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Annual reports

Trachoma surveillance in Australia, 2009

A report by the National Trachoma Surveillance and Reporting Unit

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Abstract

Trachoma is highly prevalent in remote Indigenous communities in Australia. The National Trachoma Surveillance and Reporting Unit was established in 2006 as a result of a Federal Government initiative to provide comprehensive surveillance data from regional and remote Indigenous communities considered by the jurisdictional population health staff to be 'At Risk' for endemic trachoma, defined as a trachoma prevalence of 5% or more. This report details the findings from the 2009 trachoma screening program together with trends in trachoma prevalence and screening coverage since 2006. Aboriginal children aged 1–9 years resident in At Risk communities were examined for trachoma using the World Health Organization (WHO) simplified trachoma grading criteria. In the Northern Territory, screening was conducted by staff from the Healthy School Age Kids program and the Aboriginal Community Controlled Health Services. In South Australia, screening was conducted by the Eye Health and Chronic Disease Specialist Support Program and a team of visiting ophthalmologists and optometrists. In Western Australia, screening was conducted by staff from State Government population health units and Aboriginal Community Controlled Health Services. In the Northern Territory, 53 of 86 At Risk communities were screened and data were reported for 2,283 children. In South Australia, 12 of 72 At Risk communities were screened and data were reported for 149 children. In Western Australia, 68 of 74 At Risk communities were screened and data were reported for 1,684 children. The prevalence of active trachoma ranged from 1%–44% in the Northern Territory, 0%–57% in South Australia and 13%–15% in Western Australia. Trend analysis across all three jurisdictions combined found that neither the prevalence of trachoma nor community screening coverage changed significantly between 2006 and 2009. When trend analysis was done by jurisdiction, there was a significant decrease in trachoma prevalence and a significant increase in community screening coverage only for Western Australia over the same 4 year period. The implementation of the WHO Surgery, Antibiotics, Facial cleanliness and Environmental improvement (SAFE) strategy has been variable. Surgery referral processes for trichiasis were reported as available in all screened communities in the Northern Territory and South Australia but only in 35% of screened communities in Western Australia. Antibiotics were distributed according to Communicable Diseases Network Australia guidelines in 89% of communities where treatment was indicated. Facial cleanliness programs and resources were reported as poorly implemented in South Australia and Western Australia while minimal data were reported for environmental conditions in all jurisdictions. No significant change was found in bacterial resistance to azithromycin from 2007 to 2009. Significant gaps remain in community screening coverage and in the full implementation of the SAFE strategy. However, the parallel increase in community screening coverage and decrease in trachoma prevalence in Western Australia suggests that the SAFE strategy might have had an effect in reducing trachoma prevalence in that jurisdiction. *Commun Dis Intell* 2010;34(4):375–395.

Keywords: active trachoma, antibiotic resistance, facial cleanliness, Northern Territory, SAFE control strategy, South Australia, surveillance, control activities, endemic, Western Australia, At Risk

Introduction

This is the 4th report of the National Trachoma Surveillance and Reporting Unit (NTSRU). The report presents data from the 2009 screening program conducted in At Risk communities from those Northern Territory, South Australia and Western Australia regions with endemic trachoma and compares 2009 data with those from screening conducted from 2006 to 2008 inclusive.[1–3] The report focuses on data for Aboriginal children aged 1–9 years and Aboriginal adults aged 40 years or more—unless otherwise specified—to comply with Communicable Diseases Network Australia (CDNA) guidelines.[4] It comments on each jurisdiction's implementation of the CDNA trachoma guidelines 'minimum best-practice approach', and makes recommendations regarding future reporting and management.

Methods

Screening sample

As had been the practice in past surveys, key representatives from each jurisdiction confirmed the categorisation of communities as 'At Risk' or 'Not At Risk' for trachoma, using regional historical reports of trachoma. The large urban regions were not classed as At Risk. Due to limited information on prior screening for trachoma for many remote communities in South Australia, all remote communities in that jurisdiction were categorised as At Risk.

Definitions

The World Health Organization (WHO) has defined the elimination of blinding endemic trachoma in a community as being a prevalence of active trachoma less than 5% in children aged 1–9 years or a prevalence of operable trichiasis of less than 0.1% in the population.[4] According to the CDNA guidelines, screening should be conducted annually in Communities At Risk until the prevalence of active trachoma is less than 5% for 5 consecutive years.

The WHO simplified trachoma grading system was used to report results of screening.[5] Active trachoma includes WHO grades TF (trachomatous inflammation follicular) and/or TI (trachomatous inflammation intense).

Data collection

In brief, data were reported for prevalence of active trachoma, antibiotic treatment of children, their household contacts and community members, facial cleanliness, and trachomatous trichiasis (TT). The implementation of the Surgery, Antibiotics, Facial cleanliness and Environmental improvement (SAFE) trachoma control strategy was also reported.

For the 2009 screening period, the data collection form was revised to report data for trichiasis for people aged less than 40 years, 40–54 years and 55 years and over age groups to be consistent with the CDNA recommended age groups. A single region in the Northern Territory that reported trichiasis data in 2009, used the 2008 form, which included adults aged less than 30 years and from 30–49 years.

Because of conflicting reports on the development of resistance to macrolide antibiotics in respiratory pathogens resulting from community-wide use of azithromycin to treat trachoma,[6–8] the NTSRU has monitored antibiotic resistance in Aboriginal communities for 4 years (2006 to 2009 inclusive). Three pathology services have previously assisted this monitoring by collecting and reporting data to the NTSRU: Institute of Medical Veterinary Science (IMVS), Northern Territory Government Pathology Service (NTGPS) and Western Diagnostics Pathology Service (WDPS). For the 2009 screening period, the IMVS and WDPS pathology services reported antibiotic resistance (defined as both intermediate and high level resistance to the macrolide antibiotic erythromycin) for any invasive and non-invasive isolates of *Streptococcus pneumoniae* in specimens collected from those regions or health services that predominately service Aboriginal people. Resistance to erythromycin identifies resistance to azithromycin. Specimens were collected over a 6 month period from 1 July to 30 December 2009.

Northern Territory

Screening for trachoma was conducted between February and November 2009 in 5 regions. The Healthy School Age Kids (HSAK) program conducted most of the screening in the Top End and in Central Australia in collaboration with primary health care staff from the Aboriginal Community Controlled Health Services (ACCHS).

Indigenous children at a school in Alice Springs were screened for the first time in 2008 by the trachoma coordinator in conjunction with the Australian Government Emergency Intervention (AGEI) and the Central Australian Aboriginal Congress. The school was not re-screened in 2009.

In the 2007 and 2008 screening years, health personnel from the AGEI conducted Child Health Checks throughout the Northern Territory. The data from screening for trachoma by the AGEI were not regarded as reliable or consistent by the Northern Territory authorities and were not included in any of the NTSRU reports. The communities that were visited by the AGEI were not revisited by the HSAK program in 2008 and this contributed to the smaller number of communities from which data were reported for active trachoma in 2008. In 2009, the HSAK program was responsible for the screening of all of At Risk communities throughout the Northern Territory.

Ophthalmologists examined Aboriginal adults for trichiasis when they conducted outreach visits in the regions.

South Australia

Screening for trachoma was conducted between April and December 2009 in regions serviced by 6 ACCHS. The Ceduna/Koonibba region includes communities in the Eyre school district located south-east of the Ceduna/Koonibba Health

Service. This incorporates communities serviced by the Port Lincoln ACCHS region where screening has not been conducted. The Pika Wiya region includes communities from within the Flinders school district, and two communities from the Northern Country school district that were reassigned by the Eye Health and Chronic Disease Specialist Support Program (EH&CDSSP) coordinator.

In 2006, data from communities in regions serviced by Oak Valley ACCHS were reported along with data from communities from the Tullawon ACCHS; these data have been combined in Table 1 so comparisons can be made for each year between 2006 and 2009.

It is anticipated that screening in South Australia will be expanded in the 2010 screening round with the reclassification of regions in South Australia to include urban regions and to increase their number to 11 (personal communication R Zadow, 2010).

A state-wide co-ordinated screening program was not implemented in South Australia. The project coordinator of the EH&CDSSP assisted a screening team of ophthalmologists and optometrists in recording information on active trachoma from these selected communities. Some communities were visited twice in the 2009 screening period and in these instances, data from both screening events were reported. However, only the first round of data was used for determining trachoma prevalence. Aboriginal children who were screened were usually seen in schools. Others examined were brought to the clinics by family members, Aboriginal health workers and clinic staff.

Data from the Pika Wiya region were collected by the mainstream Health Service and forwarded to the EH&CDSSP coordinator to be included in this report.

The screening team of eye specialists also visited ACCHS clinics twice in 2009 to examine adults for trichiasis.

Western Australia

Screening for trachoma was conducted between August and September 2009 in four regions. Population health units collected data in partnership with primary health care staff from state government health services and ACCHS. Adults were examined for trichiasis as part of an annual influenza vaccination program.

Data analysis and reporting

In 2009, as in the previous report, a community was defined as a group of people where there was a school; larger communities where two or more schools are located were counted as a single community instead of reporting data for each school-associated community separately. Community coverage was calculated using the number of communities that were screened as a proportion of those that were identified by each jurisdiction as At Risk for trachoma. Communities that were reported as Not At Risk were usually not screened and were not included in this report.

The 2006 Australian Bureau of Statistics (ABS) Census data for the number of Aboriginal people resident in a region were used to calculate the 2009 high and low series population projections.[9,10]

Table 1: Number of communities screened for trachoma, the Northern Territory, South Australia and Western Australia, 2009, by trachoma risk and state or territory

Communities	Northern Territory		South Australia		Western Australia		Total	
	n	%	n	%	n	%	n	%
Not At Risk								
Screened	1	20	0		1	2	2	4
Not screened	4	80	0		46	98	50	96
Total Not At Risk	5		0		47		52	
At Risk								
Screened with no trachoma found	14	16	5	7	22	30	41	18
Screened with trachoma found	39	45	7	10	47	64	93	39
Not screened	33	38	60	83	5	7	98	42
Total At Risk	86		72		74		232	
Total communities	91		72		121		284	

Source: Data were collected by the Healthy School Age Kids program in the Northern Territory, the Eye Health and Chronic Disease Specialist Support Program in South Australia and Population Health Units in Western Australia.

Screening coverage was calculated using the number of children who were examined for trachoma in 2009 as a proportion of those who were estimated to be resident in Communities At Risk.

The prevalence of active trachoma in Aboriginal children aged 1–9 years was calculated using the number of children examined as the denominator and prevalence 95% confidence intervals (CI) were calculated.

CDNA guidelines recommend providing azithromycin treatment to affected children, their households and in certain instances, to community members. In some communities, although some treatment was distributed, the treatment strategy was not reported, and it was not possible to determine whether CDNA treatment guidelines were followed. In other communities, the data indicated that treatment was only given to affected children, without household or community contacts being offered treatment. These communities were regarded as not following the CDNA guidelines.

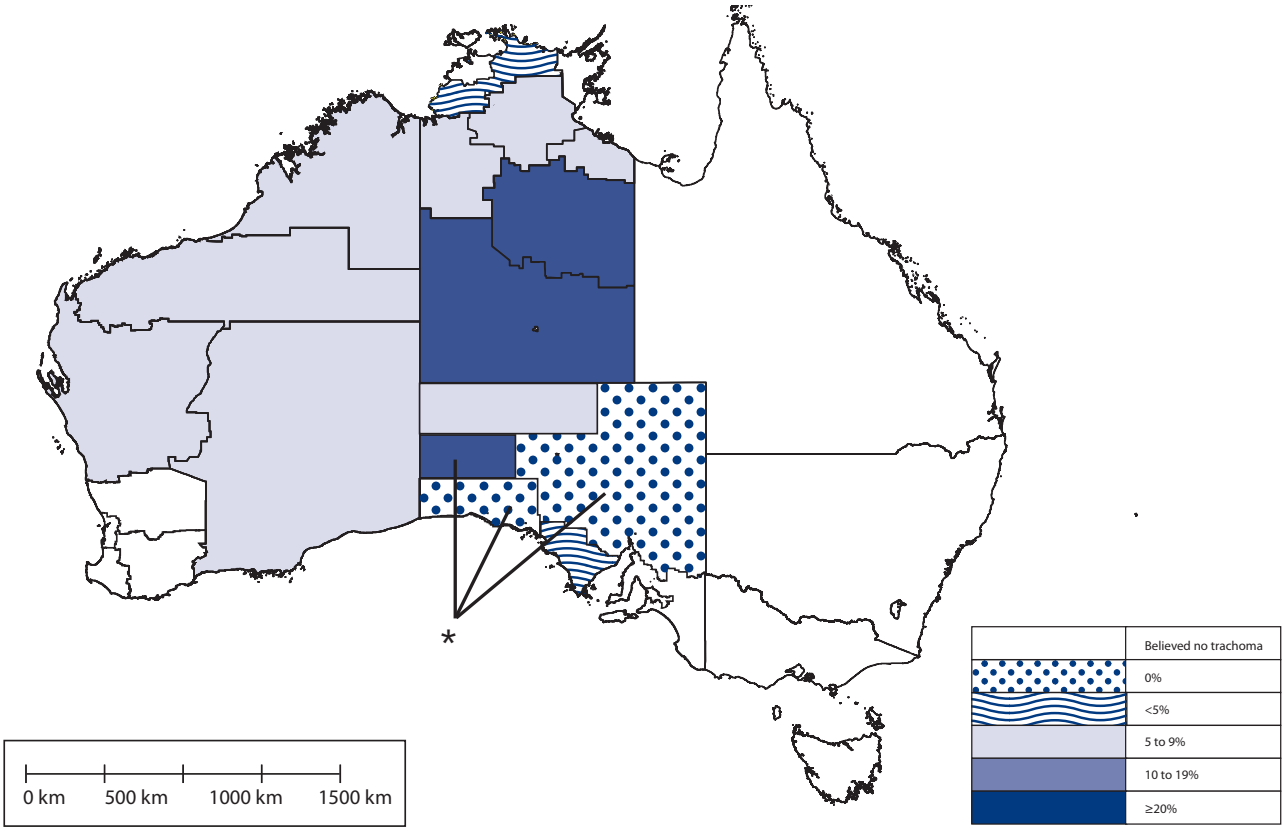
For comparisons to be made, eligible communities had to report comparable data for at least 2 years. The P-trend command in Stata[11] that calculates a chi-square statistic for trend was used to detect a trend in the prevalence of active trachoma from 2006 to 2009 for communities that examined 10 or more children. Analysis could not be conducted on 2006 data for two regions in Western Australia: the Pilbara (where follicular trachoma was not graded according to the WHO grading system) and the Kimberley region (where the number of children examined from each community was not reported). In the 2008 report, comparisons of the prevalence of active trachoma were not possible for four of the seven communities in the Katherine region in the Northern Territory as data were provided for children aged 0–15 years without separately providing data for the children in the 1–9 year age group.

Results

Overview

Data were received from a total of 15 regions with At Risk communities: five regions in the Northern Territory, six in South Australia and four in Western Australia. Other jurisdictions were not included in this screening program (Map).

Map: Prevalence of active trachoma in Australia for children 1 to 9 years, 2009, by region



Note that Pika Wiya and Umoona Tjutagku regions show a combined prevalence in the above figure.

* Less than 5 children were screened in these regions.

Trachoma screening

Of the 15 regions, nine had a prevalence of trachoma of ≥5% (Table 2). Data were reported for 134 of the 232 Communities At Risk (58%) within these regions in 2009 (Table 1). Of the 4,116 Aboriginal children aged 1–9 years for whom data were reported, 575 had active trachoma, resulting in an overall trachoma prevalence of 14% (95%CI 13%–15%) (Table 2). A total of 80 communities (60%) had a prevalence of active trachoma ≥5% (Table 3).

If those 4,116 children screened were a representative sample of all 20,155 children resident in all At Risk communities, then based on the estimated prevalence of trachoma, the additional number of children with potentially undiagnosed trachoma across the three jurisdictions lies between 2,045 and 2,448.

There has been no detectable trend in the prevalence of trachoma when data from all three jurisdictions were grouped together.

While there appeared to have been a significant rising trend in the prevalence of active trachoma in the Northern Territory since 2006, this might be misleading if the reported trachoma prevalence (29%) in the Northern Territory in the 2008 report was a biased result. Such bias could have arisen because of the absence of data from those At Risk communities screened as part of the AGEI program. Data from these communities were not reported to the NTSRU in 2008. Review of the earlier NTSRU published reports reveals that many of the communities examined as part of the AGEI program were those with a lower prevalence of trachoma. As data from these 'lower prevalence' communities were not included in the 2008 NTSRU report, the estimated trachoma prevalence of 29% could have been spuriously high.

A repeat analysis of the trend in trachoma prevalence in the Northern Territory that excluded the 2008 data indicated that the rising trend seen previously was no longer evident. Tables 4 and 5 summarise the trend in trachoma prevalence—Table 4 includes the 2008 data while Table 5 excludes 2008 data.

Trachoma prevalence in Western Australia has been observed to be decreasing over the same 4 year period and the small number of children screened in South Australia has not allowed that jurisdiction to make a useful contribution to the overall trend analysis.

Not all screened communities where data were reported implemented the components of the SAFE strategy according to the CDNA guidelines. Surgery referral processes for trichiasis were available in all communities screened in the Northern Territory and South Australia, but were only available for 35% of Western Australia communities. Use of facial cleanliness programs and resources was well documented in the Northern Territory but no data were reported for South Australia and data were reported from only 44% of screened communities in Western Australia. In only three of the 135 communities screened (2%) were the environmental conditions present in the community reported as 'good', a subjective assessment made by primary health care staff involved in the screening program (Table 6).

Antibiotic treatment was reported to have been distributed in 89 of the 100 communities (89%) in which treatment for trachoma was indicated (Table 7), including six communities where active trachoma was found in children aged 10–14 years. Overall, 70 communities (70%) were treated according to CDNA guidelines. This included treating children found to have active trachoma, their household contacts and community members. In the remaining 19 communities requiring treatment, children found to have active trachoma were treated but household or community contacts were not treated. Treating only children with active trachoma is not in accordance with CDNA guidelines. There has been no significant change in azithromycin resistance between 2005 and 2009.

In terms of overall screening coverage, a significant falling trend was noted in the Northern Territory, a significant rising trend was seen in Western Australia while no trend was detected in South Australia. No trend in community coverage over the period could be detected across all three jurisdictions combined.

Trichiasis screening

Trichiasis screening was carried out in only 49 of 232 (21%) communities in the three jurisdictions. Overall, trichiasis was found in 46 of the 1,212 adults examined giving a prevalence of 4% (95%CI 3%–5%).

If those 1,212 adults screened for trichiasis were a representative sample of the 26,382 adults at risk of trichiasis in all three jurisdictions, then the additional number of adults with undetected trichiasis in the three jurisdictions, based on the estimated prevalence of trichiasis, lies between 741 and 1,271.

Northern Territory

Data for active trachoma were reported for 5 (83%) of six regions categorised as trachoma endemic in 2009: Alice Springs Remote, Barkly, Darwin Rural, East Arnhem and Katherine. The 6th region, a town camp in Alice Springs that was screened as part of the 2008 program, was not screened in 2009 as this town camp is usually screened by Central Australian Aboriginal Congress and not by the HSAK program.

Table 2: Community coverage, screening coverage and active trachoma prevalence of Aboriginal children aged 1 to 9 years, 2006 to 2009, by state or territory, region and Aboriginal Community Controlled Health Service

State or territory and region	Number of Communities At Risk (2009)	Community coverage Number of communities screened (% of Communities At Risk)								Screening coverage Number of children examined (% of children in Communities At Risk)								Prevalence of active trachoma Children 1 to 9 years (% prevalence)							
		2006		2007		2008		2009		2006		2007		2008		2009		2006		2007		2008		2009	
		n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Northern Territory																									
Alice Springs†	–	–		–		1	100	–		–		–		45	22	–		–		–		18	40	–	
Alice Springs Remote	30	25	83	19	63	18	60	23	77	530	35	231	15	459	29	586	37	94	18	46	20	157	34	196	33
Barkly†	8	6	67	6	67	2	25	3	38	105	20	68	13	87	26	64	18	22	21	18	26	58	67	28	44
Darwin Rural	16	15	94	12	75	11	69	14	88	522	27	377	19	907	45	877	43	84	16	25	7	183	20	19	2
East Arnhem	12	12	100	12	100	4	33	6	50	879	78	465	41	232	20	250	21	22	3	23	5	10	4	2	1
Katherine†	20	11	52	11	52	7	35	7	35	218	12	562	31	732	50	506	34	65	30	104	19	287	39	64	13
Total NT	86	69	78	60	67	43	49	53	62	2,254	33	1,703	24	2,462	36	2,283	34	287	13	216	13	713	29	309	14
South Australia																									
Ceduna/ Koonibba	21	1	5	1	5	1	5	1	5	18	1	16	1	121	6	48	2	1	6	1	6	0	0	2	4
Nganampa	10	8	80	4	40	6	60	8	80	27	8	76	23	167	50	90	27	5	19	10	13	4	2	13	14
Oak Valley‡	2	2	100	2	100	2	100	2	100	28	108	34	131	25	93	7	26	7	25	7	21	2	8	4	57
Pika Wiya	33	5	15	–		1	3	1	3	51	1	–		37	1	4	0.06	6	12	–		0	0	0	0
Umoona Tjutagku	6	1	17	1	17	1	17	–		6	7	2	2	15	17	–		1	17	0	0	0	0	–	
Total SA	72	17	24	8	11	11	15	12	17	130	1	128	1	365	4	149	2	20	15	18	14	6	2	19	13

Table 2: Community coverage, screening coverage and active trachoma prevalence of Aboriginal children aged 1 to 9 years, 2006 to 2009, by state or territory, region and Aboriginal Community Controlled Health Service, *continued*

State or territory and region	Number of Communities At Risk (2009)	Community coverage Number of communities screened (% of Communities At Risk)								Screening coverage Number of children examined (% of children in Communities At Risk)								Prevalence of active trachoma Children 1 to 9 years (% prevalence)							
		2006		2007		2008		2009		2006		2007		2008		2009		2006		2007		2008		2009	
		n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Western Australia																									
Goldfields†	21	6	30	10	50	13	65	20	95	231	24	227	23	238	23	321	31	43	19	8	4	18	8	46	14
Kimberley*†	31	28	82	25	83	32	94	30	97	1,048	51	1,006	58	1,169	55	930	43	192	18	164	16	175	15	141	15
Midwest	6	6	100	5	83	6	100	6	100	167	90	127	68	122	64	177	91	32	19	28	22	12	10	23	13
Pilbara§	16	9	56	14	88	16	100	12	75	273	36	306	40	294	37	256	32	146	53	50	16	73	25	37	14
Total WA	76	49	64	54	75	67	88	68	89	1,719	43	1,666	45	1,823	44	1,684	40	413	24	250	15	278	15	247	15
Australia		**135**	**57**	**122**	**52**	**121**	**51**	**133**	**57**	**4,103**	**21**	**3,497**	**18**	**4,650**	**23**	**4,116**	**20**	**720**	**18**	**484**	**14**	**997**	**21**	**575**	**14**

– Data not reported.

* Only children aged 5-9 years were screened in this region for the 2008 reporting period.

† Barkly had 9 communities At Risk of trachoma in 2006 and 2007; Katherine had 21 communities At Risk in 2006 and 2007; Kimberley had 30 Communities At Risk in 2007 and 34 in 2008; Goldfields had 20 communities At Risk in 2008; and Alice Springs had 1 community At Rick in 2008.

‡ Communities in regions serviced by the Oak Valley Aboriginal Community Controlled Health Services were reported with communities from the Tullawon Aboriginal Community Controlled Health Services.

§ Change in grading from 2007.

Source: Data were collected by the Healthy School Age Kids program in the Northern Territory, the Eye Health and Chronic Disease Specialist Support Program in South Australia and Population Health Units in Western Australia.

Of the 91 communities in the endemic regions in the Northern Territory, 86 (95%) were categorised as being At Risk for trachoma and five (5%) as Not At Risk. Of the 86 At Risk communities, 53 (62%) were screened and reported data. No active trachoma was found in 14 communities (26%), while 30 communities (57%) had a prevalence of active trachoma ≥5% and nine communities (17%) had a prevalence less than 5% (Table 3).

Estimates of the trend in the prevalence of active trachoma in the Northern Territory were made from 2006 to 2009. Due to the uncertainty concerning the validity of the 2008 Northern Territory data, trend estimates for the Northern Territory have been made with and without the 2008 data. When the 2008 data were included in the trend estimate, a significant rising trend was evident for the Northern Territory. However when the 2008 data were excluded, that rising trend was no longer seen.

Two regions, Alice Springs Remote and Barkly, showed a significant rising trend in trachoma prevalence while Darwin Rural and Katherine showed a significant falling trend. No trend was observed in East Arnhem.

Table 3: Community prevalence of active trachoma in Aboriginal children aged 1–9 years, 2006 to 2009, by state or territory

Community prevalence of active trachoma (%)	Number and percentage of communities where trachoma data were reported						Total	
	Northern Territory		South Australia		Western Australia			
	n	%	n	%	n	%	n	%
2006 data								
0	30	42	0		5	9	35	26
1 to <5	7	10	0		3	6	10	8
5 to <10	7	10	2	25	8	15	17	13
10 to <20	6	8	3	38	6	11	15	11
20 to <50	12	17	3	38	19	36	34	26
≥ 50	10	14	0		12	23	22	17
Total	72	100	8	100	53	100	133	100
2007 data								
0	29	48	2	25	20	36	51	41
1 to <5	7	12	0		0		7	6
5 to <10	4	7	2	25	5	9	11	9
10 to <20	8	13	2	25	12	22	22	18
20 to <50	11	18	2	25	16	29	29	24
≥ 50	1	2	0		2	4	3	2
Total	60	100	8	100	55	100	123	100
2008 data								
0	4	9	7	64	16	24	27	22
1 to <5	4	9	1	9	7	10	12	10
5 to <10	4	9	2	18	8	12	14	12
10 to <20	6	14	1	9	7	10	14	12
20 to <50	16	37	0		21	31	37	31
≥ 50	9	21	0		8	12	17	14
Total	43	100	11	100	67	100	121	100
2009 data								
0	14	28	5	42	22	32	41	31
1 to <5	9	15	1	8	3	4	13	9
5 to <10	3	6	1	8	8	12	12	8
10 to <20	8	15	2	17	14	20	24	18
20 to <50	10	19	1	8	17	25	28	21
≥ 50	9	17	2	17	5	7	16	12
Total	53	100	12	100	69	100	134	100

Table 4: Prevalence of active trachoma in Aboriginal children aged 1 to 9 years, 2006 to 2009, by region and state or territory

State or territory and region	Prevalence of active trachoma												Test for trend*
	2006			2007			2008			2009			
	%	95% CI	n	%	95%CI	n	%	95%CI	n	%	95%CI	n	
Northern Territory													
Alice Springs			–			–	40	27, 55	45			–	
Alice Springs Remote	18	15, 21	530	20	15, 26	231	34	30, 39	459	33	30, 37	586	↑ p_{trend} = 0.0001
Barkly	21	14, 30	105	26	17, 38	68	67	56, 76	87	44	31, 57	64	↑ p_{trend} = 0.0001
Darwin Rural	16	13, 19	522	7	5, 10	377	20	18, 23	907	2	1, 3	877	↓ p_{trend} = 0.0001
East Arnhem	3	2, 4	879	5	3, 7	465	4	2, 8	232	1	0, 3	250	p_{trend} = 0.44
Katherine	30	24, 36	218	19	16, 22	562	39	36, 43	732	13	10, 16	506	↓ p_{trend} = 0.02†
Total NT	13	11, 14	2,254	13	11, 14	1,703	29	27, 31	2,462	14	12, 15	2,283	↑ p_{trend} = 0.0000†
South Australia													
Ceduna/Koonibba	6	1, 26	18	6	1, 28	16	0	0, 3	121	4	1, 14	48	p_{trend} = 0.57
Nganampa	19	8, 37	27	13	7, 23	76	2	1, 6	167	14	8, 23	90	p_{trend} = 0.44
Oak Valley			‡	22	9, 45	18	13	3, 36	16	100		4	
Pika Wiya	12	5, 23	51			NS	0	0, 9	37	0		4	
Tullawon	25	13, 43	28‡	19	7, 43	16	0	0, 30	9	0		3	
Umoona Tjutagku	17	3, 56	6	0		2	0	0, 20	15			–	
Total SA	15	10, 23	130	14	9, 21	128	2	1, 4	365	13	8, 19	149	↓ p_{trend} = 0.01
Western Australia													
Goldfields	19	15, 25	231	4	2, 7	227	8	5, 12	238	14	11, 19	321	p_{trend} = 0.49
Kimberley§	18	16, 20	1,048	16	14, 18	1,006	15	13, 17	1,169	15	13, 18	930§	p_{trend} = 0.56
Midwest	19	14, 26	167	22	16, 30	127	10	6, 16	122	13	8, 19	177	↓ p_{trend} = 0.03
Pilbara‖	53	47, 59	273	16	12, 21	306	25	21, 30	294	14	10, 19	256	p_{trend} = 0.69
Total WA	18	16, 20	1,446¶	15	13, 17	1,666	15	13, 17	1,823	15	13, 16	1,684	↓ p_{trend} = 0.03¶
Australia	**15**	**14, 16**	**3,830**	**14**	**13, 15**	**3,497**	**21**	**20, 23**	**4,650**	**14**	**13, 15**	**4,116‖**	**p_{trend} = 0.12****

* Test for trend in this table was done using a chi-square statistic for trend across the groups (ptrend command in Stata[11]).

† The trend analysis for Katherine did not include the communities screened in 2006 to 2009 that recorded data for children aged 0–15 years.

‡ Communities in the Oak Valley and Tullawon Aboriginal Community Controlled Health Services were reported together in 2006, therefore these data could not be compared with 2007 and 2008.

§ For 2009 the Kimberley region only reported the screening of children aged 5–9 years.

‖ The trend analysis for Pilbara did not include the data from 2006 because a different grading system was used in the Pilbara in that year.

¶ The trend analysis for Western Australia trachoma prevalence over the 4 year period used community numbers for Pilbara corrected as indicated in footnote ‖.

** The trend analysis for Australian trachoma prevalence over the 4 year period used community numbers for Katherine and Pilbara corrected as indicated in footnotes † and ‖.

n Number of children examined.

– Data not reported.

Note: For communities with ≤5 children examined 95% CI were very large and have not been included in the table.

Comparisons could not be made for regions where <10 children were examined.

Source: Data were collected by Healthy School Age Kids program in the Northern Territory, the Eye Health and Chronic Disease Specialist Support Program coordinator and the screening team in South Australia, and population health units in Western Australia.

ptrend <0.05 = statistically significant change.

Table 5: Prevalence of active trachoma in Aboriginal children aged 1 to 9 years, 2006 to 2009, but excluding 2008 data, by region and state or territory

State or territory and region	Prevalence of active trachoma									Test for trend*
	2006			2007			2009			
	%	95% CI	n	%	95%CI	n	%	95%CI	n	
Northern Territory										
Alice Springs			–			–			–	
Alice Springs Remote	18	15, 21	530	20	15, 26	231	33	30, 37	586	↑ p_{trend} < 0.0001
Barkly	21	14, 30	105	26	17, 38	68	44	31, 57	64	↑ p_{trend} = 0.002
Darwin Rural	16	13, 19	522	7	5, 10	377	2	1, 3	877	↓ p_{trend} < 0.0001
East Arnhem	3	2, 4	879	5	3, 7	465	1	0, 3	250	p_{trend} = 0.63
Katherine	30	24, 36	218	19	16, 22	562	13	10, 16	506	↓ ptrend <0.001†
Total NT	13	11, 14	2,254	13	11, 14	1,703	14	12, 15	2,283	p_{trend} = 0.31†
South Australia										
Ceduna/Koonibba	6	1, 26	18	6	1, 28	16	4	1, 14	48	p_{trend} = 0.77
Nganampa	19	8, 37	27	13	7, 23	76	14	8, 23	90	p_{trend} = 0.74
Oak Valley			‡	22	9, 45	18	100		4	
Pika Wiya	12	5, 23	51			NS	0		4	
Tullawon	25	13, 43	28‡	19	7, 43	16	0		3	
Umoona Tjutagku	17	3, 56	6	0		2			–	
Total SA	15	10, 23	130	14	9,21	128	13	8, 19	149	p_{trend} = 0.53
Western Australia										
Goldfields	19	15, 25	231	4	2, 7	227	14	11, 19	321	p_{trend} = 0.18
Kimberley§	18	16, 20	1,048	16	14, 18	1,006	15	13, 18	930§	p_{trend} = 0.09
Midwest	19	14, 26	167	22	16, 30	127	13	8, 19	177	p_{trend} = 0.13
Pilbara‖	53	47, 59	273	16	12, 21	306	14	10, 19	256	p_{trend} = 0.52
Total WA	18	16, 20	1,446¶	15	13, 17	1,666	15	13, 16	1,684	↓ ptrend = 0.03¶
Australia	**15**	**14, 16**	**3,830**	**14**	**13, 15**	**3,497**	**14**	**13, 15**	**4,116§**	**p_{trend} =0.20****

* Test for trend in this table and in Table 5 was done using a chi-square statistic for trend across the groups (ptrend command in Stata[11]).

† The trend analysis for Katherine did not include the communities screened in 2006 to 2009 that recorded data for children aged 0–15 years.

‡ Communities in the Oak Valley and Tullawon Aboriginal Community Controlled Health Services were reported together in 2006, therefore these data could not be compared with 2007 and 2008.

§ For 2009 the Kimberley region only reported the screening of children aged 5–9 years.

‖ The trend analysis for Pilbara did not include the data from 2006 because a different grading system was used in the Pilbara in that year.

¶ The trend analysis for Western Australia trachoma prevalence over the revised 3 year period used community numbers for Pilbara corrected as indicated in footnote ||.

** The trend analysis for Australian trachoma prevalence over the revised 3 year period used community numbers for Katherine and Pilbara corrected as indicated in footnotes † and ||.

n Number of children examined.

– Data not reported.

Note: For communities with ≤5 children examined 95% CI were very large and have not been included in the table.

Comparisons could not be made for regions where <10 children were examined.

Source: Data were collected by Healthy School Age Kids program in the Northern Territory, the Eye Health and Chronic Disease Specialist Support Program coordinator and the screening team in South Australia, and population health units in Western Australia.

Table 6: Implementation of trachoma control activities (SAFE strategy), 2009, by state or territory

SAFE trachoma control activities	Number of communities from which trachoma control activities were reported						Total	
	Northern Territory		South Australia		Western Australia			
	n	%	n	%	n	%	n	%
Surgery referral process for trichiasis	54	100	12	100*	24	35	90	67
Antibiotics distributed	34	63	10	77	48	70	92	68
Facial cleanliness resources used	41	76	0		31	45	72	53
Facial cleanliness programs implemented	48	89	0		30	43	78	58
Environmental conditions reported as 'good'†	0		0		3	4	3	2
Total number of communities from which trachoma screening data were reported	54		12		69		135	

* Note an extra community that screened only adults for trichiasis was found to have a surgery process for trichiasis available.

† 'Good' was a subjective assessment made by primary health care staff involved in screening.

Source: Data were collected by the Healthy School Age Kids program in the Northern Territory, the Eye Health and Chronic Disease Specialist Support Program in South Australia and Population Health Units in Western Australia.

Table 7: Reported treatment for trachoma, 2009, by state or territory

Communities	Northern Territory		South Australia		Western Australia		Total	
	n	%	n	%	n	%	n	%
Treated in compliance with CDNA guidelines*								
Community-based	9	39	0		12	26	21	30
Household-based	14	61	0		34	72	48	69
Strategy not reported†	0		0		1	2	1	1
Total treated	23		0		47		70	
Not treated in compliance with CDNA guidelines								
Children only	11	55	7	100	1	33	19	63
No treatment reported	9	45	0		2	67	11	37
Total not following CDNA	20		7		3		30	
Total communities	43		7		50		100	

* Includes three communities in the Northern Territory and three in Western Australia where active trachoma was found in children aged 10–14 years without being detected in children aged 1–9 years.

† Communities carried out treatment but the strategy was not reported.

Note: The Communicable Diseases Network Australia (CDNA) guidelines recommend that treatment of children and household or community contacts aged greater than 6 months be completed in as short a timeframe as possible where population mobility is high.

Source: Data were collected by the Healthy School Age Kids program in the Northern Territory, the Eye Health and Chronic Disease Specialist Support Program in South Australia and Population Health Units in Western Australia.

Of the 6,638 children aged 1–9 years reported to be resident in Communities At Risk (Table 8), 2,283 (34%) were examined for trachoma, and 309 had active trachoma (14%, 95% CI, 12%–15%) (Table 2). Of the 1,900 children examined for facial cleanliness 1,403 had clean faces (74%, 95% CI 72%–76%) (Table 8).

Antibiotic treatment was reported to have been distributed according to the CDNA guidelines in 23 of the 43 communities (53%) in which treatment for trachoma was indicated (Table 7). This included three communities where active trachoma was found in children aged 10–14 years and one Not at Risk community that was screened.

Table 8: Number of resident Aboriginal children aged 1 to 9 years, and number examined for facial cleanliness, Northern Territory, 2009, by region

	Alice Springs Remote	Barkly	Darwin Rural	East Arnhem	Katherine	Total
Population data						
Children resident						
in region*	1,843	670	2,176	1,943	2,019	8,651
in Communities At Risk	1,599	346	2,041	1,171	1,480	6,638
in Communities At Risk from which data were reported†	1,898	327	3,291	808	993	7,317
Facial cleanliness						
Communities from which data were reported/Communities At Risk	23/30	3/8	14/16	6/12	3/20	49/86
Children examined	586	64	877	250	123	1,900
Clean faces (%)	352 (60%)	44 (69%)	683 (78%)	223 (89%)	101 (82%)	1,403 (74%)

* Projected 2009 population data based are based on 2006 Australian Bureau of Statistics data with a 1.4% low series population growth rate in the Northern Territory.

† Number of children in Communities At Risk 'from which data were reported' may be greater than the number from the Australian Bureau of Statistics projected data as the former were provided by the Healthy School Age Kids program derived from school enrolment data or from Community Health Centre population data.

Source: Data regarding active trachoma and clean faces were collected by the Healthy School Age Kids program.

Overall, 6,457 persons were identified as requiring treatment with azithromycin according to the CDNA guidelines. Of these, 3,055 (47%) were reported to have been treated. Those treated included children found to have active trachoma, their household contacts and community members.

The reporting of trachoma control activities was similar to that reported in 2008. There is still a lack of reporting of the Environmental component of the SAFE strategy, with only one (2%) of the 53 communities having data reported in terms of current environmental conditions.

Data on trichiasis were reported for the Alice Springs Remote region only. Of the adults aged ≥30 years resident in Communities At Risk, 350 (11%) were examined and 13 (4%) had trichiasis (Table 9). Another 2 adults were reported to have undergone surgery for trichiasis within 12 months prior to the date of reporting.

South Australia

All 72 communities in the 6 ACCHS regions of South Australia that were visited (Ceduna/Koonibba, Nganampa, Oak Valley (Maralinga Tjarutja), Pika Wiya, Tullawon and Umoona Tjutagku), were categorised as being At Risk of trachoma in 2009. Of these 72 communities, 12 (17%) were visited and had data reported (Table 1). Five communities (42%) had no active trachoma while 6 (50%) had a prevalence of active trachoma ≥5% (Table 3).

Of the 9,347 children aged 1–9 years reported to be resident in these Communities At Risk (Table 10), 149 (2%) were examined for trachoma, and 19 had active trachoma (13%, 95% CI 8%–19%) (Table 2). Of the 151 children examined for facial cleanliness, 118 (78%, 95% CI 71%–84%) had clean faces (Table 10).

Antibiotic treatment was reported to have been distributed in all seven of the communities in which treatment for trachoma was indicated (Table 7). Treatment was given to 19 children aged 1–9 years found to have active trachoma. However, CDNA guidelines were not followed as household or community treatment was not given despite the presence of trachoma in these children. The same non-adherence to the CDNA treatment guidelines was noted in the 2006, 2007 and 2008 reports.

Trend in the prevalence of active trachoma was examined for two out of 12 communities where data were reported for at least two of the years between 2006 and 2009. No significant trend in trachoma prevalence was found in either community over this period (Table 4).

Data for trichiasis were reported for all 6 ACCHS regions. Of the 10,653 adults aged ≥40 years resident in Communities At Risk in these regions, 395 (4%) were examined, and 10 (3%, 95% CI 1%–5%) had trichiasis. Two adults were reported to have undergone surgery for trichiasis within 12 months prior to the date of reporting (Table 11).

Table 9: Trichiasis screening reported for Aboriginal adults aged ≥30 years, Northern Territory, 2009, by region

	Alice Springs Remote	Barkly	Darwin Rural	East Arnhem	Katherine	Total
ABS projection						
Adults resident:						
in region*	3,571	1,319	3,343	3,355	3,084	14,672
in Communities At Risk	3,052	550	3,217	2,347	2,067	11,233
Trichiasis						
Communities from which data were reported/Communities At Risk	25/30	0/8	0/16	0/12	0/20	25/86
Adults examined (% of the resident adults in Communities At Risk)	350 (11%)	–	–	–	–	350 (3%)
Trichiasis (%)	13 (4%)	–	–	–	–	13 (4%)
Trichiasis surgery within 12 months prior to the date of reporting	2	–	–	–	–	2

– Data not reported

* Projected 2009 population data based are based on 2006 Australian Bureau of Statistics data with a 1.4% low series population growth rate in the Northern Territory.

† Adults were seen by an ophthalmologist during specialist outreach visits.

Source: Data regarding trichiasis were collected by eye health professionals as part of specialist outreach visits.

Table 10: Number of resident Aboriginal children aged 1 to 9 years, and number examined for facial cleanliness in South Australia, 2009, by Aboriginal Community Controlled Health Service

	Ceduna/ Koonibba*	Nganampa	Oak Valley	Pika Wiya†	Tullawon	Umoona Tjutagku	Total
Population data							
Children resident:							
in ACCHS region‡	2,112	339	9	6,781	18	88	9,347
in Communities At Risk	2,112	339	9	6,781	18	88	9,347
in Communities At Risk from which data were reported	68	92	–	4	3	–	167
Facial cleanliness							
Communities from which data were reported/ Communities At Risk	1/21	8/10	1/1	1/33	1/1	0/6	12/72
Children examined	48	92	4	4	3	0	151
Clean faces (%)	38 (79%)	73 (79%)	0 (0%)	4 (100%)	3 (100%)	0 (0%)	118 (78%)

– Data not reported.

* Includes Aboriginal children from communities in the Eyre school district and incorporates communities serviced by the Port Lincoln Aboriginal Community Controlled Health Services region where screening has not been conducted.

† Includes Aboriginal children from communities in the Flinders school district and two from the Northern Country school district, which were reassigned by the Eye Health and Chronic Disease Specialist Support Program coordinator.

‡ Projected 2009 population data based are based on 2006 Australian Bureau of Statistics data with a 1.9% low series population growth rate in South Australia.

Note: All communities in South Australia were considered At Risk, therefore the number of children resident in the region and in Communities At Risk is the same.

Source: Data regarding clean faces and number of children in Communities At Risk from which data were reported were collected and provided by the Eye Health and Chronic Disease Specialist Support Program coordinator and the screening team.

Table 11: Trichiasis screening reported for Aboriginal adults aged ≥40 years in South Australia, 2009, by Aboriginal Community Controlled Health Service

	Ceduna/ Koonibba*	Nganampa	Oak Valley (Maralinga Tjarutja)	Pika Wiya†	Tullawon	Umoona Tjutagku	Total
Regional population							
Adults resident:							
in region‡	2,454	368	25	7,660	18	128	10,653
in Communities At Risk	2,454	368	25	7,660	18	128	10,653
Trichiasis							
Communities from which data were reported/Communities At Risk	2/21	6/10	1/1	1/33	1/1	1/6	12/72
Adults examined	85	222	12	20	35	21	395
With trichiasis	4	6	0	0	0	0	10
Prevalence of trichiasis	5%	3%	0%	0%	0%	0%	3%
Trichiasis surgery within 12 months prior to the date of reporting	1	1	0	0	0	0	21

– Data not reported.

* Regional population data of Aboriginal adults, and the number of Communities At Risk, include adults and communities in the Eyre school district in South Australia and incorporates those serviced by the Port Lincoln Aboriginal Community Controlled Health Services region where screening has not been conducted.

† Regional population data of Aboriginal adults, and the number of Communities At Risk, include adults and communities in the Flinders school district in South Australia and 2 communities from the Northern Country school district, which were reassigned by the Eye Health and Chronic Disease Specialist Support Program coordinator.

‡ Projected 2009 population data based are based on 2006 Australian Bureau of Statistics data with a 1.9% low series population growth rate in South Australia.

Note: All communities in South Australia were considered At Risk, therefore the number of adults resident in the region and in Communities At Risk is the same.

In 2009 insufficient information was reported on the implementation of the SAFE trachoma control strategy to allow a useful assessment to be made.

Western Australia

Of the regions in Western Australia, four were categorised as being trachoma endemic in 2009: Goldfields, Kimberley, Midwest and Pilbara. From these regions, 74 At Risk communities were identified and of these, 69 (93%) were screened and reported data (Table 1). Of the 69 screened communities, 22 (32%) had no active trachoma, 44 (64%) had a prevalence of active trachoma ≥5%, 2 (3%) had a prevalence of less than 5% and one community reported screening only 1 child who was in the 10–14 years age group (Table 3).

Of the 4,170 children aged 1–9 years reported as resident in Communities At Risk (Table 12), 1,684 (40%) were examined for trachoma, and 247(15%, 95% CI 13%–16%) had active trachoma (Table 2). Of the 1,576 children examined for facial cleanliness 1,228 (78%, 95% CI 76%–80%) had clean faces (Table 12). The Facial cleanliness component of the SAFE strategy was less well implemented this year than in 2008.

Data on trichiasis were reported for all four regions, and of the 4,496 adults aged ≥40 years resident in Communities At Risk, 467 (10%) were examined, and 23 (5%, 95% CI 3%–7%) had trichiasis. No adults were reported to have undergone surgery for trichiasis within 12 months prior to the date of reporting (Table 13).

The Antibiotic component of the SAFE trachoma control strategy was more comprehensively implemented in screened communities than the Surgery and Environmental improvements components.

Antibiotic treatment was reported to have been distributed according to the CDNA guidelines in 47 of the 50 communities (94%) in which treatment for trachoma was indicated (Table 7), including one community where active trachoma was found in children aged 10–14 years. The reporting of azithromycin antibiotic treatment in trachoma endemic jurisdictions has improved from 2006 to 2009 in South Australia and Western Australia (Table 14).

Overall, 1,512 persons were identified as requiring treatment according to the CDNA guidelines. Of these, 1,459 (96%) were reported to have been

Table 12: Number of resident Aboriginal children aged 1 to 9 years, and number examined for facial cleanliness, Western Australia, 2009, by region

	Goldfields	Kimberley‡	Midwest	Pilbara	Total
Population data					
Children resident:					
in region*	1,201	2,915	1,256	1,216	6,588
in Communities At Risk	1,031	2,146	195	798	4,170
in Communities At Risk from which data were reported†	343	941	234	256	1,774
Facial cleanliness					
Communities from which data were reported/Communities At Risk	20/21	29/31	6/6	12/16	67/74
Children examined	295	838	187	256	1,576
Clean faces (%)	181 (61%)	682 (81%)	152 (81%)	213 (83%)	1,228 (78%)

* Projected 2009 population data based are based on 2006 Australian Bureau of Statistics data with a 1.8% low series population growth rate in Western Australia.

† Number of children in Communities At Risk from which data were reported were provided by the Population Health Units from each region.

‡ Only children aged 5–9 years were screened in this region.

Source: Data regarding active trachoma and clean faces were collected by the Population Health Units and staff from Aboriginal Community Controlled Health Services in Western Australia.

Table 13: Trichiasis screening reported for Aboriginal adults aged ≥40 years, Western Australia, 2009, by region

	Goldfields	Kimberley	Midwest	Pilbara	Total
ABS projection					
Adults resident:					
in region*	1,316	2,940	1,421	1,446	7,123
in Communities At Risk	1,131	2,088	264	1,013	4,496
Trichiasis					
Communities from which data were reported/Communities At Risk	3/21	4/31	3/6	2/16	12/74
Adults examined (% of the resident adults in Communities At Risk)	84 (2%)	293 (14%)	47 (18%)	43 (4%)	467 (10%)
Trichiasis (%)	0 (0%)	21 (7%)	2 (4%)	0 (0%)	23 (5%)
Trichiasis surgery within 12 months prior to the date of reporting	–	–	–	–	–

– Data not reported

* Projected 2009 population data based are based on 2006 Australian Bureau of Statistics data with a 1.8% low series population growth rate in Western Australia

Source: Data regarding trichiasis were collected by the population health units and staff from Aboriginal Community Controlled Health Services in Western Australia.

Table 14: Percentage of people treated with azithromycin (total treated/total requiring treatment) in jurisdictions where trachoma is regarded as endemic, 2006, 2007, 2008 and 2009

State or territory	2006*		2007		2008†		2009	
	n	%	n	%	n	%	n	%
Northern Territory	–/287		328/533	62	3,069/4,860	63	3,055/6,457	47
South Australia‡	19/20	95	18/18	100	7/7	100	19/19	100
Western Australia§	396/471	84	1,675/2,084	80	2,917/3,013	97	1,459/1,512	96
Total	415/778	53	2,235/2,635	85	5,993/7,880	76	4,533/7,988	57

– Data not reported.

* No jurisdiction reported the number of household or community contacts treated.

† An additional 871 people were treated in 4 communities in the Katherine region (Northern Territory) and they have not been included in the total because the number of people requiring treatment was not provided.

‡ Number of children found to have active trachoma at the first screening have been reported; no household or community contacts were treated irrespective of the presence of trachoma.

§ Treatment data were reported for only two of the 4 regions in 2006.

treated with azithromycin, including children found to have active trachoma, their household contacts and community members (Table 14).

Estimates of trend in trachoma prevalence were made from 2006 to 2009 and a significant downward trend in trachoma prevalence was observed in Western Australia whether or not data from 2008 were included in the analysis. The trend in trachoma prevalence in the Pilbara was estimated with data from 2006 excluded as a non-standard definition of trachoma was used in that region in 2006. In addition, as only children aged 5–9 years were examined in the 31 communities screened in the Kimberley, these communities were not compared with communities from other regions where children aged 1–9 years were examined. After these adjustments, the Midwest region was the only region to display a significant downward trend in trachoma prevalence (Table 4).

Antibiotic resistance

Overall, 39 of the 119 *S. pneumoniae* isolates (32.8%, 95% CI, 24%–42%) were reported to be resistant to azithromycin (Table 15).

Azithromycin resistance reported from 2007 to 2009 was not significantly different from the 22.7% resistance found in isolates reported in the AGAR survey in 2005 (Table 16).[12]

Discussion

In 2009, 13 of the 15 regions screened for trachoma in Australia had a prevalence of trachoma of ≥5%. These data clearly indicate that trachoma is still endemic in regional and remote Australia. Current estimates of trachoma prevalence indicate that there was likely to be a large pool of Indigenous children with active trachoma that was undiagnosed and untreated in regional and remote Australia in 2009, unless treatment was given outside the current jurisdictional trachoma programs.

Community screening coverage has not improved overall since 2006 and remains consistently lower in South Australia than in the other jurisdictions. However, while screening coverage has fallen in the Northern Territory and South Australia since 2006, it has improved in Western Australia over this period.

Comparisons over the period from 2006 to 2009 must be interpreted with caution due to the year-to-year variation in methods, data collection and reporting, and the small numbers of children examined in some communities. Furthermore, when comparing trachoma prevalence over time for the three jurisdictions, care must be taken to ensure the screening process has been consistent across the years. During 2006, screening in the Pilbara region in Western Australia used a different trachoma grading system from that used from 2007 onwards. The 2006 grading system overestimated the prevalence of trachoma and so was excluded from the trend analysis for this region. Reports from the Katherine region in the Northern Territory for the 2006–2008 screening periods detailed combined screening data for children aged 0–15 years in some communities. In this instance, all communities where this occurred were removed from any trend analysis. After these exclusions, the trend analysis of prevalence for 2006–2009 showed a significant increase in trachoma prevalence in the Northern Territory and a significant decrease in South Australia and Western Australia. No significant change in trachoma prevalence was observed for all three jurisdictions combined.

Table 15: Azithromycin resistance and susceptibility to *Streptococcus pneumoniae* isolates collected from Aboriginal people, 2009, by pathology service and region

	Resistant		Intermediate		Susceptible		Total	
	n	%	n	%	n	%	n	%
Institute of Medical Veterinary Science								
Goldfields	1	9	0		0		1	100
Nganampa	10	91	0		11	100	21	100
Subtotal	11	50	0		11	50	22	100
Western Diagnostics Pathology Service								
Alice Springs	7	25	0		9	13	16	100
Alice Springs Remote	2	7	0		0	0	2	100
Darwin	2	7	0		9	13	11	100
Darwin Rural	5	18	0		21	30	26	100
East Arnhem	6	21	0		17	25	23	100
Katherine	5	18	0		11	16	16	100
Pilbara	1	4	0		0	0	1	100
Kimberley	0		0		1	1	1	100
Unknown	0		0		1	1	1	100
Subtotal	28	29	0		69	71	97*	100
Total	**39**	**33**	**0**	**0**	**80**	**67**	**119**	**100**

* Resistance data were not provided for 9 samples and have not been included.

Source: Data provided by the Institute of Medical Veterinary Science and Western Diagnostics Pathology Service.

Table 16: Comparison of azithromycin resistance (resistant and intermediate) to invasive and non-invasive *Streptococcus pneumoniae* isolates collected from Aboriginal people, 2005 to 2009, by state or territory

State or territory	AGAR monitoring				NTSRU monitoring			
	2005		2007		2008		2009	
	%	Number resistant/ total tested	%	Number resistant/ total tested	%	Number resistant/ total tested	%	Number resistant/ total tested
New South Wales/ACT	27.8	162/583		NR		NR		NR
Northern Territory		NR	23.4	11/47	20.9	48/230	28.7	27/94
Queensland	28.2	80/284		NR	0.0	0/1		NR
South Australia	20.9	82/392	40.0	6/15	20.0	4/20	47.6	10/21
Victoria	14.5	35/221		NR		NR		NR
Western Australia	16.2	48/296		NR	20.0	1/5	66.7	2/3
Unknown		0		0	0.0	0/5	0.0	0/1
Australia (95%CI)	22.7% (21,25)	404/1,776	27.4 (18,40)	17/62	20.7 (16,26)	53/256	33.1 (25,42)	39/118

NR Not reported

No trend was detected in azithromycin resistance between 2007 and 2009 (Ptrend=0.17).

However, when compared with data from previous screening years, data for the 2008 screening period showed an abrupt and marked increase in trachoma prevalence in the Northern Territory and a similar marked decrease in South Australia. The increase in prevalence in the Northern Territory might have been due in part to the non-inclusion of communities examined during the AGEI in 2008. These communities were not re-screened by HSAK and data collected by the AGEI were not reported to the NTSRU

due to limited training of AGEI staff collecting the data. Examination of trachoma prevalence prior to 2008 in those communities screened by AGEI showed that the majority appeared to have a very low prevalence of trachoma. Therefore, the exclusion of these lower prevalence communities from the 2008 report might have resulted in a spuriously high prevalence being observed for the Northern Territory in that year. This is supported by data from the 2009 screening period when the HSAK resumed screening of all Northern Territory communities. In 2009, the prevalence of trachoma returned to a level in keeping with the prevalence levels observed prior to the involvement of the AGEI. The sharply lower trachoma prevalence observed in South Australia in 2008 might have been influenced by the larger number of children screened in that year compared with any other year. However, the true reason for the marked fall in trachoma prevalence in South Australia in 2008 remains unclear.

Because of the uncertainty with the 2008 data, a second analysis was conducted with the 2008 data excluded to estimate the trend in trachoma prevalence from 2006 onwards. When the 2008 data were excluded, no significant trend in trachoma prevalence was observed in the Northern Territory or South Australia but a significant falling trend still occurred in Western Australia. Again, no significant change in trachoma prevalence was observed overall in the three jurisdictions. In summary, the findings indicated that Western Australia was the only jurisdiction to display a decrease in trachoma prevalence since 2006, while there was no change in trachoma prevalence in the Northern Territory, South Australia or in all three jurisdictions combined.

CDNA guidelines recommend that for trachoma control, antibiotic treatment for all children with active trachoma, and provision of treatment to their household members and community members as appropriate, is necessary. Such treatment is critical in preventing the spread of trachoma by stopping the cycle of re-infection that can occur if household or community contacts are not treated along with affected children. However, in 2009 only 70% of the 100 communities where treatment was required received it according to these guidelines. In 19% of communities, treatment was given only to children with trachoma and in the remaining 11% no data were reported on treatment of children or their contacts. South Australia is the only jurisdiction that continues to treat affected children only without providing household or community treatment. This treatment strategy is not in accord with the CDNA guidelines.

The data indicate that household and community treatment according to the CDNA guidelines has improved from 2006 to 2009 in Western Australia, with 47 of 50 (94%) communities being treated in 2009 compared with 41 of 52 (79%) in 2008. However, similar improvement in treatment strategy was not observed in South Australia or the Northern Territory. Treatment was reported to have been distributed according to the CDNA guidelines in only 23 of 43 (53%) of Northern Territory communities in 2009, compared with 35 of 41 communities (85%) in 2008. No change in antibiotic resistance of *S. pneumoniae* has been detected over this time.

Implementation of the Surgery, Antibiotics and Facial cleanliness components of the SAFE trachoma control strategy has improved since 2006; however implementation of these components could be strengthened further. In 2009, 67% of screened communities reported having an existing referral process for trichiasis surgery. All screened communities in the Northern Territory and South Australia had this referral process available, but in Western Australia it was only available in 35% of the screened communities. In 2009, 68% of the 135 screened communities treated children with trachoma but did not necessarily treat household and community contacts. The use of facial cleanliness programs and resources is a key component of the SAFE strategy. These components were reported to be present and used in just over half (56%) of the screened communities in 2009. Further improvement is necessary in the promotion of facial cleanliness, a major factor in preventing the transmission of trachoma. The Environmental improvement component of the SAFE strategy was poorly reported overall and most reports indicated that environmental conditions were poor. In only three (2%) of the 135 screened communities did reports indicate that the environmental conditions were good. Both the reporting of this component and measures to improve environmental conditions need attention.

The reporting of trichiasis data has not changed since 2008. Only 4% of eligible adults in At Risk communities were examined in each year. In 2009, trichiasis data were reported from 49 (21%) of the 232 At Risk communities in all three jurisdictions. Trichiasis data were reported from 29% of At Risk communities in the Northern Territory, with all of these communities coming from one region while data were reported from 16% of At Risk communities in Western Australia and 17% of At Risk communities in South Australia.

The number of adults found to have trichiasis was 49 from 1,212 adults screened, giving a trichiasis prevalence of 4% (95% CI 3%–5%). If these 1,212 adults are a representative sample of all 26,382 adults resident in At Risk communities, the additional number of adults with undetected trichiasis in these communities is likely to lie between 741 and

1,271. These people are at high risk of blindness and would be very likely to benefit from surgical intervention.

In summary, jurisdictions have collected data from communities where trachoma was still thought to be present. Gaps in data collection and limitations in the reporting of data remain although some improvement has occurred over the 4 year survey period.

Recommendations for the future include reviewing assumptions that communities classified as Not At Risk in 2006 remained so in 2009 and that Aboriginal children in urban communities are Not At Risk of trachoma. It is strongly recommended that at least some of the communities presently classified as Not At Risk are included in the next round of screening. Concerted efforts are needed to screen all communities classified as At Risk and to screen all children in those communities. Compliance with the CDNA guidelines for treatment of children and household and community contacts is attainable and should be emphasised. Screening for trichiasis must be improved and alternative methods for making surgery for trichiasis readily available should be a priority. In terms of data reliability, an increase in both community screening coverage and the number of children screened will enable more reliable estimates of the prevalence and distribution of trachoma. Implementation of these recommendations would be a major advance towards the elimination of blinding endemic trachoma by 2020.

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Data collection

The organisations that collected and/or reported data were:

Northern Territory

Aboriginal Community Controlled Health Services staff

Australian Government Emergency Intervention

Centre for Disease Control, Northern Territory Department of Health and Families, Northern Territory

Healthy School Age Kids program: Top End and Central Australia

South Australia

Aboriginal Health Council of South Australia, Eye Health and Chronic Disease Specialist Support Program

Country Health South Australia

Ceduna/Koonibba Health Service

Nganampa Health Council

Oak Valley (Maralinga Tjarutja) Health Service

Pika Wiya Health Service

Tullawon Health Service

Umoona Tjutagku Health Service

Western Australia

Aboriginal Community Controlled Health Services staff

Communicable Diseases Control Directorate, Western Australian Department of Health

Goldfields Population Health Unit

Kimberley Population Health Unit

Midwest Population Health Unit

Pilbara regions Population Health Unit

Antibiotic resistance

Institute of Medical Veterinary Science

Northern Territory Government Pathology Service

Western Diagnostics Pathology Service

National Trachoma Surveillance Reference Group

The NTSRU is advised by the National Trachoma Surveillance Reference Group, members of which include representatives from the following organisations:

Centre for Disease Control, Alice Springs, Northern Territory Department of Health and Families

Centre for Disease Control, Darwin, Northern Territory Department of Health and Families

Communicable Diseases Control Directorate, Western Australian Department of Health

Country Health South Australia

Eye Health and Chronic Disease Specialist Support Program, Aboriginal Health Council of South Australia

National Aboriginal Community Controlled Health Organisation

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

Western Australian Country Health Service

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Abbreviations

ABS	Australian Bureau of Statistics
ACCHS	Aboriginal Community Controlled Health Service(s)
AGEI	Australian Government Emergency Intervention
CDNA	Communicable Diseases Network Australia
CI	Confidence interval
EH&CDSSP	Eye Health and Chronic Disease Specialist Support Program
HSAK	Healthy School Age Kids program
IMVS	Institute of Medical Veterinary Science
NR	Not reported
NTGPS	Northern Territory Government Pathology Service
NTSRU	National Trachoma Surveillance and Reporting Unit
SAFE	Surgery, Antibiotics, Facial cleanliness, and Environmental improvement
TF	Trachomatous inflammation – follicular
TI	Trachomatous inflammation – intense
TT	Trachomatous trichiasis
WDPS	Western Diagnostics Pathology Service
WHO	World Health Organization

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Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet network, 2009

The OzFoodNet Working Group

Abstract

In 2009, OzFoodNet sites reported 27,037 notifications of 9 diseases or conditions that are commonly transmitted by food. The most frequently notified infections were *Campylobacter* (15,973 notifications) and *Salmonella* (9,533 notifications). Public health authorities provided complete serotype and phage type information on 92% of all *Salmonella* infections in 2009. The most common *Salmonella* serotype notified in Australia during 2009 was *Salmonella* Typhimurium, and the most common phage type was S. Typhimurium 170/108. During 2009, OzFoodNet sites reported 1,820 outbreaks of gastrointestinal illness, which affected 36,426 people and resulted in 1,240 people being hospitalised. There were 118 deaths during these outbreaks. The majority (82%, 1,496/1,820) of outbreaks were due to person-to-person spread, 9% (163/1,820) were suspected or confirmed to have been transmitted by contaminated food and 9% (161/1,820) were due to either waterborne transmission or outbreaks with an unknown mode of transmission. Foodborne outbreaks affected 2,679 persons including 342 hospitalisations. Eight deaths were reported during these foodborne outbreaks. *Salmonella* was the most common aetiological agent in foodborne outbreaks and restaurants were the most common setting where foods were prepared. Eighteen outbreaks were related to dishes containing raw or undercooked eggs; the majority (n=14) due to various phage types of S. Typhimurium. This report summarises the incidence of disease potentially transmitted by food in Australia and details outbreaks associated with various food vehicles in 2009. These data assist agencies to identify emerging sources of disease, develop food safety policies, and prevent foodborne illness. *Commun Dis Intell* 2010;34(4):396–426.

Keywords: foodborne disease, surveillance, disease outbreak

Introduction

In Australia, an estimated 5.4 million cases of foodborne disease occur annually, costing an estimated $1.2 billion per year.[1] Many of these illnesses are preventable by appropriate interventions. Foodborne disease surveillance can be used to gather evidence to help identify appropriate control measures.[2] Health departments conduct surveillance for foodborne diseases and diseases potentially transmitted by food to monitor trends in illness, detect outbreaks, inform preventative measures and to evaluate the efficacy of intervention efforts.[3,4]

Most foodborne diseases manifest as mild self-limiting gastroenteritis, with only around 20% of affected people seeking medical attention. Consequently, surveillance data collected by health departments underestimate the true burden of disease. In Australia, for every case of salmonellosis notified to a health department there are an estimated 7 infections that occur in the community, while there are approximately 8 cases in the community for every notified case of campylobacteriosis and Shiga toxin-producing *Escherichia coli* (STEC).[5,6]

Surveillance data are used to monitor trends in the incidence of disease and to detect outbreaks and clusters of disease. Long-term trends in surveillance data also enable the efficacy of public health interventions to be assessed.[7] In Australia, state and territory health departments conduct surveillance for between 10 and 15 different diseases that may be transmitted through food. Most of these diseases are transmitted by the faecal–oral route and as such may also be transmitted by contact with infected animals or people, or through consumption of contaminated water. In addition, health departments collect summary data on all outbreaks of foodborne diseases, which provide robust information on contaminated foods causing illness in Australia.

The Australian Government established OzFoodNet—Australia's enhanced foodborne disease surveillance system—in 2000 to improve national surveillance and conduct applied research into the causes of foodborne illness.[8] OzFoodNet aggregates and analyses national-level information on the incidence of diseases caused by pathogens commonly transmitted by food, as well as foodborne disease outbreaks. The OzFoodNet network includes collaborators from the Public Health Laboratory Network, Food Standards Australia New Zealand (FSANZ), the Department of

Agriculture, Fisheries and Forestry and the National Centre for Epidemiology and Population Health at the Australian National University. OzFoodNet is a member of the Communicable Diseases Network Australia (CDNA), which is Australia's peak body for communicable disease control.[9] This is the 9th annual report for the OzFoodNet network and summarises 2009 surveillance data, which include a comparison with data from previous years.

Methods

Population under surveillance

In 2009, the network covered the whole of the Australian population, which was estimated to be 21,874,920 persons.[10]

Data sources

Notified infections

All Australian states and territories have public health legislation requiring doctors and pathology laboratories to notify cases of infectious diseases that are important to public health. State and territory health departments record details of notified patients on surveillance databases. These surveillance datasets are aggregated into a national database—the National Notifiable Diseases Surveillance System (NNDSS)—under the auspices of the *National Health Security Act 2007*. OzFoodNet aggregated and analysed data from NNDSS and enhanced surveillance data from OzFoodNet sites on the following 9 diseases or conditions, a proportion of which are commonly transmitted by food:

- non-typhoidal *Salmonella* infections;
- *Campylobacter* infections (except in New South Wales);
- *Listeria* infections;
- *Shigella* infections;
- *Salmonella* Typhi;
- hepatitis A;
- botulism;
- STEC infections; and
- haemolytic uraemic syndrome (HUS).

There may be differences when comparing state and territory enhanced data totals and NNDSS notifications. This is due to amendments to notification totals by states and territories after the date of data extraction. Data for this report were extracted from NNDSS in June 2010 and were analysed by the date of diagnosis within the reporting period 1 January to 31 December 2009. Date of diagnosis was derived from the earliest date supplied from the date of onset of the case's illness, the date a specimen was collected or the date that a health department received the notification. Estimated resident populations for each state or territory as at June 2009 were used to calculate rates of notified infections.

Enhanced surveillance

OzFoodNet sites collected supplementary data on infections commonly transmitted by foods. Information on travel status was collected for cases of *Salmonella* Enteritidis, hepatitis A, *Shigella* and typhoid. The incidence of infection in returned travellers was compared with the number of travellers to that region using overseas arrivals and departures data from the Department of Immigration and Citizenship. The field 'country where you spent the most time abroad' was used as the numerator. Cases that reported overseas travel to more than one region or continent were counted against each country separately.

To examine the quality of surveillance data collected across Australia, OzFoodNet sites provided data on the completeness of notification databases for *Salmonella* notifications regarding serotype and phage type. Data from Western Australia were excluded from the analysis of phage type completeness, as isolates have not been sent routinely for phage typing since June 2007. To assess completeness, data were analysed using the date a notification was received at the health department.

OzFoodNet sites supplied data on listeriosis cases, which included whether or not a case was materno–foetal and whether the case died. Many cases have severe chronic illnesses prior to their *Listeria* infection so it is difficult to determine if listeriosis is the cause of death for fatal cases, or a contributing factor. For the purpose of surveillance, a woman and her unborn child are counted as 1 case, and where the pregnancy results in a miscarriage, the case is counted as fatal. This affects age specific notification rates for listeriosis and the proportion of reported cases that were female. *Listeria* typically infects immunocompromised patients, the elderly and pregnant women.[11]

For disease counts less than 20 only age specific rates (not and age and sex) are calculated as the low case numbers make the rates unstable.

Gastrointestinal and foodborne disease outbreaks

OzFoodNet sites collected summary information on gastrointestinal and foodborne disease outbreaks that occurred in Australia during 2009. An outbreak of foodborne disease was defined as an incident where two or more persons experience a similar illness after consuming a common food or meal and epidemiological analysis implicate the meal or food as the source of illness. A suspected foodborne

outbreak was defined as an incident where two or more persons experience illness after consuming a common meal or food and a specific meal or food is suspected, but person-to-person transmission cannot be ruled out. A cluster was defined as an increase in infections that were epidemiologically related in time, place or person where there is no common setting and investigators were unable to implicate a vehicle or determine a mode of transmission.

Summary information for foodborne and suspected foodborne outbreaks were combined for the analysis, and information collected for each outbreak included the setting where the outbreak occurred, where food was prepared, the month the outbreak occurred, the aetiological agent, the number of persons affected, the type of investigation conducted, the level of evidence obtained, and the food vehicle responsible for the outbreak. To summarise the data, outbreaks were categorised by aetiological agents, food vehicles and settings where the implicated food was prepared. Data on outbreaks due to waterborne transmission and data from clusters investigated by jurisdictional health departments were also summarised. The number of outbreaks and documented causes reported here may vary from summaries previously published by individual jurisdictions as these can take time to finalise.

Data analysis

Microsoft Excel and Stata version 10.1 were used for all analyses.

Results

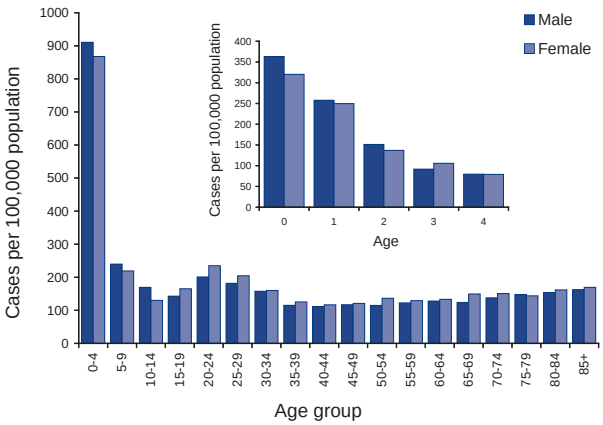
Rates of notified infections

In 2009, OzFoodNet sites reported 27,037 notifications of 9 diseases or conditions that are commonly transmitted by food (Table 1), similar to the mean of 25,637 notifications per year for the previous 5 years (2004–2008).

Salmonella infections

In 2009, OzFoodNet sites reported 9,533 cases of *Salmonella* infection, a rate of 43.6 cases per 100,000 population. The 2009 rate was a 7% increase over the mean of the previous 5 years (40.8) (Table 1). Notification rates ranged from 30 cases per 100,000 population in Victoria to 217 cases per 100,000 population in the Northern Territory, which usually has the highest rate of salmonellosis. Approximately half (49%) of *Salmonella* notifications were in males. The highest age specific rate of *Salmonella* infection was 300 cases per 100,000 population in children aged from 0–1 year (Figure 1). The notification rate decreased dramatically in children aged more than 2 years (Figure 1).

Figure 1: Salmonellosis, Australia, 2009, by age group and sex



Nationally during 2009, the most commonly notified *Salmonella* serotype was *S.* Typhimurium, which was responsible for approximately 41% of all notified infections (Tables 2 and 3). Various phage types of *S.* Typhimurium were the most commonly reported infections in all states and territories, except for Tasmania and the Northern Territory. In particular, the incidence of *S.* Typhimurium phage type 170/108* increased dramatically in several jurisdictions. This increase instigated a cluster investigation of 15 separate outbreaks of *S.* Typhimurium 170/108 occurring in Queensland, New South Wales and the Australian Capital Territory (see cluster investigations). In New South Wales there were 10.0 cases of *S.* Typhimurium 170/108 per 100,000 population, 2.8 cases per 100,000 in Queensland and 38.7 cases per 100,000 in the Australian Capital Territory (Table 2). All of the top 5 serotypes in the Northern Territory, except *S.* Litchfield, exceeded 10 cases per 100,000 population, with *Salmonella* Saintpaul the highest at 25.8 cases per 100,000 population. Tasmania also recorded a high rate for infection due to a specific serotype, *S.* Mississippi, with 14.3 cases of per 100,000 population. *S.* Mississippi is endemic in Tasmania but since 1999 the number of notifications originating from the mainland states and territories has more than doubled. Tasmania is currently conducting a case control study of people infected with *S.* Mississippi on mainland Australia to identify what proportion of infections are linked to Tasmania or to the Pacific and to investigate whether contact with native animals or birds, or consumption of seafood, native animals, untreated water or unpasteurised milk are risk factors for infection.

* Classification of this organism differs between laboratories, with the Institute of Medical and Veterinary Science using phage type 108 to classify this type of *Salmonella* Typhimurium and Microbiological Diagnostic Unit using phage type 170 due to a difference in the interpretation of one phenotypic characteristic.

Table 1: Number of notified cases, crude rate and 5-year mean (2004–2008) rate per 100,000 population of diseases or infections commonly transmitted by food, Australia, 2009, by disease and state or territory

Disease		State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Salmonella	Notified cases, 2009	225	2,736	487	2,471	681	166	1,647	1,120	9,533
	Crude rate, 2009	64.1	38.5	216.6	56.1	42.0	33.0	30.3	50.1	43.6
	Mean rate, 2004–2008	33.9	32.8	209.4	61.1	41.1	42.4	29.2	39.4	40.8
*Campylobacter**	Notified cases, 2009	357†	NN	205	4,610	1,755	626	5,838	2,582	15,973
	Crude rate, 2009	101.7	NN	91.2	104.6	108.2	124.5	107.6	115.4	108.1
	Mean rate, 2004–2008	118.3	NN	121.7	106.2	143.6	129.0	118.0	99.3	115.1
Listeria	Notified cases, 2009	2	26	0	13	4	3	27	13	88
	Crude rate, 2009	0.6	0.4	0.0	0.3	0.2	0.6	0.5	0.7	0.4
	Mean rate, 2004–2008	0.4	0.4	0.1	0.2	0.3	0.2	0.2	0.4	0.3
Shigella	Notified cases, 2009	8	156	85	115	51	2	85	120	622
	Crude rate, 2009	2.3	2.2	37.8	2.6	3.1	0.4	1.6	5.4	2.8
	Mean rate, 2004–2008	0.8	1.4	74.7	2.1	4.4	0.7	1.9	6.4	3.1
Typhoid	Notified cases, 2009	2	47	0	13	2	1	42	8	115
	Crude rate, 2009	0.6	0.7	0.0	0.3	0.1	0.2	0.8	0.4	0.5
	Mean rate, 2004–2008	0.1	0.5	0.7	0.2	0.2	0.2	0.4	0.4	0.4
Hepatitis A	Notified cases, 2009	6	98	1	56	59	5	303	35	563
	Crude rate, 2009	1.7	1.4	0.4	1.3	3.6	1.0	5.6	1.6	2.6
	Mean rate, 2004–2008	0.7	1.3	11.1	1.0	0.7	0.5	1.2	2.2	1.3
Shiga toxin-producing *Escherichia coli*	Notified cases, 2009	0	21	1	23	63	0	16	6	130
	Crude rate, 2009	0.0	0.3	0.4	0.5	3.9	0.0	0.3	0.3	0.6
	Mean rate, 2004–2008	0.1	0.2	0.5	0.4	2.3	0.1	0.2	0.2	0.4
Haemolytic uraemic syndrome	Notified cases, 2009	0	4	0	2	4	0	2	0	12
	Crude rate, 2009	0.00	0.06	0.00	0.05	0.25	0.00	0.04	0.00	0.05
	Mean rate, 2004–2008	0.06	0.18	0.19	0.06	0.10	0.08	0.05	0.02	0.10
Botulism	Notified cases, 2009	0	0	0	1	0	0	0	0	1

* *Campylobacter* is notifiable in all jurisdictions except New South Wales.

† Actual figures for the Australian Capital Territory may differ due to 1 laboratory not reporting *Campylobacter* results for 2009.

Salmonella *Enteritidis*

S. Enteritidis is an important *Salmonella* serotype that can infect the internal contents of eggs, but is not endemic in Australian egg layer flocks. To monitor the emergence of this strain in Australia, OzFoodNet conducts enhanced surveillance of locally-acquired infections of *S.* Enteritidis in humans. The majority of cases in Australia are associated with overseas travel.

During 2009, OzFoodNet sites reported 587 cases of *S.* Enteritidis infection (Table 4). Travel histories were obtained for 93% (547/587) of cases in 2009, compared with 94% (480/511) of cases in 2008. Of those cases where travel status was reported, 93% (508/547) had travelled overseas and cases often reported visiting several countries.

Of the cases that were known to have been acquired overseas, 83% (423/508) reported travel to South East Asia. This compares with only 33% (4,139, 293/12,430,460) of returning travellers coming from South East Asia in 2009 (relative risk [RR] 10, 95% confidence interval [CI] 8–13). Similar to previous years, the most common country of acquisition for overseas-acquired infections was Indonesia, with 56% (283/508) of cases reporting travel there, while comprising only 5% (618,318/12,430,460) of travel undertaken in 2009 (RR 24, 95% CI 20–29). Thailand was the second most common country of acquisition with 11% (58/508) of all notifications that were known to have been acquired overseas, followed by Malaysia with 9% (47/508) and Singapore with 4% (18/508). The most common infecting phage types amongst overseas-acquired cases were 6a (19%, 96/508) and 1b (6%, 33/508) (Table 5).

Table 2: Number, rate and proportion of the top 5 *Salmonella* infections, Australia (excluding Western Australia), 2008 to 2009, by OzFoodNet site*

OzFoodNet site	Sero/phage type	2009		Proportion	2008		Ratio 2009/2008§
		n	Rate†	%‡	n	Rate†	
ACT	*S.* Typhimurium 170/108	136	38.7	60.4	11	3.2	12.4
	S. Typhimurium 135/135a	7	2.0	3.1	12	3.5	0.6
	S. Montevideo	6	1.7	2.7	3	0.9	2.0
	S. Typhimurium 9	5	1.4	2.2	19	5.5	0.3
	S. Kiambu	4	1.1	1.8	0	0.0	
	S. Typhimurium 44	4	1.1	1.8	23	6.7	0.2
NSW	*S.* Typhimurium 170/108	710	10.0	26.0	242	3.5	2.9
	S. Typhimurium 135/135a	199	2.8	7.3	254	3.6	0.8
	S. Typhimurium 9	101	1.4	3.7	150	2.2	0.7
	S. Stanley	64	0.9	2.3	32	0.5	2.0
	S. Birkenhead	64	0.9	2.3	68	1.0	0.9
NT	*S.* Saintpaul	58	25.8	11.9	38	17.3	1.5
	S. Virchow 8	42	18.7	8.6	29	13.2	1.4
	S. Ball	36	16.0	7.4	44	20.0	0.8
	S. Lansing	30	13.3	6.2	27	12.3	1.1
	S. Litchfield	14	6.2	2.9	9	4.1	1.6
Qld	*S.* Saintpaul	207	4.7	8.4	154	3.6	1.3
	S. Birkenhead	143	3.2	5.8	119	2.8	1.2
	S. Aberdeen	123	2.8	5.0	72	1.7	1.7
	S. Typhimurium 170/108	123	2.8	5.0	53	1.2	2.3
	S Typhimurium 135/135a	127	2.9	5.1	159	3.7	0.8
SA	*S.* Typhimurium 9	71	4.4	10.4	75	4.7	0.9
	S. Typhimurium 170/108	69	4.3	10.1	24	1.5	2.9
	S. Typhimurium 193	55	3.4	8.1	27	1.7	2.0
	S. Typhimurium 135/135a	48	3.0	7.0	93	5.8	0.5
	S. Typhimurium 44	27	1.7	4.0	19	1.2	1.4
Tas	*S.* Mississippi	72	14.3	43.4	64	12.8	1.1
	S. Typhimurium 160	14	2.8	8.4	1	0.2	14.0
	S. Typhimurium 135/135a	12	2.4	7.2	58	11.6	0.2
	S. Typhimurium 170/108	9	1.8	5.4	0	0.0	–
	S. Stanley	6	1.2	3.6	3	0.6	2.0
Vic	*S.* Typhimurium 170/108	316	5.8	19.2	128	2.4	2.5
	S. Typhimurium 135/135a	173	3.2	10.5	272	5.1	0.6
	S. Typhimurium 9	124	2.3	7.5	155	2.9	0.8
	S. Typhimurium 44	103	1.9	6.3	194	3.7	0.5
	S. Infantis	44	0.8	2.7	29	0.5	1.5

* Where there were multiple 5th ranking *Salmonella* types all data have been shown; Western Australia data not included due to incomplete phage typing of *S.* Typhimurium, *S.* Enteritidis, and *S.* Virchow in 2009.

† Rate per 100,000 population.

‡ Proportion of total *Salmonella* notified for this jurisdiction in 2009.

§ Ratio of the number of cases in 2009 compared to the number in 2008.

Table 3: Numbers, rates, and proportions of top 5 *Salmonella* serotypes, 2008 to 2009, Western Australia

Serotype	2009		Proportion	2008		Ratio 2009/2008§
	n	Rate†	%‡	n	Rate†	
S. Typhimurium	362	16.2	32.3	296	13.7	1.2
S. Enteritidis	198	8.9	17.7	138	6.4	1.4
S. Saintpaul	72	3.2	6.4	25	1.2	2.9
S. Paratyphi B bv Java	38	1.7	3.4	18	0.8	2.1
S. Singapore	32	1.4	2.9	17	0.8	1.9

† Rate per 100,000 population.

‡ Proportion of total *Salmonella* notified for this jurisdiction in 2009.

§ Ratio of the number of cases in 2009 compared to the number in 2008.

Table 4: Number of *Salmonella* Enteritidis infections, Australia, 2009, by travel history and state or territory

State	Locally acquired	Overseas travel	Unknown	Total
ACT	0	2	0	2
NSW	3	101	7	111
NT	1	6	4	11
Qld	27	55	24	106
SA	1	36	0	37
Tas	0	8	0	8
Vic	3	107	4	114
WA	4	193	1	198
Total	39	508	40	587

Table 5: Number and percentage of each phage type for of overseas-acquired cases of *Salmonella* Enteritidis, Australia, 2009

Phage type	Total	Proportion (%)
6a	96	19
1b	33	6
21	26	5
1	23	5
13	22	4
21b var	18	4
4	10	2
26	9	2
Reactions do not conform (RDNC)	9	2
Untypeable	6	1
Other phage types	52	10
Unknown*	204	40
Total	508	100

* The number of overseas-acquired cases with no phage type available includes 193 cases from Western Australia, where phage typing is not routinely conducted.

Completeness of Salmonella *serotyping and phage typing*

Overall, 92% (7,598/8,250) of *Salmonella* notifications on state and territory databases contained information about serotype and/or phage type for those jurisdictions participating in this typing scheme. For several years OzFoodNet has monitored the completeness of 6 serotypes that are routinely phage typed: Bovismorbificans; Enteritidis; Hadar; Heidelberg; Typhimurium; and Virchow. In 2009, phage typing was greater than 90% complete for serotypes Typhimurium, Virchow and Enteritidis (Table 6). There was an overall decline in the percentage of notifications with phage type reported in 2009 compared with previous years. In 2009, 91.6% of *Salmonella* notifications contained complete information on phage type compared with 94% in 2008.

Table 6: Percentage of *Salmonella* notifications for 6 serotypes notified to state and territory health departments with phage type information available, Australia, 2005 to 2009

Salmonella serotype	2005	2006	2007*	2008	2009
S. Bovismorbificans	94.2	96.8	97.4	83.5	80.0
S. Enteritidis	96.6	98.3	94.5	92.3	92.2
S. Hadar	81.3	100.0	90.0	81.3	33.3†
S. Heidelberg	90.2	94.8	90.0	80.5	74.3
S. Typhimurium	98.5	98.3	98.3	94.8	92.3
S. Virchow	98.7	99.4	95.4	93.4	91.0

* Phage typing ceased in Western Australia in June 2007 and is not included in data from 2007 onwards.

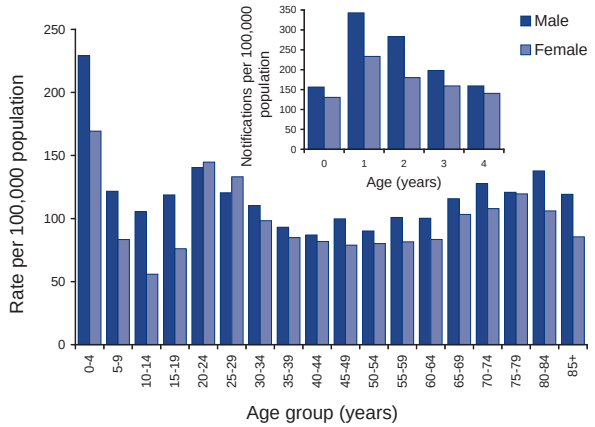
† The Microbiological Diagnostic Unit is waiting on reagents from England to type *S.* Hadar isolates. Completeness will improve as a result.

Campylobacter infections

In 2009, OzFoodNet sites (excluding New South Wales where *Campylobacter* is not notifiable) reported 15,973 cases of *Campylobacter* infection; a rate of 108 cases per 100,000 population (Table 1). The lowest and highest rates of *Campylobacter* notification were in the Northern Territory (91 cases per 100,000 population) and in Tasmania (125 cases per 100,000 population) respectively.

Fifty-four per cent of notified cases were male, which is consistent with previous years. Notification rates were highest among males in nearly all age groups. In 2009, notification rates were highest in males and females aged 0–4 years (229 and 169 notifications per 100,000 population, respectively) with additional peaks in the 20–29 and 70–84 age groups (Figure 2). Amongst children under 5 years of age, the highest notification rates were in infants aged 1 year for both males and females (343 and 233 cases per 100,000 population, respectively)(Figure 2).

Figure 2: Campylobacteriosis notification rates, Australia, 2009, by age group and sex



Listeria infections

OzFoodNet sites reported 88 cases of *Listeria monocytogenes* infection in 2009, a crude rate of 0.4 cases per 100,000 population, which was an increase over the 5-year historical mean of 0.3 cases per 100,000 (60 cases) (Table 1). This increase was due in part to a multi-jurisdictional outbreak of listeriosis associated with the consumption of chicken wraps on domestic airlines (described under multi-jurisdictional outbreak investigations).

Fourteen of these 88 cases (16%) were pregnancy related (Figure 3). Fifty-eight per cent (51/88) of notifications were in people aged 60 years or more. The highest age specific notification rate was in people aged 85 years or more (3.1 cases per 100,000 population, 12 cases) (Figure 4). Fourteen per cent (2/14) of pregnancy related cases and 14% (10/74) of the non-pregnancy associated cases in 2009 were fatal (Figure 3). In 2009, 55% (41/74) of the non-pregnancy related cases were female.

Figure 3: Notification rates and 5 year mean rate for listeriosis, Australia, 2009, by age

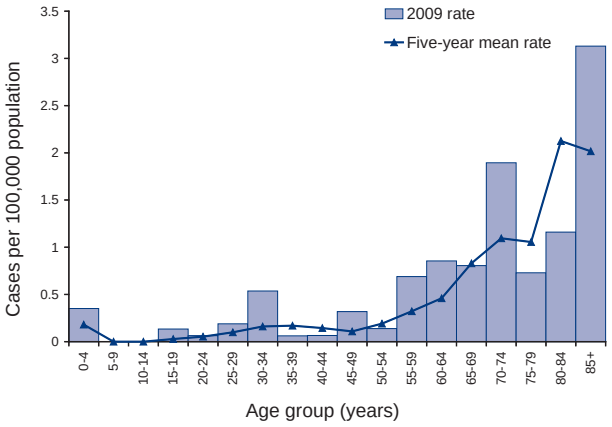
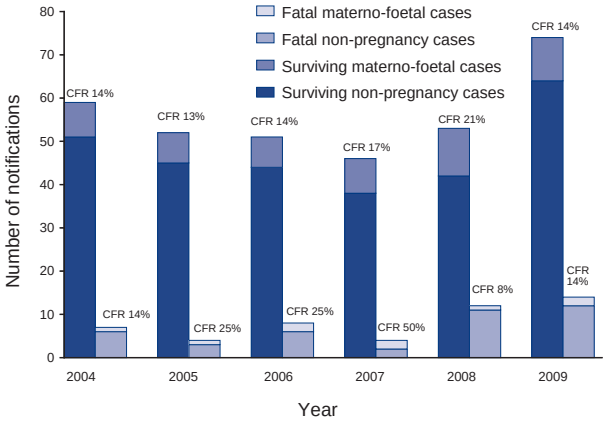


Figure 4: Notifications and case fatality ratio for fatal and surviving listeriosis cases, 2004 to 2009, by pregnancy status



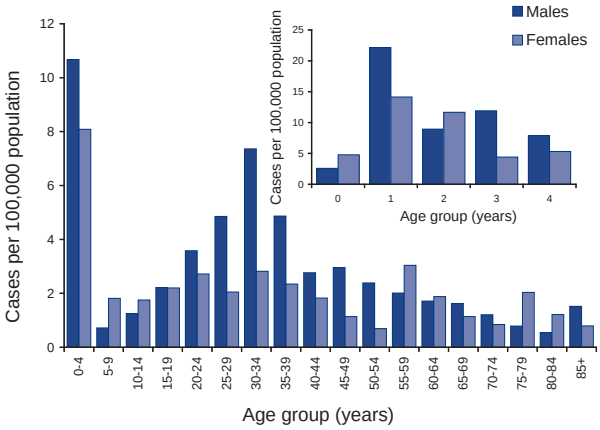
CFR Case fatalitiy rate

Shigella infections

There were 622 notifications of shigellosis in Australia in 2009, a rate of 2.8 notifications per 100,000 population compared with a mean of 645 cases (3.1 notifications per 100,000) per year between 2004 and 2008. As in previous years, the highest notification rate was in the Northern Territory, with 37.8 cases per 100,000 population compared with 74.7 cases per 100,000 population between 2004 and 2008.

In 2009, notification rates for shigellosis were highest in males and females aged 0–4 years, with 10.7 and 8.1 notifications per 100,000 population respectively. A secondary peak was observed in males aged 30–44 years, and in females aged 55–59 years. Amongst children under 5 years of age, the highest notification rates were in children aged 1 year (Figure 5).

Figure 5: Notification rates for shigellosis, Australia, 2009, by age and sex



The most frequently reported *Shigella* biotype in 2009 was *S. sonnei* biotype g, followed by *S. sonnei* biotype a. These biotypes accounted for 52.4% of all *Shigella* infections reported in 2009 (Table 7). In 2009, *S. sonnei* biotype g was more frequently reported than in 2008 and 2007, when the most common biotype was *S. sonnei* biotype a (Table 7).

In 2009, information on the following selected risk factors for *Shigella* cases were collated nationally: overseas travel; indigenous status; and whether the case was a man who has sexual contact with other men (MSM). However, the completeness of this information varied as not all jurisdictions collected information on these risk factors. Information on injecting drug use was not available for all states and territories.

Of the *Shigella* cases with known specific risk factor information, the most frequently reported risk factor was Aboriginality, at 47% (193/414), followed by overseas travel, 46% (135/295) and MSM 4.8% (30/622).

Typhoid

In 2009, there were 115 cases of typhoid (*S.* Typhi infection) in Australia, a rate of 0.5 cases per 100,000 population compared with 0.4 cases per 100,000 between 2004 and 2008 (Table 1). In 2009, 48.6% (56/115) of cases were female. Cases were reported from all Australian states and territories except for the Northern Territory. Travel status was known for all cases, with 11.3% (13/115) of cases reporting no overseas travel and 88.7% (102/115) of cases reporting infections known to have been acquired overseas.

Notification rates for typhoid in 2009 were highest in young adults, with 1.6 cases per 100,000 (26 cases)

Figure 6: Notification rates for typhoid, Australia, 2009, by age

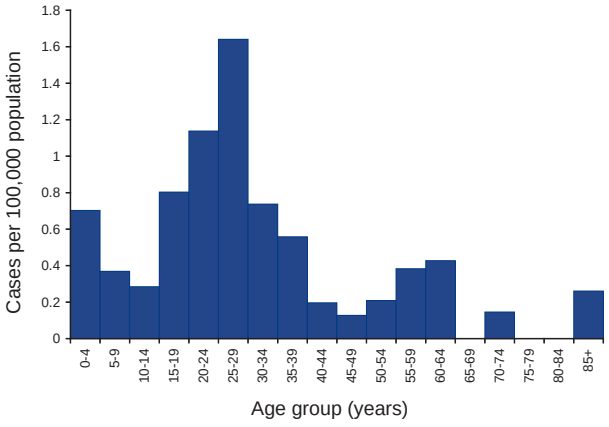


Table 7: Number, percentage and ratio of the top 10 Shigella infections, Australia, 2007 to 2009

Biotype	2007		2008		2009		2009/2007 ratio*	2009/2008 ratio*
	n	%	n	%	n	%		
S. sonnei biotype g	98	16.3	185	22.3	208	33.4	2.1	1.5
S. sonnei biotype a	134	22.3	232	28	118	19.0	0.9	0.7
S. sonnei untyped	37	6.1	48	5.8	57	9.2	1.5	1.6
S. flexneri 3a	37	6.1	41	5	43	6.9	1.1	1.4
S. flexneri 2a	64	10.6	55	6.6	39	6.3	0.6	0.9
S. flexneri untyped	20	3.3	21	2.5	22	3.5	1.1	1.4
S. flexneri 4	49	8.1	35	4.2	22	3.5	0.4	0.8
S. flexneri 4a mannitol neg	69	11.5	103	12.4	21	3.4	0.3	0.3
S. flexneri 4a	12	2.0	13	1.6	12	1.9	0.9	1.2
Shigella species	21	3.5	27	3.3	11	1.7	0.5	0.4

and 1.1 cases per 100,000 (18 cases) amongst the 25–29 years age group and the 20–24 years age group respectively (Figure 6). This is likely to reflect higher rates of overseas travel in these age groups.

India was the most frequently reported country of travel for overseas-acquired cases of typhoid in 2009, with 61.8% (63/102) of cases. Phage type E1 was the most common phage type of typhoid cases with a known travel status (Table 8). Of the cases where no travel was reported, four were suspected or confirmed long term carriers of *S.* Typhi acquiring the infection overseas during childhood, 2 cases were secondary infections acquired from long term carriers and 4 cases were clustered geographically, however no source was identified. Travel history was not available for the remaining 3 cases.

Hepatitis A

The number of hepatitis A cases in Australia in recent years has decreased markedly from over 2,000 cases per year during the 1990s to a mean of 274 cases per year (1.3 cases per 100,000) between 2004 and 2008 (Figure 7). In 2009, there was an increase in the number of infections reported, with 563 cases (2.6 cases per 100,000) due to a large outbreak of locally-acquired cases between 1 March 2009 and 18 March 2010 associated with the consumption of semi-dried tomatoes (described under multi-jurisdictional outbreaks investigations) (Table 1).

Indigenous status was known for 93% of cases in 2009 (Table 9). The proportion of cases of hepatitis A in Australia who identify themselves as Indigenous remains low, with only 1% of cases in 2009 known to have been Indigenous compared with 10%–12% (37 to 53 cases) per year between 2003 and 2006 to less than 2% between 2007 (0 cases) and 2008 (3 cases).

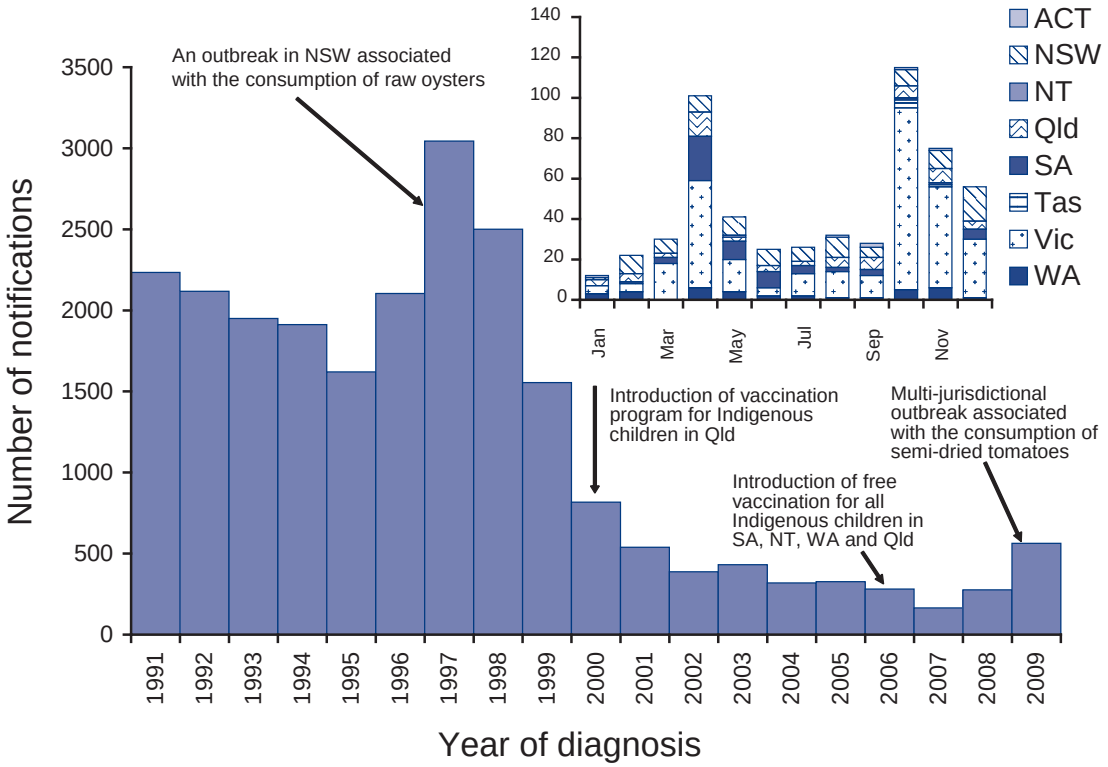
Table 9: Hepatitis A notifications, Australia, 2003 to 2009, by indigenous status

Year	Indigenous		Non-indigenous		Unknown	
	%	n	%	n	%	n
2003	12.3	53	75.4	325	12.3	53
2004	11.6	37	78.7	251	9.7	31
2005	15.0	49	70.9	232	14.1	46
2006	10.0	28	77.6	218	12.5	35
2007	0.0	0	88.5	146	11.5	19
2008	1.1	3	87.7	242	11.2	31
2009	1.4	8	91.1	513	7.5	42

Table 8: *Salmonella* Typhi phage types isolated from cases (n=115)

Country where travelled	Phage type (n)	Number of cases
India	28(1), 36(2), A(3), degraded(4), E1(30), E9(11), O var(1), unknown(3), untypable(3), blank(3), 28(1), E1 var(1)	63
Indonesia	Degraded(1), unknown(1), untypable(4)	6
Samoa	E1(3), E9(2)	5
Bangladesh	E9(1), unknown(1), untypable(2)	4
Pakistan	E1(1), E9(3)	4
Papua New Guinea	D2(3)	3
Morocco	E1(2)	2
Nepal	A(1), E1(1)	2
Thailand	E1(2)	2
Burma (Myanmar)	Untypable(1)	1
Ethiopia	36(1)	1
Fiji	E1a(1)	1
Malaysia	28(1)	1
Pakistan or Singapore	Blank(1)	1
Philippines	E1(1)	1
Somalia	Untypable(1)	1
Sri Lanka	Degraded(1)	1
Sudan	C1(1)	1
Thailand and Nepal	E1(1)	1
Uganda	E var(1)	1
No travel reported	50(1), A(5), E1(3), unknown(1), untypable(1), D1 var(2)	13

Figure 7: Notifications of hepatitis A, Australia, 1991 to 2009, by year of diagnosis[12] and inset, notifications of hepatitis A, by month and state or territory, 2009



This marked decrease in the past 3 years in the number and proportion of cases who are Indigenous is likely due to targeted vaccination programs for Indigenous children in Queensland commencing in 1999[13] and free vaccine from 2006 for Indigenous children in South Australia, the Northern Territory, Western Australia and Queensland.

Data on the place of acquisition for cases of hepatitis A was more complete than in previous years, and this may have been in part due to the multi-jurisdictional outbreak investigation, with more resources invested into complete follow-up for all cases and more complete documentation. A higher than usual proportion of cases were thought to have been locally acquired (67%, 377/563 in 2009 compared with less than 45% between 2004 and 2008), and this was also due to the outbreak. In 2009, 30% of cases (171/563) reported overseas travel during their incubation period for hepatitis A and were considered overseas acquired (Table 10). Overseas acquired cases most frequently reported travel to the Southeast Asian and South Asian regions. India was the most frequently reported country of travel, with 10% (17/171) of overseas acquired cases reporting travel to India, a higher than expected proportion since data on incoming passenger movements to Australia indicate that only 1% (73,299/12,430,460) of travellers report travel there (RR 17, 95% CI 10 to 28).

Table 10: Place of acquisition for cases of hepatitis A, 2004 to 2009, Australia

Year	Locally acquired		Acquired overseas		Unknown	
	%	n	%	n	%	n
2004	44.7	143	30.6	98	24.7	79
2005	36.7	121	31.8	105	31.5	104
2006	42.1	120	37.9	108	20.0	57
2007	30.5	50	57.9	95	11.6	19
2008	37.0	102	55.8	154	7.2	20
2009	67.0	377	30.4	171	2.7	15

Botulism

Four forms of naturally occurring botulism are recognised; adult, infant, foodborne and wound.[14] Infant botulism occurs when *Clostridium botulinum* spores are ingested, germinate in the infant's intestine and the organism produces botulinum toxin. It does not include cases where the preformed toxin is ingested: these are considered foodborne.

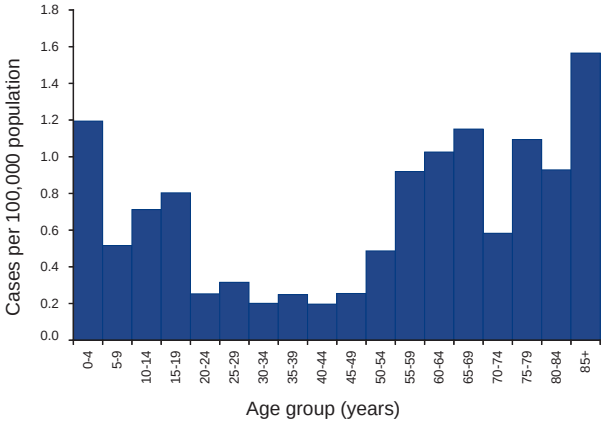
One case of infant botulism was reported in 2009, in a 5 month-old female infant from Queensland. The case was hospitalised in intensive care with onset of symptoms (acute flaccid paralysis) in March 2009. *C. botulinum* toxin was detected in a stool sample and culture by mouse bioassay, and identified as

toxin type B. The infant was entirely breast-fed. The child had not had a bowel motion for approximately 2 weeks prior to admission. It was suspected that the slow transient time within the bowel provided enough time for toxin to develop. Treatment included human immunoglobulin for infant botulism obtained from the United States of America (USA). There were no cases of botulism reported in 2008 and only 1 case was reported in 2007.

Shiga toxin-producing *Escherichia coli* infections

In 2009, there were 130 notifications of STEC in Australia, a rate of 0.6 cases per 100,000 population compared with 0.4 cases per 100,000 population between 2004 and 2008 (Table 1). The number of STEC notifications has increased over the past 5 years, from an average of 6 cases per month between 2004 and 2006 to 9 cases per month between 2007 and 2009 (Figure 8). STEC notifications have a seasonal association, tending to increase during the warmer months (November to April) (Figure 8).

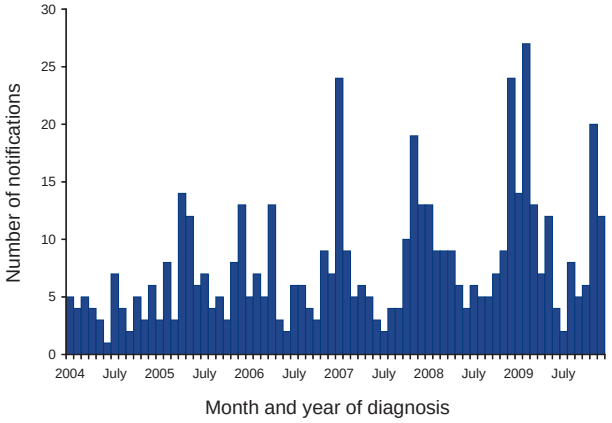
Figure 8: Shiga toxin-producing *Escherichia coli* notifications, Australia, 2004 to 2009, by month and year of diagnosis



There were no cases of STEC in the Australian Capital Territory or Tasmania in 2009. Rates of STEC infection are strongly influenced by jurisdictional practices regarding the screening of stool specimens.[15] In particular, South Australia routinely tests all bloody stools by polymerase chain reaction (PCR) for genes coding for Shiga toxins and other virulence factors, making rates for this State the highest in the country. During 2009, Queensland changed its screening procedures resulting in all stool specimens submitted for STEC testing now being screened for the presence of Shiga toxins using an enzyme immunoassay (EIA – Premier EHEC, Meridian BioScience) method in conjunction with PCR. Cases identified through this laboratory method do not meet the CDNA case definition to be included as a confirmed case. Therefore, they have been classified as 'probable' until the methodology has been reviewed and it is decided whether to include this method in the confirmed case definition. These probable cases (EIA positive only; PCR and/or culture negative) are not notified to the NNDSS.[16]

In 2009, 56.9% of cases were female. The median age of cases was 44 years (range 0–91 years). Age specific notification rates were highest in the youngest (0–19 years) with 35.4% (46/130) and oldest age groups (55 years or older) with 41.5% (54/130). The highest notification rate was amongst people aged 85 years or older (6 cases, 1.56 cases per 100,000 population) (Figure 9).

Figure 9: Age specific notification rates of STEC, Australia, 2009



In 2009, 20% (26/130) of cases were known to be outbreak associated. A further 14 cases were associated with a multi-jurisdictional cluster investigation. The STEC cluster investigation occurred between 23 March and 31 April 2009. The Microbiological Diagnostic Unit Public Health Laboratory typed isolates using pulsed field gel electrophoresis (PFGE) and phage typing and identified that amongst STEC O157 cases, there was a distinct cluster of 14 related cases. OzFoodNet epidemiologists identified several foods of interest through hypothesis generating interviews, but there were no common brands and the number of cases declined before an analytical study could be considered.

The most commonly identified serogroups (obtained by serotyping cultured isolates or by PCR targeting serotype-specific genes) of STEC cases in 2009 were O157, with 57 cases (43.8%), followed by O26 (8 cases, 6.2%) and O111 (6 cases, 4.6%). This is consistent with the serogroups reported in 2008. No organism was isolated or the serogroup was not reported for 36.9% (48/130) of cases.

Haemolytic uraemic syndrome

In 2009, OzFoodNet sites reported 12 cases of haemolytic uraemic syndrome (HUS); a rate of 0.05 cases per 100,000 population (Table 1) compared with a mean of 20 cases per year (0.10 cases per 100,000) for the years 2004 to 2008. Similar to previous years, the highest notification rate in 2009 was in children aged 0–4 years (Figure 10), with 33.3% (4 cases, 0.29 cases per 100,000) of cases notified in this age group.

Not all diagnoses of HUS are related to enteric pathogens (including those potentially transmitted by food), but in Australia cases are commonly associated with STEC. In 2009, an antecedent STEC infection was reported for 41.7% (5/12) of cases, with serogroup information reported for 80% (4/5) of these cases. *E. coli* O111 was reported in 2 instances, while serotypes ONT:H19 and OR:H25 were reported for 1 case each. For 1 HUS case for which *E. coli* infection was not confirmed, contact tracing revealed that the mother of the case was positive for *E. coli* O111 infection but was asymptomatic. Of the remaining non-STEC HUS cases, one was associated with a non-STEC *E. coli* infection, 1 case resulted from *Streptococcus pneumoniae* infection and in the remaining 4 cases, no aetiology was reported though one of these had multiple underlying health conditions.

In Australia, HUS cases show a seasonal pattern, tending to increase during late spring and early summer, with 46.5% (79/170) of cases between 1999 and 2008 occurring in November, December or January (Figure 11) compared with the expected 25% (3/12 months) of cases occurring during these months. In 2009, 33.3% (4/12) of cases occurred in November, December or January (Figure 11).

Gastrointestinal and foodborne disease outbreaks

During 2009, OzFoodNet sites reported 1,820 outbreaks of gastroenteritis, including both foodborne and non-foodborne outbreaks, which affected 36,426 people. During these outbreaks, 1,240 peo-

Figure 10: Age specific notifications of haemolytic uraemic syndrome, Australia, 2009

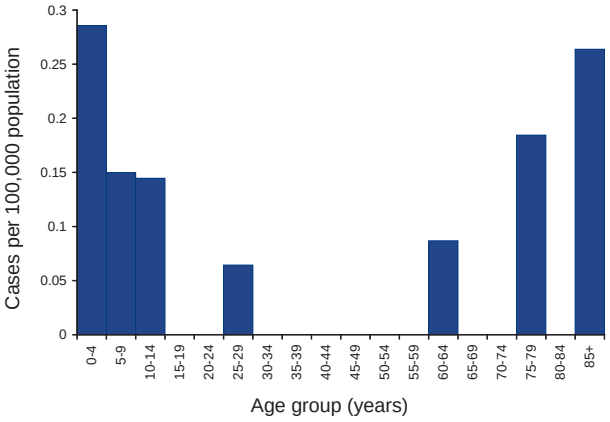


Figure 11: Notifications of haemolytic uraemic syndrome by month of diagnosis, Australia, 1999 to 2008, and inset, notifications of haemolytic uraemic syndrome by month of diagnosis, 2009

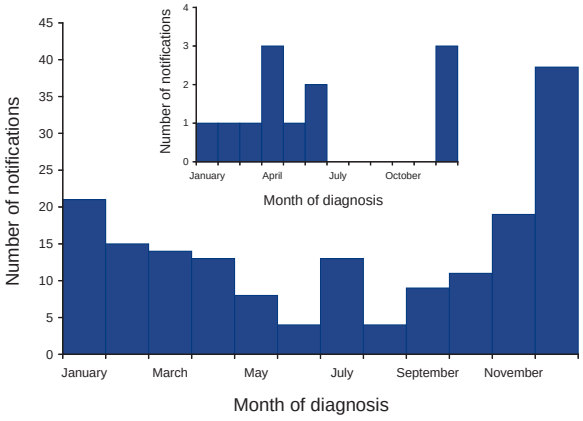


Table 11: Outbreaks of gastroenteritis including foodborne disease reported to state and territory health departments, Australia, 2009

Mode of transmission	Number of outbreaks	Number affected	Hospitalised	Fatalities
Foodborne*	163	2,679	342	8
Person-to-person	1,496	33,070	824	105
Unknown mode (*Salmonella* cluster)	15	168	14	0
Unknown mode (other pathogen cluster)	9	61	18	0
Unknown mode (unknown aetiology)	125	1,266	30	5
Waterborne	12	182	12	0
Total	1,820	36,426	1,240	118

* Includes 3 multi-jurisdictional outbreaks.

ple were hospitalised and there were 118 deaths (Table 11). This compares with the 5-year mean (2004–08) of 1,336 outbreaks reported in Australia.

Outbreaks spread person-to-person

In 2009, 82% (1,496/1,820) of all gastroenteritis outbreaks were reported as person-to-person transmission, affecting 33,070 people with 105 deaths. Aged care facilities (42%, 627/1,496) were the most frequently reported settings for person-to-person outbreaks, followed by hospitals (9%, 134/1,496) and child care centres (9%, 129/1,496). Fifty-two per cent (785/1,496) of person-to-person outbreaks were caused by norovirus and 32% (482/1,496) were of unknown aetiology. The number of person-to-person outbreaks due to norovirus does not include a small number of outbreaks of mixed aetiology that included norovirus or outbreaks where norovirus could not be confirmed as the aetiology of the outbreak. Spring was the peak season for person-to-person outbreaks, with 46% (685/1,496) of outbreaks reported in the months of September to November 2009.

Waterborne outbreaks

There were 12 outbreaks due to waterborne transmission, affecting 182 people. The largest outbreak, which had an unknown aetiology, affected 135 people and illness was suspected to have been associated with contaminated water at a school. *Cryptosporidium spp.* was the causative agent associated with 9 swimming pool outbreaks between January and March. The aetiologies of the remaining 2 outbreaks were unknown.

Outbreaks with unknown mode of transmission

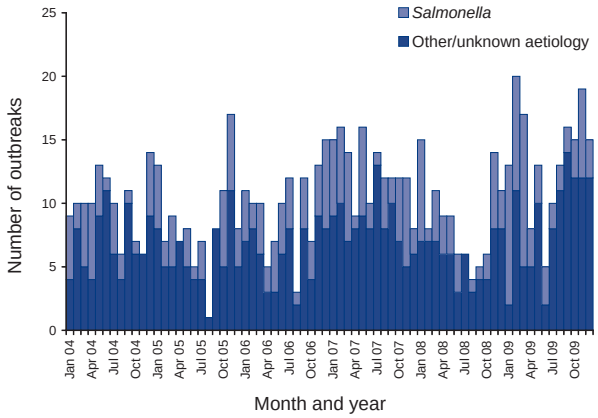
There were 149 outbreaks where the mode of transmission was not determined (clustered in time, place or person where investigators were unable to develop an adequate hypothesis for the source of illness) affecting a total of 1,495 people. There were 15 clusters of *Salmonella*, 9 clusters due to other pathogens and 125 clusters where neither the mode of transmission nor the aetiology could be determined.

Foodborne outbreaks

In 2009, there were 163 outbreaks of foodborne disease affecting 2,679 people of whom 342 people were hospitalised. There were 8 deaths reported during these outbreaks (Appendix). This compares with the 5-year mean (2004–08) of 118 foodborne outbreaks. Three of the 163 outbreaks were multi-jurisdictional, affecting 433 people with 168 hospitalisations and 4 deaths.

The overall rate of reported foodborne disease outbreaks for Australia was 7.5 outbreaks per million population in 2009 (Table 12). The highest rates of reporting were from the Australian Capital Territory (17.1 per million population) and the Northern Territory (13.3 per million population, although representing a small number of outbreaks). Outbreaks were more common in warmer months (Figure 12).

Figure 12: Outbreaks of foodborne disease reported to state and territory health departments, by aetiology month of outbreak, Australia, 2004–9 (n=751)



Aetiological agents

The mostly commonly implicated aetiological agent in outbreaks of foodborne illness was *Salmonella*, which caused 36% (59/163) of outbreaks and 80% (47/59) of these were due to *S.* Typhimurium (Table 13). The most commonly implicated *S.* Typhimurium subtype was phage type 170/108 (26 outbreaks) and included the following multi-locus variable number of tandem repeats analysis (MLVA) patterns 3-9-8-12-523 (6 outbreaks), MLVA 3-9-7-13-523 (4 outbreaks), MLVA 3-9-7-13-523 (2 outbreaks) and MLVA 3-9-7-12-532 (1 outbreak). There were also 5 outbreaks of phage type 44.

Toxin-mediated outbreaks comprised 9% (15/163) of all foodborne outbreaks, with 33% (5/15) of these due to fish toxins (2 outbreaks of ciguatera fish poisoning and 3 outbreaks of scombroid or histamine poisoning) and 67% (10/15) due to foodborne intoxications with *Clostridium perfringens*.

There were 3 foodborne outbreaks of *Campylobacter*, and norovirus was confirmed in 30 outbreaks. In the USA contamination of foods with norovirus is thought to be principally due to poor hygiene practices of foodhandlers.[17] In 2009, 27% (44/163) of foodborne outbreaks were of unknown aetiology compared with 37% in the previous year. Outbreaks

Table 12: Outbreaks of foodborne disease in Australia, 2009, by OzFoodNet site

State	Number of outbreaks	People affected	Mean size (persons)	Hospitalised	Outbreaks per million population
ACT	6	85	14.2	1	17.1
NSW	67	903	13.5	74	9.4
NT	3	2	0.7	0	13.3
Qld	20	164	8.2	11	4.5
SA	14	190	13.6	36	8.6
Tas	3	58	19.3	0	6.0
Vic	29	574	19.8	20	5.3
WA	18	270	15.0	32	8.1
Multijurisdictional	3	433	144.3	168	0.1*
Australia	163	2,679	16.4	342	7.5

* Calculated using Australia's total population.

Table 13: Aetiological agents responsible for foodborne disease outbreaks, number of outbreaks and persons affected, Australia, 2009

Agent category	Number of outbreaks	People affected	Mean size (persons)	Hospitalised
Salmonella Typhimurium	47	646	13.7	110
Norovirus	30	731	24.4	7
Other *Salmonella* serotypes	12	119	9.9	22
Foodborne intoxication	10	139	13.9	2
Ciguatera/histamine poisoning	5	15	3	6
Hepatitis A	4	411	102.8	170
Campylobacter	3	44	14.7	0
Shiga toxin-producing *Escherichia coli*	2	37	18.5	8
Listeria monocytogenes	2	38	19	4
Fish wax ester	2	30	15	0
Escherichia coli	1	Unknown	Unknown	0
Yersinia enterocolitica	1	3	3	0
Unknown	44	466	10.6	13
Total	163	2,679	16.4	342

due to hepatitis A and fish wax ester outbreaks were implicated in 2% (4/163) and 1% (2/163) of all foodborne outbreaks respectively. STEC and *L. monocytogenes* were each implicated in 1% of outbreaks (2/163) and 1 outbreak (1%, 1/163) was due to *E. coli*. *Yersinia enterocolitica* was identified in 1 outbreak (1% of all foodborne outbreaks).

Food vehicles

A wide variety of food vehicles were implicated in outbreaks of foodborne disease in 2009, and investigators were unable to identify a food vehicle in 58% (94/163) outbreaks (Table 14).

There were 18 outbreaks (11% of all foodborne outbreaks) associated with eggs (Table 15). Eight of these outbreaks were suspected or confirmed to have involved desserts that commonly contain raw egg (such as tiramisu and fried ice-cream), five were due to egg based sauces or dressings (such as aioli and mayonnaise), 4 outbreaks were suspected to have been caused by chicken and/or eggs and 1 outbreak was due to eggs as a whole food. These outbreaks affected a total of 343 people and hospitalised 54 people.

Ten (6%) of the 163 foodborne outbreaks were due to mixed dishes where investigators were unable to implicate a particular ingredient, 10 (6%) were due to or suspected to be due to fish or seafood dishes (including the multi-jurisdictional outbreak of *S.* Litchfield suspected to be associated with a barramundi meal) and 9 (6%) were confirmed or suspected to be due to be salads and/or sandwiches.

Table 14: Categories of food vehicles implicated in foodborne disease outbreaks, Australia, 2009

Vehicle category	Number of outbreaks	Number affected	Mean size (persons)	Hospitalised
Mixed dishes	10	123	12.3	15
Fish/seafood	8	47	5.9	6
Egg containing desserts	7	88	12.6	12
Suspected salad and/or sandwiches	7	75	10.7	9
Egg based sauces and dressing	5	187	37.4	28
Meat and meat containing dishes	5	75	15.0	0
Chicken and chicken containing dishes	4	103	25.8	2
Suspected chicken and/or eggs	4	23	5.8	5
Semi dried tomatoes	3	406	135.3	169
Fruit	3	36	12.0	6
Salads and/or sandwiches	2	46	23.0	0
Dessert	2	25	12.5	0
Suspected gravy	2	31	15.5	6
Suspected meat and meat containing dishes	2	11	5.5	2
Suspected seafood	2	8	4.0	1
Suspected vitamised foods	1	22	22.0	0
Eggs	1	39	39.0	7
Suspected egg containing desserts	1	6	6.0	2
Unknown	94	1,328	14.1	72
Total	163	2,679	16.4	342

Four per cent (7/163) were due to or suspected to be caused by meat or meat containing dishes. Two per cent (4/163) were due to chicken or chicken containing dishes (including the multi-jurisdictional outbreak of *Listeria* associated with chicken wraps) and 4% (6/163) were due to produce (including the multi-jurisdictional outbreak of hepatitis A associated with semi-dried tomatoes). The remaining outbreaks were suspected to have been due to gravy (2), dessert (2) and suspected vitamised foods (1) .

Settings where food was prepared

In 2009, foods implicated in outbreaks were most commonly prepared in restaurants (39%, 64/163), aged care facilities (12%, 20/163) or by commercial caterers (11%, 18/163) (Table 16).

In 2009, implicated foods that were contaminated in primary produce environments (6%, 10/163) were Spanish mackerel, escolar fish, tuna, anchovies, semi-dried tomatoes, berries, fresh chilli, and paw-paw.

Investigative methods and levels of evidence

To investigate these foodborne outbreaks, epidemiologists in the states and territories conducted 37 retrospective cohort studies and 8 case control studies. Descriptive case series investigations were conducted for 108 outbreaks. There was no patient data collected for 10 outbreaks. Analytical evidence and microbiological evidence were obtained for 5 outbreaks, analytical evidence alone was obtained for 14 outbreaks and microbiological evidence alone was obtained for 14 outbreaks. Investigators relied on descriptive evidence implicating the food vehicle in 128 outbreaks and 1 outbreak relied on descriptive and microbiological evidence.

Significant outbreaks

In 2009, there were 9 outbreaks of foodborne illness affecting 40 or more people per outbreak; 1 outbreak of *C. perfringens,* 2 outbreaks of norovirus, 2 outbreaks of *S.* Typhimurium 170 and 1 outbreak of unknown aetiology. In total these outbreaks affected 453 people, ranging between 40 and 165 people per outbreak with 19 people hospitalised. There were 3 multi-jurisdictional outbreaks that also affected more than 40 people (described under multi-jurisdictional outbreak investigations).

Victoria reported 3 significant outbreaks.

- An outbreak of gastrointestinal illness affected 165/284 people from 2 separate groups attending a restaurant on 2 consecutive nights in November. Only 1 stool sample was collected, which was positive for norovirus. The symptoms and median incubation period for cases were consistent with norovirus aetiology. A food vehicle was not identified.

Table 15: Outbreaks of foodborne illness associated with eggs, Australia, 2009 (n=18)

State	Setting prepared	Agent responsible	Number affected	Evidence	Responsible vehicles
ACT	Restaurant	*Salmonella* Typhimurium 170	20	A	Tiramisu
NSW	Restaurant	*Salmonella* Singapore	3	M	Fried ice cream prepared with raw eggs
	Restaurant	*Salmonella* Typhimurium 170 MLVA 3-9-7-12-523	33	AM	Fried ice cream prepared with raw eggs
	Restaurant	*Salmonella* Typhimurium 170 MLVA 3-9-8-12-523	40	A	Hollandaise sauce prepared with raw eggs
	Commercial caterer	*Salmonella* Typhimurium 170 MLVA 3-9-8-12-523	68	AM	Mayonnaise prepared with raw eggs
	Bakery	*Salmonella* Virchow	10	D	Suspected Margarine/butter prepared with raw eggs
	Bakery	*Salmonella* Typhimurium 170 MLVA 3-9-8-12-523	9	D	Suspected chicken and/or eggs
	Private residence	*Salmonella* Typhimurium MLVA 3-13-9-11-550	6	D	Suspected tiramisu prepared with raw eggs
	Restaurant	*Salmonella* Typhimurium 170 MLVA 3-9-7-13-523	2	D	Suspected chicken and/or eggs
NT	Private residence	*Salmonella* Typhimurium U302	2	D	Suspected Tiramisu
SA	Private residence	*Salmonella* Typhimurium 44	16	A	Tiramisu
	Restaurant	*Salmonella* Typhimurium 135	7	D	Fried ice cream
	Restaurant	*Salmonella* Typhimurium 44	30	A	Garlic aioli
Vic	Private residence	*Salmonella* Typhimurium 3	6	D	Suspected eggs
WA	Restaurant	*Salmonella* Typhimurium 170	39	A	Scrambled eggs
	Restaurant	*Salmonella* Singapore	6	D	Suspected chicken and/or eggs
	Restaurant	*Salmonella* Saintpaul	7	M	Fried ice cream
	Takeaway	*Salmonella* Typhimurium 170	39	D	Raw egg mayonnaise

D Descriptive evidence implicating the vehicle

A Analytical epidemiological association between illness and vehicle

M Microbiological confirmation of aetiology in vehicle and cases

Table 16 Food preparation settings implicated in disease outbreaks, Australia, 2009

Setting prepared	Number of outbreaks	Proportion of all outbreaks (%)	Number affected (persons)
Restaurant	64	39	921
Aged care facility	20	12	294
Commercial caterer	18	11	343
Private residence	11	7	74
Primary produce	10	6	471
Takeaway	9	5	149
Other	8	5	128
Bakery	4	2	51
Camp	4	2	89
Military	2	1	23
School	1	1	37
Child care	1	1	18
Fair/festival/mobile service	1	1	3
National franchised fast food	1	1	3
Unknown	9	5	75
Total	163	100	2,679

- An outbreak of gastrointestinal illness affected 87 people from 8 separate groups dining at the same restaurant on 3 consecutive days in August. Illness was suspected to be due to contamination of food during a smorgasbord service of meals. Two out of 7 people interviewed were food handlers at the restaurant and reported illness with onset dates consistent with the patrons. Thirteen cases were confirmed with norovirus.
- An outbreak of gastroenteritis was reported among 41/86 people after consuming foods prepared by a commercial caterer in October. The description of symptoms, incubation period, duration of symptoms and 1 secondary case was consistent with a viral aetiology. The analysis of the cohort study revealed no significant associations with any specific food or illness. There were no reports of any illness at the function and no food handlers were reported to have been ill. It is suspected that this outbreak was foodborne although there was no definitive evidence of foodborne transmission.

New South Wales reported 2 significant foodborne outbreaks.

- An outbreak of gastroenteritis was reported amongst 68 of 120 people who consumed foods prepared by a commercial caterer in January. Fourteen people were hospitalised due to illness. Twenty-four of 30 collected stool samples were positive for *S.* Typhimurium 170 MLVA 3-9-8-12-523. A homemade raw egg mayonnaise served by the commercial caterer was found to contain *S.* Typhimurium 170 MLVA 3-9-8-12-523.
- An outbreak of gastrointestinal illness affected 40 of 100 people at a restaurant in January. Five people were hospitalised due to illness. *S.* Typhimurium 170 MLVA 3-9-8-12-523 was isolated in 2 of 8 stool samples. A cohort study found an association with hollandaise sauce prepared with raw eggs; however there was no food remaining from the function that could be tested.

The Australian Capital Territory reported an outbreak of gastroenteritis illness affecting 52 of 126 residents of an aged care facility in July. Aetiology of the outbreak was confirmed as *C. perfringens* with 8 of 52 stool samples from residents positive for *C. perfringens* enterotoxin A. There were no food isolates available for testing; however a cohort study suggested an association between illness and the level of service offered to residents (which reflected meal options).

Multi-jurisdictional outbreak investigations

In 2009 there were 3 multi-jurisdictional outbreak investigations coordinated by OzFoodNet.

Salmonella Litchfield

An outbreak of *Salmonella* Litchfield occurred in June during a charity car rally travelling through Queensland and the Northern Territory.[18] The investigation team contacted 286 participants inviting them to complete an online survey. In total, 43% (76/176) of respondents were ill with gastroenteritis, including 5 confirmed cases of *S.* Litchfield across four jurisdictions; New South Wales (2), Victoria (1), Queensland (1) and Western Australia (1). Consumption of a variety of foods and meals were associated with illness, with barramundi having the highest relative risk (RR= 3.8, 95% CI 1.0–14.2) for illness. A source of illness was not definitively identified.

Listeria monocytogenes

A multi-jurisdictional outbreak of 13 laboratory-confirmed cases of invasive listeriosis (*Listeria monocytogenes* molecular serotype: 1/2c, binary gene type: 82) occurred between January and July with cases in Queensland (5), Victoria (3), New South Wales (2), South Australia (1), Western Australia (1) and Tasmania (1). In addition, there were 23 epidemiologically-linked cases of non-invasive illness associated with this outbreak, 22 with clinical symptoms of gastroenteritis only. Headaches (100%), fever (100%), diarrhoea (100%), abdominal pain (96%) and vomiting (43%) were the most commonly reported symptoms among the 22 clinical cases.

A case–case comparison study of outbreak cases and non-outbreak sporadic cases was conducted to identify potential risk factors associated with the outbreak. Eight of the 13 of the laboratory-confirmed cases were materno-foetal infections with 3 foetal deaths at 15, 20 and 40 weeks gestation. Eight of the 13 laboratory-confirmed cases and 17 of 22 clinical cases reported consuming chicken wraps on a particular domestic airline. Laboratory confirmed cases infected with the outbreak strain (n = 13) were more likely to have flown on a domestic airline in the 3 months before onset of illness (OR 30.0, 95% CI: 2.3, 885.7, $P < 0.001$) and more likely to have consumed chicken wraps (OR 27.2, 95% CI: 2.2, 758.5, $P = 0.001$), when compared with sporadic cases of *L. monocytogenes* infected with other strains (n = 40). Traceback investigation subsequently led to the isolation of the outbreak strain of *Listeria* from pre-packaged chicken sandwiches and wraps. The cooked diced chicken meat used in the sandwiches and wraps was supplied by a New South Wales food processing business to a food manufacturer in Queensland where the sandwiches and wraps were prepared. The food manufacturer in Queensland was a supplier of the chicken wraps to the domestic airline and several other food businesses. An

environmental investigation identified deficiencies in the food safety program for the production of chicken meat.

Hepatitis A

A large outbreak of locally-acquired hepatitis A (genotype 1B†) was investigated between 1 March 2009 and 18 March 2010 (the outbreak period) manifesting as 2 separate temporal clusters peaking in April and November 2009 (Figure 7).

In May 2009, OzFoodNet reported an increase in locally-acquired hepatitis A cases in Victoria, South Australia, and Queensland triggering a multi-jurisdictional outbreak investigation. The number of reported cases of hepatitis A nationally returned to expected levels by the end of May. Victoria commenced investigating a second wave of cases beginning at the end of June 2009 and the multi-jurisdictional outbreak investigation was re-opened on 2 November 2009. Three separate case control studies were conducted to investigate the outbreak, and each confirmed associations between illness and the consumption of semi-dried tomatoes.[19,20]

There were 415 locally-acquired cases of hepatitis A in Australia during the outbreak period, the majority (64%, 267/415) of them from Victoria. Cases were considered to have been locally acquired if they had no reported overseas travel in the 50 days prior to the onset of symptoms. There were 372 primary cases, 43 secondary cases (considered likely to have been due to person-to-person transmission from previously reported cases) and three were unknown. Of the 244 primary cases who were interviewed, nearly half (47%, 115/244) reported consuming semi-dried tomatoes, while 25% (61/244) could not recall. A tiered case definition was used to define outbreak cases. A confirmed outbreak case was a locally-acquired and laboratory-confirmed case of hepatitis A virus (HAV) infection with genotype 1B, a suspected outbreak case was a locally-acquired and laboratory-confirmed case of HAV infection with subtype pending or unavailable and a sporadic case was locally acquired and laboratory confirmed as infected with a HAV genotype other than 1B. There were 169 confirmed and 223 suspected outbreak cases during the outbreak period, 67% (261/392) of them from Victoria. Nearly half of all suspected and confirmed outbreak cases were hospitalised (42%, 165/392) and 1 case from Victoria was fatal.

A range of public health actions resulted from the investigations. Trade level recalls were conducted in South Australia in May 2009 following the outcomes of the first case control study and in Victoria in October 2009 after product of a particular brand tested positive for HAV genetic material. In November 2009, Victoria's Chief Health Officer exercised an emergency power under the *Victorian Food Act 1984,* requiring manufacturers of semi-dried tomatoes to either pasteurise finished semi-dried tomato products or to ensure that all tomatoes used in the production were sanitised prior to drying. Media releases were issued by Victoria, South Australia and Queensland in May 2009 advising about the first recall and not to eat specific brands and by Victoria, Western Australia and Tasmania in November 2009 advising consumers not to eat semi-dried tomatoes unless thoroughly cooked.

Information provided through a notification under the World Health Organization (WHO) *International Health Regulations* (2005), via the WHO International Food Safety Authorities Network (INFOSAN) and the European Centre for Disease Control prompted the Euro virology network to compare sequences and identify a related cluster of hepatitis A in The Netherlands. The sequences of the Australian outbreak strain and the cluster in The Netherlands were found to be identical.[21] The sequence of the hepatitis A virus from the outbreak in France was similar but not the same as the virus from The Netherlands and Australian outbreaks.[21] In an outbreak in France, investigators were alerted to the possibility of an epidemiological link with semi-dried tomatoes. Case-control studies identified semi-dried tomatoes as the source of infection in both countries.[21 22]

Cluster investigations

In 2009, OzFoodNet epidemiologists and state and territory health departments investigated 24 clusters of various aetiologies. A cluster is defined as an increase in a specific infection in terms of time, place, or person where a source and mode of transmission remains unknown. The majority of these investigations involved *Salmonella* serotypes (15) for which no common food vehicle or source of infection could be identified: *S.* Havana, *S.* Singapore, *S.* Stanley, *S.* Typhimurium (phage types 141 and 170 and MLVA type 3-9-7-13-523), *S.* Virchow, *S.* Wangata, *S.* Infantis and *S.* Heidelberg. However, a multi-jurisdictional cluster of *Salmonella* Typhimurium 170/108 occurring between 3 April and 20 May 2009 in Queensland, New South Wales and the Australian Capital Territory was suspected to have been associated with chicken and/or eggs. The multi-jurisdictional cluster consisted of 15 smaller outbreaks of *S.* Typhimurium 170/108 that were counted as individual foodborne outbreaks (described under gastrointestinal and

† The designation 1B has been agreed amongst the 3 laboratories conducting genotyping of isolates for this outbreak and may not be directly comparable to results obtained in other laboratories or over time.

foodborne outbreaks). The remaining 9 clusters were due to a variety of infections; *Campylobacter, Cryptosporidium, Shigella, Yersinia* and STEC.

Discussion

This report documents changes in the incidence of gastrointestinal diseases commonly transmitted by food in Australia. Foodborne disease surveillance provides information to assist immediate public health action, the prevention of these diseases and the assessment of food safety policies and campaigns. A national program of surveillance for foodborne diseases and outbreak investigation has many benefits including identifying foods that cause human illness through investigation of outbreaks that occur across state and territory borders. Continuing efforts to strengthen the quality of these data will ensure their use by agencies to develop food safety policy contributing to the prevention of foodborne illness.

Similar to 2008, higher rates of campylobacteriosis were observed in males than in females, particularly those over the age of 45 years.[12] In Australia, the primary source of *Campylobacter* infection is thought to be chicken consumption, causing an estimated 29.3% of all infections.[23] This is consistent with findings from other countries, although recent work in New Zealand highlights that the proportion of campylobacteriosis due to chicken meat consumption may be higher.[24] In 2009, the New Zealand Food Safety Authority announced that the poultry industry had successfully reduced the prevalence of *Campylobacter* in chicken meat, which had lead to a marked decline in human cases.[25] In March 2010, the FSANZ Board approved a draft Primary Production and Processing Standard for Poultry Meat, which will introduce new requirements within the poultry industry with the aim of reducing the prevalence of *Campylobacter* and *Salmonella* in poultry meat.[26]

In 2009, the proportion of *Salmonella* isolates that contained appropriate information on serotype and/or phage type decreased by 2.4% compared with 2008. Typing is vital for outbreak detection and monitoring trends. Western Australia's *Salmonella* isolates are phage typed in other jurisdictions and in 2007 as an alternative, Western Australia started using PFGE, which is conducted within the jurisdiction. PFGE is a discriminatory technique for typing *Salmonella* but not routinely used by other Australian laboratories.[27] Other jurisdictions used MLVA to compare strains during outbreaks, which proved rapid and very useful. Inconsistencies in typing schemes, including differences in MLVA nomenclature between states and territories, cause complexity during multi-jurisdictional investigations. Despite this there is increasing harmonisation in typing schemes used by Australian laboratories. OzFoodNet has identified the need for more complete and consistent *Salmonella* subtyping (serotypes, MLVA, phage typing and PFGE for clusters) nationally to better identify multi-jurisdictional outbreaks. The CDNA's National Surveillance Committee has acknowledged this by including salmonellosis on its national typing priority list in 2010.

The Department of Foreign Affairs and Trade provides specific country information to travellers about health risks and measures that can be taken to reduce the risk of infection.[28] In this report we summarised 3 infections commonly associated with travel overseas; typhoid (89% of cases), hepatitis A (30% of cases) and *S.* Enteritidis (87% of cases). The percentage of overseas acquired infections of hepatitis A are lower than previous years (in 2008 54.7% cases were overseas acquired) due to larger numbers of locally-acquired hepatitis A cases attributed to a large outbreak of hepatitis A associated with semi dried tomatoes. Travel to South East Asia and India were the most common place of acquisition for these infections. People who travel overseas are at a higher risk of developing infections without recommended preventative vaccinations for particular infections or appropriate caution when consuming water and food in overseas countries.

In 2009, OzFoodNet sites reported 1,820 outbreaks of gastrointestinal disease, which was 18% more than that reported in 2008 (1,545).[12] Similar to previous years, the majority of outbreaks in 2009 were transmitted from person-to-person (82%) and were most frequently caused by norovirus (52%) followed by those of unknown aetiology (44%). Aged care facilities (42%) was the most commonly reported setting for person-to-person infections, reflecting the frequency with which outbreaks of gastrointestinal illness occur, the ease of transmission in this setting and the improved reporting practices of these facilities. Case reporting of gastrointestinal outbreaks in aged care settings are well established, with outbreak preparedness enhanced through the introduction of resources, such as the Department of Health and Ageing Gastroenteritis Kit for Aged Care[29] to manage and prevent outbreaks in this setting.

Norovirus is one of the most common causes of gastroenteritis outbreaks globally. In response to increasing reports of outbreaks in 2005, CDNA proposed developing national guidance regarding outbreaks of norovirus and suspected viral gastroenteritis. A CDNA working group developed the *Guidelines for the public health management of gastroenteritis outbreaks due to norovirus or suspected viral agents in Australia*.[30] The guidelines are designed to assist state and territory health departments and public health units in managing outbreaks of gastroenteritis due to norovirus or suspected viral agents, and provide advice to aged care homes regarding management of suspected viral outbreaks. The guidelines comple-

ment existing state and territory protocols and the guidelines and were endorsed by CDNA and the Australian Health Protection Committee (AHPC) in early 2010.

In 2009, OzFoodNet sites reported 163 foodborne or suspected foodborne outbreaks (including 3 multi-jurisdictional outbreak investigations), a rate of 7.5 outbreaks per million population. This is a higher reporting rate than in 2008 with 4.9 outbreaks per million population, which was comparable with an estimated 4.18 outbreaks per million in the USA in 2006.[31] *Salmonella* continues to be the leading cause of reported outbreaks of foodborne illness in Australia, with 36% of outbreaks due to this pathogen, the majority of them due to *S.* Typhimurium (80%). In 2009, there were 9 large outbreaks of foodborne illness (affecting 40 or more people) including 3 multi-jurisdictional outbreaks. Excluding the multi-jurisdictional outbreaks, the largest of these large outbreaks was suspected to have been due to norovirus and affected 165 people who dined at the same restaurant over 2 consecutive nights. A food vehicle was not identified in this outbreak.

Eggs were suspected as the cause of 26% (18/69) of foodborne outbreaks where investigators were able to identify a food vehicle. Eggs are a commonly consumed food, used as an ingredient of many dishes, and may be served raw or lightly cooked in dishes such as aioli, sauces and desserts. It is important that egg safety continues to be improved in Australia. During 2009, FSANZ continued developing a primary production and processing standard for eggs that is considering safety of the whole production chain from farm through to retail.[32]

It is important to recognise some of the limitations of the data used in this report. Where there are small numbers of notifications, caution must be used in comparisons between jurisdictions and over time. Some of the most common enteric pathogens are not notifiable, particularly norovirus and *C. perfringens*, which is why surveillance of outbreaks is important. A limitation of the outbreak data provided by OzFoodNet sites for this report is the potential for variation in categorising features of outbreaks depending on investigator interpretation and circumstances. States and territories are working towards harmonising surveillance and outbreak data to address some of these issues.

In 2004, DoHA commissioned an evaluation of Australia's capacity to investigate outbreaks of foodborne illness. It was clear from the assessment that while there had been a marked improvement in capacity since the establishment of OzFoodNet in 2000, there was no national plan describing who would perform what functions during major national outbreaks, or during more common smaller multi-jurisdiction cluster investigations. In response to this evaluation, OzFoodNet developed its *Guidelines for the investigation and management of multi-jurisdictional outbreaks of foodborne illness* (the Guidelines) which have been used by the network in draft form since May 2009. The Guidelines provide clear guidance framework to the OzFoodNet network for national management and investigation of multi-jurisdictional outbreaks potentially linked to contaminated food sources in a timely, appropriate, consistent and coordinated manner. The Guidelines formalise current arrangements between agencies that investigate multi-jurisdictional outbreaks of foodborne illness and complement the National Food Incident Response Protocol developed by FSANZ. The Guidelines were endorsed by CDNA in September 2010 and are pending endorsement by the AHPC. The effectiveness of each multi-jurisdictional outbreak investigation is assessed by OzFoodNet, and any necessary enhancements made to the Guidelines, through a structured audit process, using the template provided in the Guidelines.

On 11 February 2010, representatives of OzFoodNet's national and jurisdictional sites, state health departments, the NSW Food Authority, FSANZ and public health laboratories met in Newcastle to conduct a debrief of the response to the 2009 multi-state outbreak of listeriosis. The debrief identified the need for nationally standardised rapid subtyping of *Listeria* isolates from humans and for the centralised collection of epidemiological data. An action arising from the meeting was to develop a conceptual plan for the surveillance of human *Listeria* isolates to ensure they are typed using a national approach and that epidemiological data are available for rapid analysis of clusters. This plan also requires states and territories to undertake molecular serogroup and binary typing of *Listeria* isolates to enable clearer identification of clusters and outbreaks. This plan has been endorsed by Public Health Laboratory Network.

While the proportion of hepatitis A infections that may be foodborne is thought to be less than 10%, it is important to keep this infection under surveillance as it can manifest in large outbreaks of foodborne disease. This was observed in the 2009 outbreak of hepatitis A associated with semi-dried tomatoes, an outbreak of international public health concern.[33,34] The outbreak was a major investigative and control effort for all of the agencies involved; state and territory health authorities, Food Standards Australia New Zealand, OzFoodNet (in the jurisdictions and in the Department of Health and Ageing) and laboratory staff. The hepatitis A outbreak occurred concurrently with the 2009 pandemic influenza A (H1N1) between mid-May and late September 2009[35] and many foodborne disease epidemiologists and laboratory technicians were also

integral to surveillance and response efforts for the pandemic. The hepatitis A outbreak highlighted the effectiveness of the OzFoodNet surveillance model, with enhanced inter-jurisdictional communication and collaboration of epidemiological laboratory and traceback evidence leading to early detection and a rapid response. Sharing information internationally about the outbreak in Australia was vital to investigators finding the source of infection for outbreaks occurring overseas. The WHO INFOSAN network proved an effective network for coordination of the international investigation, and Australia's ability to liaise and investigate potential sources of contaminated raw product overseas was greatly enhanced by the network.

In 2009, OzFoodNet provided epidemiological support to the investigation of a cluster of thyroid conditions thought to be associated with the consumption of particular seaweed and products containing seaweed that were found to contain high levels of iodine and were subsequently recalled. Between 23 December 2009 and 6 October 2010, 50 cases of thyroid dysfunction that were suspected to be associated with the consumption of products containing excessive levels of iodine, were reported to state and territory health authorities, and collected into a national database. Forty-seven of these cases were associated with Bonsoy soy milk, two were associated with an unknown brand soy milk and one was due to consumption of a dried seaweed product prepared as a soup. Cases were reported from Victoria (25), New South Wales (20), South Australia (2), Western Australia (2) and the Australian Capital Territory (1).

In May 2009, DoHA hosted the WHO's 9th annual Global Salm-Surv Steering Committee meeting in Canberra. The Steering Committee oversees WHO's international program for the enhancement of laboratory-based foodborne disease surveillance and outbreak detection and response worldwide. WHO's Global Salm-Surv Steering Committee members attended the 9th annual meeting from WHO, Switzerland; National Food Institute, Denmark; National Institute of Public Health, Japan; Public Health Agency of Canada; OzFoodNet, Australia; and the Centres for Disease Control and Prevention, United States. At the meeting, the Steering Committee agreed to change its name to the Global Foodborne Infections Network: A WHO network building capacity to detect, control and prevent foodborne and other enteric infections from farm to table.

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Appendix : Foodborne outbreak summary for OzFoodNet sites, Australia, 2009

State or territory	Month of outbreak	Setting prepared	Agent responsible	Number affected	Hospitalised	Fatalities	Evidence	Epidemiological study	Responsible vehicles	Vehicle category
ACT	February	Private residence	Scombroid	2	1	0	D	Descriptive case series	Tuna steak	Fish/seafood
	February	Restaurant	*Salmonella* Typhimurium 170	20	0	0	A	Case control study	Tiramisu	Egg containing desserts
	March	Private residence	*Salmonella* Typhimurium 170	5	0	0	D	Descriptive case series	Zucchini bake	Mixed dishes
	May	primary produce	Rudderfish/escolar	3	0	0	D	Descriptive case series	Rudderfish	Fish/seafood
	July	Aged care facility	*Clostridium perfringens*	52	0	0	A	Cohort	Sweet and sour pork probable food vehicle	Meat and meat containing dishes
	July	Restaurant	*Yersinia enterocolitica*	3	0	0	D	Descriptive case series	BBQ pork or roast pork	Meat and meat containing dishes
NSW	January	Aged care facility	*Salmonella* Typhimurium 170 MLVA 3-9-7-13-523	4	0	0	D	N	Unknown	Unknown
	January	Bakery	*Salmonella* Typhimurium 170 MLVA 3-9-8-12-523	9	1	0	D	Descriptive case series	Suspected cross-contamination with raw mince through piping bag, of chocolate, custard and cream cakes	Suspected chicken and/or eggs
	January	Commercial caterer	*Salmonella* Typhimurium 170 MLVA 3-9-8-12-523	68	14	0	AM	Cohort	Mayonnaise prepared with raw eggs	Egg based sauces and dressing
	January	National franchised fast food	*Salmonella* Typhimurium 170 MLVA 3-9-7-13-523	3	1	0	D	Descriptive case series	Suspected bacon and beef burgers	Suspected meat and meat containing dishes
	January	Primary produce	Histamine	2	1	0	M	N	Tinned anchovies imported from Morocco	Fish/seafood
	January	Private residence	*Salmonella* Typhimurium MLVA 3-15-16-14-523	4	1	0	D	Descriptive case series	Unknown	Unknown
	January	Restaurant	*Salmonella* Chester	14	2	0	M	Descriptive case series	Fresh chillies used to prepare chilli sauce	Salads and/or sandwiches
	January	Restaurant	*Salmonella* Typhimurium 170 MLVA 3-9-8-12-523	40	5	0	A	Cohort	Hollandaise sauce prepared with raw eggs	Egg based sauces and dressing
	January	Takeaway	*Salmonella* Typhimurium 170 MLVA 3-9-8-12-523	2	1	0	D	Cohort	Suspected chicken salad roll with homemade mayonnaise	Suspected salad and/or sandwiches

Appendix: Foodborne outbreak summary for OzFoodNet sites, Australia, 2009, *continued*

State or territory	Month of outbreak	Setting prepared	Agent responsible	Number affected	Hospitalised	Fatalities	Evidence	Epidemiological study	Responsible vehicles	Vehicle category
NSW, cont'd	February	Aged care facility	*Clostridium perfringens* enterotoxin A	25	0	0	M	Descriptive case series	Suspected vegetable gravy	Suspected gravy
	February	Commercial caterer	*Salmonella* Montevideo	10	2	0	D	Descriptive case series	Unknown	Unknown
	February	Restaurant	Unknown	5	0	0	D	Descriptive case series	Unknown	Unknown
	February	School	*Salmonella* Typhimurium 170 MLVA 3-9-8-12-523	37	0	0	D	Cohort	Unknown	Unknown
	February	Takeaway	*Salmonella* Typhimurium 170	3	1	0	M	Descriptive case series	Unknown	Unknown
	February	Takeaway	Unknown	6	6	0	D	N	Unknown - suspected gravy	Suspected gravy
	March	Aged care facility	*Salmonella* Typhimurium 170 MLVA 3-9-8-12-523	26	2	0	D	Descriptive case series	Unknown	Unknown
	March	Bakery	*Salmonella* Typhimurium 170 MLVA 3-9-7-13-523	8	1	0	D	Descriptive case series	Suspected chicken/pork rolls	Suspected meat and meat containing dishes
	March	Bakery	*Salmonella* Virchow	10	3	0	D	Cohort	Suspected bread rolls with pork filling (with homemade margarine/ butter with raw egg)	Egg based sauces and dressing
	March	Restaurant	*Campylobacter*	4	0	0	D	N	Suspected steak with chips and salad	Mixed dishes
	March	Restaurant	*Salmonella* Typhimurium 170 MLVA 3-9-7-12-523	33	9	0	AM	Cohort	Fried ice cream prepared with raw eggs	Egg containing desserts
	March	Restaurant	*Salmonella* Typhimurium 170 MLVA 3-9-7-13-523	2	1	0	D	Descriptive case series	Unknown - Fijian chicken suspected	Suspected chicken and/ or eggs
	March	Restaurant	*Salmonella* Virchow	3	1	0	D	N	Unknown	Unknown
	March	Restaurant	Unknown	10	0	0	D	Descriptive case series	Unknown	Unknown
	April	Aged care facility	*Clostridium perfringens*	16	2	2	D	Cohort	Unknown	Unknown
	April	Other	Unknown	7	0	0	D	Descriptive case series	Unknown	Unknown
	April	Restaurant	Norovirus	16	0	0	D	Cohort	Unknown	Unknown

Appendix: Foodborne outbreak summary for OzFoodNet sites, Australia, 2009, *continued*

State or territory	Month of outbreak	Setting prepared	Agent responsible	Number affected	Hospitalised	Fatalities	Evidence	Epidemiological study	Responsible vehicles	Vehicle category
NSW cont'd	April	Restaurant	Unknown	5	0	0	D	Case control study	Suspected lasagne, chicken Caesar salad	Mixed dishes
	May	Other	Unknown	15	0	0	D	Case control study	Unknown	Unknown
	May	Restaurant	Unknown	3	1	0	M	Descriptive case series	Unknown	Unknown
	May	Takeaway	Unknown	4	1	0	D	Descriptive case series	Unknown - sandwiches suspected	Suspected salad and/or sandwiches
	June	Commercial caterer	Unknown	23	0	0	D	Cohort	Unknown	Unknown
	June	Restaurant	Unknown	15	2	0	D	Descriptive case series	Unknown	Unknown
	July	Restaurant	Unknown	2	0	0	D	N	Unknown	Unknown
	July	Restaurant	Unknown	6	0	0	D	Descriptive case series	Unknown - sandwiches suspected	Suspected salad and/or sandwiches
	August	Commercial caterer	Norovirus	33	0	0	D	Cohort	Unknown	Unknown
	August	Private residence	Unknown	8	0	0	D	Descriptive case series	Unknown	Unknown
	August	Restaurant	Norovirus	27	0	0	D	Descriptive case series	Unknown	Unknown
	August	Restaurant	Norovirus	31	0	0	D	Cohort	Unknown	Unknown
	August	Restaurant	Unknown	2	0	0	D	N	Unknown	Unknown
	August	Restaurant	Unknown	28	2	0	D	Descriptive case series	Unknown	Unknown
	September	Aged care facility	*Salmonella* Typhimurium 170 MLVA 3-9-8-13-523	9	1	0	D	N	Unknown	Unknown
	September	Camp	Unknown	31	0	0	D	Descriptive case series	Unknown	Unknown
	September	Commercial caterer	Norovirus	Unknown	0	0	M	Descriptive case series	Unknown	Unknown
	September	Commercial caterer	Unknown	12	0	0	D	Descriptive case series	Unknown	Unknown
	September	Other	Norovirus	22	0	0	D	Cohort	Unknown	Unknown
	September	Restaurant	Norovirus	13	0	0	D	Cohort	Suspected salad items	Suspected salad and/or sandwiches

Appendix: Foodborne outbreak summary for OzFoodNet sites, Australia, 2009, *continued*

State or territory	Month of outbreak	Setting prepared	Agent responsible	Number affected	Hospitalised	Fatalities	Evidence	Epidemiological study	Responsible vehicles	Vehicle category
NSW, cont'd	September	Restaurant	Unknown	3	0	0	D	Cohort	Unknown	Unknown
	September	Restaurant	Unknown	10	0	0	D	Descriptive case series	Unknown	Unknown
	September	Unknown	*Salmonella* Heidelberg	7	1	0	D	Descriptive case series	Unknown	Unknown
	September	Unknown	Unknown	8	0	0	D	Cohort	Unknown	Unknown
	October	Commercial caterer	*Salmonella* Typhimurium 170 MLVA 3-9-8-13-532	4	0	0	M	Descriptive case series	Layered chocolate cake, prepared with cream and ganache icing (no raw eggs used)	Desserts
	October	Fair/festival/ mobile service	Unknown	3	1	0	D	Descriptive case series	Suspected prawns and calamari	Suspected seafood
	October	Other	Norovirus	20	2	0	D	N	Unknown	Unknown
	October	Other	Unknown	24	0	0	D	Descriptive case series	Unknown	Unknown
	October	Other	Unknown	24	0	0	D	N	Unknown	Unknown
	October	Private residence	Unknown	8	0	0	D	Cohort	Unknown	Unknown
	October	Restaurant	Unknown	4	0	0	D	Descriptive case series	Suspected salad items	Suspected salad and/or sandwiches
	October	Restaurant	Unknown	4	0	0	D	Descriptive case series	Unknown	Unknown
	October	Restaurant	Unknown	7	0	0	D	Descriptive case series	Unknown	Unknown
	November	Commercial caterer	Unknown	28	0	0	D	Cohort	Unknown	Unknown
	November	Private residence	*Salmonella* Typhimurium MLVA 3-13-9-11-550	6	2	0	D	Descriptive case series	Suspected tiramisu prepared with raw eggs	Suspected egg containing dessert
	November	Restaurant	*Salmonella* Typhimurium MLVA 3-12-12-13-523	3	0	0	M	Descriptive case series	Cooked pork mince and leftover food (mix of tofu, rice, duck)	Mixed dishes
	November	Restaurant	Unknown	7	0	0	D	Cohort	Unknown	Unknown
	November	Takeaway	*Salmonella* Stanley MLVA 2-15 (14)-0-0-496	32	7	0	D	Descriptive case series	Suspected salads, wraps, burgers	Suspected salad and/or sandwiches
	December	Other	Unknown	12	0	0	D	Cohort	Unknown	Unknown

Appendix: Foodborne outbreak summary for OzFoodNet sites, Australia, 2009, *continued*

State or territory	Month of outbreak	Setting prepared	Agent responsible	Number affected	Hospitalised	Fatalities	Evidence	Epidemiological study	Responsible vehicles	Vehicle category
NSW, cont'd	December	Restaurant	*Salmonella* Singapore	3	0	0	M	Descriptive case series	Fried ice cream prepared with raw eggs	Egg containing desserts
	December	Takeaway	Norovirus	30	0	0	D	Descriptive case series	Unknown	Unknown
NT	March	Private residence	*Salmonella* Typhimurium u302	2	0	0	D	Descriptive case series	Suspect tiramisu	Egg containing desserts
	November	Commercial caterer	Norovirus	Unknown	0	0	D	Descriptive case series	Unknown	Unknown
	December	Restaurant	*Escherichia coli*	Unknown	0	0	D	Descriptive case series	Unknown	Unknown
Qld	January	Aged care facility	*Salmonella* Typhimurium pt 135a	3	0	0	D	Descriptive case series	Unknown	Unknown
	January	Aged care facility	*Salmonella* Typhimurium pt 44	20	4	0	AM	Descriptive case series	Unknown	Unknown
	January	Private residence	Norovirus	10	1	0	D	Descriptive case series	Unknown	Unknown
	February	Aged care facility	*Salmonella* Typhimurium pt 170	3	Unknown	0	D	Descriptive case series	Unknown	Unknown
	February	Commercial caterer	Norovirus	20	1	0	A	Cohort	Unknown	Mixed dishes
	February	Primary produce	Ciguatera fish poisoning	3	2	0	D	Descriptive case series	Spanish Mackerel	Fish/seafood
	February	Restaurant	Unknown	6	0	0	D	Descriptive case series	Unknown	Unknown
	April	Restaurant	*Salmonella* Typhimurium pt 170	3	0	0	D	Descriptive case series	Unknown	Unknown
	May	Primary produce	Histamine	6	0	0	M	Descriptive case series	Tuna	Fish/seafood
	May	Restaurant	Norovirus	17	1	0	D	Cohort	Unknown	Unknown
	May	Takeaway	Unknown	2	0	0	D	Descriptive case series	Prawn Roll	Fish/seafood
	June	Restaurant	*Salmonella* Typhimurium pt 141	7	0	0	D	Descriptive case series	Unknown	Mixed dishes
	July	Bakery	Norovirus	24	Unknown	0	D	Descriptive case series	Sandwiches (various fillings)	Salads and/or sandwiches
	July	Restaurant	Unknown	2	0	0	D	Descriptive case series	Unknown	Unknown

Appendix: Foodborne outbreak summary for OzFoodNet sites, Australia, 2009, *continued*

State or territory	Month of outbreak	Setting prepared	Agent responsible	Number affected	Hospitalised	Fatalities	Evidence	Epidemiological study	Responsible vehicles	Vehicle category
Qld cont'd	August	Primary produce	Ciguatera fish poisoning	2	2	0	D	Descriptive case series	King snapper/jobfish green	Fish/seafood
	August	Restaurant	*Clostridium perfringens*	4	0	0	D	Descriptive case series	Unknown - suspected roast beef, vegetables and gravy	Meat and meat containing dishes
	September	Restaurant	Unknown	4	0	0	D	Descriptive case series	Unknown	Unknown
	October	Restaurant	Norovirus	3	0	0	D	Descriptive case series	Unknown	Unknown
	October	Restaurant	Norovirus	23	0	0	A	Cohort	Chicken Caesar salad; roast chicken	Chicken and chicken containing dishes
	December	Restaurant	*Clostridium perfringens*	2	0	0	D	Descriptive case series	Unknown	Mixed dishes
SA	January	Unknown	*Salmonella* Typhimurium 9	20	5	0	D	Descriptive case series	Unknown	Unknown
	February	Aged care facility	Unknown	3	Unknown	Unknown	D	Descriptive case series	Unknown	Unknown
	February	Unknown	STEC	6	3	0	D	Descriptive case series	Unknown	Unknown
	March	Unknown	*Salmonella* Typhimurium 108	11	6	0	D	Descriptive case series	Unknown	Unknown
	April	Restaurant	*Salmonella* Typhimurium 44	30	0	0	A	Cohort	Garlic aioli	Egg based sauces and dressing
	May	Restaurant	*Salmonella* Typhimurium 135	7	0	0	D	Descriptive case series	Fried ice cream	Egg containing desserts
	July	Unknown	*Salmonella* Anatum	5	2	0	D	Descriptive case series	Unknown	Unknown
	August	Commercial caterer	Norovirus	22	0	0	D	Descriptive case series	Sandwiches and baguettes	Salads and/or sandwiches
	August	Unknown	*Salmonella* Typhimurium u302	7	2	0	D	Descriptive case series	Unknown	Unknown
	November	Camp	STEC	31	5	0	A	Cohort	Potato salad and pasta salad	Mixed dishes
	November	Restaurant	Norovirus	21	0	0	A	Cohort	Berry cheesecake	Desserts
	November	Unknown	*Salmonella* Virchow 8	4	9	0	D	Descriptive case series	Unknown	Unknown

Appendix: Foodborne outbreak summary for OzFoodNet sites, Australia, 2009, *continued*

State or territory	Month of outbreak	Setting prepared	Agent responsible	Number affected	Hospitalised	Fatalities	Evidence	Epidemiological study	Responsible vehicles	Vehicle category
SA. Cont'd	December	Private residence	*Salmonella* Typhimurium 44	16	2	0	A	Cohort	Tiramisu	Egg containing desserts
	December	Unknown	*Salmonella* Typhimurium 44	7	2	0	D	Descriptive case series	Unknown	Unknown
Tas	September	Restaurant	*Campylobacter*	35	0	0	A	Cohort	chicken liver parfait	Chicken and chicken containing dishes
	September	Restaurant	Unknown	9	0	0	A	Cohort	chicken liver parfait	Chicken and chicken containing dishes
	November	Commercial caterer	Norovirus	14	0	0	A	Cohort	green salad suspected	Suspected salad and/or sandwiches
Vic	February	Commercial caterer	*Salmonella* Typhimurium 170	4	1	0	D	Descriptive case series	Unknown	Unknown
	February	Restaurant	Not further specified	10	0	0	D	Descriptive case series	Suspected stews and casseroles	Meat and meat containing dishes
	February	Restaurant	*Salmonella* Typhimurium 197	2	2	0	D	Descriptive case series	Unknown	Unknown
	March	Aged care facility	Clostridium perfringens	22	0	0	D	Descriptive case series	Vitamised meals	Suspected vitamised foods
	March	Camp	Unknown	13	0	0	D	Cohort	Unknown	Unknown
	March	Child care	*Salmonella* Typhimurium 170	18	1	0	D	Descriptive case series	Unknown	Unknown
	March	Private residence	*Salmonella* Typhimurium 44	7	1	0	D	Descriptive case series	Unknown	Unknown
	April	Aged care facility	*Salmonella* Typhimurium 170	12	0	0	D	Descriptive case series	Unknown	Unknown
	April	Restaurant	Not further specified	6	0	0	D	Descriptive case series	Unknown	Unknown
	May	Aged care facility	Norovirus	17	1	0	D	Descriptive case series	Unknown	Unknown
	May	Aged care facility	Not further specified	7	0	0	D	Descriptive case series	Unknown	Unknown
	July	Aged care facility	Unknown	4	0	0	D	Descriptive case series	Unknown	Unknown
	August	Restaurant	Norovirus	87	0	0	D	Descriptive case series	Unknown	Unknown
	August	Restaurant	Unknown	3	0	0	D	Descriptive case series	Unknown	Unknown

Appendix: Foodborne outbreak summary for OzFoodNet sites, Australia, 2009, *continued*

State or territory	Month of outbreak	Setting prepared	Agent responsible	Number affected	Hospitalised	Fatalities	Evidence	Epidemiological study	Responsible vehicles	Vehicle category
Vic, cont'd	September	Aged care facility	Unknown	7	0	0	D	Descriptive case series	Unknown	Unknown
	September	Restaurant	Norovirus	10	0	0	D	Cohort	Unknown	Unknown
	October	Commercial caterer	Not further specified	41	0	0	D	Cohort	Unknown	Unknown
	November	Aged care facility	Not further specified	6	0	0	D	Descriptive case series	Unknown	Meat and meat containing dishes
	November	Aged care facility	*Salmonella* Typhimurium 170	20	2	1	D	Descriptive case series	Unknown	Unknown
	November	Aged care facility	*Salmonella* Typhimurium 170	22	5	1	D	Descriptive case series	Unknown	Unknown
	November	Other	*Clostridium perfringens*	4	0	0	D	Descriptive case series	Unknown	Unknown
	November	Primary produce	Fish wax ester	27	0	0	D	Descriptive case series	Escolar	Fish/seafood
	November	Private residence	*Salmonella* Typhimurium 3	6	3	0	D	Descriptive case series	suspect eggs	Suspected chicken and/ or eggs
	November	Restaurant	Norovirus	165	(blank)	0	D	Descriptive case series	Unknown	Unknown
	November	Restaurant	Not further specified	17	0	0	D	Descriptive case series	Unknown	Unknown
	December	Military	*Campylobacter*	5	Unknown	0	D	Descriptive case series	Unknown	Unknown
	December	Military	Norovirus	18	1	0	D	Descriptive case series	Unknown	Unknown
	December	Restaurant	Hepatitis A	5	1	0	D	Descriptive case series	Food contaminated with HAV either from an infected food handler or SDT	Unknown
	December	Restaurant	Hepatitis A	9	2	0	D	Descriptive case series	Food contaminated with HAV either from an infected food handler or SDT	Semi dried tomatoes
WA	February	Aged care facility	Unknown	16	0	0	D	Descriptive case series	Unknown	Unknown
	February	Restaurant	*Salmonella* Saintpaul	7	1	0	M	Descriptive case series	Fried ice cream	Egg containing desserts
	March	Restaurant	*Salmonella* Singapore	6	0	0	D	Descriptive case series	Unknown - chicken suspected	Suspected chicken and/ or eggs

Appendix: Foodborne outbreak summary for OzFoodNet sites, Australia, 2009, *continued*

State or territory	Month of outbreak	Setting prepared	Agent responsible	Number affected	Hospitalised	Fatalities	Evidence	Epidemiological study	Responsible vehicles	Vehicle category
WA, cont'd	May	Community	Hepatitis A	5	1	0	M	Descriptive case series	Berries	Fruit
	May	Restaurant	*Salmonella* Typhimurium	8	2	0	D	Descriptive case series	Unknown	Unknown
	May	Restaurant	*Salmonella* Typhimurium 6	5	0	0	D	Descriptive case series	Unknown	Unknown
	June	Restaurant	*Salmonella* Typhimurium 135a	7	1	0	D	Descriptive case series	Unknown	Unknown
	July	Takeaway	*Salmonella* Typhimurium 193 var 1	31	9	0	M	Descriptive case series	Vietnamese Pork Roll	Mixed dishes
	August	Primary produce	*Salmonella* Saintpaul	17	3	0	M	Descriptive case series	Pawpaw	Fruit
	September	Commercial caterer	Norovirus	15	0	0	A	case control study	Rice paper rolls	Mixed dishes
	October	Restaurant	*Listeria*	2	2	0	DM	Descriptive case series	Unknown	Unknown
	October	Restaurant	*Salmonella* Typhimurium 170	39	7	0	A	case control study	Scrambled eggs	Eggs
	October	Takeaway	*Salmonella* Typhimurium 170	39	6	0	D	Descriptive case series	Raw egg mayonnaise	Egg based sauces and dressing
	November	Commercial caterer	Norovirus	8	0	0	D	Cohort	Unknown	Unknown
	December	Camp	Norovirus	14	0	0	D	Cohort	Unknown	Unknown
	December	Restaurant	Norovirus	11	0	0	D	Case control study	Unknown	Unknown
	December	Restaurant	Norovirus	18	0	0	D	Case control study	Unknown	Unknown
	December	Restaurant	Norovirus	22	0	0	D	Cohort	Unknown	Unknown

Evidence

D Descriptive evidence implicating the vehicle

A Analytical epidemiological association between illness and vehicle

M Microbiological confirmation of aetiology in vehicle and cases.

Epidemiological study

N Individual patient data not collected.

Australian Rotavirus Surveillance Program: Annual report, 2009/2010

Carl D Kirkwood, Karen Boniface, Ruth F Bishop, Graeme L Barnes

Abstract

The Australian Rotavirus Surveillance Program together with 15 collaborating laboratories Australia-wide conducts a laboratory based rotavirus surveillance program. This report describes the genotypes of rotavirus strains responsible for the hospitalisation of children with acute gastroenteritis during the period 1 July 2009 to 30 June 2010, the 3rd year of surveillance following introduction of rotavirus vaccines into the National Immunisation Program. Seven hundred and seventy-eight faecal samples were referred to the centre for G and P genotype analysis using hemi-nested multiplex reverse transcription-polymerase chain reaction. Of the 422 confirmed as rotavirus positive, genotype G1P[8] was the dominant type nationally, representing 49.3%, followed by genotype G2P[4] (21.1%). Genotypes G3P[8], G4P[8] and G9P[8] each represented less than 3% of circulating strains nationally. The dominance of G1P[8] was in part associated with a large outbreak of severe gastroenteritis in the Northern Territory in 2010. The identification of uncommon rotavirus genotype combinations has increased since vaccine introduction, with G1P[4], G2P[8] and G9P[4] identified during this survey. Single strains of G1P[6] and G4P[6] were identified during this study period. This survey continues to highlight the fluctuations in rotavirus genotypes, and results from this survey suggest there is limited genotype selection based on vaccine usage. However, the large G1P[8] outbreak of gastroenteritis in the Northern Territory may have resulted from vaccine pressure on wild-type strains. *Commun Dis Intell* 2010;34(4):427–434.

Keywords: Rotavirus, gastroenteritis, genotypes, disease surveillance

Introduction

Rotaviruses are the most important cause of dehydration, hospitalisation and death due to severe gastroenteritis in young children worldwide[1] Two live oral rotavirus vaccines have been developed (Rotarix® [GlaxoSmithKline] and RotaTeq® [Merck]) in an effort to decrease the large disease burden. Both vaccines were shown to be safe and highly effective in prevention of severe diarrhoea and hospitalisation due to rotavirus infections during large phase III clinical and efficacy trials, each involving over 60,000 children worldwide.[2,3]

Rotavirus vaccines have been commercially available in Australia from 2006 and were introduced into the Australian National Immunisation Program (NIP) for all infants from 1 July 2007. Each state health department made independent decisions on which vaccine to use; Victoria, South Australia, and Queensland selected RotaTeq, while New South Wales, Western Australia (changed to RotaTeq from May 2009), the Northern Territory, Tasmania and the Australian Capital Territory selected Rotarix. Introduction of rotavirus vaccines to the NIP is aimed to decrease the large social and economic burden of rotavirus disease in Australia. In the pre-vaccine era diarrhoea accounted for up to 50% of childhood hospitalisations in Australia, which represents 10,000 children hospitalised each year.[4]

The Australian Rotavirus Surveillance Program has been reporting the changing annual pattern of dominant genotypes in the Australian population since 1999. Over this period, the results have highlighted the diversity of rotavirus strains capable of causing disease in children, and provided the baseline information of the changing pattern of circulating strains, prior to vaccine introduction.[5]

The introduction of vaccines into Australia will increase population immunity. This is likely to have an impact on circulating wild-type strains. However, exactly what will happen is difficult to predict as strain replacement and changes in the prevalence of common genotypes, as well as emergence of new or rare genotypes, are all possible. Thus continuing genotype surveillance should identify the effects that each vaccine program has on circulating wild-type strains.

This report describes the surveillance and genotype characterisation of rotavirus strains causing severe gastroenteritis in young children 5 years of age or younger in Australia for the period 1 July 2009 to 30 June 2010.

Methods

Rotavirus positive specimens detected by enzyme immunoassay (EIA) or latex agglutination in collaborating laboratories across Australia were collected, stored frozen and forwarded to the National Rotavirus Reference Centre (NRRC) Melbourne, together with relevant age and sex details. Viral

RNA was extracted from each specimen using an RNA extraction kit (Qiamp Viral mini extraction kit, Qiagen) according to the manufacturers instructions. Double stranded RNA was used to determine the G and P genotype of each specimen by hemi-nested multiplex reverse transcription-polymerase chain reaction (RT-PCR) assay, using G or P specific oligonucleotide primers.[6,7]

Results

Number of isolates

A total of 778 specimens were received for analysis from 15 collaborating centres in Victoria, Western Australia, the Northern Territory, New South Wales, Queensland, South Australia and Tasmania. Samples were not obtained from the Australian Capital Territory. Thus the sample collection is likely to be highly representative of all Australian children hospitalised with acute gastroenteritis. Four hundred and twenty-two specimens from Victoria (n=55), Western Australia (n=98), New South Wales (n=35), Queensland (n= 78), South Australia (n=18), Tasmania (n=1) and the Northern Territory (n=137), were confirmed as rotavirus positive using a combination of in-house EIA and RT-PCR analysis. The remaining 356 specimens contained either insufficient specimen for genotyping (n=154), or the specimen was not confirmed to be positive for rotavirus (n=202), and were not analysed further.

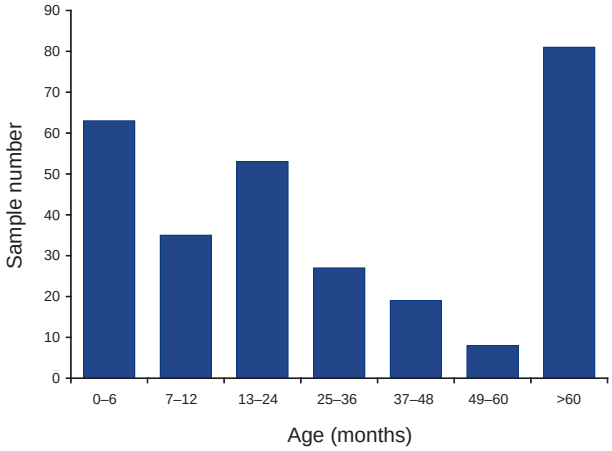
The Royal Children's Hospital in Melbourne was able to access approximately 55% of all faecal specimens identified as containing rotavirus antigen from children less than 5 years of age. However the collection rate at other centres was unknown.

Age distribution

The overall age distribution of children with acute rotavirus gastroenteritis is depicted in Figure 1. During the reporting period, 22% of cases were from infants 0–6 months of age, 12% were from infants 7–12 months of age, and 19% from patients 13–24 months of age. Overall, 71% of samples were from children aged 5 years or less. Eighty-one samples were obtained from individuals more than 5 years of age; 23 were collected from children 5–10 years of age, 11 were from individuals 10–20 years of age, 31 were from individuals 21–80 years of age, and 16 were from individuals aged 80–100 years.

During the study period, slightly more specimens from male than female children (n= 144 vs 141) were obtained for analysis.

Figure 1: Distribution of rotavirus samples, Australia, I July 2009 to 30 June 2010, by age group



Genotype distribution

The rotavirus genotypes identified in Australia from 1 July 2009 to 30 June 2010 are shown in Table 1.

G1P[8] strains were the most common genotype identified, representing 49.3% of all specimens analysed, and was identified in all collaborating centres except Tasmania. It was the dominant type in the Northern Territory and Queensland, and 2nd most common strain in Western Australia, New South Wales and Victoria. G2P[4] strains were the 2nd most common type nationally, representing 21.1% of all specimens, and was the dominant type in South Australia and Western Australia. It represented 27% of samples in New South Wales, but less than 2% in Victoria, Queensland and the Northern Territory. G3P[8] or G3Pnt strains were the dominant type in Victoria representing 60% of strains. G3P[8] were identified in the three eastern states (Victoria, New South Wales and Queensland) and Western Australia, overall however, they represented only 6.6% of strains nationally. Five G9P[8] strains, one each from Sydney, Darwin, Melbourne and Perth, comprised 1.2% of samples analysed. A single G4P[8] strain was identified in Darwin, a single G4P[6] was identified in Tasmania, and a single G1P[6] was identified in Sydney.

Fourteen strains were found to possess uncommon genotype combinations of VP4 and VP7; 5 G1P[4] strains were identified in Queensland, Perth and Darwin and 4 G2P[8] strains were identified in Newcastle, Darwin and Melbourne. Three G9P[4] strains were identified in Sydney and Alice Springs. Single G8 strains were identified in Darwin and Perth. Eleven (2.6%) rotavirus samples contained multiple types.

Table 1: . Rotavirus G and P genotypes in Australia, 1 July 2009 to 30 June 2010

Centre	Type Total	G1P[8] %	G1P[8] n	G2P[4] %	G2P[4] n	G3P[8] %	G3P[8] n	G4P[8] %	G4P[8] n	G9P[8] %	G9P[8] n	Mix* %	Mix* n	Other† %	Other† n	Non-type %	Non-type n
New South Wales																	
Sydney (POW)	7	28.6	2	28.6	2	28.6	2	–	0	14.2	1	–	0	–	0	–	0
Sydney (Westmead)	25	24.0	6	20.0	5	4.0	1	–	0	4.0	1	–	0	8.0	2	40.0	10
Newcastle	3	33.3	1	33.3	1	–	0	–	0	–	0	–	0	33.3	1	–	0
Northern Territory																	
Alice Springs	56	85.7	48	3.6	2	–	0	–	0	–	0	1.8	1	3.5	2	5.4	3
Darwin	33	72.7	24	3.0	1	–	0	–	0	–	0	–	0	3.0	1	21.3	7
Western Diagnostic (NT)	48	77.1	37	–	0	–	0	2.1	1	2.1	1	4.2	2	6.2	3	8.3	4
Queensland																	
	78	84.6	66	1.3	1	7.6	6	–	0	–	0	1.3	1	1.3	1	3.9	3
South Australia																	
Adelaide	18	5.5	1	83.5	15	–	0	–	0	–	0	5.5	1	–	0	5.5	1
Tasmania																	
Hobart	1	–	0	–	0	–	0	–	0	–	0	–	0	100.0	1	–	0
Victoria																	
Melbourne	55	14.6	8	1.8	1	29.0	16	–	0	1.8	1	3.6	2	3.6	2	45.6	25
Western Australia																	
PathWest WA	91	15.4	14	61.5	56	3.3	3	–	0	1.1	1	4.4	4	3.3	3	11.0	10
Perth	7	14.3	1	71.4	5	–	0	–	0	–	0	–	0	–	0	14.3	1
Total	**422**	**49.3**	**208**	**21.1**	**89**	**6.6**	**28**	**0.2**	**1**	**1.2**	**5**	**2.6**	**11**	**3.8**	**16**	**15.2**	**64**

* Mix

Alice Springs	G1P[4]/P[8]
Western Diagnostic (NT)	2xG1P[4]/P[8]
Queensland	G1P[4]/P[8]
Melbourne	G2P[4]/[8], G3/G2P[non-typeable]
PathWest WA	G8P[4]/P[8], 2x G1/G2P[8], G1/G4P[8]
Adelaide	G9/G2P[4]

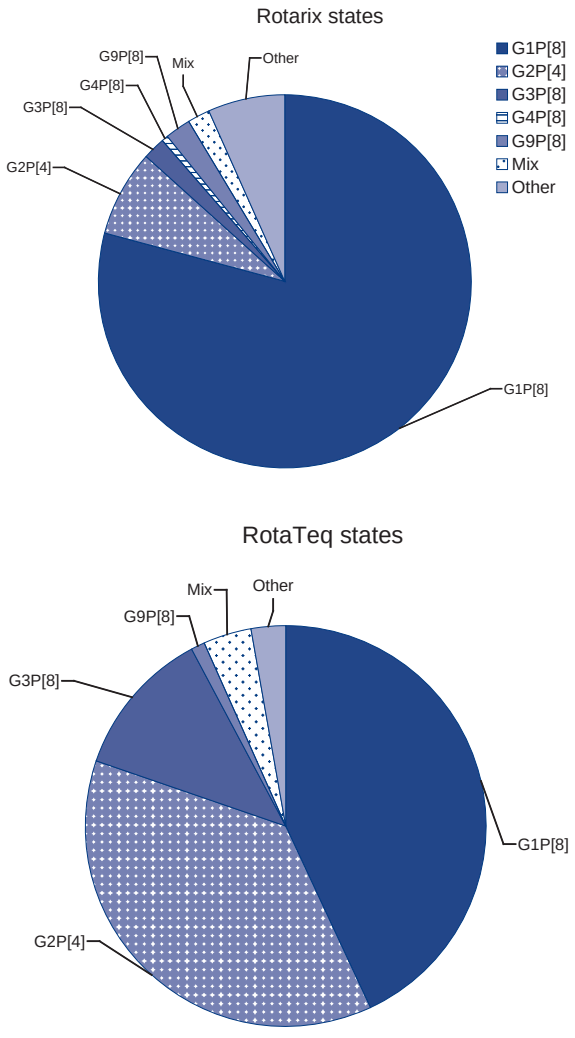
† Other

Westmead	G1P[6], G9P[4]
Newcastle	G2P[8]
Alice Springs	2x G9P[4]
Darwin	G2P[8]
Western Diagnostic	3x G1P[4]
Queensland	G1P[4]
PathWest WA	G1P[4], G8P[4]/P[8], G8 P[non-typeable]
Melbourne	2x G2P[8]

In 15.2% of samples either a G– or P-Type, or both, could not be assigned (Table 2). These are likely to be samples with virus numbers below the detection limits of our typing assays, or could have contained inhibitors in extracted RNA to prevent the function of the enzymes used in RT and/or PCR steps. Future studies will include further characterisation of the genes encoding the outer capsid proteins of these strains.

The distribution of G and P genotypes in states using Rotarix (New South Wales, the Northern Territory and Tasmania) compared with distribution in states using RotaTeq (Victoria, Queensland, South Australia and Western Australia) is shown in Figure 2.

Figure 2: Overall distribution of rotavirus G and P genotypes identified in Australian children based on vaccine usage for period 1 July 2009 to 30 June 2010



Rotarix was used in New South Wales, Tasmania, and the Northern Territory.

RotaTeq was used in Victoria, South Australia, Western Australia and Queensland

The number of samples analysed was fairly consistent, with 149 strains from Rotarix sites and 209 from RotaTeq sites. However, 83% of samples in the Rotarix sites were from the Northern Territory, whereas strains from Victoria, Queensland and Western Australia represented 14%, 36% and 42% of the total number of samples analysed from states using RotaTeq.

Analysis of fully G and P typeable samples revealed that in Rotarix states G1P[8] strains dominate (79.2%), with G2P[4] strains comprising 7.4% of specimens. In RotaTeq states, G1P[8] was also dominant, identified in 43% of strains, G2P[4] comprised 37.3% of strains and G3P[8] represented 12% of strains. A slight increase in G3P[8] (12% vs 2%) and mixed strains (3.8% vs 2%) was observed in the states that introduced RotaTeq when compared with those using Rotarix. Conversely, a slight increase in uncommon strains was observed in Rotarix states versus RotaTeq states (6.7% vs 2.9%).

Faecal specimens were received from 24 children who developed rotavirus gastroenteritis after being vaccinated with RotaTeq. Vaccine virus was identified in seven of these cases by RT-PCR and sequence analysis. In addition, Rotarix vaccine was identified in a faecal specimen received from 1 child who developed rotavirus gastroenteritis after being vaccinated with Rotarix.

Discussion

This report from the Australian Rotavirus Surveillance Program, covering the period 1 July 2009 to 31 June 2010, describes the annual epidemics and geographic distribution of rotavirus genotypes causing disease in Australian children. The surveillance program has showed that genotype G1P[8] re-emerged as the dominant genotype nationally, representing 49.3% of all strains. In part, this corresponded with a large outbreak of acute gastroenteritis in the Northern Territory during May to June 2010, as well as its emergence as the dominant type in Queensland and New South Wales. Genotype G2P[4] was the 2nd predominant type nationally, comprising 21.1% of all strains characterised. It was the dominant type in South Australia and Western Australia. Victoria was the only location where genotype G3 (G3P[8] or G3Pnt) was the dominant type, and continues to highlight its important role in acute gastroenteritis observed during past 2 surveys.[8,9,10] This survey highlights the ongoing fluctuations in the dominant genotypes, and reveals the return of G1P[8] as the dominant genotype nationally, similar to that observed in 2006–07 and 2007–08.

Similar to other reports,[5,8,9] multiple common genotypes (G1P[8], G2P[4], G3P[8], G4P[8]

Table 2: G and P genotype assignments in non-typeable specimens

Centre	Total	P non-typeable						G non-typeable			G & P non-typeable
		G1	G2	G3	G4	G8	G9	P[4]	P[6]	P[8]	NT
New South Wales											
Sydney (POW)	0	–	–	–	–	–	–	–	–	–	–
Sydney (Westmead)	10	–	–	–	–	–	–	8	–	1	1
Newcastle	0	–	–	–	–	–	–	–	–	–	–
Northern Territory											
Alice Springs	3	–	1	–	–	–	1	–	1	–	–
Darwin	7	–	1	–	–	1	1	3	–	1	–
Western Diagnostic (NT)	4	4	–	–	–	–	–	–	–	–	–
Queensland											
	3	3	–	–	–	–	–	–	–	–	–
South Australia											
Adelaide	1	–	–	–	–	–	1	–	–	–	–
Tasmania											
Hobart	0	–	–	–	–	–	–	–	–	–	–
Victoria											
Melbourne	25	2	1	19	1	–	1	–	–	–	1
Western Australia											
PathWest WA	10	2	–	–	–	1	2	–	–	1	4
Perth	1	1	–	–	–	–	–	–	–	–	–
Total (%)	**64**	**12 (18.8%)**	**3 (4.7%)**	**19 (29.7%)**	**1 (1.5%)**	**2 (3.1%)**	**6 (9.4%)**	**11 (17.2%)**	**1 (1.5%)**	**3 (4.7%)**	**6 (9.4%)**

and G9P[8]) continue to co-circulate within the Australian population causing significant disease with G1 and G2 identified in six states and territories, and G3 being identified in four states. In contrast, G4 and G9 strains each represented minor circulating strains.

The parallel usage of both vaccines in Australia provides a unique opportunity to compare the effect of each vaccine on the circulating wildtype strains. During the first 2 years post-vaccine introduction, differences have been observed in genotype distribution when vaccine usage was compared.[8,9,10] As previously reported the emergence of G2P[4] strains were more commonly identified in locations using Rotarix vaccine, while G3P[8] strains were more common in locations using RotaTeq.[10,11,12] In the current survey, the two locations where G2P[4] strains were dominant both used RotaTeq vaccine in their vaccine program. The lack of association with Rotarix vaccine seen during this survey period was supported by the emergence of G2P[4] reported in vaccinated populations in Nicaragua (RotaTeq) and to a lesser extent in non-vaccinated populations in Europe.[13,14] The association of G3P[8] and RotaTeq observed previously in our study, was not confirmed here. G3P[8] was only observed in one of the three states using RotaTeq vaccine, in the other states either G1P[8] or G2P[4] were the dominant strain. Therefore for both Rotarix and RotaTeq no clear association of vaccine driven genotype selection was observed during the 3rd year post-vaccine introduction.

The 2009–10 reporting period was also characterised by a large outbreak of acute diarrhoea in the Northern Territory between May and June 2010, caused by a G1P[8] virus. Unlike the recent G9P[8] and G2P[4] outbreaks in the Northern Territory, the G1P[8] represents an identical genotype as the Rotarix vaccine. It is unknown whether this strain emerged due to a lack of protection by the vaccine or by natural variation, since not all the subjects from whom samples were obtained were eligible for vaccination and the vaccination status of vaccine-eligible infants is unknown. However, the emergence of G1P[8] maybe an example of vaccine pressure, such that it resulted from genetic drift such that a divergent G1P[8] lineage was selected.

The rapid emergence and global spread of G9 and G12 strains in less than a decade illustrates the potential with which rotavirus can evolve.[15] Thus uncommon rotavirus types continue to be of worldwide interest because of the possible impact they could have on future rotavirus vaccine programs. This year several uncommon VP7/VP4 genotype combinations were again identified; including G1P[4], G2P[8], and G9P[4], several of which have existed in Australia for 2–3 years, albeit in low numbers. Similar to past surveys[8,9] G8 strains continue to be identified circulating in low levels in Australian children. The identification of uncommon G and P genotype combinations has increased in Australia since vaccine introduction, and may suggest that the wild-type strains are under an increased state of flux. Vaccine pressure may cause more mixing of strains with greater selection pressure occurring to identify 'fitter' strains, or natural variants that will replicate in a setting with greater immune pressure.

In Australia, where rotavirus mortality was rare prior to vaccine introduction, the decision to implement infant rotavirus vaccination was based upon the morbidity caused by rotavirus and the predicted cost-effectiveness of vaccination.[16] Recently the impact of rotavirus vaccination has shown not only reductions in rotavirus positive tests and hospital encounters, but also reductions in non-rotavirus coded episodes of gastroenteritis.[17,18] Importantly reductions in childhood gastroenteritis have been observed at all hospital levels. This impact has also been reported in others setting including the United States of America and Belgium.[19,20] These early data provide reassurance that vaccination has impacted directly and possibly indirectly upon gastroenteritis morbidity. However, in the Northern Territory vaccine effectiveness was not as high as seen elsewhere in Australia, where protection was evident for young infants with severe disease, but not for all cases resulting in hospitalisation.[21] It is possible that a waning of vaccine-induced immunity or increasing immunity from natural infection might account for the apparent decline in vaccine effectivenesss amongst older infants in this setting.

The previous report (2008/09),[9] which represented the 2nd year of vaccine usage, showed a change in age distribution of children admitted to hospital. This observed increase in infants in the 0–6 month age group was also observed in the current survey where the highest proportion of children admitted to hospital was in the 0–6 month age group. Thus since the 2007/08 survey, the proportion of hospitalisation in the 0–6 month age group has increased from 14% to 27% to 30.7%. Interestingly, 71% of the infants in this age group were less than 3 months of age, an age group too young to receive complete vaccination. The proportion of children in the 7–12 month age group remained similar since the vaccine was introduced, but importantly lower than pre-vaccine rates.

This survey has further highlighted the continued fluctuations in rotavirus genotypes across Australia, and tends to support the notion that there is limited genotype selection based on vaccine usage. However, the rapidly changing genotype patterns do illustrate a more dynamic wild-type population. The recent report that estimated that a single novel rotavirus

strain could emerge and spread worldwide in less than a decade re-enforces the need for thorough and continued rotavirus surveillance. Thus the ongoing evolution of the wild-type strains circulating in Australia, under constant vaccine pressure will require close monitoring to identify any changes that may impact on vaccine effectiveness.

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Peer-reviewed articles

Incomplete protection against hepatitis B among remote Aboriginal adolescents despite full vaccination in infancy

Elizabeth Dent, Christine E Selvey, Andrew Bell, Joshua Davis, Malcolm I McDonald

Abstract

The objective of this study was to determine long-term immunity to hepatitis B virus (HBV) in a cohort of adolescents who received plasma-derived HBV vaccine in 1989 and 1990 in a remote Australian Aboriginal community. This was done using a serological survey; primary outcome measures were cut-off titres of HBsAb, and the presence of HBcAb and/or HBsAg. Of 37 adolescents in the cohort, 4 (11%) had evidence of active infection, one with abnormal liver enzymes, 7 (19%) had evidence of past infection, 15 (41%) were HBsAb positive in low titre and 11 (30%) were classed as immune. It was concluded that there was relatively poor long-term serological immunity to HBV vaccination in this group; a finding which is in keeping with similar studies in Indigenous and remote populations elsewhere. This finding raises the concern that a significant proportion of Aboriginal adolescents in other remote communities (vaccinated in 1989 and 1990) were not adequately protected by the vaccine. If so, there will be an unexpected burden of chronic HBV infection in these settings and a substantial group who are non-immune, despite having received complete HBV vaccination courses as infants. The authors recommend follow-up serosurveys in remote Aboriginal communities to identify people with low HBsAb titres, especially those without an adequate anamnestic response to another dose of HBV vaccine. In addition, community-based active surveillance programs will be required to detect people with chronic HBV infection and provide access to monitoring and appropriate treatment. *Commun Dis Intell* 2010;34(4):435–439.

Keywords: Indigenous, Australia, immunity, hepatitis, HBV

Introduction

The hepatitis B virus (HBV) vaccine was widely accepted and distributed soon after it became available in the early 1980s.[1] Immunisation prevents HBV chronic liver disease and has dramatically reduced the incidence of hepatocellular carcinoma in vaccinated populations.[1] HBV vaccination was introduced into the Northern Territory Childhood Immunisation Schedule at birth, 1 month and 6 months of age for Aboriginal neonates in April 1988. Several years later, it became universal across Australia.

Recent studies have suggested that immune responses to the early HBV vaccines may have been suboptimal in some Aboriginal communities.[2–5] In addition to factors related to the vaccination process, investigators have suggested that genetic, developmental, and environmental factors may contribute to a poor response.[2–4]

Studies in Mongolia and Indonesia have shown that improper storage and interrupted vaccine transport to remote settings can lead to freezing; this structurally destabilises the vaccine and reduces efficacy.[6,7] A study in the Northern Territory, Australia, in 1994 documented freezing temperatures in 47.5% of vaccines, either in transfer or during storage.[8] In rural China it was thought that similar transport factors could play a part, however it was found that genetic factors played a larger role, with a specific HLA haplotype predicting poor vaccine responses among the Han Chinese.[9]

Two randomised controlled trials have reported long term follow-up of high risk populations wherein a small percentage of vaccinated persons with initial seroprotection later developed HBcAb, indicating that some individuals either did not remain, or were never protected by the vaccine.[10,11] Importantly, none of the subjects who developed HBcAb developed clinical hepatitis and the vaccine appears to have provided protection against chronic HBV disease. Maternal to infant transmission, prior to the first dose of vaccine, would also have been a plausible cause for the presence of HBcAb in these individuals.

In 2004, a well men's check in one remote Indigenous community in the Northern Territory found a 14-year-old male with active HBV infection and abnormal liver function. A review of his medical record showed that he had received all 3 recommended vaccine doses, at the correct times, as an infant. Health centre staff knew of a second child of the same age in the community who had become HBV positive following contact with an

infected individual at age three. Both young people had also been fully vaccinated against HBV at the correct times. The hepatitis serology of their mothers at birth is unknown. These findings prompted the medical staff to conduct a serosurvey of children in the community who had been vaccinated in 1989 and 1990.

This Aboriginal population was among the first to receive routine infant HBV vaccination and this is the first report of long term follow-up in a remote setting. The aim was to determine immunity to HBV in adolescents vaccinated with plasma derived HBV vaccine in infancy over a 2-year period (January 1989 to December 1990). The outcome measures were:

1. the prevalence of HBV infection as indicated by a positive test for HBV surface antigen (HBsAg),
2. the rate of past exposure to HBV or vaccine as indicated by the titre of HBV surface antibody (HBsAb),
3. the rate of past HBV infection, indicated by the titre of HBV core antibody (HBcAb).

Methods

Ethics approval was granted by the human research ethics committee of the Menzies School of Health Research and the Northern Territory Department of Health and Families. The governing Health Board gave permission to access relevant laboratory results. A clinic nurse matched pathology results to vaccination history, de-identified the data and provided it to the principal investigator. Permission was given by the appropriate Aboriginal Health Board to publish the results. The community general practitioner (GP) used the computerised clinical records system to identify children born to families in the community and neighbouring communities in 1989 and 1990. HBV vaccination details were retrieved for each individual. An Aboriginal Health Worker identified vaccinated adolescents currently living in the community and located those who had left. Forty-eight teenagers were identified but 11 could not be located, leaving a cohort group of 37. The GP obtained informed consent, collected blood for serology between May and July 2005 (15–16 years post vaccination) and counselled each person about the reasons for the test and the potential meaning of results.

All samples were tested for HBsAb, HBcAb, and HBsAg. The Table outlines the relevance of each serological test. An HBsAb titre <10 mIU/ml with a negative HBcAb was taken as indication of immunity due to vaccination. Post-vaccine immunological memory is defined as the case where HBbsAb titre < 10 mIU/ml and HBV exposure promotes a secondary immune response.[12] Low HBsAb titre could represent a lack of initial immune response, waning immunity, or a latent immune response that would be re-activated with exposure to the virus or vaccine. Immunological memory can be measured by testing HBsAb titre after a booster dose.[13]

Results of testing were explained to adolescents and their parents. Adolescents with evidence of current infection (positive HBsAg) were listed for regular follow-up. The GP arranged further liver function tests and counselled the adolescents, and their parents regarding the significance of the results.

Results

The GP screened 35 children, and two were tested by the school nurse at boarding school. The 11 adolescents who could not be located are being kept on a list for follow-up should they return to the community.

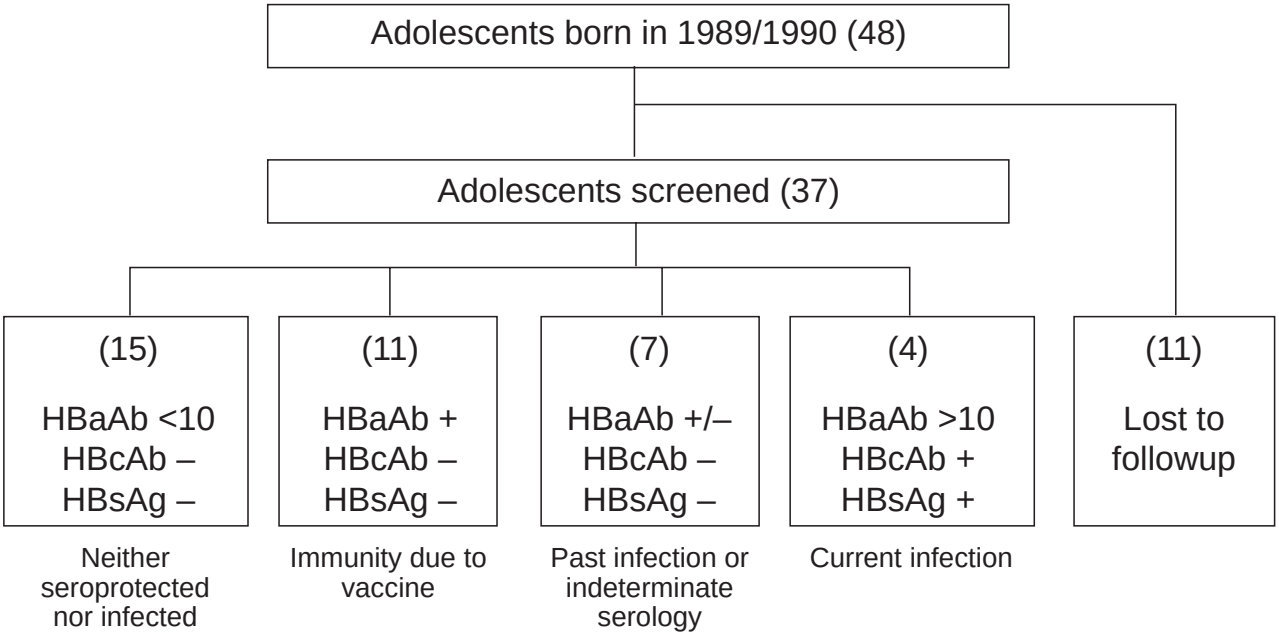
Of the 37 in the cohort, 4 (11%) had evidence of HBV infection, one with abnormal aminotransferase levels, 7 (19%) with evidence of past infection, 15 (41%) were considered not to have seroprotection (0 mIU/ml < HBsAb < 10 mIU/ml), and 11 (30%) were classed as having seroprotection according to the cut-off (Figure). Of the 11 adolescents with evidence of current or past HBV infection, all but one had received the first dose of vaccine at birth. One child had received the first dose at 1 month of age.

Table: Guide to hepatitis serology

Abbreviation	Meaning of abbreviation	Explanation of HBV serology
HBsAg	Hepatitis B surface antigen	Presence indicates current infection
HBsAb	Hepatitis B surface antibody	Presence indicates immunity
HBcAb Tot	Hepatitis B core antibody (total)	Presence indicates current or past infection
HBcAbIgM	Hepatitis B core antibody (IgM)	Presence indicates recent acute infection
HBeAg	Hepatitis B e Antigen	Presence indicates highly infective stage
HBe Ab	Hepatitis B e Antibody	Presence indicates reduced infectivity status

Adapted from Western Diagnostic Pathology

Figure: Results of screening survey



Discussion

Eleven fully vaccinated children (close to 30%) in this cohort showed evidence of past infection with HBV, and 4 had ongoing infection as determined by positive HBsAg. This compares with the study of Wood et al (2008)[5] (Australian Aboriginal Birth Cohort Study) who followed up 401 Aboriginal adolescents aged 16–20 years, vaccinated across 50 communities between 1987–1992. Evidence of past HBV infection was found in 21%, and 1.5% had persistent infection. Half were considered to have inadequate immunity and were given booster doses. In this cohort, only a third had received on-time childhood HBV vaccination in infancy.[5]

Plasma derived vaccines were used before the introduction of recombinant vaccines in 1991,[14] and when used in infancy, were accompanied by a relativity inferior anamnestic response.[15] Moreover, evidence-based guidelines for storage and transport of vaccines were not developed by the World Health Organization until 1992/1993.[16] A 1994 study conducted in the Northern Territory tagged 144 vials of HBV and 127 vials of poliomyelitis vaccine distributed to community settings. The cold chain was breached frequently with 23% of temperatures too high and 47.5% too low. The more stages there were in transportation, and the more remote the setting, the higher the risk of being exposed to freezing temperatures.[8] As a result, inclusion of freeze monitors is now considered best practice. Awareness of freezing has increased since cold chain monitoring became policy in Australia in 2001.[14] In 1989 and 1990, it is quite likely that some batches were frozen during transport or storage.

Another consideration would be persistent maternal–infant transmission despite proper vaccination. Hepatitis B immunoglobulin is recommended, in addition to infant vaccination, for the prevention of maternal–infant transmission. The HBsAg status of the mothers at the time of birth of the study adolescents is not known. However, HBV has high prevalence in Indigenous communities. In 1987, the prevalence of HBV infection among non-metropolitan Aboriginal women at prenatal screening in Western Australia was 3.6%.[17] Estimates of the population prevalence of HBV infection among Aboriginal populations in Western Australia and the Northern Territory have varied from 3%–22%.[17–21] A recent East Arnhem Land survey found a high prevalence of chronic infection, with a prevalence of HBsAg in 12% of adults.[22]

Several studies have demonstrated that infant vaccination does not always prevent vertical transmission. In China, a long-term follow-up of 95 adolescents who were vaccinated with plasma derived hepatitis vaccine but not immunoglobulin at birth, 1 and 2 months, and whose mothers had HBV infection, found 9% had evidence of past infection (positive HBcAb), though none developed clinical hepatitis.[23] In a similarly designed Canadian study, of 770 children who had been vaccinated at birth with immunoglobulin followed by the plasma derived vaccination at 0, 1 and 2 months, 5% had developed HBcAb[24] when followed up at 8 years. Another 5-year follow-up study of vaccinated infants of carrier mothers using recombinant vaccine found that 12% (19/162) subsequently developed HBcAb by age 5 years (6/19 before age one) and that maternal HBcAb disappeared from the blood of the infant

in 99% of children before age 2 years.[25] While the post-birth period is the highest risk period for seroconversion, the infection might not be established or detected until as late as age two.[25]

Low birth weight (LBW), with or without prematurity, is common among Indigenous infants, and failure to thrive occurs more commonly than in the general Australian population. In addition, many infants live in crowded conditions, exposed to multiple childhood infections and life stressors. Infant mortality is unacceptably high.[26] Rates of seroprotection are lower for preterm compared with full term infants.[27] Theoretically, lack of immunological maturity in LBW infants could compromise vaccine response; however, when infection and other co-morbid illnesses are excluded, there appears to be no difference.[27] It is possible that prematurity, recurrent infection and ongoing poor nutrition in infants contribute to a suboptimal immune response.

Some studies have suggested that lower immune response among Aboriginal infants is genetically determined.[2–4] Additionally, HBV vaccine escape mutants can lead to vaccine failure.[12,28] For practical reasons, neither HLA typing nor the presence of escape mutants could be assessed in this study setting.

The size of the cohort was small and 11 adolescents could not be located; this raises concerns of potential attrition bias. A sensitivity analysis suggests that if all the missing subjects had evidence of past infection, the proportion with hepatitis infection would be 22/48 (46%). If all 11 were immune, the proportion would have been 11/48 (23%), more in keeping with the expected infection rates in a population with a high rate of maternal infection.

The major limitation of this study is that the presence of immunological memory was not investigated for those adolescents with HBsAb <10 mIU/ml and HBsAg negative. Therefore, the immune status of this subgroup was unclear. A similar 15-year follow-up study of Micronesian infants illustrates the importance of checking for immunological memory. A group of 105 children were vaccinated with recombinant vaccine in infancy. HBcAb was determined at baseline, at 35 months, and again after 15 years.[13] The rate of HBcAb in this study was much lower at 15 years post vaccination than in our study, 7.6% compared with 30%, and none became HBsAg positive. HBV booster doses were given to 96 subjects without HBcAb to investigate immune memory; 47.9% had an anamnestic response.[13] If this study had tested immune memory in this way and found similar results, then around half the adolescents may have an anamnestic response.

No studies have demonstrated active HBV disease in vaccinated subjects (without renal failure) with initially documented seroprotection. Even though protective seroimmunity levels and the anamnestic response may wane over time,[10,11] they do not necessarily constitute vaccine failure. There is probably still protection from progressive liver disease and hepatocellular carcinoma.[10,11] Further investigation should examine the possible benefits of post-vaccination serological testing for high risk infants such as those in remote Aboriginal communities, as additional doses may result in seroconversion in a proportion of those who initially fail to respond to vaccination.

The results of this study are a cause for concern. There may be a significant proportion of Aboriginal adolescents vaccinated in 1989 and 1990 who have chronic HBV infection, and another substantial subset who are non-immune, despite having received complete HBV vaccination courses as infants. The authors recommend further investigation across remote Indigenous Australian populations to determine the proportion, location, and vaccine batch used in those whom vaccine failure has occurred and identify those who are not immune as well as those who are chronically infected. A systematic community-based program could be carried out as part of regular Adult Health Checks and is recommended to detect those with chronic HBV infection and provide access to ongoing monitoring and treatment

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Nosocomial pertussis infection of infants: still a risk in 2009

Jennifer M Paterson, Vicky Sheppeard

Abstract

The Sydney West Centre for Population Health investigated a confirmed pertussis infection in a health care worker on a maternity ward and identified pertussis infection in 4 neonates cared for by this case. This report describes the public health intervention to identify and prevent further cases. Of the 4 neonates, three were laboratory-confirmed cases and one was diagnosed on clinical grounds alone. All were cared for by the infected worker during only one shift and developed symptoms six to 16 days afterwards. No other possible source of infection was identified. This investigation highlights the need to maintain awareness, particularly amongst staff working with neonates, that pertussis infection can arise despite complete vaccination. Thus it is important to investigate new coughing illnesses and exclude symptomatic staff from contact with neonates until pertussis infection is excluded or effectively treated. The burden on the health system arising from a pertussis infection in a health care worker in a high-risk setting is also described with the hospitalisation of 4 infants, and prophylactic antibiotics given to 73 new mothers, infants and health care workers. *Commun Dis Intell* 2010;34(4):440–443.

Keywords: pertussis transmission, neonates, infants, health care worker, outbreak

Issues raised by this paper

Vaccinated health care workers can contract pertussis infection and their symptoms are likely to be modified compared to classic pertussis; nevertheless they can be responsible for transmission to others, especially vulnerable infant contacts.

Reinforces the recommendation for all exposed HCW (irrespective of immunisation status) to receive chemoprophylaxis if in contact with vulnerable patient populations.

Implications for how frequently re-vaccination is required, and the need for a monovalent acellular pertussis vaccine.

Introduction

A pertussis outbreak commenced in New South Wales in February 2008 and peaked in December of that year.[1] The peak number of notifications greatly exceeded that seen in other outbreaks during the past decade (Figure). About 54% of the cases were children under 15 years of age, with 21% of cases notified in children under 5 years.

Pertussis containing vaccine is free for Australian children at 2, 4 and 6 months of age, with a booster at 4 years and during adolescence.[2] All doses are acellular pertussis vaccine combined with diphtheria and tetanus antigens. This outbreak occurred in the context of good childhood vaccination coverage (93% at 12 months and 90% at 4 years).[3]

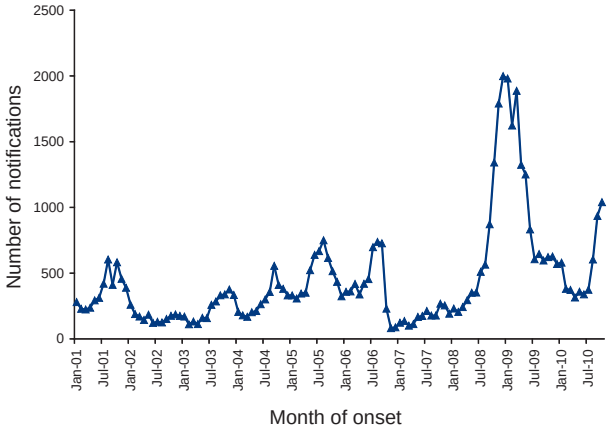
Pertussis infection is of most concern during the first year of life, particularly prior to receiving the first 2 doses of vaccine. In addition to increased susceptibility of acquiring infection, infants are also most at risk of developing severe complications, including death, from pertussis infection.[4]

This report describes the investigation of a pertussis infected health care worker (HCW), the probable transmission to 4 neonates in a maternity ward, and the public health intervention to prevent further cases.

Methods

Pertussis is a notifiable condition in New South Wales and must be notified to the local public health unit by doctors and laboratories. Pertussis infection is confirmed by isolation of *Bordetella pertussis* by culture or by detection with nucleic acid testing. Cases with clinical evidence of pertussis (a coughing illness lasting more than 2 weeks or history of paroxysmal cough, inspiratory whoops or post-tussive vomiting) are also considered confirmed cases if

Figure: Pertussis notifications, New South Wales, January 2000 to October 2010



they have a positive serological test or if they have an epidemiological link to a case with laboratory evidence of pertussis.

In this outbreak all upper pharyngeal specimens were tested by nucleic acid methods using polymerase chain reaction (PCR) testing.[5]

Public health follow up of notifications was undertaken according to New South Wales and Australian guidelines.[6] The guidelines include provision of prophylactic antibiotics to all neonates and parents directly cared for by an infectious case, if they can be commenced within 21 days of the last contact, and all health care workers who have been exposed to the infected staff member who are to care for neonates in the next 3 weeks, regardless of vaccination status.

Exposed patients and staff were identified by the maternity unit nurse manager through a review of staff rosters. Public health unit staff contacted all identified patients by phone using a standard interview format. Neonates reporting cough or apnoeas were requested to attend a paediatric emergency department for assessment. All other neonates and mothers were requested to attend a public health clinic held in the maternity unit if within 21 days of last exposure, or were provided with information about the exposure and potential risk if more than 21 days had elapsed.

Exposed HCWs were contacted by managers and assessed against the guidelines. Any symptomatic staff members had a throat swab taken for pertussis PCR testing.

Results

Index case

On 15 July 2009 the Public Health Unit was notified of a positive pertussis PCR test in a 54-year-old HCW. The HCW reported an influenza-like illness from 26 to 29 June and a coughing illness from 7 July. The HCW presented to a general practitioner on 26 June and again on 10 July when a swab was taken for pertussis testing and erythromycin treatment commenced. The HCW had received an acellular pertussis vaccine in 2006.

The HCW had worked as a registered nurse on a maternity ward on 28 and 30 June and 7 and 9 July and provided direct care to 39 mothers and newborns during those shifts. Twenty HCWs were also identified as being exposed to the index case while infectious.

Secondary cases

Four infant contacts of the HCW were confirmed cases of pertussis, three by laboratory test and one with a cyanotic episode, mild cough and epidemiological link (Table). No other potential source of infection could be identified for these cases using the standard pertussis investigation questionnaire.[7] The incubation period ranged from 6–16 days. All 4 secondary cases had short hospital stays but none required intensive care.

Two other infants who had a cough reported but negative pertussis PCR tests, received prophylactic antibiotics and information about pertussis according to the same guidelines as followed for asymptomatic infants. Their mothers also received prophylactic antibiotics.

A HCW contact who reported a spasmodic cough also tested negative for pertussis by PCR and returned to work after commencing antibiotic prophylaxis.

No secondary cases were confirmed amongst adult patients or HCWs.

Other public health interventions

Antibiotic prophylaxis was provided to another 25 infants and 26 mothers. Azithromycin was administered according to Australian Therapeutic Guidelines to all contacts except those with macrolide allergy, where trimpethoprim-sulfamethoxazole was substituted.[8] Contacts were also advised of the potential risk of developing pertussis and to seek medical review if symptoms developed.

Eight mother and infant pairs were contacted more than 21 days after exposure so were only provided with information about the exposure and potential risk.

HCW records were reviewed and all had received vaccination with acellular pertussis vaccine within

Table: Details of neonatal pertussis cases

Age at onset	Exposure date	Onset date	Symptoms	Laboratory test and date
10 days	7 July	15 July	Mild cough	PCR + 16 July
8 days	9 July	15 July	Mild cough, cyanotic episode	Not tested
16 days	30 June	16 July	Apnoeas and vomiting (rhinorrhoea noted 13 July)	PCR + 17 July
21 days	30 June	14 July	Cough	PCR + 21 July

the past 3 years. Eighteen HCW who were rostered to work on the maternity ward within 21 days of last exposure to the infectious case were administered azithromycin daily for 5 days, or trimethoprim-sulfamethoxazole twice daily for 7 days

Maternity ward managers were requested to maintain a high awareness for the symptoms of pertussis amongst their staff, and to exclude any HCW with a coughing illness until they tested negative for pertussis. Managers and HCW were also reminded that pertussis can develop despite recent vaccination or past infection.

Discussion

This incident highlights the highly transmissible nature of pertussis infection with 1 epidemiologically-linked and 3 laboratory-confirmed cases of pertussis in neonates cared for by an infected HCW. Each neonate was exposed during only 1 shift and developed symptoms six to 16 days after exposure. Fortunately none of the infants developed severe disease and to our knowledge no other cases of pertussis developed amongst the 27 neonates provided with prophylaxis or the 8 neonates who were identified too late for antibiotics.

As culture isolates were not obtained it could not be conclusively proven that the neonatal infections were acquired from the HCW. However no other possible source of infection amongst the neonates' other contacts could be identified. Transmission from HCWs to neonates is well documented, albeit, not recently in Australia.[9–13] Thorough investigations of infected HCWs in maternity wards have not always yielded clear evidence of infection transmission.[14,15]

The index case's history of symptoms was not completely typical of pertussis infection, with an initial period of fever and nasal congestion that was reported to resolve completely, followed 1 week later by the more typical coughing illness of pertussis.[16] This resulted in some uncertainty in defining the infectious period. However 2 laboratory-confirmed cases were identified as related to the earlier phase of illness, more than 7 days before the index case could recall any cough.

This outbreak also underlines the cost to the health system of a pertussis infection in a HCW in a high-risk setting such as a maternity unit. In addition to the hospitalisation of 4 infants, 73 courses of prophylactic antibiotics were administered. Providing this intervention as soon as possible after recognition of the exposure drew considerably on public health and maternity unit resources. The serious adverse health and economic burden of pertussis outbreak control in hospitals has been previously described.[17–19] Ward et al note that where infants are exposed to pertussis the costs are higher due to the higher expected hospitalisation rates.[19] Some authors have attempted to model the effectiveness of HCW vaccination compared to outbreak control measures and suggest that there is a health and economic benefit from HCW vaccination.[20,21]

In 2006 Edwards et al reviewed the challenges of controlling pertussis outbreaks in hospitals and noted the promise of adult pertussis booster vaccines to markedly reduce or eliminate this risk.[10] It is disappointing that this outbreak occurred despite the mandatory NSW Health policy for all HCWs to have received the full primary course of pertussis vaccination, and an adult booster dose.[21] Staff in maternity and paediatric units have been prioritised to achieve compliance with this policy. All the staff in this unit, including the index case, had evidence of complete pertussis vaccination.

Thus despite adoption of a mandatory HCW vaccination policy a risk to infants for nosocomial infection with pertussis continues. Several factors unique to pertussis infection and highlighted by previous authors play a part in this ongoing risk: immunity wanes over time and multiple life-time infections can occur; vaccination provides protection to only around 80% of those vaccinated; cases with prior vaccination or infection tend to have mild/atypical symptoms.[10,22] These factors lead to delay in recognition, diagnosis and treatment, potentially resulting in ongoing exposure during the most infectious period of the disease.

Hospital infection control practitioners and maternity HCW must maintain awareness that pertussis infection can arise despite complete vaccination and investigate new coughing illnesses. HCWs must be excluded from contact with neonates until pertussis infection is ruled out or effectively treated. As the early stages of pertussis can mimic many other upper respiratory conditions, access to rapid diagnostic testing is essential to protect neonates from infection while minimising disruption to staff rosters. Vigilance is particularly required during pertussis epidemics, when the likelihood of community-acquired pertussis infection in HCW increases. A flexible human resources policy that allows redeployment of maternity unit staff with cough to lower risk duties (and their replacement with suitably trained staff from other units) while awaiting test results could help reduce the risk of such incidents in the future.

Earlier development of immunity to pertussis would also assist in reducing the risk of infection during the neonatal period. Strategies such as maternal immunisation during pregnancy (to provide passive infant immunity) and a birth dose of pertussis vaccine are under investigation to determine if earlier protection can be provided safely and effectively to

neonates.[23] Of these options, only passive immunity could provide protection for neonates exposed to infected HCWs in the immediate post-natal period.

Finally, public health units have a role in ongoing raising of awareness and education of HCWs in high risk settings of the current pertussis transmission risk in the local community; effectiveness of vaccination; recognition of early symptoms; and the latest treatment and exclusion policies.

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Melioidosis in north Queensland, 2000–2009

Jeffrey N Hanna, Jan L Humphreys, Dianne L Brookes, Terrianne Messina, Alexandra Raulli

Abstract

There were 176 culture-confirmed cases of melioidosis in north Queensland over the 10 years, 2000–2009. Most (nearly 80%) occurred in the first 4 months of the year. The overall case fatality was 21%, but was 14% in 2005–2009. Of the 173 adult cases, 45% were in Indigenous adults. Both diabetes and alcohol abuse were more prevalent among Indigenous adults with melioidosis than among non-Indigenous adults. The incidences in Indigenous adults were particularly high in the Torres Strait and Northern Peninsula Area, Cape York and Mornington Island, whereas for non-Indigenous adults there appears to be a higher risk within Townsville city. *Commun Dis Intell* 2010;34(4):444–447.

Keywords: *Burkholderia pseudomallei,* Indigenous, melioidosis, Queensland, septicaemic pneumonia

Introduction

Melioidosis is an infection caused by the soil-dwelling bacterium *Burkholderia pseudomallei,* which it causes a wide range of clinical syndromes ranging from mild superficial skin infections to fulminant septicaemic pneumonia.[1] Endemic melioidosis has long been recognised in north Queensland[2] and it was a notifiable disease in Queensland in the 1980s and the first half of the 1990s. Although it was not a notifiable disease in the latter half of the 1990s, it became gazetted as a notifiable disease again in mid-1999. The objective of this study is to describe the salient features of the melioidosis cases that occurred in north Queensland over 10 years, 2000–2009.

Methods

Cases of melioidosis were defined by the isolation of *B. pseudomallei* from any clinical sample; serological diagnoses were not considered valid.[1] Relapses of the disease in those known to have had a previous culture-confirmed episode were not included, nor were cases known to have been acquired outside north Queensland. (There were several 'imported' cases from the Northern Territory and Papua New Guinea during the 10 years.)

Upon notification, a standard questionnaire was used to collect details about each case, including indigenous status, occupation, clinical details, risk factors and any apparent exposures to soil and surface waters. Retirees and pensioners were included in a single occupation category, as were manual and outdoor workers. As well as the recognised risk factors for melioidosis (i.e. diabetes, alcohol abuse, chronic renal and lung diseases, malignancy and immunosuppression[1]), 'aged' was also included. This was defined as ≥50 years and ≥65 years of age in Indigenous and non-Indigenous adults, respectively.

Acute disease was defined by duration of illness of less than 2 months but if there was uncertainty about the onset date of the more chronic cases, the first day of the apparent month of onset was used. It was assumed that the place of onset of the acute cases was likely to have been in the Health Service District that included the place of residence, with the only exception being a miner who worked remotely from his home address.

Incidence rates were calculated using the Experimental Estimated Resident Populations (ERPs) based on national census data; these ERPs have been specifically developed to define Queensland Health Service District (HSD) populations. The ERPs of Indigenous and non-Indigenous adults ≥15 years of age in north Queensland in 2006 were approximately 46,610 and approximately 463,970 respectively.

One– and two-sample tests of proportion were used as appropriate.

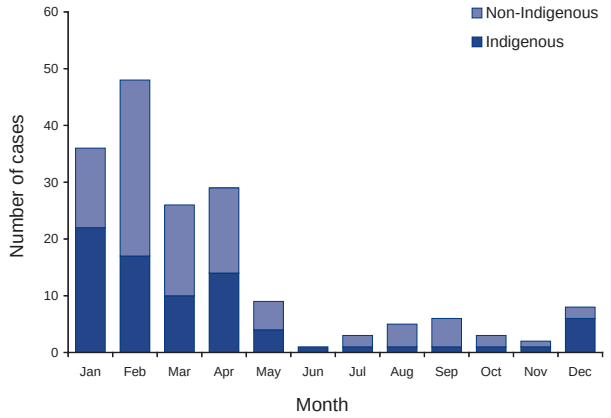
Results

There were 176 culture-confirmed cases of melioidosis in north Queensland over the 10 years, ranging from 8 (in 2003) to 38 (2000) cases per year. Of the 176, 139 (79%) had an onset in the first 4 months of the year (Figure). There were 36 melioidosis-related deaths; a case fatality of 20%. The case fatality rate in the first 5 years, 2000–2004, was 26%, compared with 14% in 2005–2009 ($P > 0.05$).

There were only 3 cases of melioidosis in children aged less than 15 years over the 10 years. An Indigenous infant, who possibly acquired melioidosis through breastfeeding, died from overwhelming sepsis at 5 days of age.[3] An 11-year-old non-Indigenous boy, in otherwise good health, developed a superficial melioidosis abscess, and a 12-year-old non-Indigenous girl developed severe neurological

melioidosis with a catastrophic outcome (i.e. quadriplegia). These paediatric cases will not be considered further.

Figure: Monthly distribution of the melioidosis cases, north Queensland, 2000 to 2009



The clinical presentations of the 173 cases in those aged ≥15 years are given in Table 1, with many cases involving multiple organs. There were 120 (69%) patients with pneumonia and/or septicaemia, which are combined here as it was often clinically difficult to distinguish between the two.

Of the 173 adult cases, 78 (45%) were in Indigenous people. Of these, 47 (60%) occurred in 2000–2004, compared with 31 (40%) in 2005–2009 (P <0.01). The melioidosis related case fatality rate in Indigenous adults was 21%.

Many patients had more than one risk factor (Table 2). The distribution of 'aged' persons was the same among Indigenous (41%) and non-Indigenous cases (41%), but there were more diabetics among the Indigenous (71%) compared with the non-Indigenous cases (33%) (P <0.01). Alcohol abuse was more prevalent among Indigenous adults with melioidosis (41%) than among non-Indigenous adults (25%) (P <0.05).

The occupations of the adult cases are given in Table 3. A number of the retirees and pensioners volunteered that they were recreational gardeners, and seven of the manual and outdoor workers were miners.

One hundred and sixty (92%) of the adult cases were acute. The distribution of these acute cases throughout the HSDs is shown in Table 4. The average annual incidence in adults in the Townsville HSD (3.3 cases (95% CI, 2.6–4.3 cases) per 100,000 adults) was greater than that in the Cairns HSD (1.4 cases (95% CI, 0.9–2.1 cases) per 100,000 adults) (P <0.05). Furthermore, whereas only 62% of the acute adult cases in the Cairns HSD were apparently acquired within Cairns city (and adjacent suburbs), 92% of the Townsville HSD cases were acquired

Table 1: Clinical presentations of melioidosis in adults, north Queensland, 2000 to 2009

Clinical presentation	Cases with the presentation*	
	n	%
Pneumonia	76	44
Septicaemia	76	44
Superficial tissue infection	28	16
Internal organ abscess	25	14
Urinary infection	15	9
Septic arthritis	11	6
Neurological disease	6	3
Other	6	3

* Numbers do not add up to 100% as many cases included multiple organs.

Table 2: Risk factors for melioidosis in adults, north Queensland, 2000 to 2009

Risk factor	Cases with the risk factor*	
	n	%
Diabetes	87	50
Aged	71	41
Alcohol abuse	56	32
Chronic respiratory disease	28	16
Chronic renal disease	21	12
Immunosuppression/transplant	19	11
No risk factors	18	10
Other	16	9
Malignancy	12	7

* Numbers do not add up to 100% as many cases included multiple risk factors.

Table 3: The occupations of the adults with melioidosis, north Queensland, 2000 to 2009

Occupation	Number of cases	%
Retiree/pensioner	68	39
Manual/outdoor worker	35	21
Unemployed	34	20
Other occupations	20	12
Home duties (females)	15	9

within Townsville city ($P < 0.01$). Seventeen (81%) of the 21 acute cases among Indigenous adults in the Mt Isa HSD were from Mornington Island.

Table 4: The distribution of acute adult melioidosis, north Queensland, 2000 to 2009, by Health Service District

Health service district	All acute cases	Indigenous acute cases
Bowen	4	0
Cairns	21	3
Cape York	17	15
Charters Towers	0	0
Innisfail	0	0
Mackay	2	0
Moranbah	1	0
Mt Isa	24	21
Tablelands	7	1
Torres Strait and Northern Peninsula Area	24	23
Townsville	60	13
Total	160	76

The average annual incidence in Indigenous adults was the same in both the Torres Strait and Northern Peninsula Area and Mt Isa HSDs: 42 cases (95% CI, 26–63 cases) per 100,000 Indigenous adults aged ≥15 years. The incidence in Cape York HSD was similar: 40 cases (95% CI, 22–66 cases) per 100,000 Indigenous adults.

Discussion

This 10-year prospective study has shown that many of the features of melioidosis in north Queensland are similar to those documented elsewhere from the tropical north of the Northern Territory.[1]

Most (nearly 80%) of the cases are compressed into the first 4 months of the year coinciding with the height of the annual monsoonal wet season throughout the region. These months also coincide with the tropical cyclone season, and several tropical cyclones were soon followed (i.e. within a week) by 'clusters' of melioidosis, for example in Townsville city in 2000 (10 cases) and Mornington Island (4 cases) in 2002. However, Cape York was affected by cyclones in 2005 and 2006, but neither cyclone was followed by such clusters.

Some wet seasons were associated with a relatively small number of cases, for example in 2001 (9 cases) and 2003 (8 cases). Furthermore, there was a relative paucity of cases throughout much of the wet tropics bioregion, from Ingham via Innisfail, Cairns and Mossman/Port Douglas north to Daintree. Indeed, no cases were reported from the Innisfail HSD (which has the highest rainfall in north Queensland) over the 10 years, even following severe Cyclone Larry in 2006.

The study has identified two very localised geographic foci, both apparently associated with increased risk of melioidosis: Townsville city (where the cases occur predominantly in non-Indigenous adults) and Mornington Island (where cases occur exclusively among Indigenous adults). The 'high-risk' focus of Townsville city has been recognised previously, and it has been suggested that some specific soil characteristics may contribute to this local risk.[4]

The study has also identified very high incidences among Indigenous adults in both the Torres Strait and Northern Peninsula Area and Cape York HSDs. However, in both there was no focal distribution of the cases, which were scattered throughout the islands and communities in these HSDs. Presumably, the high risk for Indigenous adults within these HSDs is more a reflection upon the high prevalence of co-morbidities, particularly diabetes, in these adults rather than any specific soil characteristics.

Similar to those documented in the Northern Territory,[1] the most prevalent risk factors for melioidosis in adults in north Queensland are diabetes, alcohol abuse and chronic respiratory disease. Diabetes was particularly prevalent among the Indigenous adult cases, and this may have contributed to the considerably higher overall prevalence of diabetes in melioidosis cases (50%) compared with that documented in the Northern Territory (37%).[1] Another potential risk factor is being 'aged'; although the definitions differed, the percentages of aged Indigenous and non-Indigenous adults were very similar.

Clearly, the relatively high percentage of cases that were either retirees or pensioners reflects the number of cases that were either aged or had significant co-morbidities. However, it is of concern that approximately 20% of the cases were either manual or outdoor workers, suggesting that melioidosis may be a significant occupation-related infection in north Queensland, particularly in those with underlying conditions such as diabetes.

Many of the clinical features seen in north Queensland have been described previously from the Northern Territory.[1] Paediatric melioidosis is rare in north Queensland. The 12–year-old non-Indigenous girl with neurological melioidosis was very unusual for several reasons: her young age, severe disease despite the absence of underlying risk

factors, onset during a 'low risk' month (September), and she was the only case from the farming areas near Atherton during the 10 years.

The predominant clinical presentation in adults is pneumonia and/or septicaemia. Eighty-three per cent of the 36 melioidosis-related deaths in adults occurred in those with pneumonia and/or septicaemia, but all except one had significant underlying co-morbidities. The overall melioidosis case fatality was similar to that in the Northern Territory (19%).[1] Although not statistically significant, the recent decline in the case fatality is encouraging, and may be a reflection upon the recent decline in cases in Indigenous adults.

Internal organ abscesses (liver, splenic, prostatic and lung abscesses) are not uncommon, with some requiring extensive surgery. So as to recognise clinically unapparent visceral abscesses, abdominal–pelvic CT scanning of all adult melioidosis cases is recommended in the Northern Territory regardless of clinical presentation,[1,5] but this does not yet seem to be a routine practice throughout north Queensland. Therefore there could have been an under-ascertainment of some abscesses—prostatic abscesses in particular.[5]

Superficial tissue infections (ulcers and abscesses) were also common. Twenty (77%) of those with superficial infections were non-Indigenous adults, and 9 (32%) had no underlying co-morbidities. Six of the 13 with chronic disease (duration of illness ≥2 months) had superficial tissue infections.

Although simple messages about melioidosis and measures that should be taken to reduce the risk of disease are publicised annually, the reality is that melioidosis is probably not a preventable condition at the current time. Indeed, with the aging population, the increase in chronic diseases, and an increasing population in north Queensland, it is quite plausible that melioidosis could actually increase over time. Clinicians in north Queensland need to maintain a high degree of suspicion of the disease in an acutely unwell adult with a systemic febrile illness, particularly if the adult is diabetic or aged or has alcohol-related problems, during the first 4 months of the year, and particularly if the adult is a resident of Townsville city, Mornington Island, Cape York or the Torres Strait and Northern Peninsula Area.

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Short reports

HIV, malaria and pneumonia in a Torres Strait Islander male – a case report

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Abstract

This report presents the case of a middle-aged Torres Strait Islander male with HIV who contracted Plasmodium vivax malaria in Papua New Guinea. His presentation included clinical and radiological features of pneumonia and he required inpatient treatment for 13 days. This study reviews the literature concerning co-infection with HIV and malaria, which is an uncommon combination in Australia, discusses the public health risks posed by patients with malaria in the Torres Strait, given the presence of a known vector, and suggests strategies to reduce the disease burden posed by malaria in this patient and other Torres Strait Islanders travelling to Papua New Guinea under the terms of the Torres Strait Treaty. *Commun Dis Intell* 2010;34(4):448–449.

Case history

A 57-year-old Torres Strait Islander male presented to the primary health centre on a remote island in the Torres Strait with a week-long history of fevers, myalgia and a cough productive of green sputum. He had recently returned from 3 weeks in Daru in Western Province, Papua New Guinea (PNG). The patient's past medical history was significant in that he was HIV positive on antiretroviral therapy (lamivudine, zidovudine and azatanavir); though 6 months earlier he had an undetectable viral load and a CD4 count of 0.5 x 10^9/L indicating a reasonable level of immune function. The previous year he had required hospital admission for severe community acquired pneumonia.

On presentation he was hypoxic and in septic shock with rigors. He was also noted to have an infected ulcer on his left lower leg with associated left inguinal lymphadenopathy. An initial immunochromatographic test for pan-malarial antigens was positive.

The patient was transferred to Thursday Island Hospital where upon arrival he was commenced on artemether/lumefantrine combination therapy as well as intravenous ceftriaxone and gentamicin. A chest x-ray confirmed the diagnosis of pneumonia; malaria films on day two revealed *P. vivax* malaria with an initial parasite count of 23,000/μL and numerous trophozoites and occasional schizonts present. After excluding glucose-6-phosphate dehydrogenase deficiency, primaquine was commenced; artemether/lumefantrine was ceased after 6 doses as per the Torres Strait Malaria Protocol (S. Parish, 2009[1]). Blood and sputum cultures (including for acid-fast bacilli) were negative, as was serology for dengue and melioidosis. While an inpatient the patient's white cell count reached a nadir of 3.2 (with a neutrophil count of 0.69) and platelets of 74. Repeat malaria parasite screens showed reduced trophozoite load by day two of admission and complete clearance on days four and seven. The patient showed dramatic clinical improvement over the first few days of his admission and was discharged on day thirteen.

Discussion

While in many parts of the developing world the coexistence of HIV and malaria is unfortunately common, these infections are rarely seen together in Australia, where the burden of disease for each is relatively small.[2,3] The interaction between HIV infection and malaria has been extensively studied, with current knowledge suggesting that each infection may potentiate the other, mainly via effects on T-cell activation and immunity. More specifically, HIV has been shown to increase the infection rate, parasite density and severity of clinical illness of malaria, with an inversely proportional relationship between CD4 count and incidence of severe malaria, and decreases the response to malaria treatment.[4] Conversely, malaria increases HIV viral load and transmission and contributes to the development of anaemia.[5] Research from sub-Saharan Africa indicates that HIV-infected patients living in or travelling to malarious areas benefit from chemoprophylaxis with cotrimoxazole, which has been shown to be effective in reducing the burden of disease due to malaria and HIV-associated opportunistic infections, although it should be noted that malaria infections in the African cohorts studied were due almost exclusively to *P. falciparum*.[6] Another important area of research concerns the chemotherapeutic interactions between drugs used for the treatment of HIV and malaria, with some antimalarial agents exerting a weak antiretroviral effect, and certain antiretrovirals demonstrating malarial parasite growth inhibition.[7]

In the Torres Strait, the number of cases of HIV is limited to a few individuals, and the majority of patients with malaria are from PNG, with a small number of cases in Torres Strait Islanders who acquire the disease in PNG. There have however been documented cases of locally-acquired malaria, including 2 cases of *P. vivax* on Badu Island in 1997 and one of *P. falciparum* on Darnley Island in 2001.[8,9] Australia has a potential malaria vector in the form of *Anopheles farauti sensu lato*, which exists in the Torres Strait and represents the most significant threat with respect to local transmission of disease. During investigation of the Darnley case in 2001 several female *An. farauti s.l.* were trapped. In these previously reported cases the sources of the infections were unknown; none of the 3 individuals affected had travelled to PNG. The possible explanations for these cases include transport of an infected mosquito from PNG in one of the many boats that travel between PNG and the Torres Strait Islands (so-called 'baggage malaria'), or travel to the Torres Strait by an asymptomatic PNG national with parasitaemia. In this case it is most likely that the patient acquired malaria in PNG, given the average incubation period for clinical *P. vivax* infection (12–17 days) and the patient's travel history.

Public health significance

From a communicable disease perspective, this case is significant in several respects. Firstly, the concurrent infection with HIV and malaria in this patient incurred an increased risk of morbidity due to both diseases as discussed above. Secondly, the potential for local spread of *P. vivax* on the patient's home island represented a small but significant risk to the local community, given the historical presence of a known vector on the island and the fact that the patient likely had an increased parasite load due to HIV infection. Thirdly, the particular living arrangements of this patient, namely sharing a house with approximately 15 other individuals, lend themselves to vector-borne disease transmission. Finally, it is interesting that this patient almost certainly contracted both HIV and malaria on separate visits to PNG under the terms of the Torres Strait Treaty. This treaty allows free travel for traditional purposes, without passports or visas, within the Treaty zone for residents of the Torres Strait Islands and the inhabitants of 13 coastal village communities in PNG.[10] This unique arrangement represents a potential route for the spread of communicable diseases south to the Australian mainland. There exists a strong case for recommending routine use of malaria chemoprophylaxis for Torres Strait Islanders travelling to PNG, and additional cotrimoxazole prophylaxis for those with HIV infection. The efficacy of cotrimoxazole specifically for prophylaxis against *P. vivax* malaria warrants further investigation, given the relatively high prevalence of *P. vivax* malaria in PNG.[11]

The patient gave verbal consent for publication of this case report.

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Quarterly reports

OzFoodNet quarterly report, 1 July to 30 September 2010

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. 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 2010.

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 3rd quarter of 2010, OzFoodNet sites reported 633 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 illness. In total, these outbreaks affected 12,494 people, of whom 273 were hospitalised. There were 38 deaths reported during these outbreaks. The majority of outbreaks (86.4%, n=547) were due to person-to-person transmission (Table 1).

Table 1: Mode of transmission for outbreaks and clusters of gastrointestinal illness reported by OzFoodNet, 1 July to 30 September 2010

Transmission mode	Number of outbreaks and clusters	Per cent of total
Foodborne and suspected foodborne	34	5.4
Person-to-person	547	86.4
Unknown (*Salmonella* cluster)	11	1.7
Unknown (other known pathogen cluster)	1	0.2
Unknown pathogen cluster	40	6.3
Total	633	100.0

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 (Table 2). These outbreaks affected 469 people and resulted in 22 hospitalisations. There were 3 reported deaths during these outbreaks. This compares with 28 outbreaks for the 3rd quarter of 2009[1] and a 5-year mean of 26.8 outbreaks for the 3rd quarter between 2005 and 2009.

Salmonella was the aetiological agent for 12 outbreaks during this quarter, with *S.* Typhimurium being the most common serotype (n=11). There was 1 outbreak due to *S.* Virchow phage type 8 (Table 2.)

Of the remaining 22 outbreaks, seven were due to foodborne toxins, including 3 ciguatera fish poisoning, 2 *Clostridium perfringens* outbreaks, 1 histamine poisoning and 1 *Staphylococcus aureus* outbreak. There were 3 outbreaks due to norovirus and 1 outbreak due to *Campylobacter* infection. Eleven outbreaks were of unknown aetiology.

Eleven of the foodborne or suspected foodborne outbreaks (32%) reported in this quarter were associated with food prepared in aged care facilities, 9 (27%) with food prepared in restaurants, 3 (9%) in private residence and in 3 outbreaks (9%) the foods were associated with contaminated primary produce. Single outbreaks were associated with foods prepared in a range of other settings.

To investigate these outbreaks, sites conducted 5 cohort studies, 1 case control study and collected descriptive case series data for 23 investigations. In 5 outbreaks, no individual case data were collected. As evidence for the implicated food vehicle, investigators collected microbiological evidence in 2 outbreaks and analytical epidemiological evidence in 5 outbreaks. Descriptive evidence only was obtained in 27 outbreaks.

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

Table 2: Outbreaks of foodborne disease reported, 1 July to 30 September 2010 (n=34), by OzFoodNet sites

State or territory	Month of outbreak	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
ACT	September	Restaurant	Unknown	8	0	D	Unknown
NSW	July	Aged care facility	*Salmonella* Typhimurium	7	0	A	Unknown, possibly minced or pureed diet
	July	Private residence	*S.* Typhimurium (MLVA: 3-9-7-13-523)	9	5	D	Unknown, possibly mousse cake with raw eggs
	August	Restaurant	*S.* Typhimurium phage type 9 (MLVA: 3-21-12-13-523)	9	Unknown	D	Unknown
	August	Restaurant	*S.* Typhimurium (MLVA: 3-9-7-13-523)	14	4	M	Fried ice cream
	August	Restaurant	Unknown	11	0	D	Unknown
	August	Restaurant	Unknown	27	0	A	Suspect assorted wraps
	September	Aged care facility	*Clostridium perfringens*	8	1	D	Unknown
	September	Aged care facility	Unknown	16	0	D	Unknown
	September	Restaurant	Unknown	4	0	D	Unknown
NT	July	School	Unknown	19	0	D	Unknown
	August	Other	Unknown	62	0	D	Unknown
	August	Picnic	*S.* Virchow phage type 8	6	0	D	Unknown
Qld	July	Primary produce	Ciguatera fish poisoning	2	0	D	Spanish mackerel
	August	Primary produce	Ciguatera fish poisoning	2	0	D	Coral trout
	August	Primary produce	Ciguatera fish poisoning	4	0	D	Fish head soup
	September	Fair/festival/ mobile service	*Staphylococcus aureus*	3	Unknown	M	Rice noodle
SA	August	Institution	Unknown	8	0	D	Sandwiches
	August	Restaurant	*Campylobacter*	18	2	A	Steak with chicken liver pate
	Sept	Community	*S.* Typhimurium phage type 9	10	0	D	Unknown
Tas	July	Aged care facility	Norovirus	76	Unknown	A	Suspected pork sausage and gravy meal
Vic	July	Aged care facility	*C. perfringens*	16	0	D	Unknown
	July	Private residence	Histamine (scombroid) fish poisoning	4	0	D	Tuna
	August	Aged care facility	*S.* Typhimurium phage type 186	4	2	D	Unknown
	August	Aged care facility	*S.* Typhimurium phage type 197	23	4	D	Unknown
	August	Camp	*S.* Typhimurium phage type 9	6	1	D	Unknown
	August	Private residence	*S.* Typhimurium phage type 170	4	2	D	Eggs
	September	Aged care facility	Unknown	12	0	D	Unknown

Table 2: Outbreaks of foodborne disease reported, 1 July to 30 September 2010 (n=34), by OzFoodNet sites, *continued*

State or territory	Month of outbreak	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
WA	July	Aged care facility	Unknown	6	0	D	Unknown
	July	Restaurant	Norovirus	30	0	A	Lasagne
	August	Aged care facility	*S*. Typhimurium phage type 170 PFGE 11	7	0	D	Unknown
	August	Restaurant	*S*. Typhimurium phage type 170 PFGE 11	3	1	D	Unknown
	September	Aged care facility	Unknown	10	0	D	Unknown
	September	Military	Norovirus	21	0	D	Unknown

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.

Australian Capital Territory

There was 1 reported outbreak of foodborne or suspected foodborne disease reported during the quarter.

An outbreak affected 8 people in 2 separate groups who ate a variety of foods at a Chinese restaurant in September. Cases reportedly suffered diarrhoea, vomiting, fevers, chills, abdominal pain and headaches following consuming meals served on the same evening. Symptom onset occurred around 15–21 hours after eating, with illness lasting around 5 days. Cases were unwilling to assist further in the investigation and, as no faecal specimens were submitted, the aetiology remained unknown. An environmental health inspection identified a number of potential issues including inappropriate storage of cooked and raw products in the cool room as a possible source of cross contamination.

New South Wales

There were 9 reported outbreaks of foodborne or suspected foodborne illness during the quarter.

In August, two groups of diners reported developing symptoms of gastroenteritis after consuming meals from a Chinese restaurant over a 1-week period. In addition, active case finding identified an additional 2 people (related) who also reportedly ate at the same restaurant during the exposure period. In total, 14 of 15 people developed symptoms consistent with salmonellosis, with 6 stool specimens positive for *S.* Typhimurium (MLVA: 3-9-7-13-523). Food common to all cases was deep fried ice cream, made with unpasteurised whole egg. *S.* Typhimurium (MLVA: 3-9-7-13-523) was isolated from samples of raw and cooked deep fried ice cream. The New South Wales Food Authority issued a formal warning letter to the premises requesting that raw egg no longer be used in preparing deep fried ice cream.

All 9 guests became ill a median of 10.5 hours after consuming both home-made and commercially prepared foods served at a birthday party in July. Three stool specimens collected from party attendees were positive for *S.* Typhimurium (MLVA: 3-9-7-13-523). A homemade white chocolate mousse cake made with a raw egg filling is suspected to have been the cause of illness, with eight of 9 people consuming the cake. However, no cake samples were available for microbiological analysis.

In July, an outbreak of gastroenteritis affected six of 125 residents and 1 of 160 staff from all four wards in an aged care facility. The illness onsets of cases were spread over a 1-week period. Seven stool specimens were collected, with *S.* Typhimurium (MLVA: 3-9-7-13-523) isolated in specimens from 5 residents. A cohort study found an association between the consumption of minced or pureed diet and illness (RR 9.53, 95% CI 1.11–82.12), however, caution is required when interpreting this result given a number of possible confounding factors. The New South Wales Food Authority collected a number of environmental swabs and food samples during their environmental investigation, which were negative for *Salmonella* spp. No significant food hygiene or food safety issues were identified during the inspection. The source of the outbreak remains unclear, although a foodborne source was suspected.

A local public health unit identified an outbreak of gastrointestinal illness affecting 9 people in August. One person with *S.* Typhimurium (MLVA: 3-21-12-13-523) was interviewed upon notification, as this MLVA profile had not been previously reported in New South Wales. A restaurant in rural New South Wales, close to the Victorian border was implicated as the source of the salmonellosis. Other cases included 3 employees at the same restaurant (reported by the Victorian Department of Health) and a 4-month-old baby whose parents ate at the restaurant. Five cases were confirmed as having *S.* Typhimurium phage type 9 infection. The cases ate at the restaurant over a 2-day period, with a variety of meals consumed. It was reported that the chef of the restaurant had symptoms of gastroenteritis 2 days prior to the time when cases were likely to have been exposed, but this was not confirmed. No formal epidemiological or environmental investigations were conducted due the length of time that had elapsed since the onset of cases' illness.

In September, eight of 48 residents of an aged care facility became ill with symptoms including diarrhoea and vomiting, with illness onsets spread over a 6-day period. Four stool specimens were collected with *C. perfringens* enterotoxin A detected in two and an additional specimen was culture-positive for the bacterium. Affected residents were accommodated in two separate wards and although both wards were serviced by the same kitchen, different menus were provided to each ward. The New South Wales Food Authority conducted an environmental investigation of the kitchen, but no food vehicle or contributing factors that may have allowed *C. perfringens* to multiply in food were identified.

Twenty-seven of 45 people (61%) became ill with vomiting and diarrhoea a median of 37 hours after attending a catered meeting at a licensed club in August. Two specimens were collected which were negative for viral and bacterial pathogens. A cohort study was conducted, and an association between illness and the consumption of wraps (combined variable of chicken wraps and vegetable wraps) was found (RR 2.24, 95% CI 1.21–4.16). No ill food handlers were identified. Given the clinical

profile of cases and the onset times of illness, a viral point source outbreak was suspected, quite possibly associated with the consumption of assorted wraps. However, no pathogen was detected in stool or food and the aetiology remained unknown.

Eleven of 80 people attending a 21st birthday party at a hotel in August were ill with diarrhoea and vomiting. No specimens were collected. There were no reports of illness in functions held at the same venue on the same night (some meal items shared). It is suspected that this was a foodborne outbreak, however an environmental source or person-to-person transmission cannot be discounted and the aetiology remains unknown.

In September four of 8 people who ate a meal at a club, reported symptoms of diarrhoea and abdominal pain with median illness onsets 11 hours later. No other events or meals were common to all 4 cases. No specimens were collected. A council inspection identified problems with defrosting potentially hazardous foods, and overstocking and storage of foods in a freezer and cool room. Based on the clinical profile of cases, onset times of illness and the findings of the environmental investigation, a point source outbreak of a toxin-mediated pathogen was suspected, but the aetiology remains unknown.

In September,, 16 of 150 aged care facility residents became unwell with a diarrhoeal illness over a 4-day period (onset of illness for 82% of residents were on a single day). Five specimens were negative for bacteria, viruses and toxins. The New South Wales Food Authority conducted an environmental investigation and a review of the menu, but no aetiology or particular food vehicle for the outbreak could be identified.

Northern Territory

There were 3 reported outbreaks of foodborne or suspected foodborne illness during this quarter.

Whilst participating in a rally drive held across the Top End of the Northern Territory in August, 62 of 105 participants reported a predominantly diarrhoea only illness. Food was purchased or consumed in a variety of different settings during the rally. No common pathogen was identified from the 5 stool samples submitted, but the isolation of *Hafnia alvei* from two samples was of interest. Food samples were negative for common pathogens. No formal epidemiological study was undertaken due to the poor response rate to the initial outbreak questionnaires.

An outbreak in a school holiday care program affected 19 people in July. Epidemiology was suggestive of a point source foodborne outbreak. The aetiological agent was unknown but suspected to be viral. No clinical specimens or food samples were collected.

An outbreak of *S.* Virchow phage type 8 was investigated in a family of 6 persons travelling in the Northern Territory in August. All 6 family members became ill at the same time and *S.* Virchow phage type 8 was detected in all 4 of the stool samples submitted. The food vehicle is unknown.

Queensland

There were 4 reported outbreaks of foodborne or suspected foodborne illness during this quarter.

Two cases of suspected ciguatera fish poisoning were reported in July following the consumption of Spanish mackerel. The cases experienced symptoms including vomiting, diarrhoea, reversed temperature sensation and numbness/tingling of extremities. The fish was purchased from a local seafood retailer in Townsville.

Four family members became ill in August, with suspected ciguatera fish poisoning following the consumption of a home cooked curry that was prepared using a 2 kg fish head. Onsets of illness were between 3 and 12 hours after the meal. The species of fish used in the preparation of the curry was unable to be determined during investigations. The fish head was purchased from a local retailer in Brisbane. A traceback investigation was unable to be conducted.

Two family members became ill with suspected ciguatera fish poisoning after consuming coral trout. The cases experienced symptoms including reversed temperature sensation, numbness/tingling of extremities, diarrhoea and muscle pain approximately 11 hours after the meal. The fish was purchased from a local seafood retailer in Rockhampton and had been caught off Mackay.

At least 3 people reported illness with symptoms including vomiting, diarrhoea and abdominal cramps following the consumption of rice noodles at a multi-cultural festival held in Townsville in September. The cases experienced illness approximately 2 to 6 hours after consuming the noodles. *Staphylococcus aureus* ($>2.5 \times 10^7$ org/g) and staphylococcal enterotoxin were detected in rice noodle samples collected from the venue. A moderate growth of *Staphylococcus aureus* was also detected from a single faecal specimen that was collected 3 days post onset. No enterotoxin was detected in this sample.

South Australia

There were 3 reported outbreaks of foodborne or suspected foodborne illness during this quarter.

An outbreak of foodborne illness was investigated amongst patrons who attended a function and ate from a set menu at a restaurant in August. A total of 6 confirmed cases of *Campylobacter* infection and 12 presumptive cases were identified. Results from a cohort study conducted amongst attendees (n=32) at the function indicated that there was a statistically significant association between illness and eating steak with chicken liver pate (RR= 6.65; 95% CI 1.69–26.23). Food samples and environmental swabs were negative for *Campylobacter*. It was hypothesised that *Campylobacter* may have entered the kitchen through home grown eggs that were covered with mud and chicken faeces and that cross-contamination with the steak meal may have occurred.

Eight of 11 people who attended a training function in August became ill with vomiting and diarrhoea of a short duration. Symptom onset was 24–60 hours after the lunch served at the training session. A faecal sample was collected from 1 case but no agent was identified. A cohort study failed to implicate any particular food item. One of the people who prepared the sandwiches and wraps was ill two days before the training session. It was hypothesised that either the sick food handler may have contaminated the food served at lunch or an attendee was incubating the illness on the day of the lunch.

A community outbreak was investigated in September with 10 cases of *Salmonella* Typhimurium phage type 9 reported within a 2-week period. Hypothesis generating interviews found that four of those cases were linked to a common restaurant. An environmental inspection of the premises was conducted and food samples were collected. The restaurant served battered ice cream and battered bananas (foods commonly known to contain raw or lightly cooked egg), but none of these food samples tested positive for *Salmonella*. Also within this cluster of *S*. Typhimurium phage type 9, there was a small social cluster of 3 children (2 siblings and 1 neighbour) who regularly played together. It is likely that transmission of the bacterium amongst this group was person-to-person.

Tasmania

There was 1 reported outbreak of foodborne or suspected foodborne illness during this quarter. In an aged care facility, 49 of 221 residents and 21 of 96 staff reported developing symptoms of gastroenteritis. The majority of residential cases (73%) occurred within a 7 hour period, while half the staff cases occurred 3 days later. The median duration of symptoms was approximately 24 hours. Eleven samples were collected and seven tested positive for norovirus. There was difficulty in obtaining detailed food histories from those in care due to a high prevalence of dementia. However, the aged care facility's kitchen also delivered meals to the community. Six of the 36 meal recipients interviewed, reported developing symptoms of gastroenteritis. Persons who became ill were more likely to have reported consuming pork sausages and gravy (4/7, attack rate 57%, crude relative risk 3.71%, 95% CI 0.95, 14.55). The epidemiological evidence suggests that the aged care facility was the point source of this outbreak of norovirus with the likely initial route of transmission being foodborne rather than person-to-person. The reporting of illness in delivered meal recipients and the association between illness and one of the delivered meals also point to food as being the likely route of transmission.

Victoria

There were 7 reported outbreaks of foodborne or suspected foodborne illness during this quarter.

An emergency department registrar notified 2 cases of suspected scombroid fish poisoning in July. Investigations revealed that these 2 cases and another 2 people who were friends of these cases had consumed tuna steaks that were purchased from a fish retailer. All 4 people developed typical symptoms of histamine poisoning approximately 1 hour after consuming the tuna. There were some temperature control issues at the retail premises but as it was not possible to trace the supplier of the tuna, temperature control of the fish through the supply chain was unable to be assessed.

An outbreak of diarrhoea in an aged care facility that was notified in July manifested as 2 separate clusters, 2 weeks apart. Five residents and 1 staff member were affected in the 1st cluster, and all had an onset of illness over the same 24 hour period. Two faecal specimens were collected and one was positive for *C. perfringens* enterotoxin. Two weeks later in the 2nd cluster, 9 residents and 1 staff member were affected, with eight of the cases having an onset in the same 28 hour period. One case from the 2nd cluster had a faecal specimen positive for *C. perfringens* enterotoxin. Food processes were reviewed including vitamising of meals but no food source could be determined.

In August, 3 of 21 residents and 1 staff member of an aged care facility were notified to the Communicable Disease Prevention and Control Unit (CDPCU) with vomiting and/or diarrhoea. Two of the family members were subsequently found to have

Salmonella infection, which was later typed as *S*. Typhimurium phage type 186. Investigators were unable to identify the source of the outbreak.

Four members of the same family were notified with *S*. Typhimurium phage type 170 infection in August. Investigation revealed that three of the cases (a father and his 2 children) shared a meal of eggs on toast, prepared with runny yolks, on their day of onset. *S*. Typhimurium phage type 170 was isolated from a rinse of leftover eggs sampled from the home. The 4th case was the grandmother who was likely to have been a secondary case as she had cared for her sick grandchildren and her onset was 6 days after the other cases.

The CDPCU was notified of an outbreak of gastroenteritis at an aged care facility in August. Twenty-one residents and 2 staff members were affected, and 10 were subsequently confirmed as having *S*. Typhimurium phage type 197 infection. Two residents were reported to have died during the outbreak. Onsets ranged over a 16 day period suggesting an ongoing source of contamination. Although it was not able to be proven, it was suspected that one of the blenders was not being effectively cleaned and sanitised after being used to process uncooked foods such as eggs. This potentially contaminated piece of equipment was then used to blend/whip ready to eat food such as cream.

An outbreak of gastroenteritis occurred amongst a group of attendees at a camp facility in August. Forty-one of 55 people who attended the camp were interviewed with a structured questionnaire containing information about foods consumed. Six camp participants developed symptoms of diarrhoea after the camp and two were confirmed with *S*. Typhimurium phage type 9 infection. A cohort analysis did not find any association between consumption of any of the food items and illness. The group self-catered and either prepared food at home prior to travelling to the camp or prepared some foods in the kitchen at the campsite. The menu was vegetarian but there were foods containing eggs that were either eaten uncooked (hedgehog slice) or possibly undercooked (rice balls). No leftover foods were available for testing to assist with finding a source for this outbreak.

An outbreak of diarrhoea occurred amongst 11 residents and 1 staff member of an aged care facility in September. The majority of cases had an onset of illness within a 24-hour period of each other. One faecal specimen was collected, which was positive for *C. perfringens* enterotoxin. Investigators were unable to identify a food source for this outbreak.

Western Australia

There were 6 reported outbreaks of foodborne or suspected foodborne illness during this quarter.

One outbreak resulted in 3 notified cases of *S*. Typhimurium phage type 170, PFGE type 11, in August. The 3 people became ill after eating dinner separately at an Italian restaurant with the duration of illnesses ranging between 6 and 8 days. One case could not recall what food was eaten, 2 cases ate squid and one of these cases ate raw egg aioli with the squid. The median incubation period was 14.5 hours. An environmental investigation did not detect any food handling malpractices and the raw egg aioli was made fresh each day. The eggs used at the restaurant were sourced from two egg producers, one of which had been linked previously to several other raw egg outbreaks due to *S*. Typhimurium phage type 170, PFGE type 11. The implicated egg producer is assisting the Western Australian Department of Health to assess the risk of egg contamination and the identification of any possible system improvements.

In August, seven of 63 residents at an aged care facility had diarrhoea with onsets over a 4 day period and 4 of 7 cases were diagnosed with *S*. Typhimurium phage type 170, PFGE type 11. One case was symptomatic with gastroenteritis when they died from myocardial infarction (cause of death as reported on the death certificate). This type of *S*. Typhimurium has been identified in egg-associated outbreaks linked to food premises in 2009 and 2010, which had been supplied with eggs by one Western Australian producer. However, in this instance the eggs used in the aged care facility were from a different supplier from another jurisdiction. From the information obtained, residents did not consume any raw egg food products. The ill residents lived in three different wings of the facility and it was reported that these residents would not have had contact with each other. The environmental investigation found that the head chef had unsatisfactory hand hygiene and food handling practices. No swabs of the food preparation area were positive for *Salmonella*.

An outbreak of norovirus was investigated amongst 30 of 45 people from a club who became ill after attending a dinner at a reception centre in July. One specimen was collected and was positive for norovirus. The dinner was a buffet with 38 different cold and hot food and drink exposures. Twenty-nine attendees were enrolled in a case control study which showed a significant association between illness and consumption of lasagne (OR 7.2, CI 1.2– 42.6). It was reported that the lasagne was hot when served. Diners reported that the plates used for main meals were dirty underneath. The median incubation period was 47 hours (range 11–60 hours). No staff at

the reception centre reported illness and an environmental investigation found staff had good general knowledge about safe food handling practices.

A suspected foodborne outbreak was investigated at an aged care facility in July. Six of 109 residents were unwell, with diarrhoea only. Illness onsets were over a 24-hour period with symptoms resolving within a day. Cases resided in three different wings of the facility. Two faecal samples were negative for common bacterial and viral pathogens and *C. perfringens,* and the aetiology remained unknown.

A suspected foodborne outbreak was investigated at an aged care facility in September, with 10 of 41 residents experiencing symptoms of diarrhoea only. Illness onsets were over a 12 hour period with symptoms resolving within 1 day. Ill residents lived on four different floors and ate a range of food consistencies (vitamised, soft and normal). Two faecal samples were negative for common bacterial and viral pathogens and *C. perfringens*, and the aetiology of the outbreak remained unknown.

A suspected foodborne norovirus outbreak in September affected 21 of 2,000 people who worked at a defence force base. Two stool specimens were positive for norovirus. Seventeen cases were interviewed, and the common exposure was salad sandwiches prepared in a central mess and consumed for lunch at different locations. Illness onsets ranged between 7.5 and 35 hours after consuming the sandwiches. It was suspected that a salad ingredient contaminated by an infected food handler was the source of illness, although this was not investigated further. After the outbreak, an education session on safe food handling practices was conducted with all food handlers who worked at the base.

Multi-jurisdictional outbreak investigation

A previously reported multi-jurisdictional investigation into an outbreak of *Listeria monocytogenes*[2] was stood down on 13 September 2010, with more than 2 incubation periods since the onset of the last outbreak case on 23 May 2010. The number of outbreak cases remained at nine and the suspected source being rockmelon and/or honeydew melon, eaten fresh or used in the preparation of fruit salads.

Cluster investigations

During the 3rd quarter of 2010, OzFoodNet sites investigated 52 clusters with the majority due to *S.* Typhimurium. Other clusters investigated include *S.* Montevideo, *S.* Infantis, *Campylobacter* and *S.* subspecies I ser 4,12:i:– and *S.* subspecies I ser 4,5:i:–. In forty of the clusters investigated, the causative agent remained unknown.

South Australia investigated a cluster of *S.* Typhimurium, which was untypeable. This investigation followed a notification from the Australian Salmonella Reference Centre of 6 cases confirmed in 1 week and a further case notified subsequently. Five of the 7 cases were aged 3 years or under and four of these children required hospitalisation. Hypothesis generating interviews found a link to a common event for two of the cases and it was suspected that two other cases were also linked to the event. The cluster occurred within a small ethnic community.

Comments

The number of foodborne outbreaks reported during the quarter (n = 34) exceeds the average number during the same quarter over the past 5 years (n = 26.8) but is similar to the number reported during the previous quarter (35).[2]

Of note is that there was a 47.6% increase in *Salmonella* notifications nationally during the quarter compared with the mean notifications for the same quarter between 2005 and 2009. Between 1 July and 30 September 2010, there were 1,373 cases of *Salmonella* nationally by date of diagnosis (a derived field) compared with a 5-year mean of 654 cases. Jurisdictions also reported increased *Salmonella* notifications. In Western Australia, *Salmonella* infections were 29% higher during the quarter compared with the 5-year mean (254 notifications between 1 July and 30 September compared with an average of 202 notifications). This has been attributed entirely to an increase in the number and proportion of infections that were overseas acquired. Of the 159 overseas-acquired *Salmonella* infections in Western Australia during the quarter, 73% (117) reported travel to Indonesia. These cases were commonly of *S.* Enteritidis, *S.* Weltevreden and *S.* Paratyphi bv Java. In Victoria, where *Salmonella* notifications during the quarter were increased by 73% (up to 451 from 261) compared with the 5-year mean, there were increases in the most common *S.* Typhimurium sub-types (phage types 9, 170 and 135) and *S.* Infantis.

During the quarter, the Department of Health and Human Services (DHHS), Tasmania, began sampling for the *Campylobacter* Multi-Locus Sequence Typing Project. The project is a collaboration of the Microbiological Diagnostic Unit (MDU), University of Melbourne; Department of Primary Industries, Parks, Water and Environment, Tasmania; Diagnostic Services, Tasmania; and OzFoodNet. *Campylobacter* spp. are isolated from human clinical cases, food, water and animal faecal specimens, along with obtaining clinical case data through doctor assessment forms and case interviews. From 1 August to 30 September 2010, 93 cases of *Campylobacter* infection were notified. During the

same period, *Campylobacter* spp. had been isolated from 60% of raw chicken samples (48/80), 1.3% of raw red meat samples (1/78), 16% of offal samples (11/67), and 30% of freshwater samples (10/33). All human, food, water and animal isolates will be forwarded to MDU for genotyping at the completion of the sampling period in October 2010.

OzFoodNet conducted a structured audit of the response to the multi-jurisdictional outbreak of locally-acquired hepatitis A that occurred between March 2009 and March 2010. As reported previously, the outbreak was associated with the consumption of semi-dried tomatoes.[3] The audit noted that investigators were able to find the source of the infections quite early and take appropriate actions. This was despite the difficulties of investigating an infection with a long incubation period, and that health departments and epidemiologists were already stretched with the response to 2009 pandemic influenza A (H1N1) between mid-May and late September 2009. National and international communications were important during this investigation, and the outbreak demonstrated the value of the highly functional and established national and international networks (OzFoodNet, the National Food Incident Response Protocol group and the WHO International Food Safety Authorities Network). The audit identified the need for states and territories to maintain a sustained epidemiological workforce and for surge capacity during a multi-jurisdictional outbreak investigation.

OzFoodNet and the Communicable Diseases Network Australia have developed national guidance regarding outbreaks of norovirus and suspected viral gastroenteritis, in response to increasing reports of such outbreaks. The *Guidelines for the Public Health Management of Gastroenteritis Outbreaks Due to Norovirus or Suspected Viral Agents in Australia* are designed to complement existing state and territory guidelines and are available from: http://www.health.gov.au/internet/main/publishing.nsf/content/cda-cdna-norovirus.htm

A limitation of the outbreak data provided by OzFoodNet sites for this report is the potential for variation in categorisation of the features of outbreaks depending on investigator interpretation and circumstances. OzFoodNet continues to standardise and improve practices through its Outbreak Register Working Group and workshops. The National Surveillance Committee, OzFoodNet and the Public Health Laboratory Network continue to work toward harmonisation of *Salmonella* typing practices between jurisdictions that will aid the identification of outbreaks. Changes in the incidence of foodborne outbreaks should be interpreted with caution due to the small numbers each quarter.

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OzFoodNet thanks the investigators in the public health units and state and territory departments of health, as well as public health laboratories and local government environmental health officers who provided data used in this report. We would particularly like to thank reference laboratories for conducting sub-typing of *Salmonella, Listeria* and other enteric pathogens for their continuing work and advice during the quarter.

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2. OzFoodNet Working Group. OzFoodNet quarterly report, 1 April to 30 June 2010. *Commun Dis Intell* 2010;34(3):345–354.
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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 49,583 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 July and 30 September 2010 (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 <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions except South Australia
Syphilis - congenital	All jurisdictions

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

* Notifiable in South Australia as of 1 May 2008.

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

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2010, by date of diagnosis*

Disease	State or territory								Total 3rd quarter 2010†	Total 2nd quarter 2010	Total 3rd quarter 2009	Last 5 years mean 3rd quarter	Ratio‡	Year to date 2010	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	0.2
Hepatitis B (newly acquired)	1	11	1	16	2	2	15	8	56	57	59	65.0	0.9	187	202.6
Hepatitis B (unspecified)	36	760	35	278	75	11	403	196	1,794	1,856	1,773	1,706.8	1.1	5,587	4,969.4
Hepatitis C (newly acquired)	2	14	0	NN	8	1	22	17	64	94	99	98.6	0.6	254	294.6
Hepatitis C (unspecified)	61	1,107	48	690	82	75	638	241	2,942	3,079	2,697	2,920.4	1.0	8,991	8,736.6
Hepatitis D	0	3	0	7	0	0	3	0	13	7	5	9.6	1.4	24	28.2
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	1.0
Campylobacteriosis§	111	NN	36	1,217	447	146	1,126	545	3,628	3,382	3,837	3,695.2	1.0	11,204	11,619.0
Cryptosporidiosis	2	63	9	50	3	15	80	20	242	411	258	296.0	0.8	1,166	2,601.6
Haemolytic uraemic syndrome	0	1	0	1	0	0	1	0	3	1	0	2.4	1.3	7	11.8
Hepatitis A	0	16	1	7	1	3	29	3	60	48	87	66.0	0.9	210	232.6
Hepatitis E	0	4	0	2	0	0	2	0	8	11	8	6.0	1.3	30	25.6
Listeriosis	0	0	0	2	0	0	5	0	7	13	22	14.6	0.5	54	49.2
STEC, VTEC‖	0	4	0	4	5	0	3	2	18	11	23	16.6	1.1	62	69.0
Salmonellosis	25	570	107	398	171	37	449	265	2,022	2,936	1,500	1,375.6	1.5	8,991	6,550.8
Shigellosis	0	29	10	24	19	0	24	23	129	125	121	147.4	0.9	417	511.6
Typhoid	1	4	0	3	2	0	6	2	18	30	25	18.0	1.0	78	67.6
Quarantinable diseases															
Cholera	0	2	0	0	0	0	0	1	3	0	1	0.2	15.0	3	1.8
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0

Table 2: Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2010, by date of diagnosis,* *continued*

Disease	State or territory								Total 3rd quarter 2010†	Total 2nd quarter 2010	Total 3rd quarter 2009	Last 5 years mean 3rd quarter	Ratio‡	Year to date 2010	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶,**	276	4,436	782	4,656	891	483	1,184	2,455	15,163	19,093	15,096	12,827.2	1.2	53,003	39,556.4
Donovanosis	0	0	0	1	0	0	0	0	1	0	0	1.0	1.0	1	4.2
Gonococcal infection**	10	556	557	486	63	3	415	357	2,447	2,610	1,696	1,803.0	1.4	7,401	6,107.8
Syphilis < 2 years duration**	2	61	7	41	7	0	40	16	174	273	346	282.6	0.6	746	846.4
Syphilis > 2 years or unspecified duration**	8	38	22	33	-	3	180	16	300	314	356	351.2	0.9	926	1,011.8
Syphilis - congenital**	0	0	0	1	0	0	0	0	1	1	1	1.0	1.0	3	7.0
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	4	0	3	0	0	1	0	8	6	2	5.2	1.5	17	15.2
Influenza (laboratory confirmed)	65	0	216	1,449	2,809	50	1,344	967	6,900	604	34,836	11,060.6	0.6	7,830	14,259.0
Measles	1	17	1	6	0	0	5	0	30	15	8	4.0	7.5	59	58.6
Mumps	0	6	0	5	1	0	4	1	17	24	34	81.4	0.2	59	220.8
Pertussis	113	1,849	92	2,306	1,861	59	1,511	478	8,269	5,248	6,516	3,813.6	2.2	19,054	10,007.6
Pneumococcal disease (invasive)	8	182	14	102	45	17	154	88	610	423	635	612.0	1.0	1,238	1,239.4
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.3	0.0	0	0.3
Rubella	0	2	0	2	0	0	9	0	13	12	7	11.4	1.1	40	30.8
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.6
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	1	2.2
Varicella zoster (chickenpox)	1	NN	25	153	83	5	61	154	482	319	452	478.5	1.0	1,073	1,080.8
Varicella zoster (shingles)	6	NN	27	28	244	44	94	150	593	680	636	450.8	1.3	2,045	1,373.3
Varicella zoster (unspecified)	24	NN	0	986	109	18	487	235	1,859	1,685	1,586	1,199.8	1.5	5,300	3,467.3
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	8	0	0	3	0	11	1	4	4.6	2.4	15	22.2
Barmah Forest virus infection	0	32	7	157	5	1	4	13	219	382	270	310.2	0.7	1,052	1,398.0
Dengue virus infection	2	42	6	78	5	1	23	130	287	255	91	65.6	4.4	699	442.6
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Kunjin virus infection	0	0	0	0	0	0	0	0	0	1	0	0.2	0.0	2	1.4
Malaria	1	46	1	22	3	0	23	10	106	97	149	156.6	0.7	303	511.2
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	1.8
Ross River virus infection	0	69	73	271	47	1	34	57	552	1,908	832	591.6	0.9	4,067	3,699.2

Table 2: Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2010, by date of diagnosis,* *continued*

Disease	State or territory								Total 3rd quarter 2010†	Total 2nd quarter 2010	Total 3rd quarter 2009	Last 5 years mean 3rd quarter	Ratio‡	Year to date 2010	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	1	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	0	0	6	1	0	0	0	7	3	6	10.2	0.7	16	29.4
Leptospirosis	0	3	0	15	0	0	2	1	21	43	16	17.2	1.2	85	107.4
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	1	0	0	0	0	3	0	4	8	18	29.2	0.1	23	92.0
Q fever	0	21	0	23	2	0	4	2	52	94	66	92.4	0.6	217	286.8
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Other bacterial infections															
Legionellosis	2	16	2	9	5	1	13	14	62	84	74	68.8	0.9	205	229.6
Leprosy	0	0	0	0	0	0	1	1	2	2	1	1.8	1.1	6	7.0
Meningococcal infection††	1	25	2	14	7	1	13	9	72	58	86	115.0	0.6	173	236.8
Tuberculosis	5	69	5	83	9	4	106	33	314	299	349	303.4	1.0	908	839.2
Total	**764**	**10,063**	**2,086**	**13,643**	**7,012**	**981**	**8,524**	**6,510**	**49,583**	**46,603**	**74,684**			**143,833**	

* Date of diagnosis = true onset date, or where not available, the earliest of (i) specimen date, (ii) notification date, or (iii) notification receive date. Hepatitis B and C unspecified were analysed by the notification receive date.

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

‡ Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter. Note: Ratios for varicella zoster (chickenpox), varicella zoster (shingles) and varicella zoster (unspecified) are based on 3 years of data.

§ Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

‖ 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).

†† 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 July to 30 September 2010, 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)	1.1	0.6	1.8	1.5	0.5	1.6	1.1	1.4	1.0
Hepatitis B (unspecified)	41.0	42.8	62.3	25.2	18.5	8.8	29.7	35.0	32.8
Hepatitis C (newly acquired)	2.3	0.8	0.0	NN	2.0	0.8	1.6	3.0	1.5
Hepatitis C (unspecified)	69.5	62.4	85.4	62.6	20.2	59.7	47.0	43.1	53.8
Hepatitis D	0.0	0.2	0.0	0.6	0.0	0.0	0.2	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	126.4	NN	64.0	110.5	110.2	116.2	83.0	97.5	98.2
Cryptosporidiosis	2.3	3.5	16.0	4.5	0.7	11.9	5.9	3.6	4.4
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Hepatitis A	0.0	0.9	1.8	0.6	0.2	2.4	2.1	0.5	1.1
Hepatitis E	0.0	0.2	0.0	0.2	0.0	0.0	0.1	0.0	0.1
Listeriosis	0.0	0.0	0.0	0.2	0.0	0.0	0.4	0.0	0.1
STEC, VTEC‡	0.0	0.2	0.0	0.4	1.2	0.0	0.2	0.4	0.3
Salmonellosis	28.5	32.1	190.4	36.1	42.2	29.4	33.1	47.4	37.0
Shigellosis	0.0	1.6	17.8	2.2	4.7	0.0	1.8	4.1	2.4
Typhoid	1.1	0.2	0.0	0.3	0.5	0.0	0.4	0.4	0.3
Quarantinable diseases									
Cholera	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.2	0.1
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
Sexually transmissible infections									
Chlamydial infection§	314.4	249.9	1,391.2	422.6	219.6	384.4	87.3	439.0	277.3
Donovanosis	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	11.4	31.3	990.9	44.1	15.5	2.4	30.6	63.8	44.7
Syphilis <2 years duration	9.1	2.1	39.1	3.0	-	2.4	13.3	2.9	5.9
Syphilis >2 years or unspecified duration	2.3	3.4	12.5	3.7	1.7	0.0	2.9	2.9	3.2
Syphilis - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.2	0.0	0.3	0.0	0.0	0.1	0.0	0.1
Influenza (laboratory confirmed)	74.0	0.0	384.3	131.5	692.4	39.8	99.0	172.9	126.2
Measles	1.1	1.0	1.8	0.5	0.0	0.0	0.4	0.0	0.5
Mumps	0.0	0.3	0.0	0.5	0.2	0.0	0.3	0.2	0.3
Pertussis	128.7	104.2	163.7	209.3	458.7	47.0	111.4	85.5	151.2
Pneumococcal disease (invasive)	9.1	10.3	24.9	9.3	11.1	13.5	11.3	15.7	11.2
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3: Notification rates of diseases, 1 July to 30 September 2010, by state or territory. (Annualised rate per 100,000 population), *continued*

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, *continued*									
Rubella	0.0	0.1	0.0	0.2	0.0	0.0	0.7	0.0	0.2
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	1.1	NN	44.5	13.9	20.5	4.0	4.5	27.5	13.0
Varicella zoster (shingles)	6.8	NN	48.0	2.5	60.1	35.0	6.9	26.8	16.1
Varicella zoster (unspecified)	27.3	NN	0.0	89.5	26.9	14.3	35.9	42.0	50.3
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.7	0.0	0.0	0.2	0.0	0.2
Barmah Forest virus infection	0.0	1.8	12.5	14.3	1.2	0.8	0.3	2.3	4.0
Dengue virus infection	2.3	2.4	10.7	7.1	1.2	0.8	1.7	23.2	5.2
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.1	2.6	1.8	2.0	0.7	0.0	1.7	1.8	1.9
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	0.0	3.9	129.9	24.6	11.6	0.8	2.5	10.2	10.1
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.5	0.2	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.2	0.0	1.4	0.0	0.0	0.1	0.2	0.4
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.1	0.0	0.0	0.0	0.0	0.2	0.0	0.1
Q fever	0.0	1.2	0.0	2.1	0.5	0.0	0.3	0.4	1.0
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	2.3	0.9	3.6	0.8	1.2	0.8	1.0	2.5	1.1
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.2	0.0
Meningococcal infection‖	1.1	1.4	3.6	1.3	1.7	0.8	1.0	1.6	1.3
Tuberculosis	5.7	3.9	8.9	7.5	2.2	3.2	7.8	5.9	5.7

* Rates are subject to retrospective revision.

† Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

‡ 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 Queensland, which exclude ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

‖ 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.

Laboratory Serology and Virology Reporting Scheme

There were 20.885 reports received by the Laboratory Virology and Serology Reporting Scheme (LabVISE) in the reporting period, 1 July to 30 September 2010 (Tables 4 and 5).

Table 4: Laboratory Virology and Serology reports, 1 July to 30 September 2010 and total reports for the year,* by state or territory†

	State or territory								This period 2010	This period 2009	Year to date 2010	Year to date 2009
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	1	8	2	3	2	-	3	-	19	3	31	47
Mumps virus	-	-	-	2	5	-	1	-	8	9	17	39
Rubella virus	-	1	-	-	-	1	3	-	5	4	23	13
Hepatitis viruses												
Hepatitis A virus	-	-	1	7	1	-	1	1	11	11	45	39
Hepatitis D virus	-	-	-	1	5	-	-	1	7	-	13	14
Hepatitis E virus	-	-	-	-	-	-	-	1	1	2	4	5
Arboviruses												
Ross River virus	-	4	5	65	48	-	1	13	136	143	1,094	837
Barmah Forest virus	-	2	-	27	7	-	-	4	40	30	188	179
Dengue type 1	-	-	4	-	-	-	-	26	30	-	32	-
Dengue type 2	-	-	4	1	-	-	-	33	38	-	38	-
Dengue type 3	-	-	-	-	-	-	-	10	10	-	11	-
Dengue type 4	-	-	-	-	-	-	-	7	7	-	7	-
Dengue not typed	-	-	14	1	-	-	-	113	128	-	129	-
Kunjin virus	-	-	1	-	-	-	-	-	1	-	1	-
Flavivirus (unspecified)	1	18	-	25	1	-	18	-	63	36	183	207
Adenoviruses												
Adenovirus type 40	-	-	-	-	-	-	-	9	9	-	9	-
Adenovirus type 41	-	-	-	-	-	-	-	12	12	-	12	-
Adenovirus not typed/ pending	8	204	4	144	398	2	4	92	856	299	1,381	1,162
Herpes viruses												
Herpes virus type 6	-	1	-	-	-	-	-	1	2	1	4	2
Cytomegalovirus	4	86	-	155	146	5	10	-	406	198	1,126	834
Varicella-zoster virus	4	69	2	603	267	3	5	150	1,103	556	2,813	1,939
Epstein-Barr virus	1	15	41	312	279	1	14	114	777	424	2,333	1,584
Other DNA viruses												
Molluscum contagiosum	-	-	-	-	-	-	-	10	10	-	11	-
Contagious pustular dermatitis (Orf virus)	-	-	-	-	-	-	-	2	2	-	2	-
Parvovirus	-	1	4	51	47	-	17	6	126	48	287	170
Picornavirus family												
Rhinovirus (all types)	2	206	-	1	1,468	1	1	74	1,753	39	1,888	104
Enterovirus type 71 (BCR)	-	-	-	-	-	-	-	1	1	-	1	-
Enterovirus not typed/ pending	-	48	2	5	14	-	-	23	92	20	136	72
Picornavirus not typed	-	-	3	-	-	2	-	202	207	6	215	11
Ortho/paramyxoviruses												
Influenza A virus	6	130	14	286	2,287	3	77	169	2,972	2,959	3,336	6,179
Influenza A virus H1N1	-	3	-	-	-	2	-	2	7	90	7	96
Influenza A virus H3N2	-	-	1	-	-	-	-	34	35	3	35	4
Influenza B virus	-	15	1	36	76	1	3	220	352	126	413	268

Table 4: Laboratory Virology and Serology reports, 1 July to 30 September 2010, and total reports for the year,* by state or territory † *continued*

	State or territory								This period 2010	This period 2009	Year to date 2010	Year to date 2009
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Ortho/paramyxoviruses, *cont'd*												
Newcastle disease virus	-	17	-	-	-	-	-	-	17	-	19	-
Parainfluenza virus type 1	-	4	-	6	56	-	-	10	76	12	177	23
Parainfluenza virus type 2	1	6	-	5	32	-	-	8	52	12	91	81
Parainfluenza virus type 3	-	81	-	38	278	-	1	111	509	157	585	320
Parainfluenza virus typing pending	-	-	-	-	-	1	-	-	1	1	1	2
Respiratory syncytial virus	2	421	2	99	737	19	2	350	1,632	572	3,088	2,516
Paramyxovirus (unspecified)	-	42	-	-	-	-	-	-	42	-	42	-
Other RNA viruses												
HTLV-1	-	-	-	-	19	-	-	2	21	-	66	142
Rotavirus	2	122	2	-	327	4	2	232	691	34	887	185
Calicivirus	-	-	7	-	-	-	-	218	225	-	226	-
Norwalk agent	-	43	-	-	302	-	-	-	345	46	924	78
Other												
Chlamydia trachomatis not typed	2	418	3	2,029	629	17	6	699	3,803	1,833	10,082	6,392
Chlamydia pneumoniae	-	-	-	-	-	-	-	2	2	3	33	9
Chlamydia psittaci	-	-	-	1	-	-	10	-	11	17	25	56
Chlamydia spp typing pending	-	18	-	-	-	-	-	-	18	10	31	16
Chlamydia species	-	-	-	-	-	-	3	-	3	1	5	8
Mycoplasma pneumoniae	1	11	16	66	239	2	103	214	652	249	1,207	823
Mycoplasma hominis	-	5	-	-	-	-	-	-	5	5	5	9
Coxiella burnetii (Q fever)	2	17	-	22	12	-	7	2	62	56	183	172
Orientia tsutsuganushi	-	-	-	1	-	-	1	2	4	-	4	-
Rickettsia - spotted fever group	-	4	-	15	-	1	-	2	22	23	61	99
Rickettsia spp - other	-	-	-	-	-	-	-	3	3	-	4	-
Streptococcus group A	-	7	-	179	-	-	51	1	238	164	640	479
Brucella abortus	-	-	-	-	2	-	-	-	2	-	2	1
Brucella species	-	1	-	10	-	-	-	-	11	2	15	11
Bordetella pertussis	4	51	-	831	1,496	-	126	101	2,609	620	5,775	3,554
Legionella pneumophila	-	1	-	4	2	-	8	2	17	6	29	26
Legionella longbeachae	-	-	1	-	-	-	-	8	9	1	17	11
Legionella species	-	2	-	2	-	-	5	2	11	9	33	22
Cryptococcus species	-	5	-	5	5	-	-	-	15	7	40	28
Histoplasma capsulatum	-	-	-	-	-	-	1	-	1	-	1	-
Leptospira species	-	-	-	6	3	-	-	-	9	8	33	33
Treponema pallidum	-	59	-	298	135	-	14	8	514	332	1,501	1,294
Entamoeba histolytica	-	-	-	2	-	-	-	2	4	5	10	6
Toxoplasma gondii	-	1	-	3	5	-	4	-	13	4	37	15
Echinococcus granulosus	-	-	-	-	3	-	3	4	10	-	15	14
Total	41	2,147	134	5,347	9,333	65	505	3,313	20,885	9,196	41,719	30,230

* Data presented are for reports with report dates in the current period.

† State or territory of postcode, if reported, otherwise state or territory of reporting laboratory.

– No data received this period.

Table 5: Laboratory Virology and Serology reports, 1 July to 30 September 2010,* by laboratory

State or territory	Laboratory	July 2010	August 2010	September 2010	Total
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	219	271	336	826
	New Children's Hospital, Westmead	189	125	200	514
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	19	30	32	81
	South West Area Pathology Service, Liverpool	176	111	132	419
Queensland	Queensland Medical Laboratory, West End	1,813	1,902	2,015	5,730
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	2,091	2,683	4,548	9,322
Tasmania	Northern Tasmanian Pathology Service, Launceston	14	26	17	57
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Australian Rickettsial Reference Laboratory	11	8	9	28
	Monash Medical Centre, Melbourne	–	–	–	–
	Royal Children's Hospital, Melbourne	86	59	74	219
	Victorian Infectious Diseases Reference Laboratory	66	63	86	215
Western Australia	PathWest Virology, Perth	942	1,115	1,255	3,312
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	17	75	70	162
Total		5,643	6,468	8,774	20,885

* The complete list of laboratories reporting for the 12 months, January to December 2010, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– No data received this period.

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 5 years of age, for 3-month birth cohorts of children at the stated ages between 1 April and 30 June 2010. '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-containing (MMR) vaccine. 'Fully immunised' at 5 years 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;22:36–37.*

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, E-mail: brynleyh@chw.edu.au

The percentage of children 'fully immunised' at 12 months of age for Australia increased slightly by 0.2 percentage points to 91.7% (Table 1). There were no important changes in coverage for any individual vaccines due at 12 months of age or by jurisdiction.

The percentage of children 'fully immunised' at 24 months of age for Australia increased by 0.3 percentage points 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' at 5 years of age for Australia decreased slightly by 0.5 percentage points, to sit currently at 89.1% (Table 3). There were no important changes in coverage for any individual vaccines due at 5 years 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 6 years (till December 2007). This trend continued when the age of coverage calculation was changed from 6 years to 5 years in March 2008, and then increased further in the previous quarter as outlined in the previous report.

Table 1. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 April to 30 June 2009; assessment date 30 September 2010

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,233	24,054	1,031	15,792	4,862	1,622	17,551	7,825	73,970
Diphtheria, tetanus, pertussis (%)	94.7	91.8	91.0	92.3	92.2	92.9	92.8	90.7	92.1
Poliomyelitis (%)	94.7	91.7	90.9	92.3	92.1	92.9	92.8	90.6	92.1
Haemophilus influenzae type b (%)	94.6	91.6	91.8	92.3	92.0	92.8	92.6	90.5	92.0
Hepatitis B (%)	94.2	91.5	90.7	92.1	91.7	92.8	92.3	90.3	91.8
Fully immunised (%)	94.2	91.4	90.5	92.1	91.6	92.7	92.2	90.3	91.7
Change in fully immunised since last quarter (%)	+2.0	+0.1	+0.2	+0.2	+0.3	+1.1	+0.1	+0.2	+0.2

Table 2. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 April to 30 June 2008; assessment date 30 September 2010*

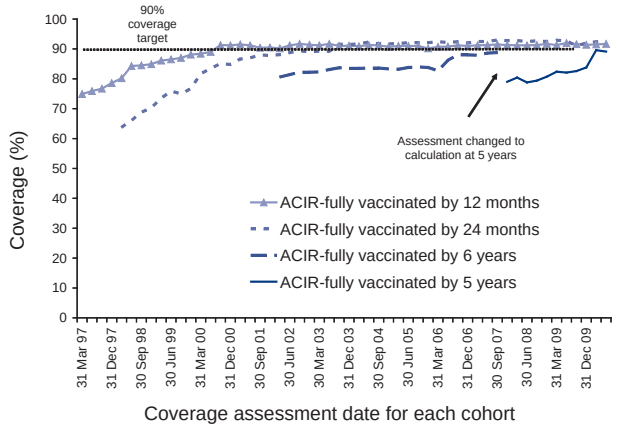
Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,160	23,847	944	15,723	4,813	1,589	17,323	7,840	73,239
Diphtheria, tetanus, pertussis (%)	97.2	95.0	96.7	94.8	94.6	95.0	95.7	94.2	95.1
Poliomyelitis (%)	97.1	94.9	96.6	94.8	94.6	95.0	95.6	94.2	95.0
Haemophilus influenzae type b (%)	96.8	95.1	92.7	94.8	94.6	95.1	95.4	93.5	94.9
Measles, mumps, rubella (%)	96.3	93.7	96.3	94.2	93.7	94.7	94.6	93.2	94.1
Hepatitis B (%)	96.1	94.5	96.5	94.3	94.2	95.0	95.1	93.5	94.6
Fully immunised (%)	94.9	92.4	92.0	92.8	92.4	93.8	93.4	90.3	92.6
Change in fully immunised since last quarter (%)	+1.1	-0.0	-1.5	+0.6	-0.0	+1.0	+0.3	-0.2	+0.2

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2009;33(4):444.

Table 3. Percentage of children immunised at 5 years of age, preliminary results by disease and state or territory for the birth cohort 1 April to 30 June 2005; assessment date 30 September 2010

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,103	23,476	950	15,489	4,663	1,571	17,003	7,426	71,681
Diphtheria, tetanus, pertussis (%)	91.1	89.5	86.3	90.6	87.2	93.2	90.9	86.1	89.6
Poliomyelitis (%)	91.2	89.5	86.3	90.5	87.2	93.1	90.9	86.0	89.6
Measles, mumps, rubella (%)	91.1	89.3	85.8	90.6	87.1	93.3	90.7	86.0	89.5
Fully immunised (%)	90.7	89.0	85.4	90.0	86.7	92.7	90.4	85.3	89.1
Change in fully immunised since last quarter (%)	+1.7	-0.5	-2.0	-0.2	-0.4	+2.1	-0.7	-1.3	-0.5

Figure: Trends in vaccination coverage, Australia, 1997 to 30 April 2010, 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.

In June 2010, ASPREN's laboratory ILI testing was implemented, allowing for viral testing of 25% of ILI

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

The list of conditions is reviewed annually by the ASPREN management committee. In 2010, 4 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 *2010;34(1):83–84.*

Reporting period 1 July to 30 September 2010

Sentinel practices contributing to ASPREN were located in all 8 jurisdictions in Australia. A total of 120 general practitioners contributed data to ASPREN in the 3rd quarter of 2010. Each week an average of 101 general practitioners provided information to ASPREN at an average of 9,821 (range 9,194–10,857) consultations per week and an average of 208 (range 149–292) notifications per week.

ILI rates reported from 1 July to 30 September 2010 averaged 14 cases per 1,000 consultations (range 9–19 cases per 1,000 consultations). The reported rates in July and August 2010 (10–11 cases per 1,000 consultations and 9–18 cases per 1,000 consultations, respectively) were significantly lower compared with rates in the same reporting period in 2009 (22–44 cases per 1,000 consultations and 21–45 cases per 1,000 consultations, respectively). ILI rates reported in September 2010 (15–19 cases per 1,000 consultations) were slightly higher than rates recorded in September 2009 (6–18 cases per 1,000 consultations) (Figure 1).

ILI swab testing commenced at the beginning of June 2010. The most commonly reported virus during this reporting period was influenza A H1N1(2009) (21% of all swabs performed), with rhinovirus the 2nd most commonly reported (9% of all swabs performed) (Figure 2). For the whole of

Figure 1: Consultation rates for influenza-like illness, ASPREN, 1 January 2009 to 30 September 2010, by week of report

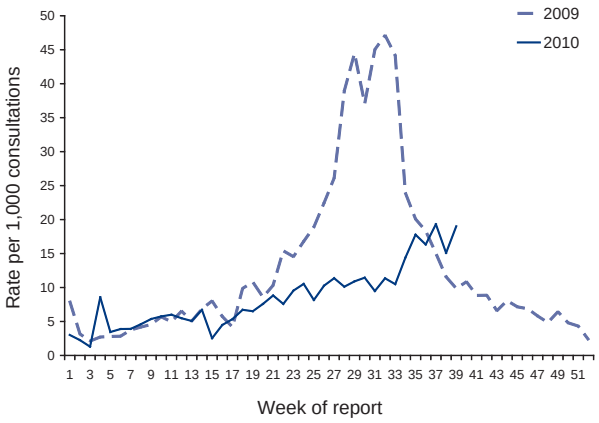
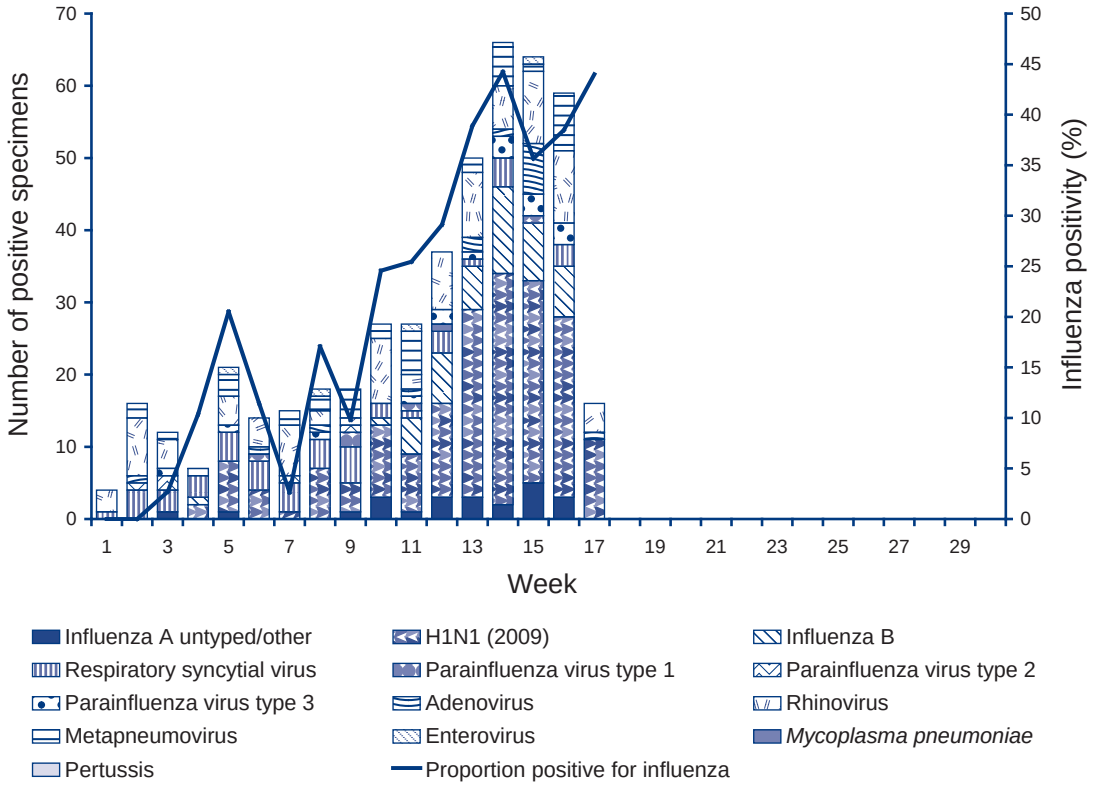


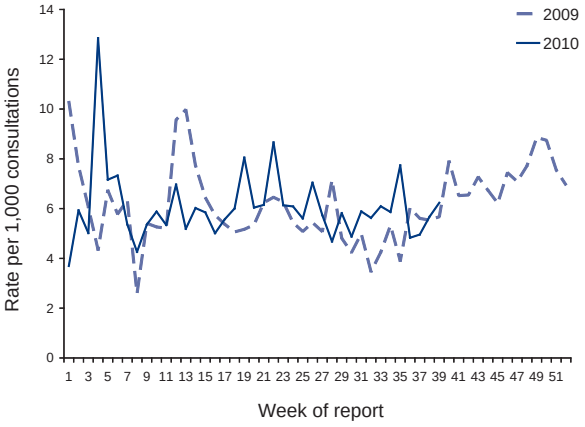
Figure 2: Influenza-like illness swab testing results, ASPREN, 1 June 2010 to 30 September 2010, by week of report



2010 to the end of week 39, 248 cases of influenza have been detected, the majority of these being H1N1(2009) (72% of all swabs performed) and the remainder were influenza B (19% of all swabs performed) and influenza A untyped or other (9% of all swabs performed).

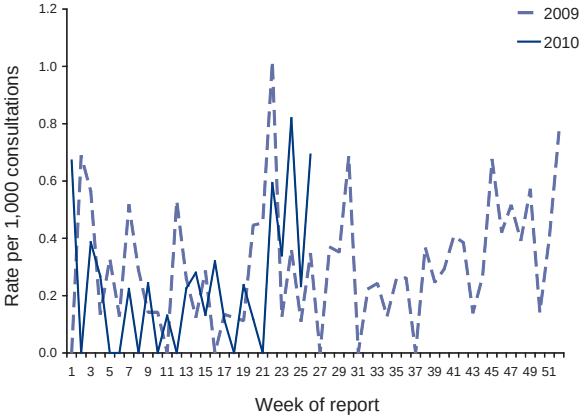
During this reporting period, consultation rates for gastroenteritis averaged 5.7 cases per 1,000 consultations (range 5–8 cases per 1,000, Figure 3). This was slightly higher compared with the same reporting period in 2009 where the average was 5.1 cases per 1,000 consultations (range 4–9 cases per 1,000).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 1 January 2009 to 30 September 2010, by week of report



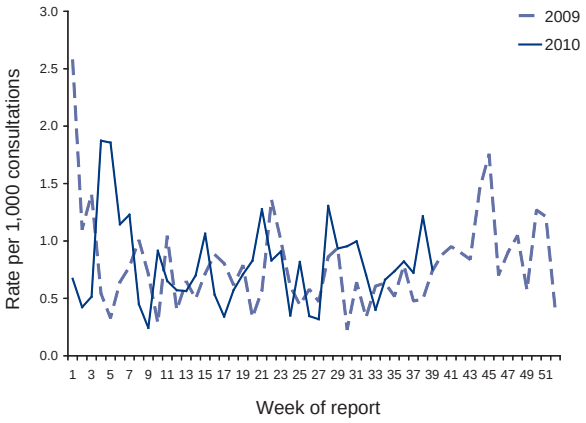
Varicella infections were reported at a slightly higher rate for the 3rd quarter of 2010 compared with the same period in 2009. From 1 July to 30 September 2010, recorded rates for chickenpox averaged 0.4 cases per 1,000 consultations (range 0.1–0.7 cases per 1,000 consultations, Figure 4).

Figure 4: Consultation rates for chickenpox, ASPREN, 1 January 2009 to 30 September 2010, by week of report



In the 3rd quarter of 2010, reported rates for shingles averaged 0.8 cases per 1,000 consultations (range 0.3–1.2 cases per 1,000 consultations, Figure 5), which was higher than the same reporting period in 2009 where the average shingles rate was 0.6 cases per 1,000 consultations (0.2–0.9 cases per 1,000 consultations).

Figure 5: Consultation rates for shingles, ASPREN, 1 January 2009 to 30 September 2010, by week of report



Gonococcal surveillance

Monica Lahra and John Tapsall, 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 on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[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 program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see* Commun Dis Intell *2010;34(1):82–83.*

Reporting period 1 April to 30 June 2010

The AGSP laboratories received a total of 1,027 isolates in the 2nd quarter of 2010, which was an increase from the 796 isolates seen in the corresponding period in 2009. Of these, 1,011 remained viable for susceptibility testing. Of the total, 328 (32%) were from New South Wales, 232 (23%) from Victoria, 224 (22%) from Queensland, 93 (9%) from the Northern Territory, 87 (8.5%) from Western Australia and 52 (5%) from South Australia. There were 11 isolates from the Australian Capital Territory and no isolates from Tasmania. The number of isolates examined in this quarter in New South Wales, Queensland, South Australia and the Australian Capital Territory was increased, the number from Victoria was similar, and there was a decline in numbers examined in Western Australia and the Northern Territory.

Penicillins

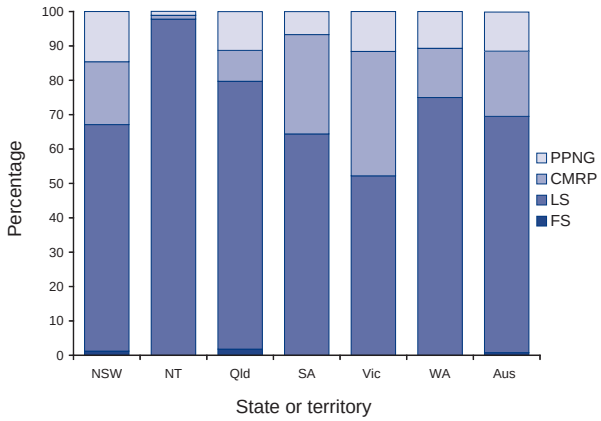
In the 2nd quarter of 2010, 307 (30%) of all isolates examined were penicillin resistant by one or more mechanisms, which was proportionally similar to the 34% reported in the same quarter in 2009. One hundred and ninety-two (19%) were resistant by chromosomal mechanisms, (CMRP) and 115 (11%) were penicillinase-producing *Neisseria gonorrhoeae* (PPNG). When compared with the same quarter in 2009, the proportion of CMRP (14%) and PPNG (20%) were also similar.

The proportion of all strains resistant to the penicillins by any mechanism ranged widely across all jurisdictions: Northern Territory 2.3%; Queensland 20%; Western Australia 25%; New South Wales 33%; South Australia 36% and Victoria 48%. There were 11 isolates from the Australian Capital Territory for this quarter and four were penicillin resistant. Of note, there was a decline in the proportion of penicillin resistance in New South Wales from the same quarter in 2009, from 49% to 33% in 2010. This decline was due to a reduction in CMRP from 30% to 18%.

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

Penicillin resistance due to CMRP predominated in isolates from Victoria (CMRP 36%: PPNG 12%), South Australia (CMRP 29%: PPNG 7%), New South Wales (CMRP 18%: PPNG 15%) and Western Australia (CMRP 14%: PPNG 11%). However, PPNG were more prominent in Queensland (PPNG 11%: CMRP 9%). There was 1 CMRP and 1 PPNG detected in the Northern Territory, and in the Australian Capital Territory there were 2 CMRP and 2 PPNG.

Figure 1: Categorisation of gonococci isolated in Australia, 1 April to 30 June 2010, 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*.

Ceftriaxone

Existing criteria: Decreased susceptibility to ceftriaxone (MIC range 0.06–0.12mg/L)

Fifty-five isolates (5.4%) with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected nationally, which was a marked increase when compared with the same quarter in 2009 or the low numbers reported in previous years. There were 22 (6.7%) isolates in New South Wales, 12 (5.2%) in Victoria, 10 (22%) in South Australia, 9 (4%) in Queensland and one each from Western Australia, and the Australian Capital Territory.

This increase in gonococci showing decreased susceptibility to ceftriaxone was reported initially by the AGSP in the 1st quarter of 2010 when there were 62 isolates (6%) with MICs in the range 0.06–0.12 mg/L. This proportion remains similar nationally for the 2nd quarter of 2010.

New criteria: Decreased Susceptibility to ceftriaxone (MIC range 0.03–0.12 mg/L)

Whilst decreased susceptibility to ceftriaxone is yet to be associated with treatment failure in genital

infection, it is both increasing and of increasing concern globally. To better monitor this, the criteria for detecting gonococci with decreased susceptibility to ceftriaxone has been adjusted to include MIC ≥ 0.03mg/L.[2]

In this quarter, data for ceftriaxone MIC ≥ 0.03 mg/L were contributed from four jurisdictions (New South Wales; Queensland; Victoria and Western Australia) with 866 isolates examined. Using the new criteria (MIC range 0.03–0.12 mg/L), 164 isolates (19% of 866 gonococci) were detected. There were 72 (22%) in New South Wales, 48 (21%) in Victoria, 33 (15%) in Queensland and 11 (13%) in Western Australia.

From the 1st quarter of 2011 AGSP reports will report gonococci with a ceftriaxone MIC range 0.03–0.12mg/L as having decreased susceptibility.

Spectinomycin

All isolates were susceptible to this injectable agent.

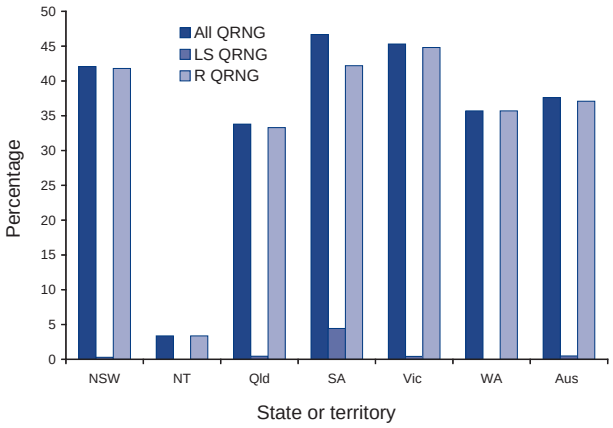
Quinolone antibiotics

Quinolone resistant *N. gonorrhoeae* (QRNG) are defined as those isolates with an 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 a total of 380 (QRNG) in this quarter for 2010, representing 38% of all gonococci tested nationally. The proportion of QRNG was reduced when compared with the corresponding quarter in 2009: 44%, and 2008: 59%.

The majority of QRNG in the current period exhibit higher-level resistance (ciprofloxacin MICs ≥ 1mg/L). QRNG were detected in all states and territories with the highest proportions in South Australia: 21 QRNG (47% of all isolates); Victoria: 105 QRNG (45% of all isolates); New South Wales 138 QRNG (42% of all isolates) (Figure 2). In Western Australia there were 30 QRNG (36%), and in Queensland there were 75 QRNG (34%). There were 8 QRNG isolates from the Australian Capital Territory, and three from the Northern Territory.

Figure 2: The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 April to 30 June 2010, by state or territory



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.

R QRNG Ciprofloxacin MICs ≥1 mg/L.

High level tetracycline resistance

There were 218 (22%) high level tetracycline resistance (TRNG) detected nationally, which was proportionally unchanged from this same quarter in 2009 (165 TRNG: 21%). The highest proportions of TRNG in any jurisdiction were reported from the Northern Territory (37 TRNG: 42%), and the Australian Capital Territory (4 TRNG: 37%). The number and proportion in the other jurisdictions were New South Wales (83 TRNG: 25%); Western Australia (TRNG 18: 21%); Victoria (TRNG 36: 16%); South Australia (TRNG 7: 16%) and Queensland (TRNG 33: 15%)

Reference

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.
2. University of New South Wales. The CDS Antibiotic Susceptibility Test. Available from: http://web.med.unsw.edu.au/cdstest/

HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. 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 three 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/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, CFI Building, Cnr Boundary and West Streets, Darlinghurst NSW 2010. Internet: www.nchecr.unsw.edu.au Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see Commun Dis Intell *2010;34(1):84.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 October to 31 December 2009, are included in this issue of Communicable Diseases Intelligence *(Tables 1 and 2).*

Table 1: New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 October to 31 December 2009, 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 2009	This period 2008	YTD 2009	YTD 2008
HIV diagnoses	Female	0	11	0	2	5	1	6	6	31	28	139	137
	Male	1	83	2	47	10	0	67	11	221	208	909	864
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	1	94	2	49	15	1	73	17	252	236	1,050	1,001
AIDS diagnoses†	Female	0	--	0	1	0	0	2	1	4	3	13	9
	Male	0	--	0	2	4	0	6	1	13	23	77	95
	Total*	0	--	0	3	4	0	8	2	17	26	90	104
AIDS deaths†	Female	0	--	0	0	0	0	0	0	0	0	2	1
	Male	0	--	0	0	0	0	1	0	1	10	7	25
	Total*	0	--	0	0	0	0	1	0	1	10	9	26

* Totals include people whose sex was reported as transgender.

† AIDS cases and deaths following AIDS occurring in New South Wales from January 2008 are not included.

Table 2: Number of new diagnoses of HIV infection since the introduction of HIV antibody testing 1985, and number of new diagnoses of AIDS and deaths following AIDS since 1981, cumulative to 31 December 2009, by sex and state or territory

		State or territory								Aust
	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
HIV diagnoses	Female	36	1,010	30	355	125	17	465	263	2,301
	Male	283	14,480	158	3,251	1,066	127	6,012	1,396	26,773
	Not reported	0	228	0	0	0	0	22	0	250
	Total*	319	15,750	188	3,615	1,192	144	6,521	1,666	29,395
AIDS diagnoses†	Female	10	265	6	77	32	4	126	48	568
	Male	95	5,513	48	1,096	426	55	2,151	458	9,842
	Total*	105	5,796	54	1,175	459	59	2,290	508	10,446
AIDS deaths†	Female	7	138	1	43	20	2	66	30	307
	Male	73	3,597	33	679	280	34	1,449	301	6,446
	Total*	80	3,746	34	724	300	36	1,524	332	6,776

* Totals include people whose sex was reported as transgender.

† AIDS cases and deaths following AIDS occurring in New South Wales from January 2008 are not included.

Meningococcal surveillance

Monica Lahra and John Tapsall, The Prince of Wales Hospital, Randwick, NSW, 2031 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 is published in Communicable Diseases Intelligence. For more information see* Commun Dis Intell *2010;34:83.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 April to 30 June 2010 and 1 July to 30 September 2010, are included in this issue of Communicable Diseases Intelligence *(Tables 1 and 2).*

Table 1: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2010, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD
Australian Capital Territory	10			1	1	0	0	0	0	0	0	0	0	1	1
	09			3	3	0	0	0	0	0	0	0	0	3	3
New South Wales	10			8	21	2	2	0	0	1	2	1	2	12	27
	09			13	25	1	4	1	1	1	2	3	3	19	35
Northern Territory	10			0	0	0	0	0	0	0	0	0	0	0	0
	09			1	3	0	1	0	0	0	0	0	0	1	4
Queensland	10			17	23	1	1	0	0	1	1	0	0	19	25
	09			6	17	0	0	0	0	0	0	0	0	6	17
South Australia	10			6	10	0	0	0	1	0	0	0	0	6	11
	09			7	11	0	0	1	1	0	0	0	0	8	12
Tasmania	10			0	1	0	0	0	0	0	0	0	1	0	2
	09			1	1	0	0	0	0	0	0	0	0	1	1
Victoria	10			7	10	0	0	1	2	2	3	0	0	10	15
	09			5	10	0	1	0	0	0	0	0	2	5	13
Western Australia	10			2	5	0	1	1	1	0	0	0	0	3	7
	09			8	10	0	2	0	0	0	0	0	0	8	12
Total	10			41	71	3	4	2	4	4	6	1	3	51	88
	09			44	80	1	8	2	2	1	2	3	5	51	97

Table 2: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 July to 30 September 2010, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD
Australian Capital Territory	10			1	2	0	0	0	0	0	0	0	0	1	2
	09			0	3	0	0	0	0	0	0	0	0	0	3
New South Wales	10			14	35	2	4	2	2	0	2	2	4	20	47
	09			24	49	3	7	2	3	2	4	0	3	31	66
Northern Territory	10			0	0	0	0	0	0	0	0	0	0	0	0
	09			0	3	0	1							0	4
Queensland	10			31	48	4	5	0	0	1	2	0	0	36	55
	09			19	36	0	0	1	1	0	0	2	2	22	39
South Australia	10			6	16	0	0	0	1	0	0	0	0	6	17
	09			4	15	0	0	1	2	0	0	0	0	5	17
Tasmania	10			0	1	0	0	0	0	0	0	0	1	0	2
	09			0	1	0	0	0	0	0	0	0	0	0	1
Victoria	10			12	32	0	0	0	2	0	3	0	0	12	37
	09			13	23	0	1					1	3	14	27
Western Australia	10			8	13	0	1	0	1	1	1	0	0	9	16
	09			6	16	0	2	1	1	0	0	0	0	7	19
Total	10			73	147	6	10	2	6	3	8	2	5	86	176
	09			66	146	3	11	5	7	2	4	3	8	79	176

Index to *Communicable Diseases Intelligence*, 2010

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Reviewers for *Communicable Diseases Intelligence* 2010

The *CDI* staff wish to thank the following reviewers for their valued assistance throughout the year.

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