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Annual reports

Invasive pneumococcal disease in Australia 2007 and 2008

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Abstract

Enhanced surveillance for invasive pneumococcal disease (IPD) was conducted in all Australian states and territories in 2007 and 2008 with comprehensive comparative data available since 2002. There were 1,477 cases of IPD notified to the National Notifiable Diseases Surveillance System in Australia in 2007; a notification rate of 7.0 cases per 100,000 population. In 2008 there were 1,628 cases; a notification rate of 7.6 cases per 100,000 population. The overall rate of IPD in Indigenous Australians was almost 6 times the rate in non-Indigenous Australians in 2007 and almost 5 times in 2008. By 2008, the 4th year of a funded universal infant 7-valent pneumococcal conjugate vaccine (7vPCV) program in Australia with a 3+0 schedule, vaccine serotype IPD notification rates in those identified as non-Indigenous decreased in all age groups compared with 2002 levels, most significantly by 96% in children aged less than 5 years. However, rates of disease in non-vaccine serotypes increased by 168% in children aged less than 5 years, including a four-fold increase in the number of cases due to serotype 19A. For the Aboriginal and Torres Strait Islander population, national pre-vaccination data are not available, as the vaccine program was funded for this group from 2001. From 2002 to 2008, the proportion of disease due to 7vPCV serotypes in children aged less than 5 years decreased by 77%, while disease due to non-7vPCV serotypes increased by 76%. In Indigenous adults (≥50 years), rates of 23vPPV serotypes increased by 92%. There were 120 deaths attributed to IPD in 2007 and 113 in 2008, although it should be noted that deaths may be under-reported. The number of invasive pneumococcal isolates with reduced penicillin susceptibility remains low and reduced susceptibility to third-generation cephalosporins is rare. *Commun Dis Intell* 2012;36(2):E151–E165.

Introduction

Streptococcus pneumoniae infection is a major cause of disease worldwide. The organism commonly colonises the nasopharynx, and can spread to the respiratory tract, causing a wide range of diseases ranging from mild, such as otits media and sinusitis, to severe—such as pneumonia, septicaemia and meningitis.[1] The burden of disease is greatest in infants and the elderly. The 23-valent pneumococcal polysaccharide vaccine (23vPPV) was first recommended in Australia in 1994 for certain high risk groups, and a 7-valent pneumococcal conjugate vaccine (7vPCV) program with a 3+0 schedule (i.e. 2, 4 and 6 month schedule without a conjugate vaccine booster) was first funded on the National Immunisation Program (NIP) for Aboriginal and Torres Strait Islander infants in mid-2001 with those in areas of very high incidence also funded for a 23vPPV booster at 18–24 months.[2] The 23vPPV is now funded nationally for all individuals aged 65 years or older and Aboriginal and Torres Strait Islander adults aged 50 years or older and in 2005 the conjugate vaccine was funded for all infants.[2] Enhanced surveillance is carried out for invasive pneumococcal disease (IPD), which has been nationally notifiable in Australia since 2001 with some states and territories having collected data from earlier years. Surveillance reports have been published in *Communicable Diseases Intelligence* for each year from 2002 to 2006.[3–7] This report focuses on the years 2007 and 2008.

Methods and materials

Data collection

IPD has been a nationally notifiable disease in Australia since 2001, with complete data being collected in all states and territories from 2002. To varying degrees across jurisdictions, medical practitioners, laboratories and other health professionals are legally required to report cases of IPD to state and territory health authorities. Information on notified cases is collated by state and territory jurisdictions under jurisdictional public health legislation. The *National Health Security Act 2007* provides the legislative basis for, and authorises the exchange of, health information between jurisdictions and the Commonwealth.[8] The Act provides for the establishment of the National Notifiable Diseases List, which specifies the diseases about which personal information can be provided.[9] IPD is one of the diseases specified in this list. De-identified data on notified cases are reported by jurisdictions electronically to the National Notifiable Diseases Surveillance System (NNDSS), managed by the Australian Government Department of Health and Ageing. National data standards ensure consistency and comparability of data

collected across Australia. Core data are collected for all notified cases such as serotype, sex, age, Indigenous status and vaccination. Enhanced data are collected for notified cases of IPD, including information relating to cases' risk factors, clinical diagnostics and antibiotic susceptibilities. Table 1 outlines the population subgroups for which enhanced data are reported in each jurisdiction.

The Enhanced Invasive Pneumococcal Disease Surveillance Working Group (EIPDSWG), a subcommittee of the Communicable Diseases Network Australia (CDNA), is responsible for finalising IPD data reported to the NNDSS. Data presented in this report were analysed by date of diagnosis. For the purposes of the NNDSS, the date of diagnosis 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. Cases presented and analysed in this report had a date of diagnosis between 1 January 2007 and 31 December 2008, inclusive.

Data presented in this report represent a point in time analysis of cases of IPD notified to the NNDSS. Analyses of these cases were finalised in May 2011. Due to the dynamic nature of the NNDSS, data in this report may vary from data reported in other NNDSS reports and reports of IPD notifications at the jurisdictional level. Notification rates were calculated using the mid-year estimated resident populations supplied by the Australian Bureau of Statistics.[10]

Case definition

Cases of IPD were determined to be notified for national notification according to the CDNA case definition of IPD.[11] A confirmed case was defined as the isolation from or detection by nucleic acid test (NAT) in blood, cerebrospinal fluid (CSF) or other sterile site of *S. pneumoniae*.

Indigenous status

Cases of IPD were reported indicating the Indigenous status of the individual. The definition of an Aboriginal or Torres Strait Islander person within the NNDSS aligns with the Commonwealth definition, that is, an Aboriginal or Torres Strait Islander is determined by descent, self-identification and community acceptance.

The small number of cases reported without an Indigenous status were excluded from analyses relating to Indigenous status in this report.

Vaccination

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, older Australians and Aboriginal and Torres Strait Islander people. Vaccination with 7vPCV was added to the NIP schedule for Indigenous and medically at-risk children in 2001 and for all children up to 2 years of age from January 2005. A primary series of 7vPCV is given at 2, 4 and 6 months of age, with medically at-risk children requiring a 4th dose of 7vPCV at 12 months of age and a booster dose of 23vPPV at 4 years of age. A 23vPPV booster was also recommended for Indigenous children at 18–24 months in the Northern Territory, Queensland, South Australia and Western Australia. Of note, subsequent to this study period, higher valency vaccines, such as Prevenar13®, have replaced 7vPCV throughout Australia.

Since 1999, the 23vPPV has been funded for Indigenous Australians aged 50 years or older and 15–49 years with risk factors, with non-Indigenous Australians aged 65 years eligible to receive the vaccine under the NIP from January 2005. Recommendations for revaccination with 23vPPV vary by age and Indigenous status, current Australian guidelines can be found on the Immunise Australia web site.[ref: http://www.immunise.health.gov.au] A detailed list of recommendations and funding initiatives for pneumococcal vaccinations in Australia is shown in Table 2.

The definitions of fully vaccinated and vaccination validation for determination of vaccine failure in this report are described in Table 3. These definitions are applied to the vaccination fields reported to the NNDSS and are agreed to by the EIPDSWG.

Vaccine coverage data (7vPCV) were provided by the Australian Childhood Immunisation Register

Table 1: Enhanced invasive pneumococcal disease surveillance data collection performed by states and territories in 2007 and 2008

Age group	Jurisdictions
Under 5 years	Australian Capital Territory, New South Wales, Queensland (South Brisbane Public Health Unit only).
Over 50 years	New South Wales.
All ages	Northern Territory, Queensland (except South Brisbane Public Health Unit), Tasmania, South Australia, Victoria, Western Australia.

Table 2: Recommendations and funding initiatives for pneumococcal vaccinations in Australia

Vaccine	23-valent polysaccharide vaccine	7-valent conjugate vaccine
Pneumococcal serotypes	1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19A, 19F, 20, 22F, 23F, 33F	4, 6B, 9V, 14, 18C, 19F, 23F
Target populations	All individuals aged 65 years or over to receive a single dose of vaccine with a booster 5 years later* Aboriginal and Torres Strait Islander people aged 50 years or over to receive a single dose of vaccine with a booster 5 years later† Aboriginal and Torres Strait Islander people aged between 15 and 49 years at high risk to receive a single dose of vaccine and appropriate booster(s)‡ Children who have underlying chronic illnesses predisposing to invasive pneumococcal disease (including asplenia and immunocompromised)§ Immunocompetent individuals with chronic illness including chronic cardiac, renal or pulmonary disease, diabetes and alcohol-related problems Individuals with cerebrospinal fluid leaks‖ Tobacco smokers¶ As a booster dose at 18 to 24 months of age following a primary course of 7vPCV in Aboriginal and Torres Strait Islander children in regions of high incidence** As a booster dose at 4 to 5 years of age following a primary course of 7vPCV in children at risk because of predisposing medical conditions**	Children at 2, 4 and 6 months of age†† Children born between 1 January 2003 and 31 December 2004‡‡ Additional booster dose for children in specific high-risk groups**

* Funded in Victoria from 1998. Funded nationally from 2005.

† Targeted funded programs in north Western Australia, Far North Queensland and the Northern Territory from 1995, Funded nationally from 1999.

‡ Funded nationally from 1999. Funded for all children aged 15 years or over in the Northern Territory from 1999.

§ Targeted funded programs for high risk children aged over 2 years in north Western Australia and the Northern Territory from 1986. Recommended nationally for children aged over 2 years (pre-July 2001) and children aged over 5 years from July 2001.

‖ Recommended nationally for children aged over 2 years (pre-July 2001) and children aged over 5 years from July 2001.

¶ Recommended nationally from 2003.

** Funded nationally from July 2001.

†† Funded nationally for Indigenous children from July 2001 and all children from 2005.

‡‡ Funded nationally as a catch-up program in 2005.

Table 3: Definitions of vaccination status and vaccine failure used in this report

Category	Definition
Fully vaccinated	Those that have completed the primary course of the relevant vaccine(s) required for their age, Indigenous status, geographical location and/or other risk factor(s) according to the most recent edition of the *Australian Immunisation Handbook,* at least 2 weeks prior to disease onset with at least 28 days between doses of vaccine. This includes the following; a child that received a vaccine as 'catch up' and therefore does not require a full three dose primary schedule. Providing they have had the number of doses required for the age they were at first dose they should be considered fully vaccinated. NB: A young child who has had all the required doses for their age but is not old enough to have completed the primary course would not be assessed as fully vaccinated.
Vaccination validation	Written confirmation of vaccination through the Australian Childhood Immunisation Register, state or territory immunisation register or health record.
Vaccine failure	A fully vaccinated person (as defined above) with disease due to a serotype found in the corresponding vaccine.

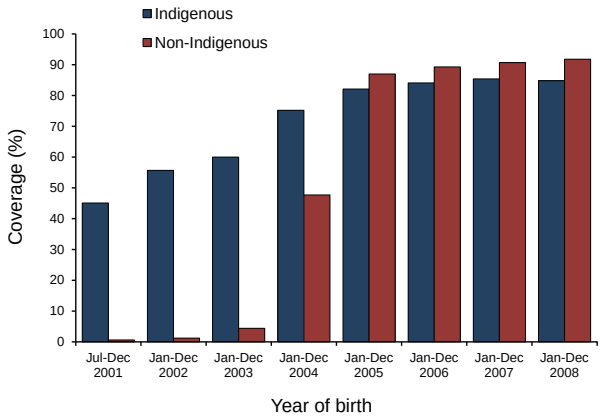
(ACIR). The ACIR records details of vaccinations given to children under the age of 7 years who live in Australia. The ACIR definition of fully vaccinated is a population-based milestone (3 doses by 12 months of age), which differs from the age-appropriate definition used by the EIPDSWG for classification of individuals with IPD.

Results

Vaccination coverage

The proportion of children who are fully vaccinated against pneumococcal disease has increased steadily since 2001 (Figure 1). In 2007, the proportion of children aged 12 months immunised with 3 doses of 7vPCV was 85% in Indigenous children and 91% in non-Indigenous children. In 2008, the proportions were 85% in Indigenous children and 92% in non-Indigenous children.

Figure 1: Proportion of children aged 12 months fully vaccinated with 7vPCV, Australia, 2001 to 2008, by Indigenous status



Source: The Australian Childhood Immunisation Register.

The Australian Childhood Immunisation Register defines fully vaccinated as aged 12 months and immunised with 3 doses of 7vPCV.

Invasive pneumococcal disease notifications

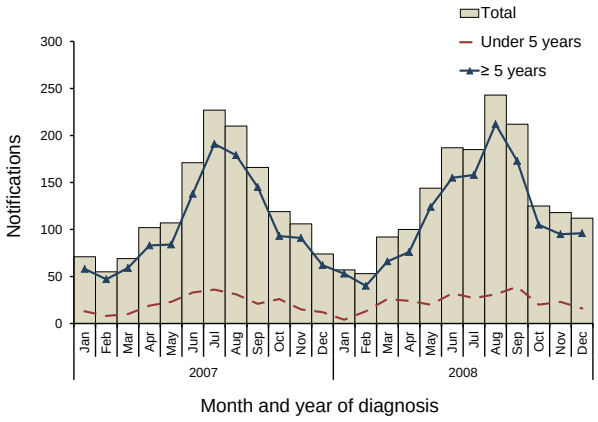
The total number of notifications of IPD in 2007 was 1,477 and in 2008 was 1,628. This represents annual notification rates of 7.0 and 7.6 cases per 100,000 population, respectively.

A summary of the number and rates of notifications by jurisdiction is shown in Table 4. The Northern Territory continued to have the highest notification rate (31.2 per 100,000 population reported in 2007 and 27.3 per 100,000 population in 2008) while the lowest notification rate for 2007 was reported in Victoria (5.4 per 100,000 population) and for 2008 in the Australian Capital Territory (5.5 per 100,000 population).

When notification rates of IPD were examined by geographical distribution, variation within states and territories was apparent (Map).

The number of cases of IPD was greatest in winter months with the peak number of notifications for 2007 reported in July (n=227) and for 2008 in August (n=243). The effect of season was more evident in the distribution of cases aged 5 years or over compared with younger children (Figure 2).

Figure 2: Notifications of invasive pneumococcal disease, Australia, 2007 and 2008, by month of diagnosis and age group

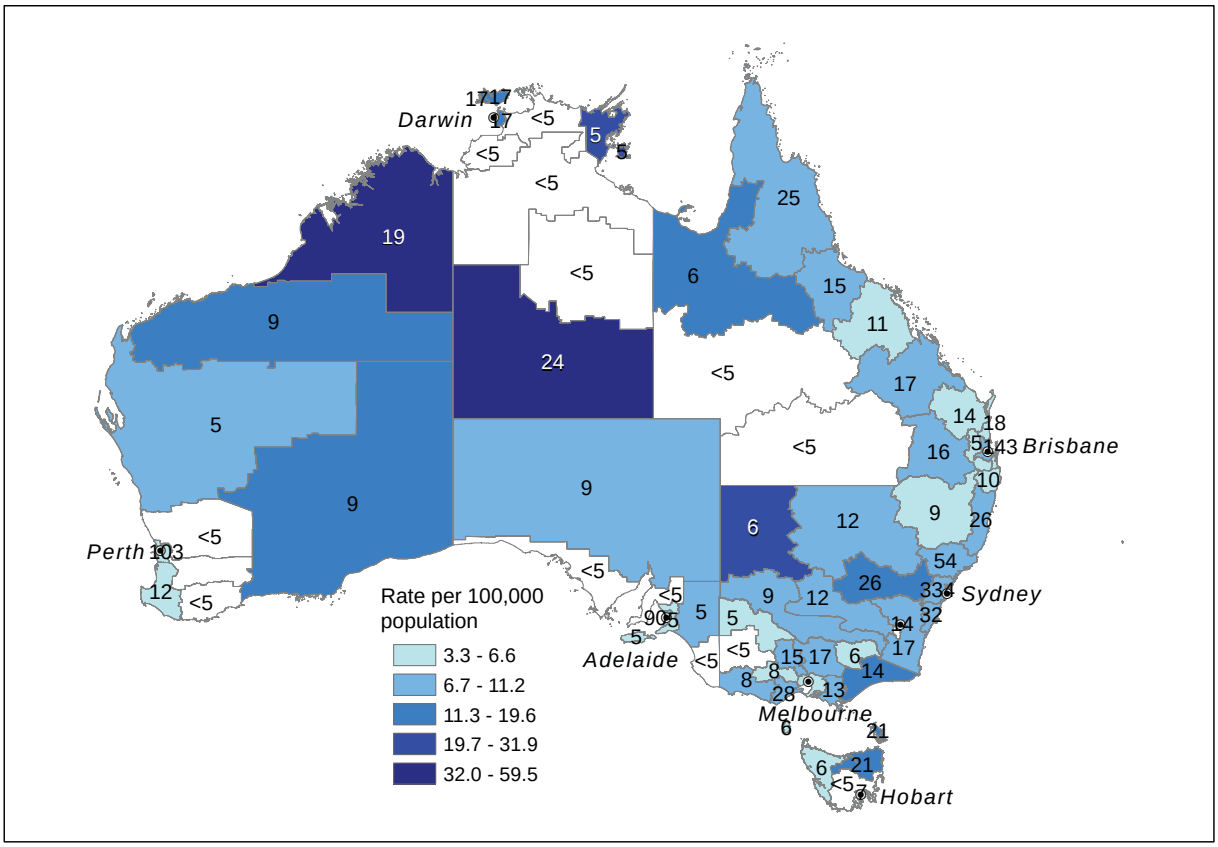


Invasive pneumococcal disease by age and sex

The overall male to female ratio in both 2007 and 2008 was 1.3:1 (Table 4). In almost all age groups there was a greater notification rate of IPD in males than females (Figure 3). The highest rates in 2007 and 2008 combined were among the elderly aged 85 years or over (35.5 per 100,000 population) and in children aged 1 year (32.8 per 100,000 population, Figure 3).

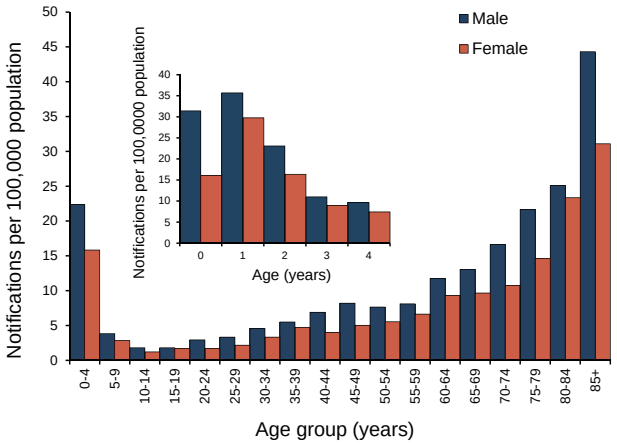
An examination of rates of IPD in different age groups from 2002 to 2008 is shown in Figure 4. There was a small increase in the rate of IPD in children aged under 2 years in 2007 and 2008 (27.5 per 100,000 population and 29.3 per 100,000 population respectively) when compared with 2006 (24.3 per 100,000 population). However, overall the rate maintains the large decrease experienced in this age group as a result of the introduction of the universal 7vPCV immunisation program in 2005. Prior to the vaccination program the notification rate in this age group was close to 100 cases per 100,000 population.

Map: Notification rates for invasive pneumococcal disease, Australia, 2008, by Statistical Division of residence



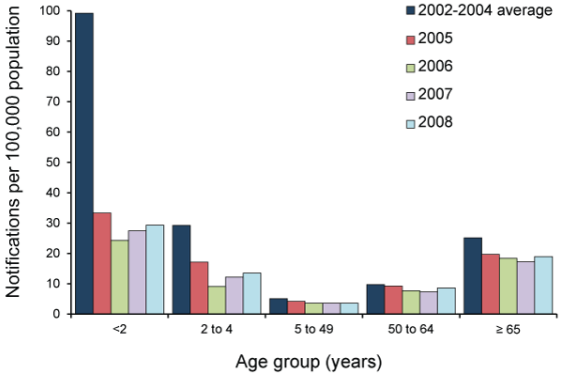
Data point labels represent the number of notifications. Notification rates in geographic areas where estimated residential population and case numbers are small should be interpreted with caution.

Figure 3: Notification rates for invasive pneumococcal disease, Australia, 2007 and 2008, by age group and sex



The rate of IPD in adults aged 65 years or over continued to decline in 2007 (18.4 per 100,000 population) when compared with 2006 (17.2 per 100,000 population), and there was an increase experienced in 2008 (19.0 per 100,000 population).

Figure 4: Notification rate for invasive pneumococcal disease, Australia, 2002 to 2008, by age group



Invasive pneumococcal disease in Aboriginal and Torres Strait Islander people

Indigenous status was reported in 88% of notifications in 2007 (1,301/1,477) and 2008 (1,435/1,628) (Table 4). This level of completeness in reporting Indigenous status was improved in the target popu-

Table 4: Notifications, rates and demographics of invasive pneumococcal disease cases, Australia, 2007 and 2008, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
2007									
All cases									
Notifications	35	523	67	318	91	31	280	132	1,477
Rate (notifications per 100,000 population)	10.3	7.6	31.2	7.6	5.7	6.3	5.4	6.2	7.0
Male:female ratio	0.8:1	1.2:1	1.2:1	1.3:1	1.4:1	1.2:1	1.3:1	1.4:1	1.3:1
Indigenous	0	18	56	51	7	0	5	32	169
Non-Indigenous	35	404	11	218	84	26	254	100	1,132
Unknown	0	101	0	49	0	5	21	0	176
Notifications aged <5 years									
Total	9	83	16	49	21	2	38	29	247
Indigenous	0	9	14	8	2	0	1	3	37
Non-Indigenous	9	72	2	34	19	1	35	26	198
Unknown	0	2	0	7	0	1	2	0	12
Notifications aged 5 to 64 years									
Total	10	261	46	172	47	14	133	71	754
Indigenous	0	7	39	37	5	0	4	28	120
Non-Indigenous	10	157	7	110	42	11	115	43	495
Unknown	0	97	0	25	0	3	14	0	139
Notifications ≥ 65 years									
Total	16	179	5	97	23	15	109	32	476
Indigenous	0	2	3	6	0	0	0	1	12
Non-Indigenous	16	175	2	74	23	14	104	31	439
Unknown	0	2	0	17	0	1	5	0	25
2008									
All cases									
Notifications	19	548	60	327	119	39	355	161	1,628
Rate (notifications per 100,000 population)	5.5	7.8	27.3	7.6	7.4	7.8	6.7	7.4	7.6
Male:female ratio	0.9:1	1.2:1	2.2:1	1.1:1	1.3:1	0.8:1	1.4:1	1.7:1	1.3:1
Indigenous	0	14	38	45	6	1	6	42	152
Non-Indigenous	19	429	22	230	112	36	316	119	1,283
Unknown	0	105	0	52	1	2	33	0	193
Notifications aged <5 years									
Total	5	96	15	60	13	10	46	30	275
Indigenous	0	5	12	13	0	0	2	6	38
Non-Indigenous	5	89	3	37	13	10	39	24	220
Unknown	0	2	0	10	0	0	5	0	17
Notifications aged 5 to 64 years									
Total	10	247	43	173	61	15	168	99	816
Indigenous	0	4	25	27	6	1	3	35	101
Non-Indigenous	10	143	18	120	54	12	145	64	566
Unknown	0	100	0	26	1	2	20	0	149
Notifications ≥ 65 years									
Total	4	205	2	94	45	14	141	32	537
Indigenous	0	5	1	5	0	0	1	1	13
Non-Indigenous	4	197	1	73	45	14	132	31	497
Unknown	0	3	0	16	0	0	8	0	27

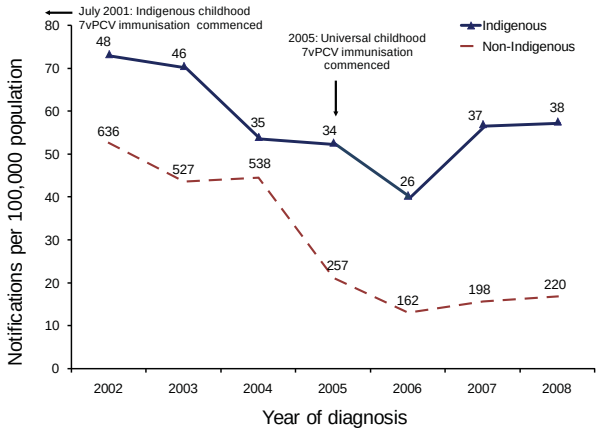
lations of children aged less than 5 years and adults aged 65 years or over, at close to 95% in each of these subgroups in 2007 and 2008.

In 2007, there were 169 cases of IPD reported among Indigenous people (11% of all cases). This represents a rate of 32.0 cases per 100,000 population—a rate almost 6 times that seen in the non-Indigenous population (5.5 per 100,000 population). In 2008, there were 152 cases of IPD reported among Indigenous people (9% of all cases), representing a rate of 28.2 cases per 100,000 population—almost 5 times that seen in the non-Indigenous population (6.1 per 100,000 population). Further analyses of the Indigenous population group are provided throughout this report.

Invasive pneumococcal disease in children

Figure 5 shows the rate of IPD in Indigenous and non-Indigenous children aged less than 5 years since 2002. Rates in Indigenous children in this age group decreased from 73.4 cases per 100,000 population in 2002 (n=48) to 57.6 cases per 100,000 population (n=38) in 2008, a decrease of 21%. In non-Indigenous children aged less than 5 years the rate decreased from 52.5 cases per 100,000 population in 2002 (n=636) to 16.8 cases per 100,000 population (n=220), a decrease of 68%.

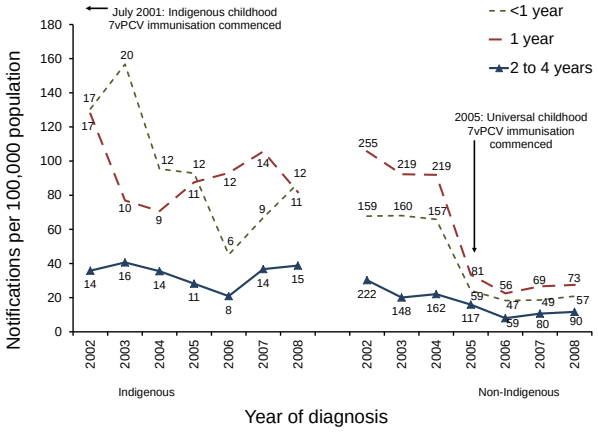
Figure 5: Notification rate for invasive pneumococcal disease in children aged less than 5 years, Australia, 2002 to 2008, by Indigenous status



Data point labels represent the number of notifications.

Figure 6 shows the rates of IPD in children aged less than 5 years by Indigenous status broken down into smaller age groups. The rate of IPD in Indigenous children fluctuated over the period due to the smaller number of notifications from a smaller population. In Indigenous children aged less than 1 year rates decreased from 130.5 cases per 100,000 population (n=17) in 2002 to 86.9 cases per 100,000 population in 2008 (n=12). In Indigenous children aged 1 year, over the same period, the IPD rate decreased from 127.7 cases per 100,000 population (n=17) to 81.8 cases per 100,000 population (n=11), while the 2–<5 years age group increased from 35.8 cases per 100,000 population (n=14) to 38.8 cases per 100,000 population (n=15).

Figure 6: Notification rate for invasive pneumococcal disease in children aged less than 5 years, Australia, 2002 to 2008, by Indigenous status and age group



Data point labels represent the number of notifications.

In non-Indigenous children less variability was seen. A clear and consistent decrease reflected the implementation of the universal 7vPCV immunisation program, introduced in 2005. The IPD rate in non-Indigenous children aged less than 1 year decreased from 67.7 cases per 100,000 population (n=159) in 2002 to 20.8 cases per 100,000 population (n=57) in 2008. In non-Indigenous children aged 1 year, over the same period, the rate decreased from 105.7 cases per 100,000 population (n=255) to 27.5 cases per 100,000 population (n=73), and in children aged 2 to less than 5 years from 30.2 cases per 100,000 population (n=222) to 11.7 cases per 100,000 population (n=90).

Mortality of invasive pneumococcal disease cases

Table 5 shows data on mortality of IPD cases reported in 2007 and 2008. Mortality data were available for 67% (985/1,477) of notifications in 2007 and 72% (1,165/1,628) notifications in 2008. Of these notifications, there were 120 deaths associated with IPD in Australia in 2007 and 113 deaths in 2008.

Overall, case fatality rates in notifications reported as non-Indigenous were higher than in those reported

as Indigenous. In 2007, death associated with IPD was reported in 9 Indigenous cases (CFR=5.3%) and in 106 non-Indigenous cases (CFR=9.4%). In 2008, death was reported overall in 6 Indigenous cases (CFR=3.9%) and in 103 non-Indigenous cases (CFR=8.0%).

In those aged less than 5 years there were 7 deaths associated with IPD in 2007 and 4 deaths in 2008, giving case fatality rates of 2.8% and 1.5%, respectively. One of those deaths was reported in an Indigenous child, occurring in 2008. Further details, including serotype and vaccination history, of the eleven children aged less than 5 years whose deaths were associated with IPD are shown in Table 6.

In the 65 years and older age group there were 63 deaths due to IPD in 2007 and 77 deaths in 2008, giving case fatality rates of 13.2% and 14.3%, respectively. Two of those deaths were reported as Indigenous, both in 2008.

Jurisdictional specific case fatality rates have not been presented in Table 5 for those jurisdictions where completeness of data was less than 50%. Rates shown should be interpreted with caution given the proportion of cases without mortality data reported to the NNDSS, as well as the variability across jurisdictions to report death as primary and secondary causes.

Risk factors for invasive pneumococcal disease

Risk factor data were provided for 60% (1,854/3,105) of cases reported in 2007 and 2008. Of the cases with risk factor data reported, 1,723 cases reported at least 1 risk factor and 131 cases reported that no risk factors were identified. Table 7 shows risk factors in children aged less than 5 years, Indigenous people aged less than 50 years and non-Indigenous people aged greater than 65 years for 2007 and 2008 combined.

In children aged less than 5 years the most frequently reported risk factor in the Indigenous

Table 5: Deaths and case fatality rates* for invasive pneumococcal disease, Australia, 2007 and 2008, by age group, Indigenous status and state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
2007									
Notifications	35	523	67	318	91	31	280	132	1,477
Deaths	0	58	4	6	7	3	19	23	120
Completeness of mortality data (%)	94.3	71.7	100.0	2.5*	75.8	87.1	97.9	100.0	66.7
Deaths in under 5 years	0	0	0	1	1	0	1	4	7
CFR under 5 years	0.0	0.0	0.0	NA	4.8	0.0	2.6	13.8	2.8
Deaths in ≥ 65 years	0	40	1	1	4	1	9	7	63
CRF ≥ 65 years	0.0	22.3	20.0	NA	17.4	6.7	8.3	21.9	13.2
Deaths in Indigenous people	0	0	2	0	1	0	0	6	9
Deaths in non-Indigenous people	0	55	2	6	6	3	17	17	106
2008									
Notifications	19	548	60	327	119	39	355	161	1,628
Deaths	0	48	3	10	14	3	21	14	113
Completeness of mortality data (%)	94.7	77.7	100.0	34.3*	11.8*	69.2	97.7	100.0	71.6
Deaths in under 5 years	0	0	0	1	0	0	1	2	4
CFR under 5 years	0.0	0.0	0.0	NA	NA	0.0	2.2	6.7	1.5
Deaths in ≥ 65 years	0	37	0	7	9	2	16	6	77
CFR ≥ 65 years	0.0	18.0	0.0	NA	NA	14.3	11.3	18.8	14.3
Deaths in Indigenous people	0	0	1	2	0	0	1	2	6
Deaths in non-Indigenous people	0	47	0	8	14	3	19	12	103

* Jurisdictional specific case fatality rates (CFRs) have not been presented for those jurisdictions where completeness of data was less than 50%, denoted as 'NA'. Rates shown should be interpreted with caution given the proportion of cases without mortality data reported to the NNDSS, as well as the variability across jurisdictions to report death as primary and secondary causes.

population was chronic illness, (28% of cases with a risk factor reported), while childcare attendee was the most frequently reported risk factor in the non-Indigenous population (28% of cases with risk factor data reported). Among the adult population groups, Indigenous cases aged greater than 50 years and non-Indigenous cases aged greater than 65 years, chronic disease was the most frequently reported risk factor, with 82% (46/56) and 79% (621/791) reported with this risk factor, respectively.

Pneumococcal serotypes causing invasive disease

Pneumococcal serotypes were identified for 90% (1,336/1,477) of all notified cases in 2007 and for 92% (1,495/1,628) in 2008. Table 8 shows the number and proportion of IPD cases due to serotypes covered by the various pneumococcal vaccines.

Of all the cases reported with a serotype in 2007 and 2008, 24% (678/2,831) were due to serotypes covered by the 7vPCV; this ranged from 10% (2/21) of Indigenous cases aged 5–14 years to 35% (27/78) of non-Indigenous cases in the same age

Table 6: Characteristics of deaths from invasive pneumococcal disease in children aged less than 5 years of age, Australia, 2007 and 2008

Patient	Year of diagnosis	Sex	Age (months)	Indigenous status	Serotype	Doses of 7vPCV	Risk factors
Deaths potentially preventable by 7vPCV							
1	2007	Male	11	non-Indigenous	19F	Unknown	No risk factors
Deaths not preventable by 7vPCV							
2	2007	Female	19	non-Indigenous	11A	3	Premature child care attendee
3	2007	Male	29	non-Indigenous	11A	Unknown	Unknown
4	2007	Male	19	non-Indigenous	15C	3	No risk factors
5	2007	Female	22	non-Indigenous	15C	3	No risk factors
6	2007	Male	5	non-Indigenous	18A	2	No risk factors
7	2007	Male	29	non-Indigenous	19A	3	Child care attendee
8	2008	Female	0	Indigenous	16F	0	Premature
9	2008	Female	0	non-Indigenous	19A	0	Congenital abnormality
10	2008	Female	23	non-Indigenous	35B	3	Premature congenital abnormality
11	2008	Male	5	non-Indigenous	Not typed	1	Unknown

Table 7: Risk factors* for invasive pneumococcal disease population sub-groups, Australia, 2007 and 2008

Risk factor	Children aged less than 5 years		Indigenous aged more than 50 years (n=64)	Non-Indigenous aged more than 65 years (n=936)
	Indigenous (n=75)	Non-Indigenous (n=418)		
Premature birth	10	33	NA	NA
Congenital or chromosomal abnormality	7	20	NA	NA
Anatomic or functional asplenia	1	0	1	13
Immunocompromised	2	8	16	199
Chronic illness	11	26	46	621
Childcare attendee	4	56	NA	NA
Previous episode of IPD	1	0	3	6
Other†	23	17	18	163
No risk factor identified	5	59	0	31
Risk factor data not reported	36	218	8	145
Total cases with risk factor data	39	200	56	791

* Case may be reported with more than one risk factor.

† Other risk factors include but are not limited to exposure to smoke, asthma and previous pneumonia.

NA Not applicable

group. The 3 additional serotypes (1, 5 and 7F) covered by the 10-valent pneumococcal conjugate vaccine (10vPCV) accounted for an additional 7% (195/2,831) of cases in 2007 and 2008; this ranged from 3% (22/868) of non-Indigenous adults aged 65 years or older and non-Indigenous children aged less than 5 years to 38% (8/21) of Indigenous cases aged 5–14 years. The 3 additional serotypes (3, 6A and 19A) covered by the 13-valent pneumococcal conjugate vaccine (13vPCV) in addition to the 10vPCV serotypes accounted for an additional 33% (936/2,831) of cases; this ranged from 0% (0/21) of Indigenous cases aged 5–14 years to 52% (200/381) of non-Indigenous cases aged less than 5 years.

Table 8: Number and proportion of invasive pneumococcal disease cases, Australia, 2007 and 2008, by pneumococcal vaccine serotypes

Age group	Vaccine type	Indigenous			Non-Indigenous		
		Number	%	Cumulative (%)	Number	%	Cumulative (%)
<5 years	7vPCV	7	9.7	9.7	47	12.3	12.3
	10vPCV (non-7vPCV)	6	8.3	18.1	10	2.6	15.0
	13vPCV (non-10vPCV)	13	18.1	36.1	200	52.5	67.5
	Non-conjugate serotypes	46	63.9	100.0	124	32.5	100.0
	Total	72	100.0		381	100.0	
	23vPPV (non-7vPCV)	43	59.7		262	68.8	
5–14 years	7vPCV	2	9.5	9.5	27	34.6	34.6
	10vPCV (non-7vPCV)	8	38.1	47.6	8	10.3	44.9
	13vPCV (non-10vPCV)	0	0.0	47.6	19	24.4	69.2
	Non-conjugate serotypes	11	52.4	100.0	24	30.8	100.0
	Total	21	100.0		78	100.0	
	23vPPV (non-7vPCV)	15	71.4		41	52.6	
15–49 years	7vPCV	16	11.0	11.0	123	29.2	29.2
	10vPCV (non-7vPCV)	27	18.5	29.5	42	10.0	39.2
	13vPCV (non-10vPCV)	30	20.5	50.0	124	29.5	68.6
	Non-conjugate serotypes	73	50.0	100.0	132	31.4	100.0
	Total	146	100.0		421	100.0	
	23vPPV (non-7vPCV)	87	59.6		258	61.3	
50–64 years	7vPCV	5	14.3	14.3	117	24.8	24.8
	10vPCV (non-7vPCV)	5	14.3	28.6	31	6.6	31.4
	13vPCV (non-10vPCV)	8	22.9	51.4	157	33.3	64.8
	Non-conjugate serotypes	17	48.6	100.0	166	35.2	100.0
	Total	35	100.0		471	100.0	
	23vPPV (non-7vPCV)	24	68.6		272	57.7	
>65 years	7vPCV	4	16.7	16.7	240	27.6	27.6
	10vPCV (non-7vPCV)	3	12.5	29.2	22	2.5	30.2
	13vPCV (non-10vPCV)	6	25.0	54.2	271	31.2	61.4
	Non-conjugate serotypes	11	45.8	100.0	335	38.6	100.0
	Total	24	100.0		868	100.0	
	23vPPV (non-7vPCV)	13	54.2		414	47.7	
Total	7vPCV	34	11.4	11.4	554	25.0	25.0
	10vPCV (non-7vPCV)	49	16.4	27.9	113	5.1	30.1
	13vPCV (non-10vPCV)	57	19.1	47.0	771	34.7	64.8
	Non-conjugate serotypes	158	53.0	100.0	781	35.2	100.0
	Total	298	100.0		2,219	100.0	
	23vPPV (non-7vPCV)	182	61.1		1,247	56.2	

Notifications with Indigenous status and/or serotype reported as unknown are excluded.

There are an additional 16 serotypes covered by the 23vPPV, over the 7 covered by the 7vPCV. Of all the cases reported with a serotype in 2007 and 2008, 57% (1618/2831) of all cases were due to these additional 16 serotypes. This ranged from 48% (414/868) of non-Indigenous adults aged 65 years or older to 71% (15/21) of Indigenous cases aged 5–14 years.

The notification rate of IPD due to 7vPCV serotypes fell considerably between 2002 and 2008 in children aged less than 5 years (Figure 7). The rate decreased by 95% in cases aged less than 2 years (70.9 to 3.4 per 100,000 population) and similarly decreased by 95% in cases aged 2–<5 years (27.3 to 1.4 per 100,000 population). The notification rate of IPD in adults aged 65 years or over decreased by 74% from 2002 to 2008 (14.2 to 3.7 per 100,000 population).

Figure 7: Notification rate for invasive pneumococcal disease caused by 7vPCV serotypes, Australia, 2002 to 2008, by age group

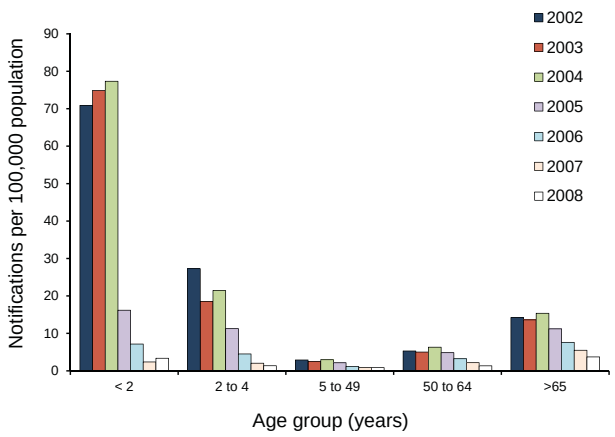
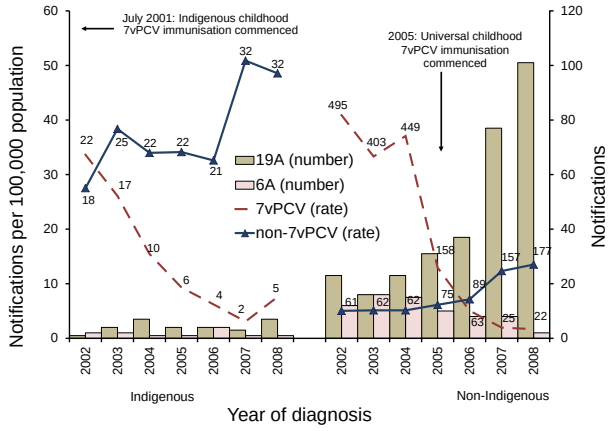


Figure 8 shows rates of IPD caused by 7vPCV serotypes in Indigenous and non-Indigenous children aged less than 5 years since 2002. Disease due to 7vPCV serotypes in Indigenous children has declined from 33.6 cases per 100,000 population in 2002 (n=22) to 7.6 cases per 100,000 population in 2008 (n=5), a decrease of 77%. Similarly in non-Indigenous children over the same period, rates fell from 40.9 (n=495) to 1.7 cases per 100,000 population (n=22), a decrease of 96%.

Rates of disease caused by non-7vPCV serotypes over the same period increased for both Indigenous and non-Indigenous children. In Indigenous children aged less than 5 years the rate increased from 27.5 to 48.5 cases per 100,000 population, an increase of 76%, and in non-Indigenous children from 5.0 to 13.5 cases per 100,000 population, an increase of 168%. The number of cases due to serotype 19A increased over this period in children aged less than 5 years, with 54% (118/221) of disease caused by non-7vPCV types due to this serotype in 2008. The number of cases due to serotype 19A in non-Indigenous children increased from 23 in 2002 to 101 in 2008. The number of cases due to serotype 6A decreased over this period in this age group, with 1% (3/221) of disease caused by non-7vPCV types due to serotype 6A in 2008.

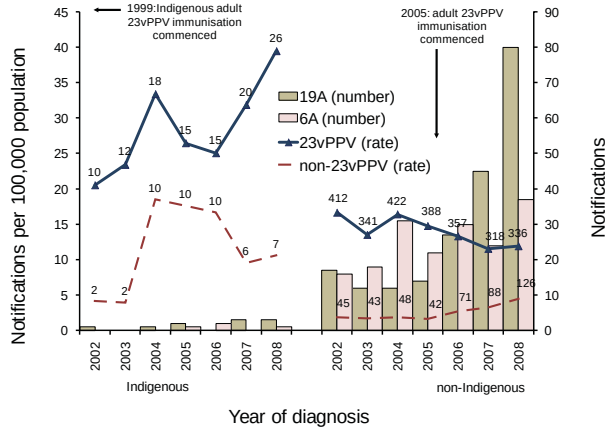
Figure 9 shows rates of IPD caused by 23vPPV serotypes in Indigenous adults aged 50 years or older and non-Indigenous adults aged 65 years or older. In Indigenous adults the rate of disease caused by 23vPPV serotypes increased from 20.5 cases per 100,000 population (n=10) in 2002 to 39.5 cases

Figure 8: Notifications and rates for 7vPCV and non-7vPCV serotypes causing cases of invasive pneumococcal disease in children aged less than 5 years, Australia, 2002 to 2008, by Indigenous status



Data point labels represent the number of notifications.

Figure 9: Notification rates for 23vPPV and non-23vPPV serotypes causing cases of invasive pneumococcal disease in Indigenous adults (aged 50 years or over) and non-Indigenous adults (aged 65 years or over), Australia, 2002 to 2008



Data point labels represent the number of notifications.

per 100,000 population (n=26) in 2008, an increase of 92%. Conversely, in non-Indigenous adults the rate decreased from 16.7 cases per 100,000 population (n=412) to 11.9 cases per 100,000 population (n=336) over the same period, a decrease of 28%. The number of cases due to serotypes 19A in non-Indigenous adults increased over this period, from 17 cases reported in 2002 to 80 cases in 2008. In 2002, serotype 19A represented 4% of all disease due to serotypes covered by the 23vPPV in non-Indigenous adults. This increased to 24% in 2008.

The rate of disease caused by non-23vPPV serotypes increased from 2002 to 2008 for both Indigenous (4.1 to 10.6 cases per 100,000 population respectively) and non-Indigenous adults (1.8 to 4.5 cases per 100,000 population, respectively). The number of cases due to serotype 6A increased over this period in non-Indigenous adults. In 2008, Serotype 6A represented 29% (37/126) of all disease in non-Indigenous adults that was caused by serotypes not covered by 23vPPV.

Vaccine failures

Table 9 shows cases of IPD due to 7vPCV serotypes in children aged less than 5 years who were fully vaccinated with 7vPCV as defined in Table 3. In 2007 and 2008 a total of 25 children who were considered fully vaccinated were notified with disease due to 7vPCV serotypes. Of these cases, 80% (n=20) were reported as non-Indigenous and 12% (n=3) were reported as Indigenous. Serotype 19F was reported in 64% (n=16) of these cases, with 6B the next most frequently reported serotype at 20% (n=5).

Antibiotic resistance

Penicillin and ceftriaxone/cefotaxime susceptibility data were analysed only from jurisdictions that reported susceptibility data for more than 50% of cases. Penicillin susceptibility completeness was suitable for reporting in both 2007 and 2008 for all jurisdictions. However, ceftriaxone/cefotaxime susceptibility completeness was suitable for report-

Table 9: Characteristics of 7vPCV vaccine failures in children aged less than 5 years, Australia, 2007 and 2008

Case	Year of diagnosis	Age (months)	Indigenous status	Serotype	Doses of 7vPCV	Doses of 23vPPV	Clinical category	Risk factors
1	2007	11	non-Indigenous	19F	3	0	Bacteraemia	yes
2	2007	14	non-Indigenous	6B	3	0	Pneumonia	unknown
3	2007	17	non-Indigenous	19F	3	0	Pneumonia	no
4	2007	24	non-Indigenous	19F	3	0	Pneumonia	no
5	2007	28	non-Indigenous	9V	3	0	Pneumonia	yes
6	2007	30	non-Indigenous	6B	3	0	Bacteraemia	no
7	2007	31	non-Indigenous	19F	3	0	Pneumonia	no
8	2007	34	non-Indigenous	19F	3	0	Pneumonia	yes
9	2007	38	non-Indigenous	19F	2	0	Meningitis	no
10	2007	38	non-Indigenous	19F	3	0	Bacteraemia	no
11	2007	58	Indigenous	19F	3	1	Bacteraemia	yes
12	2008	13	non-Indigenous	6B	3	0	Septic arthritis	no
13	2008	16	non-Indigenous	19F	3	0	Bacteraemia	no
14	2008	16	unknown	19F	3	0	Pneumonia	no
15	2008	16	non-Indigenous	4	3	0	Meningitis	no
16	2008	16	Indigenous	19F	3	0	Bacteraemia	unknown
17	2008	20	non-Indigenous	19F	3	0	Bacteraemia	yes
18	2008	22	non-Indigenous	19F	3	0	Pneumonia	no
19	2008	23	unknown	19F	3	0	Pneumonia	no
20	2008	25	non-Indigenous	6B	3	0	Bacteraemia	yes
21	2008	26	Indigenous	23F	3	1	Bacteraemia	yes
22	2008	30	non-Indigenous	19F	3	0	Pneumonia	no
23	2008	42	non-Indigenous	19F	3	0	Bacteraemia	no
24	2008	54	non-Indigenous	18C	3	0	Bacteraemia	yes
25	2008	55	non-Indigenous	6B	2	0	Unknown	yes

ing in both 2007 and 2008 in the Australian Capital Territory, New South Wales, the Northern Territory, Tasmania, Victoria and Western Australia.

In 2007, penicillin susceptibility was reported in 84% (1,246/1477) of all IPD cases (Table 10). Ceftriaxone/cefotaxime susceptibility was reported in 78% (834/1,068) of all analysed cases. A similar level of testing was reported in 2008, with 85% (1,379/1,628) of isolates tested for penicillin and 77% (910/1,182) for ceftriaxone/cefotaxime susceptibility.

In 2007 and 2008, 11% (140/1,246 and 153/1,379 respectively) of isolates with reported penicillin susceptibility had reduced susceptibility to penicillin, which was the same proportion as 2006 (11%, 143/1,351). Of the isolates in 2007 with reduced susceptibility to penicillin, 137 were serotyped with 50% (68/137) of these cases due to a serotype in the 7vPCV and 91% (125/137) of these cases due to a serotype in the 23vPPV. Of the isolates in 2008 with reduced susceptibility to penicillin, 148 were serotyped, with 36% (54/148) of these cases due to a serotype in the 7vPCV and 93% (137/148) due to a serotype in the 23vPPV. Of the isolates with reduced susceptibility to penicillin in 2007, 53 were serotype 19A, 42 were 9V, and 12 were 19F, accounting for 78% (107/137) of isolates with reduced penicillin susceptibility and with a known serotype. Of the isolates with reduced susceptibility to penicillin in 2008, 74 were serotype 19A, 17 were 9V, and 17 were 19F, accounting for 73% (108/148) of isolates with reduced penicillin susceptibility and with a known serotype.

In 2008, 2% (16/910) of isolates with reported ceftriaxone/cefotaxime susceptibility testing had reduced susceptibility to ceftriaxone/cefotaxime, which was lower than in 2007 (3%, 25/834) and in

Table 10: *Streptococcus pneumoniae* susceptibility to penicillin and ceftriaxone/cefotaxime,* for selected states and territories, 2007 and 2008

		9V	19F	All 7vPCV serotypes	19A	All 23vPPV serotypes	Not typed	All Isolates
2007								
Penicillin	Resistant	8	9	21	6	27	0	29
	Intermediate	34	3	47	47	98	3	111
	Sensitive	16	39	243	145	825	62	1106
	Total tested	58	51	311	198	950	65	1246
	Total isolates with reduced susceptibility (%)	42 (72%)	12 (24%)	68 (22%)	53 (27%)	125 (13%)	3 (5%)	140 (11%)
Ceftriaxone	Resistant	2	1	3	0	3	0	3
	Intermediate	10	6	18	4	22	0	22
	Sensitive	28	28	190	136	618	44	809
	Total tested	40	35	211	140	643	44	834
	Total isolates with reduced susceptibility (%)	12 (30%)	7 (20%)	21 (10%)	4 (3%)	25 (4%)	0 (0%)	25 (3%)
2008								
Penicillin	Resistant	3	9	18	9	28	1	31
	Intermediate	14	8	36	65	109	4	122
	Sensitive	15	31	201	213	886	68	1226
	Total tested	32	48	255	287	1023	73	1379
	Total isolates with reduced susceptibility (%)	17 (53%)	17 (35%)	54 (21%)	74 (26%)	137 (13%)	5 (7%)	153 (11%)
Ceftriaxone	Resistant	1	1	4	2	6	0	6
	Intermediate	2	1	4	2	8	0	10
	Sensitive	21	32	163	178	657	43	894
	Total tested	24	34	171	182	671	43	910
	Total isolates with reduced susceptibility (%)	3 (13%)	2 (6%)	8 (5%)	4 (2%)	14 (2%)	0 (0%)	16 (2%)

* Susceptibility data are restricted to jurisdictions with completeness suitable for reporting, that is greater than 50% completeness. Penicillin susceptibility completeness was suitable for reporting in both 2007 and 2008 for all jurisdictions. However, ceftriaxone/cefotaxime susceptibility completeness was suitable for reporting in both 2007 and 2008 in the Australian Capital Territory, New South Wales, the Northern Territory, Tasmania, Victoria and Western Australia.

2006 (3% 30/1046). All of the isolates in 2007 with reduced susceptibility to ceftriaxone/cefotaxime were serotyped with 84% (21/25) due to a serotype in the 7vPCV and 100% (25/25) to a serotype in the 23vPPV. All of the isolates in 2008 with reduced susceptibility to ceftriaxone/cefotaxime were serotyped, with 50% (8/16) due to a serotype in the 7vPCV and 88% (14/16) cases due to a serotype in the 23vPPV.

Discussion

By 2008, the 4th year of a universal funded infant 7vPCV (3+0) vaccination program in Australia, vaccine serotype IPD notification rates in those identified as non-Indigenous decreased in all age groups compared with 2002 levels; most significantly by 96% in children aged less than 5 years. However, rates of disease in non-vaccine serotypes had increased by 168% in this age group, including a 4-fold increase in serotype 19A disease. A similar increase in 19A was also seen in non-Indigenous adults. Combining these two trends, the net change in rates of all-serotype IPD was a 68% decrease in children less than 5 years. Substantial impacts on vaccine-type IPD in vaccinated age groups and herd immunity impacts on adults, as well as moderate levels of serotype replacement, have also been seen in other settings.[12, 13]

For the Aboriginal and Torres Strait Islander population, national pre-vaccination data are not available, as the vaccine was funded for this group from 2001. From 2002 to 2008, in children aged less than 5 years notifications of disease due to 7vPCV serotypes decreased by 77% and non-vaccine serotypes increased by 76%. In Indigenous adults (≥50 years), rates of 23vPPV serotypes increased by 92%. However, the substantial increases in 19A serotype disease seen in non-Indigenous children and adults were not evident in Aboriginal and Torres Strait Islander people. As in the pre-vaccine era, non-7vPCV and non-23vPPV serotypes were a greater proportion of total IPD in Aboriginal and Torres Strait Islanders than for non-Indigenous Australians.[3] Serotype 19A was a greater proportion of IPD in the pre-vaccine era in Aboriginal and Torres Strait Islander people compared with other Australians, and carriage studies have shown 19A to be relatively commonly found in Aboriginal children post-vaccination.[14, 15] Increases in serotype 19A disease in Alaskan Native children resulted in IPD rates almost returning to pre-vaccination levels, while in the Navajo, little serotype replacement was seen.[16, 17] The overall increase in IPD in Aboriginal and Torres Strait Islander adults in recent years is a concern. While coverage data are limited, the latest estimates from 2004–05 of 34% of those aged 50 years or older suggest it has been inadequate to have an impact.[1]

The proportion of IPD cases that had reduced susceptibility to penicillin did not change between 2006 and 2008 (11% in each year), although there was an increase in the proportion of these that were serotype 19A; from 25% to 50%. However, more detailed analysis and interpretation is limited by differences in methods used to assess susceptibility in different laboratories.

These data show a substantial impact in the first 4 years of a publicly-funded 3+0 7-valent conjugate pneumococcal vaccination program (that is without a booster dose). As well as the herd immunity impacts shown here, other Australian studies have shown impacts on non-invasive disease such as hospitalised pneumonia and myringotomy tube insertions.[18, 19] However, the relatively large number of vaccine failures due to serotype 19F should be closely monitored, as well as increases in disease due to serotype 19A.

Post-immunisation surveillance in Australia is essential to monitor trends in IPD, to inform future control strategies, including the targeting of existing and new vaccines and the best options for antibiotic treatment.

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Annual Report of the Australian Gonococcal Surveillance Programme, 2011

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Abstract

The Australian Gonococcal Surveillance Programme monitors antibiotic susceptibility testing of *Neisseria gonorrhoeae* isolates in all states and territories. In 2011, the *in vitro* susceptibility of 4,133 isolates of gonococci from public and private sector sources was determined by standardised methods. Varying antibiotic susceptibility patterns were again reported across jurisdictions and regions. Resistance to the penicillins nationally was 25%, and with the exception of the Northern Territory and Tasmania, ranged from 17% in South Australia and Western Australia, to 44% in Victoria. Quinolone resistance, most at high minimal inhibitory concentration (MIC) levels, was 27% nationally (except in the Northern Territory and Tasmania), ranging from 12% in the Australian Capital Territory to 40% in Victoria. Decreased susceptibility to ceftriaxone (MIC 0.06 mg/L or more), was found nationally in 3.2% of isolates, a decrease from 4.8% in 2010. There has not been an isolate of *N. gonorrhoeae* with a ceftriaxone MIC value greater than 0.125 mg/L reported in Australia. Nationally, all isolates remained sensitive to spectinomycin. Azithromycin surveillance was performed in the Australian Capital Territory; New South Wales; Queensland; Western Australia; the Northern Territory and South Australia. Resistance was found in low numbers of gonococci, with MIC values up to 16 mg/L. The source and site of the isolates referred to the program varied by geographic location. In larger urban centres the ratio of male to female cases was high, and rectal and pharyngeal isolates were common in men. In other centres, and in rural Australia, the male to female ratio was lower, and most isolates were from the genital tract. *Commun Dis Intell* 2012:36(2):E166–E173.

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

Introduction

The World Health Organization (WHO) estimates that 88 million cases of gonorrhoea (*Neisseria gonorrhoeae* infection) occur annually worldwide.[1] In Australia, the rate of gonorrhoea has increased from 35.8 per 100,000 in 2005, to 54.3 per 100,000 in 2011.[2] This increased rate of gonorrhoea was coupled with a global increase in the prevalence of antimicrobial resistance (AMR) in *N. gonorrhoeae*. The potential impact of AMR on treatment outcome is a continuing and growing concern[1,3] as effective antibiotic treatment is fundamental to disease control at the population level.[3]

Over time, the emergence of resistance to the penicillins, tetracyclines, macrolides and fluoroquinolone antibiotics has necessitated the removal of these agents from standard treatment regimens.[4] This was followed by the replacement with extended-spectrum cephalosporin antibiotics (ESCs) as the recommended first line treatment for gonorrhoea in Australia and elsewhere.[5] Unusually, but importantly in Australia however, treatments based on the penicillins remain effective in many rural centres where extremely high disease rates persist.[4]

In large centres in urban Australia, AMR in *N. gonorrhoeae* has long been influenced by the introduction of multi-resistant strains from overseas.[4] There are an increasing number of reports from overseas sources of treatment failures with orally administered ESCs.[6,7] In Australia, oral ESCs are not available, therefore the injectable form (ceftriaxone) is recommended for use in high doses.[5] No treatment failures have yet been reported following ceftriaxone treatment of genital-tract gonorrhoea. However there were 2 instances of failure of treatment of pharyngeal gonorrhoea after treatment with ceftriaxone 250 mg intramuscularly reported in Sydney where elimination of intercurrent genital-tract infection with the same organism was achieved.[8] The gonococci involved both had raised minimal inhibitory concentrations (MIC values) for ceftriaxone.

Strategies for treating and controlling gonorrhoea are based on single dose regimens effecting a cure in a minimum of 95% of cases. The formulation of these regimens is reliant on data derived from continuous AMR monitoring of gonococci to the antibiotics in clinical use.[3,9] Recently, and following the reports of treatment failures with orally administered extended-spectrum cephalosporins,[6,7] calls have been made internationally for enhanced surveillance of all forms of gonococcal AMR in order to optimise gonococcal antibiotic treatment.[1,10]

Since 1981 the Australian Gonococcal Surveillance Programme (AGSP) has continuously monitored the

susceptibility of *N. gonorrhoeae* making it the longest, continuously running national surveillance system for gonococcal AMR.[11] The emergence and spread of penicillin and quinolone resistant gonococci in major cities in Australia has been well documented.[4] This analysis of AMR in *N. gonorrhoeae* in Australia was derived from data collated by the AGSP during the 2011 calendar year. It provides information regarding the gonococcal isolates showing resistance to multiple antibiotics including those with decreased susceptibility to ceftriaxone.[4,12]

Methods

Ongoing monitoring of AMR in gonococci in Australia is performed by the AGSP through a collaborative program conducted by reference laboratories in each state and territory. The AGSP is a component of the National Neisseria Network of Australia and comprises participating laboratories in each state and territory. This collaborative network of laboratories obtains isolates for examination from as wide a section of the community as possible. Both public and private sector laboratories refer isolates to regional testing centres. The increasing use of non-culture based methods of diagnosis has the potential to reduce the size of the sample of isolates available for testing. Details of the numbers of organisms examined are provided in order to indicate the AGSP sample size.

Gonococci isolated in, and referred to, the participating laboratories are examined for antibiotic susceptibility to the penicillins; quinolones; spectinomycin and third generation cephalosporins; azithromycin and for high-level resistance to the tetracyclines, by a standardised methodology previously described.[11,13] The AGSP also conducts a program-specific quality assurance (QA) program.[14]

Antibiotic susceptibility data from each jurisdiction are submitted quarterly to the coordinating laboratory, which collates the results and provides individual feedback to each participating laboratory. Additionally, the AGSP collects data on the gender of the patient, and site of isolation of gonococcal strains. Where available, data on the geographic source of acquisition of antibiotic-resistant isolates are included in analyses.

Results

Number of isolates

There were 4,230 gonococcal isolates referred to, or isolated in, AGSP laboratories in 2011, representing 35% of the 12,118 cases of gonococcal infection notified to the Australian Government Department of Health and Ageing National Notifiable Diseases Surveillance System (NNDSS) in 2011.[2] This was lower than the 40%-42% referred in 2008–2010.

The source and site of infection of these isolates are shown in Table 1.

Isolate numbers in 2011 increased from those reported in 2010 in most jurisdictions: Victoria (from 913), the Northern Territory (from 448),

Table 1: Source and number of gonococcal isolates, Australia, 2011, by sex, site and state or territory

		State or territory								
Sex	Site	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust
Male	Urethra	21	689	304	453	74	4	499	271	2,315
	Rectal	20	248	2	73	27	1	228	24	623
	Pharynx	10	201	0	37	17	1	120	17	403
	DGI	1	2	4	3	0	0	0	2	12
	Other/NS	5	5	13	4	14	0	3	6	50
	Total	57	1,145	323	570	132	6	850	320	3,403
Female	Cervix	5	135	140	204	33	0	69	121	707
	Rectal	0	8	0	2	3	0	3	0	16
	Pharynx	0	41	0	5	2	0	13	5	66
	DGI	0	0	4	4	0	0	0	7	15
	Other/NS	2	3	5	4	5	0	2	1	21
	Total	7	187	149	219	43	0	87	134	826
Total		64	1,332	472	789	176*	6	937	454	4,230

DGI Disseminated gonococcal infection.

NS Not specified

* Total includes 1 isolate with unknown sex and unknown site for South Australia.

Western Australia (from 352) and the Australian Capital Territory (from 30). There was a decrease in isolates from Queensland (from 840) and Tasmania (from 11); and similar numbers were reported from New South Wales (from 1328), and South Australia (176).

Source of isolates

There were 3,403 isolates from men (80%) and 826 (20%) from women and 1 isolate where the gender was not recorded. This male to female (M:F) ratio was 4:1, lower than in 2010 and 2009 (4.7:1 and 4.4:1 respectively). Compared with 2010, the number of isolates increased in both men (from 3,381) and women (from 719).

There were 27 isolates from disseminated gonococcal infections. Twelve were from men (0.4% of all isolates from men), which was less than in 2010 (0.7%), and 2009 (0.9%). In females, there were 15 (1.8% of all isolates from women) isolates from disseminated gonococcal infections, a decrease from the 24 isolates (3.3%) referred in 2010 but an increase from the 4 isolates (0.7%) referred from females in 2009. The infected site was not specified for 32 isolates from males and 9 isolates from females. Isolates from urine samples were regarded as genital tract isolates.

Antibiotic susceptibility patterns

In 2011, 4,133 of the 4,230 gonococcal isolates (98%) remained viable for antibiotic susceptibility testing. These were examined by the AGSP reference laboratories for susceptibility to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics), azithromycin, spectinomycin and for high level resistance to tetracycline. As in past years the patterns of gonococcal antibiotic susceptibility differed between the various states and territories. For this reason data are presented by region as well as aggregated for Australia as a whole.

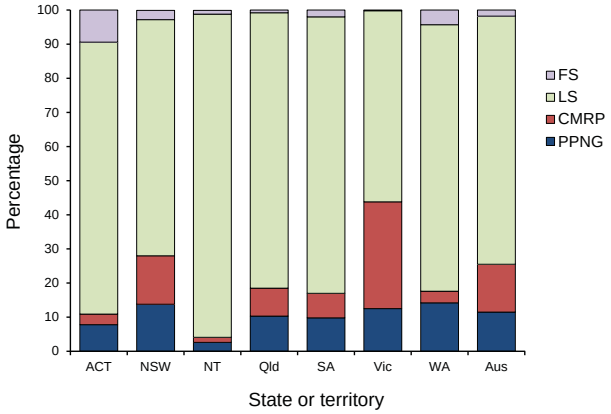
Penicillins

The categorisation of gonococci isolated in Australia in 2011 by penicillin MIC is shown in Figure 1. Infections unlikely to respond to treatment with the penicillin group of antibiotics (penicillin, ampicillin and amoxycillin with or without clavulanic acid), are caused by penicillinase-producing *N. gonorrhoeae* (PPNG) and/or *N. gonorrhoeae* that are chromosomally resistant to penicillin (CMRP). Resistance in the PPNG group results from the production of beta-lactamase, and in the CMRP group by the aggregation of chromosomally-controlled resistance mechanisms.[4] Chromosomal resistance is defined by a MIC to penicillin of 1 mg/L or more.[4,13] The MIC is the least amount of antibiotic which inhibits *in vitro* growth under defined conditions. Infections with gonococci classified as fully sensitive (FS: MIC ≤ 0.03 mg/L) or less sensitive (LS: MIC 0.06–0.5 mg/L) would be expected to respond to standard penicillin treatments, although response to treatment may vary at different anatomical sites.

Nationally, of those gonococci available for susceptibility testing 1,053 (25%) were penicillin resistant by one or more mechanism in 2011, a further decrease in the proportion of isolates resistant to this group of antibiotics recorded in 2010 (29%); 2009 (36%) and 2008 (44%). In 2011, there were 579 (14%) CMRP and 474 (11%) PPNG identified. In 2010 there were 17% CMRP and 12% PPNG demonstrating that the decrease in penicillin resistance nationally in 2011 was predominantly due to chromosomally mediated resistance, as was the case in 2008 and 2009; whereas in 2010 there was a decrease in the proportion of gonococci with both chromosomally mediated resistance and penicillinase production.

The proportion of all gonococcal isolates that were penicillin resistant was highest in Victoria with 44% (CMRP 31%: PPNG 13%) and New South Wales with 28% (CMRP 14%: PPNG 14%) (Figure 1).

Figure 1: Penicillin resistance of gonococcal isolates, Australia, 2011, by state or territory



FS	Fully sensitive to penicillin.
LS	Less sensitive to penicillin.
CMRP	Chromosomally mediated resistant to penicillin.
PPNG	Penicillinase-producing *Neisseria gonorrhoeae*

Proportions of resistant gonococci were lower than those reported in 2010 in New South Wales, Queensland, Western Australia, and South Australia. In Victoria, the proportions were essentially unchanged from 2010. There were 2 CMRP

identified in the Australian Capital Territory and 5 PPNG; and in Tasmania there were no CMRP and 3 PPNG. In the Northern Territory, there were 19 penicillin-resistant gonococci: 7 CMRP and 12 PPNG giving a total of 4.2% of strains that were penicillin-resistant in 2011; a higher proportion than in 2010 (3.6%), but similar to the proportion in 2006 to 2009 (4.2% in 2009; 3.9% in 2008; 4.1% in 2007; 4.6% in 2006).

Data on the place or mode acquisition were available for 86 (18%) infections with PPNG. Forty-one (8.6%) of the infections with PPNG were acquired locally, and 45 (9.4%) were acquired by overseas contact. These external contacts were principally in Western Pacific or South East Asian countries with those reported from Thailand, The Philippines, Indonesia and Vietnam the most numerous. Additionally, disease was also reportedly acquired in Malaysia, Papua New Guinea, Singapore, Malaysia and, more widely, Ireland and the United States of America.

Ceftriaxone

From 2001 onwards, low numbers of gonococcal isolates with decreased susceptibility to ceftriaxone (MIC 0.06–0.125 mg/L) have been found in Australia. The proportion has increased incrementally with the data from recent years showing a rise from 0.6% in 2006; 0.8% in 2007; 1.1% in 2008; 2.0% in 2009: to 4.8% in 2010. In 2011, a decreased proportion of isolates with decreased susceptibility to ceftriaxone was observed nationally: 134 of 4,129 (3.2%). There has not been an isolate of *N. gonorrhoeae* with an MIC value greater than 0.125 mg/L reported in Australia.

In Victoria, 50 of 937 isolates (5.3%) had decreased susceptibility to ceftriaxone; 58 of 1,322 (4.4%) from New South Wales; 2 of 64 (3.1%) from the Australian Capital Territory; 18 of 789 (2.8%) from Queensland; 3 of 454 (0.7%) from Western Australia; and 2 of 470 (0.4%) from the Northern Territory. There were no isolates with decreased susceptibility to ceftriaxone reported from Tasmania.

In 2011, there was a decrease in the number of gonococci with decreased susceptibility to ceftriaxone compared with 2010, in all jurisdictions with the exception of the Northern Territory and Tasmania, as shown in Table 2.

Spectinomycin

Again in 2011, all isolates from all jurisdictions were susceptible to this injectable antibiotic.

Quinolone antibiotics

Figure 2 shows the distribution of gonococci with altered susceptibility to quinolones nationally and by jurisdiction. Thus far, resistance to the quinolone antibiotics in *N. gonorrhoeae* is mediated only by chromosomal mechanisms so that incremental increases in MIC values are observed. The AGSP uses ciprofloxacin as the representative quinolone and defines altered susceptibility as an MIC of 0.06 mg/L or more.[13]

Nationally in 2011, 1,132 of gonococci examined (27%) had some level of resistance to quinolones (QRNG), a further decrease in proportion nationally of quinolone resistance from the 35% in 2010; 43% in 2009 and 54% in 2008. The majority of QRNG found in 2011 (1,099; 97%) had resistance at a higher level i.e. MICs ≥ 1 mg/L and many of these had MIC levels of 8– 64 mg/L, the same proportion reported in 2010.

Table 2: Number and per cent of gonococcal isolates with decreased susceptibility to ceftriaxone, Australia, 2009 to 2011, by state or territory

	2009		2010		2011	
State or territory	n	%	n	%	n	%
Australian Capital Territory	2	5.3	2	6.7	2	3.1
New South Wales	16	1.7	74	5.6	58	4.4
Northern Territory	1	0.2	1	0.2	2	0.4
Queensland	10	1.8	26	3.2	18	2.3
South Australia	9	5.3	19	11.6	1	0.7
Tasmania	0	0.0	0	0.0	0	0.0
Victoria	17	2.2	52	5.7	50	5.3
Western Australia	9	3.1	17	5.2	3	0.7
Australia	64	2.0	191	4.8	134	3.2

MIC value 0.06–0.125 mg/L

Figure 2: Percentage of gonococcal isolates less sensitive to ciprofloxacin or with higher level ciprofloxacin resistance and all strains with altered quinolone susceptibility, Australia, 2011, by state or territory

LS QRNG, MIC 0.06–0.5 mg/L

R QRNG, MIC 1 mg/L or more

In Victoria, 378 (40%) of all isolates examined were QRNG (2010: 44%); in New South Wales there were 453 (34%) (2010: 40%); in South Australia there were 37 (24%) (2010: 41%); in Western Australia there were 90 (22%) QRNG (2010: 40%); in Queensland there were 144 (18%) (2010: 28%); and in the Australian Capital Territory there were 12 (18%) (2010: 60%). In the other jurisdictions the numbers of QRNG remained low and essentially unchanged from 2010; with 16 (3.5%) in the Northern Territory and 2 QRNG from Tasmania.

Information on country of acquisition of QRNG was available for 167 (14.8%) of the 1,132 cases. Ninety-seven of these (8.6%) were acquired locally and 50 (3.7%) infections were acquired overseas with Thailand, The Philippines and Indonesia the most numerous. Additionally, disease was also reportedly acquired in Hong Kong, Singapore, Vietnam and South Africa.

Azithromycin

In 2011, data on azithromycin susceptibility was available from all states and territories except Victoria. Overall, nationally, the proportion of isolates exhibiting resistance (azithromycin MIC ≥1 mg/L) was low (1.1%) and the maximum MIC value recorded in Australia to date is 4 mg/L. MIC levels in azithromycin resistant gonococci have reached very high levels in Europe, but these strains have not been detected in Australia.

High-level tetracycline resistance

High-level tetracycline resistance *N. gonorrhoeae* (TRNG) is used as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea and are rarely, if ever, used for treatment of gonorrhoea in Australia. Despite the lack of use of this antibiotic group, the proportion of TRNG detected continued to increase between 2006 and 2009 (12% to 21%), and was stable in 2010 (21%). In 2011, there was a decrease in TRNG nationally with 733 of 4,129 isolates (18%) reported.

TRNG were present in all jurisdictions, with the highest proportion in Western Australia (107 TRNG, 26%); the Northern Territory (114 TRNG, 25%); Queensland (149, 19%); New South Wales (236, 18%) and South Australia (24, 16%). Lower proportions were present in Victoria (95 TRNG, 10%) and in the Australian Capital Territory there were 6 TRNG (9%) and there were 2 TRNG in Tasmania.

Discussion

The WHO recommendations for standardised treatment regimens for gonorrhoea are based on data from epidemiological surveillance of the distribution and extent of AMR in gonococci.[3] An antimicrobial resistance rate of 5% or more in gonococci isolated in a general population is the 'threshold' for removal of an antibiotic from treatment schedules and substitution with another, effective, agent.[3,15] Programs such as the AGSP are conducted to determine the proportion of antimicrobial resistance in gonococcal strains isolated in a defined patient population and relate these findings to the likely efficacy of current treatment schedules.[3,5,9,13,15]

Surveillance producing quality AMR data, on a sufficient and representative sample of isolates,[3,13,15] is a pivotal part of a strategy for disease control; however the *in vitro* growth requirements and the fastidious nature of *N. gonorrhoeae* can complicate this process. In 2011, the strains examined by the AGSP were sourced from the public and private health sectors, constituting a comprehensive sample (35% of all notifications nationally) that meets these requirements, in spite of the increasing use of nucleic acid amplification testing (NAAT) for diagnosis of gonorrhoea in Australia, which does not provide AMR testing. The AGSP distributes reference panels for use in internal quality control and provides an External Quality Assurance Programme[14,16] required for validation of gonococcal AMR data.

The overall number of gonococcal strains examined by the AGSP in 2011 (4,133) was higher than the number examined in previous years, but the proportion of isolates received from notified cases in Australia was lower than that examined in the

years 2008 to 2010, a reflection of the increase in the number of cases and the increase in NAAT testing. Isolate numbers in 2011 increased from those reported in 2010 in most jurisdictions except Queensland and Tasmania, and were unchanged in New South Wales and South Australia.

In 2011, 25% of gonococci nationally were resistant to the penicillins, and 27% to the quinolone antibiotics. These proportions were again reduced from those reported nationally in 2008 to 2010 whereas previously they had been increasing each year since 2003.[4] In 2011, there were decreased numbers of gonococci with both chromosomally mediated resistance to penicillin and penicillinase production, but predominantly chromosomally mediated resistance. Aggregated data have shown a predominant clone of CMRP coupled with high-level quinolone resistance circulating with increasing frequency annually since 2003.[4,12] It is possible that the continued reduction in resistance to both penicillin and the quinolones seen since 2008 continues to reflect a 'clonal shift' in gonococcal isolates.

The proportion of gonococci with high-level tetracycline resistance decreased in 2011 in Australia where there is a low level of exposure to these antibiotics.[4]

Evidence of the 'rural-urban divide'[4] in gonococcal resistance was maintained, (Figures 1 and 2) underscoring the need for disaggregated information rather than pooled national data to define treatment regimens appropriate for the various jurisdictions. Some remote areas with high disease rates continue to be able to use penicillin-based treatments, but effective use of this inexpensive and acceptable treatment is contingent on vigilant monitoring of resistance patterns.

The emergence and spread of gonococci with decreased susceptibility to the later generation cephalosporin antibiotics also referred to as ESCs has been documented in the AGSP reports.[12] These gonococci have also been found in rapidly increasing numbers in the WHO Western Pacific Region.[17] In 'urban' Australia, the injectable agent ceftriaxone is now the standard treatment for gonorrhoea in public sector clinics, with the recommended dosage being 500 mg by intramuscular injection. This dose is higher than the 250 mg dose that is more commonly used throughout the Western Pacific Region[18] but 500 mg is the smallest volume vial currently available in Australia.

Decreased susceptibility to the ESCs has been accompanied by increasing numbers of reports of treatment failures with the orally administered antibiotics in this group.[7,18,19] To date, there have been no strains of *N. gonorrhoeae* reported in Australia with a ceftriaxone MIC value greater than 0.125 mg/L; nor substantiated reports of treatment failure in genital tract gonorrhoea following ceftriaxone therapy. However, concerns are escalating locally and globally as increasing proportions of strains with decreased susceptibility are reported.[5]

In Australia in 2011, the proportion of gonococcal isolates with decreased susceptibility to the ESCs (3.2%) was lower than the 4.8% reported in 2010 but has markedly increased from the proportion reported in 2009 (2%). Surveillance to monitor *N. gonorrhoeae* with elevated MIC values coupled with sentinel site surveillance in high risk populations is critically important to inform therapeutic strategies and to detect instances of treatment failure. Sentinel site surveillance programs involve patient follow-up and test of cure (TOC) cultures after treatment of *N. gonorrhoeae* infections, in particular those in oropharyngeal sites. This is currently being conducted in a very limited number of settings in Australia, and needs to be expanded throughout all jurisdictions as a matter of priority.

The mechanisms of resistance responsible for the MIC increases to ceftriaxone in gonococci include the presence of 'mosaic' *penA* genes in gonococci with raised ESC MIC values. The *penA* gene encodes penicillin binding protein 2 (PBP2), the major site of action of ceftriaxone, and mosaic PBP2 are altered to reduce this activity. Additional gene polymorphisms that affect antibiotic access to the organism complement these PBP2 changes and further increase ESC MICs.[5] Of recent interest has been an extension of a study from 2001 to 2005 on the dynamics of the spread of mosaic PBP2-containing gonococci (mPBP2-GC) in Australia. Initial investigations suggested that mPBP2-GC found locally were also present in Hong Kong (where they were associated with treatment failure with an oral ESC, ceftibuten),[7] and Japan.[20] Continuing studies in 2007 and 2008 showed that the subtypes of the mPBP2-GC present in Australia had altered markedly and that these strains had increased as a proportion of all gonococci tested.[20] Also of relevance have been local studies that showed that other non-mosaic lesions in *penA* were also responsible for increases in ceftriaxone MIC values similar to those found in mosaic PBP2 containing gonococci.[21] These lesions were single nucleotide polymorphisms that represented mutations occurring in the *penA* of *N. gonorrhoeae*. This contrasted with the mosaic *penA* alteration that results from acquisition of 'foreign' DNA by the gonococcus.[22] However, not all increases detected in ESC MIC levels can be explained by the molecular mechanisms described so far. This poses difficulties in developing reliable laboratory methods for the detection of ESC 'resistant' gonococci.

All gonococcal isolates tested in Australia in 2011, including those with altered cephalosporin susceptibility, were susceptible to spectinomycin. A low proportion of gonococci were found to be resistant to azithromycin in 2011. Recently, the United States Centers for Disease Control and Prevention and United Kingdom guidelines have moved to recommend a dual therapy strategy of ceftriaxone with oral azithromycin for uncomplicated gonococcal infection.[23,24] Resistance to azithromycin, widely used as an anti-chlamydial agent in conjunction with gonococcal treatment, has been reported with increasing frequency overseas. MIC levels in azithromycin-resistant gonococci have reached very high levels in Europe, but these strains have not been detected in Australia.

The continued emergence and spread of anti-microbial resistance in *N. gonorrhoeae* is a global public health issue, and the evolution of this resistance is complex, and requires attention to both disease control strategies and rational use of antibiotics.[10,25,26] Critically, disease control strategies and the understanding of the global scope of AMR are informed by surveillance programs of AMR nationally and internationally. Continuing commitment and vigilance to surveillance of AMR in *N. gonorrhoeae* means that maintenance of culture-based systems is crucial whilst surveillance is based on testing of gonococcal isolates.

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Surveillance of Creutzfeldt-Jakob disease in Australia: update to December 2011

Genevieve M Klug, Alison Boyd, Amelia McGlade, Christiane Stehmann, Marion Simpson, Colin L Masters, Steven J Collins

Abstract

The Australian National Creutzfeldt-Jakob disease Registry (ANCJDR) is a Commonwealth Government-funded surveillance unit, responsible for the ascertainment of all cases of human transmissible spongiform encephalopathy (also known as prion diseases) in Australia. Having been in operation for 18 years, the activities of the ANCJDR have evolved and expanded over this timeframe, with the ANCJDR providing clinical, diagnostic and infection control advice and service. This update provides a review of the activities of the ANCJDR during 2011 and analysis of both prospective and retrospective (to 1970) data collected from 1993 to 31 December 2011. *Commun Dis Intell* 2012;36(2):E174–E179.

Keywords: Creutzfeldt-Jakob disease, surveillance, bovine spongiform encephalopathy, transmissible spongiform encephalopathies

Introduction

The surveillance of Creutzfeldt-Jakob disease (CJD) in Australia has been undertaken since 1993 by the Australian National CJD Registry (ANCJDR), located at The University of Melbourne. CJD is one form of the transmissible spongiform encephalopathy (TSE) family of rare neurological diseases. While the prime objective of the ANCJDR is to detect all cases of CJD and other TSE forms including Gerstmann Sträussler-Sheinker syndrome, fatal familial insomnia and variant CJD (vCJD) arising in the Australian population, the Registry also provides additional nation-wide infection control advice, diagnostic services, advice to family and clinicians and collaborates with both national and international surveillance counterparts. CJD may be acquired through medical intervention (iatrogenic CJD), inherited in an autosomal dominant pattern (familial CJD) or occur sporadically, as in the majority of cases (known as sporadic CJD). After evaluation by the ANCJDR, cases are classified in accordance with the European and Allied Countries Creutzfeldt-Jakob Disease Surveillance Consortium (EUROCJD) and World Health Organization (WHO) promulgated diagnostic criteria.[1,2] Definite cases are neuropathologically confirmed, whereas probable and possible cases are defined on the basis of clinical profile and diagnostic testing. Both definite and probable cases are considered to be confirmed CJD cases for the purposes of statistical analysis.

Methods

The ANCJDR is funded by the Australian Government Department of Health and Ageing to investigate all notified cases of suspected CJD in Australia. As of June 2006, CJD has been a notifiable disease in all Australian states and territories. Notification of suspect cases to the ANCJDR occurs through several mechanisms including referral for diagnostic testing, personal communications from clinicians, family members and hospitals, as well as searches of death certificates, hospital and health department records. These record searches were performed until 2004 and were integral in ascertaining retrospective cases of CJD in Australia between 1970 and 1993 and monitoring of prospective notifications.

After notification, the ANCJDR investigates all suspected cases for the clinical likelihood of CJD and where possible aims to classify all cases based on clinically validated criteria.[1,2] Definite cases are those that have been neuropathologically confirmed either by brain biopsy or post-mortem examination. Probable cases are classified on the basis of clinical profile, a typical electroencephalogram (EEG) and/or a positive 14-3-3 cerebrospinal fluid (CSF) test and/or characteristic MRI with high signal in the caudate/putamen. In addition to dementia, probable cases must display at least two of the following; myoclonus; visual or cerebellar signs; pyramidal or extrapyramidal features; and/or akinetic mutism with an illness duration of less than 2 years. Possible cases fulfill the same clinical profile in the absence of a typical EEG, characteristic MRI and either no 14-3-3 CSF test or a negative result. Based on data collected on definite and probable cases arising in Australia between 1970 and 2011, epidemiological analysis was performed, including age-adjusted annual mortality rates, calculated using direct standardisation and adjusted to the Australian Bureau of Statistics estimated resident population for Australia and each state and territory as at June 2000.

In this report, an update of Australian TSE surveillance will be presented for the reporting period from 1 January to 31 December 2011.

Results

Australian National Creutzfeldt-Jakob Disease Registry surveillance update to 31 December 2011

For the current reporting period of 1 January to 31 December 2011, 78 new suspect cases have been notified and evaluated by the Registry. Of these new suspect cases, nine were excluded (7 after neuropathological examination), 50 were incomplete, and 19 were classified as definite (17) or probable CJD (2). In total, 20 suspect cases were excluded from the register (13 after neuropathological confirmation) since 1 January 2011. For the same period, a total of 41 CJD cases were confirmed, with definite cases increasing from 432 to 457 and probable cases increasing from 221 to 237 cases.

As of 31 December 2011, 954 cases were included on the register with 694 of these being classified as probable or definite CJD cases; an additional 593 cases were excluded after detailed follow-up (Table 1). There are currently 14 cases of possible CJD of which 13 are sporadic and 1 iatrogenic. One of the possible sporadic cases was classified in 2011. Of the 245 incomplete cases, 167 are presently alive. The rapid annual increase in the number of incomplete cases on the register observed in recent years (20% increase per year) has been markedly reduced in 2011 (7% increase), due to the high level of cases confirmed by neuropathology coupled with a 4-fold increase in the number of probable cases classified. During 2011, the ANCJDR has made a concerted effort to focus staff on case review and classify outstanding cases.

During 2011, no further cases of iatrogenic CJD were identified. The most recent human-derived pituitary gonadotrophin-related CJD death occurred in 1991, while the most recent Lyodura-related CJD death occurred in 2000. As of 31 December 2011, no cases of vCJD have been identified in Australia.

Notifications

The level of notification of suspect CJD cases to the ANCJDR is an important factor influencing ascertainment adequacy of TSE cases in Australia. The ANCJDR currently relies on a surveillance program whereby suspect cases are notified directly to the ANCJDR through personal communication with clinicians and allied health professionals, families, health departments or through ANCJDR diagnostic services. A decline in notifications typically results in a decline in confirmed cases,[3] which underscores the importance of monitoring notification rates across Australia and by individual states and territories.

Since surveillance began in Australia, the notification of suspect cases to the ANCJDR has fluctuated, ranging from 40 to 131 cases per year. As described previously,[4] the fluctuation can be partly explained by changes in the criteria used to classify a suspect case and the inflation of notification numbers in the early years of the ANCJDR's operation when retrospective case deaths between 1970 and 1993 were actively ascertained through hospital, health department and death certificate searches. Since 2000, these factors no longer influence the annual number of cases notified, making this 12 year period a clearer representation of notifications. The average number of suspect cases notified to the ANCJDR for this period was 72 per year (range 54–89 cases).

In 2011, 78 new cases were added to the Registry as suspect cases for further investigation, which aligns with the 2000–2011 average. Over two-thirds of the 78 cases were ascertained through referral for 14-3-3 cerebrospinal fluid diagnostic testing, offered by the ANCJDR since 1997. The remainder were direct communications to the ANCJDR by clinicians, family or support services, and in a single case, notification occurred through a direct referral for genetic testing.

By state and territory, the notification of prospective suspect cases (those arising between 1993 and 2011)

Table 1: Classification of Creutzfeldt-Jakob Disease Registry cases, 1 January 1970 to 31 December 2011

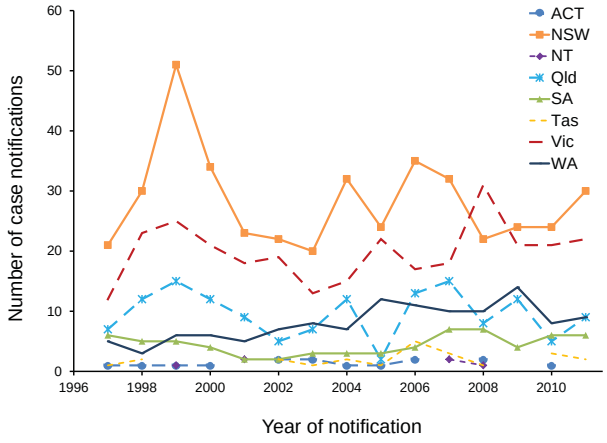
Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total
Definite	410	43	5*	0	0	458
Probable	223	10	4	0	0	237
Possible	13	0	1	0	0	14
Incomplete	0	0	0	0	245†	245
Total	646	53	10	0	245	954

* Includes 1 definite iatrogenic case who received pituitary hormone treatment in Australia but disease onset and death occurred while a resident of the United Kingdom. This case is not included in statistical analysis since morbidity and mortality did not occur within Australia.

† Includes 167 living cases.

to the ANCJDR has been relatively stable since 2006, compared with the previous years (Figure 1). Prior to this report, Tasmania was the only exception, with declining notifications since 2006, however, in 2010 and 2011, the number of notifications has returned to previously observed levels. Similarly, in 2011 notifications in New South Wales returned to pre-2008 levels of around 30 cases per year after 3 years of below average notifications.

Figure 1: Prospective, suspect Creutzfeldt-Jakob disease case notifications to the Australian National Creutzfeldt-Jakob Disease Registry, 1997 to 2011, by state or territory

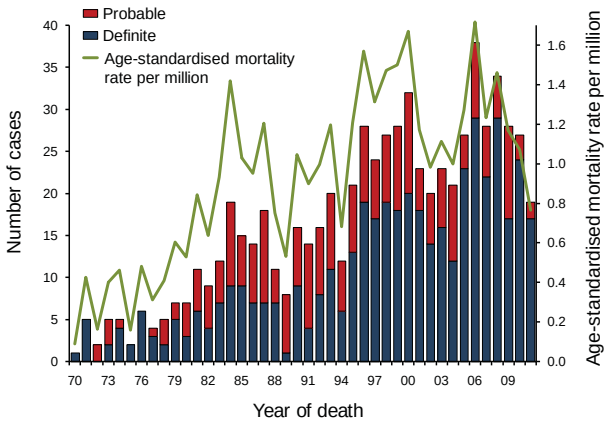


Based on the state and territory populations, Tasmania, New South Wales, Victoria and Western Australia had the highest average rates of suspect case notification with between 3.7–4.3 cases notified per million population per year for the 2000–2011 period. Western Australia had the greatest average rate of notifications at 4.3 cases per million population per year. Temporally, the annual rates in these States have remained relatively stable, with the exception of Western Australia where the rate has been increasing over the period to around 5 cases per million population per year. In the other states and territories, the average annual notification rates reside between 2.0 and 3.0 cases per million population per year with the Northern Territory having the lowest notification rate at 2.0 cases per million population per year. Minimal temporal change in notification rates was observed for the same period in Queensland, South Australia, the Northern Territory and the Australian Capital Territory.

Case outcomes

Since 1970, there has been an increasing positive trend in the annual number of TSE deaths in Australia rising from 10–15 in the mid-1980s to over 25 in the late 1990s, reaching an initial peak in 2000 (32 cases), a decline between 2001 and 2004 and then reaching over 25 cases since 2005 (Figure 2). The ANCJDR postulate that the 2001–2004 period of decline related to a reduction in overall notifications to the register and also fewer probable cases being classified during this time due to difficulties associated with case investigation. Since 2005, the annual number of confirmed definite and probable cases has been consistently between 25–35 cases with two further peaks in 2006 (38 cases) and 2008 (34 cases). It is believed that this sustained increase is a reflection of greater notifications and increased post-mortem examinations being performed on suspect CJD cases.

Figure 2: Number and age-standardised mortality rate of Australian National Creutzfeldt-Jakob Disease Registry definite and probable cases, Australia, 1970 to 2011*



Age-standardised mortality rates were calculated using the Australian Bureau of Statistics 2000 estimated resident population for Australia.

* To 31 December 2011

Although the ANCJDR retrospectively ascertained cases to 1970, it is the prospective ascertainment period from 1993 to 2011 that is considered a more meaningful period for analysis of Australian CJD incidence. For this timeframe, the average annual age-adjusted mortality rate was 1.2 deaths per million population; a slight increase from the previous reporting period and aligns closely with other EUROCJD countries where similar surveillance units are in operation.[5]

The ANCJDR has a large number of cases currently on the register that are incomplete (245) and remain under investigation. The ANCJDR aims to evaluate and classify all notified cases as definite, probable, possible or non-CJD, although obstacles such as a lack of available diagnostic information, next-

of-kin consent not being granted for investigation and staff resourcing for clinical assessment, can be encountered, which restrict the ANCJDR's ability to classify a case. During 2011, there was a concerted effort to clinically evaluate pending cases, made possible by the employment of an additional part-time neurologist to the unit. As a result, a large number of cases were classified as probable. This has affected the mortality rates for several years due to the time of death for some of these cases. Of the 41 cases confirmed as definite or probable in 2011, 17 died in 2011 and 2 were confirmed as living, definite cases by neuropathological examination of biopsy tissue. The remaining 22 cases died prior to 2011. Six died in 2010 and underwent post-mortem examination for case confirmation, while the remainder died in 2006, 2008, 2009 and 2010 and were classified as probable cases based on ANCJDR evaluation of the clinical and investigation profile. The confirmation of these previously known but unclassified cases on annual mortality rates has led to the age-adjusted mortality rate peaking at 1.7 cases per million population per year in 2006, while the rates in the other years reached 1.1–1.5 cases per million population per year. These figures provide the ANCJDR with an insight that the incidence of CJD in Australia may be closer to 2 cases per million population per year, rather than the presumed rate of 1 case per million population per year.[3] In several other countries where CJD surveillance is performed, mortality rates at this elevated level have been observed previously.[5] In some regions, this increase has been explained by increased awareness, and better case ascertainment and disease diagnosis.[6,7]

By state and territory, the 41 new cases confirmed in 2011 were identified in New South Wales (16), Victoria (11), Western Australia (8), Queensland (4) and South Australia (2). With the addition of these cases, the average age-adjusted mortality rates for the two periods of 1993–2011 and 2000–2011 shows that the majority of regions in Australia have rates above or close to 1 case per million population per year (Table 2). For the full prospective period (1993–2011), the highest mortality rates were observed in Victoria and Western Australia (1.4 and 1.6 deaths per million population per year, respectively). When analysing the incidence rates for sporadic cases, thereby excluding any influence of genetic and iatrogenic cases on incidence, Western Australia was associated with a 28% (95% CI, 2%–78%) greater rate than would be expected in the Australian general population. This greater risk could be due to optimal case ascertainment and notification to the ANCJDR and a more complete estimate of CJD incidence being achieved in this region.

Tasmania continues to have the lowest TSE mortality in Australia and it has been postulated that cases were being under-ascertained in this region. However, it should be noted that given the small population, the effect of 2 to 3 additional cases in Tasmania would result in the mortality increasing to 0.9–1.0 cases per million population per year. Furthermore, the restriction of the data to the recent 12 year period, where more uniform ascertainment, diagnostic capacity and case evaluation has been in place, the rate in Tasmania is closer to 1 case per million population per year, which is similar to the rates reported in other regions of lower population such as South Australia and the Northern Territory.

Case demographics

The 694 confirmed CJD cases comprise 8 iatrogenic cases, 53 familial cases and 633 sporadic cases. The 8 iatrogenic cases comprise 5 Lyodura-related cases and 3 pituitary-gonadotrophin associated cases.

Table 2: Transmissible spongiform encephalopathy deaths and mortality rates, by state and territory

State or territory	TSE cases by year of death													Total cases	Mean age-adjusted mortality rate (deaths/million/year)	
	00	01	02	03	04	05	06	07	08	09	10	11	Alive		00–11*	93–11*
ACT			1		1		1		2		1			6	1.4	1.3
NSW	12	9	7	7	11	10	12	10	6	10	5	10	1	110	1.3	1.2
NT							2	1						3	0.8	0.8
Qld	7	3	3	3			7	2	4	4	2	2		37	0.7	1.0
SA	2			1	2	1	1	3	5	2	4			21	1.0	1.3
Tas			2			1	2							5	0.8	0.7
Vic	9	10	5	9	5	11	9	6	13	7	11	4	1	100	1.5	1.4
WA	2	1	2	3	2	4	4	6	4	5	4	3		40	1.5	1.6
Aust	32	23	20	23	21	27	38	28	34	28	27	19	2	322	1.2	1.2

* Includes all deaths occurring between 1 January 1993 or 1 January 2000 and 31 December 2011.

A further definite pituitary-gonadotrophin associated case received pituitary hormone treatment in Australia but as disease onset and death occurred in the United Kingdom; this case was not included in the Australian statistical analysis.

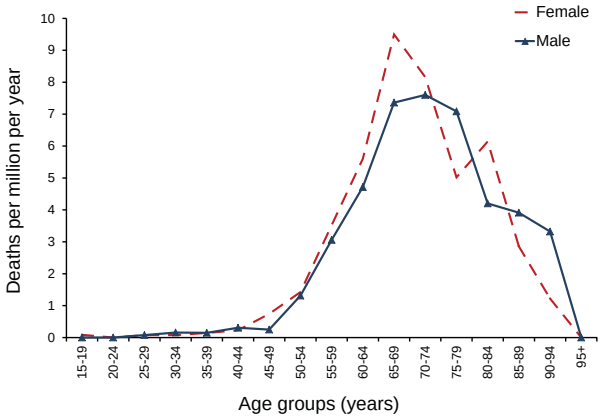
No new familial cases of CJD were identified since the last reporting period. Annually, between 1 and 4 familial cases are detected by the ANCJDR each year in Australia, although this number has been declining in the last 3 years. The ANCJDR offers genetic testing, however, as testing is dependent on consent by next-of-kin, the test is not systematically undertaken on all cases. The ANCJDR is also aware that genetic testing may be performed independently of the unit, and the results for some cases will be unknown to the ANCJDR. This raises the potential that some genetic cases may be misclassified as sporadic CJD.

All new cases confirmed during 2011 have been classified as sporadic CJD at this time. There has been no change to the median age at death or durations of all 3 CJD groups since the last reporting period, despite the inclusion of the new cases in the analysis. For all iatrogenic cases, median age at death is 39 years (range 26–62 years) and for all familial cases, 59 years (range 18–82 years). For all sporadic cases, the median remained at 66 years (range 25–90 years). Disease duration was 4.0 months for all cases, 3.7 months for sporadic cases (range 0.9–60 months) while longer in iatrogenic (6.5 months, range 2–25 months) and familial (6 months, range 1.25–192 months) cases. Within 6 months of disease onset, 73% of sporadic cases, 53% of familial cases and 56% of iatrogenic cases were deceased.

The gender breakdown of all Australian cases have remained the same as previously reported, with confirmed cases in females slightly higher for all cases (53%), sporadic cases (53%) and familial cases (55%). The iatrogenic group is divided into 3 female pituitary hormone-related cases, 1 female and 4 male Lyodura related cases. In men, incidence peaked in the 70–74 year age group with 7.6 cases per million per year; however, the incidence in both the 65–69 and 75–79 year age groups was also above 7 cases per million per year (Figure 3). In women, incidence appears to peak slightly at a younger age than men, peaking in the 65–69 year age group with almost 10 cases per million per year. It is important to note that these age groups have a 6 to 7 times greater incidence of CJD than that of the general Australian population (1.24 cases per million population per year).

As shown in Figure 2, the ascertainment of all Australian TSE cases has improved markedly since surveillance began in 1993. To investigate whether this increase correlates with an age-specific change

Figure 3: Age- and sex-specific mortality rates in all Creutzfeldt-Jakob disease cases, 1993 to 2011

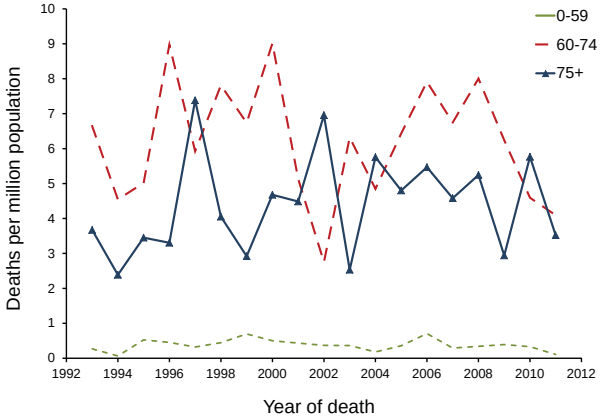


in ascertainment, the temporal changes in the annual mortality rate by specific age groups are shown in Figure 4. Since 1993, the annual rate of disease amongst the 60–74 year age group has remained relatively stable with an annual rate ranging from 3–9 case deaths per million persons. Although the 60–74 year age group constitutes the majority of cases, the rate in the older age group of 75 years or over shows a slight increasing positive trend over the 1993–2011 period and further supports the opinion that it is the detection of older age cases that has led to increased incidence over the active surveillance period.

Summary

In 2011, the ANCJDR continued surveillance activities to monitor the occurrence of all forms of TSE in Australia and also provide various services for the health sector and family members of suspect and confirmed cases, including expert advice, diagnostic tests and associated case investigation. Focused staff resources for case review and evaluation during 2011

Figure 4: Temporal change in age-specific mortality rates for Australian Creutzfeldt-Jakob disease cases, 1993 to 2011



has enabled an increased number of cases, especially probable cases, to be classified during the reporting period compared with previous years. This has increased the overall incidence rates in Australia and suggests that CJD incidence in some regions is closer to 2 cases per million population.

Acknowledgements

The ANCJDR wishes to thank families, as well as medical practitioners and associated staff for their generous support of Australian CJD surveillance. The ANCJDR also thanks Dr Handan Wand, Dr Matthew Law and Professor John Kaldor (The Kirby Centre) for their expert epidemiological and statistical support.

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Erratum

The report *Australia's notifiable disease status, 2010: Annual report of the National Notifiable Diseases Surveillance System* published in the previous issue of *Communicable Diseases Intelligence* (*Commun Dis Intell* 2012;35(1):1–69) contained an error on page 2 of the Methods section. It was incorrectly stated that the indirect method of standardisation was used for calculating age-adjusted rates for sexually transmitted diseases. The corrected sentence should read 'For some diseases, age adjusted rates were calculated using the direct method of standardisation, with 2006 census data as the standard population.'

Peer-reviewed articles

An outbreak of *Plasmodium falciparum* malaria in the Torres Strait

Annie Preston-Thomas, Richard W Gair, Kelly A Hosking, Gregor J Devine, Steven D Donohue

Abstract

This report describes the largest outbreak of *Plasmodium falciparum* malaria in the Torres Strait for more than 25 years. It details factors that may have contributed to the outbreak, the public health response and implications for the broader region. Eight cases of locally-acquired falciparum malaria occurred on Saibai and Dauan islands during March and April 2011. Including imports, there were 17 *P. falciparum* notifications between February and May 2011. Three cases of pure *P. vivax* malaria that might have been locally acquired have been omitted from this report. Malaria is endemic on the nearby coast of Papua New Guinea (PNG), and regularly imported to the Torres Strait where a competent vector exists in sufficient numbers to transmit the disease to the local population. The most common malaria vectors in northern Australia and Torres Strait are the *Anopheles farauti* complex. Factors contributing to the outbreak may include an increase in travel between the outer islands and PNG, inadequate local vector control and late or missed diagnoses of malaria. Outbreak management involved intensive case finding and treatment, vector control and health promotion. Reducing the risk of future outbreaks requires studies of vector behaviour, ecology and management, health promotion, improvements to protective infrastructure, and clinical guideline revision. Further malaria outbreaks are likely in the Torres Strait and elsewhere in northern Australia. It is important to maintain awareness and be prepared to respond rapidly. *Commun Dis Intell* 2012;36(2):E180–E185.

Keywords: *Plasmodium falciparum*, malaria, Australia, Torres Strait, *Anopheles farauti*

Introduction

The Torres Strait Islands are situated between the tip of Cape York on the Australian mainland, and Papua New Guinea (PNG). The northernmost islands Saibai, Boigu and Dauan lie within 5 kilometres of the PNG coast (Figure). The 1978 Torres Strait Treaty permits free movement for traditional activities of Torres Strait Islanders and people from 13 coastal PNG villages, in a designated cross-border zone. Traditional purposes do not include health, however it is acknowledged to be unwise for humanitarian and public health reasons to exclude PNG visitors from acute care services.[1]

Saibai Island comprises largely swampland. About 340 people live in the 1.7 km long township on the northwest coast, some of whom are PNG citizens.[2] Breeding sites of the malaria vector *Anopheles farauti* sensu lato (s.l.) include an abundance of mangrove swamps, coastal flats, and brackish pools.[3] Alternative feeding hosts on the island include feral deer and dogs.[4] Dauan is a small, rocky, volcanic island, 4.5 km south west of Saibai; the township lies along a narrow coastal strip backed by steep hills with swampland to the east, and salt marsh and mangroves to the north-west. There are far fewer suitable vector breeding sites than on Saibai.[3]

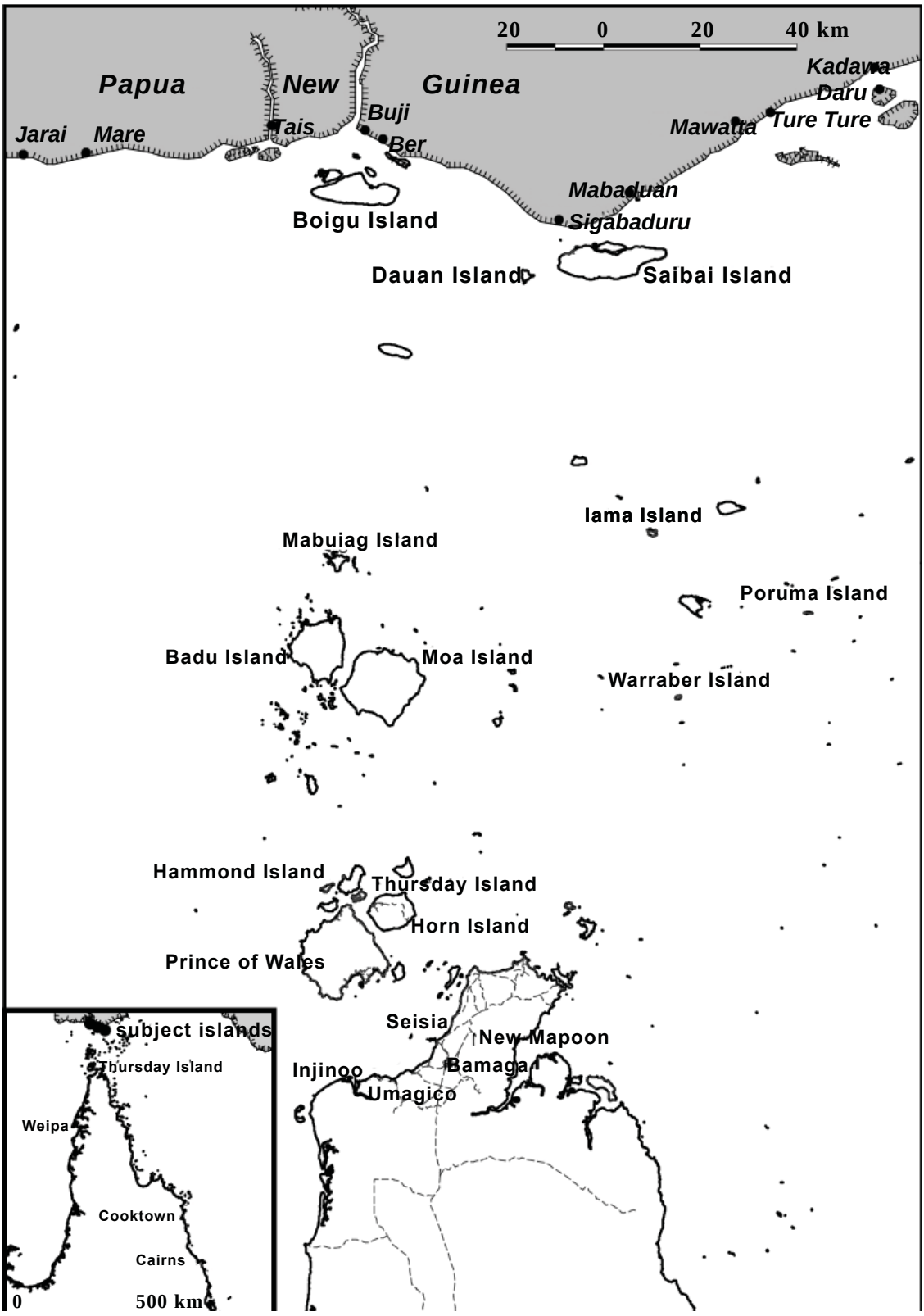
Malaria was previously endemic in the Torres Strait, but Australia was declared free of malaria in 1981.[5] *P. falciparum* and *vivax* infections are regularly diagnosed in the Torres Strait with most cases imported from PNG, and local transmission is rare. Saibai had small outbreaks of malaria in 1984, 1989, 1991, and 2004.[3,6–8] One case of locally-acquired falciparum malaria was diagnosed on Darnley Island in 2001.[9] There had been no locally transmitted malaria on Dauan in 25 years.

Malaria diagnosis was based on clinical presentation, rapid diagnostic tests (RDT, Binax Now® brand) and/or blood films. As falciparum malaria can cause serious morbidity and death, it was particularly important the chosen rapid test could detect this species. The definition of an outbreak included a single locally-acquired case of falciparum malaria. It is much more difficult to define local transmission of *P. vivax* malaria, due to relapses from hypnozoites, so 3 cases of pure vivax malaria in the study period are not described further in this paper.

The 'malaria receptive zone' of northern Australia (approximately north of the 19° parallel, corresponding to the distribution of *An. farauti* s.l.) was described over 60 years ago.[10] This report describes an outbreak in islands with frequent visitors from a country with endemic malaria.

Ethics approval was not sought for publication of this article because it is a public health report and individuals are not identified.

Figure: Map of Torres Strait



Papua New Guinea territory shaded grey

Description of outbreaks

Between 23 February and 26 May 2011, 17 cases of falciparum malaria were notified from the Torres Strait and Northern Peninsula Area Health Service District ('Torres District'). Of these, eight were definitely locally acquired, six were imported, and for three the site of acquisition was unclear (Table). Ten cases occurred in males and seven in females.

Table: Falciparum malaria cases, Torres Strait, 2011, by notification date and place of acquisition

Notification	Age	Nationality	Onset	Acquisition
23 March	3	TSI	17 March	Dauan
23 March	54	TSI	12 March	Saibai
24 March	21	TSI	17 March	Saibai
28 March	45	TSI	12 March	Saibai
31 March	24	TSI	28 March	Saibai
7 April	1	TSI	5 April	Saibai
7 April	33	PNG	5 April	Saibai
11 April	24	TSI	10 April	Saibai
20 March	28	PNG	unknown	PNG
25 March	31	PNG	unknown	PNG
1 April	25	PNG	29 March	PNG
6 April	24	PNG	4 April	PNG
3 May	35	PNG	2 May	PNG
9 May	29	PNG	2 May	PNG
23 February	22	PNG	18 February	PNG/Saibai?
6 April	13	PNG	1 April	PNG/Saibai?
26 May	29	TSI	23 May	PNG/Saibai?

PNG Papua New Guinean
TSI Torres Strait Islander

On 23 March 2011, an outbreak was declared following notification of two locally-acquired *P. falciparum* cases: a man living on Saibai, with symptom onset on 12 March, and a child on Dauan, with onset of illness on 17 March. Neither had travelled from their island in the preceding month.

On 24 March 2011 another case, living in the same house as the child, was notified with onset also 17 March. This person had left Dauan once in the preceding month to make a day trip to Saibai. Five further locally-acquired *P. falciparum* cases were reported on Saibai (one was mixed *P. falciparum* and *P. vivax* infection in a PNG national resident on Saibai for over a month). Three of these people were hospitalised; two were children, and one adult required admission to the intensive care unit because of hepatic and renal failure. One person refused transfer despite a recommendation from clinical staff.

Cases were considered imported if they had been in PNG 9 to 14 days before symptoms developed (corresponding to the incubation period).[11] One woman with imported malaria had an intrauterine foetal death in hospital attributed to malaria. One case with an uncertain site of acquisition was hospitalised for an acute abdomen, and coincidentally found to have malaria.

On Saibai, cases initially occurred at the east and west ends of the community, and later were found throughout the village. All the imported cases had acquired malaria in PNG; most came from the villages of Mabudauan and Sigabaduru. The majority of these were residing in the Saibai community west side when diagnosed (where PNG nationals live in completely unscreened houses).

Blood smears were positive for malaria in all but 1 locally-acquired case. The latter had a positive polymerase chain reaction (PCR) test, performed because of clinical suspicion. Gametocytes were not identified in blood smears of any of the locally-acquired cases, but were seen in 1 imported case notified on 6 April (symptom onset 2 days prior) and in a PNG national notified on 23 February (symptom onset 5 days prior) with uncertain place of acquisition.

Outbreak management

Measures to control the outbreak included passive and active case finding, supervised treatment with gametocytocidal therapy, vector control to reduce mosquito longevity and health promotion. During a funeral lasting several days on Saibai, local staff deemed it culturally inappropriate to commence community activities. This delayed response was a cause of concern. A formal 'Incident Management Team' convened on 8 April, was able to accelerate and coordinate these responses. The outbreak was declared over on 15 June 2011, 59 days after treatment of the last locally-acquired case.

Passive case finding and treatment

The outbreak prompted an urgent re-evaluation of clinical supervision, guidelines, recording, diagnosis and treatment at the Saibai and Dauan clinics. Nurses relatively inexperienced in the management of malaria were encouraged to ring doctors at Thursday Island Hospital and request air transfer if needed, and training was given in the rapid diagnostic test (RDT) use and its limitations. Treatment with effective, gametocytocidal artemether/lumefantrine continued, but the first doses were given under supervision. It was recognised that a problem with smear quality had arisen since on-site smear preparation had been abandoned in favour of sending venous blood specimens to the distant laboratory. This could not be addressed until after the outbreak.

Active case finding

Acute staffing and training issues resulted in a delay in active case finding until 4 April. Twice weekly checks of all 70 households were made, and then reduced to weekly. A nurse and health worker made home visits seeking persons with current fever or within the preceding 48 hours. Anyone identified through the use of this protocol attended the clinic for an RDT and a blood sample was sent for thick and thin films. Two cases were identified early by outreach. All 7 pregnant women were monitored daily for fever from 5 April.

Malaria and mosquito avoidance advice was provided and included: precautions during peak feeding times; long-sleeved, light coloured clothing; personal insect repellent; devices (plug-in or mosquito coils); impregnated bed nets; and surface spray to reduce adult mosquito numbers inside the house.

Vector control

The Cairns Public Health Unit and local district officers commenced vector control at Dauan on 25 March and Saibai 29 March. Vector surveillance methods (CO_2 baited light traps) confirmed large numbers of *An. farauti* s.l. on both islands. These were later identified by PCR as being the subspecies *An. farauti* 1. (Robert Cooper, Australian Army Malaria Institute, personal communication), capable of transmitting malaria.[12] Control strategies included residual insecticide spraying inside houses, thermal fogging in and around houses, and harbourage spraying (vegetation within a 50 m radius of houses). Fogging was timed to correspond to peak *An. farauti* flight activity. All methods utilised pyrethroid insecticides with the aim of killing the older, infected female mosquitoes.

Health promotion and public information

Malaria fact sheets were distributed, and the community was informed about vector control measures by the local public health team and a nurse. Announcements were made on local radio and published in the local Torres Strait newspaper.

Mosquito repellent was distributed and promoted from the health centre and on sale at the local shop. Pyrethrin-impregnated bed nets were supplied to households with pregnant women, children under the age of 5 years and confirmed cases. Due to logistical difficulties, nets only arrived towards the end of the outbreak. There was local support for restriction of cross border travel, but this was not a public health recommendation.

Discussion

Despite regular travel from endemic PNG, *P. falciparum* outbreaks in the Torres Strait are limited and sporadic. This could be because the human populations on outer islands are small and there is effective surveillance. Cases are generally diagnosed and treated early, leaving a limited time window for gametocytes to develop (i.e. for people to become infectious to mosquitoes). *P. falciparum* requires a longer period than *P. vivax* for gametocytes to appear.[13] Also, *An. farauti* may take a low proportion of blood meals from humans because both dogs and feral deer are alternative hosts.[4]

It remains unclear why an outbreak occurred at this time. Calls to exclude migrants from Australian clinics are frequent, and PNG nationals may be reluctant to attend if not very sick. It is likely that delayed diagnosis and treatment of early cases played a role. Whether there was an increased incidence on the PNG side, due to climatic or other factors, is unknown. Health service breakdowns in PNG were noted by Saibai nurses through radio contact. These may have temporarily increased health-seeking traffic to outer island clinics (Teresa O'Brien, Torres Strait and Northern Peninsula Area Health Service District, personal communication).

Infrequent malaria, and no deaths for many years, breeds complacency in both community and clinics. There was anecdotal evidence that mosquito avoidance behaviour (nets, spraying, cleaning yards) had declined since the old days of endemic malaria. There was clearly poor maintenance of mosquito screens, and no regular mosquito control activities by the council.

Although the standard of health care observed on Saibai was high, the outbreak prompted a search for

clinical problems. For example, some diagnostic and reporting criteria were felt to be poorly defined and requiring review.

Prior to the outbreak, two cases diagnosed in February and classified as 'possibly locally acquired' could have actually been an index local case:

- probable falciparum malaria in a Saibai resident with no history of travel, with onset on 5 February (not reported until 15 April);
- *P. falciparum* notified on 23 February (symptom onset on 18 February) in a PNG national who had been on Saibai for 3–4 four weeks beforehand.

The former tested positive on RDT, but negative on 1 smear. Treatment was commenced, but incorrectly interrupted when the microscopy result was obtained. Gametocytes were reported on smears in the second case, i.e. potentially infective to mosquitoes. It is doubtful whether these were the first local cases, with diagnostic uncertainty in the former, and a possible import in the latter (extended incubation is possible in persons from an endemic area).[14]

Data on malaria incidence in PNG are limited. Only 11% of suspected cases are tested, and 6% of reported cases are confirmed.[15] In recent limited surveys from the South Fly region where the treaty villages are situated, less than 10% prevalence of parasitaemia was reported.[16] It was not possible to check for an increased incidence in the South Fly region during or before the outbreak. Similarly, accurate head counts for travel between PNG and the outer islands are unavailable. Prior to the outbreak there appeared to be increased PNG attendances at the Saibai clinic.[1]

There were seven imported *P. falciparum* notifications in the Torres District in the first 4 months of 2011 (compared with 5 cases or less in the previous 5 years), suggesting a higher incidence. Active case finding may have identified more imports than otherwise, given that semi-immune PNG patients may not be very sick.

In the Torres Strait the main malaria vector is *An. farauti* 1.[17] The behaviour and ecology of this species varies enormously by locality. Research into these factors has not been conducted in the Torres Strait, but it would assist in managing future outbreaks by refining vector control approaches. In northern mainland Australia, *An. farauti* 1 typically feeds from 1900 hours to midnight, both outdoors and indoors.[4] Protective measures such as house screens and bed nets are of some value. Locals typically sit in cool open verandahs in the early evening, probably resulting in a greater risk of transmission. Of particular concern were the unscreened houses in west Saibai used by residents from PNG.

The excellent vector breeding conditions on Saibai make control of overall mosquito numbers impractical. Instead, vector control aims to reduce the longevity of adult females, so few would survive long enough to become infective and bite another person. Early case detection and effective gametocytocidal treatment are essential to prevent infection of local *An. farauti*.

To reduce the risk of further malaria outbreaks, the Incident Management Team has proposed: repair of mosquito screens; revised local malaria guidelines; commenced a study of the behaviour and ecology of *An. farauti* in the Torres Strait; developed local education resources; and reviewed regional vector control capacity and equipment.

Although malaria has been eliminated in northern Australia, this outbreak reminds us that cases are still a risk in the Torres Strait, and throughout the country's 'malaria receptive zone'. Travel from malaria endemic areas to this zone will always carry the potential for importation and local transmission. Mosquito elimination on the islands is not feasible because of their geography. Early diagnosis and treatment of cases, and vector control measures in their vicinity, are critical in preventing local outbreaks. Malaria does not currently pose a great public health threat in northern Australia, although there are occasional outbreaks, even on the mainland. It is however, a potentially lethal disease and timely diagnosis, treatment and public health responses are crucial.

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Short reports

Surveillance of potential rabies exposure in Australian travellers returning to South East Queensland

Heidi J Carroll, Brad J McCall, Jason C Christiansen

Introduction

Rabies is endemic in animals in many parts of the world, including much of Asia, the Americas, Africa and Europe. Each year, rabies kills approximately 55,000 people worldwide, most of whom are children.[1] Australia currently remains free of rabies. However in late-2008, rabies was confirmed for the first time in dogs with subsequent human cases in one of our most popular travel destinations, Bali, Indonesia.[2]

Health authorities have recommended post-exposure treatment for potentially exposed returned travellers from Bali, Indonesia, from 1 August 2008. These travellers should receive appropriate post-exposure treatment as per the Australian Immunisation Schedule.[3]

This report summarises potential rabies exposure incidents that have been reported to Public Health Units in the south Brisbane region of Queensland, from January 2008 to the end of April 2012.

This region covers the geographic areas of south Brisbane, Logan and West Moreton, where approximately 1.3 million people reside.

Method

Enhanced surveillance of returned travellers, resident in the geographical region of the Brisbane South and Logan West Moreton Public Health Units, who were bitten or scratched by animals in rabies endemic countries, was conducted in accordance with chapter 3 of the (Queensland) *Public Health Act 2005*.

Travellers with a rabies exposure prone injury were reported to the Public Health Unit via their health professional and/or were self-reported. Data were collected using a standard case report form and included information relating to the type and circumstances of injury, geographic location and treatment.[4] De-identified data were stored in an SQL server database and collated for analysis using Crystal Reports.

Results

Since 2008, 136 travellers with potential rabies exposure from animal bites or scratches have been reported to our public health units. Ages ranged from 2 to 65 years with 42% in the 20–29 year age range. Eighty-four per cent of travellers potentially exposed to rabies reported being bitten (n=114) while the remainder reported scratches or other non-bite exposures (n=22, 16%).

Animals most commonly responsible for injuries were monkeys (n=76, 56%) and dogs (n=41 , 30%), followed by cats (n=10, 7%). Nine people (7%) reported an injury that was due to either a rodent, squirrel, mule, tiger or antelope.

Potential exposures occurred in a wide range of geographic locations. Most were from Indonesia (n=54, 40%), Thailand (n=30, 22%), India (n=8, 7%) and China (n=7, 5%). Of those exposures that occurred in Indonesia, 52 (96%) occurred in Bali with 46 (88%) due to an encounter with a monkey and 3 (6%) due to an encounter with a dog. The remainder were due to contact with other mammals.

Of the 136 returned travellers with potential exposure to rabies, 52 (38%) encounters involved deliberate animal interaction, 38 (28%) were the result of an unprovoked animal attack and 4 (3%) were due to accidental exposure. Only 11 (8%) people had received complete pre-exposure vaccination and only 43 (32%) people were up-to-date with their tetanus vaccination at the time of exposure.

Forty-two people (31%) received appropriate first aid (washing of the wound with soap and water for 5 minutes) at the time of injury. The average time between exposure and commencement of treatment was 17 days. However, if people who received their treatment after 60 days (n=6, 4%), were removed from the analysis the average time between exposure and commencement of treatment was reduced to 7 days.

Sixty-three travellers (46%) commenced their treatment overseas and of these only 5 (4%) received immunoglobulin overseas. Seventy-three travellers

(54%) received their full course of post-exposure prophylaxis in Australia, as per the recommendations in the Australian Immunisation Schedule.

Discussion

There may be under-reporting of non-bite exposures, particularly from returned travellers who visit newly rabies endemic areas such as Bali, Indonesia. The data show that Australian tourists abroad do not understand, or choose to ignore the potential risks involved in deliberate interactions with animals, even animals that they may assume to be 'safe' (e.g. temple monkeys). This is despite travel advice that urges visitors to Indonesia, in particular Bali, to avoid direct contact with dogs, cats, monkeys and other animals.[5]

The data also show that commencement of post-exposure prophylaxis is often delayed or incompletely initiated overseas, with the consequent risk of developing rabies. This may be due to the fact that complete post-exposure rabies treatment in Indonesia may be limited, requiring travel to another country for adequate treatment.[5] Other reasons for delay may include lack of awareness among travellers about the importance of potential rabies exposures and the requirement for treatment.

Concerns around the adequacy of documentation of treatment initiated overseas, may also result in a traveller requiring further doses of rabies vaccine to ensure adequate coverage. There is also the potential risk of tetanus and wound infection if people are not adequately assessed following an exposure to a rabies prone injury.

Conclusion

We recommend that there should be better education of Australian travellers to rabies endemic areas. Travellers should be informed about the risk of bites and scratches from animals, the importance of avoidance and the opportunity for pre-exposure vaccination, including ensuring tetanus vaccination status is up-to-date. In particular, our data show that travellers to Bali should be advised about pre-exposure vaccination and associated risks with visiting monkey temples. Further research may be required to assess the best way to maximise these messages to Australian travellers.

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Quarterly reports

OzFoodNet quarterly report, 1 July to 30 September 2011

The OzFoodNet Working Group

The Australian Government Department of Health and Ageing 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, occurring in Australia from 1 July to 30 September 2011.

Data were received from OzFoodNet epidemiologists in all Australian states and territories. The data in this report are provisional and subject to change, as the results of outbreak investigations can take months to finalise.

During the third quarter of 2011, OzFoodNet sites reported 517 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 9,520 people, of whom 167 were hospitalised. There were 32 deaths reported during these outbreaks. The majority of outbreaks (81%, n=419) were due to person-to-person transmission (Table 1).

Foodborne and suspected foodborne disease outbreaks

There were 34 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission. These outbreaks affected 510 people, resulting in 13 hospitalisations and no deaths (Table 2).

Salmonella enterica was identified as the aetiological agent for 7 outbreaks during this quarter (1 *S.* Saintpaul and 6 *S.* Typhimurium). Of the remaining outbreaks, 5 (15%) were due to *Clostridium perfringens*, 3 (9%) were due to *Campylobacter*, 2 (6%) were due to ciguatera fish poisoning and 2 (6%) due to *Staphylococcus aureus*. One outbreak (3%) was due to norovirus, and 2 (6%) were due to mixed aetiological agents. There were 12 (35%) outbreaks where the aetiological agent remained unknown (Table 2).

Fifteen outbreaks (44% of foodborne outbreaks) reported in this quarter were associated with food prepared in restaurants, 5 outbreaks (15%) with food prepared by a commercial caterers, 4 outbreaks (11%) in private residences and 3 outbreaks (9%) in aged care facilities. The remaining 7 outbreaks (21%) were reported from a range of settings (Table 2).

To investigate these outbreaks, sites conducted 6 cohort studies, 2 case control studies and collected descriptive case series data for 18 investigations, while for 8 outbreaks no individual patient data were collected. As evidence for the implicated food vehicle, investigators collected both microbiological and analytical evidence for 1 outbreak, relied on microbiological evidence in 1 outbreak and analytical evidence alone in 5 outbreaks. Descriptive evidence was obtained in 27 outbreak investigations.

Table 1: Outbreaks and clusters of gastrointestinal illness reported by OzFoodNet, 1 July to 30 September 2011, by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total
Foodborne and suspected foodborne	34	7
Person-to-person	419	81
Animal-to-person	1	<1
Unknown (*Salmonella* cluster)	12	2
Unknown (other)	51	10
Total	517	100

Table 2: Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 July to 30 September 2011 (n=34)

State	Month	Setting prepared	Agent responsible	Number affected (n=510)	Hospitalised (n=13)	Evidence	Responsible vehicles
NSW	July	Restaurant	*S.* Typhimurium MLVA profile 3-9-8-14-523	13	1	D	Raw egg tiramisu
	July	Restaurant	Unknown	13	0	D	Unknown
	July	Restaurant	Unknown	2	0	D	Unknown
	July	Restaurant	Unknown	2	0	D	Unknown
	July	Takeaway	Unknown	3	1	D	Unknown
	August	Commercial caterer	Unknown	25	0	D	Unknown
	August	Restaurant	Unknown	11	0	D	Unknown
	August	Restaurant	*S.* Typhimurium MLVA profile 3-9-7-13-523	6	0	D	Raw egg dressing
	August	Restaurant	*S.* Typhimurium MLVA profile 3-9-7-15-523	3	0	D	Raw egg mayonnaise
	September	Commercial caterer	Unknown	87	0	A	Salad of poached prawns with Thai herbs
	September	Restaurant	Unknown	6	0	D	Madras chicken curry with rice
	September	Restaurant	Unknown	4	0	D	Unknown
	September	Restaurant	Unknown	3	0	D	Unknown
	September	Restaurant	*Campylobacter*	2	0	D	Unknown
NT	September	Fair/festival/mobile service	*S.* Saintpaul	3	0	D	Suspect mango or mango/banana smoothies
Qld	July	Private residence	Ciguatera fish poisoning	3	0	D	Reef fish (unknown species)
	July	Restaurant	*C. perfringens*	3	0	M	Chicken curry
	August	Primary produce	Ciguatera fish poisoning	3	0	D	Coral Trout
	September	Commercial caterer	*S. aureus*	38	1	AM	Fried rice; chicken; egg fu yung; mussels
	September	Hospital	*Campylobacter*	5	5	D	Unknown
SA	August	Institution-other	*Campylobacter*	9	0	A	Unknown
	August	Institution-other	*S.* Typhimurium PT 108 and *Campylobacter*	4	0	D	Unknown
Vic	July	Aged care	*C. perfringens*	11	1	D	Unknown
	July	Restaurant	Unknown	7	0	D	Beef rendang or pork satay
	July	Private residence	Unknown	7	0	D	Raw egg chocolate mousse
	August	Restaurant	*S.* Typhimurium PT 170	14	1	D	Raw egg chocolate mousse
	August	Aged care	*C. perfringens*	7	0	D	Unknown
	August	Private residence	*S.* Typhimurium PT 135	4	2	D	Suspected eggs

Table 2 continued: Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 July to 30 September 2011 (n=34)

State	Month	Setting prepared	Agent responsible	Number affected (n=510)	Hospitalised (n=13)	Evidence	Responsible vehicles
Vic, *cont'd*	September	Reception centre	*C. perfringens*	41	0	A	Roast beef
	September	Commercial caterer	*S. aureus*	28	1	D	Mixed curry meal
	September	Aged care	*C. perfringens*	14	0	D	Suspected roast meats
	September	Private residence	*S.* Typhimurium PT 44	11	0	D	Raw egg tiramisu
WA	July	Restaurant	Norovirus	53	0	A	Salad
	September	Commercial caterer	*Campylobacter, S.* Typhimurium PFGE type 0007, *S.* Infantis	65	0	A	Duck parfait

* No foodborne outbreaks were reported by the Australian Capital Territory or Tasmania.

A Analytical epidemiological association between illness and one or more foods.

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 of tandem repeat analysis

PFGE Pulsed-field gel electrophoresis

PT Phage type.

The following jurisdictional summaries describe key outbreaks and public health actions that occurred in this quarter.

New South Wales

There were 14 reported outbreaks of foodborne or suspected foodborne illness during the quarter.

An investigation of a complaint to the NSW Food Authority (NSWFA) identified illness in 3 restaurant patrons after consuming tiramisu (which included raw egg). One case submitted a specimen that was positive for *S.* Typhimurium multi-locus variable number of tandem repeats analysis (MLVA) profile 3-9-8-14-523. Interviews with other *S.* Typhimurium cases with the same MLVA profile identified 10 ill people who also consumed the tiramisu at this restaurant. A further 6 submitted stool specimens were positive for *S.* Typhimurium, all with the same MLVA profile. The NSWFA inspected the premises with the only food safety issue being the use of raw eggs to make tiramisu. The business agreed to use pasteurised egg product as an alternative.

Six cases of *S.* Typhimurium MLVA profile 3-9-7-13-523 infection were notified in the Newcastle area. These cases represented 3 separate groups that ate at a bakery, with 5 of 6 cases consuming a carrot salad (which contained raw egg), amongst other foods. Food and environmental samples from the bakery were negative for bacterial pathogens. Environmental samples taken during an investigation at the egg farm supplying the bakery were positive for *S.* Typhimurium with a matching MLVA profile to the human cases in this outbreak.

The NSWFA investigated a report of 2 friends who were ill with diarrhoea, abdominal pain, nausea and headache 9–10 hours after sharing a meal. One case submitted a stool sample, which was positive for *Campylobacter*. The likely exposure was a shared meal consumed 5 days prior to onset of illness. Further investigations were unable to identify a food vehicle.

A complaint by 2 separate groups (6 people each) who ate 8 days apart at the same restaurant was investigated by the NSWFA. Abdominal cramps and diarrhoea were reported symptoms in 11 of 12 people, however, no responsible food vehicle was identified. Based on the incubation period of 12 hours and symptoms reported, the outbreak was likely due to toxins from *C. perfringens* or *Bacillus cereus*.

The NSWFA were notified of 6 people from a group of 20 who developed diarrhoea 12 hours after consuming Madras chicken curry at a restaurant. Whilst a responsible vehicle could not be identified, a bacterial toxin from *C. perfringens* or *B. cereus* could have been introduced due to slow cooling.

A public health unit reported a gastrointestinal illness outbreak; with 87 of 500 people experiencing vomiting and diarrhoea 24 hours after attending a commercially catered function at a school. Participants of the dinner were surveyed (59% response rate) and a pre-prepared salad of poached prawns with Thai herbs was statistically associated with illness (odds ratio (OR) = 6.3, confidence interval (CI) 3.2–13.1). There was no food remaining for testing and there were no stool samples submitted, however, the clinical picture suggests a viral pathogen.

Following a party at a bar, 3 of 25 attendees experienced nausea, abdominal pain and diarrhoea with an incubation period ranging 7–9 hours. Based on the incubation period and symptom profile, with no food or clinical specimens collected, a bacterial toxin was suspected as the cause of the outbreak.

No cause was found for the remaining 7 outbreaks.

Northern Territory

There was a single outbreak of foodborne or suspected foodborne disease reported during the quarter.

The outbreak was identified whilst undertaking a cluster investigation into *S.* Saintpaul cases; with 5 of 8 interviewed cases consuming fruit smoothies from a local market. Locally grown mangoes were thought to be the vehicle with trace back investigations on mangoes used at the stalls attempted, although this was difficult as stall owners/operators used fruit sourced from different local suppliers. Fruit drink stall owner/operators and fruit and vegetable stall owners in Darwin were provided with advice on washing, sanitising and preparing fresh fruit.

Queensland

There were 5 reported outbreaks of foodborne or suspected foodborne disease during the quarter.

Three cases (2 adults and 1 child) of ciguatera fish poisoning were notified to authorities following the consumption of an unknown species of reef fish at a private residence in July. The fish was a private catch with no food samples available for testing. Symptoms of numbness or tingling of skin, reversed temperature sensation, diarrhoea, joint and muscle pain all indicated ciguatera fish poisoning.

Another 3 cases, from a group of six, with suspected ciguatera fish poisoning in adults were notified to authorities. The cases experienced diarrhoea, abdominal cramps, numbness and tingling of extremities, reversed temperature sensation and joint pain 24–48 hours after consumption of the

fish. Coral trout along with barracuda were submitted for testing, with the coral trout positive while the barracuda samples were negative for ciguatoxins.

Three people from the same household ate a chicken curry from a local restaurant and reported illness 8 to 14 hours after consumption of the meal. Microbiological testing of left-over curry identified the presence of *C. perfringens* toxins and coagulase positive staphylococci. One faecal specimen that was collected during the investigation was positive for *C. perfringens*. The cooked curry was reported to be placed into 4 litre capacity plastic tubs, which were held at room temperature for 30–45 minutes before being placed into a freezer. As no temperature monitoring was in place at the restaurant an improvement notice was served to ensure regular temperature monitoring is performed and to cease the process of bulk cooking, cooling and reheating of food.

Authorities investigated a suspected foodborne outbreak among 115 guests who attended a catered wedding. A retrospective cohort study was conducted as part of the investigation where clinical and food history information was obtained on 94 attendees. Of those interviewed, 38 cases (aged between 2–63 years, 1:1 male:female ratio) experienced either vomiting and/or diarrhoea following the consumption of food at the reception or left-over food from the reception on subsequent days. The median incubation period was 5 hours (range 2–38 hours) and the median duration of illness was 1 day.

The cohort study identified multiple foods served at the reception (fried rice, egg yu fung, chicken and mussels) were associated with an increased risk of illness (relative risk (RR) 1.9 to 2.1). High levels of coagulase positive staphylococci and emetic and diarrhoeal strains of *B. cereus* were detected in mixed left-over samples of prawns, pork, corned beef, mussels, noodles, curry, rice, chicken, egg fu yung, taro and seafood salad. High levels of *C. perfringens* were also reported in both samples of fried rice and staphylococcal enterotoxin detected in the fried rice and chicken samples. Both samples of corned meat contained high levels of *Escherichia coli*. Coagulase positive staphylococci were also detected on a chopping board that was used by the caterer. Five faecal specimens and 1 vomitus specimen from persons reporting illness had light to moderate growth of coagulase positive staphylococci. Staphylococcal enterotoxin and *B. cereus* were not detected in any of the clinical specimens. Inappropriate timing of food preparation resulting in long holding times, inadequate food storage, inappropriate defrosting of food and lack of knowledge in safe food handling practice were major contributing factors in this outbreak that resulted in the proliferation and survival of pathogens.

South Australia

There were 2 reported outbreaks of foodborne or suspected foodborne disease during the quarter.

Investigators were notified of a *Campylobacter* infection in a person who attended a team building work function with several other attendees reporting similar symptoms, after which a cohort study was undertaken. The team building function involved 14 people preparing and sharing a meal together. A questionnaire collecting information on demographics, illness and menu items prepared and consumed was undertaken along with discussions with the food supplier, to identify sources of infection. Nine of 12 people had symptoms of gastrointestinal illness with stool samples from 2 attendees testing positive for *Campylobacter*. No food was available for testing.

There was a cluster of 2 *Campylobacter* cases co-infected with *S.* Typhimurium phage type (PT) 108 who worked at a common workplace in remote South Australia. Active case finding further identified a case of *Campylobacter* and a case of *S.* Typhimurium PT 108. The workplace had on-site kitchen facilities, which provided the majority of the food for the workers, with an inspection identifying several food handling issues such as storing raw and cooked food in close proximity and inadequate hand washing facilities in the kitchen. Workers were also not able to maintain appropriate temperature control of their meals when they were in the field.

Victoria

There were 10 reported outbreaks of foodborne or suspected foodborne disease during the quarter.

Two separate groups of people complained of illness after eating at a restaurant with all 4 people in the first group and all 3 people in the second group reporting nausea and diarrhoea approximately 8 hours after their meal. Duration of illness was less than 24 hours. Beef curry and coconut rice were consumed by both groups, with no other common food items identified. Different batches of beef curry, rice and chicken and pork satays were sampled, with *C. perfringens* detected in the pork satay sample. No faecal specimens were collected but the incubation period and illness profile was consistent with *C. perfringens* enterotoxin as the causative agent.

Authorities were notified of 38 of 184 members of a sports club with onset of diarrhoea and abdominal pain the day after attending a dinner. Sixty-six of the 138 attendees were able to be interviewed and 41 reported symptoms. Analysis of food history information showed a statistically significant association with the consumption of the roast beef

meal and illness, RR 12.7 (CI 3.3–48.0). *C. perfringens* enterotoxin was detected in 11 of 12 faecal specimens collected. The beef was roasted the day before the dinner, then kept in the cool-room. The following day the meat was sliced thinly on a slicing machine and then placed into a warmer, without being re-heated. The meat slicer was found to be unclean with pieces of meat and meat juices behind the blade. No leftover food was available for testing.

There were 3 outbreaks in aged care facilities during this quarter where the aetiology was either confirmed or suspected as being caused by *C. perfringens* enterotoxin. Investigators were unable to identify a food source in these outbreaks.

A doctor notified a *Salmonella* case to the Communicable Disease Prevention and Control Unit reporting that 10 family members attended a dinner where chocolate mousse made with raw eggs was served for dessert. Seven of 10 people who consumed the chocolate mousse developed diarrhoea after the function. One of these cases had a faecal specimen collected that was positive for *S*. Typhimurium PT 9. The remaining cases had an illness profile consistent with this aetiology.

Following the receipt of 2 doctors' notifications of *Salmonella* from the same rural town, an investigation revealed that both cases had attended a cooking class at a restaurant prior to onset of illness. Of those interviewed, 13 of 21 attendees and 1 staff member experienced symptoms of diarrhoea and/or vomiting with a median incubation period of 24 hours. Eight cases presented to a doctor. One was admitted to hospital and 4 cases were confirmed as being *S*. Typhimurium PT 170. High risk foods served included a chicken noodle dish and chocolate mousse containing raw eggs. Fourteen of 21 people who ate the chocolate mousse were ill. The chef reported that they do not routinely make food containing raw eggs in the café and catering business. No leftover food or eggs were available for sampling.

Two year 9 students from a boarding school were notified with *Salmonella* after attending a cooking class at a teacher's home. Five students participated in the cooking class where they each made their own batch of ravioli filled with ricotta cheese, roasted pine nuts, fresh parsley, basil and lemon zest. Four of the students tasted the uncooked pasta dough containing the raw eggs. The 5th student and the teacher did not taste the uncooked dough and were not ill. The two students initially notified were confirmed with *S*. Typhimurium PT 135. There were 3 eggs leftover at the end of the class, and on the following day the teacher lightly scrambled these for her young children. Both children were then ill, and one has been confirmed with *S*. Typhimurium PT 135. The eggs used in the cooking class were traced back to a New South Wales producer and this information was forwarded to the NSW Food Authority.

During routine investigation of a single case of *Salmonella* it was found that several members of 3 families had consumed tiramisu containing raw eggs. One family made the tiramisu and ate it on the same day, and leftovers were taken to be shared with 2 other family groups on the following day. Eleven of 12 people who ate the tiramisu became ill, and a 13th person who did not eat the tiramisu was not ill. Faecal specimens were collected from 3 cases and all 3 were confirmed with *S*. Typhimurium PT 44. No leftover tiramisu or eggs were available for testing.

Ambulance Victoria notified the Victorian Department of Health when a large number of ambulances were requested to assist a number of people with vomiting after consuming lunch at a community centre. Vomiting and/or diarrhoea was reported by 28 of 46 people who were interviewed with a median incubation period of 5 hours after consumption of the lunch. Nine cases presented to hospital, with one being admitted overnight, and 9 cases presented to their general practitioner. No faecal or vomit specimens were collected. The lunch had been provided by a catering company and consisted of a mixture of beef, eggplant, fish and sauces on a bed of rice. Food preparation commenced at 6:00 am, with meals placed into containers from 9:30 am and collected for delivery by car at 11 am. Some meals were placed in a warm oven at the community centre, and consumed at approximately 12:30 pm. High levels of *S. aureus* and *S. aureus* toxin were detected in the meals from 2 unopened containers sampled from the community centre.

Western Australia

There were 2 reported outbreaks of foodborne or suspected foodborne disease during the quarter.

In 1 outbreak, there were 53 cases of gastroenteritis (15 staff and 38 patrons of a hotel), of which 48 were interviewed (13 staff and 35 patrons). All experienced similar symptoms (diarrhoea, vomiting and fever) and duration of illness. For patrons, the median incubation period was 31 hours and the median duration of diarrhoea was 11 hours. One faecal specimen from a patron was positive for norovirus. A case control study was conducted (included 33 ill and 31 not ill patrons) and multivariate analysis showed that eating any salads (OR 5.31, CI 1.31–21.57) and aioli (OR 12.75, CI 1.36–120) were significantly associated with illness. While food samples and environmental swabs were negative for routine pathogens, norovirus testing of foods is not currently available in Western Australia. At least 2 staff-members were ill prior to the date that ill

patrons attended the hotel. There were also reports of ill staff returning to work before the Western Australian Department of Health's recommended period for exclusion from work was completed (until asymptomatic, including normal stools for at least 48 hours). Most of the ill staff had also eaten meals at the hotel. It is most likely that the illness among staff and patrons was due to norovirus and the patrons became ill after eating salad and possibly other foods contaminated with norovirus. It is possible, but cannot be proven, that one or more infected food handlers may have been responsible for the contamination of food. However, other mechanisms of transmission such as person-to-person (e.g. via contaminated surfaces in toilets/bathrooms) may also have caused some illness, especially among staff.

An outbreak of gastroenteritis caused by *Campylobacter* and *Salmonella* occurred amongst attendees at a gala dinner at a function centre. Some cases had mixed infections, with 2 cases positive for *Campylobacter* and *S.* Typhimurium PFGE type 0007; one case was positive for *Campylobacter* and *S.* Infantis; and 3 cases were positive for *Campylobacter* only. A self-administered questionnaire regarding illness and food consumed was completed by 136 of 705 people who attended the dinner with 65 people reporting symptoms of gastroenteritis (minimum attack rate of 9.2%). The median incubation period was 60 hours and median duration of illness was 5 days. Symptoms reported by cases included diarrhoea, abdominal pain, nausea, chills, fever, vomiting and bloody diarrhoea. The multivariate analysis of significant food exposures using the information from 65 ill attendees and 71 non-ill attendees found that illness was statistically associated with consumption of duck parfait (OR 13, 95% CI 1.9–91.5, *P* 0.01). None of the parfait served at the dinner was available to be tested at the time of the investigation.

Australian Capital Territory and Tasmania

There were no foodborne or suspected foodborne outbreaks investigated during the quarter.

Cluster investigations

During the quarter, OzFoodNet sites investigated a number of clusters, with 7 due to *S.* Typhimurium, 2 clusters due to *S.* Saintpaul and 1 cluster due to each of *S.* Montevideo, *S.* Singapore and norovirus. A cluster of Shiga-toxin producing *E. coli* (STEC) was investigated with 10 cases linked to an agricultural show, with two petting zoos at the show being identified as possible sources.

Both Western Australia and South Australia investigated an increase in reported cases of *S.* Typhimurium PT 135a during the quarter. Western Australia was notified of an additional 15 cases associated with a cluster investigation that commenced in the first quarter of 2011. Eggs and chicken were consumed by 88% and 82% of the additional cases respectively, with an investigation to trace the origin of chicken isolates continuing. The South Australian cluster involved a primary school, with date of onset of cases spread over a month and indicated potential person-to-person transmission. An environmental inspection was conducted and the classrooms and all toys were sanitised.

Comments

The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission, and in this quarter, 81% of outbreaks (n=419) were transmitted via this route. *S.* Typhimurium continues to be a leading cause of foodborne outbreaks in Australia, with 44% (7/16) of outbreaks with a known aetiology due to this *Salmonella* serotype.

Foodborne disease outbreak investigations this quarter have highlighted a range of high risk practices, many occurring in food service settings. Fifteen foodborne disease outbreaks this quarter were associated with foods prepared in a restaurant, while a further six were associated with foods prepared by caterers. Catering for large groups presents particular challenges in ensuring the adequate temperature control of stored foods and preventing cross contamination between raw and cooked foods. There may often be inadequate facilities for the safe storage and handling of large quantities of food at the location where it is to be served.

The consumption of dishes containing raw or undercooked eggs continues to account for a large proportion of outbreaks of foodborne disease in Australia. Of the 34 foodborne or suspected foodborne outbreaks during the quarter, 21% (n=7) were egg-associated with six of these outbreaks due to consumption of ready-to-eat foods containing raw eggs, such as raw egg mayonnaise, chocolate mousse, tiramisu and dressings containing raw egg.

A limitation of the outbreak data provided by OzFoodNet sites was the potential variation in the way investigators interpreted features of the outbreak depending on the evidence available. Changes in the number of foodborne outbreaks should be interpreted with caution due to the small number each quarter.

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Communicable diseases surveillance

Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 57,857 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 January and 31 March 2012 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

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

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

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 January to 31 March 2012, by date of diagnosis

Disease	State or territory								Total 1st quarter 2012	Total 4th quarter 2011	Total 1st quarter 2011	Last 5 years mean 1st quarter	Ratio	Year to date 2012	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Hepatitis B (newly acquired)*	0	8	1	15	5	3	16	5	53	50	49	62.8	0.8	53	62.8
Hepatitis B (unspecified)†	23	624	50	219	85	13	446	195	1,655	1,573	1,696	1,734.2	1.0	1,655	1,734.2
Hepatitis C (newly acquired)*,‡	6	13	0	NN	10	6	0	24	59	44	108	94.6	0.6	59	94.6
Hepatitis C (unspecified)†	31	924	59	597	110	63	598	233	2,615	2,349	2,541	2,803.8	0.9	2,615	2,803.8
Hepatitis D	0	2	0	2	0	0	2	0	6	4	11	9.4	0.6	6	9.4
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	0.4
Campylobacteriosis§	158	NN	57	1,372	669	234	1,707	600	4,797	4,313	4,884	4,541.4	1.1	4,797	4,541.4
Cryptosporidiosis	9	211	131	848	86	2	97	59	1,443	408	637	1,180.2	1.2	1,443	1,180.2
Haemolytic uraemic syndrome	0	2	0	2	0	0	1	0	5	4	4	5.2	1.0	5	5.2
Hepatitis A	0	12	0	9	4	0	14	4	43	40	44	69.2	0.6	43	69.2
Hepatitis E	0	1	0	3	0	0	9	0	13	9	16	12.6	1.0	13	12.6
Listeriosis	0	10	0	1	2	0	10	3	26	22	19	25.2	1.0	26	25.2
STEC, VTEC‖	0	4	0	13	14	5	3	0	39	36	19	34.0	1.1	39	34.0
Salmonellosis	87	1,013	149	1,062	288	112	808	428	3,947	2,899	4,753	3,709.2	1.1	3,947	3,709.2
Shigellosis	2	42	61	18	19	0	33	15	190	139	161	184.4	1.0	190	184.4
Typhoid	1	16	2	8	1	0	18	10	56	31	57	39.4	1.4	56	39.4
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	1	1.0	0.0	0	1.0
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2012, by date of diagnosis

Disease	State or territory								Total 1st quarter 2012	Total 4th quarter 2011	Total 1st quarter 2011	Last 5 years mean 1st quarter	Ratio	Year to date 2012	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶,**	351	5,869	743	5,001	1,371	483	5,506	3,103	22,427	19,484	20,750	16,672.6	1.3	22,427	16,672.6
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.6	0.0	0	0.6
Gonococcal infection**	13	986	498	766	107	5	653	573	3,601	3,189	2,953	2,293.2	1.6	3,601	2,293.2
Syphilis – congenital**	0	0	0	0	0	0	0	0	0	1	4	1.6	0.0	0	1.6
Syphilis < 2 years duration**	5	93	4	82	15	0	109	25	333	287	370	341.6	1.0	333	341.6
Syphilis > 2 years or unspecified duration**	2	60	10	48	NN	0	146	28	294	311	324	332.2	0.9	294	332.2
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Haemophilus influenzae type b	0	0	0	1	1	0	1	0	3	2	3	4.2	0.7	3	4.2
Influenza (laboratory confirmed)	11	229	21	390	138	9	187	264	1,249	2,836	2,648	873.8	1.4	1,249	873.8
Measles	0	0	0	1	2	0	6	0	9	50	79	41.6	0.2	9	41.6
Mumps	1	15	0	7	1	0	11	3	38	41	39	60.4	0.6	38	60.4
Pertussis	90	2,037	125	1,801	167	243	1,197	1,506	7,166	10,165	10,679	5,459.2	1.3	7,166	5,459.2
Pneumococcal disease (invasive)	10	53	21	40	19	6	55	28	232	375	225	208.2	1.1	232	208.2
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	4	0	2	2	0	4	1	13	8	22	12.0	1.1	13	12.0
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	1	0	0	0	1	0	1	1.6	0.6	1	1.6
Varicella zoster (chickenpox)††	1	NN	26	49	115	4	141	60	396	646	377	334.4	1.2	396	334.4
Varicella zoster (shingles)††	7	NN	60	18	395	61	277	240	1,058	1,052	1,015	705.4	1.5	1,058	705.4
Varicella zoster (unspecified)††	22	NN	1	1,045	27	30	707	258	2,090	2,167	1,773	1,459.4	1.4	2,090	1,459.4
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	4	0	0	0	0	4	8	4	4.6	0.9	4	4.6
Barmah Forest virus infection	1	104	15	242	13	1	12	64	452	345	837	624.0	0.7	452	624.0
Dengue virus infection	5	91	51	82	18	3	96	296	642	213	366	357.4	1.8	642	357.4
Japanese encephalitis virus infection	0	0	0	1	0	0	0	0	1	0	0	0.0	0.0	1	0.0
Kunjin virus infection‡‡	0	0	0	0	0	0	0	0	0	1	0	0.6	0.0	0	0.6
Malaria	0	13	3	29	2	5	15	5	72	110	118	122.6	0.6	72	122.6
Murray Valley encephalitis virus infection‡‡	0	0	0	1	0	0	0	0	1	1	8	2.2	0.5	1	2.2
Ross River virus infection	2	198	95	797	72	14	87	1,016	2,281	747	3,047	2,045.6	1.1	2,281	2,045.6

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2012, by date of diagnosis

Disease	State or territory								Total 1st quarter 2012	Total 4th quarter 2011	Total 1st quarter 2011	Last 5 years mean 1st quarter	Ratio	Year to date 2012	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	0.4	0.0	0	0.4
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Brucellosis	0	1	0	6	0	0	0	1	8	9	12	9.2	0.9	8	9.2
Leptospirosis	0	7	1	33	0	0	4	0	45	21	125	61.2	0.7	45	61.2
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	3	0	0	0	0	4	2	9	25	23	19.8	0.5	9	19.8
Q fever	0	30	1	56	2	0	1	3	93	90	81	95.8	1.0	93	95.8
Tularaemia	0	0	0	0	0	0	0	0	0	1	1	1.0	0.0	0	1.0
Other bacterial infections															
Legionellosis	0	34	2	15	6	1	15	19	92	100	81	69.2	1.3	92	69.2
Leprosy	0	0	0	0	0	0	0	0	0	3	0	2.4	0.0	0	2.4
Meningococcal infection§§	1	6	1	13	1	0	7	3	32	48	58	47.4	0.7	32	47.4
Tuberculosis	3	60	6	50	25	1	85	38	268	343	298	289.2	0.9	268	289.2
Total	842	12,775	2,194	14,749	3,793	1,304	13,088	9,112	57,857	54,600	61,321			57,857	

* Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis.

† Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined.

‡ In Queensland, includes incident hepatitis cases.

§ Not notifiable in New South Wales.

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

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

** In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode (e.g. perinatal infections, epidemic gonococcal conjunctivitis).

†† Ratio of current quarter total to the mean of last 5 years for the same quarter. Ratios for varicella zoster (chickenpox), varicella zoster (shingles) and varicella zoster (unspecified) are based on 4 years of data.

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

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

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.

Table 3: Notification rates of diseases, 1 January to 31 March 2012, by state or territory. (Annualised rate per 100,000 population)

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)*	0.0	0.4	1.7	1.3	1.2	2.4	1.2	0.9	0.9
Hepatitis B (unspecified)†	25.6	34.5	87.1	19.4	20.7	10.2	32.2	34.0	29.6
Hepatitis C (newly acquired)*	6.7	0.7	0.0	NN	2.4	4.7	0.0	4.2	1.3
Hepatitis C (unspecified)†,‡	34.6	51.1	102.8	52.9	26.8	49.6	43.1	40.6	46.8
Hepatitis D	0.0	0.1	0.0	0.2	0.0	0.0	0.1	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis§	176.1	NN	99.3	121.5	162.7	184.4	123.1	104.5	127.0
Cryptosporidiosis	10.0	11.7	228.1	75.1	20.9	1.6	7.0	10.3	25.8
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.2	0.0	0.0	0.1	0.0	0.1
Hepatitis A	0.0	0.7	0.0	0.8	1.0	0.0	1.0	0.7	0.8
Hepatitis E	0.0	0.1	0.0	0.3	0.0	0.0	0.6	0.0	0.2
Listeriosis	0.0	0.6	0.0	0.1	0.5	0.0	0.7	0.5	0.5
STEC,VTEC‖	0.0	0.2	0.0	1.2	3.4	3.9	0.2	0.0	0.7
Salmonellosis	97.0	56.0	259.5	94.1	70.0	88.3	58.3	74.6	70.7
Shigellosis	2.2	2.3	106.2	1.6	4.6	0.0	2.4	2.6	3.4
Typhoid fever	1.1	0.9	3.5	0.7	0.2	0.0	1.3	1.7	1.0
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¶,**	391.2	324.3	1,294.0	442.9	333.4	380.6	397.0	540.5	401.5
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	14.5	54.5	867.3	67.8	26.0	3.9	47.1	99.8	64.5
Syphilis – congenital**	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years duration**	5.6	5.1	7.0	7.3	3.6	0.0	7.9	4.4	6.0
Syphilis > 2 years or unspecified duration†,**	2.2	3.3	17.4	4.3	NN	0.0	10.5	4.9	5.7
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	0.0	0.1	0.2	0.0	0.1	0.0	0.1
Influenza (laboratory confirmed)	12.3	12.7	36.6	34.5	33.6	7.1	13.5	46.0	22.4
Measles	0.0	0.0	0.0	0.1	0.5	0.0	0.4	0.0	0.2
Mumps	1.1	0.8	0.0	0.6	0.2	0.0	0.8	0.5	0.7
Pertussis	100.3	112.6	217.7	159.5	40.6	191.5	86.3	262.3	128.3
Pneumococcal disease (invasive)	11.1	2.9	36.6	3.5	4.6	4.7	4.0	4.9	4.2
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.2	0.0	0.2	0.5	0.0	0.3	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	0.2	0.0	0.0	0.0	0.0

Table 3 *continued*: Notification rates of diseases, 1 January to 31 March 2012, 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)	1.1	NN	45.3	4.3	28.0	3.2	10.2	10.5	10.5
Varicella zoster (shingles)	7.8	NN	104.5	1.6	96.1	48.1	20.0	41.8	28.0
Varicella zoster (unspecified)	24.5	NN	1.7	92.6	6.6	23.6	51.0	44.9	55.4
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.1
Barmah Forest virus infection	1.1	5.7	26.1	21.4	3.2	0.8	0.9	11.1	8.1
Dengue virus infection	5.6	5.0	88.8	7.3	4.4	2.4	6.9	51.6	11.5
Japanese encephalitis virus infection	0.0	0.0	0.0	0.1	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	0.0	0.7	5.2	2.6	0.5	3.9	1.1	0.9	1.3
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	2.2	10.9	165.5	70.6	17.5	11.0	6.3	177.0	40.8
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	0.5	0.0	0.0	0.0	0.2	0.1
Leptospirosis	0.0	0.4	1.7	2.9	0.0	0.0	0.3	0.0	0.8
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.2	0.0	0.0	0.0	0.0	0.3	0.3	0.2
Q fever	0.0	1.7	1.7	5.0	0.5	0.0	0.1	0.5	1.7
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	0.0	1.9	3.5	1.3	1.5	0.8	1.1	3.3	1.6
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection‡‡	1.1	0.3	1.7	1.2	0.2	0.0	0.5	0.5	0.6
Tuberculosis	3.3	3.3	10.4	4.4	6.1	0.8	6.1	6.6	4.8

* Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis.

† Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined.

‡ In Queensland, includes incident hepatitis C cases.

§ Not notifiable in New South Wales.

|| Infection with Shiga toxin/verotoxin-producing *Escherichia coli* (STEC/VTEC).

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

** In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode (e.g. perinatal infections, epidemic gonococcal conjunctivitis).

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

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

NEC Not elsewhere classified.

NN Not notifiable.

NDP No data provided.

Additional reports

Australian childhood immunisation coverage

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

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months of age, for 3-month birth cohorts of children at the stated ages between October and December 2011. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, pneumococcal conjugate, varicella, or meningococcal C conjugate vaccines, and is outlined in more detail below.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing Haemophilus influenzae *type b (Hib) vaccine or 3 doses of any other Hib vaccine, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of all other hepatitis B vaccines. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 or 4 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP containing Hib vaccine or 4 doses of any other Hib vaccine, 3 or 4 doses of Comvax hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of a measles, mumps and rubella (MMR)-containing vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 or 5 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.*

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: brynleyh@chw.edu.au

The percentage of children 'fully immunised' by 12 months of age for Australia decreased marginally from the previous quarter by 0.4 of a percentage point to 91.4% (Table 1). Important changes in coverage were seen only in the Northern Territory with coverage for 'fully immunised', DTP, polio, Hib and hepatitis B vaccines increasing by almost 5 percentage points. However, this apparent increase in coverage is a correction from the previous quarter where an administrative delay in data reported to the ACIR from the Northern Territory occurred.

The percentage of children 'fully immunised' by 24 months of age for Australia increased marginally from the previous quarter by 0.1 of a percentage point to 92.7% (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

The percentage of children 'fully immunised' by 60 months of age for Australia increased from the previous quarter by 0.2 of a percentage point to 90.1% (Table 3). This is the first time coverage for this milestone has reached 90% since coverage was first calculated at the 72-month age milestone in March 2002. There were no important changes in coverage for any individual vaccines due at 60 months of age or by jurisdiction.

The Figure shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007). Coverage at 60 months of age is close to the coverage levels attained at 12 and 24 months.

Table 1. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2010; assessment date 31 March 2012

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,197	23,584	889	14,505	4,842	1,527	17,673	7,603	71,820
Diphtheria, tetanus, pertussis (%)	93.6	91.6	92.6	91.9	92.1	93.1	92.7	90.6	92.0
Poliomyelitis (%)	93.6	91.6	92.6	91.8	92.1	93.1	92.7	90.6	91.9
Haemophilus influenzae type b (%)	93.4	91.5	92.5	91.7	92.1	93.0	92.5	90.5	91.8
Hepatitis B (%)	92.7	91.3	92.4	91.6	92.0	92.9	92.3	90.1	91.6
Fully immunised (%)	92.7	91.1	92.4	91.4	91.9	92.9	92.1	90.0	91.4
Change in fully immunised since last quarter (%)	-0.6	-0.5	+4.9	-0.1	+0.2	+0.0	-0.9	-0.6	-0.4

Table 2. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2009; assessment date 31 March 2012*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,343	24,754	923	15,165	4,913	1,621	18,525	8,007	75,251
Diphtheria, tetanus, pertussis (%)	96.0	94.8	95.7	95.0	94.8	95.3	95.5	93.2	94.9
Poliomyelitis (%)	96.1	94.7	95.7	95.0	94.8	95.3	95.5	93.1	94.8
Haemophilus influenzae type b (%)	95.8	95.0	96.0	95.0	94.8	95.6	95.4	93.5	94.9
Measles, mumps, rubella (%)	95.2	93.8	95.3	94.3	94.0	94.8	94.7	92.4	94.1
Hepatitis B (%)	94.9	94.3	95.5	94.5	94.5	95.2	95.0	92.7	94.4
Fully immunised (%)	93.5	92.4	94.4	93.1	92.5	93.7	93.4	90.7	92.7
Change in fully immunised since last quarter (%)	-0.1	-0.1	-0.2	+0.6	-0.1	+0.3	+0.3	-0.2	+0.1

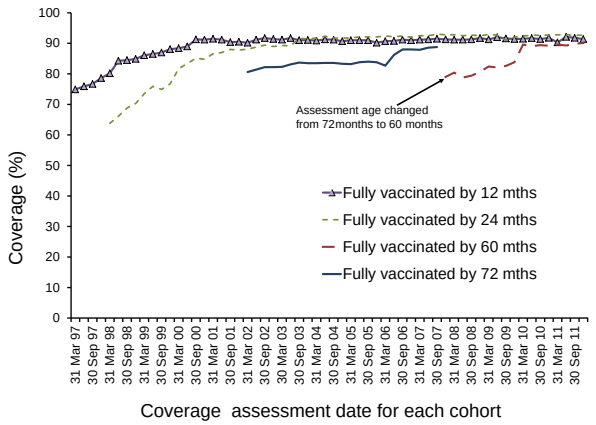
* The 12 months age data for this cohort were published in *Commun Dis Intell* 2011;35(4):328.

Table 3. Percentage of children immunised at 5 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2006; assessment date 31 March 2012

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,190	24,154	878	15,014	4,868	1,641	18,332	7,784	73,861
Diphtheria, tetanus, pertussis (%)	91.6	90.8	91.2	91.2	87.6	91.4	92.0	88.0	90.7
Poliomyelitis (%)	91.5	90.7	91.2	91.1	87.5	91.4	91.9	87.9	90.6
Measles, mumps, rubella (%)	91.5	90.7	91.1	91.0	87.3	91.5	91.8	87.7	90.5
Fully immunised (%)	91.2	90.3	90.8	90.7	86.9	91.0	91.4	87.2	90.1
Change in fully immunised since last quarter (%)	-1.3	+0.6	+3.2	+0.3	-1.2	-0.1	-0.2	+0.4	+0.2

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: brynleyh@chw.edu.au

Figure: Trends in vaccination coverage, Australia, 1997 to 31 December 2011, by age cohorts



Australian Sentinel Practices Research Network

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

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

Since 2010, ASPREN GPs have been collecting nasal swab samples for laboratory testing, allowing for viral

testing of 25% of ILI patients for a range of respiratory viruses including influenza A, influenza B and H1N1(2009).

The list of conditions reported is reviewed annually by the ASPREN management committee. In 2012, four conditions are being monitored. They include influenza-like illness (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 *2008; 32:135.*

Reporting period 1 January to 31 March 2012

Sentinel practices contributing to ASPREN were located in all 8 jurisdictions in Australia. A total of 135 general practitioners contributed data to ASPREN in the 1st quarter of 2012. Each week an average of 113 general practitioners provided information to ASPREN at an average of 10,423 (range 4,814 to 12,335) consultations per week and an average of 106 (range 72–150) notifications per week.

ILI rates reported from 1 January to 31 March 2012 averaged 4 cases per 1,000 consultations (range 2–6 cases per 1,000 consultations). This was slightly lower compared with rates in the same reporting period in 2011, which averaged 5 cases per 1,000 consultations (range 2–6 cases per 1,000 consultations) (Figure 1).

ILI swab testing has continued during 2012. The most commonly reported virus during this reporting period was rhinovirus (11% of all swabs performed), with the second most common virus being influenza A (untyped) (10% of all swabs performed).

From the beginning of 2012 to the end of week 13, 21 cases of influenza had been detected, the majority of these being influenza A (untyped) (10% of all swabs performed), influenza B (3% of all swabs performed) and the remainder H1N1(2009) (0.5% of all swabs performed) (Figure 2).

Figure 1: Consultation rates for influenza like illness, ASPREN, 1 January 2011 to 31 March 2012, by week of report

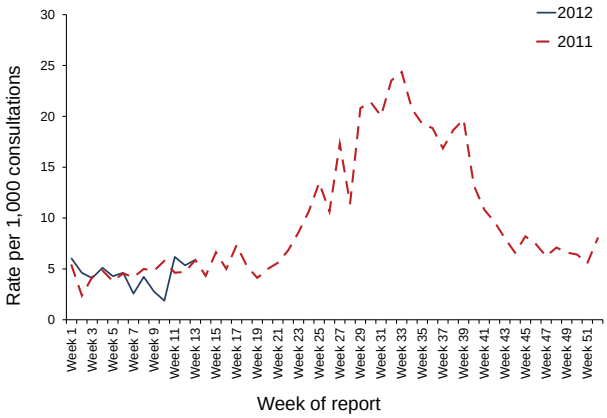
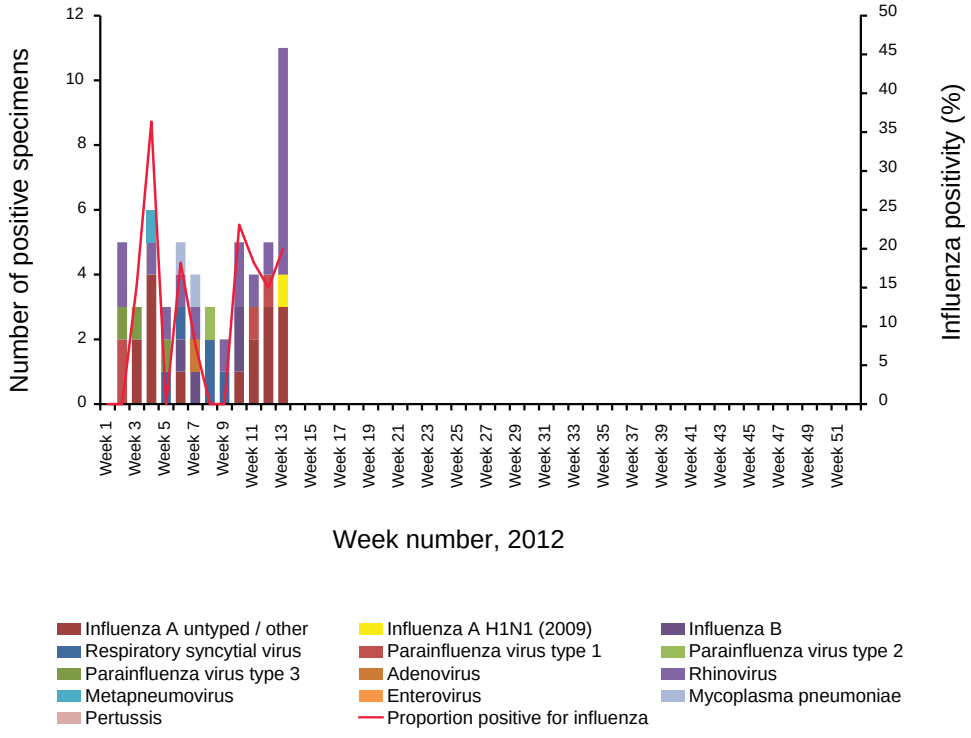
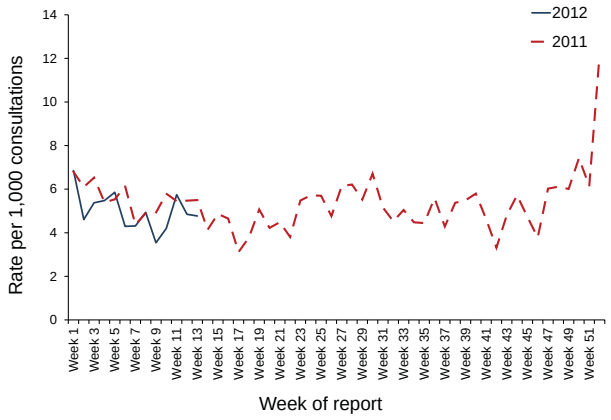


Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January to 31 March 2012, by week of report



During this reporting period, consultation rates for gastroenteritis averaged 5 cases per 1,000 consultations (range 4–7 cases per 1,000, Figure 3). This was similar to rates in the same reporting period in 2011 where the average was 6 cases per 1,000 consultations (range 4–7 cases per 1,000).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 1 January 2011 to 31 March 2012, by week of report



Varicella infections were reported at a lower rate for the 1st quarter of 2012 compared with the same period in 2011. From 1 January to 31 March 2012, recorded rates for chickenpox averaged 0.1 cases per 1,000 consultations (range 0–0.3 cases per 1,000 consultations, Figure 4).

In the 1st quarter of 2012, reported rates for shingles averaged 0.9 cases per 1,000 consultations (range 0.5–2.1 cases per 1,000 consultations, Figure 5), slightly lower compared with the same reporting period in 2011 where the average shingles rate was 1.0 case per 1,000 consultations (range 0.4–1.7 cases per 1,000 consultations).

Figure 4: Consultation rates for chickenpox, ASPREN, 1 January 2011 to 31 March 2012, by week of report

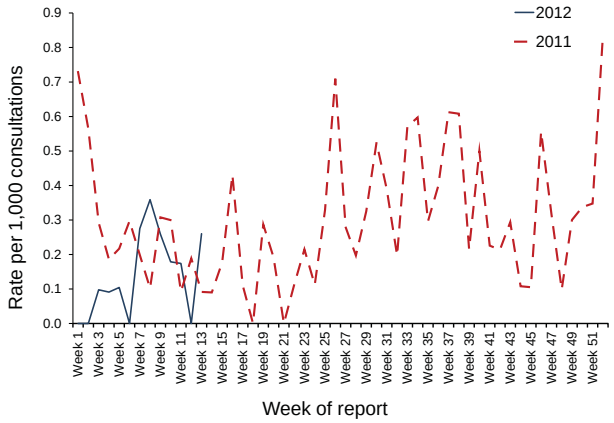
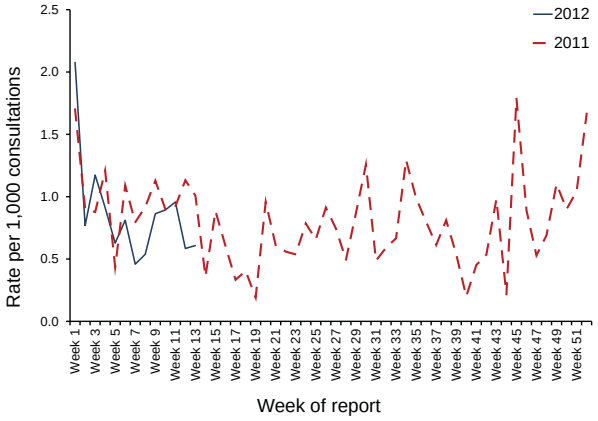


Figure 5: Consultation rates for shingles, ASPREN, 1 January 2011 to March 2012, by week of report



Gonococcal surveillance

Dr Monica M Lahra, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various states and territories report data quarterly on sensitivity to an agreed 'core' group of antimicrobial agents. The antibiotics routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, which are current or potential agents used for the treatment of gonorrhoea. When clinical 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. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a programme-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented.*

Reporting period 1 January to 31 March 2012

The AGSP laboratories received a total of 1,262 isolates in the first quarter of 2012 of which 1,238 (98%) were viable and underwent susceptibility testing. This number is higher than the 1,059 isolates referred in this period in 2011. Approximately 36% of this total was from New South Wales; 25% from Victoria; 16% from Queensland; 11% from Western Australia; 8% from the Northern Territory; 3% from

South Australia and 1% from the Australian Capital Territory. A small number of isolates were received from Tasmania.

Penicillins

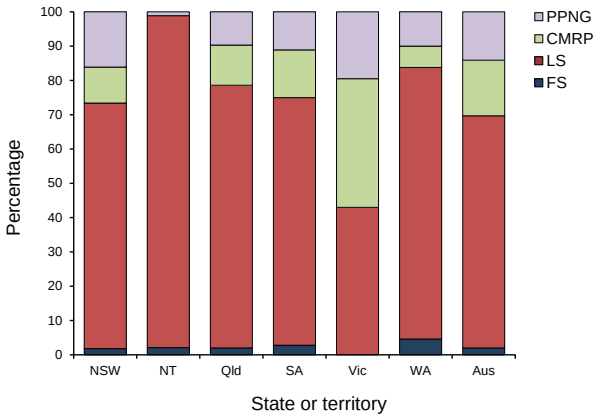
In this quarter, 375 (30%) of all isolates examined were penicillin resistant by one or more mechanisms. One hundred and seventy-four (14%) were penicillinase-producing *Neisseria gonorrhoea* (PPNG); and 201 (16%) had chromosomally mediated resistance to penicillin (CMRP). This first quarter in 2012 saw an increase in penicillin resistance in gonococci by any mechanism since the decreasing trend from 2007 (2011: 22%; 2010: 32%; 2009: 39%; 2008: 45%; and 2007: 39%). Whilst the proportion nationally of PPNG has remained stable at 11%–13% over the period 2007–2011, the proportion of gonococci with CMRP has decreased in the same quarter from 26%–32% in 2007–2009, to 20% in 2010 then to 11% in 2011. However, in the first quarter of 2012 the proportion of CMRP has increased to 16%. Penicillin resistance will continue to be monitored over 2012.

The proportion of strains in each jurisdiction resistant to the penicillins by any mechanism ranged from 1.1% in the Northern Territory to 57% in Victoria. In Victoria, there were 312 strains tested and of these there were 117 CMRP (37%) and 61 PPNG (20%); in New South Wales of 447 strains tested there were 47 CMRP (10%) and 72 PPNG (16%); in Queensland of 205 strains tested there were 24 CMRP (12%) and 20 PPNG (10%), and in Western Australia of 130 strains tested there were 8 CMRP (6%) and 13 PPNG (10%). In South Australia in this quarter, there was an increase in the proportion of penicillin resistance from 11% reported in 2011, to 25% reported in 2012 where 36 isolates tested were penicillin resistant (14% CMRP: 11% PPNG). However in South Australia in the first quarter of 2010 46% of isolates had penicillin resistance by any mechanism. No CMRP, but 1 PPNG strain was found in the Northern Territory, and the geographic acquisition of this isolate was unknown. There were 3 PPNG in the Australian Capital Territory but no CMRP and no penicillin resistance reported for the one isolate from Tasmania.

The proportions of gonococci fully sensitive (MIC ≤ 0.03 mg/L); less sensitive (MIC 0.06–0.5 mg/L); CMRP (MIC ≥ 1 mg/L) and PPNG aggregated for Australia by state or territory are shown in Figure 1. A high proportion of those strains classified as PPNG or CMRP fail to respond to treatment with penicillins (penicillin; amoxycillin; ampicillin) and early generation cephalosporins.

There was an increase in the proportion of isolates with penicillin resistance in Victoria, Queensland South Australia and the Australian Capital Territory, however in New South Wales and Western Australia the proportion was unchanged from the same quarter in 2011.

Figure 1: Categorisation of gonococci isolated in Australia, 1 January to 31 March, 2012, by penicillin susceptibility and state or territory



FS	Fully sensitive to penicillin, MIC ≤ 0.03 mg/L.
LS	Less sensitive to penicillin, MIC 0.06–0.5 mg/L.
CMRP	Chromosomally mediated resistant to penicillin, MIC ≥ 1 mg/L.
PPNG	Penicillinase producing *Neisseria gonorrhoeae.*

Quinolones

Quinolone resistant *N. gonorrhoeae* (QRNG) are defined as those isolates with a MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups.

There were 372 (30%) QRNG detected in the first quarter of 2012. All but 12 of the 372 QRNG detected had ciprofloxacin MICs of 1 mg/L or more; and 324 (87% of QRNG) had ciprofloxacin MICs of 4 mg/L or more. The proportion of QRNG (30%) in this quarter nationally was similar to the equivalent quarter in 2011 (27%); but lower than previous equivalent periods (2010: 38%; 2009: 46%; and 2008: 35%).

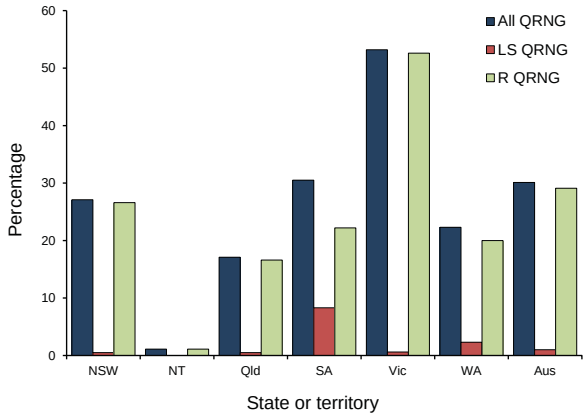
The distribution of quinolone resistant isolates of *N. gonorrhoeae* in Australia by jurisdiction is shown in Figure 2. The highest proportion of QRNG was found in Victoria with 53% of all isolates; in South Australia 31% of isolates were QRNG; in New South Wales 27% and in Western Australia 22% of isolates were QRNG.

The increase in QRNG in Victoria, Queensland and South Australia parallels the increase in penicillin

resistance noted in these jurisdictions in this quarter, whereas in New South Wales the proportion of penicillin resistance remained similar and it was decreased in Western Australia.

There were 8 QRNG detected in the Australian Capital Territory; one in Tasmania; and there were none in the Northern Territory.

Figure 2: The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 January to 31 March, 2012, by state or territory



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.
R QRNG Ciprofloxacin MICs ≥ 1 mg/L.

Ceftriaxone

Forty-four gonococcal isolates (3.5%) with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected nationally, which was slightly higher than the 2.7% detected in the same quarter in 2011 but markedly less than the proportion (6.1%) detected in the same quarter in 2010. There were 21 in Victoria; 17 in New South Wales; 3 in Queensland; 2 in Western Australia; and 1 in the Austalian Capital Territory. There were no isolates with decreased susceptibility to ceftriaxone detected in South Australia; the Northern Territory or Tasmania. The small increase in the proportion of isolates with decreased susceptibility to ceftriaxone (MIC ≥ 0.06 mg/L) corresponds with the increase in CMRP resistant gonococci and QRNG also reported in this first quarter of 2011. It is possible that the small increase in numbers of isolates with decreased susceptibility to ceftriaxone together with an increase in CMRP and QRNG, reflects a clonal shift from that which was evident in 2010 and 2011.

Spectinomycin

All isolates were susceptible to this injectable agent. This antibiotic is not readily available in Australia.

Tetracycline

The following data relate to a form of high-level plasmid mediated resistance to the tetracyclines, and gonococcal isolates possessing this plasmid are known as tetracycline resistant *N. gonorrhoeae* (TRNG). Nationally, the number (168) and the proportion (14%) of TRNG detected in the first quarter of 2012 was lower than that reported in the same quarter of 2010 (TRNG:20%) and 2011 (TRNG: 21%). TRNG were found in all states and territories except Tasmania; and proportions ranged from 8% in the Australian Capital Territory to 22% of isolates in Western Australia. In the Northern Territory, the proportion of TRNG was (10%) markedly lower than for the same quarter in 2011 (TRNG: 28%) and 2010 (TRNG: 18%).

Reference

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health and Ageing. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the state and territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV,

viral hepatitis and sexually transmissible infections in Australia, Annual Surveillance Report'. The reports are available from the Kirby Institute, CFI Building, Cnr Boundary and West Streets, Darlinghurst NSW 2010. Internet: http://www.kirby.unsw.edu.au/ Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see Commun Dis Intell 2012;36(1):123.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 July to 30 September 2011, are included in this issue of Communicable Diseases Intelligence (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 2011, by sex and state or territory of diagnosis

		State or territory								Totals for Australia			
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2011	This period 2010	YTD 2011	YTD 2010
HIV diagnoses	Female	0	11	1	2	4	1	8	9	36	36	105	114
	Male	1	85	4	47	16	4	73	10	240	223	766	700
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	1	96	5	49	20	5	81	19	276	260	871	819
AIDS diagnoses	Female	0	0	0	0	0	0	2	0	2	2	11	10
	Male	0	10	1	0	0	1	15	1	28	25	77	78
	Total*	0	10	1	0	0	1	17	1	30	27	88	88
AIDS deaths	Female	0	1	0	0	0	0	1	0	2	0	3	1
	Male	0	4	0	0	0	0	4	0	8	4	15	15
	Total*	0	5	0	0	0	0	5	0	10	4	18	16

* 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 2011, by sex and state or territory

		State or territory								
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
HIV diagnoses	Female	40	1,066	32	417	144	25	526	323	2,573
	Male	299	15,113	170	3,602	1,146	151	6,478	1,522	28,481
	Not reported	0	227	0	0	0	0	22	0	249
	Total*	339	16,441	202	4,028	1,291	176	7,050	1,852	31,379
AIDS diagnoses	Female	10	288	6	80	32	4	134	50	604
	Male	95	5,669	53	1,115	427	56	2,240	472	10,127
	Total*	105	5,976	59	1,197	460	60	2,387	524	10,768
AIDS deaths	Female	7	144	1	44	20	2	67	30	315
	Male	73	3,618	33	687	281	34	1,472	301	6,499
	Total*	80	3,773	34	733	301	36	1,548	332	6,837

* Totals include 76 HIV diagnoses, 37 AIDS diagnoses and 23 deaths in people whose sex was reported as transgender.

Meningococcal Surveillance Australia

Monica M Lahra, Rodney Enriquez for the Australian Meningococcal Surveillance Programme

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 (NAA) 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 numbers 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 *2012;36(1):121.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 January to 31 March 2012 are included in this issue of Communicable Diseases Intelligence *(Table).*

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2012, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD
Australian Capital Territory	12	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	11	0	0	2	2	0	0	0	0	0	0	0	0	2	2
New South Wales	12	0	0	6	6	0	0	0	0	0	0	3	3	9	9
	11	0	0	10	10	0	0	3	3	2	2	7	7	22	22
Northern Territory	12	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	11	0	0	1	1	0	0	0	0	0	0	0	0	1	1
Queensland	12	0	0	10	10	1	1	0	0	0	0	0	0	11	11
	11	0	0	8	8	1	1	1	1	0	0	1	1	11	11
South Australia	12	0	0	0	0	1	1	0	0	0	0	0	0	1	1
	11	0	0	4	4	0	0	0	0	1	1	0	0	5	5
Tasmania	12	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	11	0	0	0	0	1	1	0	0	1	1	0	0	2	2
Victoria	12	0	0	7	7	0	0	0	0	0	0	0	0	7	7
	11	0	0	10	10	0	0	0	0	0	0	0	0	10	10
Western Australia	12	0	0	1	1	1	1	1	1	0	0	1	0	4	4
	11	0	0	4	4	0	0	0	0	0	0	0	0	4	4
Total	12	0	0	24	24	3	3	1	1	0	0	4	4	32	32
	11	0	0	39	39	2	2	4	4	4	4	8	8	57	57

Administration

New features for the *Communicable Diseases Intelligence* web site

The CDI Editorial Team has been working to introduce some exciting new changes to improve access to articles on the Department of Health and Ageing's web site. CDI is no longer being produced in hard copy and is available in HTML and PDF format from: www.health.gov.au/cdi You can now subscribe to the new electronic notification system from the 'Subscribe link' on any CDI web page. Subscribers will be sent an email when new content is added to the web site.

The second new feature is the ability to search exclusively for CDI articles without the need to retrieve content from the department's web site. Both of these new features can be accessed from any CDI web page.

CDI will also be moving to an Online first format. This format will allow articles to be published online as soon as the editorial process is complete, and will allow for timely publication of information on recent outbreaks. Articles will be consolidated into a single issue at the end of each quarter.

We welcome articles on recent outbreaks or trends in communicable diseases in Australia. Authors should send their manuscripts for consideration to cdi.editor@health.gov.au taking note of the Instructions for authors available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-cdi-auth_inst.htm These instructions will be reviewed and updated in the coming months.

Margaret Curran
Editor
Communicable Diseases Intelligence