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Communicable Diseases Intelligence is produced every quarter by:
Health Emergency Management Branch
Office of Health Protection
Australian Government Department of Health
GPO Box 9848, (MDP 6)
CANBERRA ACT 2601;
Telephone: +61 2 6289 2717
Facsimile: +61 2 6289 2700
Email: cdi.editor@health.gov.au

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Annual report

Immunisation coverage annual report, 2011

Brynley P Hull, Aditi Dey, Rob I Menzies, Julia M Brotherton, Peter B McIntyre

Abstract

This, the 5th annual immunisation coverage report, documents trends during 2011 for a range of standard measures derived from Australian Childhood Immunisation Register data, and National Human Papillomavirus (HPV) Vaccination Program Register data. The proportion of children 'fully vaccinated' at 12, 24 and 60 months of age was 91.4%, 92.2% and 89.5% respectively. Although pneumococcal vaccine had similar coverage at 12 months to other vaccines, coverage was lower for rotavirus at 12 months (83.8%) and varicella at 24 months (83.9%). By late 2011, the percentage of children who received the 1st dose of DTPa vaccine dose at less than 8 weeks of age was greater than 50% in 3 jurisdictions, the Australian Capital Territory, Victoria, and Queensland and at 70% for New South Wales and Tasmania. Although coverage at 12 months of age was lower among Indigenous children than non-Indigenous children in all jurisdictions, the extent of the difference varied. Overall, coverage at 24 months of age exceeded that at 12 months of age nationally. At 60 months of age, there was a marked variation between individual jurisdictions, ranging from coverage 8% lower in Indigenous children in South Australia to 6% higher in the Northern Territory. As previously documented, vaccines recommended for Indigenous children only (hepatitis A and pneumococcal polysaccharide vaccine) had suboptimal coverage at 60% and 68%, respectively. On-time receipt (before 49 months of age) of vaccines by Indigenous children at the 60-month milestone age improved between 2010 (18%) and 2011 (19%) but the disparity in on-time vaccination between Indigenous and non-Indigenous children increased at all 3 age milestones. The percentage of vaccine objectors in 2011 (1.7%) increased from 2007 when it was 1.1%. Coverage data for the 3rd dose of HPV from the national HPV register in the school catch up program was 71% but was substantially lower for the catch-up program for women outside school (39%–67%), although this was an improvement from 2010. *Commun Dis Intell* 2013;37(4):E291–E312.

Keywords: immunisation coverage, immunisation delay, small area coverage reporting, human papilloma virus vaccine

Introduction

This is the 5th annual immunisation coverage report, with the 1st report being in 2007.[1,2,3] It consolidates regular reports produced by the National Centre For Immunisation Research and Surveillance[4–16] using Australian Childhood Immunisation Register (ACIR) data and highlights important trends and significant issues over the preceding 12 months. It follows the format of the previous reports, providing a detailed summary for 2011. It includes vaccination coverage at standard milestone ages, coverage for vaccines not included in standard coverage assessments, timeliness of vaccination, coverage for Indigenous children and data for small geographic areas on vaccination coverage and prevalence of vaccine objectors. This report also includes data on adolescents who are not on the ACIR, from previously published sources. Readers are referred to the 2007 report for a more detailed explanation of the background to this series of annual reports and the range of analyses presented.[1] This report uses the long-standing international practice of reporting at key milestone ages, to measure coverage against national targets and to track trends over time. From July 2011, 13vPCV replaced 7vPCV on the National Immunisation Program (NIP) for all Australian children at 2, 4 and 6 months of age. In addition, a single supplementary dose of 13vPCV was funded for children aged 12–35 months who had not received a dose of 13vPCV or 10vPCV in their primary course. This 'supplementary dose' was available from October 2011 through to end of September 2012. It was also available through the NIP to medically at-risk and Indigenous children on the 3+1 schedule, to be given at least 2 months after the booster dose.

Incentives for vaccination and reporting to the Australian Childhood Immunisation Register up to December 2011

The ACIR makes information payments (up to $6) to all immunisation providers. Additional payments to general practitioners (GPs) for the provision of data to the ACIR have been in place since its inception in 1996. In the 2008–09 Budget one of the components of the General Practice Immunisation Incentives Scheme (GPII), the Service Incentive Payment (SIP), was discontinued effective from 1 October 2008. SIP payments of $18.50 had

previously been made to GPs for reporting all required vaccines on the NIP, at 6, 12, 18 months and 60 months.[17] However, the GPII Outcomes Payments, which pays practices that achieve levels of full immunisation amongst patients of 90% or greater, were maintained. The vaccines/antigens required for full immunisation in assessment for the outcomes payment in 2011 were the same as in recent years, i.e. diphtheria, *Haemophilus influenzae* type b (Hib), hepatitis B, measles, mumps, pertussis, polio, rubella and tetanus. Vaccines included in the NIP in 2011 but not required for the completed schedule assessment for provider payments were: meningococcal C vaccine (Men C); pneumococcal conjugate vaccine (PCV); and rotavirus vaccine. Varicella vaccine was also not included for coverage assessment but eligible providers received an information and SIP payment[1] (up to October 2008) for reporting, as varicella vaccine was the only vaccine required for completion of the 18-month schedule point. While the ACIR records vaccines available under the NIP only for Indigenous children in Queensland, the Northern Territory, Western Australia and South Australia (hepatitis A and 23vPPV) and vaccines not included in the NIP such as Bacille Calmette–Guérin, reporting of these does not attract a GPII payment.

In July 2004, the means test required to qualify for the Maternity Immunisation Allowance (MIA) was removed. This payment of $233 per child in 2008, was intended to provide motivation both to complete immunisation and for parents to prompt their provider to notify any outstanding reports to the ACIR before the child reached 24 months of age. In the 2008–09 budget, in addition to the changes mentioned above, it was announced that the MIA payment would be paid in 2 equal amounts of $167, with eligibility for the 2nd payment assessed between 48 and 60 months of age. This came into effect in January 2009, through a change in the National Due and Overdue Rules for Childhood Immunisation for all children born from 1 January 2005 onwards. This change stated that a child was due for their 48-month vaccinations at 48 months and overdue at 49 months of age, instead of overdue at 60 months of age. In July 2012, the MIA payment was ceased and the immunisation status of children aged 12, 24 and 60 months was linked to the existing Family Tax Benefit Part A supplement.

Table 1 shows the Australian National Immunisation Program Schedule for 2011.

Table 1: Australian National Immunisation Program Schedule in 2011

Age	Vaccine										
Childhood vaccines											
Birth	Hep B										
2 months	Hep B	DTPa	Hib	Polio				13vPCV		Rotavirus	
4 months	Hep B	DTPa	Hib	Polio				13vPCV		Rotavirus	
6 months	Hep B	DTPa	Hib	Polio				13vPCV		Rotavirus*	
12 months			Hib		MMR		Hep A†		Men C		
18 months						VZV	Hep A†‡	23vPPV†			
24 months							Hep A‡	23vPPV‡			
48 months		DTPa		Polio	MMR						
Adolescent vaccines											
12 years	Hep B§	dTpa				VZV§					HPV‖
15 years		dTpa								Flu¶,**	23vPPV††
Adult vaccines											
≥ 50 years										Flu¶,**	23vPPV¶
65 years										Flu**	23vPPV

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

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

‡ Aboriginal and Torres Strait Islander children in Queensland and South Australia

§ Catch-up only

‖ Females only

¶ For Aboriginal people only

** Annual vaccination, all aged ≥6 months with medical risk factors, non-Aboriginal adults ≥65 years

†† Aboriginal adults with medical risk factors

Methods

The Australian Childhood Immunisation Register

The ACIR was established on 1 January 1996, by incorporating demographic data from Medicare for all enrolled children under the age of 7 years.[5] Participation in the ACIR is opt-out so it constitutes a nearly complete population register, as approximately 99% of children are registered with Medicare by 12 months of age.[5] Children not enrolled in Medicare can also be added to the ACIR via a supplementary number. Since 2001, immunisations given overseas may be recorded if a provider endorses their validity. Data are transferred to the ACIR when a recognised immunisation provider supplies details of an eligible immunisation either automatically from medical practice software or through the Internet using the Medicare Australia web site or by submitting paper encounter forms. The existence of medical contraindications and conscientious objection to immunisation is also recorded on the ACIR. All vaccination records for a child remain on the register indefinitely, but no new immunisation encounter records are added after the 7th birthday.

Immunisations recorded on the register must be given in accordance with the guidelines issued by the National Health and Medical Research Council as stated in *The Australian Immunisation Handbook*.[18] Notifications falling outside these guidelines or duplicate notifications prompt an enquiry with the provider and, if their validity cannot be established, they are rejected.

Measuring immunisation coverage using the Australian Childhood Immunisation Register

The cohort method has been used for calculating coverage at the population level (national and state or territory)[19] since the ACIR's inception. Cohort immunisation status is assessed at 12 months of age (for vaccines due at 6 months), 24 months of age (for vaccines due at 12 months), and 60 months of age (for vaccines due at 48 months). A minimum 3-month lag period is allowed for late notification of immunisations to the register, but only immunisations given on or before a child's 1st, 2nd or 5th respective birthdays are considered.[19] If a child's records indicate receipt of the last dose of a vaccine that requires more than 1 dose to complete the series, it is assumed that earlier vaccinations in the sequence have been given. This assumption has been shown to be valid.[7,8]

Three-month birth cohorts are used for time trend analyses, while 12-month wide cohorts are used for other analyses in this report such as for small area coverage analysis and mapping of coverage estimates. The 12-month wide cohorts used in this report are children born between 1 January and 31 December 2010 for the 12-month milestone age; children born between 1 January and 31 December 2009 for the 24-month milestone age; and children born between 1 January and 31 December 2006 for the 5-year (60-month) milestone age.

The proportion of children designated as being 'fully immunised' is calculated using the number of children completely immunised with the vaccines of interest by the designated age as the numerator, and the total number of Medicare-registered children in the age cohort as the denominator. 'Fully immunised' at 12 months of age was defined as a child having a record on the ACIR of a 3rd dose of the combined DTPa-hepB-IPV-Hib vaccine. 'Fully immunised' at 24 months of age was defined as a child having a record on the ACIR of a 3rd dose of the combined DTPa-hepB-IPV-Hib vaccine, a 4th dose of *Haemophilus influenzae* type b (PRP-T) vaccine, and a 1st dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 60 months of age was defined as a child having a record on the ACIR of a 4th dose of combined DTPa-IPV vaccine, and a 2nd dose of an MMR-containing vaccine.

Immunisation coverage estimates were also calculated for individual NIP vaccines, including the 6 NIP vaccines not routinely reported in *Communicable Disease Intelligence* (CDI) and not part of 'fully immunised' calculations at 12 and 24 months of age. They were: a 3rd dose of PCV and 2nd or 3rd dose of rotavirus vaccine by 12 months of age; a 1st dose of varicella vaccine and a 1st dose of Men C vaccine by 24 months of age; a 2nd dose of hepatitis A vaccine in Indigenous children by 30 or 36 months of age; and a dose of 23vPPV vaccine in Indigenous children by 36 months of age.

Changes to immunisation policy and changes to the 'fully immunised' coverage algorithms have had an impact on vaccination coverage presented in this report. In December 2007, the coverage algorithm for immunisations due at 48 months of age was changed to assess children at 60 months, not 72 months of age. In January 2009, changes were made to the overdue rules so that children were classified as overdue for pre-school boosters at 49 months instead of the previous 60 months of age. This applied to parental and provider incentive payments. In March 2009, a recommendation was made by the Australian Technical Advisory Group on Immunisation (ATAGI) to parents and immunisation providers to consider bringing the 1st dose of DTPa forward to 6 weeks of age to

provide earlier protection against pertussis infection. From the September 2009 coverage assessment date onwards, changes were made in the coverage calculation algorithms that tightened the rules regarding receipt of Hib and hepatitis B vaccines for children aged 12 and 24 months of age. Prior to September 2009, if a child aged 12 months of age had a record on the ACIR of a 2nd or 3rd dose of any child Hib vaccine, he or she was considered 'fully vaccinated'. From September 2009, a child needed a record on the ACIR of a 3rd dose of any Hib vaccine or a 2nd dose of either PedvaxHIB or Comvax to be assessed as 'fully vaccinated'. Prior to September 2009, if a child aged 12 months of age had a record on the ACIR of a 2nd or 3rd dose of any hepatitis B vaccine, he or she was considered 'fully vaccinated'. From September 2009, a child needed a record on the ACIR of a 3rd dose of any hepatitis B vaccine or a 2nd dose of either Engerix B (paediatric), Comvax, or HBVAX II (paediatric), to be assessed as 'fully vaccinated'. In October 2009, a recommendation was made by the Australian Technical Advisory Group on Immunisation that the 4th dose of DTPa containing vaccine can be given from 42 months of age instead of the previously recommended 48 months of age.

Timeliness

Age-appropriate immunisation was defined as receipt of a scheduled vaccine dose within 30 days of the recommended age. For example, a child who received the 1st dose of DTPa (due at 60 days of age) when he or she was more than 90 days of age was classified as late for that dose. For descriptive purposes, we categorised the outcome measure for each dose as either vaccine dose 'no delay', 'delay of between 1 to 6 months', or 'delay greater than 6 months'. Doses received 'too early' (more than 30 days prior to when it was due), and doses never administered or recorded were excluded. Timeliness is measured in 12-month birth cohorts. Children included in the timeliness analysis were assessed at 1–2 years after doses were due, to allow time for late vaccinations to be recorded. Therefore, cohorts assessed for timeliness are not the same as those assessed for coverage milestones. The interval between doses was not evaluated. Timeliness of different vaccines and doses was also compared by plotting the cumulative percentage receiving each vaccine dose by age, with the proportion ever immunised set at 100%.

Remoteness status

The area of residence of children was defined as accessible or remote using the Accessibility/ Remoteness Index of Australia (ARIA), which was developed by the then Department of Health and Aged Care, and proposed as the national standard measure of remoteness for inclusion in the Australian Bureau of Statistics (ABS) 2001 census.[20] For the timeliness analysis, we defined the 2 ARIA categories with most restricted access to services as 'remote' (approximately 2.6% of the Australian population) and all other areas as 'accessible'.

Indigenous status

Indigenous status on the ACIR is recorded as 'Indigenous', 'non-Indigenous' or 'unknown', as reported by the child's carer to Medicare, or by the immunisation provider to the ACIR. This report considers 2 categories of children: 'Indigenous' and 'non-Indigenous': children with unknown Indigenous status were presumed to be 'non-Indigenous'. Coverage estimate time trends are presented from 2004 only, due to poor rates of reporting of Indigenous status prior to then.[21]

Small area coverage

Coverage was calculated for ABS-defined Statistical Subdivisions (SSD),[22] chosen because each is small enough to show differences within jurisdictions but not too small to render maps unreadable. Maps were created using version 10 of the MapInfo mapping software[23] and the ABS Census Boundary Information. As postcode is the only geographical indicator available from the ACIR, the ABS Postal Area to Statistical Local Area Concordance 2006 was used to match ACIR postcodes to SSDs, in order to create a SSD field for each child in the relevant study cohorts.[24]

Vaccine objection / no vaccines recorded

A child must be registered with Medicare before their parent(s) can lodge an official objection to immunisation. Parents can also object to immunisation and also object to lodging any official objection to the ACIR. This report uses the percentage of children with no vaccines recorded on the ACIR as a proxy measure of the number of these children.[16] Some children with no vaccines recorded on the ACIR are officially registered as 'vaccine objectors' and some are not registered as such. Registered vaccine objectors are eligible for parent incentive payments even if their children are unvaccinated. The proportion of vaccine objectors and children with no vaccines recorded by region were calculated from the cohort of children registered with Medicare, and born between 1 January 2005 and 31 December 2010. At the time of data extraction on 31 March 2012, they were between 12 and 72 months of age. This cohort was chosen when calculating proportions so that children under the age of 12 months

were not included, to allow sufficient time for registration of objection and exclude infants late for vaccination.

Human papillomavirus vaccine coverage

The human papillomavirus (HPV) vaccination program is listed on the NIP Schedule, funded under the Immunise Australia Program, and delivered to girls through an ongoing school-based program usually in the first year of secondary school. From 2007 to 2009 there was a time-limited catch-up program delivered through schools, general practices and community immunisation services for girls up to age 26. Immunisation against HPV is achieved with a course of 3 doses of vaccine, over a 6 month period. Data on the National HPV Vaccination Program are provided by the National HPV Vaccination Program Register, which is operated by the Victorian Cytology Service. The purpose of this legislated register is to support the implementation of the vaccination program and to provide data for monitoring and evaluation.[25] States and territories provide data to the Register from their school based programs. Doses administered in general practice or by community providers outside of the school program are notified on a voluntary basis, with a notification payment provided only to GPs during the 2007–2009 catch up program. The World Health Organization proposes using 15 years as the reference age for HPV vaccination coverage for the purposes of international comparison. Data on HPV coverage as notified to the HPV Register was obtained from the Immunise Australia web site.[26]

Coverage in the elderly

As there has not been an Adult Vaccination Survey (AVS)[27] undertaken in Australia since 2009, no data are presented in this 2011 report on influenza and pneumococcal (23vPPV) vaccination coverage in the elderly. The next AVS is planned for 2014.

Results

Coverage estimates

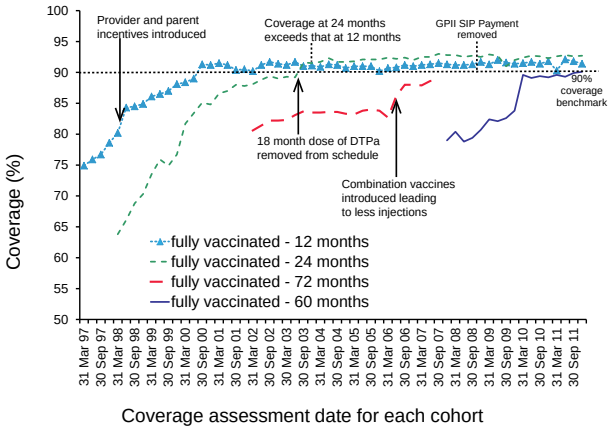
Overall

Nationally and for most jurisdictions, 'fully immunised' coverage and coverage for all individual vaccines (except rotavirus and varicella vaccines) for the 12-month and 24-month age groups exceed the 1993 Immunise Australia Program's target of 90% (Tables 2 and 3). However, coverage in Western Australia was below this target for hepatitis B and 'fully immunised' at 12 months of age. Recorded national 'fully immunised' coverage for the 60-month age group is marginally below the target, at 89.5% for all vaccines, and lower in particular jurisdictions such as Queensland, South Australia, and Western Australia (Table 4). For individual vaccines for this age group, coverage is below 90.0% for all vaccines in the Northern Territory, South Australia, and Western Australia and over 90% in all other jurisdictions.

There is a clear trend of increasing vaccination coverage over time for all age groups assessed, with the 2 youngest age cohorts having the highest coverage (Figure 1). The proportion 'fully immunised' at 12 months of age increased steadily from 75% for the 1st cohort in 1997 to 91.4% by 31 December 2011. At the 24-month milestone, 'fully immunised' coverage estimates also increased steadily from 64% for the 1st cohort to 92.2% by December 2011. 'Fully immunised' coverage estimates assessed at 72 months of age, for vaccines due at 48 months, were first reported in *CDI* in 2002, and increased steadily from 80.6% in early 2002 to 87.3% in late 2007, including a noticeable increase in June 2006, corresponding with the introduction of combination vaccines. However, from the beginning of 2008, when the assessment age was changed from 72 months to 60 months, 'fully immunised' coverage was substantially lower at 80.7% in December 2008, related to delayed immunisation. However, during 2009 and 2010, coverage for this age group rose substantially. Coverage calculated at 60 months was largely unchanged during the latter half of 2011 at 89.5% (Figure 1).

Coverage estimates for the 24-month age group increased substantially and suddenly in September 2003 to 91.6%, following the removal from the immunisation schedule of the 4th dose of DTPa (due at 18 months of age) from this quarter onwards (Figure 1). Coverage estimates for the

Figure 1: Trends in 'fully immunised' vaccination coverage estimates, Australia, 1997 to 2011, by age cohort



By 3-month birth cohorts born between 1 January 1996 and 31 December 2010. Coverage assessment date was 12 months after the last birth date of each cohort.

Source: Australian Childhood Immunisation Register.

Table 2: Percentage of children in 2011 vaccinated by 12 months of age, by vaccine and state or territory*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,110	96,942	3,692	61,789	19,468	6,058	71,943	31,551	296,553
Diphtheria, tetanus, pertussis (%)	94.2	92.1	91.7	91.8	92.1	92.4	93.1	90.5	92.1
Poliomyelitis (%)	94.1	92.1	91.7	91.7	92.1	92.4	93.1	90.4	92.1
Haemophilus influenzae type b (%)	93.8	91.7	91.3	91.5	91.8	92.2	92.7	90.1	91.8
Hepatitis B (%)	93.3	91.6	91.4	91.4	91.8	92.2	92.5	89.7	91.6
Fully immunised† (%)	92.9	91.3	91.3	91.2	91.6	92.0	92.2	89.5	91.4
Rotavirus (%)	89.0	86.5	84.0	80.9	84.0	87.1	83.5	77.5	83.8
PCV (%)	93.2	91.3	91.3	91.1	91.4	91.7	92.1	89.2	91.3

* For the birth cohort born in 2010

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

Table 3: Percentage of children in 2011 vaccinated by 24 months of age, by vaccine and state or territory*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,149	98,290	3,790	62,626	19,785	6,455	72,551	32,025	300,671
Diphtheria, tetanus, pertussis (%)	95.4	94.5	95.2	94.5	94.6	95.1	95.2	93.3	94.6
Poliomyelitis (%)	95.4	94.4	95.2	94.5	94.6	95.1	95.2	93.2	94.5
Haemophilus influenzae type b (%)	95.4	94.7	94.8	94.4	94.4	95.6	95.1	93.4	94.6
Hepatitis B (%)	94.3	94.0	94.8	94.0	94.0	94.9	94.6	92.6	94.0
Measles, mumps, rubella (%)	94.7	93.7	94.8	93.9	93.7	95.0	94.5	92.6	93.9
Fully immunised† (%)	92.9	92.0	92.8	92.4	92.2	93.7	92.9	90.6	92.2
Varicella (%)	87.1	82.8	85.7	86.7	83.2	84.1	84.3	80.7	83.9
MenC (%)	94.2	93.3	94.4	93.6	93.6	95.0	94.1	91.8	93.5

* For the birth cohort born in 2009.

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

Table 4: Percentage of children vaccinated by 60 months of age, 2011, by vaccine and state or territory*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	4,928	96,218	3,497	61,704	19,403	6,462	71,939	31,653	295,804
Diphtheria, tetanus, pertussis (%)	91.9	90.1	89.4	90.4	87.5	91.2	91.5	86.6	90.0
Poliomyelitis (%)	91.9	90.1	89.5	90.4	87.5	91.3	91.6	86.6	90.0
Measles, mumps, rubella(%)	91.6	90.0	89.4	90.2	87.3	91.2	91.4	86.5	89.9
Fully immunised (%)	91.2	89.6	88.9	89.9	86.9	90.7	91.0	85.9	89.5

* For the birth cohort born in 2006.

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

12-month age group have, however, remained steady over the past 10 years, fluctuating around the 91% level.

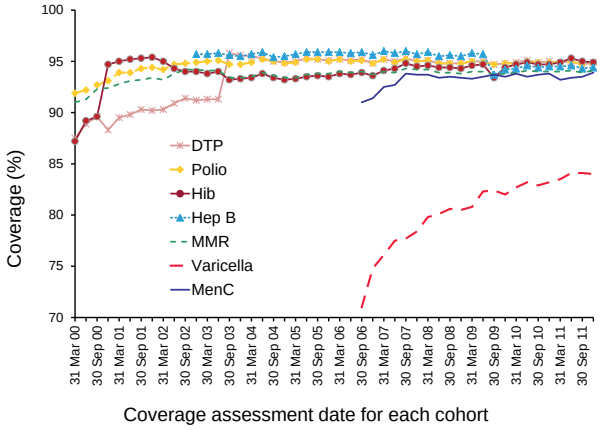
Individual vaccines

DTPa and polio coverage at 12 months of age remained relatively stable from the latter part of 2001 until 2011 (Figure 2). Coverage for the Hib and hepatitis B vaccines at 12 months of age (prior to the change in algorithm to measure coverage that occurred in the latter half of 2009) are becoming similar to those for DTPa and polio in the last 2 cohorts of 2009 and all of 2010 and 2011 and to more accurately reflect the situation (Figure 2). Coverage for PCV rose steadily from below 90% in mid-2007 to be just below that for all other vaccines due at this age at around 91%, except for rotavirus vaccine. Rotavirus vaccine coverage rose steeply from late 2008 from below 70% to almost 84% in late 2011 (Figure 2).

For most of the study period, at 24 months of age, hepatitis B coverage was higher than for all other vaccines at just under 95%, due to the different coverage algorithm described above (Figure 3). Coverage was lowest for MMR and Hib, the only vaccines that have a 12-month dose used in calculations, but in 2011 coverage is similar for all vaccines at around 94%–95%, except for varicella vaccine.

There was a marked increase in coverage for individual vaccines at 60 months of age following the change in the due or overdue rules in January 2009, with coverage increasing to levels similar to when coverage was assessed at 72 months of age

Figure 3: Trends in vaccination coverage estimates for individual vaccines at 24 months of age (DTP, polio, Hib, hepatitis B, measles, MMR, meningococcal C, and varicella),* Australia, 1999 to 2011

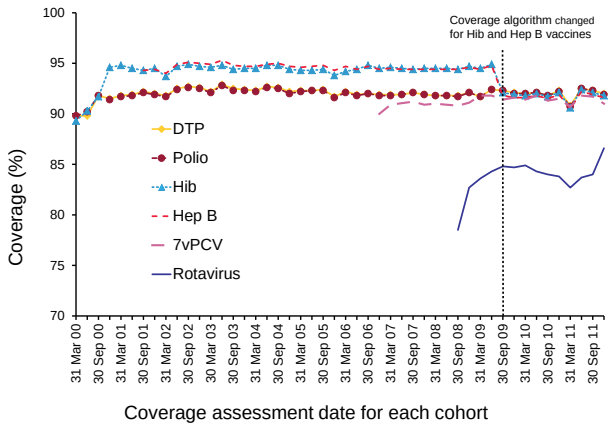


* 3rd or 4th dose of DTP, 3rd dose of polio, 3rd or 4th dose of Hib, 2nd or 3rd dose of Hep B, 1 dose of MMR, meningococcal C, and varicella.

By 3-month birth cohorts born between 1 January 1997 and 31 December 2009. Coverage assessment date was 24 months after the last birth date of each cohort.

Source: Australian Childhood Immunisation Register.

Figure 2: Trends in vaccination coverage estimates for individual vaccines at 12 months of age (DTP, polio, Hib, hepatitis B, 7-valent pneumococcal, and rotavirus),* Australia, 1999 to 2011

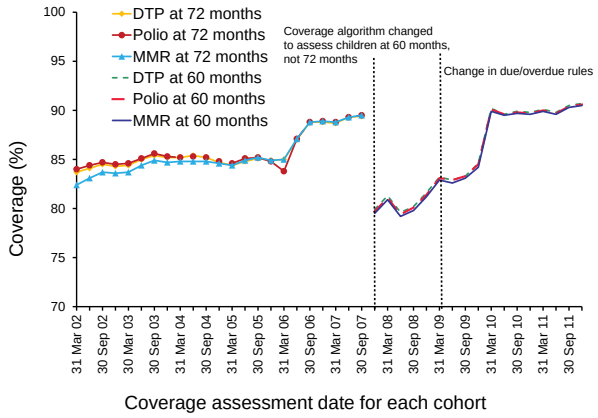


* 3rd dose of DTP, polio, and 7-valent pneumococcal, 2nd or 3rd dose of Hib, Hep B, and rotavirus.

By 3-month birth cohorts born between 1 January 1999 and 31 December 2010. Coverage assessment date was 12 months after the last birth date of each cohort.

Source: Australian Childhood Immunisation Register.

Figure 4: Trends in vaccination coverage estimates for individual vaccines (DTP, polio, and MMR)* at 60 months of age (6 years prior to December 2007), Australia, 2002 to 2011



* 4th dose of DTP and polio, 2nd dose of MMR

By 3-month birth cohorts born between 1 January 1996 and 31 December 2006. Coverage assessment date was 72 months after the last birth date of each cohort up to December 2007 and then 60 months after the last birth date of each cohort.

Source: Australian Childhood Immunisation Register.

(Figure 4). Coverage for all individual vaccines is at 90% in 2011 (Table 4), probably related, in part, to completed immunisation by 48 months of age being introduced in 2009 as a requirement for GP incentive payments.

Coverage estimates for Indigenous children

Immunisation coverage is lower for Indigenous children than non-Indigenous, particularly at 12 months and 60 months, with little or no difference at 24 months of age (Table 5). The difference in coverage at 12 months of age has been relatively consistent for the past 4 years.[1,2,3] The coverage differential between Indigenous and non-Indigenous children for individual vaccines varies, with coverage at 24 months of age for most vaccines being almost identical for both groups and greater among Indigenous children for Hib, hepatitis B, MMR and meningococcal C vaccines.

The proportion of children 'fully immunised' by 24 months of age has consistently remained higher than at 12 months and 60 months of age (Figure 5). As for non-Indigenous children, coverage at 60 months of age for Indigenous children increased following the change in due or overdue rules. Coverage at the end of 2011 was higher at 60 months than at 12 months.

Although coverage was lower among Indigenous children in all jurisdictions, the extent of the difference varied, reaching a 15 percentage point differential in South Australia and a 9 percentage

Table 5: Vaccination coverage estimates, 2011, by age, vaccine and Indigenous status

Vaccine	Milestone age	Indigenous	Non-Indigenous
DTP	12 months*	85.3	92.5
	24 months†	94.2	94.6
	60 months‡	87.3	90.1
Polio	12 months*	85.3	92.4
	24 months†	94.1	94.6
	60 months‡	87.3	90.1
Hib	12 months*	85.2	92.1
	24 months†	94.8	94.6
	60 months‡	N/A§	N/A§
Hep B	12 months*	85.3	92.0
	24 months†	94.1	94.0
	60 months‡	N/A§	N/A§
MMR	12 months*	N/A§	N/A§
	24 months†	94.7	93.8
	60 months‡	87.5	90.0
Varicella	12 months*	N/A§	N/A§
	24 months†	82.1	84.0
	60 months‡	N/A§	N/A§
Meningococcal C	12 months*	N/A§	N/A§
	24 months†	94.3	93.4
	60 months‡	N/A§	N/A§
PCV	12 months*	85.2	91.6
	24 months†	N/A§	N/A§
	60 months‡	N/A§	N/A§
Rotavirus	12 months*	71.1	84.8
	24 months†	N/A§	N/A§
	60 months‡	N/A§	N/A§

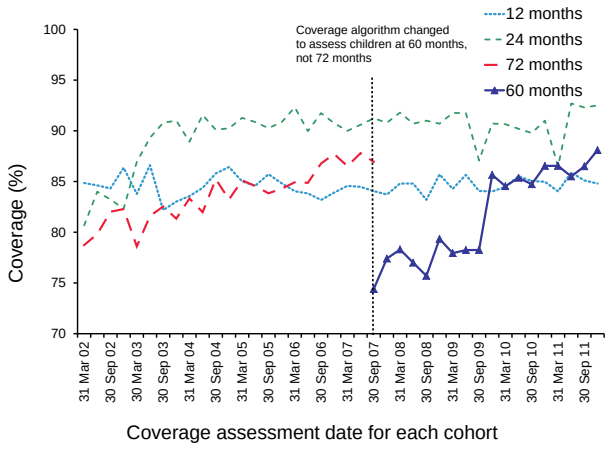
* Birth cohort born 1 January – 31 December 2010.

† Birth cohort born 1 January – 31 December 2009.

‡ Birth cohort born 1 January – 31 December 2006.

§ Not included in coverage estimates for that group.

Figure 5: Trends in 'fully immunised' vaccination coverage estimates for Indigenous children in Australia, 2002 to 2011, by age cohorts



point differential in Western Australia (Table 6). By age 24 months, the coverage disparity between Indigenous and non-Indigenous children ranged from 4 percentage points higher in the Northern Territory to 5 percentage points lower in South Australia (Table 6).

At 60 months of age, there was large variation between individual jurisdictions, ranging from coverage 8 percentage points lower in Indigenous children in South Australia to 6 percentage points higher in the Northern Territory, compared to non-Indigenous children (Table 6).

Coverage for National Immunisation Program vaccines not routinely reported elsewhere

Pneumococcal conjugate vaccine and rotavirus

7vPCV was first added to the NIP in January 2005 and was replaced in July 2011 by 13vPCV for all Australian children at 2, 4 and 6 months of age. Since coverage was first calculated for this vaccine in early 2006, it has remained high, with a slight increase from 89% to 91.3% (Figure 2). Coverage is greater than the 1993 Immunise Australia Program target of 90% in all jurisdictions except for Western Australia where it is very close to 90% (Table 2).

Rotavirus vaccine was added to the NIP in July 2007, so coverage for 2 or 3 doses (depending on vaccine) at 12 months of age could be calculated only from the December 2008 quarter onwards. Rotavirus coverage was lower nationally (Figure 2), and had greater variation between

Table 6: Percentage of children fully immunised by 12 months, 24 months and 60 months of age, 2011, by Indigenous status and state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
12 months – fully immunised (%)*									
Indigenous	86.7	86.7	89.8	84.8	77.6	91.9	85.4	80.6	85.2
Non-Indigenous	93.1	91.5	92.2	91.7	92.1	92.0	92.3	90.0	91.7
12 months – fully immunised (incl rotavirus & PCV) (%)									
Indigenous	79.7	75.0	74.3	65.5	64.7	82.5	68.0	59.1	69.0
Non-Indigenous	87.0	83.8	85.3	83.5	85.4	83.6	84.2	79.7	83.6
24 months – fully immunised (%)†									
Indigenous	91.9	91.7	95.2	92.9	87.8	94.0	90.9	90.4	92.2
Non-Indigenous	92.9	92.0	91.2	92.4	92.4	93.6	92.9	90.6	92.2
24 months – fully immunised (incl varicella & MenC) (%)									
Indigenous	84.9	78.0	85.9	82.9	72.3	82.3	76.6	74.7	79.9
Non-Indigenous	85.2	80.9	82.5	85.4	82.1	82.6	82.7	79.0	82.3
60 months – fully immunised (%)‡									
Indigenous	90.3	86.1	92.2	88.8	79.0	90.2	86.9	81.6	86.8
Non-Indigenous	91.2	89.8	86.5	89.9	87.1	90.8	91.1	86.2	89.6

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

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

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

jurisdictions compared to other vaccines given at 2, 4 and 6 months, which may be due to the strict upper age limits for this vaccine. Reported coverage in 2011 for 2 doses of Rotarix® or 3 doses of Rotateq® vaccine at 12 months of age varied from 77.5% in Western Australia (Rotateq®) to 87.1% and 89% in Tasmania and the Australian Capital Territory (both Rotarix®) respectively (Table 2).

Meningococcal C and varicella

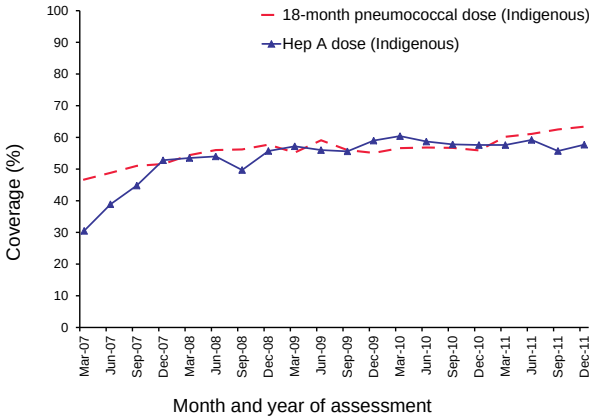
Meningococcal C vaccine was added to the NIP in January 2003. Since coverage was first calculated for this vaccine in early 2006, it has remained at high levels, with an increase over 2 years from 88% to almost 94% (Figure 3). There was little variation in 2011 by jurisdiction with all jurisdictions experiencing coverage levels greater than 91% and some, the Northern Territory and Tasmania, approaching or at 95% (Table 3).

Varicella vaccine was added to the NIP in November 2005. Reported coverage for this vaccine has consistently been 10 to 15 percentage points lower than that for all the other vaccines assessed at the 24-month milestone, being 84% for the latest assessment in 2011 (Figure 3). This is probably partly due to the shorter time varicella has been on the NIP and the age of administration (18 months). The 18-month schedule point was historically associated with lower coverage when there was an 18-month pertussis booster prior to 2003. Between 2003 and 2005, there was a gap of over 2 years when no vaccine was administered at 18 months. Also there is a shorter time period to catch up for varicella vaccination (6 months) compared with other vaccines. Reported varicella vaccine coverage in 2011 also shows considerable variation by jurisdiction from 80.7% in Western Australia to 87.1% in the Australian Capital Territory (Table 3). Data are also available from the ACIR on the number of reports from GPs stating that children born since May 2004 have natural immunity to varicella and do not require varicella vaccination. Reports of natural immunity to varicella total greater than 20,000 since May 2004 (not shown), corresponding to approximately 1.1% of the cohort. It is likely that there is under-reporting of presumed natural immunity by GPs but this is unlikely to fully account for lower varicella coverage.

Hepatitis A and 23vPPV

Hepatitis A vaccine was available in Australia prior to the inception of the ACIR in 1996 and has been included on the NIP for Indigenous children in the Northern Territory, South Australia, Western Australia, and in Queensland since November 2005, but was used earlier than this in north Queensland. Since March 2007, coverage of 2 doses of hepatitis A vaccine for Indigenous children by 30 months of age in Western Australia and the Northern Territory and 36 months of age in Queensland and South Australia has increased from 31% to 58% in December 2011 (Figure 6). An additional 8% of children had received 1 dose of hepatitis A vaccine by 18 or 24 months of age, putting national coverage for at least 1 dose of hepatitis A vaccine for 2012 at 66% in Indigenous children compared with 58% for 2 doses (Table 7). There is a variation in reported hepatitis A vaccine coverage by jurisdiction, from a low of 33% in South Australia to a high of 81.2% in the Northern Territory (Table 7).

Figure 6: Trends in coverage estimates for hepatitis A* and pneumococcal polysaccharide (23vPPV) vaccines for Indigenous children, Australia, 2007 to 2011



* Two doses assessed at 30 months for Western Australia and the Northern Territory. Two doses assessed at 36 months for Queensland and South Australia.

Table 7: Vaccination coverage* for hepatitis A and 23vPPV, Northern Territory, Queensland, South Australia and Western Australia, 2011, by state or territory

	Vaccine type	
State or territory	Hep A	23vPPV
NT	81.2 (84.6)	83.7
Qld	53.1 (62.0)	62.8
SA	33.0 (52.7)	47.6
WA	55.5 (66.5)	54.9
Aust	57.7 (66.0)	63.4

* For the last 3-month cohorts assessable in 2011.

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

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

The 23vPPV has been recommended for Indigenous children in the Northern Territory, South Australia, Western Australia, and Queensland as a booster at 18–24 months of age since 2001. Coverage has gradually increased from 47% in March 2007 to 63% in December 2011 (Figure 6). From 2010 to 2011, coverage increased by 7 percentage points. There is a large variation in 23vPPV coverage by jurisdiction from a low of 48% in South Australia to a high of 84% in the Northern Territory (Table 7).

Timeliness of immunisation

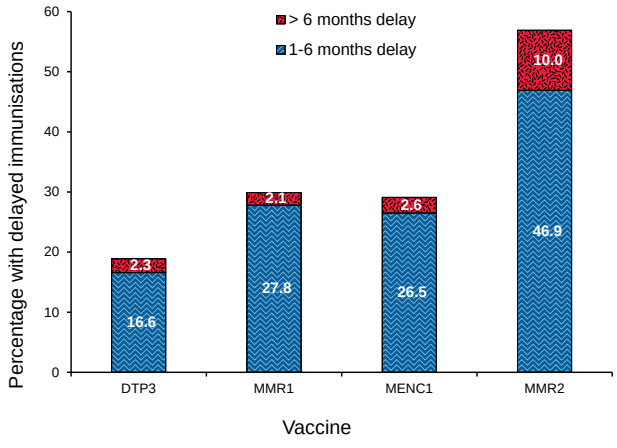
Timeliness has been examined for vaccines requiring both multiple doses (DTPa, 7vPCV and MMR) and a single dose (Men C) at 12 and 24 months of age.

Since 2004, the proportion of children with timely receipt of the 3rd dose of DTP vaccine has remained at 88% (not shown). Across the 5-year period, 2004 to 2008, timely receipt of 1 dose of MMR rose 3 percentage points, although estimated coverage by 24 months of age remained stable at almost 94% (not shown).

As demonstrated in previous studies, the proportion of children with vaccination delay increased with older age (Figure 7). The greatest proportion with any delay was seen with the 2nd dose of MMR vaccine with 57% of doses given late and 10% given more than 6 months late. This analysis is for doses due in 2009 allowing up to 3 years for capturing delayed doses, as explained in the methods. This is a considerable improvement on the 2010 report where the corresponding figures were 65% and 24%. Further improvements are expected in future analyses that reflect more recent improvements in timely receipt of the 2nd dose of MMR (Figure 4).

For the 3rd dose of DTPa, there was greater delay for Indigenous children than non-Indigenous children, with a 22% differential in on-time vaccination at < 7 months of age (Figure 8). The same pattern was found for timeliness of the 1st dose of MMR, but with a smaller differential in on-time vaccination at < 13 months of age of 13% (Figure 9). Although Indigenous children had only slightly lower coverage than non-Indigenous chil-

Figure 7: Vaccination delay for cohorts born in 2009 (DTP3, MMR1, MENC1) and 2005 (MMR2)



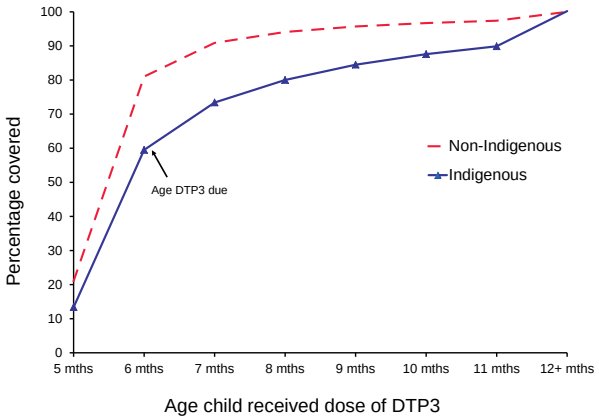
DTP3 = 3rd dose of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine

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

MENC1 = 1st dose of a meningococcal C vaccine

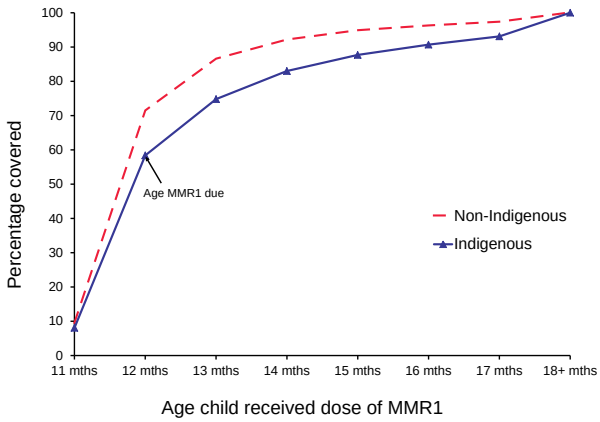
MMR2 = 2nd dose of a measles, mumps and rubella vaccine

Figure 8: Timeliness* of the 3rd dose of DTP vaccine (DTP3) for the cohort born in 2009, by Indigenous status



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

Figure 9: Timeliness* of the 1st dose of MMR vaccine (MMR1) for the cohort born in 2009, by Indigenous status



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

dren by 24 months of age, they were more likely to have delayed vaccination and this differential in on-time vaccination between Indigenous and non-Indigenous continues to increase (the corresponding differential for the 1st dose of MMR from the 2010 report was 11%).

Vaccination with the 3rd dose of DTPa and the 1st dose of MMR was delayed by more than 1 month for 29%–38% of Indigenous children and 16%–29% of non-Indigenous children (Table 8). The proportion with long delays (i.e. greater than 6 months) was 2 to 4 times higher in Indigenous children than in non-Indigenous children, with no great differences between accessible and remote areas or vaccines. Delays of 1 to 6 months were also more frequent for Indigenous children, although less marked, especially for the 1st dose of MMR. The proportion with short delays was greater among Indigenous children residing in remote areas than in accessible areas for the 3rd dose of DTP vaccine (37% versus 29%), but not for the 1st dose of MMR.

Vaccination delay for Indigenous children by jurisdiction was measured for the 3rd dose of PCV, with greater delays in Western Australia (45.8%) and the Northern Territory (41.5%) (Figure 10). The proportion of children with long delays in South Australian Indigenous children increased from the previous report in 2010 (from 5.8% to 6.2%) but decreased in Indigenous children from the Northern Territory (6.9% to 4.4%). There were no important differences in vaccination delay for non-Indigenous children by jurisdiction (not shown).

In contrast to earlier reports, analysis of timeliness of immunisation for a vaccine due at 48 months of age, the 2nd dose of MMR, showed a large difference in delay in receiving this vaccine for non-Indigenous children and Indigenous children, with a 11.0% differential at 51 months of age (Figure 11). However, timeliness for both groups was improved from the previous report in 2010.

Figure 10: Vaccination delay for Indigenous children for the 3rd dose of pneumococcal conjugate vaccine for the cohort born in 2009, by state or territory

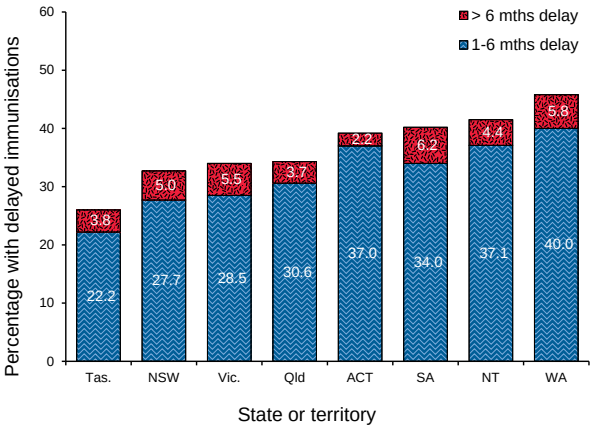
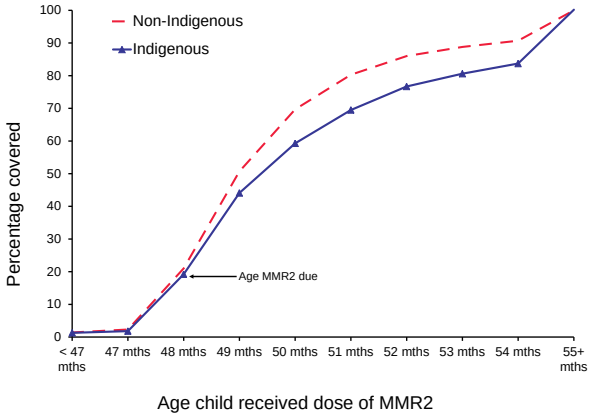


Figure 11: Timeliness* of the 2nd dose of MMR vaccine (MMR2) for the cohort born in 2005, by Indigenous status



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

Table 8: Vaccination delay for the cohort of children born in 2009, Australia, by Indigenous and remoteness status

Vaccine dose	Indigenous status	Remoteness	1–6 months delay %	> 6 months delay %
DTP3	Indigenous	Accessible	29.2	8.7
		Remote	37.2	7.1
	Non-Indigenous	Accessible	15.9	2.0
		Remote	15.7	1.9
MMR1	Indigenous	Accessible	37.8	5.4
		Remote	36.3	3.9
	Non-Indigenous	Accessible	27.3	2.0
		Remote	28.7	1.5

In response to a pertussis epidemic and to provide early protection to young infants, it was recommended by the ATAGI in March 2009, and promoted in that year during epidemics in New South Wales and Tasmania (later in other jurisdictions), that immunisation providers give the 1st dose of DTPa vaccine at 6 weeks of age instead of 8 weeks of age. Prior to this, very few children received the vaccine dose at less than 8 weeks of age, but for New South Wales and Tasmania the percentage rose over the 2 years with more than 60% of children receiving the dose prior to 8 weeks of age in December 2010 (Figure 12). By late 2011, this percentage was greater than 50% in 3 jurisdictions, the Australian Capital Territory, Victoria, and Queensland and at 70% for New South Wales and Tasmania.

Small area coverage

Immunisation coverage in Australia in 2011 varied substantially within jurisdictions, with some areas substantially below the national averages, potentially putting them at risk of outbreaks (Figures 13–15). In particular, there are 12 Statistical Subdivisions with coverage at 60 months of age below 85% (Figure 15).

The proportions of children whose parents are recorded as vaccine objectors, and the proportion of children with no vaccines recorded are presented by SSD in Figures 16 and 17, respectively. No vaccines recorded may represent either non-immunisation (parents refusing any vaccines) or, and probably much less commonly, non-reporting by a provider. The percentage of children with no vaccines recorded nationally (3.0%) is greater than those recorded as vaccine objectors (1.7%) but the percentage of vaccine objectors has increased from 2007 when it was 1.1%. The map of the proportion of vaccine objectors (Figure 16) shows pockets of high levels of objection within jurisdictions in 2011, particularly in coastal areas of South East Queensland, northern New South Wales, the Mount Lofty Ranges region in South Australia, and south west Western Australia, which also appear with low coverage in Figures 13–15. These areas have had consistently high levels of objection over many years.

The map of the proportion of children with no vaccines recorded (Figure 17) shows some additional areas not evident from, but usually adjacent to, maps of official conscientious objection. Children with no vaccines recorded and children who have parents who register as a conscientious objector are not mutually exclusive groups. Only 30% of children with no vaccines recorded were registered vaccine objectors, whilst 45% of vaccine objectors have vaccines recorded on the ACIR (not shown). Areas with low coverage that do not have high proportions of official vaccine objection nor high levels of no vaccines recorded are more likely to reflect access issues.

Figure 12: The percentage of children who received their 1st dose of DTP/Hexa vaccine at age 6 to < 8 weeks, January 2009 to December, 2011 by state or territory and month of receipt

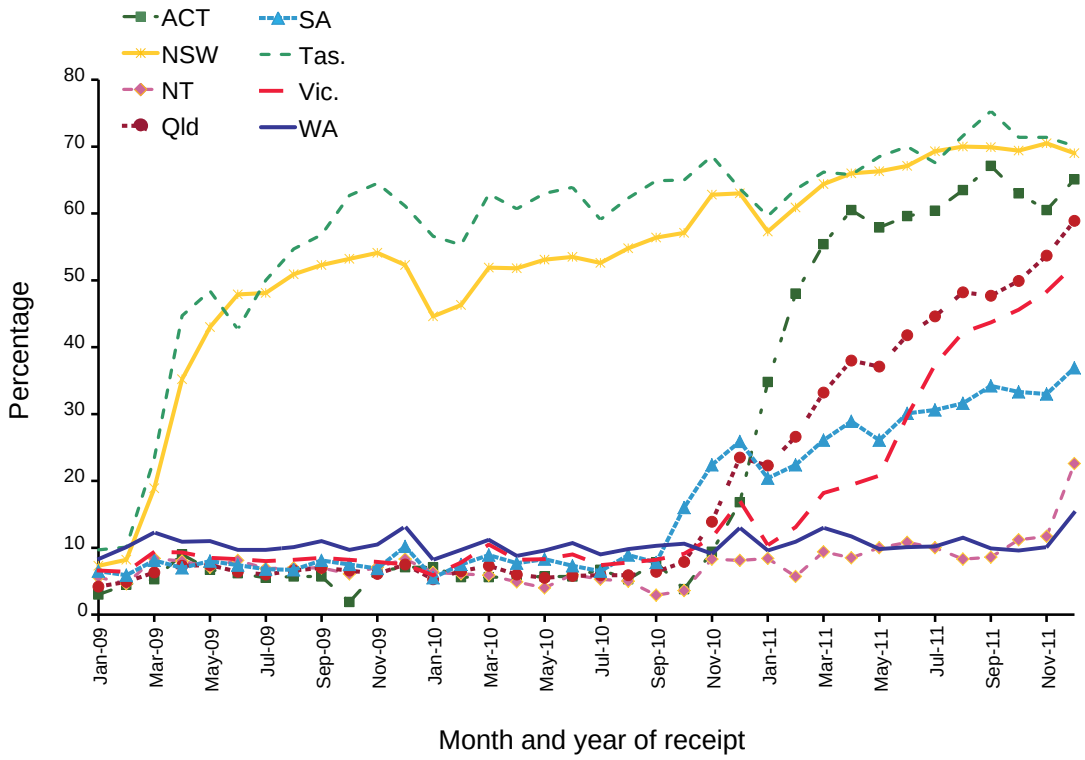
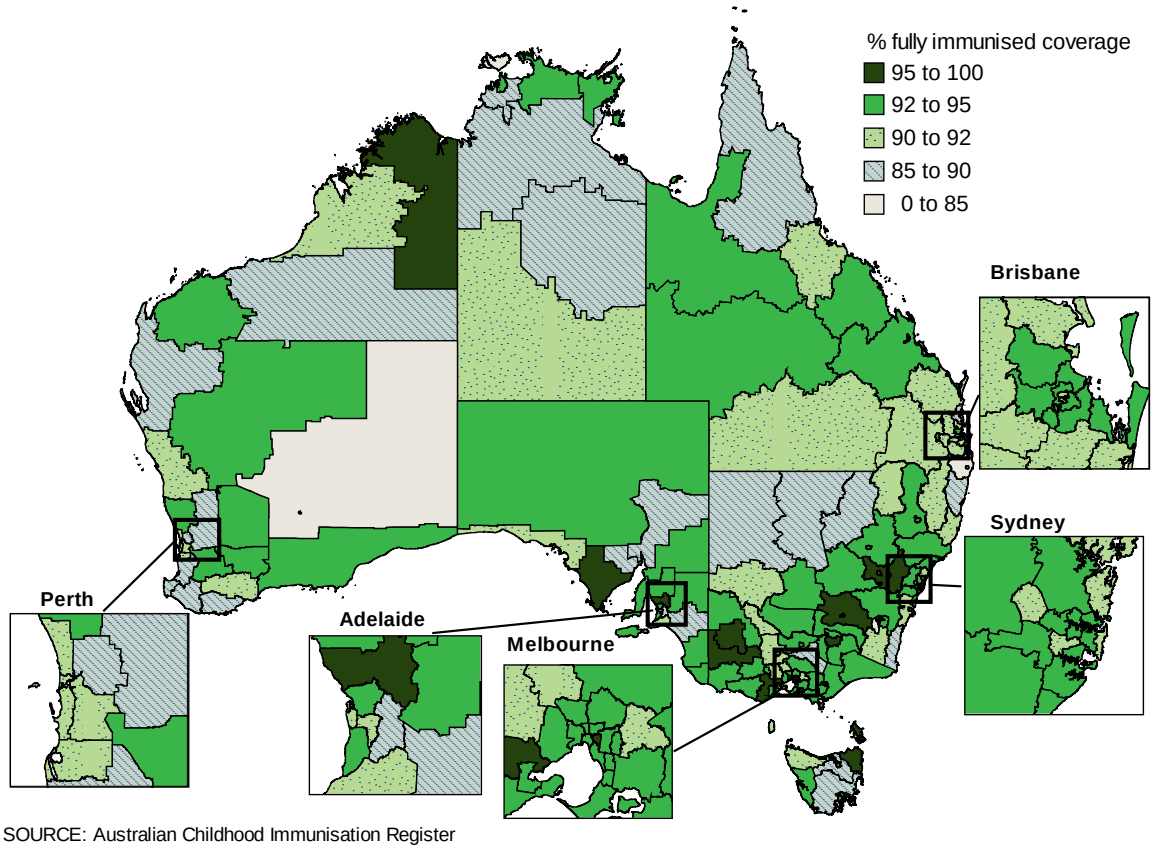
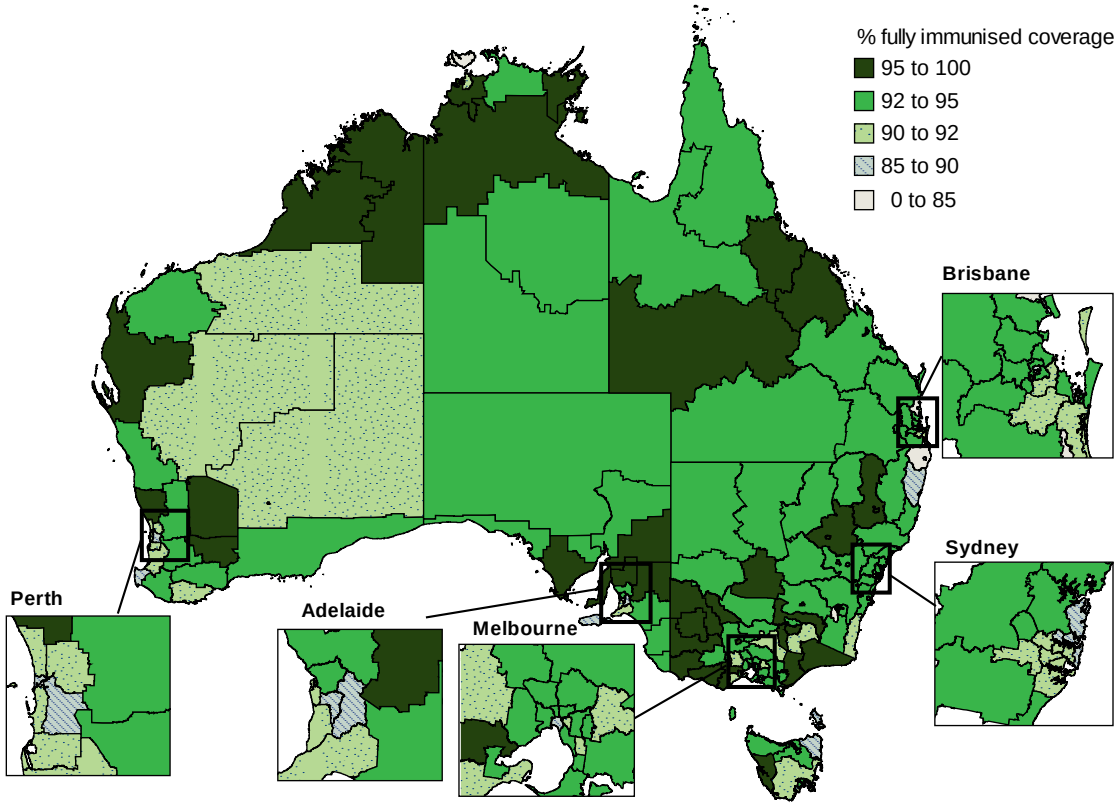


Figure 13: 'Fully immunised' coverage at 12 months of age, 2011, Australia, by Statistical Subdivision



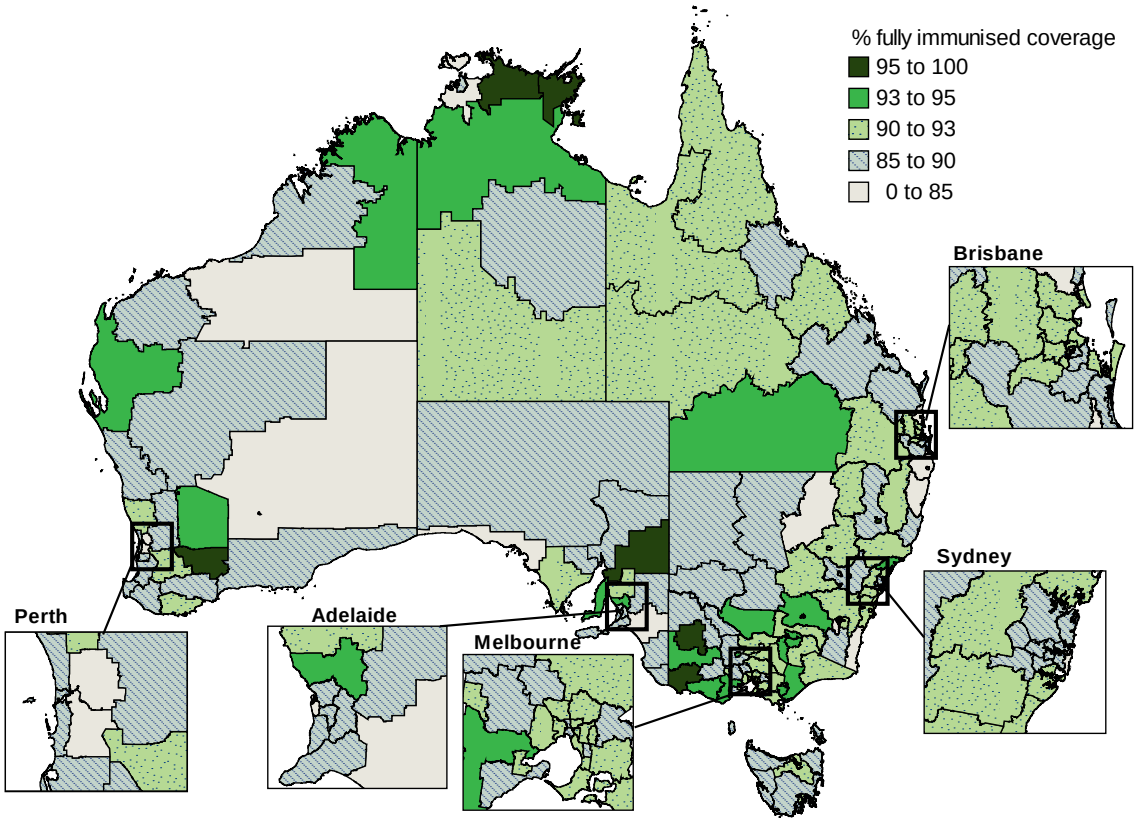
SOURCE: Australian Childhood Immunisation Register

Figure 14: 'Fully immunised' coverage at 24 months of age, Australia, 2011, by Statistical Subdivision



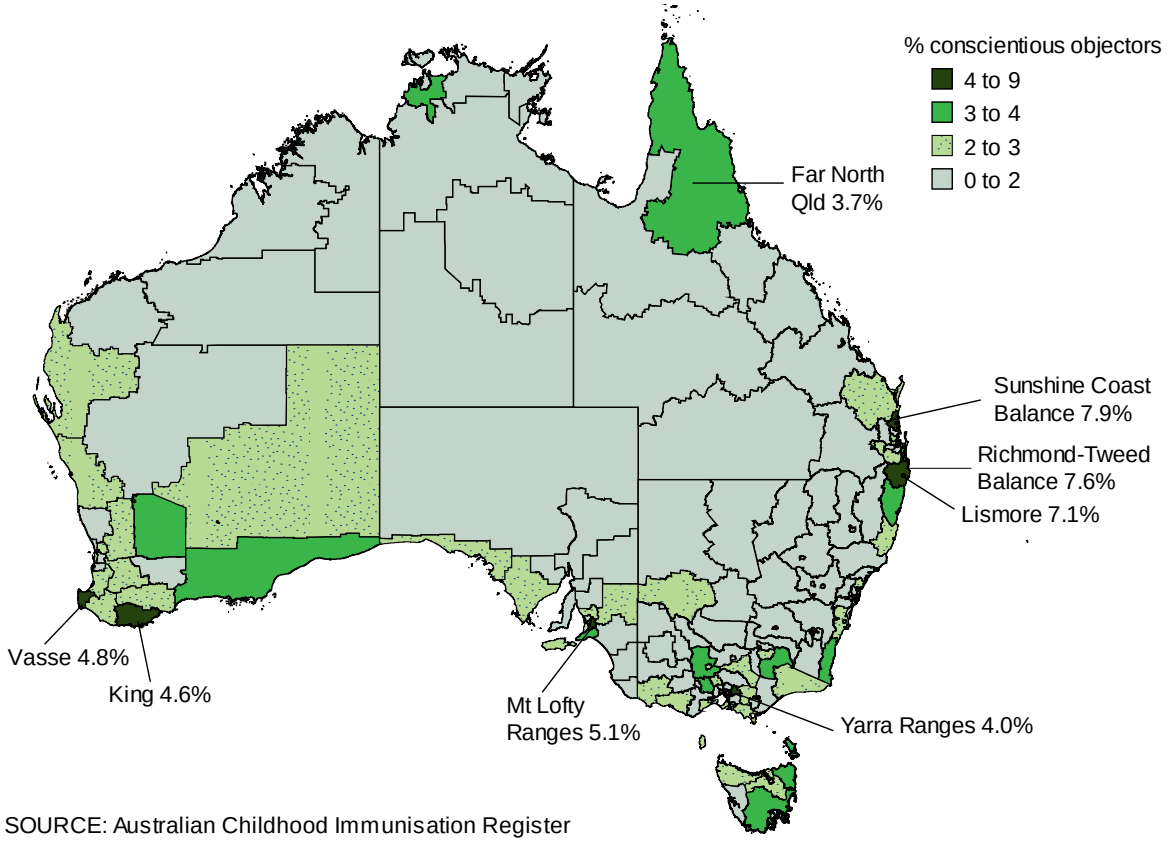
SOURCE: Australian Childhood Immunisation Register

Figure 15: 'Fully immunised' coverage at 60 months of age, Australia, 2011, by Statistical Subdivision



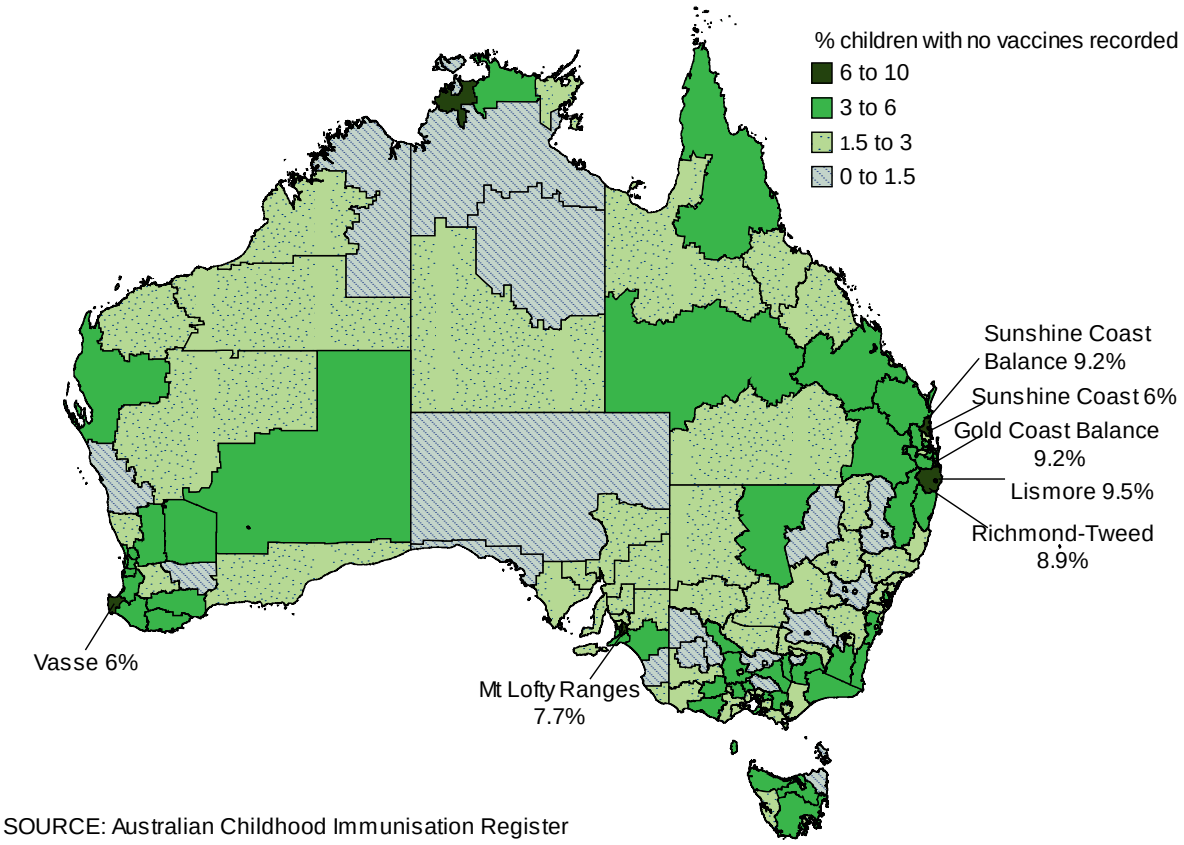
SOURCE: Australian Childhood Immunisation Register

Figure 16: Proportion registered as official conscientious objectors to immunisation, Australia, 2010 (cohort born 1 January 2005 to 31 December 2010)



SOURCE: Australian Childhood Immunisation Register

Figure 17: Proportion of children with no vaccines recorded on the Australian Childhood Immunisation Register, Australia, 2010 (cohort born 1 January 2005 to 31 December 2010)



SOURCE: Australian Childhood Immunisation Register

Provider type

GPs administer the large majority of immunisations in Australia (Figure 18); the proportion given by GPs has increased over the past 11 years by almost 5% (not shown). Local government clinics also administer a substantial proportion of immunisations, especially in some jurisdictions. The only other category of provider administering major numbers of immunisations nationally is community health centres. Regional differences are marked, with immunisations almost entirely administered by GPs in some jurisdictions (New South Wales, Queensland, South Australia, Tasmania and Western Australia), while in others a majority are given by local government (Victoria) and community health clinics (the Northern Territory).

Human papillomavirus vaccine coverage

Vaccination coverage, as notified to the HPV Register, for dose 3 of the HPV vaccine for girls

Table 9: Vaccination coverage for dose 3 of HPV vaccine for girls turning 15 years in 2011, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
HPV	73.2	72.7	79.5	70.2	66.0	64	74.5	64.8	71.2

Source: Human papillomavirus vaccination coverage data. Australian Government. Department of Health and Ageing, February 2013. Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/immunise-hpv

Includes valid doses and too close doses for Clinically Complete Consumers.

Population is Estimated Resident Population provided by the Australian Bureau of Statistics – Cat 3101.0 Australian Demographic Statistics, Tables 51 to 58: Estimated Resident Population By Single Year of Age by State and Territory, published June 2011.

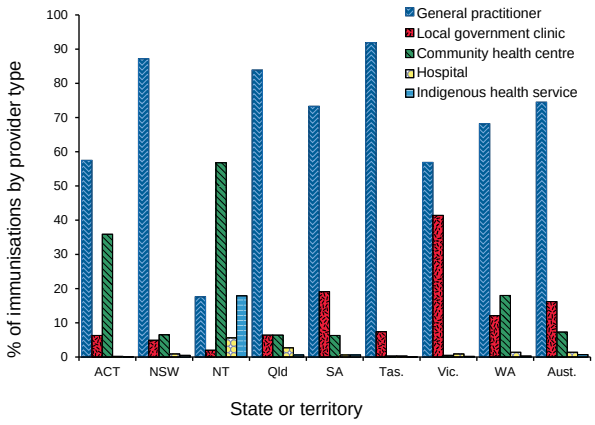
Age is age as at date of Estimated Resident Population estimate.

Coverage is calculated as doses administered and reported to the HPV Register / estimated resident population expressed as a percentage.

Excludes consumers who do not wish their details to be recorded on the HPV Register.

aged 15 years in 2011 is shown in Table 9. For Australia, 71% of girls completed a full course of the vaccine. Coverage varied by jurisdiction from a low of 64% in Tasmania to a high of 79.5% in the Northern Territory. Coverage in all age groups was higher for earlier doses, as high as 81% for the 1st dose in girls aged 14–15 (Figure 19). Coverage was higher in the younger age groups than in the older age groups, with only 39% of girls aged 20–26 years fully vaccinated according to data notified to the Register. HPV coverage by Indigenous status is not available due to limitations in Indigenous status reporting on the HPV Register.

Figure 18: Proportion of immunisations on the Australian Childhood Immunisation Register given by various provider types from 1 January to 31 December 2011, by state or territory

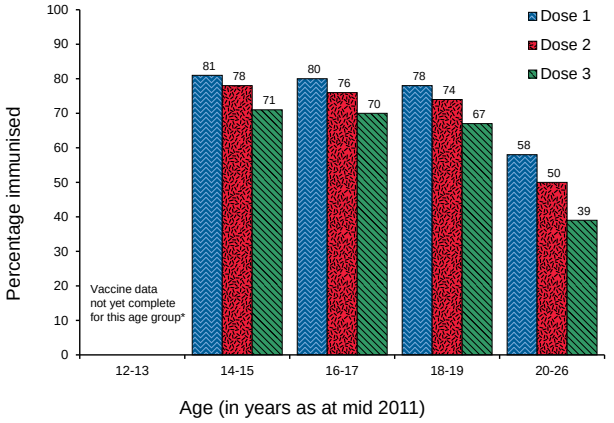


Discussion

These data show that 1993 Immunise Australia Program coverage targets (90%) were reached for children both 12 and 24 months of age in 2011. However, this is not the case for children 60 months of age where coverage, whilst much improved, is below the target in some jurisdictions.

'Fully immunised' coverage at 24 months of age exceeded that at 12 months of age, and this is likely related to the longer time available for late vaccinations to be assessed due to the exclusion of varicella vaccine at 18 months from the calculation of 'fully vaccinated', and also the absence of any other vaccines administered between those ages. There may also be an impact of immunisation incentives. National coverage for vaccines due at 48 months of age improved considerably during 2011 approaching 90% for all 4 cohorts. This increase is due to improved timeliness of vaccination, and is probably related to the change to the overdue rules

Figure 19: Human papillomavirus vaccination coverage for females, Australia, April 2007 and June 2012, by dose number



* In some states those aged 12–13 in 2011 were not eligible for vaccination until 2012. Notification of 2012 doses to the Register is in progress. Data will be published with the 2012 HPV vaccination coverage update.

Source: Human papillomavirus vaccination coverage data. Australian Government. Department of Health and Ageing, February 2013. Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/immunise-hpv

Technical notes:

12–13 years – School program – routinely vaccinated in 2011 and 2012

14–15 years – School program– routinely vaccinated in 2009 and 2010

16–17 years – Vaccinated in school catch up program 2007–2009

18–19 years – Vaccinated in school catch up program 2007–2009

20–26 years – Vaccinated in GP/community catch up program 2007–2009

Includes valid doses and too close doses for Clinically Complete Consumers

Population is Estimated Resident Population provided by the Australian Bureau of Statistics – Cat 3101.0 Australia Demographic Statistics, Tables 51 to 58.: Estimated Resident Population By Single Year of Age by State and Territory, published 2011.

Age is age as at date of Estimated Resident Population estimate

Coverage is calculated as doses administered and reported to the HPV Register/ estimated resident population expressed as a percentage

Excludes consumers who do not wish their details to be recorded on the HPV Register

in January 2009, where children became overdue for their pre-school boosters at 49 months of age instead of the previous 60 months. This change had an impact on eligibility for child care benefits for parents and outcome payments for providers. It was accompanied by a letter from Medicare Australia advising parents of the change, and the follow-up of overdue children by local health authorities. It is possible that the splitting of the

Maternity Immunisation Allowance at that time could have had an impact in these data, as it applies to children turning 48 months from 2011 onwards.

There is earlier evidence that immunisation incentives to providers positively impacted on coverage estimates.[15] However, the initial analyses in this report provide no evidence of a reduction in coverage associated with the removal of SIP payments in October 2008, while coverage at 60 months has increased following the due and overdue rules changes. However, more analysis is required to examine the impact of these changes in more detail.

A number of vaccines that are included in the NIP are not included when calculating 'fully immunised' status or in eligibility for incentive payments. Coverage estimates for PCV and meningococcal C vaccines are comparable with estimates for vaccines that are included in 'fully vaccinated' calculations, but estimates for varicella and rotavirus are still substantially lower. During 2011, there were only slight changes in coverage for varicella (from 83% to 83.9%) and rotavirus vaccine (from 85% to 84%). For rotavirus vaccines, strict upper age limits for administration may explain lower coverage, whilst varicella is the only vaccine due at 18 months, and this milestone was historically problematic and lapsed for a 2 year period (2003–2005). The implications also vary. In the case of rotavirus vaccine, coverage of 80% or greater has been associated with substantial herd immunity and decreases in rotavirus hospitalisations in Australia and elsewhere.[28,29] In contrast, modelling studies suggest that low coverage with varicella vaccine may result in a shift of disease to older age groups with higher disease severity.[29] This has changed from July 2013 with the inclusion of PCV, Men C, and varicella (as MMRV) in the algorithms used to calculate fully immunised coverage at 12 and 24 months of age.

Coverage for vaccines recommended for Indigenous children only (i.e. hepatitis A and 23vPPV) remained sub-optimal during 2011 but increased substantially for the 23vPPV vaccine (nationally, from 56% to 63%). The extent of under-reporting to the ACIR for these vaccines is unknown but may be more than for 'universal' vaccines, given the lack of incentive payments for notification to the ACIR. However, lower coverage for vaccines targeted at Indigenous people has been a relatively consistent finding using a range of different methods for both children[14] and adults.[31] Both a lack of provider knowledge about the recommendations for high risk groups, and poor identification of Indigenous children by immunisation providers are likely to be important contributing factors. Differences in schedules between jurisdictions may also contribute. During 2011, coverage for both vaccines was still higher in the Northern Territory and Western Australia, which give the vaccines 6 months younger (hepatitis A, 12 and 18 months, 23vPPV 18 months), than in South Australia (18 and 24, and 24 months). However, coverage for both vaccines in 2011 for Queensland was similar to that in Western Australia even though the vaccines are given at 6 months older in Queensland. The presence of other vaccines on the schedule at the same age may assist achieving higher coverage at 12 months and 18 months of age. Failure to receive a 2nd dose by 6% of children also contributed to the low coverage for hepatitis A vaccine. However, a protective antibody response after 1 dose is expected from a majority of children.[32]

Although coverage data reveal that most children eventually complete the scheduled vaccination series by the 24-month milestone, many still do not do so in a timely manner. On-time vaccination in 2011 as measured in this report for vaccines assessed at 12 and 24 months of age has improved only marginally. However, timeliness cannot be measured in the most recent cohort, as time must be allowed for late vaccination to be received. Poorer timeliness in Indigenous children has been noted previously in infants. Timeliness has improved markedly at 60 months of age for both Indigenous and non-Indigenous children. However, as coverage and timeliness of vaccines assessed at 60 months of age has improved, the disparity in timeliness between Indigenous and non-Indigenous children has increased, as improvements in non-Indigenous children were not fully duplicated in Indigenous children. Delayed vaccination is a concern, especially for diseases where multiple vaccine doses are required for protection and the disease risk among young infants is significant (e.g. pertussis). Immunisation at the earliest appropriate age should be a public health goal for countries such as Australia where high levels of vaccine coverage at milestone ages have been achieved.

The ACIR has shown the rapid uptake of new vaccines and consistently high coverage for all vaccines, unlike some other developed countries.[33,34] In comparison with similar countries, reported coverage at 12 months of age is higher,[33] and, with almost 2% of children not vaccinated due to parental objection, targeting of on time vaccination is required to significantly improve the current levels of greater than 91% 'fully immunised' at 12 months of age. Areas of low coverage have been identified in many remote areas and areas containing higher proportions of vaccine objectors. Further vaccination coverage estimates in small areas has been provided by the National Health Performance Authority for children in 2011–12.[35]

Coverage data for HPV from the national HPV register reflect a successful school-based program

with lower coverage for the catch-up program.[36,37] Under-notification to the HPV Register of doses administered in general practice and the community contributes to the apparently lower coverage in women currently aged over 20 years, with independent coverage estimates from population surveys in this age group suggesting under-notification of around 5%–15%.[37,38] The approximate 10% drop in coverage between dose 1 and 3 may also reflect under-notification of doses missed in school and caught up in general practice but not notified to the register, as well as demonstrating the challenges in delivering a three dose vaccination course to adolescents.

Australia's HPV vaccination program remains the most broadly targeted program in the world, with no other country having provided a free catch up program up to the age of 26 years. The coverage achieved in the program has been sufficient to result in demonstrable decreases in HPV prevalence in young women,[39] genital warts[40] and cervical abnormalities.[41]

Unfortunately, coverage data are not available for Indigenous adolescents. For adults, data are only available from the Aboriginal and Torres Strait Islander Health Survey, last conducted in 2004/05.[42]

Data provided in this report reflect continuing successful delivery of the NIP in Australia, while identifying some areas for improvement. Coverage for varicella and rotavirus vaccines is below that for other vaccines, and is low in some small geographic areas. Timeliness of vaccination could be improved, particularly for Indigenous infants, and coverage for vaccines recommended only for Indigenous infants is lower than for other vaccines. From July 2013, varicella and other NIP vaccines (meningococcal C and pneumococcal conjugate vaccines) will be included in coverage assessments for 'fully immunised', and thereby in eligibility for provider and parent incentives.[43] It will be important to evaluate the impact of this change in coming years and given the encouraging improvements in timely coverage seen with the changes to reimbursement introduced in 2009 for the 48-month milestone, this promises to have a favourable impact especially for varicella vaccine where high coverage is crucial to long-term outcomes of the program.

Author details

Brynley P Hull[1]
Aditi Dey[1]
Rob I Menzies[1]
Julia M Brotherton[2]
Peter B McIntyre[1]

1. National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, The Children's Hospital at Westmead and University of Sydney, Westmead, New South Wales
2. Victorian Cytology Service Registries: (National HPV Vaccination Program Register and Victorian Cervical Cytology Registry)

Corresponding author: Mr Brynley Hull, National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, The Children's Hospital at Westmead and University of Sydney, Locked Bag 4001, WESTMEAD NSW 2145. Email: brynley.hull@health.nsw.gov.au

Abbreviations

7vPCV	7-valent pneumococcal conjugate vaccine
10vPCV	10-valent pneumococcal conjugate vaccine
13vPCV	13-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
ABS	Australian Bureau of Statistics
ACIR	Australian Childhood Immunisation Register
ARIA	Accessibility/Remoteness Index of Australia
ATAGI	Australian Technical Advisory Group on Immunisation
AVS	Adult Vaccination Survey
DTP	diphtheria-tetanus-pertussis
DTPa	Diphtheria-tetanus-pertussis (acellular) (child formulation)
DTP/Hexa	Diphtheria-tetanus-pertussis-inactivated polio-Hib-hepatitis B vaccine
GP	general practitioner
GPII	General Practice Immunisation Incentives Scheme
HBVAX II	hepatitis B (paediatric) vaccine
Hep A	hepatitis A virus
Hep B	hepatitis B virus
Hib	*Haemophilus influenzae* type b
HPV	human papillomavirus
IPV	inactivated poliovirus vaccine
MenC	meningococcal C conjugate vaccine
MIA	Maternity Immunisation Allowance
MMR	measles-mumps-rubella
MMRV	measles-mumps-rubella-varicella
NIP	National Immunisation Program
PCV	pneumococcal polysaccharide vaccine
PRP-D	*Haemophilus influenzae* type b polysaccharide conjugated to diphtheria toxoid
PRP-OMP	*Haemophilus influenzae* type b polysaccharide conjugated to the outer membrane protein of *Neisseria meningitidis* vaccine
PRP-T	*Haemophilus influenzae* type b polysaccharide conjugated to tetanus toxoid
SIP	Service Incentive Payment
SSD	Statistical subdivisions
VZV	Varicella-zoster virus

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Australia's notifiable disease status, 2011: Annual report of the National Notifiable Diseases Surveillance System

NNDSS Annual Report Writing Group

Abstract

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

Keywords: Australia, communicable diseases, epidemiology, surveillance

Introduction

Australia's notifiable diseases status, 2011, is an annual surveillance report of nationally notifiable communicable diseases. Communicable disease surveillance in Australia operates at the national, jurisdictional and local levels. Primary responsibility for public health action lies with the state and territory health departments. The role of communicable disease surveillance at the national level includes:

- identifying national trends;
- providing guidance for policy development and resource allocation at the national level;
- monitoring the need for and impact of national disease control programs;
- coordinating the response to national or multi-jurisdictional outbreaks;
- describing of the epidemiology of rare diseases that occur infrequently at state and territory levels;
- meeting various international reporting requirements, such as providing disease statistics to the World Health Organization (WHO); and
- supporting quarantine activities, which are the responsibility of the Commonwealth government.

Methods

Australia is a federation of 6 states (New South Wales, Queensland, South Australia, Tasmania, Victoria and Western Australia) and 2 territories (the Australian Capital Territory and the Northern Territory).

State and territory health departments collect notifications of communicable diseases under their respective public health legislation. In September 2007, the National Health Security Act 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 2011 states and territories forwarded de-identified data on the nationally agreed set of 65 communicable diseases to the Australian Government Department of Health for the purposes of national communicable disease surveillance, although not all 65 diseases were notifiable in each jurisdiction. Data were updated electronically from states and territories, daily or several times a week. The system was complemented by other surveillance systems, which provided information on various diseases, including four that are not reported to National Notifiable Diseases Surveillance System (NNDSS) (HIV, AIDS and the classical and variant forms of Creutzfeldt-Jakob disease (CJD)).

In 2011, the NNDSS core dataset included the following 5 mandatory data fields: unique record reference number; notifying state or territory; disease code; confirmation status and the date when the jurisdictional health department was notified (notification receive date). In addition, the following core but non-mandatory data fields were supplied where possible: date of birth; age at onset;

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

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

Newly diagnosed HIV infection and AIDS were notifiable conditions in each state or territory health jurisdiction in 2011. These were forwarded to the Kirby Institute for infection and immunity in society. Further information can be found in the Kirby Institute's annual surveillance report.[4]

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

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

Notification rates for each notifiable disease were calculated using the estimated 2011 mid-year resident population supplied by the Australian Bureau of Statistics (Appendix 1 and Appendix 2).[6] Where diseases were not notifiable in a state or territory, national rates were adjusted by excluding the population of that jurisdiction from the denominator. For some diseases, age adjusted rates were calculated using the direct method of standardisation, with 2006 census data as the standard population. All rates are represented as the rate per 100,000 unless stated otherwise.

Notes on interpretation

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

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

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

Methods of surveillance vary between states and territories, each having different requirements for notification by medical practitioners, laboratories and hospitals. Although the National Notifiable Diseases List[2] was established, some diseases are not notifiable in all 8 jurisdictions (Table 1).

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

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

Figure 1: Communicable diseases notifiable fraction

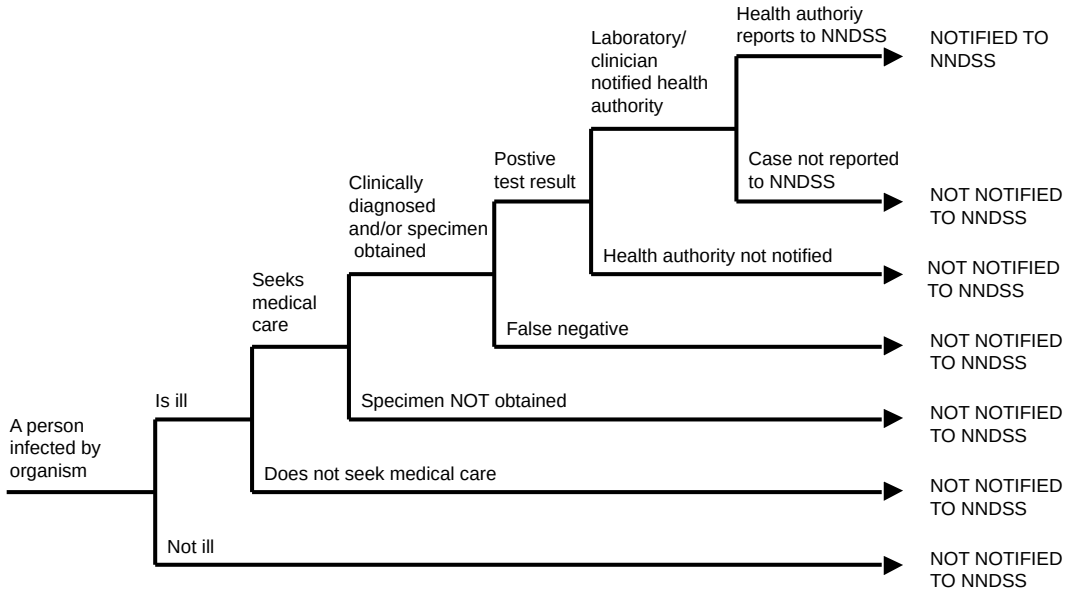


Table 1: Diseases notified to the National Notifiable Diseases Surveillance System, Australia, 2011

Disease	Data received from
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions, except Western Australia
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions, except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions, except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
STEC, VTEC*	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions

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

Disease	Data received from
Sexually transmissible infections	
Chlamydial infections	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years or unspecified duration	All jurisdictions, except South Australia
Syphilis – congenital	All jurisdictions
Vaccine preventable diseases	
Diphtheria	All jurisdictions
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.

Data completeness was assessed for cases' sex, age at onset, and Indigenous status, and reported as the proportion of complete notifications. The completeness of data in this report is summarised in the Results.

The per cent of data completeness was defined as:

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

The Indigenous status was defined by the following nationally accepted values:[7]

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

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

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

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

9=Not stated

Notes on case definitions

Each notifiable disease is governed by a national surveillance case definition for reporting to the NNDSS. These case definitions were agreed by CDNA and implemented nationally in January 2004 and were used by all jurisdictions for the first time in 2005. These case definitions are reviewed by the Case Definitions Working Group (CDWG) as required.

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

Results

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

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

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

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

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

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

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

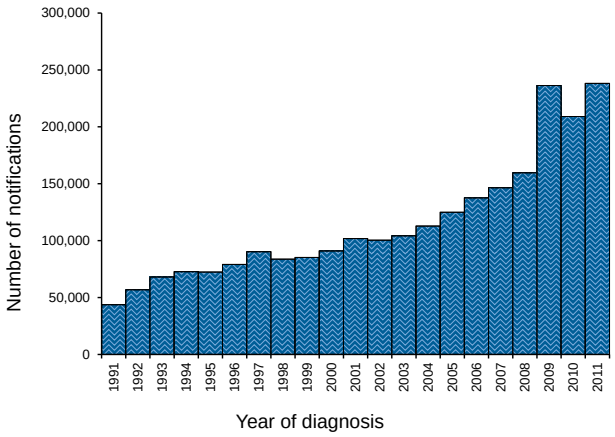


Table 3: Notifications of communicable diseases, Australia, 2011, by state or territory

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

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

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

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

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

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

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

|| The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

¶ Does not include congenital syphilis.

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

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

NEC Not elsewhere classified.

NN Not notifiable.

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

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

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

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

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

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

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

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

|| The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

¶ Does not include congenital syphilis.

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

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

NEC Not elsewhere classified.

NN Not notifiable.

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

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

Disease	Number of notifications						5-year mean	Ratio	Notification rate per 100,000 population					
	2006	2007	2008	2009	2010	2011			2006	2007	2008	2009	2010	2011
Bloodborne diseases														
Hepatitis (NEC)	1	0	1	0	0	0	0.4	<0.1	<0.1	–	<0.1	–	–	–
Hepatitis B (newly acquired)*	291	296	259	241	228	190	263.0	0.7	1.4	1.4	1.2	1.1	1.0	0.8
Hepatitis B (unspecified)†	6,168	6,783	6,444	7,015	6,960	6,629	6674.0	1.0	29.8	32.2	30.0	32.0	31.2	29.3
Hepatitis C (newly acquired)*	437	379	363	398	401	400	395.6	1.0	2.6	2.2	2.1	2.3	2.3	2.2
Hepatitis C (unspecified)†	11,689	11,675	10,956	10,871	10,916	9,861	11221.4	0.9	56.5	55.4	51.0	49.5	49.0	43.6
Hepatitis D	29	33	41	35	34	43	34.4	1.3	0.1	0.2	0.2	0.2	0.2	0.2
Gastrointestinal diseases														
Botulism	1	1	0	1	0	2	0.6	3.3	<0.1	<0.1	–	<0.1	–	<0.1
Campylobacteriosis	15,416	16,980	15,539	16,075	16,968	17,717	16,195.6	1.1	111.1	119.9	107.3	108.4	112.5	115.7
Cryptosporidiosis	3,201	2,808	2,003	4,624	1,478	1,808	2,822.8	0.6	15.5	13.3	9.3	21.1	6.6	8.0
Haemolytic uraemic syndrome	14	19	32	13	9	13	17.4	0.7	0.1	0.1	0.1	0.1	<0.1	0.1
Hepatitis A	281	166	277	564	267	144	311.0	0.5	1.4	0.8	1.3	2.6	1.2	0.6
Hepatitis E	24	18	44	33	37	40	31.2	1.3	0.1	0.1	0.2	0.2	0.2	0.2
Listeriosis	61	50	68	92	71	70	68.4	1.0	0.3	0.2	0.3	0.4	0.3	0.3
Salmonellosis	8,215	9,461	8,289	9,509	11,924	12,267	9,479.6	1.3	39.7	44.9	38.6	43.3	53.5	54.2
Shigellosis	544	596	828	616	551	494	627.0	0.8	2.6	2.8	3.9	2.8	2.5	2.2
STEC,VTEC‡	67	105	98	128	80	95	95.6	1.0	0.3	0.5	0.5	0.6	0.4	0.4
Typhoid	77	90	105	115	96	134	96.6	1.4	0.4	0.4	0.5	0.5	0.4	0.6
Quarantinable diseases														
Cholera	3	4	4	5	3	6	3.8	1.6	<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	–	–	–	–	–	–	–
Plague	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Rabies	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Severe acute respiratory syndrome	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Smallpox	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Viral haemorrhagic fever	0	0	0	0	0	0	0.0	–	–	–	–	–	–	–
Yellow fever	0	0	0	0	0	2	0.0	–	–	–	–	–	–	<0.1

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

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

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

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

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

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

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

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

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

|| The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

¶ Does not include congenital syphilis.

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

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

NEC Not elsewhere classified.

NN Not notifiable.

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

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

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

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

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

Disease	Year in which data first sent to Commonwealth								Period of national reporting	Exceptions to national reporting
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA		
Sexually transmissible infections										
Chlamydial infection (NEC)	1993	1991	1991	1991	1993	1991	1991	1993	1994 to present	NSW did not report 1994–1998
Donovanosis	1991	2002	1991	1991	2002	1993	1991	1991	1991 to present	NSW and SA did not report 1991–2001 Tasmania did not report 1991–1992
Gonococcal infection§	1991	1993	1991	1991	1991	1991	1991	1991	1991 to present	
Syphilis – all‖	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Syphilis < 2 years	2004	2004	2004	2004	2004	2004	2004	2004	2004 to present	
Syphilis > 2 years or unspecified duration	2004	2004	2004	2004	–	2004	2004	2004	2004 to present	South Australia do not report
Syphilis – congenital	2003	2003	2003	2003	2003	2003	2003	2003	2003 to present	
Vaccine preventable diseases										
Diphtheria	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Haemophilus influenzae type b	1991	1991	1991	1991	1991	1991	1991	1994	1991 to present	WA did not report 1991–1993
Influenza (laboratory confirmed)	2001	2001	2001	2001	2001	2001	2001	2001	2001 to present	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	1997–1998; 2002	1994	1995	1992	1994	1995 to present	Queensland did not report (1995–1996 & 1999–2000)
Pertussis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Pneumococcal disease (invasive)	2001	2001	2001	1997	2001	2001	2001	2001	2001 to present	
Poliomyelitis	1991	1991	1991	1991	1991	1991	1991	1991	1991 to present	
Rubella¶	1991	1991	1993	1991	1993	1995	1992	1994	1993 to present	Tasmania did not report 1993–1994
Rubella – congenital	2003	2003	2003	1997	2003	2003	2003	2003	2003 to present	
Tetanus	1991	1991	1991	1985	1991	1991	1991	1991	1991 to present	Qld did not report 1991–1993
Varicella zoster (chickenpox)	2006	NN	2006	2006	2006	2006	2008	2006	2006 to present	All jurisdictions except NSW Reported by Victoria in September 2008
Varicella zoster (shingles)	2006	NN	2006	2006	2006	2006	2008	2006	2006 to present	All jurisdictions except NSW Reported by Victoria in September 2008
Varicella zoster (unspecified)	2006	NN	2006	2006	2006	2006	2008	2006	2006 to present	All jurisdictions except NSW Reported by Victoria in September 2008

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

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

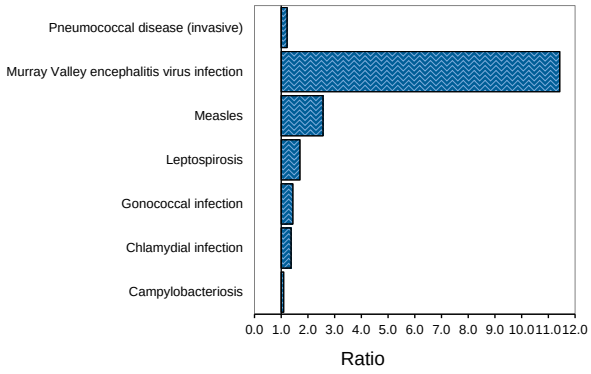
** Before 1997, includes Ross River virus infection, dengue virus infection and Barmah Forest virus infection.

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

NN Not notifiable

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

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



Data completeness

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

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

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

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

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

Bloodborne diseases

In 2011, the bloodborne viruses reported to the NNDSS were hepatitis B, C, and D. Both hepatitis B and C cases were notified to the NNDSS as either 'newly acquired', where evidence was available that the infection was acquired within 24 months prior to diagnosis; or 'greater than 2 years or unspecified' period of infection. These categories were reported from all states and territories except Queensland where all cases of hepatitis C, including newly acquired, were reported as 'greater than 2 years or unspecified'. The determination of a case as being 'newly acquired' was heavily reliant on public health follow-up, with the method and intensity of follow-up varying by jurisdiction and over time.

In interpreting these data it is important to note that changes in notifications over time may not solely reflect changes in disease prevalence or incidence. Testing policies[8] and screening programs, including the preferential testing of high risk populations such as persons in prison, injecting drug users and persons from countries with a high prevalence of hepatitis B or C, may contribute to these changes.

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

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

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

Hepatitis B

Hepatitis B notifications are classified as either 'newly acquired' or 'unspecified' as described above. The classification of hepatitis B cases is primarily based on serological evidence or evidence of a previously negative test within the 24 months prior to diagnosis.

Epidemiological situation in 2011

In 2011, there were 6,819 notifications of hepatitis B (both newly acquired and unspecified), equating to a rate of 30.1 per 100,000 (Figure 4). The Northern Territory recorded the highest hepatitis B rate in 2011 (70.8 per 100,000), followed by Victoria (35.3 per 100,000) and New South Wales (34.7 per 100,000).

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

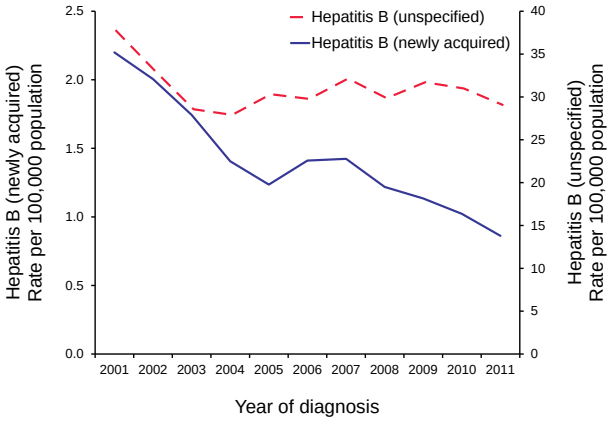
Newly acquired hepatitis B

Epidemiological situation in 2011

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

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

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



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

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

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

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

Geographic distribution

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

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

Age and sex distribution

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

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

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

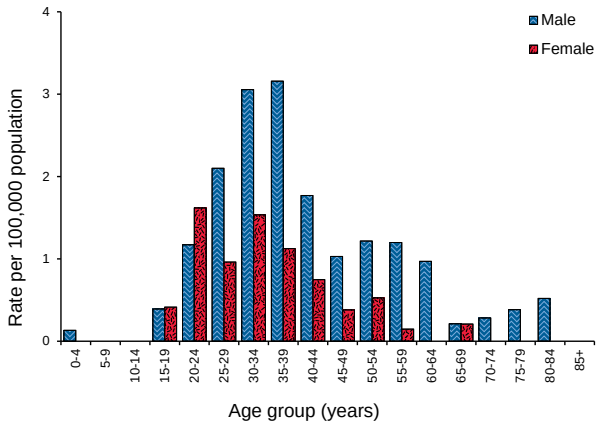
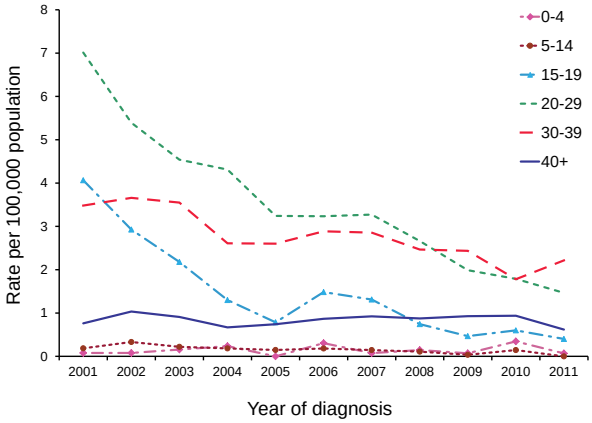


Figure 6: Notification rate for newly acquired hepatitis B,* Australia, 2001 to 2011, by year and age group



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

Risk groups

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

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

Unspecified hepatitis B notifications

Epidemiological situation in 2011

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

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

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

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

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

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

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

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

‖ Includes both occupational and non-occupational exposures.

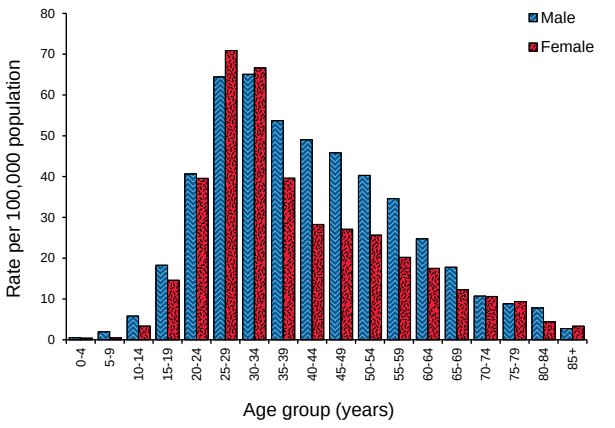
the rate has remained relatively stable with an average annual rate of 31 per 100,000 during this time. (Figure 4).

Age and sex distribution

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

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

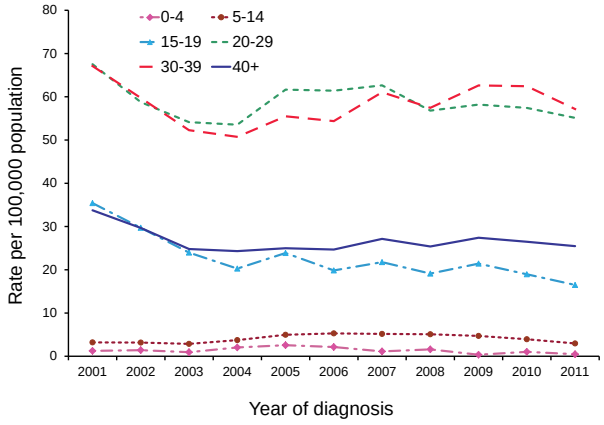
Figure 7: Notification rate for unspecified hepatitis B,* Australia, 2011, by age group and sex



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

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

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



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

† Excludes notifications for whom age was not reported.

Hepatitis C

Hepatitis C notifications are classified as either 'newly acquired' (infection acquired within 24 months prior to diagnosis) or 'unspecified' (infection acquired more than 24 months prior to diagnosis or not able to be specified). Current testing methods cannot distinguish between newly acquired (incident) and chronic infections (greater than 2 years or unspecified). The identification of newly acquired cases is therefore dependent on evidence of a negative test result within 24 months prior to laboratory diagnosis or clinical hepatitis within the 24 month prior to a positive diagnostic test where other causes of acute hepatitis have been excluded. Ascertainment of a person's hepatitis C testing and clinical history usually requires active follow-up by public health units. Although initial infection with the hepatitis C virus is asymptomatic or mildly symptomatic in more than 90% of cases, approximately 50%–80% of cases will go on to develop a chronic infection. Of those who develop a chronic infection, half will eventually develop cirrhosis or cancer of the liver.[4]

Epidemiological situation in 2011

Between 2001 and 2011, total hepatitis C notification rates declined by 51% (93 to 45 per 100,000), with the greatest reductions observed in the earlier years, (a 16% decline between 2001 and 2002) (Figure 9). In 2011, it was estimated that 304,000 people living in Australia had been exposed to the hepatitis C virus. Of these, approximately 179,900 had chronic hepatitis C infection and early liver disease, 49,500 had chronic hepatitis C infection with moderate liver disease, 6,300 were living with hepatitis C related cirrhosis and 77,300 had cleared their infection.[4]

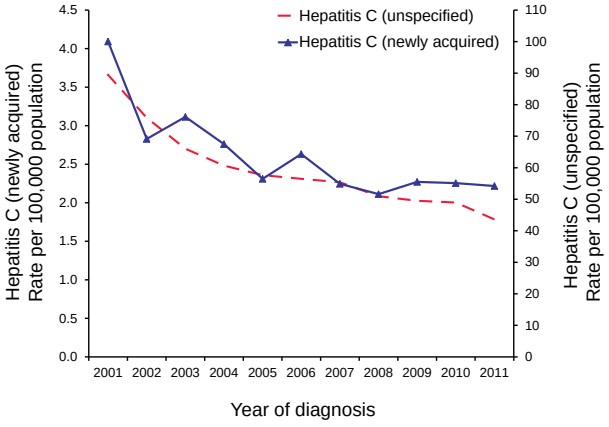
Newly acquired hepatitis C notifications

Cases of newly acquired hepatitis C were reported from all states and territories except Queensland, where all cases of hepatitis C are reported as unspecified.

Epidemiological situation in 2011

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

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



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

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

Geographic distribution

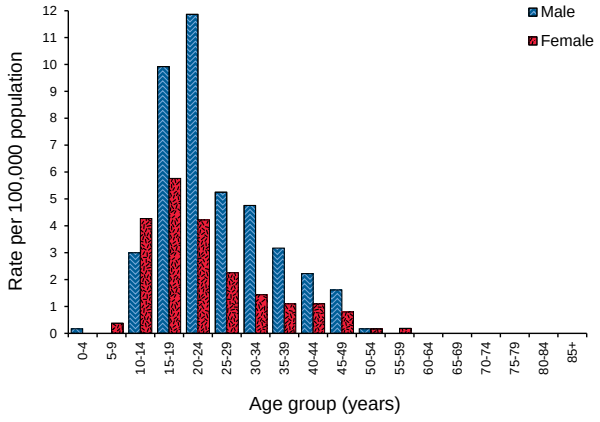
The proportion of infections that were newly acquired compared with total hepatitis C diagnoses varied substantially between the states and territories ranging from 1.4% in the Northern Territory to 11.8% in Tasmania. The highest rates of newly acquired hepatitis C infection were reported in Tasmania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

(2.9 per 100,000). The identification and classification of newly acquired hepatitis C is reliant upon public health follow-up to identify testing and clinical histories. The method and extent of case follow-up and the population groups targeted vary between states and territories, with newly acquired infection more likely to be detected in population groups that are tested frequently, such as those in prison settings.

Age and sex distribution

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

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



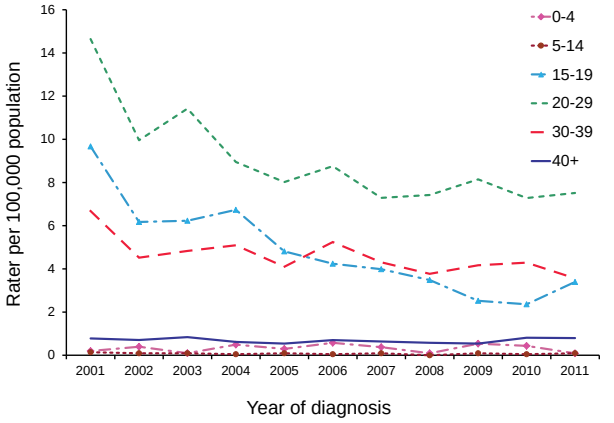
* Data from all states and territories except Queensland.

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

Risk groups

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

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



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

factors respectively noting that screening rates are generally higher in the prison entry population than the general population. A screening survey of prison entrants conducted over a 2-week period in 2010 found that the prevalence of hepatitis C based on hepatitis C antibody detection was 22%, a decrease compared with the 35% reported in 2007.[11]

Unspecified hepatitis C notifications

Epidemiological situation in 2011

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

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

Geographic distribution

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

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

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

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

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

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

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

‖ Includes both occupational and non-occupational exposures.

Age and sex distribution

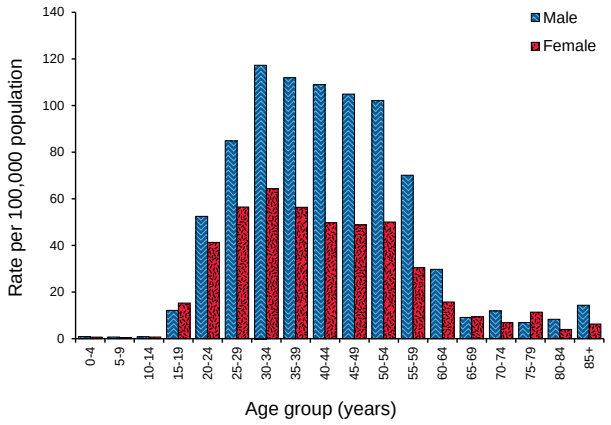
The male to female ratio remained consistent with historical trends at 1.8:1 in 2011. Amongst males, rates were highest across age groups between 30 and 54 years ranging from 102 to 118 per 100,000. Similarly, rates for females were highest amongst adults in the 30–34 year age group (64 per 100,000)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

Between 2001 and 2011 the rate of unspecified hepatitis C has declined in all age groups with the biggest decreases occurring in the 15–19 year (81%), 20–29 year (70%) and the 30–39 year (50%) age groups; the majority of this decline occurred in the early part of the decade (Figure 13). Trends in the 0–4, 5–14 and the 40 years or over age groups have remained relatively stable over this time (Figure 13).

Hepatitis D

Hepatitis D is a defective single-stranded RNA virus that replicates in the presence of the hepatitis B virus. Hepatitis D infection can occur either as a co-infection with hepatitis B or as a super-infection with chronic hepatitis B infection.[14] The modes of hepatitis D transmission are similar to those for hepatitis B. In countries with low hepatitis B prevalence, injecting drug users are the main group at risk for hepatitis D.

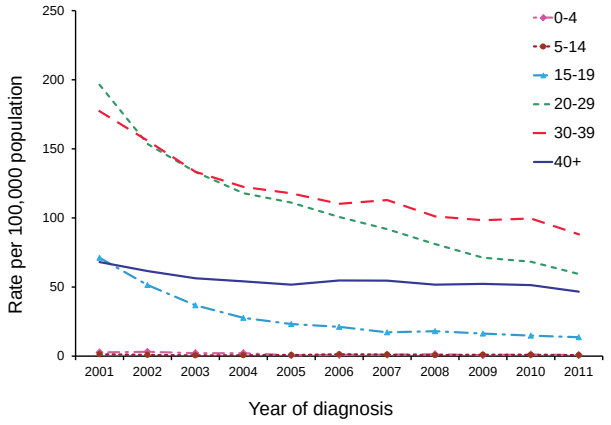
Figure 12: Notification rate for unspecified hepatitis C,*,† Australia, 2011, by age group and sex



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

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

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



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

† Excludes notifications for whom age was not reported.

Epidemiological situation in 2011

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

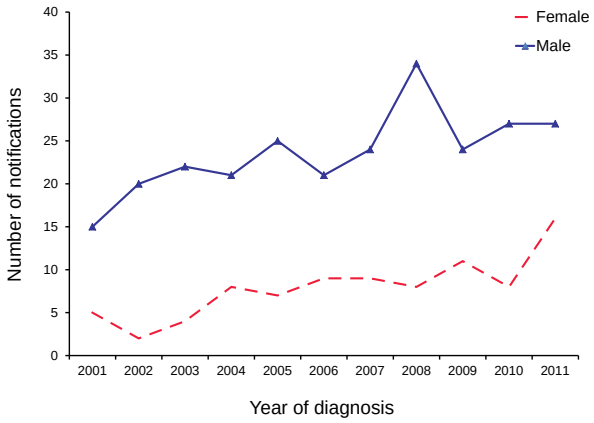
Geographic distribution

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

Sex distribution

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

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



Gastrointestinal diseases

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

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

Surveillance system overview

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

Botulism

Botulism is a rare but extremely serious intoxication resulting from toxins produced by *Clostridium botulinum* (commonly toxin types A, B and E). Three forms of botulism are recognised; infant, foodborne and wound.[14]

Epidemiological situation in 2011

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

Campylobacteriosis

The bacterium *Campylobacter* is a common cause of foodborne illness (campylobacteriosis) in humans. The severity of this illness varies and is characterised by diarrhoea (often bloody stools), abdominal pain, fever, nausea and/or vomiting.[14] Campylobacteriosis is notifiable in all Australian jurisdictions, except New South Wales.

Epidemiological situation in 2011

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

Age and sex distribution

Notification rates were highest amongst males in nearly all age groups. The highest age-specific rate for both males and females was in the 0–4 year age group (221.6 and 165.2 per 100,000, respectively) with secondary peaks occurring in the 20–24 year and 70 years or over age groups (Figure 15).

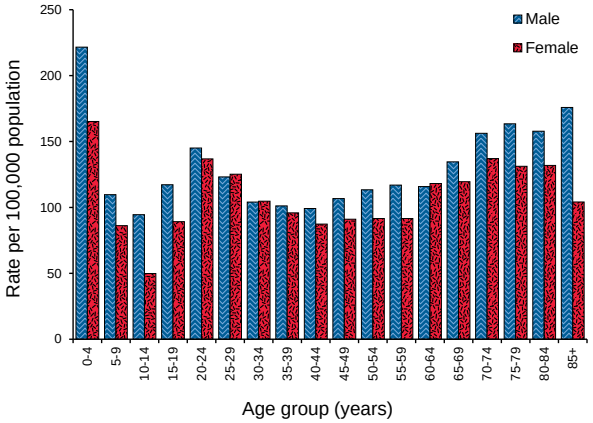
Cryptosporidiosis

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

Epidemiological situation in 2011

In 2011, there were 1,808 notifications of cryptosporidiosis; a national rate of 8 per 100,000. This represents a 22% increase over the 1,479 notifications reported in 2010; however it is below the 5-year historical mean of 2,823 notifications. Notification rates ranged from 3.6 per 100,000 in the Australian Capital Territory to 40.8 per 100,000 in the Northern Territory.

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



Age and sex distribution

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

Haemolytic uraemic syndrome

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

Epidemiological situation in 2011

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

Age and sex distribution

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

Hepatitis A

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

Epidemiological situation in 2011

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

Age and sex distribution

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

Indigenous status

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

Place of acquisition

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

In 2011, 39 notified cases were locally acquired. This was a decrease from 2010 where 111 notified cases were locally acquired (Table 10). In 2009–2010 an outbreak associated with the consumption of semi-dried tomatoes contributed to an increase in locally acquired hepatitis A cases in both 2009 and 2010.[16]

Hepatitis E

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

Epidemiological situation in 2011

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

Age and sex distribution

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

Place of acquisition

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

Listeriosis

Invasive listeriosis is caused by a bacterial infection that commonly affects the elderly or immunocompromised, and typically occurs amongst people with serious underlying illnesses. Listeriosis can also affect pregnant women and infect their unborn baby. Laboratory-confirmed infections in a mother and unborn child or a neonate are notified separately in the NNDSS.

Epidemiological situation in 2011

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

Age and sex distribution

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

Enhanced surveillance in 2011

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

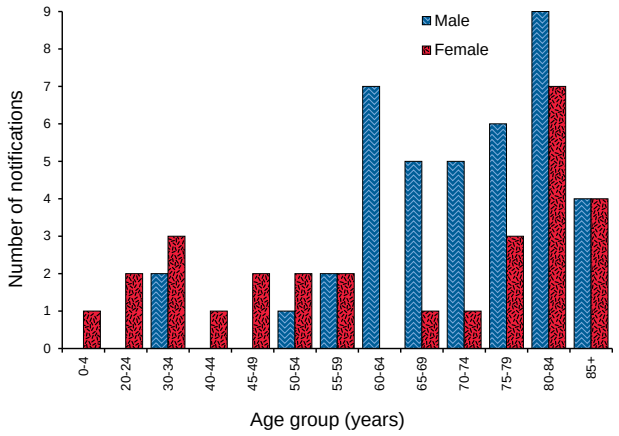
Table 10: Notifications of hepatitis A, Australia, 2006 to 2011, by place of acquisition

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

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

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

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



Salmonellosis (non-typhoidal)

Salmonellosis is a bacterial disease characterised by the rapid development of symptoms including abdominal pain, fever, diarrhoea, muscle pain, nausea and/or vomiting. People can become infected via faecal-oral transmission, ingesting contaminated food, through animal contact and from environmental exposures.[14]

Epidemiological situation in 2011

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

Age and sex distribution

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

Shigellosis

Shigellosis is a bacterial disease characterised by acute abdominal pain and fever, small-volume loose stools, vomiting and tenesmus. *Shigella* is transmitted via the faecal-oral route, either directly (such as male-to-male sexual contact) or indirectly through contaminated food.[14]

Epidemiological situation in 2011

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

Age and sex distribution

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

Indigenous status

Information on Indigenous status was available for 89% (n=429) of shigellosis notifications. This proportion varied by state or territory, with New South Wales, Queensland and Victoria being less than 85% complete. Amongst jurisdictions with greater than 85% completeness, the proportion of notified cases who identified as being of Aboriginal or Torres Strait Island origin was 55% (114/206).

Place of acquisition

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

Shiga toxin-producing *Escherichia coli* infections

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

Epidemiological situation in 2011

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

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

Age and sex distribution

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

Typhoid

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

Epidemiological situation in 2011

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

Age and sex distribution

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

Place of acquisition

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

Quarantinable diseases

Human diseases covered by the Quarantine Act 1908, and notifiable in Australia and to the WHO in 2011 were cholera, plague, rabies, yellow fever, smallpox, highly pathogenic avian influenza in humans (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 to avoid infection. More information on quarantinable diseases and travel health can be found on the Department of Health's web site (www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-strateg-quaranti-index.htm) and the Department of Foreign Affairs and Trade's Smartraveller web site (www.smartraveller.gov.au).

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

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

Disease	Status	Date of last record and notes
Cholera	Free	Small number of cases are reported annually and related to overseas travel or imported food products
Plague	Free	Last case recorded in Australia in 1923[17]
Rabies	Free	Last case (overseas acquired) recorded in Australia in 1990[18]
Smallpox	Free	Last case recorded in Australia in 1938, last case world-wide in 1977, declared eradicated by the World Health Organization 1980[19, 20]
Yellow fever	Free	Two cases in 2011 are the first recorded, related to overseas travel
SARS	Free	Last case recorded in Australia in 2003[21]
HPAIH	Free	No cases recorded[22]
Viral haemorrhagic fevers		
Ebola	Free	No cases recorded
Marburg	Free	No cases recorded
Lassa	Free	No cases recorded
Crimean–Congo	Free	No cases recorded

Cholera

There were 6 notifications of cholera in Australia in 2011, five from Queensland and one from Western Australia. The 5 cases notified in Queensland all acquired their infection in Papua New Guinea and were overseas residents, while the case in Western Australia was acquired in the Philippines. There were 19 cases of cholera in Australia between 2006 and 2010 (Table 5).

All cases of cholera reported since the commencement of the NNDSS in 1991 were acquired outside Australia except for 1 case of laboratory-acquired cholera in 1996[23] and 3 cases in 2006 linked to imported whitebait.[24]

Yellow fever virus infection

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

Sexually transmissible infections

Introduction

In 2011, the sexually transmissible infections (STIs) reported to the NNDSS were chlamydial infection, donovanosis, gonococcal infection and syphilis. Other national surveillance systems that monitor STIs in Australia include the Australian Gonococcal Surveillance Programme (AGSP), which is a network of specialist laboratories monitoring antimicrobial susceptibility patterns of gonococcal infection; and the Kirby Institute, which maintains the National HIV Registry and the National AIDS Registry.

In interpreting these data it is important to note that changes in notifications over time may not solely reflect changes in disease prevalence as changes in screening programs,[25,26] the use of less invasive and more sensitive diagnostic tests and periodic public awareness campaigns may influence the number of notifications that occur over time. Rates for STIs, are particularly susceptible to overall rates of testing, with low testing rates resulting in an underestimation of disease and increased testing potentially causing an increase in notifications.[27] For some diseases, changes in surveillance practices may also need to be taken into account when interpreting national trends.

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

Direct age standardised notification rates, using the method described by the Australian Institute of Health and Welfare[28] were calculated for Aboriginal and Torres Strait Islander and non-Indigenous notifications for jurisdictions that had Indigenous status data completed for more than 50% of notifications over the period 2006–2011. Where the Indigenous status of a case was not completed, these notifications were counted as non-Indigenous in the analyses. These data, however, should be interpreted with caution, as STI screening occurs predominately in specific high risk groups, including in Aboriginal and Torres Strait Islander populations. The differences in rates between females and males should be interpreted with caution, as rates of testing for STIs, symptom status, health care-seeking behaviours, and partner notification differ between the sexes.[29]

In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Infections in children may be acquired perinatally (e.g. gonococcal conjunctivitis).[30] Notifications of chlamydial, gonococcal and non-congenital syphilis infections were excluded from analysis where the case was aged less than 13 years and the infection was able to be determined as non-sexually acquired.

Chlamydial infection

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

Epidemiological situation in 2011

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

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

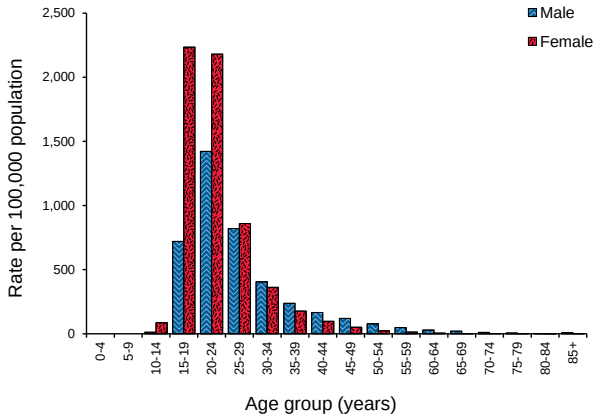
Geographical distribution

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

Age and sex distribution

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

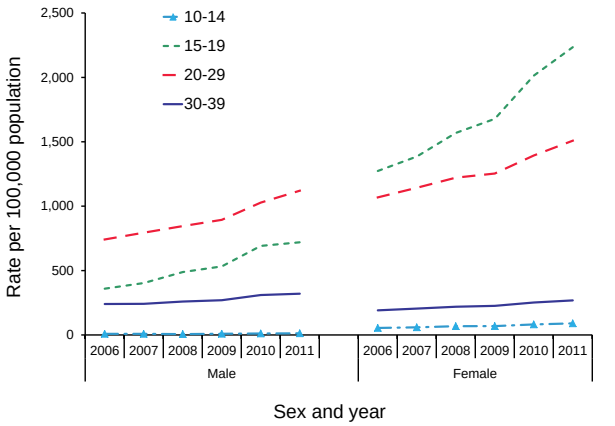
Figure 17: Notification rate for chlamydial infection, Australia, 2011, by age group and sex*



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

Between 2006 and 2011, there was an increasing trend in chlamydia notification rates across both sexes and in all age groups (Figure 18). The greatest increase in rates amongst those aged between 15 and 39 years occurred in both males and females in the 15–19 (100% and 76% respectively) age group. Those between 15 and 29 years of age accounted for approximately 80% of the annual number of reported cases during the period 2006–2011.

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



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

Indigenous populations

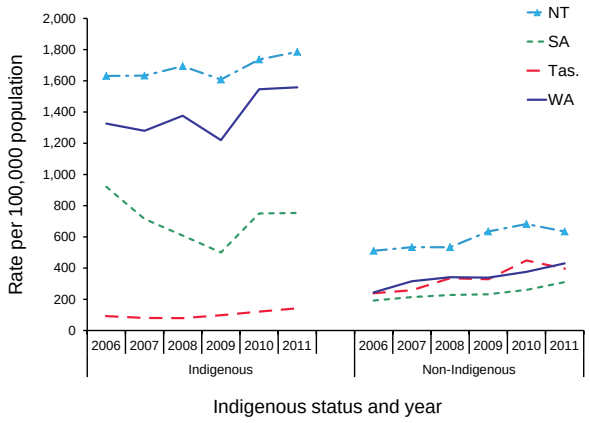
Data on Indigenous status were complete in 51% of notifications in 2011, higher than the preceding 5-year average of 47% (range: 44%–51%). It should be noted that the completeness of Indigenous status identification in the notification data varies by year and by jurisdiction. Four jurisdictions had greater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

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

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

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

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



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

Donovanosis

Donovanosis, caused by the bacterium *Klebsiella granulomatis,* is a chronic, progressively destructive infection that affects the skin and mucous membranes of the external genitalia, inguinal and anal regions.[33] Donovanosis was targeted for elimination in Australia through the National Donovanosis Elimination Project, which commenced in 2001.[34] It predominantly occurred in rural and remote Aboriginal and Torres Strait Islander communities in central and northern Australia and is now relatively uncommon.

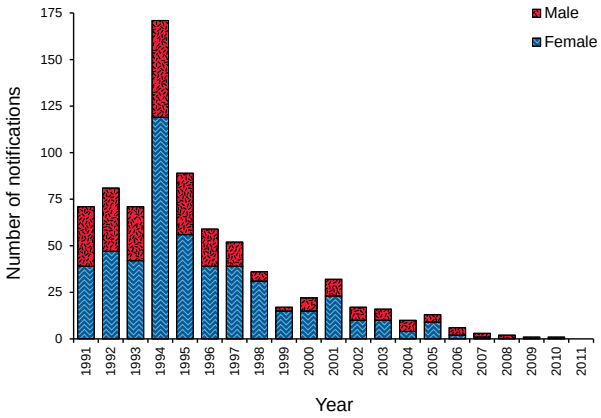
Epidemiological situation in 2011

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

Gonococcal infections

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

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



Epidemiological situation in 2011

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

Geographical distribution

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

Age and sex distribution

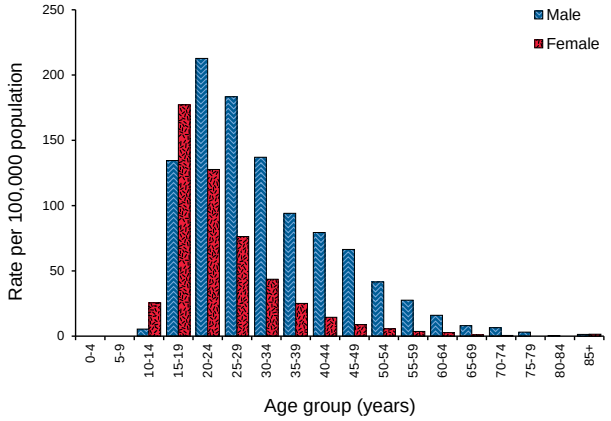
Nationally, there was an increase in gonococcal infection rates in both males (18%) and females (27%) compared with 2010. The male to female rate ratio in 2011 was 2.0:1 (72 and 35 per 100,000 respectively), which is similar to the previous 5 years. The rate of gonococcal infection in males exceeded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

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

Indigenous populations

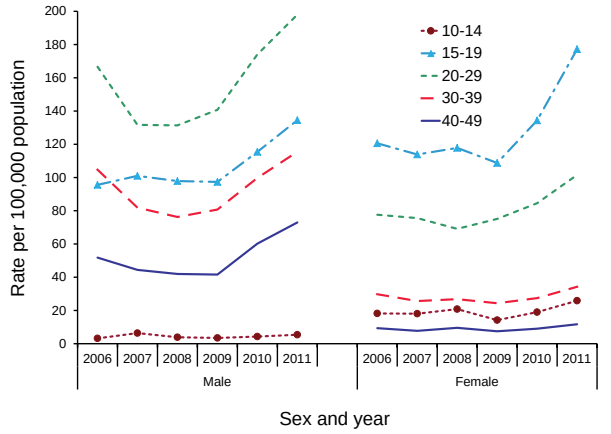
In 2011, the data completeness of the Indigenous status field for gonococcal infection notifications was 68%, slightly higher than 2010 (65%) but a decrease compared with the previous few years (average completeness 69%). All jurisdictions except New South Wales and the Australian Capital Territory had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

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



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

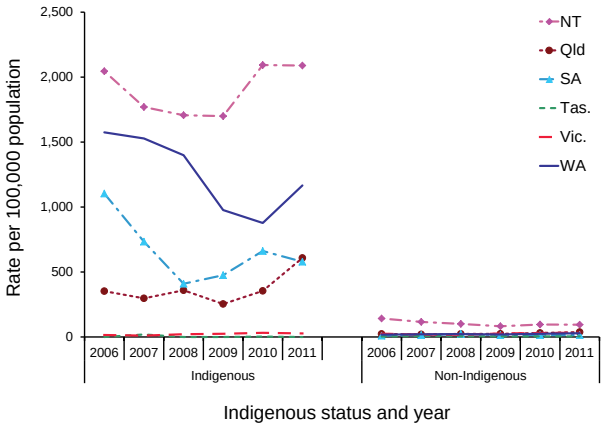
Figure 22: Notification rate for gonococcal infection in persons aged 10–49 years, Australia, 2006 to 2011, by age group and sex*



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

ness of the Indigenous status field. Amongst these jurisdictions with adequate completeness, the combined age standardised notification rate for gonococcal infection in the Aboriginal and Torres Strait Islander population had been steadily declining from 923 per 100,000 in 2006 to 642 per 100,000 in 2009 before increasing to 746 per 100,000 in 2010 and 894 per 100,000 in 2011. In the non-Indigenous population, rates remained stable at around 22 to 23 per 100,000 between 2006 and 2009 before also increasing by 35% to 31 per 100,000 in 2011. Between 2006 and 2011 the Aboriginal and Torres Strait Islander to non-Indigenous rate ratio decreased 30% from 41:1 to 28:1. In 2011, rates of gonococcal infection in the Aboriginal and Torres Strait Islander and non-Indigenous populations decreased or remained relatively stable compared with 2010 in all jurisdictions except Queensland and Western Australia (Figure 23).

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



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

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

Microbiological trends

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

In 2011, the AGSP annual report for 2011[35] reported a total of 4,230 gonococcal isolates were tested for antibiotic susceptibility, representing approximately 35% of gonococcal infection notifications, which was a slightly lower proportion to 2010 (41%) and 2009 (40%).

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

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

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

Discussion

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

Syphilis (non-congenital categories)

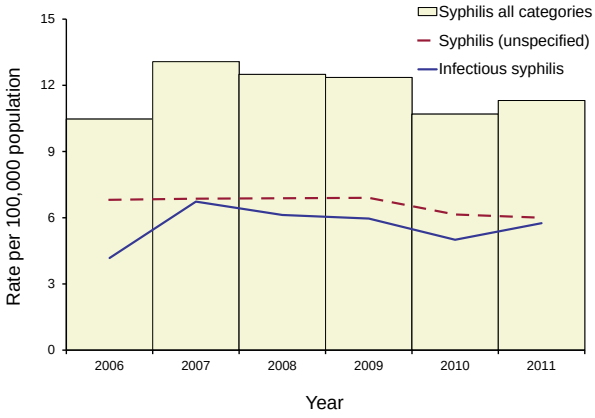
Syphilis, caused by the bacterium *Treponema palladium*, is characterised by a primary lesion, a secondary eruption involving skin and mucous membrane, long periods of latency and late lesions of skin, bone, viscera, cardiovascular and nervous systems.[14]

In 2004, all jurisdictions except South Australia began reporting to the NNDSS of non-congenital syphilis infections categorised as: infectious syphilis (primary, secondary or early latent) of less than 2 years duration; and syphilis of more than 2 years or unknown duration. South Australia, only report cases of infectious syphilis. Detailed analyses are reported for these 2 categories, as well as for syphilis of the combined categories (syphilis – all categories) for the purpose of showing trends in previous years. Data for all states and territories are reported by diagnosis date, except Queensland, which is reported by notification receive date. During this reporting period, the syphilis case definition was applied differently in Queensland compared with the rest of Australia. The aggregated data are presented with this variation in place.

Epidemiological situation in 2011

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

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



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

Geographical distribution

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

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

Epidemiological situation in 2011

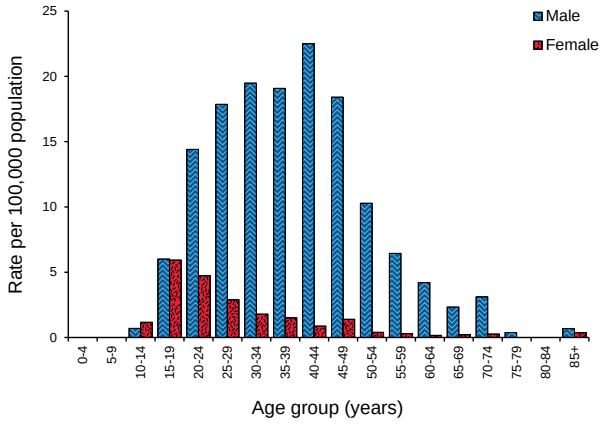
In 2011, there were 1,303 notifications of infectious syphilis (primary, secondary and early latent), of less than 2 years duration. This represents a notification rate of 5.8 per 100,000, an increase of 13.7% compared with 2010 (5.1 per 100,000) (Table 5). The rate of infectious syphilis notifications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

Age and sex distribution

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

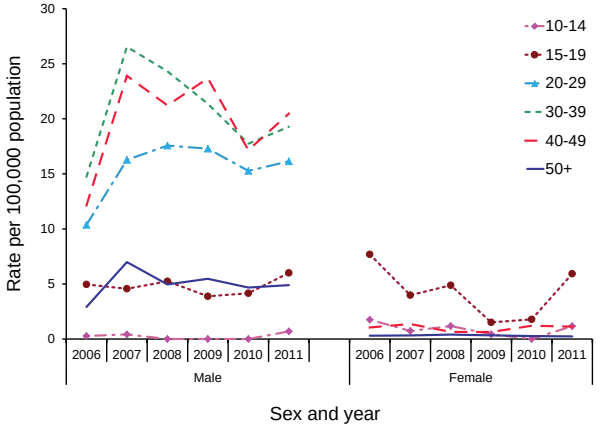
From 2006 to 2007 notification rates amongst males increased substantially in the 20–49 year age range. Since 2007, 20–29 and 30–39 year age groups have either decreased or remained relatively stable. The 40–49 year age group experienced a marked increase between 2010 and 2011. In females, for the 2006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

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



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

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



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

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

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

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

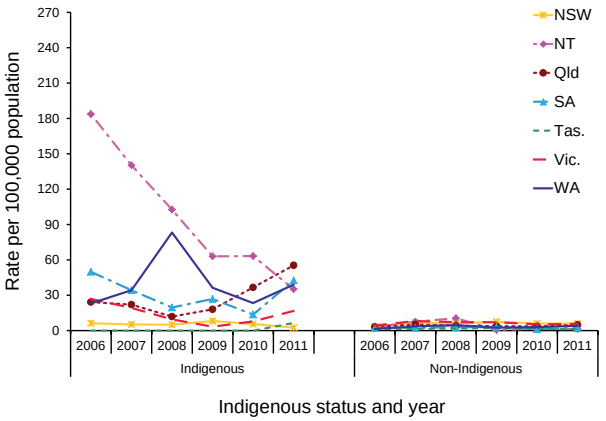
† Notification rate per 100,000 population.

‡ Includes notifications that have missing sex.

Indigenous populations

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

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



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

Syphilis of more than 2 years or unknown duration

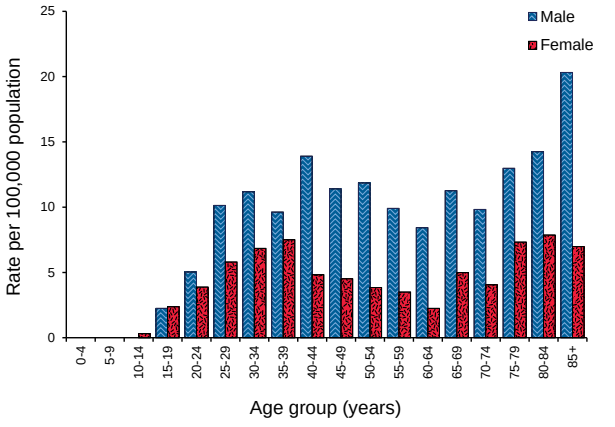
Epidemiological situation in 2011

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

Age and sex distribution

In 2011, notification rates of syphilis of more than 2 years or unknown duration in males and females were 7.9 and 4.0 per 100,000, respectively (Table 13), with a male to female rate ratio of 2.0:1 (Figure 28). Age group specific rates in males were higher than in females except in the 10–14 and 15–19 age groups, and were more than 3 times higher amongst males in the 50–54 and 60–64 year age groups than in females. The distribution of notification rates across age groups in females was bimodal, with the highest rate (7.9 per 100,000) amongst those in the 80–84 year age group, followed by those in the 35–39 year age group (7.5 per 100,000). In males, rates remained high in those aged 25 years or over with peaks occurring in the 40–44 year and 85 years or over age groups (13.9 and 20.3 per 100,000 respectively).

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



* Data from all states and territories except South Australia.

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

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

Congenital syphilis

Epidemiological situation in 2011

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

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

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

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

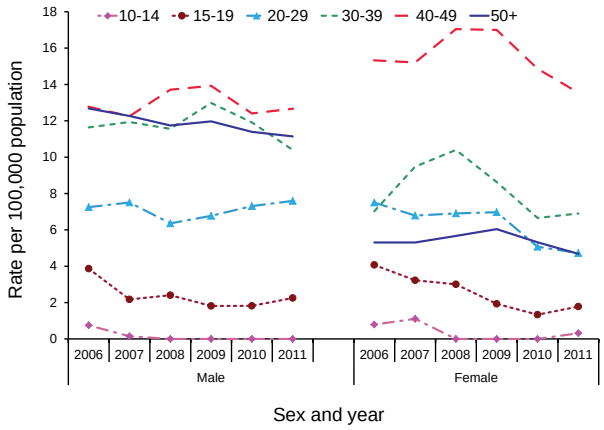
† Notification rate per 100,000 population.

‡ Data from all states and territories except South Australia.

§ Includes notifications missing sex.

NDP No data provided.

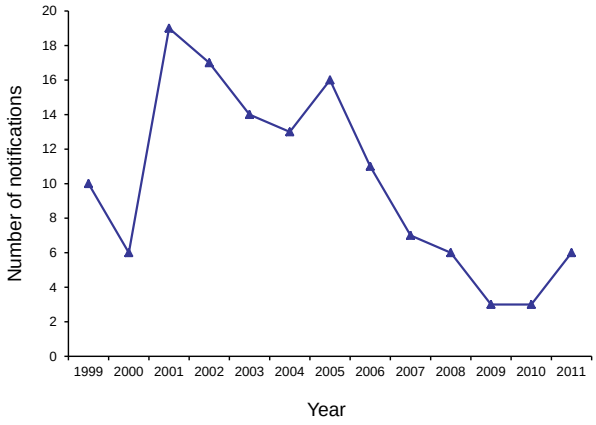
Figure 29: Notification rate for syphilis of more than 2 years or unknown duration, Australia,* 2006 to 2011, by age group and sex†



* Data from all states and territories except South Australia.

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

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



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

Vaccine preventable diseases

Introduction

This section summarises the national surveillance data for notifiable diseases targeted by the National Immunisation Program (NIP) in 2011. These include diphtheria, invasive *Haemophilus influenzae* type b infection, laboratory-confirmed influenza, measles, mumps, pertussis, invasive pneumococcal disease, poliomyelitis, rubella, tetanus and varicella zoster infections (chickenpox, shingles and unspecified). Data on hepatitis B and invasive meningococcal disease, which are also targeted by the NIP, can be found in this report under 'Bloodborne diseases' and 'Other bacterial infections' respectively. Other vaccine preventable diseases (VPDs) presented in this report include hepatitis A and Q fever under the 'Gastrointestinal' and 'Zoonoses' sections respectively. For more detailed reports on historical data, including notifications, hospitalisations and deaths, readers are referred to the regular CDI supplements 'Vaccine Preventable Diseases in Australia', the latest of

which was published as the December 2010 supplement issue of CDI.[38] Additionally, a similar report which analyses the impacts of vaccines on the health of Aboriginal and Torres Strait Islander people between 2007 and 2010 was published in the November 2013 supplement to CDI.[39]

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

New vaccines added to the National Immunisation Program in 2011

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

Vaccination coverage is an important factor influencing the incidence of vaccine preventable diseases. Since the commencement of the Australian Childhood Immunisation Register in 1996, immunisation coverage in Australian children has been high by international standards, although geographical pockets of lower coverage remain, in which there is an increased potential for VPDs to occur and circulate. No vaccine is 100% effective, and therefore infections sometimes do occur in fully vaccinated people, and some are reported later in this section. However, effective vaccines do provide a substantially lower chance of becoming infected, and/or reduced the severity of the disease.

Information on the receipt of vaccines has historically been recorded in NNDSS using the 'vaccination status' field (fully or partially vaccinated for age or unvaccinated), plus a field capturing the number of doses. In January 2008 new, more detailed fields were added for recording 'vaccine type' and 'vaccination date' for each dose. The new fields were intended to replace the old fields, with a transition period allowing either field to be utilised. In 2011, 4 jurisdictions were using the new fields (the Northern Territory, Queensland, Tasmania and New South Wales for selected diseases), while the remaining jurisdictions continued to use the old fields. In this report, data on receipt of vaccines is presented for each disease, combining data provided by the states and territories from the two different formats.

Diphtheria

Diphtheria is an acute toxin-mediated systemic disease caused by the bacterium C*orynebacterium diphtheriae*. Infection is usually localised to the throat (pharyngeal diphtheria) in which a membranous inflammation of the upper respiratory tract can cause airway obstruction, or the skin (cutaneous diphtheria). Systemic complications caused by the bacterium's exotoxin can occur in both pharyngeal and cutaneous diphtheria. Diphtheria is spread by respiratory droplets or by direct contact with skin lesions or articles soiled by infected individuals.[16] While there are non-toxigenic strains of *C. diphtheriae,* they usually only cause mild throat or skin infection and are not nationally notifiable.

Epidemiological situation in 2011

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

Age and sex distribution

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

Vaccination status

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

Discussion

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

theria in 2001 acquired in East Timor and the last deaths, of which there were two, occurred between 1986 and 1995. In Australia, serosurveillance data indicate that childhood immunity to diphtheria is greater than 99%. However, waning immunity amongst adults may result in this population being susceptible, with the most likely source of exposure being through overseas travel to countries where diphtheria remains endemic.[40] The 2011 Queensland cluster highlights the importance of maintaining high vaccination coverage to protect against vaccine preventable diseases that remain endemic in many other countries around the world.

Influenza

Epidemiological situation in 2011

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

Notification rates were highest in South Australia (286 per 100,000), followed by the Northern Territory (259 per 100,000) and Queensland (227 per 100,000). Notification rates in the remaining jurisdictions were all substantially lower than the national notification rate of 120 per 100,000. In 2011, Queensland reported a larger number of influenza cases than any other jurisdiction, comprising 38% of all notifications, which was consistent with previous years with the exception of 2010 (Figure 31).

Age and sex distribution

Females accounted for 14,323 (53%) of the 27,119 influenza notifications for which sex was reported in 2011. Notifications rates were higher amongst females in most age groups, with the primary exception of those aged less than 15 years where the rates were higher for males (Figure 32). This likely reflects the health seeking behaviour of adult females as they tend to account for a greater proportion of encounters in general practice.[41]

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

Seasonality

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

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

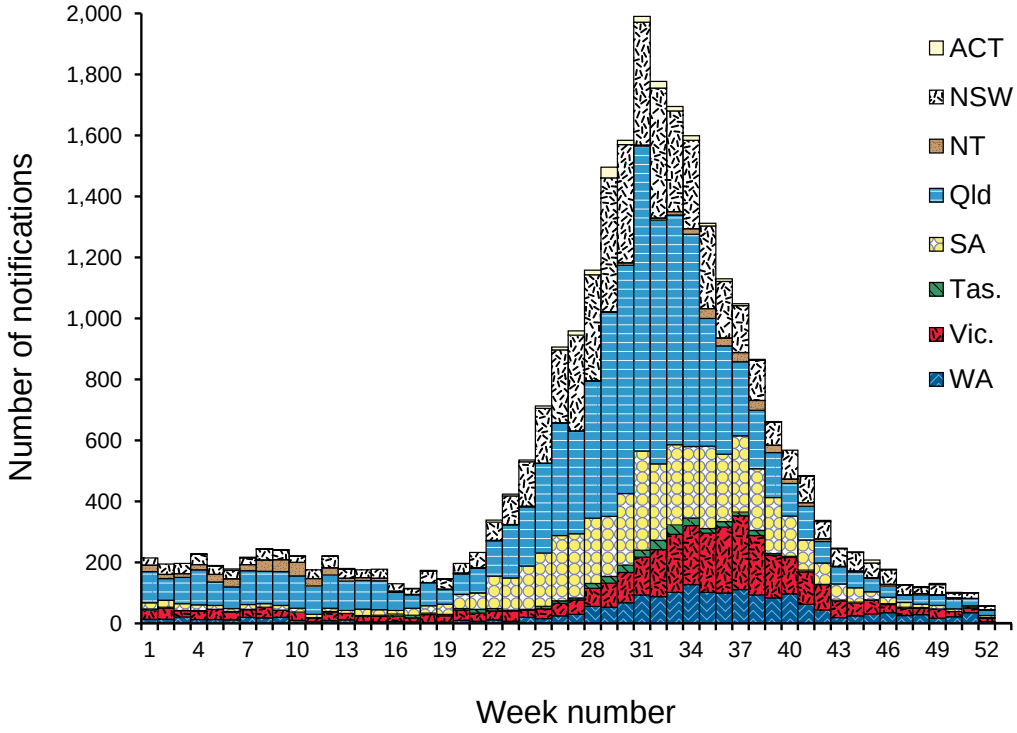
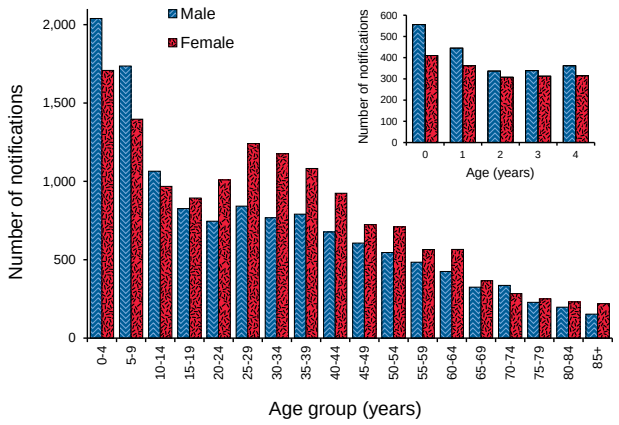
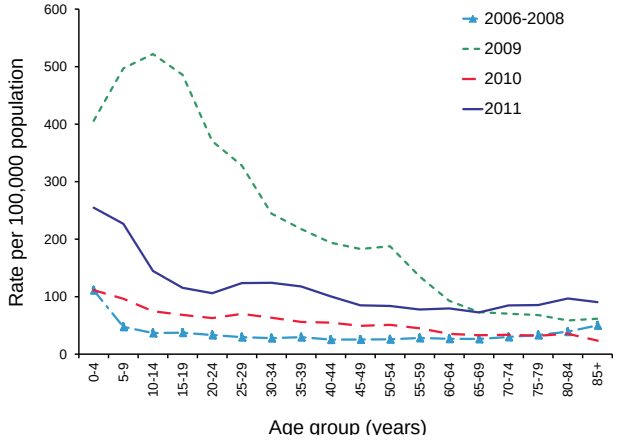


Figure 32: Notifications of laboratory-confirmed influenza, Australia, 2011, by age group and sex*



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

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

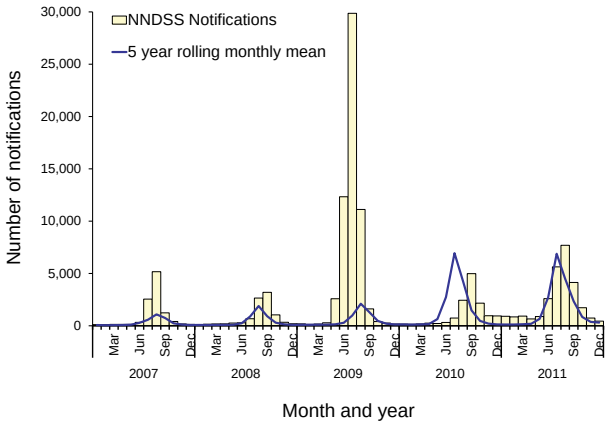


season peaked in August with 7,690 cases for the month, compared with the lower peak of 4,981 notifications during September 2010 (Figure 34). Notifications fell substantially through October and returned to typical inter-seasonal levels by late November 2011.

Virological surveillance

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

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

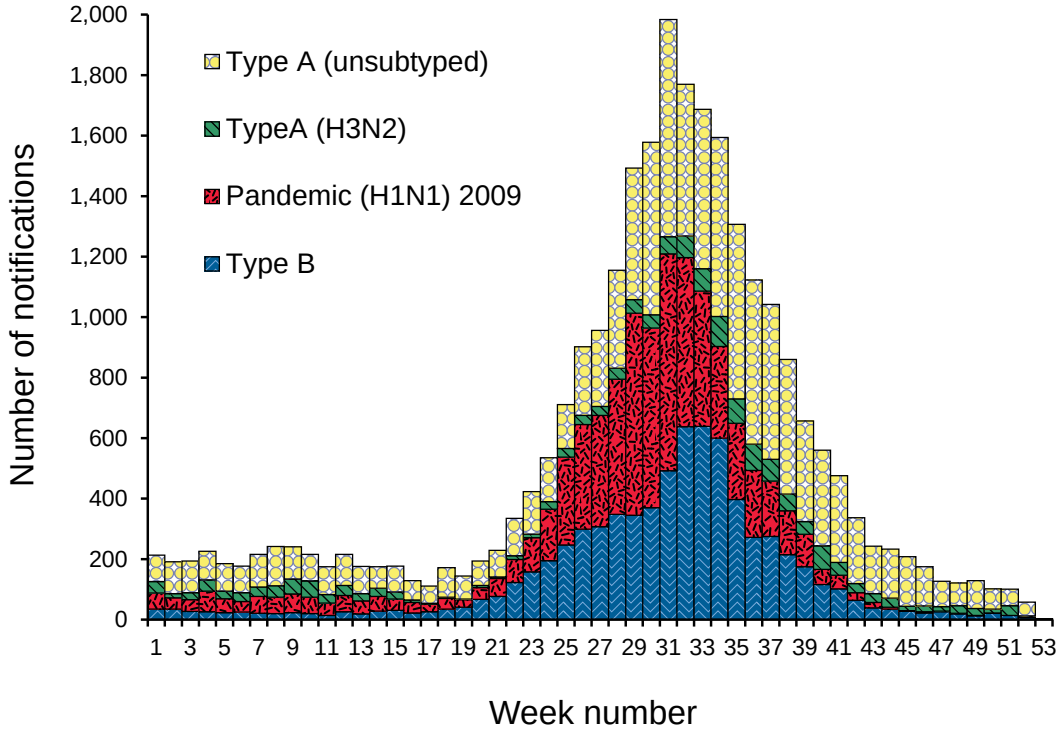


proportion of notifications reported as influenza type A was much higher (90%), with the majority of these (56%) reported as A(H1N1)pdm09, followed by A(unsubtyped) (30%), with very few A(H3N2) (4%). Additionally, the proportion of influenza B notifications in 2010 was substantially less (10%) than in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

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

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

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



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

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

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

Discussion

Higher than usual levels of influenza activity characterised the 2010–11 inter-seasonal period and contributed to the increase in notifications, compared with the previous year. There were 4,207 notifications in the first 5 months of 2011, compared with just 934 in the same period of 2010. Most of the influenza activity during this period was attributed to A(H1N1)pdm09 and A(H3N2) infections. The reason for the unusually high activity is not clear but does not appear to be solely due to increased testing. It is worth noting that the 2010–11 inter-seasonal period was characterised by extensive flooding, particularly in Queensland, which may have been associated with increases in influenza A virus survival rates,[42] and time spent indoors.

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

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

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

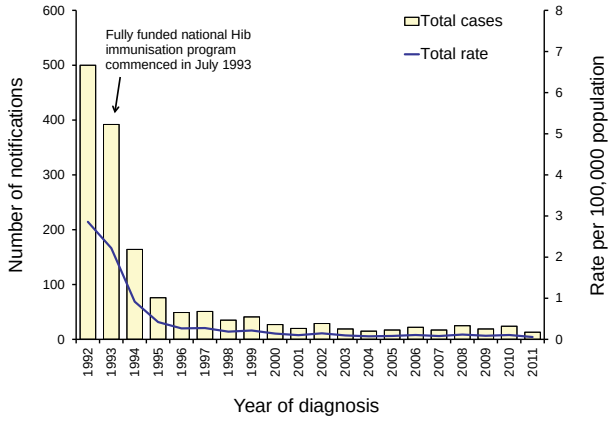
Invasive *Haemophilus influenzae* type b disease

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

Epidemiological situation in 2011

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

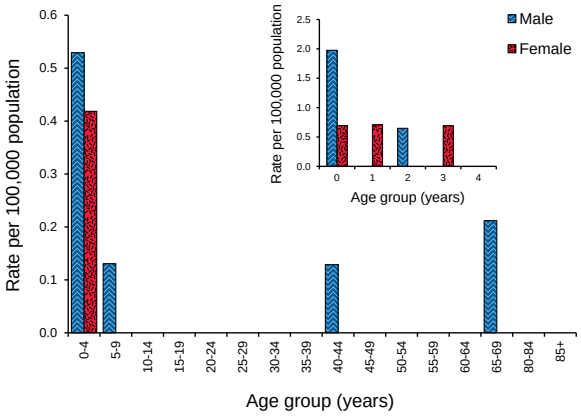
Figure 36: Notifications and notification rate for invasive *Haemophilus influenzae* type b infection, Australia, 1992 to 2011



Age and sex distribution

The male to female ratio was 1.6:1 in 2011 with 8 males and 5 females overall. The majority of cases (n=7) were in children less than 5 years of age, 57% of which were in infants aged less than 1 year. Age group specific rates were highest in the 0–4 year age group (Figure 37).

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



Vaccination status

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

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

Invasive pneumococcal disease

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

From 1 October 2011 until 30 September 2012 a supplementary dose of the 13vPCV was made available under the NIP to eligible children who had completed their primary pneumococcal vaccination course with the 7vPCV. Vaccination with the 23-valent pneumococcal polysaccharide vaccine (23vPPV) was added to the NIP schedule for Aboriginal and Torres Strait Islander peoples aged 50 years or over in 1999 and non-Indigenous Australians aged 65 years from January 2005.[43]

Epidemiologic situation in 2011

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

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

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

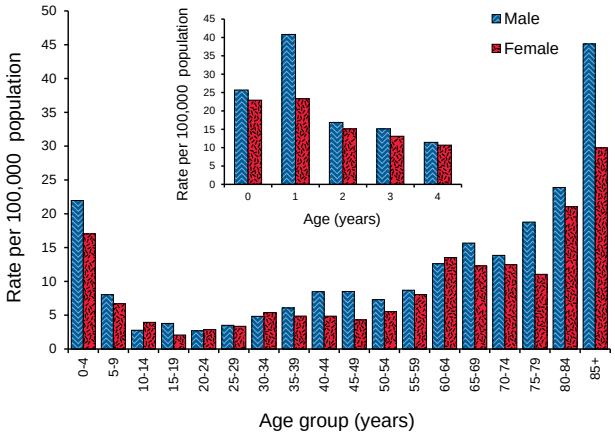
Each jurisdiction experienced an increase on the number of cases reported in the previous year. The largest increase at the jurisdictional level was in cases resident in the Northern Territory, where the number of cases increased by 126%, from 57 cases in 2010 to 129 cases in 2011. Most of this increase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

Age and sex distribution

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

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

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



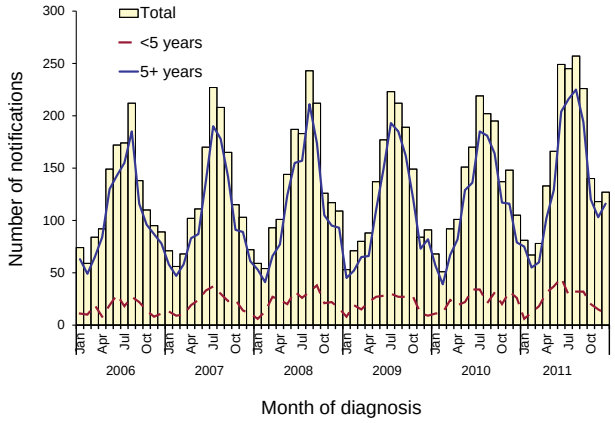
Seasonality

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

Indigenous status

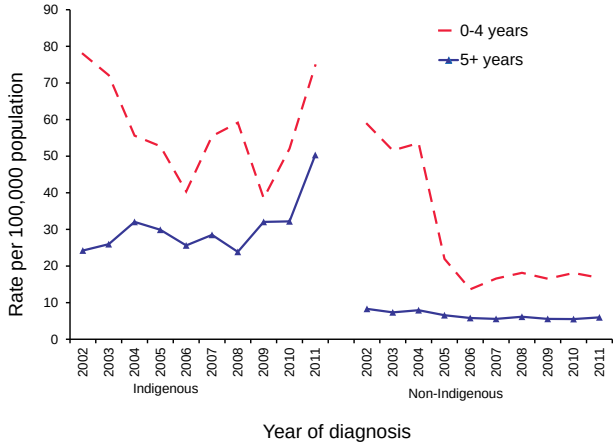
Completeness of Indigenous status reporting in 2011 was high, with 94% (n=1778) of cases reported with a known Indigenous status. Indigenous people made up 17% (n=307) of all notifications. In 2011, the rate of IPD in Indigenous people (53.3 per 100,000) was 8 times the rate of non-Indigenous people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

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



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

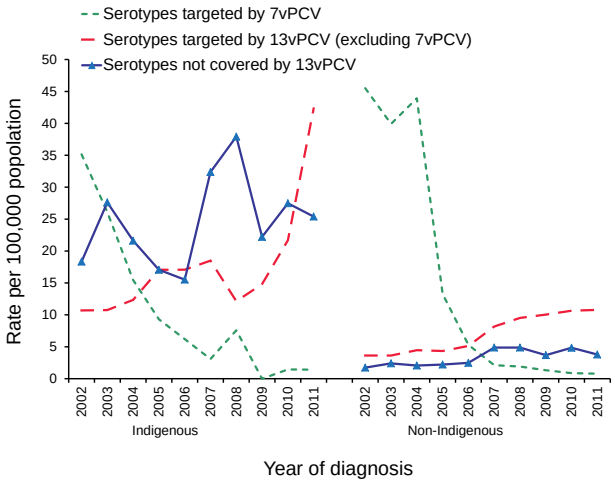
Figure 40: Notification rate for invasive pneumococcal disease, Australia, 2002 to 2011, by Indigenous status and age group



Serotype

An identified serotype was reported in 93% (n=1746) of notified cases of IPD in 2011. In children aged less than 5 there has been a dramatic reduction in disease due to serotypes targeted by the 7vPCV in both the Indigenous and non-Indigenous populations, with this reduction being maintained in 2011 (Figure 41). As few as 5% (n=12) of notifications in children aged less than 5 with a serotype identified were caused by those targeted by the 7vPCV in 2011. In this same age group there was an increase in disease due to the 6 additional serotypes targeted by the 13vPCV, with 63% (n=181) of notifications in 2011 due to one of these 6 serotypes. In Indigenous children, this was largely (67%) due to serotype 1, while in non-Indigenous children serotype 19A was the most common serotype reported (77%).

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



Discussion

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

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

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

Measles

Measles is a highly infectious, acute viral illness spread by respiratory secretions, including air-borne transmission via aerosolised droplets. The prodrome, lasting 2–4 days, is characterised by fever and malaise followed by a cough, coryza and conjunctivitis. It is usually followed by a maculopapular rash, which typically begins on the face, and then becomes generalised. Measles can be a severe disease, with complications such as otitis media, pneumonia, and acute encephalitis. Subacute sclerosing panencephalitis is a late, rare (approximately 1 in 100,000 cases) complication of measles, which is always fatal.[44]

Epidemiological situation in 2011

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

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

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

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

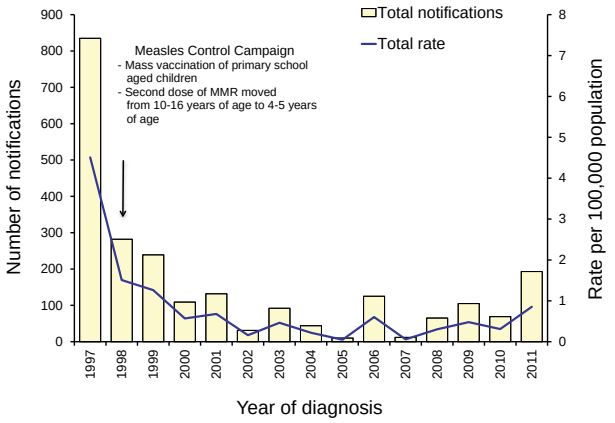
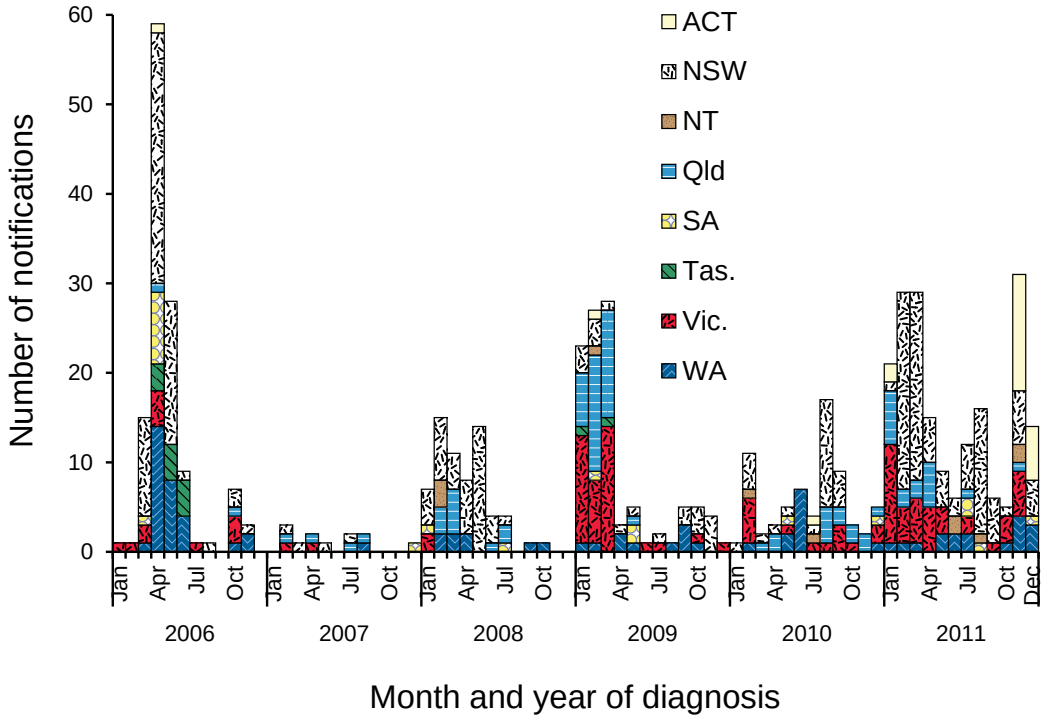


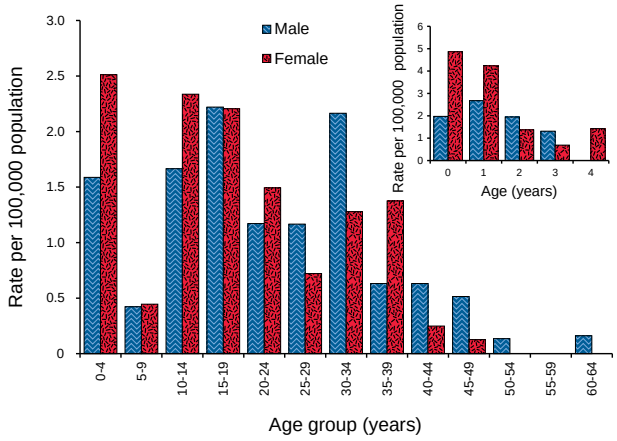
Figure 43: Notifications of measles, Australia, 2006 to 2011, by state and territory and month of diagnosis



Age and sex distribution

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

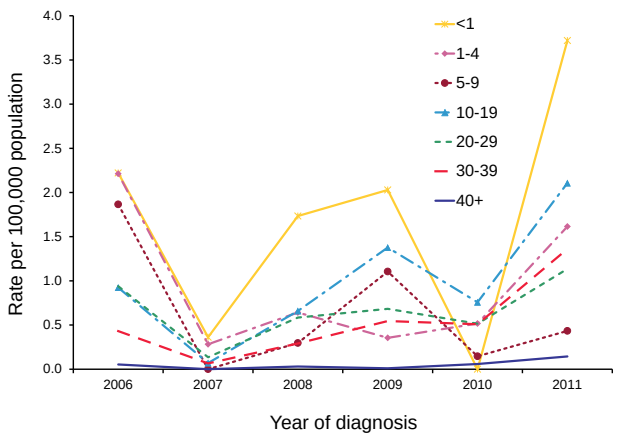
Figure 44: Notification rate for measles, Australia, 2011, by age group and sex



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

Measles rates remained below 2.5 per 100,000 in all age groups between 2006 and 2011 with the exception of infants aged less than 1 year in 2011 (Figure 45). The fluctuating nature of these rates can be attributed to a general trend of sporadic imported cases that occasionally result in outbreaks of locally acquired infection amongst susceptible contacts.

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



Vaccination status

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

Of the 193 cases notified in 2011, 172 (89%) were born after 31 December 1969 and eligible for a publicly funded measles-containing vaccine. Of the 18 cases aged between 1 and 3 years of age who were eligible for 1 dose of a measles-containing vaccine, one was fully vaccinated for age and the remaining 17 were not vaccinated. Of the remaining 154 cases 4 years of age or over and eligible for 2 doses, 81 were not vaccinated, 17 were partially vaccinated, 5 were fully vaccinated and 51 were of unknown vaccination status. The 10–19 year age group accounted for 51% (n=41) of the unvaccinated cases amongst those 4 years of age or over. Twenty-two cases occurred amongst those born between 1978 and 1982 (29–33 years in 2011). This cohort has previously been identified as susceptible to measles infection as during their childhood a second dose of a measles containing vaccine was not yet recommended and they were not targeted as part of the 1998 measles control campaign.[46] Of these cases, 6 were at least partially vaccinated for age, 5 were not vaccinated and 11 were of unknown vaccination status. Eleven cases occurred in adults born before 1968, a cohort that is considered to have high levels of natural immunity,[47] all of them were either unvaccinated or of unknown vaccination status.

Source of infection and outbreaks

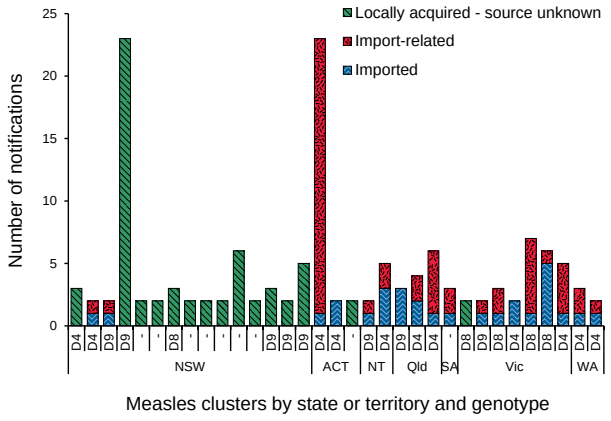
The majority of cases in 2011 were either imported (32%, n=61) or import-related (27%, n=53). The remaining 79 cases (41%) were locally acquired with the original source of infection unknown. Eighty four per cent of all imported cases were either from the South East Asia Region (n=26) or the Western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

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

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

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

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



Discussion

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

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

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

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

Mumps

Mumps is an acute viral illness transmitted by the respiratory route in the form of air-borne droplets or by direct contact with saliva of an infected person. The characteristic bilateral, or occasionally unilateral, parotid swelling occurs in 60%–70% of clinical cases. However, a high proportion have non-specific symptoms including fever, headache, malaise, myalgia and anorexia, with approximately one-third of infections being asymptomatic. Mumps is a multi-system infection, with orchitis occurring in 20% to 30% of post-pubertal males.[14]

Epidemiological situation in 2011

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

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

Age and sex distribution

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

There were cases of mumps notified across most age groups with age at diagnosis ranging from 3 to 80 years and with a median age of 30 years. All age group rates in 2011 were higher than in 2010 except those less than 1 year of age, in which there were no cases. The biggest increase in age group rates occurred amongst young adults between 20 and 39 years of age, although they remained lower compared with the peak amongst this cohort in 2007–2008 (Figure 49).

Vaccination status

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

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

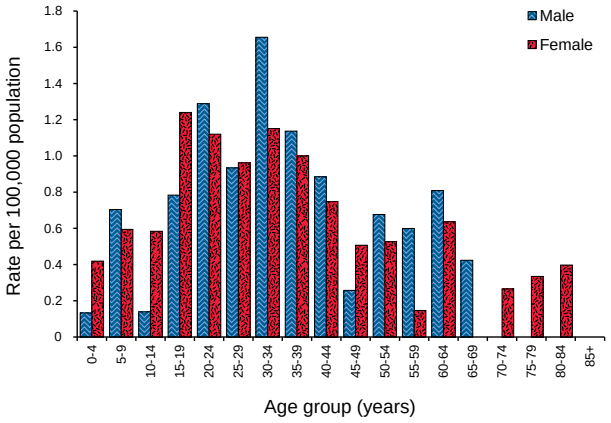


Figure 47: Notifications of mumps, Australia, 2006 to 2011, by state or territory and month of diagnosis

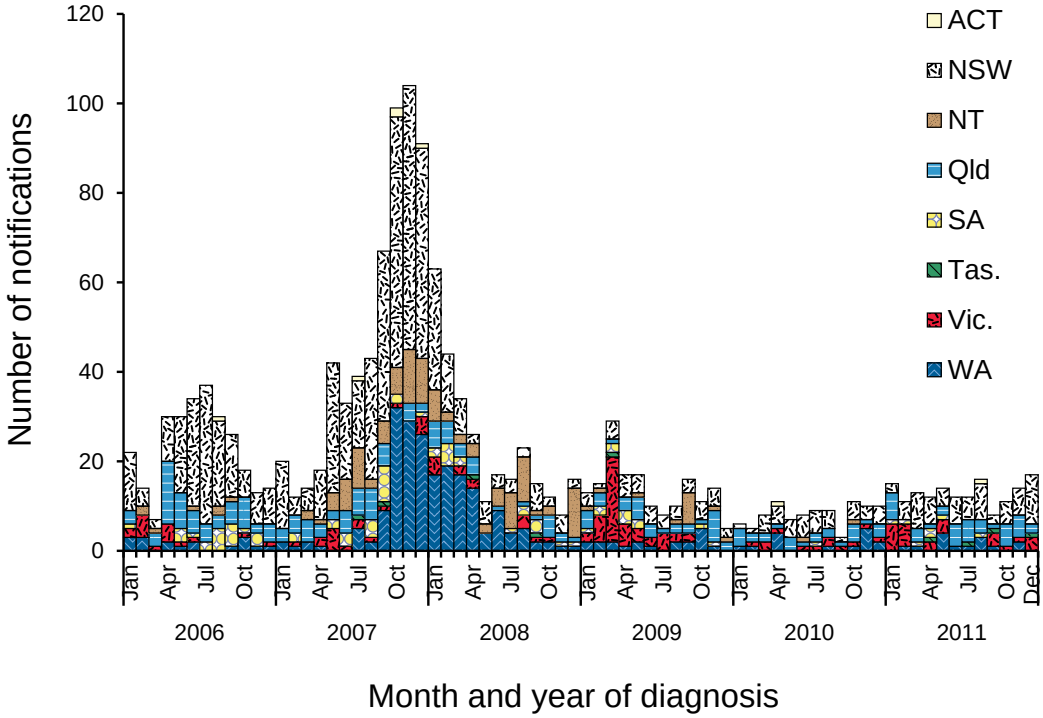
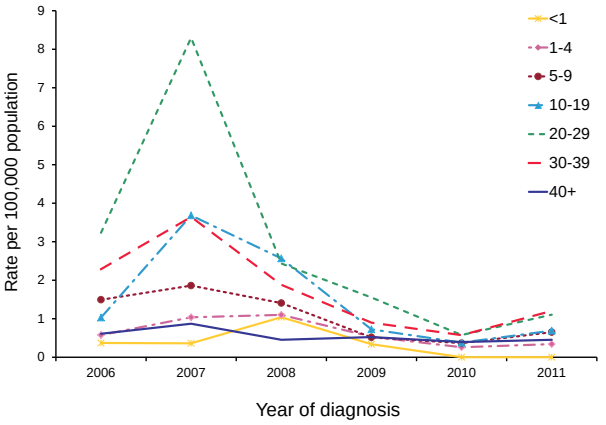


Figure 49: Notification rate for mumps, Australia, 2006 to 2011, by age group



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

The mumps vaccine was first funded on the NIP available in Australia in 1981 with people born since then eligible for 2 doses of a mumps-containing vaccine.[54] In 2011, there were 75 cases of mumps in individuals born after 31 December 1980. One case was aged between 1 and 3 years and eligible for 1 dose and was fully vaccinated for age. The remaining 74 cases were aged 4 years or over. Of these, 9% (n=7) were fully vaccinated for age, 9% (n=7) were partially vaccinated for age, 15% (n=11) were unvaccinated. As mumps notifications are not routinely followed up by all public health units, a further 66% (n=49) had an unknown vaccination status reported.

Pertussis

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

Epidemiological situation in 2011

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

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

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

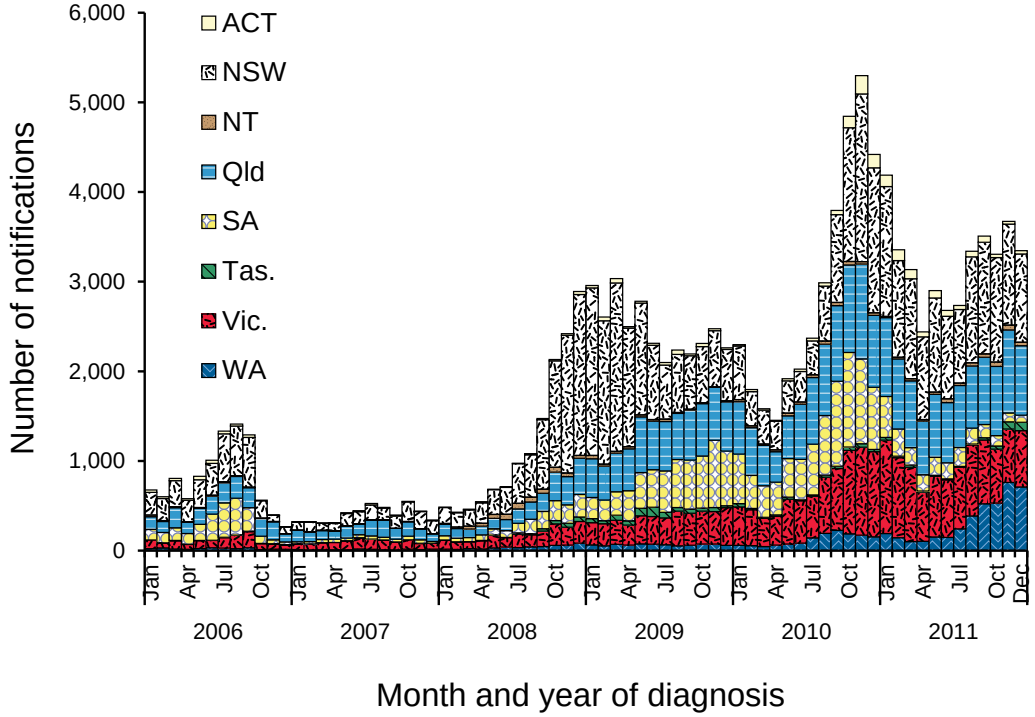
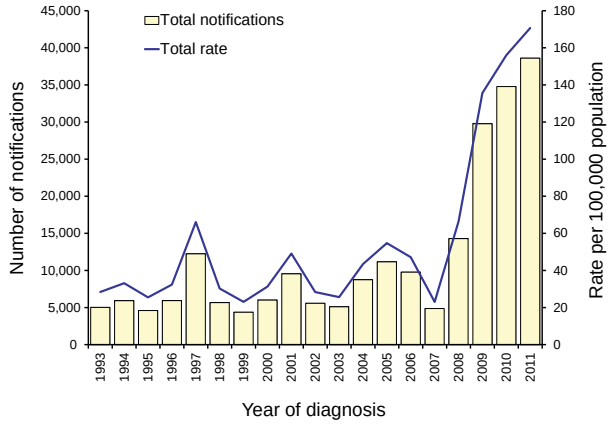
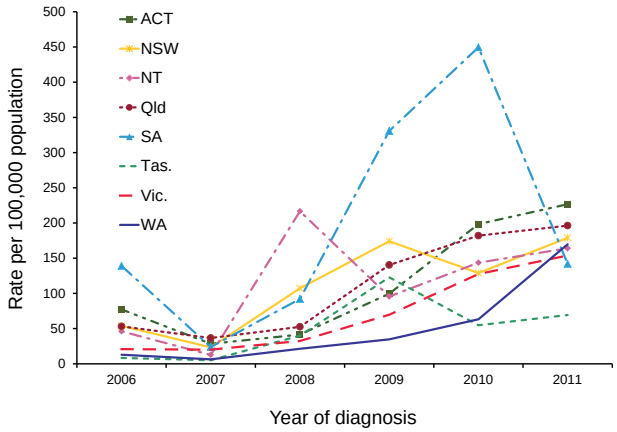


Figure 51: Notifications and notification rate for pertussis, Australia, 1993 to 2011



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

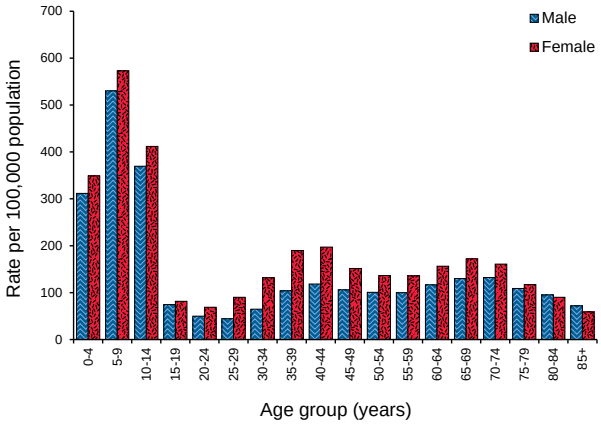
Figure 52: Notification rate for pertussis, 2006 to 2011, by state and territory



Age and sex distribution

In 2011, females accounted for 56% (n=21,512) of cases, resulting in a male to female ratio of 0.8:1. Forty-one cases had no sex specified and an additional 20 had no age provided. Females had higher rates across all age groups except for those adults 80 years of age or over (Figure 53). The highest rate in both males and females occurred in the 5–9 year age group (530 and 573 per 100,000 respectively). The largest difference in sex distribution occurred in the 25–29, 30–34 and 35–39 year age groups where rates in females were 2 times that of males, likely representing the increased health seeking behaviour noted in adult females compared with males.[41]

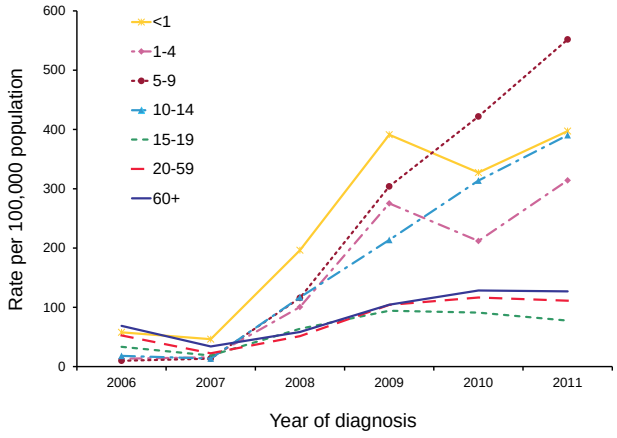
Figure 53: Notification rate for pertussis, Australia, 2011, by age group and sex



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

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

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



Vaccination status

Pertussis vaccine effectiveness amongst Australian children has been estimated to range from 82% to 89% with the lower figure representing the cohort of children who would not have been eligible for the 18-month booster dose, which was removed from the NIP in 2003.[55] Immunity to disease decreases over time post-vaccination with estimates of protection remaining for 4–12 years.[56] The current vaccine schedule for pertussis under the NIP includes a dose provided at 2, 4 and 6 months of age followed by a booster at 4 years of age and again at 12–17 years of age (the timing of this last booster dose varies by jurisdiction). In response to the ongoing epidemic in 2011, some infants were given their first vaccination at 6 weeks of age and their fourth from 3.5 years.

Follow-up is required in order to determine the vaccination status of individual cases. In a large outbreak follow-up of all cases is not possible and as per national guidelines jurisdictions prioritised follow-up to those less than 5 years of age. This age group made up 12.6% (n=4,865) of all notified cases in 2011.

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

Discussion

Pertussis was the most commonly notified vaccine preventable illness in Australia in 2011 reflecting the ongoing epidemic activity across the country in this year. Epidemics of pertussis occur at regular intervals of approximately 3 to 5 years on a background of endemic circulation in Australia.[57] The timing of this epidemic activity was not uniform across the country. States and territories experienced peak levels of pertussis at varying intervals as evidenced in 2011 when South Australia had its lowest rate since 2008, while the Australian Capital Territory, New South Wales, Queensland, Victoria and Western Australia all experienced the highest rates since the epidemic began.

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

The causes of this epidemic are likely to be multifactorial. A widespread shift in diagnostic practice associated with the increased use of PCR for pertussis diagnosis in all age groups[61,62] and increased case ascertainment during the epidemic period both serve to amplify the number of reported cases. Additional contributory factors may also include waning immunity levels in the vaccinated population including amongst children following their booster vaccination at 4 years of age,[63,64] reduced vaccine efficacy of the acellular vaccine compared with the whole cell vaccine,[65] the removal of the 18-month dose from the routine schedule[66] and adaptation of *Bordetella pertussis* to the acellular vaccine.[67]

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

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

Poliomyelitis

Poliomyelitis is a highly infectious disease caused by gastrointestinal infection by poliovirus. Transmission occurs primarily person-to-person via the faecal-oral route. In most cases poliovirus infection is not symptomatic but in less than 1% of cases the virus may invade the nervous system and cause acute flaccid paralysis (AFP).[14]

In 2011, there were no notifications of poliomyelitis in Australia, which along with the Western Pacific Region remained poliomyelitis free. Poliomyelitis is a notifiable disease in Australia with clinical and laboratory investigation conducted for cases involving patients of any age with a clinical suspicion of poliomyelitis. Australia follows the WHO protocol for poliomyelitis surveillance and focuses on investigating cases of AFP in children under 15 years of age. The WHO target for AFP surveillance in a polio non-endemic country is 1 case of AFP per 100,000 children aged less than 15 years, which in 2011 Australia achieved for the fourth consecutive year in a row. More details can be found in the annual report of the Australian National Polio Reference Laboratory published in the CDI.

Rubella and congenital rubella

Rubella is generally a mild and self-limiting viral infectious disease. It is spread person-to-person through contact with respiratory secretions directly or via air-borne droplets. Clinically, rubella can be difficult to distinguish from other diseases that cause a febrile rash, such as measles, and is asymptomatic in up to 50% of cases. Rubella infection in pregnancy can result in foetal infection resulting in congenital rubella syndrome (CRS). CRS occurs in up to 90% of infants born to women who are infected during the first 10 weeks of pregnancy and may result in foetal malformations and death.[14]

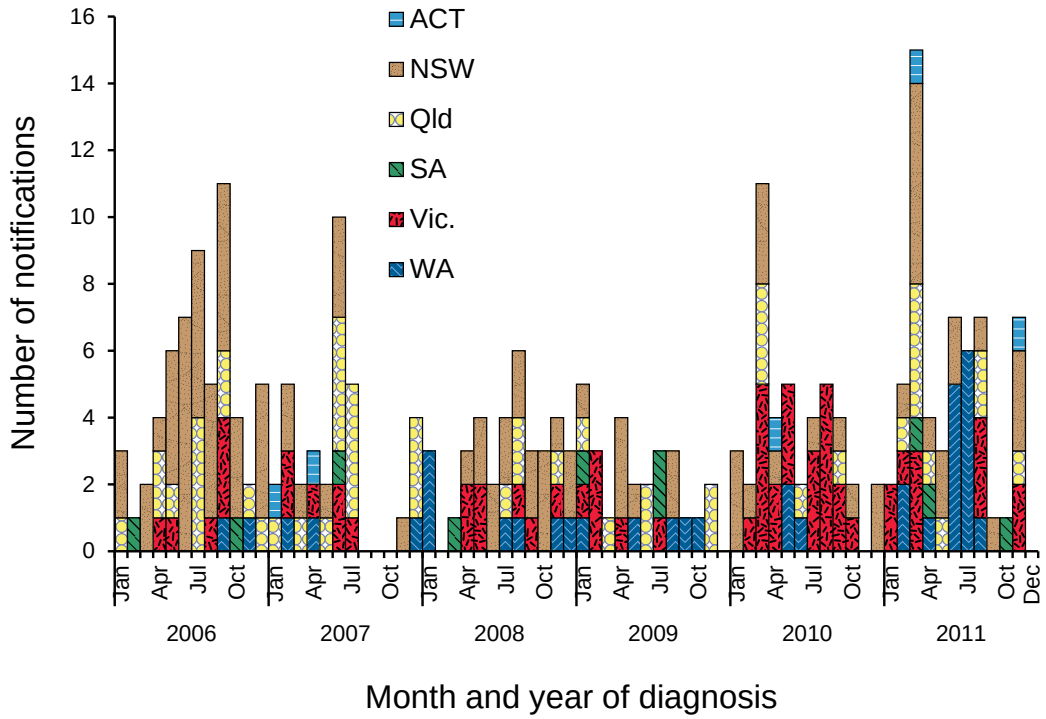
Epidemiological situation in 2011

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

Source of infection

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

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

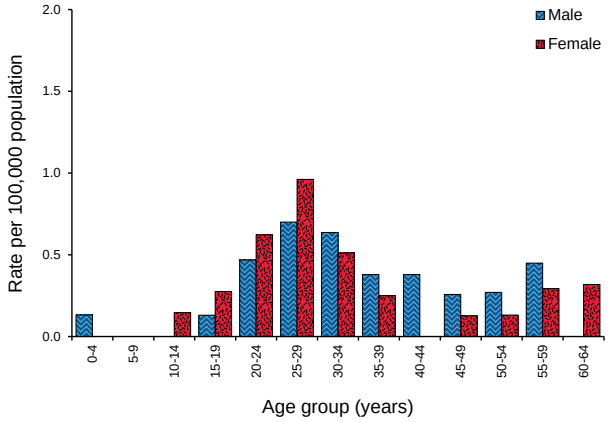


tion unknown. The majority of imported cases were from Asia, South East Asia (n=9), China (n=3) and India (n=1).

Age and sex distribution

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

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



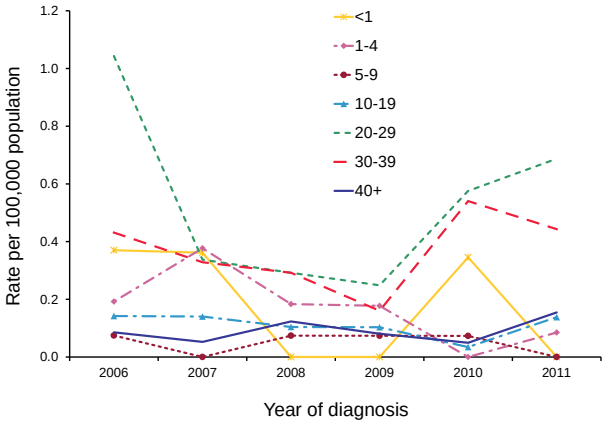
The majority of cases cluster around the young adult age groups with 74% of cases aged between 20 and 49 years of age and a median age of 31 years. The majority (75%) of female cases were notified in women of child-bearing age ranging from 15 to 36 years of age.

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

Vaccination status

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

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



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

Tetanus

Tetanus is an acute, often fatal disease caused by the toxin produced by the bacterium *Clostridium tetani*. Tetanus spores usually enter the body through contamination of a wound with soil, street dust or animal or human faeces.[14] The neurotoxin acts on the central nervous system to cause muscle rigidity with painful spasms. Generalised tetanus, the most common form of the disease, is characterised by increased muscle tone and generalised spasms. Early symptoms and signs include increased tone in the jaw muscles, difficulty in swallowing, stiffness or pain in the neck, shoulder and back muscles. In Australia, tetanus is rare, occurring primarily in older adults who have never been vaccinated or were vaccinated in the remote past.[44]

Tetanus vaccination stimulates the production of antitoxin, which protects against the toxin produced by the organism. Complete immunisation (3 primary doses and 2 boosters included for children on the NIP) induces protective levels of antitoxin lasting throughout childhood but by middle age, about 50% of vaccinees have low or undetectable levels. It is recommended, though not funded under the NIP, that all adults who reach 50 years of age and have not received a booster of a tetanus-containing vaccine in the previous 10 years should do so.[44] Results from the 2006 Adult Vaccination Survey indicate that uptake of this booster vaccine is likely to be low and decrease with increasing age

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

Epidemiological situation in 2011

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

Varicella zoster virus infections

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

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

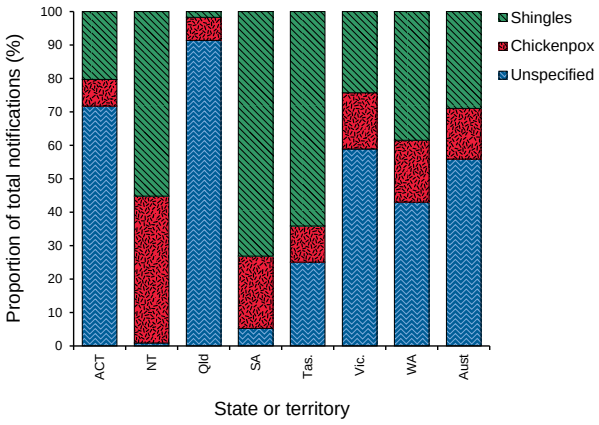
Epidemiological situation in 2011

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

Varicella zoster virus infection (unspecified)

Notifications of unspecified VZV infections are laboratory specimens that are positive for VZV but have not been followed up by the local health authority and distinguished clinically as either chickenpox or shingles.

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



* Excluding New South Wales.

Epidemiological situation in 2011

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

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

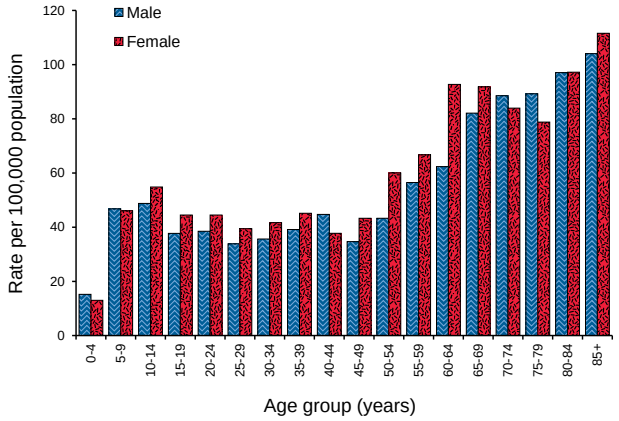
Age and sex distribution

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

Chickenpox

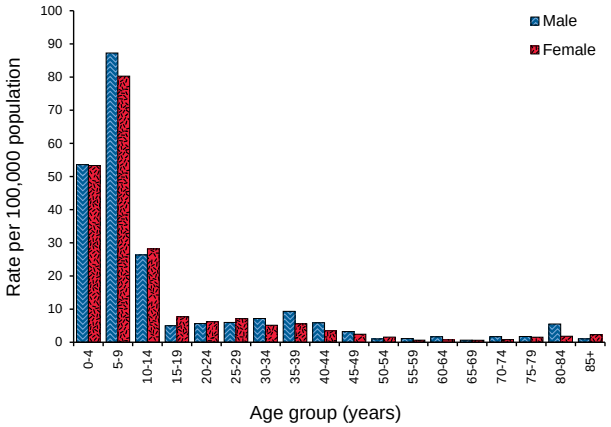
Chickenpox is a highly contagious infection spread by air-borne transmission of droplets from the upper respiratory tract or from the vesicle fluid of the skin lesions of a patient with chickenpox or shingles infection. Chickenpox is usually a mild disease of childhood; however, complications occur in approximately 1% of cases. It is more severe in

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



* Excluding New South Wales.

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



* Excluding New South Wales.

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

Epidemiological situation in 2011

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

Age and sex distribution

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

Vaccination status

In November 2005, the monovalent varicella zoster vaccine was added to the NIP as a single dose due at 18 months of age (for children born on or after 1 May 2004), or as a catch-up dose at 10–13 years of age. In 2011, children born in 2004 and eligible for the 18-month dose would be 7 years of age or younger and as follow-up of cases does not routinely occur in those older than 7 years, and analysis of vaccination status is restricted to this cohort. Information was available for 51% (n=525) of the 1,028 children less than 8 years of age. Thirty-one per cent (n=165) were vaccinated and 69% were either not vaccinated (n=126) or less than 18 months of age and ineligible for vaccination (n=234).

Shingles

Shingles occurs most commonly with increasing age, impaired immunity, and a history of chickenpox in the first year of life. Reactivation of VZV causing shingles is thought to be due to a decline in cellular immunity to the virus, and in the majority of cases presents clinically as a unilateral vesicular rash in a dermatomal distribution. Associated symptoms may include headache, photophobia, malaise, and itching, tingling, or severe pain in the affected dermatome. In the majority of patients, shingles is an acute and self-limiting disease but complications develop in approximately 30% of cases, the most common of which is chronic severe pain or post-herpetic neuralgia.[14]

Epidemiological situation in 2011

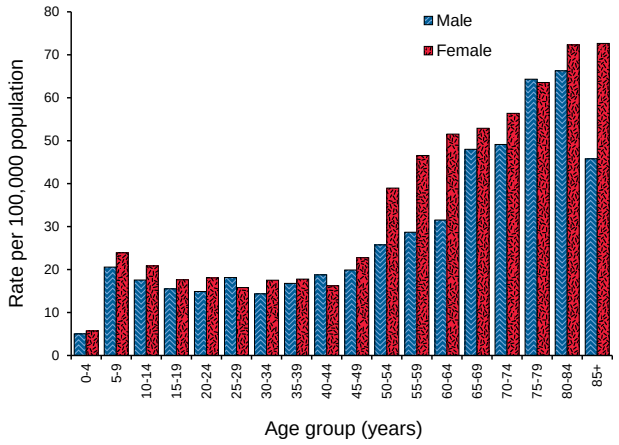
There were 3,999 notifications of shingles reported to NNDSS in 2011; a rate of 26 per 100,000 and a 34% increase compared with 2010. The highest rates of shingles occurred in South Australia, 97 per 100,000 (n=1,614) and the Northern Territory, 81 per 100,000 (n=186). High rates in these jurisdictions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

Age and sex distribution

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

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

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



* Excluding New South Wales.

Vectorborne diseases

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

Alphaviruses

Viruses in the genus *Alphavirus* that are notifiable in Australia are BFV and RRV. These viruses are unique to the Australasian region.[71] Infection can cause a clinical illness, which is characterised by fever, rash and polyarthritis. The viruses are transmitted by numerous species of mosquito that breed in diverse environments.[72] The alphavirus chikungunya is not nationally notifiable, and thus not included in this annual report, but it is notifiable in all states and territories except the Australian Capital Territory, and states and territories send information about cases to the Commonwealth for national collation and analysis.[70,73]

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

Barmah Forest virus infection

Epidemiological situation in 2011

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

Seasonality and place of acquisition

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

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

Age and sex distribution

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

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

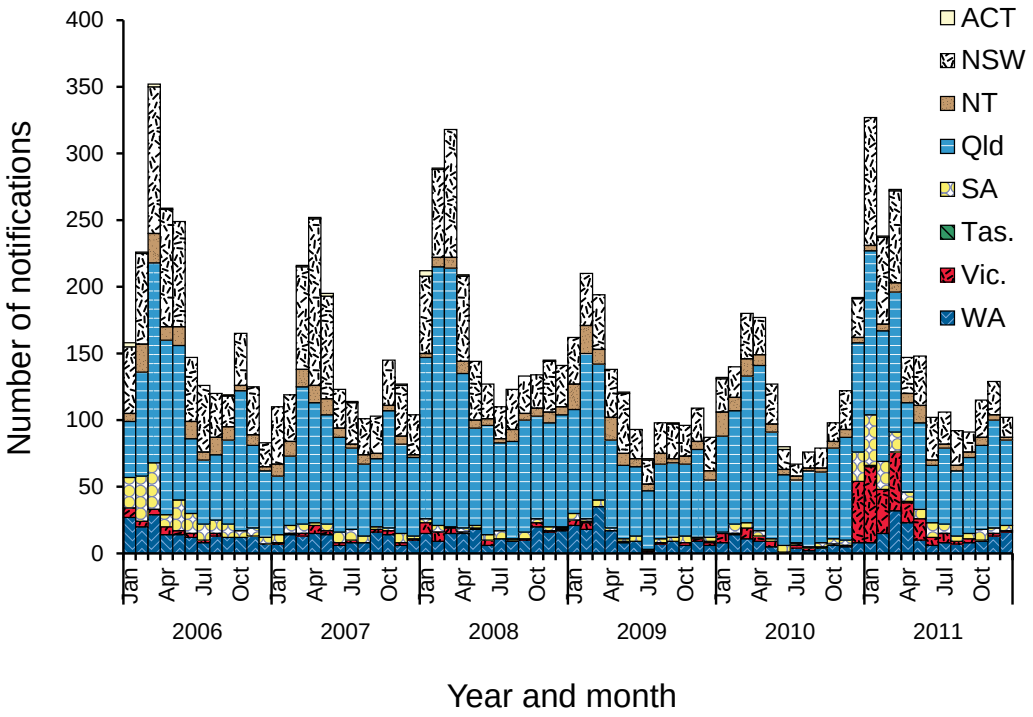
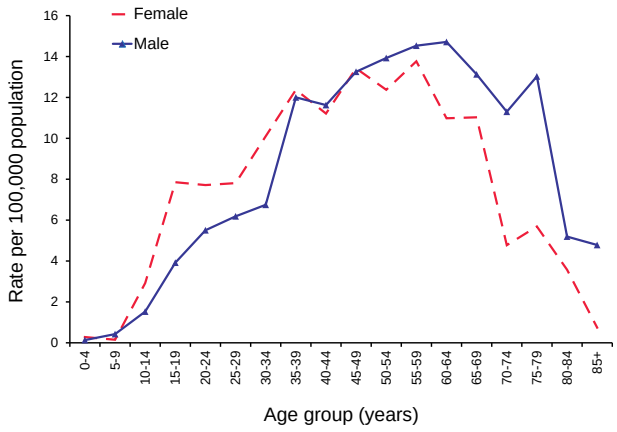


Figure: 63: Notification rates for Barmah Forest virus infection, 2011, by age and sex (n=1,869)*



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

Ross River virus infection

Epidemiological situation in 2011

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

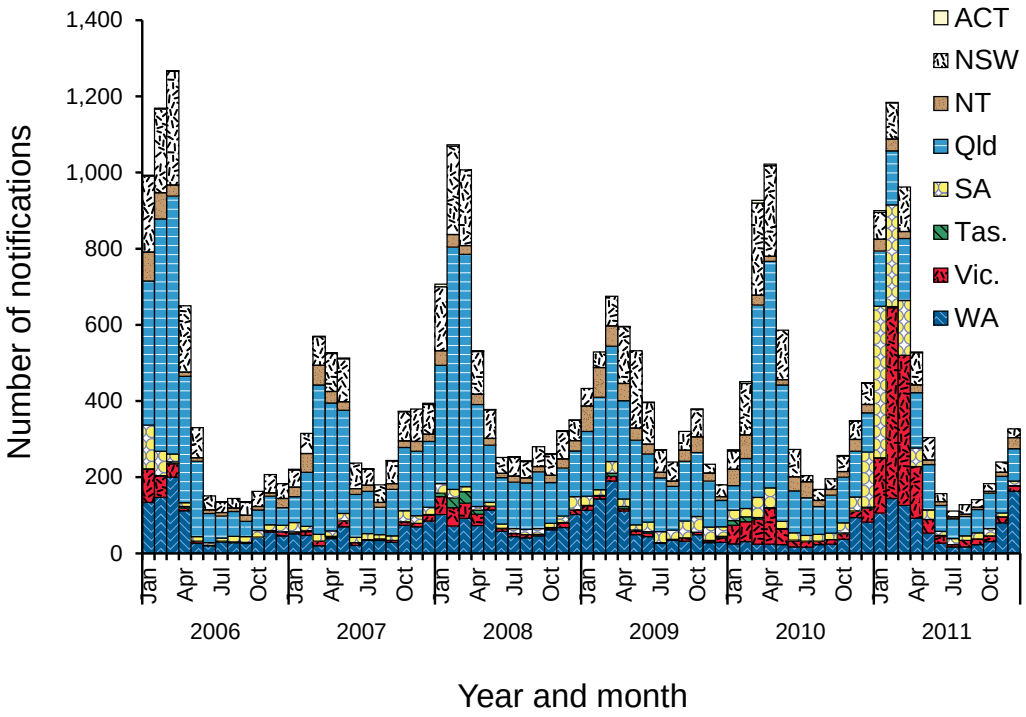
Seasonality

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

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

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

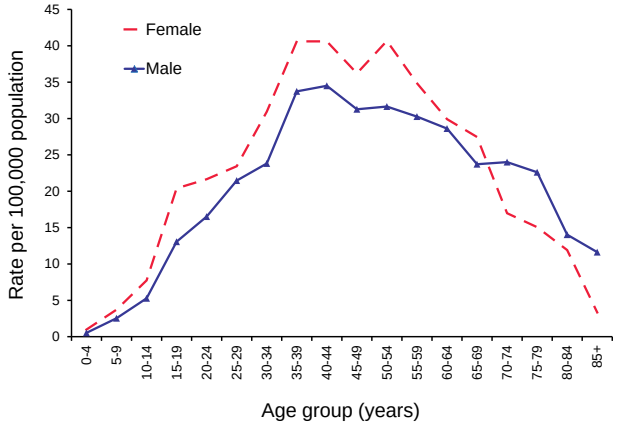
Figure: 64: Notifications of Ross River virus infection, Australia, 2006 to 2011, by month and year and state or territory



Age and sex distribution

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

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



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

Flaviviruses

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

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

Infection with MVEV, KUNV or JEV is usually asymptomatic or produces a non-specific illness, but a small percentage of cases progress to encephalomyelitis of variable severity. *Culex annulirostris* is the major vector of MVEV, JEV and KUNV. No specific treatment is available for these diseases and

care is largely supportive. A vaccine is available to prevent JEV infection,[44] but there are no vaccines currently for DENV, MVEV or KUNV infection.

Arbovirus NEC

Epidemiological situation in 2011

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

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

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

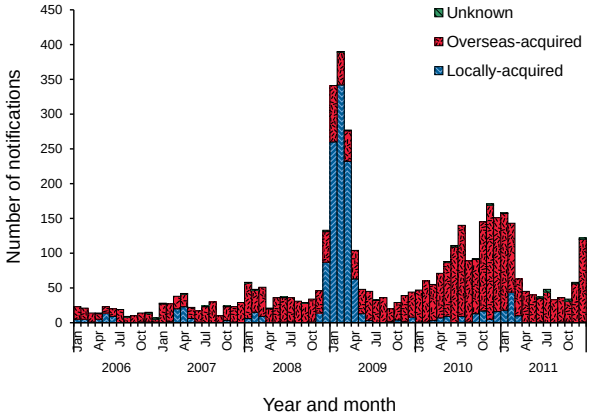
Dengue virus infection

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

Epidemiological situation in 2011

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

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



Serotype of dengue virus infections

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

Seasonality and place of acquisition

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

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

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

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

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

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

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

Most of the 76 locally-acquired cases in 2011 were known to have been associated with one of 3 outbreaks of locally-acquired infection that occurred in Queensland in 2011. The largest of these was an outbreak of DENV 2 in Cairns and Innisfail, and was related to an importation from Papua New Guinea in 2010.[70] An outbreak of 13 cases of DENV 4 occurred in Innisfail between January and March, but the source of the outbreak was unknown and an outbreak of 9 cases of DENV 1 in Townsville was linked to an importation from Bali.[82] One locally-acquired case in Western Australia was health-care associated; a physician in Perth sustained a needle-stick injury whilst taking blood, 5 days prior to symptom onset.[83]

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

Age and sex distribution

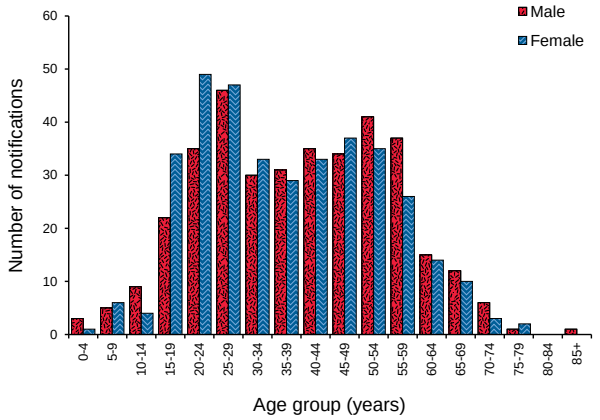
DENV infections acquired overseas in 2011 were most commonly reported amongst younger and middle aged adults (median 37.5 years, range 0–86 years), with a peak of notifications amongst males aged 20–24 and 25–29 years and females aged 25–29 years (Figure 67). Males and females each comprised 50% of overseas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

Kunjin virus infection

Epidemiological situation in 2011

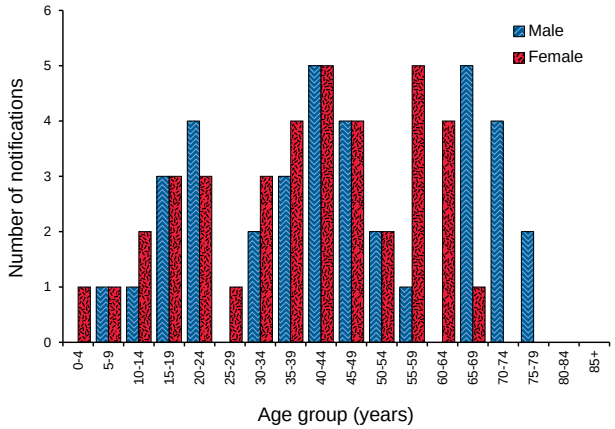
In 2011, there were 2 notifications of KUNV infections in Australia, compared with an average of 1.8 cases per year between 2006 and 2010.

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



* Age was not available for 1 case.

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



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

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

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

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

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

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

Japanese encephalitis virus infection

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

Murray Valley encephalitis virus infection

Epidemiological situation in 2011

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

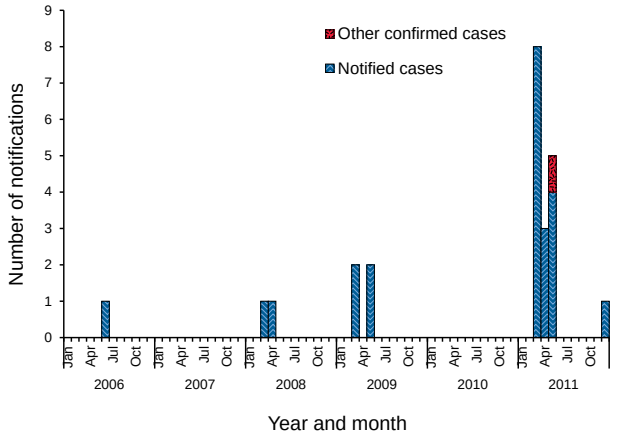
The outbreaks of MVEV infection in humans occurred in the context of the previously mentioned widespread evidence of seroconversions in sentinel chickens to flaviviruses and outbreaks of arboviral disease (KUNV and RRV) in horses (see RRV infections, Epidemiological situation).[75]

Seasonality and place of acquisition

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

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

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



Age and sex distribution

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

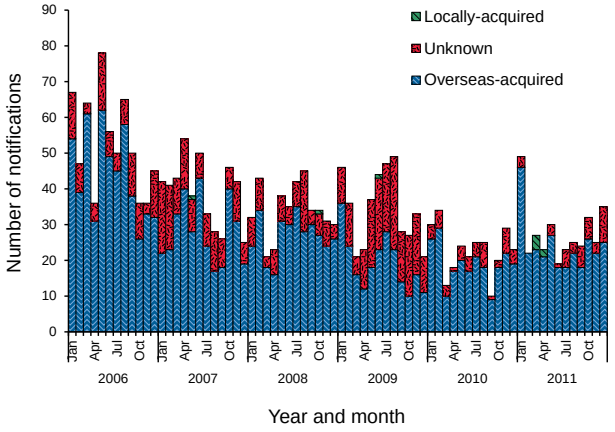
Malaria

Malaria is caused by a protozoan parasite in the genus *Plasmodium*, and 5 species are known to infect humans; *Plasmodium vivax, Plasmodium falciparum, Plasmodium malariae, Plasmodium ovale* and *Plasmodium knowlesi,*[14,84] Malaria is a serious acute febrile illness that is transmitted from person-to-person via the bite of an infected mosquito of the genus *Anopheles*. Malaria is the most frequently reported cause of fever in returned travellers world-wide.[85] Australia was declared free of malaria in 1981,[86] but suitable vectors are present in Northern Australia, and the area remains malaria-receptive. A recent case series in the Northern Territory showed that malaria cases were reported in travellers returning from endemic areas, but also reflected current events such as military operations and increased refugee arrivals from particular areas.[87]

Epidemiological situation in 2011

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

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



Seasonality and place of acquisition

Most infections in 2011 were acquired overseas, but 6 locally-acquired cases were associated with an outbreak in the Torres Strait in April 2011.[88] The last outbreak of locally-acquired infection on the mainland was in North Queensland in 2002.[89]

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

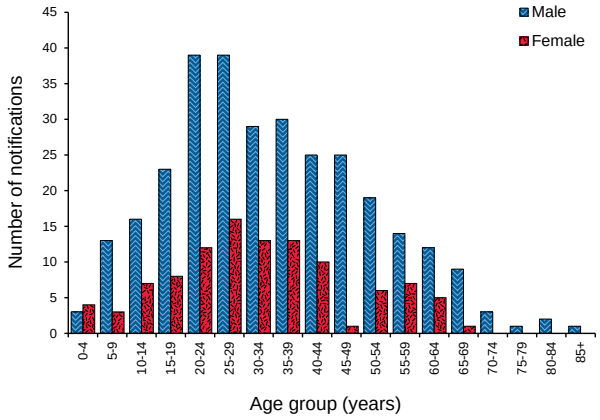
There was no discernible seasonality in notifications between 2006 and 2010, but in 2011, 13% of notifications were for cases diagnosed in January,

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

Infecting species for malaria infections

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

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



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

Age and sex distribution

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

Zoonoses

Zoonoses are those diseases and infections that are naturally transmitted between vertebrate animals and humans.[90] Approximately 60%–70% of emerging human infectious diseases are zoonoses [91,92,93] and more than 70% of emerging zoonoses originate from wildlife.[92] An emerging zoonosis is defined by WHO as 'a zoonosis that is newly recognised or newly evolved, or that has occurred previously but shows an increase in incidence or expansion in geographical, host or vector range'.[94]

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

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

Anthrax

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

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

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

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

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

Epidemiological situation in 2011

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

There were no reports of anthrax in livestock in Australia in 2011, and the last reported case of anthrax in livestock was in November 2010.[99]

Australian bat lyssavirus and lyssavirus (unspecified) infections

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

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

Epidemiological situation in 2011

There were no notifications of ABLV or lyssavirus (unspecified) in Australia in 2011. Subsequent to this reporting period, a fatal case of ABLV infection was reported in Queensland in 2013, for a total of 3 cases of ABLV infection in humans (1996, 1998 and 2013). All cases occurred after close contact with an infected bat and all were fatal.[104–106] In 2013, the Queensland Department of Agriculture, Fisheries and Forestry confirmed ABLV infection in 2 horses on a Queensland property. These were the first known equine cases of ABLV infection.[107,108] The Bat Health focus group in the Australian Wildlife Health networks gathers and collates information from a range of organisations on testing of bats for ABLV. In 2011 there were 7 ABLV detections compared with 9 detections in bats during 2010.[109]

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

Brucellosis

Brucella species that can cause illness in humans include *Brucella melitensis* acquired from sheep and goats, *Brucella suis* from pigs and *Brucella abortus* from cattle. *B. abortus* was eradicated from Australian cattle herds in 1989 and *B. melitensis* has never been reported in Australian sheep or goats.[95] Therefore, all cases of *B. melitensis* or *B. abortus* in Australia are related to overseas travel. *B. suis* is confined to some areas of Queensland, where it occurs in feral pigs. Eales et al (2010) found that feral pig hunting was the most common risk factor for infection for brucellosis cases in Townsville during 1996 to 2009.[110]

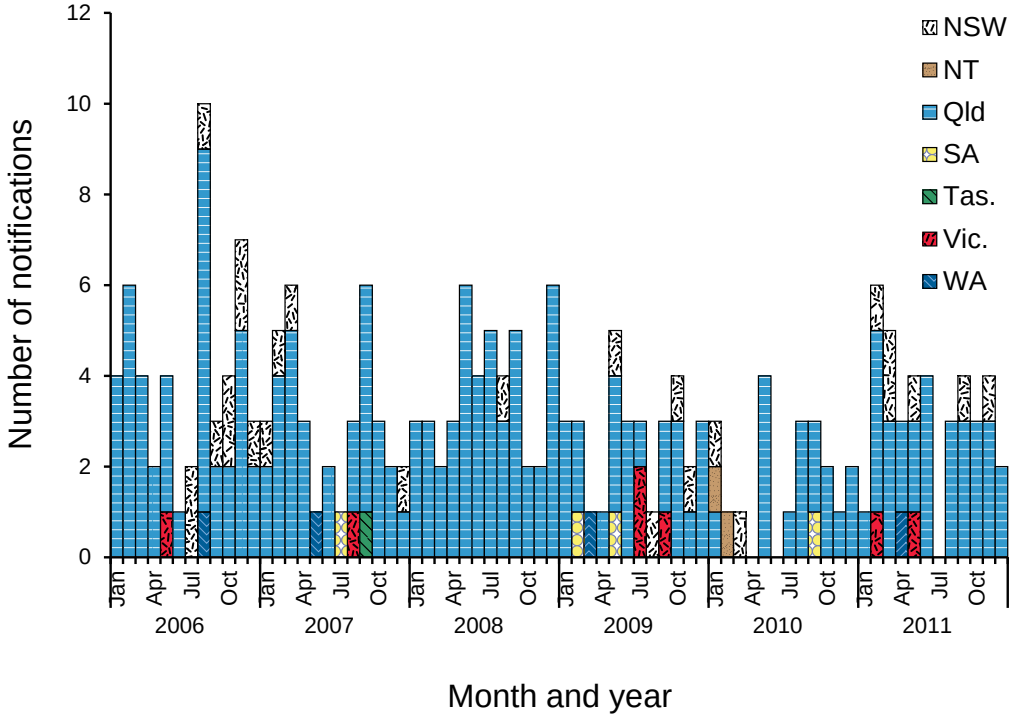
Internationally, brucellosis is mainly an occupational disease of farm workers, veterinarians, and abattoir workers who work with infected animals or their tissues.[14]

Epidemiological situation in 2011

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

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

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



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

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

Leptospirosis

Leptospirosis is caused by spirochaetes of the genus *Leptospira,* which is found in the genital tract and renal tubules of domestic and wild animals. In affected areas, where there is exposure to infected urine of domestic and wild animals, this disease can be an occupational and recreational hazard (such as in certain agricultural sectors and swimming or wading in contaminated water).[111,112] The last reported death in Australia attributed to leptospirosis was in 2002.[113]

Epidemiological situation in 2011

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

Age and sex distribution

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

Typing information

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

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

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

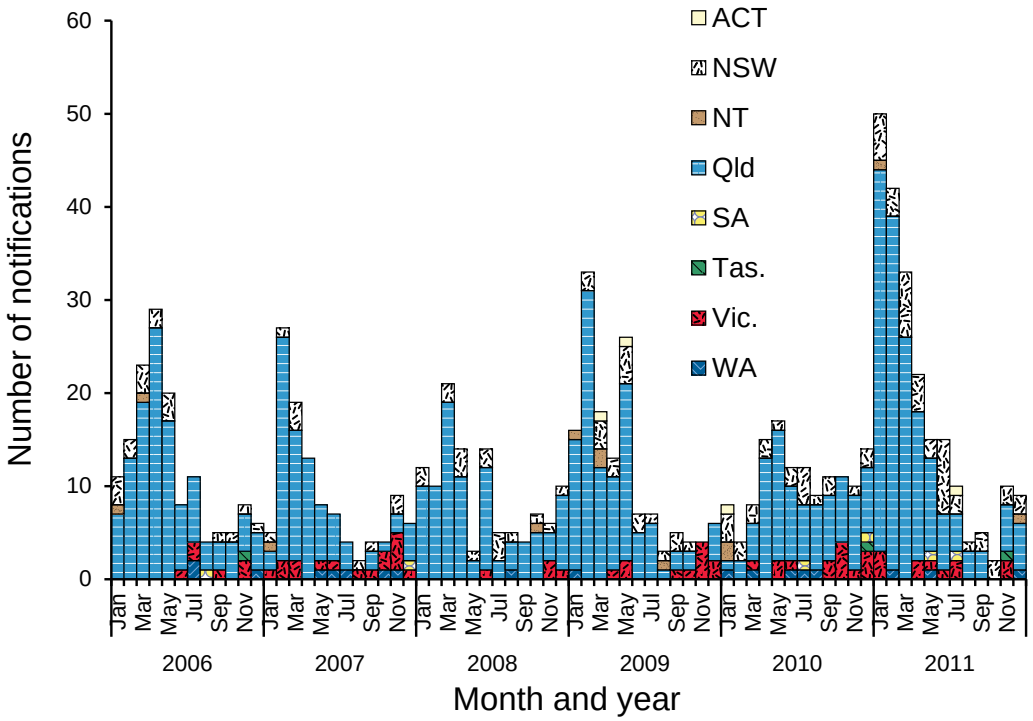
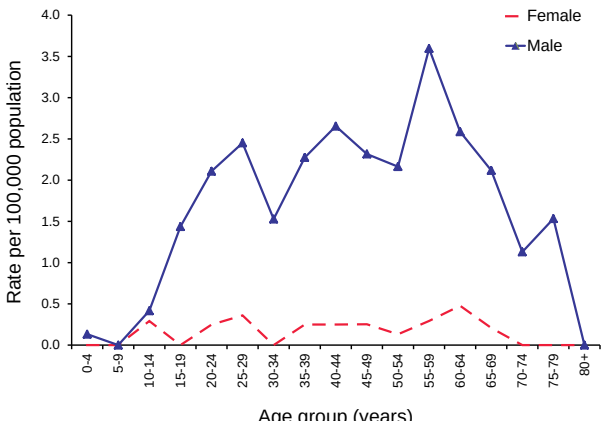


Figure 74: Notification rate for leptospirosis, Australia, 2011, by age group and sex (n=217)



Ornithosis

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

Epidemiological situation in 2011

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

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

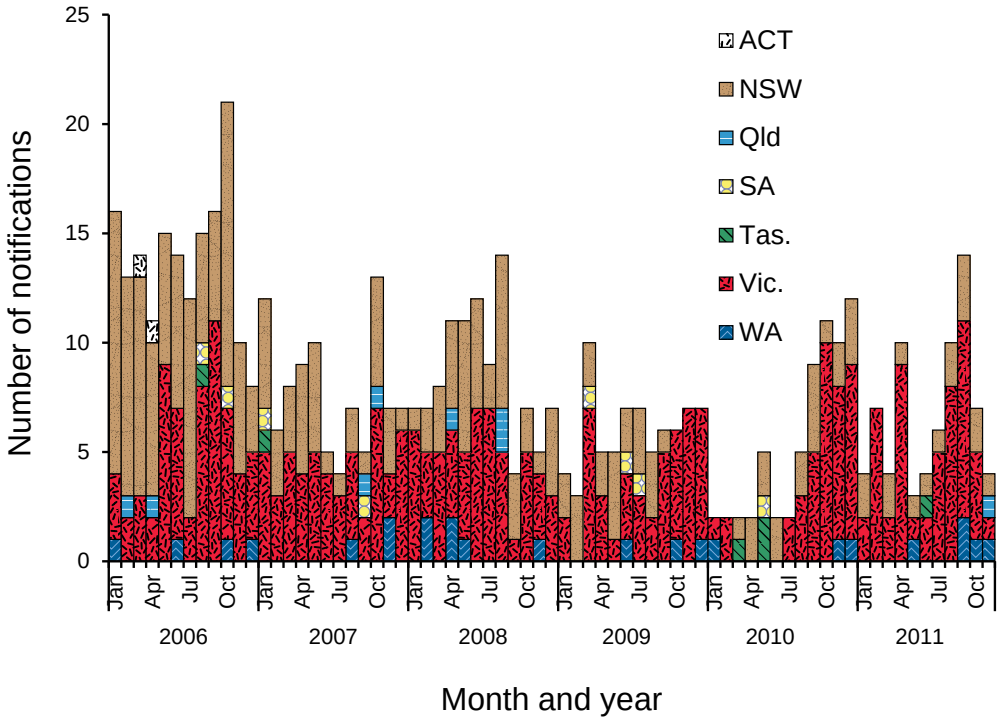
Age and sex distribution

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

Q fever

Q fever is caused by infection with the bacterium, *Coxiella burnetii*. The primary reservoirs of these bacteria are cattle, sheep and goats. *C. burnetii* is resistant to environmental conditions and many common disinfectants.[119] Q fever is most commonly transmitted via the airborne route, where the organism is carried in dust contaminated

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



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

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

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

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

Epidemiological situation in 2011

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

Between 1991 and 2001, and prior to the introduction of the NQFMP, Q fever notification rates ranged from between 2.5 and 4.9 per 100,000.[124] In 2011, the highest notification rates were from Queensland (3.6 per 100,000, n=164) and New South Wales (1.8 per 100,000, n=131). Cases also occurred in Victoria (n=24), Western Australia (n=10) and South Australia (n=7). There was 1 notification each from the Australian Capital Territory and the Northern Territory and none from Tasmania (Figure 76).

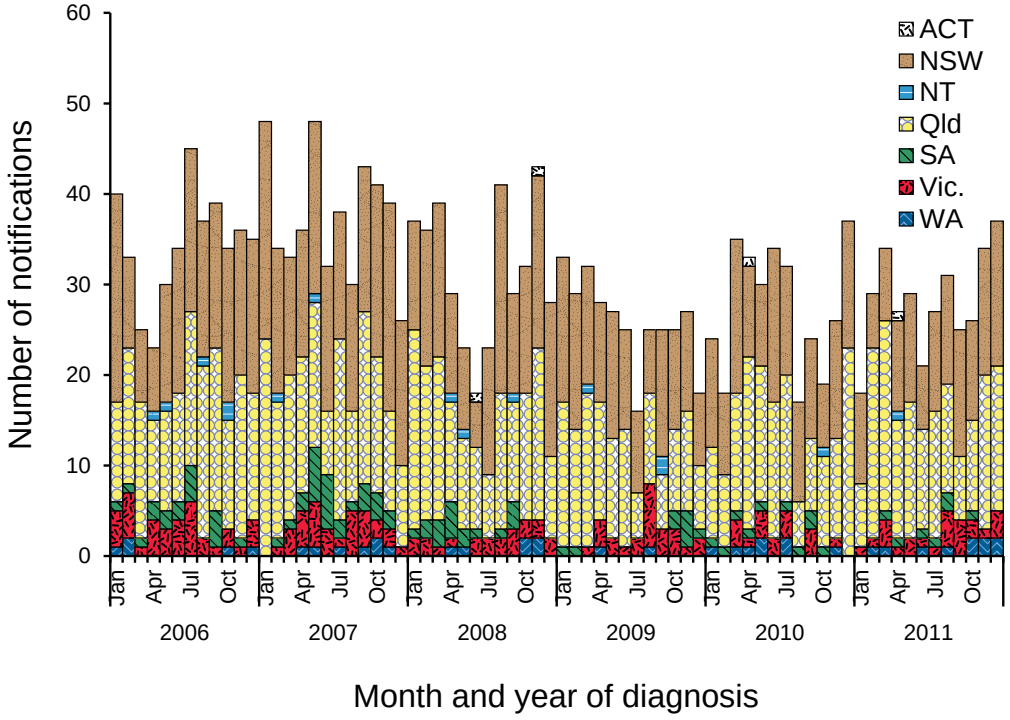
Age and sex distribution

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

Tularaemia

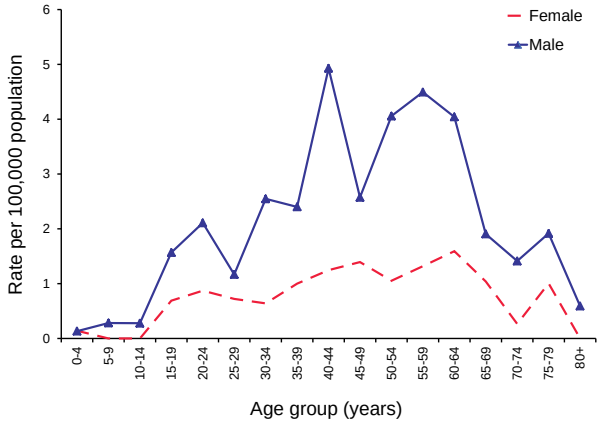
Tularaemia is caused by infection with the bacterium *Francisella tularensis*. The most common modes of transmission are through arthropod bites, handling infected animals, inhalation of infectious aerosols or exposure to contaminated food or water. Small mammals such as rodents, rabbits and hares are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

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



* No notifications from Tasmania in 2011.

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



Epidemiological situation in 2011

In 2011, there were 2 notifications of tularaemia, both from Tasmania with exposure to sick or injured wildlife. This is the first time that *F. tularensis* type B had been detected in the Southern Hemisphere.[126] Both cases were in women who had been bitten by possums along the same stretch of road in a remote location, one in February and one in September 2011.[127]

Other bacterial infections

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

Legionellosis

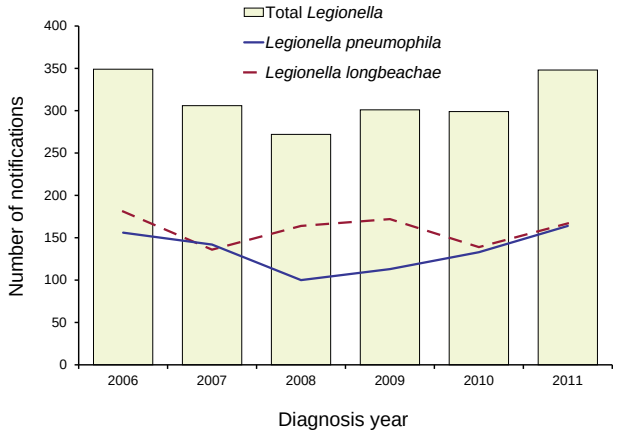
Legionellosis, caused by the bacterium *Legionella,* can take the form of either Legionnaires' disease, a severe form of infection of the lungs or Pontiac fever, a milder influenza-like illness. The species that are most commonly associated with human disease in Australia are *L. pneumophila* and *L.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

Epidemiological situation in 2011

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

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

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



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

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

Mortality data were available for 62% of notifications in 2011. There were 8 reported deaths due to legionellosis, which was an increase on the 7 deaths reported in 2010. Half of the deaths were associated with *L. pneumophila* infection and the remaining half was associated with *L. longbeachae* (Table 18). Mortality data should be interpreted with caution given the large proportion of cases reported without death data to the NNDSS.

Geographical distribution

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

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

Age and sex distribution

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

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

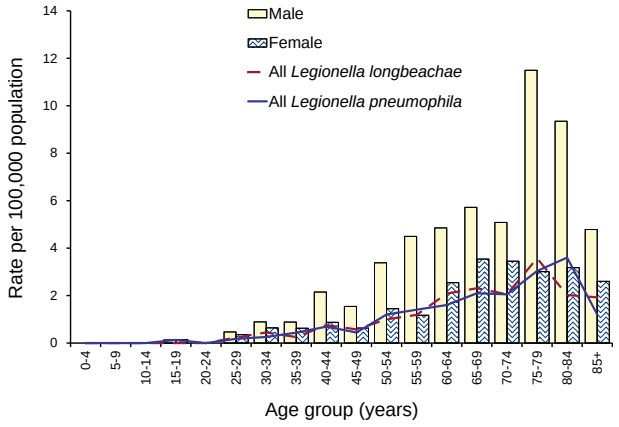
Table 18: Notifications of legionellosis, Australia, 2011, by species and state or territory

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

* 4 deaths.

† 4 deaths.

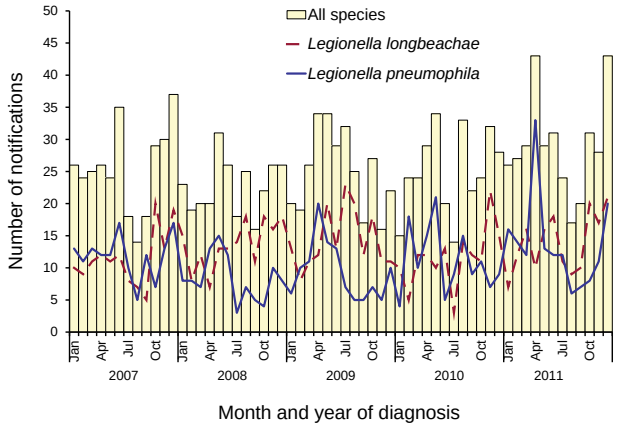
Figure 79: Notification rate for legionellosis, Australia, 2011, by age group and sex



Seasonality

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

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



Place of acquisition

Place of acquisition was reported for 68% (n=235) of legionellosis cases notified in 2011. Of cases with a place of acquisition reported, most (n=218, 93%) were reported as having been acquired within Australia. A small number (n=17) of cases was reported as acquired overseas. Indonesia (n=9) and China (n=3) were the most commonly reported overseas place of acquisition.

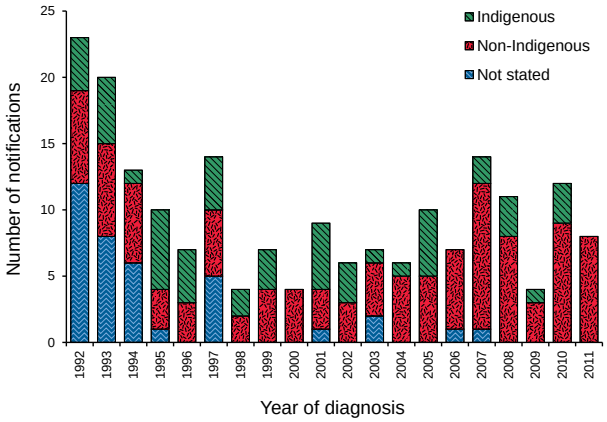
Leprosy

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

Epidemiological situation in 2011

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

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



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

Geographical distribution

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

Age and sex distribution

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

Meningococcal disease (invasive)

Meningococcal disease is caused by the bacterium *Neisseria meningiditis* and becomes invasive when bacteria enter a normally sterile site, usually the blood (septicaemia), cerebrospinal fluid (meningitis) or both. The bacterium is carried by about 10% of the population without causing disease, and is transmitted via respiratory droplets. It occasionally causes a rapidly progressive serious illness, most commonly in previously healthy children and young adults. There are 13 known serogroups of meningococcus. Globally, serogroups A, B, C, W135 and Y most commonly cause disease.[14] Historically, *N. meningitidis* serogroups B and C have been the major cause of invasive meningococcal disease (IMD) in Australia.

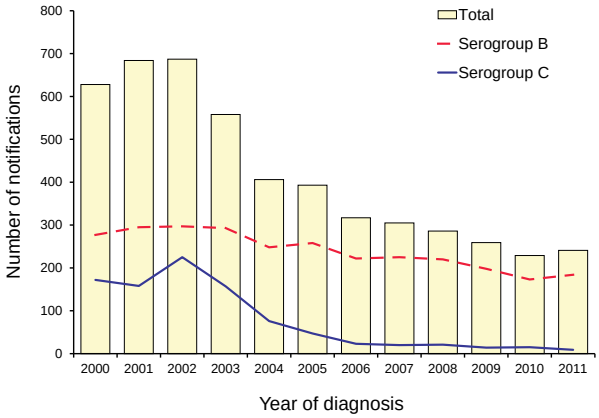
Epidemiological situation in 2011

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

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

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

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



Geographical distribution

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

Age and sex distribution

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

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

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

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

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

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

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

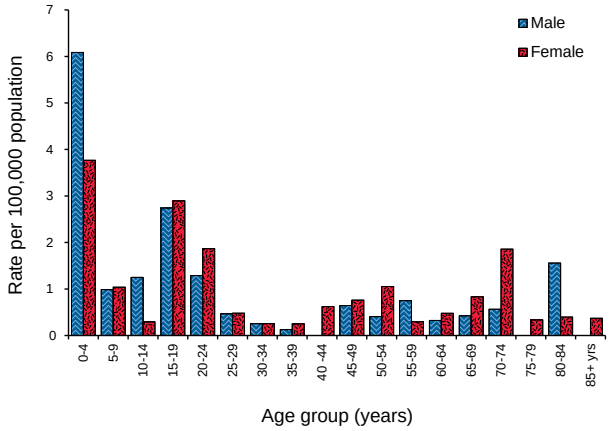
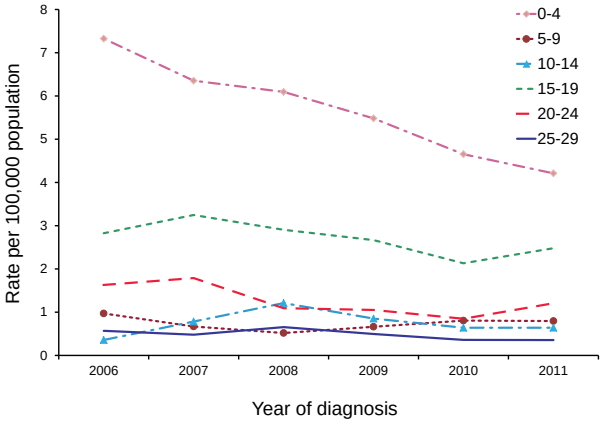


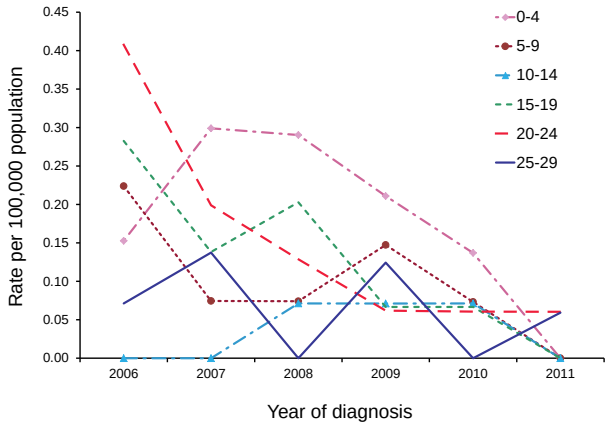
Figure 84: Notification rate for serogroup B invasive meningococcal disease, Australia, 2006 to 2011, by select age group



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

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

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



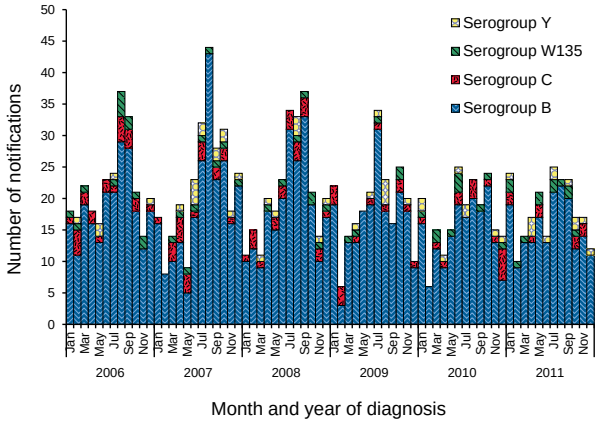
Seasonality

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

Vaccination status

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

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



Laboratory based meningococcal disease surveillance

The Australian Meningococcal Surveillance Programme (AMSP) was established in 1994 for the purpose of monitoring and analysing isolates of *N. meningitidis* from cases of IMD in Australia. The program is undertaken by a network of reference laboratories in each state and territory, using standardised methodology to determine the phenotype (serogroup, serotype and serosubtype) and the susceptibility of *N. meningitidis* to a core group of antibiotics. Annual reports of the AMSP are published in CDI with the latest report published for 2012.[128]

Tuberculosis

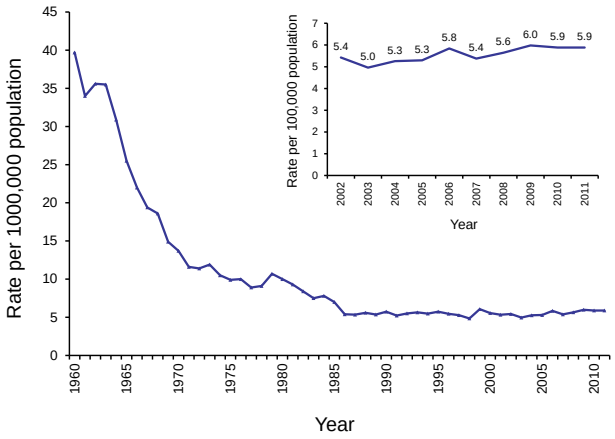
Tuberculosis (TB) is an infection caused by the bacterium *Mycobacterium tuberculosis*. TB is transmitted by airborne droplets produced by people with pulmonary or respiratory tract TB during coughing or sneezing. While Australia has one of the lowest rates of tuberculosis in the world, the disease remains a public health issue in overseas-born and Indigenous communities.

Epidemiological situation in 2011

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

At the time the annual report snap shot was agreed, the New South Wales TB data was affected by a data quality issue, resulting in an undercount of the number of TB cases in New South Wales and consequently nationally. The issue was identified and subsequently resolved in the NNDSS. For the revised case totals please refer to the NNDSS data on the Department of Health's web site (www.health.gov.au/nndssdata). The analysis presented in this report relates to data agreed in the creation of the snap shot.

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



Geographical distribution

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

Age and sex distribution

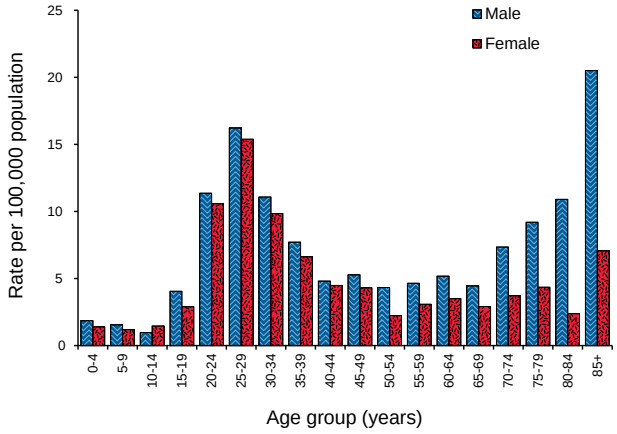
In 2011, TB was predominantly seen in young adults and older males. Males accounted for more than half (56%) of the notifications of TB, resulting in a male to female ratio of 1.3:1. Overall, the age group with the highest notification rate was the 25–29 year age group (15.8 per 100,000). The highest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

Enhanced surveillance

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

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

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



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Author details

Coordinating author: Rachael Corvisy
Data management: Mark Trungove
Bloodborne diseases: Amy Bright
Gastrointestinal diseases: Rachael Corvisy, Gerard Fitzsimmons
Quarantinable diseases: Katrina Knope
Sexually transmissible infections: Amy Bright
Vaccine preventable diseases: Nicolee Martin, Kellie Gavin, Christina Bareja
Vectorborne diseases: Katrina Knope
Zoonoses: Timothy Sloan-Gardner, Katrina Knope
Other bacterial infections: Nicolee Martin, Christina Bareja

With contributions from:

National organisations

Communicable Diseases Network Australia and subcommittees
Australian Childhood Immunisation Register
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Australian Sentinel Practice Research Network
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The Kirby Institute
National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases
National Enteric Pathogens Surveillance Scheme
OzFoodNet Working Group
World Health Organization Collaborating Centre for Reference and Research on Influenza

State and territory health departments

Communicable Diseases Control, ACT Health, Australian Capital Territory
Communicable Diseases Surveillance and Control Unit, NSW Ministry of Health, New South Wales
Centre for Disease Control, Northern Territory Department of Health and Community Services, Northern Territory
Communicable Diseases Branch, Queensland Health, Queensland
Communicable Disease Control, South Australian Department of Health, South Australia
Communicable Diseases Prevention Unit, Department of Health and Human Services, Tasmania
Health Protection Branch, Department of Health, Victoria
Communicable Diseases Control Directorate, Department of Health, Western Australia

Abbreviations

7vPCV	7 valent pneumococcal conjugate vaccine
13vPCV	13 valent pneumococcal conjugate vaccine
23vPPV	23 valent pneumococcal polysaccharide vaccine
ABLV	Australian bat lyssavirus
AFP	acute flaccid paralysis
AGSP	Australian Gonococcal Surveillance Programme
AIDS	acquired immunodeficiency syndrome
AMSP	Australian Meningococcal Surveillance Programme
ANCJDR	Australian National Creutzfeldt-Jakob Disease Registry
ATAGI	Australian Technical Advisory Group on Immunisation
BFV	Barmah Forest virus
CDI	Communicable Diseases Intelligence
CDNA	Communicable Diseases Network Australia
CDWG	Case Definitions Working Group
CJD	Creutzfeldt-Jakob disease
COB	Country of birth
CRS	congenital rubella syndrome
DENV	dengue virus
Hib	*Haemophilus influenzae* type b
HIV	human immunodeficiency virus
HPAIH	highly pathogenic avian influenza in humans
HRIG	human rabies immunoglobulin
HUS	haemolytic uraemic syndrome
IMD	invasive meningococcal disease
IPD	invasive pneumococcal disease
JEV	Japanese encephalitis virus
KUNV	Kunjin virus
MMR	measles-mumps-rubella
MVEV	Murray Valley encephalitis virus
NAI	Neuraminidase inhibition
NAMAC	National Arbovirus and Malaria Advisory Committee
NDP	no data provided
NEC	not elsewhere classified
NIP	National Immunisation Program
NN	not notifiable
NNDSS	National Notifiable Diseases Surveillance System
NQFMP	National Q Fever Management Program
NSC	National Surveillance Committee
PCR	polymerase chain reaction
RRV	Ross River virus
RVC	Regional Verification Commission
SARS	severe acute respiratory syndrome
STEC	Shiga toxin-producing *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
YFV	yellow fever virus

Appendices

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

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

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

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

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

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

Appendix 3: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2011, by notifiable disease*

Disease name	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
Arbovirus infection (NEC)	0	0	0	20	4	0	24	86.5	20	4
Barmah Forest virus infection	37	8	1	623	810	391	1,870	62.4	669	1,201
Botulism	0	0	0	1	0	1	2	50.0	1	1
Brucellosis	0	0	0	23	16	0	39	86.7	23	16
Campylobacteriosis	200	24	16	9,070	8,148	259	17,717	52.3	9,310	8,407
Chlamydial infection	5,938	767	339	33,955	20,882	18,919	80,800	58.7	40,999	39,801
Cholera	0	0	0	2	4	0	6	60.0	2	4
Cryptosporidiosis	208	3	4	992	466	135	1,808	73.9	1,207	601
Dengue virus infection	3	1	1	663	123	26	817	83.1	668	149
Diphtheria	0	0	0	1	3	0	4	50.0	1	3
Gonococcal infection	4,045	345	145	3,705	1,688	2,159	12,087	79.1	8,240	3,847
Haemolytic uraemic syndrome	1	0	0	12	0	0	13	100.0	13	0
Haemophilus influenzae type b	2	0	0	11	0	0	13	100.0	13	0
Hepatitis A	1	0	1	133	9	0	144	95.9	135	9
Hepatitis B (newly acquired)	13	1	1	145	27	3	190	91.0	160	30
Hepatitis B (unspecified)	206	27	6	2,066	1,992	2,332	6,629	65.1	2,305	4,324
Hepatitis C (newly acquired)	67	0	0	287	39	7	400	91.2	354	46
Hepatitis C (unspecified)	550	12	23	3,156	3,497	2,623	9,861	55.9	3,741	6,120
Hepatitis D	1	0	0	32	2	8	43	70.6	33	10
Hepatitis E	0	0	0	37	3	0	40	93.2	37	3
Influenza (laboratory confirmed)	1,033	46	31	10,738	10,233	5,068	27,149	46.9	11,848	15,301
Kunjin virus infection	0	0	0	2	0	0	2	100.0	2	0
Legionellosis	6	0	0	307	30	5	348	87.8	313	35
Leprosy	0	0	0	8	0	0	8	100.0	8	0
Leptospirosis	9	1	0	164	42	1	217	95.1	174	43
Listeriosis	2	0	0	59	8	1	70	92.8	61	9
Malaria	1	9	2	334	59	6	411	91.7	346	65
Measles	10	0	0	173	8	2	193	93.6	183	10
Meningococcal disease (invasive)	22	2	0	206	11	0	241	97.5	230	11

Appendix 3 *continued*: Indigenous status, National Notifiable Diseases Surveillance System, Australia, 2011, by notifiable disease*

Disease name	Aboriginal but not TSI origin	TSI but not Aboriginal origin	Aboriginal and TSI origin	Not Indigenous	Not stated	Blank/ missing	Total	% complete	Number complete	Number incomplete
Mumps	2	0	0	96	31	26	155	72.8	98	57
Murray Valley encephalitis virus infection	4	0	0	12	0	0	16	100.0	16	0
Ornithosis	0	0	0	76	6	3	85	75.1	76	9
Pertussis	673	55	61	18,771	12,515	6,527	38,602	68.4	19,560	19,042
Pneumococcal disease (invasive)	294	6	7	1,309	162	109	1,887	91.6	1,616	271
Q fever	11	0	1	239	75	12	338	89.0	251	87
Ross River virus infection	79	9	2	2,795	1,711	570	5,166	58.7	2,885	2,281
Rubella	1	0	0	44	7	6	58	76.1	45	13
Salmonellosis	360	15	17	5,915	2,976	2,984	12,267	70.8	6,307	5,960
Shigellosis	133	2	2	293	39	25	494	91.3	430	64
STEC, VTEC	2	0	0	85	5	3	95	92.3	87	8
Syphilis - congenital	3	0	0	3	0	0	6	100.0	6	0
Syphilis < 2 years	191	3	2	1,038	54	15	1,303	95.3	1,234	69
Syphilis > 2 years or unspecified duration	134	7	6	817	284	12	1,260	87.6	964	296
Tetanus	0	0	0	3	0	0	3	100.0	3	0
Tuberculosis	32	3	1	1,277	18	0	1,331	98.5	1,313	18
Tularaemia	0	0	0	2	0	0	2	100.0	2	0
Typhoid	0	0	0	127	6	1	134	90.5	127	7
Varicella zoster (chickenpox)†	145	4	0	1,727	206	12	2,094	76.3	1,876	218
Varicella zoster (shingles)†	110	3	1	3,349	504	32	3,999	73.7	3,463	536
Varicella zoster (unspecified)†	97	12	2	1,844	5,558	202	7,715	30.1	1,955	5,760
Yellow fever	0	0	0	1	1	0	2	50.0	1	1
Total	14,626	1,365	672	106,748	72,262	42,485	238,158	80.4	123,411	114,747

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

TSI Torres Strait Islander

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Australian Paediatric Surveillance Unit annual report, 2012

Marie Deverell, Yvonne Zurynski, Elizabeth Elliott, on behalf of all chief investigators of APSU surveillance studies

Introduction

This report provides an update on the surveillance conducted by the Australian Paediatric Surveillance Unit (APSU) during the period 1 January to 31 December 2012. The APSU, now in its 20th year of operation, continues to facilitate national active surveillance of uncommon communicable diseases of childhood. In 2012, the APSU conducted national surveillance for acute flaccid paralysis (AFP), congenital cytomegalovirus (cCMV), congenital rubella, perinatal exposure to HIV and HIV infection, neonatal herpes simplex virus (HSV) infection, congenital and neonatal varicella, severe complications of varicella and juvenile onset recurrent respiratory papillomatosis (JoRRP). Surveillance for the severe complications of influenza was undertaken during the influenza season June to September.

Methods

Australian Paediatric Surveillance Unit

The APSU uses standardised protocols and case definitions, which are developed in collaboration with the study investigators who provide specialised clinical expertise for each of the conditions studied. This methodology has been previously described in detail.[1] Protocols and case definitions for all conditions under surveillance are available from the APSU web site (www.apsu.org.au). Currently, 1,396 paediatricians and other child health clinicians participate in active reporting in response to a monthly report card sent by the APSU. Response rates for participating paediatricians in 2012 have remained above 90%.

Paediatric active enhanced disease surveillance

The Paediatric Active Enhanced Disease Surveillance (PAEDS) system is a joint initiative between the APSU and the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. PAEDS is a hospital-based surveillance system reliant on active case ascertainment by specialist surveillance nurses, has operated in four tertiary paediatric hospitals in four states since 2007: New South Wales, Victoria, South Australia and Western Australia. The Royal Children's Hospital, Brisbane joined PAEDS in mid 2013. PAEDS complements surveillance conducted by APSU for acute flaccid paralysis.[2]

Results

Acute flaccid paralysis

Data from APSU and the PAEDS system are pooled and submitted regularly to the Polio Expert Committee. The target of a non-polio AFP rate of ≥1 per 100,000 children less than 15 years of age has been reached for the last 5 years (2008–2012). These data have contributed to Australia fulfilling its requirements as stipulated by the World Health Organization (WHO) required AFP surveillance as part of the Global Polio Elimination Strategy and maintenance of Polio-Free Certification by the WHO.

Congenital cytomegalovirus

There was a total of 231 confirmed cases of cCMV by the end of 2012 for the total study period. Reports of cCMV have decreased over the last few years with 31 confirmed cases reported in 2010, 24 cases in 2011 and 16 cases in 2012. McMullan et al have reported that cCMV infection is underreported in Australia. Infected infants may be asymptomatic at birth and are unlikely to be identified without screening; therefore, early screening in pregnancy and neonates is vital.[4]

Congenital rubella

During 2012 there were no notifications of congenital rubella to the APSU. The last confirmed case of congenital rubella was reported to the APSU in 2008. However, the risk of congenital rubella remains, particularly among immigrant women born in countries with poorly developed vaccination programs. We need to remain vigilant with regards to vaccination to ensure there is no resurgence of disease in those unprotected in the community, as seen with the recent outbreak of measles in New South Wales. Even though there are high immunisation rates for measles, those who were unvaccinated were more susceptible to outbreaks.[5]

Table: Confirmed cases identified in 2012 and for the total study period for each condition, and reported rates per 100,000 for the relevant child population

Condition	Date study commenced	Questionnaire returned (%)	Number of confirmed cases 2012	Reported rate for 2012 (per 100,000)	Number of confirmed cases for total study period	Reported rate for total study period (per 100,000 per annum)
Acute flaccid paralysis*	Mar 1995	100	57	1.34†	717*	0.99†
Congenital cytomegalovirus	Jan 1999	71	16	5.30‡	231	6.59‡
Congenital rubella (with defects)	May 1993	No notifications	Nil	Nil	51	0.07§
Juvenile onset recurrent respiratory papillomatosis‖	Oct 2011	57	4	0.09†	4	0.05†
Perinatal exposure to HIV	May 1993	90	75	24.87‡	544	10.81‡
HIV Infection	May 1993	No notifications	Nil	Nil	83	0.11§
Neonatal herpes simplex virus infection	Jan 1997	73	9	2.98‡	138	3.45‡
Congenital varicella	May 2006	No notifications	Nil	Nil	2	0.11‡
Neonatal varicella	May 2006	50	1	0.33	19	1.09‡
Severe complications of varicella	May 2006	50	2	0.04†	49	0.17†
Severe complications of influenza¶	Influenza season each year since 2008	95	56	1.23†	276	1.29†

* Includes all cases of acute flaccid paralysis reported via the Australian Paediatric Surveillance Unit or the Paediatric Active Enhanced Disease Surveillance. All cases have been classified by the Polio Expert Panel as 'non-polio AFP' according to World Health Organization criteria.

† Based on population of children aged less than 15 years.

‡ Based on number of births.

§ Based on population of children aged less than 16 years.

‖ Cases confirmed by clinical diagnosis.

¶ Influenza surveillance was conducted each year since 2008 during the influenza season, July to September except in the pandemic year (2009) when surveillance occurred from June to October.

All reported rates based on child population estimates published by the Australian Bureau of Statistics.[3]

All of the figures were correct at the time of submission and agreed by the chief investigators for each condition. As additional information becomes available cases may be reclassified for the current year and for previous years.

Juvenile onset recurrent respiratory papillomatosis

The APSU commenced surveillance for JoRRP in 2011. To date there have been 7 notifications to the APSU with 4 confirmed clinical cases. JoRRP is a very rare condition which usually develops in childhood and is typically found in children aged less than 12 years, with a median age of 4 years. It is the most common cause of benign neoplasm of the larynx in children and is caused by human papillomavirus (HPV) infection, with HPV 6 and HPV 11 being the 2 most common causative genotypes.[6]

Perinatal exposure to HIV and HIV infection

During 2012, 75 confirmed cases of perinatal exposure to HIV were reported to the APSU. Since May 1993 there have been a total of 544 cases of perinatal exposure to HIV infection and 83 cases

of HIV infection in neonates. There were no reports of HIV infection in children reported to the APSU during 2012.

Neonatal herpes simplex virus

In 2012, there were a total of 9 cases of neonatal herpes simplex virus (HSV), and over the total study period (January 1997–December 2012) a total of 138 confirmed cases of HSV were reported to the APSU. During 2012, the HSV study results were reviewed and the case definition was amended to include disease in infants up to 1 year of age, whereas the previous study definition only included newborns. This will enable capture of late presentations and re-presentations of HSV disease.[7]

Congenital, neonatal and severe complications of varicella

No cases of congenital varicella were reported to the APSU during 2012. There was 1 confirmed case of neonatal varicella and 2 confirmed cases of children hospitalised with severe complications of varicella reported to APSU during this period, supporting the effectiveness of the varicella vaccination program which commenced in 2005.

Severe complications of influenza

A total of 56 cases of severe complications of influenza were reported to the APSU and of these 40 (71%) were male. Most cases (70%) had influenza A. A range of complications were reported: pneumonia, encephalitis, rhabdomyolysis and seizures. Fifty-four per cent of cases were admitted to the paediatric intensive care unit and a total of 6 deaths were reported in 2012 compared with 3 deaths in 2011 and 6 deaths reported to APSU in 2009 during the H1N1 pandemic. Only 4 reported paediatric cases were vaccinated for influenza in 2012. Twenty-one cases (37.5%) had received Oseltamivir in 2012; this is much lower than during the H1N1 2009 pandemic (64%).

Conclusions and future directions

Next year will mark 20 years of national surveillance by the APSU. The APSU continues to provide valuable national surveillance data on a number of serious rare childhood diseases and in some cases is the only source of national data. The information collected by the APSU informs clinicians, policy makers and the wider community.

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Chief investigators of APSU surveillance studies were:

Acute flaccid paralysis: Dr Bruce Thorley, Victorian Infectious Diseases Reference Laboratory;

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Author details

Marie Deverell[1,2]
Yvonne Zurynski[1,2]
Elizabeth Elliott[1,2,3]

1. Australian Paediatric Surveillance Unit, Kids Research Institute, Westmead, New South Wales
2. Discipline of Paediatrics and Child Health, Sydney Medical School, The University of Sydney, New South Wales
3. The Sydney Children's Hospitals Network (Randwick and Westmead), New South Wales

Corresponding author: Dr Yvonne Zurynski, Assistant Director, Australian Paediatric Surveillance Unit, The Children's Hospital at Westmead, Locked Bag 4001, WESTMEAD NSW 2145. Telephone: +61 2 9845 1202/3005. Facsimile: +61 2 9845 3082. Email: yvonne.zurynski@health.nsw.gov.au

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Flutracking weekly online community survey of influenza-like illness annual report 2011 and 2012

Sandra J Carlson, Craig B Dalton, Michelle T Butler, John Fejsa, Elissa Elvidge, David N Durrheim

Abstract

Flutracking is a national online community influenza-like illness (ILI) surveillance system that monitors weekly ILI activity and field vaccine effectiveness (FVE). This article reports on the 2011 and 2012 findings from Flutracking. There was a 22% increase in participants to 16,046 who completed at least one survey in 2012, compared with 2011 (13,101). By October 2012 (the end of the 2012 season), 54.2% of participants had received the 2012 seasonal vaccine, while by the end of the 2011 season, 55.9% of participants had received the 2011 seasonal vaccine. From 2007 to 2012 the FVE calculation for New South Wales participants demonstrated that the seasonal vaccine was effective except in 2009 when a novel H1N1 virus was dominant. The 2012 Flutracking ILI weekly incidence peaked in mid-July at 4.9% in the unvaccinated group, 1 month earlier than laboratory confirmed influenza. The 2011 Flutracking ILI weekly incidence peaked in mid-August at 4.1% in the unvaccinated group, 1 week later than laboratory confirmed influenza. Similar to laboratory notifications, there was an increase in ILI activity from 2010 to 2012, with the peak weekly ILI prevalence for 2012 Flutracking data, (unstratified by vaccination status), being higher (4.7%) than the peak weekly prevalence for 2011 (3.8%) and 2010 (3.7%). The 2012 Flutracking influenza season showed moderate levels of ILI, compared with lower levels of ILI seen in 2011 and 2010, and consistent with the increase in national influenza laboratory notifications. *Commun Dis Intell* 2013;37(4):E398–E406.

Keywords: influenza, surveillance, syndromic surveillance, influenza-like illness, survey, Flutracking.

Background

There are a number of surveillance methods that contribute to influenza surveillance in Australia each year.[1] Integrating data from each of these systems is vital to create a timely and accurate picture of influenza activity, as each surveillance method has its strengths and limitations. The Flutracking surveillance system makes an important contribution to Australian influenza surveillance by providing weekly community level influenza-like illness (ILI) attack rates that are not biased by health seeking behaviour and clinician testing practices.[2–5] The Flutracking surveillance system has been incorporated into the weekly Australian influenza report since 2009.[1]

The main aims of Flutracking have been to:

1. compare ILI syndrome rates between vaccinated and unvaccinated participants to detect inter-pandemic and pandemic influenza and provide early confirmation of vaccine effectiveness or failure;
2. provide consistent surveillance of influenza activity across all jurisdictions and over time; and
3. provide year to year comparison of the timing, incidence, and severity of influenza.

In 2011 new questions were added to the Flutracking surveillance system to document health seeking behaviour amongst participants. This enabled regular calculation of influenza burden of illness pyramids to examine the proportion of participants with ILI that sought medical care, the type of medical care sought, and the proportion tested for influenza.

This article reports on the 2011 and 2012 findings from the Flutracking ILI surveillance system.

Methods

We report on participation numbers compared with previous years, socio-demographic data, vaccination uptake for the seasonal influenza vaccine amongst participants, field vaccine effectiveness (FVE) estimates, weekly ILI estimates and comparison of these estimates with Australian laboratory influenza notifications.

Survey methodology

The Flutracking surveillance system was in operation for 24 weeks in 2011, from the week ending 8 May to the week ending 16 October 2011, and for 24 weeks in 2012, from the week ending 6 May to the week ending 14 October 2012. The recruitment methods in 2011 and 2012 were similar to those used in 2007–2010.[2] In 2011, the focus of recruitment was state-based government organisations

in Western Australia, Queensland and Victoria, with a view to expanding Flutracking to improve its national representativeness of ILI weekly prevalence comparison across states and territories. In 2012, the focus of recruitment was also to improve state-based representativeness (focusing on Victoria and Western Australia), as well as boost national participation further by contacting large government and private organisations. In 2011 and 2012, 168 and 279 organisations respectively were contacted and requested to participate in Flutracking. Social media tools, including Facebook and Twitter, as well as media releases were also used to communicate with current participants and recruit new participants.

The weekly survey in 2011 and 2012 was similar to that used in 2007 to 2010.[2] However, in 2011 the following questions were added to the initial questionnaire that participants receive upon registration (existing participants were also asked to complete these new questions):

- gender of participant
- highest level of educational attainment (for participants 15 years of age or older).

In 2011, additional questions on health seeking behaviour that allow a surveillance pyramid to be constructed were also added. These findings have been published.[6]

Aboriginal and Torres Strait Islander status was also added to the questionnaire in 2012. In addition, participants selecting both fever and cough for a particular week were asked whether they had experienced a sore throat in the 2012 survey.

Participation and vaccination rate

Peak weekly participation numbers were reported for 2012 at the national and state or territory level and compared with the participation numbers in 2008, the first year that Flutracking was expanded nationally. The participation rate (per 100,000) was calculated using the number of participants in the peak week and the March 2012 estimated resident population for each state and territory from the Australian Bureau of Statistics.[7]

The percentage of participants who completed at least one survey in 2012 and identified as Aboriginal and/or Torres Strait Islander was calculated nationally.

The percentage of participants who completed the final survey of the season and who were vaccinated with the seasonal influenza vaccine was calculated nationally for 2011 and 2012.

The proportion of participants less than 10 years of age whose parents completed the final survey of the season on their behalf and who were vaccinated with the seasonal influenza vaccine, was calculated nationally for 2011 and 2012.

The proportion of participants reporting a sore throat in the national peak week of ILI for 2012 (peak week determined using the number of participants with fever and cough divided by the total number of participants for that week), were compared with the proportion of participants in this same week with fever, cough, and fever and cough.

The proportion of participants with a sore throat in the peak 4 weeks of ILI for 2012 were compared with the proportion of participants in this same 4 weeks with fever, cough, and fever and cough.

Field vaccine effectiveness for influenza-like illness

A FVE analysis for New South Wales participants 18 years of age or older was conducted for 2011 and 2012 using a similar method to 2010.[8]

Vaccine effectiveness (VE) was calculated as follows:

VE = 100 x (1 – relative risk)

= 100 x (1 – (ILI rate in vaccinated group/ILI rate in unvaccinated group))

The peak influenza period was defined as the 4 consecutive weeks with the highest weekly Flutracking ILI rates for unvaccinated participants. Table 1 shows peak influenza periods used in yearly vaccine effectiveness calculations.

Weekly influenza-like illness prevalence and national laboratory influenza notifications

An analysis of the difference in weekly ILI prevalence amongst vaccinated and unvaccinated participants was conducted at both the national

Table 1: Peak influenza periods used in yearly vaccine effectiveness calculations

Year	Peak influenza period (week ending)
2007	29 July – 19 August
2008	17 August – 7 September
2009	5 July – 26 July
2010	15 August – 6 September
2011	22 May – 12 June
2012	27 May – 17 June

level and state or territory level for states and territories with greater than 1,000 participants from 2010 to 2012. Vaccination was defined as having received seasonal vaccine in the year of participation. Weekly ILI prevalence was reported using a definition of fever and cough in the preceding week. The unstratified ILI rates were compared with national laboratory influenza notifications for 2009 to 2012.

Results

Participation in 2011 and 2012

Amongst the 14,467 participants in the first 4 weeks of the survey in 2012, the median weekly participation rate during the 2012 survey period was 96%. Amongst the 12,109 participants who participated in the first 4 weeks of the survey in 2011, the median weekly participation rate during the 2011 survey period was 96%. Nationally, participation has more than doubled from 2008 to 2012 (Table 2). At a state or territory level, increases were most marked in the Northern Territory, South Australia, and Queensland. Tasmania had the highest rate of Flutracking participation per 100,000 persons, followed by the Northern Territory and South Australia. There were 16,046 participants who completed at least one survey in 2012, compared with 13,101 in 2011 (a 22% increase); 12,581 in 2010; 8,546 participants in 2009; 4,827 in 2008; 982 in 2007; and 394 in 2006.

The most successful recruitment strategy in 2012 was through existing participants. A *Welcome back to Flutracking for 2012* email sent to all active participants on 2 May included a suggestion that participants invite 3 people to join the survey using an email link to 'Tell-a-Friend'. There were significant increases in participant numbers on 2 May (1,032 participants enrolled), 3 May (242 participants enrolled) and 4 May (105 participants enrolled). The second successful recruitment strategy was through the first survey email (sent on 7 May), which also included the 'Tell-a-Friend' email recruitment link. Spikes in recruitment of participants with email domains of organisations that had recently distributed Flutracking recruitment invitations via email demonstrate the effectiveness of this strategy (Figure 1). A large increase in recruitment of participants immediately followed an interview discussing the Flutracking surveillance system on national radio in April 2011 (Figure 2).

Socio-demographic data

Amongst Flutracking participants who completed at least one survey each year, 66% and 64% were female in 2011 and 2012 respectively, and 24% of participants had a postgraduate degree (Table 3.)

Of those who completed at least one survey in 2012, 8,800 participants (54.8%) completed the Aboriginal/Torres Strait Islander status question. Of those who completed the question, 102 (1.2%) identified as either Aboriginal, Torres Strait Islander, or both Aboriginal and Torres Strait Islander, and 98.8% (8,698 participants) identified as neither Aboriginal or Torres Strait Islander.

Survey response time

In 2011, 42% of participants responded to the survey by the end of the first business day (5:00 pm AEST), 87% by the end of the second business day, and 93% by the end of the third business day. In 2012, 39% of participants responded to the survey

Table 2: Recruitment to Flutracking, 2008 and 2012, by state or territory

State or territory	Number of respondents (peak week) 2008	Number of respondents (peak week) 2012	Percentage positive change	Rate of Flutracking participation per 100,000 population
ACT	159	371	133	99
NSW	2,689	4,328	61	60
NT	2	587	29,250	252
Qld	158	1,315	732	29
SA	52	2,555	4,813	155
Tas	1,235	1,804	46	352
Vic	404	2,029	402	36
WA	128	718	461	30
Total	4,827	13,707	184	61

by the end of the first business day (5:00 pm), 88% by the end of the second business day, and 93% by the end of the third business day.

Proportion of participants with influenza-like illness

By the end of the 2012 season (week ending 14 October 2012), 54.2% (7,071/13,050) of participants had received the 2012 seasonal vaccine, compared with 55.9% (5,950/10,643) of participants by the end of 2011. Seasonal vaccination levels were similar each year, with 64.8% of participants vaccinated in 2010, 59.5% vaccinated in 2009, 50.7% vaccinated in 2008, and 52.9% vaccinated in 2007. Of the 2,735 participants who identified as working face-to-face with patients in 2012, 2,007 (73.4%) received the vaccine compared with 73.0% by the end of 2011, and 77.5% by the end of 2010. In 2012, 11.6% of participants less than 10 years

Figure 1: Significant recruitment events for Flutracking and impact, 2012

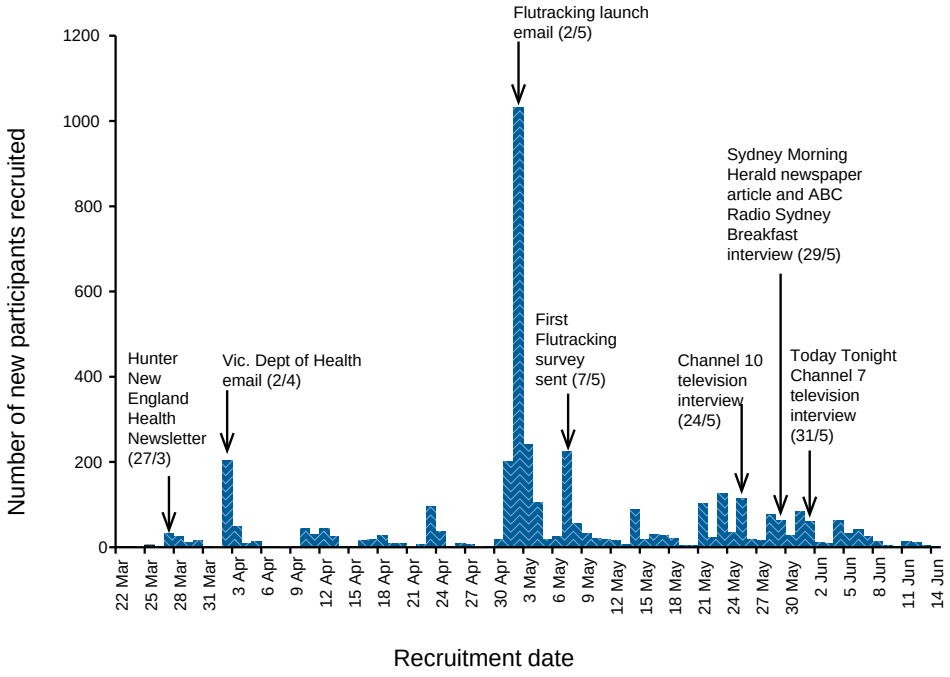


Figure 2: Significant recruitment events for Flutracking and impact, 2011

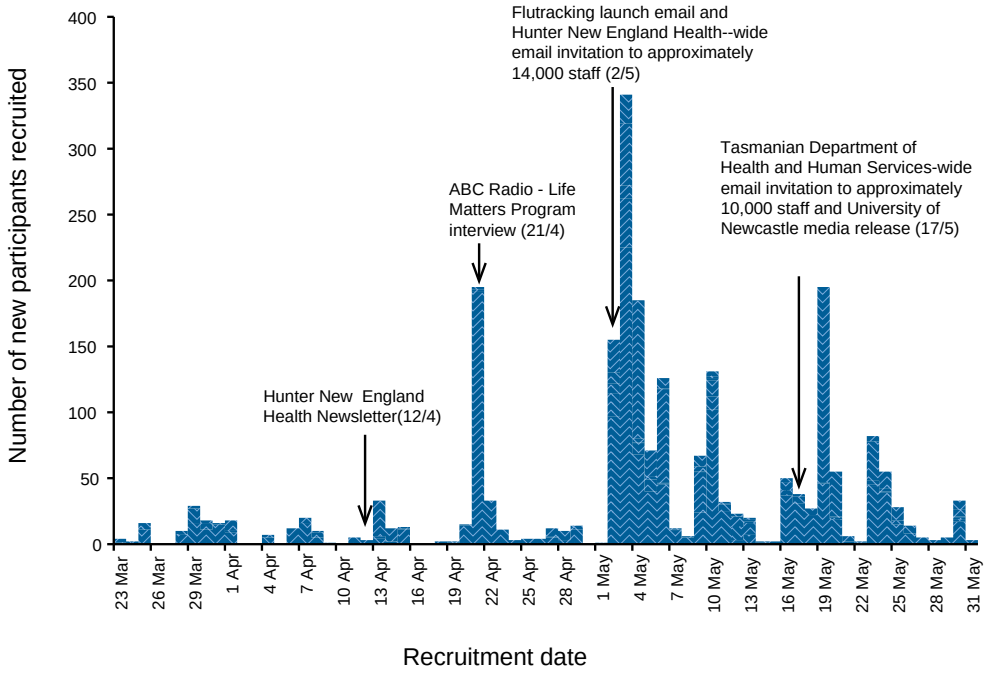


Table 3: Socio-demographic characteristics of Flutracking participants, 2011 and 2012

Age (years)	2011		2012	
	Frequency	Per cent	Frequency	Per cent
0–15	1,404	10.7	1,854	11.6
16–34	2,290	17.5	2,902	18.1
35–49	3,928	30.0	4,544	28.3
50–64	4,722	36.0	5,623	35.0
65 and over	757	5.8	1,123	7.0
Total participants	13,101	100.0	16,046	100.0
Gender				
Male	2,737	33.8	4,882	36.4
Female	5,354	66.2	8,516	63.6
Total reported	8,091	100.0	13,398	100.0
Education				
Year 10 or below (or equiv)	504	6.8	918	7.2
Year 11 (or equivalent)	225	3.1	392	3.1
Year 12 (or equivalent)	500	6.8	912	7.2
Certificate I/II/III/IV	683	9.3	1,211	9.5
Advanced Diploma/Diploma	731	9.9	1,190	9.3
Enrolled Bachelor Degree	208	2.8	428	3.4
Completed Bachelor Degree	1,674	22.7	2,871	22.5
Grad Diploma/Grad Certificate	1,079	14.6	1,762	13.8
Postgraduate Degree	1,782	24.1	3,071	24.1
Total reported (15 years and over only)	7,386	100.0	12,755	100.0
Aboriginal and/or Torres Strait Islander				
Yes	N/A	N/A	102	1.2
No	N/A	N/A	8,698	98.8
Total reported	N/A	N/A	8,800	100.0

Table 4: Count of participants vaccinated with the seasonal influenza vaccine at the final survey of each year, for all participants, participants working face-to-face with patients, and participants less than 10 years of age

Participant group	Year					
	2007	2008	2009	2010	2011	2012
All participants						
Received vaccine	52.9%	50.7%	59.5%	64.8%	55.9%	54.2%
Number of participants	726	3,893	5,216	9,109	10,643	13,050
Participants working face to face with patients						
Received vaccine	73.3%	71.2%	76.8%	77.5%	73.0%	73.4%
Number of participants	221	1,144	1,360	2,059	2,497	2,735
Participants less than 10 years of age						
Received vaccine	N/A	15.8%	18.7%	15.8%	10.6%	11.6%
Number of participants	N/A	202	284	501	623	850

of age whose parents completed a survey on their behalf were vaccinated with the seasonal influenza vaccine by the end of the season, compared with 10.6% in 2011, and 15.8% in 2010 (Table 4).

Proportion of participants with sore throat

Of the 13,707 participants who completed a survey in the national peak week of ILI for 2012 (week ending 15 July 2012), 449 participants (3.3%)

Table 5: Percentage of participants with influenza-like illness symptoms who completed a survey either in the national peak influenza-like illness week, or completed at least one survey in the national peak four weeks of influenza-like illness, 2012

Influenza-like illness symptoms	Participants who completed survey in national peak influenza-like illness week*	Participants who completed at least one survey during national peak 4 weeks influenza-like illness
Fever, cough and sore throat	3.3%	9.3%
Fever	5.7%	14.9%
Cough	16.4%	32.4%
Fever and cough	4.7%	12.1%

* Week ending 15 July 2012, N= 13,707

† Weeks ending 1 to 22 July 2012, N=14,851

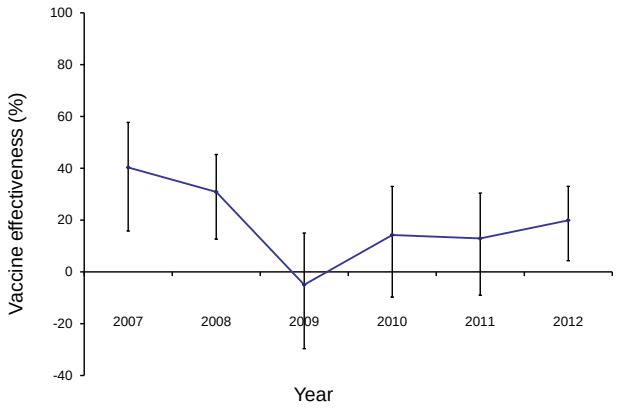
reported fever, cough and sore throat. In this same week, 785 participants (5.7%) reported fever, 2,254 participants (16.4%) reported cough, and 646 participants (4.7%) reported fever and cough (Table 5).

Of the 14,851 participants who completed at least one survey in the national peak 4 weeks of ILI for 2012 (weeks ending 1–22 July 2012, 1,377 participants (9.3%) reported fever, cough and sore throat. In the same peak 4 weeks, 2,206 participants (14.9%) reported fever, 4,814 participants (32.4%) reported cough, and 1,795 participants (12.1%) reported fever and cough (Table 5).

Field vaccine effectiveness for influenza-like illness

From 2007 to 2012 the FVE calculation for New South Wales participants demonstrated that the seasonal vaccine was effective in reducing the risk of ILI except in 2009 during the pandemic (Figure 3).

Figure 3: Field vaccine effectiveness against all influenza-like illness for peak 4 weeks in New South Wales, participants greater than 18 years of age, 2007 to 2012

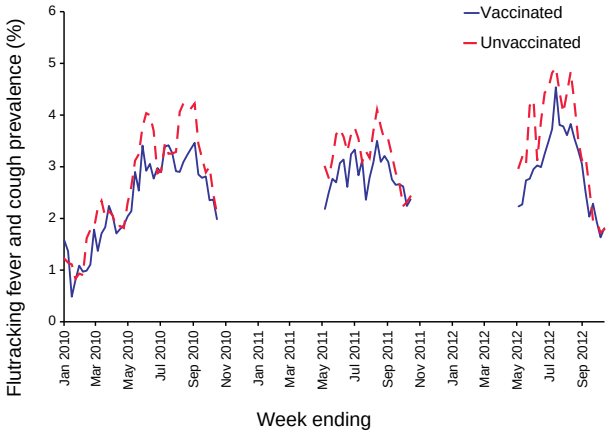


95% confidence intervals are represented by the bars in the figure

Detection of influenza-like illness

Peak ILI activity for 2012 was in mid-July (4.9% in the unvaccinated group) (Figure 4). However, the divergence between ILI prevalence for the vaccinated and unvaccinated participants was highest in early June and mid-August. The 2012 season was moderate, but higher levels of ILI were seen than in 2011 and 2010. Peak ILI activity for 2011 was in mid-August (4.1% in the unvaccinated group). However, the divergence between ILI prevalence in vaccinated and unvaccinated participants was greatest between late May and early June. Peak weekly ILI prevalence in 2011 (4.1% amongst unvaccinated participants) was similar to peak weekly ILI prevalence in 2010 (4.2% amongst unvaccinated participants).

Figure 4: Weekly national fever and cough prevalence, 2010 to 2012, by vaccination status

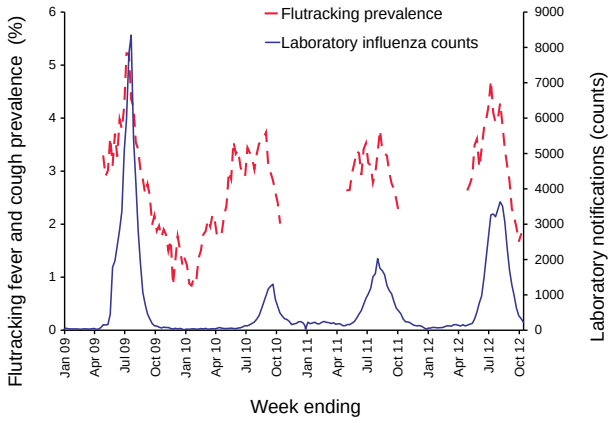


Comparison with national laboratory influenza notifications

There was an increase in the number of laboratory confirmed cases of influenza between 2010 and

2012 (from 1,301 in the peak week of 2010 to 2,026 in the peak week of 2011 to 3,631 in the peak week of 2012). The peak weekly ILI prevalence for 2012 Flutracking data unstratified by vaccination status was also higher (4.7%) than the peak weekly prevalence for 2011 (3.8%) and 2010 (3.7%). However, the increase in Flutracking ILI prevalence was not as large between 2010 and 2011 as it was for laboratory confirmed cases of influenza (Figure 5).

Figure 5: Flutracking weekly national fever and cough prevalence, April through October 2009 to 2012, compared with national influenza laboratory notifications



Discussion

With each additional year of Flutracking surveillance, the number of participants has steadily grown. Media releases are useful and high profile media programs can result in significant recruitment as demonstrated by the increase in participant numbers seen in 2011, but an invitation email to a potential participant has the advantage of being immediately clickable to initiate the enrolment process. While media coverage on television and radio does provide enhanced awareness of Flutracking, unless a potential participant is actually at a computer during the program they cannot immediately enrol and must remember the Internet address for later enrolment. Newspapers that include online coverage with hyperlinks to the Flutracking web site (Flutracking.net) may be more effective.

Flutracking participants have high levels of educational attainment with 60.4% of Flutracking participants aged 15 years or over holding a bachelor's degree or higher post school qualification compared with 25.4% of Australians aged 15–64 years.[9]

In 2011 and 2012, Flutracking was the only surveillance system providing weekly updates of vaccination uptake. Although the Flutracking sample may not be representative of the Australian population, results from this system are still useful for monitoring long term trends or detecting changes in vaccine uptake. The decreased influenza vaccination coverage of participants under 10 years of age after 2010 may be due to decreased concern about influenza after a relatively mild influenza pandemic or increased concern about adverse reactions to the vaccine following media coverage of adverse events in 2010 associated with the CSL Biotherapies Fluvax and Fluvax Junior vaccines.[10]

Sore throat was asked about amongst those who reported cough and fever. The addition of sore throat as a separate question in the survey is being further evaluated but our preliminary analysis is that it is not justified.

The FVE for influenza vaccine preventing ILI calculated amongst Flutracking participants is much lower than the pooled influenza vaccine efficacy estimate of 59% in those aged 18–65 years, from a recent meta-analysis.[11] The FVE calculated for 2011 and 2012 nevertheless demonstrated a greater protective effect than during the 2009 influenza pandemic year and the 2012 estimate was higher than the estimates for 2011 and 2010. A symptom based case definition cannot be expected to provide the same quantitative estimates of FVE provided by a laboratory confirmed outcome as the cases are a mixture of both influenza and other pathogens.[12] A sister system in the United Kingdom, however, found that vaccination with the 2010 seasonal influenza vaccine was significantly protective against ILI during the 2010–2011 influenza season with a vaccine effectiveness of 52% (95% CI 27–68).[13] Their markedly higher estimate may be due to the greater number of symptom questions and recording of temperature in the European Influenzanet system, which likely allows a more specific case definition for influenza. It will be interesting to follow their assessment of vaccine effectiveness in subsequent years. The main benefit of Flutracking's FVE calculations are that they can provide a rapid qualitative indication of FVE as was provided during the pandemic. While increasing the number of symptoms reported through our survey could optimise the case definition for calculating FVE, it may negatively impact on participation numbers and frequency of response.

Based on Flutracking data, the community attack rates in the 2012 season were higher than the attack rates in the 2011 and 2010 seasons, which were lower than most other Flutracking surveillance

years. National influenza laboratory notifications showed an increase in cases of influenza from 2010 to 2011. However, consistent with Flutracking data, the number of cases was still much lower than 2009. This suggests the 2010 and 2011 influenza seasons have been mild—perhaps due to higher rates of influenza vaccination in the community since 2009 and widespread exposure and subsequent immunity to the influenza A(H1N1) pdm09 virus. The 2012 influenza season was a more moderate influenza season, perhaps due to a returning dominance of the H3N2 influenza strain and influenza B.[1]

Flutracking and other syndromic surveillance systems can provide situational awareness to assist with the interpretation of the more specific influenza surveillance provided by laboratories; the latter being subject to changes in testing practices by clinicians and laboratories. Flutracking's surveillance method was unaffected by the changes in testing practices during the 2009 influenza pandemic and was able to demonstrate that the pandemic was not as severe as first anticipated.[5]

Online surveillance of influenza is a relatively young methodology. At the second International Workshop on Participatory Surveillance held in Amsterdam in April 2013, Influenzanet Europe, Flutracking Australia, and FluNearYou in the United States of America signed an agreement to share data on a standard platform internationally and to standardise methods and case definitions where possible to compare data internationally.[14,15]

Competing Interests

All authors declare that they have no competing interests.

Authors' contributions

Sandra Carlson led the writing of the manuscript and the statistical analysis, Craig Dalton conceived and designed the Flutracking program, oversaw the statistical analysis, and contributed to writing of the manuscript, Michelle Butler contributed to the statistical analysis, John Fejsa, contributed to the design of the project and had primary responsibility for the online software and database development, as well as questionnaire design, Elissa Elvidge contributed to the daily operational running of the system, David Durrheim contributed to the design of the project, statistical analysis, and writing of the manuscript.

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Author details

Ms Sandra J Carlson,[1] Research and Evaluation Officer
Dr Craig B Dalton,[1,2] Public Health Physician
Ms Michelle T Butler,[1] Research and Evaluation Officer
Mr John Fejsa,[1] Applications Developer
Ms Elissa Elvidge,[1] Project Officer
Dr David N Durrheim,[1,2] Public Health Physician, Conjoint Professor of Public Health Medicine.

1. Hunter New England Population Health, Wallsend, New South Wales
2. University of Newcastle, Callaghan, NSW and Hunter Medical Research Institute, Newcastle, New South Wales

Corresponding author: Dr Craig B Dalton, Public Health Physician, Hunter New England Population Health, Locked Bag 10, WALLSEND NSW 2287. Facsimile: +61 2 4924 6490. Email: craig.dalton@hnehealth.nsw.gov.au

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Original article

Epidemiology of sexually transmissible infections in New South Wales: are case notifications enough?

Teresa M Wozniak, Helen A Moore, C Raina MacIntyre

Abstract

Background: Surveillance of sexually transmissible infections (STI)s is important to assess the disease burden in the population and to monitor and evaluate changes in trends over time. Routinely collected surveillance data in New South Wales are reliant on case reporting, which for many infections is an inadequate mechanism for capturing incidence and prevalence. Increasing rates of chlamydia over the past decade have sparked intense debate as to whether the current notification system is optimal and whether the true burden of infection are being measured. This study describes the current surveillance for STIs in New South Wales.

Methods: New South Wales-specific data for the years 2000–2009 were analysed. Notification data were used to examine the rate of the 4 STIs that are notifiable in New South Wales; chlamydia, gonorrhoea, infectious syphilis and HIV notifications. Hospital admissions and chlamydia-associated pelvic inflammatory disease were analysed using admitted patient data.

Results: Chlamydia was the most frequently reported of the notifiable STIs in New South Wales. Despite the higher rates of notification compared with other STIs, chlamydia-related hospitalisations contribute less than a 5th of all STI-related hospital admissions. Infectious syphilis contributed to the highest proportion of all STI-related hospitalisations in New South Wales and rates increased from 2000 to 2009. For other STIs such as anogenital herpes and gonorrhoea, hospital admissions remained stable for the same period.

Conclusions: Notifications data for STIs should be complemented with hospital admission and other data sources to better describe STI morbidity. A synthesis of these data sources is needed to improve current surveillance and allow for better comparisons and trend analysis of STIs in New South Wales. *Commun Dis Intell* 2013;37(4):E407–E414.

Keywords: sexually transmissible infections, surveillance, hospitalisation, notifications, New South Wales

Introduction

Sexually transmissible infections (STI)s can be transferred from one person to another through sexual contact. STIs are a significant cause of preventable morbidity worldwide, mainly attributed to syphilis, gonorrhoea and chlamydia. Despite the greatest burden of infection being in developing countries,[1] either social, demographic or migratory trends have resulted in infection spreading to other countries. In Australia over the past decade, chlamydia was the most frequently notified STI followed by gonorrhoea, HIV and syphilis. Non-notifiable conditions such as genital warts have declined in Australia.[2]

Currently in Australia, surveillance for STIs is a combination of routine case notification, surveillance for antimicrobial resistant isolates, enhanced surveillance systems and a collection of special interest studies. Notifications capture incident and / or prevalent cases of chlamydia, gonorrhoea, syphilis and HIV in the population and are reported through the National Notifiable Diseases Surveillance System. Enhanced surveillance systems include the continuous randomised study of national general practice termed 'Bettering the Evaluation And Care of Health' (BEACH), the Australian Collaboration for Chlamydia Enhanced Sentinel Surveillance (ACCESS) and the Australian Gonococcal Surveillance Programme These systems serve to enrich notifications data by collecting additional information such as general practitioner (GP) encounters and frequency of testing for STIs. Special-interest studies serve to monitor a representative sample of the population, and in Australia, a number of such studies exist.[3,4] Smaller studies may focus on gaining an understanding of gay health issues, and these include the Gay Community Periodic Survey[5] and the Health in Men cohort study of HIV antibody negative men who have sex with men (MSM), in Sydney.[6] Hospital admissions may inform disease trends and measure the additional health care costs associated with severe disease, but limited published data are available in Australia.[7–9]

The aim of this study was to examine the current surveillance systems for STIs and to describe the epidemiology of STIs in New South Wales using notifications and hospital admissions data.

Methods

In New South Wales, notifiable diseases are reported to the NSW Notifiable Conditions Information Management System (NCIMS). Under the authority of the *NSW Public Health Act 2010,* the NSW Ministry of Health receives notifications of communicable disease through public health units from GPs, hospitals, and pathology laboratories.

The following notifiable conditions were analysed from NCIMS for the years 2000 to 2009:

Communicable disease group	Condition
Chlamydia trachomatis	*Chlamydia trachomatis* (non-lymphogranuloma venereum), congenital *Chlamydia trachomatis*
Gonorrhoea	Gonorrhoea; including site of infection: of the eye, pharynx, genitourinary system, anus/rectum and other or unspecified sites
Syphilis	Infectious syphilis (less than 2 years duration)
HIV	HIV

New South Wales Admitted Patient Data Collection (APDC) was utilised to extract records for hospital separations for the following ICD10-AM codes:

Early congenital syphilis, symptomatic	A50.0
Early congenital syphilis, latent	A50.1
Early congenital syphilis, unspecified	A50.2
Late congenital syphilitic oculopathy	A50.3–A50.7
Congenital syphilis, unspecified	A50.9
Primary genital syphilis	A51.0
Primary anal syphilis	A51.1
Primary syphilis of other sites	A51.2
Secondary syphilis of skin and mucous membranes	A51.3
Other secondary syphilis	A51.4
Early syphilis, latent	A51.5
Infectious syphilis	A51.9
Cardiovascular syphilis (I98.0*)	A52.0
Symptomatic neurosyphilis	A52.1
Asymptomatic neurosyphilis	A52.2
Neurosyphilis, unspecified	A52.3
Other symptomatic late syphilis	A52.7
Late syphilis, latent	A52.8
Late syphilis, unspecified	A52.9
Latent syphilis, unspecified as early or late	A53.0
Syphilis, unspecified	A53.9
Gonococcal infection of lower genitourinary tract without periurethral or accessory gland abscess	A54.0
Gonococcal infection of lower genitourinary tract with periurethral and accessory gland abscess	A54.1
Gonococcal pelviperitonitis and other gonococcal genitourinary infections	A54.2
Gonococcal infection of eye	A54.3
Gonococcal infection of musculoskeletal system	A54.4
Gonococcal pharyngitis	A54.5
Gonococcal infection of anus and rectum	A54.6
Other gonococcal infections	A54.8
Gonococcal infection, unspecified	A54.9
Chlamydial lymphogranuloma (venereum)	A55
Chlamydial infection of lower genitourinary tract	A56.0
Chlamydial infection of pelviperitoneum and other genitourinary organs	A56.1
Chlamydial infection of genitourinary tract, unspecified	A56.2
Chlamydial infection of pharynx	A56.4
Sexually transmitted chlamydial infection of other sites	A56.8
Chancroid	A57
Granuloma inguinale	A58
Urogenital trichomoniasis	A59.0
Herpesviral infection of genitalia and urogenital tract	A60.0
Herpesviral infection of perianal skin and rectum	A60.1
Anogenital herpesviral infection, unspecified	A60.9
Anogenital (venereal) warts	A63.0
Other specified predominantly sexually transmitted diseases	A63.8
Unspecified sexually transmitted disease	A64

The APDC was used to extract records for hospital separations for pelvic inflammatory disease (PID) ICD10-AM codes: N70.0, N70.1, N70.9, N71.0, N71.1, N71.9, N73.0–N73.2, N73.8, N73.9, N74.4.

Rates of hospital admission measures separations, not individuals, and one person may have multiple separations within a reporting period. The principal cause of the hospitalisation was defined as the diagnosis that caused the patient's episode of care in hospital. A hospitalisation rate is an estimate of the proportion of a population that was hospitalised during a specified period and is the number of hospitalisations per 100,000 population per year.

Notifications are a measure of incident cases: that is, the number of new cases in a specified period. A

notification rate is an estimate of the proportion of the population who have been diagnosed with the condition per 100,000 population per year.

All rates were age-standardised to adjust for differences in the age structure between populations using the direct method to compare between study groups. The Australian Bureau of Statistics estimated residential population as at 30 June 2001 was used as the standard population. All analyses were conducted using SAS software (SAS Institute, Inc., Cary, North Carolina).

The study was conducted by the NSW Ministry of Health and used de-identified aggregated data for which ethics committee approval was not required.

Results

Notifications in New South Wales

In New South Wales in 2009, chlamydia was the most commonly notified STI, with rates notably higher when compared with infectious syphilis, HIV or gonorrhoea (Figure 1). The largest increases in rates from 2000 to 2009 were for chlamydia (53.7–215.4 per 100,000) and infectious syphilis (1.2–7.4 per 100,000) whilst the rate of gonorrhoea and HIV notification remained stable over this period. In the study period, gonorrhoea and chlamydia notifications were most common amongst persons aged 15–34 years (Table 1). Rates of chlamydia notification among women in this age group were double that of males (Table 1).

Notification rates for infectious syphilis (range 7.8–26.8 per 100,000), HIV (range 13.2–28.1 per 100,000) and gonorrhoea (range 82.5–102.6 per 100,000) were 5 to 10 times higher amongst males aged 20–39 years compared with females. More specifically, between 2000 and 2009 there was

Figure 1: Age-standardised rate for notifiable sexually transmissible infections, New South Wales, 2000 to 2009, by year

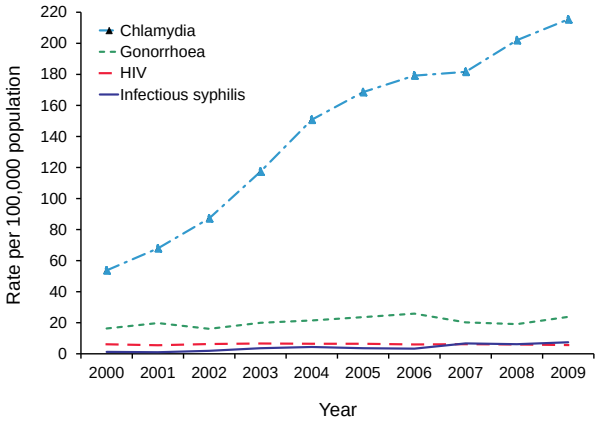
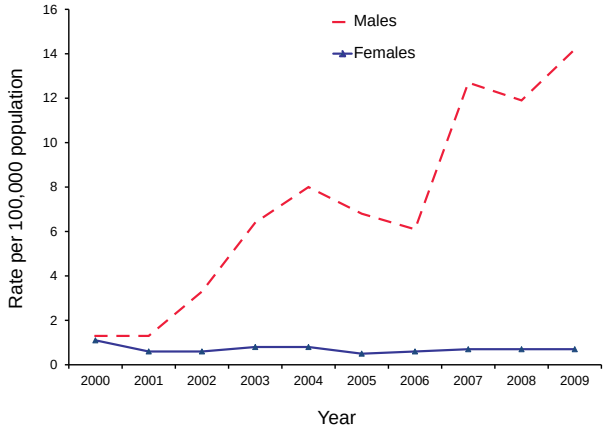


Table 1: Age-specific rate for notifiable sexually transmissible infections, New South Wales, 2000 to 2009, by age and sex

	Chlamydia		Gonorrhoea		HIV		Infectious syphilis	
Age group	Females	Males	Females	Males	Females	Males	Females	Males
0–4	9.4	8.5	0.3	0.1	0.3	0.1	0.0	0.0
5–9	0.3	0.0	0.2	0.0	0.1	0.1	0.0	0.0
10–14	28.0	2.4	1.4	0.0	0.2	0.0	0.0	0.0
15–19	880.6	237.6	18.1	27.6	0.7	0.9	0.6	1.4
20–24	1,093.8	661.6	22.8	92.7	3.6	13.2	1.6	7.8
25–29	489.7	471.6	15.5	102.6	3.8	23.5	2.0	15.7
30–34	213.2	273.4	10.3	90.4	3.5	25.6	1.7	20.0
35–39	103.8	177.1	6.7	82.5	1.8	28.1	0.6	26.8
40–44	54.1	119.1	4.4	56.1	1.8	22.5	1.0	22.5
45–49	28.5	73.6	3.1	33.3	1.0	14.8	0.7	13.9
50–54	17.3	48.3	1.6	22.6	1.1	8.8	0.4	9.0
55–59	6.4	26.9	1.3	12.1	0.6	5.5	0.4	4.7
60–64	3.0	15.5	1.0	8.1	0.5	4.1	0.1	3.6
65–69	1.9	8.2	0.3	4.2	0.5	1.1	0.2	1.9
70–74	1.2	4.4	0.4	1.3	0.4	0.6	0.4	1.0
75–79	0.6	2.3	0.2	1.2	0	0.9	0.0	1.6
80–84	0.2	1.0	0.2	0.0	0.2	0.7	0.2	0.4
85+	1.1	2.2	0.3	0.0	0.0	0.0	0.0	0.0

an 11-fold increase in infectious syphilis notifications amongst males. The rate increased from 1.3 per 100,000 in 2000 to 14.2 per 100,000 in 2009 (Figure 2).

Figure 2: Age-standardised rates of infectious syphilis, New South Wales, 2000 to 2009, by sex



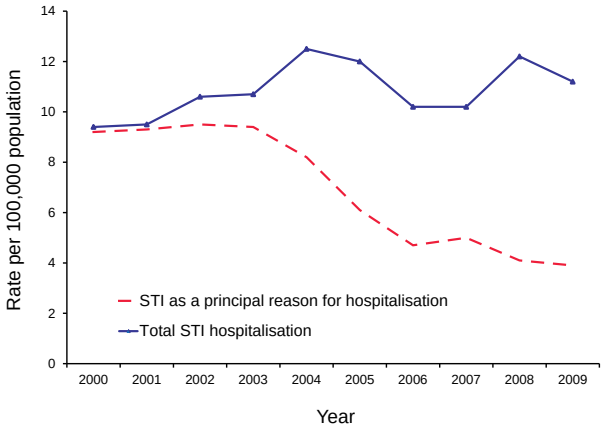
Gonorrhoea most commonly caused infection of the genitourinary system in all ages except in the 0–14 years age range (n=6) where infection of the eye or 'other' were more common (Table 2). Anogenital (genitourinary combined with infection of the anus or rectum) infection accounted for approximately 72% (n=10,239) of the gonococcal notifications of people aged 15 years or over. Amongst those younger than 15 years, there were 16 cases of anogenital gonorrhoea. Pharyngeal infection due to gonorrhoea was highest in the 30–34 years age group where it contributed to 11% (n=285) of total gonorrhoea infections (Table 2).

Hospital admissions in New South Wales

The rate of hospital separations in which an STI was recorded as the principal diagnosis or comorbid condition can provide a measure of the burden of more serious STI complications. In New South Wales, the total rate of STI admissions has gradually increased from a rate of 9.4 per 100,000 in 2000 to 11.2 per 100,000 in 2009 (Figure 3). Females were more likely to be hospitalised for an STI than males at a ratio of 1.3:1 (data not shown). Over the same period, there was a greater than 2-fold decrease in hospital admissions with an STI as the principal cause of the hospitalisation (Figure 3).

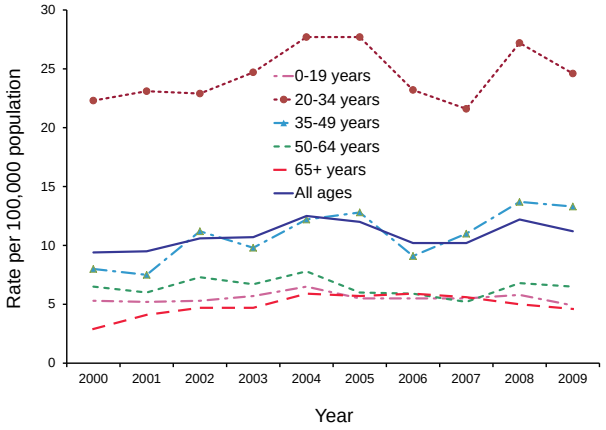
Syphilis, anogenital herpes and chlamydia represented the greatest proportion of total STI-related hospital admissions (Table 3). An analysis of changes over time shows that hospitalisations for chlamydia and syphilis increased between 2000 and 2009, while hospitalisations for conditions related to anogenital herpes and gonorrhoea remained stable.

Figure 3: Age-standardised rate of sexually transmissible infection hospitalisations, New South Wales 2000 to 2009, by year



Young adults experienced the highest rate of STI-related hospital admissions, with the number of STI-related admissions amongst those aged 20–34 years being approximately double that for those aged 35–49 years and approximately 5 times that of people in the other age groups (0–19, 50–64, and 65+) (Figure 4).

Figure 4: Rate for sexually transmissible infection hospitalisation,* New South Wales, 2000 to 2009, by age group and year



* Primary and secondary diagnoses

Table 2: Site of gonorrhoea infection, New South Wales, 2000 to 2009, by age group

	<15		15–19		20–24		25–29		30–34		35–39		40–44		45–49		50–54		55–59		>60	
Site of infection	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Eye	6	15	5	1	5	0	2	0	3	0	2	0	3	1	1	0	0	0	0	0	0	0
Genitourinary	16	39	584	59	1,472	59	1,609	58	1,399	56	1,275	58	830	58	521	65	314	63	166	69	147	65
Anus and rectum	0	0	50	5	317	13	379	14	392	16	360	16	235	16	101	13	55	11	16	7	1	7
Pharynx	0	0	42	4	198	8	293	10	285	11	213	10	117	8	62	8	24	5	11	5	11	5
Other	19	46	302	31	509	20	495	18	433	17	356	16	246	17	118	14	103	21	48	19	52	23

Total number of notifications for site of infections in the eye n=27; in genitourinary site n=8,334; anus/ rectum site n=1,922; pharynx site n=1,256; and other n=2,681. Total number of notifications n=14,220.

Table 3: Sexually transmissible infection-related hospitalisations * where a sexually transmissible infection was the primary cause of hospitalisation, New South Wales, 2000 to 2009

	2000		2001		2002		2003		2004		2005		2006		2007		2008		2009	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Syphilis	48	35	42	29	42	29	41	30	60	34	46	28	36	28	46	29	57	36	66	40
Gonorrhoea	12	9	19	13	31	22	18	13	14	18	17	10	16	12	19	12	14	9	21	13
Chlamydia	15	11	26	18	25	17	21	16	37	21	46	28	32	25	36	23	37	23	31	19
Anogenital herpes	48	35	44	30	36	25	33	24	45	26	45	27	40	31	51	32	46	29	45	28
Other†	15	11	15	10	9	6	22	16	20	11	11	7	6	5	8	8	5	3	0	0

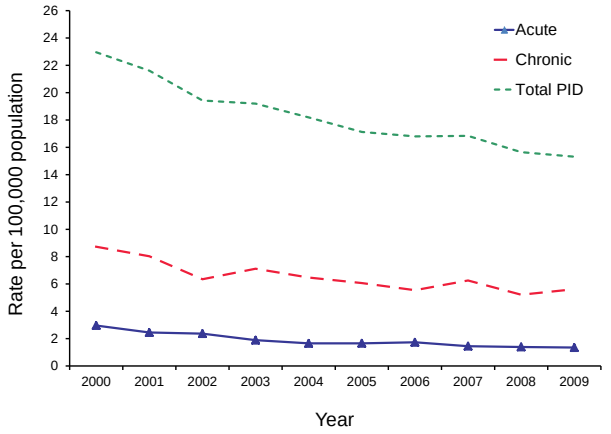
The total hospitalisations where a sexually transmissible infection was the primary diagnosis for the period 2000 to 2009 was 1,515.

* does not include co-morbid conditions (n=2,359) and conditions coded as 'other predominantly sexually transmissible infections not elsewhere specified' (n=3,092).

† 'Other' refers to hospitalisations for chancroid, granuloma inguinale and urogenital trichomoniasis.

Rates of total PID due to chlamydia infection declined over the past decade from 22 per 100,000 population in 2000 to 15 per 100,000 population in 2009 (Figure 5). Of these, the largest decline since 2000 was for chronic PID hospitalisation, whereas acute PID hospitalisations were stable.

Figure 5: Age-standardised rate of chlamydia-associated pelvic inflammatory disease diagnosis,* New South Wales, 2000 to 2009



* Coded as a principal diagnosis. Pelvic inflammatory disease of unspecified duration, n=6663.

Discussion

STIs remain a significant public health burden in New South Wales. Much of what informs STI disease trends in New South Wales is generated from the case notification system. Chlamydia is the most highly reported notifiable STI in New South Wales, followed by gonorrhoea, with infectious syphilis and HIV remaining stable and low for the period 2003–2009. However, these notification data do not establish the detail essential to understanding the true burden of disease, such as the rates of undiagnosed infections and the severity of disease. Much of the current focus has been on the increase in chlamydia notification rates in young people and in particular amongst young females. However, there needs to be a more wide reaching discussion as to how suitable the current notification based surveillance system is at measuring rates of STIs in New South Wales, in the absence of an evaluation of the NSW Notifiable Conditions Information Management System. Clearly, notifications are important for ascertaining the overall number of identified cases, but as is the case for chlamydia, infectious syphilis and gonorrhoea notifications, they do not provide a measure of incidence and overall burden of disease. Other data sources are required to complement notifications data and to more appropriately reflect changes in rates of infection and associated disease complications. These include data generated by surveys,[3] sentinel surveillance systems,[11] testing data[10,12] and inpatient hospital statistics. Currently, hospitalisation data are under utilised but can provide valuable information on the more severe consequences of undiagnosed or untreated infection.

Chlamydia notification rates in New South Wales increased rapidly between 2000 and 2009, with the highest proportion increase amongst young women aged 15–24 years. These apparent trends in increased notification rates may be a result of increasing transmission rates or an increase in targeted testing activity,[10,11,12] which is detecting and treating previously asymptomatic persons, or a combination of both. Currently, there is no systematic reporting of chlamydia testing patterns. However, studies have shown that the testing rates for chlamydia and other STIs have increased 10-fold since 1998 and testing is undertaken most frequently amongst the 15–24 years age group.[10] Similarly, increased rates of chlamydia testing were also evident in rural New South Wales, though the proportion of people tested in this region remained low (below 2% per GP visit).[12] Measures of chlamydia testing rates in priority populations are needed, more specifically, the number of young people tested for chlamydia in the past 12 months. These data could be collected through Medicare reports or alternatively by establishing an enhanced surveillance system for the regular collection and reporting of these data. The Australian Government Department of Health is in negotiations with Medicare for the routine release of testing data.[13]

While chlamydia notification rates were the highest of all STIs in New South Wales, chlamydia-associated PID rates have been declining, and chlamydia was not the leading primary diagnosis for STI-related hospitalisations. Similar discordances between chlamydia notifications and PID rates have been previously reported.[9,14] The discordance may reflect early detection and treatment of chlamydia, preventing the subsequent complications. Although difficulties with the diagnosis of PID may lead to differences in the rate of diagnosis between clinicians,[15,16] few studies have examined these differences. Without a co-ordinated approach and a clear understanding of the complexity of chlamydia and its associated disease development, notifications alone cannot provide evidence for appropriate public health actions.

Over the past decade there has been an 11-fold increase in infectious syphilis notification rates amongst males and this may in part explain the large proportion of STI-related hospitalisations that are due to syphilis. In New South Wales, infectious

syphilis occurs predominantly amongst MSM, approximately half of whom are HIV co-infected.[17] The increase in the number of male cases of syphilis may be a consequence of factors other than an increase in prevalence, such as increasing testing,[18] which disproportionately affects some populations. Notifications data and published studies support a predominance of syphilis transmission amongst males and amongst those co-infected with HIV. However, more detailed socio-demographic and behavioural predictors are needed to draw appropriate conclusions about disease trends. In the future, information pertaining to 'gender of partner' and or 'HIV status' would be necessary to complement notifications in understanding the priority populations most affected by infection.[13] Furthermore, hospitalisation data would improve the understanding of the impact of infection in the more severe cases of untreated infectious syphilis.

Despite an increase in antimicrobial resistance amongst gonococci isolates in New South Wales, which might be expected to lead to a higher number of infections,[19] gonorrhoea notifications remained stable from 2003 until 2009. The anogenital region accounted for the majority of gonorrhoea notifications, followed by infection of the pharynx. Pharyngeal gonorrhoea is most common amongst MSM,[20] and can be transmitted to other sites.[21] Currently, there are limited data on the transmissibility of gonorrhoea, particularly by anatomical sites. Reporting the site of gonorrhoea infection is important for determining appropriate diagnosis and treatment and in some populations. It may also reflect changes in sexual behaviour.

With changes in STI epidemiology, it is important to examine whether notifications alone are reliable enough to monitor trends in a population. In many instances, case notifications measure the rate of infection in those individuals who present with symptoms, those who have been tested opportunistically or have requested to be tested. It does not however, provide a measure of population-level rates of STIs and can only approximate trends over time as it is highly dependent on who presents with symptoms and who is tested. To better understand the timing and duration of infection, data relating to testing and positivity rates are needed. As GPs are the first point of contact with approximately 80% of the Australian population,[22] surveillance data from GPs could estimate population-level testing patterns. Information relating to GP practices can be found in the BEACH program or by requesting data from Medicare. The BEACH data show that the rate of GP management for all STI-related problems increased by two-thirds between 1998 and 2007.[10] Other than GPs, STIs can also be investigated in sexual health clinics, and data from this setting suggests chlamydia rates to be at least a third of that currently notified, when adjusted for testing patterns.[11] No similar studies have been conducted for other STIs but comparable trends may be evident when testing data becomes more readily available.

There are several limitations to the data presented. Persons with asymptomatic disease or with mild symptoms of the infection are less likely to seek health care than those with symptoms. Site of infection for gonorrhoea notifications does not take into account multiple sites infected but rather the site where the organism was first isolated. Persons may have several hospital admissions, which may overestimate rates of hospitalisation. This may occur for infections such as chlamydia in particular where repeat infections are common, or syphilis, where positive serology may have been from previously treated individuals.

Despite the substantial heterogeneity in diagnosis, treatment and surveillance systems available data suggest that STIs are on the rise. Changes in the epidemiology of STIs presents challenges to surveillance both in understanding the true burden of disease and for service provision. Understanding the true burden of disease needs to comprise of morbidity measures to effectively monitor the spread of disease and establish true patterns of progression. Morbidity is currently routinely reported using case notification, whilst other data measuring rates of STI-related hospitalisations are under utilised. Both data sources are important and should not be used in isolation but rather combined, and complemented with data on testing patterns, diagnosis, treatment, and aspects of sexual behaviour. A synthesis of these systems is needed to improve current surveillance which in turn would allow for better comparisons and trend analysis of STIs in New South Wales.

Author details

Dr Teresa M Wozniak[1,2]
Mrs Helen A Moore[3]
Professor C Raina MacIntyre[2]

1. New South Wales Public Health Officer Training Program, NSW Ministry of Health, Sydney, New South Wales
2. School of Public Health and Community Medicine, University of New South Wales, Sydney, New South Wales
3. Centre for Epidemiology and Evidence, NSW Ministry of Health, Sydney, New South Wales

Corresponding author: Dr Teresa Wozniak. Email: twozn@doh.health.nsw.gov.au

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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 2013.

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 (DoH), the Public Health Laboratory Network (PHLN), OzFoodNet, the Kirby Institute, the National Centre for Immunisation Research and Surveillance (NCIRS) and other communicable disease experts. CDWG develops and revises surveillance case definitions for all diseases reported to the National Notifiable Diseases Surveillance System. Surveillance (NNDSS) 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 will be implemented on 1 January 2014 and supersede any previous versions.

Poliovirus (non-paralytic) infection

(Effective 1 January 2014)

Reporting

Isolation or detection of poliovirus from clinical specimens with laboratory definitive evidence should be notified.

This case definition should be used for asymptomatic patients or patients with illness not consistent with acute flaccid paralysis.

Laboratory definitive evidence

Wild poliovirus infection

Isolation of wild poliovirus (confirmed in the National Enterovirus Reference Laboratory) OR detection of wild poliovirus by nucleic acid testing (confirmed in the National Enterovirus Reference Laboratory).

Sabin-like poliovirus infection

Isolation of Sabin-like poliovirus (confirmed in the National Enterovirus Reference Laboratory) OR detection of Sabin-like poliovirus by nucleic acid testing (confirmed in the National Enterovirus Reference Laboratory) except where there has been vaccination with Sabin oral polio vaccine in the six weeks* prior to the date of specimen collection.

* Note: This period may be longer for immunocompromised individuals

Vaccine derived poliovirus (VDPV) infection

Isolation of poliovirus (confirmed in the National Enterovirus Reference Laboratory) OR detection of poliovirus by nucleic acid testing (confirmed in the National Enterovirus Reference Laboratory), characterised as a vaccine derived poliovirus according to the current definition of the World Health Organization (reported by the National Enterovirus Reference Laboratory).

Poliovirus (non paralytic) infection changes

Laboratory definitive evidence

Changed National Poliovirus Reference Laboratory to National Enterovirus Reference Laboratory.

Sabin-like poliovirus infection

Added 'except where there has been vaccination with Sabin oral polio vaccine in the six weeks* prior to the date of specimen collection.'

Added * Note: This period may be longer for immunocompromised individuals.

Poliomyelitis (paralytic infection)

Reporting

Both confirmed cases and probable cases should be notified.

Confirmed case

A confirmed case requires laboratory definitive evidence AND clinical evidence.

Laboratory definitive evidence

Wild poliovirus infection

Isolation of wild poliovirus (confirmed in the National Enterovirus Reference Laboratory) OR detection of wild poliovirus by nucleic acid testing (confirmed in the National Enterovirus Reference Laboratory).

Vaccine-associated paralytic poliomyelitis (VAPP)

Isolation of Sabin-like poliovirus (confirmed in the National Enterovirus Reference Laboratory) OR detection of Sabin-like poliovirus by nucleic acid testing (confirmed in the National Enterovirus Reference Laboratory).

Vaccine derived poliovirus (VDPV) infection

Isolation of poliovirus (confirmed in the National Enterovirus Reference Laboratory) OR detection of poliovirus by nucleic acid testing (confirmed in the National Enterovirus Reference Laboratory), characterised as a vaccine derived poliovirus according to the current definition of the World Health Organization (reported by the National Enterovirus Reference Laboratory).

Clinical evidence

Any child under 15 years of age with acute flaccid paralysis* (including Guillain-Barré syndrome) or any person of any age with paralytic illness if polio is suspected.

For a case to be classified as VAPP the determination must be made by the Polio Expert Panel.

Probable case

A probable case of poliomyelitis (paralytic infection) requires clinical evidence AND the case not discarded as non-polio paralytic illness by the Polio Expert Panel.

Clinical evidence

As with confirmed case.

Acute flaccid paralysis syndrome is characterised by rapid onset of weakness of an individual's extremities, often including weakness of the muscles of respiration and swallowing, progressing to maximum severity within 1–10 days. The term "flaccid" indicates the absence of spasticity or other signs of disordered central nervous system (CNS) motor tracts such as hyperflexia, clonus, or extensor plantar responses. (Excerpt from Acute onset flaccid paralysis; World Health Organization 1993; WHO/MNH/EPI/93.3. Geneva)

Poliovirus (non paralytic) infection changes	**Laboratory definitive evidence**
	Changed National Poliovirus Reference Laboratory to National Enterovirus Reference Laboratory and changed Polio Expert Committee to Polio Expert Panel.

Viral haemorrhagic fever

(Effective 1 January 2014)

(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 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 CDC, Atlanta or NIV, Johannesburg

OR

Detection of specific virus by nucleic acid testing, pending confirmation by 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 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 (VHF)-infected blood or tissues.

Viral haemorrhagic fever changes	**Laboratory definitive evidence**
	Added ‘or the Special Pathogens Laboratory, National Institute of Virology (NIV), Johannesburg’.
	Removed ‘viral haemorrhagic fever’ virus.
	Added ‘specific’ virus.
	Added ‘or’ antigen detection assay.
	Removed ‘or electron microscopy’

Quarterly report

OzFoodNet quarterly report, 1 October to 31 December 2012

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. In each Australian state and territory, OzFoodNet epidemiologists investigate outbreaks of enteric infection. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, which occurred in Australia between 1 October and 31 December 2012.

Data were received from OzFoodNet epidemiologists in all Australian states and territories. The data in this report are provisional and subject to change.

During the 4th quarter of 2012, OzFoodNet sites reported 647 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports may be delayed, meaning that these figures under-represent the true burden of enteric disease outbreaks. In total, these outbreaks affected 13,058 people, of whom 297 were hospitalised. There were 48 deaths reported during these outbreaks. The majority of outbreaks (86%, n=559) were due to person-to-person transmission (Table 1), with 60% (335/559) of these occurring in residential aged care facilities.

Table 1: Outbreaks and clusters of gastrointestinal illness reported by OzFoodNet, 1 October to 31 December 2012 by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total
Foodborne and suspected foodborne	37	6
Waterborne and suspected waterborne	1	<1
Person-to-person	559	86
Unknown (*Salmonella* cluster)	5	<1
Unknown (other pathogen cluster)	1	<1
Unknown	44	7
Total	647	100*

* Percentages do not add up due to rounding.

Foodborne and suspected foodborne disease outbreaks

There were 37 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission (Table 2). These outbreaks affected 815 people and resulted in 59 hospitalisations. There were 6 deaths and 1 miscarriage reported during these outbreaks. This compares with 31 outbreaks in the 3rd quarter of 2012[1] and a 5-year mean of 35 outbreaks for the 4th quarter between 2007 and 2011. A limitation of the outbreak data provided by OzFoodNet sites for this report was the potential for variation in the categorisation of the features of outbreaks depending on circumstances and investigator interpretation. Changes in the number of foodborne outbreaks should be interpreted with caution due to the small number each quarter.

Salmonella Typhimurium was identified as the aetiological agent in 10 (27%) foodborne or suspected foodborne outbreaks during this quarter. The aetiological agent in the remaining outbreaks included 8 (22%) norovirus, 2 (5%) *Clostridium perfringens*, 2 (5%) histamine fish poisoning (previously referred to as scombroid poisoning), and one each (3%) due to *S.* Singapore, *Listeria monocytogenes* and a suspected bacterial toxin. In 12 outbreaks (32%), the aetiological agent was unknown.

Twenty-one outbreaks (57% of foodborne or suspected foodborne outbreaks) reported in this quarter were associated with food prepared in restaurants (Table 3).

To investigate these outbreaks, sites conducted 4 cohort studies, 4 case control studies, 2 case-case analyses and collected descriptive case series data for 26 investigations, while for 2 outbreaks, no individual patient data were collected. The evidence

Table 2. Outbreaks of foodborne or suspected foodborne disease reported, 1 October to 31 December 2012, by OzFoodNet site* (n=37)

State	Month†	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
Multi-jurisdictional	October	Primary production	*Salmonella* Typhimurium PT 3	43	7	M	Raw almonds
Multi-jurisdictional	December	Commercially manufactured	*Listeria monocytogenes* PFGE type 119A:44A:1	34	34	A	Brie and/or camembert cheese
Multi-jurisdictional	December	Commercially manufactured	*S.* Typhimurium MLVA profile 03-16-09-12-523 & 03-17-09-12-523 (historically PT 135)	391	Unknown	A	Suspected fresh pre-cut chicken pieces
ACT	November	Takeaway	Suspected bacterial toxin	3	0	D	Sashimi
NSW	October	Restaurant	*Clostridium perfringens*	5	0	M	Chicken burrito
NSW	October	Community	Norovirus genotype II.4 New Orleans 2009	8	0	M	Raw oysters
NSW	October	Restaurant	Unknown	20	0	D	Unknown
NSW	November	Restaurant	*S.* Singapore	7	3	D	Unknown
NSW	November	Restaurant	*S.* Typhimurium MLVA profile 03-09-08-13-523 (historically PT 170)	3	1	D	Unknown
NSW	November	Restaurant	Unknown	9	0	D	Unknown
NSW	December	Restaurant	*S.* Typhimurium MLVA profile 03-09-08-13-523 (historically PT 170)	4	0	D	Unknown
NSW	December	Restaurant	Unknown	16	0	D	Unknown
NSW	December	Restaurant	Unknown	12	0	D	Unknown
NSW	December	Restaurant	Unknown	8	0	D	Unknown
NSW	December	Restaurant	Unknown	7	0	D	Unknown
NSW	December	Restaurant	*C. perfringens*	13	0	D	Roast beef
Qld	October	Restaurant	Unknown	12	0	D	Unknown
Qld	November	Restaurant	Norovirus genotype II.4 New Orleans 2009	4	0	D	Raw oysters
Qld	November	Private residence	Histamine fish poisoning	3	0	M	Mahi Mahi
Qld	December	Hospital	*S.* Typhimurium MLVA profile 03-09-07-12-524	6	3	D	Unknown
Qld	December	Restaurant	*S.* Typhimurium MLVA profile 03-09-07-15-524 (PT 170)	11	3	D	Sushi
Qld	December	Restaurant	Histamine fish poisoning	3	0	D	Mahi Mahi
SA	October	Aged care facility	Unknown	20	0	D	Unknown
Vic.	October	Restaurant	Norovirus	17	1	D	Salads prepared by an ill food handler
Vic.	October	Private residence	*S.* Typhimurium PT 12a	7	1	D	Noodles with chicken and egg
Vic.	November	Private residence	Norovirus	10	0	A	Cake

Table 2 *continued*. Outbreaks of foodborne or suspected foodborne disease reported, 1 October to 31 December 2012, by OzFoodNet site,* (n=37)

State	Month†	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
Vic.	November	Restaurant	Norovirus	13	0	D	Multiple foods contaminated by infectious food handler/s
Vic.	November	Private residence	*S.* Typhimurium PT 135a	5	1	D	Suspected chocolate mousse made with raw eggs
Vic.	December	Restaurant	Norovirus	32	1	D	Suspected salad
Vic.	December	Restaurant	Norovirus	35	0	D	Unknown
Vic.	December	Unknown	*S.* Typhimurium PT 170	3	1	D	Scrambled eggs or chicken teriyaki
Vic.	December	Private residence	*S.* Typhimurium PT 170	3	3	D	Raw egg drink
Vic.	December	Aged care facility	Unknown	10	0	D	Unknown
WA	October	Restaurant	Unknown	9	0	A	Pork belly meal
WA	November	Restaurant	Norovirus	13	0	A	Pickled octopus, prawns, asparagus
WA	December	Commercial caterer	Unknown	9	0	A	Unknown
WA	December	Bakery	Unknown	7	0	D	Assorted sandwiches/rolls
Total				815	59		

* No foodborne or suspected foodborne outbreaks were reported by the Northern Territory and Tasmania.

† Month of outbreak is the month of onset of first case or month of notification/investigation of the outbreak

A Analytical epidemiological association between illness and 1 or more foods

BT Binary type

D Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission

M Microbiological confirmation of agent in the suspected vehicle and cases

MLVA Multi-locus variable number tandem repeat analysis

PFGE Pulsed-field gel electrophoresis

PT Phage type

ST Serotype

Table 3: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet, 1 October to 31 December 2012 by food preparation setting

Food preparation setting	Outbreaks
Restaurant	21
Private residence	5
Aged care	2
Commercially manufactured	2
Bakery	1
Commercial caterer	1
Community	1
Hospital	1
Primary production	1
Takeaway	1
Unknown	1
Total	37

used to implicate food vehicles included analytical evidence in 6 outbreaks and microbiological evidence in 4 outbreaks. Descriptive evidence alone was obtained for 27 outbreak investigations.

The following jurisdictional summaries describe key outbreaks and public health actions that occurred during the quarter.

Australian Capital Territory

There was 1 reported outbreak of foodborne or suspected foodborne illness during the quarter. A bacterial toxin was the suspected cause of the outbreak, which was associated with the consumption of salmon and tuna sashimi.

New South Wales

There were 12 reported outbreaks of foodborne or suspected foodborne illness during the quarter. The aetiological agent was identified in six of these outbreaks: two each were due to *S.* Typhimurium and *C. perfringens*; and one each due to *S.* Singapore and norovirus.

Description of key outbreaks

Five people from a group of seven developed diarrhoea and abdominal cramps 12–15 hours after eating a meal at a Mexican restaurant. The 5 ill people had all consumed a chicken burrito, which included shredded chicken, cheese, rice and beans. Left-over and newly cooked food samples were taken from the restaurant and an inspection indicated poor temperature control may have been a contributing factor. Samples of the cooked chicken were positive for *C. perfringens*. New South Wales Food Authority (NSWFA) issued the restaurant with an improvement notice.

Six people from a group of 30 developed symptoms of diarrhoea, vomiting and stomach cramps after participating in a 5 day social event. A husband and wife who were not a part of the group of six were also ill. All 8 cases consumed oysters from the local area. One stool sample was collected, which was positive for norovirus genotype II.4 New Orleans 2009 variant.[2] An environmental investigation identified a damaged sewerage pipe that had been leaking into the waterway where the local oysters were harvested. Oyster samples from this waterway subsequently tested positive for norovirus genogroup II. The waterway was temporarily closed to oyster harvesting and the broken pipe was repaired.[3]

A complaint of illness was made to the NSWFA following a meal at a restaurant. Thirteen people from a group of 16 developed diarrhoea 12 hours after eating various dishes from a buffet. All interviewed cases reported eating roast beef. Stool samples were submitted for 3 cases and *C. perfringens* enterotoxin type A was identified in one sample. The NSWFA inspected the premises and issued improvement notices for storage and holding temperatures of food. The lack of temperature controls suggests the possibility of *C. perfringens* being able to proliferate to sufficient levels to cause illness.

Eight people from a group of 17 who had eaten at a restaurant reported symptoms of fever, nausea, vomiting, diarrhoea and abdominal cramps. Foods consumed by all 8 cases were tiramisu and panna cotta, though it was not known if the people who were not unwell also consumed these items. No clinical specimens were provided. While the symptoms and onsets could be representative of a salmonellosis outbreak, no positive samples were found from sampling of left-over foods to confirm this as the cause of illness. The cases did eat a raw egg tiramisu and whilst the use of raw eggs in the making of the tiramisu may have been a vehicle for *Salmonella*, in the absence of any further information, the cause of the illness for these cases could not be determined.

Northern Territory

There were no reported outbreaks of foodborne or suspected foodborne illness during the quarter.

Queensland

There were 6 reported outbreaks of foodborne or suspected foodborne illness during the quarter. The aetiological agent was identified in five of these

outbreaks: two each were due to *S.* Typhimurium and histamine fish poisoning; and one due to norovirus.

Description of key outbreaks

Four cases of suspected foodborne illness were reported following the consumption of a seafood buffet meal at a sports club. The cases became ill approximately 30 to 41 hours following the meal with symptoms including diarrhoea, vomiting and abdominal cramps. The consumption of raw oysters was common to all cases. Norovirus genotype II.4 2010 variant (also referred to as genotype II.4 New Orleans 2009 variant)[2] was detected in 2 faecal specimens, a strain that was genetically identical (by genome sequencing) to a human case from a New South Wales outbreak that was also associated with oyster consumption (reported above and in Table 2), suggesting that the source of the virus for these cases was likely to be the same.

Two outbreaks of histamine fish poisoning associated with the same fish species, Mahi Mahi, were investigated. The first outbreak involved 3 cases who consumed Mahi Mahi fish fillets purchased from a seafood vendor. The cases became ill within 1 hour of consuming the fish fillets with symptoms including vomiting, diarrhoea, fever, breathing difficulty, rash and headaches. High levels of histamine (1,600–2,050 mg per kg) were detected on samples of fillets collected from the retailer who sold the fish to the cases. The wholesaler and supplier of the Mahi Mahi were also investigated to ensure food handlers were aware of risks associated with poorly handled fish and inappropriate storage and transport of seafood. The second outbreak involved 3 cases who consumed Mahi Mahi fish at a restaurant. The cases became ill within 1 hour of consuming the fish. All 3 cases presented with symptoms including red rash and tachycardia. Environmental investigations of the restaurant and supplier of the Mahi Mahi were conducted and advice provided on the importance of correct food handling techniques for seafood. No leftover fish fillets were available for laboratory testing.

Twenty-six cases of *S.* Typhimurium multi-locus variable number tandem repeat analysis (MLVA) profile* 03-09-07-15-524 (phage type 170 [PT 170]) were investigated. Seventeen of the 26 cases were interviewed with 11 reporting the consumption of sushi meals from the same sushi restaurant prior to illness. Three of the cases were hospitalised. No specific sushi meal was common among the cases. The investigation identified numerous food hygiene and handling issues at the sushi restaurant including poor temperature control of ready to eat food and the potential for cross contamination. Extensive environmental sampling was conducted on-site including environmental swabs of food preparation surfaces and the collection of sushi, chicken, eggs, mayonnaise and cleaning cloths for microbiological analysis. The same strain of *Salmonella* as that found in cases was cultured from two cleaning cloths, however, all other environmental samples tested negative for bacterial pathogens. The investigation was still ongoing at the time of this report as further cases with this particular strain of *Salmonella* were found to be associated with this same sushi venue. No single vehicle or source of infection has been identified.

South Australia

There was 1 reported outbreak of foodborne or suspected foodborne illness during the quarter. No aetiological agent was identified.

Nineteen residents from an aged care facility reported diarrhoea. Twelve of the 19 cases were reportedly on a vitamised diet and two on a minced diet (these diets include the same foods). The illness was short-lived and no cases were hospitalised. Ten faecal samples were submitted, and no common viral or bacterial pathogens were detected. Due to the short duration of symptoms and only one person reporting vomiting, a bacterial toxin was suspected as the cause. Four faecal samples were submitted to an interstate laboratory for *C. perfringens* and *Bacillus cereus* testing. *C. perfringens* was detected in all faecal samples, but was below the diagnostic level for *C. perfringens* food poisoning (>10^6 cfu/g). No *B. cereus* was detected in any of the faecal samples. An environmental investigation identified that the dishwasher was not working prior to residents becoming unwell, and food reheating practices were of concern to investigators. *C. perfringens* and *Bacillus* species (not able to be further classified) were detected in food samples. Actions undertaken by the facility included fixing the dishwasher, and kitchen staff at the facility underwent food safety training.

Tasmania

There were no reported outbreaks of foodborne or suspected foodborne illness during the quarter.

Victoria

There were 10 reported outbreaks of foodborne or suspected foodborne illness during the quarter. The aetiological agent was identified for nine of these outbreaks with four due to *S.* Typhimurium and five due to norovirus.

* MLVA profiles are reported using the Australian coding convention agreed at a MLVA typing harmonisation meeting in Sydney in November 2011.[4]

An outbreak of salmonellosis affecting 3 of 5 teachers and 4 of 7 students who shared a meal at a secondary school was investigated. A meal consisting of spring rolls and a chicken and egg noodle dish was prepared at home by one of the students and brought to school to share with teachers and classmates. The noodle dish was suspected as the source as only the 3 cases consumed this food item. All 3 cases were confirmed with *S.* Typhimurium PT 12a.

Five family members were affected by an outbreak of *S.* Typhimurium PT 135a. The group had shared a meal of slow-cooked chicken curry and chocolate mousse made with raw eggs. Chocolate mousse was considered to be the most likely source based on information about how the food was prepared.

An outbreak of gastroenteritis affecting a group of people who had eaten dinner together at a restaurant was investigated. Twenty people from the group of 36 were interviewed and 10 reported an onset of vomiting and/or diarrhoea between 24 and 48 hours after eating. The group ate a variety of different meals. During the investigation it was revealed that one of the attendees had prepared a cake at home and brought this to share for dessert. This person, who subsequently became unwell approximately 24 hours after the dinner, had a child at home who had been ill with diarrhoea during the period when the cake was made. Analysis of this exposure (eating cake) for those interviewed suggested that the cake may have been the source (odds ratio [OR] 8.0; 95% confidence interval [CI] 0.8 to 235.5; $P=0.04$). Four cases were confirmed with norovirus including the person who made the cake.

An outbreak of salmonellosis affecting 3 children in a family was investigated. Two days prior to their illness onset, the children had shared scrambled eggs for breakfast. This meal was eaten at a hotel buffet. That same day the children also shared a chicken teriyaki and rice meal from a noodle bar. Neither of these meals was eaten by the children's parents who remained well. The children were all confirmed with *S.* Typhimurium PT 170. No other cases linked to either premise were identified.

Another outbreak of *S.* Typhimurium PT 170 affecting 3 family members was investigated. All 3 family members shared a raw egg milk drink the day prior to onset. The eggs were obtained from their own backyard chickens.

An outbreak of gastroenteritis affecting 2 separate groups of people who dined at a restaurant on the same evening was investigated. An analytical study was conducted for the larger group of 45 people (28 cases) who ate the set menu. A statistically significant association between illness and consumption of food from one of three platters was observed (OR 4.7; 95% CI 1.0 to 25.8; $P=0.026$). There were no common food items consumed by those who became unwell in the second smaller group of 20 attendees (7 cases). None of the 23 staff interviewed reported illness in the week prior to the groups dining at the restaurant. Seven cases (cases from both groups) were confirmed with norovirus. As two separate groups were affected, and the median incubation period for both groups was consistent with a point source exposure at the hotel, the source was suspected to be norovirus contaminated food. However, the investigation was unable to determine how the food became contaminated.

Thirty-two people reported vomiting and/or diarrhoea after attending a restaurant for a function. Over 70 people dined at the restaurant that night. Detailed questionnaires were not completed for the majority of attendees, but some data were collected for 44 attendees. Analysis of food exposures suggested that consumption of salad with the main meal was associated with illness (OR 5.6; 95% CI 1.2 to 27.4; $P=0.02$). Although this was one large work function, 3 separate groups reported illness and the median incubation period for 12 cases and their onset dates and times were indicative of a point source exposure at the restaurant suspected to be norovirus contaminated food. Seven cases were confirmed with norovirus.

Western Australia

There were 4 reported outbreaks of foodborne or suspected foodborne illness during the quarter. Norovirus was identified as the aetiological agent for one of these outbreaks.

Investigators were notified that people within a group became ill after attending a lunch at a restaurant. There were at least 24 people in the group and information on illness and food exposures was obtained from 21 people using a structured questionnaire. Of these, 13 were ill with diarrhoea (n=11), vomiting (n=8), and fever (n=9), with a median illness duration of 24 hours. One person submitted a faecal specimen that was positive for norovirus. None of the people reported diarrhoea and/or vomiting prior to, or during, the meal. The incubation period was 31 hours. A univariate analysis found an association between illness and eating either marinated octopus (risk ratio [RR] and CI not defined; $P<0.01$), prawns (RR 7.3; 95% CI 1.2–46.2; $P<0.01$) and asparagus (RR 2.4; 95% CI 1.1–5.5; $P=0.04$). The environmental investigation identified no immediate or critical risks to food safety at the time of assessment. The marinated octopus was prepared by a Western Australia company using local octopus. There were no reports of illness among restaurant staff

prior to the meal. Whilst the source of norovirus contamination was not identified, the outbreak was suspected to be foodborne, with a number of foods possibly contaminated.

At least 9 of the 110 people who attended a wedding became ill following the reception, with a median incubation period of 7 hours and a duration of one day. An analytical study found a statistical association between illness and eating the pork belly meal (OR: not defined; CI: not defined; $P=0.002$), and two of the individual components of the pork belly meal – pork belly (OR not defined; CI not defined; $P=0.013$) and polenta (OR 13.3; 95% CI 1.4 to 124; $P=0.017$). The shallot tart, which was also part of the pork belly meal, had an elevated OR of 5.8, but this was not statistically significant (95% CI 1.0 to 34.4, $P=0.057$). A logistic regression model was unable to identify a source of infection. While the environmental health investigation identified a potential food safety risk from an inadequate cooking-cooling process, there was insufficient evidence to determine an environmental cause of the outbreak. The symptoms experienced by cases, together with the incubation period and the duration of illness, were suggestive of toxin mediated bacterial food poisoning. However, without a positive faecal specimen or food samples, the aetiological agent could not be confirmed.

Multi-jurisdictional investigations

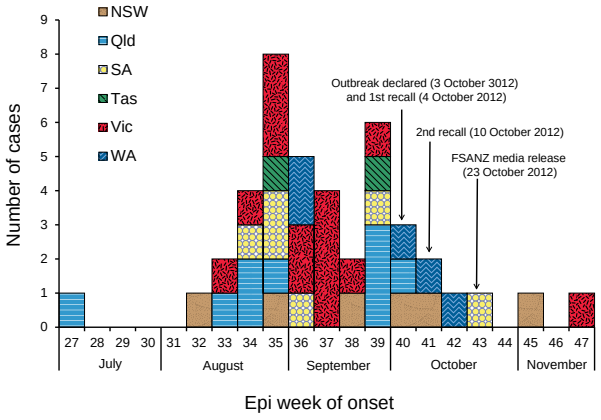
Salmonella Typhimurium PT 3 associated with the consumption of raw almonds

OzFoodNet commenced a multi-jurisdictional outbreak investigation on 3 October 2012 upon identifying *S.* Typhimurium PT 3 infection among cases from 3 jurisdictions. A confirmed outbreak case was defined as any person with a specimen collection date from 1 July 2012 with an isolate that had been confirmed as PT 3 and either MLVA profile 03-13-08-11-523/524 or 03-13-10-11/12-523/524, or pulsed-field gel electrophoresis (PFGE) type 0434. Of 39 interviewed cases, 37 (95%) reported consuming raw almonds in the week before onset of illness, either as a single food or part of a mixed nut product. Thirty-two of 37 (87%) cases who reported consuming almonds had purchased them from a retailer known to have been supplied with almonds from a single Victorian producer. The Victorian health department had received 3 separate notifications of *S.* Typhimurium PT 3 in raw almonds from this producer in July 2012. In total, 116 samples of implicated almond products were collected for microbiological testing including 13 samples from the homes of cases. Fourteen of 116 (12%) samples tested positive for *Salmonella* spp. of which 7 (6%) were confirmed with the outbreak strain *S.* Typhimurium PT 3.

An extensive environmental investigation was unable to determine the exact source of contamination of the almonds. However, it is known that the harvesting method for almonds exposes them to a risk of environmental contamination, especially from orchard soils that may be contaminated with bacteria including *Salmonella*. Almond trees are shaken during harvesting and the almonds lie on the ground for several days/weeks prior to being mechanically picked up for processing. It is known that the almond orchards in northern Victoria and southern New South Wales were subject to heavy rainfall during the 2012 harvest season. Under moist conditions there is potential for the growth of *Salmonella*.[5] Media releases and consumer level recalls of the implicated brands of almonds were conducted during the course of this investigation. As a result of this outbreak investigation all batches of raw almonds processed by this company now undergo pasteurisation prior to sale.

By the close of the investigation on 29 November 2012, 40 confirmed and 3 suspected cases were reported from 6 jurisdictions. For the 43 cases, the median age was 33 years (age range: 1 to 78 years), and there were approximately equal numbers of males and females. Seven cases were hospitalised with their illness, and there were no deaths. Onset of illness was from 2 July 2012 to 26 November 2012 (Figure).

Figure: Epidemic curve of Salmonella Typhimurium PT 3 infections, Australia, July to November 2012, by week of onset and jurisdiction



Listeria monocytogenes associated with the consumption of brie and/or camembert cheese

OzFoodNet, food safety officers and public health laboratories collaborated on a multi-jurisdictional outbreak investigation of listeriosis commencing on 10 December 2012. The outbreak involved 34 con-

firmed cases of infection with the same strain of *L. monocytogenes*: serotype 4b, 4d, 4e; binary type 254/255 and PFGE type 119A:44A:1.† Cases were reported from 6 jurisdictions, and had onset of illness between 18 August 2012 and 19 April 2013. There were 6 deaths and 1 miscarriage reported during the outbreak. Brie and camembert cheeses produced by a Victorian manufacturer were implicated, and 2 recalls of a range of soft cheese products from this manufacturer were conducted in December 2012 and January 2013. The outbreak strain of *L. monocytogenes* was detected in brie and camembert produced by the implicated manufacturer, and from product sampled from retailers in Victoria, New South Wales, Queensland and South Australia. Dairy Food Safety Victoria worked closely with the cheese manufacturer to improve processes, and more stringent routine test and hold protocols were implemented, as well as an extensive environmental testing protocol. The investigation closed on 22 August 2013.

Salmonella Typhimurium PT 135 associated with an unknown source

Two novel strains of *S.* Typhimurium PT 135 (MLVA profile 03-16-09-12-523 and 03-17-09-12-523) emerged in New South Wales and Queensland in significant numbers from July 2012. OzFoodNet commenced a multi-jurisdictional outbreak investigation on 10 December 2012 and declared the investigation closed on 10 January 2013. Nationally, 391 cases were identified between May and December 2012, with cases reported from 6 jurisdictions. The same *S.* Typhimurium MLVA profiles were identified in raw chicken meat sampled from the same chicken producer in both New South Wales and Queensland. A joint case control study in New South Wales and Queensland was conducted in December 2012 which included 22 cases and 45 controls; however the results of the study failed to identify any significant association with chicken consumption and illness.

Based on the laboratory evidence, food safety regulators and industry representatives implemented control measures at the farm and processing levels with the aim of reducing the prevalence of carcass contamination. Control measures included culling of chicken flocks from the implicated supplier, the inclusion of *S.* Typhimurium PT 135 organisms into vaccines for day old chickens and breeders, maintaining recommended chlorine levels in spin chillers at the processing plants, enhancing surveillance on-farm and diverting contaminated chickens to cooked product such as nuggets or schnitzels. Nationally, notifications peaked in October 2012 and began to decrease following the implementation of control measures.

Cluster investigations

During the quarter, OzFoodNet sites conducted investigations into a number of clusters of infection for which no common food vehicle or source of infection could be identified. Aetiological agents identified during the investigations included *S.* Typhimurium (PT 135, PT 8 and PFGE type 1), *S.* Chester and *Shigella sonnei* biotype a.

Comments

The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission, and in this quarter, 86% of outbreaks (n=559) were transmitted via this route. The number of foodborne outbreaks this quarter (n=37) was similar to the previous quarter (n=31) and was consistent with the 5-year mean (n=35, 2007–2011). Of the 21 foodborne outbreaks for which a source of the outbreak was identified, 7 (63.6%) were associated with the consumption of dishes containing seafood including oysters, Mahi Mahi, sashimi and sushi.

Salmonella species were identified as the aetiological agent in 11 (29.7%) of the 37 foodborne or suspected foodborne outbreaks during this quarter (Table 2), with 10 of 11 outbreaks being due to *S.* Typhimurium. Of the 11 outbreaks where *Salmonella* was implicated as the responsible agent, 45% (5/11) were associated with dishes containing eggs and/or chicken. To reduce the incidence of salmonellosis, Australian states and territories commenced implementation of the Primary Production and Processing Standard for Eggs and Egg Products[7] in November 2012, and continue to implement the Primary Production and Processing Standard for Poultry Meat[8] since May 2012.

OzFoodNet provided evidence to the Food Safety Information Council to inform the Council's annual Australian Food Safety Week, held in November. The theme of Food Safety Week 2012 was cross-contamination; with a specific focus on practices that increase the risk of cross-contamination from raw chicken.[9]

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† PFGE patterns were interpreted with reference to the guidelines proposed by Tenover et al,[6] the 3 restriction enzymes used for *L. monocytogenes* PFGE typing were: *Apa*1 : *Sma*1 : *Not*1 and a discrete PFGE type was assigned using a nomenclature defined by the Microbiological Diagnostic Unit Public Health Laboratory.

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OzFoodNet contributors to this report include *(in alphabetical order)*: Robert Bell (Qld), Barry Combs (WA), Anthony Draper (NT), Emily Fearnley (SA), Tove Fitzgerald (NSW), Gerard Fitzsimmons (Health), Neil Franklin (NSW), Robyn Gibbs (WA), Joy Gregory (Vic,), Michelle Green (Tas.), Karin Lalor (Vic.), Megge Miller (SA), Cameron Moffatt (ACT), Jennie Musto (NSW), Nevada Pingault (WA), Ben Polkinghorne (NSW) and Russell Stafford (Qld).

Correspondence

Ms Robyn Leader, OzFoodNet, Office of Health Protection, Australian Government Department of Health, GPO Box 9848, MDP 14, Canberra, ACT 2601. Telephone: +61 2 6289 2750. Email: ozfoodnet@health.gov.au

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National Notifiable Diseases Surveillance System, 1 July to 30 September 2013

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 64,122 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 July and 30 September 2013 (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 July to 30 September 2013, by date of diagnosis*

Disease	State or territory								Total this quarter 2013	Total last quarter 2012	Total this quarter 2012	Last 5 years mean this quarter	Ratio	Year to date 2013	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)†	2	5	1	8	1	1	6	10	34	44	55	60.0	0.6	124	173.0
Hepatitis B (unspecified)‡	33	707	65	243	87	12	448	292	1,887	1,773	1,755	1,760.0	1.1	5,287	5,103.4
Hepatitis C (newly acquired)†	1	16	0	NN	14	5	28	32	96	74	112	97.4	1.0	285	306.2
Hepatitis C (unspecified)‡	53	940	61	626	116	66	583	331	2,776	2,518	2,492	2,705.6	1.0	7,726	7,962.4
Hepatitis D	0	2	0	1	0	0	5	0	8	17	5	8.6	0.9	40	28.6
Gastrointestinal diseases															
Botulism	0	1	0	0	0	0	0	0	1	1	0	0.2	5.0	4	0.6
Campylobacteriosis	90	NN	44	860	506	124	1,391	509	3,524	3,221	3,681	3,871.4	0.9	10,207	12,001.2
Cryptosporidiosis	1	74	12	94	20	4	170	45	420	1,138	334	289.4	1.5	3,276	2,219.2
Haemolytic uraemic syndrome	0	1	0	1	0	0	0	0	2	2	3	3.2	0.6	8	12.4
Hepatitis A	3	12	0	13	0	0	14	3	45	42	39	55.2	0.8	156	198.0
Hepatitis E	0	0	0	0	0	0	1	1	2	6	4	7.4	0.3	23	31.4
Listeriosis	0	7	0	0	1	1	5	1	15	17	18	15.0	1.0	61	59.4
STEC, VTEC§	0	0	0	64	10	1	2	1	78	28	19	19.2	4.1	149	69.8
Salmonellosis	47	534	94	479	226	34	566	275	2,255	3,046	2,026	1,789.4	1.3	9,443	8,013.0
Shigellosis	1	40	22	15	9	0	27	15	129	116	105	130.2	1.0	387	462.6
Typhoid	1	6	0	2	1	0	2	4	16	29	17	20.4	0.8	112	87.6
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	1	1	1.2	0.0	1	3.8
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	1.0

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2013, by date of diagnosis*

Disease	State or territory								Total this quarter 2013	Total last quarter 2012	Total this quarter 2012	Last 5 years mean this quarter	Ratio	Year to date 2013	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection[‖,¶]	304	5,079	808	4,932	1,312	344	4,663	2,977	20,419	20,794	20,192	17,760.4	1.1	62,192	54,535.6
Donovanosis	0	0	0	0	0	0	0	0	0	0	1	0.4	0.0	0	1.0
Gonococcal infection[¶]	24	1,069	464	685	213	15	709	447	3,626	3,858	3,196	2,430.6	1.5	11,318	7,804.6
Syphilis – congenital	0	1	0	0	0	0	0	2	3	2	0	1.0	3.0	6	3.0
Syphilis < 2 years duration[¶]	3	144	10	80	10	3	192	18	460	408	404	330.2	1.4	1,316	1,016.4
Syphilis > 2 years or unspecified duration[‡,¶]	3	108	14	76	37	4	140	87	469	462	328	348.4	1.3	1,322	1,020.4
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	1	0.8
Haemophilus influenzae type b	0	3	0	2	0	0	3	0	8	5	5	4.2	1.9	16	15.4
Influenza (laboratory confirmed)	388	6,288	114	2,589	2,381	162	3,777	1,295	16,994	2,522	32,898	21,526.0	0.8	21,842	28,208.8
Measles	0	8	0	15	0	0	19	1	43	17	129	41.2	1.0	70	106.4
Mumps	0	12	1	13	2	1	9	5	43	63	52	39.8	1.1	178	145.0
Pertussis	60	560	24	901	224	63	705	442	2,979	2,554	5,480	6,881.4	0.4	9,137	19,309.8
Pneumococcal disease (invasive)	1	173	20	93	34	13	146	73	553	447	730	668.0	0.8	1,208	1,343.2
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	7	0	2	0	0	1	1	11	7	9	11.2	1.0	19	34.2
Rubella – congenital	0	0	0	0	0	0	0	0	0	1	1	0.2	0.0	1	0.2
Tetanus	0	0	0	0	0	0	0	0	0	1	2	0.6	0.0	4	3.0
Varicella zoster (chickenpox)	2	NN	23	127	72	9	229	105	567	433	601	553.8	1.0	1,370	1,308.6
Varicella zoster (shingles)	12	NN	62	12	403	60	284	303	1,136	1,284	1,083	788.6	1.4	3,619	2,434.8
Varicella zoster (unspecified)	41	NN	2	1,483	35	25	829	302	2,717	2,240	2,035	1,720.0	1.6	7,189	5,004.6
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	8	0	0	0	0	8	4	2	3.2	2.5	17	9.0
Barmah Forest virus infection	2	76	79	461	20	1	13	147	799	1,559	284	284.6	2.8	3,784	1,297.6
Dengue virus infection	5	85	19	80	20	6	138	155	508	511	201	165.4	3.1	1,509	862.6
Japanese encephalitis virus infection	0	0	0	2	0	0	0	0	2	2	0	0.2	10.0	4	0.4
Kunjin virus infection**	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	1.2
Malaria	1	28	8	22	1	3	31	13	107	85	100	122.2	0.9	330	334.6
Murray Valley encephalitis virus infection**	0	0	0	1	0	0	0	0	1	0	0	0.0	0.0	1	4.4
Ross River virus infection	2	85	71	321	41	0	26	132	678	1,316	484	602.8	1.1	3,339	4,220.6

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2013, by date of diagnosis

Disease	State or territory								Total this quarter 2013	Total last quarter 2012	Total this quarter 2012	Last 5 years mean this quarter	Ratio	Year to date 2013	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	1	0.0
Brucellosis	0	1	0	0	0	0	0	0	1	4	13	9.6	0.1	10	25.4
Leptospirosis	0	4	0	16	1	0	2	0	23	40	11	18.4	1.3	77	121.6
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	1	0	0	0	0	8	0	9	16	16	18.0	0.5	32	54.0
Q fever	0	31	0	67	3	0	12	2	115	141	89	82.0	1.4	356	258.8
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.5
Other bacterial infections															
Legionellosis	0	33	1	80	10	0	14	33	171	113	99	73.8	2.3	381	237.6
Leprosy	1	0	0	0	0	0	1	1	3	4	2	2.0	1.5	9	6.0
Meningococcal infection††	1	20	1	5	6	2	10	4	49	27	83	87.2	0.6	115	193.4
Tuberculosis	5	113	12	29	19	5	105	44	332	290	335	342.2	1.0	943	936.6
Total	1,087	16,274	2,032	14,508	5,835	964	15,314	8,108	64,122	51,283	79,531			169,005	

* 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 and tuberculosis, the earliest of specimen date, health professional notification date or 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, 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).

** 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 July to 30 September 2013, 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)‡	2.1	0.3	1.7	0.7	0.2	0.8	0.4	1.6	0.6
Hepatitis B (unspecified)§	35.2	38.7	110.6	21.3	21.0	9.4	31.8	48.0	33.2
Hepatitis C (newly acquired)‡	1.1	0.9	0.0	NN	3.4	3.9	2.0	5.3	2.1
Hepatitis C (unspecified)§	56.5	51.5	103.7	54.8	28.0	51.5	41.4	54.4	48.9
Hepatitis D	0.0	0.1	0.0	0.1	0.0	0.0	0.4	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	96.0	NN	74.8	75.3	122.2	96.8	98.8	83.7	91.5
Cryptosporidiosis	1.1	4.1	20.4	8.2	4.8	3.1	12.1	7.4	7.4
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Hepatitis A	3.2	0.7	0.0	1.1	0.0	0.0	1.0	0.5	0.8
Hepatitis E	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.2	0.0
Listeriosis	0.0	0.4	0.0	0.0	0.2	0.8	0.4	0.2	0.3
STEC, VTEC‖	0.0	0.0	0.0	5.6	2.4	0.8	0.1	0.2	1.4
Salmonellosis	50.1	29.3	159.9	42.0	54.6	26.5	40.2	45.2	39.7
Shigellosis	1.1	2.2	37.4	1.3	2.2	0.0	1.9	2.5	2.3
Typhoid fever	1.1	0.3	0.0	0.2	0.2	0.0	0.1	0.7	0.3
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¶,**	324.3	278.3	1,374.3	432.1	316.9	268.6	331.3	489.5	359.6
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	25.6	58.6	789.2	60.0	51.4	11.7	50.4	73.5	63.9
Syphilis – congenital	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.3	0.1
Syphilis < 2 years duration**	3.2	7.9	17.0	7.0	2.4	2.3	13.6	3.0	8.1
Syphilis > 2 years or unspecified duration§,**	3.2	5.9	23.8	6.7	8.9	3.1	9.9	14.3	8.3
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.2	0.0	0.2	0.0	0.0	0.2	0.0	0.1
Influenza (laboratory confirmed)	414.0	344.5	193.9	226.8	575.0	126.5	268.4	212.9	299.3
Measles	0.0	0.4	0.0	1.3	0.0	0.0	1.4	0.2	0.8
Mumps	0.0	0.7	1.7	1.1	0.5	0.8	0.6	0.8	0.8
Pertussis	64.0	30.7	40.8	78.9	54.1	49.2	50.1	72.7	52.5
Pneumococcal disease (invasive)	1.1	9.5	34.0	8.1	8.2	10.1	10.4	12.0	9.7
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.4	0.0	0.2	0.0	0.0	0.1	0.2	0.2
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 *continued*: Notification rates of diseases, 1 July to 30 September 2013, 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)	2.1	NN	39.1	11.1	17.4	7.0	16.3	17.3	14.7
Varicella zoster (shingles)	12.8	NN	105.5	1.1	97.3	46.8	20.2	49.8	29.5
Varicella zoster (unspecified)	43.7	NN	3.4	129.9	8.5	19.5	58.9	49.7	70.5
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.1
Barmah Forest virus infection	2.1	4.2	134.4	40.4	4.8	0.8	0.9	24.2	14.1
Dengue virus infection	5.3	4.7	32.3	7.0	4.8	4.7	9.8	25.5	8.9
Japanese encephalitis virus infection	0.0	0.0	0.0	0.2	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	1.1	1.5	13.6	1.9	0.2	2.3	2.2	2.1	1.9
Murray Valley encephalitis virus infection††	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	2.1	4.7	120.8	28.1	9.9	0.0	1.8	21.7	11.9
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Leptospirosis	0.0	0.2	0.0	1.4	0.2	0.0	0.1	0.0	0.4
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.1	0.0	0.0	0.0	0.0	0.6	0.0	0.2
Q fever	0.0	1.7	0.0	5.9	0.7	0.0	0.9	0.3	2.0
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	0.0	1.8	1.7	7.0	2.4	0.0	1.0	5.4	3.0
Leprosy	1.1	0.0	0.0	0.0	0.0	0.0	0.1	0.2	0.1
Meningococcal infection‡‡	1.1	1.1	1.7	0.4	1.4	1.6	0.7	0.7	0.9
Tuberculosis	5.3	6.2	20.4	2.5	4.6	3.9	7.5	7.2	5.8

* 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 and tuberculosis, the earliest of specimen date, health professional notification date or 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, 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).

†† 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 2013

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

Introduction

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

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months, for 3-month birth cohorts of children at the stated ages between 1 January and 31 March 2013. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, pneumococcal conjugate, 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 three doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of all other hepatitis B vaccines. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 or 4 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP Hib vaccine or 4 doses of any other Hib vaccine, 3 or 4 doses of Comvax hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine.

'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 or 5 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

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

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

Results

The percentage of children 'fully immunised' by 12 months of age for Australia decreased from the previous quarter by 1.1 percentage points to 90.4% (Table 1). Except for the 2 territories, all jurisdictions experienced decreases in coverage for all individual vaccines due at 12 months of age, ranging from –0.7 of a percentage point to –1.7 percentage points. This decrease was likely due to the cessation of an ACIR mail-out to parents in late 2012. Prior to December 2012, the ACIR conducted a mail-out every quarter to parents of children who are identified as not up-to-date at 9 months of age according to ACIR records. The children targeted are those who would have been assessed against the 12–15 month age cohort for the following coverage quarter.

Table 1. Percentage of children immunised at 12 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 2013

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,370	25,059	1,022	16,094	4,976	1,484	18,790	8,536	77,331
Diphtheria, tetanus, pertussis (%)	94.4	90.4	91.7	91.8	91.0	91.1	91.4	90.6	91.1
Poliomyelitis (%)	94.3	90.3	91.7	91.7	90.9	91.1	91.3	90.6	91.0
Haemophilus influenzae type b (%)	94.0	90.0	91.6	91.6	90.5	91.0	91.0	90.4	90.8
Hepatitis B (%)	93.9	90.0	91.5	91.5	90.4	91.1	90.8	90.0	90.6
Fully immunised (%)	93.4	89.7	91.4	91.4	90.2	91.0	90.5	89.8	90.4
Change in fully immunised since last quarter (%)	+1.0	-1.2	+0.4	-0.9	-1.2	-1.4	-1.3	-0.8	-1.1

Table 2. Percentage of children immunised at 24 months of age for the birth cohort 1 January to 31 March 2011, preliminary results, by disease and state or territory; assessment date 30 June 2013*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,396	24,586	940	15,853	4,857	1,579	18,143	8,438	75,792
Diphtheria, tetanus, pertussis (%)	95.4	94.7	95.2	94.2	94.7	96.1	95.4	93.4	94.7
Poliomyelitis (%)	95.4	94.7	95.2	94.2	94.7	96.1	95.3	93.4	94.6
Haemophilus influenzae type b (%)	94.3	93.6	94.4	93.4	93.8	95.7	94.0	92.3	93.6
Measles, mumps, rubella (%)	94.4	93.6	94.9	93.6	94.0	95.3	94.2	92.4	93.7
Hepatitis B (%)	95.1	94.2	95.0	93.9	94.3	96.1	94.8	92.7	94.2
Fully immunised (%)	93.3	91.9	93.1	92.3	92.7	94.2	92.7	90.5	92.1
Change in fully immunised since last quarter (%)	+0.4	-0.0	-0.0	-0.3	+0.4	-0.7	+0.0	+0.1	-0.1

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2012;36(3):E308.

Table 3. Percentage of children immunised at 60 months of age for the birth cohort 1 January to 31 March 2008, preliminary results, by disease and state or territory; assessment date 30 June 2013

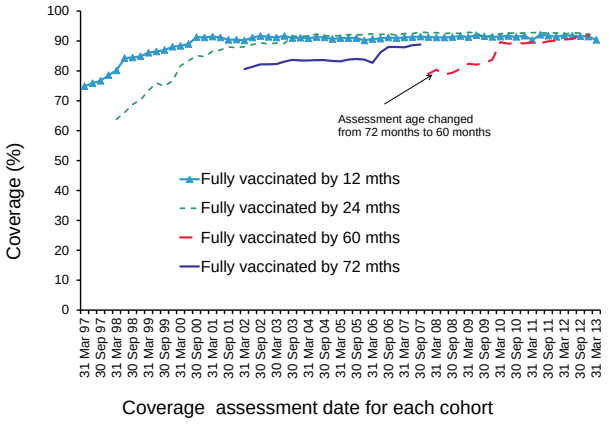
Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,289	24,798	904	16,275	5,074	1,628	18,554	8,567	77,089
Diphtheria, tetanus, pertussis (%)	91.8	92.6	91.4	92.0	92.0	94.0	92.9	90.2	92.3
Poliomyelitis (%)	91.7	92.5	91.6	92.0	91.9	93.9	92.9	90.2	92.2
Measles, mumps, rubella (%)	91.8	92.5	91.9	91.9	91.9	94.7	92.8	90.1	92.2
Fully immunised (%)	91.2	92.2	91.2	91.5	91.6	93.6	92.4	89.7	91.8
Change in fully immunised since last quarter (%)	-2.5	+0.5	-0.2	-0.4	+0.5	+1.8	-0.5	+0.4	+0.0

The percentage of children 'fully immunised' by 24 months of age for Australia decreased marginally from the previous quarter by 0.1 of a percentage point to 92.1% (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

The percentage of children 'fully immunised' by 60 months of age for Australia remained at 91.8% (Table 3). This maintains the improvement in coverage for this age milestone. There were no important changes in coverage for any individual vaccines due at 60 months of age or by jurisdiction.

The Figure shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007). However, for this quarter, a decrease was observed for 12 month coverage. Coverage at 24 and 60 months is still higher than coverage at 12 months of age.

Figure: Trends in vaccination coverage, Australia, 1997 to 31 March 2013, by age cohorts



Australian Gonococcal Surveillance Programme, 1 July to 30 September 2013

Monica M Lahra for the Australian Gonococcal Surveillance Programme

Introduction

The Australian National Neisseria Network reference laboratories in each State and Territory report data quarterly on sensitivity to an agreed group of antimicrobial agents, for the Australian Gonococcal Surveillance Programme (AGSP). The antibiotics routinely tested and reported are penicillin, ceftriaxone, ciprofloxacin and spectinomycin which are current or potential agents used for the treatment of gonorrhoea. Azithromycin testing is now performed by all states and territories as it has a role as part of a dual therapy regimen in the treatment of gonorrhoea. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatments.[1] Additional data are also provided on other antibiotics from time to time. These data are reported in the AGSP annual report. The AGSP has a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. The AGSP data are presented quarterly in tabulated form, as well as in the AGSP annual report.

Results

Penicillin resistant *Neisseria gonorrhoeae* (NG) is defined as those isolates with a minimum inhibitory concentration (MIC) to penicillin equal to or greater than 1.0 mg/L. Total penicillin resistance includes penicillinase producing NG (PPNG) and NG that have chromosomally mediated resistance to penicillin (CMRP).

Quinolone resistant NG is defined as those isolates with a MIC to ciprofloxacin equal to or greater than 0.06 mg/L.

Azithromycin resistance is defined as a MIC to azithromycin equal to or greater than 1.0 mg/L. There were no isolates reported in Australia with high level resistance (HLR) with an azithromycin (MIC value >256 mg/L) in this quarter.

Ceftriaxone MIC values in the range 0.06–0.250 mg/L have been reported in the category decreased susceptibility (DS) since 2005. To date there has not been an isolate reported in Australia with a ceftriaxone MIC value >0.125 mg/L.

Over the 1st half of 2013, a sustained increase was reported in the proportion of NG isolates with DS to ceftriaxone, predominantly from New South Wales and Victoria, when compared with the same period in 2012. This increase was again evident in the 3rd quarter of 2013, compared with the same quarter in 2012. When compared with the 2nd quarter of 2013, there was a decrease from 10.9% to 8.8% in the proportion of NG isolates with DS to

Table: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to ciprofloxacin, azithromycin and penicillin, Australia, 1 July to 30 September 2013, by state or territory

State or territory	Number of isolates tested	Decreased susceptibility		Resistance					
		Ceftriaxone		Ciprofloxacin		Azithromycin		Penicillin	
		n	%	n	%	n	%	n	%
ACT	9	0	0.0	3	33.0	0	0.0	2	22.0
NSW	408	45	11.0	139	34.0	1	0.3	177	43.0
NT	78	0	0.0	3	3.9	0	0.0	3	3.9
Qld	165	9	5.5	49	30.0	16	10.0	47	28.5
SA	73	0	0.0	7	9.6	1	1.4	6	8.2
Tas.	10	4	40.0	5	50.0	0	0.0	6	60.0
Vic.	344	47	14.0	133	39.0	6	1.8	118	34.0
WA	111	0	0.0	30	27.0	4	3.6	33	30.0
Aust.	1,198	105	8.8	369	30.8	28	2.3	392	33.0

ceftriaxone nationally, but this is more than double that reported in the 3rd quarter of 2011 and 2012 (3.4% and 3.6%, respectively).

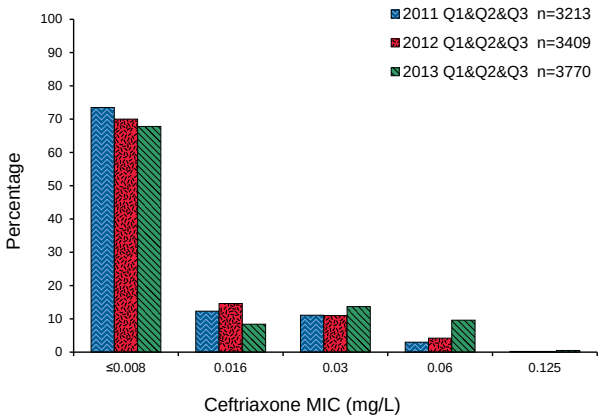
The highest proportions of isolates with decreased susceptibility to ceftriaxone were reported from the eastern states: Victoria, New South Wales and Queensland. In Victoria there were 47 strains with DS to ceftriaxone and of those, 42/47 (89%) were multiple drug resistant; 38/47 (81%) were from males; and 22/47 (46%) were isolated from extra genital sites (rectal and pharyngeal). In New South Wales there were 45 strains with decreased susceptibility to ceftriaxone and of those, 31/45 (69%) were multiple drug resistant; 40/45 (89%) were from males; and 23/45 (51%) were isolated from extra genital sites (rectal and pharyngeal). In contrast there were no gonococci with DS to ceftriaxone reported from the Northern Territory, the Australian Capital Territory, South Australia or Western Australia.

There are recent reports of ceftriaxone 500 mg treatment failure from Victoria and New South Wales. These patients had pharyngeal infections where the gonococcal strains with ceftriaxone MIC values in the range 0.03–0.06 mg/L.[2,3] In addition, in 2013 the first reports of strains with azithromycin HLR were reported from Victoria and Queensland. These are the first reports of HLR to azithromycin reported in Australia.

In response to concerns over the increasing proportions of NG strains with DS to ceftriaxone, dual therapy (ceftriaxone plus azithromycin) is recommended as a strategy to temper development of more widespread resistance.[4] Patients with infections in extra genital sites, where the isolate has decreased susceptibility to ceftriaxone, are recommended to have a test of cure.[4]

The proportion of NG strains at each ceftriaxone MIC value is shown in the Figure, where it can be seen that the greatest increase is in the proportion of isolates with a ceftriaxone MIC value of 0.06–0.25 mg/L.

Figure: Distribution of ceftriaxone MIC values in gonococcal isolates tested by the Australian Gonococcal Surveillance Programme, 1 January to 30 September 2011 to 2013



Reference

1. Management of Sexually Transmitted Diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.
2. Read PJ, Limnios EA, McNulty A, Whiley D, Lahra MM. One confirmed and one suspected case of pharyngeal gonorrhoea treatment failure following 500 mg ceftriaxone in Sydney, Australia. *Sex Health* 2013;10(5):460–462.
3. Chen M, Stevens K, Tideman R, Zaia A, Fairley CK, Lahra MM, Hogg G. Failure of 500 mg of ceftriaxone to eradicate pharyngeal gonorrhoea, Australia. *J Antimicrob Chemother* 2013;68(6):1445–1447.
4. Australasian Sexual Health Alliance. *Australian Sexually Transmitted Infection Treatment Guidelines*. [online] Available from: www.sti.guidelines.org.au

Australian Meningococcal Surveillance Programme, 1 July to 30 September 2013

Monica M Lahra, Rodney Enriquez for the Australian Meningococcal Surveillance Programme

Introduction

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of cases confirmed by laboratory testing using culture and by non-culture based techniques. Culture positive cases, where *Neisseria meningitidis* is grown from a normally sterile site or skin lesions, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in quarterly reports are restricted to a description of the number of cases by jurisdiction and serogroup, where known. Some minor corrections to data in the Table may be made in subsequent reports if additional data are received. A full analysis of laboratory confirmed cases of IMD in each calendar year is contained in the annual reports of the programme published in *Communicable Diseases Intelligence*. For more information see *Commun Dis Intell* 2013;37(1):E61.

Results

Laboratory confirmed cases of invasive meningococcal disease for the period 1 July to 30 September 2013 are shown in the Table.

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 July to 30 September 2013, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD
Australian Capital Territory	2013	0	0	1	2	0	0	0	1	0	0	0	0	1	3
	2012	0	0	0	2	0	0	0	0	0	0	0	0	0	2
New South Wales	2013	0	0	7	16	0	2	3	4	5	5	1	2	16	29
	2012	0	0	16	36	1	1	3	4	2	2	4	8	26	51
Northern Territory	2013	0	0	1	2	0	0	0	0	0	0	0	0	1	2
	2012	0	0	0	2	0	1	0	0	0	0	0	1	0	4
Queensland	2013	0	0	4	19	1	2	0	2	1	3	1	1	7	27
	2012	0	0	16	36	1	2	3	3	2	3	4	4	26	48
South Australia	2013	0	0	7	17	0	0	0	1	0	1	0	0	7	19
	2012	0	0	10	17	0	1	0	0	0	0	0	0	10	18
Tasmania	2013	0	0	1	2	0	0	0	0	0	0	1	1	2	3
	2012	0	0	2	3	0	0	1	1	0	0	0	1	3	5
Victoria	2013	0	0	8	16	0	1	0	0	1	1	0	1	9	19
	2012	0	0	7	20	0	0	2	4	0	0	1	1	10	25
Western Australia	2013	0	0	3	11	1	2	0	0	0	1	0	0	4	14
	2012	0	0	4	11	1	2	0	1	0	0	0	1	5	15
Australia	2013	0	0	32	85	2	7	3	8	7	11	3	5	47	116
	2012	0	0	55	127	3	7	9	13	4	5	9	16	80	168

Australian Sentinel Practices Research Network, 1 October to 31 December 2012

Monique Chilver, Daniel Blakeley 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.

In June 2010, ASPREN's laboratory influenza-like illness (ILI) testing was implemented, allowing for viral testing of 25% of ILI patients for a range of respiratory viruses including influenza A, influenza B and influenza A(H1N1)pdm09.

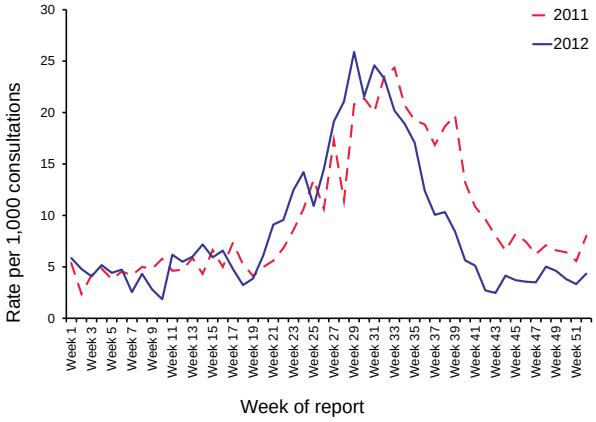
The list of conditions is reviewed annually by the ASPREN management committee. In 2013, 4 conditions were monitored. They included ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, published in *Commun Dis Intell* 2013;37(1):E62.

Results

Sentinel practices contributing to ASPREN were located in all 8 states and territories in Australia. A total of 244 general practitioners contributed data to ASPREN in the 4th quarter of 2012. Each week an average of 199 general practitioners provided information to ASPREN at an average of 16,388 (range 13,438–19,063) consultations per week and an average of 163 (range 143–205) notifications per week.

ILI rates reported from 1 October to 31 December 2012 averaged 4 cases per 1,000 consultations (range 3–6 cases per 1,000 consultations). This was lower than the same reporting period in 2011, which averaged 8 cases per 1,000 consultations (range 6–13 cases per 1,000 consultations) (Figure 1).

Figure 1: Consultation rates for influenza-like illness, ASPREN, 2011 and 2012, by week of report



ILI swab testing has continued during 2012. From the beginning of 2012 to the end of week 52, 1,277 cases of influenza have been detected, the majority of these being influenza A (untyped) (26% of all swabs performed), influenza B (11% of all swabs performed) and the remainder A(H1N1) pdm09 (0.5% of all swabs performed) (Figure 2).

During this reporting period, consultation rates for gastroenteritis averaged 5 cases per 1,000 consultations (range 3–6 cases per 1,000 consultations, Figure 3). This was lower than the same reporting period in 2011 where the average was 6 cases per 1,000 consultations (range 3–12 cases per 1,000 consultations).

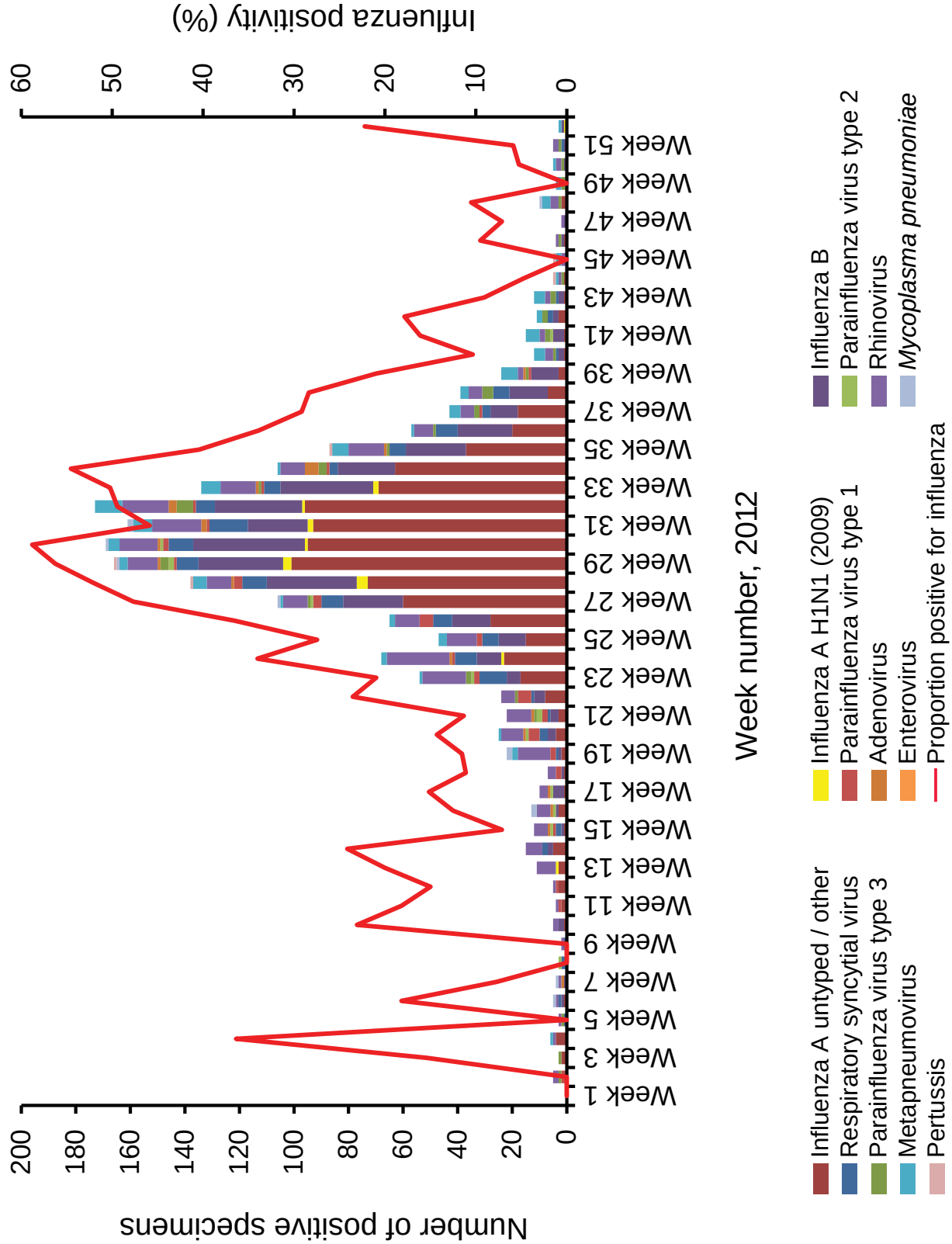
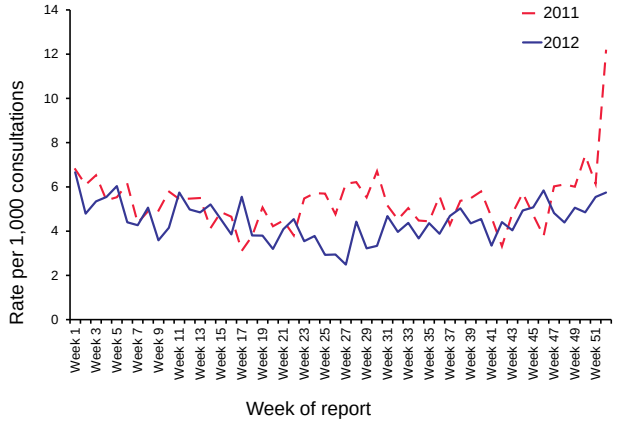


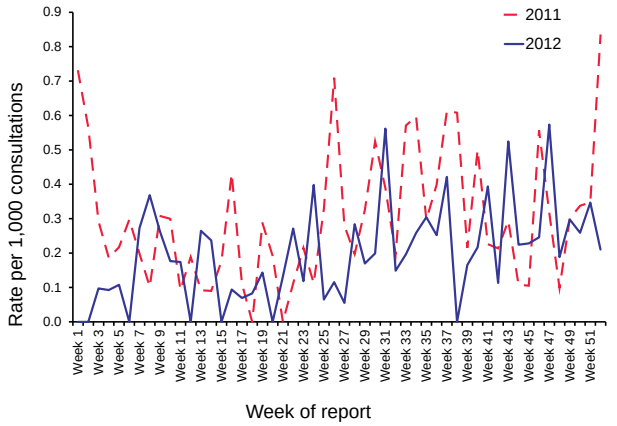
Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January to 31 December 2012, by week of report

Figure 3: Consultation rates for gastroenteritis, ASPREN, 2011 and 2012, by week of report



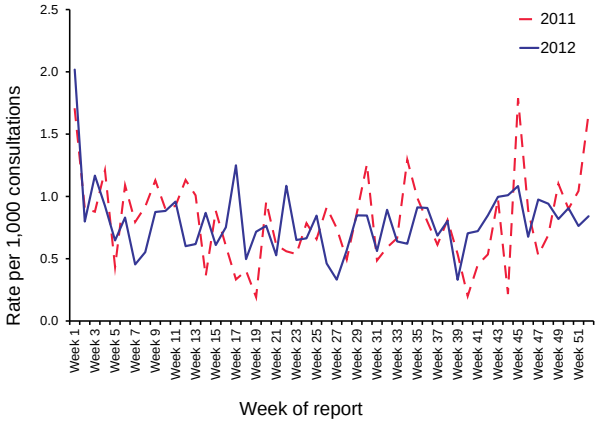
Varicella infections were reported at a lower rate for the 4th quarter of 2012 compared with the same period in 2011. From 1 October to 31 December 2012, recorded rates for chickenpox averaged 0.3 cases per 1,000 consultations (range 0.1–0.6 cases per 1,000 consultations, Figure 4).

Figure 4: Consultation rates for chickenpox, ASPREN, 2011 and 2012, by week of report



In the 4th quarter of 2012, reported rates for shingles averaged 0.9 cases per 1,000 consultations (range 0.7–1.1 cases per 1,000 consultations, Figure 5), unchanged from the same reporting period in 2011 where the average shingles rate was 0.9 case per 1,000 consultations (range 0.2–1.8 cases per 1,000 consultations).

Figure 5: Consultation rates for shingles, ASPREN, 2011 and 2012, by week of report



HIV and AIDS surveillance, 1 April to 30 June 2012

Kirby Institute

Introduction

National surveillance for HIV infection is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the state and territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the Kirby Institute, University of New South Wales, SYDNEY NSW 2052. Internet: http://hiv.cms.med.unsw.edu.au/ Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see *Commun Dis Intell* 2013;37(1):E63.

Results

HIV and AIDS diagnoses and deaths following AIDS reported for 1 April to 30 June 2012, are shown in Tables 1 and 2).

Table 1: Number of new diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 April to 30 June 2012, by sex and state or territory of diagnosis

		State or territory								Totals for Australia			
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2012	This period 2011	YTD 2012	YTD 2011
HIV diagnoses	Female	2	13	1	9	2	0	10	4	41	44	85	69
	Male	4	80	5	55	2	2	60	21	229	262	526	524
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	6	93	6	64	4	2	71	25	271	306	612	593
AIDS diagnoses	Female	0	0	0	0	0	0	2	0	2	4	5	9
	Male	0	4	4	0	0	0	4	1	13	23	36	49
	Total*	0	4	4	0	0	0	6	1	15	27	41	58
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	0	0	1
	Male	0	1	0	0	0	0	0	0	1	5	3	11
	Total*	0	1	0	0	0	0	0	0	1	5	3	12

* Totals include people whose sex was reported as transgender.

Table 2: Number of new diagnoses of HIV infection since the introduction of HIV antibody testing 1985, and number of new diagnoses of AIDS and deaths following AIDS since 1981, cumulative to 30 June 2012, by sex and state or territory of diagnoses

		State or territory								
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
HIV diagnoses	Female	42	1,102	37	444	153	26	555	348	2,707
	Male	325	16,211	183	3,793	1,189	163	6,865	1,617	30,346
	Not reported	0	227	0	0	0	0	22	0	249
	Total*	367	17,580	220	4,246	1,343	189	7,470	1,972	33,387
AIDS diagnoses	Female	10	291	7	81	32	4	138	51	614
	Male	95	5,683	58	1,120	427	60	2,262	482	10,187
	Total*	105	5,993	65	1,203	460	64	2,413	535	10,838
AIDS deaths	Female	7	144	1	44	20	2	67	30	315
	Male	73	3,627	33	687	281	34	1,472	301	6,508
	Total*	80	3,782	34	733	301	36	1,548	332	6,823

* Totals include people whose sex was reported as transgender.

HIV and AIDS surveillance, 1 July to 30 September 2012

Kirby Institute

Introduction

National surveillance for HIV infection is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the state and territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the Kirby Institute, University of New South Wales, SYDNEY NSW 2052. Internet: http://hiv.cms.med.unsw.edu.au/ Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see *Commun Dis Intell* 2013;37(1):E63.

Results

HIV and AIDS diagnoses and deaths following AIDS reported for 1 July to 30 September 2012, are shown in Tables 1 and 2).

Table 1: Number of new diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 July to 30 September 2012, by sex and state or territory of diagnosis

		State or territory								Totals for Australia			
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2012	This period 2011	YTD 2012	YTD 2011
HIV diagnoses	Female	0	15	1	7	3	0	6	10	42	37	127	106
	Male	4	111	5	48	14	6	70	27	285	240	811	764
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	4	126	6	55	17	6	76	37	327	277	939	870
AIDS diagnoses	Female	0	2	0	0	0	0	1	0	3	2	8	11
	Male	0	7	1	3	0	0	5	0	16	30	52	79
	Total*	0	9	1	3	0	0	6	0	19	32	60	90
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	2	0	3
	Male	0	2	1	0	0	0	3	0	6	10	9	21
	Total*	0	2	1	0	0	0	3	0	6	12	9	24

* Totals include people whose sex was reported as transgender.

Table 2: Number of new diagnoses of HIV infection since the introduction of HIV antibody testing 1985, and number of new diagnoses of AIDS and deaths following AIDS since 1981, cumulative to 30 September 2012, by sex and state or territory

		State or territory								
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
HIV diagnoses	Female	42	1,117	38	451	156	26	561	358	2,749
	Male	329	16,322	188	3,841	1,203	169	6,935	1,644	30,631
	Not reported	0	227	0	0	0	0	22	0	249
	Total*	371	17,706	226	4,301	1,360	195	7,546	2,009	33,714
AIDS diagnoses	Female	10	293	7	81	32	4	139	51	617
	Male	95	5,690	59	1,123	427	60	2,267	482	10,203
	Total*	105	6,002	66	1,206	460	64	2,419	535	10,857
AIDS deaths	Female	7	144	1	44	20	2	67	30	315
	Male	73	3,624	33	687	281	34	1,472	301	6,505
	Total*	80	3,779	34	733	301	36	1,548	332	6,843

* Totals include people whose sex was reported as transgender.

HIV and AIDS surveillance, 1 October to 31 December 2012

Kirby Institute

Introduction

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Results

HIV and AIDS diagnoses and deaths following AIDS reported for 1 October to 31 December 2012, are shown in Tables 1 and 2).

Table 1: Number of new diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 October to 31 December 2012, by sex and state or territory of diagnosis

		State or territory								Totals for Australia			
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2012	This period 2011	YTD 2012	YTD 2011
HIV diagnoses	Female	0	13	3	5	3	0	5	3	32	37	159	143
	Male	3	102	5	66	9	2	76	18	281	232	1,092	996
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	3	116	8	71	12	2	81	21	314	270	1,253	1,140
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	5	8	16
	Male	0	4	1	10	0	0	0	0	15	20	67	99
	Total*	0	4	1	10	0	0	0	0	15	25	75	115
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	0	0	3
	Male	0	0	0	0	0	0	0	0	0	0	9	21
	Total*	0	0	0	0	0	0	0	0	0	0	9	24

* Totals include people whose sex was reported as transgender.

Table 2: Number of new diagnoses of HIV infection since the introduction of HIV antibody testing 1985, and number of new diagnoses of AIDS and deaths following AIDS since 1981, cumulative to 31 December 2012, by sex and state or territory

		State or territory								
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
HIV diagnoses	Female	42	1,130	41	456	159	26	566	361	2,781
	Male	332	16,424	193	3,907	1,212	171	7,011	1,662	30,912
	Not reported	0	227	0	0	0	0	22	0	249
	Total*	374	17,822	234	4,372	1,372	197	7,627	2,030	34,028
AIDS diagnoses	Female	10	293	7	81	32	4	139	51	617
	Male	95	5,694	60	1,133	427	60	2,267	482	10,218
	Total*	105	6,006	67	1,216	460	64	2,419	535	10,872
AIDS deaths	Female	7	144	1	44	20	2	67	30	315
	Male	73	3,629	34	687	281	34	1,475	301	6,514
	Total*	80	3,784	35	733	301	36	1,551	332	6,852

* Totals include people whose sex was reported as transgender.

Invasive pneumococcal disease surveillance Australia, 1 July to 30 September 2013

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

Introduction

Invasive pneumococcal disease (IPD) is caused by the bacterium *Streptococcus pneumoniae* and results in illnesses 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. The Communicable Diseases Network Australia established the Enhanced Invasive Pneumococcal Disease Surveillance Working Group (EIPDSWG) in 2000 to assist in developing and implementing a nationally standardised approach to the enhanced surveillance of IPD in Australia. This quarterly report documents trends in notified cases of IPD occurring in Australia in the 3rd quarter of 2013.

Notification data are collected by all Australian states and territories under jurisdictional public health legislation and are forwarded to the Commonwealth under the *National Health Security Act 2007*. Notified cases are collated nationally in the National Notifiable Diseases Surveillance System (NNDSS). The data in this report are provisional and subject to change as laboratory results and additional case information become available. The data are analysed by diagnosis date, which is the onset date or where the onset date was not known, the earliest of the specimen collection date, the notification date, and the notification receive date. Data for this report were extracted on 31 October 2013. Crude rates were calculated using the Australian Bureau of Statistics estimated resident populations for Australia at 30 June of each year. Consideration of vaccination status of cases is outside the scope of this report. For more detailed reports readers are referred to the regular *Communicable Diseases Intelligence* supplements *Vaccine Preventable Diseases in Australia*.

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk and older Australians. 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 older in 1999 and for non-Indigenous Australians aged 65 years or older from January 2005.

Results

There were 550 cases of IPD reported to the NNDSS in the 3rd quarter of 2013, bringing the year to date total to 1,202 cases (Table). While the number of cases notified in the reporting period is the highest so far this year, it was a 24% reduction on the number of cases reported in the same period in 2012 (n=726).

Overall, Aboriginal and Torres Strait Islander status was reported for 82% (n=452) of cases, ranging from 68% of cases reported by Victoria to 100% of cases reported by the Australian Capital Territory, the Northern Territory, Tasmania and Western Australia. Victoria and New South Wales only actively follow up notified cases of IPD aged 5 years or under, and 50 years or over for core and enhanced data, whereas follow up of all cases is undertaken in other states and territories. This may account for missing data among cases falling outside these age groups. Of cases with a reported Indigenous status, Aboriginal and Torres Strait peoples accounted for 14% (n=62) of all cases notified in the quarter (Table).

Serotype information was available for 92% (n=508) of all cases reported in the quarter, ranging from 85% of cases reported by South Australia to 100% of cases reported by the Australian Capital Territory and the Northern Territory. There was a single case reported in the quarter that was deemed by the reference laboratory as non-typable. Cases such as these are included in the vaccine serotype group in figures of this report as serotype not specified.

In the 3rd quarter of 2013, notified cases were highest in children aged under 5 years (n=65), followed by the 60–64 years age group (n=55) and the 65–69 years age group (n=50). This age distribution was evident in cases reported as non-Indigenous Australian (Figure 1). However in cases reported as Indigenous, the most prevalent age groups were those under 5 years (n=9) and the 45–49 years age group (n=8). Three age groups

Table: Notified cases of invasive pneumococcal disease, Australia, 1 July to 30 September 2013, by Indigenous status, serotype and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 3rd qtr 2013	Total 2nd qtr 2013	Total 3rd qtr 2012	Year to date 2013
Indigenous	0	5	18	8	2	1	3	25	62			
Non-Indigenous	1	132	2	69	30	12	96	48	390			
Not stated/unknown	0	31	0	19	2	0	46	0	98			
Total	1	168	20	96	34	13	145	73	550	438	726	1,202
Indigenous status completeness* (%)	100	82	100	80	94	100	68	100	82			
Serotype completeness† (%)	100	87	100	96	85	92	95	96	92			

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

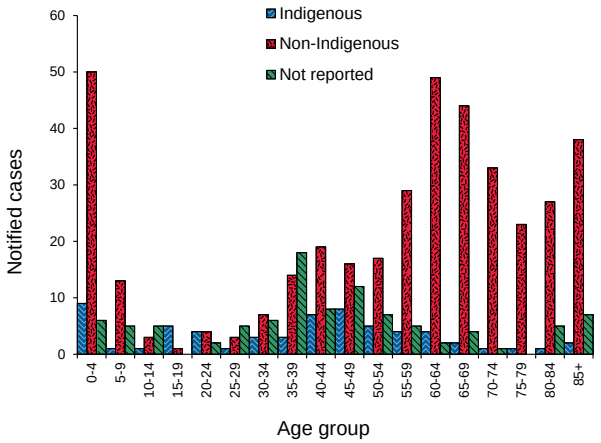
† Serotype completeness is the proportion of all cases of IPD that were reported with a serotype or reported as non-typable. Serotype incompleteness may include when no isolate was available as diagnosis was by polymerase chain reaction and no molecular typing was performed; the isolate was not referred 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. In this report, the category 'Serotype not specified' includes notified cases reported with an incomplete serotype or reported as non-typable.

were selected for focused analyses in this quarterly report. These age groups align with groups that carry the greatest burden of disease and against which the NIP is targeted.

Invasive pneumococcal disease in children aged less than 5 years

In the 3rd quarter of 2013, 12% (n=65) of notified cases were aged less than 5 years. This was a small increase on the number of cases reported in the previous quarter (n=59) and to the number reported during the same period of 2012 (n=58) (Figure 2).

Figure 1: Notified cases of invasive pneumococcal disease, Australia, 1 July to 30 September 2013, by Indigenous status and age group



The majority (88%, n=57) of cases aged less than 5 years were reported with serotype information. Of these, 39% (n=22) were reported with a serotype included in the 7vPCV or the 13vPCV.

Notified cases aged less than 5 years with disease caused by the 6 additional serotypes targeted by the 13vPCV increased steadily over the period 2007 to 2011, particularly those caused by serotype 19A (Figure 3). However, cases of this type have decreased since the 4th quarter of 2011, reflecting the introduction of the 13vPCV on the universal childhood immunisation program in mid-2011. In the 3rd quarter of 2013, there were 9 cases aged less than 5 years with disease due to serotype 19A, 5 cases due to serotype 3, 2 cases of serotype 7F and 1 case due to serotype 1. Similar to the previous quarter, no cases in this age group were reported with disease caused by serotypes 5 or 6A.

Invasive pneumococcal disease in Indigenous Australians aged 50 years or over

In the 2nd quarter of 2013, 4% (n=20) of notified cases were reported in Indigenous Australians aged 50 years or over. This was the highest reported so far this year (1st quarter n=8; 2nd quarter n=16) and was similar to the number reported during the same period in 2012 (n=21)(Figure 4). The annual rate of IPD in this group has tended to increase over time, with an outbreak of disease caused by serotype 1 in Central Australia that commenced in late 2010, contributing in part to this increase.

Figure 2: Notified cases and rates of invasive pneumococcal disease in those aged less than 5 years, Australia, 2002 to 30 September 2013, by vaccine serotype group

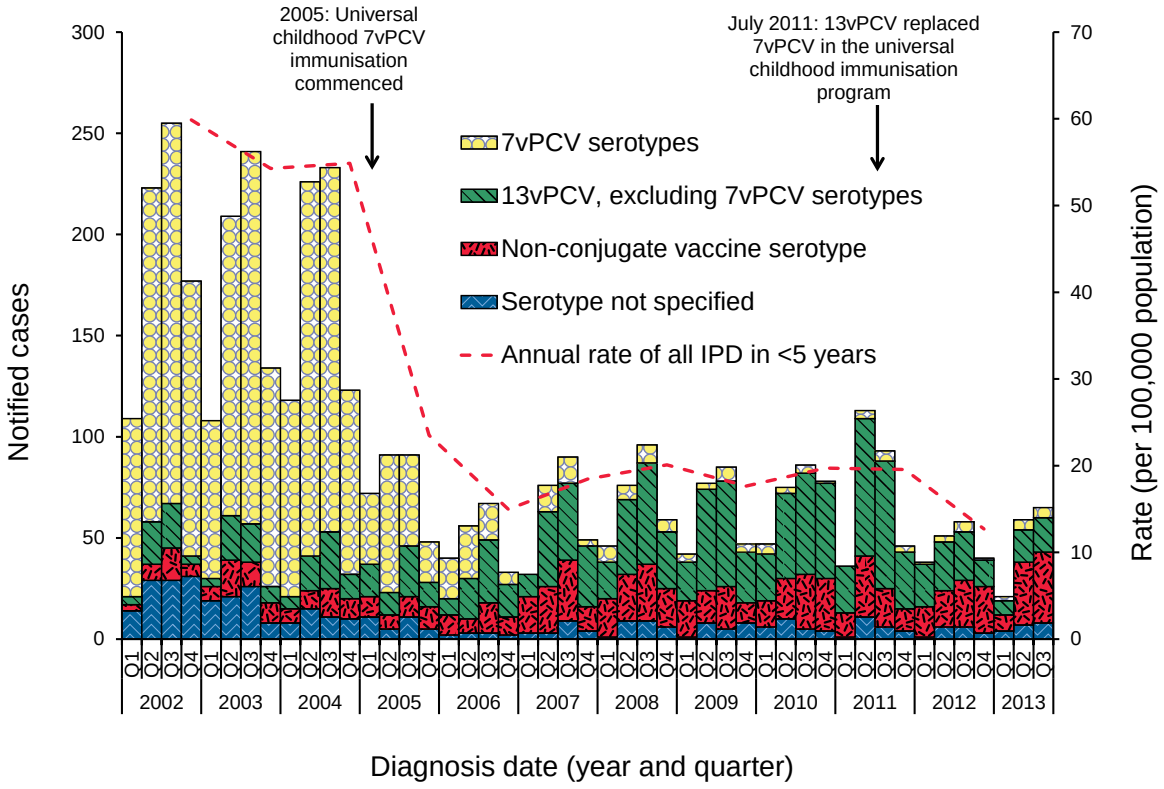


Figure 3: Notified cases of invasive pneumococcal disease caused by serotypes targeted by the 13-valent pneumococcal conjugate vaccine (excluding those targeted by 7-valent pneumococcal conjugate vaccine) and rates of all invasive pneumococcal disease, aged less than 5 years, Australia, 2002 to 30 September 2013

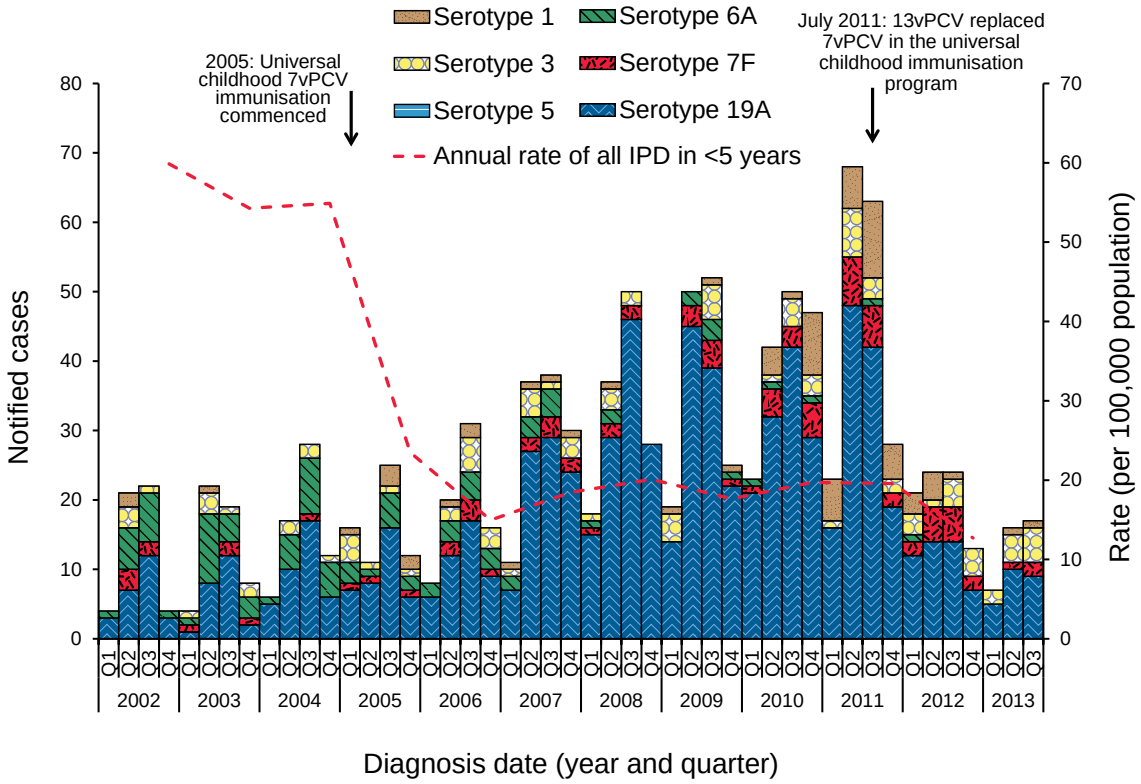
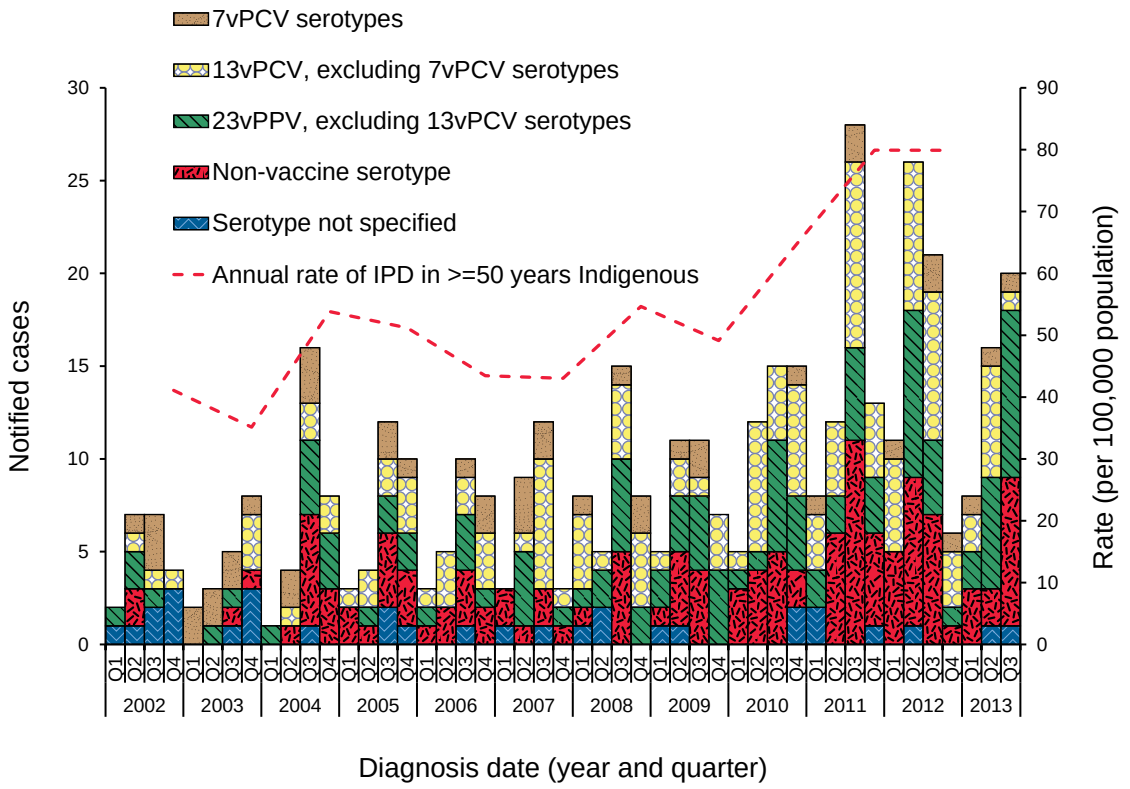


Figure 4: Notified cases and rates of invasive pneumococcal disease in Indigenous Australians aged 50 years or over, Australia, 2002 to 30 September 2013, by vaccine serotype group



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

All but one of the cases were reported with serotype information. Of these, 58% (n=11) were reported with disease due to serotypes targeted by the 23vPPV; the remaining reported disease due to a non-vaccine serotype (n=8).

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

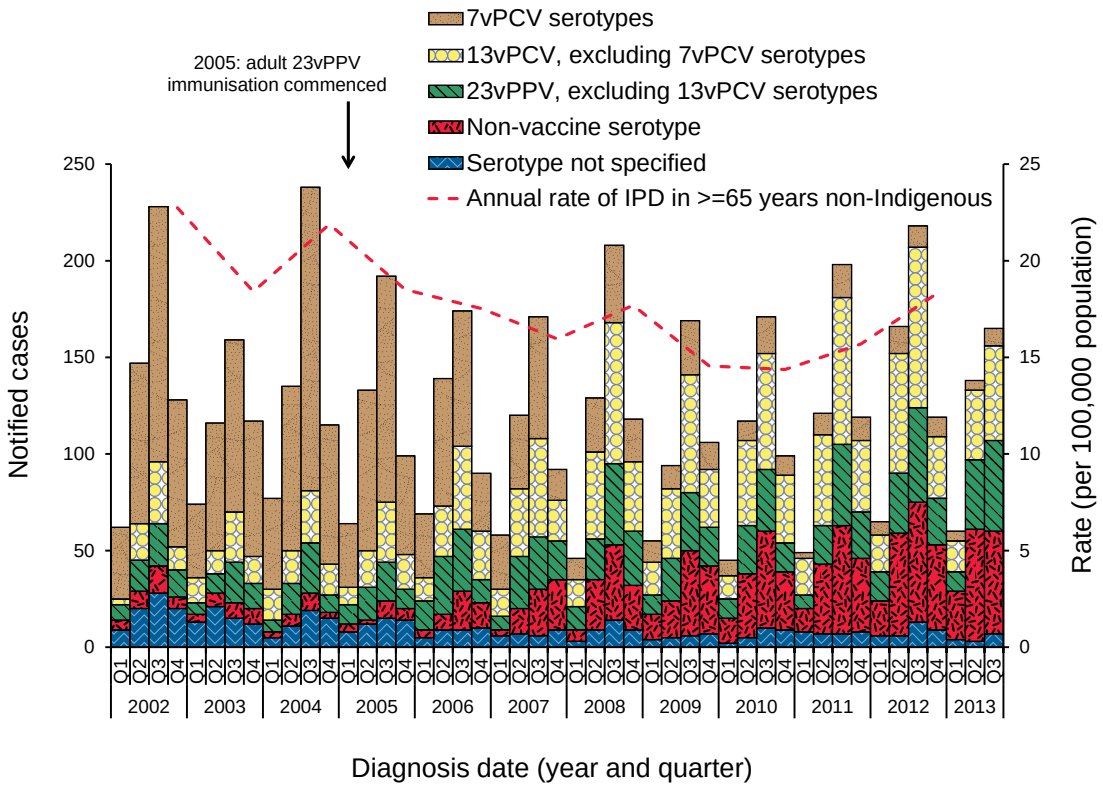
In the 3rd quarter of 2013, 30% (n=165) of notified cases were reported as non-Indigenous Australians aged 65 years or over. This was a moderate increase in the number of cases reported in the previous quarter (n=138), but a 24% decrease on the number reported during the same period of 2012 (n=218) (Figure 5).

The majority (96%, n=158) of cases reported in this quarter were reported with serotype information. Of these cases, 66% (n=105) 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.

Conclusion

While the number of notified cases of IPD in the 3rd quarter of 2013 was the highest so far this year and a 26% increase on the previous quarter, it represented a decline on the incidence reported in the same quarter in 2012. Elevated numbers of IPD notifications during the 2nd and 3rd quarters are consistent with annual winter seasonality observed for many infectious diseases. Nationally, the pattern of disease has not changed from the 2nd quarter this year. Specifically, disease due to the serotypes targeted by the 13vPCV has continued to decline since the 13vPCV replaced the 7vPCV in the childhood immunisation program from July 2011. Notified cases of IPD in Indigenous Australians aged 50 years or over have tended to increase over time, whereas disease in non-Indigenous Australians aged 65 years or over has remained relatively stable but the profile of serotypes causing disease has diversified.

Figure 5: Notified cases and rates of invasive pneumococcal disease in non-Indigenous Australians aged 65 years or over, Australia, 2002 to 30 September 2013, by vaccine serotype group



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EIPDSWG contributors to this report include (in alphabetical order): Christina Bareja (Health), David Coleman (Tas), Rachel de Kluyver (Health), Heather Cook (NT), Lucinda Franklin (Vic), Carolien Giele (WA), Robin Gilmour (NSW), Michelle Green (Tas), Geoff Hogg (Microbiological Diagnostic Unit, University of Melbourne), Vicki Krause (NT), Rob Menzies (NCIRS), Shahin Oftadeh (Centre for Infectious Diseases and Microbiology- Public Health, Westmead Hospital), Sue Reid (ACT), Stacey Rowe (Vic), Vitali Sintchenko (Centre for Infectious Diseases and Microbiology- Public Health, Westmead Hospital), Helen Smith (Queensland Health Forensic and Scientific Services), Janet Strachan (Microbiological Diagnostic Unit, University of Melbourne), Hannah Vogt (SA), Angela Wakefield (Qld).

Author details

Rachel de Kluyver[1]

1. Vaccine Preventable Diseases Surveillance Section, Health Emergency Management Branch, Office of Health Protection, Department of Health, Canberra ACT

Corresponding author: Ms Rachel de Kluyver, Vaccine Preventable Diseases Surveillance Section, Office of Health Protection, Australian Government Department of Health, GPO Box 9484, MDP 14, Canberra, ACT 2601. Telephone: +61 2 6289 1463. Facsimile: +61 2 6289 1070. Email: Rachel.de.kluyver@health.gov.au

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