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Original article

Australian vaccine preventable disease epidemiological review series: measles 2000–2011

May Chiew, Aditi Dey, Nicolee Martin, Han Wang, Stephanie Davis, Peter B McIntyre

Abstract

Background: Since the introduction of measles vaccine to the vaccination schedule, the burden of measles has substantially fallen in Australia. Despite this, a number of recent measles outbreaks have occurred. The aim of this study was to examine the burden of measles in Australia using notification, hospitalisation and mortality data with the objectives of setting a baseline for comparison prior to the introduction of the combined measles-mumps-rubella-varicella vaccine.

Methods: Data were obtained from the Australian National Notifiable Diseases Surveillance System, the National Hospital Morbidity Database and the National Mortality Database to obtain notification, hospitalisation and death data, respectively from 2000 to 2011. Rates were calculated and compared over time by age group and jurisdiction.

Results: Since 1993, measles notifications have fallen considerably in Australia. However, between 2000 and 2011, measles notification rates and hospitalisation rates fluctuated. Between 2000 and 2011, there were 990 measles notifications in Australia. The average annual notification rate was 0.4 per 100,000 population. Children aged 0–4 years were the most susceptible group, particularly infants less than 1 year of age (average annual rate, 1.6 per 100,000 population). High incidence was also observed in adolescents (average annual rate, 0.7 per 100,000 population) and young adults (average annual rate, 0.8 per 100,000 population). Jurisdictional variation occurred with differing patterns of notifications and hospitalisations.

Conclusions: Although a marked reduction in measles notifications and hospitalisations has occurred in the past decade, susceptible individuals should be vaccinated to prevent outbreaks and to maintain a low incidence of measles and Australia's elimination status. *Commun Dis Intell* 2015;39(1):E1–E9.

Keywords: epidemiology; measles; vaccine preventable diseases

Introduction

This report is part of an ongoing series produced by the National Centre for Immunisation Research and Surveillance documenting vaccine preventable disease epidemiological trends.

Measles is an acute and highly infectious disease caused by a paramyxovirus. Infection is characterised by cough, coryza, fever and the onset of a generalised maculopapular rash several days after initial symptom onset. Although most individuals recover from infection, complications can occur including otitis media, pneumonia, croup, diarrhoea, encephalitis and, very rarely, subacute sclerosing panencephalitis.[1]

A live attenuated measles vaccine was first licensed in 1968 in Australia.[2] Although the vaccine was introduced in all states and territories of Australia by 1972, it was not included in the first national childhood vaccination schedule at 12 months of age until 8 years later.[2] A 2nd dose was introduced for those aged 10–16 years at the end of 1993, following a large measles epidemic that resulted in approximately 10,000 cases and 4 deaths.[3,4] By 1995, measles incidence and hospitalisations had decreased substantially and the last death was recorded during this year.[5–8]

Despite these reductions in measles incidence, modelling studies indicated that gaps in immunity due to suboptimal vaccine coverage and the large gap between the 1st and 2nd doses of vaccine were likely to result in further outbreaks, leading to the 1998 National Measles Control Campaign.[5] The campaign included school-based mass vaccination of children 5–12 years of age[8] and lowering the age for the 2nd dose to 4–5 years.[3] Following the campaign, population immunity increased substantially.[8] In 1999, following a large Victorian outbreak that predominantly affected young adults, the Australian Government funded measles vaccine for young adults aged 18–30 years.[9] However, this campaign had suboptimal uptake; no subsequent increase in immunity among young adults could be demonstrated and outbreaks continued in

the young adult age group.[10] Despite this, overall measles incidence continued to fall in Australia since 2005.[3]

In 2010, coverage for the 1st dose of a measles-containing vaccine, usually given as measles-mumps-rubella vaccine (MMR), was estimated at 94% for children at 24 months of age, compared with 90% for the 2nd dose at 60 months of age.[11] Based on some degree of under-reporting to the Australian Childhood Immunisation Register, these are likely to be minimum estimates[12] and from July 2013, a combined measles-mumps-rubella-varicella vaccine (MMR-V) was included in the National Immunisation Program for children aged 18 months, which is expected to increase 2nd dose measles coverage.[13]

This epidemiologic review documents age-specific trends in measles infection using data on measles notifications, hospitalisations and mortality with the objectives of setting a baseline for comparison prior to the introduction of the combined MMR-V vaccine.

Methods

Data sources

Notifications

Measles is a notifiable condition in all jurisdictions in Australia and both confirmed and probable cases are notified to health authorities.[14] A confirmed case of measles requires laboratory definitive evidence or clinical evidence with an established epidemiological link. Alternatively, a probable case of measles requires clinical and laboratory evidence suggesting measles infection.[15]

De-identified national data for both confirmed and probable measles notifications were obtained from the National Notifiable Diseases Surveillance System (NNDSS) from January 2000 to December 2011.[16] Notification data from 1993 to 1999 were also obtained to show historical trends prior to the reporting period of the study. The following fields were included in the analysis: date of diagnosis, age at onset, state or territory of residence and genotype of measles specimen. The date of diagnosis is a NNDSS derived field and is defined as the onset date (if known) or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date.

Hospitalisations

Data coded for measles using the International Statistical Classification of Diseases and Related Health Problems, Tenth Revision, Australian Modification International Classification of Disease (ICD)-10-AM/ICD-10 code B05 were obtained from the Australian Institute of Health and Welfare National Hospital Morbidity Database.[17] The database collects information on patients admitted to public and private hospitals in Australia. Hospital admissions between January 2000 and December 2011 were analysed. Our analysis includes measles hospitalisations recorded in any field as well as by principal diagnosis (i.e. the diagnosis primarily responsible for prompting an episode of admitted or residential care or presentation at a healthcare institution). The variables used in the analysis included date of admission, diagnosis, age on admission, state or territory of residence, length of stay and the mode of separation (i.e. the process by which an admitted patient is discharged e.g. discharge, death, transfer or change in care type).

Deaths

De-identified aggregated mortality data were obtained from the Australian Bureau of Statistics (ABS) National Mortality Database for this reporting period.[17] Registered deaths with measles as the underlying cause based on the cause of death classification ICD-10 were analysed. A more detailed explanation of the methodologies used has been previously described.[8]

No ethics approval was sought as de-identified aggregate population based data were used in this epidemiological review.

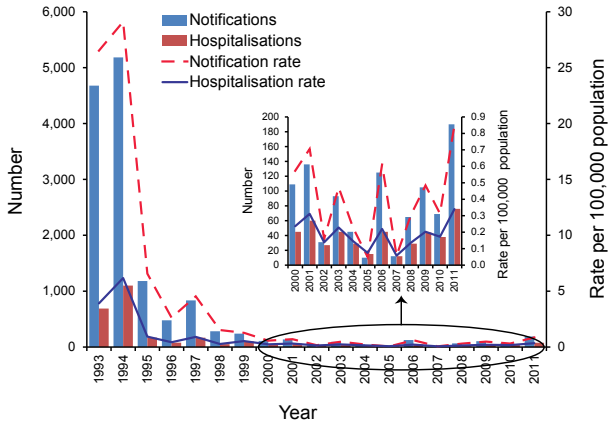
Data analysis

Crude and age-specific annual rates were calculated using mid-year population estimates obtained from the ABS.[18] Median length of stay was calculated for hospital admissions by days. The analyses focused on the most recent period, January 2007 to December 2011.

Results

Historical measles data show that notified cases peaked at 4,678 in 1993 and 5,184 in 1994, and since then measles notifications dropped dramatically to as low as 10 cases during 2005 (Figure 1). For the reporting period of the study, between 2000 and 2011, small peaks were observed in 2001 (n = 136; 0.7 per 100,000 population), 2006 (n = 125; 0.6 per 100,000 population) and, more recently 2011 (n=190; 0.9 per 100,000 population). The number of hospitalisations also decreased since 1993, with peaks corresponding to notifications in 2001 (n=60; 0.3 per 100,000 population); 2006 (n = 45; 0.2 per 100,000 population) and 2011 (n = 76; 0.3 per 100,000 population).

Figure 1: Number and rates of notifications and hospitalisations,* Australia, 1993 to 2011, by year of diagnosis or admission



* Hospitalisations (all diagnoses of measles).

Source: Australian Institute of Health and Welfare National Hospital Morbidity Database and the National Notifiable Diseases Surveillance System.

Age distribution

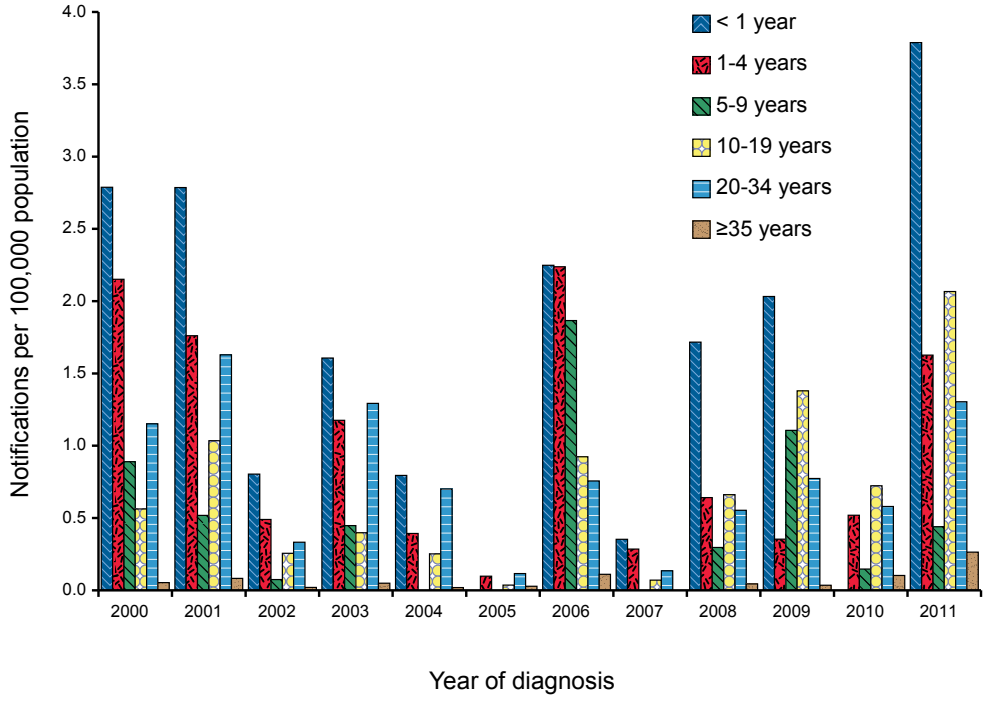
Between 2000 and 2011, no specific age group consistently had the highest annual notification rate, which fluctuated considerably due to the small number of cases (Figure 2). Infants less than 12 months of age, who were not eligible to receive the vaccine, had the highest notification rates in most years, except during 2005 and 2010, when no infant cases were notified. In 2011, the notification rate in infants was 3.8 per 100,000 population, the highest notification rate of all age groups during the 12-year period. During most years, children 1–4 years of age, who were eligible for 2 doses of the vaccine, had the second highest notification rate followed by adolescents aged 10–19 years of age and young adults aged 20–34 years since 2009 (Figure 2).

State and territory variations

Over the time period studied, there was considerable variation in notification rates among state and territories (Figure 3). Between 2000 and 2011, the 3 most populous states (New South Wales, Victoria and Queensland) accounted for 76% of all measles cases nationwide. New South Wales reported the highest rate of measles notifications, of which 27% occurred during 2011 (1.2 per 100,000 population). Only in 2009 did all states and territories report at least 1 measles case. The trend in hospitalisation rates follows a similar pattern to the rates of notifications (Figure 3).

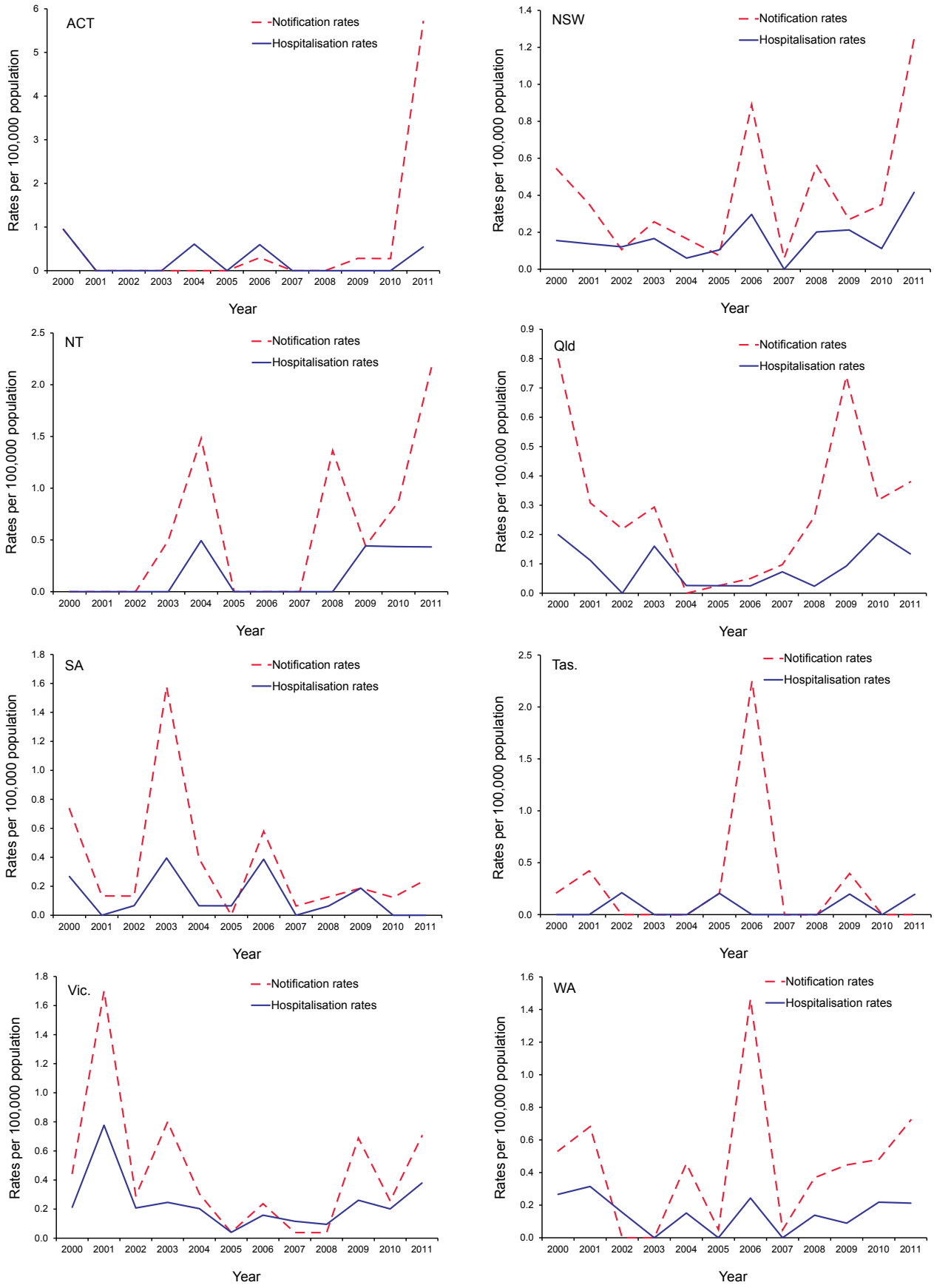
Overall, notification and hospitalisation rates remained low throughout the time period considered. Nationally, notification rates remained below 1 per 100,000 population for 2007 to 2011, except in New South Wales, the Northern Territory and the Australian Capital Territory in 2011.

Figure 2: Measles notification rates, Australia 2000–2011, by age group and year of diagnosis



Source: National Notifiable Diseases Surveillance System.

Figure 3: Measles notification and hospitalisation rates, 2000 to 2011,*,† by state or territory



* Scales vary between jurisdictions.

† Hospitalisations by principal diagnosis only.

Source: Australian Institute of Health and Welfare National Hospital Morbidity Database and the National Notifiable Diseases Surveillance System.

Genotypes

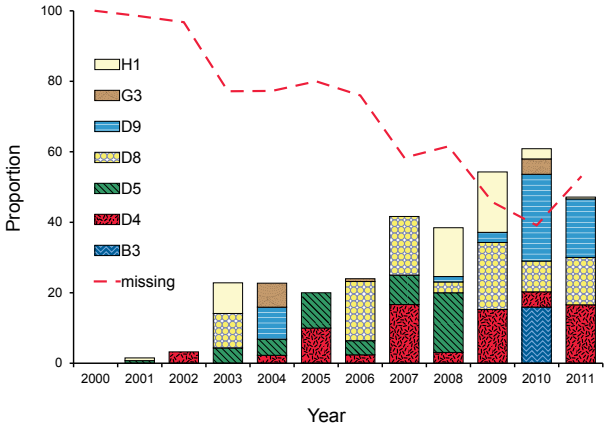
At the national level, the proportion of measles notifications with data on genotype increased from zero (no genotype data available) in 2000 to 61% in 2010 (Figure 4). There was no single dominant genotype over this period. The genotypes detected were B3, D4, D5, D8, D9, G3 and H1 and the majority of reported cases were imported or linked to imported cases. During this period, sporadic unknown source cases were also identified.

Severe morbidity and mortality

As observed with notifications, the number of hospitalisations has substantially declined over the past 2 decades, with the less than 1 year age group having the highest hospitalisation rates for most years followed by children aged 1–4 years and young adults (aged 20–34 years) (Figure 5). Overall, adults aged 35 years or more had the lowest hospitalisation rates.

During 2000 to 2011, the all age average ratio of notification to hospitalisation was 1.9. For the same period, the average age specific notification to hospitalisation ratio was 0.9 for infants (< 1 year); 1.7 for 1–4 years; 4.7 for 5–9 years; 4.4 for 10–19 years; 2.1 for 20–34 years and 1.3 for 35 years and older.

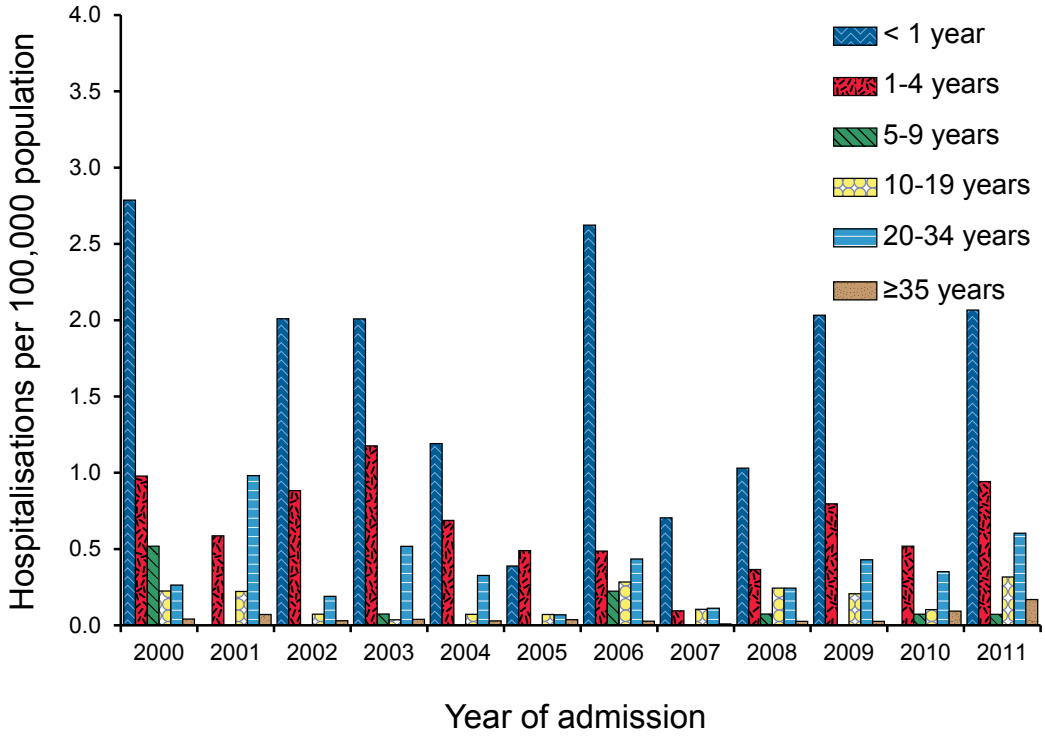
Figure 4: Proportion of measles notification, Australia, 2000 to 2011, by genotype



Source: National Notifiable Diseases Surveillance System.

In 2007 to 2011, of the 199 measles-related hospital admissions, 173 (87%) had measles recorded as the principal diagnosis and complications were recorded for 26 (15 %) admissions (Table 1). Of these, 10 were coded as having pneumonia and 16 as complications other than pneumonia, otitis media, encephalitis and meningitis such as

Figure 5: Measles hospitalisation rates, Australia, 2000 to 2011, by age group and year of admission



Source: Australian Institute of Health and Welfare National Hospital Morbidity Database

keratitis, keratoconjunctivitis and intestinal complications. No deaths were recorded in patients hospitalised with measles.

Length of stay per admission by age group is displayed in Table 2. Between 2007 and 2011, the total number of 173 hospital admissions by principal diagnosis accounted for 491 bed days and the median length of stay was 3 days.

Between 2002 and 2011, the National Mortality Database recorded measles as the underlying cause of death in 1–4 cases. These deaths were registered in 2010 and occurred in males only. To protect confidentiality, cells in the dataset with non-zero values of less than five are assigned a value of 1–4 by the ABS.[17]

Discussion

Our results provide an overview of the burden of measles in Australia over the last 12 years demonstrating that measles incidence is low and consistent with the elimination of indigenous measles. Overall, the notification rate fluctuated, falling to less than 1 case per million population twice during the study period, a previous indicator of low incidence set by the World Health Organization, required to reach elimination.[19] More recently, however, this indicator was updated to not include a threshold. The revised indicator states countries where measles has been eliminated should have a very low incidence of confirmed cases and absence of seasonality, a situation which our study support.

It has previously been accepted that measles is predominantly a childhood disease. During the period reported, despite overall low incidence, the

Table 1: Selected indicators of severe morbidity for hospitalised cases of measles, Australia, 2007 to 2011,* by age group

Age group (years)	Measles complicated by pneumonia		Measles with complications other than pneumonia, otitis media, encephalitis or meningitis		Measles without complications	
	n	% total†	n	% total†	n	% total†
<1	1	6.7	2	13.3	12	80.0
1–4	0	–	0	–	28	100.0
5–9	0	–	0	–	2	100.0
10–19	1	3.8	1	3.8	24	92.3
20–34	3	4.2	7	9.9	60	84.5
35+	5	16.1	6	19.4	20	64.5
All ages	10	5.8	16	9.2	146	84.4

* Hospitalisations by principal diagnosis only.

† Per cent of total in the age group.

Table 2: Length of stay per admission of measles hospitalisation, 2007 to 2011,* by age group

Age group (years)	Hospital admissions		Length of stay (days)	
	n	Rate per 100,000†	Median number of days	Range
< 1	15	1.03	1	1–6
1–4	28	0.50	1.5	1–5
5–9	2	0.03	1	1–1
10–19	26	0.18	2	1–6
20–34	71	0.31	3	1–8
35+	31	0.05	3	1–10
Total	173	0.16	3	1–10

* Hospitalisations by principal diagnosis only.

† Average annual age-specific rate per 100,000 population.

0–4 years age group remain a vulnerable population for measles infection and hospitalisations and infants younger than 12 months of age had the highest incidence in most years, highlighting their susceptibility. One possible explanation for this could be the decline in maternal antibodies in women with vaccine-acquired immunity, reaching a lower nadir at 7–9 months of age than those with infection-acquired immunity.[20,21] It has been postulated that because measles is becoming rare, the lack of natural boosting through exposure to wild virus in both vaccinated women and women with past infection has consequently resulted in infants becoming more susceptible. It is thus important, to avert preventable cases, that timely vaccine uptake among infants occurs at the recommended 12 months of age. During outbreaks, the 1st dose of measles can be administered early, for example at 9 months of age, with 2 subsequent doses required after 12 months of age due to concerns of interference with maternal antibodies. Previously under the NIP, the 2nd MMR dose (MMR2) was administered at 4 years of age. In 2008, it was recommended that MMR2 should be brought forward to 18 months given the number of notifications in the 1–4 years age group and the potential to improve vaccine uptake.[13] This recommendation was implemented under the NIP in July 2013 and aims to protect children against measles at an earlier age.[13]

Between 2009 and 2010, it was notable that individuals aged 10–19 years had high notification rates that further increased in 2011. It is likely that a proportion of this age group were too young to have been eligible for MMR2 given at 10–14 years between 1995 and 1998 and too old for MMR2 when the dose was brought forward to 4–5 years of age in 1998, and thus missed out on MMR altogether. This was highlighted during a recent high-school based outbreak (10 cases in a single high-school).[22] Additionally, collection and analysis of detailed demographic information on the 10–19 years age group may assist in considering risk factors that may be associated with infection such as ethnicity (8 of the 10 cases were of Pacific Islander origin[22]). This would allow for more targeted interventions to be piloted, promoting vaccination uptake in this age group.

Our study supports previous literature that identified young adults as a susceptible cohort. [23] The authors from the serosurveillance study reported highest immunity (98.3%) in subjects born before 1968 reflecting greater exposure to the wild type measles virus in older adults. Those born between 1968 and 1982 are particularly susceptible as low vaccine coverage existed when they were infants and circulation of wild virus was becoming less common.[23] Furthermore, many in this cohort were ineligible for MMR2 as they exceeded the 10–16 year eligibility age when it was offered between 1994 and 1998. Young adults are increasingly well-travelled and a number of more recent outbreaks have occurred following the importation of measles by a young adult traveller from an endemic country.[24–26] Although a targeted young adult measles campaign was conducted in 2001, it did not achieve high uptake.[27]

In Aboriginal and Torres Strait Islander people, notification and hospitalisation rates for measles have remained low across all age groups.[28] The highest notification and hospitalisation rate among Aboriginal and Torres Strait Islander people occurred in children less than 5 years of age.[28] With respect to hospitalisations, there were no hospitalisations recorded for Aboriginal and Torres Strait Islander people ≥15 years of age.[28] Vaccination status of all reported measles cases should be checked and validated.[30] As most outbreaks in Australia begin with an importation of measles from an endemic country, it is essential that measles immunity status be assessed when patients attend clinics to receive vaccinations for international travel. Currently, all individuals born during or after 1966 who have not acquired natural immunity or received 2 doses of MMR are recommended to be vaccinated before travelling overseas.[13] Of further concern is whether travellers present to a healthcare provider for pre-travel advice. In a study of 17,353 ill returned travellers who presented to one of the 30 participating travel or tropical medicine clinic around the globe, 50% had documented pre-travel health advice.[29] An Australian-based study found that 4% of 917 recently returned travellers who presented to 2 hospitals over a 6-year period (1998–2004) were vaccinated against MMR.[30] Hence, it is necessary to raise awareness of the risk of measles in under- or unvaccinated individuals travelling to measles endemic countries as part of pre-travel health advice. Currently, comprehensive information is provided on the Department of Foreign Affairs and Trade Smartraveller web site.[31]

Clearly, age-specific vulnerability (derived from interpretation of age-specific seropositivity to measles antibodies) of populations exist, even though measles is so rare in Australia and consequently this may lead to outbreaks in these populations. To determine whether cases are linked, it is important that genotyping of specimens occurs. Additionally, genotyping indicates the origins of the virus and gives information as to whether there are particular strains circulating in a country– especially important for ensuring measles elimination status.[32]

A number of limitations in the analysis warrant caution in interpreting the results. In general, notification data are considered not representative

of all cases in the community as not all cases may present to a medical practitioner or be recognised. However, this is unlikely to occur for measles as it is assumed most cases would attend a medical practitioner due to the severity of the symptoms of the disease.

Hospitalisations for measles should be interpreted with caution due to possible coding errors. A Victorian based study found that the discrepancy rate in coding fields among hospital morbidity data increased the rarer the condition.[33] Our data also highlighted these discrepancies, as in some instances the number of hospitalisations exceeded the number of notifications suggesting either the under-reporting of measles notifications and/or the miscoding of hospitalisations as measles.

There were reporting issues with mortality data too and paucity of information on complications, pre-existing co-morbidities and/or extremes of age.[34]

Lastly, the case definition for measles was amended in 2004. Prior to 2004, a confirmed case of measles could include a diagnosis based on an illness clinically consistent with measles. As many conditions may present with similar symptoms to measles, the specificity of this earlier case definition is likely to have been low and subsequently led to an overestimation of true measles cases. [35]

Conclusions

Measles is rare in Australia. The incidence of measles has fluctuated over the last 12 years, with presence of age-specific vulnerability in populations, even though measles incidence is so low in Australia. Children less than 5 years of age, and more recently, adolescents and young adults have been susceptible and hence, there is an ongoing need to improve vaccine uptake in vulnerable populations to prevent outbreaks. Overall, in this period, measles incidence has remained low with cases being mainly imported or imported-related, whilst only limited secondary spread has been documented, which together provide evidence consistent with elimination of indigenous measles in the country.

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Australian vaccine preventable disease epidemiological review series: mumps 2008-2012

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Abstract

In 2007, Australia recorded the highest notification rate (2.8 per 100,000) for mumps since it became notifiable, with outbreaks in Western Australia and the Northern Territory. Of particular concern was the number of cases seen in vaccinated individuals. The aim of this study was to review subsequent epidemiological data. Notification, hospitalisation and mortality data from the National Notifiable Diseases Surveillance System, the National Hospital Morbidity Database and Australian Bureau of Statistics (ABS) respectively, from 2008 to 2012 for notifications and 2008 to 2011 for hospitalisations and deaths, were analysed by age, year and jurisdiction. ABS population data were used to calculate rates. National mumps notification rates decreased from 1.3 per 100,000 in 2008 to 0.4 per 100,000 in 2010, but then increased to 0.9 per 100,000 in 2012, predominantly due to increased notifications in New South Wales (1.4 per 100,000). Hospitalisation rates remained stable at 0.4 per 100,000 over the 2008–2011 period. The median age of notified cases was 30 years and for hospitalisations, 27 years. The highest rate of notifications and hospitalisations was in the 25–34 years age group. Completeness of vaccination status ranged from 16% to 39%. The increasing trend in mumps notifications needs to be closely monitored. Improved data quality, in particular on vaccination status, is needed to inform the monitoring of vaccine effectiveness. In March 2014 the World Health Organization certified that Australia had achieved measles elimination. Greater availability of case history (vaccination status and place of acquisition) and genotyping data would facilitate an assessment of Australia's progress in relation to mumps elimination. *Commun Dis Intell* 2015;39(1):E10–E18.

Keywords: mumps, disease surveillance, immunisation

Introduction

Mumps is a paramyxovirus in the genus Rubulavirus. Humans are the only known reservoir for the virus. Symptoms of the disease include fever and other non-specific symptoms including headache, malaise, myalgia and anorexia. One-third of infected individuals have subclinical disease.[1,2] The characteristic feature of mumps is swelling and inflammation of one or more salivary glands, most commonly the parotid gland.[1] Up to 10% of cases develop meningitis and 0.1% encephalitis. The case fatality rate for encephalitis is around 1.5%.[1,2] Unilateral orchitis occurs in 25% of post-pubertal males with clinical mumps, however, infertility is rare.[1] In early pregnancy, mumps can cause spontaneous abortion, but there is no evidence of infection leading to increased risk of congenital malformation or low birth weight.[2]

The incubation period of mumps is usually 16–18 days but can range from 12–25 days.[3] Transmission occurs mainly through contact with respiratory droplets or saliva, with peak infectiousness just before or at the onset of parotitis.[1]

A single dose of mumps (as measles-mumps) vaccine was funded for all children aged 12 months of age on the national immunisation schedule in 1982.[4] In 1989, the vaccine was changed to the measles-mumps-rubella (MMR) vaccine. A 2nd dose of MMR was introduced at 10–14 years of age in 1992. This dose was brought forward to 4–5 years of age in 1998, and then to 4 years in 2000. There were 2 major MMR catch-up campaigns. The Measles Control Campaign in 1998 targeted primary school children. A young adult MMR vaccination campaign in 2001 targeted individuals born in the late 1960s to the mid-1980s who may have missed being vaccinated or acquiring measles, mumps and rubella infection as an infant.[4] While the Measles Control Campaign reached 96% of the target age group,[5] the uptake in the young adult campaign was likely to have been poor, based on the lack of any significant improvement in immunity in this age group in pre– and post-serosurvey data.[6,7]

Since the introduction of universal childhood vaccination, a shift in the age distribution of mumps cases from younger children to adolescents and young adults occurred in Australia and overseas from the 1980s to the early 2000s, with the exact period of age shift varying from country to country.[8–10]

Previous reviews of mumps epidemiology in Australia documented an increase in mumps notifications from 2003 to 2007, contributed to by epidemiologically-linked outbreaks in Indigenous communities in the Northern Territory and

Western Australia in 2007.[11,12] Concerns were raised about the number of cases seen in vaccinated individuals.[11] Increasing numbers of mumps notifications were also observed in the United States of America and the United Kingdom in the first half of the 2000s,[13–15] and in some countries in the World Health Organization Western Pacific Region, including China, Japan, Mongolia and Korea, to 2007.[16]

This study aims to review subsequent Australian mumps epidemiology for the 2008 to 2012 period, analysing by age, gender, state or territory, Indigenous status and vaccination status, and to place this in historical and international context by comparison with published data.

Methods

Study period

For notifications, we analysed data from 1 January 2008 to 31 December 2012. For hospitalisations and deaths, we analysed data from 1 January 2008 to 31 December 2011 (the latest data available at the time of analysis). We reviewed published data from the pre-2008 period for comparison purposes.[10–12,17]

Data sources

Notifications

In Australia, cases of mumps are notified under the public health legislation in each state and territory. De-identified data on confirmed cases are submitted to the National Notifiable Diseases Surveillance System (NNDSS). The NNDSS was established in 1991, with regular reporting by all 8 states and territories on a consistent basis occurring from mid-2001 onwards. A national case definition has been in place since 2004, which includes a combination of laboratory, clinical and epidemiological evidence.[18]

For this analysis, notifications with a diagnosis date between 1 January 2008 and 31 December 2012 were obtained from the NNDSS for all Australian jurisdictions. The diagnosis date is derived from the date of onset, or, where not supplied, the earliest date recorded among these fields: date of specimen, date of notification, or date when the notification was received.[11] Vaccination status was only assessed for individuals born after 31 December 1980 i.e. the population eligible for mumps vaccination under the National Immunisation Program (NIP) since it was introduced onto the NIP in 1982. Vaccination status is categorised as fully or partially vaccinated for age, unvaccinated, unknown or missing.[19]

Hospitalisations

Hospital admissions to public and private hospitals in Australia are captured through an administrative database, the National Hospital Morbidity Database, maintained by the Australian Institute of Health and Welfare (AIHW). Demographic and clinical information about patients are captured. For this analysis, all hospitalisations with admission dates between 1 January 2008 and 31 December 2011 were included. Eligible hospitalisation admissions were identified using the International Statistical Classification of Diseases and Related Health Problems, 10th revision, Australian Modification (ICD-10-AM) code B26 (mumps), where listed as principal or other diagnosis. Hospitalisations from Tasmania and the Australian Capital Territory were excluded from the analysis of Indigenous status, as recommended by the AIHW on the basis that completeness in these jurisdictions is below the level (80%) considered acceptable for inclusion in national analysis.[20]

Mortality

Deaths are registered with the Registry of Births, Deaths and Marriages in each state and territory. Data from the registries and state and territory coroners are collated by the Australian Bureau of Statistics (ABS). Mortality data for mumps were obtained from the ABS for the period 2008 to 2011. Data where the underlying cause of death was recorded as mumps, using ICD-10-AM code B26, were included in this analysis.

Population estimates

National, jurisdictional and age-specific mid-year estimated resident population data were obtained from the ABS.[21]

Data analysis

For notifications, variables extracted for analysis included year of diagnosis, age, sex, state or territory of residence, vaccination status, Indigenous status and place of acquisition. For hospitalisations, variables extracted for analysis included primary or other diagnosis, date of admission, age, sex, state or territory of residence, Indigenous status, complications and length of stay (bed days). Data fields were assessed for completeness. Notification and hospitalisation rates were calculated using ABS population data, and are presented as average annual rates per 100,000 total population or population in age, sex or geographical subgroups as appropriate, with age groups selected based on epidemiological relevance and usage in previous published reports. Summary statistics including median and range were calculated for age and length of hospital stay.

P-values were derived using t-test for proportions. Analysis was conducted using Microsoft Excel 2010 and SAS version 9.3 (SAS Institute Inc, Cary, NC, USA).

Results

Secular trends

Nine hundred and four mumps notifications were recorded in the NNDSS between 2008 and 2012. The number of notifications decreased progressively from a peak of 582 in 2007 (notification rate 2.8 per 100,000) to 98 (0.4 per 100,000; $P<0.001$) in 2010 but then increased to 155 (0.7 per 100,000; $P<0.001$) in 2011 and 200 (0.9 per 100,000; $P<0.001$) in 2012 (Figure 1).

There were 356 hospitalisations between 2008 and 2011. Following the 2007 peak (104 hospitalisations; 0.5 per 100,000), the number of hospitalisations and hospitalisation rate remained stable at around 89 and 0.4 per 100,000, respectively, over the 2008 to 2011 period (Figure 2).

During the 2008 to 2011 period, there were twice as many notifications of mumps (704) as there were hospitalisations (356). The ratio of notifications to hospitalisations declined from 5.7 in 2007 to 3.2 in 2008 and 1.1 in 2010 (Figure 3).

No deaths with mumps coded as the underlying cause of death were recorded during the 2008 to 2011 period.

Age and sex distribution

The age distribution for notifications from 2008 to 2012 was similar to that in the preceding 3-year period (Figure 1). The highest age-specific notification rate was in the 25–34 years age group (1.7 per 100,000), and the lowest in the 0–4 years age group (0.5 per 100,000; data not shown). The median age of notifications was 30 years (range: 0–88 years). The overall male:female ratio over the 5–year period was 1.2:1 with some variation by year (lowest 0.7:1 in 2010; highest 1.5:1 in 2009) but without notable variation by age group.

The age distribution for hospitalisations from 2008 to 2011 was similar to that in the preceding 3-year period (Figure 2). The highest age specific hospitalisation rate was in the 15–24 years and 25–34 years age groups (0.6 per 100,000), and the lowest in the ≥35 year age group (0.3 per 100,000). The median age for hospitalisations was 27 years (range: 0–97 years). The overall male:female ratio was 1:1, with variation by year (lowest 0.6:1 in 2010; highest 1.4:1 in 2009)

Figure 1: Mumps notification rates, Australia, 1999 to 2012, by age group and year of diagnosis

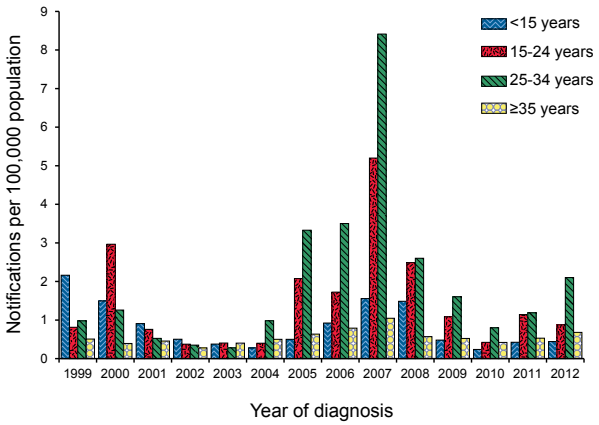


Figure 2: Mumps hospitalisation rates, Australia, 1999 to 2011, by age group and year of admission

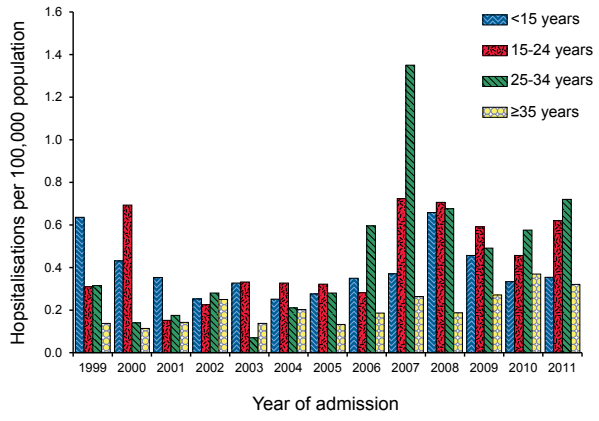
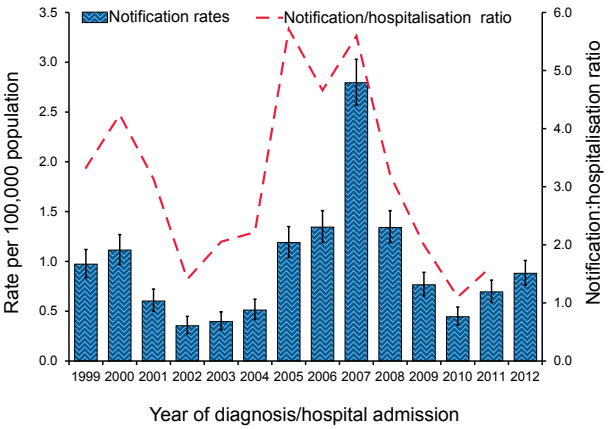


Figure 3: Mumps notifications and notification:hospitalisation ratio, Australia,* 1999 to 2012†



* From July 1999 until June 2001 mumps was not notifiable in Queensland.

† Hospitalisation data available to 2011.

and age group (1.6:1 in 0–4 years age group;1.4:1 in 5–14 years age group; 0.8:1 in 15–34 years age group; 0.9:1 in ≥35 years age group).

Seasonality

No seasonal pattern was apparent over the 2008 to 2012 period (data not shown).

Indigenous status

During the 2008 to 2012 period, 52% (468/904) of notifications were recorded as non-Indigenous, 14% (127/904) as Indigenous, and 34% (309/904) had unknown or missing Indigenous status. Completeness of Indigenous status was highest in 2008 (77%), and varied between 51% and 63% in subsequent years. The proportion of notifications recorded as Indigenous was highest in 2008 (39%, 110/285), decreasing to 7% (11/166) in 2009 and 1% (1/200) in 2012.

From 2008 to 2011, 88% (306/349) of hospitalisations (Tasmania and the Australian Capital Territory excluded) were recorded as non-Indigenous, 10% (36/349) as Indigenous, and 2% (7/349) had unknown or missing Indigenous status. Completeness of Indigenous status varied between 97% and 99% by year. The proportion of hospitalisations recorded as Indigenous decreased from 24% (21/88) in 2008 to 2% (2/92) in 2011.

State and territory variations

Notification and hospitalisation rate trends varied across states and territories (Figure 4). The highest average annual notification rate was in the Northern Territory (5.9 per 100,000), with the 2nd highest in Western Australia (1.4 per 100,000). Average annual notification rates were below 1.0 per 100,000 in all other states and territories. Of note, 96% (106/110) of the notifications recorded as Indigenous in 2008 were notified in either Western Australia or the Northern Territory. The number of notifications in the Northern Territory decreased from 52 in 2008 (23.7 per 100,000) to 2 (0.9 per 100,000) in 2010 ($P<0.001$), while the number of notifications in Western Australia decreased from 95 in 2008 (4.4 per 100,000) to 20 in 2009 (0.9 per 100,000; $P<0.001$). The number of notifications in New South Wales increased from 40 (0.6 per 100,000) in 2010 to 105 (1.4 per 100,000) in 2012 ($P<0.001$).

The highest average annual hospitalisation rate was in the Northern Territory (2.4 per 100,000, 22/356). The average annual hospitalisation rate in all other states and territories was below 0.5 per 100,000. The number of hospitalisations in the Northern Territory decreased from 11 in 2008 (5.0 per 100,000) to 1 in 2011 (0.4 per 100,000; $P<0.001$). Ninety-five per cent (20/21) of the hospitalisations recorded as Indigenous in 2008 were notified in either Western Australia or the Northern Territory.

Vaccination status

Between 2008 and 2012, there were 435 notifications in individuals born after 31 December 1980. Of these individuals, 410 were aged ≥4 years, who should have received 2 doses of mumps-containing vaccine according to current recommendations. Of these, 18% (74/410) were reported as fully vaccinated, 4% (15/410) as partially vaccinated for age, and 6% (24/410) as unvaccinated; vaccination status was missing or unknown for most (72%, 297/410). Completeness of vaccination status in individuals born after 31 December 1980 and aged ≥4 years was highest in 2008 (39%) and varied between 16% and 28% in subsequent years.

Between 2008 and 2012, there were 119 notifications in individuals born after 31 December 1995, eligible to have their immunisation information recorded on the Australian Childhood Immunisation Register. Of the 94 of these aged ≥4 years, 28% (26/94) were reported as fully vaccinated, 5% (5/94) as partially vaccinated for age and 10% (9/94) as unvaccinated, with 57% (54/94) having missing or unknown vaccination status. Completeness of vaccination status in individuals born after 31 December 1995 and aged ≥4 years was highest in 2008 (53%), and varied between 27% to 41% in subsequent years.

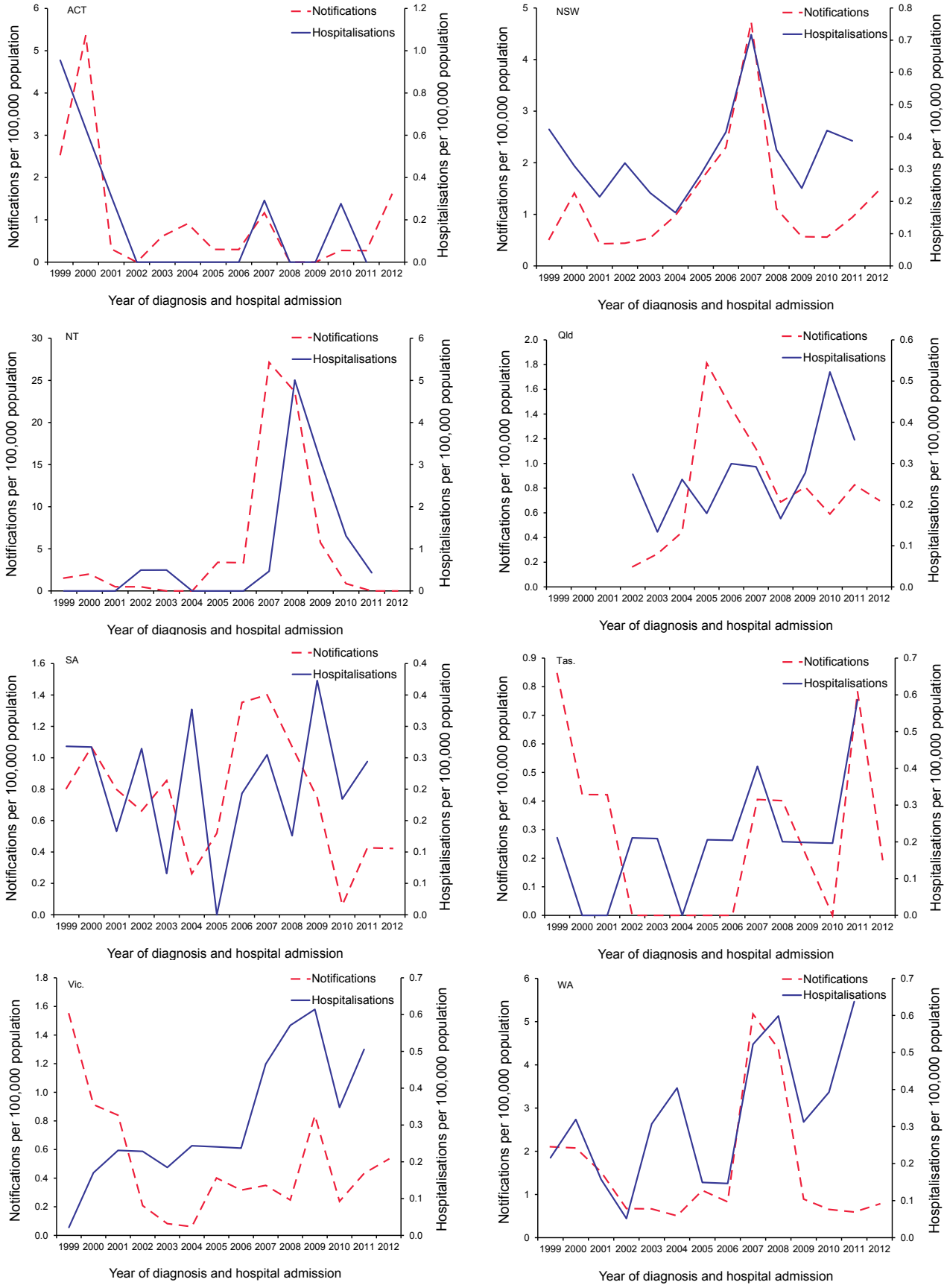
Place of acquisition

Place of acquisition was missing or unknown for 97% (879/904) of notifications between 2008 and 2012. As data quality was poor, further data analysis was not conducted.

Severe morbidity

One thousand four hundred and ninety-three hospital bed days (average 373.3 per year) were recorded for patients with an ICD-10-AM code for mumps. The median length of stay per admission ranged from 1 to 3 days, and was longest (3 days) in the ≥35 years age group. The overall median length of stay was 2 days (range: 1–119 days) (Table 1). Complications arising from mumps infection were recorded for 45 hospitalisations (12.6%). The most common complication recorded was orchitis (23 hospitalisations), predominantly in individuals aged ≥15 years (Table 2). There was 1 hospitalisation coded as mumps meningitis and two coded as pancreatitis. Nineteen hospitalisations were coded as 'other complications', with most of these in individuals aged ≥15 years (Table 2).

Figure 4: Mumps notification and hospitalisation rates, Australia, 1999 to 2012,* by state or territory† and year of diagnosis or admission



Scales vary between jurisdictions.

* From July 1999 until June 2001 mumps was not notifiable in Queensland.

† Hospitalisation data available to 2011.

Table 1: Mumps notification and hospitalisation counts and rates, and hospitalisation length of stay, Australia, 2008 to 2012,* by age group

Age (years)	Notifications (2008–2012)		Hospitalisations (2008–2011)			
					Length of stay (bed days)	
	n	Rate	n	Rate	Median	Total
0–4	38	0.53	31	0.54	1	60
5–14	107	0.78	44	0.40	2	189
15–24	182	1.20	72	0.59	1	131
25–34	258	1.65	76	0.62	1	171
≥35	318	0.55	133	0.29	3	942
Missing	1		0			
All ages	904	0.82	356	0.41	2	1,493

* Hospitalisation and death data not available for 2012.

Table 2: Indicators of severe morbidity in hospitalised cases of mumps,* Australia, 2008 to 2011, by age group

Age group (years)	Mumps orchitis		Mumps meningitis		Mumps encephalitis		Mumps pancreatitis		Mumps with other complications†	
	n	%‡	n	%‡	n	%‡	n	%‡	n	%‡
0–4	1	3.2	0	0.0	0	0.0	0	0.0	0	0.0
5–14	3	6.8	0	0.0	0	0.0	0	0.0	1	2.3
15–24	7	9.7	0	0.0	0	0.0	0	0.0	4	5.6
25–34	6	7.9	0	0.0	0	0.0	0	0.0	4	5.3
≥35	6	4.5	1	0.8	0	0.0	2	1.5	10	7.5
All ages	23	6.5	1	0.3	0	0.0	2	0.6	19	5.3

* From the Australian Institute of Health and Welfare hospitalisation data based on ICD-AM-10 codes.

† Other complications include mumps associated arthritis, myocarditis, neuritis and polyneuropathy.

‡ Percentage of total in the age group

Discussion

After 2007, when the highest number of annual mumps notifications was recorded since mumps became notifiable across all states and territories from July 2001,[17] mumps notifications in Australia decreased between 2008 and 2010 but then increased in 2011 and 2012, predominantly due to increased notifications in New South Wales. Age distribution during the 2008 to 2012 period was broadly similar to the preceding 3-year period, with approximately half of notifications in adolescents and young adults aged 15–34 years compared with greater than 60% during the 2005 to 2007 period.[10] The highest age-specific notification rate during the 2008 to 2012 period was in the 25–34 years age group. Serosurveillance data have highlighted the susceptibility of individuals in this age group i.e. those born in the 1980s when exposure to wild type virus was decreasing but before good levels of vaccine coverage were achieved.[22]

For hospitalisations, following the 2007 peak, the annual number and rate of hospitalisations in Australia remained stable during the 2008 to 2011 period. While the average annual number of hospitalisations was higher for the 2008 to 2012 period compared with the previous 3-year period, the proportion with complications recorded was lower. The average number of hospital bed days (373) was considerably higher for the 2008 to 2011 period compared with the previous 3-year period (171.5).[11] The reason for these differences is unclear. No deaths were recorded from mumps during the 2008 to 2011 period.

The notification:hospitalisation ratio in Australia decreased from 2007 to 2011. This is consistent with increased identification of cases with mild disease during outbreaks, due to active case finding by public health authorities.

There were no reports of large mumps outbreaks in Australia between 2008 and 2012. Several small outbreaks involving 2 to 10 cases were reported in Victoria, in 2009, 2011 and 2012; however, limited information is available on age and vaccination status for the majority of these.[23–27] Following on

from the epidemiologically-linked outbreaks in Indigenous communities in the Northern Territory and Western Australia in 2007, higher numbers of notifications in the Northern Territory continued into 2008 and 2009. These were predominantly in Indigenous people, although it is not definitively known whether these cases were linked to the 2007 outbreak (Peter Markey, Public Health Physician, Communicable Diseases Centre, Darwin, personal communication December 2013). It is also not clear whether the higher number of notifications in Western Australia in 2008, predominantly in Indigenous people, was linked to the 2007 outbreak. However, this possible linkage may account for the decrease in the proportion of notifications and hospitalisations recorded as Indigenous in Australia. From 1984 to 1998, the Northern Territory vaccinated Indigenous infants with a mumps-containing vaccine at 9 months of age, due to their higher risk of measles at the time. This is thought to have played an important role in the 2007 Northern Territory outbreak, as immune response is poorer in infants immunised at under 12 months of age due to interference from maternal antibodies.[28,29]

Concerns about high rates of mumps in fully vaccinated individuals have been raised in relation to recent outbreaks, both in Australia[11] and overseas.[30–33] However, poor completeness of vaccination status in NNDSS data limits the ability to assess vaccine effectiveness. During the 2008 to 2012 period, vaccination status was known for less than a third of notifications in individuals born after 31 December 1980 and aged ≥4 years, with over two-thirds of these recorded to be fully vaccinated. For notifications in individuals born after 31 December 1995 and aged ≥4 years, for whom Australian Childhood Immunisation Register data are potentially available, vaccination status was known for less than half, with over two-thirds of these recorded as fully vaccinated.

Infection in fully vaccinated individuals can be due to a range of reasons including primary vaccine failure, waning immunity, and immune escape.[34–36] Clinical trials of MMR vaccine have shown 95% mumps seroconversion after 1 dose and up to 100% after 2 doses.[37] However, outbreak investigations and post-marketing studies have reported 1-dose vaccine effectiveness (VE) to be between 60% and 90%.[38,39] In some recent outbreaks cases in 2-dose vaccine recipients have been reported to be common, particularly in young adults who were vaccinated more than 10 years earlier.[33,40,41] Two-dose VE, while higher than 1-dose VE,[35] has been shown to decline over time, suggesting waning immunity.[35,42] Whether immune escape of the mumps virus, in response to vaccine-related selection pressure, has been an issue in Australia is difficult to assess due to the paucity of genotyping data, which are only available from outbreak reports.[27,28,34] It is also difficult to assess the relative contributions to mumps disease of suboptimal 2-dose vaccine coverage and primary vaccine failure in 2-dose recipients, given the poor completeness of vaccination status in mumps notification data.

Australia has recently been certified by the World Health Organization as having eliminated endemic measles transmission, on the basis of documented interruption of endemic measles virus transmission for more than three years in the presence of a well-performing surveillance system and supportive genotyping evidence.[43] Available data also suggest that Australia may be close to elimination of rubella.[44] It is therefore of interest to ask what the situation is in regards to mumps. The threshold for herd immunity for mumps has been estimated as 75%–86%, compared with 92%–94% for measles and 83%–85% for rubella.[45] While Australia has achieved high vaccination coverage for MMR (94% for the 1st dose and 90% for the 2nd dose in 201146) vaccine effectiveness is considerably lower for mumps than measles.[40,47] Finland is the only country to have reported elimination of endemic mumps transmission (in the mid-1990s, along with measles and rubella), on the basis of sustained high (>95%) 2-dose MMR vaccine coverage and enhanced surveillance showing the vast majority of cases confirmed to be imported.[48,49] However, it is possible that other countries such as Australia may also have achieved or come close to mumps elimination, and that sporadic outbreaks in highly vaccinated populations may be due to the force of infection after virus introduction from an endemic area into high-density, high contact environments.[42] Greater availability of case history (vaccination status and place of acquisition) and genotyping data would assist in answering this question. Nationally standardised methods of data collection, follow-up and reporting could help to facilitate improved completeness of key data fields.

The figures presented in this report are likely to underestimate the true burden of mumps in Australia, as not all mumps cases are diagnosed and notified. A limitation of notification data is that they may be affected by changes in diagnostic and public health follow-up practices, particularly in outbreak settings, over time and across jurisdictions. Data quality was poor for a number of fields in notification data, including Indigenous status, vaccination status and place of acquisition, presumably reflecting variable levels of follow-up by public health authorities with clinicians and patients. Hospitalisation data may be influenced by access to hospitals and changes in admission practices. ICD-10-AM codes used to identify cases of mumps in the National Hospital Morbidity Database were

assigned for hospital billing purposes, have not been validated to clinical diagnoses or case definitions, and may be susceptible to misclassification.

In conclusion, after peaking in 2007 at their highest level since 2001, mumps notifications in Australia decreased progressively through to 2010, but then increased in 2011 and 2012, while hospitalisations remained stable over the 2008 to 2011 period. The increasing trend in mumps notifications will require continued close monitoring to determine if it is sustained. Improvements in data quality, particularly in terms of completeness of vaccination status and place of acquisition, are required to inform monitoring of vaccine effectiveness. Along with greater availability of genotyping data, this would also facilitate assessment of Australia's progress in relation to mumps elimination.

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Australian vaccine preventable disease epidemiological review series: rubella 2008–2012

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Abstract

Introduction: Since the introduction of universal rubella vaccination in 1989, the incidence of rubella and congenital rubella syndrome (CRS) in Australia has declined significantly. Worldwide, there has been a focus on elimination, with the region of the Americas declaring rubella elimination in 2011. This study aims to review Australian rubella epidemiology for the 2008–2012 period, in the context of historical and international trends.

Methods: Notification, hospitalisation and mortality data were sourced from the National Notifiable Diseases Surveillance System, the National Hospital Morbidity Database and the Australian Bureau of Statistics (ABS). Data analysis focused on 2008–2012 for notifications and 2008–2011 for hospitalisations and deaths. ABS population data were used to calculate rates.

Results: The average annual rubella notification rate in Australia from 2008–2012 was 0.18 per 100,000 and the average annual hospitalisation rate was 0.03 per 100,000 from 2008–2011. One case of CRS was notified in 2012 and 1 hospitalisation with a principal diagnosis of CRS was recorded in 2008. The median age of rubella notifications was 29 years and 37% of notifications were for infections acquired overseas.

Discussion: Rubella continues to be well controlled in Australia and CRS is rare. The low incidence and increasing proportion of imported cases and other evidence suggest that elimination has been achieved; however, for formal verification of rubella elimination the expansion of genotypic surveillance will be required. Ongoing rubella control needs to focus on improved surveillance, maintenance of high levels of vaccine coverage, vaccination of at-risk populations in Australia, and regional and global efforts towards rubella elimination. *Commun Dis Intell* 2015;39(1):E19–E26.

Keywords: epidemiology; rubella; vaccine preventable diseases

Introduction

Rubella is a vaccine preventable, usually mild infection. However, infection during pregnancy is associated with a range of congenital abnormalities known as congenital rubella syndrome (CRS). The principal aim of vaccination campaigns worldwide is to prevent CRS, which remains a common cause of fetal abnormalities in rubella endemic regions.[1]

Rubella is caused by rubella virus, which is a member of the Togaviridae family, genus *Rubivirus*. Rubella is transmitted by contact with nasopharyngeal secretions, either through droplet or direct contact. The incubation period ranges from 12 to 23 days.[2,3] Rubella usually manifests as a mild non-specific febrile illness characterised by a non-confluent maculopapular rash, lymphadenopathy, headache, sore throat, cough and conjunctivitis. Arthralgia and arthritis are the most common complications. More serious complications, including encephalopathy and haemorrhage, occur rarely. Up to 50% of infections are asymptomatic. Infection during the first trimester of pregnancy is associated with spontaneous abortion or CRS in 85% of cases. Congenital abnormalities associated with CRS include cataracts, heart defects, sensorineural deafness, low birth weight and neurological defects.[3,4]

Rubella vaccines have been licensed in Australia since 1969, initially as a monovalent vaccine containing attenuated rubella virus, given to schoolgirls aged 10–14 years and susceptible women prior to pregnancy. From 1989, the measles-mumps-rubella (MMR) vaccine was introduced to the National Immunisation Program (NIP) schedule for all infants. From 1993, a 2nd dose of MMR vaccine was added to the NIP schedule for both boys and girls at 10–14 years of age, with a school-based catch-up program for this age group from 1993 to 1998.[5] The 2nd dose of MMR was moved to 4–5 years of age in 1998 and then replaced in 2013 with a dose of measles-mumps-rubella-varicella (MMRV) vaccine given at 18 months of age (Table 1 for full details of funded rubella vaccination programs in Australia).[6] Protection against rubella is long-term.[7]

The epidemiology of rubella in Australia changed following the introduction of universal rubella vaccination. There was a marked decline in the average annual notification rate from 14.8 per 100,000 population between 1993 and 1998 to 0.23 per 100,000 between 2005 and 2007. This was

Table 1: Nationally funded rubella vaccination programs in Australia[5]

Year	Program	Target age
1971–1993	Selective vaccination of schoolgirls	12–14 years
1989–1992	Universal 1-dose MMR	1 year
1993–1998	Universal 2-dose MMR	1 and 10-14 years
1993–1994	Selective school based catch-up for one cohort of males and females	10-14 years
1998-2013	Universal 2-dose MMR	1 and 4-5 years
1998	Measles Control Campaign – one-off school-based catch-up MMR	5-12 years
2013 onwards	Universal MMR + MMRV	1 year and 18 months

MMR Measles-mumps-rubella vaccine.
MMRV Measles-mumps-rubella-varicella vaccine.

accompanied by an increase in the median age of notifications and a decrease in the male to female notification rate ratio.[8–11]

This study aims to review Australian rubella epidemiology from 2008 to 2012, in the context of historical and international trends.

Methods

Data sources

Notifications

The National Notifiable Diseases Surveillance System (NNDSS), established in 1991, collects de-identified information about notifiable diseases from Australian states and territories. Confirmed and probable cases of rubella and CRS are notifiable under public health legislation in each state and territory. A confirmed case of rubella requires laboratory definitive evidence including either detection of rubella virus (viral culture or nucleic acid testing) or confirmatory serology (IgG seroconversion or ≥ 4-fold rise in IgG titre). A probable case of rubella requires clinical evidence and an epidemiological link to a laboratory confirmed case. A confirmed case of CRS requires definitive laboratory evidence in the infant (detection of rubella virus by culture or nucleic acid testing, or positive IgM confirmed by a reference laboratory). A probable case of CRS requires clinical evidence (compatible defects in the infant) and laboratory suggestive evidence in the mother or infant.[12] For this analysis, data were obtained from NNDSS for all notifications with a diagnosis date between 1 January 1993 and 31 December 2012. The diagnosis date is derived from the date of onset, or, where not supplied, the earliest date recorded among these fields: date of specimen, date of notification, or date when the notification was received. Analysis focused primarily on the 1 January 2008 to 31 December 2012 period, with notifications from 1 January 1993 included where relevant for trends.

Hospitalisations

Hospitalisation data were obtained from the National Hospital Morbidity Database, maintained by the Australian Institute of Health and Welfare. Administrative, demographic and clinical information about patients admitted to public and private hospitals in Australia are collected from hospital discharge summaries. For this analysis, all hospitalisations with admission dates between 1 January 2008 and 31 December 2011 (the most recent data available) were included. Eligible hospitalisation admissions were identified using the International Statistical Classification of Diseases and Related Health Problems, 10th revision, Australian Modification (ICD-10-AM) code B06 (rubella) and P350 (CRS), where listed as the principal or other diagnosis.

Mortality

Mortality data were obtained from the Australian Bureau of Statistics (ABS). Data where the underlying cause of death was recorded as rubella, using ICD-10 code B06 (rubella) were included in this analysis.

Population estimates

National, jurisdictional and age-specific mid-year estimated resident population data were obtained from the ABS.

Data analysis

For notifications, variables extracted for analysis included confirmed or probable status, year of diagnosis, age, sex, state or territory of residence, laboratory diagnosis method, vaccination status, Indigenous status and place of acquisition. For hospitalisations, variables extracted for analysis included primary or other diagnosis, year of admission, age, sex, state or territory of residence,

Indigenous status, complications and length of hospital stay. The outbreak reference field was not included in the analysis due to poor data quality.

Rates were calculated using ABS population data and are presented as annual average rates per 100,000 total population or population in age, sex or geographical subgroups as appropriate. Male to female notification rate ratios were calculated by age group with 95% confidence intervals. Trends in median age were analysed using the Kruskal-Wallis rank test. Summary statistics including median and range were calculated for age and length of hospital stay. Analysis and presentation of data was conducted using SAS, Microsoft Excel 2010 and Stata.

Ethics approval was not required for this review as de-identified aggregate population based data were summarised for routine public health surveillance only.

Results

Rubella

Notification and hospitalisation trends

From January 2008 to December 2012, there were 201 notified cases of rubella, of which 185 (92%) were confirmed and 16 (8%) were probable cases. The average overall annual notification rate (probable and confirmed cases combined) was 0.18 per 100,000. The annual number of rubella notifications has remained stable and low since 2004 (Figure 1). No seasonal pattern was seen over the 2008–2012 period (data not shown).

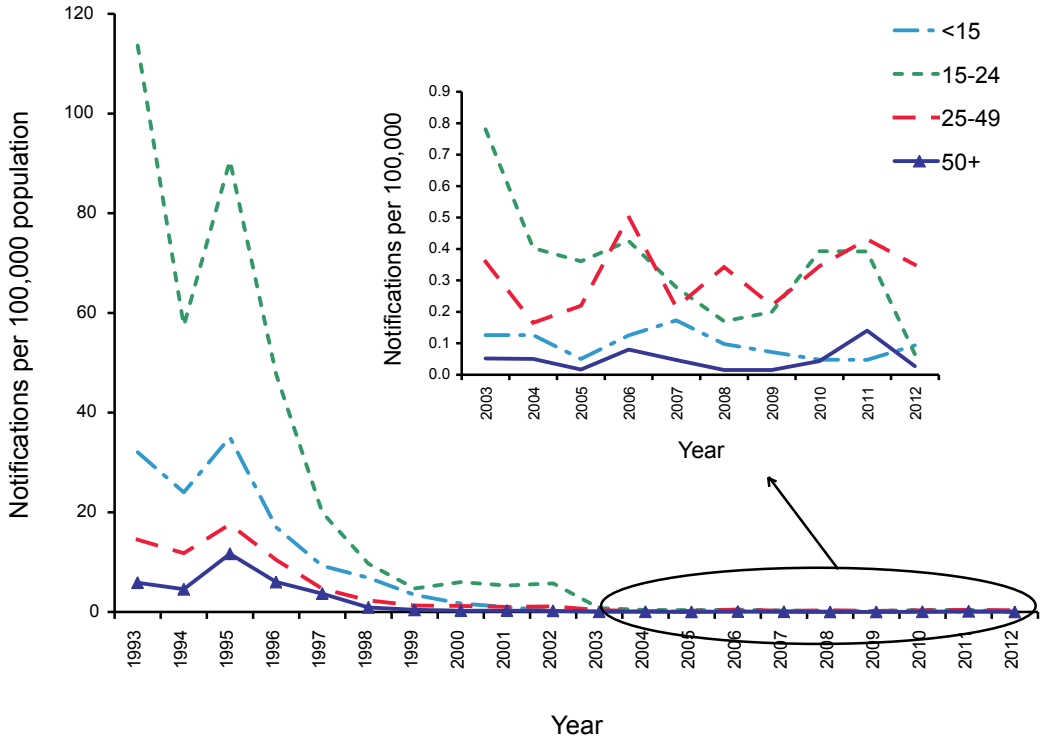
There were 30 hospitalisations due to rubella infection between January 2008 and December 2011; an average annual rate of 0.03 per 100,000 (Table 2). Of the 30 hospital separations, 15 had a principal diagnosis of rubella, an average annual rate of 0.02 per 100,000.

Age and sex distribution

Rubella notification rates have declined markedly in all age groups since 1993 (Figure 1). The median age of notified cases between 2008 and 2012 was 29 years. Between 1993 and 2012 there was an increase in the median age from 18 to 32 ($P < 0.001$).

From 2008 to 2012, the highest age-specific average annual rate of rubella notifications in men was in the 30–39 years age group (0.53 per 100,000); in women it was in the 20–29 years age group (0.52 per 100,000). The overall male to female notification rate ratio was 1.3:1 (95% CI 1.0–1.8) from 2008 to 2012. However, the rate of notifications in males was significantly higher than females only in the 30–39 years age group (RR = 2.3, 95% CI 1.3–4.2) (Figure 2).

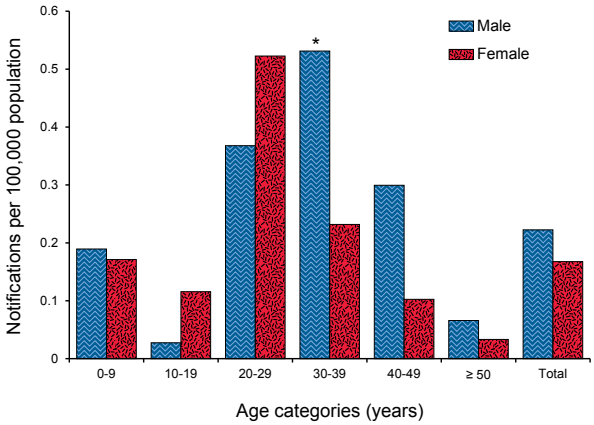
Figure 1: Rubella notification rates, Australia, 1993 to 2012, by age group



There were 69 notified cases of rubella in women of child bearing age (15–44 years) from 2008 to 2012 at an average annual rate of 0.30 per 100,000. Within this group the highest average annual rate of notifications was in women in the 25–29 years age group (0.63 per 100,000).

The median age of hospitalisations with any diagnosis of rubella from 2008 to 2011 was 26 years. The median age of hospitalisations with a principal diagnosis of rubella was 15 years. The male to female rate ratio of hospitalised cases with any diagnosis of rubella from 2008 to 2011 was 1.4:1 (95% CI 0.6–3.0).

Figure 2: Rubella notification, Australia, 2008 to 2012, by age category and sex



* Statistically significant difference.

Geographical distribution

From 2008 to 2012, notifications of rubella were distributed across the jurisdictions, except in the Northern Territory, where no cases were notified. The average annual jurisdiction-specific notification rate from 2008 to 2012 ranged from 0.00 per 100,000 in the Northern Territory to 0.28 per 100,000 in Western Australia. The average annual hospitalisation rate from 2008 to 2012 varied by jurisdiction ranging from 0.00 per 100,000 in Western Australia to 0.33 per 100,000 in the Northern Territory.

The apparent discrepancy between rubella notification and hospitalisation rates in the Northern Territory may be due to the different methods of data collection and classification used for these datasets, in the context of a small number of cases.

Severe morbidity and mortality

From 2008 to 2011, 156 hospital bed days were associated with diagnostic codes for rubella. Thirty-seven of these bed days were recorded for hospitalisations where the principal diagnosis was rubella. The median length of stay in hospital was 2 days for all rubella hospitalisations and 1 day for hospitalisations with a principal diagnosis of rubella.

From 2008 to 2011, the hospitalisation to notification rate ratio was 0.25 (95% CI 0.16–0.37). The average annual hospitalisation rate was highest in the 0–9 years age group (0.07 per 100,000, 8 hospitalisations). Complications arising from rubella infection were recorded for 7 out of 30 hospitalisations. The complications were distributed throughout different age groups (Table 2).

Table 2: Rubella hospitalisations, complications and length of stay , Australia, 2008 to 2011, by age group

Age	Rubella (any diagnosis) hospitalisations n	Rubella (any diagnosis) hospitalisations Rate per 100,000 population	Rubella with neurological complications n	Rubella with other complications n	Rubella without complications n	Median LOS per admission Days
0-9	8	0.07	0	1	7	1
10-19	2	0.01	0	0	2	2.5
20-29	8	0.05	0	2	6	2.5
30-39	5	0.03	0	0	5	3
40-49	2	0.01	0	1	1	5
50+	5	0.01	1	2	2	5
Total	30	0.03	1	6	23	2

LOS Length of stay.

Deaths

There were no deaths recorded as due to rubella from 2008 to 2011.

Place of acquisition

Place of acquisition was documented for 143 of 201 rubella notifications (71%) from 2008 to 2012. Of the 201 notifications, 55 (27%) were imported from overseas and 88 (44%) were locally acquired. Countries of acquisition included Vietnam, Indonesia, England, The Philippines, China, Sri Lanka, India, South Africa and Zimbabwe.

Indigenous status

Indigenous status was known for 155 of 201 (77%) rubella notifications from 2008 to 2012. Two (1%) notifications of rubella were recorded as Aboriginal or Torres Strait Islander.

Indigenous status was recorded for 28 of 30 rubella hospitalisations from 2008 to 2011. Two hospitalised cases were recorded as Aboriginal or Torres Strait Islander. Neither of these cases had a principal diagnosis of rubella.

Vaccination status

Vaccination status was recorded for 142 of 201 (71%) notifications between 2008 and 2012. Of these, 51 (36%) were recorded as unvaccinated, 16 (11%) as fully vaccinated, 7 (5%) as partially vaccinated, 2 (1%) as 'not applicable', and 66 (46%) as having unknown vaccination status. Completeness of vaccination status by state or territory during the 2008–2012 period ranged from all 34 of Queensland notifications to 7 of 64 New South Wales notifications. Data completeness for this field at the national level was above 70% in each year over this period.

Laboratory diagnosis method

The laboratory diagnosis method was recorded for 188 of 201 (93%) notifications between 2008 and 2012. Of these 169 (89%) were diagnosed by serology only, 17 (9%) by both nucleic acid testing and serology, and 1 (1%) by both culture and serology.

Congenital rubella syndrome

There was 1 notification of CRS between 2008 and 2012: a male aged less than 1 year of age notified in 2012 from the Northern Territory. The place of acquisition was recorded as Indonesia.

There was 1 hospitalisation with a principal diagnosis of CRS between 2008 and 2011, in a 1-month-old male in Victoria hospitalised for 2 days.

There were 72 hospitalisations with a secondary or other diagnosis of CRS from 2008 to 2011 contributing to a total of 608 bed days. The median length of stay was 4.5 days with a range from 1 day to 57 days. The median age at separation was 41 years, with a range of 0 to 71 years (the youngest case being a readmission of the 2008 case noted above, with the next youngest being 2 years of age).

Discussion

Between 2008 and 2012, rubella remained well controlled in Australia with an average annual notification rate of 0.18 per 100,000. The annual rubella notification rate has remained below 0.3 per 100,000 since 2003, following a marked decline in the late 1990s and early 2000s. This is well below the goal for rubella control of 1 per 100,000 endorsed by the Western Pacific Regional Office of the World Health Organization (WHO).[13]

The median age of rubella notifications in Australia between 2008 and 2012 was 29 years, compared with a median age of 27 years between 2005 and 2007 and 25 years between 2003 and 2005.[10,11] This trend of increasing age of notifications likely reflects the declining rates of rubella in children since routine MMR immunisation in infants was implemented.

The rate ratio of male to female rubella notifications during the 2008–2012 period overall was 1.3:1 (95% CI 1.0–1.8). However, this conceals differences between age categories. The male notification rate was significantly higher than the female notification rate in the 30–39 years age group (RR = 2.3, 95% CI 1.3–4.2). Men within this age group were not vaccinated in school based immunisation programs and are also likely to have had less opportunity to acquire natural immunity because of declining wild-type virus circulation due to the immunisation programs targeted at their female peers. This susceptible male population has been demonstrated in national serosurveillance data showing significant differences in seropositivity between males and females aged 25 to 40 years in 2007. The difference in seropositivity (female minus male) was statistically significant ($P < 0.001$) in the 25–29 years age group, the 30–34 years age group and the 35–39 years age group. The differences ranged from 8.7%–15.8%.[14] An over-representation of men in these age groups has also been described in recent outbreaks in Poland, Romania and Japan, which also had selective schoolgirl vaccination

policies.[15–17] Other under-vaccinated groups in Australia at risk of rubella include migrant and refugee populations.[18–20]

The average annual rubella notification rate in women of child-bearing age (15–44 years) during the 2008–2012 period was low—0.3 per 100,000—similar to that during the 2003–2007 period.[11,10] Rubella screening in pregnancy, a part of routine antenatal practice in Australia, is recommended to identify women who are non-immune, so that they can be vaccinated prior to future pregnancies.[21] Serological studies in pregnant Australian women report seropositivity of over 90%, but lower rates have been observed in women born in Asia, nulliparous women, women over 35 years of age and Aboriginal and Torres Strait Islander women living in rural areas.[22,23]

CRS is now rare in Australia. We identified 2 cases between 2008 and 2012: 1 CRS hospitalisation in 2008 and 1 notified case in 2012. This compares with a total of 9 CRS cases notified in the previous 6 year period, from 2002 to 2007.[11] The Australian Paediatric Surveillance Unit, which conducts active surveillance among paediatricians, as opposed to the passive surveillance that occurs via NNDSS, also identified 2 cases of CRS between 2008 and 2012; however both were reported in 2012.[24–28]

The low number of CRS cases documented underlines the success of the Australian rubella vaccination program. The incidence of CRS is estimated to have been 200 cases (1 in 2000 live births) annually between 1968 and 1976.[29] However, the global burden of CRS remains high, principally in countries yet to introduce rubella-containing vaccines. It is estimated that 103,068 CRS cases occurred globally in 2010, with the greatest burden in the African and South East Asian WHO regions (40,680 and 47,527 cases, respectively).[30]

Rubella elimination is a recent focus of the WHO. Elimination was announced in the Americas in 2011.[31] The European region has a revised target of elimination by 2015.[31] The Western Pacific Region, of which Australia is a member state, has endorsed a regional accelerated rubella control and CRS prevention goal to decrease rubella incidence to less than 10 cases per million population and CRS incidence to less than 10 cases per million live births by 2015 and has recently proposed a regional goal of rubella elimination, with the target year to be defined in the near future.[14,32]

The lines of evidence required to verify the elimination of rubella have been established by the American and European regions. These include epidemiology suggestive of elimination (low incidence of rubella and CRS, and the shift towards the predominance of sporadic, imported cases with limited spread) in the presence of quality surveillance; molecular epidemiology documenting interruption of endemic transmission; high levels of population immunity; and a sustainable national immunisation program.[33,34]

It can be argued that Australia meets all of these criteria, with the exception of molecular epidemiology documenting interruption of endemic transmission, due to the limited genotyping currently conducted. Our study shows low incidence of rubella and CRS. The proportion of imported cases increased from 9% to 27% of rubella notifications between 2005 and 2007[11] and 2008 and 2012 in Australia. We identified only 1 published report of a rubella cluster in Australia during the 2008–2012 period.

Immunisation coverage for rubella in Australia is high. In 2012, the percentage of children immunised with their 1st dose of rubella vaccine by 2 years of age and their 2nd dose by 5 years of age was 93.9% and 91.6% respectively.[35] Serosurveys confirm high levels of population immunity.[35] A study using Australian rubella notification, vaccine coverage, and serosurvey data calculated a reproduction number (R) less than 0.5, well below the epidemic threshold of 1.[14] A modelling study using multiple data sources, including Australian serosurvey data, estimated a 99% reduction in both rubella and CRS incidence and $R \leq 0.28$, consistent with Australia having achieved rubella elimination.[36]

Formal verification of elimination of rubella in Australia will require improvements in surveillance for rubella and CRS. Nationally consistent follow-up of notifications is needed, with improved data quality on the place of acquisition and outbreak reference fields to demonstrate the source of infection and chains of transmission. The expansion of genotype surveillance will be needed to demonstrate the absence of endemic strains. Wider use of nucleic acid testing or culture will be required to generate specimens suitable for genotyping, given that most rubella notifications are currently diagnosed by serology.

Modelling suggests that rubella control in Australia is much less vulnerable to reductions in vaccine coverage compared with measles, with decreases in coverage of up to 15% estimated to have minimal impact before 2060,[36] However, high levels of vaccine coverage still need to be maintained, particularly to protect against the potentially severe consequences of CRS, as sporadic cases of imported rubella will continue to occur as long as rubella continues to circulate in other countries.

As of 2012, 62 of 194 (32%) WHO member states have yet to introduce rubella-containing vaccines in their immunisation schedule.[31]

There are a number of limitations of this study. The true burden of rubella in Australia is likely to be underestimated since up to 50% of rubella is asymptomatic[3] and not all symptomatic cases are diagnosed and notified. The current case definition for CRS in the NNDSS does not include spontaneous abortion and hence is unable to capture the full spectrum of disease due to congenital rubella infection. However, since methods of case ascertainment and definitions have remained constant since 2004, the trends described remain valid. In rubella notification data, completeness was poor for a number of fields including country of acquisition and vaccination status. Where the vaccination status field was complete, it was often recorded as unknown. This may reflect the difficulty in obtaining an accurate vaccination history from adults in the absence of a whole-of-life immunisation register. The discrepancy in CRS cases identified in NNDSS and APSU data may be due to different methods of case ascertainment and case definitions. ICD-10-AM codes used identify cases of rubella and CRS in the National Hospital Morbidity Database were assigned for hospital billing purposes and have not been validated to clinical diagnoses or case definitions, so may be susceptible to misclassification. However, again, methodology has remained consistent over time.

In conclusion, rubella incidence remains low in Australia and CRS is increasingly rare. Formal verification of rubella elimination in Australia will require the expansion of genotype surveillance. Ongoing rubella control requires good surveillance, maintenance of high levels of vaccine coverage, and support to regional and global efforts towards rubella control or elimination.

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A FIELD STUDY OF HOUSEHOLD ATTACK RATES AND THE EFFECTIVENESS OF MACROLIDE ANTIBIOTICS IN REDUCING HOUSEHOLD TRANSMISSION OF PERTUSSIS

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Abstract

Bordetella pertussis (whooping cough) is an endemic, highly contagious bacterial respiratory infection, which is notifiable to Australian state and territory health departments. Between 2008 and 2011 there was a substantial outbreak in New South Wales with an initial increase in cases occurring in North Coast New South Wales from late 2007. During September and October 2011 the North Coast Public Health Unit conducted a household study of secondary attack rates to assess the effectiveness of pertussis vaccination as well as the timely use of antibiotics in preventing household transmission. At the time the study was commenced, notified cases included a large proportion of individuals with a documented history of vaccination against pertussis. We found lower attack rates amongst vaccinated compared with non-vaccinated subjects in all age groups, with the exception of the 5–11 years age group, who were also primarily responsible for the introduction of pertussis into the household. There was an increased risk of pertussis transmission from the household first primary case to contacts when antibiotic treatment was commenced later than 7 days after the onset of symptoms compared with within 7 days. This protective effect of timely antibiotic treatment in relation to transmission highlights the need to control for antibiotic treatment in field studies of pertussis. The benefits of timely diagnosis and use of antibiotics in preventing household transmission underscore the importance of early presentation and diagnosis of pertussis cases, particularly in households with susceptible occupants. *Commun Dis Intell* 2015;39(1):E27–E33.

Keywords: *Bordetella pertussis,* household transmission, secondary attack rates, antibiotics, waning immunity, vaccine failure

Introduction

Bordetella pertussis (whooping cough) is a highly contagious bacterial respiratory infection endemic in Australia and notifiable in each of the states and territories under the respective public health acts.[1] In New South Wales the average crude notification rate for pertussis during the years from 2004 to 2007 was 59 per 100,000 population. New South Wales experienced an outbreak of pertussis from 2008 to 2011 when the crude notification rate increased to 154 per 100,000. This state-wide outbreak was initially apparent in the northern New South Wales region covered by the North Coast Area Health Service (NCAHS) and also in Western Sydney.[2] Within the NCAHS, the crude notification rates increased from 28 per 100,000 in 2007 to 206 per 100,000 in 2011. At the same time, the proportion of notified cases followed up by the public health unit with a documented history of complete pertussis vaccination increased from 29% of cases in 2007 to 82% in the outbreak years of 2011–2012. During this time there was increased media coverage of the outbreak and also a substantial increase in the use of polymerase chain reaction (PCR) as a method of diagnosis.[2] In response to the increasing number of pertussis cases who were not fully vaccinated the NCPHU conducted a study during late 2011 to investigate the household secondary attack rates of pertussis as well as the importance of timeliness in the use of macrolide antibiotic treatment in reducing household transmission.

Methods

Following ethical approval by the North Coast Area Health Service Human Research Ethic Committee, the NCPHU recruited households within the North Coast Area of New South Wales via routine follow up of laboratory notified cases during September and October 2011. Data were collected for all household members by telephone at the initial contact, and a follow-up interview took place within 28 days after the illness onset of the 1st case in the household.

Household members were classified according to the onset date of their illness. The first primary case (FP) was the person with the earliest onset date in the household. Household contacts whose onset of symptoms was within 7 days of the FP case were classified as co-primary (CP) cases. Those with symptom onset from 7 to 28 days of the FP or CP case in the household were classified as secondary (SEC) cases. Second primary (SP) cases were those individuals whose symptoms developed

28 days after any previously identified case in the household during the study period. Those individuals who remained symptom free during the study period were classified as non-cases. Cases were identified by the following methods:

- notified to the NCPHU by the laboratory following either a positive PCR or serology test;
- clinically diagnosed by the treating doctor;
- identified at household follow up with a clinical diagnosis using the New South Wales Department of Health pertussis response protocol for Public Health Units.[3]

The vaccination status of all children in the study was checked against the Australian Childhood Immunisation Register.[4] Adults and children over 11 years were asked about history of receiving the adolescent or adult formulated pertussis vaccine (dTpa) and confirmation of vaccination was obtained through either the school-based records held at the NCPHU or from the person's general practitioner.

Fully vaccinated individuals under 12 years of age were defined as subjects who had completed the 3 course primary vaccination schedule and any booster doses required according to age by the Australian immunisation schedule.[5] We considered subjects aged 6 months and under as not vaccinated (n=4). Study subjects aged 12 years or over who had not received a dTpa vaccine in accordance with the Australian immunisation schedule between the ages of 10–17 years or as an adult as recommended between 50–65 years of age, or as parents or carers in contact with infants, were classified as not vaccinated regardless of their childhood vaccination history.[1] Recall was used to indicate the vaccination status of adults and children over the age of 11 years where immunisation records were not available (n=6).

The following households were excluded prior to analysis: households with CP cases (to avoid issues related to multiple sources of exposure to household contacts); 1 household with a PCR positive asymptomatic case; households where the immunisation status of some members was unknown; and households where some individuals had a history of receiving some but not all of the required vaccinations for their age (partial vaccination). In order to avoid potential confounding of our analysis by age, we stratified the data by age groups that reflected the number of pertussis vaccines required according to the Australian immunisation schedule.

To assess the protection of vaccination in preventing household transmission, we measured the secondary attack rate in vaccinated household contacts compared with non-vaccinated contacts following exposure to a FP case.

We estimated the relative risk (RR) of contracting pertussis among household contacts (all ages) of FP cases whose treatment with antibiotics was commenced within 7 days, compared with commencement of treatment later than 7 days, between 8–14 days, between 15–21 days and later than 21 days after onset of symptoms. Logistic regression was used to identify factors related to the risk of a household contact becoming infected with pertussis. All analysis was conducted using StataSE 9 statistical software.[6]

Results

During the 2 month study period (September and October 2011) the NCPHU received 242 pertussis notifications and completed the household follow-up for 142 (58%) of these cases. During this time, an additional 6 cases were diagnosed by a doctor on clinical grounds, bringing the total number of initial cases to 148. During the course of household follow up an additional 48 cases were identified. The final study population included 454 individuals residing in 111 households, of these 196 were classified as cases (FP, CP, SEC, SP) and 258 were non-cases.

We excluded the following cases and their household from the analysis (Figure) (19 excluded households and 89 excluded subjects):

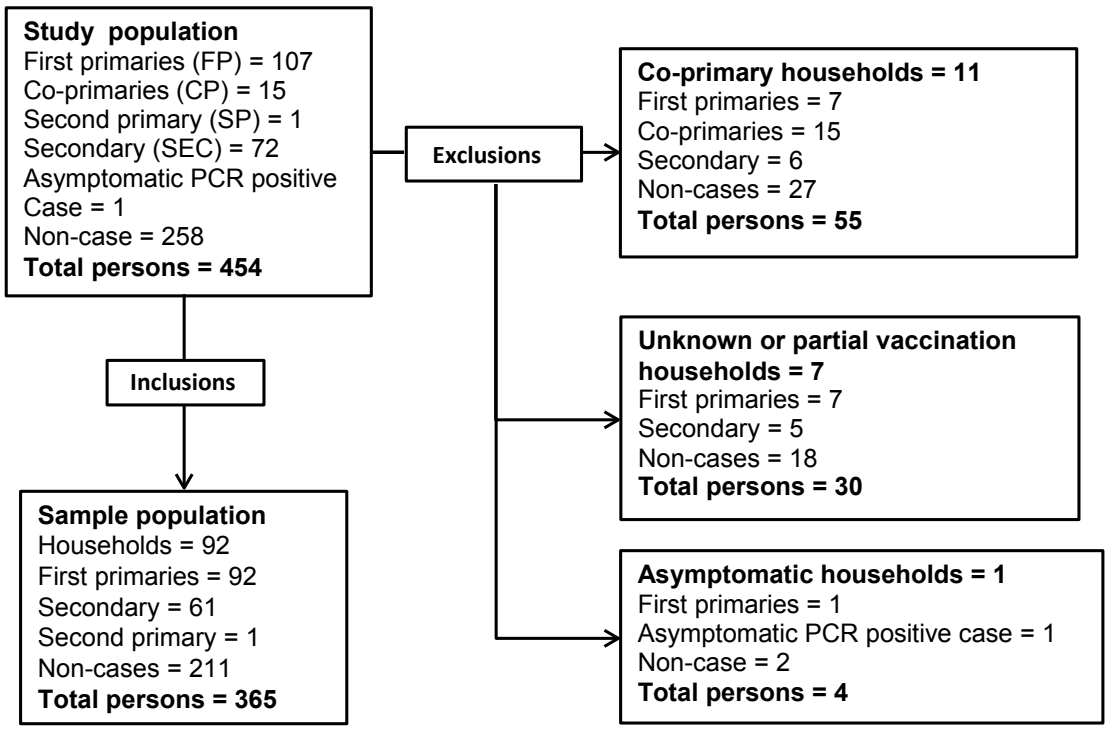
- 11 households of the 15 CP cases;
- 7 households where the vaccination status of a person was unknown or partial;
- 1 household where there was an asymptomatic PCR positive case.

Following these exclusions there were 92 households with 365 study subjects, which consisted of 92 FP, 61 SEC and 212 non-cases.

Description of study population

Table 1 summarises the study population by age group, case classification, and vaccination status. Overall, 52% of study subjects were vaccinated. There was no significant difference in the proportion of vaccinated non-cases (46%) compared with vaccinated SEC cases (54%), (difference = –8%, $P = 0.25$, 95% CI –23% to 6%). In comparison to the younger age group of 1–4 years (96% vaccinated), the 5–11 years age group (83% vaccinated) had a similar proportion of vaccinated subjects (difference = –13% $P = 0.08$, 95% CI –23% to –3%), but the proportion of vaccinated subjects decreased in the older age groups (12–19 years = 46%, difference = –50%, $P < 0.001$, 95% CI –68% to –33%), (20+ years = 29%, difference = –67%, $P < 0.001$, 95% CI –77% to –57%).

Figure: Summary of inclusions and exclusions for study subjects, North Coast household transmission study, 2011



The 20 years or over age group comprised 51% (186/365) of the total study subjects, 23% (21/92) of the FP cases, 38% (23/61) of SEC cases and 67% (142/212) non-cases. Compared with the 20 years or over age group, the 5–11 years age group comprised a smaller percentage of total study subjects at 30% (108/365), but they accounted for a significantly higher percentage of FP cases at 54% (50/92) ($P < 0.001$, 95% CI 44% to 65%) a similar percentage of SEC cases at 34% (21/61) ($P = 0.56$, 95% CI 23% to 46%) and a significantly smaller proportion of non-cases at 18% (37/212) ($P < 0.001$, 95% CI 12% to 22%). This indicated that the 5–11 years age group was predominantly responsible for introducing pertussis into the household.

Secondary attack rates

The secondary attack rate for all household contacts was 22.3%. The secondary attack rate for non-vaccinated household contacts was 19.6% compared with 25.4% for vaccinated contacts. However, the secondary attack rate was higher for non-vaccinated subjects compared with vaccinated subjects in all age groups, except for the 5–11 years age group where the attack rate was similar between vaccinated and non-vaccinated groups (Table 1).

Use of antibiotic treatment for pertussis

Thirty-four of the 92 FP cases commenced antibiotic treatment within 7 days of illness onset, 20 commenced treatment between 8–14 days of onset, 15 commenced treatment between 15–21 days of onset, 12 commenced treatment outside the recommended cut off of 21 days, 8 cases were not prescribed treatment and for the remaining 3 cases treatment was unknown. The 3 households where the FP treatment was unknown were excluded from the analysis of antibiotic treatment.

There was a statistically significantly higher proportion of vaccinated FP cases that received antibiotic treatment within 7 days compared with non-vaccinated FP cases (difference = 26% $P = 0.02$, 95% CI 6.2% to 44.8%). If the FP was vaccinated they were more than twice as likely to receive antibiotic treatment within 7 days of illness onset (RR = 2.17, $P = 0.02$, 95% CI 1.06 to 4.4). To ensure that the reduction in transmission of pertussis to household members was due to the antibiotic usage we compared the vaccination status of the contacts exposed to the FP who received timely antibiotic treatment. Within this cohort there was no difference in the proportion of vaccinated contacts compared with non-vaccinated contacts whose FP received macrolide treatment within 7 days of illness onset. ($P = 0.103$, 95% CI –2% to 22%). There was also no difference in the proportion of vaccinated contacts whose FP received macrolide treatment within 7 days compared with the proportion of vaccinated contacts whose FP had not received antibiotic treatment within 7 days of illness onset ($P = 0.10$, 95% CI –2% to 22%).

Table 1: Pertussis classification and vaccination status stratified by age, North Coast household transmissions study, 2011

Age (years)	Classification	Vaccination		Total number	SARv*	SARnv†
		Yes	No			
< 12 months	First primary	0	2	2	1	1
	Secondary	2	3	5		
	Non-case	0	0	0		
	Total	2	5	7		
1–4	First primary	10	0	10	0.56	1.00‡
	Secondary	9	1	10		
	Non-case	7	0	7		
	Total	26	1	27		
5–11	First primary	41	9	50	0.37	0.33
	Secondary	18	3	21		
	Non-case	31	6	37		
	Total	90	18	108		
12–19	First primary	0	9	9	0.06	0.09
	Secondary	1	1	2		
	Non-case	16	10	26		
	Total	17	20	37		
20+	First primary	7	14	21	0.07	0.17
	Secondary	3	20	23		
	Non-case	43	99	142		
	Total	53	133	186		
All ages	First primary	58	34	92		
	Secondary	33	28	61		
	Non-case	97	115	212		
	Total	188	177	365		

* SARv = Secondary attack rate for vaccinated.

† SARnv = Secondary attack rate for non-vaccinated.

‡ This estimated based on only 1 non-vaccinated subject.

The risk of illness was significantly increased for those contacts (all ages) exposed to a FP case whose treatment was delayed beyond 7 days compared with those exposed to a case treated within 7 days of their illness onset, RR = 3.89 (95% CI 2.00–7.55, $P < 0.001$). The risk of illness remained similar for those household contacts exposed to a FP case treated between 8–14 days and those whose FP case was treated between 15–21 days or over. Therefore in this study there was no reduction in household transmission when the FP case treatment was delayed beyond 7 days (Table 2).

Regression analysis

Logistic regression analysis investigated the odds of a contact being infected with pertussis or not. Univariate analysis found that the risk of a household contact becoming ill with pertussis was significantly associated with the household FP case not being prescribed antibiotics within 7 days of the onset of illness (OR = 5.3, 95% CI 2.5–11.3, $P < 0.001$) (Table 3). We intended to build the model using household contacts becoming ill with pertussis (non-case / secondary case) as the outcome variable, and the following predictor variables: macrolide use, age, contacts immunised and FP immunised. However, the high correlation between a contact's age and contact's immunisation status ($r^2 = -0.49$, $P < 0.001$) meant it was not possible to include both these covariates in the same multivariate model.

From the multivariate analysis, the odds of a household contact getting pertussis was found not to be significantly associated with the FP immunisation status ($P = 0.12$), but was associated with FP antibiotic use and contact age. The odds of a contact getting pertussis was more than 6 times greater (OR = 6.7, 95% CI 2.9–15.4, $P < 0.001$) if

Table 2: Risk of pertussis for contacts exposed to a first primary case treated within 7 days of onset compared with longer time periods, North Coast household transmission study, 2011

Macrolide treatment	Classification		Relative risk	95% CI
	Secondary	Non-case		
Within 7 days	9	98	3.89	2.00–7.55
> 7 days	52	107		*P*<0.001
Within 7 days	9	98	4.04	1.95–8.37
8–14 days	18	35		*P*<0.001
Within 7 days	9	98	3.15	1.45–6.88
15–21 days	13	36		*P*=0.005
Within 7 days	9	98	4.38	2.15–8.92
>21 days	21	36		*P*<0.001

Table 3: Logistic regression models for odds of household contact getting pertussis

Model	Variable	OR	Std error	*P* value	95% CI
1	Macrolide*	5.29	2.048	*P*<0.001	2.48–11.3
2	Macrolide*	5.88	2.442	*P*<0.001	2.61–13.27
	Age†	5.66	1.957	*P* <0.001	2.88–11.15
3	Macrolide*	6.69	2.849	*P* <0.001	2.91–15.41
	Age†	5.55	1.928	*P* <0.001	2.81–10.96
	FP immunised‡	1.76	0.644	*P* =0.121	0.86–3.61

* Macrolide: 0 = contact first primary (FP) received antibiotic treatment within 7 days / contact FP did not receive antibiotic treatment within 7 days.

† Age: <12 year / 12+ years.

‡ FP immunised: yes or no.

the FP did not have antibiotic treatment (i.e. treatment commenced greater than 7 days after disease onset) compared with timely treatment (within 7 days of disease onset) with antibiotics, controlling for the contacts age and FP immunisation status. The odds of a household contact getting pertussis was more than 5 times greater (OR = 5.6, 95% CI 2.8–11.0, $P < 0.001$) for contacts aged less than 12 years compared with contacts aged 12 years or over, controlling for FP antibiotic use and FP immunisation status (Table 3).

Discussion

Within our study population we found that vaccination status decreased sharply by age from a high of 96% in the 1–4 years age group, to 83% in the 5–11 years age group, and 29% in the 20 years or over age group. The 5–11 years age group was predominately responsible for introducing pertussis into the household. The secondary attack rates for vaccinated and non-vaccinated subjects in the 5–11 years age group were similar, while for the 1–4 years and 20 years or over age groups the secondary attack rates were lower in the vaccinated household contacts compared with those who were not vaccinated. These results suggest waning immunity in the 5–11 years age group and possibly a greater opportunity for exposure to pertussis at school. The vaccination rates coupled with the low secondary attack rates in adults may reflect past priming with whole cell vaccine,

We were unable to calculate vaccine efficacy due to the uneven distribution and low number of subjects across age groups, case classification and vaccination status.

The higher number of overall pertussis cases in the 5–11 years age group is consistent with a study by Fine et al (1988) who conducted a much larger household study of the protective effects of pertussis vaccine against household transmission. These authors found the pertussis vaccine provided a low protective effect to household contacts aged 5–12 years.[7]

Although Fine et al suggest this may be due to vaccine failure, more recent studies of the 2010 pertussis outbreak in California, United States of

America, identify an increase in disease incidence in this age group as being associated with waning immunity suggesting problems with durability of the acellular pertussis vaccine and the need to adjust vaccination schedules accordingly.[8–10] Although post licensure field evaluations of vaccine effectiveness have been encouraged, such studies have various methodological difficulties that may underestimate or overestimate the benefit of the pertussis vaccine.[1,7,11] Selection bias against such studies is introduced when they utilise laboratory reported cases to estimate secondary attack rates among household contacts, as our study has.[7] It is generally accepted that vaccination within households is non-random and that risk factors for vaccine failure is intra familial in respect to deficiencies in the vaccine provider and genetics.[7] Therefore, selection of immunised cases via routine pertussis reporting likely represents increased selection for vaccine failure of the entire household.

In our study, vaccine failure would likely have a disproportionate effect on estimates of secondary attack rates in the younger age groups due to these age groups having substantially higher proportions of vaccinated subjects compared with older age groups. Secondary attack rates would also be exacerbated by the problem of waning immunity in the 5–11 years age group, as there would already be a baseline proportion of the 5–11 years age group who are vaccine failures, therefore not developing immunity. Also, non-vaccinated cases that became infected with pertussis early in the outbreak would have developed natural immunity thus reducing the number of susceptible non-vaccinated people in the community later in the outbreak. Another issue is that our study was conducted late into a protracted 4-year long state wide pertussis outbreak and followed intensive media coverage. The percentage of vaccinated notified cases increased from 29% at the commencement of the outbreak period in 2008, to 82% at the time of the study in 2011. Possible reasons for the increase of notified vaccinated cases later in the outbreak may have been as a result of media attention on the outbreak leading to heightened concerns and increased reporting of pertussis by physicians and parents. Another possibility is there was an increase in testing due to the advent of the less invasive PCR test. All these issues could confound the estimation of secondary attack rates in our study.[11,12]

Few studies have analysed timely antibiotic treatment of household FP cases and the subsequent effects on pertussis transmission rates. Previous work indicates that treatment of FP cases with antibiotics (erythromycin) has reduced infection rates.[13–15] Within our study population, we found that treatment of FP cases with macrolide antibiotics or trimethoprim+sulfamethoxazol substantially decreased pertussis transmission if commenced within 7 days of the onset of symptoms. This suggests medication use is an important potential confounder in studies of pertussis transmission and such studies need to control for medication use in the study design and analysis. We detected no difference in the proportions of non-vaccinated and vaccinated contacts of the FP case who received treatment within 7 days, iIndicating that the protective effect of timely treatment was not subject to bias by the vaccination status of the contacts. In addition to this, we found no statistically significant protective effects of vaccination against transmission from a FP case as there was no difference in the secondary attack rates from vaccinated FP cases compared with non-vaccinated FP case, suggesting the reduction in transmission from these cases was due to antibiotic treatment.

These results reinforce the benefit of prompt initiation of antibiotic treatment within 7 days of the onset of symptoms to reduce pertussis transmission to household contacts. Our analysis suggests that when the treatment of pertussis with antibiotics is delayed for longer than 7 days the amount of exposure to the transmission of pertussis from the FP case to contacts negates the reduction in transmission that the antibiotic treatment provides. It is important to emphasise that antibiotic treatment is still likely to be beneficial to the person infected with pertussis in reducing symptoms and severity, and our findings regarding prescription after 7 days are in relation to reducing transmission of the disease to contacts.

The results of our regression analysis reinforced our stratified analysis that the risk of household transmission of pertussis was related to timely antibiotic use and the contact's age. The risk of a contact getting pertussis was more than 6 times greater if the FP did not have timely antibiotic treatment (within 7 days) compared with those contacts whose FP case was commenced treatment within 7 days. The risk of contracting pertussis following exposure to FP case was more than 4 times greater for contacts aged less than 12 years compared with contacts aged 12 years or over.

Vaccination status was assessed using immunisation records except for 6 subjects where subject recall was used. Of those 6 individuals, one was a member of a household excluded due to the presence of a co-primary case and the remaining 5 individuals were distributed throughout the age strata, indicting minimal change in the secondary attack rates for the two affected age groups. While we acknowledge this may have introduced some recall bias we believe any impact on our study would be negligible.

The case definitions applied in our study were based on either laboratory evidence or clinical evidence and there may have been some misclassification in the application of clinical evidence. This may have resulted in different determinations of cases among vaccinated and non-vaccinated groups resulting in inaccurate attack rate estimates.[8,13] Factors that may have resulted in case misclassification of individuals in our study include:

1. reliance on clinical history to determine case status and potential differences in reporting symptoms between immunised and non-immunised households; and
2. more likely reporting of a history of cough by parents of young children compared with older age groups resulting in a more sensitive case definition.

Our study found that children aged 5–11 years of age were the primary source of pertussis in the household and that timely antibiotic treatment of the primary household case substantially reduce pertussis household transmission. Interestingly, the use of antibiotic treatment is not generally documented in studies of household transmission estimating vaccine effectiveness and we recommend future studies assess this issue.[13] The consequential confounding effects along with methodological problems associated with using notified cases and households as the study populations make field evaluation of pertussis vaccine difficult. It is unclear if our findings on increased risk of household transmissions among younger age groups may be due to higher susceptibility, more sensitive case diagnosis, waning immunity or other factors and further research is required to clarify these issues.

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Norovirus genotype diversity associated with gastroenteritis outbreaks in Victoria in 2013

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Abstract

The noroviruses are now considered a leading cause of outbreaks of non-bacterial gastroenteritis worldwide. Vaccine strategies against norovirus are currently under consideration but depend on a detailed knowledge of the capsid genotypes. This study examined the incidence of norovirus outbreaks in Victoria over 1 year (2013) and documented the genotypes occurring in the different outbreak settings (healthcare and non-healthcare) and age groups. It was found that 63.1% of gastroenteritis outbreaks were associated with norovirus, thereby showing norovirus to be the major viral cause of illness in gastroenteritis outbreaks. Sixteen capsid genotypes were identified and included GI.2, GI.3, GI.4, GI.6, GI.7, GI.8, GI.9, GII.1, GII.2, GII.3, GII.4, GII.5, GII.6, GII.7, GII.13 and an as yet unclassified GII genotype. All genotypes found in the study, with the exception of GI.9, were detected in the elderly, indicating prior exposure to a norovirus genotype did not appear to confer long term immunity in many cases. The incidence of genotypes GII.1, GII.4 and GII.7 was linked with setting and age. As setting and age were correlated it was not possible to determine which variable was critical with the exception of GII.7, which appeared to be linked to age. The findings indicate that norovirus vaccine strategies should encompass a broad range of genotypes and, as setting or age may be important in determining genotype incidence, this should be taken into account as well. *Commun Dis Intell* 2015;39(1):E34–E41.

Keywords: norovirus, outbreaks, genotypes, vaccine strategies, healthcare, non-healthcare, setting, age

Introduction

The noroviruses, which are single-stranded positive sense RNA viruses classified in the genus *Norovirus* within the family Caliciviridae,[1] are now considered a leading cause of outbreaks of non-bacterial gastroenteritis worldwide.[1–3] Noroviruses are currently classified into 6 genogroups[1] and three of these, genogroups I, II and IV (GI, GII and GIV), occur in human infections,[1] although little is known concerning the incidence and clinical significance of GIV noroviruses in human infections.[4]

The norovirus genome comprises three open reading frames (ORFs).[1] ORF 1 encodes the non-structural polyprotein, ORF 2 encodes the major capsid protein and ORF 3 encodes the minor capsid protein.[1] Norovirus genotype classification using nucleic acid sequencing can be based on the ORF 1 region or the ORF 2 region.[5] Currently, 31 ORF 2 genotypes have been identified in norovirus genogroups I and II.[1]

The GII.4 genotype appears to be the most common in humans[6] and has been further subdivided into 'variants' or 'strains'[7,8] and successive variants are typically given a specific name.[7] In Australia, some recent GII.4 variants have been named 'Hunter' (2004–2005), 2006a (2006–2007) and 2006b (2006–2007).[7] In the period 2009–2012 the GII.4 'New Orleans 2009' variant appeared to be predominant and in late 2012 the GII.4 variant 'Sydney 2012' emerged as a major GII.4 variant in Australia.[9,10]

An understanding of norovirus genotypes is important in the ultimate management and eradication of norovirus infections. There are 2 main reasons for this. Firstly, genotype analysis enables an understanding of how noroviruses circulate throughout the community. Secondly, the development of vaccine strategies against norovirus depends on a knowledge of ORF 2 (capsid) genotypes in the community,[11,12] so documentation here is critical.

The current study documents the ORF 2 norovirus genotypes associated with norovirus gastroenteritis outbreaks in Victoria over 1 calendar year (2013) and represents the first detailed overview of ORF 2 norovirus genotypes associated with gastroenteritis outbreaks in Australia. The findings are discussed in terms of the great diversity of norovirus genotypes found and their relationship with the setting of the outbreak (healthcare vs non-healthcare) and the age of the patient. The significance of these findings for developing vaccine strategies against norovirus is discussed.

Materials and methods

Definition of gastroenteritis outbreak

For the purposes of this study an outbreak was defined as a gastroenteritis cluster, apparently associated with a common event or location, in which four or more individuals had symptoms of gastroenteritis. For an outbreak in a particular setting to be so defined at least 2 individuals had to develop gastroenteritis within 4 days of each other and for an outbreak linked to a suspect food source at least 2 individuals had to develop gastroenteritis within 4 days of consuming the suspect food.

Specimens

The faecal specimens included in this study were those sent to the Victorian Infectious Diseases Reference Laboratory (VIDRL) for norovirus testing from outbreaks that occurred during 2013. VIDRL is the main public health laboratory for viral identification in the State of Victoria. As such, it receives faecal material from gastroenteritis outbreaks reported to the Victorian health department. Outbreak specimens are also occasionally sent by other institutions such as hospitals. Only outbreaks that occurred in Victoria, were included in the study.

The date of an outbreak was taken as the onset date. If this was not available, the date the outbreak was first notified or the earliest date of collection of a specimen from the outbreak was taken as the date of the outbreak.

Outbreak setting

For data analysis, norovirus outbreaks were divided into the 2 groups of healthcare and non-healthcare. Healthcare settings included aged care facilities, disabled care facilities, hospitals, hospital geriatric ward, hospital palliative care and hospital rehabilitation unit. Non-healthcare settings included child care centres, children's activity centres, gatherings, a navy base, restaurants, schools and suspect food.

Faecal processing and RNA extraction prior to polymerase chain reaction testing

Faecal specimens were prepared as a 20% (vol/vol) suspension in Hanks' complete balanced salt solution (Sigma-Aldrich Company, Irvine, UK) and the suspension clarified with a single 10 min centrifugation spin as previously described.[13] This clarified extract was then used for RNA extraction followed by reverse-transcription polymerase chain reaction (RT-PCR). RNA extraction was carried out using the Corbett automated extraction procedure (now Qiagen Sciences, Germantown, MD, USA) essentially as described previously.[14]

Reverse-transcription polymerase chain reaction, nucleotide sequencing and phylogenetic analysis

Three 2 round RT-PCR protocols were used in the study (Table 1). For the first round of each of the 3 protocols the Qiagen (Qiagen GmbH, Hilden, Germany) OneStep RT-PCR kit that combined the RT step and the first round of the PCR was utilised. For the second round PCR the Qiagen *Taq* DNA polymerase kit was used. ORF 1 RT-PCR for GI and GII norovirus was carried out using a 2 round RT-PCR (Table 1). For studies on region C of ORF 2, GI and GII 2-round RT-PCR protocols were used (Table 1).

Nucleotide sequencing and phylogenetic analysis were carried out essentially as described previously.[18] The regions used for sequencing analysis are given in Table 1. Genotype analysis also made use of the norovirus automated genotyping tool (http://www.rivm.nl/mpf/norovirus/typingtool).[19]

ORF 2 GII.4 variant status was determined in a 3 step process. Firstly, norovirus ORF 2 sequences were tested by the norovirus automated genotyping tool.[19] If the genotyping tool assigned a variant form, it was accepted. Secondly, if the typing tool classified a variant as 'unknown' it was classified as a 'like' variant if the ORF 2 sequence of 195 bases used was no more than 2 bases different from the accepted variant reference strain; this approach yielded the 'GII.4 New Orleans_2009-like' and 'GII.4 Sydney_2012-like' GII.4 forms found in the study. Thirdly, if an ORF 2 sequence had more than 2 base changes from the known variant reference strain it was classified as 'GII.4 (unknown)'. The reference strains used for this analysis comprised GII.4 New Orleans_2009 (GU445325) and GII.4 Sydney_2012 (JX459908).

Statistical analysis

Statistical significance of differences in genotype incidence between different groups was determined by the chi-square test or by Fisher's exact two-tailed test[20] as appropriate. Only sequenceable outbreaks were used to calculate the proportions. The significance of norovirus outbreak seasonality trends was evaluated by the method of partitioning of chi-square as given by Agresti.[21] The significance of differences in average age for different settings was determined by Student's t-test with the Welch approximation.[22] The correlation between setting and age was determined using Cohen's w index from Cramer's phi coefficient.[23]

Table 1: RT-PCR protocols used

Genogroup detected	ORF	Primers (5' to 3')*	Comments	References	Fragment size for phylogenetic analysis (position relative to reference strain)
GI and GII	ORF 1	**NV 4562** GAT GCD GAT TAC ACA GCH TGG G **NV 4611** CWG CAG CMC TDG AAA TCA TGG **NV 4692** GTG TGR TKG ATG TGG GTG ACT TC **NV 5296** CCA YCT GAA CAT TGR CTC TTG **NV 5298** ATC CAG CGG AAC ATG GCC TGC C **NV 5366** CAT CAT CAT TTA CRA ATT CGG	Two-round hemi-nested RT-PCR both detects and distinguishes between GI and GII noroviruses.	Yuen et al.[15] Bruggink et al.[16]	NA
GI	ORF 2	**COG1F** CGY TGG ATG CGN TTY CAT GA **G1SKR** CCA ACC CAR CCA TTR TAC A	Two-round RT-PCR.	McIver et al[17] Bruggink et al[16]	198bp (5415–5612†)
GII	ORF 2	**G2F3** TTG TGA ATG AAG ATG GCG TCG A **G2SKR** CCR CCN GCA TRH CCR TTR TAC AT	Two-round RT-PCR.	McIver et al. [17] Bruggink et al. [9]	195bp (5133–5327‡)

* D=AGT, H=ACT, W=AT, M=AC, R=AG, K=GT, Y=CT, N=AGCT.
† Reference strain Norwalk (accession number M87661).
‡ Reference strain Camberwell (accession number AF145896).
NA Not applicable.
RT-PCR Reverse-transcription polymerase chain reaction.

Experimental plan

All faecal specimens received at VIDRL for norovirus testing were initially screened by the ORF 1 RT-PCR, which detects and distinguishes between GI and GII noroviruses. If more than 1 specimen from a particular individual in a given outbreak was received, only the 1st specimen from that individual was tested. In addition, 1 specimen from every outbreak, chosen without bias, was also tested by both the ORF 2 GI and ORF 2 GII RT-PCRs and nucleotide sequencing performed if a positive result was obtained. The data were then analysed as follows: an outbreak was classified as norovirus positive if at least 1 specimen from the outbreak was positive by the ORF 1 and/or the ORF 2 assays. For the genotype analysis performed in the current study only sequences generated by the ORF 2 assays were used.

Results

Norovirus outbreak incidence, setting and temporal variation

For the calendar year 2013 faecal specimens from 301 gastroenteritis outbreaks were received for testing, and of these, 170 outbreaks were found to be positive for norovirus by the ORF 1 PCR and a further 20 were negative by the ORF 1 PCR but positive by an ORF 2 PCR. Thus a total of 190 outbreaks (63.1% of all outbreaks) were positive for norovirus, indicating it was the major cause of gastroenteritis.

Of these 190 outbreaks, 165 (86.8%) could be classified as healthcare and 25 (13.2%) as non-healthcare. A breakdown of the individual outbreak settings within these 2 categories is given in Table 2.

The monthly incidence of norovirus outbreaks and all outbreaks tested for 2013 is given in the Figure. It was noted that the 2-monthly incidence of norovirus outbreaks in January–February (i.e. late summer) was significantly higher than in any of the other 2-monthly periods in the year ($P < 0.005$, partitioning of chi-square).

Figure: The monthly incidence of norovirus outbreaks and all outbreaks tested, Victoria, 2013

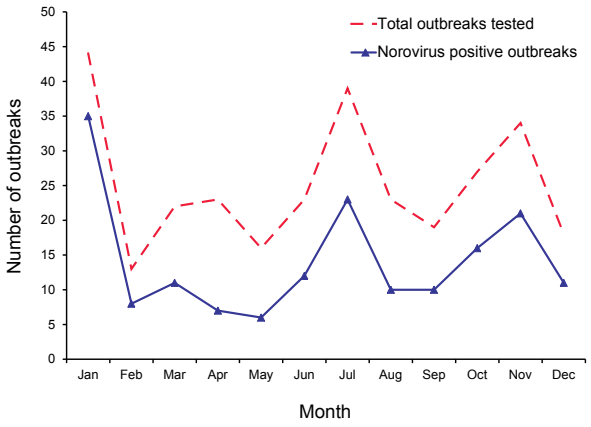

Table 2: Settings of norovirus outbreaks, Victoria, 2013

Healthcare	Number of norovirus outbreaks	Percentage of healthcare	Percentage of all norovirus outbreaks
Aged care facility	135	81.8	71.1
Disabled care facility	4	2.4	2.1
Hospital	21	12.7	11.1
Hospital – geriatric ward	2	1.2	1.1
Hospital – palliative care	1	0.6	0.5
Hospital – rehabilitation unit	2	1.2	1.1
Total	165	100.0	86.8
Non-healthcare	**Number of norovirus outbreaks**	**Percentage of non-healthcare**	**Percentage of all norovirus outbreaks**
Child care centre	8	32.0	4.2
Children's activity centre	2	8.0	1.1
Gathering	5	20.0	2.6
Navy base	1	4.0	0.5
Restaurant	5	20.0	2.6
School	1	4.0	0.5
Suspect food	3	12.0	1.6
Total	25	100.0	13.2

ORF 2 genotypes and settings

Sixteen ORF 2 genotypes were identified in the study (Table 3). Of these, the GII.4 genotype was the most common in both healthcare settings (63%) and non-healthcare settings (32%). However, GII.4 was significantly more common in outbreaks in healthcare settings than in non-healthcare settings ($P < 0.001$, chi-square test). A number of GII.4 variants were identified including GII.4 New Orleans_2009-like, GII.4 Sydney_2012 and GII.4 Sydney_2012-like. It was also noted that the genotypes GII.1 and GII.7 were significantly more common in non-healthcare settings than in healthcare settings ($P < 0.0012$ and $P < 0.032$, respectively, Fisher's exact two-tailed test).

Using the norovirus automated genotyping tool, an 'untypeable' GII ORF 2 norovirus sequence was detected in a 90-year-old individual associated with a gastroenteritis outbreak in November 2013. A BLAST search indicated the sequence had 99% nucleotide identity with the 2013 Korean strain KF774001. The strain identified in the current study has been lodged in GenBank as KM025343.

Table 3: ORF 2 norovirus genotypes and settings

Norovirus ORF 2 genotypes seen in healthcare settings	Number of norovirus outbreaks	Percentage of healthcare	Percentage of all norovirus outbreaks
GI.2	1	0.6	0.5
GI.3	2	1.2	1.1
GI.4	4	2.4	2.1
GI.6	2	1.2	1.1
GI.7	1	0.6	0.5
GI.8	1	0.6	0.5
GII.1	1	0.6	0.5
GII.2	1	0.6	0.5
GII.3	1	0.6	0.5
GII.4 New Orleans_2009-like	1	0.6	0.5
GII.4 Sydney_2012	67	40.6	35.3
GII.4 Sydney_2012-like	25	15.2	13.2
GII.4 unknown	11	6.7	5.8
GII.5	4	2.4	2.1
GII.6	2	1.2	1.1
GII.7	3	1.8	1.6
GII.13	8	4.8	4.2
GII.unknown	1	0.6	0.5
No sequence available	29	17.6	15.3
Total	165	100.0	86.8
Norovirus ORF 2 genotypes seen in non-healthcare settings	**Number of norovirus outbreaks**	**Percentage of non-healthcare**	**Percentage of all norovirus outbreaks**
GI.2	1	4.0	0.5
GI.9	1	4.0	0.5
GII.1	4	16.0	2.1
GII.3	1	4.0	0.5
GII.4 Sydney_2012	4	16.0	2.1
GII.4 Sydney_2012-like	4	16.0	2.1
GII.6	2	8.0	1.1
GII.7	3	12.0	1.6
GII.13	1	4.0	0.5
No sequence available	4	16.0	2.1
Total	25	100.0	13.2

ORF 2 genotypes and age

The relationship between age and norovirus ORF 2 genotype is presented in Table 4. It can be seen that healthcare settings tended to represent an older age demographic whereas non-healthcare settings tended to represent a younger age demographic. The average age of individuals (of known age) in healthcare settings was 82.6 years (standard deviation = 12.6 years, range = 23–98 years, n = 163) whereas the average age of individuals (of known age) in non-healthcare settings was 26.0 years (standard deviation = 25.2 years, range = 0–77 years, n = 24). This difference in average ages was statistically significant ($P < 0.001$, Student's t test with the Welch approximation).

All genotypes found in the study occurred in the older age group (66 years of age or over) with the exception of GI.9. Many of the genotypes found in the older age group were also detected in younger individuals (Table 4).

GII.4 norovirus was significantly more common in individuals 66 years of age or over than in those 65 years of age or under ($P < 0.001$, chi-square test). In contrast, GII.1 and GII.7 were significantly more common in those 65 years of age or under than in those 66 years of age and over ($P < 0.031$ and $P < 0.0006$ respectively, Fisher's exact two-tailed test).

Table 4: Age ranges and ORF 2 genotypes found in one representative individual from each outbreak

ORF 2 genotypes	Age ranges				
	0–15 years	16–45 years	46–65 years	>65 years	Unknown
Healthcare settings					
GI.2				1	
GI.3				2	
GI.4				4	
GI.6		1		1	
GI.7				1	
GI.8				1	
GII.1				1	
GII.2				1	
GII.3				1	
GII.4		2	7	94	1
GII.5				4	
GII.6			1	1	
GII.7		1	1	1	
GII.13			1	7	
GII.unknown				1	
No sequence available			2	26	1
Total	0	4	12	147	2
Non-healthcare settings					
GI.2	1				
GI.9			1		
GII.1	2		1		1
GII.3	1				
GII.4	3	3	2		
GII.6	1			1	
GII.7	1	2			
GII.13	1				
No sequence available	1	2		1	
Total	11	7	4	2	1

ORF 2 genotypes, setting and age

The incidence of GII.1 and GII.4 was linked to both setting and age. However, setting and age were closely correlated; chi-square analysis of setting vs age gave a value for Cohen's w index of 0.8, which indicates a strong correlation between setting and age.[19] Thus the data does not permit discrimination as to whether the critical variable was setting or age for these genotypes.

The data also indicates that there was a significant relationship between the incidence of the genotype GII.7 and setting and age. However, in this case it is possible to discriminate which variable is the critical one. In healthcare settings, GII.7 was significantly more common in those 65 years of age or under ($P < 0.029$, Fisher's exact two-tailed test). In non-healthcare settings, GII.7 was only found in individuals 65 years of age or under. These results suggest that for this genotype age was the critical variable.

Discussion

The current study indicates that in 2013 norovirus outbreaks occurred throughout the year although they were found to peak in warmer months of the year. Previous studies on the periodicity of norovirus outbreaks in Victoria have shown a similar trend,[24] indicating that 2013 was a typical year for norovirus incidence. The outbreaks occurred in a broad range of settings and individuals of all ages were affected.

This report is the first to systematically examine ORF 2 (capsid) genotypes associated with gastroenteritis outbreaks in Australia and a marked diversity of genotypes was found. Sixteen capsid genotypes were identified and included GI.2, GI.3, GI.4, GI.6, GI.7, GI.8, GI.9, GII.1, GII.2, GII.3, GII.4, GII.5, GII.6, GII.7, GII.13 and an unknown GII genotype. This unknown strain probably represents a new, as yet unclassified, norovirus genotype.

The current study indicates that the incidence of some genotypes, notably GII.1, GII.4 and GII.7, was linked to both setting and age. Since setting and age were linked to each other it is difficult to determine which was the more important. However, for one genotype, GII.7, age appeared to be the critical variable, with significantly greater incidence in younger age groups, possibly indicating long term immunity with this genotype. The relationship between the incidence of GII.7 and the age of infected individuals does not appear to have been examined in detail in the mainstream literature and warrants further study.

Of the genotypes detected, GII.4 was by far the most common in both healthcare settings and non-healthcare settings but was significantly more common in healthcare settings. This finding reinforces the findings of an earlier study in this laboratory, which examined the incidence of GII.4 norovirus, classified on the basis of ORF 1 sequencing,[25] in Victoria. In contrast to GII.4, genotypes GII.1 and GII.7 were found to be more common in non-healthcare settings than in healthcare settings. The precise relationship between the frequency of detection of these latter 2 genotypes and outbreak setting does not appear to have been examined in the mainstream literature and also warrants further study.

Although GII.4 (ORF 2) norovirus was the most common genotype in this and related studies[1], the findings of the current report indicate that norovirus vaccine strategies that target only GII.4 could exclude numerous other norovirus genotypes in both healthcare and non-healthcare settings. Norovirus vaccine strategies should encompass a broad range of genotypes and, as setting/age may be important in determining genotype incidence, this should be taken into account as well.

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Short report

Review of 2005 Public Health Laboratory Network *Neisseria gonorrhoeae* nucleic acid amplification tests guidelines

David M Whiley, Monica M Lahra on behalf of the National Neisseria Network

Abstract

At the request of the Public Health Laboratory Network (PHLN), the National Neisseria Network (NNN) met to discuss the 2009 PHLN *Neisseria gonorrhoeae* nucleic acid amplification test (NAAT) guidelines and the need for supplementary testing. A central point of discussion at this NNN meeting, which took place in May 2013, was the potential for *N. gonorrhoeae* supplementary testing to lead to false-negative results. Data were presented at the meeting that questioned the sensitivity of commonly used in-house supplementary methods as compared with later generation commercial NAAT systems. It was the opinion of the NNN that supplementary testing remains best practice, but that caution should be used when reporting negative results. The NNN recommends that urogenital samples providing a positive result in a screening method and a negative result by a supplemental method should not be reported as negative for *N. gonorrhoeae* without an appropriate explanatory comment indicating that gonococcal infection cannot be excluded. *Commun Dis Intell* 2015;39(1):E42–E45.

Keywords: gonorrhoea, nucleic acid amplification tests, NAAT, supplementary

Nucleic Acid Amplification Tests (NAATs) for diagnosis of gonococcal infection are increasingly utilised in laboratories but their use has been complicated by specificity problems since their introduction. This is mostly due to frequent exchange of genetic material between commensal *Neisseria* species and *Neisseria gonorrhoeae*[1–3] and cross-reactivity with commensal *Neisseria* strains has been observed with most *N. gonorrhoeae* NAAT methods.[3] Given the potential medical, legal, social or psychological implications that may arise from an incorrect gonorrhoea diagnosis, laboratories have a duty to avoid issuing false positive results. This has prompted the development of the Australian Public Health Laboratory Network's (PHLN) *Guidelines for the use and interpretation of nucleic acid detection tests for Neisseria gonorrhoeae in Australia* in 2005.[4] Briefly, the 2005 Guidelines state that all *N. gonorrhoeae* NAAT positive results should also test positive on a reliable supplemental assay before a positive result is reported. In response, most clinical laboratories in Australia implemented supplemental NAAT methods (typically in-house polymerase chain reaction (PCR) methodology) for this purpose.

Whilst the implementation of supplemental testing has largely resolved the specificity problems associated with *N. gonorrhoeae* NAAT-based testing, new concerns have been raised about the overall sensitivity of the NAAT algorithm (i.e. false-negative results). At the National Neisseria Network (NNN) annual meeting in Canberra on 30–31 May 2013, three key data sets were presented as follows:

1. Data showing that sequence target variability may impact upon the sensitivity of in-house real-time polymerase chain reaction methods

In recent years there have been several reports of genetic mutations in gonococcal strains that have led to false-negative results in some in-house real-time PCR methods. Such problems have been observed in Australia for assays targeting the *porA* pseudogene, the *opa* genes and *cppB* gene.[5–7] Data kindly provided by the Royal College of Pathologists Quality Assurance Programs (RCPAQAP) indicate that *porA* and *opa*-based PCR methods are widely used by Australian laboratories for supplementary testing, and so there is potential for *porA* or *opa* variant strains to cause false-negative results in testing algorithms. However, a recent nationwide analysis of Australian gonococci (n = 2,455 isolates) conducted by the NNN showed that the prevalence of *porA, opa* and *cppB* variant strains is low (0.12%, 0.04% and 1.14 % of gonococci respectively) and not widespread throughout Australia at this point in time.[7] Hence, the overall impact of such variants may in fact be minimal. Ongoing monitoring of strains for genetic variation in sequences targeted by NAAT assays is critical.

2. Data showing that supplementary testing may lead to false-negative results for 'low load' samples

Data kindly provided by the RCPAQAP indicate that it is not uncommon for laboratories to correctly detect *N. gonorrhoeae* nucleic acid in a quality assurance program sample by a screening NAAT method, but then fail to detect *N. gonorrhoeae* in the same sample by a supplementary NAAT method. Such discrepancies are typically observed for samples that provide the highest cycle threshold (Ct) values in the screening methods (where such data are available), suggesting that the issue relates to low DNA loads. It should be noted that RCPAQAP does occasionally deliberately select gonococcal strains that are known to lack certain sequence targets (e.g. *porA* pseudogene variants) to use in their panels, and that this does explain some of the RCPAQAP discrepancies. However, for this point we are primarily concerned with RCPAQAP samples that are known to contain gonococcal nucleic acids, for which there are no known sequence target issues, yet provide positive results in screening methods and negative results in supplementary methods. For example, for one 2013 sample there were 11 laboratories that obtained a positive result by a commercial screening method and that also separately reported the individual results for their supplementary methods; of these, 6 laboratories obtained negative results in the supplementary tests.

Unpublished data from NNN laboratories also show that up to 5% of urogential samples and 20% of pharyngeal samples positive by a later generation NAAT, are not detected by a supplemental assay. Examples of these data are provided in Tables 1 and 2; samples are from The Canberra Hospital (n = 369) and The Prince of Wales Hospital (n = 1,174) where in-house real-time PCR (targeting the gonococcal *opa* genes and/or *porA* pseudogene) were used to confirm samples testing positive by the Roche 4800 NG PCR Assay. Further data were shown indicating that the majority of samples that are 'screen positive/supplementary negative' typically provide the highest Ct values in the screening methods, again suggesting low DNA loads are involved. For example, a subset of samples (n = 427) from The Prince of Wales Hospital sample set showed that 'screen positive/supplementary negative' samples (n = 98) provided an average Ct value of 38.3 cycles by the Roche 4800 NG PCR, whereas those that were positive by supplementary PCR (n = 329 samples) provided an average Ct value of 32.2 cycles.

Table 1: Results of supplementary testing of samples providing positive results by the Roche 4800 NG PCR Assay, The Canberra Hospital, 2011 to 2013

Sample site	Roche 4800 *Neisseria gonorrhoeae* positive	Supplementary PCR (*porA* pseudogene)		
		Positive	Negative	Confirmation rate (%)
Urogenital	152	146	6	96.1
Rectal	106	95	11	89.6
Throat	110	88	22	80.0
Eye	1	1	0	100.0

PCR Polymerase chain reaction

Table 2: Results of supplementary testing of samples providing positive results by the Roche 4800 NG PCR Assay, The Prince of Wales Hospital, 2011 to 2013

Sample site	Roche 4800 *Neisseria gonorrhoeae* positive	Supplementary PCR (*opa* genes and *porA* pseudogene)		
		Positive	Negative	Confirmation rate (%)
Urogenital	245	234	11	95.5
Rectal	601	562	39	93.5
Throat	325	276	49	84.9
Eye	3	2	1	66.6

PCR Polymerase chain reaction

3. Data showing later generation commercial *Neisseria gonorrhoeae* nucleic acid amplification tests methods are more specific

Published data by Tabrizi et al.[8] and others indicate that later generation *N. gonorrhoeae* NAAT methods have substantially less cross-reactivity with non-gonococcal *Neisseria* species compared with earlier generation methods. It was however noted that cross-reactions were still possible, as evidenced by recent studies.[9,10]

The above data are highly suggestive that true gonococcal infections (particularly those with low bacterial load) are providing positive results in a screening method but negative results upon supplemental testing. Whilst sampling issues at low load leading to 'hit and miss' results are a well-recognised limitation of NAAT technology, this is not of primary concern. The key issue here is how such results are being interpreted and reported. The NNN discussions revealed that different laboratories handle such results in different ways; including:

a.) issuing the results as negative i.e. *gonococcal infection not detected;*

b.) issuing the results as *equivocal* or *indeterminate;*

c.) reporting both the screening and supplementary results; or

d.) a, b or c but with a comment discussing the discrepancy.

At the Canberra NNN meeting there was considerable discussion over how these issues should be addressed. These discussions included debate over whether the 2005 *N. gonorrhoeae* NAAT PHLN guidelines remain relevant, particularly in light of recent improvements in the specificity of the commercial systems. It was also highlighted that the Australian guidelines are amongst the most stringent in the world and that other regions (e.g. *United Kingdom National Guideline for Gonorrhoea Testing 2012*[11]), only recommend the use of supplementary testing for extra-genital samples (frequented by commensal *Neisseria* species, being the key source of *N. gonorrhoeae* NAAT cross-reaction) and not urogenital samples. The consensus opinion of the NNN was that (1) the PHLN guidelines remain best practice for *N. gonorrhoeae* NAAT testing, and that (2) the requirement for supplementary testing should not be relaxed, even for urogenital samples.

It was also the opinion of the NNN that gonococcal infection cannot be excluded for urogenital samples that provide positive results in a later generation *N. gonorrhoeae* NAAT method, but negative results upon supplementary testing. In the light of escalating rates of gonorrhoea infection in Australia and elsewhere, combined with concerns over emerging antimicrobial resistance, it is the opinion of the NNN that laboratories should err on the side of caution when issuing such results for urogenital samples. In such instances a laboratory should not issue a negative result in the absence of an appropriate explanatory comment. While the precise wording of the comment may be determined by the respective laboratory, at a minimum the comment should indicate that *N. gonorrhoeae* infection cannot be excluded and that re-collection should be considered where warranted. At the NNN meeting there was no consensus as to whether discrepant results should be issued as 'negative' or 'indeterminate'; however the US Centers for Disease Control and Prevention[12] suggests that an interpretation of 'inconclusive', 'equivocal', or 'indeterminate' would be most appropriate. Again, this may depend on local requirements.

Furthermore, the above also highlights that some laboratories may need to change their supplementary NAAT methods so as to improve assay performance. A review of recent results in *N. gonorrhoeae* quality assurance panels would help ascertain if individual laboratories have a potential problem with assay performance.

In summary, the NNN advocates ongoing adherence to the guidelines laid out in the 2005 PHLN *N. gonorrhoeae* NAAT document; however it recommends that appropriate explanatory comments are provided with results for urogenital samples so as to negate any potential negative impacts that may arise through the use of supplementary testing.

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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 multi-jurisdictional 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 Department of Health web site (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 2007*[1] received royal assent. This Act provides a legislative basis for and authorises the exchange of health information, including personal information, between jurisdictions and the Commonwealth. The Act provides for the establishment of the National Notifiable Diseases List,[2] which specifies the diseases about which personal information can be provided. The National Health Security Agreement,[3] which was 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 Welfare[7] 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

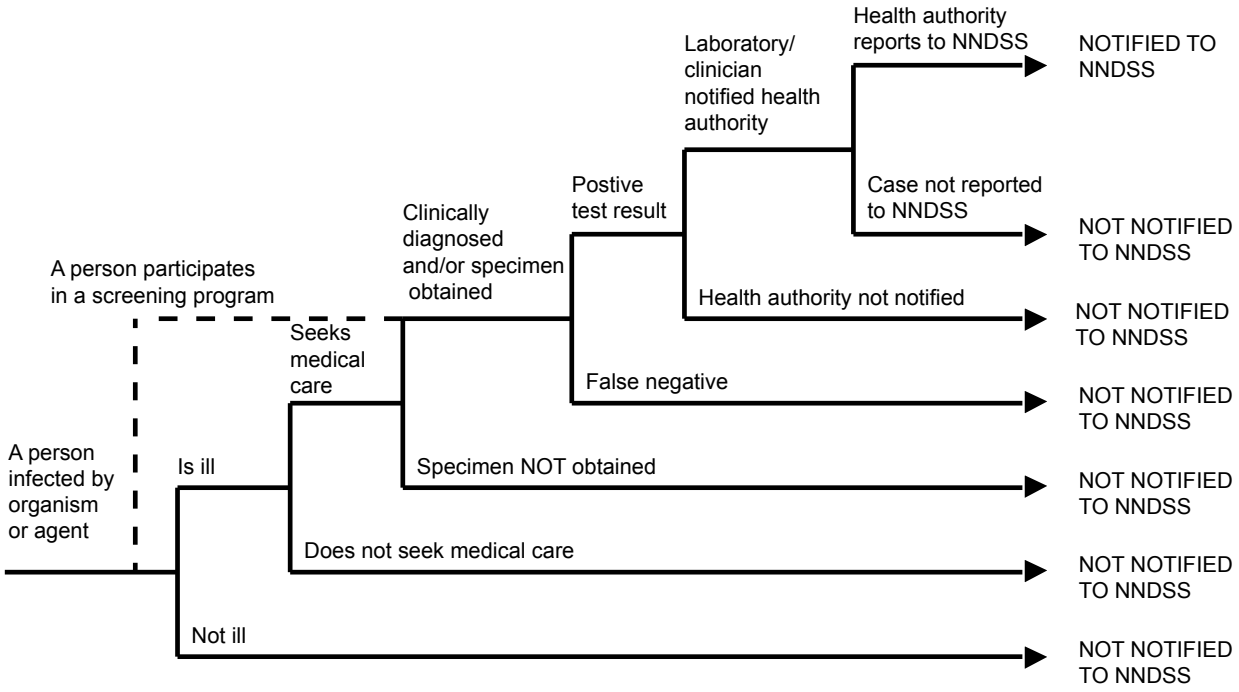


Table 1: Percentage* of notifications from different sources, 2012, by state or territory

State or territory	Source of notifications		
	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

* 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
Tuberculosis	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 tests[13] and periodic public awareness campaigns[14] 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 Department of Health web site (http://www.health.gov.au/casedefinitions).

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

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

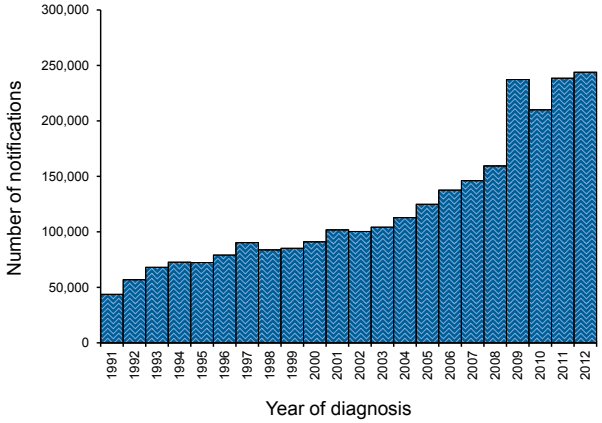


Table 4: Notifications of nationally notifiable communicable diseases, Australia, 2012, by state or territory

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Bloodborne diseases									
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0
Hepatitis B (newly acquired)*	2	29	5	55	16	10	52	24	193
Hepatitis B (unspecified)†	104	2,298	200	808	383	62	1,855	799	6,509
Hepatitis C (newly acquired)*	15	47	0	NN	77	23	179	125	466
Hepatitis C (unspecified)†,‡	132	3,243	195	2,376	394	242	2,055	1,011	9,648
Hepatitis D	0	5	0	6	8	0	9	2	30
Gastrointestinal diseases									
Botulism	0	0	0	0	0	0	0	0	0
Campylobacteriosis	477	NN	175	4,182	2,161	882	5,870	1,906	15,653
Cryptosporidiosis	19	687	234	1,371	162	42	460	168	3,143
Haemolytic uraemic syndrome	0	10	0	4	0	1	5	0	20
Hepatitis A	1	42	3	34	7	2	62	14	165
Hepatitis E	1	10	0	6	0	0	17	1	35
Listeriosis	0	39	0	5	4	3	34	8	93
Salmonellosis	241	2,951	407	2,825	845	280	2,547	1,169	11,265
Shigellosis	6	124	107	82	48	7	120	53	547
STEC,VTEC§	5	13	2	27	45	7	11	1	111
Typhoid fever	1	43	4	15	3	1	38	18	123
Quarantinable diseases									
Cholera	0	2	0	1	2	0	0	0	5
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0
Plague	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0
Yellow fever	0	0	0	0	0	0	0	0	0
Sexually transmissible infections									
Chlamydial infection‖,¶	1,283	21,293	2,532	18,849	4,848	1,787	20,312	11,803	82,707
Donovanosis	0	0	0	0	0	0	0	1	1
Gonococcal infection¶	92	4,129	1,536	2,700	499	35	2,543	2,115	13,649
Syphilis – congenital¶	0	0	0	0	0	0	0	0	0
Syphilis – all¶,**,††	28	793	81	639	131	24	980	217	2,893
Syphilis < 2 years duration¶,††	15	510	14	383	52	14	474	77	1,539
Syphilis > 2 years or unknown duration†,¶,††	13	283	67	256	79	10	506	140	1,354
Vaccine preventable diseases									
Diphtheria	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	2	0	5	2	1	4	1	15
Influenza (laboratory confirmed)	666	7,998	435	16,853	6,288	1,093	5,990	5,240	44,563
Measles	0	170	2	4	6	0	11	6	199
Mumps	6	105	0	32	7	1	30	19	200
Pertussis	429	5,828	298	7,536	904	1,276	4,423	3,375	24,069
Pneumococcal disease (invasive)	27	579	72	348	131	45	385	235	1,822
Poliomyelitis	0	0	0	0	0	0	0	0	0
Rubella	1	10	0	9	2	1	11	2	36
Rubella – congenital	0	0	1	0	0	0	0	0	1
Tetanus	0	1	0	2	1	0	2	1	7
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	4,481
Varicella zoster (unspecified)	121	NN	4	4,414	92	84	2,633	1,105	8,453

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

Disease	State or territory								
	ACT	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

* 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 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.

‡‡ 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	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Bloodborne diseases									
Hepatitis (NEC)	–	–	–	–	–	–	–	–	–
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	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)†,‡	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
Gastrointestinal diseases									
Botulism	–	–	–	–	–	–	–	–	–
Campylobacteriosis	127.2	NN	74.4	91.6	130.5	172.2	104.3	78.3	101.6
Cryptosporidiosis	5.1	9.4	99.5	30.0	9.8	8.2	8.2	6.9	13.8
Haemolytic uraemic syndrome	–	0.1	–	0.1	–	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
Listeriosis	–	0.5	–	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
STEC,VTEC§	1.3	0.2	0.9	0.6	2.7	1.4	0.2	<0.1	0.5
Typhoid	0.3	0.6	1.7	0.3	0.2	0.2	0.7	0.7	0.5
Quarantinable diseases									
Cholera	–	<0.1	–	<0.1	0.1	–	–	–	<0.1
Highly pathogenic avian influenza in humans	–	–	–	–	–	–	–	–	–
Plague	–	–	–	–	–	–	–	–	–
Rabies	–	–	–	–	–	–	–	–	–
Severe acute respiratory syndrome	–	–	–	–	–	–	–	–	–
Smallpox	–	–	–	–	–	–	–	–	–
Viral haemorrhagic fever	–	–	–	–	–	–	–	–	–
Yellow fever	–	–	–	–	–	–	–	–	–
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	<0.1
Gonococcal infection¶	24.5	56.6	653.1	59.1	30.1	6.8	45.2	86.9	60.1
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
Vaccine preventable diseases									
Diphtheria	–	–	–	–	–	–	–	–	–
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
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
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
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

Disease	State or territory								
	ACT	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.

¶ 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.

‡‡ 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

Disease	Number of notifications								Notification rate per 100,000					
	2007	2008	2009	2010	2011	2012	5-year mean	Ratio (2012 : 5-year mean)	2007	2008	2009	2010	2011	2012
Bloodborne diseases														
Hepatitis (NEC)	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
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)†‡	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	1	0	1	0	2	0	0.8	0.0	<0.1	–	<0.1	–	<0.1	–
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	9	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	90	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	5	3	6	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	–	–	–	–	–	–
Plague	0	0	0	0	0	0	0.0	0.0	–	–	–	–	–	–
Rabies	0	0	0	0	0	0	0.0	0.0	–	–	–	–	–	–
Severe acute respiratory syndrome	0	0	0	0	0	0	0.0	0.0	–	–	–	–	–	–
Smallpox	0	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	0	0	0	2	0	0.4	0.0	–	–	–	–	<0.1	–

Table 6 *continued:* Notifications and notification rate for communicable diseases, Australia, 2007 to 2012

Disease	Number of notifications								Notification rate per 100,000					
	2007	2008	2009	2010	2011	2012	5-year mean	Ratio (2012 : 5-year mean)	2007	2008	2009	2010	2011	2012
Sexually transmissible infections														
Chlamydial infection[‖,¶]	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	3	2	1	1	0	1	1.4	0.7	0.014	0.009	0.005	0.005	–	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	6	3	3	7	0	5.2	0.0	0.03	0.03	0.01	0.01	0.03	–
Syphilis – all[¶,**,††]	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[†,¶,††]	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	–	–	–	–	<0.1	–
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	1	0	0	0	0	0	0.2	0.0	<0.1	–	–	–	–	–
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	2	0	0	0	0	1	0.4	2.5	<0.1	–	–	–	–	<0.1
Tetanus	3	4	3	2	3	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	8	24	20	9	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	1	0	0	0	1	0.2	5.0	–	<0.1	–	–	–	<0.1
Kunjin virus infection[‡‡]	1	1	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	1	4.6	0.2	–	<0.1	<0.1	–	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

Table 6 *continued:* Notifications and notification rate for communicable diseases, Australia, 2007 to 2012

Disease	Number of notifications						5-year mean	Ratio (2012 : 5-year mean)	Notification rate per 100,000					
	2007	2008	2009	2010	2011	2012			2007	2008	2009	2010	2011	2012
Zoonoses														
Anthrax	1	0	0	1	0	0	0.4	0.0	<0.1	–	–	<0.1	–	–
Australia bat lyssavirus	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Brucellosis	37	45	32	21	39	29	34.8	0.8	0.2	0.2	0.1	0.1	0.2	0.1
Leptospirosis	108	111	141	132	215	116	141.4	0.8	0.5	0.5	0.7	0.6	1.0	0.5
Lyssavirus (NEC)	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Ornithosis	93	102	65	61	90	75	82.2	0.9	0.4	0.5	0.3	0.3	0.4	0.3
Q fever	449	378	311	335	352	358	365.0	1.0	2.1	1.8	1.4	1.5	1.6	1.6
Tularaemia	0	0	0	0	2	0	0.4	0.0	–	–	–	–	<0.1	–
Other bacterial diseases														
Legionellosis	303	272	301	302	358	382	307.2	1.2	1.4	1.3	1.4	1.4	1.6	1.7
Leprosy	14	11	5	13	8	4	10.2	0.4	0.1	0.1	<0.1	0.1	<0.1	<0.1
Meningococcal infection§§	305	286	259	228	241	223	263.8	0.8	1.4	1.3	1.2	1.0	1.1	1.0
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.

† 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.

¶ 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.

‡‡ 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.

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

Table 7: Year which diseases become notifiable to the National Notifiable Diseases Surveillance System, Australia, by state or territory*

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA		
Bloodborne diseases										
Hepatitis 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	NN	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	

Table 7 *continued*: Year which diseases become notifiable to the National Notifiable Diseases Surveillance System, Australia, by state or territory*

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA		
Sexually transmissible infections										
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	
Gonococcal infection§	1991	1993	1991	1991	1991	1991	1991	1991	1991 to present	
Syphilis – all‖	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Syphilis < 2 years	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Syphilis > 2 years or unspecified duration	2004	2004	2004	2004	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	NN	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
Ross River virus infection	1993	1993	1991	1991	1993	1993	1991	1991	1993 to present	

Table 7 *continued*: Year in which diseases become notifiable to the National Notifiable Diseases Surveillance System, Australia, by state or territory*

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA		
Zoonoses										
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	

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

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

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

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

|| Includes syphilis – congenital from 1991 to 2002.

¶ Includes rubella – congenital from 1991 to 2002.

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

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

NN Not notifiable

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.

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

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	NDP	NDP	NDP	NDP	NDP	NDP	NDP	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

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 The Kirby Institute web site (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]

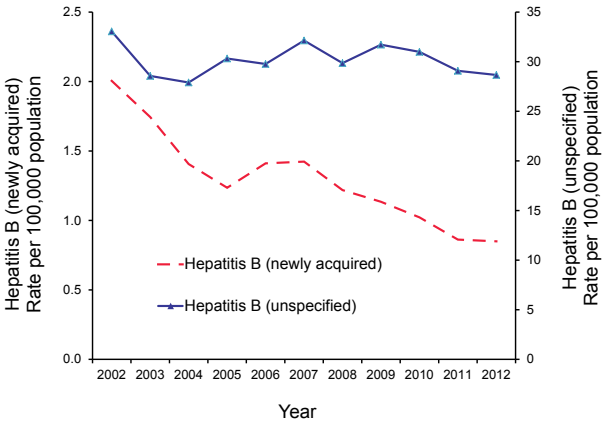
Newly acquired hepatitis B

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).

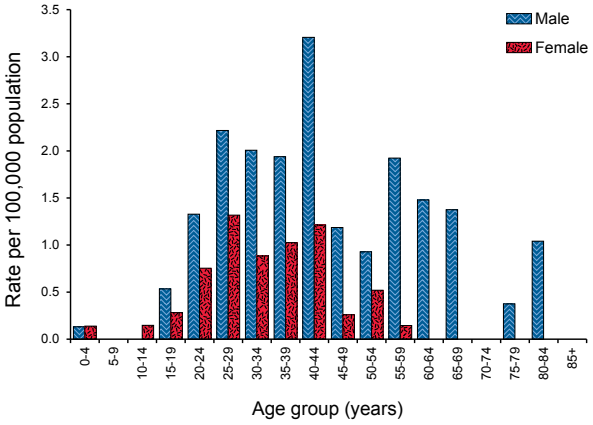
Figure 3: Notification rate for newly acquired hepatitis B* and unspecified hepatitis B,† Australia, 2002 to 2012, by year



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

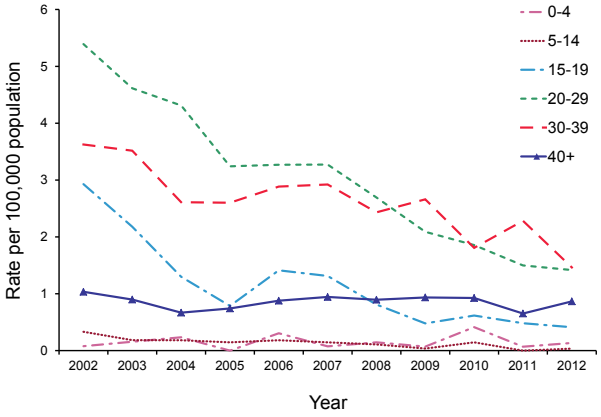
† Data for unspecified hepatitis B for all states and territories, excluding the Northern Territory between 2002 and 2004

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.

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]

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.

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

Risk factors	Number of risk factors reported			Percentage of total cases§ (n=108)
	Male	Female	Total	
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‖	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	–

* 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

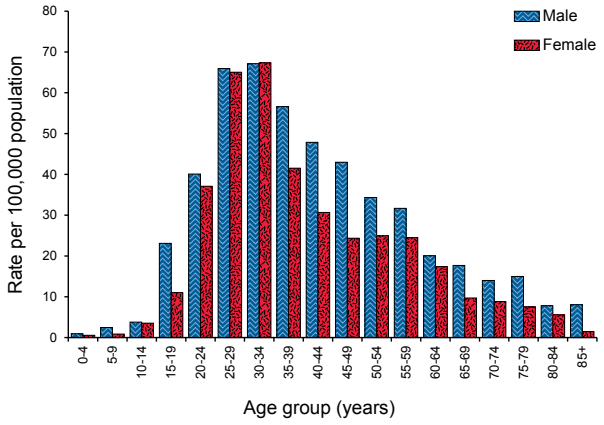
Epidemiological situation in 2012

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).

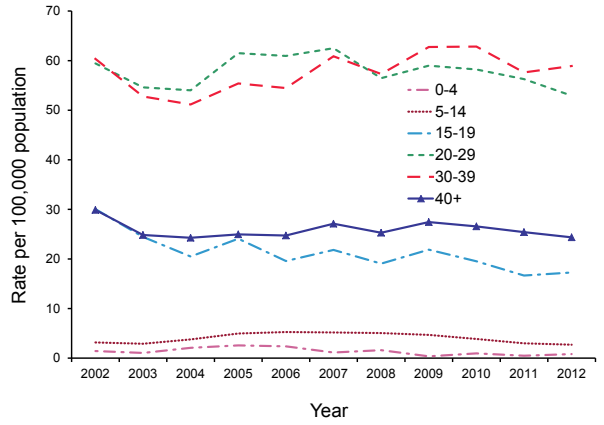
Figure 6: Notification rate for unspecified hepatitis B,* Australia, 2012, by age group† and sex



* 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%.

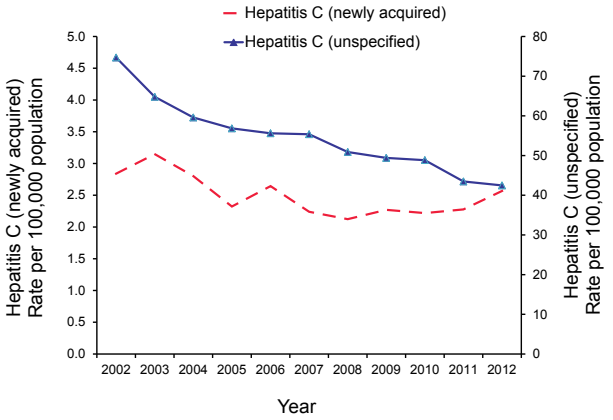
Hepatitis C causes inflammation of the liver. In more than 90% of cases initial infection with hepatitis C virus is asymptomatic or mildly symptomatic. Approximately 50%–80% of cases go on to develop a chronic infection. Of those who develop a chronic infection, half will eventually develop cirrhosis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

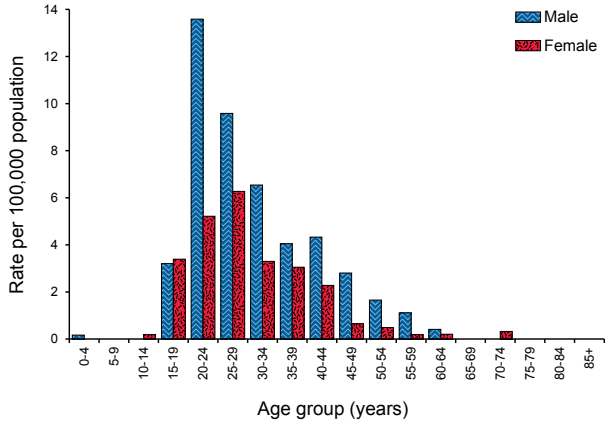
Newly acquired hepatitis C

- 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†



* Data from all states and territories except Queensland.

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

Geographical distribution

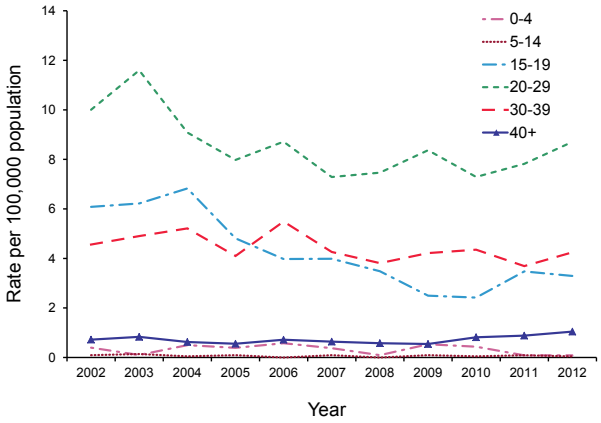
The highest rates of newly acquired hepatitis C infection were reported in Western Australia (5.1 per 100,000), South Australia (4.6 per 100,000) and Tasmania (4.5 per 100,000) (Table 5). The proportion of newly acquired infections compared with total hepatitis C diagnoses varied substantially among 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.

Age and sex distribution

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†,‡

Risk factors	Number of risk factors reported			Percentage of total cases (n=341)§
	Male	Female	Total	
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‖	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.[26]

Geographical distribution

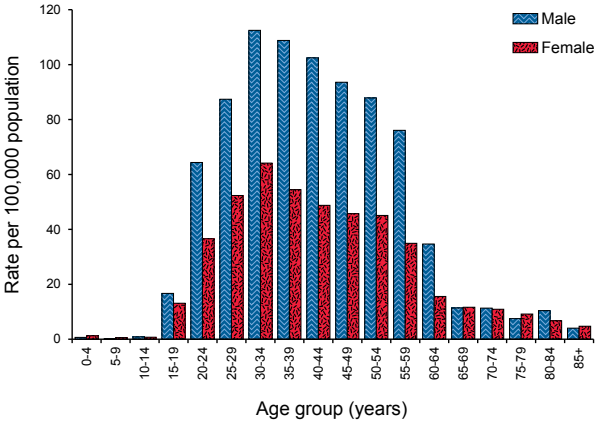
In 2012, the Northern Territory continued to have the highest notification rate (82.9 per 100,000) followed by Queensland (52.0 per 100,000) (Table 5).

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 (54.5 per 100,000) and 25–29 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.

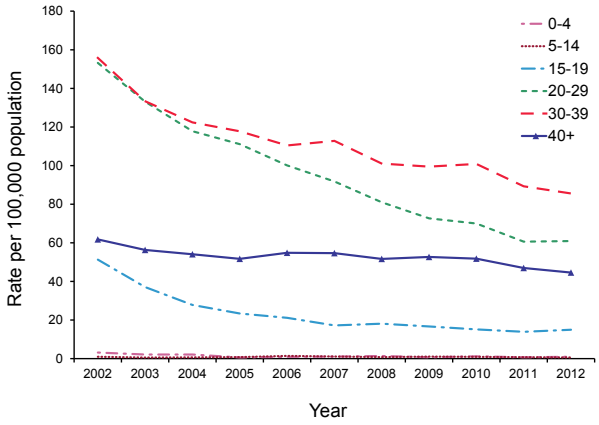
Figure 11: Notification rate for unspecified hepatitis C,*† Australia, 2012, by age group and sex



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

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

Figure 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).

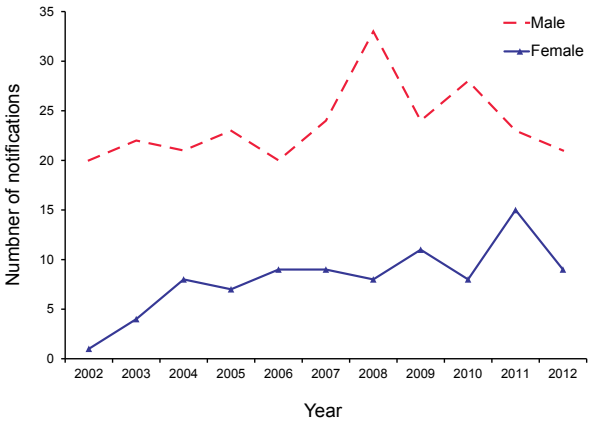
Geographical distribution

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).

Figure 13: Notifications of hepatitis D, Australia, 2002 to 2012, by year and sex



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

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

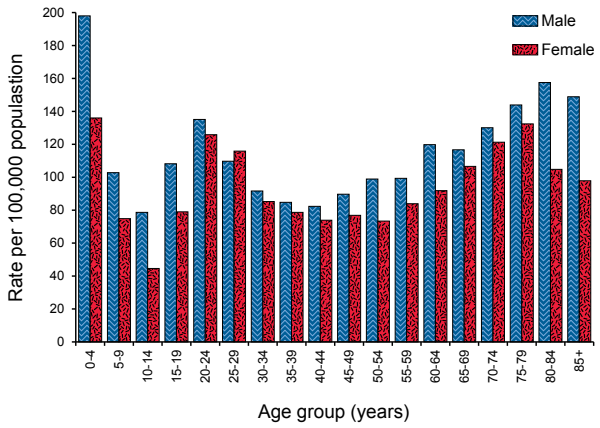
Epidemiological situation in 2012

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).

Figure 14: Notification rate for campylobacteriosis, Australia, 2012, by age group and sex



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 large-volume watery diarrhoea. Ingesting contaminated water, typically from a recreational source like a community swimming pool or lake, is a major risk factor for infection.[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

	Locally		Overseas		Unknown		
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 faecal-oral 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.

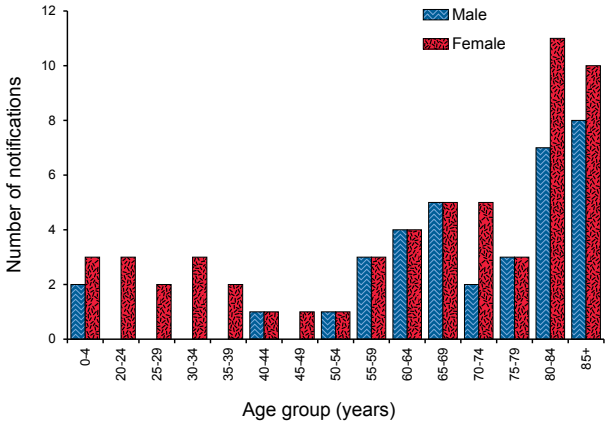
Epidemiological situation in 2012

There were 93 notified cases of invasive *L. monocytogenes* 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 non-typhoidal 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 Department of Health web site (www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-quaranti-index.htm) and on the Department of Foreign Affairs and Trade Smartraveller web site (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 1996[36] 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

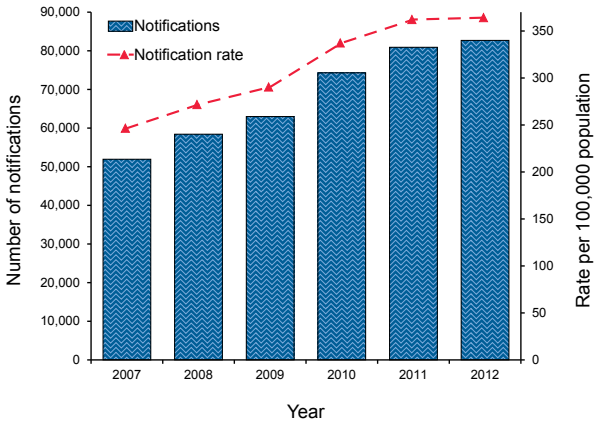
- 82,707 cases of chlamydial infection were notified in 2012.
- 2012 notification rates were similar to 2011.
- Women under 25 years of age and Indigenous people were disproportionately represented in the notifications of chlamydial infection.

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

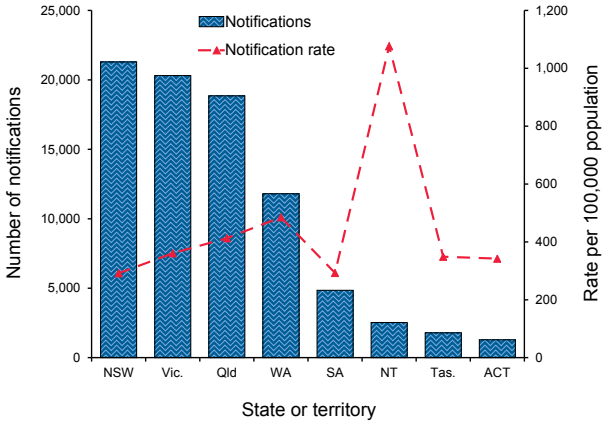


Geographical distribution

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.

Figure 17: Notifications and notification rates for chlamydial infection, Australia, 2012, by state or territory



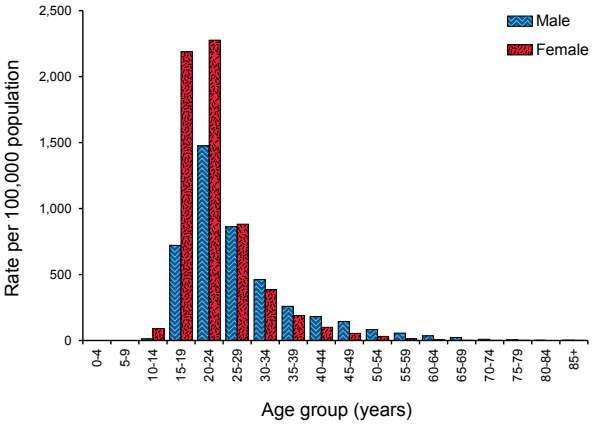
Age and sex distribution

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]

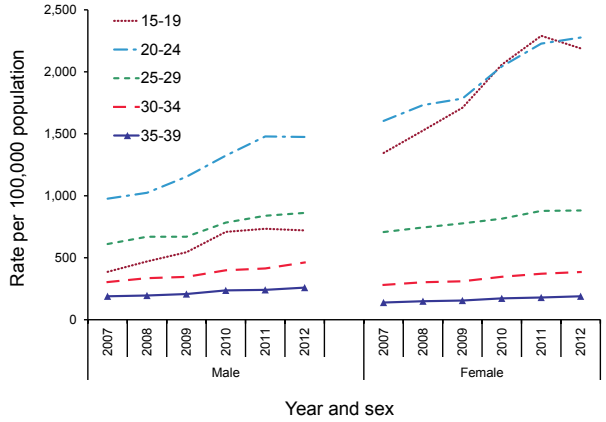
When considering trends over time in those aged 15–39 years, notification rates increased almost every year, for all age groups and for both sexes (Figure 19). The exceptions were between 2011 and 2012, when rates declined in males aged 15–19 years and 20–25 years, and females aged 15–19 years.

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).

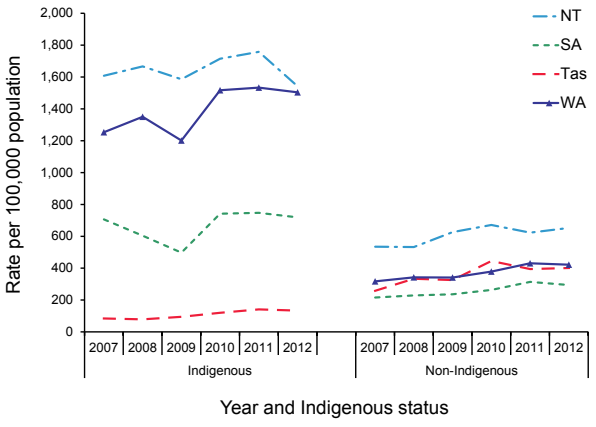
Among the Indigenous population, the age-standardised notification rate declined from 1,344 per 100,000 in 2011 to 1,252 per 100,000 in 2012. This followed increases in 2009, 2010 and 2011 (1,115, 1,321 and 1,344 per 100,000 respectively), which in turn followed a decline in 2008 (1,180 per 100,000).

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).

Figure 20: Age standardised notification rates of chlamydial 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, South Australia, Tasmania and Western Australia.

Donovanosis

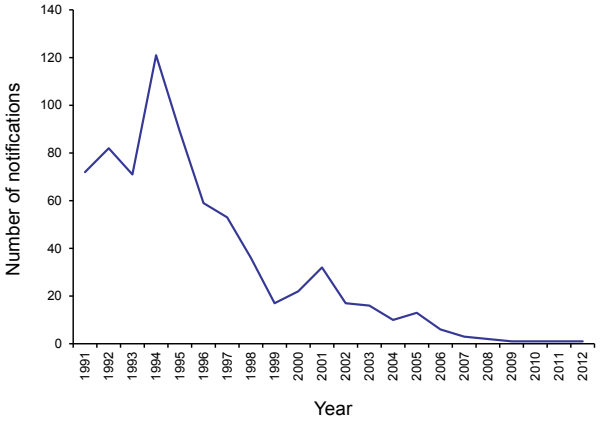
- One case of donovanosis was notified in 2012.
- This disease is now rare in Australia.

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

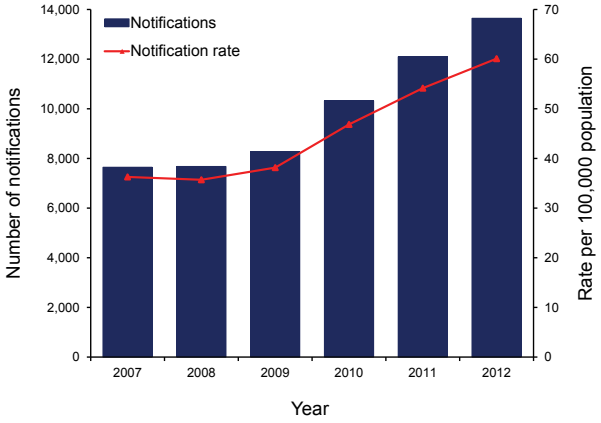
- 13,649 cases of gonococcal infection were notified in 2012.
- Notification rates of gonococcal infection continue to increase.
- Notifications in 2012 occurred predominately in males aged 20 years or over.

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

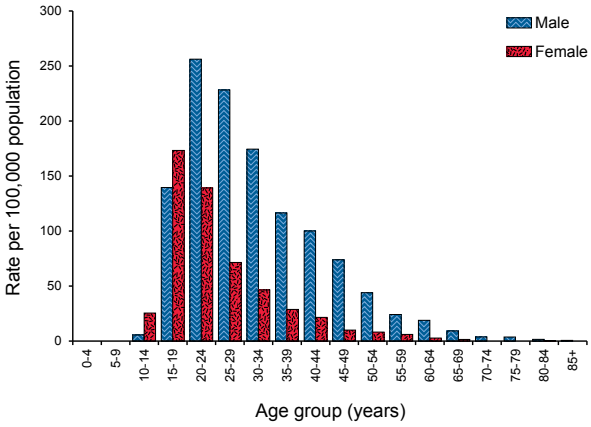
653 per 100,000), and Queensland (from 66 to 59 per 100,000). Between 2007 and 2012, all states and territories have seen overall increasing rates of gonococcal infection have been observed in all states and territories, except for the Northern Territory and Tasmania.

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).

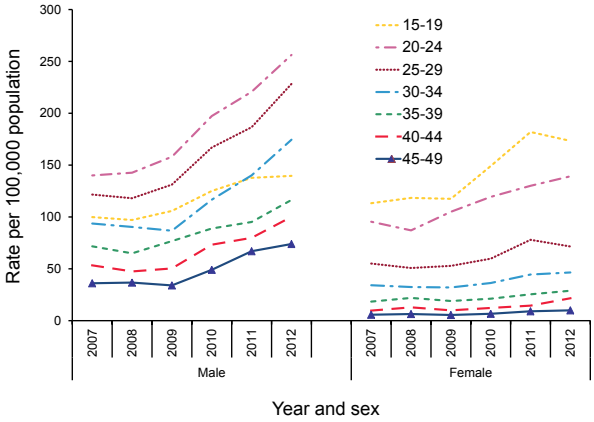
Figure 23: Notification rate for gonococcal infection, Australia, 2012, by age group and sex*



* 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).

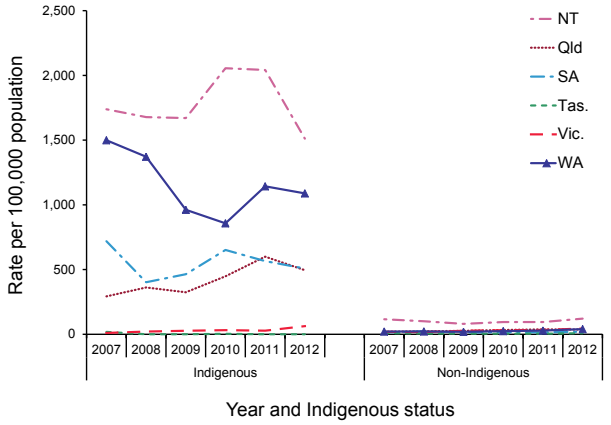
The age-standardised notification rate among the non-Indigenous population almost doubled from 2007 to 2012 (20 and 38 per 100,000 respectively). The average annual increase over this period was 14% (range: 5%–21%).

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. gonorrhoeae* 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 (non-congenital) 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.

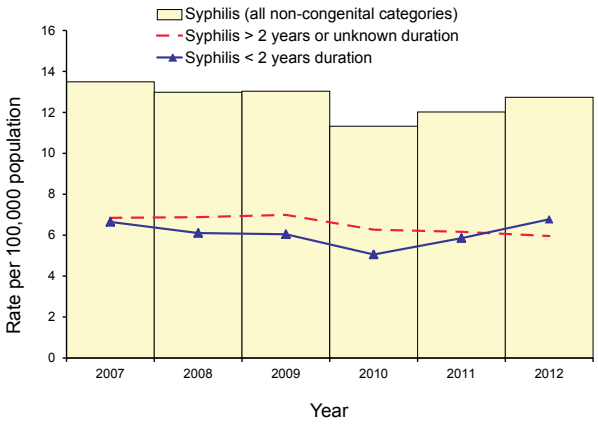
Epidemiological situation in 2012

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 non-congenital 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.

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

State or territory	Male		Female		Total*	
	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

* Includes notifications for whom sex was not reported.

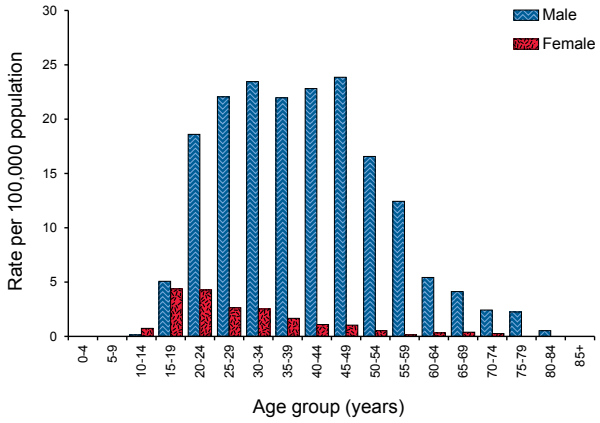
† Per 100,000 population.

‡ 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*

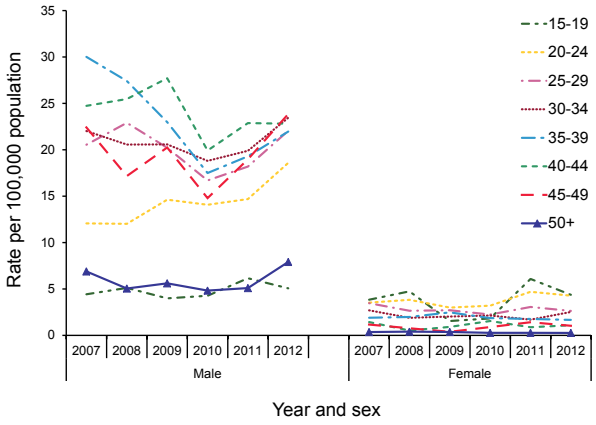


* Excludes notifications for whom age and/or sex were not reported and notifications where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

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.

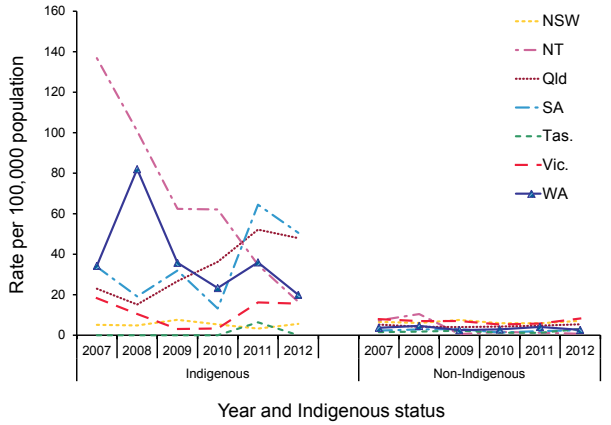
Among these states and territories, the combined age standardised notification rate ratio between the Indigenous and non-Indigenous populations in 2012 was 4.0:1, which is lower than the preceding 5-year mean of 5.4:1 (range: 4.4–6.0).

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).

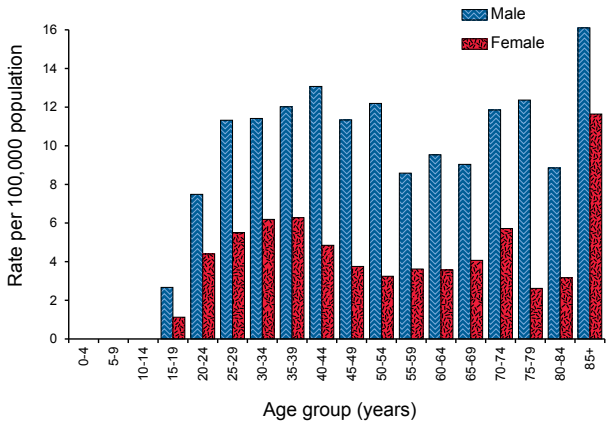
Geographical distribution

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.

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

State or territory	Male		Female		Total*	
	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

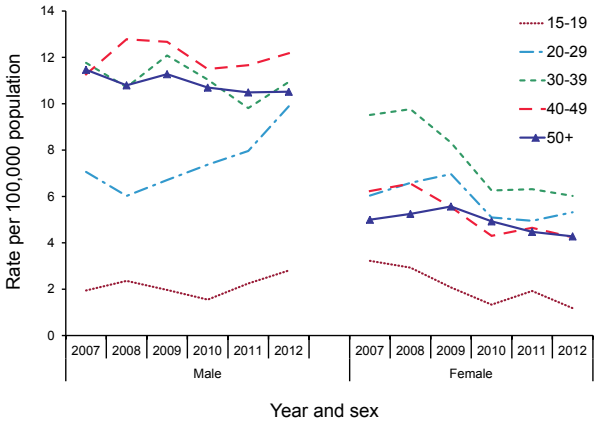
* Includes notifications for whom sex was not reported.

† 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

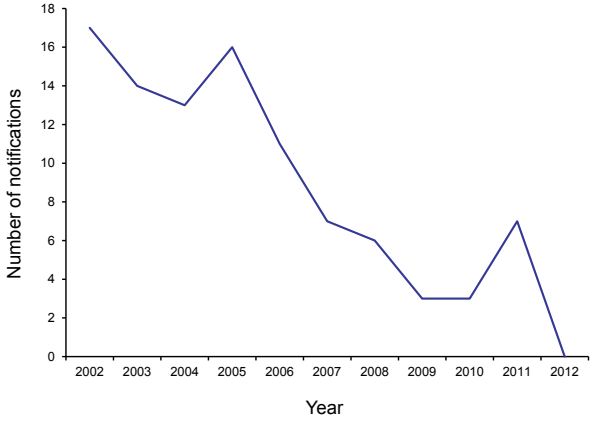
- No cases of congenital syphilis were notified in 2012.
- Congenital syphilis remains rare in Australia.

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]

Figure 32: Notifications of congenital syphilis, Australia, 2002 to 2012, by year



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 pneumococcal disease (IPD), poliomyelitis, rubella, tetanus and varicella zoster infections (chickenpox, shingles and unspecified). Data on hepatitis B and invasive meningococcal disease, which are also targeted by the NIP, 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 Handbook*[20] 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 asymptomatic[51] 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]

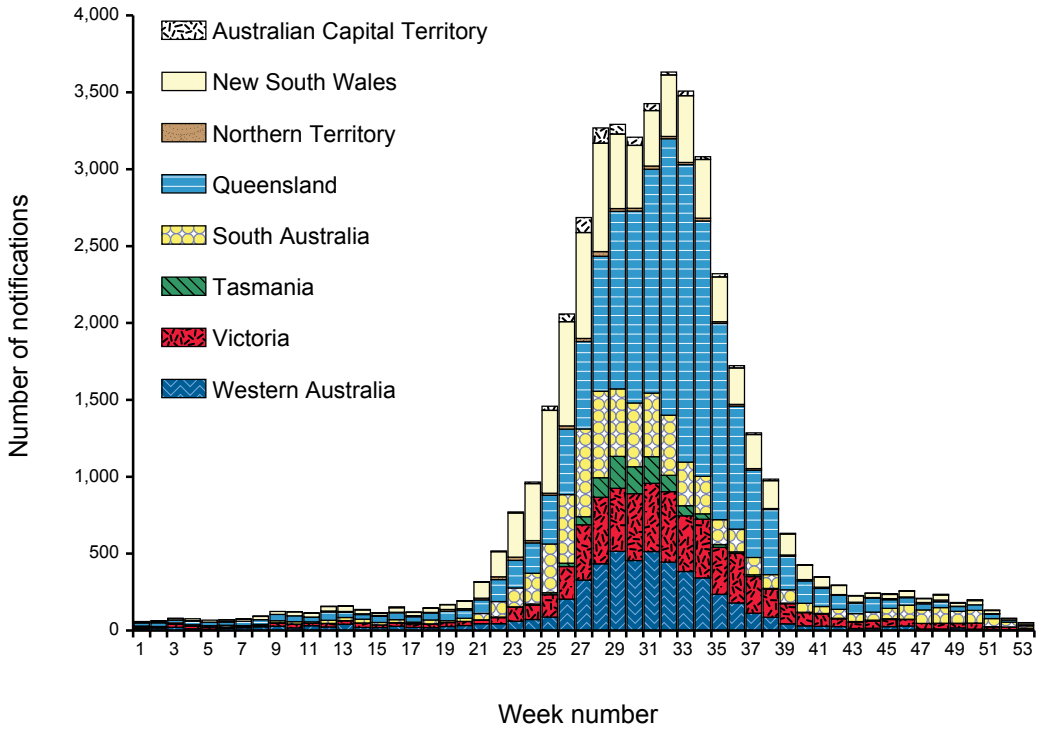
Vaccination

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).

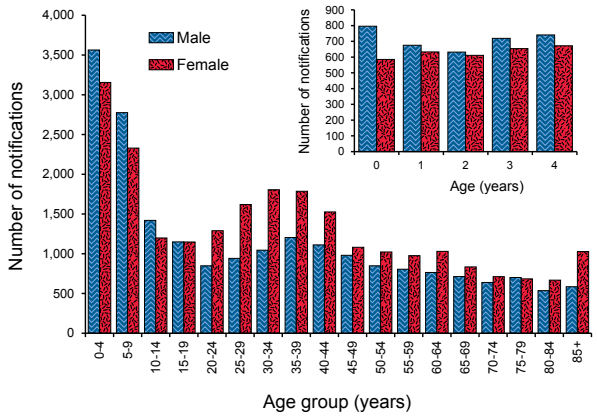
Figure 33: Notifications of laboratory confirmed influenza, Australia, 2012, by week and state or territory



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]

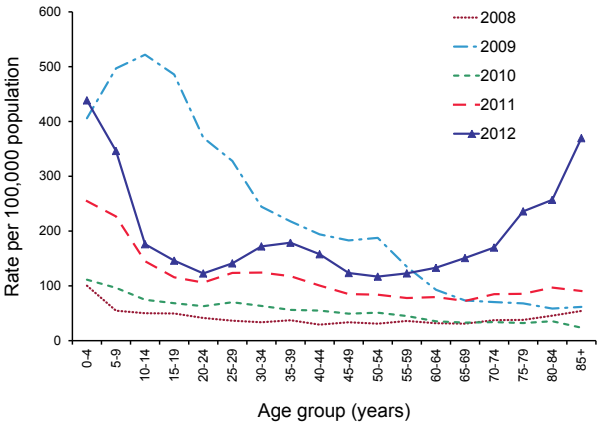
Figure 34: Notifications of laboratory confirmed influenza, Australia, 2012, by age group and sex*



* 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).

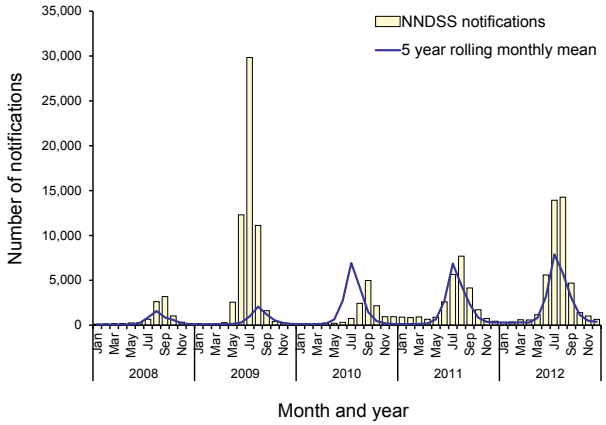
Figure 35: Notification rate for laboratory confirmed influenza, Australia, 2008 to 2012, by age group and year



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

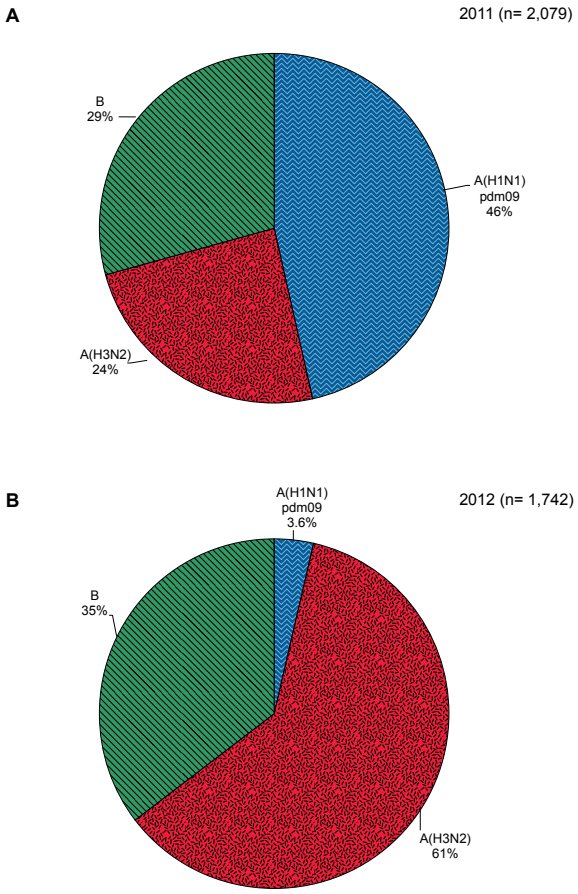
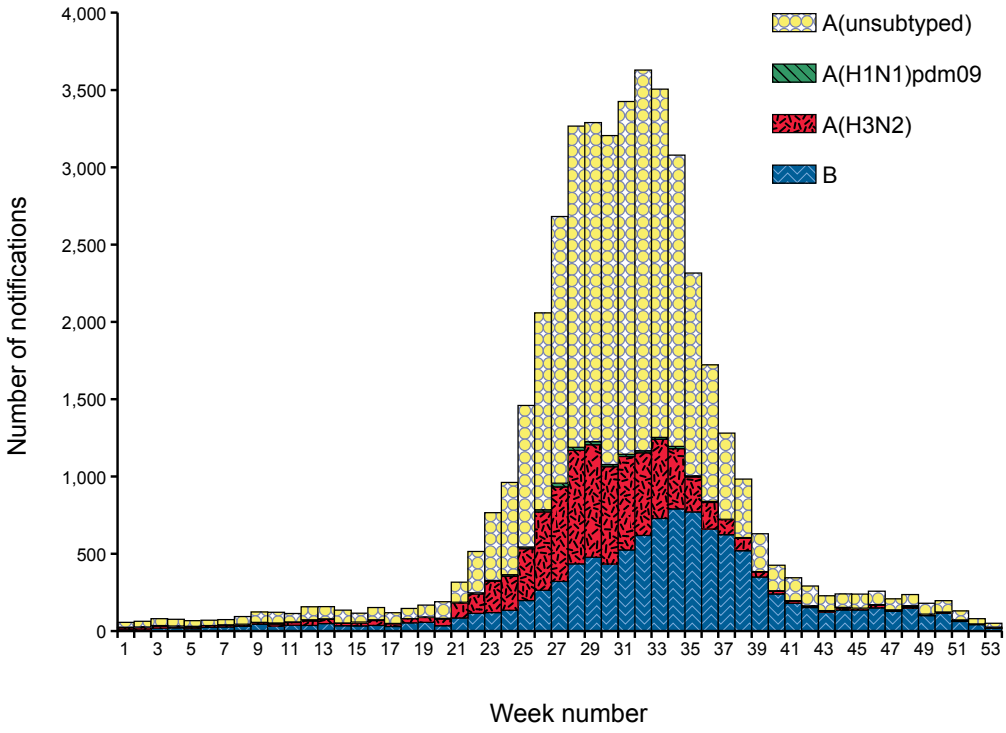


Figure 37: Notifications of laboratory confirmed influenza,* Australia, 2012, by week and subtype



* 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/60/2008-like (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.

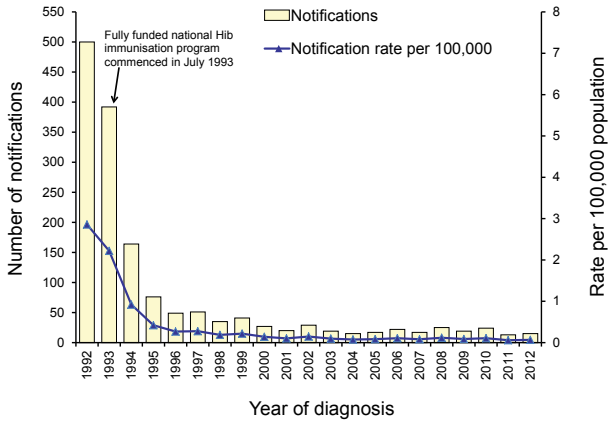
Invasive Hib is a bacterium that causes disease with symptoms dependant on which part of the body is infected. These include: septicaemia (infection of the blood stream); meningitis (infection of the membranes around the brain and spinal cord); epiglottitis (severe swelling of the epiglottis at the back 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.

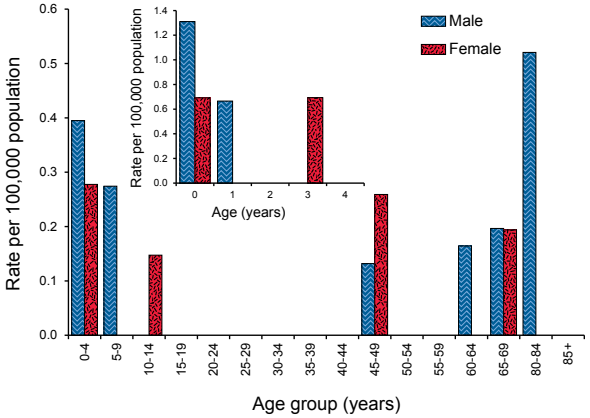
Figure 39: Notifications and notification rates for invasive *Haemophilus influenzae* type b infection, Australia, 1992 to 2012, by year of diagnosis



Age and sex distribution

In 2012, the male to female ratio was 1.5:1. One-third 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

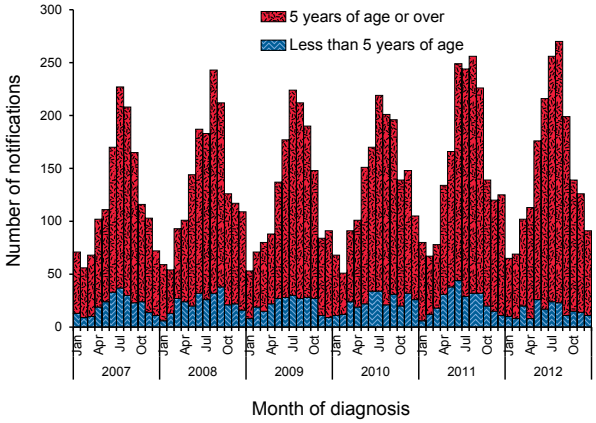
There were 1,822 notifications of IPD reported in 2012, representing a rate of 8.0 per 100,000 Compared with 2011, the national number of IPD notifications in 2012 decreased by 3.3% but was the 2nd highest reported in any year since the introduction of the universal pneumococcal conjugate vaccine program 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).

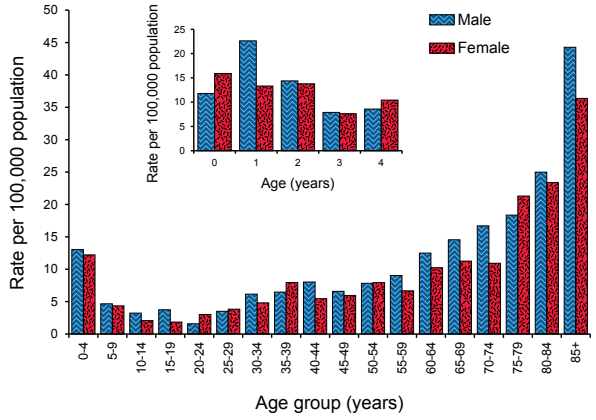
Figure 41: Notifications of invasive pneumococcal disease, Australia, 2012, by month of diagnosis and age group



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-

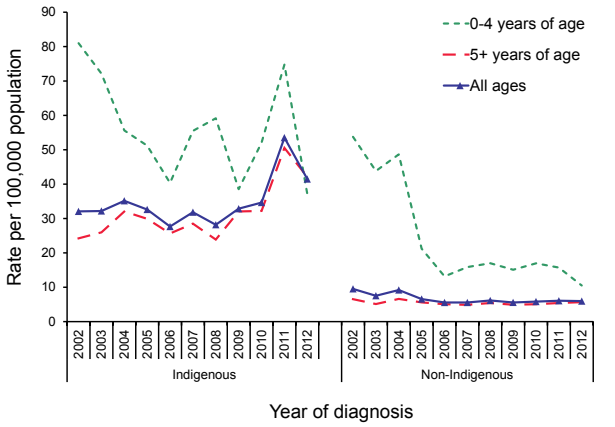
lation (41 per 100,000) was almost 7 times that for non-Indigenous people (6 per 100,000). In 2012, the notification rate for IPD among Indigenous children aged less than 5 years (37.1 cases per 100,000) remained almost 3-fold that of the general population (10.5 per 100,000) (Figure 43).

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 – Introduction of universal childhood 7vPCV 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

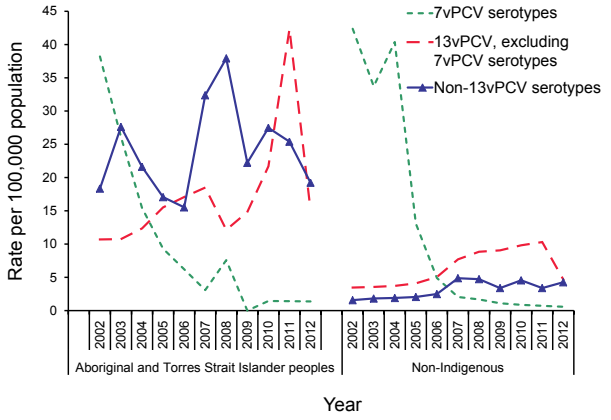
Vaccine type	National Immunisation Program pneumococcal vaccination schedule
7-valent pneumococcal conjugate vaccine (7vPCV)	From 2005 to July 2011, 7vPCV was funded nationally for all infants as a 3-dose primary vaccination schedule consisting of doses at 2, 4 and 6 months of age without a booster in 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 conjugate vaccine (13vPCV)	From July 2011, the 13vPCV replaced the 7vPCV for all infants. 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 Immunise Australia web site (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.

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]

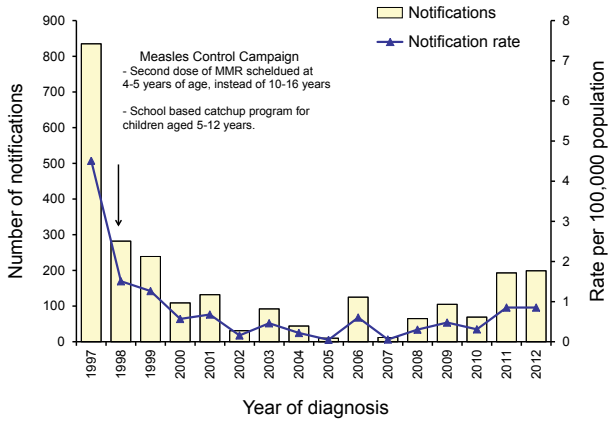
In 2012, measles vaccine was available in the combined measles-mumps-rubella (MMR) vaccine and provided under the NIP schedule to children at 12 months and 4 years of age. Two doses of a measles containing vaccine are recommended for all non-immune persons born during or since 1966 and who are 18 months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

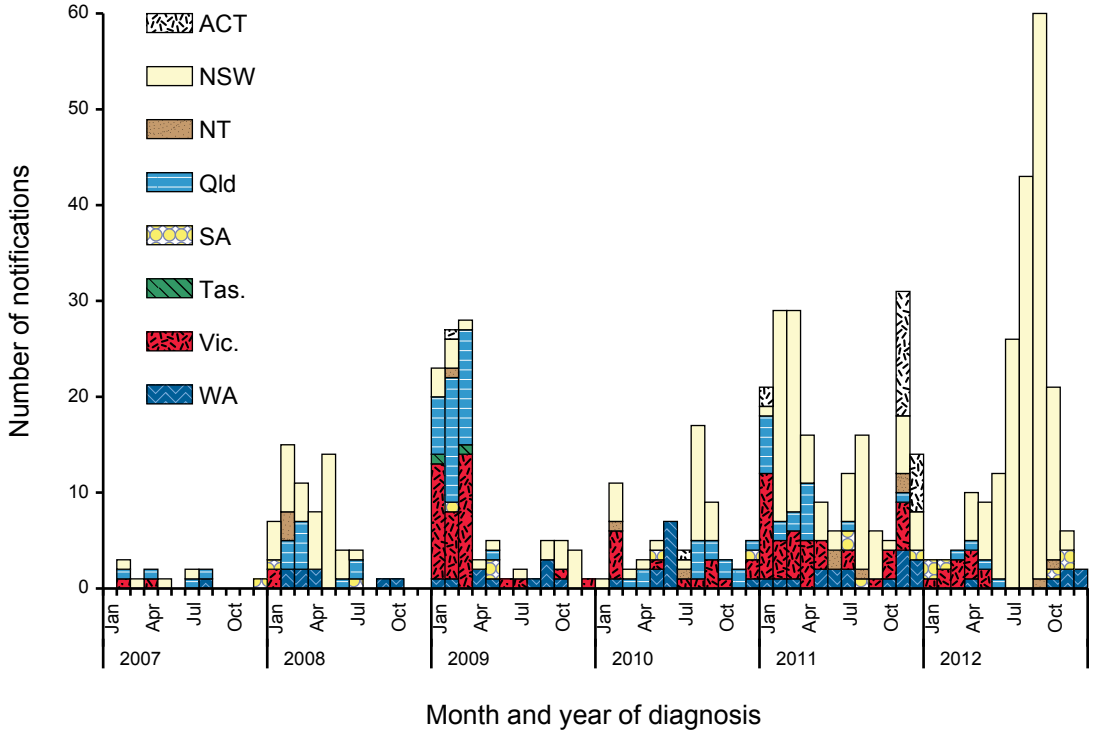
In Australia, a seasonal pattern is no longer evident as the virus is not endemic (Figure 46). In temperate climates where measles transmission remains endemic, the majority of cases occur in late winter to early spring.[19]

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



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

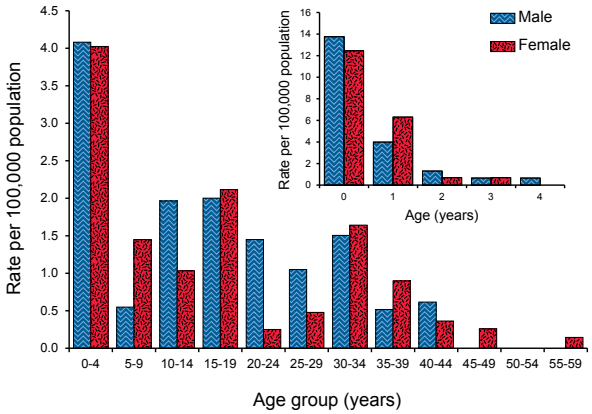
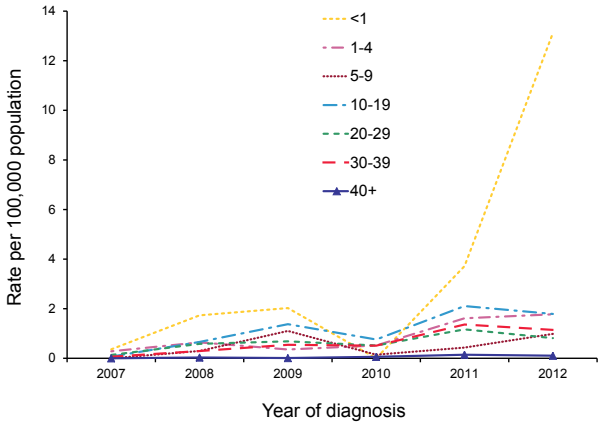


Figure 48: Notification rate for measles, Australia, 2007 to 2012, by age group



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]

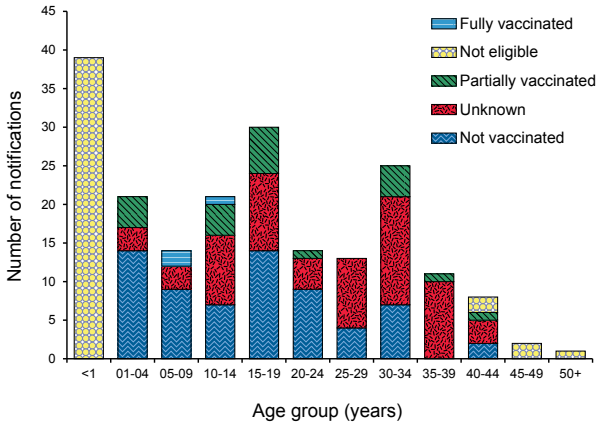
Indigenous status

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).

Figure 49: Notifications of measles, Australia, 2012, by age group and vaccination status

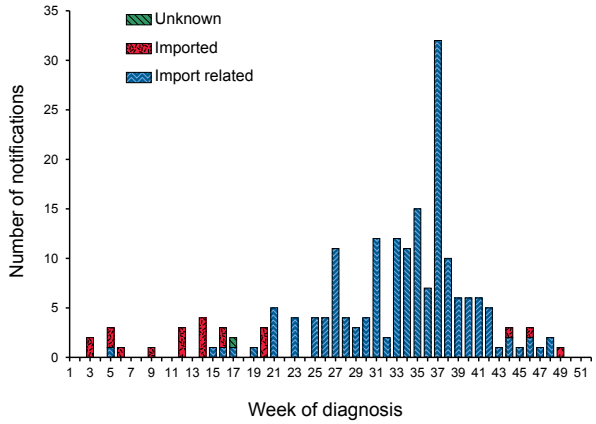


A high proportion of unvaccinated cases (48%, n=21) occurred in adolescents in the 10–19 years age group or young adults 20–29 years of age. Twenty-five cases occurred in those born between 1978 and 1982 (30–34 years age group) of which three were reported as receiving 1 dose of vaccine and the remainder 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.

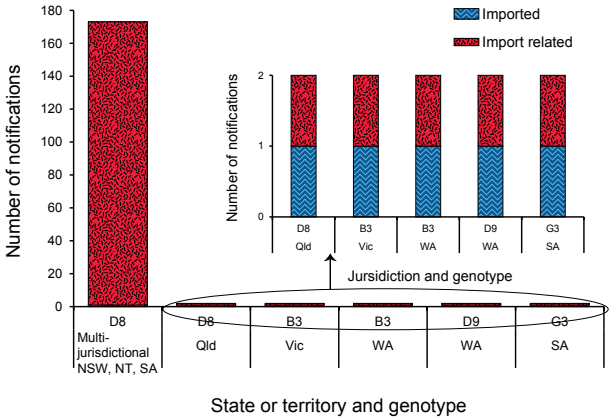
There were 6 clusters of two or more epidemiologically linked cases in 2012, all of which were import-related. In all except 1 cluster, transmission was interrupted quickly resulting in only 2 cases for each of these clusters. The largest outbreak of measles occurred predominately in Western and South 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.

Figure 51: Measles clusters, Australia, 2012, by state or territory, genotype and source of infection



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 excep-

tion 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

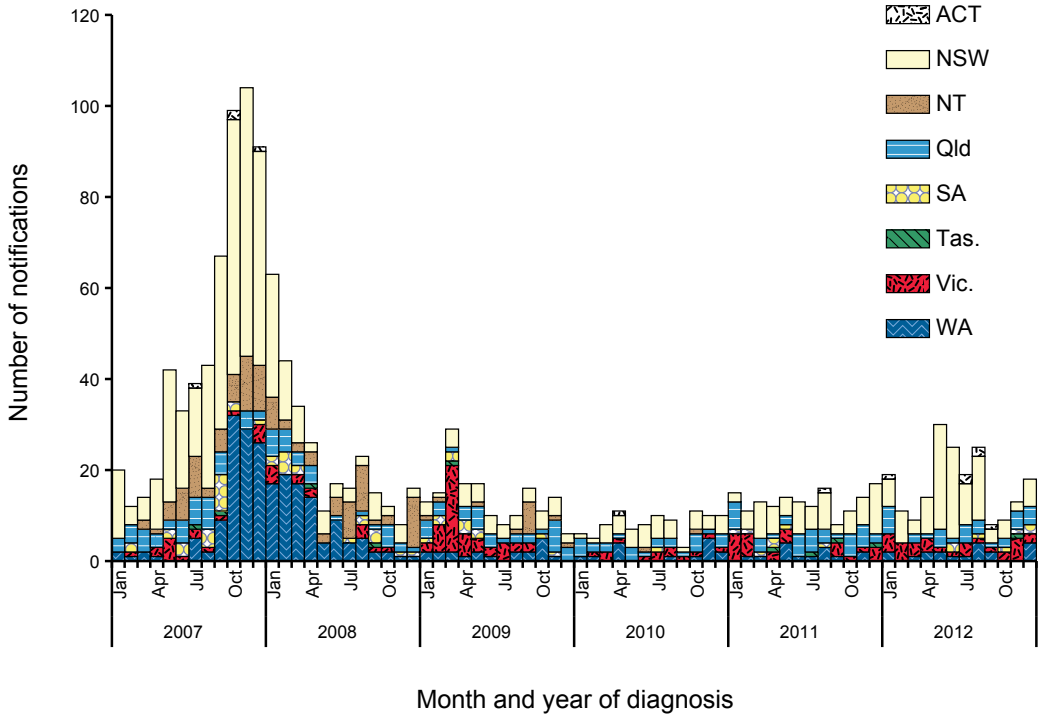
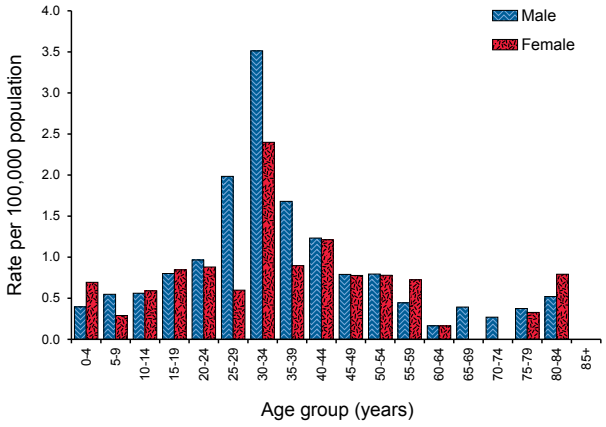
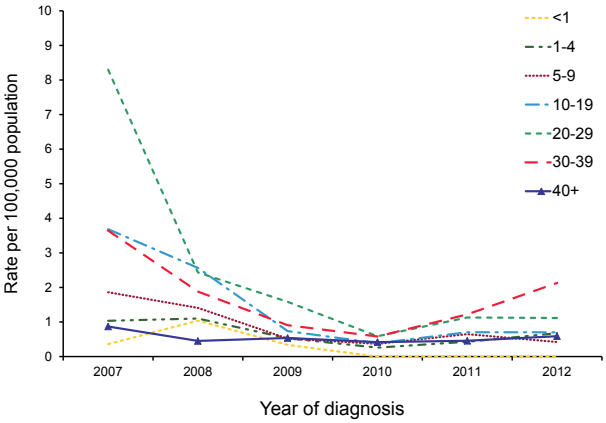


Figure 53: Notification rate for mumps, Australia, 2012, by age group and sex



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

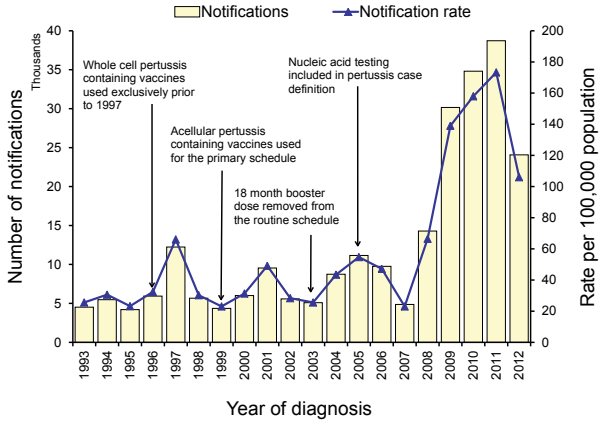


Figure 57: Notification rates for pertussis, 2007 to 2012, by state or territory

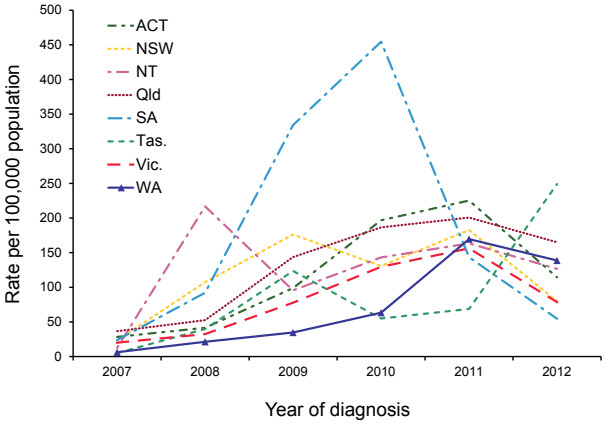
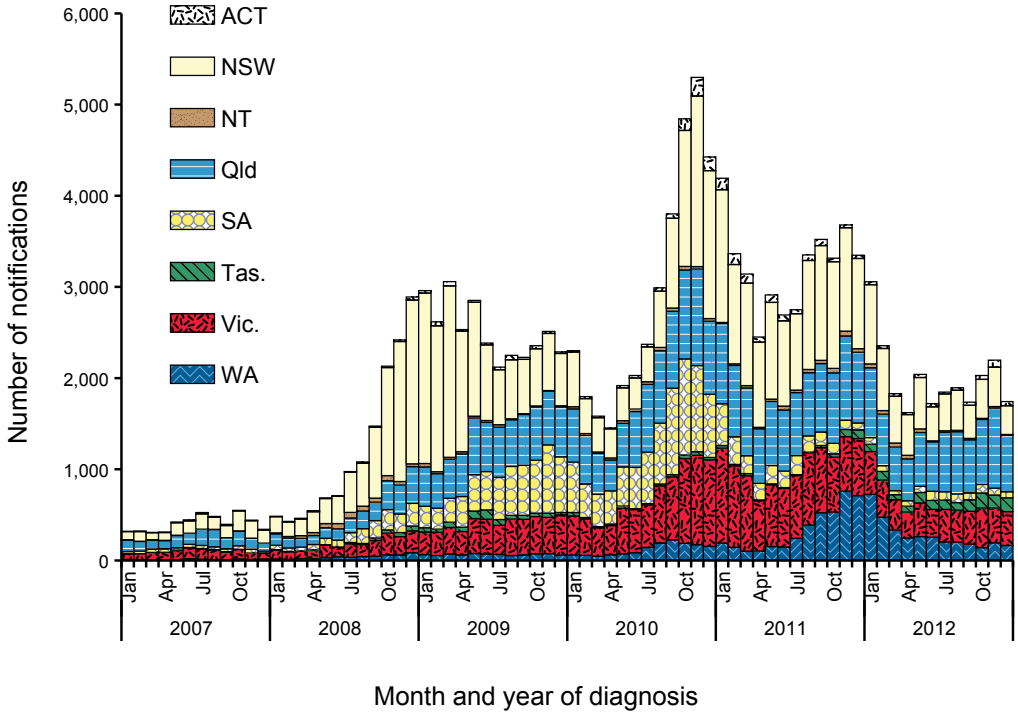


Figure 56: Notifications of pertussis, Australia, 2007 to 2012, by month and year of diagnosis and and state or territory

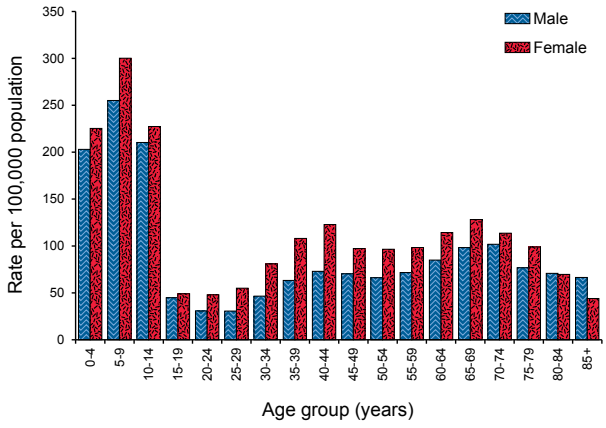


breaks, varying by geographical location, size and timing across jurisdictions, were the main cause of the varying rates for this period.

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, Australia, 2007 to 2012, by age group and sex

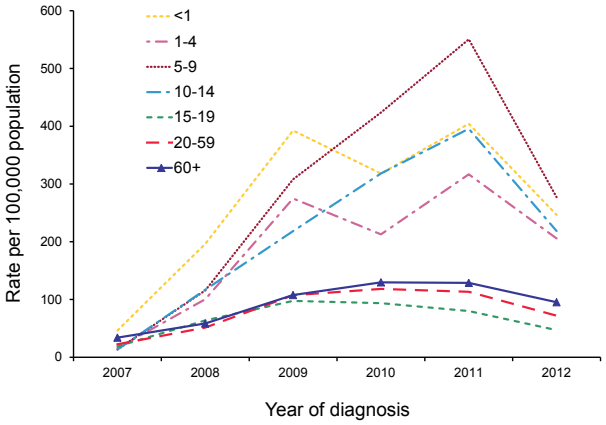


In 2012, females accounted for 56% (n=13,497) of cases, resulting in a male to female ratio of 1.2:1. Females had higher rates across all age groups, except adults aged 80 years or over (Figure 59). The highest notification rate for both males and females occurred in the 5–9 years age group (255 and 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]

Figure 59: Notification rate for pertussis, Australia, 2012, by year and age group



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,

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

Age group	Number of vaccine doses					Unknown	Total
	0	1	2	3	4		
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

* 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

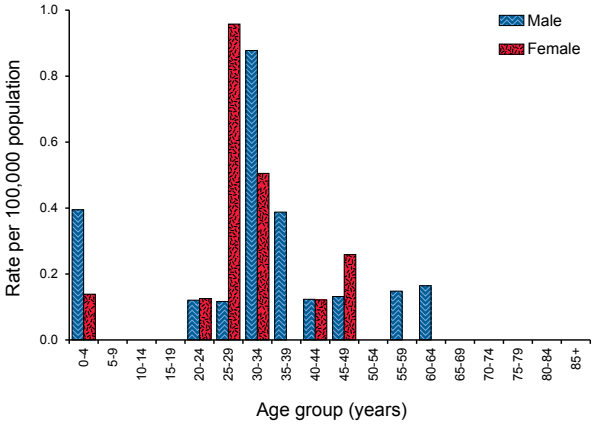


Figure 60: Notifications of rubella, Australia, 2007 to 2012, by month of diagnosis

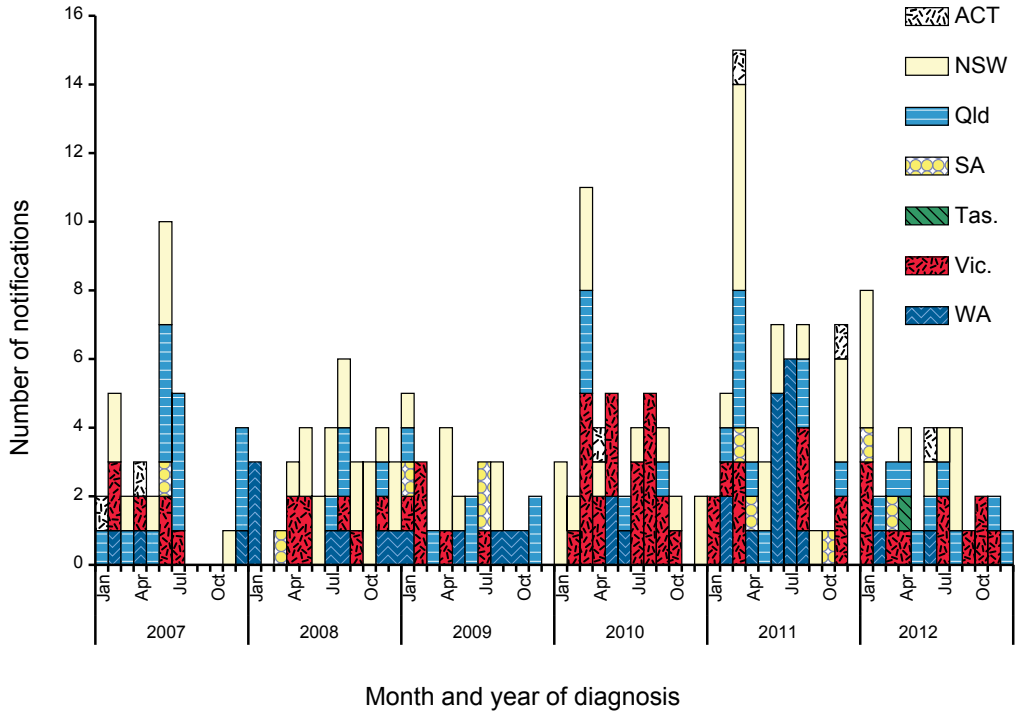
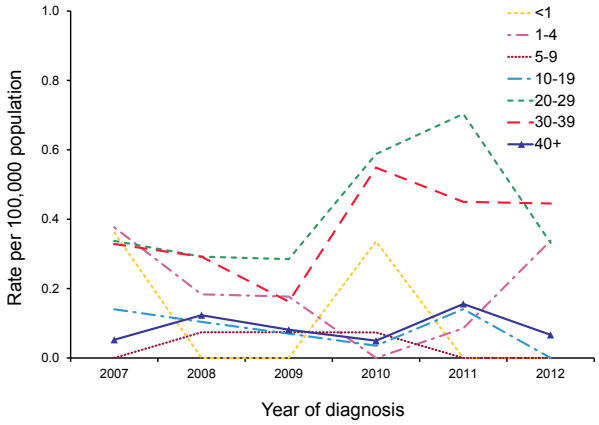


Figure 62: Notification rate for rubella, Australia, 2007 to 2012, by age group



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.

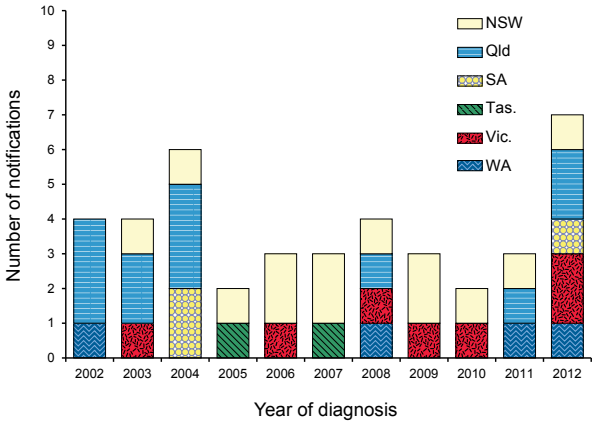
Tetanus is an acute, often fatal, disease caused by the toxin produced by the bacterium *Clostridium tetani*. Tetanus spores usually enter the body through contamination of a wound with manured soil.[19] The neurotoxin acts on the central nervous system to cause muscle rigidity with painful spasms. Generalised 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.

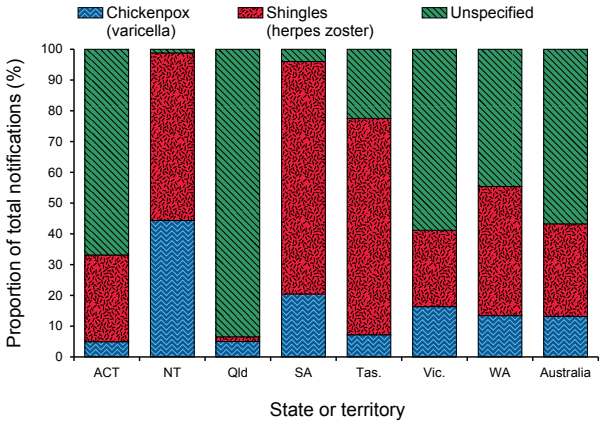
The ability to categorise a VZV infection as chickenpox or shingles depends largely on clinical evidence. Due to the absence of information on clinical presentation for many cases, the majority of VZV notifications nationally are reported as being unspecified.

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.

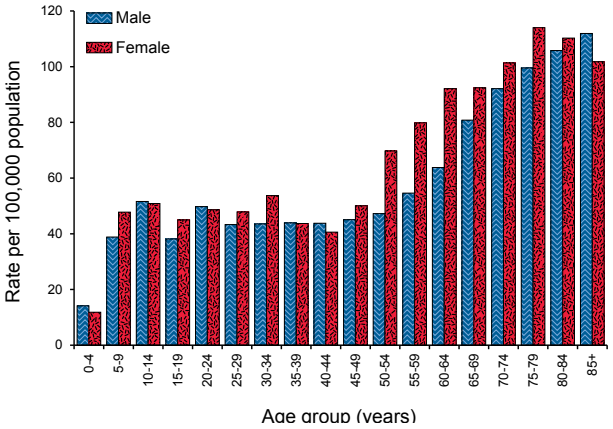
Epidemiological situation in 2012

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 self-limiting 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

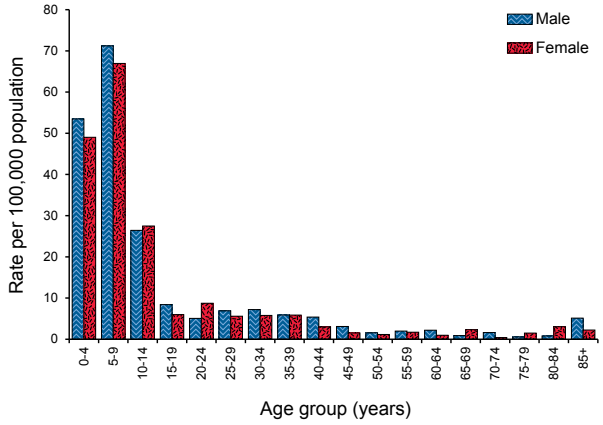
In 2012, there were 4,481 cases of shingles notified; a notification rate of 29 per 100,000 and similar to 2011. The highest rates of shingles occurred in South Australia, (106 per 100,000, n=1,761) followed by the Northern Territory, (78 per 100,000, n=183). The high rates in these jurisdictions likely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

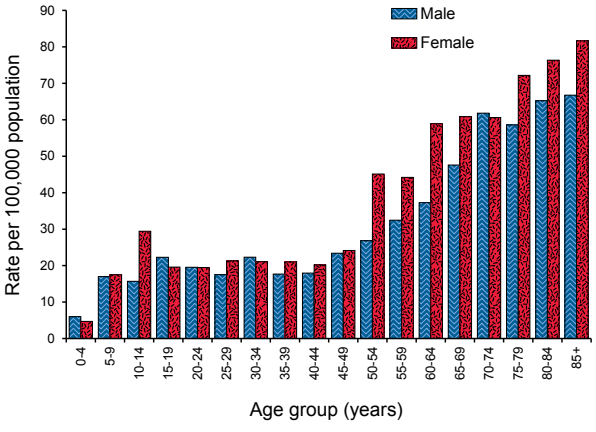
Geographical distribution

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

Figure 67: Notification rate for varicella zoster virus (unspecified), Australia,* 2012, by age group and sex



* 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

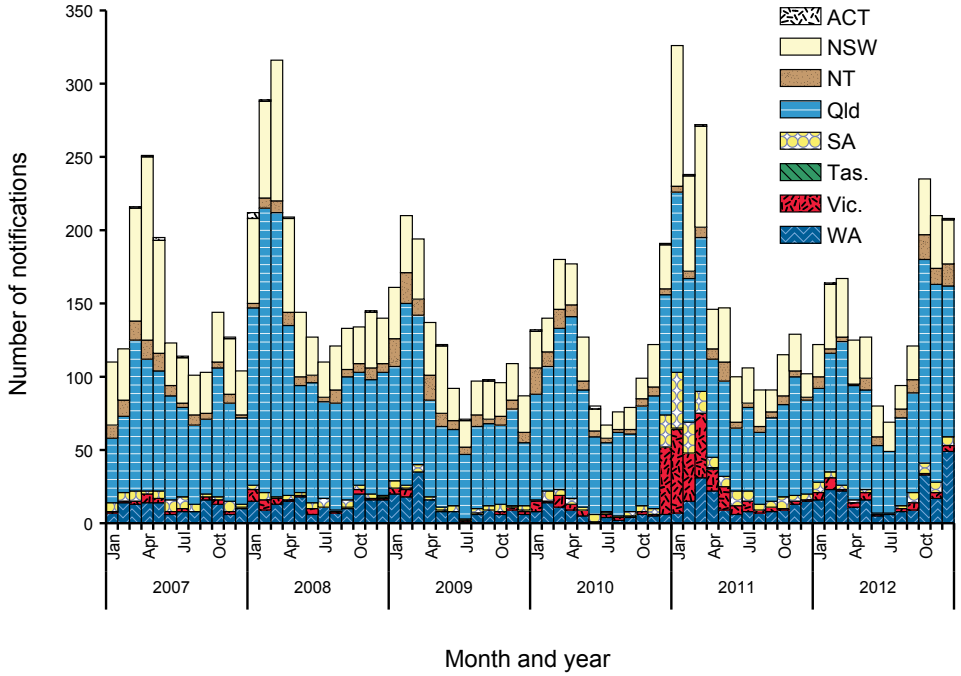
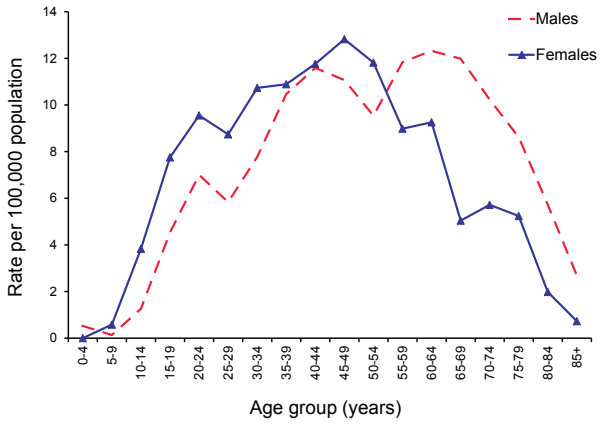


Figure 69: Notification rates for Barmah Forest virus infection, Australia, 2012, by age group and sex



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

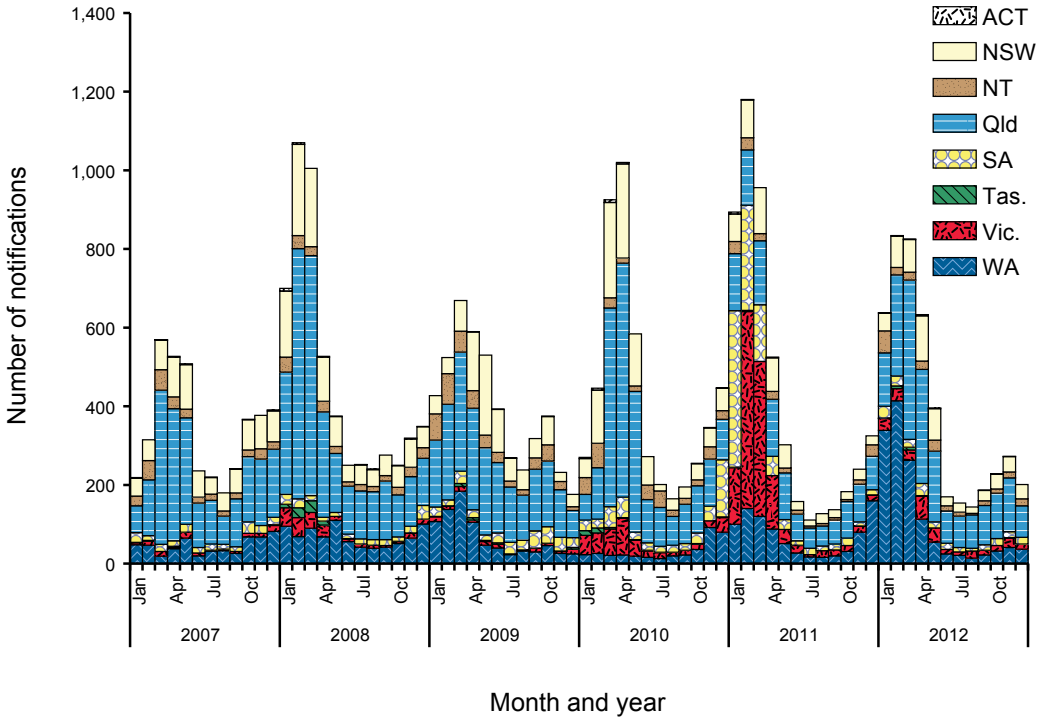
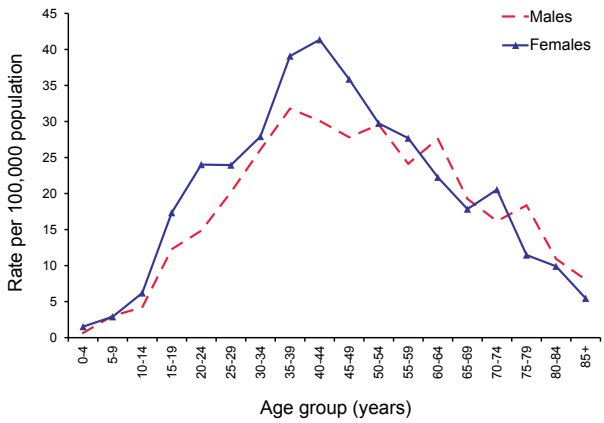


Figure 71: Notification rates for Ross River virus infection, Australia, 2012, by age group and sex



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.

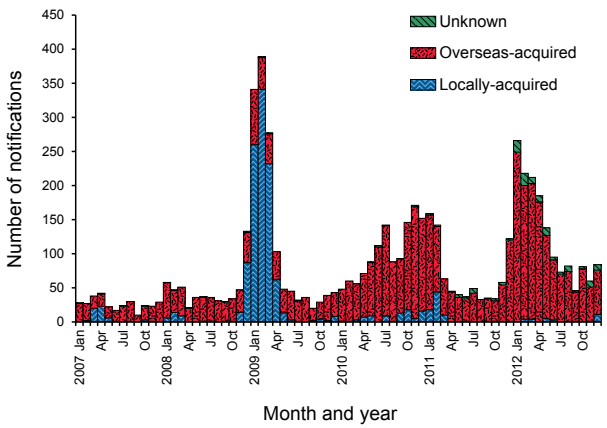
Serotype of dengue virus infections

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

Place of acquisition	Serotype					Untyped/ unknown	Total
	DENV 1	DENV 1 and 4	DENV 2	DENV 3	DENV 4		
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.

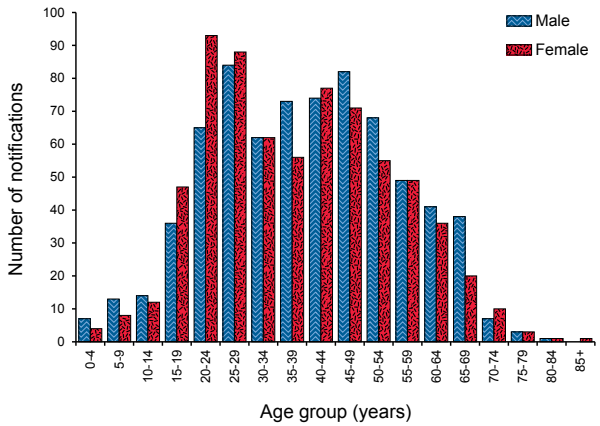
All but one of the 29 locally-acquired DENV in 2012 were known to have been associated with one of the 5 outbreaks of locally-acquired infection in Queensland in 2012 that were notified to NNDSS. The largest number of notified cases during the year was during an outbreak of DENV 1 and 2 in Townsville 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

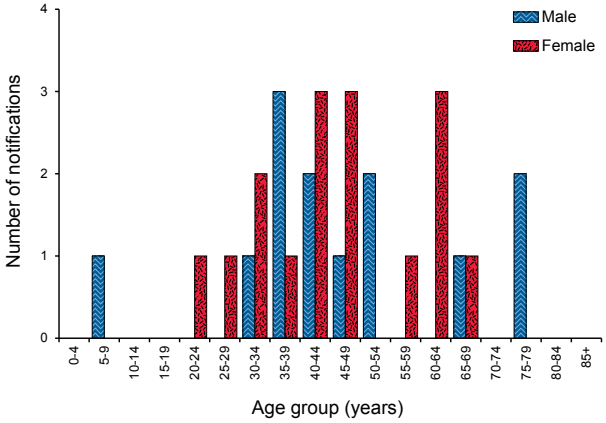
DENV infections acquired overseas in 2012 were most commonly reported among younger and middle aged adults (median 39 years, range 2–85 years), with a peak of notifications among males aged 20–29 years and females aged 25–29 years (Figure 73). Males comprised 51% of cases with overseas-acquired DENV. For locally-acquired DENV, infections were more commonly reported among middle aged and older adults (median 44 years, range 5–76 years), with peak notifications among males and females aged 40–44 years (Figure 74). Males comprised 45% of cases with locally-acquired DENV.

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



* Sex was not available for 2 cases.

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

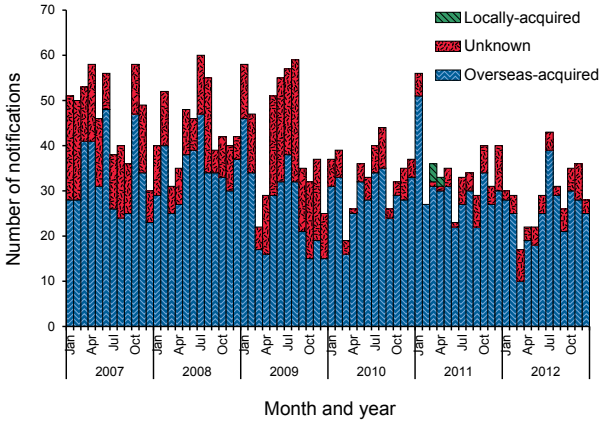
- Notifications continued the gradual decline observed since 2005.
- No cases were known to have been acquired in Australia in 2012.

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 locally-acquired 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

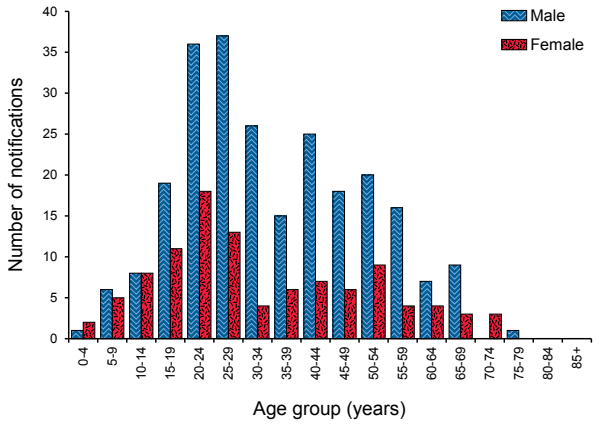
Country and region	*P. falciparum*	*P. vivax*	*P. ovale*	*P. malariae*	Mixed species infection	*Plasmodium* species	Total
Oceania							
Papua New Guinea	20	25	0	0	0	1	46
Solomon Islands	0	7	0	0	0	0	7
South East Asia							
Indonesia	8	8	0	0	0	1	17
Cambodia	2	4	1	0	0	0	7
Philippines	1	2	0	0	0	0	3
Laos	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 East							
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 and region not stated/unknown							
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 north-eastern 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.

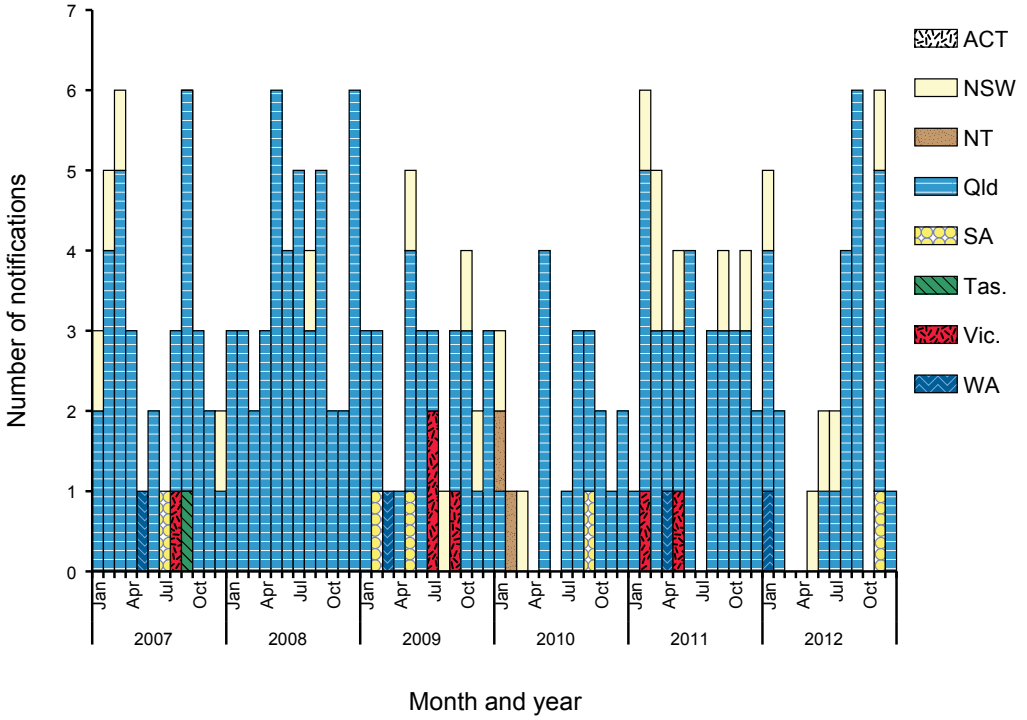
Brucella species that can cause illness in humans include *Brucella melitensis* acquired from sheep and goats, *B. suis* from pigs and *B. abortus* from cattle. *B. abortus* was eradicated from Australian cattle herds in 1989 and *B. melitensis* has never been reported in Australian sheep or goats.[124] 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]

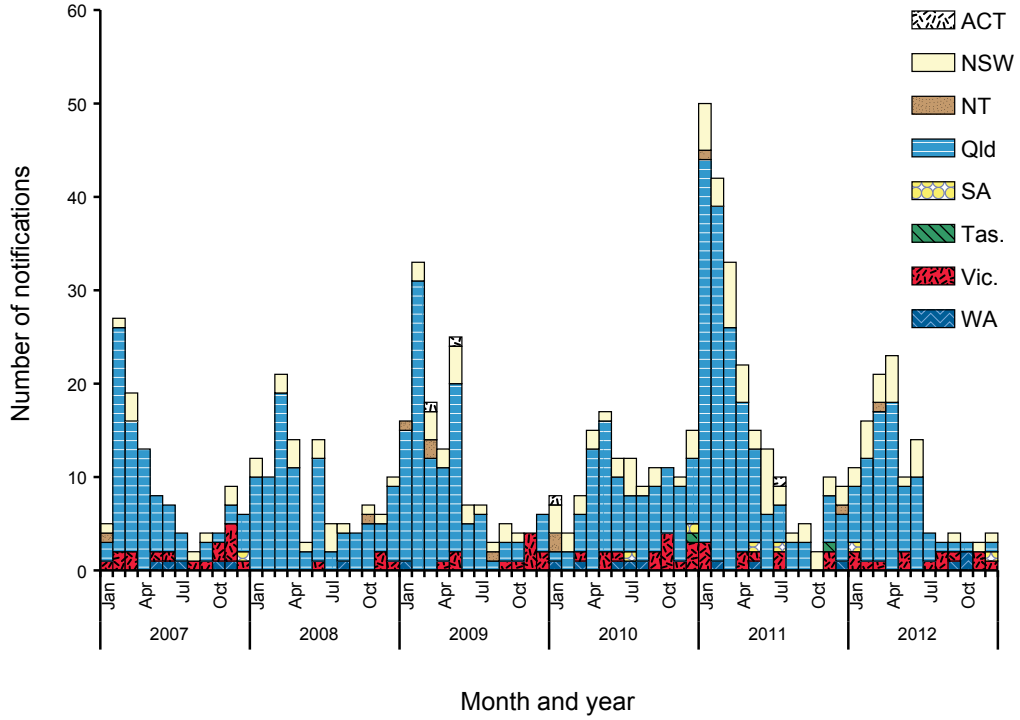
Epidemiological situation in 2012

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



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.

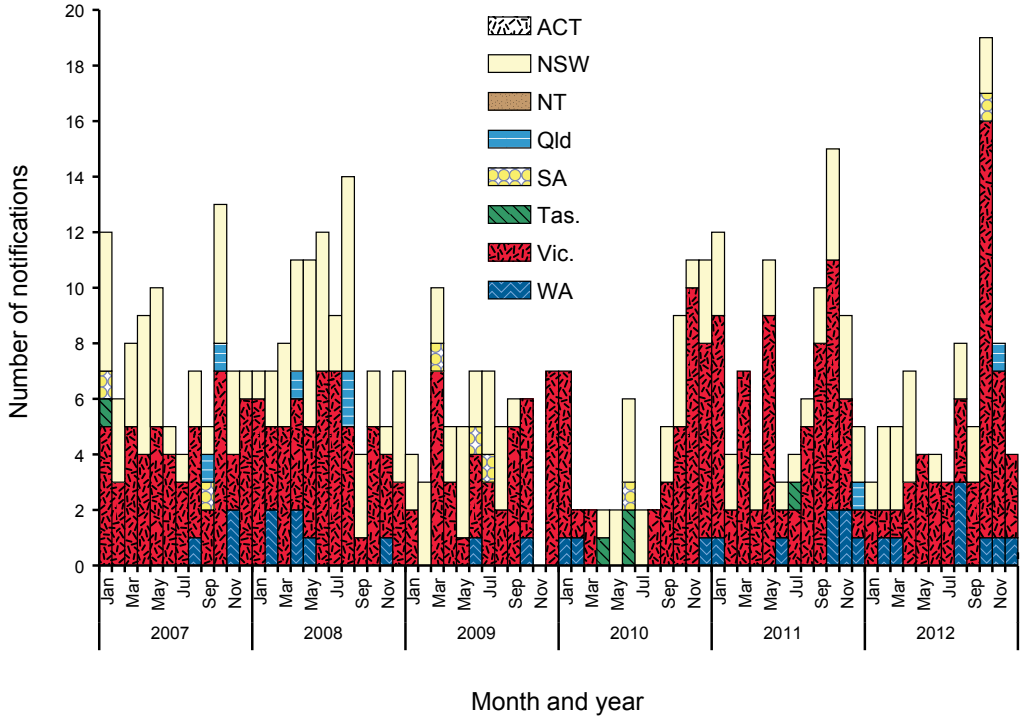
Ornithosis (or psittacosis) is caused by infection with the bacterium *Chlamydophila psittaci*. It is transmitted to humans primarily from infected parrots of many species, but also poultry and a range of other birds.[144] Transmission to humans can occur via the inhalation of contaminated dried faeces, 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



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]

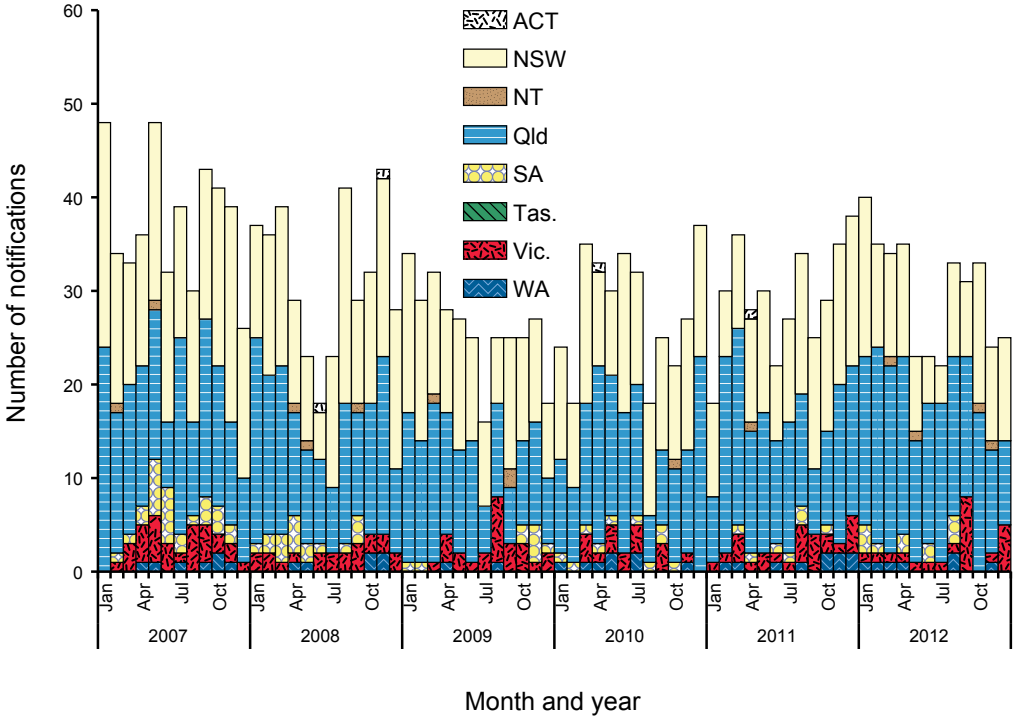
Adults at risk of Q fever infection, including abattoir workers, farmers, veterinarians, stockyard workers, shearers and animal transporters should be considered for vaccination. The administration of the Q fever vaccine requires a pre-vaccination screening test to exclude those recipients with a previous (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



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

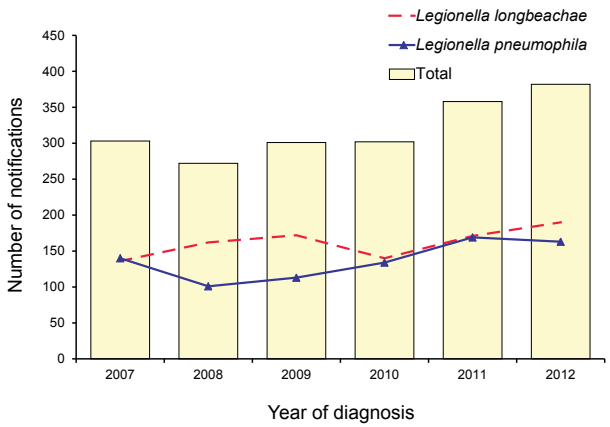
- 382 cases of legionellosis were notified in 2012.
- Since 1991, the number of legionellosis notifications has continued to rise.
- *Legionella longbeachae,* traditionally associated with potting mix, was more frequently reported as the causative species in 2012.

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

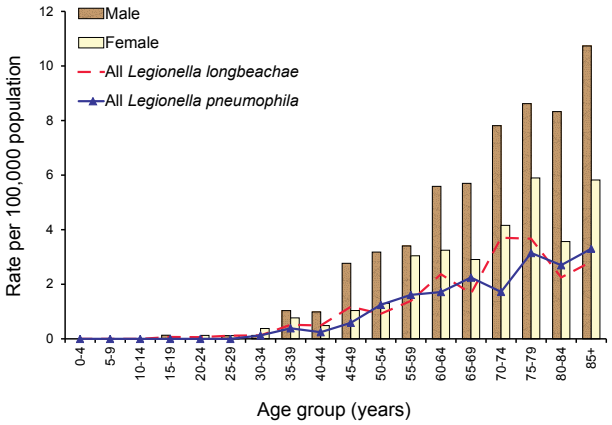


Figure 83: Notifications of legionellosis, Australia, 2007 to 2012, by month and year of diagnosis and species

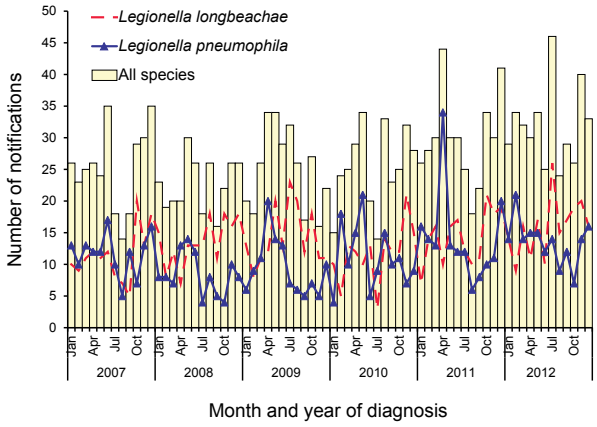


Table 24: Notifications of legionellosis, Australia, 2011, by species and state or territory

Species	State or territory									Deaths due to legionellosis
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.	
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]

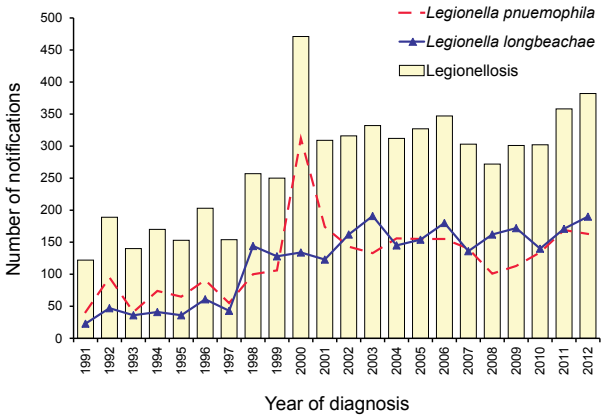
The Queensland cluster consisted of 2 cases diagnosed in January and February of 2012. The cases were identified in residents of a retirement village in South East Queensland. An environmental investigation of the facility was undertaken with water samples collected from the spa, pool and resident showers. 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.

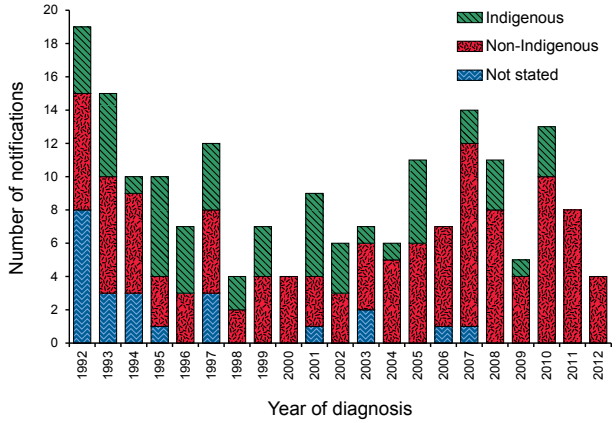
Epidemiological situation in 2012

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.

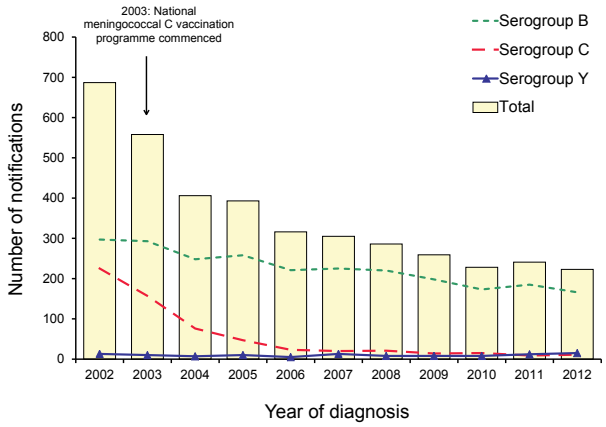
Epidemiological situation in 2012

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

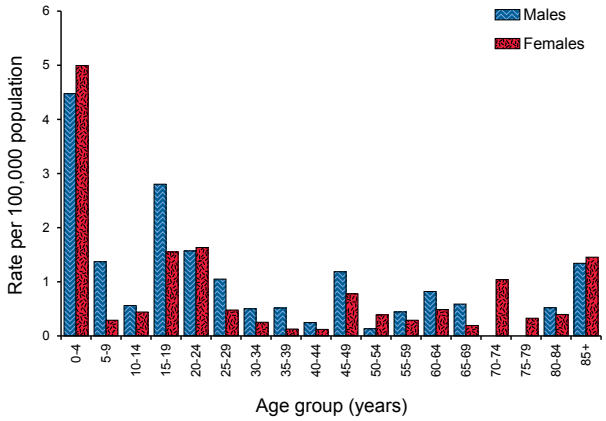
More males than females were reported with IMD in 2012, with a male to female ratio of 1:0.8. Two-thirds of cases (n=147) were less than 25 years of age, of which those less than 5 years of age made up almost half (n=70). Cases aged less than 5 years had the highest age-specific rate at 4.7 cases per 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									
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.	Deaths
B	1	43	3	45	26	4	29	15	166	10
C	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	–

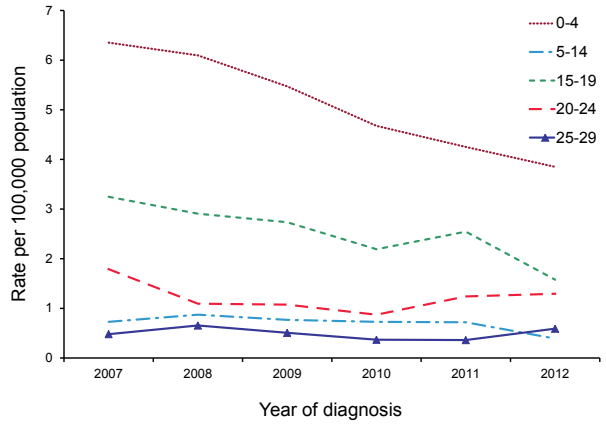
15–19 years age group (2.2 per 100,000) followed by the 20–24 years age group (1.6 per 100,000) (Figure 87).

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



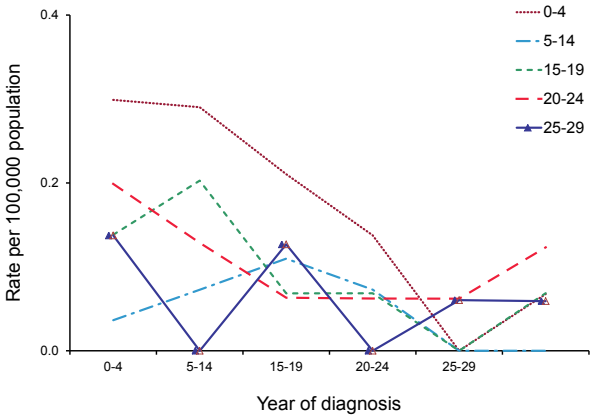
Serogroup B accounted for the majority of cases across all age groups including those aged less than 25 years, where it accounted for 85% of cases with serogroup information available. While the age-specific rates of serogroup B infection in 2012 remain high compared with other serogroups, they continue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

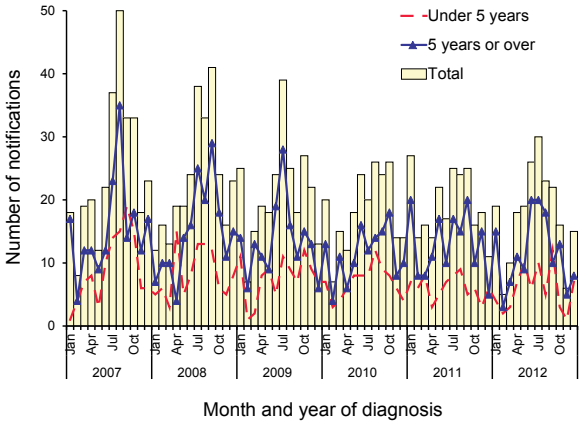
Figure 89: Notification rate for serogroup C invasive meningococcal disease, Australia, 2007 to 2012, by selected age group



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.

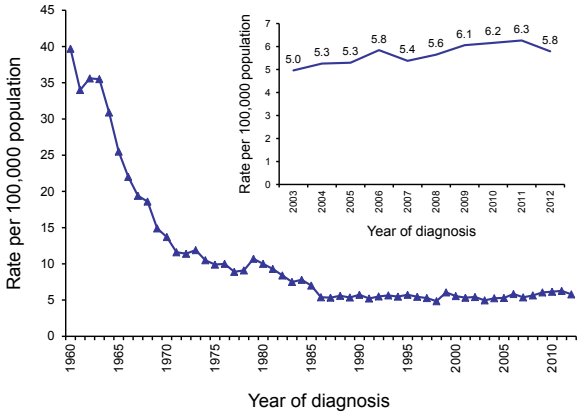
TB is an infection predominantly caused by the bacterium *Mycobacterium tuberculosis*. It is transmitted by airborne droplets produced by people with pulmonary TB during coughing or sneezing. About one-third of the world's population has latent TB infection, which means people have been infected with 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



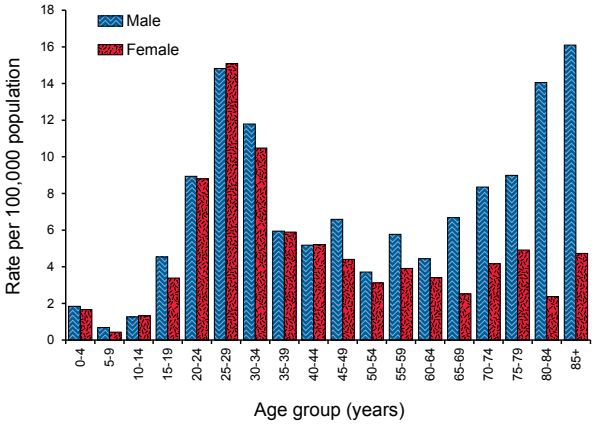
Geographical distribution

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 group	State or territory								
	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

Appendix 3: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2012, by notifiable disease*

Disease name	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
Arbovirus infection (NEC)	0	0	0	4	5	0	9	44	4	5
Barmah Forest virus infection	27	3	3	579	811	299	1,722	36	612	1,110
Brucellosis	1	0	0	19	9	0	29	69	20	9
Campylobacteriosis	143	9	11	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	1	0	0	1	100	1	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	1	20	95	19	1
Haemophilus influenzae type b	2	0	0	13	0	0	15	100	15	0
Hepatitis A	0	0	0	148	16	1	165	90	148	17
Hepatitis B (newly acquired)	17	0	2	146	25	3	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	1	321	50	11	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	2	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	1	0	0	1	100	1	0
Legionellosis	6	0	2	327	42	5	382	88	335	47
Leprosy	0	0	0	4	0	0	4	100	4	0
Leptospirosis	2	0	0	98	14	2	116	86	100	16
Listeriosis	1	1	0	87	3	1	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	3	0	187	11	0	223	95	212	11
Mumps	1	0	0	119	49	31	200	60	120	80

Appendix 3 *continued*: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2012, by notifiable disease*

Disease name	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
Murray Valley encephalitis virus infection	0	0	0	1	0	0	1	100	1	0
Ornithosis	1	0	0	39	34	1	75	53	40	35
Pertussis	512	20	17	11,086	9,674	2,760	24,069	48	11,635	12,434
Pneumococcal disease (invasive)	230	5	9	1,320	143	115	1,822	86	1,564	258
Q fever	12	1	0	263	74	8	358	77	276	82
Ross River virus infection	78	5	1	2,230	1,965	404	4,683	49	2,314	2,369
Rubella	1	0	0	25	10	0	36	72	26	10
Rubella – congenital	0	0	0	1	0	0	1	100	1	0
Salmonellosis	350	12	9	5,849	2,824	2,221	11,265	55	6,220	5,045
Shigellosis	147	0	0	332	40	28	547	88	479	68
STEC, VTEC*	3	0	0	91	16	1	111	85	94	17
Syphilis < 2 years	153	1	4	1,226	146	9	1,539	90	1,384	155
Syphilis > 2 years or unspecified duration	162	16	6	842	322	6	1,354	76	1,026	328
Tetanus	0	0	0	6	1	0	7	86	6	1
Tuberculosis	28	3	0	1,276	6	2	1,315	99	1,307	8
Typhoid fever	0	0	0	113	6	4	123	92	113	10
Varicella zoster (chickenpox)	121	3	5	1,654	181	0	1,964	91	1,783	181
Varicella zoster (shingles)	115	3	6	3,823	534	0	4,481	88	3,947	534
Varicella zoster (unspecified)	121	18	13	2,142	6,159	0	8,453	27	2,294	6,159
Total	13,855	1,101	662	109,918	79,971	38,366	243,872	51	125,536	118,337

* 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

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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	*Haemophilus influenzae* 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 *Escherichia coli*
STI(s)	sexually transmissible infections(s)
TB	tuberculosis
TSI	Torres Strait Islander
VPD(s)	vaccine preventable disease(s)
VTEC	verotoxigenic *Escherichia coli*
VZV	varicella zoster virus
WHO	World Health Organization
WHOCC	World Health Organization Collaborating Center

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Australian Gonococcal Surveillance Programme annual report, 2013

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Abstract

The Australian Gonococcal Surveillance Programme has continuously monitored antimicrobial resistance in clinical isolates of *Neisseria gonorrhoeae* from all states and territories since 1981. In 2013, 4,897 clinical isolates of gonococci from public and private sector sources were tested for *in vitro* antimicrobial susceptibility by standardised methods. Decreased susceptibility to ceftriaxone (MIC value 0.06–0.125 mg/L) was found nationally in 8.8% of isolates, double that reported in 2012 (4.4%). The highest proportions were reported from New South Wales and Victoria (both states reporting 11.8%), with a high proportion of strains also reported from Tasmania but a low number of isolates were tested. In addition, there was a multi-drug-resistant strain of *N. gonorrhoeae* isolated from a traveller to Australia, with a ceftraixone MIC value of 0.5 mgL—the highest ever reported in Australia. These antimicrobial resistance data from Australia in 2013 are cause for considerable concern. With the exception of remote Northern Territory where penicillin resistance rates remain low (1.3%) the proportion of strains resistant to penicillin remained high in all jurisdictions ranging from 15.6% in the Australian Capital Territory to 44.1% in Victoria. Quinolone resistance ranged from 16% in the Australian Capital Territory to 46% in Victoria. Azithromycin susceptibility testing was performed in all jurisdictions and resistance ranged from 0.3% in the Northern Territory to 5.7% in Queensland. High level resistance to azithromycin (MIC value was > 256 mg/L) was reported for the first time in Australia, in 4 strains: 2 each from Queensland and Victoria. Azithromycin resistant gonococci were not detected in the Australian Capital Territory, Tasmania or from the remote Northern Territory. Nationally, all isolates remained susceptible to spectinomycin. *Commun Dis Intell* 2015;39(1):E137–E145.

Keywords: antimicrobial resistance; disease surveillance; gonococcal infection; *Neisseria gonorrhoeae*

Introduction

Gonococcal disease rates have markedly increased in Australia in recent years.[1] Whilst this may be in part due to increased use of nucleic acid amplification testing (NAAT), this situation is coupled with significant concerns regarding gonococcal antimicrobial resistance.[2] The current linchpin of treatment, ceftriaxone, has no ideal replacement.[3] Over recent years increasing proportions of gonococcal isolates with raised ceftriaxone MIC values strains have been reported in Australia,[3] and this reflects the situation globally. Strategies for gonococcal disease treatment; prevention and control are placed high on the agenda of the World Health Organization (WHO)[4] and the Centers for Disease Control and Prevention (CDC) in the United States of America.[5]

Over the period 2007–2012 there was a 65% increase in gonococcal disease notifications in Australia,[1] with the disease rate rising from 35.4 to 58.4 per 100,000 population. The greatest increases in notifications were observed in both males and females in the eastern states (Victoria, New South Wales and Queensland), and males in the Australian Capital Territory. The highest notification rates remain in Indigenous people in the Northern Territory and Western Australia. The overall Australian age standardised gonorrhoea notification rates in 2012 for Indigenous compared with non-Indigenous Australians were 933.4 per 100,000 population and 38.5 per 100,000 population, respectively.[1]

Gonococcal antimicrobial surveillance programs both in Australia and internationally, have reported over time, the emergence and spread of antimicrobial resistance in gonorrhoea.[3,6] In recent years, ceftriaxone treatment failure has occurred in Australia and elsewhere, in strains with raised MIC values (decreased susceptibility).[2] In 2010, the 1st ceftriaxone-resistant strain (H041) (MIC = 2.0 mg/L), was found in Japan,[7] with no evidence of spread. However, in 2011, a 2nd ceftriaxone-resistant *Neisseria gonorrhoeae* strain (F89), was initially observed in France and subsequently identified in Spain, with a MIC value 2.0 mg/L.[8,9]

In response to increasing MIC values to the extended spectrum cephalosporin antibiotics, the United Kingdom, the United States of America, and European gonococcal treatment guidelines moved to recommend a dual therapy strategy comprising ceftriaxone plus azithromycin, in a bid to stem the development of *N. gonorrhoeae* antimicrobial resistance (AMR).[10–12]

Of the WHO estimated 106 million new *N. gonorrhoeae* infections reported in those aged 15–49 years

annually worldwide[13] almost two-thirds (67.4 million, 63%) occur in the Asia–Pacific region.[13] The WHO gonococcal antimicrobial surveillance data from the Asia–Pacific region indicate that there are high levels of gonococcal AMR in the region, which is densely populated and has a disproportionate burden of gonococcal disease. In many countries there is uncontrolled antimicrobial use providing ideal conditions for the development of AMR.[14] This is of continuing concern to Australia where, in urban centres, AMR in *N. gonorrhoeae* has long been influenced by the introduction of multi-resistant strains from overseas.[15]

In contrast, the highest reported rate of gonococcal infection occurs amongst the Indigenous populations in some remote regions of Australia, where there are low rates of AMR. In these remote regions, gonococcal infection acquired locally or in an endemic region, can still be effectively treated with oral antibiotics (amoxycillin 3 g, probenecid 1 g and azithromycin 1 g).[6]

Strategies for treating and controlling gonorrhoea are based on regimens effecting cure in a minimum of 95% of cases. The formulation of these regimens is reliant on data derived from continuous monitoring of resistance to the antibiotics in clinical use.[16] The current global situation has raised concerns for gonococcal disease treatment, prevention and control.[17] The WHO has called for enhanced surveillance as a fundamental component of the Global Action Plan to control the spread and impact of gonococcal AMR.[4]

In Australia, the National Neisseria Network (NNN) is a collaboration of reference laboratories in each state and territory that monitor clinical isolates of pathogenic *Neisseria* species nationally from public and private sector laboratories, representing as wide a section of the community as possible, for phenotypic and genotypic characteristics, including antimicrobial resistance. The Australian Gonococcal Surveillance Programme (AGSP) is a key activity of the NNN and has continuously monitored the susceptibility of *N. gonorrhoeae* since 1981, making it the longest, continually running, national surveillance system for gonococcal AMR.[18]

Methods

The NNN AMR data for gonococcal isolates are collated for the AGSP quarterly and annual reports. Gonococcal infection is a notifiable disease in Australia and each case is notified to the National Notifiable Diseases Surveillance System (NNDSS). The number of isolates tested by the NNN and reported by the AGSP represents a proportion of the number of cases reported to the NNDSS. The increasing use of non-culture based methods of diagnosis has reduced the number of isolates available for AMR testing, however, the NNN still tests approximately one third of the number of notified cases in Australia.

The NNN laboratories test gonococcal isolates for antibiotic susceptibility to penicillin (representing this group of antibiotics); ceftriaxone (representing later generation cephalosporin antibiotics); ciprofloxacin (representing quinolone antibiotics); azithromycin; spectinomycin; and for high level plasmid mediated resistance to tetracycline using previously described standardised methodology to determine the minimum inhibitory concentration (MIC) values.[18,19] The MIC value is the least amount of antibiotic that inhibits *in vitro* growth under defined conditions. The AGSP conducts a program-specific quality assurance program.[20]

Antibiotic susceptibility data from each jurisdiction are submitted quarterly to the coordinating laboratory (the Neisseria Reference Laboratory and WHO Collaborating Centre for Sexually Transmitted Diseases, Sydney), which collates the results for reporting. Where available, the AGSP collects data on the gender of the patient, country of acquisition, and site of isolation of gonococcal strains. Data from isolates from all jurisdictions are predominantly from urban centres, except for the Northern Territory where the data are further divided into urban versus remote.

Statistics

Statistical analysis was performed using Prism version 5.0d. Results were compared using Fisher's exact test for proportional differences.

Results

Number of isolates

There were 4,897 gonococcal isolates tested in NNN laboratories in 2013, representing 33% of the 14,933 cases of gonococcal infection notified to the NNDSS in 2013 (Table 1). This was slightly lower than the proportion tested in 2012 (35%); and a further decrease from the 40%–42% referred between 2008 and 2010.

Source of isolates

There were 4,032 isolates from men (82%) and 863 (18%) from women (Table 2). There were 2 isolates from patients of unknown gender. The proportion of gonococcal isolates from males and females tested by the AGSP has remained stable over recent years (2009–2012); ranging between 18%–20% for women and 80%–82% for men. The infected site was reported as 'other' or not speci-

fied for 94 isolates from males and 23 isolates from females (Table 2). Isolates from urine samples were regarded as genital tract isolates.

Antibiotic susceptibility patterns

As in past years the patterns of gonococcal antibiotic susceptibility differed between the various states and territories, thus the data are presented by region as well as aggregated for Australia (Table 3).

Table 1: Number of Australian Gonococcal Surveillance Programme gonococcal isolates tested as a proportion of National Notifiable Diseases Surveillance System gonorrhoea notifications, Australia, 2013, by state or territory

State or territory	Number of isolates tested	Number of cases notified*	Number of isolates tested/ number of cases notified %
ACT	44	114	39
NSW	1,555	4,237	37
NT	344	1,985	17
Qld	670	2,734	25
SA	212	808	26
Tas.	45	71	63
Vic.	1,539	3,012	51
WA	488	1,972	25
Aust.	4,897	14,933	33

Penicillin

Resistance to the penicillin group of antibiotics (penicillin, ampicillin and amoxycillin with or without clavulanic acid) in gonococci is a result of the production of a specific beta-lactamase, penicillinase, and/or by the aggregation of chromosomally-controlled resistance mechanisms. These are denoted respectively, as penicillinase-producing *N. gonorrhoeae* (PPNG); and chromosomally mediated resistant to penicillin (CMRP).[15] Chromosomal resistance is defined by an MIC to penicillin of 1 mg/L or more.[15,19]

In 2013 in Australia, 1,700/4,897 (35%) isolates were penicillin resistant; a proportional increase from 2012 (32%), and higher than that reported in 2010–2011 (25%–29%), but lower than 2008–2009 (36%–44%). In 2013, there were 978 (20%) isolates with CMRP; and 722 (15%) with PPNG. In 2012, the proportion of isolates with CMRP was 17%, and 15% were PPNG. Thus the increase in penicillin resistance nationally in 2013 was due to an increase in the proportion of isolates with CMRP.

Penicillin resistance in the Northern Territory

In 2013 there were 344 isolates tested from the Northern Territory. There were 105 from Darwin, and 239 from the remote Northern Territory comprising 205 from Alice Springs, 19 isolates from Katherine and 15 from other areas.

Of the isolates tested from the Northern Territory, 21/105 (20%) from the city of Darwin were penicil-

Table 2: Gonococcal isolates tested, Australia, 2013, by sex, site and state or territory

Sex	Site	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Male	Genital	23	790	208	376	120	24	695	276	2,512
	Rectal	12	292	2	67	22	12	383	48	838
	Pharynx	6	224	2	36	14	4	253	27	566
	DGI	0	4	7	5	0	0	0	6	22
	Other/NS	0	17	2	6	2	0	62	5	94
	Total	41	1,327	221	490	158	40	1,393	362	4,032
Female	Genital	3	169	115	166	42	4	129	117	745
	Rectal	0	2	0	3	5	0	1	2	13
	Pharynx	0	44	0	2	4	1	9	2	62
	DGI	0	5	7	5	0	0	0	3	20
	Other/NS	0	6	1	4	3	0	7	2	23
	Total	3	226	123	180	54	5	146	126	862
Unknown	Total		2							2
Total		44	1,555	344	670	212	45	1,539	488	4,897

DGI Disseminated gonococcal infection.
NS Not specified.

Table 3: Proportion of gonococcal isolates with resistance to penicillin, ciprofloxacin and azithromycin and decreased susceptibility to ceftriaxone reported, Australia, 2013, by state or territory

State or territory	Number of isolates tested	Decreased susceptibility Ceftriaxone		Resistance Ciprofloxacin		Azithromycin		Penicillin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	44	0	0.0	9	20.0	1	2.2	7	16.0
New South Wales	1,555	183	11.8	553	35.0	14	0.9	593	38.0
Darwin, Northern Territory	105	2	1.9	24	23.0	1	1.0	21	20.0
Remote, Northern Territory	239	2	0.8	5	2.1	0	0.0	3	1.3
Queensland	670	33	4.9	194	29.0	38	5.7	209	31.0
South Australia	212	4	1.9	56	26.0	6	2.8	39	18.0
Tasmania	45	11	25.0	22	49.0	0	0.0	17	38.0
Victoria	1,539	181	12.0	683	44.0	35	2.3	678	44.0
Western Australia	488	13	2.7	123	25.0	9	1.9	133	27 .0
Australia	4,897	429	8.8	1,669	34.0	104	2.1	1,700	35.0

lin resistant: (3 CMRP and 18 PPNG) (Table 3), and 2/21 also had decreased susceptibility to ceftriaxone. Of these 21 strains 19 (90%) were isolated from an urban STD clinic. In contrast, from the remote regions of the Northern Territory, 3/239 (1.3%) strains tested were penicillin resistant (1 PPNG and 2 CMRP).

Ceftriaxone

From 2001 onwards, gonococcal isolates with ceftriaxone MIC values 0.06 to 0.125 mg/L, and categorised as having decreased susceptibility by the AGSP, have been reported in Australia. The proportion has increased incrementally from 0.6% in 2006, to 4.4% in 2012. From 2012 to 2013, the proportion of gonococci with decreased susceptibility to ceftriaxone doubled from 4.4% to 8.8%. An increase in proportion was reported from all states and territories except the Australian Capital Territory (Table 4).

Ceftriaxone decreased susceptibility includes MIC values 0.06 and 0.125 mg/L. The right shift in the distribution of ceftriaxone MIC values over recent years (Table 5), is statistically significant with a sustained increase in the proportion of strains with an MIC value of 0.06 mg/L (2011–2012: [$P = 0.02$, 95% CI: 1.04–.62], and 2012–2013 [$P < 0.0001$, 95% CI: 1.70–2.38]). In 2010, the proportion of strains with ceftriaxone decreased susceptibility was higher than that reported in 2011. This proportion has subsequently increased as described. The proportion of strains with a ceftriaxone MIC 0.125 mg/L has also increased from 0.1% in 2010 and 2011, to 0.3% in 2012 to 0.6% in 2013 (Table 5).

Table 4: Number and rate of gonococcal isolates with decreased susceptibility to ceftriaxone (MIC 0.06–0.125 mg/L), Australia, 2009 to 2013, by state or territory

State or territory	Decreased susceptibility to ceftriaxone 2009		2010		2011		2012		2013	
	n	%	n	%	n	%	n	%	n	%
Australian Capital Territory	2	5.3	2	6.7	2	3.1	2	3.6	0	0.0
New South Wales	16	1.7	74	5.6	58	4.4	76	4.5	183	11.8
Northern Territory	1	0.2	1	0.2	2	0.4	0	0.0	4	1.5
Queensland	10	1.8	26	3.2	18	2.3	17	2.4	33	4.9
South Australia	9	5.3	19	11.6	1	0.7	1	0.7	4	1.9
Tasmania	0	0.0	0	0.0	0	0.0	0	0.0	11	24.4
Victoria	17	2.2	52	5.7	50	5.3	105	8.4	181	11.8
Western Australia	9	3.1	17	5.2	3	0.7	6	1.2	13	2.7
Australia	64	2.0	191	4.8	134	3.2	207	4.4	429	8.8

These differences were not significant, which may be attributable to the low number of strains in this MIC category. In December 2013, a gonococcal isolate (termed the A8806 strain) from a young female European traveller was found to have a ceftriaxone MIC value of 0.5 mgL—the highest ever reported in Australia. This isolate was also resistant to penicillin and ciprofloxacin but sensitive to azithromycin. Of concern was that genetic analysis showed that the A8806 strain had a mosaic penicillin binding protein 2 (PBP2) and other key similarities to the ceftriaxone resistant H041 strain reported from Japan in 2009. [21]

Table 5: Proportion of gonococcal isolates tested with MIC values at 0.06 mg/L and 0.125 mg/L, Australia, 2010 to 2013

Ceftriaxone MIC mg/L	2010	2011	2012	2013
0.06	4.8%	3.2%	4.1%	8.2%
0.125	0.1%	0.1%	0.3%	0.6%

Azithromycin

Nationally, the proportion of isolates exhibiting any resistance (2.1%) was higher than that reported in 2011–2012 (1.1%–1.3%) (Table 3). There were marked increases in the proportion of strains with resistance to azithromycin in 2012 from Queensland (from 2.7% to 5.7%), South Australia (from 0.7% to 2.8%) and Western Australia (from 0.6% to 1.9%).[2] In 2013, there were 4 isolates, 2 from Queensland and 2 from Victoria, that exhibited high level resistance to azithromycin (MIC value $>$ 256 mg/L).

Quinolone antibiotics

The AGSP uses ciprofloxacin as the representative quinolone. Quinolone resistant *N. gonorrhoeae* is defined as MICs $\geq$ 1 mg/L. The resistance mechanism in *N. gonorrhoea* has thus far been mediated only by chromosomal mechanisms so that incremental changes in MIC values are observed.

In 2013, 1,669 of the 4,897 gonococci examined (34%) were resistant to ciprofloxacin (Table 3). The proportion reported by the AGSP in 2012 (30%) was lower; however overall, there has been a trend of decreasing proportions since 2008 when 54% isolates were reported as ciprofloxacin resistant.

High-level tetracycline resistance

High-level tetracycline resistant *N. gonorrhoeae* (TRNG) is used as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea and are rarely, if ever used for treatment of gonococcal infection in Australia. The proportion of TRNG detected nationally increased between 2006 and 2011 from 12% to 21% and decreased to 14% in 2012, and was again reported as 14% in 2013.

TRNG were present in all jurisdictions in 2013, with the highest proportions in Western Australia (21%), Queensland (19%), South Australia (17%), New South Wales (14%) and the Northern Territory (14%).

Spectinomycin

In 2013, all isolates from all jurisdictions were susceptible to spectinomycin.

Discussion

High quality, representative AMR data are critical for public health security and effective antibiotic treatments for infections, including gonococcal infection, are essential for disease control. The WHO recommends that treatment regimens for gonococcal infection are based on epidemiological surveillance of the distribution and extent of AMR.[22] An AMR rate of 5% or more is the nominal threshold for change of treatment recommendations.[22] Programs such as the AGSP are conducted to determine the proportion of antimicrobial resistance in gonococcal strains isolated in a defined patient population and relate these findings to the likely efficacy of current treatment schedules.[19,22,23] For quality assurance and quality control of gonococcal AMR data, the AGSP provides the NNN laboratories with the AGSP External Quality Assurance Program, and WHO *N. gonorrhoeae* reference strains.[20,24]

The overall number of gonococcal strains examined by the AGSP in 2013 was higher in number but proportionally lower than previous years. These clinical isolates were from both the public and private health sectors, constituting a comprehensive sample of 33% of all notifications nationally. Of concern for gonococcal AMR surveillance programs worldwide, is the increasing use of NAAT for diagnosis, both in urban and remote settings. Whilst NAAT has an advantage over culture in terms of sensitivity and is more robust and reliable for remote settings where cultures may not survive, they have the distinct disadvantage in that they cannot test broadly for AMR. However, currently molecular AMR testing strategies can give targeted and specific information.[6,25,26] At this stage however, NAAT is unable to provide definitive data for predicting AMR, thus the continued commitment to the support of surveillance programs such as the

AGSP is vital. Culture based AMR surveillance is a foundation component for disease control strategies, essential in the current context of emerging gonococcal AMR globally.[4]

In the AGSP, decreased susceptibility to ceftriaxone is reported as an MIC value in the range 0.06–0.125 mg/L.[27] In 2013, in Australia, the proportion of strains in this MIC range doubled from 4.4% in 2012 to 8.8%, with the highest rates (11.8%) reported from the eastern states of Victoria, New South Wales and Queensland, where the largest increases in notifications were observed. Twenty–four per cent of the isolates from Tasmania were in this category, but the number of isolates tested was low (n=45). The data showed that the proportion of strains tested with an MIC value of 0.06 mg/L was 8.2% in 2013, significantly higher than that reported at that MIC value in 2012. The proportion of strains tested that had a ceftriaxone MIC value of 0.125 mg/L also doubled from 0.3% in 2012 to 0.6% in 2013. The multi-drug-resistant A8806 strain reported in late 2013, with a ceftriaxone MIC of 0.5 mgL is cause for considerable public health concern, particularly if spread occurs into remote Indigenous communities where disease rates are high, and extreme remoteness limits access to medical and diagnostic services. Enhanced surveillance strategies have been put in place by the NNN and to date no evidence of spread has been detected.

International and national surveillance programs define decreased susceptibility to ceftriaxone differently. For example, Public Health England's Gonococcal Resistance to Antimicrobials Surveillance Programme (GRASP), which reports AMR data from England and Wales; and the CDC gonococcal AMR surveillance programs define decreased susceptibility to ceftriaxone as MIC $\geq$ 0.125 mg/L. In recent years, both these programs have reported low proportions of strains with ceftriaxone decreased susceptibility by their criteria. In 2012, the GRASP reported 0.2% of isolates; and the CDC reported 0.3% with decreased susceptibility to ceftriaxone. In contrast, the European Centre for Disease Prevention and Control Gonococcal (ECDC), defines decreased susceptibility to ceftriaxone as MIC $>$ 0.125 mg/L.[28,29] The most recent (2011) ECDC gonococcal antimicrobial susceptibility surveillance reported 10 of 1,902 isolates with decreased susceptibility to ceftriaxone (MIC $>$ 0.125mg/L) and all 10 isolates were from a total of 214 strains tested in Austria and Germany.[29] The absence of an international standard definition of decreased susceptibility to ceftriaxone, and non-uniform methods of AMR testing confound comparison of surveillance data. However, in 2012 the WHO Global Action Plan nominated the criteria for decreased susceptibility to ceftriaxone as an MIC value $\geq$ 0.125 mg/L.[4] The 2013 surveillance data from the GRASP; the CDC and the ECDC are yet to be published. Interestingly, comparison of 2011 and 2012 data between these international surveillance programs shows that the rates of gonococci with reduced susceptibility to ceftriaxone were very similar to that reported in Australia (Table 6). It remains to be seen if the substantial increase in the proportion of strains with decreased susceptibility seen in Australian gonococci in 2013 is also reported in the United States of America, the United Kingdom and Europe.

Table 6: Proportion of gonococcal isolates with decreased susceptibility to ceftriaxone reported in the gonococcal antimicrobial resistance programs of the United States Centres for Disease Control, the European Centres for Disease Control and Public Health England's Gonococcal Resistance to Antimicrobials Surveillance Programme, 2011 to 2012

Surveillance program criteria for decreased susceptibility MIC mg/L	2011 % Decreased susceptibility	2012 % Decreased susceptibility
ECDC MIC >0.125 mg/L	0.5	NA
CDC MIC > 0.125 mg/L	0.4	NA
AGSP MIC 0.125 mg/L	0.1	0.3
PHE GRASP MIC ≥0.125 mg/L	0.0	0.2

Gonococci with decreased susceptibility to ceftriaxone have also been reported in increasing numbers in the WHO Western Pacific Region;[14] however, the scope of this is not known as wide scale MIC based data from this region are not available.[14] Decreased susceptibility to the cephalosporin antibiotics has been accompanied by increasing numbers of reports of treatment failures.[8,30–33]

The primary concern is that molecular studies have shown that many circulating gonococcal strains harbour a mosaic penicillin binding protein 2 (PBP2). This mosaic PBP2 sequence has been shown to be a stepping stone for ceftriaxone resistance: the 2010 Japan H041 strain had only 3 important additional amino acid substitutions, the A8806 strain had 2 of the 3 amino acid substitutions found in the H041 strain, and the F89 strain, found in France, then Spain, had only 1 additional amino acid substitution to the mosaic PBP2.[6]

In essence therefore, there are a significant proportion of circulating strains that likely harbour the key foundation elements for ceftriaxone resistance (mosaic PBP2). Moreover, the difference between decreased susceptibility and resistance is now known to be only a few point mutations, and further, these strains are under constant selection pressure.[3,6] Given this, the level of concern about the development of ceftriaxone resistance is growing globally.

A dual therapy strategy of ceftriaxone with oral azithromycin for uncomplicated gonococcal infection is now in use in many states and territories of Australia. Resistance to azithromycin has been reported with very high MIC levels overseas.[34,35] In 2013, for the first time in Australia, there were 4 strains with high level resistance to azithromycin reported, two from Victoria and two from Queensland, and of these, two were likely acquired from China.[36]

In 2013, in the majority of Australia, with the exception of remote Northern Territory, 35% of gonococci were resistant to penicillin and 34% were resistant to quinolone antibiotics. These proportions were higher than those reported in 2012, where there was 32% resistance to penicillin and 30% to the quinolone antibiotics. Prior to 2012, there was a reduction in penicillin and quinolone resistance nationally from 2008 to 2011, whereas previously, resistance to both classes of antibiotics had been increasing annually since 2003.[15] Fluctuations in penicillin and quinolone resistance have been reported over time by the AGSP. Since 2003, aggregated data has shown a predominant clone of CMRP coupled with high-level quinolone resistance circulating with increasing frequency annually.[2,15] In 2012, the increase in the proportion of isolates with penicillin and quinolone resistance is likely to be a further reflection of the clonal shift in gonococcal isolates nationally.

The proportion of gonococci with high-level tetracycline resistance in Australia increased from 2006 to 2008 and stabilised at 21% in 2009 to 2010. The proportion of TRNG decreased to 18% in 2011, then to 14% in 2012 and remained unchanged (14%) in 2013.

In the remote areas of the Northern Territory, low rates of penicillin and ciprofloxacin resistance continue to be reported. This underscores the continued need for disaggregated surveillance data, as these data are used to define treatment regimens appropriate for the various jurisdictions. Remote areas in some jurisdictions with high disease rates continue to be able to use penicillin-based treatments. However, effective use of this treatment is contingent on continued, timely and vigilant monitoring of resistance patterns. In some regions culture and AMR testing are logistically difficult. A PPNG assay developed in Australia by members of the NNN[26] is currently being utilised in Western Australia to enhance surveillance for penicillin resistance and to inform local gonorrhoea treatment guidelines.[6]

The continued emergence and spread of AMR in *N. gonorrhoeae* is widely recognised as a global public health threat, and in 2013 this threat was rated as urgent by the CDC.[5] Broad based disease control strategies including the rational use of antibiotics have been called for.[3,22,37,38] The WHO Global Action Plan states that disease control strategies and the understanding of the global scope of AMR need to continue to be informed by surveillance programs of AMR, nationally and internationally.[4]

Enhanced surveillance to monitor *N. gonorrhoeae* with elevated MIC values coupled with sentinel site surveillance in high risk populations remains critically important to inform our therapeutic strategies and to detect instances of treatment failure. Sentinel site surveillance programs involve patient follow up and test of cure cultures after treatment of *N. gonorrhoeae* infections, in particular those in oropharyngeal sites. This is currently conducted in a very limited number of settings in Australia, and needs to be expanded throughout all jurisdictions as a matter of priority.

In summary, gonococcal infection rates and AMR rates are increasing in Australia. In 2013 the proportion of strains with elevated ceftriaxone MIC values doubled from 2012. A multi-drug-resistant strain with high level ceftriaxone MIC has now been reported and, in addition, in another first for Australia, in 2013, high level resistance to azithromycin was reported.[36] The next direction for treatment is uncertain, but what is clear is that additional and renewed efforts for disease prevention and disease control are urgently called for, and that continued monitoring of AMR to inform treatment and monitor interventions is paramount.

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National trachoma surveillance annual report, 2012

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Abstract

Australia remains the only developed country to have endemic levels of trachoma (a prevalence of 5% or greater among children) in some regions. Endemic trachoma in Australia is found predominantly in remote and very remote Aboriginal communities. The Australian Government funds a National Trachoma Surveillance and Reporting Unit to collate, analyse and report trachoma prevalence data and document trachoma control strategies in Australia through an annual surveillance report. This report presents data collected in 2012. Data are collected from Aboriginal and Torres Strait communities designated as at-risk for endemic trachoma in the Northern Territory, Queensland, South Australia and Western Australia. The World Health Organization grading criteria were used to diagnose cases of trachoma in Aboriginal children with jurisdictions focusing screening activities on the 5–9 years age group; however, some children in the 1–4 and 10–14 years age groups were also screened. The prevalence of trachoma within a community was used to guide treatment strategies as a public health response. Aboriginal adults aged 40 years or older were screened for trichiasis. Community screening coverage of the designated at-risk communities was 96%. Screening coverage of the estimated population of children aged 5–9 years and adults aged 40 years or older in at-risk communities was 71% and 31%, respectively. Trachoma prevalence among children aged 5–9 years who were screened was 4%. Of communities screened, 63% were found to have no cases of active trachoma and 25% were found to have endemic levels of trachoma. Treatment was required in 87 at-risk communities screened. Treatment coverage of active cases and their contacts varied from 79%–97% between jurisdictions. Trichiasis prevalence was 2% within the screened communities. *Commun Dis Intell* 2015;39(1):E146–E157.

Keywords: active trachoma, control activities, endemic, facial cleanliness, Northern Territory, Queensland, SAFE control strategy, South Australia, surveillance, Western Australia

Introduction

This is the 7th national trachoma surveillance annual report prepared by the National Trachoma Surveillance Reporting Unit.[1-6] Trachoma screening and management data for 2012 were provided to the National Trachoma Surveillance and Reporting Unit (NTSRU) by the Northern Territory, South Australia, Western Australia and Queensland.

Trachoma is one of the major causes of preventable blindness globally.[7] Trachoma is an eye infection caused by *Chlamydia trachomatis* serotypes A, B, Ba and C. Infection with the relevant *C. trachomatis* serotypes causes inflammation of the conjunctiva. Diagnosis of trachoma is by visual inspection, and the detection of follicles (white spots) and papillae (red spots) on the inner upper eye lid. Repeated infections, especially during childhood, may lead to scarring with contraction and distortion of the eyelid, which may in turn cause the eyelashes to rub against the cornea. This condition is known as trichiasis and can lead to blindness.[8,9] Scarring of the cornea due to trichiasis is irreversible. If early signs of in-turned eyelashes are found, surgery is usually effective in preventing further damage to the cornea. The infection can be transmitted through close facial contact, hand-to-eye contact, via fomites (towels, clothing and bedding) or by flies. Trachoma generally occurs in dry, dusty environments and is linked to poor living conditions. Overcrowded households, limited water supply for bathing and general hygiene, poor waste disposal systems and high numbers of flies have all been associated with trachoma. Children generally have the highest prevalence of trachoma and are believed to be the main reservoirs of infection because the infection has a longer duration in children than in adults[10]

Trachoma is usually treated by a single dose of the antibiotic, azithromycin. Best public health practice involves treatment of all members of the household in which a person with clinically active trachoma resides, whether or not they have evidence of trachoma. Depending on the prevalence of trachoma in a community, treatment may also be extended to all children aged 6 months to 14 years, or to all members of the community, excluding or including infants less than 6 months of age.[10]

The Alliance for the Global Elimination of Blinding Trachoma by 2020 (GET 2020)[11] initiative, supported by the World Health Organization (WHO) advocates the implementation of the SAFE strategy, with its key components of surgery (to correct trichiasis), antibiotic treatment, facial cleanliness and environmental improvements.[12]

This strategy is ideally implemented through a primary care model within a community framework, ensuring consistency and continuity in screening, control measures, data collection and reporting, as well as the building of community capacity. The target set by both WHO and the Communicable Diseases Network Australia (CDNA) for elimination of blinding trachoma is community prevalence in children aged 1–9 years of less than 5% over a period of 5 years.[8,11,13,14]

Trachoma control in Australia

Australia is the only high income country where trachoma continues to be endemic.[15] It occurs primarily in remote and very remote Aboriginal communities in the Northern Territory, South Australia and Western Australia. In 2009, the Australian Government invested in the *Closing the Gap – Improving Eye and Ear Health Services for Indigenous Australians* measure, which included committing $16 million over 4 years towards eliminating trachoma in Australia. The funding is for improving and expanding screening and control activities, as well as establishing a strong framework for monitoring and evaluation of trachoma control activities. As a result, increased funding was provided to the Northern Territory, South Australia and Western Australia for trachoma control activities from 1 July 2010 and to Queensland and New South Wales in 2012.

The surveillance and management of trachoma is guided by the CDNA *Guidelines for the public health management of trachoma*.[16] These guidelines were recently revised and released in January 2014.[16.] This document was developed in the context of the WHO SAFE strategy and make recommendations for improving data collection, collation and reporting systems.

CDNA guidelines recommend the treatment of active cases and their household contacts. When prevalence is greater than 10% and cases are not clustered within a few households, community-wide treatment is advised. The approach to community-wide treatment differs across jurisdictions. In the Northern Territory, the recommendation is taken to mean the entire community, whereas South Australia and Western Australia choose to treat all children aged between 6 months and 14 years. The differences in approach are a response to the average number of contacts per active case in each jurisdiction: in the Northern Territory 1:24; 1:7 in South Australia; and 1:6 in Western Australia.

Methods

Each participating jurisdiction undertook screening and treatment for trachoma according to its respective protocols, and in the context of the national 2006 CDNA *Guidelines for the public health management of trachoma in Australia* recommend specific treatment strategies depending on the prevalence of trachoma detected through screening.[8]

In 2006, when the National Trachoma Management Program was initiated,[1] participating jurisdictions identified at-risk communities from historical prevalence data and other knowledge, including known transiency into endemic communities. Over time, additional communities have been reclassified by the jurisdictions as being at-risk due to prevalence rates of over 5%. Screening for trachoma focuses on at-risk communities, but a small number of other communities designated as not-at-risk have also been screened, generally if there is anecdotal information suggesting the presence of active trachoma. Communities that undertook screening and were found not to have trachoma are then classified as being not at-risk.

The WHO trachoma grading criteria[17] were used to diagnose and classify individual cases of trachoma in all jurisdictions except Queensland who also utilised polymerase chain reaction (PCR) testing in children identified with follicles. Forms for data collection at the community level were developed by the NTSRU based on the CDNA guidelines. Completed forms were forwarded from the jurisdictional coordinators to the NTSRU for checking for completeness and accuracy and analysis. Information provided to the NTSRU at the community level for each calendar year included:

- number of Aboriginal children aged 1–14 years screened for clean faces and the number with clean faces, by age group;
- number of Aboriginal children aged 1–14 years screened for trachoma and the number with trachoma, by age group;
- number of episodes of treatment for active trachoma, household contacts and other community members, by age group;
- number of Aboriginal adults (over 40 years of age) screened for trichiasis, number with trichiasis, and the number who had surgery for trichiasis; and
- community level implementation of WHO SAFE strategies.

Northern Territory

Trachoma screening and management in the Northern Territory was undertaken through collaboration between the Department of Health (Centre for Disease Control and Health Development) and Aboriginal Community Controlled Health

Services (ACCHS). Trachoma screening was incorporated into the Healthy School-Age Kids program[18] annual check and conducted by either local primary health-care services or community-controlled services, with support from the Centre for Disease Control Trachoma Team. Following screening, treatment was generally undertaken by primary health-care services with support from the trachoma team when requested.

In 2012, community screening for trichiasis was undertaken primarily by clinic staff, ACCHS, or by optometrists or ophthalmologists from the Regional Eye Health Service based in Alice Springs. In 2 large communities in the Northern Territory, mass trichiasis screening of all Indigenous adults aged over 40 years was conducted with assistance from the Centre for Disease Control Trachoma Team staff. Data relating to trichiasis was received in 42 communities in the Northern Territory. The Northern Territory Department of Health advised that a further 28 communities were screened for trichiasis but data were not made available to the NTSRU.

South Australia

In 2012, Country Health South Australia was responsible for managing the South Australia, trachoma screening and treatment program. Country Health South Australia contracted with local health service providers, ACCHS, the Aboriginal Health Council of South Australia and Nganampa Health Service to ensure coverage of screening services in all at-risk rural and remote areas. Additional screening activities were undertaken by the Eye Health and Chronic Disease Specialist Support Program (EH&CDSSP), coordinated by the Aboriginal Health Council of South Australia and supported by the Medical Specialist Outreach Assistance Program. This program provides regular visits to South Australia, remote Aboriginal communities by optometrists and ophthalmologists. Trichiasis screening was undertaken opportunistically for adults by both the EH&CDSSP team and the trachoma screening service providers, and is also undertaken routinely as part of the Adult Annual Health Checks. Country Health South Australia advised that regional boundary structures had changed in 2 regions in 2011 and 2012 and therefore data from these reports cannot be directly compared with previous reports.

Western Australia

Trachoma screening and management in Western Australia is the responsibility of population health units in the Kimberley, Goldfields, Pilbara and Midwest health regions. In collaboration with the local primary health-care providers, the population health units screened communities in each region within a 2-week period, usually at the end of August or early September. People identified with active trachoma were treated at the time of screening. In 2012, two communities in Western Australia, one each in the Goldfields and Pilbara regions did not screen children in the 5–9 years age group, as children in this age group were not present at the time of screening.

Trichiasis screening was undertaken in conjunction with adult influenza vaccinations. Screening of the target population also occurs with the Visiting Optometrist Scheme in the Kimberley region. This amalgamation alters trends presented in reports from 2011 from current and consecutive reports.

Queensland

In 2012, Queensland joined the trachoma screening program and screened 6 remote communities in 3 regions that were considered to be potentially at-risk. This screening was undertaken by the Queensland Health's Deadly Ears Program and supported by an ophthalmologist. Queensland screened according to national guidelines for areas of low prevalence or endemicity, which recommend using the WHO grading system,[15] to identify possible trachoma cases. Children identified with follicles consistent with *C. trachomatis* were further tested using PCR tests unlike other jurisdictions.

Under the contract between Queensland and the Australian Government Department of Health trichiasis screening was not required to be undertaken.

Data analysis

For the purpose of this report, a community is defined as 'a specific location where people reside and where there is at least 1 school.' At-risk communities are classified by jurisdictions as being at higher risk of trachoma (generally based on prevalence above 5% in age group 5–9 years). Communities are defined as being not-at-risk by having a baseline prevalence of below 5%; if previously at-risk, 5 years of a prevalence below 5%; or no historical evidence of trachoma prevalence. Community coverage is defined as the number of at-risk communities screened for trachoma as a proportion of those that were identified to possibly have trachoma. Individual screening coverage is the proportion of children in the respective target age groups, i.e. 1–4, 5–9 and 10–15 years, in a region that was actually screened. Active trachoma is defined as the presence of chronic inflammation of the conjunctiva caused by infection with *C. trachomatis*. This includes the WHO grades of trachomatous inflammation – follicular (TF) and

trachomatous inflammation – intense (TI).[15] Clean face is defined as the absence of dirt, dust and crusting on cheeks and forehead. The clean face target is at least 80% of children within the community having a clean face at the time of screening. The presence of nasal and ocular discharge significantly correlates to the risk for both acquiring and transmitting trachoma.[10,19] Trachomatous trichiasis (TT) is defined as the evidence of the recent removal of in-turned eyelashes or at least one eyelash rubbing on the eyeball.

In 2012, population data for trachoma screening coverage were provided by each jurisdiction, which provides a more accurate estimate of population than Australian Bureau of Statistics (ABS) data. The manner in which the populations were calculated differed among jurisdictions, with some jurisdictions using school enrolment lists, Health Information populations lists, or a combination of both and local knowledge. The 2011 ABS census projected population estimates were included in the tables to provide a comparison. For communities where population data were not provided, coverage estimates were based on the 2011 Australian census projected forward.[8] The population for communities in previous years was derived from projected data from the 2006 Australian census using the ABS standard estimates of population increase (1.6%, 1.8%, 2.1% and 2.6% in the Northern Territory, Western Australia, South Australia and Queensland respectively).[20] Population estimates for trichiasis screening coverage were based on the projected 2011 Australian census data. Population estimates based on the 2011 census do not account for population movements within communities, regions and jurisdictions. Prevalence of active trachoma was calculated using the number of children screened as the denominator.

Trachoma data were analysed in the key screening age groups 1–4, 5–9 and 10–14 years. Comparisons over time were limited to the 5–9 years age group, for which screening coverage has been consistently high. Data from 2006 were excluded from assessment of time trends as collection methods in this first year of the surveillance program differed from those subsequently adopted. Trachoma prevalence in the 1–9 years age group was calculated by weighting the population provide by the jurisdictions. For population averaging we took the prevalence for the for ages 1–4 and 5–9 years and calculate a weighted average given the populations in those 2 groups, to better reflect the prevalence of the 1–9 years age group.

The NTSRU in 2011 developed a web-based data entry system that minimised duplicates and inconsistent entry. This database is being enhanced to allow improved accessibility in the field and report generation for jurisdictions.

Results

National results

In 2012, 96% (195/204) of designated at-risk communities were screened for trachoma across 16 regions in the Northern Territory, South Australia, Western Australia and Queensland (Figure 1, Table 1). Within regions screened for trachoma, 71% (5,426/7,676) of the estimated resident children at-risk aged 5–9 years were screened. Screening coverage in children aged 5–9 years in at-risk regions increased since the last report in the Northern Territory, South Australia and Western Australia to 67%, 79% and 73% respectively. Screening was conducted in Queensland for the first time in 2012 with a screening coverage of 64%. (Table 1, Figure 2).

The overall national prevalence of active trachoma among children aged 5–9 years in screened communities was 4%, with 4% in the Northern Territory and Western Australia, 1% in South Australia, and nil in Queensland (Table 1). Follicles consistent with TF were observed in 1 community in Queensland; however, PCR tests results were negative for *C. trachomatis*. The prevalence of trachoma in children aged 5–9 years decreased since 2009 in South Australia and from the previous 4 years in the Northern Territory and Western Australia (Figure 3). In 25% (48/193) of all communities where children aged 5–9 years were screened, endemic levels of trachoma (over 5%) were found. Hyperendemic levels of trachoma (over 20% prevalence of trachoma) were found in 8% (15/193) (Figure 4). Of all communities that screened children aged 5–9 years, 63% (121/193) had no trachoma detected (Figure 4). The proportion of screened communities with no trachoma detected increased in the Northern Territory by 20%, in South Australia by 21% and in Western Australia by 3% in 2012 compared with 2011 (Figures 3 and 5) and the proportion of screened communities with endemic trachoma (greater than 5% prevalence) decreased in the Northern Territory (28%), South Australia (9%) and Western Australia (29%) in 2012 compared with 2011 (Figures 4 and 6). Trachoma prevalence was found to be marginally higher in boys compared with girls in all jurisdictions with endemic trachoma (Northern Territory male: 5.3% female: 3.7%; South Australia male: 1.8% female: 1%; Western Australia male: 3.9% female: 3.1% (Figure 7).

Active trachoma cases requiring treatment were detected in 87 of 195 communities screened, with

Table 1: Trachoma screening coverage, trachoma prevalence and clean face prevalence among at-risk Aboriginal communities, Australia, 2012, by state or territory

	Northern Territory				South Australia				Western Australia				Queensland				Total			
Number of communities at risk	82				38				78				6				204			
Number of communities screened	76				36				77				6				195			
Age group (years)	**1–4**	**5–9**	**10–14**	**1–14**	**1–4**	**5–9**	**10–14**	**1–14**	**1–4**	**5–9**	**10–14**	**1–14**	**1–4**	**5–9**	**10–14**	**1–14**	**1–4**	**5–9**	**10–14**	**1–14**
ABS estimated number of Aboriginal children at risk	3,492	3,981	3,359	10,832	942	961	855	2,758	1,965	2,097	1,896	5,958	380	414	286	1,080	6,779	7,453	6,396	20,628
Jurisdiction estimated number of Aboriginal children at risk	3,091	3,893	3,733	10,717	1,176				524	2,306	1,387	4,217	224	385	96	705	3,839	7,760	5,216	15,639
Children examined for clean face	446	2,640	1,895	4,981	14	930	323	1,267	254	1,709	811	2,774	1	194	52	247	715	5,473	3,081	9,269
Children with clean face	293	1,972	1,719	3,984	9	843	320	1,172	179	1,379	713	2,271	1	136	50	187	482	4,330	2,802	7,614
Clean face prevalence	66%	75%	91%	80%	64%	91%	99%	93%	70%	81%	88%	82%	100%	70%	96%	76%	67%	79%	91%	82%
Children examined for trachoma	362	2,610	1,908	4,880	14	933	322	1,269	244	1,689	793	2,726	1	194	52	247	621	5,426	3,075	9,122
Trachoma screening coverage	12%	67%	51%	46%	79%				47%	73%	57%	65%	0.4%	50%	54%	35%	16%	70%	59%	58%
Children with active trachoma	24	117	35	176	0	13	1	14	6	71	34	111	0	0	0	0	30	201	70	301
Active trachoma prevalence	7%	4%	2%	4%	0%	1%	0.3%	1%	2%	4%	4%	4%	0%	0%	0%	0%	5%	4%	2%	3%
Trachoma prevalence 1-9 years (weighted by population)*	6%				1%				3%				0%				4%			

* Calculated as the proportion of children with active trachoma in age groups 1–4 and 5–9 years, weighted by the estimated population sizes of each age group. This calculation accounts for uneven coverage with respect to age groups.

Figure 1: Proportion of at risk communities screened and trachoma prevalence in children aged 5–9 years in screened communities, Australia, 2012, by region

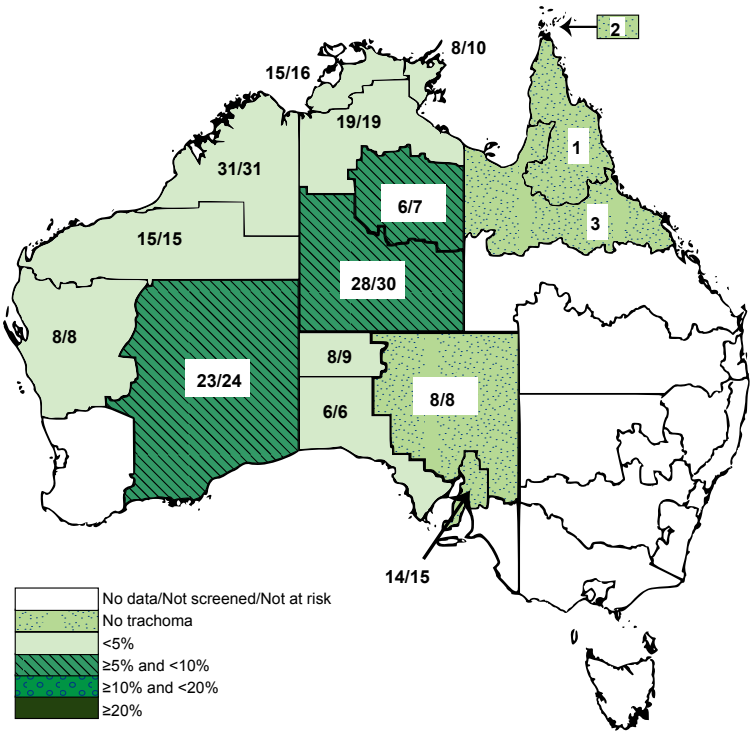
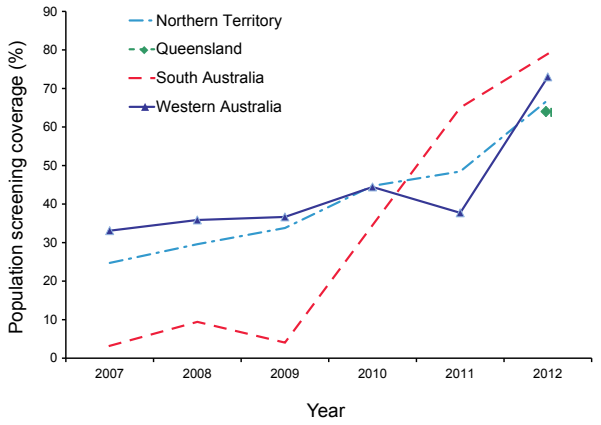
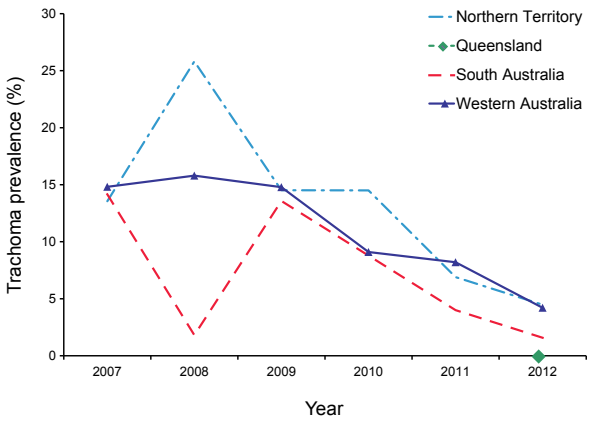


Figure 2: Population screening coverage of children aged 5–9 years, Australia, 2007 to 2012, by year and jurisdiction



* Queensland data only available for 2012.

Figure 3: Trachoma prevalence among screened children aged 5–9 years, Australia,* 2007 to 2012, by year and jurisdiction



* Queensland data only available for 2012.

95% of those with active trachoma reported to be appropriately treated (Table 2). Estimated treatment coverage of contacts of those with active trachoma was 81% overall, 79% in the Northern Territory, 97% in South Australia and 95% in Western Australia (Table 2). Estimates of appropriate contacts requiring treatment were provided by the jurisdictions. Six communities in Western Australia did not provide estimates for contacts to be treated and treated active cases only. No treatment was required for Queensland (Table 2).

Trichiasis screening coverage of adults over 40 years of age increased in the Northern Territory, Western Australia and South Australia, both in terms of the number of communities screened and population screened from 2011 to 2012. A total of 4,468 (33%) adults of an estimated at-risk population of 13,406, were reported to have been screened for trichiasis across the Northern Territory, South Australia and Western Australia (Table 3). Overall trichiasis prevalence among those screened was 2% with 61 cases reported in the Northern Territory,

Table 2: Trachoma treatment coverage among at-risk Aboriginal communities, Australia, 2012, by state or territory

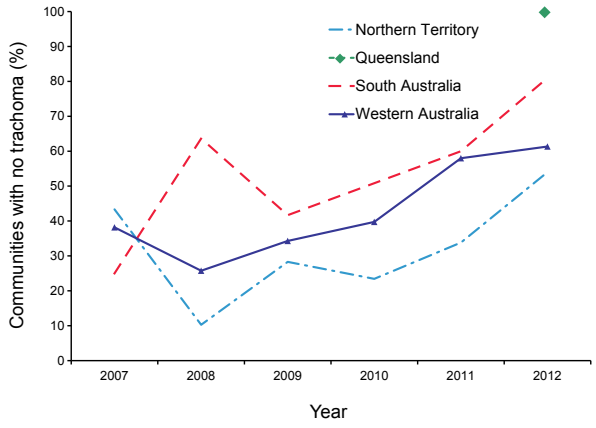
	Northern Territory					South Australia					Western Australia					Queensland					Total				
Number of communities at risk	82					38					78					6					204				
Number of communities screened	76					36					77					6					195				
Number of communities requiring treatment	43					9					35					0					87				
Number of communities treated according to CDNA guidelines*	41					8					26										75				
Age group (years)	**0-4**	**5-9**	**10-14**	**15+**	**All**	**0-4**	**5-9**	**10-14**	**15+**	**All**	**0-4**	**5-9**	**10-14**	**15+**	**All**	**0-4**	**5-9**	**10-14**	**15+**	**All**	**0-4**	**5-9**	**10-14**	**15+**	**All**
Active cases requiring treatment	24	115	35		174	0	13	1		14	6	71	34		111						30	199	70		299
Active cases who received treatment	23	114	33		170		13	1		14	6	68	29		103						29	195	63		287
% Active cases received treatment	96%	97%	94%		98%		100%	100%		100%	100%	96%	85%		93%						97%	98%	90%		96%
Estimated contacts requiring treatment	572	619	581	2,532	4,304	9	21	10	63	103	91	135	112	301	639						672	775	703	2,896	5,046
Number of contacts who received treatment	450	557	474	1,911	3,392	9	20	10	61	100	75	126	103	290	594						534	703	587	2,262	4,086
% estimated contacts received treatment	79%	90%	82%	75%	79%	100%	95%	100%	97%	97%	82%	93%	92%	96%	93%						79%	91%	83%	78%	81%
Estimated overall treatment coverage	79%	91%	82%	75%	80%	100%	97%	100%	97%	97%	84%	94%	90%	96%	93%						80%	92%	84%	78%	82%

* Communicable Diseases Network Australia (CDNA), *Guidelines for the public health management of trachoma in Australia*. March 2006

11 in South Australia, and 22 in Western Australia. Trichiasis surgery was reported to have been undertaken for 16 people with trichiasis in 2012; 5 in the Northern Territory, 2 in South Australia and 9 in Western Australia. Queensland was not required in the program agreement with the Australian Government Department of Health and Queensland to screen for trichiasis in 2012.

The overall prevalence of clean faces in children aged 5–9 years was 79%. The prevalence was 75% in the Northern Territory, 90% in South Australia, 81% in Western Australia and 70% in Queensland (Table 1). The proportion of screened communities with over 80% of children aged 5–9 years reported as having a clean face was 58% in the Northern Territory, 89% in South Australia, 57% in Western Australia and 33% in Queensland (Figure 8). Health promotion activities were reported to be taking place in 133 at-risk communities across all jurisdictions. These activities included education sessions regarding trachoma and facial hygiene, distribution of print material and social marketing campaigns to increase the knowledge and risk factors of trachoma in communities.

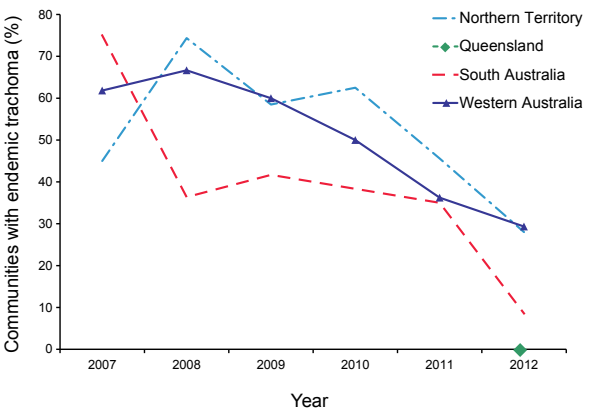
Figure 4: Proportion of screened at-risk communities* according to level of trachoma prevalence in children aged 5–9 years, Australia, 2012, by jurisdiction



* Two communities in Western Australia did not screen children in the 5–9 years age group.

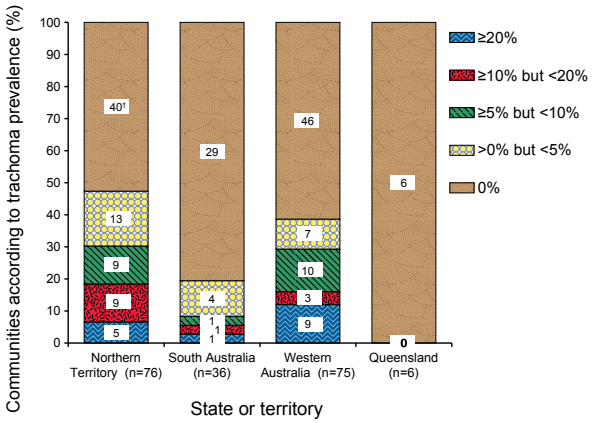
† Number of communities

Figure 5: Proportion of screened communities in which no trachoma was reported among children aged 5–9 years, Australia, 2007 to 2012, by year and jurisdiction



* Queensland data only available for 2012.

Figure 6: Proportion of screened communities with endemic trachoma* among children aged 5–9 years, Australia, 2007 to 2012, by year and jurisdiction



* Prevalence greater than 5%.

† Queensland data only available for 2012.

Figure 7: Trachoma prevalence among children aged 5–9 years in screened at-risk communities, Australia, 2012, by sex and jurisdiction

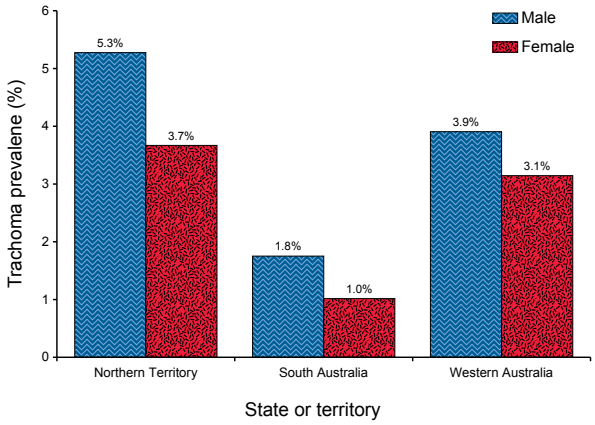
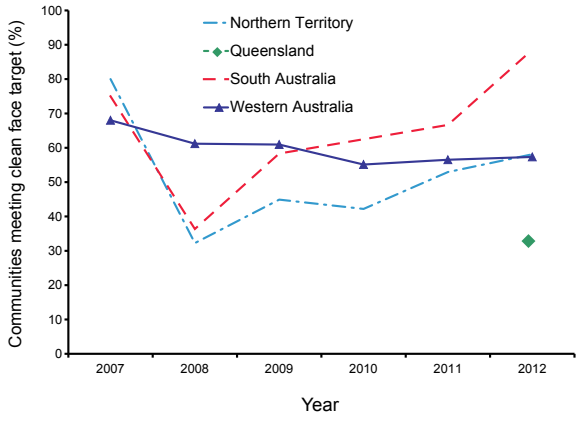


Figure 8: Proportion of screened communities meeting clean face target* in children aged 5–9 years, Australia,† 2007 to 2012, by year and jurisdiction



* Clean face target is at least 80% of children within the community having a clean face at time of screening.

† Queensland data only available for 2012.

Discussion

Screening coverage

Population estimates were generally similar for the 5–9 years age group except in Queensland. In 2012, all regions increased trachoma screening coverage of children aged 5–9 years except the Darwin Rural region in the Northern Territory and the Far North region in South Australia, compared with 2011 data.

The newly revised *Guidelines for the public health management of trachoma in Australia*[16] allow endemic communities to focus resources on treatment without the need for repeat screening for up to 3 years. This strategy will affect the number of communities screened and the regional screening coverage of children. The impact of this strategy may not be apparent for several years.

Trachoma prevalence

NTSRU has been able to estimate the prevalence using population weights. In Australia, the prevalence in the 5–9 years age group is accepted as a sufficient measure of the prevalence of trachoma within at-risk communities.

Across all 4 jurisdictions in 2012, the prevalence of trachoma in children aged 5–9 years in screened at-risk communities was 4%. This was lower than the prevalence of 7% in this age group in 2011.[9]

Subsequently, Queensland has concluded that trachoma is not a public health concern for Queensland and will review further screening activities.

In all other reporting jurisdictions, a decreasing trend in trachoma prevalence among screened individuals has been observed since 2009. Decreasing trends in the Northern Territory, South Australia and Western Australia were also observed in the number of communities found to have prevalence of greater than 5% among screened children aged 5–9 years (endemic trachoma), while there was an increasing trend in the number of communities that reported no trachoma in screened children aged 5–9 years. It may be timely to review the risk classifications of communities that have reported no evidence of trachoma, such as three in the

Table 3: Trichiasis screening coverage, prevalence and treatment among Aboriginal adults aged over 40 years, Australia, 2012

	Northern Territory		South Australia		Western Australia		Total	
	n	%	n	%	n	%	n	%
Number of communities at risk	82		38		78		198	
Number of communities screened for trichiasis	42*	51	14	37	52	69	108	52
Adult population of at-risk communities	7,030		2,246		4,130		13,406	
Adults examined (% of estimated population at risk)	1,278	18	1,061	47	2,129	52	4,468	3
With trichiasis (% of adults examined)	61	5	11	1	22	1	94	2
Offered ophthalmic consultation	49		11		22		82	
Surgery in past 12 months	5		2		9		16	

* Twenty-eight further communities were screened in the Northern Territory, but findings were not made available to the National Trachoma Surveillance and Reporting Unit.

Darwin Rural region, four in the Katherine region (Northern Territory); one in Eyre and Western region, seven in the Far North region, all of the York and Mid North region (South Australia); two in the Goldfields region, two in the Midwest region and three in the Kimberley region (Western Australia). This process will allow resources to be better targeted to endemic communities.

The target set by both WHO and CDNA for elimination of blinding trachoma is community prevalence in children aged 1–9 years of less than 5% over a period of 5 years.[8,11]

As communities are reclassified as being not at-risk due to prevalence rates below 5% at either baseline screen or consistently over 5 years, future prevalence trends may increase for a period of time as the at-risk population becomes more concentrated.

In 2012, the NTSRU collected prevalence data by sex. These data had not been collected in previous years. There is evidence in many trachoma endemic countries that women are disproportionately more likely to be at-risk of trachoma, and become blind due to trichiasis.[10] However, the national results from 2012 illustrate that males in all Australian jurisdictions had a higher prevalence of trachoma compared with females.

Trachoma treatment

Ninety-five per cent of active cases received treatment in 2012.

In 2012, jurisdictions supplied estimates of the populations requiring treatment. These estimates were influenced by the interpretation of the current treatment guidelines. For 6 communities in Western Australia in which only active cases were treated, estimates of the number of household contacts or community members requiring treatment were not obtained; therefore treatment coverage was overestimated for Western Australia. Nationwide, 75 of the 87 communities that required treatment were treated according to their jurisdictional interpretation of the current CDNA treatment guidelines.

The Northern Territory also undertook 6-monthy treatment of all members of the community in 6 communities that detected hyperendemic levels of trachoma and achieved an overall coverage level of 70% for the second treatment.

Trichiasis

Screening for trichiasis among Aboriginal adults aged over 40 years in the Northern Territory, South Australia and Western Australia increased in 2012. However, coverage remained low, with screening rates of 18% in the Northern Territory, 47% in South Australia and 52% in Western Australia. Prevalence of trichiasis of adults screened in communities designated as at-risk for trachoma was 2% (94/4,468). These prevalence levels include data collected in communities currently designated as at-risk, and do not take into account the possibility that endemic areas may have changed over time.

Facial cleanliness

Facial cleanliness is a major component of the SAFE strategy, recognising that the presence of nasal and ocular discharge significantly correlates with the risk for both acquiring and transmitting trachoma.[10] The proportion of children aged 5–9 years screened who had clean faces increased slightly in the Northern Territory, Western Australia and South Australia compared with 2011.

Program delivery and monitoring

Significant improvements in program delivery have been reported in 2012 with increased coverage of screening and treatment delivery and health promotion activities. Data quality also improved in all jurisdictions; however, as many regions chose to focus on the 5–9-years age group, data regarding the 1–4-years age group were not comprehensive.

Progress towards Australia's elimination target

As a signatory to the WHO Alliance of the Global Elimination of Blinding Trachoma by the year 2020 (GET 2020), Australia is committed to ensuring that trachoma levels continue to decrease to below endemic levels in at-risk communities. This report has shown significant decreases in trachoma prevalence in the Northern Territory, South Australia and Western Australia. With the implementation of new guidelines in 2014 and sustained efforts, as reported in this report, Australia remains on course to eliminate trachoma by 2020.

Acknowledgements

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Jurisdictional contributors to trachoma data collection:

Queensland

Deadly Ears Program, Department of Health

Northern Territory

Aboriginal Community Controlled Health Services

Aboriginal Medical Services Alliance of the Northern Territory

Centre for Disease Control, Northern Territory Department of Health

Healthy School Age Kids Program: Top End and Central Australia

South Australia

Aboriginal Community Controlled Health Services

Aboriginal Health Council of South Australia

Country Health South Australia

Western Australia

Aboriginal Community Controlled Health Services

Communicable Diseases Control Directorate, Health Department of Western Australia

Goldfields Population Health Unit

Kimberley Population Health Unit

Midwest Population Health Unit

Pilbara Population Health Unit

The National Trachoma Surveillance and Control Reference Group

The NTSRU is guided by the National Trachoma Surveillance and Control Reference Group, members of which include the following representatives and organisations:

Greg Lemmon: Office for Aboriginal and Torres Strait Islander Health, Australian Government Department of Health and Ageing

Stephanie Mackney: Office for Aboriginal and Torres Strait Islander Health, Australian Government Department of Health and Ageing

David Scrimgeour: Aboriginal Health Council of South Australia

Renee Williams: National Aboriginal Community Controlled Health Organisation

National Aboriginal Community Controlled Health Organisations

Vicki Krause: Communicable Disease Network Australia

Stephen Lambert: Queensland Department of Health

Paula Spokes: New South Wales Department of Health

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Quarterly report

National Notifiable Diseases Surveillance System, 1 October to 31 December 2014

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 57,995 notifications to the National Notifiable Diseases Surveillance System with a notification received date between 1 October to 31 December 2014 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

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

Table 1 *continued*: Reporting of notifiable diseases by jurisdiction

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

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

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2014, by date of diagnosis*

Disease	State or territory								Total 4th quarter 2014	Total 3rd quarter 2014	Total 4th quarter 2013	Last 5 years mean 4th quarter	Ratio	Year to date 2014	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Hepatitis B (newly acquired)†	0	5	1	12	0	0	9	6	33	36	44	50.2	0.7	173	208.8
Hepatitis B (unspecified)‡	24	593	31	251	94	15	432	151	1,591	1,708	1,730	1,657.0	1.0	6,562	6,815.4
Hepatitis C (newly acquired)†	0	4	0	0	7	2	40	31	84	97	117	106.0	0.8	387	424.8
Hepatitis C (unspecified)‡	38	847	48	642	115	56	513	279	2,538	2,666	2,595	2,526.6	1.0	10,259	10,387.4
Hepatitis D	0	7	1	1	0	0	0	2	11	12	13	9.2	1.2	50	38.8
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	1	1.4
Campylobacteriosis	125	NN	84	1,785	540	265	2,015	883	5,697	4,758	4,491	4,486.8	1.3	19,898	16,235.4
Cryptosporidiosis	2	123	18	87	54	6	107	69	466	414	574	417.2	1.1	2,403	2,981.0
Haemolytic uraemic syndrome	0	2	0	0	0	0	1	0	3	4	6	3.8	0.8	20	14.0
Hepatitis A	0	25	0	6	2	0	14	4	51	51	33	82.6	0.6	227	266.0
Hepatitis E	0	4	0	3	0	0	5	0	12	9	12	7.2	1.7	53	35.4
Listeriosis	1	5	2	4	0	2	4	2	20	18	15	20.6	1.0	80	80.4
STEC, VTEC§	0	3	0	6	9	0	2	0	20	22	29	31.0	0.6	116	118.6
Salmonellosis	59	1,092	113	1,323	294	71	948	303	4,203	2,745	3,390	2,887.2	1.5	16,370	11,545.8
Shigellosis	0	39	28	68	11	1	110	33	290	249	159	135.6	2.1	1,063	549.6
Typhoid	1	13	0	2	3	1	5	4	29	22	39	30.6	0.9	120	124.6
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	2	0.4	0.0	2	4.2
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.7

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2014, by date of diagnosis*

Disease	State or territory								Total 4th quarter 2014	Total 3rd quarter 2014	Total 4th quarter 2013	Last 5 years mean 4th quarter	Ratio	Year to date 2014	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection[‖,¶]	284	5,483	761	4,736	1,156	417	4,508	2,732	20,077	20,651	20,287	18,603.4	1.1	85,662	76,758.6
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.6
Gonococcal infection[¶]	28	1,167	380	621	147	20	885	511	3,759	3,653	3,622	2,998.0	1.3	15,703	11,900.4
Syphilis – congenital	0	0	1	0	0	0	0	0	1	4	1	0.6	1.7	5	4.2
Syphilis < 2 years duration[¶]	3	152	27	76	10	6	180	22	476	538	440	336.4	1.4	1,975	1,417.4
Syphilis > 2 years or unspecified duration[‡,¶]	9	124	34	57	23	6	161	18	432	453	404	337.6	1.3	1,822	1,422.8
Vaccine preventable diseases															
Diphtheria	0	0	0	1	0	0	0	0	1	1	1	0.2	5.0	2	1.2
Haemophilus influenzae type b	0	1	0	3	0	0	2	0	6	6	4	3.6	1.7	21	18.2
Influenza (laboratory confirmed)	113	1,124	112	1,742	1,396	59	1,123	620	6,289	53,141	6,457	3,465.6	1.8	67,854	34,522.8
Measles	0	1	4	18	1	0	8	4	36	57	87	37.2	1.0	340	145.0
Mumps	0	15	0	7	11	0	1	5	39	46	40	36.8	1.1	187	167.4
Pertussis	56	1,421	16	215	146	10	1,844	530	4,238	3,137	3,203	8,257.6	0.5	11,830	28,029.6
Pneumococcal disease (invasive)	3	123	9	41	23	11	88	53	351	589	341	360.0	1.0	1,562	1,690.6
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	3	0	0	0	0	2	0	5	3	6	5.4	0.9	17	38.2
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	1	0.2	0.0	0	0.6
Tetanus	0	0	0	1	0	0	0	1	2	0	0	0.8	2.5	3	3.8
Varicella zoster (chickenpox)	12	NN	13	70	91	15	251	114	566	540	689	570.4	1.0	2,082	1,959.4
Varicella zoster (shingles)	38	NN	67	11	504	69	344	385	1,418	1,328	1,403	1,034.4	1.4	5,531	3,877.8
Varicella zoster (unspecified)	34	NN	3	1,416	59	36	977	300	2,825	2,703	2,710	2,175.2	1.3	10,778	7,997.6
Vectorborne diseases															
Arbovirus infection (NEC)	1	22	7	56	0	0	7	8	101	90	459	432.4	0.2	739	2,154.8
Barmah Forest virus infection	0	10	0	18	2	0	13	5	48	17	11	14.2	3.4	106	56.6
Dengue virus infection	0	0	0	3	0	0	0	0	3	2	4	3.2	0.9	29	13.6
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	1	0	0.0	0.0	1	1.0
Kunjin virus infection**	0	0	0	0	0	0	0	0	0	0	2	0.6	0.0	1	1.6
Malaria	1	11	3	22	1	1	13	11	63	81	88	98.6	0.6	318	418.4
Murray Valley encephalitis virus infection**	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	4.4
Ross River virus infection	3	218	65	796	23	2	80	332	1,519	879	966	849.6	1.8	5,329	4,800.6

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2014, by date of diagnosis

Disease	State or territory								Total 4th quarter 2014	Total 3rd quarter 2014	Total 4th quarter 2013	Last 5 years mean 4th quarter	Ratio	Year to date 2014	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Brucellosis	0	0	0	3	0	0	0	0	3	5	6	7.2	0.4	17	27.0
Leptospirosis	0	4	1	3	0	0	1	1	10	17	20	20.6	0.5	85	137.8
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	5	0	2	0	0	6	0	13	9	15	23.8	0.5	39	66.6
Q fever	1	50	0	36	1	0	9	1	98	117	124	94.0	1.0	459	373.2
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.3	0.0	0	0.7
Other bacterial infections															
Legionellosis	2	20	0	22	9	1	15	44	113	111	124	95.8	1.2	425	370.4
Leprosy	0	0	0	0	1	0	0	1	2	0	4	2.8	0.7	9	9.4
Meningococcal infection††	0	14	0	7	10	0	10	5	46	57	34	48.4	1.0	169	220.4
Tuberculosis	10	120	9	47	13	4	133	31	367	351	325	378.6	1.0	1,337	1,326.8
Total	**848**	**12,850**	**1,838**	**14,220**	**4,756**	**1,076**	**14,866**	**7,501**	**57,955**	**101,398**	**55,127**			**272,221**	

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.

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

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

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

|| Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens. From 1 July 2013 case definition changed to exclude all 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).

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

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

NN Not notifiable

NEC Not elsewhere classified

Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

Table 3: Notification rates of diseases, 1 October to 31 December 2014, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)‡	0.0	0.3	1.7	1.0	0.0	0.0	0.6	1.0	0.6
Hepatitis B (unspecified)§	25.2	32.0	51.4	21.6	22.5	11.7	30.1	24.0	27.5
Hepatitis C (newly acquired)‡	0.0	0.2	0.0	0.0	1.7	1.6	2.8	4.9	1.5
Hepatitis C (unspecified)§	39.8	45.7	79.6	55.2	27.5	43.7	35.8	44.3	43.9
Hepatitis D	0.0	0.4	1.7	0.1	0.0	0.0	0.0	0.3	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	131.1	NN	139.3	153.4	129.3	206.6	140.4	140.1	144.9
Cryptosporidiosis	2.1	6.6	29.9	7.5	12.9	4.7	7.5	10.9	8.1
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Hepatitis A	0.0	1.3	0.0	0.5	0.5	0.0	1.0	0.6	0.9
Hepatitis E	0.0	0.2	0.0	0.3	0.0	0.0	0.3	0.0	0.2
Listeriosis	1.0	0.3	3.3	0.3	0.0	1.6	0.3	0.3	0.3
STEC,VTEC‖	0.0	0.2	0.0	0.5	2.2	0.0	0.1	0.0	0.3
Salmonellosis	61.9	59.0	187.4	113.7	70.4	55.3	66.1	48.1	72.7
Shigellosis	0.0	2.1	46.4	5.8	2.6	0.8	7.7	5.2	5.0
Typhoid fever	1.0	0.7	0.0	0.2	0.7	0.8	0.3	0.6	0.5
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Human pathogenic avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmitted infections									
Chlamydial infection¶,**	297.8	296.0	1,262.0	407.0	276.8	325.1	314.2	433.6	347.1
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	29.4	63.0	630.2	53.4	35.2	15.6	61.7	81.1	65.0
Syphilis – congenital	0.0	0.0	1.7	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years duration**	3.1	8.2	44.8	6.5	2.4	4.7	12.5	3.5	8.2
Syphilis > 2 years or unspecified duration§,**	9.4	6.7	56.4	4.9	5.5	4.7	11.2	2.9	7.5
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.3	0.0	0.0	0.1	0.0	0.1
Influenza (laboratory confirmed)	118.5	60.7	185.7	149.7	334.2	46.0	78.3	98.4	108.7
Measles	0.0	0.1	6.6	1.5	0.2	0.0	0.6	0.6	0.6
Mumps	0.0	0.8	0.0	0.6	2.6	0.0	0.1	0.8	0.7
Pertussis	58.7	76.7	26.5	18.5	35.0	7.8	128.5	84.1	73.3
Pneumococcal disease (invasive)	3.1	6.6	14.9	3.5	5.5	8.6	6.1	8.4	6.1
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.2	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.2	0.0

Table 3 *continued*: Notification rates of diseases, 1 October to 31 December 2014, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, *cont'd*									
Varicella zoster (chickenpox)	12.6	NN	21.6	6.0	21.8	11.7	17.5	18.1	14.4
Varicella zoster (shingles)	39.8	NN	111.1	0.9	120.7	53.8	24.0	61.1	36.1
Varicella zoster (unspecified)	35.7	NN	5.0	121.7	14.1	28.1	68.1	47.6	71.9
Vectorborne diseases									
Arbovirus infection (NEC)	1.0	1.2	11.6	4.8	0.0	0.0	0.5	1.3	1.7
Barmah Forest virus infection	0.0	0.5	0.0	1.5	0.5	0.0	0.9	0.8	0.8
Dengue virus infection	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection††	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.0	0.6	5.0	1.9	0.2	0.8	0.9	1.7	1.1
Murray Valley encephalitis virus infection††	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	3.1	11.8	107.8	68.4	5.5	1.6	5.6	52.7	26.3
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.2	1.7	0.3	0.0	0.0	0.1	0.2	0.2
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.3	0.0	0.2	0.0	0.0	0.4	0.0	0.2
Q fever	1.0	2.7	0.0	3.1	0.2	0.0	0.6	0.2	1.7
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	2.1	1.1	0.0	1.9	2.2	0.8	1.0	7.0	2.0
Leprosy	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.2	0.0
Meningococcal infection‡‡	0.0	0.8	0.0	0.6	2.4	0.0	0.7	0.8	0.8
Tuberculosis	10.5	6.5	14.9	4.0	3.1	3.1	9.3	4.9	6.3

* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.

† Rate per 100,000 of population. Annualisation Factor was 4.0

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

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

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

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens. From 1 July 2013 case definition changed to exclude all 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).

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

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

NEC Not elsewhere classified.

NN Not notifiable.

Australian childhood immunisation coverage, 1 January to 31 March cohort, assessed as at 30 June 2014

Brynley P Hull for the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

Introduction

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) provides commentary on the trends in ACIR data. For further information please contact NCIRS at: telephone +61 2 9845 1435, email: brynley.hull@health.nsw.gov.au

Tables 1, 2 and 3 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR) for all children.

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months, for 3-month birth cohorts of children assessed at the stated ages between 1 January and 31 March 2014 using ACIR data as at 30 June 2014. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, varicella, and meningococcal C conjugate vaccines.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, and 3 doses of 13-valent pneumococcal conjugate vaccine. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP Hib vaccine or 4 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

A full description of the basic methodology used can be found in *Commun Dis Intell* 1998;22(3):36–37.

Table 1: Percentage of children immunised at 12 months of age for the birth cohort 1 January to 31 March 2013, preliminary results, by disease and state or territory; assessment date 30 June 2014

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,359	24,758	982	15,787	5,014	1,490	18,925	8,683	76,998
Diphtheria, tetanus, pertussis (%)	94.1	91.0	90.7	92.1	90.9	91.7	92.5	91.8	91.8
Poliomyelitis (%)	94.0	90.9	90.7	92.1	90.9	91.7	92.5	91.8	91.7
Haemophilus influenzae type b (%)	93.7	90.8	90.8	92.0	90.9	91.5	92.3	91.7	91.6
Hepatitis B (%)	94.0	90.6	90.4	91.9	90.7	91.4	92.0	91.3	91.4
Pneumococcal	93.9	90.6	90.5	91.9	90.6	91.5	92.0	91.2	91.3
Fully immunised (%)	93.4	90.1	90.1	91.6	90.3	90.9	91.4	90.7	90.9
Change in fully immunised since last quarter (%)	+0.7	+1.0	–0.6	+0.9	+0.8	+3.3	+1.6	+1.2	+1.2

Table 2: Percentage of children immunised at 24 months of age for the birth cohort 1 January to 31 March 2012, preliminary results, by disease and state or territory; assessment date 30 June 2014*

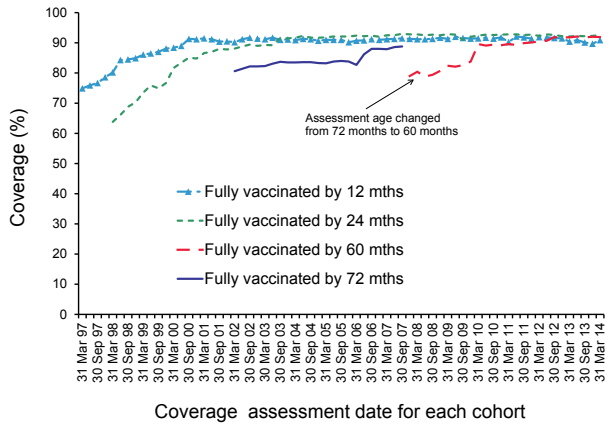
Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,387	25,281	961	15,992	4,957	1,479	18,951	8,655	77,663
Diphtheria, tetanus, pertussis (%)	96.3	94.7	95.6	95.2	95.1	95.1	95.5	94.5	95.0
Poliomyelitis (%)	96.3	94.7	95.7	95.2	95.0	95.1	95.5	94.4	95.0
Haemophilus influenzae type b (%)	95.5	93.8	94.9	94.4	93.9	94.2	94.4	93.3	94.1
Measles, mumps, rubella (%)	95.7	94.3	95.5	94.9	94.6	94.5	94.8	93.4	94.5
Hepatitis B (%)	95.5	94.3	95.5	94.9	94.5	94.9	94.9	93.8	94.6
Fully immunised (%)	93.8	92.1	94.2	93.6	92.4	92.8	92.9	91.5	92.6
Change in fully immunised since last quarter (%)	+0.9	+0.3	+1.5	+0.1	+0.4	+0.2	+0.5	+0.4	+0.3

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2013;37(4):E435.

Table 3: Percentage of children immunised at 60 months of age for the birth cohort 1 January to 31 March 2009, preliminary results, by disease and state or territory; assessment date 30 June 2014

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,209	24,617	915	16,272	5,118	1,563	18,313	8,400	76,407
Diphtheria, tetanus, pertussis (%)	95.3	92.8	92.8	92.7	91.2	93.0	92.6	91.0	92.5
Poliomyelitis (%)	95.3	92.7	92.5	92.7	91.1	92.8	92.6	91.0	92.4
Measles, mumps, rubella (%)	95.0	92.7	92.8	92.7	91.4	93.0	92.5	90.8	92.4
Fully immunised (%)	94.8	92.3	92.0	92.3	90.5	92.3	92.0	90.3	91.9
Change in fully immunised since last quarter (%)	+2.6	+0.1	+1.1	–0.5	–0.5	–0.3	–0.6	+1.4	–0.1

Figure: Trends in vaccination coverage, Australia, 1997 to 31 March 2014 2014, by age cohorts



Australian childhood immunisation coverage, 1 April to 30 June cohort, assessed as at 30 September 2014

Brynley P Hull for the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

Introduction

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The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months, for 3-month birth cohorts of children assessed at the stated ages between 1 April and 30 June 2014 using ACIR data as at 30 September 2014. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, varicella, and meningococcal C conjugate vaccines, and is outlined in more detail below.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, and 3 doses of 13-valent pneumococcal conjugate vaccine. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP Hib vaccine or 4 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

A full description of the basic methodology used can be found in *Commun Dis Intell* 1998;22(3):36–37.

Results

The percentage of children 'fully immunised' by 12 months of age for Australia increased from the previous quarter by 0.6 of a percentage point to 91.5% (Table 1). Most jurisdictions experienced small increases in the percentage of children 'fully immunised' by 12 months of age. For individual vaccines due by 12 months of age a majority of jurisdictions achieved coverage greater than 91%.

The percentage of children 'fully immunised' by 24 months of age for Australia increased marginally from the previous quarter by 0.2 of a percent-

Table 1: Percentage of children immunised at 12 months of age for the birth cohort 1 April to 30 June 2013, preliminary results, by disease and state or territory; assessment date 30 September 2014

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,379	24,627	1,037	15,966	4,975	1,485	18,797	8,536	76,802
Diphtheria, tetanus, pertussis (%)	94.0	92.0	88.1	92.7	92.0	92.3	92.7	92.7	92.4
Poliomyelitis (%)	94.0	92.0	88.1	92.7	92.0	92.3	92.6	92.7	92.4
Haemophilus influenzae type b (%)	93.6	91.9	88.0	92.6	91.9	92.3	92.4	92.4	92.2
Hepatitis B (%)	93.2	91.7	88.1	92.4	91.8	92.0	92.2	92.2	92.0
Pneumococcal	93.5	91.7	88.5	92.5	91.8	92.1	92.3	92.2	92.1
Fully immunised (%)	92.5	91.1	87.7	92.1	91.4	91.7	91.7	91.7	91.5
Change in fully immunised since last quarter (%)	–0.9	+1.0	–2.4	+0.5	+1.1	+0.8	+0.3	+1.0	+0.6

Table 2: Percentage of children immunised at 24 months of age for the birth cohort 1 April to 30 June 2012, preliminary results, by disease and state or territory; assessment date 30 September 2014*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,298	25,283	967	15,987	4,964	1,440	18,747	8,565	77,251
Diphtheria, tetanus, pertussis (%)	95.5	95.0	95.6	95.1	94.9	95.2	95.6	94.8	95.2
Poliomyelitis (%)	95.5	95.0	95.8	95.1	94.9	95.2	95.6	94.8	95.2
Haemophilus influenzae type b (%)	94.5	94.1	95.8	94.5	93.7	94.4	94.6	93.7	94.3
Measles, mumps, rubella (%)	94.9	94.6	96.1	95.0	94.2	94.7	95.1	93.9	94.7
Hepatitis B (%)	95.2	94.6	95.6	94.7	94.5	94.9	95.1	94.1	94.7
Fully immunised (%)	93.7	92.4	94.7	93.3	92.3	93.0	93.3	92.0	92.8
Change in fully immunised since last quarter (%)	–0.1	+0.3	+0.5	–0.3	–0.1	+0.2	+0.4	+0.5	+0.2

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2014;38(1):E86.

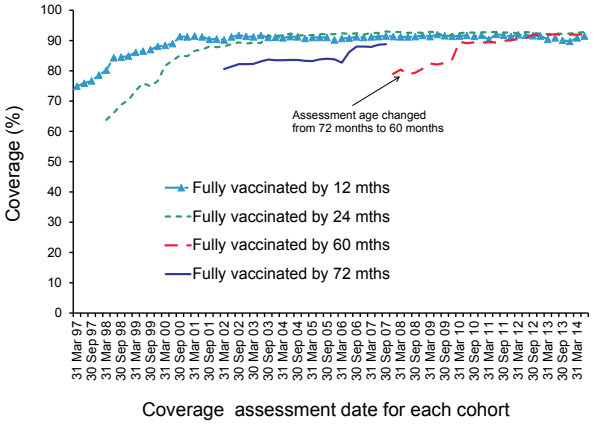
Table 3: Percentage of children immunised at 60 months of age for the birth cohort 1 April to 30 June 2009, preliminary results, by disease and state or territory; assessment date 30 September 2014

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,333	24,716	945	16,475	5,006	1,629	18,425	8,519	77,048
Diphtheria, tetanus, pertussis (%)	93.8	93.0	92.3	92.9	92.2	93.7	93.2	90.7	92.7
Poliomyelitis (%)	93.6	93.0	92.3	92.9	92.2	93.7	93.2	90.7	92.7
Measles, mumps, rubella (%)	93.5	93.1	92.6	92.8	91.9	93.5	93.1	90.5	92.7
Fully immunised (%)	93.0	92.6	91.6	92.5	91.6	92.9	92.6	90.1	92.2
Change in fully immunised since last quarter (%)	–1.8	+0.3	–0.4	+0.2	+1.1	+0.6	+0.6	–0.2	+0.3

age point to 92.8 (Table 2). There were also only marginal changes in fully immunised coverage at 24 months of age in all jurisdictions. Coverage for individual vaccines due by 24 months remained high in all jurisdictions.

The percentage of children 'fully immunised' by 60 months of age for Australia increased marginally from the previous quarter by 0.3 of a percentage point to 92.0% (Table 3). This maintains the improvement in coverage for this age milestone. There were also only marginal changes in fully immunised coverage at 24 months of age in all jurisdictions. Coverage for individual vaccines due by 60 months remained greater than 90% in all jurisdictions.

Figure: Trends in vaccination coverage, Australia, 1997 to 30 June 2014, by age cohorts



Australian Sentinel Practices Research Network, 1 October to 31 December 2013

Monique B-N Chilver, Daniel Blakeley, Nigel P Stocks for the Australian Sentinel Practices Research Network

Introduction

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is funded by the Australian Government Department of Health, owned and operated by the Royal Australian College of General Practitioners and directed through the Discipline of General Practice at the University of Adelaide.

The network consists of general practitioners who report presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Electronic, web-based data collection was established in 2006.

Since 2010, ASPREN GPs have been collecting nasal swab samples for laboratory testing, allowing for viral testing of 25% of ILI patients for a range of respiratory viruses including influenza A, influenza B and A(H1N1)pdm09.

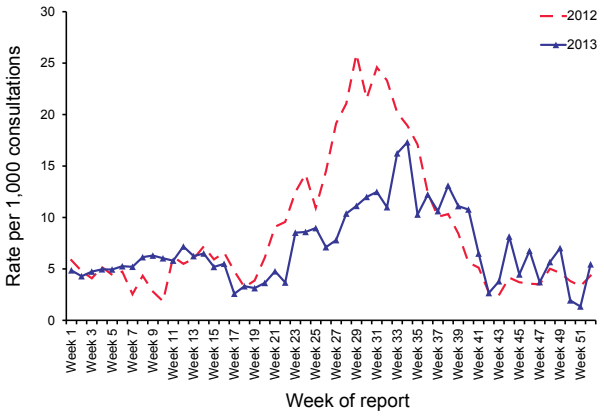
The list of conditions is reviewed annually by the ASPREN management committee. In 2013, 4 conditions are being monitored. They include ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, published in *Commun Dis Intell* 2015;39(1):E180.

Results

ILI rates reported from 1 October to 31 December 2013 averaged 5 cases per 1,000 consultations (range 1–11 cases per 1,000 consultations)This was higher compared with rates in the same reporting period in 2012, which averaged 4 cases per 1,000 consultations (range 2–6 cases per 1,000 consultations, Figure 1).

The ASPREN ILI swab testing program continued in 2013 with 2,397 tests being undertaken from 1 January to 31 December. The most commonly reported virus during this reporting period was rhinovirus (12.3% of all swabs performed, Figure 2), with the 2nd most common virus being influenza A (12.1% of all swabs performed).

Figure 1: Consultation rates for influenza-like illness, ASPREN, 2012 and 1 January to 31 December 2013, by week of report



From the beginning of 2013 to the end of week 52, 446 cases of influenza were detected, with 291 of these typed as influenza A (12.1% of all swabs performed) and the remaining 155 being influenza B (6.5% of all swabs performed) (Figure 2).

During this reporting period, consultation rates for gastroenteritis averaged 6 cases per 1,000 consultations (range 4–9 cases per 1,000 consultations, Figure 3). This was higher compared with rates in the same reporting period in 2012 where the average was 5 cases per 1,000 consultations (range 3–6 cases per 1,000).

Varicella infections were reported at a lower rate for the 4th quarter of 2013 compared with the same period in 2012. From 1 October to 31 December 2013, recorded rates for chickenpox averaged 0.22 cases per 1,000 consultations (range 0.05–0.48 cases per 1,000 consultations, Figure 4).

In the 4th quarter of 2013, reported rates for shingles averaged 1.02 cases per 1,000 consultations (range 0.53–1.57 cases per 1,000 consultations, Figure 5), higher compared with the same reporting period in 2012 where the average shingles rate was 0.87 case per 1,000 consultations (range 0.68–1.08 cases per 1,000 consultations).

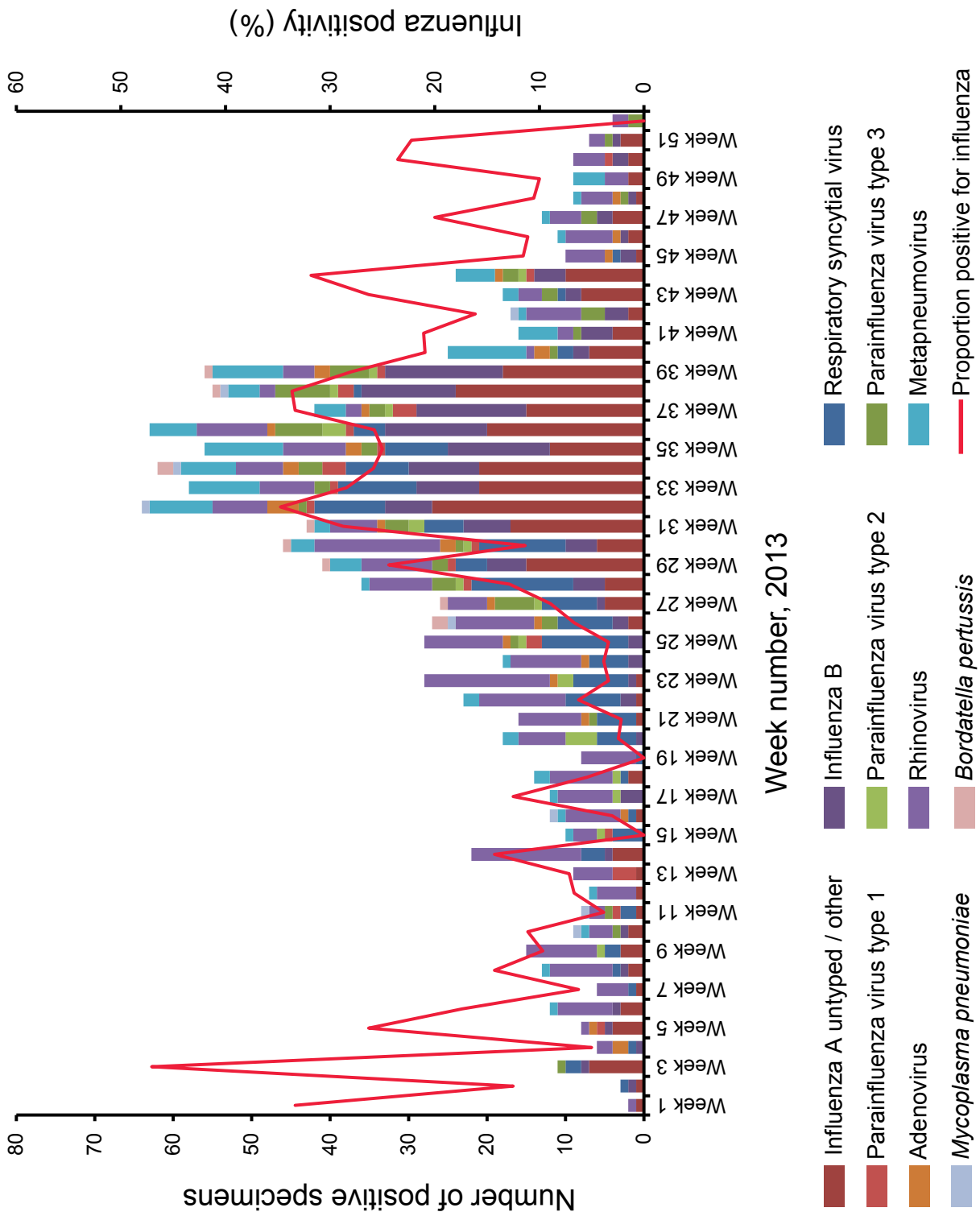


Figure 2: Influenza-like illness swab testing results, ASPREN, 2012 and 1 January to 31 December 2013, by week of report

Figure 3: Consultation rates for gastroenteritis, ASPREN, 2012 and 1 January to 31 December 2013, by week of report

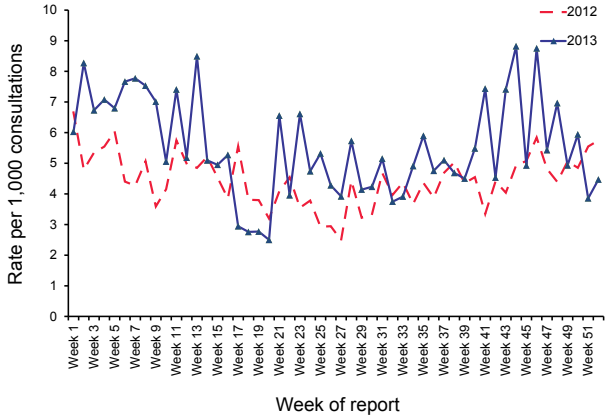


Figure 4: Consultation rates for chickenpox, ASPREN, 2012 and 1 January to 31 December 2013, by week of report

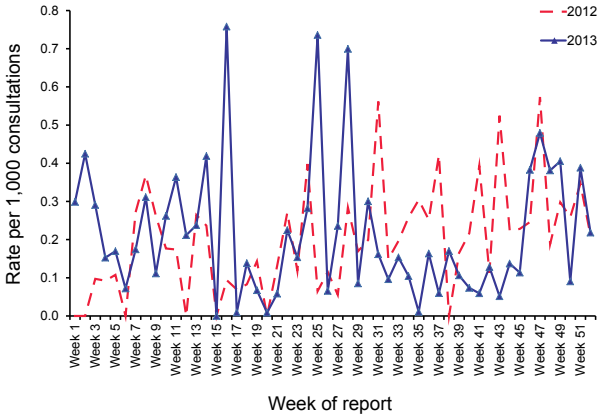
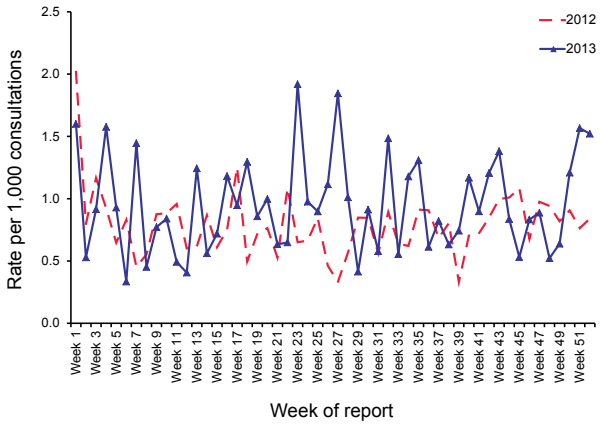


Figure 5: Consultation rates for shingles, ASPREN, 2012 and 1 January to 31 December 2013, by week of report



Invasive pneumococcal disease surveillance Australia, 1 July to 30 September 2014

Rachel de Kluyver for the Enhanced Invasive Pneumococcal Disease Surveillance Working Group

- There were 588 cases of invasive pneumococcal disease reported to the National Notifiable Diseases Surveillance System in the 3rd quarter of 2014, bringing the year-to-date total to 1,211 cases.
- The total number of cases in the year-to-date was similar to the number of cases reported for the same period in 2013.
- Aboriginal and Torres Strait Islander peoples accounted for 13% of all cases with a reported Indigenous status.

Introduction

Invasive pneumococcal disease (IPD) is caused by the bacterium *Streptococcus pneumoniae* and results in conditions such as pneumonia, bacteraemia and meningitis. There are currently more than 90 serotypes recognised worldwide, approximately half of which are found in Australia where IPD has been a nationally notifiable disease since 2001. This quarterly report documents trends in notified cases of IPD occurring in Australia in the 3rd quarter of 2014 (1 July to 30 September 2014). In this quarterly report, 3 age groups have been selected for focused analyses. These age groups align with groups that carry the greatest burden of disease and against which the National Immunisation Program is targeted. The data in this report are provisional and subject to change as laboratory results and additional case information become available.

Detailed IPD surveillance methodology is described each year in the 1st quarter report and in the annual reports published in *Communicable Diseases Intelligence*.

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk and older Australians.*

Results

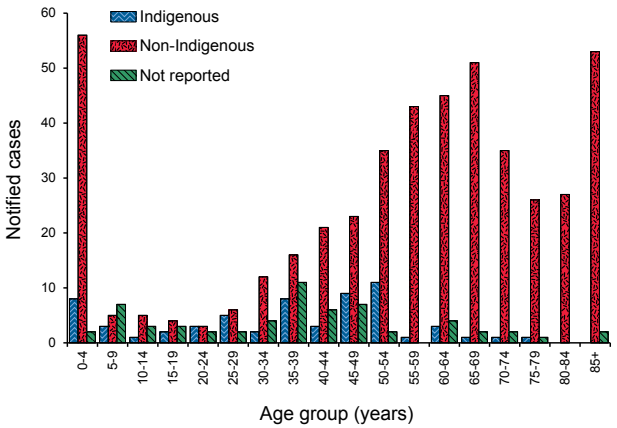
There were 588 cases of IPD reported to the National Notifiable Diseases Surveillance System in the 3rd quarter of 2014, bringing the year-to-date total to 1,211 cases. Similar to many infectious diseases, the number of cases of IPD is highest in the winter months and this is particularly evident for cases aged greater than 5 years. This seasonal trend was observed in all analyses included in this report. For the year to 30 September, the total number of cases was similar to the number of cases reported for the same period in 2013 (n=1,208) (Table).

Overall, notified cases were highest in the under 5 years age group followed by the over 85 years age group (Figure 1). In cases reported as Indigenous, the most prevalent age group was the 50–54 years age group (n=11) followed by the 45–49 years age group (n=9).

Data completeness

During the reporting period, Indigenous status was reported for 90% (n=528) of cases and serotype information was available for 96% (n=567) of all cases reported (Table).

Figure 1: Notifications of invasive pneumococcal disease, Australia, 1 July to 30 September 2014, by Indigenous status and age group



* The 7-valent pneumococcal conjugate vaccine (7vPCV) was added to the National Immunisation Program (NIP) schedule for Indigenous and medically at-risk children in 2001 and for all children up to 2 years of age in 2005. The 13-valent pneumococcal conjugate vaccine (13vPCV) replaced the 7vPCV in the childhood immunisation program from July 2011. The 23-valent pneumococcal polysaccharide vaccine (23vPPV) was added to the NIP schedule for Aboriginal and Torres Strait Islander peoples aged 50 years or over in 1999 and for non-Indigenous Australians aged 65 years or over from January 2005.

Table: Notified cases of invasive pneumococcal disease, Australia, 1 July to 30 September 2014, by Indigenous status, serotype completeness and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total 3rd qrt 2014	2nd qrt 2014	3rd qrt 2013	Year to date 2014
Indigenous	1	11	14	13	3	1	0	19	62	45	64	107
Non-Indigenous	4	158	2	86	61	11	91	53	466	315	422	781
Not stated/ unknown	0	16	0	2	0	0	42	0	60	48	68	108
Total	5	185	16	101	64	12	133	72	588	408	554	1,211
Indigenous status completeness* (%)	100	91	100	98	100	100	68	100	90			–
Serotype completeness† (%)	100	95	100	95	94	83	99	100	96			–

* Indigenous status completeness is defined as the reporting of a known Indigenous status, excluding the reporting of not stated or unknown Indigenous status.

† Serotype completeness is the proportion of all cases of invasive pneumococcal disease that were reported with a serotype or reported as non-typeable. Serotype incompleteness may include when no isolate was available as diagnosis was by polymerase chain reaction and no molecular typing was attempted or was not possible due to insufficient genetic material; the isolate was not referred to the reference laboratory or was not viable; typing was pending at the time of reporting; or no serotype was reported by the notifying jurisdiction to the National Notifiable Diseases Surveillance System.

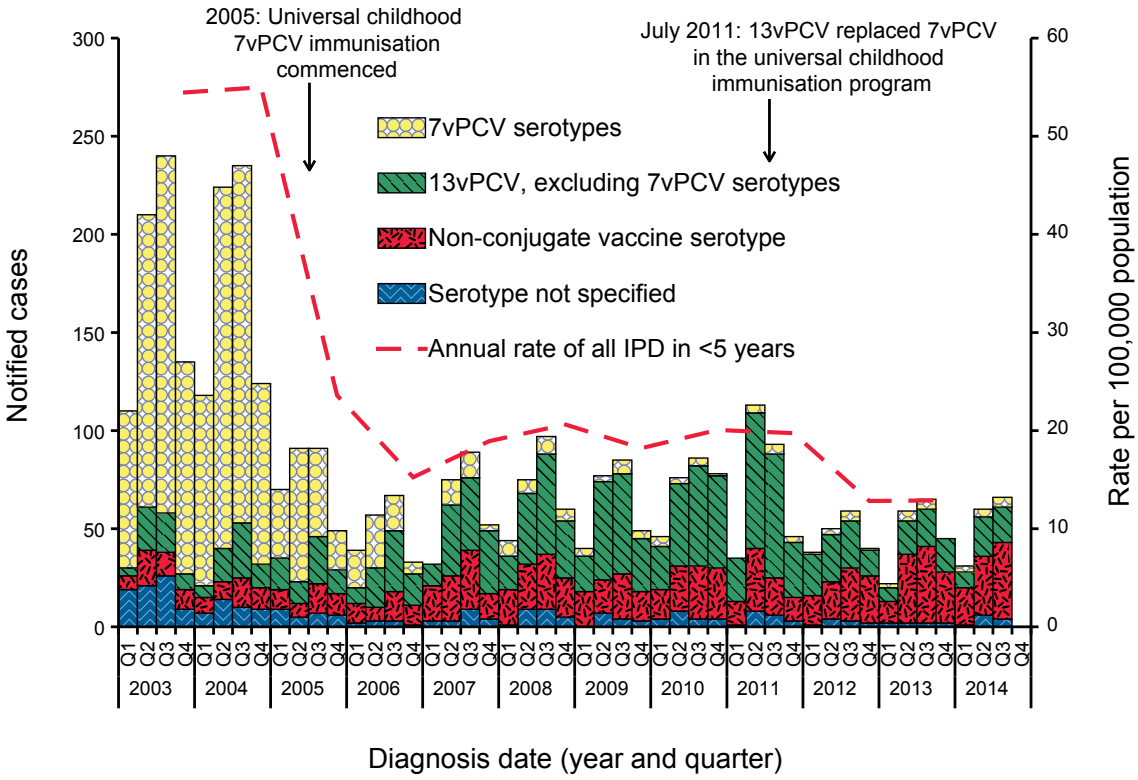
Invasive pneumococcal disease in children aged less than 5 years

In the 3rd quarter of 2014, 11% (n=66) of all notified cases were aged less than 5 years, which was similar to the number reported during the same period of 2013 (n=65).

The majority (94%, n=62) of cases aged less than 5 years were reported with serotype information. Of these, 37% (n=23) were reported with a serotype included in the 7vPCV or the 13vPCV (Figure 2).

Over the period 2007 to 2011 notified cases aged less than 5 years with disease caused by the

Figure 2: Notifications (2003 to 30 September 2014) and annual rates (2003 to 2013) of all invasive pneumococcal disease in those aged less than 5 years, Australia, by vaccine serotype group



6 additional serotypes (1, 3, 5, 6A, 7F and 19A) that would be covered by the 13vPCV, increased steadily, particularly those caused by serotype 19A (Figure 3). However, cases of serotype 19A have decreased since the 4th quarter of 2011, reflecting the introduction of the 13vPCV into the universal childhood immunisation program in mid-2011. In the 3rd quarter of 2014, there were 11 cases aged less than 5 years with disease due to serotype 19A and 6 cases due to serotype 3. In this age group, 1 case was reported with disease caused by serotype 7F, a previously common serotype.

Invasive pneumococcal disease in Indigenous Australians aged 50 years or over

In the 3rd quarter of 2014, 3% (n=18) of notified cases were reported as Indigenous Australians aged 50 years or over (Figure 4). This was slightly lower than the number reported during the same period in 2013 (n=21).

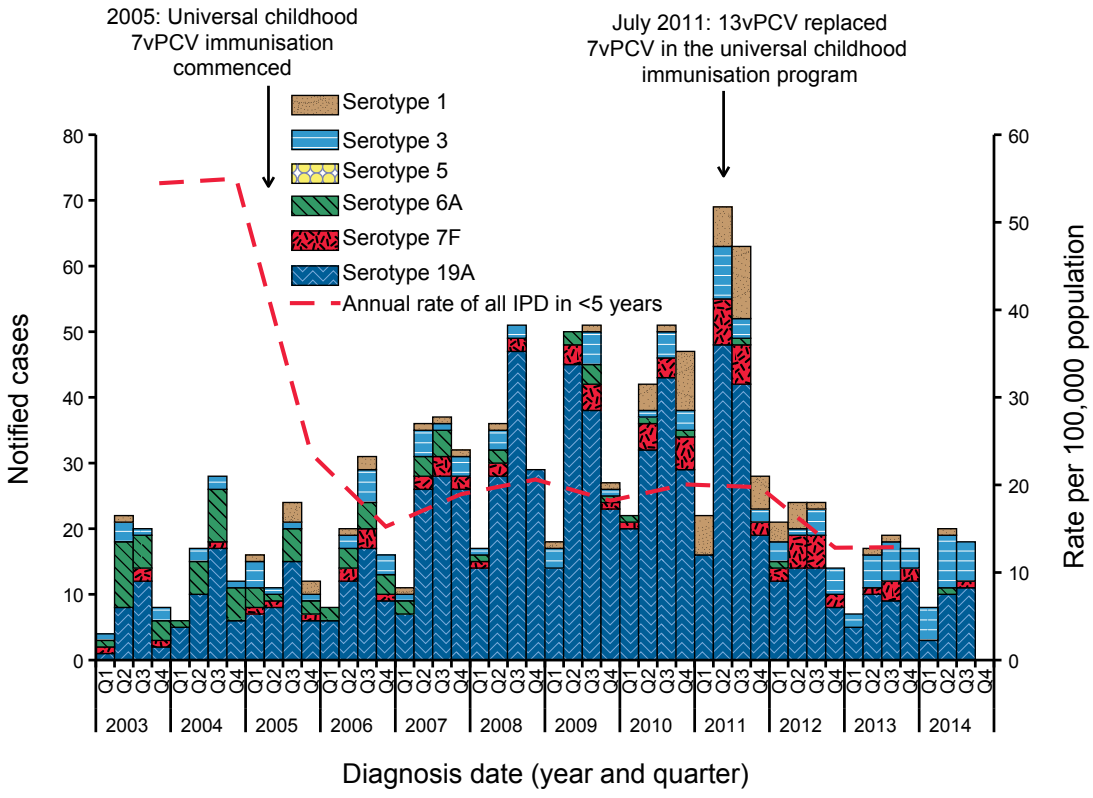
All but one of the cases notified in the 3rd quarter of 2014 were reported with serotype information. Of these, approximately 70% (n=12) were reported with disease due to serotypes targeted by the 23vPPV. The remaining cases reported disease due to a non-vaccine serotype (n=5).

Invasive pneumococcal disease in non-Indigenous Australians aged 65 years or over

In the 3rd quarter of 2014, 34% (n=198) of notified cases were reported as non-Indigenous and aged 65 years or over. This was similar to the number reported during the same period of 2013 (n=184) (Figure 5).

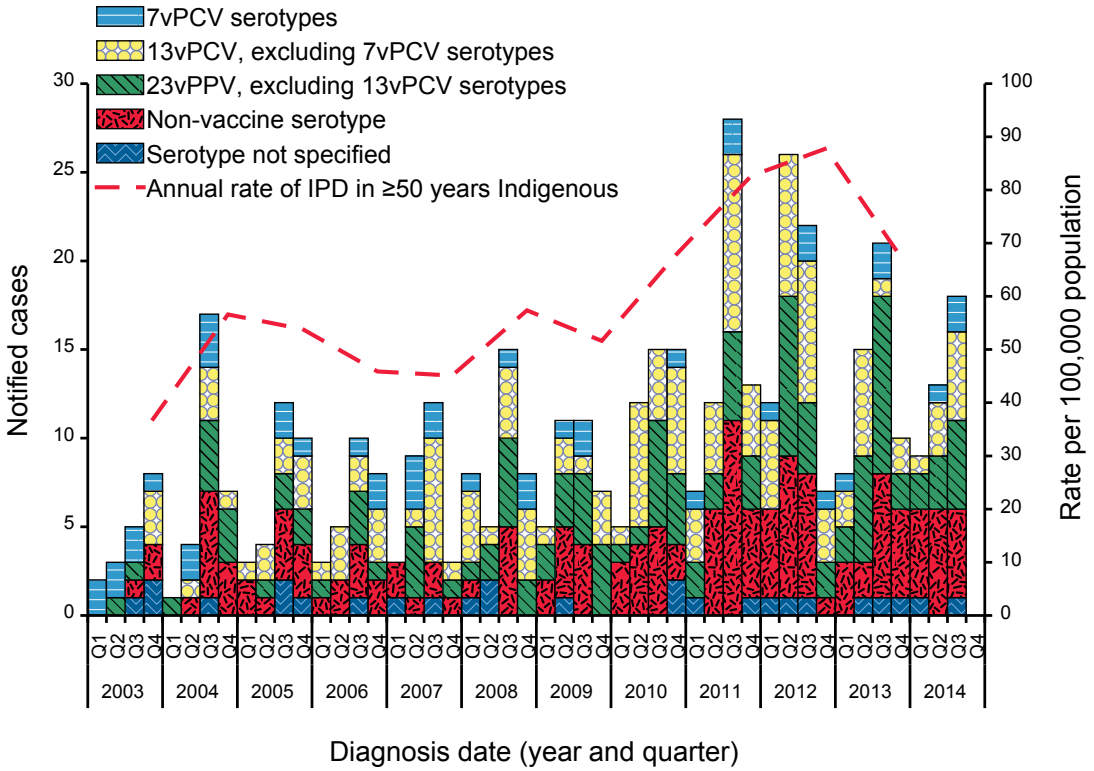
The majority (97%, n=193) of cases reported in this quarter were reported with serotype information. Of these cases, 60% (n=116) were reported with a serotype targeted by the 23vPPV. While the burden of disease in this age group has remained relatively stable, the profile of serotypes causing disease has changed over time. Disease due to serotypes targeted by the 7vPCV has reduced substantially in this age group, which is likely to be due to herd immunity impacts from the childhood immunisation program.

Figure 3: Notifications of invasive pneumococcal disease caused by serotypes targeted by the 13-valent pneumococcal conjugate vaccine* and annual rates of all invasive pneumococcal disease aged less than 5 years, Australia, 2003 to 2013



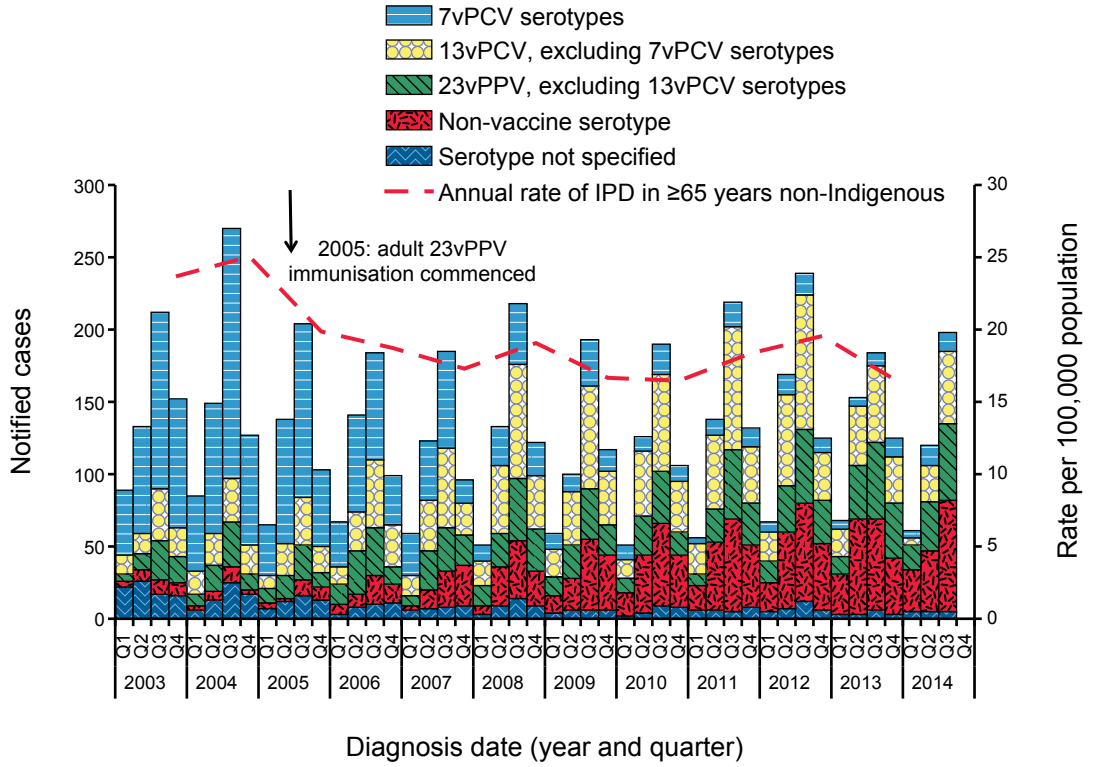
* Excluding those targeted by 7-valent pneumococcal conjugate vaccine from 2003 to 30 September 2014.

Figure 4: Notifications (2003 to 30 September 2014) and annual rates of all invasive pneumococcal disease (2003 to 2013) in Indigenous Australians aged 50 years or over, Australia, by vaccine serotype group*



* In 1999 23vPPV immunisation commenced for Indigenous Australians aged 50 years or over.

Figure 5: Notifications (2003 to 30 September 2014) and annual rates of all invasive pneumococcal disease (2003 to 2013) in non-Indigenous Australians aged 65 years or over, Australia, by vaccine serotype group



Mortality due to invasive pneumococcal disease

Nationally, there were 48 deaths attributed to 19 different IPD serotypes during this reporting period. No deaths were reported in the under 5 years age group.

Conclusion

The number of notified cases of IPD in the 3rd quarter of 2014 was an increase on the previous quarter, which was consistent with the seasonal increase of IPD during winter. To 30 September, the total number of cases in 2014 was almost identical to the number of cases reported for the same period in 2013. Nationally, the pattern of disease has not changed from the 2nd quarter of 2013. Specifically, the decline in disease due to the serotypes targeted by the 13vPCV has been maintained since the 13vPCV replaced the 7vPCV in the childhood immunisation program from July 2011. Similarly, IPD associated with non-vaccine serotypes has remained unchanged in all groups targeted for IPD vaccination. Disease in non-Indigenous Australians aged 65 years or over has remained relatively stable but the profile of serotypes causing disease has diversified.

Acknowledgements

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Surveillance summaries

Surveillance systems reported in *Communicable Diseases Intelligence*, 2015

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

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

- detecting outbreaks and identifying national trends;
- providing guidance for policy development and resource allocation at the national level;
- monitoring the need for and impact of national disease control programs;
- coordinating a response to national or multi-jurisdictional 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; and
- supporting quarantine activities, which are the responsibility of the Australian government.

State and territory health departments collect notifications of communicable diseases under their public health legislation. In September 2007, the *National Health Security Act 2007* (*National Health Security Act , No 174*) received royal assent. This Act provides the 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, which specifies the diseases about which personal information can be provided. The National Health Security Agreement, 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. States and territories voluntarily forward de-identified data on a nationally agreed group of communicable diseases to the Department of Health (Health) for the purposes of national communicable disease surveillance.

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

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

Other surveillance schemes for which CDI publishes annual reports include tuberculosis notifications (*Commun Dis Intell* 2014;38(1):E3–E15), the Australian Mycobacterium Reference Laboratory Network (*Commun Dis Intell* 2014;38(4):E356–E368), and the Australian Rotavirus Surveillance Program (*Commun Dis Intell* 2014;38(1):E29–E35).

Arbovirus and malaria surveillance

The National Arbovirus and Malaria Advisory Committee (NAMAC) collates data and reports on the epidemiology of mosquito-borne diseases of public health importance in Australia by financial year (which represents the cycle of mosquito-borne disease activity in most parts of Australia). The reports include data from the National Notifiable Diseases Surveillance System (NNDSS) on notified cases of disease caused by the alphaviruses: Barmah Forest virus, chikungunya virus and Ross River virus; the flaviviruses: dengue virus, Murray Valley encephalitis virus (MVEV), the Kunjin strain of West Nile virus, Japanese encephalitis virus and yellow fever virus; and the protzoan infection, malaria. Both locally acquired and overseas acquired cases are described. Vector, climate and sentinel animal surveillance measures for arboviruses (in particular for MVEV) conducted

by states and territories, and also at the border are described. Sentinel chicken, mosquito surveillance, viral detection in mosquitoes and climate modelling are used to provide early warning of arboviral disease activity in Australia. Sentinel chicken programs for the detection of flavivirus activity are conducted in most states at risk of arboviral transmission. Other surveillance activities to detect the presence of arboviruses in mosquitoes or mosquito saliva or for surveying mosquito abundance included honey-baited trap surveillance, surveys of household containers that may provide suitable habitat for the dengue vector, *Aedes aegypti,* and carbon dioxide baited traps.

NAMAC provides expert technical advice on arboviruses and malaria to the Australian Health Protection Principal Committee through the Communicable Diseases Network Australia (CDNA). Members of the Committee have expertise in virus and disease surveillance, epidemiology, virology, vector ecology, vector control and quarantine, and represent agencies with a substantial interest in this area. NAMAC makes recommendations about surveillance and reporting systems, strategic approaches for disease and vector management and control, laboratory support, development of national guidelines and response plans and research priorities. NAMAC assists in the detection, management and control of real or potential outbreaks of arboviruses or malaria and provides advice on the risk of these diseases or exotic vectors being imported from overseas. NAMAC members participate in outbreak management teams as required.

Further details are provided in the NAMAC annual report (*Commun Dis Intell* 2014;38(2):E122–E142).

Australian Childhood Immunisation Register

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

From the data finally entered onto the ACIR, Medicare Australia provides regular quarterly coverage reports at the national and state level. Coverage for these reports is calculated using the cohort method previously described (*Commun Dis Intell* 1998;22:36–37). With this method, a cohort of children is defined by date of birth in 3-month groups. This birth cohort has the immunisation status of its members assessed at the 3 key milestones of 12 months, 24 months and 60 months of age. Analysis of coverage is undertaken 3 months after the due date for completion of each milestone, so that time is available for processing notifications and the impact on coverage estimates of delayed notification to the ACIR is minimised. Only children enrolled with Medicare are included, in order to minimise inaccuracies in coverage estimates due to duplicate records.

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

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

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

Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) is a continuing program to monitor antimicrobial resistance in *Neisseria gonorrhoeae* and includes the reference laboratories in all states and territories. These laboratories report data on sensitivity to an agreed core group of antimicrobial agents on a quarterly basis and provide an expanded analysis as

an annual report in CDI *(Commun Dis Intell* 2013;37(3):E233–E239). The antibiotics that are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. A major purpose of the AGSP is to help define standard protocols for antibiotic treatment of gonococcal infection. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates, it is usual to reconsider the inclusion of that agent in current treatment schedules. Additional data are also provided on other antibiotics from time to time. At present, all laboratories also test isolates for the presence of high level resistance to the tetracyclines and intermittent surveys of azithromycin resistance are conducted. Comparability of data is achieved by means of a standardised system of minimal inhibitory concentration (MIC) testing and a program-specific quality assurance process.

Australian Meningococcal Surveillance Programme

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

Data are reported annually and quarterly in CDI. Data in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup where known. A full analysis of laboratory-confirmed cases of IMD, including phenotyping and antibiotic susceptibility data are published annually (*Commun Dis Intell* 2014;38(4):E301–E308).

Australian National Creutzfeldt-Jakob Disease Registry

Surveillance for Creutzfeldt-Jakob disease (CJD) in Australia is conducted through the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR). CJD is listed as a notifiable disease in all Australian states and territories. The ANCJDR is under contract to the Commonwealth to identify and investigate all suspect cases of transmissible spongiform encephalopathy in Australia. An annual update is published in CDI *(Commun Dis Intell* 2014;38(4):E348–E355).

Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit (APSU) is an active surveillance mechanism for prospective, national identification and study of children aged <15 years, newly diagnosed with uncommon conditions including rare infectious and vaccine preventable diseases, genetic disorders, child mental health problems, rare injuries and other rare chronic childhood conditions. Up to 16 different conditions are studied simultaneously. The APSU relies on monthly reporting by ~1,400 paediatricians and other child health clinicians and over 85% of clinicians respond via e-mail. Clinicians reporting cases are asked to provide details about demographics, diagnosis, treatments and short-term outcomes. All negative and positive reports are logged into a database and the report card return rate has been maintained at over 90% for the last 20 years. The APSU, together with the National Centre for Immunisation Research and Surveillance jointly provide coordination for the Paediatric Active Enhanced Disease Surveillance (PAEDS). PAEDS is currently operational in 5 paediatric referral centres in 5 states and collects detailed information on relevant admitted cases (www.paeds.edu.au).

Communicable diseases currently under surveillance include: acute flaccid paralysis (to identify potential cases of poliovirus infection); congenital cytomegalovirus infection; congenital rubella; perinatal exposure to HIV, and HIV infection; neonatal herpes simplex virus infection; neonatal varicella, congenital varicella, severe complications of varicella and juvenile onset recurrent respiratory papillomatosis. After demonstrating feasibility in 2007, the APSU has conducted seasonal surveillance for severe complications of influenza each year. In 2009 APSU contributed to the national surveillance effort during the influenza A(H1N1) pdm09 pandemic.

The activities of the APSU are funded in part by the Australian Government Department of Health, and the NHMRC Practitioner Fellowship No: 1021480 (E Elliott). The Faculty of Medicine, The University of Sydney, and the Royal Australasian College of Physicians, Division of Paediatrics and Child Health, and the Kids Research Institute, Sydney Children's Hospitals Network provide in-kind support. APSU publishes an annual report *(Commun Dis Intell* 2014;38(4):E343–E347). For further information please contact the APSU Director, Professor Elizabeth Elliott on telephone: +61 2 9845 3005, facsimile +61 2 9845 3082 or email: apsu@chw.edu.au; Internet: http://www.apsu.org.au

Australian Sentinel Practice Research Network

The Discipline of General Practice at the University of Adelaide operates the Australian Sentinel Practices Research Network (ASPREN). ASPREN is a national network of general practitioners who report presentations of defined medical conditions each week. The main aims of ASPREN are to provide an indicator of disease burden and distribution in the community and to be an early indicator of pandemic influenza.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2014, 4 conditions are being monitored; all of which are related to communicable diseases. These are influenza like illness (ILI), gastroenteritis, chickenpox and shingles.

Laboratory testing of ILI cases was implemented in 2010, allowing for viral testing of 25% of ILI patients for a range of respiratory viruses including influenza A, influenza B and A(H1N1)pdm09.

There are currently 210 general practitioners registered with the network from all jurisdictions. Fifty-eight per cent of these are in metropolitan areas, 32% in rural and 10% in remote areas of Australia. Approximately 15,000 consultations are recorded by these general practitioners each week.

Data for communicable diseases are published in CDI each quarter. Data are presented in graphical format with the rate reported as the number of conditions per 1,000 consultations per week. The conditions are defined as:

Influenza-like illness – record once only per patient

Must have the following: fever, cough and fatigue.

Gastroenteritis – record once only per patient

Three or more loose stools, and/or 2 vomits in a 24 hour period excluding cases who have a known cause, for example bowel disease, alcohol, pregnancy.

Chickenpox – record once only per patient

An acute, generalised viral disease with a sudden onset of slight fever, mild constitutional symptoms and a skin eruption which is maculopapular for a few hours, vesicular for three to 4 days and leaves a granular scab.

Shingles – record once only per patient

Recurrence, recrudescence or re-activation of chickenpox infection. Vesicles with any erythematous base restricted to skin areas supplied by sensory nerves of a single or associated group of dorsal root ganglia. Lesions may appear in crops in irregular fashion along nerve pathways, are usually unilateral, deeper seated and more closely aggregated than those of chickenpox.

Note: Those conditions which show 'record once only per patient' are to have each occurrence of the condition recorded on 1 occasion no matter how many patient contacts are made for this episode of illness. If the condition recurs at a later date it can be recorded/counted again.

HIV surveillance

National surveillance for newly diagnosed HIV infection is coordinated by the Kirby Institute, in collaboration with state and territory health authorities, the Australian Government Department of Health, the Australian Institute of Health and Welfare and other collaborating networks in surveillance for HIV, viral hepatitis and sexually transmissible infections.

Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, either by the diagnosing laboratory (Australian Capital Territory and Tasmania), by doctor notification (Western Australia) or by a combination of laboratory and doctor sources (New South Wales, Northern Territory, Queensland, South Australia and Victoria). Diagnoses of HIV infection are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Currently, 2 tables presenting the number of new diagnoses of HIV infection in Australia in the most recent quarter and cumulatively are published in CDI. The tabulations are based on data available 3 months after the end of the reporting period, to allow for reporting delay and to incorporate newly available information.

An annual surveillance report, *HIV, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report* has been published by the Kirby Institute since 1997. The Annual Surveillance Report (http://www.kirby.unsw.edu.au) provides a comprehensive analysis and interpretation of surveillance data on HIV, viral hepatitis and sexually transmissible infections in

Australia. The report *Bloodborne viral and sexually transmitted infections in Aboriginal and Torres Strait Islander people: Surveillance and Evaluation Report* has been published from 2007, as an accompanying document to the annual surveillance report. The Surveillance and Evaluation Report provides detailed analysis and interpretation of the occurrence of these infections in Aboriginal and Torres Strait Islander communities in Australia.

Invasive Pneumococcal Disease Surveillance Program

The Commonwealth has developed the Invasive Pneumococcal Disease (IPD) Surveillance Program as part of the NNDSS Program. The objectives and outcomes of the IPD Surveillance Program are to:

- record every case of IPD occurring in Australia;
- collect detailed information on each case of IPD as set out in the NNDSS Invasive Pneumococcal Infection Enhanced Surveillance Form;
- collate nationally this information in the NNDSS dataset for enhanced IPD surveillance;
- measure the impact of conjugate pneumococcal vaccination on the rates and types of pneumococcal disease, the prevalence of circulating pneumococcal serotypes and levels of antibiotic resistance; and
- assess whether cases or deaths in children under 5 years and adults over 65 years are due to IPD vaccine failure or antibiotic resistance.

The Commonwealth funds four laboratories to perform the laboratory component of enhanced surveillance of IPD, which consists of the serotyping all isolates of *Streptococcus pneumoniae* from cases of IPD.

IPD data are reported annually (*Commun Dis Intell* 2012;36(2):E151–E165) and quarterly in CDI. These reports include analysis notification and laboratory data collected through the NNDSS.

IPD surveillance is overseen by the Enhanced Invasive Pneumococcal Disease Surveillance Working Group (EIPDSWG), a subcommittee of the CDNA. The EIPDSWG assists in developing and implementing a nationally standardised approach to the enhanced surveillance of IPD in Australia.

National Influenza Surveillance Scheme

Australian influenza activity and severity in the community are monitored using a number of indicators and surveillance schemes:

- Notifications of laboratory-confirmed influenza are reported from all Australian states and territories and included in the NNDSS.
- Community level ILI is monitored through two sentinel systems, Flutracking, a weekly online survey integrating syndromic information with participant influenza immunity status; and data from the National Health Call Centre Network.
- Reports on general practice ILI consultations are provided through the Australian Sentinel Practice Research Network and the Victorian Sentinel General Practice Scheme. Additionally, data on ILI presentations to hospital emergency departments are collected from sentinel hospital sites in Western Australia and New South Wales.
- Hospitalised cases of laboratory-confirmed influenza are reported through the Influenza Complications Alert Network (FluCAN); and severe complications in children are monitored by the APSU.
- Information on influenza subtypes and positivity are provided by sentinel laboratories, including the national influenza centre laboratories and some state public health laboratories. Additional virology and antiviral resistance data are also provided from the World Health Organization Collaborating Centre for Reference and Research on Influenza in Melbourne.

During the influenza season, data from each of these surveillance systems are compiled and published fortnightly in the Australian influenza surveillance report, which is generally available from May to October on the department's web site. These reports include the above data as well as additional mortality and international surveillance data.

Annual reports on the National Influenza Surveillance Scheme are published in the CDI each year (*Commun Dis Intell* 2010;34(1):8–22).

National Notifiable Diseases Surveillance System

National compilations of notifiable diseases have been published intermittently in a number of publications since 1917.[2] The NNDSS was established in 1990 under the auspices of CDNA.

More than 60 communicable diseases agreed upon nationally are reported to NNDSS, although not all are notifiable in each jurisdiction. Data are sent electronically from states and territories daily (business days only in some jurisdictions). The system is complemented by other surveillance

systems, which provide information on various diseases, including three that are not reported to NNDSS (HIV, and the classical and variant forms of CJD).

The NNDSS core dataset includes data fields for a unique record reference number; notifying state or territory, disease code, age, sex, Indigenous status, postcode of residence, date of onset of the disease, death, date of report to the state or territory health department and outbreak reference (to identify cases linked to an outbreak). Where relevant, information on the species, serogroups/subtypes and phage types of organisms isolated, and on the vaccination status of the case is collected. Data quality is monitored by Health and the National Surveillance Committee and there is a continual process of improving the national consistency of communicable disease surveillance.

While not included in the core national dataset, enhanced surveillance information for some diseases (hepatitis B [newly acquired], hepatitis C [newly acquired], invasive pneumococcal disease, donovanosis, gonococcal infection, syphilis < 2 years duration and tuberculosis) is obtained from states and territories.

Aggregated data are presented on the department's Internet site under *Communicable Diseases Surveillance* and updated daily (http://www.health.gov.au/nndssdata). A summary report and data table are also published on the Internet each fortnight (http://www.health.gov.au/cdnareport).

Data are published in CDI each quarter and in an annual report. The reports include numbers of notifications for each disease by state and territory, and totals for Australia for the current period, the year to date, and for the corresponding period of the previous year. The national total for each disease is compared with the average number of notifications over the previous 5 years in the same period. A commentary on the notification data is included with the tables in each issue of CDI and graphs are used to illustrate important aspects of the data.

OzFoodNet: enhanced foodborne disease surveillance

The Australian Government Department of Health established the OzFoodNet network in 2000 with epidemiologists in every Australian State and Territory to collaborate nationally in the investigation of foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease.

OzFoodNet reports quarterly on investigations of outbreaks and clusters of gastroenteritis potentially related to food. Annual reports have been produced and published in CDI since 2001 with the most recent being the 2010 annual report (*Commun Dis Intell* 2012;36(3):E213–E241). Data are reported from all Australian jurisdictions.

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Policy and guidelines

REVISED SURVEILLANCE CASE DEFINITIONS

This report provides the revised Surveillance case definitions approved by the Communicable Diseases Network Australia (CDNA) since 1 July 2014.

The Case Definitions Working Group (CDWG) is a subcommittee of the CDNA and comprises members representing all states and territories, the Australian Government Department of Health, the Public Health Laboratory Network, OzFoodNet, the Kirby Institute, the National Centre for Immunisation Research and Surveillance and other communicable disease experts. CDWG develops and revises surveillance case definitions for all diseases reported to the National Notifiable Diseases Surveillance System. Surveillance case definitions incorporate laboratory, clinical and epidemiological elements as appropriate.

The following case definitions have been reviewed by CDWG and endorsed by CDNA.

These case definitions were implemented on 1 January 2015 and supersede any previous versions.

Hepatitis C – newly acquired

Reporting

Only confirmed cases should be notified.

Confirmed case

A confirmed case requires either:

Laboratory definitive evidence

OR

Laboratory suggestive evidence AND clinical evidence.

Laboratory definitive evidence

Detection of anti-hepatitis C antibody from a person who has had a negative anti-hepatitis C antibody test recorded within the past 24 months

OR

Detection of hepatitis C virus by nucleic acid testing from a person who has a negative anti-hepatitis C antibody test result currently, or has had, within the past 24 months.

OR

Detection of anti-hepatitis C antibody from a child aged 18 months to 24 months

OR

Detection of hepatitis C virus by nucleic acid test-ing in a child aged 3 months to 24 months.

Laboratory suggestive evidence

Detection of anti-hepatitis C antibody, or hepatitis C virus by nucleic acid testing in a patient with no prior evidence of hepatitis C infection.

Clinical evidence

Clinical hepatitis within the past 24 months (where other causes of acute hepatitis have been excluded) defined as

1. Jaundice

OR

2. Bilirubin in urine

OR

3. Alanine transaminase (ALT) ten times upper limit of normal.

Hepatitis C – newly acquired	**Laboratory suggestive evidence** Added '…in a patient with no prior evidence of hepatitis C infection.' **Clinical evidence** Changed Alanine transaminase (ALT) from seven to ten times upper limit of normal.

Viral haemorrhagic fevers (quarantinable)

(Quarantinable – includes Ebola, Marburg, Lassa and Crimean-Congo fevers)

Reporting

Both confirmed cases and probable cases should be notified.

Confirmed case

A confirmed case requires laboratory definitive evidence only.

Laboratory definitive evidence

Laboratory definitive evidence requires confirmation by the Victorian Infectious Diseases Reference Laboratory (VIDRL), Melbourne* or the Special Pathogens Laboratory, CDC, Atlanta, or the Special Pathogens Laboratory, National Institute of Virology (NIV), Johannesburg

Isolation of a specific virus

OR

Detection of specific virus by nucleic acid testing or antigen detection assay

OR

IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to specific virus.

Probable case

A probable case requires laboratory suggestive evidence AND clinical evidence AND epidemiological evidence.

Laboratory suggestive evidence

Isolation of virus pending confirmation by VIDRL, Melbourne, or CDC, Atlanta or NIV, Johannesburg

OR

Detection of specific virus by nucleic acid testing, pending confirmation by VIDRL, Melbourne, or CDC, Atlanta or NIV, Johannesburg

OR

IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to specific virus pending confirmation by VIDRL, Melbourne, or CDC, Atlanta or NIV, Johannesburg

OR

Detection of IgM to a specific virus.

Clinical evidence

A compatible clinical illness as determined by an infectious disease physician. Common presenting complaints are fever myalgia, and prostration, with headache, pharyngitis, conjunctival injection, flushing, gastrointestinal symptoms. This may be complicated by spontaneous bleeding, petechiae, hypotension and perhaps shock, oedema and neurologic involvement.

Epidemiological evidence

History of travel to an endemic/epidemic area within 9 days (Marburg), 13 days (Crimean Congo) or 21 days (Lassa, Ebola) of illness onset. Filoviruses are endemic in Sub-Saharan Africa, Lassa in Western Africa, Crimean Congo in Africa and the Middle East to West China;

OR

Contact with a confirmed case

OR

Exposure to viral haemorrhagic fever-infected blood or tissues.

* The first case in any outbreak in Australia will also be confirmed by CDC, Atlanta or NIV, Johannesburg.

Viral haemorrhagic fevers (quarantinable)	**Laboratory definitive evidence** Include the Victorian Infectious Diseases Reference Laboratory (VIDRL) as an additional laboratory where laboratory definitive evidence can be confirmed. Include footnote that the first case in Australia in any given outbreak will also be confirmed by CDC or NIV.

Administration

Communicable Diseases Intelligence
INSTRUCTIONS FOR AUTHORS

Communicable Diseases Intelligence (CDI) is a peer-reviewed scientific journal published quarterly by the Office of Health Protection, Department of Health. The journal aims to disseminate information on the epidemiology, surveillance, prevention and control of communicable diseases of relevance to Australia.

The objectives of CDI are to:

- report on surveillance of communicable diseases of relevance to Australia
- publish high quality original articles relevant to communicable disease epidemiology in Australia, and
- provide information on activities relevant to the surveillance, prevention and control of communicable disease in Australia.

CDI is listed on MEDLINE and indexed by PubMed, an online searchable index of published articles and authors. CDI is open access. All articles published are made available free of charge.

CDI encourages submissions consistent with the objectives from practitioners in all disciplines across the public health field. Advanced trainees and post graduate students are also encouraged to submit to CDI. CDI publishes original articles, short reports, annual reports and quarterly reports, letters to the editor and editorials. Original articles and short reports are peer-reviewed.

Manuscripts for submission

Manuscripts submitted to CDI must be offered exclusively to the journal. All manuscripts should be accompanied by a covering letter that should include:

- confirmation that the manuscript content (in part or in full) has not been submitted or published elsewhere; and
- whether the manuscript is being submitted as an article, short report, surveillance summary, outbreak report or case report.

In addition, manuscripts should include a title page that should contain the following information:

- title (e.g. Prof, Dr, Ms, Miss, Mrs, Mr), full name including middle initial, position held, and institution at the time the article was produced, of each author;
- name of corresponding author, including current postal address, telephone, and email; and
- word count of the main text and of the abstract.

On receipt of a manuscript, authors will be sent a brief acknowledgment. Accepted manuscripts are edited for style and clarity and final proofs are returned to the corresponding author for checking prior to publication.

Authorship

Authorship should be based on substantial contribution to the article. Each author should have participated sufficiently to take public responsibility for the article. Others contributing to the work should be recognised in the acknowledgments.

Types of manuscript

Original articles

The text of articles must be structured to contain an abstract, introduction, methods, results, discussion, acknowledgments and references. Manuscripts submitted as articles must be 3,000 words or less and will be peer-reviewed.

Original articles may be submitted at any time and will be included in an issue once their review and revision has been completed. Articles may be published ahead of the scheduled issue, in the 'early release' format.

Systematic reviews submitted to CDI will be expected to conform to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (http://www.prisma-statement.org/).

Letters to the Editor

The editorial team welcome comments on articles published in CDI in the form of letters to the Editor. Letters should normally be less than 500 words, include no more than a single chart and less than six references.

Short reports

Short reports may be submitted for peer review or for publication without peer review, depending

on the content. Articles of particular relevance for rapid dissemination (such as timely outbreak reports) may be fast-tracked for early release prior to the next issue of CDI. Please discuss your requirements with the editorial team. Short reports may include an abstract. Types of short reports include:

Surveillance summaries

A report of 1,000 words or less that briefly reports on changes in the local epidemiology of a communicable disease, changes in surveillance systems, or new interventions, such as introducing vaccination in an at-risk group. Surveillance summaries should provide a brief description of the setting and a discussion of the significance of the events, changes or interventions.

Case reports

Brief reports of 500 to 1,000 words on cases of communicable disease will be considered based on their public health significance. Authors must note the instructions on the protection of patient's right to privacy (refer to the Ethics committee approvals and patients' right to privacy below). Some discussion of the significance of the case for communicable disease control should be included.

Outbreak reports

Reports of communicable disease outbreaks of 500 to 1,000 words will be considered for publication based on their public health significance. Reports should include details of the investigation, including results of interventions and the significance of the outbreak for public health practice. More comprehensive reports on outbreaks should be submitted as articles.

An outbreak report may be structured as below (the subheadings can be adjusted to suit), or may be unstructured if very brief.

Most outbreak reports will present only the descriptive epidemiology of the outbreak, with suspected risk factors for infection. The findings of any analytic study would usually be presented in an article at a later date, though authors may choose to present preliminary analyses from analytic studies.

Suggested structure

Abstract

A very brief unstructured abstract should be included

Background and methods

Including initial detection of the outbreak, case finding and interview techniques, study design and any statistical methods

Description of outbreak

Case definition, number of cases, number laboratory confirmed, symptoms. Time, place and person, epidemic curve

A maximum of two tables and/or figures is suggested.

Laboratory, trace back and environmental investigations

Details of the proportion of laboratory confirmation of cases

Public health response

A very brief description of any actions taken to prevent further cases may be included

Discussion

Including the significance of the outbreak for public health practice

References

A maximum of 20 references is suggested

Peer review process

Articles provisionally accepted for publication will undergo a peer review process and articles may be rejected without peer review. Short reports may be submitted for peer review, or may be reviewed at the discretion of the Editor. Articles will be subject to review by two experts in the field and short reports by one or two reviewers (if any).

When submitting your manuscript, you may specify reviewers who are qualified to referee the work, who are not close colleagues and who would not have a conflict of interest. Suggestions regarding reviewers will be considered, however, the Editor has the final decision as to who to invite to review a particular article.

Authors may be asked to revise articles as a result of the review process before the final decision about publication is made by the Editor. Revised articles are to be returned with a covering letter addressing each comment made by each reviewer.

Annual reports and quarterly reports are not subject to peer review.

Document preparation

Articles and reports must be written in clear, comprehensible English. Authors should pay particular attention to the style guides, web accessibility requirements and table and figure formatting requirements provided on these pages.

Articles are only accepted in electronic form, in Microsoft Word and Microsoft Excel. Graphics may be provided in a range of other formats (see section below on illustrations). In addition:

- Arial font is preferred but if not available use Times New Roman.
- Abstracts should not exceed 250 words. Do not cite references in abstracts.
- Structured abstracts are acceptable.
- Include up to 10 keywords.
- Avoid too many abbreviations.
- Use sentence case for all headings.

Manuscripts should be submitted with a one or two sentence summary of the article.

Tables

Tables and table headings should be located within the body of the manuscript and all tables should be referred to within the results section.

Information in tables should not be duplicated in the text.

Headings should be brief.

Simplify the information as much as possible, keeping the number of columns to a minimum and avoid merged cells as much as possible.

Separate rows or columns are to be used for each information type (e.g. percentage and number should be in separate columns rather than having one in parentheses in the same column).

If abbreviations are used these should be explained in a footnote.

Footnotes should use the following symbols in sequence:

* † ‡ § || ¶ ** †† ‡‡

Do not use blank rows or blank columns for spacing.

A short summary of each table should be included to satisfy government accessibility requirements (refer to Web accessibility requirements).

Figures and illustrations

Figures and illustrations, including headings, should be provided in the body of the manuscript and should be referred to within the results section. They should also be provided as a separate file.

Examples of each of the following can be found in the on-line version of Instructions to authors (http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-pubs-cdi-auth_inst.htm)

A long text description should be included to satisfy government accessibility requirements (refer to Web accessibility requirements).

Figures

Use Microsoft Excel.

Each figure should be created as a separate worksheet rather than as an object in the datasheet (use the 'as new sheet' option for chart location).

The numerical data used to create each figure must be included on a separate worksheet (see example on the Department of Health web site).

Worksheets should be appropriately titled to distinguish each graph (e.g. Figure 1, Figure 2; Figure 1 data, Figure 2 data).

Do not include the graph heading on the Excel worksheet.

Graphs should be formatted to CDI requirements as much as possible. These requirements are available on the Health web site (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-cdi-auth_excel_fig.htm).

Illustrations

Illustrations or flow charts can be included if required.

Images should preferably be at least 300 dpi.

Electronic copies of computer-generated illustrations should preferably be saved in a vector image program such as Adobe Illustrator or other similar graphic but charts created in either Word or PowerPoint are acceptable. Use a sans serif font for figures (e.g. Arial). Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

Photographs

Photographs may be submitted if required.

Photos need to be at least 300 dpi.

Electronic copies should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats (in preferential order):

- PSD
- TIFF
- EPS
- JPEG (JPG).

Maps

Maps created by mapping programs such as MapInfo or ArcGIS should be saved at 300 dpi and in one of the following graphic formats (in preferential order) to allow editing of font size and colours:

- AI
- EMF

If this is not possible the following graphic formats should be used (in preferential order):

- TIFF
- EPS
- GIF.

Other images

Other images may be submitted in one of the following graphic formats (in preferential order):

- PSD
- TIFF
- EPS, or
- GIF.

Authors should aim for maximum levels of contrast between shaded areas. Use a sans serif font for text. Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

Web accessibility requirements

The Australian Government is required to meet level AA of the Web Content Accessibility Guidelines version 2.0 (WCAG 2.0). These guidelines include the need for alternate methods of presenting the information depicted in images—including figures and maps—for readers with vision impairment and other disabilities using text readers. Complex tables also present challenges for text readers.

Articles and reports should be submitted with:

- a short summary of any tables
- a long text description of any figures;
- a long text description of any maps, flowcharts, or other images. For thermal maps showing disease rates by statistical location, a data table may be a preferred alternative.

Keep in mind that the description should be sufficient for a sight impaired person to understand what the information image is trying to convey,

Samples of descriptors for tables and figures can be found on the Department of Health web site (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-cdi-auth_web.htm).

Further information about WCAG 2.0 is available from the Australian Government Information Management Office (http://agimo.gov.au/)

References

References should be identified consecutively in the text using the Vancouver reference style. Any punctuation should precede the reference indicators.

Abbreviate journal names as in the PubMed journal database (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=journals) (e.g. *Commun Dis Intell*). Include the surnames and initials of all authors (or only the first six authors, et al, if there are more than six). Cite the first and last page numbers in full, and specify the type of reference (e.g. letter, editorial).

Examples of the Vancouver reference style are available on the Health web site. See also the International Committee of Medical Journal Editors Uniform requirements for manuscripts submitted to biomedical journals *Ann Intern Med* 1997;1126:36–47 (http://www.nlm.nih.gov/bsd/uniform_requirements.html).

Cite personal communications and unpublished papers in the text, not in the reference list, with the exception of material that has been accepted for publication (in press). Obtain written permission from people cited, and include their title, position and affiliation.

The accuracy of references is the responsibility of authors.

Ethics committee approvals and patients' rights to privacy

All investigations on human subjects must include a statement that the subjects gave their written informed consent, unless data collection was covered by public health legislation or similar studies have been considered by a relevant ethics committee and a decision made that its approval was not required. The name of the ethics committee that gave approval for the study should be included in the text. Alternatively, if approval is not required a statement to this effect should appear in the manuscript.

Ethical approval and patient consent may also be required for case reports. Identifying details about patients should be omitted if they are not essential, but data should never be altered or falsified in an attempt to attain anonymity.

Copyright

All authors are asked to transfer copyright to the Commonwealth before publication. A copyright form will be sent to the corresponding author. All authors are required to sign the copyright release. The Commonwealth copyright will be rescinded if the article is not accepted for publication.

Submission of manuscripts

Manuscripts should be provided electronically by email to: cdi.editor@health.gov.au

Please contact the editorial team at cdi.editor@health.gov.au if you require any further information.

Communicable Diseases Intelligence

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