Annual report

Australia's notifiable disease status, 2012: Annual report of the National Notifiable Diseases Surveillance System

NNDSS Annual Report Writing Group

Abstract

In 2012, 65 diseases and conditions were nationally notifiable in Australia. States and territories reported a total of 243,872 notifications of communicable diseases to the National Notifiable Diseases Surveillance System, an increase of 2% on the number of notifications in 2011. In 2012, the most frequently notified diseases were sexually transmissible infections (99,250 notifications, 40.7% of total notifications), vaccine preventable diseases (85,810 notifications, 35.2% of total notifications), and gastrointestinal diseases (31,155 notifications, 12.8% of total notifications). There were 16,846 notifications of bloodborne diseases; 8,305 notifications of vectorborne diseases; 1,924 notifications of other bacterial infections; 578 notifications of zoonoses; and 5 notifications of quarantinable diseases. Commun Dis Intell 2015;39(1):E46-E136.

Keywords: Australia, communicable diseases, epidemiology, surveillance

Introduction

Australia's notifiable diseases status, 2012, 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 multijurisdictional outbreaks;
- describing 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.

Information on communicable diseases surveillance is communicated through several means. 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 online from the <u>Department of Health</u> <u>web site</u> (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.

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 2012 states and territories forwarded de-identified notification 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 electronically updated daily from states and territories. The system was complemented by other surveillance systems, which provided information on various diseases, including four that are not reported through the National Notifiable Diseases Surveillance System (NNDSS): HIV, AIDS and the classical and variant forms of Creutzfeldt-Jakob disease (CJD).

Newly diagnosed HIV infection and AIDS were notifiable conditions in all states and territories in 2012. 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

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

In 2012, 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 pathology service authorised a report or a 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 agents detected, or isolated, and on the vaccination status of the case were collected and reported to NNDSS. Data quality was monitored by the Office of Health Protection and the National Surveillance Committee (NSC). There was a continual process of improving the national consistency of communicable disease surveillance through the daily, fortnightly and quarterly reviews 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, donovanosis, gonococcal infection and syphilis < 2 years duration) were reported from states and territories to NNDSS. With the exception of hepatitis B and hepatitis C these enhanced data are 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 report.

Notification rates for each disease were calculated using the estimated 2012 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 population unless stated otherwise.

Direct age standardised notification rates, using the method described by the Australian Institute of Health and Welfare⁷ were calculated for Aboriginal and Torres Strait Islander and non-Indigenous notifications for relevant sexually transmissible infections (STIs) for jurisdictions that had Indigenous status data completed for more than 50% of notifications over the period from 2007 to 2012. Where the Indigenous status of a notification was not completed, these notifications were counted as non-Indigenous in the analyses. These data, however, should be interpreted with caution, as STI screening may occur predominately in specific high risk groups, including in remote Aboriginal and Torres Strait Islander populations. Recent studies have suggested that higher rates in Aboriginal and Torres Strait Islander populations may be attributable to higher prevalence and reinfection rates while others have suggested that it may be due to increased testing and contract tracing.8

In the national case definitions for chlamydial infection, gonococcal infection and syphilis, the mode of transmission cannot be inferred from the site of infection. Infections in children may be acquired perinatally (e.g. congenital chlamydia).⁹ Notifications of chlamydial, gonococcal and non-congenital syphilis infections were excluded from analysis of age and sex distribution where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired through enhanced surveillance data, where available.

Notes on interpretation

This report is based on 2012 data from each state and territory, agreed upon in August 2013. It represents a snapshot of the data for 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 are based on the date of diagnosis in an attempt to estimate disease activity within the reporting period, with the exception of syphilis in Queensland, which is reported by the notification received date. The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the following dates: specimen collection date, the notification date, or the notification receive date. In January 2014, the NSC redefined the diagnosis date methodology for hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (unspecified) and tuberculosis. As a considerable amount of time can elapse between the initial infection, the onset of symptoms and the subsequent diagnosis, the diagnosis date for these 5 diseases is derived from the notification receive date. This new definition was applied retrospectively to all notifications of hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (unspecified) and tuberculosis.

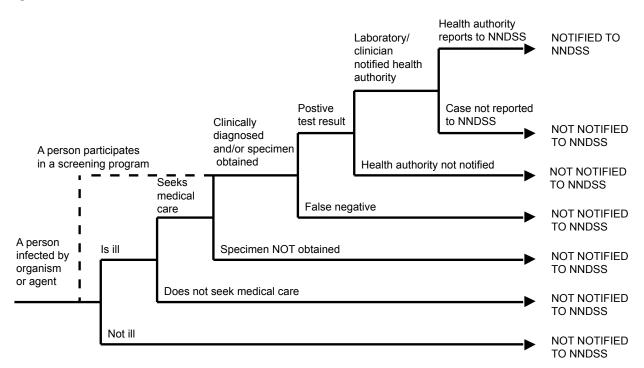
When referring to NNDSS notification data throughout this report, the terms 'cases', 'notified cases' and 'notifications' should be considered interchangeable. Notifications only represent a proportion (the 'notified fraction') of the total incidence of any disease (Figure 1). This needs to be taken into account when interpreting NNDSS data. Moreover, the notified fraction varies by disease, jurisdiction and over time.

A survey of state and territory public health departments was conducted in 2013 to ascertain the source of each notification (Table 1). Whilst most jurisdictions collect laboratory notification data, the percentage of notifications attributed to doctor only and laboratory and doctor for each state and territory is based on estimates deduced from the data that are available. Only Western Australia and New South Wales maintain data on the source of notifications as being from laboratories and/or doctors.

Methods of surveillance vary between states and territories, each having different requirements for notification by medical practitioners, laboratories and hospitals. Some diseases are not notifiable in all 8 jurisdictions (Table 2).

Over time, changes in surveillance practices may have been made in some states and territories but not others. This must be taken into consideration when comparing data between jurisdictions. In this report, some additional information was obtained from states and territories, to assist in the interpretation of the 2012 data. These include changes in surveillance, screening and laboratory practices, and disease control and prevention initiatives.

Figure 1: Communicable diseases notifiable fraction



		Source of notifications	
State or territory	Laboratory only	Doctor only	Laboratory and doctor
ACT	95.0	<1	~4.0
NSW	98.3	0.3	0.4
NT	98.0	0.7	1.3
Qld	99.5	0.2	0.2
SA	5.0	2.2	92.8
Tas.	98.0	<1.0	1.0
Vic.	38.0	5.0	52.0
WA	33.5	1.4	65.3

Table 1: Percentage* of notifications from different sources, 2012, by state or territory

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

Table 2: Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2012

Disease	Data received from
Bloodborne diseases	
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions, except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions, except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
STEC, VTEC*	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infections	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years or unknown duration	All jurisdictions
Syphilis – congenital	All jurisdictions

Table 2 continued: Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2012

Disease	Data received from
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Rubella – congenital	All jurisdictions
Tetanus	All jurisdictions
Varicella zoster (chickenpox)	All jurisdictions, except New South Wales
Varicella zoster (shingles)	All jurisdictions, except New South Wales
Varicella zoster (unspecified)	All jurisdictions, except New South Wales
Vectorborne diseases	
Arbovirus infection (NEC)	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal disease (invasive)	All jurisdictions

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

NEC Not elsewhere classified.

Postcode information usually reflects the place of residence of the case. However, it does not necessarily represent the place where the infection was acquired. Data completeness was assessed for the sex, age at onset, and Indigenous status fields, 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

Indigenous status was defined by the following nationally accepted criteria:¹⁰

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

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

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

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

9=Not stated

In interpreting STI notification 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,^{11,12} the use of less invasive and more sensitive diagnostic tests¹³ and periodic public awareness campaigns¹⁴ 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.¹⁵ For some diseases, changes in surveillance practices may also need to be taken into account when interpreting national trends.

The differences in rates between females and males for STIs should be interpreted with caution, as rates of testing, symptom status, health care-seeking behaviours, and partner notification differ between the sexes.¹⁶

Notes on case definitions

Each notifiable disease is reported to the NNDSS in accordance with the national surveillance case definition. These were agreed by CDNA and implemented nationally in January 2004 and were used by all jurisdictions for the first time in 2005. They are reviewed by the Case Definitions Working Group^{*} as required.

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

Results

There were 243,872 notifications received by NNDSS in 2012.

In 2012, the most frequently notified diseases were sexually transmissible infections (99,250 notifications, 40.7% of total notifications), vaccine preventable diseases (85,810 notifications, 35.2% of total notifications), and gastrointestinal diseases (31,155 notifications, 12.8% of total notifications) (Table 3).

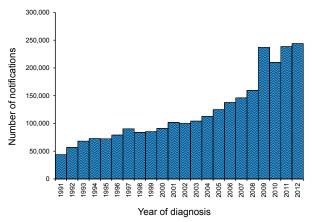
Table 3: Notifications to the National Notifiable Diseases Surveillance System, Australia, 2012, by disease category rank order

Disease Category	Number	%
Sexually transmissible infections	99,250	40.7
Vaccine preventable diseases	85,810	35.2
Gastrointestinal diseases	31,155	12.8
Bloodborne diseases	16,846	6.9
Vectorborne diseases	8,305	3.4
Other bacterial diseases	1,924	0.8
Zoonoses	578	0.2
Quarantinable diseases	5	<0.1
Total	243,872	100.0

There was an overall increase of 2% in notifications nationally compared with the total number of notifications in 2011 (Figure 2).

Notifications and notification rates per 100,000 for each disease notified to each state and territory in 2012, are shown in Table 4 and Table 5, respectively. Notifications and rates per 100,000 for the period 2007 to 2012 are shown in Table 6.

Figure 2: Trends in notifications received by the National Notifiable Diseases Surveillance System, Australia, 1991 to 2012



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

Table 4: Notifications of nationally notifiable communicable diseases, Australia, 2012, by state or territory

Disease ACT NSW NT Old SA Tas. Vic. WA Aut Bloodborne diseases Hepattis (NEC) 0					State or	terri <u>torv</u>				
Hepatitis (NEC) 0	Disease	АСТ	NSW	NT			Tas.	Vic.	WA	Aust.
Hepatitis B (newly acquired)* 2 29 5 55 16 10 52 24 Hepatitis C (newly acquired)* 15 47 0 NN 77 23 179 125 Hepatitis C (newly acquired)* 132 3,243 195 2,376 394 242 2,055 1,011 9, Hepatitis C (newly acquired)* 132 3,243 195 2,376 394 242 2,055 1,011 9, Hepatitis C (newly acquired)* 132 3,243 195 2,76 394 242 2,055 1,011 9, Hepatitis A 1 77 NN 175 4,182 2,161 88 5,870 1,906 15, Hepatitis A 1 42 3 34 7 2 62 14 14 Hepatitis A 1 42 3 4 15 3 1 35 116 11 15 14 15 14 <	Bloodborne diseases									
Hepatitis B (unspecified)* 104 2.298 200 808 383 62 1,855 799 6, Hepatitis C (newly acquired)* 15 47 0 NN 77 23 179 125 - Hepatitis D 0 5 0 6 8 0 9 2 2 Gastrointestinal diseases - - 6 8 0 0 0 0 0 0 0 0 0 15, Campvlobacteriosis 477 NN 175 4,182 2,161 882 5,870 1,906 15, Campvlobacteriosis 19 687 234 1,371 162 42 460 18 3, Hepatitis A 1 142 3 34 7 2 62 14 16 3 34 8 3 Batemolytic uraemic syndrome 0 39 0 5 4 3 3 <td< td=""><td>Hepatitis (NEC)</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td></td<>	Hepatitis (NEC)	0	0	0	0	0	0	0	0	0
Hepatitis C (newly acquired)* 15 47 0 NN 77 23 179 125 Hepatitis C (unspecified) ^{1,1} 132 3,243 195 2,376 394 2,055 1,011 9 Gastrointestinal diseases 0 5 0 6 8 0 9 2 Gatrointestinal diseases 0	Hepatitis B (newly acquired)*	2	29	5	55	16	10	52	24	193
Hepatitis C (unspecified):+ 132 3.243 195 2.376 394 242 2.055 1.011 9, Hepatitis D 0 5 0 6 8 0 9 2 Gastrointestinal diseases U 0	Hepatitis B (unspecified) [†]	104	2,298	200	808	383	62	1,855	799	6,509
Hepatitis D 0 5 0 6 8 0 9 2 Gastrointestinal diseases Botulism 0	Hepatitis C (newly acquired)*		47	0	NN	77	23	179	125	466
Gastrointestinal diseases 0 1 <td>Hepatitis C (unspecified)^{†,‡}</td> <td>132</td> <td>3,243</td> <td>195</td> <td>2,376</td> <td>394</td> <td>242</td> <td>2,055</td> <td>1,011</td> <td>9,648</td>	Hepatitis C (unspecified) ^{†,‡}	132	3,243	195	2,376	394	242	2,055	1,011	9,648
Botulism 0<	Hepatitis D	0	5	0	6	8	0	9	2	30
Campylobacteriosis 477 NN 175 4,182 2,161 882 5,870 1,906 15, Cryptosporidiosis Haemolytic uraemic syndrome 0 10 0 4 0 1 5 0 Hepatitis A 1 42 3 34 7 2 62 14 Hepatitis E 1 10 0 6 0 0 17 1 Listeriosis 0 39 0 5 4 3 34 8 Salmonellosis 241 2,951 407 2,825 845 280 2,547 1,169 11, Shigellosis 6 124 107 82 48 7 120 53 STEC,VTEC [§] 5 13 2 27 45 7 11 1 Typhoid fever 0 0 0 0 0 0 0 0 0 Plague 0 0 0<	Gastrointestinal diseases									
Cryptosporidiosis 19 687 234 1,371 162 42 460 168 3, 4aemolytic uraemic syndrome 0 10 0 4 0 1 5 00 Hepatitis A 1 42 3 34 7 2 62 14 Hepatitis E 1 0.0 39 0.5 4 3 34 88 Salmonellosis 241 2,951 407 2,825 845 280 2,547 1,169 11 Shigellosis 6 124 107 82 48 7 120 53 STEC,VTEC ⁵ 5 13 2 27 445 7 11 11 Typhoid fever 1 43 4 15 3 1 38 18 Cholara 0 2 0 1 2 0 0 0 Hagatifishe patient extensinatinfluenza in humans 0 0 0	Botulism	0	0	0	0	0	0	0	0	0
Haemolytic uraemic syndrome 0 10 0 4 0 1 5 0 Hepatitis A 1 42 3 34 7 2 62 14 Hepatitis E 1 10 0 6 0 17 1 Listeriosis 0 39 0 5 4 3 34 48 11.05 Salmonellosis 241 2,951 407 2,825 845 280 2,547 1,169 11. Shigellosis 6 124 107 82 48 7 120 53 STEC,VTEC [§] 5 13 2 27 45 7 11 1 Typhoid fever 0 2 0 1 2 0 0 0 0 Cholera 0 2 0 1 2 0 0 0 0 0 0 0 0 0 0 0	Campylobacteriosis	477	NN	175	4,182	2,161	882	5,870	1,906	15,653
Hepatitis A 1 42 3 34 7 2 62 14 Hepatitis E 1 10 0 6 0 0 17 1 Listeriosis 0 39 0 5 4 3 34 8 Salmonellosis 241 2,951 407 2,825 845 280 2,547 1,169 11 Shigellosis 6 124 107 82 48 7 11 1 Typhoid fever 1 43 4 15 3 1 38 18 Cuarantinable diseases 0 2 0 1 2 0	Cryptosporidiosis	19	687	234	1,371	162	42	460	168	3,143
Hepatitis E 1 10 0 6 0 17 1 Listeriosis 0 39 0 5 4 33 34 8 Salmonellosis 241 2,951 407 2,825 845 280 2,547 1,169 11 Shigellosis 6 124 107 82 48 7 10 53 STEC,VTEC ⁵ 13 2 27 45 7 11 1 Typhoid fever 1 43 4 15 3 1 38 18 Curantinable diseases 0 2 0 1 2 0 0 0 Highly pathogenic avian influenza in humans 0	Haemolytic uraemic syndrome	0	10	0	4	0	1	5	0	20
Listeriosis 0 39 0 5 4 3 34 8 Salmonellosis 241 2,951 407 2,825 845 280 2,547 1,169 11, Shigellosis 6 124 107 82 48 7 120 53 STEC,VTEC [§] 5 13 2 27 45 7 11 1 Typhoid fever 1 43 4 15 3 1 38 1 Cholera 0 2 0 1 2 0 0 0 Plague 0 0 0 0 0 0 0 0 0 Rabies 0	Hepatitis A	1	42	3	34	7	2	62	14	165
Salmonellosis 241 2,951 407 2,825 845 280 2,547 1,169 11, Shigellosis STEC,VTEC ⁵ 5 13 2 27 45 7 11 1 Typhoid fever 1 43 4 15 3 1 38 18 Quarantinable diseases Cholera 0 2 0 1 2 0 0 0 Highly pathogenic avian influenza in humans 0	Hepatitis E	1	10	0	6	0	0	17	1	35
Shigellosis 6 124 107 82 48 7 120 53 STEC,VTEC [§] 5 13 2 27 45 7 11 1 Typhoid fever 1 43 4 15 3 1 38 18 Quarantinable diseases 0 2 0 1 2 0 0 0 0 Highly pathogenic avian influenza in humans 0 2 0 1 2 0	Listeriosis	0	39	0	5	4	3	34	8	93
STEC,VTEC ³ 5 13 2 27 45 7 11 1 Typhoid fever 1 43 4 15 3 1 38 18 Quarantinable diseases 0 2 0 1 2 0 0 0 Highly pathogenic avian influenza in humans 0 2 0 1 2 0 0 0 Plague 0 <td>Salmonellosis</td> <td>241</td> <td>2,951</td> <td>407</td> <td>2,825</td> <td>845</td> <td>280</td> <td>2,547</td> <td>1,169</td> <td>11,265</td>	Salmonellosis	241	2,951	407	2,825	845	280	2,547	1,169	11,265
Typhoid fever 1 43 4 15 3 1 38 18 Quarantinable diseases 0 2 0 1 2 0 0 0 Cholera 0 2 0 1 2 0 0 0 Highly pathogenic avian influenza in humans 0	Shigellosis	6	124	107	82	48	7	120	53	547
Quarantinable diseases 0 2 0 1 2 0 0 0 Highly pathogenic avian influenza in humans 0 </td <td>STEC,VTEC§</td> <td>5</td> <td>13</td> <td>2</td> <td>27</td> <td>45</td> <td>7</td> <td>11</td> <td>1</td> <td>111</td>	STEC,VTEC§	5	13	2	27	45	7	11	1	111
Cholera 0 2 0 1 2 0 0 0 Highly pathogenic avian influenza in humans 0 <td< td=""><td>Typhoid fever</td><td>1</td><td>43</td><td>4</td><td>15</td><td>3</td><td>1</td><td>38</td><td>18</td><td>123</td></td<>	Typhoid fever	1	43	4	15	3	1	38	18	123
Highly pathogenic avian influenza in humans 0 <td>Quarantinable diseases</td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td>	Quarantinable diseases									
humans Image Image <t< td=""><td>Cholera</td><td>0</td><td>2</td><td>0</td><td>1</td><td>2</td><td>0</td><td>0</td><td>0</td><td>5</td></t<>	Cholera	0	2	0	1	2	0	0	0	5
Rabies 0 <td></td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td>		0	0	0	0	0	0	0	0	0
Rabies 0 <td>Plague</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td>	Plague	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 0 Yellow fever 0 <t< td=""><td>-</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td></t<>	-	0	0	0	0	0	0	0	0	0
Smallpox 0 0 0 0 0 0 0 0 0 Viral haemorrhagic fever 0	Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever 0 11,803 82, Donovanosis 0		0	0	0	0	0	0	0	0	0
Yellow fever 0 <t< td=""><td>-</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td><td>0</td></t<>	-	0	0	0	0	0	0	0	0	0
Chlamydial infection 1,283 21,293 2,532 18,849 4,848 1,787 20,312 11,803 82, Donovanosis 0 0 0 0 0 0 0 0 1,283 21,293 2,532 18,849 4,848 1,787 20,312 11,803 82, Donovanosis 0 0 0 0 0 0 0 1 13, Gonococcal infection 92 4,129 1,536 2,700 499 35 2,543 2,115 13, Syphilis – congenital 0 1, 2, 5, 5, 14 4, 4, 4, 4, 4, 1, 1, 1, 1, 1, 1, 1, 1, <td>Yellow fever</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td>	Yellow fever	0	0	0	0	0	0	0	0	0
Donovanosis 0 0 0 0 0 0 0 0 1 Gonococcal infection [¶] 92 4,129 1,536 2,700 499 35 2,543 2,115 13, Syphilis - congenital [¶] 0 13 28 52 14 474 77 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, 1, <t< td=""><td>Sexually transmissible infections</td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td></t<>	Sexually transmissible infections									
Gonococcal infection [¶] 92 4,129 1,536 2,700 499 35 2,543 2,115 13, Syphilis – congenital [¶] 0 1,	Chlamydial infection ^{II,1}	1,283	21,293	2,532	18,849	4,848	1,787	20,312	11,803	82,707
Syphilis - congenital000000000Syphilis - all $1**$. ^{††} 2879381639131249802172,Syphilis < 2 years duration	Donovanosis	0	0	0	0	0	0	0	1	1
Syphilis - all ^{1,*,t††} 2879381639131249802172,Syphilis < 2 years duration ^{1,††} 15510143835214474771,Syphilis > 2 years or unknown duration ^{1,1,††} 132836725679105061401,Vaccine preventable diseasesDiphtheria00000000Haemophilus influenzae type b02052141	Gonococcal infection [®]	92	4,129	1,536	2,700	499	35	2,543	2,115	13,649
Syphilis < 2 years duration ^{11,++} 15 510 14 383 52 14 474 77 1, Syphilis > 2 years or unknown duration ^{11,+++} 13 283 67 256 79 10 506 140 1, Vaccine preventable diseases 0 0 0 0 0 0 0 0 0 0 Haemophilus influenzae type b 0 2 0 5 2 1 4 1	Syphilis – congenital [¶]	0	0	0	0	0	0	0	0	0
Syphilis > 2 years or unknown duration ^{1,¶,1†} 13 283 67 256 79 10 506 140 1, Vaccine preventable diseases 0	Syphilis – all ^{¶,**,††}	28	793	81	639	131	24	980	217	2,893
duration ^{1,1,1+1} Vaccine preventable diseases Diphtheria 0 0 0 0 0 0 Haemophilus influenzae type b 0 2 0 5 2 1 4 1	Syphilis < 2 years duration ^{1,††}	15	510	14	383	52	14	474	77	1,539
Vaccine preventable diseases Diphtheria 0	Syphilis > 2 years or unknown	13	283	67	256	79	10	506	140	1,354
Diphtheria 0	duration ^{†,¶,††}									
Haemophilus influenzae type b 0 2 0 5 2 1 4 1	Vaccine preventable diseases									
	-	0	0	0	0	0	0	0	0	0
Influenza (laboratory confirmed) 666 7,998 435 16,853 6,288 1,093 5,990 5,240 44,	Haemophilus influenzae type b	0	2	0	5		1	4	1	15
	Influenza (laboratory confirmed)	666		435	16,853	6,288	1,093	5,990	5,240	44,563
Measles 0 170 2 4 6 0 11 6	Measles	0	170	2	4	6	0	11	6	199
Mumps 6 105 0 32 7 1 30 19	Mumps	6	105	0	32	7	1	30	19	200
Pertussis 429 5,828 298 7,536 904 1,276 4,423 3,375 24,	Pertussis	429	5,828	298	7,536	904	1,276	4,423	3,375	24,069
Pneumococcal disease (invasive) 27 579 72 348 131 45 385 235 1,	Pneumococcal disease (invasive)	27	579	72	348	131	45	385	235	1,822
Poliomyelitis 0 0 0 0 0 0 0 0	Poliomyelitis	0	0	0	0	0	0	0	0	0
Rubella 1 10 0 9 2 1 11 2	Rubella	1	10	0	9	2	1	11	2	36
Rubella – congenital 0 0 1 0	Rubella – congenital	0	0	1	0	0	0	0	0	1
Tetanus 0 1 0 2 1 0 2 1	Tetanus	0	1	0	2	1	0	2	1	7
Varicella zoster (chickenpox) 9 NN 149 238 476 27 732 333 1,	Varicella zoster (chickenpox)	9	NN	149	238	476	27	732	333	1,964
Varicella zoster (shingles) 51 NN 183 72 1,761 263 1,111 1,040 4,	Varicella zoster (shingles)	51	NN	183	72	1,761	263	1,111	1,040	4,481
Varicella zoster (unspecified) 121 NN 4 4,414 92 84 2,633 1,105 8,	Varicella zoster (unspecified)	121	NN	4	4,414	92	84	2,633	1,105	8,453

				State or	territory				
Disease	АСТ	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Vectorborne diseases									
Arbovirus infection (NEC)	0	0	0	8	1	0	0	0	9
Barmah Forest virus infection	2	348	87	982	48	0	40	215	1,722
Dengue virus infection	22	288	68	243	51	8	332	528	1,540
Japanese encephalitis virus infection	0	0	0	1	0	0	0	0	1
Kunjin virus infection ^{‡‡}	0	0	0	0	0	0	0	0	0
Malaria	11	71	17	100	8	7	84	50	348
Murray Valley encephalitis virus infection	0	0	0	1	0	0	0	0	1
Ross River virus infection	11	603	227	1,947	219	18	282	1,376	4,683
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	5	0	22	1	0	0	1	29
Leptospirosis	0	22	1	75	2	0	13	3	116
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0
Ornithosis	0	18	0	1	1	0	47	8	75
Q fever	0	122	4	192	11	0	22	7	358
Tularaemia	0	0	0	0	0	0	0	0	0
Other bacterial diseases									
Legionellosis	2	102	3	70	39	12	69	85	382
Leprosy	0	0	0	0	0	0	4	0	4
Meningococcal infection§§	1	66	4	63	29	7	35	18	223
Tuberculosis	18	467	28	176	82	6	366	172	1,315
Total	3,784	52,564	7,064	67,328	19,795	6,257	53,775	33,255	243,872

Table 4 *continued:* Notifications of nationally notifiable communicable diseases, Australia, 2012, by state or territory

* Newly acquired hepatitis and syphilis 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.

t Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.

‡ In Queensland, includes newly acquired hepatitis C cases.

§ Infection with Shiga toxin/verotoxin producing Escherichia coli.

|| Includes 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.

+ Data for all states and territories are reported by diagnosis date, except Queensland which is reported by notification receive date.

1 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 the Australian Capital Territory also reports conjunctival cases.

NEC Not elsewhere classified.

NN Not notifiable.

Table 5: Notification rates for nationally notifiable communicable diseases, Australia, 2012, by state or territory

Disease ACT NSW NT Qld SA Tas. Vic. WA Ausd Biodborne diseases				S	tate or t	territory				
Hepatitis (NEC) - 0	Disease	ACT	NSW					Vic.	WA	Aust.
Hepatitis B (newly acquired)* 0.5 0.4 2.1 1.2 1.0 2.0 0.9 1.0 0.8 Hepatitis B (unspecified)* 27.7 31.5 85.0 17.7 23.1 12.1 32.0 32.8 28.7 Hepatitis C (unspecified)*+ 4.0 0.6 - NN 4.6 4.5 3.2 6.4 42.5 Hepatitis C (unspecified)*+ - 0.1 0.1 - 0.1 5.7 0.2 0.1 1.7 Bottistis C (unspecified)*+ - - 0.1 - 0.1 - 0.2 0.1 1.1 Gatrointestinal diseases - - - - - - - 0.2 0.1 - 0.1 - 0.2 0.1 - 0.1 - 0.2 0.1 - 0.1 - 0.2 0.1 0.1 - - 0.3 0.1 - - 0.1 - - 0.1 0.1 -<	Bloodborne diseases									
Hepatitis B (unspecified)? 27.7 31.5 85.0 17.7 23.1 12.1 33.0 32.8 28.7 Hepatitis C (newly acquired)* 4.0 0.6 - NN 4.6 4.5 3.2 5.1 2.6 Hepatitis C (unspecified) ¹¹ 35.2 44.4 82.9 52.0 23.8 47.2 36.5 41.6 42.5 Hepatitis C (unspecified) ¹¹⁴ 36.2 44.4 82.9 52.0 23.8 47.2 36.5 41.6 42.5 Gastrointestinal diseases - - - - - - - - - - - - 10.6 10.6 10.6 10.8 10.6 17.7 9.3 9.0 9.8 8.2 8.2 6.9 38.8 Hepatitis C 0.3 0.1 - 0.1 -0.7 0.4 0.1 0.2 0.0 10.2 10.6 7.4 8.2 8.4 40.6 0.5 0.1 0.2 0.	Hepatitis (NEC)	-	-	-	-	-	-	-	-	-
Hepatitis C (newly acquired)* 4.0 0.0 - NN 4.6 4.5 3.2 5.1 2.6 Hepatitis C (unspecified) ^{1,4} - 0.1 - 0.1 0.5 - 0.2 0.1 0.1 0.5 - 0.2 0.1 0.1 Gattritis C (unspecified) ^{1,4} - 0.1 - 0.1 0.5 - 0.2 0.1 0.1 Gattritis C (unspecified) ^{1,4} - 0.1 - - - 0.1 - 0.2 0.1 0.7 Camylobacteriosis 10.1 - 0.2 0.1 - 0.1 - 0.2 0.1 - 0.1 - 0.2 0.1 - 0.1 - 0.3 0.1 - 0.3 0.1 - 0.3 0.1 - 0.3 0.1 - 0.3 0.1 - 0.3 0.1 - 0.3 0.1 - 0.3 0.1 0.1 0.1 - 0.1	Hepatitis B (newly acquired)*	0.5	0.4	2.1	1.2	1.0	2.0	0.9	1.0	0.8
Hepatitis C (unspecified) ¹⁺¹ 35.2 44.4 82.9 52.0 23.8 47.2 36.5 41.6 42.5 Hepatitis D - 0.1 - 0.1 0.5 - 0.2 0.1 0.1 Gattrintestinal diseases - - 0.1 - 0.1 0.5 7.2 0.13 0.1 - 0.2 0.1 0.1 Campylobacteriosis 127.2 NN 74.4 91.6 10.0 9.8 8.2 6.3 13.8 Haemolytic uraemic syndrome - 0.1 - 0.1 - 0.2 0.1 - 0.1 Hepatitis E 0.3 0.1 - 0.1 0.2 0.1 0.2 0.1 0.2 0.1 0.2 0.1 0.2 0.1 0.2 0.2 0.1 0.2 0.2 0.1 0.2 0.2 0.2 0.1 0.2 0.2 0.1 0.2 0.2 0.2 0.2 0.2 0.2 <td>Hepatitis B (unspecified)[†]</td> <td>27.7</td> <td>31.5</td> <td>85.0</td> <td>17.7</td> <td>23.1</td> <td>12.1</td> <td>33.0</td> <td>32.8</td> <td>28.7</td>	Hepatitis B (unspecified) [†]	27.7	31.5	85.0	17.7	23.1	12.1	33.0	32.8	28.7
Hepatitis D0.10.10.5-0.20.10.1Gatrointestinal diseasesBotulism <t< td=""><td>Hepatitis C (newly acquired)*</td><td>4.0</td><td>0.6</td><td>-</td><td>NN</td><td>4.6</td><td>4.5</td><td>3.2</td><td>5.1</td><td>2.6</td></t<>	Hepatitis C (newly acquired)*	4.0	0.6	-	NN	4.6	4.5	3.2	5.1	2.6
Gastrointestinal diseases Image: Constraint of the synthesis of the	Hepatitis C (unspecified) ^{†,‡}	35.2	44.4	82.9	52.0	23.8	47.2	36.5	41.6	42.5
Botulism -<	Hepatitis D	-	0.1	_	0.1	0.5	-	0.2	0.1	0.1
Campylobacteriosis 127.2 NN 74.4 91.6 130.5 172.2 104.3 78.3 11.6 Cryptosporidiosis 5.1 9.4 99.5 30.0 9.8 8.2 8.2 6.9 13.8 Haemolytic uraemic syndrome - 0.1 - 0.2 0.1 - 0.1 Hepatitis A 0.3 0.6 1.3 0.7 0.4 0.4 1.1 0.6 0.7 Hepatitis E 0.3 0.1 - 0.1 0.2 0.6 0.6 0.3 0.4 Salmonellosis 64.3 40.4 173.1 61.9 51.0 54.7 45.2 48.1 49.6 Shigellosis 1.6 1.7 45.5 1.8 2.9 1.4 2.2 24.4 Stepore Sattististististististististististististis	Gastrointestinal diseases									
Cryptosporidiosis 5.1 9.4 99.5 30.0 9.8 8.2 8.2 6.9 1.3.8 Haemolytic uraemic syndrome - 0.1 - 0.1 - 0.2 0.1 - 0.1 Hepatitis A 0.3 0.6 1.3 0.7 0.4 0.4 1.1 0.6 0.7 Hepatitis E 0.3 0.1 - 0.1 - - 0.3 0.1 2 0.6 0.6 0.7 Hepatitis E 0.3 0.1 - 0.1 0.2 0.6 0.6 0.3 0.4 Salmonellosis 64.3 40.4 173.1 61.9 51.0 54.7 45.2 48.1 49.6 Shigellosis 1.6 1.7 45.5 1.8 2.9 1.4 2.1 2.2 2.4 Stepore active assignmentions 1.6 1.7 45.5 1.8 2.9 1.4 2.1 2.2 2.4 Highly pathogenic avian	Botulism	-	-	_	_	-	-	-	-	-
Haemolytic uraemic syndrome - 0.1 - 0.1 - 0.1 - 0.2 0.1 - 0.1 Hepatitis A 0.3 0.6 1.3 0.7 0.4 0.4 1.1 0.6 0.7 Hepatitis E 0.3 0.1 - 0.1 - - 0.3 0.1 0.2 0.6 0.3 0.4 Listeriosis - 0.5 - 0.1 0.2 0.6 0.3 0.4 Salmonellosis - 0.5 - 0.1 0.2 0.6 0.6 0.3 0.4 Shigellosis 1.6 1.7 45.5 1.8 2.9 1.4 2.1 2.2 2.4 STEC,VTEC [§] 1.3 0.2 0.9 0.6 1.7 1.4 0.2 0.0 0.5 Typhoid 0.3 0.6 1.7 0.3 0.2 0.2 0.1 0.5 Typhoid 1.3 0.2 0.1 </td <td>Campylobacteriosis</td> <td>127.2</td> <td>NN</td> <td>74.4</td> <td>91.6</td> <td>130.5</td> <td>172.2</td> <td>104.3</td> <td>78.3</td> <td>101.6</td>	Campylobacteriosis	127.2	NN	74.4	91.6	130.5	172.2	104.3	78.3	101.6
Hepatitis A0.30.61.30.70.40.41.10.60.7Hepatitis E0.30.1-0.10.30.10.2Listeriosis-0.5-0.10.20.60.60.30.4Salmonellosis-0.540.4173.161.951.054.745.248.149.6Shigellosis1.61.745.51.82.91.42.12.22.4STEC,VTEC [§] 1.30.20.90.62.71.40.20.50.5Typhoid0.30.61.70.30.20.20.70.50.5Charantinable diseases0.11.40.20.10.5Plague0.10	Cryptosporidiosis	5.1	9.4	99.5	30.0	9.8	8.2	8.2	6.9	13.8
Hepatitis E 0.3 0.1 - 0.1 - - 0.3 0.1 Listeriosis - 0.5 - 0.1 0.2 0.6 0.3 0.4 Salmonellosis 64.3 40.4 173.1 61.9 51.0 54.7 45.2 48.1 49.6 Shigellosis 1.6 1.7 45.5 1.8 2.9 1.4 2.2 2.4 STEC,VTEC [§] 1.3 0.2 0.9 0.6 2.7 1.4 0.2 <0.1	Haemolytic uraemic syndrome	-	0.1	-	0.1	-	0.2	0.1	_	0.1
Listeriosis-0.5-0.10.20.60.60.30.4Salmonellosis64.340.4173.161.951.054.745.248.149.6Shigellosis1.61.745.51.82.91.42.22.44STEC,VTEC ⁵ 1.30.20.90.62.71.40.22.00.5Typhoid0.30.61.70.30.20.20.70.70.5Cholera0.10.10.10.1Highly pathogenic avian influenza in humans	Hepatitis A	0.3	0.6	1.3	0.7	0.4	0.4	1.1	0.6	0.7
Listeriosis-0.5-0.10.20.60.60.30.4Salmonellosis64.340.4173.161.951.054.745.248.149.6Shigellosis1.61.745.51.82.91.42.22.42.4STEC,VTEC [§] 1.30.20.90.62.71.40.22.00.5Typhoid0.30.61.70.30.20.20.70.70.5CholeraPlague <td< td=""><td>Hepatitis E</td><td>0.3</td><td>0.1</td><td>_</td><td>0.1</td><td>_</td><td>_</td><td>0.3</td><td><0.1</td><td>0.2</td></td<>	Hepatitis E	0.3	0.1	_	0.1	_	_	0.3	<0.1	0.2
Shigellosis1.61.745.51.82.91.42.12.4STEC,VTEC [§] 1.30.20.90.62.71.40.20.70.5Typhoid0.30.20.20.70.70.70.70.70.70.7Cholera1<0.1		_	0.5	_	0.1	0.2	0.6		0.3	
Shigellosis1.61.745.51.82.91.42.12.4STEC,VTEC§1.30.20.90.62.71.40.2<0.1	Salmonellosis	64.3	40.4	173.1	61.9	51.0	54.7	45.2	48.1	49.6
STEC,VTEC [§] 1.3 0.2 0.9 0.6 2.7 1.4 0.2 <0.1 Typhoid 0.3 0.6 1.7 0.3 0.2 0.2 0.7 0.7 0.5 Quarantinable diseases U U 0.1	Shigellosis	1.6	1.7	45.5	1.8	2.9	1.4	2.1	2.2	2.4
Typhoid0.30.61.70.30.20.20.70.70.5Guarantinable diseasesCholera-<	-	1.3	0.2	0.9	0.6	2.7	1.4	0.2	<0.1	0.5
Cholera-<0.1-<0.10.1<0.1Highly pathogenic avian influenza in humans		0.3	0.6	1.7	0.3	0.2	0.2	0.7	0.7	0.5
Highly pathogenic avian influenza in humans -	Quarantinable diseases									П
Plague $ -$ <t< td=""><td>Cholera</td><td>-</td><td><0.1</td><td>-</td><td><0.1</td><td>0.1</td><td>-</td><td>-</td><td>-</td><td><0.1</td></t<>	Cholera	-	<0.1	-	<0.1	0.1	-	-	-	<0.1
Rabies - <td>Highly pathogenic avian influenza in humans</td> <td>-</td> <td>-</td> <td>_</td> <td>_</td> <td>-</td> <td>-</td> <td>-</td> <td>-</td> <td>_</td>	Highly pathogenic avian influenza in humans	-	-	_	_	-	-	-	-	_
Severe acute respiratory syndrome -	Plague	-	-	-	-	-	-	-	_	-
Smallpox -<	Rabies	-	-	_	_	-	-	-	-	_
Viral haemorrhagic fever - </td <td>Severe acute respiratory syndrome</td> <td>-</td> <td>-</td> <td>-</td> <td>-</td> <td>-</td> <td>-</td> <td>_</td> <td>_</td> <td>-</td>	Severe acute respiratory syndrome	-	-	-	-	-	-	_	_	-
Yellow fever $ -$ <	Smallpox	-	-	_	_	-	-	-	-	_
Sexually transmissible infections Chlamydial infection ^{.¶} 342.2 291.6 1,076.6 412.9 292.7 348.8 360.8 485.2 364.2 Donovanosis - - - - - - - <0.1	Viral haemorrhagic fever	-	-	-	-	-	-	_	_	-
Chlamydial infection 342.2 291.6 1,076.6 412.9 292.7 348.8 360.8 485.2 364.2 Donovanosis - - - - - - - <0.1	Yellow fever	-	-	_	-	-	-	-	-	-
Donovanosis $ -$ </td <td>Sexually transmissible infections</td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td>	Sexually transmissible infections									
Gonococcal infection [¶] 24.5 56.6 653.1 59.1 30.1 6.8 45.2 86.9 60.1 Syphilis - congenital [¶] - </td <td>Chlamydial infection 11.11</td> <td>342.2</td> <td>291.6</td> <td>1,076.6</td> <td>412.9</td> <td>292.7</td> <td>348.8</td> <td>360.8</td> <td>485.2</td> <td>364.2</td>	Chlamydial infection 11.11	342.2	291.6	1,076.6	412.9	292.7	348.8	360.8	485.2	364.2
Syphilis - congenital [¶] - </td <td>Donovanosis</td> <td>-</td> <td>_</td> <td>-</td> <td>-</td> <td>-</td> <td>-</td> <td>-</td> <td><0.1</td> <td><0.1</td>	Donovanosis	-	_	-	-	-	-	-	<0.1	<0.1
Syphilis - all ^{¶,**,††} 7.5 10.9 34.4 14.0 7.9 4.7 17.4 8.9 12.7 Syphilis < 2 years duration ^{¶,††} 4.0 7.0 6.0 8.4 3.1 2.7 8.4 3.2 6.8	Gonococcal infection [¶]	24.5	56.6	653.1	59.1	30.1	6.8	45.2	86.9	60.1
Syphilis < 2 years duration ^{11,++} 4.0 7.0 6.0 8.4 3.1 2.7 8.4 3.2 6.8	Syphilis – congenital [¶]	-	-	-	-	-	-	-	-	-
	Syphilis – all ^{¶,**,††}	7.5	10.9	34.4	14.0	7.9	4.7	17.4	8.9	12.7
	Syphilis < 2 years duration ^{¶,††}	4.0	7.0	6.0	8.4	3.1	2.7	8.4	3.2	6.8
Syphilis > 2 years or unspecified duration 3.5 3.9 28.5 5.6 4.8 2.0 9.0 5.8 6.0	Syphilis > 2 years or unspecified duration ^{†,¶,††}	3.5	3.9	28.5	5.6	4.8	2.0	9.0	5.8	6.0
Vaccine preventable diseases	Vaccine preventable diseases									
Diphtheria	Diphtheria	_	_	_	_	_	_	_	_	-
Haemophilus influenzae type b - <0.1 0.1 0.2 0.1 <0.1 0.1	Haemophilus influenzae type b	-	<0.1	_	0.1	0.1	0.2	0.1	<0.1	0.1
Influenza (laboratory confirmed) 177.6 109.5 185.0 369.1 379.6 213.3 106.4 215.4 196.2	Influenza (laboratory confirmed)	177.6	109.5	185.0	369.1	379.6	213.3	106.4	215.4	196.2
Measles – 2.3 0.9 0.1 0.4 – 0.2 0.2 0.9	Measles	-	2.3	0.9	0.1	0.4	-	0.2	0.2	0.9
Mumps 1.6 1.4 - 0.7 0.4 0.2 0.5 0.8 0.9	Mumps	1.6	1.4	_	0.7	0.4	0.2	0.5	0.8	0.9
Pertussis 114.4 79.8 126.7 165.1 54.6 249.1 78.6 138.7 106.0	Pertussis	114.4	79.8	126.7	165.1	54.6	249.1	78.6	138.7	106.0
Pneumococcal disease (invasive) 7.2 7.9 30.6 7.6 7.9 8.8 6.8 9.7 8.0	Pneumococcal disease (invasive)	7.2	7.9	30.6	7.6	7.9	8.8	6.8	9.7	8.0
Poliomyelitis	Poliomyelitis	-	-	-	-	-	_	_	_	-
	Rubella	0.3	0.1	_	0.2	0.1	0.2	0.2	0.1	0.2

Table 5 continued: Notification rates for nationally notifiable communicable diseases, Australia,2012, by state or territory

			5	State or t	territory				
Disease	АСТ	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Rubella – congenital	-	_	0.4	_	-	-	-	-	<0.1
Tetanus	-	<0.1	-	<0.1	0.1	-	<0.1	<0.1	<0.1
Varicella zoster (chickenpox)	2.4	NN	63.4	5.2	28.7	5.3	13.0	13.7	12.7
Varicella zoster (shingles)	13.6	NN	77.8	1.6	106.3	51.3	19.7	42.8	29.1
Varicella zoster (unspecified)	32.3	NN	1.7	96.7	5.6	16.4	46.8	45.4	54.9
Vectorborne diseases									
Arbovirus infection (NEC)	-	-	-	0.2	0.1	-	-	-	<0.1
Barmah Forest virus infection	0.5	4.8	37.0	21.5	2.9	-	0.7	8.8	7.6
Dengue virus infection	5.9	4.0	28.9	5.3	3.1	1.6	5.9	21.7	6.8
Japanese encephalitis virus infection	-	-	-	<0.1	-	-	-	-	<0.1
Kunjin virus infection ^{‡‡}	-	-	-	-	-	-	-	-	-
Malaria	2.9	1.0	7.2	2.2	0.5	1.4	1.5	2.1	1.5
Murray Valley encephalitis virus infection	-	-	-	<0.1	-	-	-	-	<0.1
Ross River virus infection	2.9	8.3	96.5	42.6	13.2	3.5	5.0	56.6	20.6
Zoonoses									
Anthrax	-	-	-	-	-	-	-	-	-
Australia bat lyssavirus	-	-	-	-	-	-	-	-	-
Brucellosis	-	0.1	-	0.5	0.1	-	-	<0.1	0.1
Leptospirosis	-	0.3	0.4	1.6	0.1	-	0.2	0.1	0.5
Lyssavirus (NEC)	-	-	-	-	-	-	-	-	-
Ornithosis	-	0.2	-	<0.1	0.1	-	0.8	0.3	0.3
Q fever	-	1.7	1.7	4.2	0.7	-	0.4	0.3	1.6
Tularaemia	-	-	-	-	-	-	-	-	-
Other bacterial diseases									
Legionellosis	0.5	1.4	1.3	1.5	2.4	2.3	1.2	3.5	1.7
Leprosy	-	-	-	-	-	-	0.1	-	<0.1
Meningococcal infection ^{§§}	0.3	0.9	1.7	1.4	1.8	1.4	0.6	0.7	1.0
Tuberculosis	4.8	6.4	11.9	3.9	5.0	1.2	6.5	7.1	5.8

* Newly acquired hepatitis and syphilis 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.

- ‡ In Queensland, includes newly acquired hepatitis C 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.

1 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.

†† Data for all states and territories are reported by diagnosis date, except Queensland which is reported by notification receive date.

1 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 the Australian Capital Territory also reports conjunctival cases.

NEC Not elsewhere classified.

NN Not notifiable.

Table 6: Notifications and notification rate for communicable diseases, Australia, 2007 to 2012	ation rate	e for com	municat	ole diseas	es, Aust	ralia, 2(007 to 20	12						
		Nu	Number of no	otifications	0			Ratio		Notif	Notification rate per 100,000	te per 100	,000	
Disease	2007	2008	2009	2010	2011	2012	5-year mean	(zu1z : 5-year mean)	2007	2008	2009	2010	2011	2012
Bloodborne diseases														
Hepatitis (NEC)	0	0	0	0	0	0	0.0	I	I	I	I	I	I	I
Hepatitis B (newly acquired)*	300	262	249	228	195	193	246.8	0.8	1.4	1.2	1.1	1.0	0.9	0.8
Hepatitis B (unspecified) [†]	6,772	6,419	6,961	6,910	6,578	6,509	6,728.0	1.0	32.1	29.9	32.1	31.4	29.4	28.7
Hepatitis C (newly acquired)*	378	365	400	397	413	466	390.6	1.2	2.2	2.1	2.3	2.3	2.3	2.6
Hepatitis C (unspecified) ^{t,‡}	11,667	10,943	10,846	10,887	9,832	9,648	10,835.0	0.9	55.4	50.9	50.0	49.4	44.0	42.5
Hepatitis D	33	41	35	36	38	30	36.6	0.8	0.2	0.2	0.2	0.2	0.2	0.1
Gastrointestinal diseases	_					-	-							
Botulism	-	0	-	0	7	0	0.8	0.0	<0.1	I	<0.1	I	<0.1	I
Campylobacteriosis	16,989	15,548	16,098	16,986	17,725	15,653	16,669.2	0.9	119.9	107.3	110.0	114.1	117.2	101.6
Cryptosporidiosis	2,808	2,001	4,624	1,479	1,810	3,143	2,544.4	1.2	13.3	9.3	21.3	6.7	8.1	13.8
Haemolytic uraemic syndrome	19	32	13	6	13	20	17.2	1.2	0.1	0.1	0.1	<0.1	0.1	0.1
Hepatitis A	166	276	564	266	145	165	283.4	0.6	0.8	1.3	2.6	1.2	0.6	0.7
Hepatitis E	18	44	33	37	41	35	34.6	1.0	0.1	0.2	0.2	0.2	0.2	0.2
Listeriosis	50	68	92	71	70	93	70.2	1.3	0.2	0.3	0.4	0.3	0.3	0.4
Salmonellosis	9,465	8,286	9,506	11,922	12,270	11,265	10,289.8	1.1	44.9	38.5	43.8	54.1	54.9	49.6
Shigellosis	596	828	616	551	494	547	617.0	0.9	2.8	3.9	2.8	2.5	2.2	2.4
STEC,VTEC [§]	105	98	128	80	95	111	101.2	1.1	0.5	0.5	0.6	0.4	0.4	0.5
Typhoid	06	105	115	96	135	123	108.2	1.1	0.4	0.5	0.5	0.4	0.6	0.5
Quarantinable diseases														
Cholera	4	4	£	ო	9	5	4.4	1.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0.0	0.0	I	I	I	I	I	I
Plague	0	0	0	0	0	0	0.0	0.0	I	I	I	I	I	I
Rabies	0	0	0	0	0	0	0.0	0.0	I	I	I	I	I	I
Severe acute respiratory syndrome	0	0	0	0	0	0	0.0	0.0	I	I	I	I	I	I
Smallpox	0	0	0	0	0	0	0.0	0.0	I	I	I	I	I	I
Viral haemorrhagic fever	0	0	0	0	0	0	0.0	0.0	I	I	I	I	I	I
Yellow fever	0	0	0	0	N	0	0.4	0.0	I	I	I	I	<0.1	I

		M	a jo soqu							Noti		101 404 404		
		N		ouncations	'n			(2012 :		NOU	Nouncation rate per juu,uuu	nu jed en	000,0	
Disease	2007	2008	2009	2010	2011	2012	5-year mean	5-year mean)	2007	2008	2009	2010	2011	2012
Sexually transmissible infections														
Chlamydial infection ^{ll,¶}	51,945	58,427	62,997	74,306	80,922	82,707	65,719.4	1.3	246.5	271.8	290.4	337.3	362.2	364.2
Donovanosis	З	2	-	-	0	-	1.4	0.7	0.014	0.009	0.005	0.005	I	0.004
Gonococcal infection [¶]	7,647	7,679	8,276	10,322	12,099	13,649	9,204.6	1.5	36.3	35.7	38.2	46.9	54.2	60.1
Syphilis – congenital [¶]	7	9	S	с	7	0	5.2	0.0	0.03	0.03	0.01	0.01	0.03	I
Syphilis – all¶**:tt	2,779	2,704	2,743	2,417	2,574	2,893	2,643.4	1.1	13.2	12.6	12.6	11.0	11.5	12.7
Syphilis < 2 years duration ^{¶,+†}	1,424	1,332	1,335	1,142	1,294	1,539	1,305.4	1.1	6.8	6.2	6.2	5.2	5.8	6.8
Syphilis > 2 years or unspecified duration ^{±⊈t†}	1,355	1,372	1,408	1,275	1,280	1,354	1,338.0	1.0	7.0	6.9	7.0	6.2	6.2	6.0
Vaccine preventable diseases														
Diphtheria	0	0	0	0	4	0	0.8	0.0	I	I	I	I	<0.1	I
Haemophilus influenzae type b	17	25	19	24	13	15	19.6	0.8	0.1	0.1	0.1	0.1	0.1	0.1
Influenza (laboratory confirmed)	10,586	9,174	59,024	13,470	27,225	44,563	23,895.8	1.9	50.2	42.7	272.1	61.1	121.9	196.2
Measles	12	65	105	69	194	199	89.0	2.2	0.1	0.3	0.5	0.3	0.9	0.9
Mumps	582	285	166	98	156	200	257.4	0.8	2.8	1.3	0.8	0.4	0.7	0.9
Pertussis	4,862	14,284	30,158	34,809	38,721	24,069	24,566.8	1.0	23.1	66.4	139.0	158.0	173.3	106.0
Pneumococcal disease (invasive)	1,469	1,628	1,556	1,642	1,884	1,822	1,635.8	1.1	7.0	7.6	7.2	7.5	8.4	8.0
Poliomyelitis	-	0	0	0	0	0	0.2	0.0	<0.1	I	I	I	I	I
Rubella	34	36	27	44	58	36	39.8	0.9	0.2	0.2	0.1	0.2	0.3	0.2
Rubella – congenital	7	0	0	0	0	~	0.4	2.5	<0.1	I	I	I	I	<0.1
Tetanus	З	4	с	2	ო	7	3.0	2.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Varicella zoster (chickenpox)	1,669	1,800	1,795	1,786	2,099	1,964			18.7	19.7	12.3	12.0	13.9	12.7
Varicella zoster (shingles)	1,565	2,337	2,780	3,044	4,023	4,481			17.5	25.5	19.0	20.4	26.6	29.1
Varicella zoster (unspecified)	4,278	4,412	6,763	7,269	7,691	8,453			47.8	48.2	46.2	48.8	50.9	54.9
Vectorborne diseases														
Arbovirus infection (NEC)	17	12	80	24	20	o	16.8	0.5	0.1	0.1	0.04	0.1	0.1	0.04
Barmah Forest virus infection	1,707	2,080	1,474	1,470	1,863	1,722	1,718.8	1.0	8.1	9.7	6.8	6.7	8.3	7.6
Dengue virus infection	314	561	1,402	1,227	817	1,540	864.2	1.8	1.5	2.6	6.5	5.6	3.7	6.8
Japanese encephalitis virus infection	0	~	0	0	0	~	0.2	5.0	I	<0.1	I	I	I	<0.1
Kunjin virus infection ^{##}	-	~	2	2	2	0	1.6	0.0	<0.1	<0.1	<0.1	<0.1	<0.1	0
Malaria	565	530	507	404	417	348	484.8	0.7	2.7	2.5	2.3	1.8	1.9	1.5
Murray Valley encephalitis virus infection	0	2	4	0	17	-	4.6	0.2	I	<0.1	<0.1	I	0.1	<0.1
Ross River virus infection	4,150	5,614	4,741	5,126	5,138	4,683	4,953.8	0.9	19.7	26.1	21.9	23.3	23.0	20.6

2012	
ia, 2007 to 2012	
Australi	
s and notification rate for communicable diseases, <i>i</i>	
Table 6 continued: Notifications ar	

Tabl	TADIC O COMMINGCU: INOUTICATIONS AND INOUTICATION FAILS FOR COMMINGUM CADIC DESCASS, 720514014, 2007 FO 2012		ICALIOII I	alc IUI C	IIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIIII	capic dist	Casco, A	usu alla,		7107					
			N	imber of n	Number of notifications	SL			Ratio (2012 :		Notif	ication ra	Notification rate per 100,000	0000	
	Disease	2007	2008	2009	2010	2011	2012	5-year mean	5-year mean)	2007	2008	2009	2010	2011	2012
Zoonoses	oses														
Anthrax	ax	~	0	0	-	0	0	0.4	0.0	<0.1	I	I	<0.1	I	I
Austra	Australia bat lyssavirus	0	0	0	0	0	0	0.0	I	I	I	I	I	I	I
Bruce	Brucellosis	37	45	32	21	39	29	34.8	0.8	0.2	0.2	0.1	0.1	0.2	0.1
Lepto	Leptospirosis	108	111	141	132	215	116	141.4	0.8	0.5	0.5	0.7	0.6	1.0	0.5
Lyssa	Lyssavirus (NEC)	0	0	0	0	0	0	0.0	I	I	I	I	I	I	I
Ornithosis	losis	93	102	65	61	06	75	82.2	0.9	0.4	0.5	0.3	0.3	0.4	0.3
Q fever	er	449	378	311	335	352	358	365.0	1.0	2.1	1.8	1.4	1.5	1.6	1.6
Tularé	Tularaemia	0	0	0	0	2	0	0.4	0.0	I	I	I	I	<0.1	I
Othe	Other bacterial diseases														
Legio	Legionellosis	303	272	301	302	358	382	307.2	1.2	1.4	1.3	1.4	1.4	1.6	1.7
Leprosy	isy	14	1	5	13	8	4	10.2	0.4	0.1	0.1	<0.1	0.1	<0.1	<0.1
Menir	Meningococcal infection ^{ss}	305	286	259	228	241	223	263.8	0.8	1.4	1.3	1.2	1.0	1.1	1.0
Tuber	Tuberculosis	1,133	1,214	1,314	1,357	1,399	1,315	1,283.4	1.0	5.4	5.6	6.1	6.2	6.3	5.8
Total		146,119	159,408	237,268	209,967	238,519	243,872								
*	Newly acquired hepatitis and syphilis 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.	includes ca	ises where	the infectio	n was dete	rmined to be	e acquired	within 24 m	ronths prio	r to diagnos	sis. Queent	sland repor	rts hepatitis	s C newly a	cquired
≁	Unspecified hepatitis and syphilis includes cases where the duration of	udes cases	where the	duration of	infection c	infection could not be determined or is greater than 24 months	determinec	d or is great	er than 24	months.					
++	In Queensland, includes newly acquired hepatitis C cases	ed hepatitis	C cases.												
Ś	Infection with Shiga toxin/verotoxin producing Escherichia coli.	oducing Es	cherichia cu	oli.											
=	Includes <i>Chlamydia trachomatis</i> identified from cervical, rectal, urine, un the Northern Territory and Western Australia exclude ocular infections.	ified from c stralia excl	ervical, reclude ocular	tal, urine, u infections.	rrethral, thro	rethral, throat and eye samples, except for South Australia, which reports only cervical, urine and urethral specimens;	samples, ε	except for S	outh Austr	alia, which	reports only	y cervical,	urine and u	urethral spe	cimens;
F	The national case definitions for chlamydial, gonococcal and syphilis di	Jydial, gon	ococcal anc	ł syphilis di	iagnoses in	agnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal	ons that m	ay be acqu	lired throug	jh a non-se	xual mode	(especially	/ in childrer	1 – e.g. per	inatal

infections, epidemic gonococcal conjunctivitis).

Does not include congenital syphilis. *

Data for all states and territories are reported by diagnosis date, except Queensland which is reported by notification receive date.

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 the Australian Capital Territory also reports conjunctival cases. Ś

Not elsewhere classified. Not notifiable. NEC NN

Table 7: Year which diseases become notifiable to the National Notifiable Diseases Surveillance System, Australia, by state or territory*	me not	ifiable	to the l	Vationa	l Notif	iable L	iseases	Survei	llance System,	Australia, by state or territory [*]
		Year in	which d	Year in which data first sent to Commonwealth	sent to C	tommon	wealth		Period of	
Disease	ACT	NSN	NT	QId	SA	Tas.	Vic.	WA	national reporting	Exceptions to national reporting
Bloodborne diseases										
Hepatitis B (newly acquired)	1995	1993	1993	1994	1993	1993	1993	1994	1995 to present	
Hepatitis B (unspecified)	1991	1991	2004	1994	1991	1991	1991	1991	1991 to present	
Hepatitis C (newly acquired)	1995	1993	2005	NN	1993	1995	1997	1995	1993 to present	Reported under hepatitis C (unspecified) in Qld
Hepatitis C (unspecified)	1991	1991	1991	1991	1994	1991	1991	1993	1995 to present	Includes reports of incident hepatitis C, 1991 to 1994
Hepatitis D	1999	1999	1999	1997	1999	1999	1999	2001	1999 to present	
Gastrointestinal diseases										
Botulism	1992	1998	1998	1997	1993	1992	1992	2001	1992 to present	
Campylobacteriosis	1991	ZZ	1991	1991	1991	1991	1991	1991	1991 to present	
Cryptosporidiosis	2001	2001	2001	1996	2001	2001	2001	2001	2001 to present	
Haemolytic uraemic syndrome	1999	1999	1999	1997	1999	1999	1999	1999	1999 to present	
Hepatitis A	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Hepatitis E	1999	1999	1999	1999	1999	1999	1999	2001	1999 to present	
Listeriosis	1991	1991	1994	1991	1992	1991	1991	1991	1991 to present	
Salmonellosis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Shigellosis	1991	2001	1991	1997	1991	1991	1991	1991	1991 to present	Qld did not report 1997–2006
STEC, VTEC ⁺	1999	1999	1999	2002	1999	1999	1999	2001	1999 to present	
Typhoid [‡]	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Quarantinable diseases										
Cholera	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Highly pathogenic avian influenza in humans	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	Reported under influenza in WA
Plague	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Rabies	1993	1997	1991	1991	1991	1991	1991	1991	1991 to present	
Severe acute respiratory syndrome	2003	2003	2003	2003	2003	2003	2003	2003	2003 to present	
Smallpox	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Viral haemorrhagic fever	1993	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Yellow fever	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	

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

		Year ir	Year in which data fir		st sent to Commonwealth	Common	wealth		Period of	
Disease	ACT	NSN	NT	QId	SA	Tas.	Vic.	WA	national reporting	Exceptions to national reporting
Sexually transmissible infections										
Chlamydial infection	1993	1991	1991	1991	1993	1991	1991	1993	1994 to present	NSW did not report 1994–1998
Donovanosis	1991	2002	1991	1991	2002	1993	1991	1991	1991 to present	
Gonococal infection [§]	1991	1993	1991	1991	1991	1991	1991	1991	1991 to present	
Syphilis – all'	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Syphilis < 2 years	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Syphilis > 2 years or unspecified duration	2004	2004	2004	2004	2012	2004	2004	2004	2004 to present	
Syphilis – congenital	2003	2003	2003	2003	2003	2003	2003	2003	2003 to present	
Vaccine preventable diseases										
Diphtheria	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Haemophilus influenzae type b	1991	1991	1991	1991	1991	1991	1991	1994	1991 to present	
Influenza (laboratory confirmed)	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	Influenza became legally notifiable in SA in May 2008
Measles	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Mumps	1992	1992	1995	1997	1994	1995	1992	1994	1995 to present	Qld did not report 1999–2000
Pertussis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Pneumococcal disease (invasive)	2001	2001	2001	1997	2001	2001	2001	2001	2001 to present	
Poliomyelitis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Rubella¶	1991	1991	1993	1991	1993	1995	1992	1994	1993 to present	
Rubella – congenital	2003	2003	2003	1997	2003	2003	2003	2003	2003 to present	
Tetanus	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	Qld did not report 1991–1993
Varicella zoster (chickenpox)	2006	NN	2006	2006	2006	2006	2009	2006	2006 to present	
Varicella zoster (shingles)	2006	ZZ	2006	2006	2006	2006	2002	2006	2006 to present	
Varicella zoster (unspecified)	2006	NN	2006	2006	2006	2006	2009	2006	2006 to present	
Vectorborne diseases										
Barmah Forest virus infection	1995	1995	1997	1995	1995	1995	1995	1995	1995 to present	
Dengue virus infection	1993	1991	1991	1991	1991	1991	1991	1995	1991 to present	
Arbovirus infection (NEC)** ^{,††}	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	Includes JEV, MVEV and Kunjin 1991–2000
Japanese encephalitis virus infection	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	
Kunjin virus	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	Reported under MVE in ACT
Malaria	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Murray Valley encephalitis virus infection	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	Combined with Kunjin in ACT
Doce Divervirue infection	1002	1993	1001	1001	1003	000	1001	1001		

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

CDI

Vol 39

No 1

2015

0 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. + ++ ∞ = **⊨** ‡ ^Z

Not notifiable

E61

Data completeness

In 2012, sex and age at onset was complete for 99.9% of notifications in NNDSS (Table 8).

Indigenous status

Indigenous status was complete for 51.5% of notifications, and varied by jurisdiction. Indigenous status was complete for 94.7% of data reported in the Northern Territory, 93% in Western Australia and 91.5% in South Australia. In the remaining jurisdictions, Indigenous status completeness ranged from 16.8%–50.1% (Table 9).

Data completeness on Indigenous status also varied by disease as summarised in Appendix 3. There were 7 diseases for which notifications were 100% complete for Indigenous status. A further 25 diseases equalled or exceeded 80% completeness for Indigenous status.

In 2012, CDNA set target thresholds of 95% completeness for 18 priority diseases (Table 10) and 80% completeness for the remainder of the notifiable diseases. In 2012, there were 8 priority diseases for which Indigenous status completeness exceeded 95% (donovanosis, *Haemophilus influenzae* type b, hepatitis A, measles, meningococcal infection, syphilis < 2 years duration, leprosy, and tuberculosis).

Bloodborne viruses

In 2012, 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 in the 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 being 'greater than 2 years or unspecified'. The determination of a case as 'newly acquired' is heavily reliant on public health follow-up, with the method and intensity of follow-up varying by jurisdiction and over time.

In interpreting these data it is important to note that changes in the number of notified cases over time may not solely reflect changes in disease prevalence or incidence. National testing policies developed by the Australian Society for HIV Medicine^{17,18} and screening programs, including the preferential testing of high risk populations such as prisoners, injecting drug users and persons from countries with a high prevalence of hepatitis B or C, may contribute to these changes.

Table 8: Completeness of National Notifiable Diseases Surveillance System data, Australia, 2012, by state or territory

				State or	territory				
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total notifications	3,784	52,564	7,064	67,328	19,795	6,257	53,775	33,255	243,872
Sex									
Unknown/ missing	2	96	0	8	0	0	197	1	304
Per cent complete	99.9	99.8	100.0	>99.9	100.0	100.0	99.6	>99.9	99.9
Age at onset									
Unknown/ missing	0	21	0	33	0	0	135	3	192
Per cent complete	100.0	>99.9	100.0	>99.9	100.0	100.0	99.7	>99.9	99.9

Table 9: Indigenous status completeness of National Notifiable Diseases Surveillance System data, Australia, 2012, by state or territory*

				State or	territory				
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total notifications	3,784	52,564	7,064	67,328	19,795	6,257	53,775	33,255	243,872
Indigenous status									
Unknown/ missing	2,869	43,742	376	36,775	1,685	3,667	26,847	2,325	118,286
Per cent complete	24.2	16.8	94.7	45.4	91.5	41.4	50.1	93.0	51.5

* 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.

Priority disease	ACT	NSW	NT	Qld	SA	Tas.	Vic	WA	Aust.
Dengue virus (locally acquired)	-	100.0	-	89.3	-	-	34.0	-	54.2
Donovanosis	-	-	-	-	-	-	-	100.0	100.0
Gonococcal infection	100.0	26.2	97.1	70.9	96.6	91.4	64.2	99.9	64.7
Haemophilus influenzae type b	-	100.0	-	100.0	100.0	100.0	100.0	100.0	100.0
Leprosy	-	-	-	-	-	-	100.0	-	100.0
Measles	-	97.6	100.0	75.0	100.0	-	100.0	100.0	97.5
Meningococcal disease (invasive)	100.0	100.0	100.0	93.7	100.0	100.0	80.0	100.0	95.1
Pertussis <5 years	93.8	88.3	100.0	58.5	98.2	98.3	88.3	97.1	83.6
Shigellosis	83.3	67.7	100.0	75.6	100.0	71.4	95.8	100.0	87.6
Tuberculosis	94.4	99.6	100.0	97.2	100.0	100.0	100.0	100.0	99.4
Hepatitis A	100.0	97.6	100.0	73.5	100.0	100.0	88.7	100.0	89.7
Hepatitis B (newly acquired)	100.0	86.2	100.0	63.6	100.0	100.0	92.3	100.0	85.5
Hepatitis C (newly acquired)	100.0	59.6	-	-	97.4	95.7	78.2	100.0	86.9
Syphilis – congenital	-	-	-	-	-	-	-	-	_
HIV	NDP								
Pneumococcal disease <5 years	100.0	98.5	100.0	76.5	100.0	100.0	91.9	100.0	93.6
Pneumococcal disease ≥ 50 years	100.0	95.1	100.0	84.5	100.0	100.0	94.1	100.0	94.2
Syphilis < 2 years	100.0	91.2	100.0	96.3	96.2	100.0	89.2	100.0	92.7

Table 10: Percentage completeness of Indigenous status for priority diseases in National Notifiable Diseases Surveillance System data, Australia, 2012, by state or territory

NDP No data provided.

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

Notifications of HIV and AIDS were reported directly to The Kirby Institute, which maintains the National HIV Registry. Information on national HIV and AIDS surveillance can be obtained from <u>The Kirby Institute web site</u> (http:// www.kirby.unsw.edu.au/).

Hepatitis **B**

- 6,702 cases of hepatitis B were notified in 2012.
- Over the past 10 years, notifications of hepatitis B have declined.

Hepatitis B virus causes inflammation in the liver.¹⁹ Notifications of acute hepatitis B are classified as 'newly acquired' and chronic infections as 'unspecified'.

Epidemiological situation in 2012

In 2012, there were 6,702 notified cases of hepatitis B (both newly acquired and unspecified), equating to a rate of 29.5 cases per 100,000 (Figure 3). The Northern Territory reported the highest hepatitis B rate in 2012 (87.2 per 100,000), followed by Victoria (33.9 per 100,000), Western Australia (33.8 per 100,000) and New South Wales (31.9 per 100,000) (Table 1).

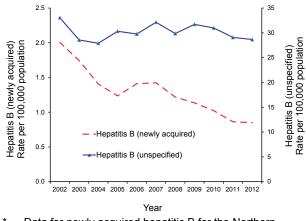
Between 2002 and 2012, unspecified hepatitis B rates decreased by 13.3% from 33.0 to 28.7 per 100,000, while newly acquired hepatitis B rates decreased from 2.0 to 0.8 per 100,000 (Figure 3). The continued decline in hepatitis B notifications may be attributed to the hepatitis B vaccination program, which was introduced nationally for infants in 2000, and the adolescent hepatitis B vaccination program, which was introduced in 1997.²⁰ In 2012, approximately 94% of children 12–24 months of age were assessed as being fully immunised against hepatitis B.²¹

Newly acquired hepatitis B

Epidemiological situation in 2012

In 2012, 193 newly acquired hepatitis B notifications (0.8 per 100,000) were reported to the NNDSS, a 1.0% decrease compared with the 195 cases (0.9 per 100,00) reported in 2011 and a continuation of the downward trend in notification rates (Figure 3).

Figure 3: Notification rate for newly acquired hepatitis B* and unspecified hepatitis B,[†] Australia, 2002 to 2012, by year



- Data for newly acquired hepatitis B for the Northern Territory (2002–2004) includes some unspecified hepatitis B cases
- † Data for unspecified hepatitis B for all states and territories, excluding the Northern Territory between 2002 and 2004

Geographical distribution

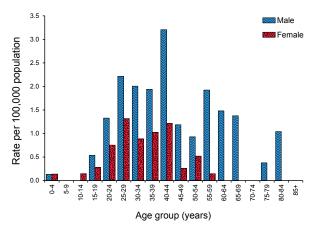
The highest rates were reported from the Northern Territory (2.1 per 100,000) and Tasmania (2.0 per 100,000) (Table 5).

Age and sex distribution

Overall, notification rates were higher among males than females, with a male to female ratio of 2.6:1. In 2012, the highest rate of newly acquired hepatitis B infection was observed among males aged 40–44 and 25–29 years (3.4 and 2.6 per 100,000 respectively) (Figure 4).

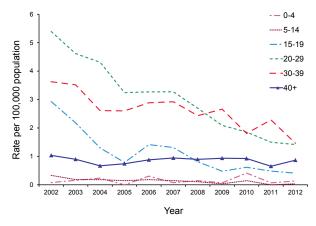
Between 2002 and 2012, most age group specific notification rates were trending downwards. The most marked decreases occurring among those aged 15–19 years and 20–29 years. During this period, notification rates among the 15–19 years age group declined by 86% from 2.9 to 0.4 per 100,000 and notification rates among the 20–29 years age group declined by 74% from 5.4 to 1.4 per 100,000 (Figure 5). These declines are likely to be attributable in part to the adolescent hepatitis B vaccination program. The notification rates among people aged 40 years or over have stabilised, which may be attributable to rates of testing or immigration from countries with higher prevalence of hepatitis B.²²

Figure 4: Notification rate of newly acquired hepatitis B, Australia, 2012, by age group and sex



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

Figure 5: Notification rate of newly acquired hepatitis B,* Australia, 2002 to 2012, by year and age group



* Data for newly acquired hepatitis B for the Northern Territory (2002–2004) includes some unspecified hepatitis B cases.

Risk groups

Enhanced data on risk factors and country of birth was provided by New South Wales, Victoria, Tasmania and the Australian Capital Territory (Table 11).[†] In 2012, 81.5% (n=88) of these cases had at least 1 risk factor recorded, with a potential source of exposure not recorded or unable to be determined for the remainder. Sexual contact was the most frequently reported potential source of infection (40.7%), followed by injecting drug use (30.6%), which remained stable from 2011 (31.0%).

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

	Numbe	r of risk factors	reported	Percentage of total cases [§]
Risk factors	Male	Female	Total	(n=108)
Injecting drug use	21	12	33	30.6
Imprisonment	3	0	3	2.8
Skin penetration procedure	6	4	10	9.3
Tattoos	3	2	5	4.6
Ear or body piercing	2	2	4	3.7
Acupuncture	1	0	1	0.9
Healthcare exposure	10	4	14	13.0
Surgical work	5	1	6	5.6
Major dental surgery	4	3	7	6.5
Blood/tissue recipient (Australia)	1	0	1	0.9
Sexual exposure	29	15	44	40.7
Hepatitis B positive partner – opposite sex	13	11	24	22.2
Hepatitis B positive partner – same sex	4	0	4	3.7
Partner with unknown hepatitis B status – opposite sex	3	0	3	2.8
Partner with unknown hepatitis B status – same sex	1	0	1	0.9
Unprotected sex – partner sex not recorded	6	3	9	8.3
Unprotected sex with a sex worker	2	0	2	1.9
Protected sex with a sex worker	0	1	1	0.9
Other	4	3	7	6.5
Needlestick or biohazardous injury ^{II}	2	0	2	1.9
Household contact	2	3	5	4.6
Cases with at least 1 exposure recorded	63	25	88	81.5
Undetermined	15	3	18	16.7
Unknown (not recorded)	2	0	2	1.9
Total exposures reported [†]	90	41	131	_
Total number of cases	80	28	108	_

Table 11: Newly acquired hepatitis B cases,* selected jurisdictions, 2012, by sex and risk factors^{†,‡}

* Cases from New South Wales, the Australian Capital Territory, Tasmania 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 Australian Capital Territory, Tasmania 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.

Of the 93 cases for which the country of birth was reported, 62 were in Australian born persons (66.7%, n=62) and 31 cases were born overseas.

Unspecified hepatitis B

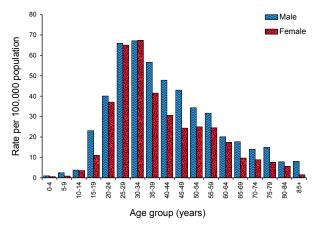
Epidemiological situation in 2012

In 2012, 6,509 cases of unspecified hepatitis B infection were notified to the NNDSS, a rate of 28.7 per 100,000, compared with 6,578 cases (29.1 per 100,000) reported in 2011.

Age and sex distribution

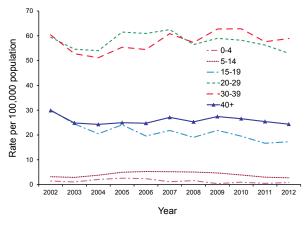
In 2012, the overall male rate (32.0 per 100,000) was higher than for females (24.9 per 100,000), a rate ratio of 1.28:1. Notification rates were higher among males in all age groups, except those aged 30–34 years where females (67.3 per 100,000) had slightly higher rates than males (67.1 per 100,000). For both males and females, the peak notification rate occurred among those aged 30–34 years (Figure 6). Between 2002 and 2012, notification rates across all age groups have declined, with the biggest decrease (42%) among the 15–19 years age group; declining from a rate of 30.0 in 2002 to 17.3 per 100,000 in 2012 (Figure 7).

Figure 6: Notification rate for unspecified hepatitis B,* Australia, 2012, by age group[†] and sex



- * Data for unspecified hepatitis B for all states and territories, excluding the Northern Territory between 2002 and 2004.
- † Excludes notifications for whom age was not reported.

Figure 7: Notification rate for unspecified hepatitis B,* Australia, 2002 to 2012, by year and age group



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

Hepatitis C

- 10,114 cases of hepatitis C were notified in 2012.
- Over the past 10 years, notifications of hepatitis C have declined by 42%.

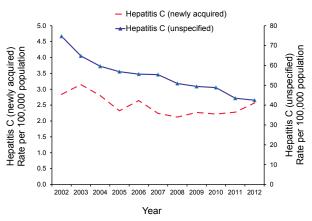
Hepatitis C causes inflammation of the liver. In more than 90% of cases initial infection with hepatitis C virus is asymptomatic or mildly symptomatic. Approximately 50%–80% of cases go on to develop a chronic infection. Of those who develop a chronic infection, half will eventually develop cirrhosis or cancer of the liver.¹⁹

Hepatitis C notifications are classified as being either 'newly acquired' (evidence that infection was acquired within the 24 months prior to diagnosis) or 'unspecified' (infection acquired more than 24 months prior to diagnosis or not able to be specified). Ascertaining a person's hepatitis C serostatus and clinical history usually requires active follow-up by public health units.

Epidemiological situation in 2012

Between 2002 and 2012, hepatitis C notifications declined by 42% from 15,126 (78 per 100,000) to 10,114 (45 per 100,000). This declining trend is reflected in both newly acquired and unspecified hepatitis C notifications (Figure 8).

Figure 8: Notification rate for newly acquired hepatitis C* and unspecified hepatitis C,[†] Australia, 2002 to 2012, by year



- * Data for newly acquired hepatitis C from all states and territories except Queensland 2002–2012 and the Northern Territory 2002–2004.
- † Data for unspecified hepatitis C provided from Queensland (2002–2012) and the Northern Territory (2002–2004) includes both newly acquired and unspecified hepatitis C cases.

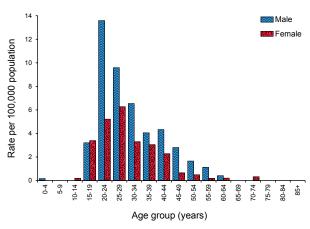
Newly acquired hepatitis C

- 466 cases of newly acquired hepatitis C were notified in 2012.
- The majority of notified cases in 2012 had a history of injecting drug use.
- The highest notification rates in 2012 were among males aged between 20 and 30 years of age.

Epidemiological situation in 2012

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, and the Northern Territory, where there were no notifications in 2012. Nationally, there were 466 notifications in 2012 (2.6 per 100,000) compared with 413 notifications in 2011 (2.3 per 100,000) (Figure 9). Of all hepatitis C cases in 2012, 4.6% were identified as having been newly acquired infections, a slighter higher proportion than the average of 3.5% reported since 2002 (range: 3.0%–4.0%).

Figure 9: Notification rate of newly acquired hepatitis C, Australia,* 2012, by age group and sex^{\dagger}



* Data from all states and territories except Queensland.

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

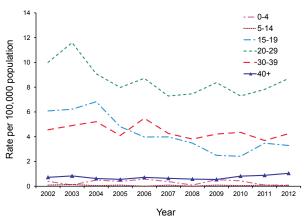
Geographical distribution

The highest rates of newly acquired hepatitis C infection were reported in Western Australia (5.1 per 100,000), South Australia (4.6 per 100,000) and Tasmania (4.5 per 100,000) (Table 5). The proportion of newly acquired infections compared with total hepatitis C diagnoses varied substantially among the states and territories ranging from less than 1% in the Northern Territory to 16% in South Australia. The identification and classification of newly acquired hepatitis C is reliant upon public health follow-up to identify testing and clinical histories. The method and extent of case follow-up, and the population groups targeted, vary among 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.

Nationally in 2012, the notification rate of newly acquired hepatitis C in males was 3.4 per 100,000 and in females 1.8 per 100,000. The male to female ratio was 1.9:1. Notification rates in males exceeded those in females across almost age groups. The highest notification rates were among males aged 20–24 years (13.6 per 100,000) and 25–29 years (9.6 per 100,000), and females aged 25–29 years (6.3 per 100,000) and 20–24 years (5.2 per 100,000) (Figure 9).

Between 2002 and 2012, notification rates declined overall among those aged 15–19, 20–29 and 30–39 years. However rates among the 20–29 years age group have risen since 2010 (from 7.3 to 8.7 per 100,000), and rates among those aged 30–39 years have risen since 2011 (from 3.7 to 4.2 per 100,000). Notification rates among those in the under 15 years and 40 years or over age groups have remained relatively low and stable during the period 2002–2012 (Figure 10).

Figure 10: Notification rates for newly acquired hepatitis C, Australia,* 2002 to 2012, by year and age group[†]



* Data from all states and territories except Queensland (2002–2012) and the Northern Territory (2002–2004).

† Excludes notifications for whom age was not reported.

Risk groups

Exposure histories for newly acquired hepatitis C cases reported in 2012 were analysed for all jurisdictions except Queensland (notified as unspecified hepatitis C) and Western Australia (no exposure data notified, n=125) (Table 12). In 2012, 86% of cases had at least 1 risk factor recorded, with the potential source of exposure not recorded or unable to be determined for the remainder. Approximately 98% of notifications had a history of injecting drug use, almost 65% of whom reported injecting drug

use in the 24 months prior to diagnosis. Skin penetration procedures and imprisonment accounted for approximately 22% and 17% of reported exposures 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 found that the prevalence of hepatitis C based on hepatitis C antibody detection was 22% in 2012, a decrease from 35% in 2007.²³

Table 12: Newly acquired hepatitis C notifications, selected jurisdictions,* 2012, by sex and risk factor^{†,‡}

	Number o	of risk factor	s reported	Percentage of total cases
Risk factors	Male	Female	Total	(n=341)§
Injecting drug use	205	128	333	97.7
Imprisonment	48	11	59	17.3
Skin penetration procedure	44	32	76	22.3
Tattoos	33	13	46	13.5
Ear or body piercing	9	18	27	7.9
Acupuncture	2	1	3	0.9
Health care exposure	4	2	6	1.8
Major dental surgery	1	1	2	0.6
Surgical work	1	1	2	0.6
Blood/tissue recipient	1	0	1	0.3
Healthcare worker with no documented exposure ^{II}	1	0	1	0.3
Sexual exposure	27	22	49	14.4
Hepatitis C positive partner – opposite sex	8	16	24	7.0
HIV positive men who have sex with men	13	_	13	3.8
Hepatitis C positive partner – same sex	4	4	8	2.3
Hepatitis C positive partner – sex of partner unknown	1	1	2	0.6
Sex worker	0	1	1	0.3
Unprotected sexual contact – status and sex of partner unknown	1	0	1	0.3
Other	16	20	32	9.4
Household contact	6	12	18	5.3
Needlestick or biohazardous injury [¶]	6	5	7	2.1
Other – not further categorised	2	3	5	1.5
Perinatal transmission	2	0	2	0.6
Cases with at least 1 exposure recorded	189	105	294	86.2
Undetermined	6	5	11	3.2
Unknown (not recorded)	22	14	36	10.6
Total exposures reported	344	215	555	_
Total number of cases	217	124	341	_

* Includes data from all states and territories except Queensland (not notified), Northern Territory (no cases) and Western Australia (no enhanced data on risk factors).

† 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 (notified as unspecified hepatitis C), the Northern Territory (n=0) and Western Australia (no exposure data notified, n=125). As more than 1 exposure category for each case could be recorded, the total percentage does not equate to 100%.

|| Healthcare worker with no recall of needlestick or biohazardous injury in the past 24 months prior to diagnosis.

¶ Includes both occupational and non-occupational exposures.

Unspecified hepatitis C

- 9,648 cases of unspecified hepatitis C were notified in 2012.
- The highest notification rates in 2012 were among males aged between 30 and 40 years.

Epidemiological situation in 2012

In 2012, 9,648 cases of unspecified hepatitis C infections were notified to the NNDSS (45.1 per 100,000), which was similar to the 9,832 cases in 2011 (45.7 per 100,000). Notification rates have decreased annually since 2002, with an overall decline of 42% between 2002 (77.5 per 100,000) and 2012 (45.1 per 100,000) (Figure 8). Several factors may account for the decrease including changes in surveillance practices, removal of duplicate notifications and a gradual decline in the prevalent group of hepatitis C cases accumulated prior to the introduction of hepatitis C testing in the early 1990s.^{24,25} The continuing decline in the notification rate may also be attributable to reductions in risk behaviours related to injecting drug use, especially among young people, and increased access to sterile injecting equipment through needle and syringe programs.²

Geographical distribution

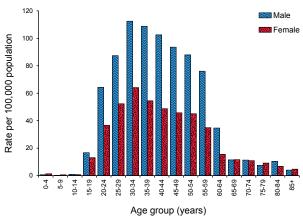
In 2012, the Northern Territory continued to have the highest notification rate (82.9 per 100,000) followed by Queensland (52.0 per 100,000) (Table 5).

Age and sex distribution

Nationally in 2012, the notification rate of unspecified hepatitis C in males was 55.1 per 100,000 and in females was 29.6 per 100,000, a male to female ratio of 1.9:1. Notification rates in males exceeded those in females across almost all age groups. The highest notification rates were among males aged 30–34 years (112.5 per 100,000), 35–39 years (108.8 per 100,000) and 40–44 years (102.5 per 100,000). The highest notification rates among females were in those aged 30–34 years (64.2 per 100,000), 35–39 years (52.3 per 100,000) (Figure 11).

Between 2002 and 2012, the notifications rates of unspecified hepatitis C declined overall across all age groups (Figure 12). The largest decreases occurred in those aged 20–29 years (from 153.2 to 60.9 per 100,000), 30–39 years (155.8 to 85.5 per 100,000) and 15–19 years (51.3 to 14.9 per 100,000). Notification rates in the 0–4, 5–14 and the 40 years or over age groups remained relatively stable over this time.

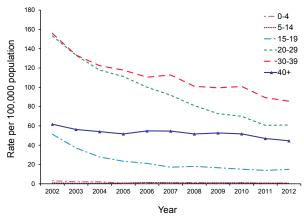




Data provided from Queensland includes both newly acquired and unspecified hepatitis C cases.

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

Figure 12: Notification rate for unspecified hepatitis C,*[†] Australia, 2002 to 2012, by year and age group



* Data provided from Queensland (2002–2012) and the Northern Territory (2002–2004) includes both newly acquired and unspecified hepatitis C cases.

† Excludes notifications for whom age was not reported.

Hepatitis D

- 30 cases of hepatitis D were notified in 2012.
- Hepatitis D is always associated with a hepatitis B co-infection.

Hepatitis D is a defective single-stranded RNA virus that replicates in the presence of the hepatitis B virus. Hepatitis D infection can occur as either an acute co-infection with hepatitis B or as a super-infection with chronic hepatitis B infection. The modes of hepatitis D transmission are similar to those for hepatitis B: exposure to infected blood or blood products, using contaminated needles or via sexual transmission. Household contact with people who are hepatitis B surface antigen positive is a major risk factor for transmission of hepatitis D.¹⁹

Epidemiological situation in 2012

In Australia, the notification rate of hepatitis D remains low. In 2012, there were 30 notified cases of hepatitis D; a rate of 0.14 per 100,000. Over the preceding 5 years, notifications of hepatitis D remained relatively stable with an average of 37 cases notified per year (range: 33–41).

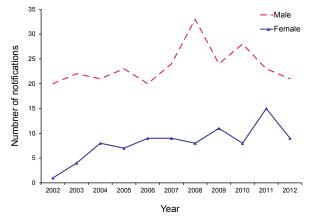
Geographical distribution

In 2012, Victoria reported the highest number of cases (9) followed by South Australia (8), Queensland (6), New South Wales (5) and Western Australia (2). Between 2007 and 2012, the majority of cases were from Victoria (67), Queensland (62) and New South Wales (59), with fewer cases reported from Western Australia (14), South Australia (10) and the Northern Territory (1). No cases were reported from the Australian Capital Territory or Tasmania during this period.

Sex distribution

The male to female ratio in 2012 was 2.3:1. This was less than the average ratio of 2.8:1 over the preceding 5 years, but greater than the 1.5:1 ratio reported in 2011 (Figure 13).





Gastrointestinal diseases

In 2012, gastrointestinal diseases notified to the 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 decreased from 32,784 in 2011 to 31,155 in 2012. None of the rates of gastrointestinal disease notified to NNDSS in 2012 were notably higher compared with the 5-year mean (exceeded the mean by more than 2 standard deviations).

Surveillance systems overview

Government established The Australian 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 2012.

Botulism

• No cases of botulism were notified in 2012.

Botulism is a rare but extremely serious intoxication resulting from the ingestion of toxins produced by *Clostridium botulinum* (commonly toxin types A, B and E). Four forms of botulism are recognised; infant, foodborne, wound and adult intestinal toxaemia.¹⁹

Epidemiological situation in 2012

There were no notifications of botulism in 2012. This compared with 2 notified cases in 2011 (both were infant botulism) and no notified cases in 2010.

Campylobacteriosis

- 15,653 cases of campylobacteriosis were notified in 2012.
- Campylobacter was the most frequently notified enteric infection in 2012.

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.¹⁹ Campylobacteriosis is notifiable in all Australian states and territories, except New South Wales.

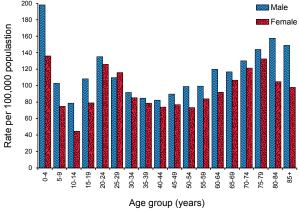
Epidemiological situation in 2012

There were 15,653 notifications of campylobacteriosis in 2012 making it the most frequently notified enteric infection (101.6 per 100,000). This was a decrease of 12% on the number of notifications received for 2011 (n=17,725) and a 6% decrease on the 5-year mean (n=16,669). Notification rates ranged from 74.4 per 100,000 in the Northern Territory to 172.2 per 100,000 in Tasmania.

Age and sex distribution

Campylobacteriosis was most frequently notified among the 0-4 years age group for both males (198 per 100,000) and females (136 per 100,000). The median age of notified cases was 34.5 years (range 0-101 years) and 54% (n=8,522) were male. Notification rates were higher among males compared with females in nearly all age groups (Figure 14).





Cryptosporidiosis

- 3,124 cases of cryptosporidiosis were notified in 2012.
- There was a 73% increase over 2011 notifications.

Cryptosporidiosis is a parasitic infection characterised by abdominal cramping and usually largevolume watery diarrhoea. Ingesting contaminated water, typically from a recreational source like a community swimming pool or lake, is a major risk factor for infection.¹⁹

Epidemiological situation in 2012

There were 3,124 notifications of cryptosporidiosis in 2012 (13.8 per 100,000). This represents a 73% increase over the 1,810 notifications reported in 2011, and a 23% increase over the 5-year mean of 2,544 notifications. Notification rates ranged from 5.1 per 100,000 in the Australian Capital Territory to 99.5 per 100,000 in the Northern Territory. Increases in notifications over 2011 levels were seen in most jurisdictions, particularly in Queensland and the Northern Territory. Queensland's increase was all in sporadic notifications whereas the Northern Territory reported an increase in sporadic notifications as well as 6 outbreaks, spread person-to-person in the child care setting.

Age and sex distribution

In 2012, notifications of cryptosporidiosis most frequently occurred in the 0–4 years age group (46%, n=1,437), and of these 59% (n=848) were male. This was consistent with 2011 where notifications of cryptosporidiosis were also most frequent in the 0–4 years age group (43%, n=780), and the majority of these were male (57%, n=446).

Haemolytic uraemic syndrome

- 20 cases of haemolytic uraemic syndrome were notified in 2012.
- Notifications were highest among the 0–4 years age group.

HUS is a rare but serious illness that is characterised by acute renal impairment; with 50% of patients requiring dialysis and about 5% resulting in death.¹⁹ Whilst not all diagnoses of HUS are related to enteric pathogens, Australian cases are commonly associated with STEC infection.²⁷

Epidemiological situation in 2012

There were 20 notifications of HUS in 2012 compared with 13 in 2011 and a mean of 17.2 notifications per year between 2007 and 2011.

Age and sex distribution

In 2012, HUS was most frequently notified among the 0-4 years age group (n=6). The median age of all notified HUS cases was 13 years (range 1-87 years) and 70% (n=14) were male, including all cases in the 0-4 years age group.

Hepatitis A

- 165 cases of hepatitis A were notified in 2012.
- Overseas travel was the primary risk factor for infection.

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 from person to person via the faecal-oral route but can also be foodborne or waterborne.¹⁹

Epidemiological situation in 2012

There were 165 notified cases of hepatitis A in 2012 (0.7 per 100,000). This was a 14% increase on the number of notifications in 2011 (n=145), but 42% less than the 5-year mean of 283. The mean reflects the impact of a 2009–2010 outbreak of hepatitis A associated with the consumption of semi-dried tomatoes.

Age and sex distribution

Hepatitis A was most frequently notified among the 25-29 years age group (16%, n=27) in 2012. The median age of notified cases was 28 years (range 0–92 years), and 52% (n=85) were female.

Indigenous status

Indigenous status was known for 90% (n=148) of cases of hepatitis A. However, none of these identified as being Indigenous.

Place of acquisition

Overseas travel was the primary risk factor for notified cases in 2012. Infection was considered to be overseas acquired in 66% (n=109) of notified cases.

In 2012, 18% (n=30) of notified cases were locally acquired. This was a decrease from 2011 where 27% (n=39) of notified cases were locally acquired

(Table 13). The 2009–2010 multi-state outbreak associated with the consumption of semi-dried tomatoes contributed to an increase in locally acquired hepatitis A cases in both 2009 and 2010.²⁸

Table 13: Notifications of hepatitis A, Australia, 2007 to 2012, by place of acquisition

	Loc	ally	Over	seas	Unkı	nown	
Year	n	%	n	%	n	%	Total
2007	65	39.2	77	46.4	24	14.5	166
2008	91	33.0	128	46.4	57	20.7	276
2009	349	61.9	137	24.3	78	13.8	564
2010	111	41.7	144	54.1	11	4.1	266
2011	39	26.9	97	66.9	9	6.2	145
2012	30	18.2	109	66.1	26	15.8	165

Hepatitis E

- 32 cases of hepatitis E were notified in 2012.
- Overseas travel was the primary risk factor for notified cases.

Hepatitis E is an acute viral infection primarily of the liver. The virus is transmitted by the faecaloral route, most often via food or water.¹⁹ Infection is usually acquired overseas among travellers to endemic areas.

Epidemiological situation in 2012

There were 32 notified cases of hepatitis E in 2012, compared with a 5-year mean of 34.6 notifications.

Age and sex distribution

Hepatitis E was most frequently notified among the 25-39 years age group (60%, n=19), the median age of notified cases was 30 years (range 24–61 years), and 75% (n=24) of total notifications were male.

Place of acquisition

Hepatitis E in Australia has traditionally been associated with overseas travel. In 2012, 84% of cases (n=27) reported overseas travel during their incubation period and were considered overseas acquired. Of these, 59% (n=16) reported travel to India. The place of acquisition for the remaining 5 cases was inadequately described or unknown.

Listeriosis

- 93 cases of listeriosis were notified in 2012.
- Notifications were highest in the 80+ years age group.

Invasive listeriosis is caused by infection with *Listeria monocytogenes*. It commonly affects the elderly or immunocompromised, typically among people with serious underlying illnesses. Listeriosis can also affect pregnant women and their unborn babies, sometimes resulting in miscarriage or foetal death. Laboratory confirmed infections in a mother and her unborn child or neonate are notified separately in the NNDSS.

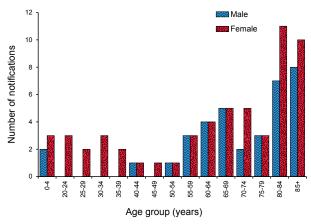
Epidemiological situation in 2012

There were 93 notified cases of invasive *L. mono-cytogenes* infection in 2012 (0.4 per 100,000). This was a 33% increase over 2011 (n=70) and a 32% increase compared with the 5-year mean of 70.2 notifications.

Age and sex distribution

Notifications for listeriosis were highest in the 80 years or over age group (41%, n=38), with 61% (n=57) of all notified cases being female (Figure 15).

Figure 15: Notifications of listeriosis, Australia, 2012, by age group and sex



Enhanced surveillance in 2012

OzFoodNet collects enhanced surveillance data on all notified cases of listeriosis in Australia. The information collected includes the characterisation of *L. monocytogenes* isolates by molecular subtyping methods, food histories and exposure data. The overall aim of this enhanced surveillance is to enable timely detection of outbreaks and subsequent public health response.²⁷

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

Salmonellosis (non-typhoidal)

- 11,265 cases of salmonellosis were notified in 2012
- Notifications were highest among the 0–4 years age group.

Salmonellosis is a bacterial disease characterised by 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.

Epidemiological situation in 2012

There were 11,265 notified cases of salmonellosis in 2012 (49.6 per 100,000). This represents an 8.2% decrease in notifications compared with 2011 (n=12,270 and the highest yearly notifications since salmonellosis became nationally notifiable in 1991), but a 9.5% increase compared with the 5-year mean of 10,289 notifications. In 2012, notification rates ranged from 40.4 per 100,000 in New South Wales to 173.1 per 100,000 in the Northern Territory.

Age and sex distribution

Salmonellosis was most frequently notified among the 0-4 years age group (25%, n=2,771). The median age of notified cases was 25 years (range 0-100 years) and 50% (n=5,673) of notifications were in females.

Shigellosis

- 547 cases of shigellosis were notified in 2012.
- Thirty-one per cent of notified cases were reported as being acquired overseas.

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 or water.¹⁹

Epidemiological situation in 2012

There were 547 notified cases of shigellosis in 2012 (2.4 per 100,000) with the number of notifications being less than the 5-year mean of 617 notifications. As in previous years, the highest notification rate was in the Northern Territory (45.5 per 100,000).

Age and sex distribution

Notifications for shigellosis were highest in the 0-4 years age group (18%, n=100). In 2012, the median age of notified cases was 27 years (range 0-85 years) and 51% (n=277) were male.

Indigenous status

Information on Indigenous status was available for 88% (n=479) of shigellosis notifications. This proportion varied by state or territory, with data for New South Wales, Queensland and Tasmania being less than 80% complete. Among states and territories with greater than 80% completeness, the proportion of notified cases who identified as being of Aboriginal or Torres Strait Islander origin was 36% (119/334).

Place of acquisition

Thirty-one per cent (n=167) of notified cases of shigellosis were reported as being acquired overseas. The most frequently reported countries of acquisition for imported cases were Indonesia (22%, n=37) and India (22%, n=37). Twenty-seven per cent of notified cases of shigellosis (n=147) were acquired in Australia and the place of acquisition for the remaining 43% of notified cases (n=233) was inadequately described or unknown.

Shiga toxin-producing *Escherichia coli* infections

- 111 cases of Shiga toxin-producing Escherichia coli were notified in 2012.
- Detection is strongly influenced by jurisdictional practices regarding the screening of stool specimens.

STEC is a cause of diarrhoeal illness in humans. People can become infected via faecal-oral transmission, ingesting contaminated food, through animal contact and from environmental exposures. Severe illness can progress to HUS. Children under 5 years of age are the most frequently diagnosed with STEC infection and are at greatest risk of developing HUS.¹⁹

Epidemiological situation in 2012

There were 111 notified cases of STEC in 2012 (0.5 per 100,000); a 10% increase compared with the 5-year mean of 101 notifications. Detection of STEC infection is strongly influenced by jurisdictional practices regarding the screening of stool specimens.²⁷ South Australia tests all bloody stools for Shiga toxin encoding genes and subsequently has the highest notification rate in Australia; 2.7 cases per 100,000, compared with 0.0–1.4 per 100,000 in other states and territories. Comparison of STEC notification data between jurisdictions and over time requires careful interpretation.

Age and sex distribution

In 2012, 53% (n=59) of notified STEC cases were male. The median age of notified cases was 46 years (range 1–95 years).

Typhoid

- 123 cases of typhoid were notified in 2012.
- As in previous years, overseas travel was the primary risk factor for infection.

Typhoid is a bacterial disease caused by *Salmonella enterica* serotype Typhi. Symptoms include sustained fever, marked headache, malaise and constipation more often than diarrhoea in adults. The transmission mode is the same as for nontyphoidal salmonellosis, however humans are the only reservoir for *S*. Typhi.¹⁹

Epidemiological situation in 2012

There were 123 notifications of typhoid in 2012 (0.5 per 100,000); a 14% increase compared with the 5-year mean of 108.2 cases, but a 9% decrease on the number of notifications in 2011 (n=135).

Age and sex distribution

Typhoid was most frequently notified among the 20-34 years age group (51%, n=63), the median age of notified cases was 26 years (range 0–61 years), and 60% (n=74) were male.

Place of acquisition

As in previous years, overseas travel was the primary risk factor for notified cases. In 2012, 89% (n=109) of notified cases reported overseas travel during their incubation period and were considered overseas acquired. India was the most frequently reported country of acquisition, accounting for 56% (n=61) of overseas-acquired cases in 2012.

Quarantinable diseases

Human diseases covered by the *Quarantine Act* 1908, and notifiable in Australia and to the WHO in 2012 were cholera, plague, rabies, yellow fever, smallpox, highly pathogenic avian influenza in humans (HPAIH), severe acute respiratory syndrome (SARS) and 4 viral haemorrhagic fevers (Ebola, Marburg, Lassa and Crimean–Congo). 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. More information on quarantinable diseases and travel health can be found on the <u>Department of Health</u> <u>web site</u> (www.health.gov.au/internet/main/ publishing.nsf/Content/health-pubhlth-strategquaranti-index.htm) and on the Department of Foreign Affairs and Trade <u>Smartraveller web site</u> (www.smartraveller.gov.au/).

There were no cases of plague, rabies, smallpox, SARS, HPAIH or viral haemorrhagic fevers reported in Australia in 2012. While there were cases of cholera (n=5) reported in 2012, Australia retained its official status as being free of all the listed quarantinable diseases (Table 14).

Cholera

- 5 cases of cholera were notified in 2012.
- All cases were acquired overseas.

Epidemiological situation in 2012

In 2012, there were 5 notifications of cholera in Australia. Between 2007 and 2011 there were 17 cases of cholera in total in Australia. The following details relate to the exposures or place of acquisition for the 5 cases in 2012:

- Two cases were reported by South Australia. Both had travelled separately to Phuket, Thailand during their incubation period, with visits to Phi Phi Island.
- New South Wales reported 2 cases, one with place of acquisition being Bangladesh and the other India.
- Queensland reported a case acquired in India.
- Cases ranged in age between 0 and 59 years.

All cases of cholera reported since the commencement of the NNDSS in 1991 to 2012 have been acquired outside Australia except for a single case of laboratory-acquired cholera in 1996³⁶ and 3 cases in 2006 linked to imported whitebait.³⁷

Sexually transmissible infections

Introduction

In 2012, the STIs reported to the NNDSS were chlamydia, donovanosis, gonorrhoea 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 for Infection and Immunity in Society.

Table 14: Australia's status for human quarantinable diseases, 2012

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 ²⁹
Rabies	Free	Last case (overseas acquired) recorded in Australia in 1990 ³⁰
Smallpox	Free	Last case recorded in Australia in 1938, last case world-wide in 1977, declared eradicated by the World Health Organization 1980 ^{31,32}
Yellow fever	Free	Two cases in 2011 are the first recorded, related to overseas travel ³³
Severe acute respiratory syndrome	Free	Last case recorded in Australia in 2003 ³⁴
Highly pathogenic avian influenza in humans	Free	No cases recorded ³⁵
Viral haemorrhagic fevers		
Ebola	Free	No cases recorded
Marburg	Free	No cases recorded
Lassa	Free	No cases recorded
Crimean–Congo	Free	No cases recorded

Chlamydial infection

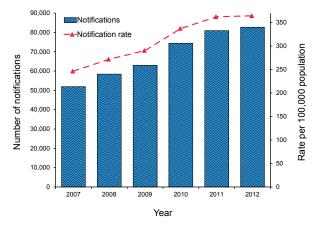
- 82,707 cases of chlamydial infection were notified in 2012.
- 2012 notification rates were similar to 2011.
- Women under 25 years of age and Indigenous people were disproportionately represented in the notifications of chlamydial infection.

Genital chlamydia infection is caused by the bacterium *Chlamydia trachomatis* serogroups D to K. Screening is important in detecting chlamydia infections, as a large proportion of infections are asymptomatic.³⁸ If infection is left untreated, complications such as epididymitis in men and infertility and pelvic inflammatory disease in females can arise.¹⁹

Epidemiological situation in 2012

Chlamydial infection was the most frequently notified disease to the NNDSS (34% of all notifications in 2012), with 82,707 cases (364 per 100,000) notified in 2012. Between 2011 and 2012, the notification rate of chlamydial infection increased by less than 1% (362 and 364 per 100,000 respectively), while between 2007 and 2011, notification rates increased by 47% (247 and 362 respectively) (Figure 16).

Figure 16: Notifications and notification rates per 100,000 for chlamydial infection, Australia, 2007 to 2012, by year

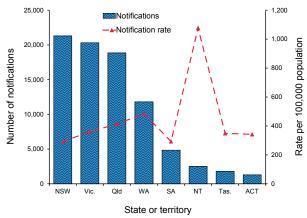


Geographical distribution

In 2012, the notification rate of chlamydial infection was almost 3 times higher in the Northern Territory (1,077 per 100,000) than nationally (364 per 100,000). In the remaining jurisdictions notification rates ranged between 292 per 100,000 in New South Wales and 485 per 100,000 in Western Australia (Figure 17).

All states and territories have seen overall increases in notification rates from 2007 to 2012, but only New South Wales and Victoria have seen increases in every year. During the same period, only the Northern Territory and Queensland have maintained a decline in rates over more than 1 year.





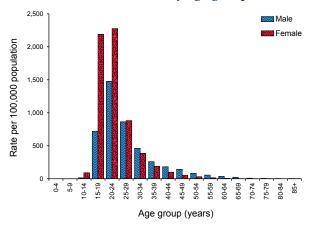
Age and sex distribution

Nationally in 2012, the notification rate for chlamydial infection was 307 per 100,000 in males, and 419 per 100,000 in females. In 2012, chlamydial infection occurred predominately among those aged 15–29 years, accounting for 80% of notified cases.

In total, the female to male rate ratio in 2012 was 1.36:1, slightly lower than the preceding 5-year mean of 1.43:1. In 2012, notification rates in females exceeded those in males under the age of 30 years, especially in the 10–14 years age group (Figure 18). The overall higher rate among females may be partly attributable to preferential testing of women attending health services compared with men.^{8,26}

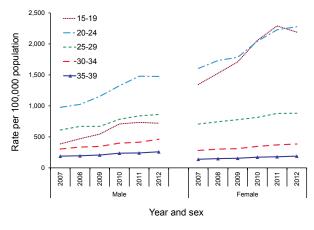
When considering trends over time in those aged 15–39 years, notification rates increased almost every year, for all age groups and for both sexes (Figure 19). The exceptions were between 2011 and 2012, when rates declined in males aged 15–19 years and 20–25 years, and females aged 15–19 years.

Figure 18: Notification rate for chlamydial infection, Australia, 2012, 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.

Figure 19: Notification rate for chlamydial infection in persons aged 15–39 years, Australia, 2007 to 2012, by year, sex* and age group



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

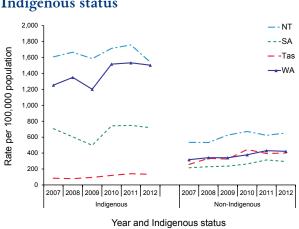
Indigenous population

The completeness of Indigenous status identification in the notification data varies by year and by jurisdiction. Nationally in 2012, data on Indigenous status were complete in 51% of notifications, slightly higher than the preceding 5-year mean of 49% (range: 44%–51%). Four jurisdictions had greater than 50% completeness of the Indigenous status field across the 2007 to 2012 period: the Northern Territory, South Australia, Tasmania, and Western Australia. Among these jurisdictions, the combined age-standardised notification rate ratio between Indigenous and non-Indigenous populations in 2012 was 3.6:1. Overall, this rate ratio has declined by 28% from 2007 (4.9:1) to 2012 (3.6:1). Among the Indigenous population, the age-standardised notification rate declined from 1,344 per 100,000 in 2011 to 1,252 per 100,000 in 2012. This followed increases in 2009, 2010 and 2011 (1,115, 1,321 and 1,344 per 100,000 respectively), which in turn followed a decline in 2008 (1,180 per 100,000).

Age-standardised notification rates among the non-Indigenous population have increased by 47% from 2007 (240 per 100,000) to 2012 (352 per 100,000). The average annual increase over this period was 8% (range: 2%–13%).

In terms of geographical trends, age-standardised notification rates of chlamydial infection in the Indigenous population declined from 2011 to 2012, in all 4 states and territories in which Indigenous status was more than 50% complete across 2007 to 2012. Age-standardised notification rates decreased in the Northern Territory by 12% (from 1,758 to 1,542 per 100,000), in Tasmania by 5% (from 141 to 134 per 100,000), in South Australia by 4% (from 748 to 719 per 100,000), and in Western Australia by 2% (from 1,533 to 1,504 per 100,000).

Between 2011 and 2012, the age-standardised notification rates of chlamydial infection in the non-Indigenous population increased by 5% in the Northern Territory (from 623 to 653 per 100,000) and by 2% in Tasmania (from 395 to 401 per 100,000). During the same period, age-standardised non-Indigenous notification rates decreased by 6% in South Australia (from 314 to 294 per 100,000) and by 2% in Western Australia (from 430 to 422 per 100,000) (Figure 20).



Includes the states and territories where Indigenous status was reported for more than 50% of cases between 2007 and 2012: the Northern Territory, South Australia, Tasmania and Western Australia.

Figure 20: Age standardised notification rates of chlamydial infection, selected states and territories,* 2007 to 2012, by year and Indigenous status

Donovanosis

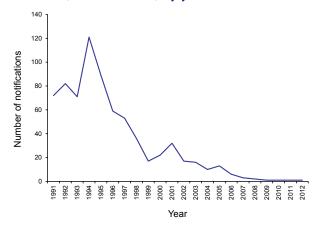
- One case of donovanosis was notified in 2012.
- This disease is now rare in Australia.

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.³⁹ Donovanosis was targeted for elimination in Australia through the National Donovanosis Elimination Project 2001–2004.⁴⁰ The disease predominantly occurred in Aboriginal and Torres Strait Islander females in rural and remote communities in central and northern Australia. It is now rare, with an average of 7 cases notified each year since 2002, and only 5 cases notified in the 5 years from 2008 to 2012.

Epidemiological situation in 2012

In 2012, 1 case of donovanosis was notified in a non-Indigenous male (Figure 21).

Figure 21: Notifications of donovanosis, Australia, 1991 to 2012, by year



Gonococcal infection

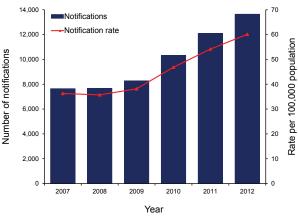
- 13,649 cases of gonococcal infection were notified in 2012.
- Notification rates of gonococcal infection continue to increase.
- Notifications in 2012 occurred predominately in males aged 20 years or over.

Gonorrhoea is caused by the bacterium Neisseria gonorrhoeae, which infects mucous membranes causing symptomatic and asymptomatic genital and extragenital tract infections.¹⁹ If left untreated, it can lead to pelvic inflammatory disease in women and infertility in both men and women. Gonococcal infection also increases the risk of both acquisition and transmission of HIV.³⁹

Epidemiological situation in 2012

In 2012, there were 13,649 cases of gonococcal infection reported to the NNDSS, a notification rate of 60 per 100,000. This was an 11% increase compared with the rate reported in 2011 (54 per 100,000). Notification rates were stable from 2007 to 2008 (36 per 100,000) and then increased in all subsequent years to 2012 by an average of 14% each year (range: 7%–23%). Overall, notification rates increased by 66% from 2007 (36 per 100,000) to 2012 (60 per 100,000) (Figure 22).

Figure 22: Notifications and notification rate for gonococcal infection, Australia, 2007 to 2012, by year



Geographical distribution

In 2012, the notification rate of gonococcal infection was more than 18 times higher in the Northern Territory (653 per 100,000) than nationally (36 per 100,000). The next highest notification rates were in Western Australia (87 per 100,000), then Queensland (59 per 100,000), New South Wales (57 per 100,000), Victoria (45 per 100,000), South Australia (30 per 100,000), the Australian Capital Territory (25 per 100,000), and Tasmania (9 per 100,000).

Between 2011 and 2012, rates increased in New South Wales (from 40 to 57 per 100,000), South Australia (from 27 to 30 per 100,000), Victoria (from 34 to 45 per 100,000) and Western Australia (from 78 to 87 per 100,000) and declined in the Australian Capital Territory (from 35 to 25 per 100,000), the Northern Territory (from 844 to

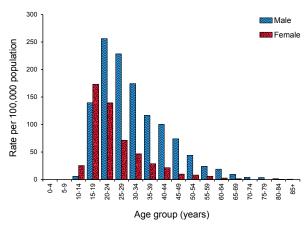
653 per 100,000), and Queensland (from 66 to 59 per 100,000). Between 2007 and 2012, all states and territories have seen overall increasing rates of gonococcal infection have been observed in all states and territories, except for the Northern Territory and Tasmania.

Age and sex distribution

Nationally in 2012, the notification rate for gonococcal infection was 84 per 100,000 in males and 36 per 100,000 in females. In males, this was an increase of 16% compared with the 2011 notification rate (73 per 100,000) and in females, this was an increase of 2% compared with the 2011 notification rate (35 per 100,000). In 2012, gonococcal infection occurred predominately among those aged 15–34 years, who accounted for 72% of notified cases.

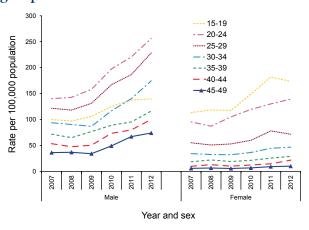
Across all age groups, the male to female ratio was 2.4:1 in 2012. This was similar to the ratios in the past 5 years. In 2012, notification rates in males exceeded those in females in all age groups above 20 years, especially in the 40–45 years age group (Figure 23).

Figure 23: Notification rate for gonococcal infection, Australia, 2012, 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 2010 to 2012, notification rates then increased in all age groups across both sexes, with the exception of females aged 15–19 years and 25–29 years where rates declined from 2011 to 2012 (Figure 24). Figure 24: Notification rate for gonococcal infection in persons aged 15–49 years, Australia, 2007 to 2012, by year, sex and age group*



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

Indigenous population

The completeness of Indigenous status information in the notification data varies by year and jurisdiction. Nationally in 2012, data on Indigenous status were complete for 65% of notifications, which was lower than the preceding 5-year mean of 69% (range: 68%–73%). All states and territories except New South Wales and the Australian Capital Territory had greater than 50% completeness for the Indigenous status field across the 2007 to 2012 period. The Australian Capital Territory has had greater than 50% completeness since 2008, with 100% completeness from 2010 to 2012. Among these states and territories, the combined age-standardised notification rate ratio between Indigenous and non-Indigenous populations in 2012 was 18.9:1, declining from 27.7:1 in 2011. Overall, the rate ratio has declined by 53% from 2007 to 2012 (from 40.2:1 to 18.9:1).

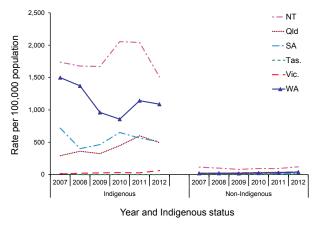
Among the Indigenous population, the age-standardised notification rate declined in 2012 from 2011 (from 876 to 724 per 100,000). The rates in 2012 were 9% lower than in 2007 (793 per 100,000).

The age-standardised notification rate among the non-Indigenous population almost doubled from 2007 to 2012 (20 and 38 per 100,000 respectively). The average annual increase over this period was 14% (range: 5%–21%).

In terms of geographical trends, age-standardised notification rates of gonococcal infection in the Indigenous population declined between 2011 and 2012 in most states and territories in which Indigenous status was more than 50% complete. Rates decreased in the Northern Territory by 26% (from 2,042 to 1,511 per 100,000), in Queensland by 17% (from 600 to 495 per 100,000), in South Australia by 10% (from 565 to 508 per 100,000), and in Western Australia by 5% (from 1,143 to 1,088 per 100,000). Tasmania reported no cases in Indigenous people in 2011 or 2012. The Indigenous notification rate in Victoria increased by 126% (from 28 to 63 per 100,000) (Figure 25).

Between 2011 and 2012, the age-standardised rates of gonococcal infection in the non-Indigenous population increased by 28% in the Northern Territory (from 95 to 121 per 100,000), by 32% in South Australia (from 15 to 19 per 100,000), by 92% in Tasmania (from 4 to 8 per 100,000), by 33% in Victoria (from 33 to 44 per 100,000), and by 41% in Western Australia (from 28 to 40 per 100,000) (Figure 25).

Figure 25: Age-standardised notification rate for gonococcal infection, selected states and territories,* 2007 to 2012, by year and Indigenous status



* Includes the states and territories where Indigenous status was reported for more than 50% of cases between 2007 and 2012: the Northern Territory, Queensland, South Australia, Tasmania, Victoria, and Western Australia.

Microbiological trends

The AGSP is the national surveillance system for monitoring the antimicrobial resistance of *N. gonorhoeae* isolates. These results are published in more details in the AGSP annual report in CDI.⁴¹

In 2012, the AGSP reported that a total of 4,784 gonococcal isolates were referred for antibiotic susceptibility testing, representing 35% of gonococcal infections notified to the NNDSS. This was similar to the proportion of NNDSS cases tested

in 2011, but lower than the 40%–42% referred in 2008–2010. Of the 4,784 referred isolates, 4,718 remained viable for antibiotic susceptibility testing.

Eighty-one per cent of the viable isolates (n=3,860) were from males and 19% (n=924) were from females (M:F, 4.18:1). The proportion of gonococcal isolates from males and females tested by the AGSP has remained similar over recent years (<1% variation).

In 2012, all isolates from all states and territories were susceptible to the injectable antibiotic spectinomycin.

Syphilis (non-congenital categories)

- 2,893 cases of syphilis (non-congenital categories) were notified in 2012; a rate of 12.7 per 100,000.
- In 2012, the notification rate for infectious syphilis was 6.8 per 100,000.
- The notification rate for syphilis of more than 2 years or unspecified duration was 6.0 per 100,000.

Syphilis, caused by the bacterium *Treponema pallidum*, is characterised by a primary lesion, a secondary eruption involving skin and mucous membranes, long periods of latency and late lesions of skin, bone, viscera, cardiovascular and nervous systems.¹⁹

In 2004, all jurisdictions except South Australia began reporting non-congenital syphilis infections to the NNDSS separately 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. From 2004 to 2011, South Australia reported only cases of infectious syphilis, and then in 2012 commenced reporting syphilis of more than 2 years or unknown duration. Data for all states and territories are reported by diagnosis date, except Queensland, which is reported by notification receive date.

Epidemiological situation in 2012

In 2012, a total of 2,893 cases of syphilis (noncongenital) were reported. This represents a rate of 12.7 per 100,000, a 6% increase compared with 2011 (12.0 per 100,000) (Table 6, Figure 26). A very small portion of this increase was due to the fact that in 2012 South Australia commenced reporting syphilis cases of more than 2 years or unknown duration. In 2012, 47% of syphilis notifications were categorised as greater than 2 years or unknown duration, and 53% of cases were categorised as less than 2 years duration.

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

- 1,539 cases of infectious syphilis were notified in 2012.
- In 2012, 78% of all notifications occurred in males aged 30 years or over. Notification rates in males exceeded those in females in almost all age groups.
- Cases of infectious syphilis were almost completely in men who have sex with men.

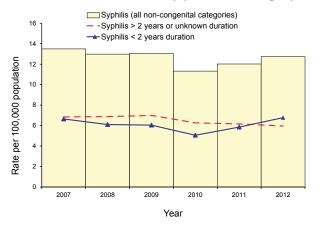
Epidemiological situation in 2012

In 2012, 1,539 notified cases of infectious syphilis (primary, secondary and early latent), less than 2 years duration, were reported to the NNDSS, representing a rate of 6.8 per 100,000. This was a 16% increase compared with the rate reported in 2011 (5.9 per 100,000) (Table 6, Figure 26). The notification rate for infectious syphilis declined by 26% from 2007 to 2010 (from 6.6 to 5.1 per 100,000), increased by 16% in 2011, and again by 16% in 2012 (Figure 26).

Geographical distribution

In 2012, notification rates of infectious syphilis (less than 2 years duration) were highest in Queensland and Victoria (both 8.4 per 100,000) (Table 15). Between 2007 and 2011, the Northern Territory consistently reported the highest rate of notifications compared with other states and territories. However, rates in the Northern Territory declined by almost 90% from 2007 (54.9 per 100,000) to 2011 (13.0 per 100,000) before halving again in 2012 (6.0 per 100,000).

Figure 26: Notification rate for noncongenital syphilis infection* (all categories), Australia,[†] 2007 to 2012, by year and category



- * For infectious syphilis, excludes notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired. For syphilis of more than 2 years or unknown duration, excludes all notifications where the case was aged less than 13 years.
- † For syphilis of more than 2 years or unknown duration, excludes South Australia from 2007 to 2011.

	Ma	le	Fem	ale	Tot	al*
State or territory	Notifications	Notification rate [†]	Notifications	Notification rate [†]	Notifications	Notification rate [†]
ACT	15	8.0	0	0.0	15	4.0
NSW	490	13.5	20	0.5	510	7.0
NT	9	7.3	5	4.5	14	6.0
Qld‡	306	13.4	77	3.4	383	8.4
SA	41	5.0	11	1.3	52	3.1
Tas.	13	5.1	1	0.4	14	2.7
Vic.	441	15.8	30	1.1	474	8.4
WA	69	5.6	8	0.7	77	3.2
Total	1,384	12.2	152	1.3	1,539	6.8

Table 15: Notifications and notification rates for syphilis less than 2 years duration, Australia, 2012, by state or territory and sex

* Includes notifications for whom sex was not reported.

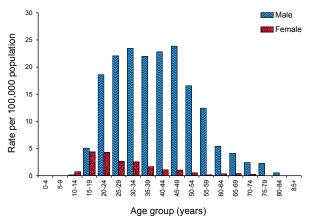
† Per 100,000 population.

‡ Data reported by notification received date.

Age and sex distribution

Nationally in 2012, the notification rate of infectious syphilis was 12.2 per 100,000 in males and 1.3 per 100,000 in females, equating to a male to female ratio of 9.2:1. In males, this was an increase of 21% when compared with the 2011 rate (10.1 per 100,000) and in females this was a decrease of 10% compared with the 2011 rate (1.5 per 100,000). The ratio of male to female notification rates increased by 35% compared with the 2011 ratio (6.8:1). In 2012, 78% of all notifications occurred in males aged 30 years or over, and notification rates in males exceeded those in females in almost all age groups (Figure 27). Diagnoses of infectious syphilis in 2012 were almost completely confined to men who have sex with men.⁴²

Figure 27: Notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, Australia, 2012, by age group and sex*

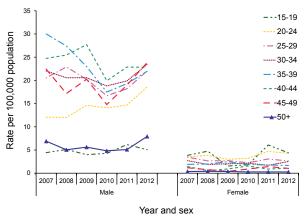


* Excludes notifications for whom age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

Notification rates for males aged 15 years or over declined overall among most age groups from 2007 to 2010. In 2011, notification rates in all age groups increased, and then in 2012, notification rates increased in all age groups except those aged 15–19 and 40–45 years (<1% increase) (Figure 28).

In females, notification rates between 2007 and 2012 have averaged 1.3 per 100,000 (range: 1.1–1.5). There was a notable increase among those aged 15–19 years from 2010 (1.8 per 100,000) to 2011 (6.1 per 100,000).

Figure 28: Notification rate for infectious syphilis (primary, secondary and early latent), less than 2 years duration, in persons aged 15 years or over,* Australia, 2007 to 2012, by year, sex* and age group



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

Indigenous population

The completeness of Indigenous status identification in the notification data varies by year and by jurisdiction. Nationally in 2012, data on Indigenous status were complete for 93% of notifications, a slight decrease compared with 2011 (95% complete) and slightly lower than the preceding 5-year mean of 95% (range: 94.6%–96.5%). All states and territories except the Australian Capital Territory had greater than 50% completeness for the Indigenous status field across the 2007 to 2012 period.

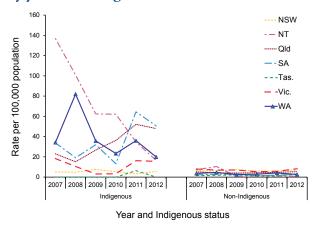
Among these states and territories, the combined age standardised notification rate ratio between the Indigenous and non-Indigenous populations in 2012 was 4.0:1, which is lower than the preceding 5-year mean of 5.4:1 (range: 4.4–6.0).

The age-standardised notification rate in the Indigenous population declined from 30.0 per 100,000 in 2011 to 24.1 per 100,000 in 2012. This follows decreases in 2008, 2009 and 2010 (31.7, 24.8 and 24.2 per 100,000 respectively). Overall, the rate in 2012 was 29% lower than the 2007 rate (33.7 per 100,000). The age-standardised notification rate among the non-Indigenous population increased from 5.1 per 100,000 in 2011 to 6.2 per 100,000 in 2012. This follows a decrease in 2010 (4.5 per 100,000), an increase in 2009 (5.6 per 100,000), and a decline in 2008 (5.4 per 100,000). The rate in 2012 is 6% higher than it was in 2007 (5.9 per 100,000).

In terms of geographical trends, from 2011 to 2012, age-standardised rates of syphilis in the Indigenous population declined in all states and territories except New South Wales (Figure 29). Between 2007 and 2012, the Northern Territory was the only jurisdiction to report declining Indigenous age-standardised notification rates every year. The increase evident in Indigenous notification rates in Western Australia in 2008 was largely attributable to an outbreak that occurred in the Pilbara region among Aboriginal people during that year.⁴³

Among the non-Indigenous population between 2011 and 2012, age-standardised rates of syphilis infection increased in all jurisdictions, except the Northern Territory and Western Australia (Figure 29).

Figure 29: Age-standardised notification rates of infectious syphilis (primary, secondary and early latent), less than 2 years duration, selected states and territories,* 2007 to 2012, by year and Indigenous status



* Includes the states and territories where Indigenous status was reported for more than 50% of cases between 2007 and 2012: New South Wales, Northern Territory, Queensland, South Australia, Tasmania, Victoria, and Western Australia.

Syphilis of more than 2 years or unknown duration

- 1,354 cases of syphilis of more than 2 years or unknown duration were notified in 2012.
- Overall, notification rates declined from 6.8 per 100,000 in 2007 to 6.0 per 100,000 in 2012.
- The notification rate among males (8.2 per 100,000) was more than double that in females (3.7 per 100,000) in 2012.

Epidemiological situation in 2012

In 2012, 1,354 cases of syphilis of more than 2 years or unknown duration were reported to the NNDSS. This represents a notification rate of 6.0 per 100,000, a decrease of 3% compared with 2011 (6.2 per 100,000) (Table 6, Figure 26). The notification rate of syphilis of more than 2 years or unknown duration increased by 1% between 2007 and 2008 (6.8 and 6.9 respectively), by 2% in 2009 (7.0 per 100,000), then declined by 10% in 2010 (6.3 per 100,000), and by 2% in 2011 (6.2 per 100,000) (Figure 26). Overall, notification rates have declined by 13% from 2007 to 2012 (6.8 to 6.0 per 100,000).

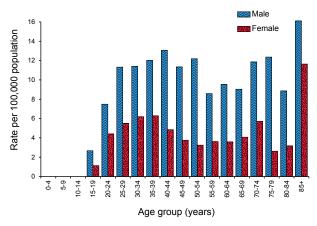
Geographical distribution

In 2012, notification rates of syphilis of more than 2 years or unknown duration were highest in the Northern Territory (28.5 per 100,000), followed by Victoria (9.0 per 100,000) (Table 16).

Age and sex distribution

Nationally in 2012, the notification rate of syphilis of more than 2 years or unknown duration was 8.2 per 100,000 in males and 3.7 per 100,000 in females; a male to female ratio of 2.2:1. In males, this was an increase of 8% when compared with the 2011 rate (7.6 per 100,000), and in females this was a decrease of 4% compared with the 2011 rate (3.8 per 100,000). Almost 70% of all notifications occurred in males aged 20 years or over, and notification rates in males exceeded those in females in all age groups (Figure 30).

Figure 30: Notification rate for syphilis of more than 2 years or unknown duration, Australia,* 2012, 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.

	Ма	Male		ale	Total*		
State or territory	Notifications	Notification rate [†]	Notifications	Notification rate [†]	Notifications	Notification rate [†]	
ACT	11	5.9	2	1.1	13	3.5	
NSW	195	5.4	88	2.4	283	3.9	
NT	62	50.2	5	4.5	67	28.5	
Qld‡	158	6.9	98	4.3	256	5.6	
SA	49	6.0	30	3.6	79	4.8	
Tas.	5	2.0	5	1.9	10	2.0	
Vic.	350	12.6	149	5.2	506	9.0	
WA	98	8.0	42	3.5	140	5.8	
Total	928	8.2	419	3.7	1,354	6.0	

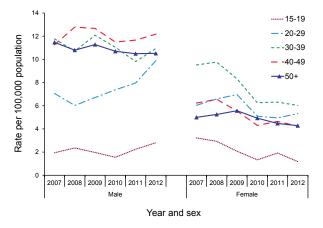
Table 16: Notifications and notification rates for syphilis (more than 2 years or unknown duration), Australia, 2012, by state or territory and sex

* Includes notifications for whom sex was not reported.

- † Per 100,000 population.
- ‡ By notification received date.

Notification rates for those aged 15 years or over from 2007 to 2012 increased overall in most age groups for males, and declined overall across age groups for females (Figure 31).

Figure 31: Notification rate for syphilis of more than 2 years or unknown duration, in persons aged 15 years or over,* Australia,[†] 2007 to 2012, by year, sex and age group



- Excludes notifications for whom age and/or sex were not reported.
- Data from all states and territories except South Australia in 2007–2011.

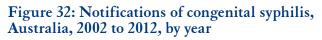
Congenital syphilis

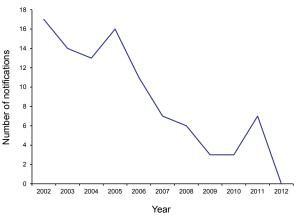
- No cases of congenital syphilis were notified in 2012.
- Congenital syphilis remains rare in Australia.

Congenital syphilis is caused by foetal infection with the bacterium *T. pallidum*. Syphilis is acquired by infants either in-utero or at birth from women with untreated early infection. Infections commonly result in abortion or stillbirth and may cause the death of a newborn infant. Congenital syphilis can be asymptomatic, especially in the first weeks of life.¹⁹

Epidemiological situation in 2012

There were no notifications of congenital syphilis in 2012, continuing the downward trend observed over the past decade (Figure 32). Antenatal screening for syphilis with follow up and adequate treatment is considered to be a contributor to this decline.⁴⁴





Vaccine preventable diseases

Surveillance objectives

This section summarises the national surveillance data for notifiable diseases targeted by the National Immunisation Programme (NIP) in 2012. These include diphtheria, invasive Haemophilus influenzae (Hib) type b infection, laboratory confirmed influenza, measles, mumps, pertussis, invasive (IPD), poliomyelitis, pneumococcal disease 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, are reported under bloodborne diseases and other bacterial infections, respectively. Other vaccine preventable diseases (VPDs) presented include hepatitis A and Q fever reported under the gastrointestinal and zoonoses sections, respectively. More detailed reports on historical data, including notifications, hospitalisations and deaths, have been published in the regular CDI Vaccine Preventable Diseases in Australia supplements.45 The more recent Australian vaccine preventable diseases epidemiological review series published in CDI, contain additional analysis on individual diseases.46-49

In 2012, there were 85,810 notifications of VPDs reported to the NNDSS, representing 35% of all notifications and a 5% increase compared with 2011 (81,872 cases) (Table 3). Influenza was the most commonly notified VPD with 44,563 (52%) cases reported, followed by pertussis (24,069 cases, 28%). The number of notifications and notification rates for VPDs in Australia are shown in Tables 4 and 5.

Vaccination coverage

Vaccination coverage is an important factor influencing the incidence of VPDs. Since the commencement of the Australian Childhood Immunisation Register in 1996, immunisation coverage in children has been high by international standards, although geographical pockets of lower coverage, in which there is an increased potential for VPD cases, remain. As no vaccine is 100% effective, infections with these diseases sometimes do occur in fully vaccinated people. Nonetheless, vaccination substantially lowers the chance of becoming infected and/or reduces the severity of disease.

Information on a case's vaccination history was previously recorded in the NNDSS using the vaccination status field (fully or partially vaccinated for age or not vaccinated), plus a field capturing the number of doses. In January 2008 new, more detailed fields were incorporated for recording vaccine type, vaccination validation 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 2012, 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 the vaccination status of a case is interpreted according to the data provided by the states and territories from the 2 different formats. A case is described as fully vaccinated if they have received all doses of the relevant vaccine according to the most recent edition of The Australian Immunisation Handbook²⁰ and at least 14 days prior to disease onset. In contrast, fully vaccinated for age describes a case that has received all recommended doses of a vaccine for their age but may not yet have received the full course of vaccinations required to be considered fully vaccinated.

Diphtheria

- There were no cases of diphtheria reported in Australia in 2012.
- Diphtheria is now rare in Australia.

Diphtheria is an acute toxin-mediated systemic disease caused by the toxigenic strains of *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 direct contact with skin lesions, or articles soiled by infected individuals.¹⁶ Non-toxigenic strains of *C. diphtheriae* usually only cause mild throat or skin infection and are not nationally notifiable.¹⁹

The NIP schedule in 2012 recommended a primary course of 3 doses at 2, 4, and 6 months of age, with additional booster doses at 4 years and between 10 and 15 years, delivered through school based programs.²⁰

In 2012, there were no notifications of diphtheria reported to the NNDSS. Whilst diphtheria is now rare in Australia, in 2011 there were 4 cases reported and prior to this, 1 case of cutaneous diphtheria reported in 2001. All these cases were associated with imported infections from countries where diphtheria remains endemic.

Influenza

- In 2012, notifications of laboratory confirmed influenza increased by almost 40% from 2011 making it the highest year since the 2009 pandemic year.
- Children aged 4 and under, middle aged and elderly adults, as well as those with underlying medical conditions were most affected.
- 2012 was the most severe influenza season since 2009.

Influenza is a common, highly infectious acute respiratory disease caused by infection with influenza viruses. The virus is transmitted from person to person by airborne droplets of exhaled respiratory secretions, especially by coughing or sneezing.⁵⁰ The disease caused by infection with influenza viruses ranges from asymptomatic⁵¹ through mild upper respiratory tract illness to severe complications including pneumonia. The severity of disease is determined by features intrinsic to the virus including its similarity to previous circulating and vaccine strains and by host factors including the presence of chronic conditions, pregnancy and smoking.⁵² The goals of influenza surveillance are to determine the severity, intensity and distribution of illness, detect outbreaks, monitor for changes in the virus and to facilitate policy development and planning.53

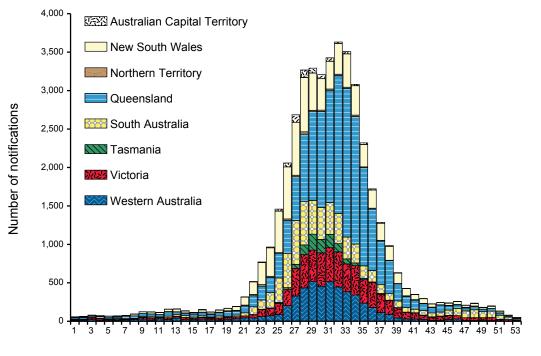
Vaccination

Seasonal influenza vaccination is the primary means of preventing influenza and its complications and is included in the NIP for specific groups of the population. In 2012, the NIP funded influenza vaccine for people aged 6 months or over with medical conditions placing them at risk of severe disease. It was also included for Aboriginal and Torres Strait Islander people aged 15 years or over, pregnant women and those aged 65 years or over.

Epidemiological situation in 2012

In 2012, there were 44,563 notifications of laboratory confirmed influenza. This was almost twice the number of notified cases reported the previous year and a more than 3-fold increase from 2010. Notification rates were highest in South Australia (380 per 100,000) and Queensland (369 per 100,000). Notifications in Western Australia, Tasmania, the Northern Territory and the Australian Capital Territory were similar to the national notification rate of 196 per 100,000, while the Victorian notification rate was substantially lower than the national notification rate at 106 per 100,000. Queensland reported the highest number of influenza cases of any jurisdiction, comprising 38% of all notifications, which was consistent with previous years with the exception of 2010 (Figure 33).

Figure 33: Notifications of laboratory confirmed influenza, Australia, 2012, by week and state or territory

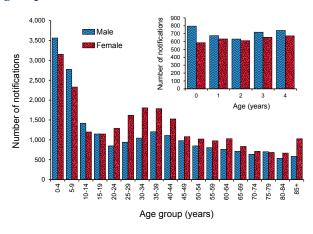


Week number

Age and sex distribution

The highest number of influenza notifications occurred in the 0-4 years age group, accounting for 26% of all notifications (Figure 34). Notification rates were highest in the 0-4 years and 85 years or over age groups (454 and 380 per 100,000 respectively) (Figure 34). The overall age distribution was characteristic of previous A(H3N2)-dominated seasons where preschool-age children and older adults were particularly affected (Figure 34).⁵⁴

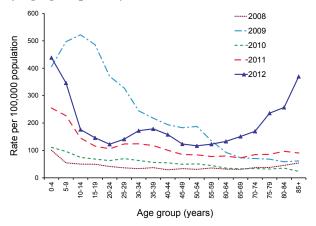
Figure 34: Notifications of laboratory confirmed influenza, Australia, 2012, by age group and sex*



* Excludes 224 notifications for which age and/or sex were not reported.

In 2012, females accounted for 23,890 (54%) of the influenza notifications for which sex was reported. Notification rates per 100,000 were higher among females in the 15–74 years age groups whereas males dominated the younger (0–14 years) and older (over 75 years) age groups (Figure 35).

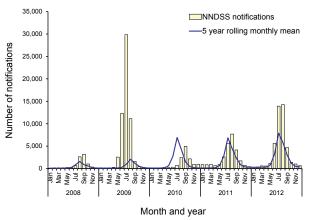
Figure 35: Notification rate for laboratory confirmed influenza, Australia, 2008 to 2012, by age group and year



Seasonality

Influenza activity during the 2011–2012 inter-seasonal period was the 2nd highest on record behind that observed in 2010–2011. Excluding 2009, notifications of influenza in 2012 started their seasonal increase earlier, rose sharply and peaked higher compared with previous years. Activity in the majority of jurisdictions peaked around mid-July. However, ongoing increased activity continued to be reported in Queensland, which peaked in mid-August and South Australia, which had a distinct second peak in late November (Figure 36).

Figure 36: Notifications of laboratory confirmed influenza, Australia,* 2007 to 2012, by month and year



 In South Australia, influenza was not made notifiable through legislation until May 2008.

Mortality

Nationally, there were 85 influenza-associated deaths notified to the NNDSS, with a median age of 80 years (range < 1–102). Approximately 88% (n=81) of those who died were reported as having influenza A (unsubtyped) or A(H3N2). Indigenous status was reported for 69% (n=67) of influenza-associated deaths notifications; Aboriginal and Torres Strait Islander peoples accounted for 9% (n=6) of influenza-associated deaths notifications. The number of influenza associated deaths reported to the NNDSS is reliant on the follow up of cases to determine the outcome of their infection and most likely underestimates the true mortality impact associated with this disease.

Virological surveillance

In 2012, typing data were reported for all but 4 laboratory-confirmed influenza notifications. Of the notifications with typing information, 76% were type A, (59% A (unsubtyped), 16% A(H3N2) and <1% A(H1N1)pdm09) and 24% were type B. Mixed influenza type A and B infections, and influenza type C together accounted for <1% of notifications (Figure 37). The ratio of influenza A to B was similar in 2011 and 2012. However, the distribution of A subtypes was markedly different, with 2012 being the first year since the 2009 pandemic not dominated by the H1N1 pandemic strain.

For 2012, the WHO Collaborating Centre for Reference and Research on Influenza (WHOCC) analysed 2,226 specimens from Australian influenza cases. This represented approximately 5% of the 44,563 laboratory confirmed cases reported to the NNDSS. Influenza A(H3N2) comprised 61% (n=1,357) of influenza viruses followed by influenza B (35% n=788) and influenza A(H1N1) pdm09 3.6% (n=81) (Figure 38).

The WHOCC assessed the antigenic similarity of circulating influenza virus isolates to reference strains by haemagglutination inhibition (HI) (n=1,742 influenza virus isolates). The majority of A(H3N2) isolates (1,115 of 1,118) were characterised as A/Victoria/361/2011-like, while the remainder were A/Perth/16/2009-like. No 'low reactor' A(H3N2) isolates were identified. All of the A(H1N1) viruses circulating (n=38), were antigenically similar to A/California/7/2009-like virus with 24% (n=9) characterised as 'low reactors'. Many of the low reactors had changes in the

Figure 38: WHO Collaborating Centre for Reference and Research on Influenza subtyped influenza virus samples, Australia, 2011 and 2012

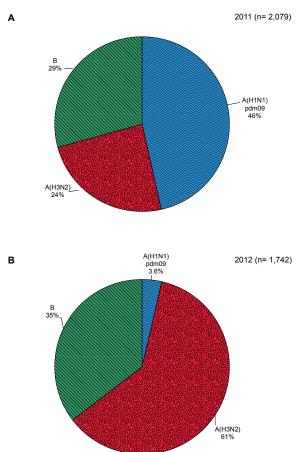
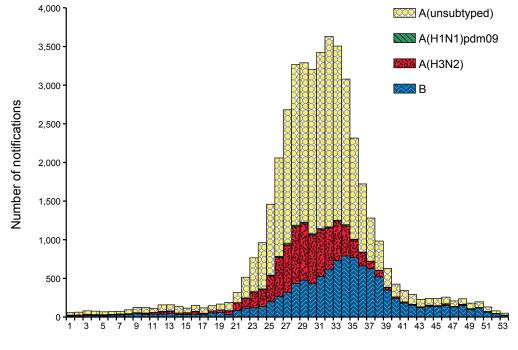


Figure 37: Notifications of laboratory confirmed influenza,* Australia, 2012, by week and subtype



Week number

* Excludes 81 mixed type A and B, type C and untyped influenza infections.

153–158 amino acid region of the haemagglutinin (HA) gene, which have been shown to reduce reactivity in HI assays. Comparison of HA genes from the original clinical samples suggest that the mutations are artefacts caused by isolation in Madin Darby canine kidney (MDCK) cells or in eggs.⁵⁵ Similarly, 90% (n=528) of influenza B viruses were closely related to the B/Brisbane/60/2008like (B/Victoria lineage) virus with 25% (n=131) characterised as 'low reactors'. The remaining 10% (n=58) of influenza B viruses were characterised as B/Wisconsin/1/2010-like, which belong to the B/Yamagata lineage. Except for the A(H3N2) viruses, the majority of influenza type A(H1N1) and B viruses that circulated during 2012, were antigenically similar to the 2010, 2011 and 2012 vaccine viruses.

Viruses collected in 2012 were also tested for sensitivity to the neuraminidase inhibitor class of antiviral drugs. A neuraminidase inhibition assay was performed on 1,715 virus isolates consisting of 1,126 A(H3N2), 43 A(H1N1)pdm09 and 546 B viruses. Resistance to oseltamivir was detected in a single A(H1N1)pdm09 isolate and was mediated by the well characterised H275Y mutation. All influenza B isolates examined were sensitive to oseltamivir. Further, all isolates were sensitive to zanamivir.

Additional surveillance activities

In addition to NNDSS data, a series of targeted influenza surveillance systems operated during 2012. Together these systems collected data that were used to describe the season under the areas of epidemiology, morbidity, mortality and virology and supported the conclusions drawn from analyses of NNDSS notification data. Enhanced influenza surveillance was based on the following sources of data:

- the number and proportion of calls to the National Health Call Centre Network related to influenza or influenza-like illness (ILI);
- rates of ILI and absence from work from a community survey;
- consultation rates for ILI identified by sentinel general practitioners;
- consultation rates for ILI identified by sentinel hospital emergency departments;
- hospitalised cases of influenza from 15 sentinel hospitals across Australia;
- mortality data from the New South Wales Registry of Births, Deaths and Marriages; and
- typing and subtyping for influenza from sentinel laboratories in New South Wales, Victoria, Western Australia and Tasmania.

Discussion

The 2012 influenza season in Australia began in May, peaked in mid-July and was largely concluded by the end of September. Australia experienced sustained virus circulation until late August, particularly in Queensland before steadily decreasing. Peak NNDSS notifications in 2012 occurred approximately 5 weeks earlier than the median week of peak transmission for the period of 2003 through 2011.⁵⁶ The most commonly detected virus was influenza A(H3N2), however influenza type B was a significant virus later in the year and was almost wholly responsible for South Australia's second wave of infections. The resurgence of A(H3N2) in Australia was associated with a shift in the age distribution of disease, compared with recent years when A(H1N1)pdm09 was the predominant virus circulating. The dominance of A(H3N2) coincided with a return to the more typical seasonal influenza pattern where the elderly and young infants are disproportionately affected.

Taken together, data from most influenza surveillance systems showed that the overall impact of influenza in 2012 was somewhat greater than average. At the seasonal peak, the number of influenza notifications reported per week and ILI consultation rates were higher than in any previous season since 2007, except for the 2009 pandemic.^{57,58} In the New South Wales Registered Death Certificates data, the rate of deaths classified as influenza and pneumonia met or exceeded the epidemic threshold for most of July, which was higher than in the previous 2 years, but lower than in 2007 and 2008.⁵⁹

In summary, notifications of influenza in 2012 started their seasonal increase earlier, rose sharply and peaked higher and for longer in comparison with previous years. When NNDSS notification data are combined with companion influenza surveillance systems, notification data supports the observation that 2012 was the most severe season since the beginning of notification in NNDSS, with the exception of 2009.

Invasive *Haemophilus influenzae* type b disease

- Hib continues to be a rare disease in Australia, with only 15 cases reported in 2012
- Notifications of Hib disease have remained relatively stable since 2000.
- Since the introduction of the Hib vaccine onto the NIP in 1993, there has been a reduction of more than 95% in notified cases of Hib disease.

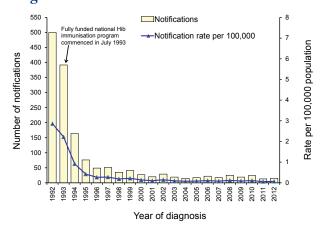
Invasive Hib is a bacterium that causes disease with symptoms dependant on which part of the body is infected. These include: septicaemia (infection of the blood stream); meningitis (infection of the membranes around the brain and spinal cord); epiglottitis (severe swelling of the epiglottis at the back of the throat); pneumonia (infection of the lungs); osteomyelitis (infection of the bones and joints); and cellulitis (infection of the tissue under the skin, usually on the face).

In 2012, the NIP schedule included 3 doses of a conjugate Hib vaccine at 2, 4 and 6 months of age, followed by a booster dose at 12 months of age.²⁰

Epidemiological situation in 2012

In 2012, there were 15 notifications of Hib disease. This was similar to the number of cases reported in 2011 (n=13), and less than the mean of the previous 5 years (n=20). The 2012 notification rate was 0.07 per 100,000 and was consistent with the very low rates that have been seen since the introduction of the vaccine on the NIP in July 1993 (Figure 39). Cases occurred in all jurisdictions, except the Australian Capital Territory and the Northern Territory. The notification rates vary widely because of the low overall number of notifications. There were 2 deaths reported in 2012, one in a partially vaccinated infant and one in an unvaccinated adult over 60 years of age.

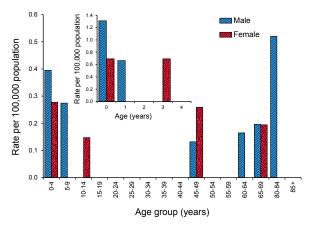
Figure 39: Notifications and notification rates for invasive Haemophilus influenzae type b infection, Australia, 1992 to 2012, by year of diagnosis



Age and sex distribution

In 2012, the male to female ratio was 1.5:1. Onethird of cases (n=5) were in children aged less than 5 years and 60% of these were among infants aged less than 1 year of age. The 0-4 years age group also had the highest notification rate (0.34 per 100,000). The remaining cases were among adults, ranging in age from 45–84 years (Figure 40).

Figure 40: Notification rate for invasive Haemophilus influenzae type b infection, Australia, 2012, by age group and sex



Indigenous status

Indigenous status was 100% complete in 2012. Two cases were reported as being Indigenous in 2012; a notification rate of 0.34 per 100,000. This rate was consistent with 2011, but much lower than 2010 (1.42 per 100,000). High routine Hib vaccination coverage has been achieved in Indigenous populations.²⁰

Vaccination status

In 2012, persons aged less than 20 years had been eligible for Hib vaccination through the NIP during infancy, following addition of the vaccine to the NIP in 1993. Eight of the 15 Hib notified cases reported in 2012 were aged less than 20 years. Of these cases, five were aged over 12 months and eligible for the full vaccine course, of which 2 cases were fully vaccinated, two were not vaccinated and one was partially vaccinated. The remaining 3 cases were aged less than 12 months and although they were fully vaccinated for their age, they had not yet completed the full course.

Discussion

Since the introduction of the Hib vaccine on to the NIP in 1993, there has been a reduction of more than 95% in notified cases of Hib disease in Australia (Figure 39). Australia now has one of the lowest rates of this disease in the world.⁴⁵

Invasive pneumococcal disease

 Notification data for 2012 shows early signs of a reduction in IPD disease due to 13v-non7v serotypes, most likely associated with the introduction of the 13vPCV vaccine.

IPD is a disease in which *Streptococcus pneumoniae* is isolated from a normally sterile site such as blood, cerebrospinal fluid or pleural fluid. Many of the signs and symptoms of IPD are non-specific including fever, chills, headache, stiff neck and a general feeling of being 'out-of-sorts' through to seizures and sometimes coma. *S. pneumoniae* is part of the normal bacterial flora in the throat and nose of infants and young children, where it does not cause disease. The bacterium is spread to people in close proximity through inhalation of respiratory droplets containing live bacteria that are produced when an infected person coughs or sneezes.

Epidemiological situation in 2012

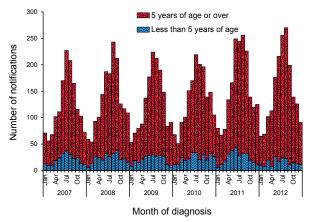
There were 1,822 notifications of IPD reported in 2012, representing a rate of 8.0 per 100,000 Compared with 2011, the national number of IPD notifications in 2012 decreased by 3.3% but was the 2nd highest reported in any year since the introduction of the universal pneumococcal conjugate vaccine program for young children in 2005 (Figure 41). The notification rate for IPD varied from 6.8 per 100,000 in Victoria to 30.6 per 100,000 in the Northern Territory.

The number of notifications in New South Wales and Queensland increased while they remained constant in the Australian Capital Territory, and all other jurisdictions reported fewer notifications. The largest change in IPD notification rates was in the Northern Territory where the rate (31 per 100,000) declined to levels similar to that seen prior to the 2011 serotype 1 outbreak (56 per 100,000).⁶⁰ The increase in notifications from Queensland, which commenced in 2011, continued with 348 notifications reported in 2012. Further, notifications in New South Wales increased by 9% from 529 notifications in 2011 to 579 in 2012.

Seasonality

Many respiratory diseases including IPD, are known to show distinct seasonality peaking during the winter months). The number of IPD cases in 2012 was greatest in the winter months, with the peak in August (n=270) (Figure 41).

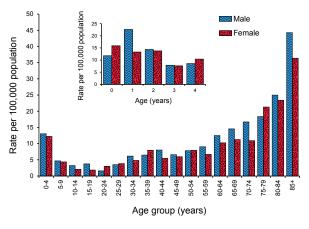
Figure 41: Notifications of invasive pneumococcal disease, Australia, 2012, by month of diagnosis and age group



Age and sex distribution

The age-specific notification rate for IPD in 2012 was trimodal, with the highest rates being in young children under the age of 5 years and older Australians (60 years or over) with a smaller peak in the 35–44 years age group (Figure 42).⁶¹ In older Australians, the highest notification rate was in those aged 85 years or over (39 per 100,000) while the highest rate in children aged less than 5 years was in those aged 1 year (18 per 100,000). In 2012, males accounted for 51% of all cases of IPD (Figure 42).

Figure 42: Notification rate for invasive pneumococcal disease, Australia, 2012, by age group and sex



Indigenous status

Completeness of Indigenous status reporting in 2012 was high, with 86% (n=1,564) of cases having known Indigenous status, of which 16% were reported as being Indigenous (n=244). In 2012, the notification rate for IPD in the Indigenous popu-

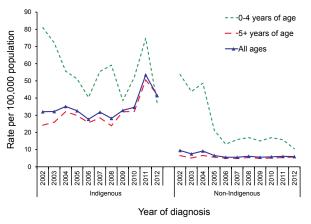
lation (41 per 100,000) was almost 7 times that for non-Indigenous people (6 per 100,000). In 2012, the notification rate for IPD among Indigenous children aged less than 5 years (37.1 cases per 100,000) remained almost 3-fold that of the general population (10.5 per 100,000) (Figure 43).

Vaccination

There are 4 pneumococcal vaccines available in Australia, each targeting multiple serotypes (Table 17). In Australia, pneumococcal vaccination is included on the NIP schedule and recommended for all infants, Australians aged 65 years or over, Aboriginal and Torres Strait Islander peoples aged 50 years or over and the medically at-risk.²⁰

There were several amendments to the NIP schedule in 2011 and 2012 with the most notable being the July 2011 replacement of the 7-valent pneumococcal conjugate vaccine (7vPCV) and the 10-valent pneumococcal conjugate vaccine (10vPCV) for all infants with the 13-valent pneumococcal conjugate vaccine (13vPCV) (Table 18).

Figure 43: Notification rate for invasive pneumococcal disease, Australia, 2002 to 2012, by Indigenous status and age group



 $2005-\mbox{Introduction}$ of universal childhood $7\nu\mbox{PCV}$ immunisation program.

July 2011 – The 13vPCV immunisation replaced the 7vPCV component in the universal childhood immunisation program.

Table 17: Streptococcus pneumoniae serotypes targeted by pneumococcal vaccines

Vaccine type	Serotypes targeted by the vaccine
7-valent pneumococcal conjugate vaccine (7vPCV)	4, 6B, 9V, 14, 18C, 19F and 23F
10-valent pneumococcal conjugate vaccine (10vPCV)	1, 4, 5, 6B, 7F, 9V, 14, 18C, 19F and 23F
13-valent pneumococcal conjugate vaccine (13vPCV)	1, 3, 4, 5, 6A, 6B, 7F, 9V, 14, 18C, 19A, 19F and 23F
23-valent pneumococcal polysaccharide vaccine (23vPPV)	1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19F, 19A, 20, 22F, 23F and 33F

Table 18: Amendments to the National Immunisation Program pneumococcal vaccination schedule for 2011 and 2012

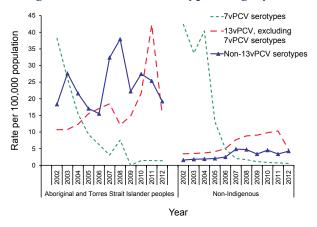
Vaccine type	National Immunisation Program pneumococcal vaccination schedule
7-valent pneumococcal conjugate vaccine (7vPCV)	From 2005 to July 2011, 7vPCV was funded nationally for all infants as a 3-dose primary vaccination schedule consisting of doses at 2, 4 and 6 months of age without a booster in the 2nd year of life.
10-valent pneumococcal conjugate vaccine (10vPCV)	From October 2009 to September 2011, 10vPCV replaced the use of the 7vPCV in all children aged <2 years in the Northern Territory.
13-valent pneumococcal	From July 2011, the 13vPCV replaced the 7vPCV for all infants.
conjugate vaccine (13vPCV)	From October 2011, the 13vPCV replaced the 10vPCV for infants in the Northern Territory.
	From October 2011 to September 2012, a single supplementary dose of 13vPCV for children aged 12–35 months who completed primary vaccination with either 7vPCV or 10vPCV was made available for 12 months.
	From October 2012, a booster dose of 13vPCV was made available for Aboriginal and Torres Strait Islander children at 12–18 months of age.
23-valent pneumococcal polysaccharide vaccine (23vPPV)	From October 2011, the 23vPPV booster dose for Aboriginal and Torres Strait Islander children aged 18–24 months living in the Northern Territory, South Australia, Queensland and Western Australia ceased.

More information on the current pneumococcal vaccination schedule in Australia can be found on the <u>Immunise Australia web site</u> (www.immunise. health.gov.au) and a detailed history of pneumococcal vaccination practices is available through the National Centre for Immunisation Research and Surveillance.⁶²

Serotype

Data on *S. pneumoniae* serotypes is important for understanding the effectiveness of vaccination programs. IPD serotypes were reported for 93%(n=1,690) of notified cases in 2012. The marked reduction in IPD due to serotypes targeted by the 7vPCV vaccine, seen in both Indigenous and non-Indigenous children aged less than 5 years has continued in 2012 (Figure 44). The 7vPCV serotypes accounted for only 6% (n=10) of IPD notifications where the serotype was known for children aged less than 5 years in 2012.

Figure 44: Notification rate of invasive pneumococcal disease in children aged less than 5 years, Australia, 2002 to 2012, by Indigenous status and serotype category



2001 – Introduction of 7vPCV immunisation for Aboriginal and Torres Strait Islander and medically at-risk children and 23vPPV booster for Aboriginal and Torres Strait Island children in the Northern Territory, Western Australia, South Australia and Queensland.

 $2005-Introduction of universal childhood 7vPCV immunisation program. \end{tabular}$

July 2011 – The 13vPCV vaccine replaced the 7vPCV component in the universal childhood immunisation program.

From 2008 to 2011, there was an increase in the incidence of IPD due to the 6 additional serotypes targeted by the 13vPCV (13v-non-7v) vaccine in children under 5 years of age. This indicates that the serotypes of circulating *S. pneumoniae* had been replaced. In 2012, and following the July 2011 introduction of 13vPPV to the NIP, this trend was

reversed with 13v-non-7v serotypes accounting for only 48% (n= 82) of IPD notifications compared with 68% (n=182) in 2011. For Aboriginal and Torres Strait Islander children, the most common 13v-non-7v serotype causing disease was due to serotype 1 (45% of 13v-non-7v serotypes), while in non-Indigenous children serotype 19A was the most common serotype reported (62%).

More detailed analyses of notification data can be found in the IPD annual reports published in CDI.⁶³

Measles

- Measles is no longer endemic in Australia, with no endemic measles for several years.
- Almost all cases of measles in Australia are either imported from overseas, or are related to transmission both directly and indirectly from an imported case.
- In 2012, there were 199 cases of measles, with 173 being associated with a large outbreak that originated from an imported case from Thailand.
- Over 80% of cases eligible for vaccination were either not vaccinated (43%) or their vaccination status could not be established (42%).

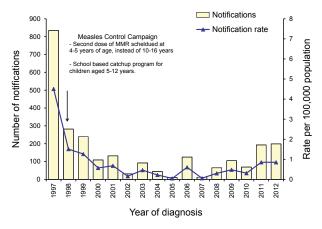
Measles is a highly infectious, acute viral illness spread by respiratory secretions, including aerosol transmission.⁶⁴ The incubation period is usually 10-14 days and it is infectious from around 4 days before and 4 days after the appearance of a characteristic rash. Initial symptoms last 2–4 days and are characterised by fever and malaise, followed by a cough, coryza and conjunctivitis. This is usually followed by a red blotchy rash, which typically begins on the face and then becomes generalised. Measles may be a severe disease with complications, which are more common in the chronically ill, children under 5 years of age and in adults over 20 years of age. Symptoms include otitis media, pneumonia, diarrhoea and acute encephalitis.^{65,66} Subacute sclerosing panencephalitis is a late, rare (approximately 1 in 100,000 cases) manifestation of measles caused by persistent infection and is always fatal.²⁰

In 2012, measles vaccine was available in the combined measles-mumps-rubella (MMR) vaccine and provided under the NIP schedule to children at 12 months and 4 years of age. Two doses of a measles containing vaccine are recommended for all non-immune persons born during or since 1966 and who are 18 months of age or over. The MMR vaccine induces long term immunity to measles virus in 95% of recipients after a single dose and 99% of recipients after the 2nd dose.²⁰

Epidemiological situation in 2012

In 2012, there were 199 notifications of measles. This represents a notification rate of 0.90 per 100,000, which is 2.2 times the mean of the previous 5 years. The number of cases in 2012 was similar to that in 2011 when 193 cases were reported (Figure 45).

Figure 45: Notifications and notification rate for measles, Australia, 1997 to 2012, by year of diagnosis



Geographical distribution

In 2012, cases of measles occurred in all states and territories, except the Australian Capital Territory and Tasmania (Table 4). The majority of these and the largest increase compared with 2011, occurred in New South Wales (n=170) (Figure 46). Over 86% of cases were associated with a large outbreak that occurred in Western and South Western Sydney and was linked to an imported case from Thailand.

Seasonality

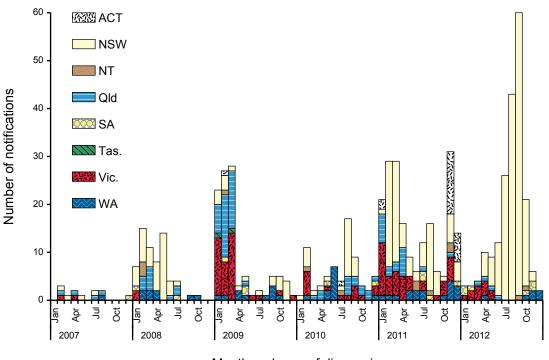
In Australia, a seasonal pattern is no longer evident as the virus is not endemic (Figure 46). In temperate climates where measles transmission remains endemic, the majority of cases occur in late winter to early spring.¹⁹

Age and sex distribution

The male to female ratio was 1.1:1 in 2012, however there was a wide variation in this ratio across the age groups (Figure 47).

In 2012, the age of measles cases ranged from 0-61 years with a median age of 15 years. Whilst notification rates increased across all age groups compared with previous years there were a higher proportion of cases aged less than 10 years of age (Figure 48). The highest age specific rates occurred

Figure 46: Notifications of measles, Australia, 2007 to 2012, by month and year of diagnosis and state or territory



Month and year of diagnosis

in the less than 1 year age group at 13 per 100,000 (n=39), with rates also high in the 1–4 years and 10–19 years age groups (1.8 per 100,000 in each).

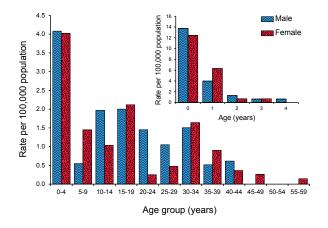
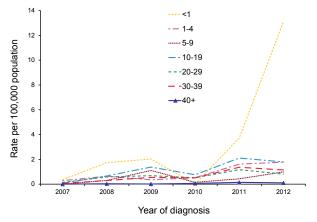


Figure 47: Notification rate for measles, Australia, 2012, by age group and sex





The notification rates for measles remained below 2.5 per 100,000 for all age groups from 2007 to 2012. The exception to this was the under 1 year age group in 2011 and 2012 (Figure 48).

There were 39 cases aged less than 1 year and therefore too young to have received measles vaccine. The majority of these cases (92%) were aged between 6 and 12 months highlighting the loss of maternal antibody.⁶⁷

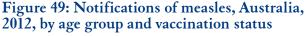
Indigenous status

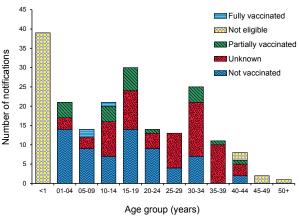
Indigenous status was reported for 98% of cases during 2012 (n=194). Of these 6% (n=12) were reported

as being Indigenous. All of these were reported from New South Wales, where Indigenous Australians had a notification rate 3 times higher than non-Indigenous people in that state (6.96 compared with 2.22 per 100,000 respectively).⁶⁸

Vaccination status

Of the 199 cases notified in 2012, 78% (n=155) were born after 1967 (or 1969 for New South Wales) and were over 12 months of age. This cohort was eligible for at least 1 dose of a publicly funded measles vaccine either during childhood or as a result of later measles vaccination catch up campaigns. Over 80% of vaccine eligible cases were either not vaccinated (43%, n=68) or of unknown vaccination status (42%, n=65). Of the remaining 15% (n=24) who were vaccinated, only three had received the full course of 2 doses of a measles vaccine (Figure 49).



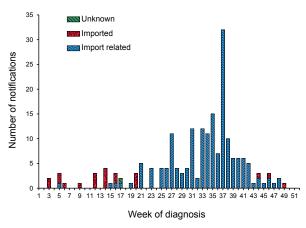


A high proportion of unvaccinated cases (48%, n=21) occurred in adolescents in the 10-19 years age group or young adults 20-29 years of age. Twenty-five cases occurred in those born between 1978 and 1982 (30-34 years age group) of which three were reported as receiving 1 dose of vaccine and the remainder were either not vaccinated (n=7)or were of unknown vaccination status (n=15). This cohort has previously been identified as being susceptible to measles virus infection as the second childhood measles vaccine now recommended at 18 months was not available to them and they were not targeted as part of the 1998 Measles Control Campaign.⁶⁹ In 2012, three of the cases were born before 1966, a cohort considered to have high levels of natural immunity. All three of these cases were either unvaccinated or of unknown vaccination status (Figure 49).⁷⁰

Source of infection and outbreaks

All but 1 case in 2012 were imported (10%, n=21) or linked to an imported case (89%, n=177). For the locally acquired case an epidemiological or virological link to an imported case could not be established (Figure 50).

Figure 50: Notifications of measles, Australia, 2012, by week of diagnosis and source of infection



Imported cases were either from the WHO South East Asia Region (81%), the majority of which were from Thailand (n=12) or the WHO Eastern Mediterranean Region (10%). A single case was reported from Uganda.

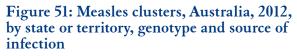
There were 6 clusters of two or more epidemiologically linked cases in 2012, all of which were import-related. In all except 1 cluster, transmission was interrupted quickly resulting in only 2 cases for each of these clusters. The largest outbreak of measles occurred predominately in Western and South Western Sydney. The outbreak comprised 173 cases in total, including 2 associated cases in the Northern Territory and three in South Australia. This outbreak began in April 2012 with an imported case from Thailand and peaked in September, with the last case reporting onset of symptoms on 29 November 2012. This outbreak included 24 generations of spread, lasting 33 weeks between the onset of symptoms of the first and last cases. NNDSS data indicate that of the 173 cases, 59% (n=102) were not vaccinated, 15% (n=26) had received 1 dose of a measles containing vaccine and 2 cases had received 2 doses, with the remaining cases being of unknown vaccination status. The median age of outbreak cases was 14 years of age (range 0–61 years).

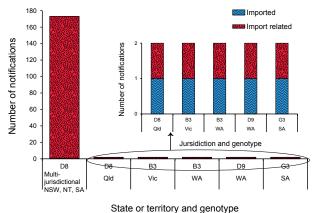
Although there were several separate chains of transmission identified during the outbreak,

all cases in each of the geographic areas in New South Wales had direct epidemiological links to the larger outbreak. In addition, there were no new importations identified during the outbreak period and the genetic sequences of measles virus isolates from cases in these clusters were identical, thus establishing the link to the larger outbreak.

Genotype

Genotyping data were available for all 6 clusters, accounting for 92% (n=183) of cases in 2012. Genotypes B3D8 and D9 were identified among the clusters across Australia (Figure 51). The largely New South Wales based outbreak was due to measles virus genotype D8.





Discussion

The fluctuating nature of measles rates over time can be attributed to sporadic imported cases that occasionally result in clusters of locally acquired infection among susceptible contacts.

Evidence suggests that endemic measles was eliminated from Australia in 2005 and possibly earlier.⁷¹ Based on the WHO definition, Australia has continued to maintain this status over time. In 2012, none of the outbreaks persisted for more than 12 months with the longest lasting 33 weeks. Additionally, there was no evidence that a single genotype was continuously circulating for 12 months or more. Ongoing evidence of high population immunity was demonstrated by the rapid cessation of the majority of the outbreaks. Only one of the outbreaks in 2012 involved more than 3 generations of transmission, with there being 5–7 weeks between the onset of disease in the first case and the last cases.⁷² With the exception of the single case for which the source of infection could not be established, all of the 2012 cases were associated with an index case that was imported from overseas.

Mumps

- 200 cases of mumps were notified in 2012.
- Following a peak in the rate of mumps notification in 2007, notifications have been less than 1 per 100,000 since 2009.

Mumps is an acute viral illness with an incubation period of 12–25 days. Transmission is via respiratory secretions, including aerosol transmission, or by direct contact with saliva. Asymptomatic infections occur in one-third of cases. Symptomatic disease ranges from mild upper respiratory tract infections to systemic involvement. 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.⁷³ Mumps encephalitis has been estimated to occur in 1 to 2 per 10,000 cases, with a case a fatality rate of around 1%.

In 2012, mumps vaccine was included in the combined MMR and provided under the NIP schedule at 12 months and 4 years of age. Two doses of a mumps containing vaccine are recommended for all non-immune persons born during or since 1966 and who are 18 months of age or over.

The mumps vaccine was first funded on the NIP schedule for infants of 12 months of age in 1982. Those born since that time are eligible for 2 doses of a mumps vaccine.⁷⁴

Epidemiological situation in 2012

In 2012, there were 200 notifications of mumps; a notification rate of 0.88 per 100,000. This represents a 28% increase compared with the 156 cases reported in 2011. Since 2009, mumps notifications have declined (Figure 52).

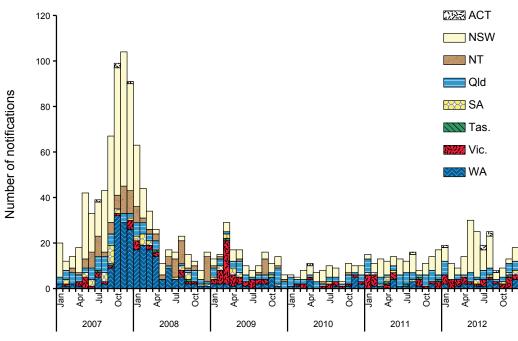
Geographical distribution

Notifications were received from all states and territories except the Northern Territory. Notification rates were highest in the Australian Capital Territory (1.6 per 100,000) followed by New South Wales (1.4 per 100,000).

Age and sex distribution

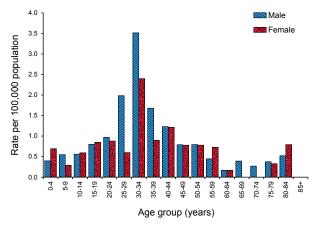
In 2012, the male to female ratio was 1.3:1 with some variation between age groups. The highest rates for both males and females occurred in the 30–34 years age group at 3.5 and 2.4 per 100,000 respectively. The male specific rates were highest in the 25–39 years age group (Figure 53).

Figure 52: Notifications of mumps, Australia, 2007 to 2012, by month and year of diagnosis and state or territory



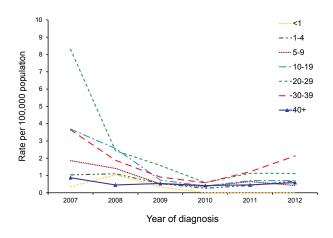
Month and year of diagnosis

Figure 53: Notification rate for mumps, Australia, 2012, by age group and sex



Cases of mumps were notified in most age groups, the median age at diagnosis being 30 years (range 1–84 years). The most notable increase in age group specific rates occurred among adults in the 20–39 years age group, although their overall rates in 2012 remained low compared with the peak experienced in this age group in 2007 to 2008 (Figure 54).

Figure 54: Notification rate for mumps, Australia, 2007 to 2012, by age group



Indigenous status

Indigenous status was reported for 60% (n=120) of mumps cases in 2012, which was relatively consistent with the level of completeness over the previous 5-year period (mean 65%, range 51%-77%). Of these, 1 case was reported as being Indigenous.

Vaccination status

Of the 200 notified cases in 2012, 45% (n=89) were born after 1980 and were more than 12 months of age. This cohort was eligible for at least 1 dose of a publicly funded mumps-containing vaccine. In 2012, 64% (n=57) of cases were of unknown vaccination status and a further 20% (n=18) were unvaccinated. Of the remaining 16% (n= 14), 6 cases were fully vaccinated having received 2 doses of a mumps vaccine and 4 cases were partially vaccinated with 1 dose of a mumps vaccine. Four cases were reported as having been vaccinated with no information on the number of doses provided.

Discussion

The mumps component of the MMR vaccine is considered to be the least effective of the 3 components. This is based on outbreak investigations and post marketing studies that report that 1 dose of vaccine provides 60%–90% protection, which varies depending on the virus strain used in the vaccine.75-77 Outbreaks have been reported among 2 dose recipients, particularly young adults who received their vaccines more than 10 years previously, suggesting that 2 doses may not be sufficient to prevent outbreaks in this cohort.78,79 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.⁸⁰

Pertussis

- Pertussis is the least well controlled of all childhood VPDs and remains highly prevalent in Australia.
- 24,069 cases of pertussis were reported in 2012, representing a notification rate of 106 per 100,000 population.
- 3,160 cases were reported in children less than 5 years of age.

Pertussis, commonly known as whooping cough, is a highly infectious respiratory disease caused by *Bordetella pertussis* and is spread by respiratory droplets. The characteristic paroxysmal cough with inspiratory whoop seen among unvaccinated children is less common in individuals who have some acquired immunity from vaccination or previous infection.⁸¹ Most deaths occur in unvaccinated infants under 6 months of age. Complications include pneumonia, atelectasis, seizures, encephalopathy, and hernias, with pneumonia as the most common cause of death.¹⁹

The NIP schedule in 2012 included a primary course of 3 doses of vaccine at 2, 4, and 6 months of age, with additional booster doses at 4 years and between 10 and 15 years of age, the latter being delivered through school based programs.²⁰

Epidemiological situation in 2012

In 2012, there were 24,069 notifications of pertussis including 2 deaths in infants aged less than 8 weeks who were too young to be vaccinated. Although declining, there continued to be large numbers of cases associated with the Australia-wide epidemic that began in mid-2008 and peaked in early 2011 (Figure 55). While pertussis remains endemic in Australia with a cyclical pattern of epidemic activity occurring approximately every 3–4 years, this most recent epidemic has been much larger and more prolonged than previous outbreaks (Figure 56). In 2012, jurisdiction specific rates varied considerably with the Australian Capital Territory (225 per 100,000), Queensland (201 per 100,000) and New South Wales (181 per 100,000), all having notification rates higher than the national notification rate (173 per 100,000) (Figure 57). Since 2008, the timing of epidemic activity has varied across all jurisdictions. In all states and territories, except Tasmania, notification rates decreased in 2012 compared with 2011. In Tasmania, the notification rate increased more than 3-fold, from 69 per 100,000 in 2011 to 249 per 100,000 in 2012 (Figure 57). Between 2008 and 2012, multiple out-

Figure 55: Notifications and notification rates for pertussis, Australia, 1993 to 2012

Figure 57: Notification rates for pertussis, 2007 to 2012, by state or territory

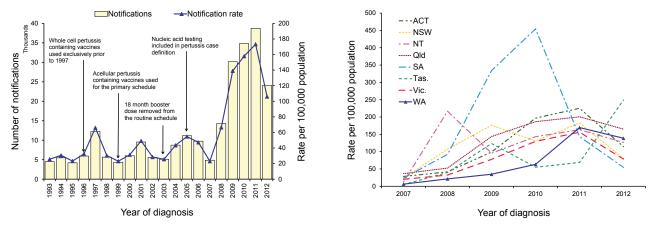
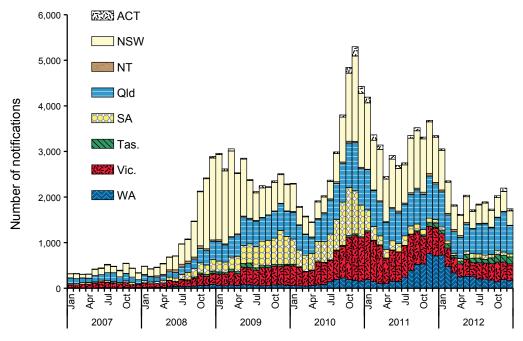


Figure 56: Notifications of pertussis, Australia, 2007 to 2012, by month and year of diagnosis and and state or territory



Month and year of diagnosis

breaks, varying by geographical location, size and timing across jurisdictions, were the main cause of the varying rates for this period.

Age and sex distribution

Following the peak in pertussis notifications in 2011, notifications decreased across all age groups in 2012. The highest notification rates were in children less than 15 years of age (236 per 100,000), accounting for 42% of all notifications. The highest age specific notification rate occurred in the 5–9 years age group (Figure 58). This was consistent with the overall trend of higher notification rates among children during the recent epidemic period, but differs from the trends observed prior to the epidemic in which children had much lower rates relative to adolescents and adults.

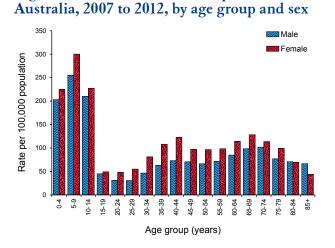


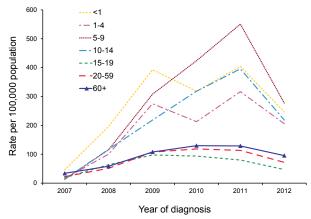
Figure 58: Notification rate for pertussis,

In 2012, females accounted for 56% (n=13,497) of cases, resulting in a male to female ratio of 1.2:1. Females had higher rates across all age groups, except adults aged 80 years or over (Figure 59). The highest notification rate for both males and females occurred in the 5–9 years age group (255 and 300 per 100,000 respectively) (Figure 59). In the 25–44 years age groups notification rates in females were more than 2 times that of males, which was likely due to the generally higher health seeking behaviour among adult females compared with males.⁸²

Vaccination status

In order to determine the vaccination status of cases, public health follow up is required. States and territories prioritise case follow up to those less than 5 years of age.⁸³





In response to the ongoing epidemic in 2012, some infants were provided their first vaccination at 6 weeks of age and young children their fourth from 3.5 years. During 2012, those aged less than 5 years and eligible for a pertussis-containing vaccine, accounted for 13% of all notified cases and information about vaccination status was available for 91% of these cases.

Of the children eligible to have received their full primary course, 52% (n=1,172) had received their scheduled 3 vaccinations and 37% (n=164) had received their full scheduled course of 4 doses (Table 19).

During the recent epidemic period between 2008 and 2012, there were 10 pertussis associated deaths reported to the NNDSS all of whom were 8 weeks of age or less. Two of these cases had received 1 dose of a pertussis containing vaccine.

Discussion

In Australia, epidemics of pertussis have historically occurred at regular intervals of approximately 4 years on a background of endemic circulation.⁸⁴ The timing of the recent multi-year epidemic was not uniform across the country with the Australian Capital Territory, Queensland, New South Wales, Victoria and Western Australia all experiencing their highest notification rates in 2011 while the Northern Territory, South Australia and Tasmania experienced peak levels of pertussis in 2008, 2010 and 2012 respectively.

The most important factors that have likely contributed to the baseline increase include more sensitive diagnostic techniques,^{85,86} increased awareness and testing for pertussis in adolescents and adults,

		Number of vaccine doses					
Age group	0	1	2	3	4	Unknown	Total
Less than 6 weeks of age (not eligible for vaccination)	47					42	89
6 weeks to <4 months (eligible for 1 dose of vaccine)	51	141	4			35	234
4 to < 6 months (eligible for 2 doses of vaccine)	21	66	41			8	136
6 months to < 4 years (eligible for 3 doses of vaccine)	205	468	259	1,150	22	154	2,255
4 to 5 years (eligible for 4 doses of vaccine)	51	75	44	74	164	38	446
Total	375	750	348	1,224	186	277	3,160

Table 19: Notifications of pertussis in persons aged 0–5 years, Australia, 2012, by age group and number of doses of vaccine*

* Excludes 6 notifications for whom age in months could not be determined.

reduced effectiveness of the newer acellular vaccines, $^{87-89}$ and the removal of the 18-month booster dose from the routine schedule in 2003. 90

Strategies to reduce pertussis infection in young children, particularly among those less than 6 months of age continued in 2012. States and territories continued to provide ongoing public awareness campaigns including extended funding during 2012 for a 'cocooning' program giving booster vaccinations to pregnant women, parents and carers of infants. The Australian Technical Advisory Group on Immunisation also recommend bringing forward the 1st dose of the pertussis containing vaccine from 8 weeks to 6 weeks and scheduling the 5th (adolescent booster) dose at 11–13 years of age to better protect siblings, especially newborns, in response to outbreak settings.⁹¹

Poliomyelitis

- There were no cases of poliomyelitis identified in Australia in 2012.
- Australia was certified as having eradicated Indigenous poliovirus by the WHO in 2000.

Poliomyelitis is a highly infectious disease caused by gastrointestinal infection with poliovirus. Transmission occurs primarily from person to person via the faecal-oral route. In most cases, poliovirus infection is not symptomatic. However, in less than 1% of cases the virus may invade the nervous system and cause acute flaccid paralysis (AFP).¹⁹

Vaccines formulated with inactivated poliovirus, are available in combination with diphtheria toxin, tetanus and other antigens. The NIP schedule in

2012 recommended a primary course of 3 doses at 2, 4, and 6 months of age, with additional booster doses at 4 years and between 10 and 15 years, delivered through school based programs.²⁰

In 2012, there were no notifications of poliomyelitis. The last case of poliomyelitis was an imported case in 2007. There has not been a case caused by a locally acquired wild poliovirus in Australia since 1972.

Australia, along with the WHOs Western Pacific Region, remains poliomyelitis free. Clinical and laboratory investigation is conducted for all cases in patients 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 Australia has achieved in all years since 2008. More details can be found in the annual reports of the Australian National Enterovirus Reference Laboratory published in CDI.⁹²

Rubella

- Rubella is now a rare disease in Australia.
- Since 2003, the rubella notification rate has been less than 0.3 per 100,000.
- In 2012, 36 cases of rubella were reported.
- Almost a quarter of cases were reported as having been acquired overseas, primarily in Asia.

Rubella is generally a mild self-limiting viral disease. It is spread from person to person through contact with respiratory secretions, including aerosol transmission. Clinically, rubella can be difficult to distinguish from other diseases that cause a fever and 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 manifest as foetal malformation or result in the death of the foetus.¹⁹

The main aim of immunisation for rubella is to prevent cases of CRS.⁹³ Rubella vaccine is included in the combined MMR vaccine. In 2012, it was provided under the NIP schedule at 12 months and 4 years of age.²⁰

Epidemiological situation in 2012

In 2012, 36 notifications of rubella were reported, representing a notification rate of 0.16 per 100,000 and a decrease compared with 2011 (n=58) and the 5-year mean. Cases were reported from all jurisdictions except the Northern Territory in 2012 (Table 4) (Figure 60). There was 1 case of CRS reported in 2012. Indigenous status was recorded for all cases; and none were reported as being Indigenous.

Age and sex distribution

The male to female ratio in 2012 was 1.1:1 comprising 19 males and 17 females. The highest rates for females occurred in the 25–29 years age group (0.96 per 100,000) and for males in the 30–34 years age group (0.88 per 100,000) (Figure 61).

The majority of cases (69%) continued to occur among adults aged 20–39 years, with a median age of 31 years (Figure 62).

Figure 61: Notification rate for rubella, Australia, 2012, by age group and sex

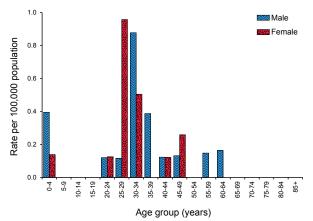
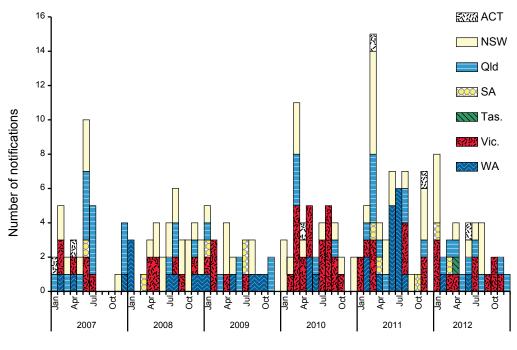
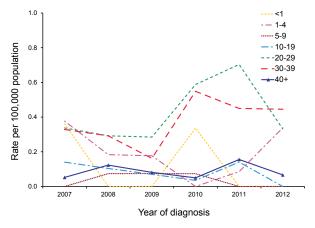


Figure 60: Notifications of rubella, Australia, 2007 to 2012, by month of diagnosis



Month and year of diagnosis

Figure 62: Notification rate for rubella, Australia, 2007 to 2012, by age group



Vaccination status

Of the 36 cases notified in 2012, 64% (n=23) were of unknown vaccination status and 19% (n=7) were reported as being unvaccinated. The remaining 6 cases were reported as having been vaccinated. Of these five were partially vaccinated having received 1 dose of a rubella-containing vaccine and 1 case had no vaccine dose information provided.

The vaccination status of those cases in women of child-bearing age and in adult men was unknown in most cases. Of the 11 female cases 15–44 years of age and the 16 adult males, four were reported as having been vaccinated, two were partially vaccinated and for two the number of vaccine doses was not reported.

Source of infections

In 2012, almost a quarter of rubella virus infections (n=8) were imported from overseas. There were three each from India and Indonesia, and one each from Germany and South East Asia. A 3rd of cases were reported as having been acquired in Australia. The place of acquisition was not reported for the remaining 44% of cases.

Discussion

Goals for the elimination of rubella and CRS have been set by a number of World Health Organization regions. Elimination has been declared by the Pan American Health Organization. The WHO Western Pacific Region, of which Australia is a member, has set goals for increased rubella and CRS elimination efforts, including the strengthening of immunisation and surveillance activities to confirm the absence of endemic strains.

Evidence suggests that rubella is well controlled in Australia. Measures implemented in the late 1990s

under the Measles Control Campaign, which included lowering the age for the 2nd dose of the combined MMR vaccine to 4 years and a catch-up program, resulted in high levels of vaccine coverage and sustained low incidence of rubella disease since that time. Now almost a quarter of infections are imported from overseas. Young men, historically a more susceptible cohort due to the delayed introduction of universal vaccination, no longer appear to be at greater risk of infection compared with females. However, the majority of cases, although small, continue to occur among adults of child-bearing age.

Tetanus

- Cases of tetanus are uncommon in Australia.
- Cases generally occur in older unvaccinated people or in those who have not received a booster dose in the last 10 years.
- 7 cases of tetanus were notified in 2012, including 2 reported deaths.

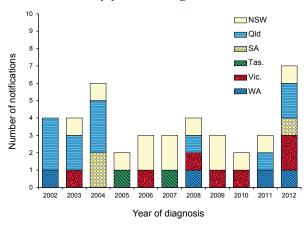
Tetanus is an acute, often fatal, disease caused by the toxin produced by the bacterium *Clostridium tetani*. Tetanus spores usually enter the body through contamination of a wound with manured soil.¹⁹ The neurotoxin acts on the central nervous system to cause muscle rigidity with painful spasms. Generalised tetanus, the most common form of the disease, is characterised by increased muscle tone and generalised spasms. The disease usually occurs after an incubation period of 3 to 21 days (ranging from 1 day to several months), with a median time of onset at 10 days post injury.

Tetanus vaccination stimulates the production of antitoxin, which protects against the toxin produced by the organism. Tetanus toxoid is available in combination with diphtheria and other antigens. The NIP schedule in 2012 recommends a primary course of 3 doses at 2, 4, and 6 months of age, with additional booster doses at 4 years and between 10 and 15 years delivered through school based programs. Booster doses are recommended for all adults at the age of 50 years who have not received a booster dose in the previous 10 years.

Epidemiological situation in 2012

In 2012, there were 7 notifications of tetanus, which was consistent with the low numbers seen in recent years. As laboratory confirmation of tetanus is usually not possible, notification of cases relies on reports from clinicians, resulting in the potential for under-reporting.⁴⁵ Indigenous status was complete for 6 of the 7 cases. None of these cases were reported as being Aboriginal or Torres Strait Islander (Figure 63).

Figure 63: Notifications of tetanus, Australia, 2002 to 2012, by year of diagnosis



Age and sex distribution and vaccination status

There were 2 male and 5 female cases reported in 2012. Two cases were in the 20–29 years age group and the remaining 5 cases were over 75 years of age. One case had received 1 dose of a tetanus containing vaccine, the remaining 6 cases were not vaccinated or were of unknown vaccination status. Two deaths occurred in unvaccinated adults over 75 years of age.

Discussion

Tetanus in Australia is rare, occurring primarily in older adults who have never been vaccinated or were vaccinated in the remote past. A high level of diagnostic awareness is important in the elderly, as most deaths occur in those over 70 years of age, especially women, particularly given that the infection may be associated with a minor injury.²⁰

Varicella zoster virus infections

- 14,898 cases of varicella zoster virus infection were notified in 2012, representing an increase of 7% from 2011.
- 57% of cases were reported as being unspecified varicella zoster infection, 30% of cases were reported as shingles and 13% as chickenpox.

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 (herpes zoster), which has a 20%–30% risk of occurring following reactivation of the latent virus. Shingles occurs more frequently among older adults (most commonly after 50 years of age) and in immunocompromised people.¹⁹ In 2006, CDNA agreed to the 3 categories of VZV infection being nationally notifiable: chickenpox, shingles and varicella infection unspecified. By 2009, all jurisdictions were notifying VZV to the NNDSS with the exception of New South Wales, where VZV is not notifiable.

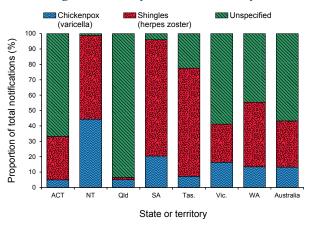
The ability to categorise a VZV infection as chickenpox or shingles depends largely on clinical evidence. Due to the absence of information on clinical presentation for many cases, the majority of VZV notifications nationally are reported as being unspecified.

An analysis of South Australian VZV infections, where the majority of cases are followed up to establish the clinical presentation, highlights that since 2006 notifications of clinically confirmed chickenpox have remained relatively stable overall with declining trends among those births cohorts targeted by vaccination. Over this period notifications of shingles have increased.

Epidemiological situation in 2012

In 2012, there were 14,898 VZV notifications from the 7 reporting jurisdictions. This was 7% higher than the number notified in 2011 (n=13,808). This upward trend in the total number of notifications has been observed since 2009 and is most likely due to increased awareness of the requirement to notify and diagnostic laboratory testing by health care practitioners. Of the total VZV notifications in 2012, 57% (n=8,453) of cases were reported as unspecified varicella infection, 30% (n=4,481) as shingles and 13% (n=1,964) as chickenpox (Figure 64).

Figure 64: Proportion of notified cases of varicella zoster virus unspecified, chickenpox and shingles, 2012, by state or territory*



* Excluding New South Wales.

Chickenpox

- The primary purpose of the vaccination is to prevent deaths, reduce the severity of disease and in the longer term reduce rates of VZV reactivation as shingles.
- 1,964 cases of chickenpox were notified in 2012, representing a 6% decrease from 2011.

Chickenpox is an illness due to a highly contagious virus, varicella zoster, which is spread by respiratory secretions, including aerosol transmission, or from the vesicle fluid of skin lesions from a patient with chickenpox or shingles. Chickenpox is usually a mild disease of childhood, but complications occur in approximately 1% of cases. It is more severe in adults and in persons of any age who are immunocompromised, in whom complications, disseminated disease, and fatal illness are more likely to occur.²⁰

Routine use of a varicella-containing vaccine in children was first recommended in Australia in 2003. In November 2005 the vaccine was funded under the NIP for all children at 18 months of age, with a school based catch-up program for children 10–13 years of age with no history of disease or previous vaccination.

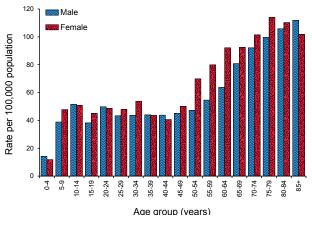
Epidemiological situation in 2012

In 2012, there were 1,964 cases of chickenpox reported; a notification rate of 13 per 100,000 and a 6% decrease in the number of notifications compared with 2011 (n=2,099). The highest notification rate, 63 per 100,000, was reported from the Northern Territory (n=149), followed by South Australia, 29 per 100,000 (n=476), reflecting the higher case ascertainment in these jurisdictions.

Age and sex distribution

The male to female ratio in 2012 was 1:1.1, with a slight variation particularly in the older age groups where reported case numbers were small. Sixty-one per cent of notified chickenpox cases (n=1,185) occurred in children aged less than 10 years. The 5–9 years age group had the highest notification rate for both sexes (71 per 100,000 for males and 67 per 100,000 for females) (Figure 65). Although higher rates among children compared with adults is expected for chickenpox, the distribution of cases by age group also reflects general jurisdictional practice of limiting follow up of laboratory notified cases of younger children.

Figure 65: Notification rate for chickenpox, Australia,* 2012, by age group and sex



Excluding New South Wales.

Vaccination status

In 2012, the oldest cohort of children eligible for varicella vaccination at 18 months of age under the NIP would now be 8 years of age. The analysis of vaccination status is therefore restricted to this cohort. Vaccination status information was available for 54% (n=387) of cases in this cohort with 87% having been vaccinated (n=337) and 13% not vaccinated (n=50).

Shingles

• 4,481 cases of shingles were notified in 2012, less than 1% variation from 2011.

Shingles occurs most commonly with increasing age, impaired immunity, and a history of chickenpox in the first year of life.²⁰ Reactivation of VZV resulting in shingles is thought to be due to a decline in cellular immunity to the virus. Shingles typically presents as a unilateral vesicular rash localised 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 selflimiting disease. However, complications develop in approximately 30% of cases, the most common of which is chronic severe neuropathic pain or post herpetic neuralgia.¹⁹

Zostavax is a live attenuated viral vaccine formulated from the same VZV vaccine strain as currently licensed varicella vaccines. However, it is of a higher potency that is designed to elicit a boost in the immune response for the prevention of VZV reactivation to cause shingles. A single dose of zoster vaccine is recommended, but not presently funded through the NIP, for adults aged 60 years or over who have not previously received a dose of zoster vaccine.²⁰

Epidemiological situation in 2012

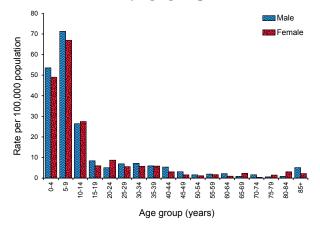
In 2012, there were 4,481 cases of shingles notified; a notification rate of 29 per 100,000 and similar to 2011. The highest rates of shingles occurred in South Australia, (106 per 100,000, n=1,761) followed by the Northern Territory, (78 per 100,000, n=183). The high rates in these jurisdictions likely reflect their higher levels of case ascertainment compared with other jurisdictions.

Age and sex distribution

The notification rate was lower in males at 26 per 100,000 compared with females at 33 per 100,000, representing a male to female ratio of 0.8:1.

As expected, rates increased with age with the highest rates in the 85 years or over age group for both males and females, at 67 per 100,000 and 82 per 100,000 respectively (Figure 66).

Figure 66: Notification rate for shingles, Australia,* 2012, by age group and sex



* Excluding New South Wales.

Discussion

It is estimated that 150,000 new cases of shingles occur each year in Australia with the majority of cases in adults over 50 years of age.^{94–96} Analysis of the South Australian data, where the majority of cases have been followed up to establish clinical diagnosis, shows an increase in shingles notifications since 2006. As noted for chickenpox, the increasing trend in shingles incidence, also observed in several other settings, is likely due to multiple factors including changes in health care seeking behaviour, clinical practice, and awareness of reporting requirements, as well as an ageing population.

Varicella zoster virus (unspecified)

• 8,453 cases of varicella zoster virus (unspecified) were notified in 2012, representing an increase of 9% from 2011.

Notifications of unspecified VZV are laboratory specimens that are positive for VZV but do not have the clinical diagnosis available to distinguish between chickenpox and shingles.

Epidemiological situation in 2012

In 2012, there were 8,453 cases of unspecified VZV infections reported, representing a notification rate of 55 per 100,000 and a 10% increase in notifications compared with 2011 (n = 7,691).

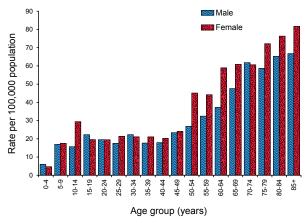
Geographical distribution

The highest notification rate for unspecified VZV was from Queensland at 97 per 100,000 (n=4,414) due to the mainly laboratory based notification of VZV in that state (Table 5). VZV unspecified rates should be interpreted with caution as they are dependent on the individual jurisdictional practice of following up laboratory notifications to establish clinical presentation.

Age and sex distribution

The male to female ratio in the unspecified VZV notifications was 0.8:1. Females have an overall higher notification rate (59 cases per 100,000) compared with males (50 per 100,000), which is consistent across most age groups. The highest age group specific notification rates occurred in the 75–79 years age group for females (114 per





* Excluding New South Wales.

100,000) and in the 85 years or over age group for males (112 per 100,000). The lowest age group specific notification rates were in the 0-4 years age group for both males and females. This was likely reflecting increased jurisdictional follow up to determine clinical presentation in children of this age (Figure 67).

Vectorborne diseases

Vectorborne diseases are infections transmitted by arthropods such as such as mosquitoes and ticks. A vectorborne disease may involve a simple transfer via the arthropod, or may involve replication of the disease-causing agent in the vector.¹⁹ Vectorborne diseases of public health importance in Australia listed in this chapter are: arbovirus 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 infection, plague and certain viral haemorrhagic fevers are listed under quarantinable diseases. The National Arbovirus and Malaria Advisory Committee (NAMAC) provide 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.⁹⁷

Alphaviruses

Viruses in the genus Alphavirus that are notifiable in Australia are BFV and RRV. These viruses are unique to the Australasian region.⁹⁸ Infection can cause a clinical illness, which is characterised by fever, rash and polyarthritis. The viruses are transmitted by numerous species of mosquito that breed in diverse environments.⁹⁹ The alphavirus chikungunya is not presently nationally notifiable, and thus not included in this annual report. However, 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.^{97,100}

The national case definitions for RRV and BFV require only a single IgM positive test to one virus, in the absence of IgM to the other.¹⁰¹ False positive IgM diagnoses for BFV in particular are a known issue, thus it is unclear what proportion of notifications represent true cases. There was a large

increase in notifications of BFV nationally from October 2012, which was likely to have been due to false positive notifications.

Barmah Forest virus infection

- There was a sharp increase in notifications from October 2012 due to false positive diagnoses.
- BFV was most frequently notified among middle aged to older adults.
- Queensland accounted for more than half of all notifications.

Epidemiological situation in 2012

In 2012, there were 1,722 notifications of BFV infection, equating to a rate of 7.6 per 100,000 population. This compares with a 5-year mean of 1,718 notifications and a 5-year mean rate of 7.9 per 100,000. The number of notifications of Barmah Forest virus increased sharply from October 2012 (Figure 68). This increase continued into late 2013 and beyond for some jurisdictions. The increase was considered likely to have been due to a high rate of false positive IgM test results from the use of a commercial test kit in private laboratories, and resulted in a recall of the affected kits in September 2013.¹⁰²

Seasonality and place of acquisition

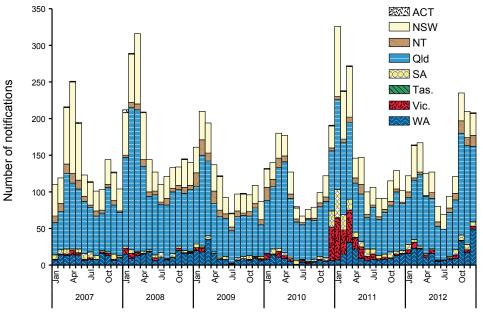
The seasonality of BFV notifications is less marked than for RRV, and a high proportion of interseasonal notifications are thought to be due to false positive diagnoses. The peak notifications of BFV between 2007 and 2012 was between January and April, and 45% of cases were diagnosed during these months (compared with 55% for RRV). The increase from October 2012 was earlier than the expected seasonal increase.

More than half of all BFV notifications in 2012 were from Queensland (57%) and rates were highest in the Northern Territory (37.0 per 100,000) and Queensland (21.5 per 100,000).

Age and sex distribution

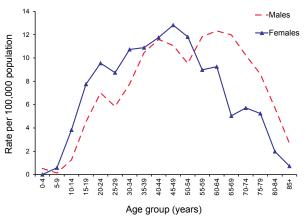
BFV was most frequently reported in middle aged adults (median 46 years, range 0–86 years). Age and sex specific rates were highest among males aged 45–49 years females aged 60–64 years (Figure 69).

Figure 68: Notifications of Barmah Forest virus infection, Australia, 2007 to 2012, by month and year and state or territory



Month and year

Figure 69: Notification rates for Barmah Forest virus infection, Australia, 2012, by age group and sex



Ross River virus infection

- Notification rates in 2012 were similar to the 5-year mean.
- RRV infections were mostly frequently notified in adults aged in their 30s or middle aged.
- Queensland accounted for nearly half of all cases in 2012.

Epidemiological situation in 2012

In 2012, there were 4,683 notifications of RRV; a rate of 20.6 per 100,000. This compares with a 5-year mean of 4,953 notifications and a 5-year mean rate of 22.8 per 100,000.

Seasonality

The peak in notifications for RRV from 2007 to 2012 occurred between January and April, and 55% of cases were diagnosed during these months (Figure 70).

Between 2007 and 2012, nearly half of all RRV infections were from Queensland (42% of all cases, 42.6 cases per 100,000), but population rates were highest in the Northern Territory (96.5 per 100,000).

Age and sex distribution

RRV was most frequently reported in adults aged in their 30s or 40s (median 42 years, range 0–85 years). Age specific rates were highest among females in the 40–44 years age group and for males in the 35–39 years age group (Figure 71).

Flaviviruses

In Australia, flavivirus infections of particular public health importance are DENV, KUNV,

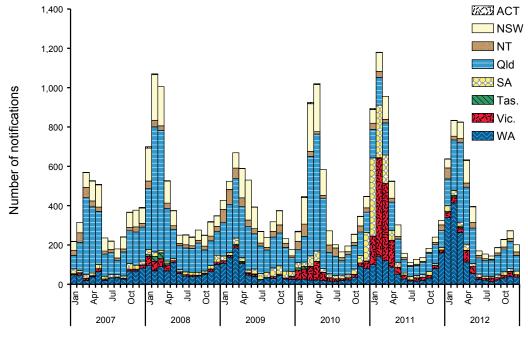
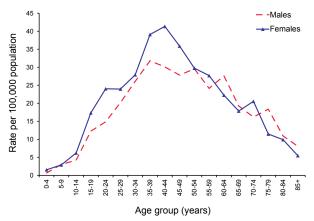


Figure 70: Notifications of Ross River virus infection, Australia, 2006 to 2012, by month and year and state or territory

Month and year

Figure 71: Notification rates for Ross River virus infection, Australia, 2012, by age group and sex



MVEV and JEV. Unspecified flavivirus infections are reported under arbovirus NEC. These infections are nationally notifiable.

DENV has 4 serotypes, each containing numerous genotypes. 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,¹⁹ 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.^{19,103–105} The clinical illness is characterised by mild to severe febrile illness with fever, headache, muscle or 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,²⁰ but there are no vaccines currently for DENV, MVEV or KUNV infection.

Arbovirus NEC

- Notifications in 2012 were below the 5-year mean.
- All cases in 2012 were in adults.
- There were a range of different infections, which were frequently acquired in South East Asia.

Epidemiological situation in 2012

In 2012, there were 9 notifications of arbovirus NEC, compared with an average of 16.2 during the previous 5 years. These notifications comprised Alfuy (1 case), flavivirus unspecified (4 cases), Zika (1 case), Kokobera (2 cases) and Stratford (1 case), (Table 20).

Table 20: Notifications of arbovirus NEC, Australia, 2012, by infecting agent and state or territory

State	Organism	Country of acquisition	Age
Qld	Alfuy	Unknown	55
Qld	Kokobera	Unknown	51
Qld	Kokobera	Unknown	68
Qld	Untyped	Australia	20
Qld	Untyped	Cambodia	43
Qld	Untyped	Thailand	22
Qld	Untyped	Philippines	56
Qld	Stratford	Unknown	70
SA	Zika	Indonesia	53

Information on the place of acquisition was available for 44% of cases (4/9), and three of these were acquired overseas.

The median age of cases was 53 years (range 20-70 years).

Dengue virus infection

- Notifications in 2012 were 1.8 times the 5-year mean.
- Larger number of overseas-acquired cases than in any previous year.
- Only 29 locally-acquired cases were reported.
- 54% of all cases in 2012 were acquired in Indonesia.

Local transmission of dengue in Australia is normally restricted to areas of northern Queensland where the key mosquito vector, *Ae. aegypti* is present.¹⁰⁶ 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.¹⁰⁷ The CDNA case definition for dengue was changed in 2013 to accept dengue non-structural protein 1 (NS1) antigen in blood as laboratory definitive evidence for infection; however, it should be noted that a number of states and territories had been sending notifications based on a positive NS1 antigen prior to this change.

Epidemiological situation in 2012

There were 1,540 notifications of dengue in 2012, with 817 in 2011. This was 1.8 times the 5-year mean of 864 notifications. Most infections were acquired overseas (n=1,410) (Figure 72). There were 29 infections acquired in Australia. For 101 cases, no information was supplied on the place of acquisition.

Serotype of dengue virus infections

In 2012, serotype information was available for 18% of notifications (282/1,540), which was a decrease compared with the 5-year mean of 50% (Table 21). The decreased reporting of a serotype may reflect the increasing use of NS1 antigen detection and/ or other diagnostic methods that do not provide a serotype. In 2012, 49% (137/282) of cases with a known serotype were due to DENV serotype 2 and 28% (79/282) were DENV 1 (Table 21).

Seasonality and place of acquisition

There were 1,412 DENV infections known to have been acquired overseas in 2012, up from 714 in 2011 and the largest number ever reported. Between 2007 and 2010, the number of DENV cases known to have been acquired overseas increased each year, from 254 in 2007 to 1,137 in 2010 (Figure 72). In recent years, improved diagnostic techniques, in particular the availability of the rapid NS1 antigen detection kit, have improved detection and

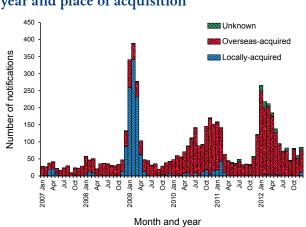


Figure 72: Notifications of dengue virus infection, Australia, 2007 to 2012, by month, year and place of acquisition

would have contributed to the observed increase in reported numbers of overseas-acquired dengue in Australia,¹⁰⁸ along with the dramatic re-emergence and geographical expansion of dengue overseas over the past 50 years and explosive outbreaks.¹⁰⁵ For 99 cases (6%), no information on the place of acquisition was available, and no particular country or region of acquisition was stated for 5 cases that were known to have been acquired overseas, (Table 22). Cases acquired in Indonesia continue to account for the largest number and proportion of all notifications, accounting for 54% (778/1,441)

Table 21: Serotype of dengue virus infections, Australia, 2007 to 2012

Serogroup	2007	2008	2009	2010	2011	2012
Virus 1	36	40	82	190	139	79
Virus 1 and 4	1					1
Virus 2	14	32	54	255	153	137
Virus 2 and 3	1					
Virus 3	52	143	771	106	78	57
Virus 4	7	37	43	47	43	8
Untyped/unknown	203	309	452	629	404	1,258
Total	314	561	1,402	1,227	817	1,540
% with a serotype supplied	35.4	44.9	67.8	48.7	50.6	18.3

Table 22: Serotype of dengue virus notifications, Australia, 2012, by serotype and place of acquisition

	Serotype						
Place of acquisition	DENV 1	DENV 1 and 4	DENV 2	DENV 3	DENV 4	Untyped/ unknown	Total
Locally acquired							
Australia	14	0	2	7	0	6	29
Unknown							
Unknown/not stated	1	0	1	0	0	97	99
Overseas acquired							
Indonesia	20	0	101	8	3	646	778
Thailand	21	0	17	11	2	204	255
India	3	0	2	9	0	44	58
Philippines	3	1	2	1	1	45	53
East Timor	1	0	1	12	0	36	50
Fiji	6	0	2	0	0	22	30
Cambodia	1	0	0	0	0	28	29
Sri Lanka	2	0	1	2	0	20	25
Vietnam	1	0	2	1	0	16	20
Malaysia		0	2	0	1	15	18
Papua New Guinea	1	0	1	4	0	10	16
Bangladesh		0	0	0	0	12	12
Kiribati	2	0	0	0	0	5	7
Maldives	1	0	0	0	0	3	4
Burma (Myanmar)	1	0	0	0	0	2	3
Other countries	1	0	2	2	1	43	49
Country not stated	0	0	1	0	0	4	5
Total overseas acquired	64	1	134	50	8	1,155	1,412
Total	79	1	137	57	8	1,258	1,540

of all cases in 2012, but down from the 58% in 2010 and compared with an average of 33% over the previous 5 years. DENV acquired in Indonesia was frequently serotype 2, comprising 76% of cases with a known serotype (101/132 cases). Other frequently reported source countries in 2012 included Thailand, India, the Philippines and East Timor.

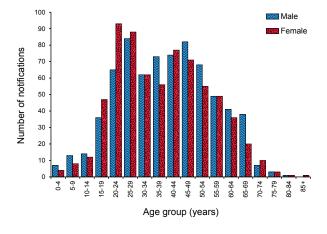
All but one of the 29 locally-acquired DENV in 2012 were known to have been associated with one of the 5 outbreaks of locally-acquired infection in Queensland in 2012 that were notified to NNDSS. The largest number of notified cases during the year was during an outbreak of DENV 1 and 2 in Townsville with 8 cases notified to NNDSS in 2012. An outbreak in Cairns, which began in late 2012 was larger, with a total of 146 cases, but most of these (141) were notified in 2013.

The peak months for overseas-acquired dengue in 2012 were January to April, together accounting for 58% (821/1,412) of cases. No particular pattern was evident with the small number of locally-acquired cases; however, there was only 1 case between July and October 2012, demonstrating that outbreaks are not continuing through the cooler months.

Age and sex distribution

DENV infections acquired overseas in 2012 were most commonly reported among younger and middle aged adults (median 39 years, range 2–85 years), with a peak of notifications among males aged 20–29 years and females aged 25–29 years (Figure 73). Males comprised 51% of cases with overseas-acquired DENV. For locally-acquired DENV, infections were more commonly reported among middle aged and older adults (median 44 years, range 5–76 years), with

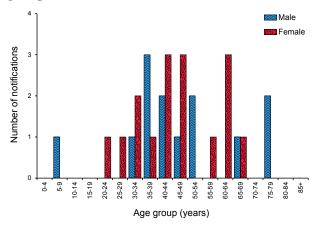
Figure 73: Notifications of overseas-acquired dengue, Australia, 2012,* by age groupand sex



* Sex was not available for 2 cases.

peak notifications among males and females aged 40–44 years (Figure 74). Males comprised 45% of cases with locally-acquired DENV.

Figure 74: Notifications of locally-acquired dengue virus infection, Australia, 2012, by age group and sex



Kunjin virus infection

• No cases of Kunjin were notified in 2012.

Epidemiological situation in 2012

In 2012, there were no notified KUNV infections in Australia, compared with 2 cases in 2011 and an average of 1.6 cases per year between 2007 and 2011.

Japanese encephalitis virus infection

- JEV is a rare disease, acquired overseas.
- The last locally-acquired case was in 1998.
- One case of JEV was notified in 2012.

Epidemiological situation in 2012

There was 1 notification of JEV infection in 2012, in a 16-year-old female who acquired the infection in the Philippines. Prior to this case there was 1 notification of JEV infection in 2008, which was also acquired overseas. The last locally-acquired case was in 1998.¹⁰⁹

Murray Valley encephalitis virus infection

- MVEV is a rare disease in Australia, and also acquired overseas in the region.
- One case of MVEV was notified in 2012.

Epidemiological situation in 2012

In 2012, there was 1 notification of MVEV infection, in a 14-year-old who acquired the infection in Papua New Guinea and was diagnosed in Queensland. In the past 5 years there were 2 cases in 2008, 4 cases in 2009 and 17 cases in 2011. The cases notified in 2011, including an outbreak in south east Australia, have been described elsewhere.^{97,110–112}

Malaria

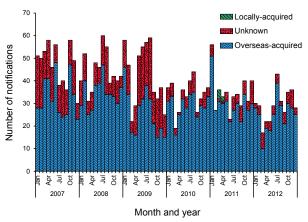
- Notifications continued the gradual decline observed since 2005.
- No cases were known to have been acquired in Australia in 2012.

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. 19,113 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. Australia was declared free of malaria in 1981,¹¹⁴ but suitable vectors are present in northern Australia, and the area remains malaria-receptive. Malaria is the most frequently reported cause of fever in returned travellers worldwide.¹¹⁵ 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.¹¹⁶ Malaria cases in Australia can be found either through testing of symptomatic persons with a compatible travel history, or through screening of refugees who may be asymptomatic.

Epidemiological situation in 2012

There were 348 cases of malaria notified in Australia in 2012; a 28% decrease compared with the 5-year mean of 484 notifications, and continuing the trend of gradually decreasing notifications since 2005 (Figure 75). The largest number of cases was reported by Queensland (100 cases).

Figure 75: Notifications of malaria, Australia, 2007 to 2012, by month, year and place of acquisition



Seasonality and place of acquisition

The place of acquisition was listed as overseas for 297 cases, while for the remaining 51 cases, no place of acquisition information was supplied to NNDSS, but none were known to have been acquired in Australia. The last known locallyacquired infections were in 2011 in an outbreak in the Torres Strait,¹¹⁷ and the last cases acquired on the mainland were during an outbreak in North Queensland in 2002.¹¹⁸

Complete information on the country or region of acquisition was supplied for all but six of the cases known to have been acquired overseas, and these remaining cases were notified as being overseas acquired, country unknown or not stated. The most frequent countries of acquisition were Papua New Guinea (16% of cases with complete information) and India (16%) (Table 23). Most cases acquired in Papua New Guinea were reported by Queensland (31 cases). Increases in notifications or an observable pattern of seasonality in a predominantly overseas-acquired infection can relate to the seasonality of travel patterns or to local disease epidemiology in the source countries. There was no discernible pattern of seasonality in notifications between 2007 and 2011, or in 2012.

Infecting species

The infecting species was supplied for 99% (343/348) of notifications in 2012 (Table 23). The most frequent infecting species was *P. falciparum* (reported in 54% of notifications with complete information). *P. vivax* was associated with Asia and the Pacific, whilst most infections acquired in African countries were *P. falciparum*. In infections acquired in Papua New Guinea however, *P. falciparum* and *P. vivax* infections were reported in similar numbers (20 and 25 cases respectively).

Table 23: Notifications of malaria, Australia, 2012, by infecting species and region and country of	
acquisition	

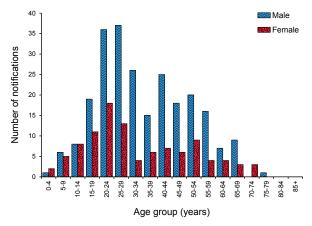
	Р.			Р.	Mixed species	Plasmodium	
Country and region	falciparum	P. vivax	P. ovale	malariae	infection	species	Total
Oceania			-				
Papua New Guinea	20	25	0	0	0	1	46
Solomon Islands	0	7	0	0	0	0	7
South East Asia	1				1		
Indonesia	8	8	0	0	0	1	17
Cambodia	2	4	1	0	0	0	7
Philippines	1	2	0	0	0	0	3
Laos	2		0	0	0	0	2
Thailand	0	1	0	0	0	0	1
Malaysia	0	1	0	0	0	0	1
Mainland southeast Asia, nfd*	0	1	0	0	0	0	1
North-east Asia							
China	0	1	0	0	0	0	1
Southern and Central Asia							
India	4	39	1	0	0	2	46
Pakistan	0	17	0	0	0	0	17
Bangladesh	0	1	0	0	0	0	1
Americas							
Guyana	2	1	0	0	0	0	3
Brazil	0	2	0	0	0	0	2
South America, nfd	0	1	0	0	0	0	1
South America, not elsewhere classified	0	1	0	0	0	0	1
North Africa and the Middle Eas	t				"	"	
Sudan	33	1	0	0	0	0	34
North Africa, nfd	2	1	0	0	0	0	3
Iran	1	0	0	0	0	0	1
Sub-Saharan Africa						"	
Ghana	14	0	0	0	0	0	14
Sierra Leone	9	1	1	1	0	0	12
Tanzania	9	0	0	1	0	0	10
Uganda	7	0	1	1	0	1	10
Kenya	6	0	0	2	0	0	8
Nigeria	7	0	0	0	0	0	7
Guinea	6	0	0	0	0	0	6
Other sub-Saharan Africa countries	19	1	2	0	2	0	24
Sub-Saharan Africa countries, nfd	5	0	0	0	0	0	5
Overseas acquired – country an	d region not s	stated/unkr	nown			n 	
Unknown country	5	0	1	0	0	0	6
Overseas-acquired total	162	116	7	5	2	5	297
Place of acquisition unknown	23	23	3	2	0	0	51
Total	185	139	10	7	2	5	348

nfd Not further defined.

Age and sex distribution

In 2012, sex was stated for all but 1 case. Malaria was most commonly reported in males (70%, 244/347 cases) with a peak of notifications in males aged 20–24 years and 25–29 years (Figure 76). The median age of cases was 31 years (range 1–77 years).

Figure 76: Notifications of malaria, Australia, 2012, by age group and sex*



* Sex was not stated for 1 case, and this case has been excluded.

Zoonotic diseases

Zoonoses are those diseases and infections that are naturally transmitted between vertebrate animals and humans.¹¹⁹ Approximately 60%–70% of emerging human infectious diseases are zoonoses^{120–122} and more than 70% of emerging zoonoses originate from wildlife.¹²¹ 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'.¹²³

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

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

• No cases of anthrax were notified in 2012.

Anthrax is caused by the bacterium *Bacillus anthracis* and mainly causes cutaneous infection. 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 livestock workers who handle infected animals or by-products.

In Australia, the areas of anthrax risk are well defined and include the northern and northeastern districts of Victoria and central New South Wales.¹²⁴ Anthrax occurs only sporadically in livestock in the at-risk areas. Rare or isolated incidents or cases have historically occurred in Queensland, South Australia, Tasmania and Western Australia.¹²⁴

Epidemiological situation in 2012

In 2012, there were no notified cases of anthrax in Australia. Over the previous 10 years, only 3 human cases of anthrax were reported in Australia; in 2006, 2007 and 2010.^{125–127} 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 4 anthrax incidents reported in livestock in Australia in 2012, with all properties located within the known New South Wales anthrax endemic area.¹²⁴

Australian bat lyssavirus and lyssavirus (unspecified) infections

• No cases of Australian bat lyssavirus or lyssavirus (unspecified) infection were notified in 2012.

ABLV belongs to the genus lyssavirus, which also includes the rabies virus. Both invariably result in progressive, fatal encephalomyelitis in humans.¹²⁸ ABLV was identified in Australia in 1996 ^{129,130} 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.¹³¹ Post-exposure prophylaxis for ABLV consists of wound care and administration of a combination of rabies virus vaccine and human rabies virus immunoglobulin, depending on exposure category and prior vaccination or antibody status.^{20,131}

Epidemiological situation in 2012

In 2012, there were no notified cases of ABLV or lyssavirus (unspecified) infection in Australia. There were also no cases of rabies in 2012. Rabies is reported in more detail in the quarantinable diseases section.

There have been 3 fatal cases of ABLV infection in humans, in 1996, 1998 and 2013. All cases occurred after close contact with an infected bat and all were fatal.^{132–134} 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.^{135,136}

The bat health focus group in the Australian Wildlife Health Network gathers and collates information from a range of organisations on opportunistic testing of bats for ABLV. In 2012, there were 5 ABLV detections compared with 6 detections in bats during 2011.¹³⁷

Brucellosis

- 29 cases of brucellosis were notified in 2012.
- 2 cases of Brucella melitensis, and 1 case of B. abortus were reported and all were acquired overseas.

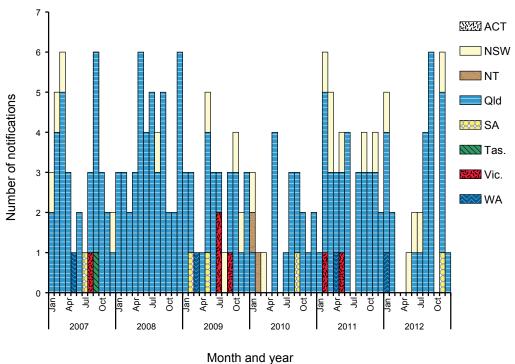
Brucella species that can cause illness in humans include *Brucella melitensis* acquired from sheep and goats, *B. suis* from pigs and *B. abortus* from cattle. *B. abortus* was eradicated from Australian cattle herds in 1989 and *B. melitensis* has never been reported in Australian sheep or goats.¹²⁴ 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)¹³⁸ found that feral pig hunting was the most common risk factor for infection for brucellosis cases in Townsville during 1996 to 2009.

Internationally, brucellosis is mainly an occupational disease of farm workers, veterinarians, and abattoir workers who work with infected animals or their tissues.¹⁹

Epidemiological situation in 2012

In 2012 there were 29 notified cases of brucellosis in Australia (a rate of 0.1 per 100,000), compared

Figure 77: Notifications of brucellosis, Australia, 2007 to 2012, by month and year of diagnosis and state or territory



with the 5-year mean of 35 notifications (2007 to 2011). Seventy-six per cent of notifications (22/29) were from Queensland (Figure 77), with a rate of 0.5 per 100,000. Since 1991, 84% of notifications have been from Queensland.

The species of the infecting organism was available for 40% of notifications (12/29). Of these, 9 notifications were for *B. suis*; eight from Queensland and one from South Australia (abattoir worker), and all were males aged between 20 and 36 years. There were 2 notifications of *B. melitensis*, with the country of acquisition listed as Iraq and Lebanon. There was also 1 notification for *B. abortus* from New South Wales, which was listed as having been acquired overseas, but the specific county was unknown.

The median age of notified cases of brucellosis was 36 years (range 18–72 years) and 90% of cases (26/29) were male.

Leptospirosis

- 116 cases of leptospirosis were notified in 2012.
- Notifications in 2012 returned to expected levels after an increase in 2011.

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).^{139,140} The last reported death in Australia attributed to leptospirosis was in 2002.¹⁴¹

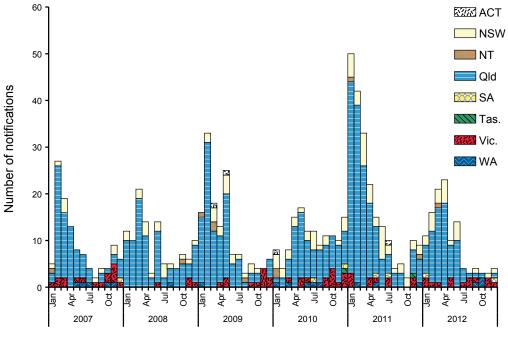
Epidemiological situation in 2012

In 2012 there were 116 notified cases of leptospirosis in Australia (a rate of 0.5 per 100,000), compared with the 5-year mean of 141 notifications (2007 to 2011). Notifications in 2012 returned to expected levels after an increase in 2011 (Figure 78), which was largely attributed to extensive flooding in central and southern Queensland.^{142,143} In 2012, Queensland accounted for 65% (75/116) of notifications.

Age and sex distribution

The median age of leptospirosis notifications was 34 years (range 6–82 years) and 88% of cases (102/116) were male. The highest notification rate was observed in males in the 25–29 years age group (2.1 per 100,000 male population).

Figure 78: Notifications of leptospirosis, Australia, 2007 to 2012, by month and year of diagnosis and state or territory



Month and year

Typing information

The WHO/FAO/OIE Collaborating Centre for Reference and Research on Leptospirosis routinely conducts polymerase chain reaction-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. At the time of compiling this report, data for 2012 were not publicly available.

Typing information from NNDSS was available for 84% (97/116) of notifications. Of those with typing information, the most common serovar was Arborea (23%, 22/97), followed by Hardjo (22%, 21/97), Australis (20%, 19/97) and Zanoni (20%, 19/97).

Ornithosis

- 75 cases of ornithosis were notified in 2012.
- The majority of notifications in 2012 were from Victoria, with half of these notified in the last quarter of 2012.

Ornithosis (or psittacosis) is caused by infection with the bacterium *Chlamydophila psittaci*. It is transmitted to humans primarily from infected parrots of many species, but also poultry and a range of other birds.¹⁴⁴ 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.¹⁴⁵

Epidemiological situation in 2012

In 2012, there were 75 notified cases of ornithosis in Australia (a rate of 0.3 per 100,000), compared with the 5-year mean of 82 notifications (2007– 2011). The majority of notifications in 2012 were from Victoria (63%, 47/75), this is a decrease compared with the number reported in 2011 (n=58)¹⁴⁶ (Figure 79).

Just over half (51%, 24/47) of the 2012 Victorian cases were notified in the last quarter, with 15 notified in October. Following an increase in notified cases (n=7) in the Yarra Ranges Shire in Victoria during the 4th quarter, the Victorian Department of Primary Industries investigated a bird feeding area in the Dandenong Ranges, with reports of sick and dying birds in the area. The public health actions taken include supplying an information leaflet on ornithosis with packets of bird seed sold and logging the number of sick and dying birds surrendered to the Parks Victoria staff.¹⁴⁷

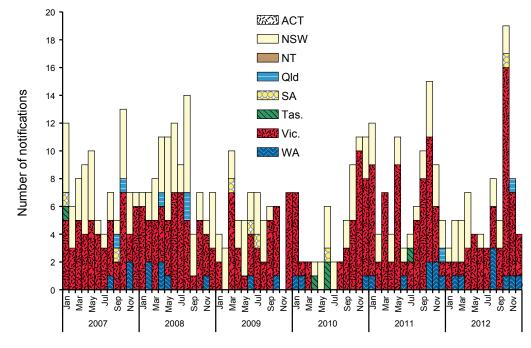


Figure 79: Notifications of ornithosis, Australia, 2007 to 2012, by month and year of diagnosis and state or territory

Month and year

Age and sex distribution

The median age of ornithosis notifications was 55 years (range 30-79 years) and 56% (42/75) of notified cases were male.

Q fever

- 358 cases of Q fever were notified in 2012.
- 78% of cases were male and the highest notification rate was observed in males in the 55–59 years age group.

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.¹⁴⁸ Q fever is most commonly transmitted via the airborne route, where the organism is carried in dust contaminated with tissue, birth fluids or excreta from infected animals.¹⁴⁹ Prior to the commencement of vaccination programs in Australia, approximately half of all cases in New South Wales, Queensland and Victoria were among abattoir workers.^{150,151}

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).¹⁵²

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.²⁰

Epidemiological situation in 2012

In 2012, there were 358 notified cases of Q fever in Australia (a rate of 1.6 per 100,000), compared with the 5-year mean of 365 notifications (2007–2011).

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 $100,000.^{152}$ In 2012, the highest notification rate was in Queensland (4.2 per 100,000, n=192). Cases were reported in all jurisdictions except the Australian Capital Territory and Tasmania (Figure 80).

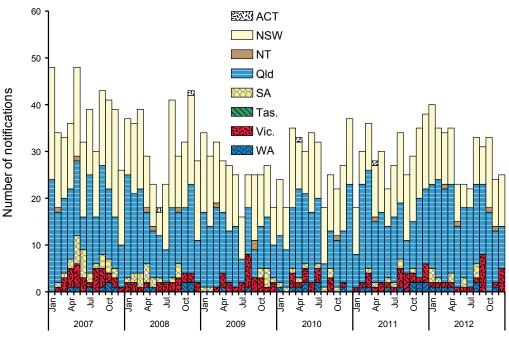


Figure 80: Notifications of Q fever, Australia, 2007 to 2012, by month and year of diagnosis and state or territory

Month and year

Age and sex distribution

The median age of Q fever notifications was 48 years (range 8–82 years) and 78% of cases (279/358) were male. The highest notification rate was observed in males in the 55–59 years age group (5.2 per 100,000 male population).

Tularaemia

• No cases of tularaemia were notified in 2012.

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.¹⁵³

Tularaemia was last notified in 2011, with 2 cases from Tasmania.³³ This was the first time that *F. tularensis* type B had been detected in the Southern Hemisphere.^{154,155}

Epidemiological situation in 2012

In 2012, there were no notified cases of Tularaemia in Australia.

Other bacterial infections

Surveillance objectives

Other bacterial diseases in the national notifiable disease list are legionellosis, leprosy, invasive meningococcal disease and tuberculosis.

In 2012, there were 1,924 cases of other bacterial diseases notified to the NNDSS, representing less than 1% of all reported cases and a 4% decrease compared with 2011 (n=2,006).

Common objectives for the surveillance of diseases in this section are to monitor their epidemiology and to identify risk groups to accurately target control strategies.

Legionellosis

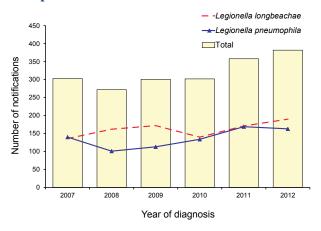
- 382 cases of legionellosis were notified in 2012.
- Since 1991, the number of legionellosis notifications has continued to rise.
- Legionella longbeachae, traditionally associated with potting mix, was more frequently reported as the causative species in 2012.

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 most commonly associated with human disease in Australia are *Legionella pneumophila* and *Legionella longbeachae*. Legionella bacteria are found naturally in low levels in the environment. In the absence of effective environmental treatment *Legionella* organisms can grow to high numbers in air conditioning cooling towers, hot water systems, showerheads, spa pools, fountains or potting mix.

Epidemiological situation in 2012

A total of 382 cases of legionellosis were notified in 2012, representing a rate of 1.7 per 100,000. Compared with 2011 the overall number of legionellosis cases increased in 2012 by 7%. This was the highest since 2007 (Figure 81).

Figure 81: Notifications of legionellosis, Australia, 2007 to 2012, by year of diagnosis and species



Data on the causative species were available for 93% (n=355) of notifications in 2012. Of the notifications with a reported species, proportionally there were slightly more cases of *L. longbeachae* (54%) than *L. pneumophila* (46%). There was a single confirmed case of *L. micdadei*. Serogroup data were available for 121 (74%) of the 163 *L. pneumophila* cases. Of these, 119 (98%) were due to serogroup 1 and the remainder were serogroup 2.

From 2007 to 2012, the annual number of notifications of *L. longbeachae* ranged from 136 to 190 cases and for *L. pneumophila* from 101 to 169 cases (Figure 81). In 2012, when compared with 2011, the number of cases of *L. pneumophila* decreased by 4% whilst case numbers of *L. longbeachae* increased by 11%. Mortality data were available for 66% (n=252) of notifications in 2012 and of those, there were 11 deaths reported due to legionellosis. Most of these deaths were attributed to *L. pneumophila* (82%, n=9) (Table 24).

Geographical distribution

In 2012, rates of legionellosis varied from 0.5 per 100,000 in the Australian Capital Territory to 3.5 per 100,000 in Western Australia (Table 24). In 2012, the geographical distribution of *L. longbeachae* and *L. pneumophila* across jurisdictions mirrored that in 2011, with the exception of Queensland. The majority of notifications in South Australia, Queensland and Western Australia were attributed to *L. longbeachae*, whilst in New South Wales and Victoria *L. pneumophila* was the most common infecting species.

Age and sex distribution

In 2012, legionellosis was predominantly seen in older males. Overall, males accounted for the majority (61%) of the notifications with a male to female ratio of 1.6:1. There were no notifications in people under the age of 15 years. The age group with the highest notification rate was the 85 years or over group (7.5 per 100,000). The highest age and sex specific rates were observed in men aged 85 years or over (10.7 per 100,000, 16 notifications) and women aged 74–79 years (5.9 per 100,000, n=18) (Figure 82).

The 11 cases that were reported to have died due to legionellosis ranged in age from 38–87 years (median 70 years); 9 deaths were males and 2 were female. The majority of legionellosis notifications were in people aged 40 years or over (Figure 82).

Seasonality

In 2012, diagnoses of legionellosis (all species) were highest in July, with 46 cases notified in that month (Figure 83). *L. pneumophila* occurred

most frequently in the summer months, with the highest number of notifications being recorded in February (n=21). *L. longbeachae* cases occurred most frequently in the spring months. However, the highest number of *L. longbeachae* cases reported in any 1 month occurred in July (n=26) of which half (n=13) were notified in Western Australia.

Figure 82: Notification rate of legionellosis, Australia, 2012, by age group and sex

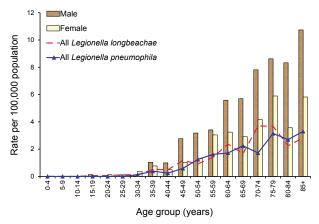


Figure 83: Notifications of legionellosis, Australia, 2007 to 2012, by month and year of diagnosis and species

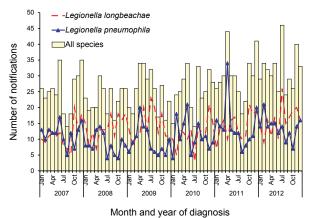


Table 24: Notifications of legionellosis, Australia, 2011, by species and state or territory

			S	State or t	territory	1				Deaths due to
Species	АСТ	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.	legionellosis
L. longbeachae	1	29	3	37	26	5	16	73	190	2
L. pneumophila	0	64	0	23	13	6	45	12	163	9
L. micdadei	0	0	0	0	0	0	1	0	1	0
L. micdadei or pneumophila	0	0	0	0	0	1	0	0	1	0
Unknown species	1	9	0	10	0	0	7	0	27	0
Total	2	102	3	70	39	12	69	85	382	11
Rate (per 100,000 population)	0.5	1.4	1.3	1.5	2.4	2.3	1.2	3.5	1.7	

The seasonal pattern of *L. longbeachae* in 2012 reflected the seasonal pattern observed for this species over the previous 5 years, with the exception of 2009 when *L. longbeachae* occurred more frequently in the winter months. The seasonal pattern of *L. pneumophila* differed from the seasonal pattern observed for this species over the previous 4 years, with *L. pneumophila* occurring more frequently over the summer months as opposed to the autumn months. In 2007, *L. pneumophila* occurred more frequently in the summer months (Figure 83).

Place of acquisition

Place of acquisition was reported for 73% (n=280) of legionellosis notifications in 2012. Of these, 96% (n=267) were reported to have been acquired in Australia and 4% (n=13) overseas. Indonesia (n=3) and Thailand (n=2) were the most commonly reported places of acquisition for infections acquired overseas.

Outbreaks and clusters

In 2012, there were 5 *L. pneumophila* clusters and 1 outbreak of *L. pneumophila* notified to the NNDSS. Two clusters were reported in New South Wales and one each in Queensland, Victoria and South Australia with an outbreak reported in Victoria.

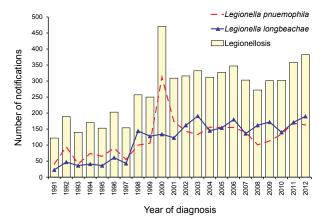
In New South Wales, 14 legionellosis notifications due to L. pneumophila serogroup 1 were reported from February to April in the Western Sydney and Nepean Blue Mountains Local Health Districts. This was approximately twice the number of cases usually seen in this period. The cases were clustered in 3 time periods; early February, mid-March and late April. Extensive investigations into these clusters were unable to determine any common sources for the infections.¹⁵⁶ An additional cluster in New South Wales was identified in November and December with 4 notified cases, but no common source was identified. One cluster and 1 outbreak were reported in Victoria in 2012, involving a total of 7 cases from the Northern and Western Metropolitan region. Both investigations were unable to definitively identify the source of infection.157

The Queensland cluster consisted of 2 cases diagnosed in January and February of 2012. The cases were identified in residents of a retirement village in South East Queensland. An environmental investigation of the facility was undertaken with water samples collected from the spa, pool and resident showers. The water samples were negative for *L. pnuemophila* and no source of the infection was identified. The cluster in South Australia formed part of an investigation that was conducted from January to March 2013. In total, there were 13 cases identified as the same cluster with 3 cases from South Australia and 3 cases from Victoria.

Change in the epidemiology of species from 1991 to 2012

Since 1991, the number of legionellosis notifications has continued to rise (Figure 84). Before 1998, legionellosis notifications were more likely to be attributed to *L. pneumophila*. However, since 1998, the most common infective species has alternated between *L. pneumophila* and *L. longbeachae*.

Figure 84: Notifications of legionellosis, Australia, 1991 to 2012, by year of diagnosis and species



Discussion

Since reporting began in 1991, the number of notifications reported annually for legionellosis has increased by two-thirds from 122 notifications in 1991 to 382 notifications in 2012. The increased use of more sensitive diagnostic testing may have contributed to this rise in notifications.

The demographic profile of legionellosis cases since 1991 has remained consistent with the recognised epidemiology of the disease.^{158–160} Less than 7% of notified cases are in persons under the age of 30 years or over 70% are in those aged 50 years or over. However, since reporting began in 1991 there has been a change in the predominant notified species. Whilst *L. pnuemophila* was the predominate species notified between 1991 and 1997, since 1998 (with the exception of the 2000 *L. pneumophila* outbreak) the most commonly reported species of *Legionella* has alternated between *L. pnuemophila* and *L. longbeachae*.

Leprosy

- A total of 4 cases of leprosy were notified in 2012.
- Since 1992 annual notifications of leprosy have ranged from 4 to 19 cases.
- All cases were acquired overseas

Leprosy is a chronic infection of the skin and peripheral nerves due to the bacterium Mycobacterium leprae. Leprosy is an uncommon disease in Australia, although, a very small number of people are diagnosed each year, with the majority of cases acquiring the infection overseas. The incidence of leprosy worldwide is declining due to various factors including economic development, the use of Bacillus Calmette-Guérin vaccine and high coverage with multi-drug therapy.¹⁹ Leprosy is not highly infectious. People at risk are generally in close and frequent contact with leprosy patients or living in countries where the disease is more common. The disease is curable and once a person with leprosy begins appropriate treatment, they quickly become non-infectious.

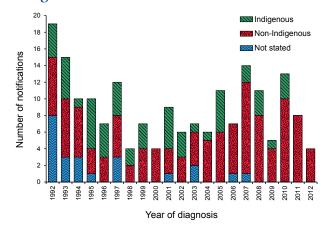
Epidemiological situation in 2012

There were 4 notifications of leprosy in 2012, representing a rate of 0.02 per 100,000.

All cases were residents of Victoria and were reported as being non-Indigenous. Cases ranged from 29–72 years of age. In 2012, 2 cases were male and 2 female. All four of these cases were reported as having acquired leprosy overseas.

The number of leprosy cases decreased in 2012 by half, from the 8 cases reported in 2011 (Figure 85). Since 1992, the annual number of notifications of leprosy has ranged from 4 to 19 cases.

Figure 85: Notifications of leprosy, Australia, 1992 to 2012, by year of diagnosis and Indigenous status



Meningococcal disease (invasive)

- Notification rates for invasive meningococcal disease (IMD) continue to be low in Australia, being only 1.0 per 100,000 population in 2012.
- Since the introduction of meningococcal C vaccine to the NIP in 2003, notifications of IMD due to serogroup C have reduced considerably with only 11 cases reported this year. Fewer than half of these were of an age eligible for vaccination.
- A primary peak in notification rates of IMD was reported in young children, aged less than 5 years with a smaller secondary peak in young adults aged 15–19 years.

Meningococcal disease is caused by the bacterium Neisseria meningiditis. Invasive disease occurs when bacteria infect a normally sterile site, usually the blood (septicaemia), cerebrospinal fluid (meningitis) or both. Asymptomatic respiratory tract carriage of meningococci occurs in 5%-10% of the population and prevalence may be higher when groups of people occupy small areas of any space.^{19,20} The disease is transmitted via respiratory droplets and has an incubation period of between 1 and 10 days, most commonly 3 to 4 days.^{20,161} Infection occasionally causes a rapidly progressive serious illness, most commonly in previously healthy children and young adults. Globally, serogroups A, B, C, W135 and Y most commonly cause disease.¹⁹ Historically, N. meningitidis serogroups B and C have been the major cause of IMD in Australia.

Since 2003, meningococcal C vaccine has been available for those 12 months of age as part of the childhood immunisation schedule funded under the NIP. Additionally, a catch-up program provided access to the meningococcal C vaccine for children and adolescents born between 1984 and 2001. There was a staged implementation, ending in 2006, with a funded vaccine made available through general practitioners for the 1–5 years age group and through school based clinics for the 6–19 years age group.

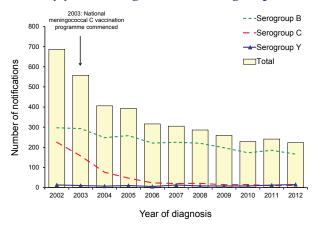
Epidemiological situation in 2012

In 2012, there were 223 cases of IMD, representing a rate of 1.0 per 100,000. This was a decrease of 8% on the cases notified in 2011 (n=241) and the lowest number of cases notified annually compared with the preceding 10 years (Figure 86).

Most cases (n=219) notified in 2012 met the definition for a confirmed case, that is, diagnosed

based on laboratory definitive evidence, or laboratory suggestive evidence and clinical evidence.¹⁶² A small number of cases (n=4) met the definition for a probable case, that is, diagnosed based on clinical evidence only.

Figure 86: Notifications of invasive meningococcal disease, Australia, 2002 to 2012, by year of diagnosis and serogroup



Data on serogroup were available for 89% of cases in 2012. Seventy-four per cent of cases were due to serogroup B, 7% serogroup Y, 5% serogroup C and 3% serogroup W135. The number of cases of IMD caused by serogroup B notified in 2012 was the lowest compared with the number of cases notified annually in the preceding 10 years. Notifications of IMD caused by serogroup C organisms have decreased substantially since the introduction of the meningococcal C vaccine on the NIP in 2003, with fewer than 25 cases reported annually. Notifications of IMD caused by serogroup Y peaked in 2012 with 15 cases, which was the highest number of cases reported annually compared with the preceding 10 years.

Mortality data were available for 60% (n=133) of cases reported to the NNDSS in 2012. Twelve

cases were reported as having died from IMD; 10 due to serogroup B and 2 due to serogroup C (Table 25). Of the deaths due to serogroup B, five were children aged less than 2 years, two were young adults and the remaining three were adults aged over 25 years. Of the 2 serogroup C related deaths, one occurred in an unvaccinated person in the 15–19 years age group, who was eligible for vaccination. The second death was an infant too young for vaccination.

Of the 11 cases of IMD due to serogroup C in 2012, four were aged between 1 and 29 years and therefore would have been eligible for vaccination either through routine childhood immunisation or under the meningococcal C catch up program. All four of these cases were reported as not vaccinated with meningococcal C vaccine.

Geographical distribution

All jurisdictions reported in accordance with the national case definition for IMD, except the Australian Capital Territory and New South Wales where conjunctival cases were also reported. Conjunctival cases cannot be distinguished from invasive cases in the national dataset.

In 2012, cases of IMD were reported from all states and territories, ranging from 1 case from the Australian Capital Territory to 66 from New South Wales (Table 25). Notification rates ranged from 0.3 per 100,000 in the Australian Capital Territory to 1.8 per 100,000 in South Australia.

Age and sex distribution

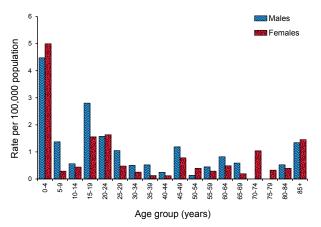
More males than females were reported with IMD in 2012, with a male to female ratio of 1:0.8. Twothirds of cases (n=147) were less than 25 years of age, of which those less than 5 years of age made up almost half (n=70). Cases aged less than 5 years had the highest age-specific rate at 4.7 cases per 100,000. High rates also occurred among the

Table 25: Notifications of invasive meningococcal disease and deaths due to invasive meningococcal disease, Australia, 2012, by serogroup and state or territory

				State or	territory					
	АСТ	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.	Deaths
В	1	43	3	45	26	4	29	15	166	10
С	0	2	1	3	1	1	1	2	11	2
W135	0	4	0	3	0	0	0	0	7	0
Y	0	5	0	4	1	1	4	0	15	0
Unknown	0	12	0	8	1	1	1	1	24	0
Total	1	66	4	63	29	7	35	18	223	12
Rate (per 100,000)	0.3	0.9	1.7	1.4	1.8	1.4	0.6	0.7	1.0	_

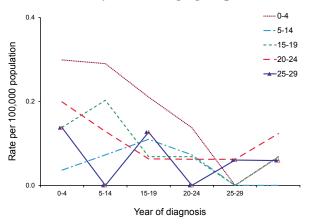
15-19 years age group (2.2 per 100,000) followed by the 20-24 years age group (1.6 per 100,000) (Figure 87).

Figure 87: Notification rates of invasive meningococcal disease, Australia, 2012, by age and sex



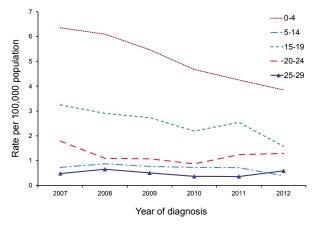
groups, age-specific rates have been maintained at very low levels, with no age group exceeding 0.2 cases per 100,000 in 2012 (Figure 89).

Figure 89: Notification rate for serogroup C invasive meningococcal disease, Australia, 2007 to 2012, by selected age group



Serogroup B accounted for the majority of cases across all age groups including those aged less than 25 years, where it accounted for 85% of cases with serogroup information available. While the age-specific rates of serogroup B infection in 2012 remain high compared with other serogroups, they continue to trend downward across all age groups (Figure 88).

Figure 88: Notification rate for serogroup B invasive meningococcal disease, Australia, 2007 to 2012, by selected age groups

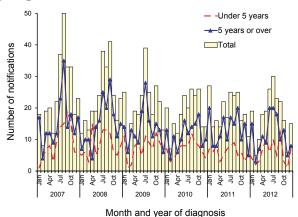


Of the 11 cases of IMD due to serogroup C notified in 2012 only 4 were among children and young adults aged less than 25 years of age. While there was an increase in the rate of IMD due to serogroup C in the 15–19 years and 20–24 years age

Seasonality

An average of 17 cases of IMD was reported each month in 2012, with a range of 5–30 cases per month. A clear seasonal pattern was apparent, with the highest number of notifications reported in the winter months, which is consistent with the normal seasonal pattern for this disease (Figure 90). The seasonal trend was more marked in cases aged 5 years or over.

Figure 90: Notifications of invasive meningococcal disease, Australia, 2007 to 2012, by month and year of diagnosis and age group



Vaccination

Coverage of the meningococcal C vaccine has remained at high levels since its introduction, with the latest data indicating that in 2010 almost 94% of Australian children were immunised by 24 months of age.¹⁶³

Laboratory based meningococcal disease surveillance

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

Annual reports of the AMSP are published in CDI. The 2012 data from AMSP showed that 82% of isolates tested demonstrated decreased susceptibility to the penicillin group of antibiotics, and just 1 isolate exhibited resistance to penicillin.⁴¹ While all isolates remained susceptible to third generation cephalosporins and ciprofloxacin, a small number of isolates were reported with an altered susceptibility to rifampicin.

Discussion

In Australia in 2012, IMD has reached its lowest level since national notifications began in 1991. This reduction has been seen most markedly with disease due to serogroup C. However, declines in disease caused by serogroup B are also evident. The slight increase in disease incidence caused by serogroup Y organisms should be closely monitored to determine whether this is an increasing trend.

Tuberculosis

- A total of 1,315 cases of tuberculosis (TB) were notified in 2012.
- Notification rates in the last decade have increased slightly overall.
- TB was predominantly seen in young adults and older males in 2012.

TB is an infection predominantly caused by the bacterium *Mycobacterium tuberculosis*. It is transmitted by airborne droplets produced by people with pulmonary TB during coughing or sneezing. About one-third of the world's population has latent TB infection, which means people have been infected with TB bacteria but are not ill with

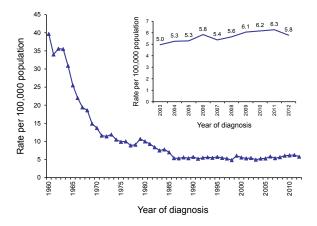
disease and cannot transmit it. Generally healthy people infected with TB bacteria have a 10% lifetime risk of progressing to disease. However, persons with a compromised immune system, such as those living with HIV, suffering from malnutrition or diabetes, or people who use tobacco, have a much higher risk of falling ill.¹⁶⁴

While Australia has one of the lowest rates of TB in the world, the disease remains a public health issue. Further analyses, including the identification of risk groups and reporting on treatment outcomes, are reported in the TB annual reports also published in CDI.¹⁶⁵

Epidemiological situation in 2012

In 2012, a total of 1,315 cases of TB were notified to the NNDSS, representing a rate of 5.8 per 100,000. This was similar to the number of cases reported in the previous year (n=1,399). While the substantial decline in the rate of TB since the 1960s has been maintained, notification rates in the last decade have tended to increase, with the previous 3 years exceeding 6 cases per 100,000 (Figure 91).

Figure 91: Notification rate for tuberculosis, Australia, 1960 to 2012



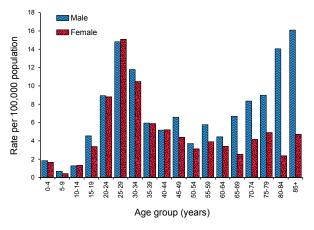
Geographical distribution

New South Wales (n=467), Victoria (n= 366), Queensland (n=176) and Western Australia (n=172) accounted for 90% of all cases of TB diagnosed in Australia. Notification rates were highest in the Northern Territory (11.9 per 100,000), Western Australia (7.1 per 100,000), Victoria (6.5 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 5.8 per 100,000.

Age and sex distribution

In 2012, TB was predominantly seen in young adults and older males. Males accounted for more than half (55%) of the notifications of TB, resulting in a male to female ratio of 1.2:1. The age group with the highest notification rate was the 25–29 years age group (15.0 per 100,000). The highest age and sex specific rates were observed for men aged 85 years or over (16.1 per 100,000) and in women aged 25–29 years (15.1 per 100,000) (Figure 92).

Figure 92: Notification rate for tuberculosis, Australia, 2012, by age group and sex



Appendices

Appendix 1: December estimate of Australian population, 2012, by state or territory

	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
Males	186,598	3,624,791	123,542	2,278,280	820,358	255,419	2,785,448	1,227,524	11,304,018
Females	188,314	3,676,343	111,640	2,287,249	835,941	256,914	2,843,674	1,205,182	11,406,334
Total	374,912	7,301,134	235,182	4,565,529	1,656,299	512,333	5,629,122	2,432,706	22,710,352

Source: ABS 3101.0 Table 4, Estimated Resident Population, State and Territories. Australian.

Appendix 2: December estimate of Australian population, 2012, by state or territory and age

Age				State or	territory				
group	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
0-4	25,195	480,038	18,781	311,523	99,079	31,700	360,280	162,596	1,489,345
5–9	22,507	455,948	17,670	301,108	96,396	31,081	340,546	154,160	1,419,580
10–14	21,139	445,081	16,778	296,587	97,808	32,522	330,483	151,020	1,391,602
15–19	23,923	462,924	16,183	304,996	105,080	33,905	355,158	157,336	1,459,675
20–24	33,667	501,133	19,310	327,882	115,164	31,779	412,430	182,303	1,623,931
25–29	33,720	527,163	22,688	334,501	114,359	30,153	434,210	199,306	1,696,561
30–34	30,258	510,800	20,077	310,952	104,359	28,895	406,697	178,784	1,591,154
35–39	27,700	499,769	18,188	313,767	104,210	30,313	391,802	170,380	1,556,350
40–44	27,400	513,719	18,096	331,264	116,468	35,224	411,666	181,443	1,635,528
45–49	24,654	488,206	15,975	308,608	113,425	34,719	378,544	168,351	1,532,695
50–54	24,334	493,244	15,300	304,242	115,408	37,857	371,151	161,965	1,523,710
55–59	20,982	443,610	12,994	269,621	105,640	35,320	333,395	144,353	1,366,102
60–64	18,487	397,667	9,899	245,049	97,240	32,973	297,065	125,461	1,224,010
65–69	14,272	338,845	6,327	205,263	82,689	27,937	249,660	98,553	1,023,622
70–74	9,712	252,602	3,775	146,489	61,301	20,615	188,476	72,402	755,425
75–79	6,997	195,763	1,943	104,763	48,369	15,171	146,607	53,271	572,906
80–84	5,225	154,209	1,190	78,889	39,517	11,431	115,351	39,964	445,791
85+	5,011	146,462	707	72,701	39,523	10,511	109,000	36,346	420,267
Total	375,183	7,307,183	235,881	4,568,205	1,656,035	512,106	5,632,521	2,437,994	22,728,254

Source : ABS 3101.0 Australian Demographic Statistics Tables, Dec 2012

E128

Annual report

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	4	5	0	6	44	4	£
Barmah Forest virus infection	27	ę	e	579	811	299	1,722	36	612	1,110
Brucellosis	-	0	0	19	6	0	29	69	20	o
Campylobacteriosis	143	6	1	8,085	7,405	0	15,653	53	8,248	7,405
Chlamydial infection	5,760	685	347	34,762	22,673	18,480	82,707	50	41,554	41,153
Cholera	0	0	0	5	0	0	5	100	5	0
Cryptosporidiosis	171	10	8	1,487	1,242	225	3,143	53	1,676	1,467
Dengue virus infection	10	0	2	1,215	276	38	1,541	80	1,227	314
Donovanosis	0	0	0	-	0	0	~	100	-	0
Gonococcal infection	3,529	233	142	4,930	2,533	2,282	13,649	65	8,834	4,815
Haemolytic uraemic syndrome	0	0	0	19	0	-	20	95	19	-
Haemophilus influenzae type b	N	0	0	13	0	0	15	100	15	0
Hepatitis A	0	0	0	148	16	~	165	06	148	17
Hepatitis B (newly acquired)	17	0	2	146	25	З	193	85	165	28
Hepatitis B (unspecified)	154	20	7	2,342	1,902	2,084	6,509	39	2,523	3,986
Hepatitis C (newly acquired)	83	0	-	321	50	1	466	87	405	61
Hepatitis C (unspecified)	620	10	17	3,123	3,474	2,404	9,648	39	3,770	5,878
Hepatitis D	N	0	0	24	4	0	30	87	26	4
Hepatitis E	0	0	0	31	4	0	35	89	31	4
Influenza (laboratory confirmed)	1,258	38	50	19,060	17,221	6,936	44,563	46	20,406	24,157
Japanese encephalitis virus infection	0	0	0	~	0	0	-	100	-	0
Legionellosis	9	0	2	327	42	5	382	88	335	47
Leprosy	0	0	0	4	0	0	4	100	4	0
Leptospirosis	N	0	0	98	14	2	116	86	100	16
Listeriosis	-	-	0	87	З	~	93	96	89	4
Malaria	0	2	0	280	62	4	348	81	282	66
Measles	12	0	0	182	5	0	199	97	194	5
Meningococcal disease (invasive)	22	ю	0	187	11	0	223	95	212	11
Mumps	-	0	0	119	49	31	200	60	120	80

*	Scase
	3
110 110	JIC
	9
5:1	Ξ
(
~	1
Ē	5
1 1100 -	1
	n
<	5
J	5
	2
_	3
	Ę
J	n
0	S
	SPO
Ë	
	đ
102110	
7	2
	1a
	5
	a
-	4
	2
	2
	E
1	
r	<i>a</i>
	1110
	1111
	co)
0	2
	KIN
-	Ġ
4	dd.
<	

CDI

Vol 39

No 1

2015

* Infection with Shiga toxin/verotoxin producing Escherichia coli.

Indigenous status is usually obtained from medical notification and completeness varies by disease and by state and territory. TSI Torres Strait Islander

National Notifiable Diseases Surveillance System, 2012

đ

Acknowledgements

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

Members of the National Surveillance Committee

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

Author details

Coordinating author: Rachael Corvisy Data management: Mark Trungove Bloodborne diseases: Amy Bright, Natasha Wood Gastrointestinal diseases: Debra Gradie, Ben Polkinghorne Quarantinable diseases: Katrina Knope Sexually transmissible infections: Amy Bright, Natasha Wood Vaccine preventable diseases: Kate Pennington, Bethany Morton, Rachel de Kluyver, Nicolee Martin Vectorborne diseases: Katrina Knope Zoonoses: Timothy Sloan-Gardner, Katrina Knope Other bacterial infections: Cindy Toms, Kara Lengyel, Anna Glynn-Robinson With contributions from:

National organisations

Communicable Diseases Network Australia and subcommittees Australian Childhood Immunisation Register Australian Gonococcal Surveillance Programme Australian Meningococcal Surveillance Programme Australian Sentinel Practice Research Network Australian Quarantine Inspection Service The Kirby Institute for Infection and Immunity in Society National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases National Enteric Pathogens Surveillance Scheme OzFoodNet Working Group World Health Organization Collaborating Centre for Reference and Research on Influenza **State and territory health departments**

Communicable Diseases Control, ACT Health, Australian Capital Territory Communicable Diseases Surveillance and Control Unit, NSW Ministry of Health, New South Wales Centre for Disease Control, Northern Territory Department of Health and Community Services, Northern Territory Communicable Diseases Branch, Queensland Health, Queensland 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 and Human Services, 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
BFV	Barmah Forest virus
CDI	Communicable Diseases Intelligence
CDNA	Communicable Diseases Network Australia
CJD	Creutzfeldt-Jakob disease
CRS	congenital rubella syndrome
DENV	dengue virus
HA	haemagglutinin
HI	haemagglutination inhibition
Hib	<i>Haemophilus influenzae</i> type b
HIV	human immunodeficiency virus
HPAIH	highly pathogenic avian influenza in humans
HUS	haemolytic uraemic syndrome
ILI	influenza-like illness
IMD	invasive meningococcal disease
IPD	invasive pneumococcal disease
JEV	Japanese encephalitis virus
KUNV	Kunjin virus
MMR	measles-mumps-rubella
MVEV	Murray Valley encephalitis virus
NAMAC	National Arbovirus and Malaria Advisory Committee
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
NS1	non-structural protein 1
RNA	ribonucleic acid
RRV	Ross River virus
SARS	severe acute respiratory syndrome
STEC	Shiga toxin-producing <i>Escherichia coli</i>
STLC STI(s)	sexually transmissible infections(s)
TB	tuberculosis
TSI	Torres Strait Islander
VPD(s) VTEC	vaccine preventable disease(s)
VIEC VZV	verotoxigenic <i>Escherichia coli</i> varicella zoster virus
VZV WHO	
WHOCC	World Health Organization
WINCE	World Health Organization Collaborating Center
	N 1 0015

CDI Vol 39 No 1 2015

References

- National Health Security Act, 2007. Accessed on November 2009. Available from: <u>http://www.comlaw.gov.au/Details/C2007A00174</u>
- 2. National Health Security (National Notifiable Disease List) Instrument 2008. Available from: <u>http://www.comlaw.gov.au/ComLaw/legislation/</u> LegislativeInstrument1.nsf/0/7162D634C6DD1BAACA 25740B0079D6B8?OpenDocument
- 3. National Health Security Agreement 2008. Accessed on November 2009. Available from: <u>http://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-nhs-agreement.htm</u>
- 4. The Kirby Institute. HIV/AIDS, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report, 2012: The Kirby Institute, the University of New South Wales, Sydney; 2012.
- Klug GM, Boyd A, McGlade A, Stehmann C, Simpson M, Masters CL, et al. Surveillance of Creutzfeldt-Jakob disease in Australia: update to December 2011. Commun Dis Intell 2012;36(2):E174–E179.
- Australian Bureau of Statistics. Australian Demographic Statistics. Canberra: Australian Bureau of Statistics; 2011. Report No.: 3101.0.
- Australian Institute of Health and Welfare. Agestandardised rate—Identifying and definitional attributes. 2005. Accessed on 17 March 2010. Available from: <u>http://meteor.aihw.gov.au/content/index.phtml/</u> itemId/327276
- Graham S, Guy RJ, Donovan B, McManus H, El-Hayek C, Kwan K, et al. Epidemiology of chlamydia and gonorrhea among Indigenous and non-Indigenous Australians, 2000–2009. Med J Aust 2012;197(11):642–646.
- 9. Hammerschlag M. Sexually transmitted diseases in sexually abused children: medical and legal implications. Sex Transm Infect 1998;74(3):167–174.
- 10. Australian Institute of Health and Welfare. National Health Data Dictionary 13.3; 2008.
- Chen MY, Fairley CK, Donovan B. Nowhere near the point of diminishing returns: correlations between chlamydia testing and notification rates in New South Wales. Aust N Z J Public Health 2005;29(3):249–253.
- Hocking J, Fairley C, Counahan M, Crofts N. The pattern of notification and testing for genital *Chlamydia* trachomatis infection in Victoria, 1998–2000: an ecological analysis. Aust N Z J Public Health 2003;27(4):405–408.
- Burckhardt F, Warner P, Young H. What is the impact of change in diagnostic test method on surveillance data trends in Chlamydia trachomatis infection? Sex Transm Infect 2006;82(1):24–30.
- Chen MY, Karvelas M, Sundararajan V, Hocking JS, Fairley CK. Evidence for the effectiveness of a chlamydia awareness campaign: increased population rates of chlamydia testing and detection. *Int J STD AIDS* 2007;18(4):239–243.
- Hammad A, Guy RJ, Fairley C, Wand H, Chen MY, Dickson B, et al. Understanding trends in genital *Chlamydia trachomatis* can benefit from enhanced surveillance: findings from Australia. Sex Transm Infect 2012;88(7):552–557.

- Stephens N, O'Sullivan M, Coleman D, Shaw K. Chlamydia trachomatis in Tasmania 2001–2007: rising notification trends. Aust N Z J Public Health 2010;34(2):120–125.
- National HBV Testing Policy Expert Reference Committee. National hepatitis B testing policy 2012 v1.1. ASHM, Darlinghurst, NSW: Commonwealth of Australia; 2012.
- National HCV Testing Policy Expert Reference Committee. National hepatitis C testing policy 2012 v1.1. ASHM, Darlinghurst, NSW: Commonwealth of Australia; 2012.
- Heymann DL. Control of Communicable Diseases Manual. 19th edn. Washington: American Public Health Association, USA; 2008.
- 20. Australian Technical Advisory Group on Immunisation. The Australian Immunisation Handbook. 10th edn. Canberra, Australia: National Health and Medical Research Council and the Department of Health and Ageing; 2013.
- 21. Hull BP, Dey A, Menzies RI, Brotherton JM, McIntyre PB. Immunisation coverage, 2012. Commun Dis Intell 2014;38(3):E208–231.
- 22. The Kirby Institute. National Blood-borne Virus and Sexually Transmissible Infections Surveillance and Monitoring Report 2013. Sydney, NSW: The Kirby Institute, the University of New South Wales; 2013.
- Butler T, Lim D, Callander D. National Prison Entrants' Bloodborne Virus and Risk Behaviour Survey Report 2004, 2007 and 2010. Prevalence of HIV, hepatitis C, hepatitis B, sexually transmissible infections, and risk behaviours among Australian prison entrants; 2011.
- 24. Gidding HF, Warlow M, MacIntyre CR, Backhouse J, Gilbert GL, Quinn HE, et al. The impact of a new universal infant and school-based adolescent hepatitis B vaccination program in Australia. *Vaccine* 2007;25(51):8637–8641.
- 25. Razali K, Thein HH, Bell J, Cooper-Stanbury M, Dolan K, Dore G, et al. Modelling the hepatitis C virus epidemic in Australia. Drug Alcohol Depend 2007;91(2–3):228–235.
- 26. The Kirby Institute. National Blood-borne Virus and Sexually Transmissible Infections Surveillance and Monitoring Report 2011. Sydney, NSW: The Kirby Institute, the University of New South Wales; 2012.
- 27. OzFoodNet Working Group. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet Network, 2010. Commun Dis Intell 2012;36(3):E213–E241.
- 28. Donnan EJ, Fielding JE, Gregory JE, Lalor K, Rowe S, Goldsmith P, et al. A multistate outbreak of hepatitis A associated with semidried tomatoes in Australia, 2009. *Clin Infect Dis* 2012;54(6):775–781.
- 29. Cumpston JHL. Health and disease in Australia. Canberra: Australian Government Publishing Service; 1989.
- Grattan-Smith PJ, O'Regan WJ, Ellis PS, O'Flaherty SJ, McIntyre PB, Barnes CJ. Rabies. A second Australian case with a long incubation period. *Med J Aust* 1992;156(9):651–654.
- Fenner F, Henderson D, Arita I, Jezek Z, Ladnyi I. Smallpox and its eradication. Geneva, Switzerland; 1988.

- 32. Australian Government Department of Health and Ageing. Guidelines for smallpox outbreak, preparedness, response and management. 2004. Available from: <u>http://www.health.gov.au/internet/main/publishing.nsf/</u> <u>Content/health-publith-publicat-document-metadatasmallpox.htm</u>
- NNDSS Annual Report Writing Group. Australia's notifiable disease status, 2011: Annual report of the National Notifiable Diseases Surveillance System. Commun Dis Intell 2013;37(4):E313–393.
- Miller M, Roche P, Yohannes K, Spencer J, Bartlett M, Brotherton J, et al. Australia's notifiable diseases status, 2003: Annual report of the National Notifiable Diseases Surveillance System. Commun Dis Intell 2005;29(1):1–61.
- 35. World Health Organization. Cumulative number of confirmed human cases of avian influenza A(H5N1) reported to WHO. 2013. Available from: <u>http://www.who.int/influenza/human_animal_interface/H5N1_cumulative_table_archives/en/index.html</u>
- Curran M, Harvey B, Crerar S. Annual report of the National Notifiable Diseases Surveillance System, 1996. Commun Dis Intell 1997;21(20):281–307.
- Forssman B, Mannes T, Musto J, Gottlieb T, Robertson G, Natoli JD, et al. Vibrio cholerae O1 El Tor cluster in Sydney linked to imported whitebait. Med J Aust 2007;187(6):345–347.
- Regan DG, Wilson DP, Hocking JS. Coverage is the key for effective screening of Chlamydia trachomatis in Australia J Infect Dis 2008;198(3):349–358.
- Victoria Department of Health. Blue book: Guidelines for the control of infectious diseases. 2009. Accessed on 3 April 2014. Available from: <u>http://ideas.health.vic.</u> <u>gov.au/bluebook/</u>
- 40. Bowden FJ, on behalf of the National Donovanosis Eradication Advisory Committee. Donovanosis in Australia: going, going... Sex Transm Infect 2005;81(5):365–366.
- 41. Lahra MM, Enriquez RP. Australian Meningococcal Surveillance Programme annual report, 2012. Commun Dis Intell 2013;37(3):E224–E232.
- 42. The Kirby Institute. HIV, viral hepatitis and sexually transmissable infections in Australia Annual Surveillance Report 2013. Sydney: The Kirby Institute, The University of New South Wales; 2013.
- 43. Government of Western Australia. Department of Health. The Epidemiology of Notifiable Sexually Transmitted Infections and Blood-Borne Viruses in Western Australia 2009. Perth: Department of Health, Western Australia; 2010.
- Ward J, Guy RJ, Akre S, Middleton M, Giele C, Su J, et al. Epidemiology of syphilis in Australia: moving toward elimination of infectious syphilis from Aboriginal and Torres Strait Islander communities? *Med J Aust* 2011;194(10):525–529.
- Chiu C, Dey A, Wang H, Menzies R, Deeks S, Mahajan D, et al. Vaccine Preventable Diseases in Australia, 2005 to 2007. Commun Dis Intell 2010;34(Suppl):S1–S172.
- Pillsbury A, Quinn HE, McIntyre PB. Australian vaccine preventable disease epidemiological review series: pertussis, 2006–2012. Commun Dis Intell 2014;38(3):E179–194.
- Chiew M, Dey A, Martin N, Wang H, Davis S, McIntyre PB. Australian vaccine preventable disease epidemiological review series: measles 2000–2011. Commun Dis Intell 2015;39(1):E1–E9.

- Bag SK, Dey A, Wang H, Beard F. Australian vaccine preventable disease epidemiological review series: mumps 2008–2012. Commun Dis Intell 2015;39(1):E10–E18.
- Chan J, Dey A, Wang H, Martin N, Beard F. Australian vaccine preventable disease epidemiological review series: rubella 2008–2012. Commun Dis Intell 2015;39(1):E19–E26.
- 50. Punpanich W, Chotpitayasunondh T. A review on the clinical spectrum and natural history of human influenza. *Int J Infect Dis* 2012;16(10):e714–e723.
- Carrat F, Vergu E, Ferguson NM, Lemaitre M, Cauchemez S, Leach S, et al. Time lines of infection and disease in human influenza: a review of volunteer challenge studies. Am J Epidemiol 2008;167(7):775–785.
- 52. Mauskopf J, Klesse M, Lee S, Herrera-Taracena G. The burden of influenza complications in different high-risk groups: a targeted literature review. J Med Economics 2013;16(2):264–277.
- 53. Sosin DM. Draft framework for evaluating syndromic surveillance systems. J Urban Health 2003;80(Suppl 1):8–13.
- 54. Turbelin C, Souty C, Pelat C, Hanslik T, Sarazin M, Blanchon T, et al. Age distribution of influenza like illness cases during post-pandemic A(H3N2): comparison with the twelve previous seasons, in France. PLoS One 2013;8(6):e65919.
- 55. Schepetiuk SK, T. K. The use of MDCK, MEK and LLC-MK2 cell lines with enzyme immunoassay for the isolation of influenza and parainfluenza viruses from clinical specimens. *J Virol Methods* 1993;42(2–3):241–250.
- Review of the 2012 winter influenza season, southern hemisphere. Wkly Epidemiol Rec 2012;87(44):422–431.
- 57. Stocks N. Australian Sentinel Practice Research Network. In: Surveillance systems reported in Communicable Diseases Intelligence, 2013. Commun Dis Intell 2013;37(1):E62.
- Carlson SJ, Dalton CB, Butler MT, Fejsa J, Elvidge E, Durrheim DN. Flutracking weekly online community survey of influenza-like illness annual report, 2011 and 2012. Commun Dis Intell 2013;37(4):E398–E406.
- Health Protection NSW. Influenza epidemiology report: December 2012 and summary for 2012. In: Influenza Monthly Epidemiology Report, NSW. 2012 Influenza Reports: NSW Government; 2013.
- 60. Northern Territory Department of Health and Community Services. Comments on notifications. Northern Territory Disease Control Bulletin 2012;19(1):29.
- Hjuler T, Poulsen G, Wohlfahrt J, Kaltoft M, Biggar RJ, Melbye M. Genetic susceptibility to severe infection in families with invasive pneumococcal disease. *Am J Epidemiol* 2008;167(7):814–819.
- 62. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. Significant events in pneumococcal vaccination practice in Australia. Accessed on 20 January 2014. This document was available from: <u>http://www.ncirs.edu.au/ immunisation/history/Pneumococcal-history-July-2014.</u> <u>pdf</u> at the time of wrting.
- 63. Bareja C, Toms C, Lodo K, de Kluvyer R, Enhanced Invasive Pneumococcal Disease Surveillance Working Group. Invasive pneumococcal disease in Australia, 2009 and 2010. Commun Dis Intell 2015;39(2):In Press.

- 64. Centers for Disease Control and Prevention. Measles. In: Atkinson W, Wolfe C, Hamborsky J, eds. *Epidemiology* and prevention of vaccine preventable diseases. 12th edn. Washington, D.C.: Public Health Foundation; 2011.
- 65. World Health Organization. Measles. Geneva; 2014.
- 66. Strebel P, Papania M, Dayan G, Halsey N. Vaccines. Philadelphia: Saunders Elsevier; 2008.
- Leuridan E, Hens N, Hutse V, leven M, Aerts M, Van Damme P. Early waning of maternal measles antibodies in era of measles elimination: longitudinal study. *BMJ* 2010;340:10.1136/bmj.c1626.
- Rosewell A, Reinten-Reynolds T, Spokes P. Measles in NSW, 2002–2011. N S W Public Health Bull 2012;23(9–10):201–207.
- 69. Gidding H, Wood J, MacIntyre CR, Kelly H, Lambert SB, Gilbert GL, et al. Sustained measles elimination in Australia and priorities for long-term maintenance. Vaccine 2007;25(18):3574–3580.
- Gidding HF, Gilbert GL. Measles immunity in young Australian adults. Commun Dis Intell 2001;25(3):133–136.
- Heywood AE, Gidding HF, Riddell MA, McIntyre PB, MacIntyre CR, Kelly HA. Elimination of endemic measles transmission in Australia. Bull World Health Organ 2009;87(1):64–71.
- 72. Gay NJ, De Serres G, Farington P, Redd SB, Papania MJ. Assessment of the status of measles elimination from reported outbreaks: United States, 1997–1999. J Infect Dis 2004;189(Suppl 1):S36–S42.
- 73. Plotkin SA. The history of rubella and rubella vaccination leading to elimination. *Clin Infect Dis* 2006;43(Suppl 3):S164–S168.
- 74. Aratchige PE, McIntyre PB, Quinn HE, Gilbert GL. Recent increases in mumps incidence in Australia: the "forgotten" age group in 1998 Australian Measles Control Campaign. Med J Aust 2008;189(8):4.
- 75. Gupta R, Best J, MacMahon E. Mumps and the UK epidemic 2005. *BMJ* 2005;330:1132–1135.
- Deeks S, Lim G, Simpson M, Gagné L, Kristjanson E, Fung C, et al. An assessment of mumps vaccine effectiveness by dose during an outbreak in Canada. Can Med Assoc J 2011;183(9):1014–1020.
- 77. Demicheli V, Rivetti A, Debalini M, Di Pietrantonj C. Vaccines for measles, mumps and rubella in children. Chochrane Database Sys Rev 2012;2:CD004407.
- Dayan GH, Quinlisk MP, Parker AA, Barskey AE, Harris ML, Schwartz JM, et al. Recent resurgence of mumps in the United States. N Engl J Med 2008;358(15):1580–1589.
- 79. Whelan J, Van Binnendijk R, Greenland K, Fanoy E, Kharqi M, Yap K, et al. Ongoing mumps outbreak in a student population with high vaccination coverage, Netherlands. *Euro Surveill* 2010;15(17):pii:19554.
- Cohen C, White JM, Savage EJ, Glynn JR, Choi Y, Andrews N, et al. Vaccine effectiveness estimates, 2004–2005 mumps outbreak, England. *Emerg Infect Dis* 2007;13(1):12–17.
- Pertussis. In: Atkinson W, Wolfe C, Hamborsky J, eds. Epidemiology and prevention of vaccine preventable diseases. 12th edn. Washington, DC: Public Health Foundation, Centers for Disease Control and Prevention; 2011.
- Britt H, Miller GC, Charles J, Henderson J, Bayram C, Pan Y, et al. General practice activity in Australia 2009–10. Canberra: Australian Institute of Health and Welfare; 2010.

- 83. Communicable Diseases Network Australia. Series of National Guidelines: Pertussis. 2013. Available from: <u>https://www.health.gov.au/internet/main/publishing.</u> <u>nsf/Content/cdna-song-pertussis.htm</u>
- 84. Quinn HE, McIntyre PB. Pertussis epidemiology in Australia over the decade 1995–2005—trends by region and age group. Commun Dis Intell 2007;31(2):205–215.
- Quinn HE, Mahajan D, Hueston L, Campbell P, Menzies RI, Gilbert GL, et al. The seroepidemiology of pertussis in NSW: fluctuating immunity profiles related to changes in vaccination schedules. N S W Public Health Bull 2011;22(11–12):224–229.
- Kaczmarek M, Lambert S, Kelly H, Ware R, Valenti L, Britt H. Seven-fold rise in likelihood of pertussis-test requests during Australian GP encounters, 2000–2011. In: 13th National Immunisation Conference. Darwin Convention Centre, Darwin, NT: Public Health Association of Australia; 2012.
- 87. Sheridan S, Ware R, Grimwood K, Lambert S. Number and order of whole cell pertussis vaccines in infancy and disease protection. *JAMA* 2012;308(5):454–456.
- Misegades L, Winter K, Harriman K, Talarico J, Messonnier NE, Clark T, et al. Association of childhood pertussis with receipt of 5 doses of pertussis vaccine by time since last vaccine dose, California, 2010. JAMA 2012;308(20):2126–2132.
- Tartof SY, Lewis M, Kenyon C, White K, Osborn A, Liko J, et al. Waning immunity to pertussis following 5 doses of DTaP. *Pediatrics* 2013;131(4):e1047–e1052.
- 90. Quinn HE. Pertussis vaccine effectiveness in Australia [abstract]. In: National pertussis workshop: strategies to prevent severe pertussis in the next decade. Sydney Australia: National Centre for Immunisation and Research of Vaccine Preventable Diseases; 2011.
- 91. Australian Technical Advisory Group on Immunisation. Bulletin 44th meeting 24–25 February 2011.
- 92. Roberts J, Hobday L, Ibrahim A, Aitken T, Thorley B. Annual report of the Australian National Enterovirus Reference Laboratory 2012. Commun Dis Intell 2013;37(2):E97–E104.
- Song N, Gao Z, Wood JG, Hueston L, Gilbert GL, MacIntyre CR, et al. Current epidemiology of rubella and congenital rubella syndrome in Australia: progress towards elimination. Vaccine 2012;30(27):4073–4078.
- Nelson MR, Britt HC, Harrison CM. Evidence of increasing frequency of herpes zoster management in Australian general practice since the introduction of a varicella vaccine. *Med J Aust* 2010;193(2):110–113.
- 95. Stein AN, Britt H, Harrison C, Conway EL, Cunningham A, Macintyre CR. Herpes zoster burden of illness and health care resource utilisation in the Australian population aged 50 years and older. Vaccine 2009;27(4):520–529.
- 96. Carville KS, Grant KA, Kelly HA. Herpes zoster in Australia. *Epidemiol Infect* 2012;140(04):599–601.
- Knope K, Whelan P, Smith D, Johansen C, Moran R, Doggett S, et al. Arboviral diseases and malaria in Australia, 2010–11: annual report of the National Arbovirus and Malaria Advisory Committee. Commun Dis Intell 2013;37(1):E1–E20.
- Mackenzie JS, Lindsay MD, Coelen RJ, Broom AK, Hall RA, Smith DW. Arboviruses causing human disease in the Australasian zoogeographic region. *Arch Virol* 1994;136(3–4):447–467.

- 99. Russell RC, Dwyer DE. Arboviruses associated with human disease in Australia. *Microbes Infect* 2000;2(14):1693–1704.
- 100. Viennet E, Knope K, Faddy H, Williams C, Harley D. Assessing the threat of chikungunya emergence in Australia. Commun Dis Intell 2013;37(2):E136–E143.
- 101. Communicable Diseases Network Australia. Australian National Notifiable Diseases Case Definitions. 2013. Accessed on 5 September 2013. Available from: <u>http://www.health.gov.au/casedefinitions</u>
- 102. Therapeutic Goods Administration. Product recall, Panbio Barmah Forest Virus IgM ELISA. An *in vitro* diagnostic medical device (IVD). Recall no. RC-2013-RN-00967-1,13/09/2013. 2013. Accessed on 6 May 2014. Available from: <u>http://www.tga.gov.au/</u> <u>SARA/arn-detail.aspx?k=RC-2013-RN-00967-1</u>
- 103. Guzman MG, Kouri G, Martinez E, Bravo J, Riveron R, Soler M, et al. Clinical and serologic study of Cuban children with dengue hemorrhagic fever/dengue shock syndrome (DHF/DSS). Bull Pan Am Health Organ 1987;21(3):270–279.
- 104. Vaughn DW, Green S, Kalayanarooj S, Innis BL, Nimmannitya S, Suntayakorn S, et al. Dengue viremia titer, antibody response pattern, and virus serotype correlate with disease severity. J Infect Dis 2000;181(1):2–9.
- 105. Mackenzie JS, Gubler DJ, Petersen LR. Emerging flaviviruses: the spread and resurgence of Japanese encephalitis, West Nile and dengue viruses. Nature Medicine 2004;10(12 Suppl):S98–S109.
- 106. Hanna JN, Ritchie SA, Richards AR, Humphreys JL, Montgomery BL, Ehlers GJ, et al. Dengue in north Queensland, 2005–2008. Commun Dis Intell 2009;33(2):198–203.
- 107. Queensland Health. Queensland Dengue Management Plan 2010–2015, 2011. Queensland: Queensland Health.
- 108. Knope K, National Arbovirus and Malaria Advisory Committee, Giele C. Increasing notifications of dengue related to overseas travel, 1991 to 2012. Commun Dis Intell 2013;37(1):E55–E59.
- 109. Hanna JN, Ritchie SA, Phillips DA, Lee JM, Hills SL, van den Hurk AF, et al. Japanese encephalitis in north Queensland, Australia, 1998. Med J Aust 1999;170(11):533–536.
- 110. Roche S WR, Garner M, East I, Paskin R, Moloney B, Carr M, Kirkland P. Descriptive overview of the 2011 epidemic of arboviral disease in horses in Australia. *Aust Vet J* 2013;91(1–2):5–13.
- Knox J, Cowan RU, Doyle JS, Ligtermoet MK, Archer JS, Burrow JN, et al. Murray Valley encephalitis: a review of clinical features, diagnosis and treatment. *Med J Aust* 2012;196(5):322–326.
- 112. Frost MJ, Zhang J, Edmonds JH, Prow NA, Gu X, Davis R, et al. Characterization of virulent West Nile virus Kunjin strain, Australia, 2011. *Emerg Infect Dis* 2012;18(5):792–800.
- 113. Plasmodium knowlesi malaria in humans is widely distributed and potentially life threatening. Clin Infect Dis 2008;46(2):165–171. doi: 110.1086/524888.
- 114. World Health Organization. Synopsis of the world malaria situation in 1981. Wkly Epidemiol Rec 1983;58(26):197–199.

- 115. Leder K, Torresi J, Brownstein JS, Wilson ME, Keystone JS, Barnett E, et al. Travel-associated illness trends and clusters, 2000–2010. Emerg Infect Dis 2013;19(7):1049–1073.
- 116. Gray TJ, Trauer JM, Fairley M, Krause VL, Markey PG. Imported malaria in the Northern Territory, Australia—428 consecutive cases. Commun Dis Intell 2012;36(1):107–113.
- 117. Preston-Thomas A, Gair RW, Hosking KA, Devine GJ, Donohue SD. An outbreak of *Plasmodium falciparum* malaria in the Torres Strait. Commun Dis Intell 2012;36(2):E180–E185.
- Hanna JN, Ritchie SA, Eisen DP, Cooper RD, Brookes DL, Montgomery BL. An outbreak of *Plasmodium vivax* malaria in Far North Queensland, 2002. *Med J Aust* 2004;180(1):24–28.
- World Health Organization. Zoonoses. Technical report series no. 169. Geneva, Switzerland: World Health Organization; 1959.
- 120. Taylor LH, Latham SM, Woolhouse ME. Risk factors for human disease emergence. *Philos Trans R Soc Lond B Biol Sci* 2001;356(1411):983–989.
- 121. Jones KE, Patel NG, Levy MA. Global trends in emerging infectious diseases. *Nature* 2008(451):990–994.
- 122. Woolhouse MEJ, Gowtage-Sequeria S. Host range and emerging and re-emerging pathogens. *Emerg Infect Dis* 2005;11(12):1842–1847.
- 123. World Health Organization. Report of the WHO/FAO/ OIE joint consultation on emerging zoonotic diseases. Geneva, Switzerland: World Health Organization; 2004.
- 124. Animal Health Australia. Animal Health in Australia 2012. Canberra; 2013.
- 125. Kolbe A, Yuen M, Doyle B. A case of human cutaneous anthrax. *Med J Aust* 2006;185(5):281–282.
- 126. Fielding J. Zoonoses: Anthrax. Vic Infect Dis Bull 2007;10(2):47.
- 127. NSW Department of Health. Communicable Diseases Report, NSW, January and February 2010. *N S W Public* Health Bull 2010;21(3–4):103–107.
- 128. Calisher CH, Ellison JA. The other rabies viruses: The emergence and importance of lyssaviruses from bats and other vertebrates. *Travel Med Infect Dis* 2012;10(2):69–79.
- 129. Fraser GC, Hooper PT, Lunt RA, Gould AR, Gleeson LJ, Hyatt AD, et al. Encephalitis caused by a lyssavirus in fruit bats in Australia. *Emerg Infect Dis* 1996;2(4):327–331.
- 130. Hooper PT, Lunt RA, Gould AR, Samaratunga H, Hyatt AD, Gleeson LJ, et al. A new lyssavirus — the first endemic rabies-related virus recognized in Australia. Bulletin de l'Institut Pasteur 1997;95(4):209–218.
- 131. Communicable Diseases Network Australia. Series of National Guidelines: Rabies virus and other lyssavirus including Australian bat lyssavirus) exposures and infections. 2013. Accessed on 7 June 2013. Available from: <u>http://www.health.gov.au/internet/main/publishing.nsf/</u> <u>Content/cdna-song-abvl-rabies.htm</u>
- 132. Allworth A, Murray K, Morgan J. A human case of encephalitis due to a lyssavirus recently identified in fruit bats. Commun Dis Intell 1996;20(24):504.
- 133. Hanna JN, Carney IK, Smith GA, Tannenberg AEG, Deverill JE, Botha JA, et al. Australian bat lyssavirus infection: a second human case, with long incubation period. *Med J Aust* 2000;172(12):597–599.

- 134. Francis JR, Nourse C, Vaska VL, Calvert S, Northill JA, McCall B, et al. Australian bat lyssavirus in a child: The first reported case. *Pediatrics* 2014;133(4):e1063–1067.
- 135. Queensland Department of Agriculture Fisheries and Forestry. Australian bat lyssavirus veterinarian communiqué, 21 May 2013. Accessed on 7 July 2013. Available from: <u>http://www.vsbsa.org.au/images/File/ABLV%20</u> vet%20communique%2021%20May%202013.pdf
- 136. Queensland Department of Agriculture Fisheries and Forestry. Australian bat lyssavirus update communiqué 7 June 2013. Accessed on 7 July 2013. Available from: <u>http://www.ava.com.au/sites/default/files/ Lyssavirus%20communique%20130607.pdf</u>
- Australian Bat Lyssavirus Focus Group. Australian Bat Lyssavirus report, December 2012: Australian Wildlife Health Network; 2012.
- Eales KM, Norton RE, Ketheesan N. Brucellosis in northern Australia. Am J Trop Med Hyg 2010;83(4):876–878.
- 139. World Health Organization. Human leptospirosis : guidance for diagnosis, surveillance and control. Geneva, Switzerland: World Health Organization; 2003.
- 140. Levett PN. Leptospirosis. Clin Microbiol Rev 2001;14(2):296–326.
- 141. O'Leary FM, Hunjan JS, Bradbury R, Thanakrishnan G. Fatal leptospirosis presenting as musculoskeletal chest pain. Med J Aust 2004;180(1):29–31.
- 142. Smith JK, Young MM, Wilson KL, Craig SB. Leptospirosis following a major flood in Central Queensland, Australia. *Epidemiol Infect* 2012;141(3):1–6.
- 143. Queensland Health. Statewide Weekly Communicable Diseases Surveillance Report, 4 April 2011: Epidemiology, Surveillance and Research Unit; 2011.
- 144. Beeckman DS, Vanrompay DC. Zoonotic Chlamydophila psittaci infections from a clinical perspective. Clin Microbiol Infect 2009;15(1):11–17.
- 145. Deschuyffeleer TP, Tyberghien LF, Dickx VL, Geens T, Saelen JM, Vanrompay DC, et al. Risk assessment and management of *Chlamydia psittaci* in poultry processing plants. Ann Occup Hyg 2012;56(3):340–349.
- 146. Department of Health Victoria. Communicable disease surveillance October–December 2011. Victorian Infectious Diseases Bulletin 2012;15(1):21–39.
- 147. Department of Health Victoria. Communicable disease surveillance October–December 2012. Victorian Infectious Diseases Bulletin 2013;16.
- 148. McCaul TF, Williams JC. Developmental cycle of Coxiella burnetii: structure and morphogenesis of vegetative and sporogenic differentiations. J Bacteriol 1981;147(3):1063–1076.
- 149. Lowbridge CP, Tobin S, Seale H, Ferson MJ. Notifications of Q fever in NSW, 2001–2010. N S W Public Health Bull 2012;23(1–2):31–35.
- 150. Bell M, Patel M, Sheridan J. Q fever vaccination in Queensland abattoirs. Commun Dis Intell 1997;21(3):29–31.

- 151. Lin M, Delpech V, McAnulty J, Campbell-Lloyd S. Notifications of Q fever in New South Wales, 1991–2000: EpiReview. N S W Public Health Bull 2001;12(6):172–175.
- 152. Gidding HF, Wallace C, Lawrence G, McIntyre PB. Australia's National Q Fever Vaccination Program. Vaccine 2009;27(14):2037–2041.
- 153. Ellis J, Oyston PC, Green M, Titball RW. Tularemia. *Clin Microbiol Rev* 2002;15(4):631–646.
- 154. Jackson J, McGregor A, Cooley L, Ng J, Brown M, Ong CW, et al. Francisella tularensis subspecies holarctica, Tasmania, Australia, 2011. Emerg Infect Dis 2012;18(9):1484–1486.
- 155. Veitch M. The public health response to tularaemia in Tasmania. Communicable Disease Control Conference 2013. Canberra, Australia; 2013.
- 156. Health Protection NSW. Year in review: Health Protection in NSW, 2012. NSW Public Health Bulletin 2013;24(3):107–118.
- 157. Victorian Department of Health. Communicable disease surveillance, Victoria, October–December 2012. Victorian Infectious Diseases Bulletin 2013;16(1):27–47.
- 158. Campese C, Bitar D, Jarraud S, Maine C, Forey F, Etienne J, et al. Progress in the surveillance and control of Legionella infection in France, 1998–2008. Int J Infect Dis 2011;15(1):e30–37.
- Hartemann P, Hautemaniere A. Legionellosis prevention in France. Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz 2011;54(6):724–727.
- Legionellosis. In: Heymmann D, ed. Control of Communicable Diseases Manual. 19th edn. Washington: American Public Health Association; 2008. p. 337–340.
- 161. Communicable Diseases Network Australia. Guidelines for the early clinical and public health management of meningococcal disease in Australia. Revised edition 2007. Accessed on 6 November 2013. Available from: http://webarchive.nla.gov.au/gov/20140801082945/ http://www.health.gov.au/internet/main/publishing.nsf/ Content/cda-pubs-other-mening-2007.htm
- 162. Communicable Diseases Network Australia. Surveillance Case Definitions for the Australian National Notifiable Diseases Surveillance System. 2004. Accessed on 25 June 2012. Available from: <u>http://www.health.gov.</u> <u>au/casedefinitions</u>
- 163. Hull B, Dey A, Menzies R, McIntyre P. Annual immunisation coverage report, 2010. Commun Dis Intell 2013;37(1):E21–E39.
- 164. World Health Organization. Tuberculosis. Fact Sheet number 104. 2014. Accessed on 13 October 2014. Available from: <u>http://www.who.int/mediacentre/</u><u>factsheets/fs104/en/</u>
- 165. Toms C, Stapledon R, Waring J, Douglas P, National Tuberculosis Advisory Committee for the Communicable Diseases Network Australia. Tuberculosis notifications in Australia, 2012 and 2013. Commun Dis Intell 2015;39(2):In Press.