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Annual reports

Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet network, 2010

The OzFoodNet Working Group

Abstract

This report summarises the incidence of diseases potentially transmitted by food in Australia and details outbreaks associated with food in 2010. OzFoodNet sites reported 30,035 notifications of 9 diseases or conditions that are commonly transmitted by food. The most frequently notified infections were *Campylobacter* (16,968 notifications) and *Salmonella* (11,992 notifications). The most frequently notified *Salmonella* serotype was *Salmonella* Typhimurium, accounting for 44% of all *Salmonella* notifications. OzFoodNet sites also reported 1,640 outbreaks of gastrointestinal illness affecting 30,193 people and resulting in 722 people being hospitalised. There were 89 deaths associated with these outbreaks. The majority of outbreaks (81%, 1,330/1,640) were due to person-to-person spread, 9% (154/1,640) were suspected or confirmed to have been transmitted by contaminated food, 9% (155/1,640) had an unknown mode of transmission and 1 outbreak was due to transmission from animal to person. Foodborne and suspected foodborne outbreaks affected 2,146 persons and included 157 hospitalisations. Fifteen deaths were reported during these outbreaks. *Salmonella* was the most common aetiological agent identified in foodborne outbreaks and restaurants were the most frequently reported food preparation setting. A single food source was identified for 43 outbreaks, 21 of which were associated with the consumption of dishes containing raw or minimally cooked eggs; the majority (n=20) due to *S.* Typhimurium. These data assist agencies to document sources of foodborne disease, develop food safety policies, and prevent foodborne illness. *Commun Dis Intell* 2012;36(3):E213–E241.

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 inform 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 interventions.[3,4]

Most foodborne diseases manifest as mild self-limiting gastroenteritis, with 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]

Public Health authorities use surveillance data to detect outbreaks and clusters of disease. Trends in surveillance data also contribute to the assessment of the efficacy of public health interventions.[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 also transmitted by the faecal–oral route and as such may be transmitted by contact with infected animals or people. They may also be transmitted by contaminated food or surfaces, or through the consumption of contaminated water. In addition, health departments collect summary data on notified outbreaks of foodborne diseases, providing 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 food-borne disease outbreaks. The OzFoodNet network includes collaborators from the Public Health Laboratory Network, Food Standards Australia New Zealand, 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, which is Australia's peak body for communicable disease control.[9] This is the 10th annual report for the OzFoodNet network and summarises the 2010 surveillance data including a comparison with data from previous years.

Methods

Population under surveillance

In 2010, the network covered the whole of the Australian population, which was estimated to be 22,342,398 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 cases 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*.[11] In 2010, OzFoodNet aggregated and analysed data from NNDSS and enhanced surveillance data from OzFoodNet sites on the following 9 diseases or conditions, which are commonly transmitted by food:

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

There may be differences when comparing OzFoodNet enhanced data state totals and NNDSS derived notifications. This is due to amendments to notification totals by states and territories after the date of data extraction. Also, some jurisdictions report on notification date rather than onset date. Data for this report were extracted from NNDSS in November 2011 and were analysed by the date of diagnosis within the reporting period 1 January to 31 December 2010. Date of diagnosis was derived for each case from the earliest date supplied by the jurisdiction, which could be 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 2010 were used to calculate rates of notified infections.[12]

Enhanced surveillance for listeriosis

Commencing in 2010, OzFoodNet collected enhanced surveillance data on all notified cases of listeriosis in Australia. This enhanced surveillance system adds to the routinely collected data within NNDSS. It is a centralised national database that includes detailed information regarding the characterisation of *Listeria monocytogenes* isolates by molecular subtyping methods, food histories and exposure data on all notified listeriosis cases in Australia. The overall aim of this enhanced surveillance is to enable timely detection of illness and subsequent public health response. Local public health unit staff interview all cases with a standard national listeriosis questionnaire. Interviews are conducted as individual cases are reported to improve accurate recall of foods consumed during the incubation period. Data are collated nationally via an online open-source database using NetEpi Case Manager. This is a secure web-based reporting system used by OzFoodNet epidemiologists for the enhanced surveillance of listeriosis and multi-jurisdictional outbreaks in Australia. NetEpi allows data to be entered from multiple sites and promotes nationally consistent data collection and analysis by OzFoodNet epidemiologists.

Supplementary surveillance

OzFoodNet sites collected supplementary data on infections commonly transmitted by food. Information on travel status was collected for cases of *Salmonella* Enteritidis, hepatitis A, *Shigella* and typhoid.

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 pulsed-field gel electrophoresis (PFGE) is used for typing *S.* Typhimurium in that state, and 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 by a health department.

Gastrointestinal and foodborne disease outbreaks

OzFoodNet sites collected summary information on gastrointestinal and foodborne disease outbreaks that occurred in Australia during 2010. 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 analytical epidemiological and/or microbiological evidence implicated 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 descriptive epidemiological evidence implicated the meal or food as the suspected source of illness, including outbreaks where food-to-person-to-food transmission is involved. 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 has been combined for the analysis. Information collected on each outbreak included the setting where the outbreak occurred, where the 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 agent, food vehicle and the setting 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 2010, OzFoodNet sites reported 30,035 notifications of 9 diseases or conditions that are commonly transmitted by food (Table 1), which is an increase compared with the mean of 26,190 notifications per year for the previous 5 years (2005–2009).

Salmonella infections

In 2010, OzFoodNet sites reported 11,992 cases of *Salmonella* infection, a rate of 53.7 cases per 100,000. This was an increase compared with the mean rate for the previous 5 years (41.8 cases per 100,000). Notification rates ranged from 40.6 cases per 100,000 in South Australia to 249.5 cases per 100,000 in the Northern Territory, which usually has the highest rate of salmonellosis. Approximately half (48.7%) of salmonellosis notifications were in males.

Nationally during 2010, the most commonly notified *Salmonella* serotype was *S.* Typhimurium, which was responsible for approximately 44% (5,241/11,992) of all notified *Salmonella* infections (Table 2). The serotype with the largest percentage increase was *S.* Infantis with 2.2 times more notifications nationally in 2010 than in 2009.

Completeness of Salmonella *serotyping and phage typing*

Overall, 97.2% (11,651/11,992) of *Salmonella* notifications contained information about serotype. OzFoodNet monitors the completeness of 6 serotypes that are routinely phage typed: Bovismorbificans; Enteritidis; Hadar; Heidelberg; Typhimurium; and Virchow, in those jurisdictions participating in this typing scheme. In 2010, phage typing was greater than 90% complete for only *S.* Enteritidis (Table 3), and across all 6 serotypes, completeness declined from 93.8% in 2009 to 86.3% in 2010.

Salmonella Enteritidis

S. Enteritidis is a globally 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 2010, OzFoodNet sites reported 835 cases of *S.* Enteritidis infection (Table 4). Travel histories were obtained for 94.9% of cases in 2010 (792/835), compared with 75% of cases in 2009 (443/591). Of those cases where travel status was reported, 92.9% (736/792) had travelled overseas and cases often reported visiting several countries. Western Australia reported the highest number of notified cases compared with other jurisdictions in 2010 and infection was mainly acquired overseas. Queensland reported the largest number of locally-acquired cases.

Of the cases that were known to have been acquired overseas, 84.9% (625/736) reported travel to South East Asia. Similarly to previous years, the most common country of acquisition for overseas-acquired infections was Indonesia, with 64.3% (473/736) of cases reporting travel there. Malaysia was the second most common country of acquisition with 7.9% (58/736) of all notifications that were known to have been acquired overseas, followed by Thailand with 6.9% (51/736).

Table 1: Number of notified cases, crude rate and 5-year mean (2005–2009) rate per 100,000 of diseases or infections commonly transmitted by food, Australia, 2010, by disease and state or territory

Disease		State or territory								Aust
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Salmonella	Notified cases, 2010	212	3,813	573	2,928	668	236	2,284	1,278	11,992
	Crude rate, 2010	59.1	52.7	249.5	64.8	40.6	46.5	41.2	55.7	53.7
	Mean rate, 2005–2009	40.7	34.3	213.9	57.9	42.6	44.1	30.7	43.0	41.8
*Campylobacter**	Notified cases, 2010	552	NN	166	4,789	1,770	726	6,641	2,324	16,968
	Crude rate, 2010	153.8	NN	72.3	106.0	107.6	143.0	119.7	101.2	112.3
	Mean rate, 2005–2009	121.8	NN	117.7	105.9	139.4	129.0	113.7	102.9	113.4
Listeria	Notified cases, 2010	2	26	0	9	1	3	27	3	71
	Crude rate, 2010	0.6	0.4	0.0	0.2	0.1	0.6	0.5	0.1	0.3
	Mean rate, 2005–2009	0.4	0.4	0.0	0.2	0.3	0.2	0.3	0.4	0.3
Shigella	Notified cases, 2010	7	118	75	93	54	5	87	113	552
	Crude rate, 2010	2.0	1.6	32.7	2.1	3.3	1.0	1.6	4.9	2.5
	Mean rate, 2005–2009	1.2	1.6	70.1	2.3	4.3	0.6	1.9	6.4	3.1
Typhoid	Notified cases, 2010	2	30	2	21	5	1	24	11	96
	Crude rate, 2010	0.6	0.4	0.9	0.5	0.3	0.2	0.4	0.5	0.4
	Mean rate, 2005–2009	0.1	0.5	0.6	0.2	0.2	0.2	0.5	0.4	0.4
Hepatitis A	Notified cases, 2010	5	83	3	41	4	4	95	32	267
	Crude rate, 2010	1.4	1.1	1.3	0.9	0.2	0.8	1.7	1.4	1.2
	Mean rate, 2005–2009	1.0	1.2	9.6	1.1	1.3	0.6	2.0	1.9	1.5
Shiga toxin-producing *Escherichia coli*	Notified cases, 2010	0	10	0	17	33	0	12	8	80
	Crude rate, 2010	0.0	0.1	0.0	0.4	2.0	0.0	0.2	0.3	0.4
	Mean rate, 2005–2009	0.1	0.3	0.6	0.5	2.7	0.1	0.2	0.2	0.5
Haemolytic uraemic syndrome	Notified cases, 2010	0	3	0	3	0	0	3	0	9
	Crude rate, 2010	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0
	Mean rate, 2005–2009	0.1	0.2	0.1	0.1	0.1	0.1	0.0	0.0	0.1

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

Table 2: Number, rate and proportion of the top 5 *Salmonella* infections, Australia, 2010, by serotype

Serotype	n	Rate*	%†	Ratio‡
S. Typhimurium	5,241	23.5	44	1.3
S. Enteritidis	836	3.7	7	1.0
S. Virchow	571	2.6	5	1.9
S. Saintpaul	422	1.9	4	1.0
S. Infantis	323	1.4	3	2.2

* Rate per 100,000.

† Proportion of total *Salmonella* notified in 2010.

‡ Ratio of the number of cases in 2010 compared with the number in 2009.

Table 3: Percentage of *Salmonella* notifications with phage type information available for 6 serotypes notified to state and territory health departments, Australia, 2007 to 2010

Salmonella serotype	2007* %	2008 %	2009 %	2010 %
S. Bovismorbificans	100.0	87.0	84.7	75.0
S. Enteritidis	97.0	91.7	91.6	94.8
S. Hadar	87.5	87.5	80.8	63.4
S. Heidelberg	90.2	80.5	75.0	64.9
S. Typhimurium	99.3	97.6	94.8	87.3
S. Virchow	97.2	95.0	91.0	74.4

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

Table 4: Number of *Salmonella* Enteritidis infections, Australia, 2010, by travel history and state or territory

	Locally-acquired	Overseas-acquired	Unknown	Total
ACT	0	14	3	17
NSW	12	133	3	148
NT	1	9	5	15
Qld	24	66	27	117
SA	3	54	1	58
Tas	0	6	0	6
Vic	8	129	2	139
WA	8	325	2	335
Total	56	736	43	835

Amongst locally-acquired *S.* Enteritidis cases in 2010, the most common phage types were 26, 13 and 6A (Table 5).

Phage types 26 and 6A have been the most common amongst locally-acquired cases since 2007. However, in 2010 phage type 13 was the second most commonly reported phage type. There was only 1 notified case of locally-acquired phage type 13 reported in 2008 (Figure 1).

Table 5: Number and percentage of the top 3 phage types and those where typing information was unknown of locally-acquired cases of *Salmonella* Enteritidis, Australia, 2010

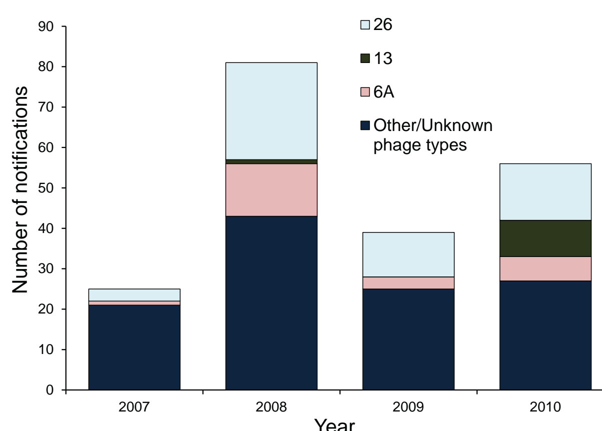
Phage type	n	%*
26	14	25.0
13	9	16.1
6A	6	10.7
Unknown	4	7.1
Untypable	3	5.4

* Proportion of total locally-acquired *Salmonella* Enteritidis (56) notified in 2010.

Campylobacter infections

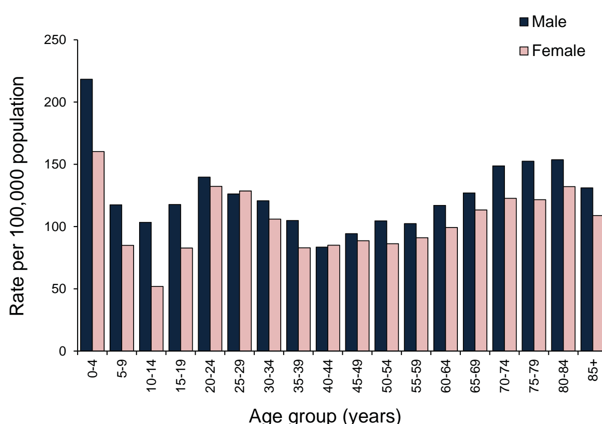
In 2010, OzFoodNet sites (excluding New South Wales) reported 16,968 cases of *Campylobacter* infection; a rate of 112.3 cases per 100,000 (Table 1). The lowest and highest rates of *Campylobacter* infection were in the Northern Territory (72.3 cases per 100,000) and in the Australian Capital Territory (153.8 cases per 100,000) respectively.

Figure 1: Number of notifications of the top 3 phage types (2010) of locally-acquired cases of *Salmonella* Enteritidis, Australia, 2007 to 2010



Overall, 54 per cent of notified cases were in males, consistent with previous years. In 2010, notification rates were highest in children aged 0–4 years for both males and females (218.3 and 160.2 notifications per 100,000, respectively) with additional peaks in the 20–24 and 80–84 years age groups (Figure 2).

Figure 2: Notification rates for campylobacteriosis, Australia, 2010, by age group and sex



Listeria infections

OzFoodNet sites reported 71 cases of *Listeria monocytogenes* infection in 2010, a rate of 0.3 cases per 100,000, which is consistent with the 5-year historical mean of 0.3 cases per 100,000 (65 cases) (Table 1). The 2010 notifications included a multi-jurisdictional outbreak linked to melons that affected at least 9 people. This outbreak is discussed later in this report.

Seventy-six per cent (54/71) of notifications in 2010 were in people aged 60 years or more and males accounted for 54% (38/71) of all notifications. Twenty-one per cent of cases died in 2010 (15/71). There was 1 materno-foetal case in 2010, which was not fatal.

The most commonly reported strain of *Listeria monocytogenes* was serotype 1/2b, 3b, 7 binary type 158 (24%, 17/71) (Table 6).

Table 6: Top 4 listeriosis strains – molecular serotype and binary type, Australia, 2010

Serotype	Binary type	n
1/2b, 3b, 7*	158*	17
4b, 4d, 4e	254	10
1/2a,3a	155	6
1/2b, 3b, 7	159	4

Source: OzFoodNet Enhanced National Listeriosis Surveillance System

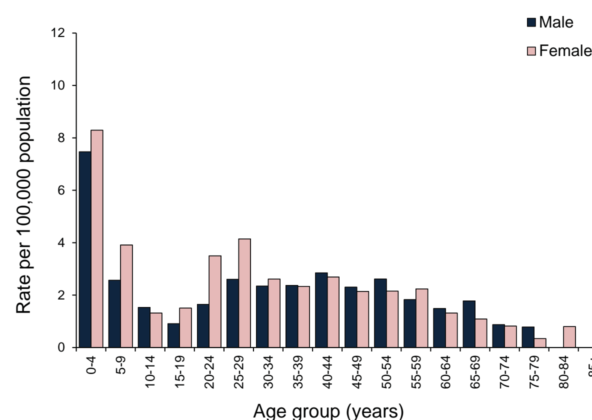
* This strain was associated with the *Listeria* outbreak discussed later in this report.

Shigella infections

There were 552 notifications of *Shigella* infection in Australia in 2010, a rate of 2.5 notifications per 100,000 compared with a mean of 655 cases (3.1 notifications per 100,000) per year between 2005 and 2009.

In 2010, notification rates for shigellosis were highest in males and females aged 0–4 years, with 7.5 and 8.3 notifications per 100,000 respectively (Figure 3). The overall rate for males decreased from 3.4 notifications per 100,000 in 2009 to 2.3 in 2010. This decrease was most prominent in the 0–4, and 20–44 year age groups.

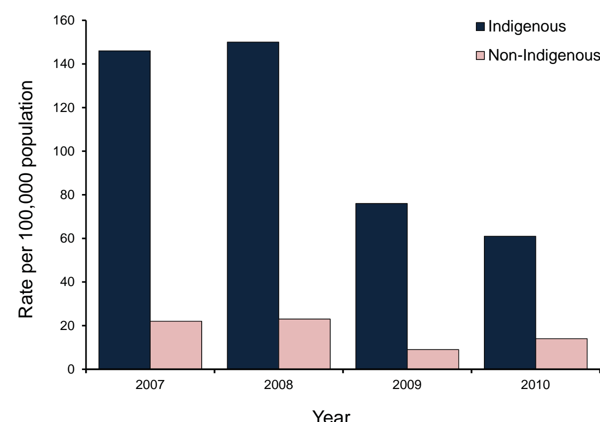
Figure 3: Notification rates for shigellosis, Australia, 2010, by age and sex



As in previous years, the highest notification rate was in the Northern Territory, with 32.7 cases per 100,000 and a decline compared with an average of 70.1 cases per 100,000 between 2005 and 2009. One factor that may have influenced the decline in cases of shigellosis in the Northern Territory since 2008 (Figure 4) was the 'No germs on me' campaign, first implemented in October 2007. This social marketing campaign targeted remote communities and Indigenous people to raise awareness about the importance of hand washing. An urban campaign was implemented in October 2008, which primarily targeted the non-Indigenous population.

The most frequently reported *Shigella* biotype in 2010 was *S. sonnei* biotype g, followed by *S. sonnei* biotype a. Together these biotypes accounted for 55.6% of all *Shigella* infections reported in 2010 (Table 7).

Figure 4: Notification rates for shigellosis, Northern Territory, 2007 to 2010, by Indigenous status



Typhoid

In 2010, there were 96 cases of *Salmonella* Typhi infection (typhoid) in Australia, a rate of 0.4 cases per 100,000, the same as the 5 year mean between 2005 and 2009 (Table 1). In 2010, 42.7% (41/96) of cases were female and cases were reported from all Australian states and territories. Travel status was known for 99.0% (95/96) of cases, with 93 cases reporting infections likely to be acquired overseas.

India was the most frequently reported country of travel for overseas-acquired cases of typhoid in 2010, with 52.7% (49/93) of cases. E1 was the most common phage type for typhoid cases with a known travel status (Table 8).

Table 7: Number, percentage and ratio of the top 10 *Shigella* infections, Australia, 2009 to 2010

Biotype	2009 n	2009 %*	2010 n	2010 %†	Ratio‡
Shigella sonnei biotype g	207	33.3	191	34.6	0.9
Shigella sonnei biotype a	119	19.2	116	21.0	1.0
Shigella flexneri 4a	33	5.3	38	6.9	1.2
Shigella flexneri 3a	44	7.1	37	6.7	0.8
Shigella flexneri 2a	38	6.1	36	6.5	0.9
Shigella sonnei untyped	56	9.0	32	5.8	0.6
Shigella flexneri 4	24	3.9	22	4.0	0.9
Shigella flexneri 2b	10	1.6	18	3.3	1.8
Shigella flexneri untyped	21	3.4	13	2.4	0.6
Shigella flexneri 6	8	1.3	11	2.0	1.4

* Proportion of total *Shigella* notified in 2009.

† Proportion of total *Shigella* notified in 2010.

‡ Ratio of the number of cases in 2010 compared with the number in 2009.

Table 8: *Salmonella* Typhi phage types acquired overseas from cases notified in 2010

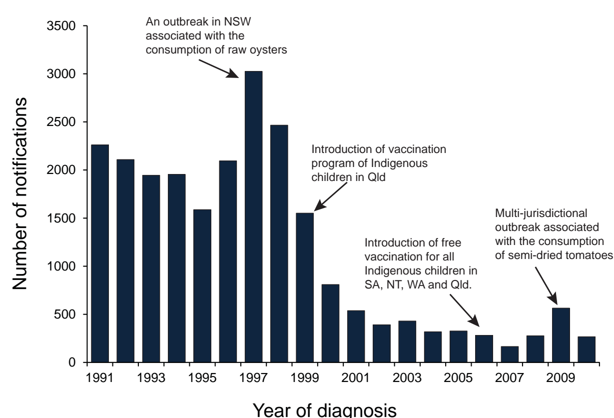
Pace of acquisition	Phage type	n
India	Unknown	17
	E1	15
	Untypable	9
	E9	5
	J1	2
	A	1
Bangladesh	Unknown	5
	Untypable	1
	E9	3
	46 VAR	3
Other countries	Unknown	10
	E1	8
	D2	5
	E9	2
	Untypable	3
	40	1
	A	1
	D1	1
	O VAR	1
Total		93

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 323 cases per year between 2005 and 2009 (1.5 cases per 100,000) (Figure 5). In 2010, the number of hepatitis A notifications decreased to 267 (1.2 cases per 100,000) from 564 cases (2.6 cases per 100,000) in 2009. The majority of cases notified in 2009 were part of a large outbreak of locally-acquired hepatitis A associated with the consumption of semi-dried tomatoes. This outbreak occurred over a 12 month period between 1 March 2009 and 18 March 2010.[13,14]

Indigenous status was known for 94.0% (251/267) of cases in 2010 (Table 9). In 2010, 1 case was identified as Indigenous (0.4%), similar to the small percentages reported between 2007 and 2009, and a decrease compared with 10%–15% (28–49 cases) per year between 2004 and 2006. This marked decrease in the past 4 years in the number and proportion of cases who are Indigenous is likely due to targeted vaccination programs. The first state program for Indigenous children commenced in Queensland in 1999.[15] The Commonwealth Government expanded the program by providing free hepatitis A vaccine for all Indigenous

Figure 5: Notifications of hepatitis A infections, Australia, 1991 to 2010, by year of diagnosis



children aged up to 5 years living in Queensland, the Northern Territory, Western Australia and South Australia from 1 November 2005.[16]

In 2010, the number of locally-acquired hepatitis A cases was similar to the numbers prior to the multi-jurisdiction outbreak of 2009, with 43.8% (117/267) of cases. The number of cases reported as overseas acquired in 2010 increased to 55.1% (147/267) of all cases being locally acquired (Table 10). Overseas-acquired cases most frequently reported travel to the South Asian and Polynesian regions. Fiji (22/147) and India (21/147) were the most frequently reported countries of travel.

Table 9: Hepatitis A notifications, Australia, 2004 to 2010, by Indigenous status

	Indigenous		Non-Indigenous		Unknown	
Year	n	%*	n	%*	n	%*
2004	37	11.6	251	78.7	31	9.7
2005	49	15.0	232	70.9	46	14.1
2006	28	10.0	219	77.9	34	12.1
2007	0	0.0	148	89.7	17	10.3
2008	3	1.1	247	89.2	27	9.7
2009	8	1.4	515	91.3	41	7.3
2010	1	0.4	250	93.6	16	6.0

* Proportion of total hepatitis A cases notified in that year.

Table 10: Place of acquisition for cases of hepatitis A, 2004 to 2010, Australia

	Locally-acquired		Overseas-acquired		Unknown	
Year	n	%*	n	%*	n	%*
2004	143	44.8	91	28.5	85	26.6
2005	140	42.8	151	46.2	36	11.0
2006	102	36.3	69	24.6	110	39.1
2007	74	44.8	38	23.0	53	32.1
2008	99	35.7	53	19.1	125	45.1
2009	417†	73.9	68	12.1	79	14.0
2010	117	43.8	147	55.1	3	1.1

* Proportion of total hepatitis A notified in that year.

† High proportion of locally-acquired hepatitis A cases in 2009 due to outbreak associated with the consumption of semi-dried tomatoes.

Botulism

There were no cases of botulism reported in 2010. The most recent notified case was reported in 2009.[14]

Shiga toxin-producing *Escherichia coli* infection

In 2010, there were 80 notifications of STEC in Australia, a rate of 0.4 cases per 100,000 compared with a mean of 0.5 cases per 100,000 between 2005 and 2009 (Table 1). These numbers include a case of HUS where an STEC organism was isolated. Under the Australian National Notifiable Disease Surveillance System surveillance case definitions, these are notified separately.[17] There were no cases of STEC in the Australian Capital Territory, the Northern Territory or Tasmania in 2010.

Notified cases of STEC infection are strongly influenced by jurisdictional practices regarding the screening of stool specimens.[18] In particular, South Australia routinely tests all bloody stools and use polymerase chain reaction (PCR) for genes coding for Shiga toxins for diagnosis, making rates for this State the highest in the country. In 2009, Queensland changed its screening procedures resulting in all stool specimens submitted for STEC testing 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 the EIA method do not meet the surveillance case definition, therefore these cases were classified as 'probable'. These probable cases (EIA positive only; PCR and/or culture negative) are not notified to the NNDSS.[19]

In 2010, 61.3% of cases were females (49/80). The median age of cases was 44 years (range 1–98 years) (Figure 6).

Figure 6: Notifications of Shiga toxin-producing *Escherichia coli*, Australia, 2010, by age group

In 2010, serotype O157 accounted for 58.8% (30/51) of STEC cases with available serotype information (obtained by serotyping cultured isolates or by

PCR targeting serotype-specific genes), followed by O111 (5 cases, 9.8%). A serotype was not identified in 36% (29/80) of cases. This is consistent with the serogroups reported in 2009.

Haemolytic uraemic syndrome

In 2010, OzFoodNet sites reported 9 cases of HUS (Table 1), compared with a mean of 20 cases per year between 2005 and 2009. Similarly to previous years, the majority of notifications were in children, with 66.7% (6 cases) of cases aged 0–4 years.

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 2010 however, an antecedent STEC infection was reported in only 1 case. In 5 cases *Streptococcus pneumoniae* was detected and for the remaining 3 cases no organism was identified as the causative agent.

Outbreaks of gastrointestinal illness

In 2010, OzFoodNet sites reported 1,640 outbreaks of gastrointestinal illness (including foodborne disease), affecting 30,193 people, of whom 722 were hospitalised (Table 11). There were 89 deaths during these outbreaks. This compares with a 5 year mean of 1,483 outbreaks.

Person-to-person outbreaks

In 2010, 81% of reported outbreaks were transmitted from person-to-person. There were 26,661 illnesses associated with these outbreaks, 519 people were hospitalised and 71 people died. Outbreaks were most commonly reported from aged care facilities (57%, 752/1,330) or child-care centres (22%, 297/1,330). Outbreaks were most commonly due to norovirus (42%, 564/1,330) or were of unknown aetiology (36%, 485/1,330).

Animal-to-person outbreaks

One outbreak was reported to have been due to animal-to-person transmission. The outbreak affected 10 children in a child-care centre following a visit to a farm, and was caused by *Cryptosporidium* sp.

Outbreaks with unknown mode of transmission

There were 155 outbreaks in which cases were clustered in time, place or person, but investigators were unable to determine the mode of transmission (Table 11). These outbreaks affected 1,376 people, 45 of whom were hospitalised and three died. Outbreaks were most commonly reported from aged care facilities (50%, 78/155), the community (11%, 17/155) and child-care centres (10%, 15/155). *Salmonella* was the aetiological agent in 15 of these outbreaks and norovirus in 15 outbreaks. In 118 outbreaks (76%), both the aetiology and the transmission mode remain unknown.

Foodborne outbreaks

In 2010, OzFoodNet sites reported 154 outbreaks of foodborne and suspected foodborne illness. These outbreaks affected 2,146 people, of whom 157 were hospitalised and 15 died (Table 11). This compares with a 5 year mean of 127 outbreaks. The overall rate of foodborne disease outbreaks in 2010 was 6.9 per million population (Table 12). The highest rates were in the Northern Territory (30.5 per million) and Tasmania (11.8 per million), although these jurisdictions reported only 7 and 6 outbreaks respectively. The largest number of outbreaks was reported from New South Wales (55 outbreaks).

Aetiologies

More than one-third of all foodborne and suspected outbreaks (34%, 53/154) were due to *S.* Typhimurium. Other frequently reported aetiologies were *Campylobacter* and *Clostridium perfringens*

Table 11: Outbreaks of gastroenteritis reported to state and territory health departments, Australia, 2010

Transmission mode	Number of outbreaks	Number ill	Number hospitalised	Number died
Foodborne and suspected foodborne	154	2,146	157	15
Person-to-person	1,330	26,661	519	71
Animal-to-person	1	10	1	0
Unknown mode (*Salmonella* cluster)	15	87	12	0
Unknown mode (other pathogen)	22	251	15	0
Unknown mode (unknown pathogen)	118	1,038	18	3
Total	1,640	30,193	722	89

Table 12: Outbreaks of foodborne disease, Australia, 2010, by OzFoodNet site

State or territory	Number of outbreaks	Number ill	Mean size (persons)	Number hospitalised	Outbreaks per million population
ACT	3	59	19.7	5	8.4
NSW	55	641	11.7	80	7.6
NT	7	121	17.3	4	30.5
Qld	23	184	8.0	13	5.1
SA	8	134	16.8	6	4.9
Tas	6	157	26.2	2	11.8
Vic	39	399	10.2	30	7.0
WA	11	128	11.6	9	4.8
Multi-jurisdictional	2	323	161.5	8	N/A
Total	154	2,146	13.9	157	6.9

(6% each, 9/154) (Table 13). More than a third of all outbreaks were of unknown aetiology (36%, 55/154) compared with 27% (44/163) in 2009.

Food vehicles

Outbreaks were categorised as being attributable to one of 18 foods (17 described by Painter et al[20] with an additional category for lamb) if a single contaminated ingredient was identified or if all ingredients belonged to that food category. Outbreaks that could not be assigned to one of the 18 categories, or for which the report contained insufficient information for food category assignment were not attributed to any food category.[21]

In 43 foodborne outbreaks (28%), investigators attributed the outbreak to a single food (Table 13),

Table 13: Number of reported foodborne disease outbreaks and number affected, Australia, 2010, by aetiology and food category

Agent category	Total number of outbreaks	Total number ill	Attributed to a single food category		Attributed to >1 food category		Not attributed to a food category	
			Number of outbreaks	Number ill	Number of outbreaks	Number ill	Number of outbreaks	Number ill
Salmonella Typhimurium	53	746	23	416	10	169	20	161
Clostridium perfringens	10	134	1	4	0	0	9	130
Campylobacter	9	103	2	15	1	18	6	70
Ciguatera fish poisoning	6	22	6	22	0	0	0	0
Norovirus	8	117	0	0	1	17	7	100
Other *Salmonella* serotypes	5	47	2	31	1	7	2	9
Listeria monocytogenes	2	15	1	9	1	6	0	0
Staphylococcus aureus	2	9	2	9	0	0	0	0
Cyclospora sp.	1	314	0	0	0	0	1	314
Bacillus cereus	1	24	1	24	0	0	0	0
Scrombroid confirmed	1	4	1	4	0	0	0	0
Other viral	1	19	0	0	0	0	1	19
Unknown	55	592	4	21	9	88	42	483
Total	154	2,146	43	555	23	305	88	1,286

in another 23 outbreaks (15%), the implicated dish contained a mix of ingredients, and no single ingredient was implicated. The majority of outbreaks (57%, 88/154) could not be attributed to a particular food due to a lack of evidence.

In outbreaks attributed to a single food (n=43), the foods most frequently implicated were eggs (49%, 21/43), fish (19%, 8/43), poultry (9%, 4/43) and fruits/nuts (7%, 3/43). In these outbreaks, 71% of those affected were in outbreaks involving eggs (394/555) whilst outbreaks involving poultry accounted for a further 8% (45/555) of cases.

Nearly one-third of foodborne outbreaks with a known food vehicle (32%, 21/66) were suspected or confirmed to have been associated with the consumption of eggs and egg-based dishes (Table 14). These egg-associated outbreaks comprised 14% (21/154) of all foodborne outbreaks, 36% (21/58) of all *Salmonella* outbreaks and 49% (21/43) of the outbreaks that were attributed to a single commodity. In these outbreaks, eggs were served as a whole food (4 outbreaks), in sauces and dressings such as hollandaise and aioli (8 outbreaks), in desserts (6 outbreaks), in a milkshake (1 outbreak), in salads or wraps (1 outbreak) and as a binding ingredient of salmon patties (1 outbreak). Investigators frequently reported that eggs and egg-based dishes included

Table 14: Outbreaks of foodborne illness associated with egg-based dishes (n=21), Australia, 2010

State or territory	Setting prepared	Aetiology	Number affected	Evidence	Food vehicle
ACT	Private residence	*S.* Typhimurium 170/108*	4	D	Chocolate mousse
NSW	Private residence	*S.* Typhimurium 170/108	5	D	Suspected mayonnaise prepared with raw eggs
	Private residence	*S.* Typhimurium 170/108	9	D	Suspected raw eggs contained in one batch of individual servings of tiramisu
	Private residence	*S.* Typhimurium 170/108	9	D	Suspected mousse cake with raw eggs
	Restaurant	*S.* Typhimurium 170/108	2	D	Suspected salmon patties made with egg
	Restaurant	*S.* Typhimurium 170/108	6	M	Tartare sauce prepared with raw egg
	Restaurant	*S.* Typhimurium 170/108	14	M	Fried ice cream
	Restaurant	*S.* Typhimurium 9	168	A	Aioli prepared with raw egg
	Takeaway	*S.* Singapore	5	D	Suspect foods containing eggs (egg and salad wrap, egg salad)
	Takeaway	*S.* Typhimurium 170/108	9	M	Mayonnaise made with raw egg
Qld	Private residence	*S.* Typhimurium	4	D	Banana milkshake containing raw egg
	Restaurant	*S.* Typhimurium 135a	34	AM	Citrus aioli containing raw egg
	Restaurant	*S.* Typhimurium 170/108	3	M	Deep fried ice cream
Tas	Restaurant	*S.* Typhimurium 170/108	43	A	Homemade ice cream
Vic	Private residence	*S.* Typhimurium 170/108	4	M	Eggs (fried soft)
	Private residence	*S.* Typhmurium 170/108	12	D	Raw egg mayonnaise
	Restaurant	*S.* Typhmurium 9	8	D	Suspected eggs
	Restaurant	*S.* Typhmurium 9	10	D	Hollandaise sauce
	Restaurant	*S.* Typhmurium 9	13	D	Uncooked egg
WA	Restaurant	*S.* Typhimurium 170/108	7	D	Scrambled eggs
	Restaurant	*S.* Typhimurium 170/108	25	D	Aioli and Caesar salad

Evidence

D Descriptive evidence implicating the vehicle

A Analytical epidemiological association between illness and vehicle

M Microbiological confirmation of aetiology in vehicle and cases.

* Classification of this phage type differs between laboratories, with the Institute of Medical and Veterinary Science using phage type 108 to classify this type of *S.* Typhimurium and Microbiological Diagnostic Unit using phage type 170 due to a difference in the interpretation of 1 phenotypic characteristic.

raw eggs and/or were insufficiently cooked (95%, 20/21). In 8 of these outbreaks, eggs were confirmed as the source of illness through microbiological or analytical evidence or both, whilst in the remaining 13 outbreaks, eggs were suspected as the food vehicle due to descriptive evidence collected during the course of the outbreak.

Settings

Implicated foods were most commonly prepared in restaurants (39%, 60/154), in aged care facilities (21%, 33/154), private residences (9%, 14/154) or at takeaway premises (8%, 12/154) (Table 15).

Evidence

To investigate these outbreaks, state and territory investigators conducted 23 retrospective cohort studies and 3 case-control studies (including 1 outbreak for which both a case-control study and a retrospective cohort study were conducted) (Appendix). Descriptive case series were collected for a further 100 outbreaks. In 29 outbreaks, no individual patient data were collected.

For 1 outbreak, there was an analytical association between illness and the implicated food as well as microbiological evidence of the aetiological agent in the epidemiologically implicated food. Investigators relied on analytical evidence alone for 24 outbreaks and microbiological (or toxicological for non-microbial outbreaks) evidence alone for 18 outbreaks. These confirmed outbreaks comprised 28% (43/154) of all foodborne outbreaks.

Contributing factors

Investigators collect information about factors that are likely to have contributed to a foodborne outbreak occurring. Contributing factors may be based on measured evidence, inspections, interview data, observations or investigator suspicion. Contamination factors are those contributing factors that led to the food becoming contaminated or to contaminated products being consumed. Contamination factors for confirmed foodborne outbreaks were most commonly stated to have been unknown (42%, 18/43) (Table 16). Contamination factors varied by the aetiology of outbreaks. In norovirus outbreaks, investigators reported that person-to-food-to-person transmission (2/3) and foodhandler contamination (1/3) were involved, while for *S.* Typhimurium outbreaks, ingestion of raw products (8/18) and cross-contamination from raw ingredients (4/18) were reported.

Significant outbreaks and multi-jurisdictional outbreaks investigated

In 2010, there were 8 outbreaks that each affected more than 40 people. Four outbreaks were due to *S.* Typhimurium, one was due to *Cyclospora cayetanensis* (a multi-jurisdictional outbreak) and 3 outbreaks were of unknown aetiology. These out-

Table 15: Food preparation setting implicated in disease outbreaks, Australia, 2010

Setting	Number of outbreaks	Per cent of outbreaks	Number affected
Restaurant	60	39	842
Aged care	33	21	425
Private residence	14	9	93
Takeaway	12	8	156
Primary produce	5	3	25
Commercial caterer	4	3	40
Institution	4	3	35
Camp	3	2	62
Other	5	3	56
Unknown	3	2	16
Fair/festival/mobile service	2	1	10
National franchised fast food	2	1	10
Bakery	2	1	13
Commercially manufactured	1	1	3
Cruise/airline	1	1	314
Military	1	1	21
Picnic	1	1	6
School	1	1	19
Total	154	100	2,146

Table 16: Factors reported as leading to the contamination of food vehicles in confirmed foodborne disease outbreaks, Australia, 2010, by aetiology

Agent	Contamination factor	Total
Bacillus cereus	unknown	1
Campylobacter	unknown	1
	ingestion of contaminated raw products	1
Clostridium perfringens	not applicable	1
Cyclospora sp.	ingestion of contaminated raw products	1
Listeria monocytogenes	unknown	1
	ingestion of contaminated raw products	1
Norovirus	Person-to-food-to-person	2
	food handler contamination	1
Other *Salmonella* serotypes	cross contamination from raw ingredients	1
Salmonella Typhimurium	ingestion of contaminated raw products	8
	unknown	5
	cross-contamination from raw ingredients	4
	other source of contamination	1
Scombroid	not applicable	1
Staphylococcus aureus	other source of contamination	1
	inadequate cleaning of equipment	1
Unknown	unknown	10
	poisonous substance	1
Total		43

breaks affected at least 687 people of whom 34 were hospitalised. There were no reported deaths. Two multi-jurisdictional outbreaks were investigated; an outbreak of listeriosis affecting 9 people, and the *Cyclospora* outbreak noted above.

An outbreak of *S.* Typhimurium 170/108 in Tasmania in December was linked to the consumption of restaurant prepared ice cream containing raw egg yolk. There were 19 microbiologically confirmed cases linked to the outbreak and at least 2 people were hospitalised. Of those initially contacted, 38/70 (54%) reported symptoms and investigations identified a further 5 cases. The attack rate among interviewees who had eaten ice cream was 100%. Approximately 400 diners ate at the restaurant over the 5 day risk period and many consumed ice cream. A sample of ice cream tested positive for *S.* Typhimurium 170/108. The restaurant received eggs from several suppliers during the period of interest and detailed trace-back was not possible.

An outbreak of suspected foodborne gastroenteritis was reported amongst 43 of 90 attendees at a church camp in April 2010 in South Australia. A cohort study was conducted, and rice was identified as the likely food vehicle due to biological plausibility and high attack rate (68.2%), but the risk ratio (RR) could not be calculated as all attendees consumed this food. No leftover food was available for testing and the 3 clinical specimens submitted were negative for pathogens.

An outbreak of 31 confirmed cases of *S.* Typhimurium 170/108 was detected through follow-up of 2 separate complaints to the NSW Food Authority, enhanced surveillance of gastroenteritis cases presenting to local emergency departments, and enhanced surveillance of laboratory notifications of *Salmonella* infection. Cases were infected with one of 3 outbreak multi-locus variable number of tandem repeats analysis (MLVA) profiles, 3-9-7-13-523* (n=1), 3-9-7-14-523 (n=16) and 3-9-7-15-523 (n=14). Illness amongst confirmed cases was associated with consuming kebabs (30 cases), mainly those filled with chicken, hummus, tabouli, lettuce, and tomato from a food outlet in a shopping centre. A further 14 probable cases were linked to the food outlet. Samples of cooked chicken kebab, hummus and tabouli and several environmental samples were positive for *S.* Typhimurium MLVA profile 3-9-7-15-523. One environmental swab was positive for both *S.* Typhimurium 170/108 MLVA profile 3-9-7-15-523 and *S.* Typhimurium 193. A sample of marinated raw chicken was positive for

* Reported in the nomenclature used by the Institute of Clinical Pathology and Medical Research (ICPMR), New South Wales.

Salmonella Infantis. The business temporarily closed and stopped preparing chicken kebab logs on site to reduce the risk of cross-contamination.

Public health staff in the Australian Capital Territory identified a link between cases and a local takeaway salad bar after investigating a higher than expected number of *Salmonella* infections, including hospitalised cases. Investigators identified 47 outbreak cases, 41 of which were laboratory confirmed with *S.* Typhimurium 170/108 infection (MLVA 3-9-7-13-523* or MLVA 3-9-7-14-523). Cases reported eating a variety of salads purchased from the salad bar, including tandoori chicken, chicken and avocado, chicken pesto, roast pumpkin fetta and baby spinach, green beans and asparagus, and Caesar and Greek salads. *Salmonella* was isolated from 2 food samples; a chicken pesto salad and a Greek salad. Environmental swabs yielded *Klebsiella oxytoca* and *Enterobacter cloacae* and an environmental health inspection identified issues including inadequate cleaning and disinfection, and ready-to-eat foods being held at inappropriate temperatures. Cross-contamination of ready-to-eat foods from an unknown source was the suspected cause.

In July 2010, an outbreak of gastroenteritis was reported in an aged care facility in Tasmania with 49/221 (22%) residents and 21/96 (22%) staff becoming ill. Seven out of 11 stool samples collected tested positive for norovirus. No food samples were available for testing as leftover food was disposed of at the end of each day. Many cases suffered from dementia therefore detailed food histories could not be collected for the majority of residents. Food histories were only obtained from 4 residents; 3 cases and 1 non-case. The aged care facility also delivered meals to the community and 82% (36/44) of the meal recipients were interviewed by phone. Six recipients reported developing symptoms of gastroenteritis. Those 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% confidence interval: [CI] 0.95, 14.55), but numbers were small and it is unclear if this meal was also consumed by the residents who became ill.

An outbreak of *S.* Typhimurium 9 (MLVA 2-27-16-12-526) in regional New South Wales was associated with eating products containing aioli prepared with raw eggs from a takeaway burger business. Interviews were conducted with 189 people who ate at the outlet over a period of 6 days and 168 of these reported symptoms of diarrhoea and/or vomiting, fever, abdominal pain, myalgia and bloody stools. Stool specimens for 104 of these people were laboratory confirmed *S.* Typhimurium 9 MLVA type 2-27-16-12-526. The outbreak strain was also isolated from aioli prepared with raw egg and from swabs of 2 chopping boards. The business was closed under a NSW Food Authority prohibition order and reopened after implementation of revised cleaning and sanitising procedures and on the provision that they would cease the production of raw egg sauces. The egg farm that supplied the eggs used to prepare the aioli was inspected but no *Salmonella* was detected on the farm.

An outbreak of *Cyclospora cayetanensis* affected 314 people, the majority from Western Australia, but also from New South Wales (1 case), South Australia (1 case), Victoria (4 cases) and Queensland (2 cases). Cases were amongst passengers and crew of 2 successive cruises on the same ship that departed from and returned to Fremantle, Western Australia in May and June 2010, visiting south-east Asian destinations.[22] Follow-up of laboratory confirmed cases and passenger enquiries identified 34 ill passengers associated with the first cruise, with 26 of these being laboratory confirmed. From the second cruise 232 passengers and 48 crew members were reported to have been affected, with 46 passengers and 1 crew member laboratory confirmed. A case-control study conducted among crew members focused on fresh produce and water consumed on board, and on-shore visits. In a univariate analysis, lettuce was mostly strongly associated with illness (Odds Ratio [OR] = 4.7, 95% CI 1.7–14.1, $P = 0.0005$). Eating rockmelon, chives and lettuce were significantly associated with illness ($P < 0.05$) in a multivariate analysis. It was concluded that illness was most likely related to eating fresh produce items taken on board in a south-east Asian port during the first cruise and also used during the second voyage. However, the case-control study did not provide enough evidence to definitively determine which fresh produce item was the likely cause of illness. Australia advised the International Health Regulations (IHR) National Focal Point of the relevant country as per Article 44 (Collaboration and Assistance) of the IHR (2005) to facilitate any local epidemiological investigations or follow-up.

An increase in a common strain of invasive *L. monocytogenes* (PCR serogroup 1/2b, 3b, 7, binary gene type 158 and PFGE 121:119:1†) infection was observed on the eastern seaboard of Australia, and a multi-jurisdictional outbreak investigation was commenced. Between January and August 2010, 9 cases of listeriosis met the case definition (cases occurring since January 2010 that were serogroup 1/2b, 3b, 7, binary gene type 158 and the Medical Diagnostic Unit designated PFGE 121:119:1 or serogroup not established, binary gene type 158 and Medical Diagnostic Unit designated PFGE 122:4N:1). A case–case analysis using non-outbreak cases as controls found that outbreak-associated

* Reported in the nomenclature used by the Institute of Clinical Pathology and Medical Research (ICPMR), New South Wales.

† Reported in the nomenclature used by the Microbiological Diagnostic Unit, University of Melbourne, Victoria.

cases were more likely to have consumed rockmelons (OR = 11.1, 95% CI 1.0–550.8, $P = 0.02$). As part of a separate investigation in one jurisdiction, 3 samples taken from a fruit salad manufacturer (honey dew melon washings, fruit rinse water and juice from mixed fruit waste) were positive for a combination of the multi-jurisdictional outbreak strains of *L. monocytogenes*. In addition, 2 samples of fruit salad sampled in May were also positive for the outbreak strains. These samples were taken by local government authorities from two separate food premises as part of their routine food sampling program. These 2 food premises reported that they purchased a combination of different whole fruits (including honey dew and rockmelon) to make the fruit salad at their retail food outlets. Trace-back of the melons used by the fruit salad manufacturer found that they were produced in a particular region of New South Wales, where melons are harvested between January and April each year. Following this outbreak, the NSW Food Authority assisted the New South Wales Department of Primary Industries to develop an information package for producers and packers increasing awareness of the outbreak and the risks of *L. monocytogenes* on melons as well as recommending effective mitigation strategies to prevent contamination.

Discussion

This report documents the incidence of gastrointestinal diseases commonly transmitted by food in Australia during 2010. The OzFoodNet surveillance network concentrates its efforts on the surveillance and outbreak investigation of foodborne diseases. This is based on partnerships with a range of stakeholders, including state and territory health departments, food safety regulators, public health laboratories, and government departments of primary industries. These partnerships and the analysis of data on notified cases and outbreaks contribute to public health action, the prevention of disease and the assessment of food safety policies and campaigns. A national program of surveillance for foodborne diseases and outbreak investigation such as OzFoodNet has many benefits including identifying foods that cause human illness through investigation of outbreaks that occur across state and territory borders. Continuing 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. This aims to reduce the cost of foodborne illness to the community, such as healthcare costs and lost productivity, and those to industry such as product recalls and loss of reputation.

Campylobacter continues to be the most frequently notified enteric pathogen under surveillance of OzFoodNet despite not being notifiable in New South Wales. The number of annual notifications has fluctuated between 14,000 and 17,000 annually over the past 10 years. However, *Campylobacter* was identified as the aetiological agent in only nine of 154 foodborne disease outbreaks reported by OzFoodNet during 2010. Three of these were attributed to foodborne transmission through contaminated chicken. In the remaining 6 outbreaks, investigators were unable to identify a food vehicle or source of infection.

There is likely to be under-reporting of *Campylobacter* outbreaks in Australia due to the lack of an efficient standardised discriminatory typing scheme and the likely under-reporting of smaller household outbreaks by the public and/or treating medical practitioner to public health authorities. A more rapid and sustainable typing method would assist OzFoodNet's activities given the large burden of *Campylobacter* infection in the community.[23–25] Evidence from these outbreak investigations would provide important risk factor information for public health action by food safety authorities to prevent further cases of disease.

In Australia, poultry is the primary source of *Campylobacter* infection. OzFoodNet estimates that about 75% of *Campylobacter* infections in the general population are acquired through foodborne transmission with approximately 30% of infections attributed to eating chicken.[26] Other cases of foodborne infection are likely to occur through food vehicles other than chicken, including foods subject to cross-contamination from raw products, especially chicken.[27,28]

The value of collaboration between public health authorities, food safety regulators and industry to reduce the incidence of foodborne *Campylobacter* infection has been recently demonstrated in New Zealand.[29] The notification rate of campylobacteriosis in New Zealand declined from an average annual rate of 353.8 per 100,000 population for the period 2002–2006 to an annual rate of 161.5 per 100,000 in 2008. A similar decline was seen for hospitalisations. This reduction was attributed to the introduction of a range of voluntary and regulatory interventions implemented as part of the risk management strategy introduced by the New Zealand Food Safety Authority in late 2006.[30] Control of *Campylobacter* in poultry meat is a major challenge for food safety authorities, regulators, agencies and industry representatives. The Primary Production Standard for Poultry Meat was implemented in May 2012 to assist in reducing the incidence of campylobacteriosis and salmonellosis in Australia.[31] The standard requires poultry growers to identify and control food safety hazards, verify the effectiveness of the control measures, and have the capacity to trace their products. Public health strategies aimed at educating the consumer should also be included as one of the interventions in a combined approach to reduce the

disease burden from *Campylobacter*. Monitoring the incidence of notified campylobacteriosis to the NNDSS together with poultry consumption rates in the Australian population would assist in measuring the effectiveness of these interventions. Any decline in the incidence of campylobacteriosis attributed to public health or primary industry interventions in Australia would need to account for trends in poultry consumption rates.

In 2010, OzFoodNet sites reported 154 foodborne or suspected foodborne outbreaks, including 2 multi-jurisdictional outbreak investigations. *Salmonella* continues to be the leading cause of reported outbreaks of foodborne illness in Australia, with 58 outbreaks due to this pathogen, the majority of them due to *S.* Typhimurium (n = 53).

Past OzFoodNet annual reports identified fish as the most common food vehicle for identified outbreaks in Australia[32] although they usually only affected small numbers of people. The most common intoxications associated with fish were ciguatera and histamine poisoning. It was encouraging to note that there were only 8 outbreaks, including six from ciguatera poisoning, associated with fish in 2010; a reduction from 16 in 2005.[32]

OzFoodNet has identified a national increase since late 2008, in the number of *Salmonella* outbreaks associated with the consumption of raw or minimally cooked eggs. These outbreaks are usually associated with *S.* Typhimurium, most commonly phage type 170/108 and related MLVA types. In 2010, investigators identified 21 outbreaks associated with raw or minimally cooked egg dishes. *S.* Typhimurium 170/108 was identified in 13 of these outbreaks across a range of settings and food vehicles. Food vehicles included desserts commonly made with raw eggs, such as chocolate mousse and tiramisu, sauces (mayonnaise, aioli), milkshakes and cake mixture. Outbreaks were also associated with food items suspected to be cross-contaminated with eggs during their preparation.

These outbreak investigations highlight the continued importance of eggs as a source of salmonellosis. A challenge in these outbreaks is to identify the factors that led to the outbreak. While the source of many of these outbreaks is likely to be from surface contamination of an egg,[33] the challenges are to determine if factors at time of food preparation were the main contributor to an outbreak. Authorities recognise that it is difficult to confidently identify the factors that lead to such outbreaks and continue to work towards a better understanding of the cause of contamination.[34] Further limitations of these investigations includes the difficulty in establishing a link between the outbreak setting or premises and egg suppliers, as trace-back to producer or farm level was not always possible. Investigations are also limited, in some cases, by poor recall of food consumption. Associations between illness and the consumption of specific food items were sometimes difficult to establish, particularly because food items such as egg and chicken are commonly consumed in the community. In addition, eggs (especially raw eggs) as ingredients of food such as desserts and dressings are not always apparent to the consumers of these foods. To contribute to the prevention of further outbreaks, regulators could consider prohibiting the sale of raw or minimally cooked egg products in commercial settings or recommend the use of pasteurised egg products in dishes that are to be served raw or lightly cooked.

Food Standards Australia New Zealand has coordinated the development of the *Primary Production and Processing Standard for Eggs and Egg Products,* which will be implemented from 26 November 2012.[35] The work has involved a risk assessment of egg production and processing in Australia and extensive consultation with industry, scientists, government agencies and the public. The new Standard places legal obligations on egg producers and processors to introduce measures to reduce food safety hazards. It also includes traceability of individual eggs for sale or used to produce egg pulp. While the onus is on the food business to have systems in place that demonstrate compliance with the Standard, the egg industry must be encouraged to work even more closely with health departments and food safety regulators to achieve demonstrable decreases in the incidence of salmonellosis.

OzFoodNet has shown that use of raw or minimally cooked eggs is currently the single largest cause of foodborne *Salmonella* outbreaks and therefore likely to be a significant source of the national increase in 'sporadic' salmonellosis seen in recent years. Measures to address this burden of illness require the collaboration of industry, food safety regulators and health representatives.

Cases of hepatitis A continued to be associated with a sustained outbreak of 415 locally-acquired cases of hepatitis A that commenced in 2009 and lasted until March 2010.[14] Detailed investigations implicated semi-dried tomatoes as the likely source of this outbreak.[13,14] Issues associated with this outbreak will continue to provide challenges for public health agencies, laboratories, industry, and food regulators. The challenges include laboratory capacity to detect viruses in food and trace-back complexities associated with international food distribution.[13] Jurisdictions investigate locally-acquired cases of hepatitis A with the aim of identifying cases associated with foodborne transmission or other risk factors for illness, and to offer contacts post-exposure prophylaxis.

Notified cases of hepatitis A infection in Indigenous people decreased from 49 cases in 2005 to a single case in 2010. This decrease occurred following the targeted hepatitis A vaccination program, which was introduced at the end of 2005. We now see the near elimination of notified cases of locally-acquired hepatitis A from the Indigenous population in Australia.[36]

In 2010, OzFoodNet continued to investigate a cluster of cases of thyroid dysfunction associated with a soy milk product that was fortified with seaweed and first reported in 2009.[14] Testing of product samples showed unusually high levels of iodine as a cause of thyroid dysfunction leading to a recall of these products. OzFoodNet coordinated the national collection of epidemiological and clinical data on cases (n=50). The response to this incident included the provision of advice to medical practitioners on hyperthyroidism in infants, developed in consultation with endocrinologists and public health physicians. This advice was circulated through professional networks to physicians and general practitioners.

This report summarises 3 preventable infections more commonly associated with travel overseas; typhoid (97% of cases), hepatitis A (55% of cases) and *S.* Enteritidis (88% of cases). Travellers are encouraged to consider the information available on the Smartraveller travel health web site (www.smartraveller.gov.au) and to seek medical advice prior to travel. Smartraveller provides specific country information to travellers about health risks. This advice, including vaccination where available, can minimise or prevent the risk of these and other infections commonly associated with travel overseas.

OzFoodNet recognises 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 *Clostridium perfringens*, which is why investigation of outbreaks is important. A further limitation relates to the outbreak data provided by OzFoodNet sites for this report and the potential for variation in categorising features of outbreaks depending on investigator interpretation and circumstances. State and territory representatives are involved in a continuous program aimed at harmonising the collection and recording of the outbreak data via the Outbreak Register Working Group.

In 2009, OzFoodNet began requesting molecular subtyping (including at least PCR serogroup and binary type) for all cases of listeriosis. National collation of subtyping information and interview data allow OzFoodNet epidemiologists to rapidly detect clusters and analyse exposure information for any possible common source(s). The OzFoodNet plan for the National Surveillance of human *Listeria monocytogenes* infection was endorsed by the Public Health Laboratory Network on 21 September 2010. It is a flexible and stable system that is able to produce timely surveillance updates and analysis. Cooperation between OzFoodNet epidemiologists and public health laboratories will continue to be an important foundation of this system. The enhanced listeriosis surveillance system identified one outbreak during 2010 with an epidemiological link to melons. Pre-cut melons and pre-made fruit salad may present higher risks of foodborne transmission of *Listeria* because once the bacterium is introduced into a food (from the surface or through processing equipment) it can proliferate under cold storage. People who may be at higher risk of infection (the elderly, pregnant women and other persons who are immunocompromised) should avoid these foods. While the scale of the outbreak investigated in 2010 was small, it was an excellent 'proof of concept' for the *Listeria* surveillance plan. During an outbreak, effective partnerships between OzFoodNet epidemiologists, public health laboratory staff and food regulatory personnel facilitated sharing of epidemiological and microbiological intelligence that enabled the early detection and characterisation of this outbreak leading to timely public health action.

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Appendix

Foodborne outbreak summary for OzFoodNet sites, Australia, 2010

State or territory	Month	Setting	Agent responsible	Ill	Hospitalised	Fatalities	Evidence	Epidemiological study	Food vehicle	Commodity[20]	Contamination factor
Qld	Jan	Restaurant	*Salmonella* Typhimurium 170/108	3	0	0	M	Case series	Deep fried ice cream	Eggs	Other source of contamination
Vic	Jan	Restaurant	*Salmonella* Typhimurium 9	13	1	0	D	No formal study	Uncooked egg	Eggs	Ingestion of contaminated raw products
WA	Jan	Restaurant	*Salmonella* Typhimurium 170/108	25	5	0	D	Case series	Aioli and caesar salad	Eggs	Other source of contamination
NSW	Jan	Restaurant	*Salmonella* Typhimurium 9	168	19	0	A	Point source cohort	Aioli prepared with raw egg	Eggs	Ingestion of contaminated raw products
NSW	Jan	Takeaway	*Salmonella* Singapore	5	0	0	D	Case series	Unknown: suspect foods containing eggs (egg and salad wrap, egg salad)	Eggs	Unknown
NSW	Jan	Private residence	*Salmonella* Typhimurium 170/108	5	4	0	D	Case series	Probably mayonnaise prepared with raw eggs	Eggs	Ingestion of contaminated raw products
Qld	Jan	Private residence	Ciguatera fish poisoning	4	4	0	D	Case series	Mackerel	Fish	Toxic substance or part of tissue
Qld	Jan	Primary produce	Ciguatera fish poisoning	6	0	0	D	Case series	Fish (unspecified)	Fish	Toxic substance or part of tissue
NSW	Jan	Restaurant	Unknown	5	0	0	D	Case series	Mehi-mehi fish fillets	Fish	Toxic substance or part of tissue
SA	Jan	Other	*Salmonella* Typhimurium 9	20	1	0	D	Case series	Bakery products, no specific item identified	Not assigned	Unknown
SA	Jan	Private residence	*Salmonella* Typhimurium 9	6	0	0	A	Point source cohort	Unknown	Not assigned	Unknown
Qld	Jan	Private residence	Norovirus	6	1	0	D	No formal study	No vehicle identified	Not assigned	Unknown

Appendix *continued:* Foodborne outbreak summary for OzFoodNet sites, Australia, 2010

State or territory	Month	Setting	Agent responsible	Ill	Hospitalised	Fatalities	Evidence	Epidemiological study	Food vehicle	Commodity[20]	Contamination factor
Qld	Jan	Restaurant	*Salmonella* Typhimurium 170/108	6	1	0	D	Case series	No vehicle identified	Not assigned	Unknown
Qld	Jan	Restaurant	Unknown	4	0	0	D	No formal study	Unknown	Not assigned	Unknown
Qld	Jan	Restaurant	*Salmonella* Typhimurium 89	4	0	0	M	Case series	No vehicle identified	Not assigned	Unknown
Vic	Jan	Private residence	Unknown	19	0	0	A	Point source cohort	Unknown	Not assigned	Unknown
Vic	Jan	Aged care	Unknown	9	0	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Jan	Private residence	*Salmonella* Typhimurium 135a	5	0	0	D	Case series	Unknown	Not assigned	Unknown
NSW	Jan	Takeaway	Unknown	3	0	0	D	Case series	Assorted pizzas (beef, cheese, chicken)	Not assigned	Unknown
NSW Jan		Restaurant	*Salmonella* Typhimurium 9	2	1	0	D	Case series	Probably a pork bun	Not assigned	Inadequate cleaning of equipment
NSW	Jan	Aged care	*Salmonella* Typhimurium 170/108	2	0	0	D	Case series	Unknown pureed food	Not assigned	Unknown
NSW	Jan	Restaurant	Unknown	25	0	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Feb	Restaurant	*Salmonella* Typhimurium 9	8	1	0	D	Case series	Suspect eggs	Eggs	Ingestion of contaminated raw products
MJOI	Feb	Primary produce	*Listeria monocytogenes*	9	8	2	M	Case series	Melons	Fruits/nuts	Ingestion of contaminated raw products
Qld	Feb	Restaurant	*Salmonella* Typhimurium 135a	9	1	0	D	Case series	Unknown	Not assigned	Cross contamination from raw ingredients
Vic	Feb	Commercial caterer	*Salmonella* Typhimurium 141	15	1	0	A	Point source cohort	Pasta salad	Not assigned	Cross contamination from raw ingredients
Vic	Feb	Aged care	*Clostridium perfringens*	9	0	0	D	No formal study	Unknown	Not assigned	Unknown
Vic	Feb	Aged care	*Campylobacter*	5	0	0	D	No formal study	Unknown	Not assigned	Ingestion of contaminated raw products
Vic	Feb	Unknown	*Listeria monocytogenes*	6	6	4	M	Case series	Cold meat	Not assigned	Unknown

Appendix *continued:* Foodborne outbreak summary for OzFoodNet sites, Australia, 2010

State or territory	Month	Setting	Agent responsible	Ill	Hospitalised	Fatalities	Evidence	Epidemiological study	Food vehicle	Commodity[20]	Contamination factor
NSW	Feb	Restaurant	*Salmonella* Typhimurium 170 var	3	0	0	D	Case series	Unknown	Not assigned	Unknown
Tas	Feb	Restaurant	Unknown	26	0	0	A	Point source cohort	Chicken mushroom and bacon cream sauce	Not assigned	Unknown
NSW	Feb	Restaurant	Unknown	3	0	0	D	Case series	Probably chicken or beef	Not assigned	Unknown
NSW	Feb	Restaurant	Unknown	4	0	0	D	No formal study	Possibly lamb, beef & chicken skewers and an assortment of vegetables	Not assigned	Unknown
NSW	Feb	Restaurant	Unknown	4	0	0	D	No formal study	Unknown	Not assigned	Not reported
NSW	Feb	Takeaway	*Salmonella* Typhimurium 204	4	3	0	M	Case series	Barbecued pork	Pork	Cross contamination from raw ingredients
ACT	Mar	Private residence	*Salmonella* Typhimurium 170/108	4	0	0	D	Case series	Chocolate mousse	Eggs	Ingestion of contaminated raw products
NSW	Mar	Restaurant	*Salmonella* Typhimurium 170/108	6	3	0	M	Case series	Tartare sauce, prepared with raw egg	Eggs	Ingestion of contaminated raw products
NSW	Mar	Private residence	*Salmonella* Typhimurium 170/108	9	1	0	D	Case series	Probably raw eggs contained in one batch of individual servings of tiramisu	Eggs	Ingestion of contaminated raw products
NSW	Mar	Commercially manufactured	Unknown	3	0	0	D	Case series	Orange and mango fruit drink	Fruits/nuts	Not applicable
NSW	Mar	Restaurant	*Salmonella* Typhimurium 170/108	19	0	0	M	Case series	Suspected peanut/cashew mixture	Fruits/nuts	Unknown
Vic	Mar	Aged care	Unknown	4	0	0	D	No formal study	Unknown	Not assigned	Unknown

Appendix *continued:* Foodborne outbreak summary for OzFoodNet sites, Australia, 2010

State or territory	Month	Setting	Agent responsible	Ill	Hospitalised	Fatalities	Evidence	Epidemiological study	Food vehicle	Commodity[20]	Contamination factor
Vic	Mar	Aged care	*Clostridium perfringens*	17	0	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Mar	Aged care	*Clostridium perfringens*	16	0	0	D	Case series	Unknown	Not assigned	Unknown
WA	Mar	Restaurant	Unknown	12	0	0	D	No formal study	Karage chicken and rice	Not assigned	Unknown
NSW	Mar	Takeaway	Unknown	3	1	0	D	Case series	Unknown	Not assigned	Unknown
NSW	Mar	National franchised fast food	*Salmonella* Typhimurium 9	4	1	0	D	Case series	Possibly chicken pieces from franchised restaurant	Poultry	Unknown
NSW	Apr	Takeaway	*Salmonella* Typhimurium 170/108	9	0	0	M	Case series	Mayonnaise made with raw egg	Eggs	Ingestion of contaminated raw products
SA	Apr	Camp	Unknown	43	0	0	A	Point source cohort	Unknown	Not assigned	Unknown
Vic	Apr	Aged care	Unknown	6	0	0	D	Case series	Unknown	Not assigned	Unknown
NSW	Apr	Restaurant	*Salmonella* Typhimurium 170/108	16	9	0	D	Case series	Suspected fried rice	Not assigned	Not applicable
NSW	Apr	Aged care	*Salmonella* Infantis	26	5	2	A	Point source cohort	Suspected fluid thickener contaminated by raw chicken mince	Poultry	Cross contamination from raw ingredients
Qld	May	Restaurant	Norovirus	11	0	0	D	Case series	Unknown	Not assigned	Person-to-food-to-person
Qld	May	Restaurant	Norovirus	12	0	0	D	Case series	Unknown	Not assigned	Person-to-food-to-person
Vic	May	Aged care	Unknown	9	0	0	D	Case series	Unknown	Not assigned	Unknown
MJOI	May	Cruise/airline	*Cyclospora cayetanensis*	314	0	0	A	Case control study		Not assigned	Ingestion of contaminated raw products
NSW	May	Takeaway	Unknown	2	0	0	D	Case series	Suspect Mongolian lamb or fried rice	Not assigned	Unknown
NSW	May	Restaurant	Unknown	26	1	0	A	Point source cohort	Unknown	Not assigned	Unknown
NSW	May	Restaurant	Unknown	7	0	0	D	Case series	Unknown	Not assigned	Unknown

Appendix *continued:* Foodborne outbreak summary for OzFoodNet sites, Australia, 2010

State or territory	Month	Setting	Agent responsible	Ill	Hospitalised	Fatalities	Evidence	Epidemiological study	Food vehicle	Commodity[20]	Contamination factor
NSW	May	Fair/festival/ mobile service	*Salmonella* Saintpaul	7	3	0	D	Case series	Suspected salmon & couscous dish	Not assigned	Unknown
NSW	May	Restaurant	*Campylobacter jejuni*	10	0	0	A	Point source cohort	Raw chicken	Poultry	Ingestion of contaminated raw products
Qld	Jun	Restaurant	*Salmonella* Typhimurium 135a	34	1	0	AM	Point source cohort	Citrus aioli	Eggs	Ingestion of contaminated raw products
NSW	Jun	Private residence	*Salmonella* Typhimurium 170/108	9	5	0	D	Case series	Unknown: possibly mousse cake with raw eggs	Eggs	Unknown
Qld	Jun	Restaurant	*Clostridium perfringens*	4	0	0	M	Case series	Rotti curry lamb	Lamb	Not applicable
SA	Jun	Other	Unknown	10	0	0	Other lab evidence	No formal study	Milk	Milk	Poisonous substance
NSW	Jun	Restaurant	Unknown	3	0	0	D	Case series	Suspected oysters	Molluscs	Unknown
Vic	Jun	Aged care	*Campylobacter*	15	1	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Jun	Aged care	Unknown	8	0	0	D	Case series	Unknown	Not assigned	Unknown
WA	Jun	Aged care	*Clostridium perfringens*	10	0	0	D	Case series	Unknown	Not assigned	Not applicable
NT	Jun	Restaurant	Norovirus	19	0	0	A	Point source cohort		Not assigned	Person to food to person
NSW	Jun	Restaurant	Unknown	10	0	0	A	Point source cohort	Unknown	Not assigned	Unknown
NSW	Jun	Takeaway	*Salmonella* Typhimurium 170/108	45	8	0	M	Case series	Chicken, hommus, tabouli	Not assigned	Cross contamination from raw ingredients
NSW	Jun	Restaurant	Unknown	15	2	0	D	Case series	Unknown	Not assigned	Not applicable
NSW	Jun	Restaurant	Unknown	7	0	0	D	Case series	Suspected chicken in cheese sauce, mixed vegetables	Not assigned	Ingestion of contaminated raw products
NSW	Jun	Restaurant	Unknown	12	0	0	D	Case series	Unknown	Not assigned	Not reported
NSW	Jun	Restaurant	Unknown	4	0	0	D	No formal study	Suspected beef pie	Not assigned	Not reported

Appendix *continued:* Foodborne outbreak summary for OzFoodNet sites, Australia, 2010

State or territory	Month	Setting	Agent responsible	Ill	Hospitalised	Fatalities	Evidence	Epidemiological study	Food vehicle	Commodity[20]	Contamination factor
NSW	Jun	Takeaway	Unknown	9	0	0	D	Case series	Unknown	Not assigned	Not reported
Vic	Jul	Private residence	*Salmonella* Typhimurium 170/108	4	2	0	M	Case series	Eggs (fried soft)	Eggs	Ingestion of contaminated raw products
Vic	Jul	Private residence	Scrombroid confirmed	4	0	0	Other lab evidence	Case series	Tuna	Fish	Not applicable
Vic	Jul	Aged care	*Clostridium perfringens*	16	0	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Jul	Aged care	*Salmonella* Typhimurium 186	4	2	0	D	Case series	Unknown	Not assigned	Unknown
WA	Jul	Restaurant	*Salmonella* Typhimurium	3	1	0	D	Case series	Unknown	Not assigned	Unknown
WA	Jul	Other	Unknown	6	0	0	D	Case series	Unknown	Not assigned	Unknown
WA	Jul	Restaurant	Norovirus	17	0	0	A	Case control study	Lasagne	Not assigned	Food handler contamination
NT	Jul	School	Viral	19	0	0	D	No formal study	Unknown	Not assigned	Unknown
NT	Jul	Unknown	*Salmonella* Typhimurium 135a	7	4	0	D	Case series	Unknown	Not assigned	Cross contamination from raw ingredients
NSW	Jul	Restaurant	*Salmonella* Typhimurium 9	9	Unknown	0	D	No formal study	Unknown	Not assigned	Not reported
Tas	Jul	Aged care	Unknown	70	0	0	D	Case series	Unknown	Not assigned	Unknown
Tas	Jul	Aged care	Unknown	6	0	0	A	Point source cohort	Unknown	Not assigned	Unknown
NSW	Jul	Aged care	*Salmonella* Typhimurium 170/108	7	0	0	A	Point source cohort	Unknown, possibly minced or pureed diet	Not assigned	Unknown
NSW	Aug	Restaurant	*Salmonella* Typhimurium 170/108	14	4	0	M	Case series	Fried ice cream	Eggs	Ingestion of contaminated raw products
Qld	Aug	Primary produce	Ciguatera fish poisoning	4	0	0	D	Case series	Fish head soup	Fish	Toxic substance or part of tissue
Qld	Aug	Primary produce	Ciguatera fish poisoning	2	0	0	D	Case series	Coral trout	Fish	Toxic substance or part of tissue
SA	Aug	Restaurant	*Campylobacter jejuni*	18	2	0	A	Point source cohort	Steak with chicken liver pate	Not assigned	Unknown

Appendix *continued:* Foodborne outbreak summary for OzFoodNet sites, Australia, 2010

State or territory	Month	Setting	Agent responsible	Ill	Hospitalised	Fatalities	Evidence	Epidemiological study	Food vehicle	Commodity[20]	Contamination factor
SA	Aug	Institution	Unknown	8	0	0	A	Point source cohort	Unknown	Not assigned	Unknown
Vic	Aug	Aged care	*Salmonella* Typhimurium 197	23	4	2	D	Case series	Unknown	Not assigned	Cross contamination from raw ingredients
Vic	Aug	Camp	*Salmonella* Typhimurium 9	6	1	0	A	Point source cohort	Unknown	Not assigned	Unknown
WA	Aug	Aged care	*Salmonella* Typhimurium	7	0	1	D	Case series	Unknown	Not assigned	Unknown
NT	Aug	Picnic	*Salmonella* Virchow 8	6	0	0	D	Case series	Unknown	Not assigned	Not reported
NSW	Aug	Restaurant	Unknown	27	0	0	A	Point source cohort	Suspect assorted wraps	Not assigned	Unknown
NSW	Aug	Aged care	*Clostridium perfringens*	8	1	0	D	No formal study	Unknown	Not assigned	Unknown
Vic	Sept	Restaurant	*Salmonella* Typhimurium 9	10	2	0	D	Case series	Hollandaise sauce	Eggs	Ingestion of contaminated raw products
Qld	Sept	Fair/festival/ mobile service	*Staphylococcus aureus*	3	Unknown	0	M	Case series	Rice noodle	Grains/beans	Other source of contamination
SA	Sept	Other	*Salmonella* Typhimurium 9	10	0	0	D	No formal study	None implicated	Not assigned	Not applicable
Qld	Sept	Aged care	*Campylobacter*	7	0	0	D	No formal study	Unknown	Not assigned	Unknown
ACT	Sept	Restaurant	Unknown	8	0	0	D	No formal study	Unknown	Not assigned	Cross contamination from raw ingredients
Vic	Sept	Aged care	Unknown	12	0	0	D	Case series	Unknown	Not assigned	Unknown
WA	Sept	Other	Unknown	10	0	0	D	Case series	Unknown	Not assigned	Unknown
WA	Sept	Military	Norovirus	21	0	0	D	Case series	Unknown	Not assigned	Food handler contamination
NT	Sept	Camp	Unknown	13	0	0	D	No formal study	Unknown	Not assigned	Unknown
NSW	Sept	Restaurant	Unknown	4	0	0	D	Case series	Unknown	Not assigned	Unknown
Tas	Sept	Commercial caterer	Unknown	6	Unknown	0	A	Point source cohort	Unknown	Not assigned	Unknown
NSW	Sept	Takeaway	*Salmonella* Typhimurium 170/108	15	3	0	M	Case series	Unknown	Not assigned	Cross contamination from raw ingredients

Appendix *continued:* Foodborne outbreak summary for OzFoodNet sites, Australia, 2010

State or territory	Month	Setting	Agent responsible	Ill	Hospitalised	Fatalities	Evidence	Epidemiological study	Food vehicle	Commodity[20]	Contamination factor
Vic	Oct	Restaurant	*Bacillus cereus*	24	0	0	A	Point source cohort	Rice (and /or beef curry)	Grains/beans	Unknown
Qld	Oct	Restaurant	Unknown	9	0	0	D	Case series	Unknown	Not assigned	Unknown
Qld	Oct	Institution	*Campylobacter jejuni*	17	1	0	D	No formal study	Unknown	Not assigned	Unknown
ACT	Oct	Takeaway	*Salmonella* Typhimurium 170/108	47	5	0	D	Case series	Assorted salads	Not assigned	Cross contamination from raw ingredients
Vic	Oct	Aged care	Unknown	7	0	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Oct	Aged care	*Clostridium perfringens*	28	0	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Oct	Aged care	Unknown	10	0	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Oct	Aged care	Unknown	5	0	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Oct	Restaurant	*Salmonella* Typhimurium 9	2	2	0	D	Case series	Broken rice	Not assigned	Unknown
Vic	Oct	Aged care	*Clostridium perfringens*	11	0	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Oct	Unknown	*Campylobacter jejuni*	3	0	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Oct	Restaurant	*Salmonella* Typhimurium 9	4	2	0	D	Case series	Mixed dishes	Not assigned	Unknown
NSW	Oct	Restaurant	Unknown	5	0	0	A	Point source cohort	Unknown	Not assigned	Unknown
NSW	Oct	Takeaway	Unknown	6	0	0	D	No formal study	Unknown	Not assigned	Unknown
NSW	Nov	Restaurant	*Salmonella* Typhimurium 170/108	2	1	0	D	No formal study	Suspected salmon patties made with egg	Eggs	Unknown
Qld	Nov	Primary produce	Ciguatera fish poisoning	4	0	0	D	Case series	Passionfruit trout	Fish	Toxic substance or part of tissue
Qld	Nov	Aged care	*Campylobacter jejuni*	23	1	1	D	No formal study	Unknown	Not assigned	Unknown
Vic	Nov	Aged care	Unknown	7	0	0	D	Case series	Unknown	Not assigned	Unknown
NT	Nov	Commercial caterer	Norovirus	12	0	0	A	Point source cohort	Unknown	Not assigned	Person-to-food-to-person
NT	Nov	Restaurant	Unknown	45	0	0	D	No formal study	Unknown	Not assigned	Unknown

Appendix *continued:* Foodborne outbreak summary for OzFoodNet sites, Australia, 2010

State or territory	Month	Setting	Agent responsible	Ill	Hospitalised	Fatalities	Evidence	Epidemiological study	Food vehicle	Commodity[20]	Contamination factor
NSW	Nov	Commercial caterer	*Salmonella* Typhimurium	7	2	0	D	No formal study	Unknown	Not assigned	Unknown
NSW	Nov	Restaurant	Unknown	6	0	0	D	Case series	Unknown	Not assigned	Unknown
NSW	Nov	Restaurant	Unknown	3	0	0	D	No formal study	Unknown	Not assigned	Unknown
NSW	Nov	Bakery	*Salmonella* Typhimurium	10	0	0	D	Case series	Probably pork roll	Not assigned	Unknown
Qld	Dec	Private residence	*Salmonella* Typhimurium	4	2	0	D	Case series	Banana milkshake	Eggs	Ingestion of contaminated raw products
Vic	Dec	Private residence	*Salmonella* Typhmurium 170/108	12	1	0	D	Case series	Raw egg mayonnaise	Eggs	Ingestion of contaminated raw products
WA	Dec	Restaurant	*Salmonella* Typhimurium 170/108	7	1	0	D	Case series	Scrambled eggs	Eggs	Unknown
Tas	Dec	Restaurant	*Salmonella* Typhimurium 170/108	43	2	0	A	Case control and cohort	Homemade ice cream	Eggs	Ingestion of contaminated raw products
Qld	Dec	Private residence	Ciguatera fish poisoning	2	0	0	D	Case series	Mangrove jack fish	Fish	Toxic substance or part of tissue
Qld	Dec	National franchised fast food	*Staphylococcus aureus*	6	1	0	M	Case series	Milkshake	Milk	Inadequate cleaning of equipment
SA	Dec	Restaurant	Norovirus	19	3	0	D	Case series	Unknown	Not assigned	Food handler contamination
Vic	Dec	Aged care	*Salmonella* Typhimurium 170/108	18	3	3	D	Case series	Unknown	Not assigned	Unknown
Vic	Dec	Aged care	Unknown	5	0	0	D	Case series	Unknown	Not assigned	Unknown
Vic	Dec	Aged care	*Clostridium perfringens*	15	1	0	D	Case series	Unknown	Not assigned	Unknown
WA	Dec	Restaurant	*Salmonella* Typhimurium 170/108	10	2	0	D	Case series	Unknown	Not assigned	Not applicable
NSW	Dec	Institution	Unknown	5	0	0	D	Case series	Unknown	Not assigned	Unknown
NSW	Dec	Bakery	*Salmonella* Infantis	3	Unknown	0	D	Case series	Bakery products	Not assigned	Unknown

Appendix *continued:* Foodborne outbreak summary for OzFoodNet sites, Australia, 2010

State or territory	Month	Setting	Agent responsible	Ill	Hospitalised	Fatalities	Evidence	Epidemiological study	Food vehicle	Commodity[20]	Contamination factor
Tas	Dec	Restaurant	Unknown	6	Unknown	0	D	No formal study	Unknown	Not assigned	Not applicable
NSW	Dec	Restaurant	Unknown	3	0	0	D	No formal study	Unknown	Not assigned	Unknown
NSW	Dec	Restaurant	Unknown	5	0	0	D	No formal study	Unknown	Not assigned	Unknown
NSW	Dec	Takeaway	*Salmonella* Typhimurium 170/108	8	3	0	M	No formal study	Suspected pork rolls	Not assigned	Ingestion of contaminated raw products
Vic	Dec	Institution	*Campylobacter jejuni*	5	0	0	D	Case series	Chicken	Poultry	Ingestion of contaminated raw products

MJOI Multijurisdictional outbreak investigation.

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 trachoma surveillance annual report, 2010

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Abstract

Endemic trachoma continues to exist in remote Aboriginal communities in Australia. The National Trachoma Surveillance and Reporting Unit, established in 2006, is responsible for the collation, analysis and reporting of trachoma prevalence data and the documentation of trachoma control strategies in Australia. Data were collected from Aboriginal communities designated at-risk for endemic trachoma (defined as prevalence of 5% or greater among children) within the Northern Territory, South Australia and Western Australia. This report presents data collected in 2010. Aboriginal children aged 1–14 years were screened using the World Health Organization grading criteria to diagnose and classify individual cases of trachoma. Aboriginal adults aged 40 years or older were screened for trichiasis. Community screening coverage of the designated at-risk communities was 60% in 2010. Screening coverage of the estimated population of children aged 1–14 years and of adults aged 40 years or older in at-risk communities was 11.5% and 5%, respectively. Trachoma prevalence among children aged 1–14 years who were screened was 11%. Of the communities screened, 36% were found to have no cases of active trachoma and 55% were found to have endemic levels of trachoma. Treatment coverage of active cases and their contacts varied between jurisdictions from 64% to 90%. Trichiasis prevalence was 4% within the screened communities. *Commun Dis Intell* 2012;36(3):E242–E250.

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

Introduction

This is the fifth national trachoma surveillance annual report. Trachoma screening and management data for 2010 were provided to the National Trachoma Surveillance and Reporting Unit (NTSRU) by the health authorities in the Northern Territory, South Australia and Western Australia. Data were analysed by region: five in the Northern Territory, six in South Australia and four in Western Australia. Jurisdictional authorities designated 243 remote communities in these regions as being at-risk of endemic trachoma in 2010.

Trachoma is an eye infection caused by the bacterium *Chlamydia trachomatis*. The infection can be transmitted through close facial contact, hand-to-eye contact, via fomites (towels, clothing and bedding) or by flies. Repeated infections with *C. trachomatis*, especially during childhood, may lead to scarring and distortion of the eyelid, which may in turn cause the eyelashes to rub against the cornea; this is known as trichiasis, and can lead to blindness. The Global Elimination of Blinding Trachoma (GET) 2020 initiative, supported by the World Health Organization (WHO) Alliance, aims to eliminate blinding trachoma by the year 2020, via implementation of the S.A.F.E. strategy; the key components of which are (S) surgery (to correct trichiasis), (A) antibiotic treatment, (F) facial cleanliness and (E) environmental improvements. The Australian Government, in accordance with the GET 2020 initiative and through the *Improving Eye and Ear Health Services for Indigenous Australians for Better Education and Employment Outcomes* measure, committed $16 million over a 4-year period from 2009, toward eliminating trachoma in Australia. The funding is for the improvement and expansion of screening and control activities, as well as the establishment of a strong framework for monitoring and evaluation.

Methods

Each jurisdiction undertook trachoma screening and treatment according to their respective state and territory protocols, broadly following Communicable Diseases Network Australia (CDNA) guidelines.[1] At the commencement of the National Trachoma Management Program in 2006, representatives from each jurisdiction identified at-risk communities based on historical data and other knowledge. Since then, some communities have been reclassified. Screening for trachoma focuses on these communities designated at-risk, but a small number of other communities may also be screened each year, generally because of anecdotal information suggesting the presence of cases.

The WHO trachoma grading criteria[2] were used to diagnose and classify individual cases of trachoma. The CDNA guidelines recommend treatment strategies according to the active trachoma prevalence within the community. For each jurisdiction, screening within at-risk communities used a sampling method whereby all children attending school during the screening period were examined. Data collection forms were developed by the National

Trachoma Surveillance Reference Group based on the CDNA guidelines. Jurisdictions agreed that data would be collected on the forms, entered into a database and forwarded to the NTSRU for checking and analysis. Only community-level information was provided to the NTSRU and included:

- the number of children aged 1–14 years with clean faces* (defined as the absence of dirt, dust and crusting on cheeks and forehead) and the number screened;
- the number of children aged 1–14 years with trachoma* and the number screened;
- the number of treatment episodes of active trachoma, their household contacts and community members;
- the number of adults with trichiasis and the number screened, as well as the number undergoing trichiasis surgery; and
- community level implementation of WHO SAFE strategies.

Northern Territory

Trachoma screening and management in the Northern Territory is undertaken through collaboration between the Centre for Disease Control (CDC) and the Child Health Program in the Northern Territory Department of Health and Families. Trachoma screening is incorporated into the Healthy School Age Kids (HSAK)[3] annual check and is conducted either by local primary health care units or by Aboriginal community controlled health services (ACCHS). Following screening, treatment is generally delivered by the CDC and public health units.

In 2010, systematic trichiasis screening of adults did not occur, but some adult screening took place during community visits by optometrists or ophthalmologists from the Regional Eye Health Service based in Alice Springs.

South Australia

In 2010, Country Health South Australia was responsible for trachoma screening and management, and activities such as conducting visits, were undertaken by the Eye Health and Chronic Disease Specialist Support Program (EH&CDSSP), Aboriginal Health Council of South Australia. Regular visits to South Australian Aboriginal communities were made by visiting optometrists, ophthalmologists and the project coordinator of EH&CDSSP. These visits incorporated trachoma screening and management. Trichiasis screening was undertaken opportunistically for adults who consulted with the EH&CDSSP team.

* Previous reports have reported on children aged 1-9 years; however, in 2010 one jurisdiction only reported aggregated data for children in the age range 1-14 years.

Western Australia

Trachoma screening and management is the responsibility of population health units (PHUs) in Western Australia in the Kimberley, Goldfields, Pilbara and Mid West Health Regions. In collaboration with local primary health care units, PHUs screen all communities in each region within a 2-week period, usually in late August or early September. Treatment is delivered at the time of screening. Trichiasis screening was performed in conjunction with the delivery of annual seasonal influenza vaccinations.

Data analysis

For the purpose of the National Trachoma Management Program, a community is defined as a specific location where people reside and where there is at least one school. Community coverage is defined as the proportion of at-risk communities that are screened for trachoma. Individual screening coverage is the proportion of children in the target age group in a community who were actually screened.

As in previous reports, population data were based on the 2006 census conducted by the Australian Bureau of Statistics (ABS).[4] The census counts for communities were projected forward for subsequent years using the ABS median series projected increase (1.6%, 1.8% and 2.1% in the Northern Territory, Western Australia and South Australia, respectively). The prevalence of active trachoma was calculated using the total number of children screened as the denominator.

Trachoma data were collated in the 0–4, 5–9 and 10–15 year age groups. Comparisons with earlier time points were limited to the 5–9 year age group due to the consistently greater screening coverage across all jurisdictions in this group. Data from 2006 were excluded from the assessment of time trends as the collection methods in that year differed from the methods subsequently adopted. Statistical significance in prevalence trend rates for communities that screened consistently from 2007 was tested with the chi-square test for trend.

For treatment coverage, adherence to the CDNA guidelines was assessed as being the proportion of active cases and contacts requiring treatment who were treated within 2 weeks of the screening of the index case. The data provided did not include information on treatment of active cases outside the 2-week period after screening. The proportion of contacts treated, regardless of when their treatment took place, was also estimated.

Due to differences in the interpretation of the treatment guidelines, if the prevalence of trachoma reached the level at which mass community treat-

ment was indicated, two methods were used to estimate the number of individuals requiring treatment in that community.

- Method 1 (targeted treatment) was based on the number of cases of trachoma detected through screening plus the number of contacts reported as requiring treatment. If mass treatment was required but the number of contacts requiring treatment was not reported then it was estimated as being the number of children in the community aged 6 months to 14 years plus the number of household contacts of active cases.
- Method 2 (whole community treatment) was based on the assumption that when mass treatment was required, all members of the community should be treated.

Results

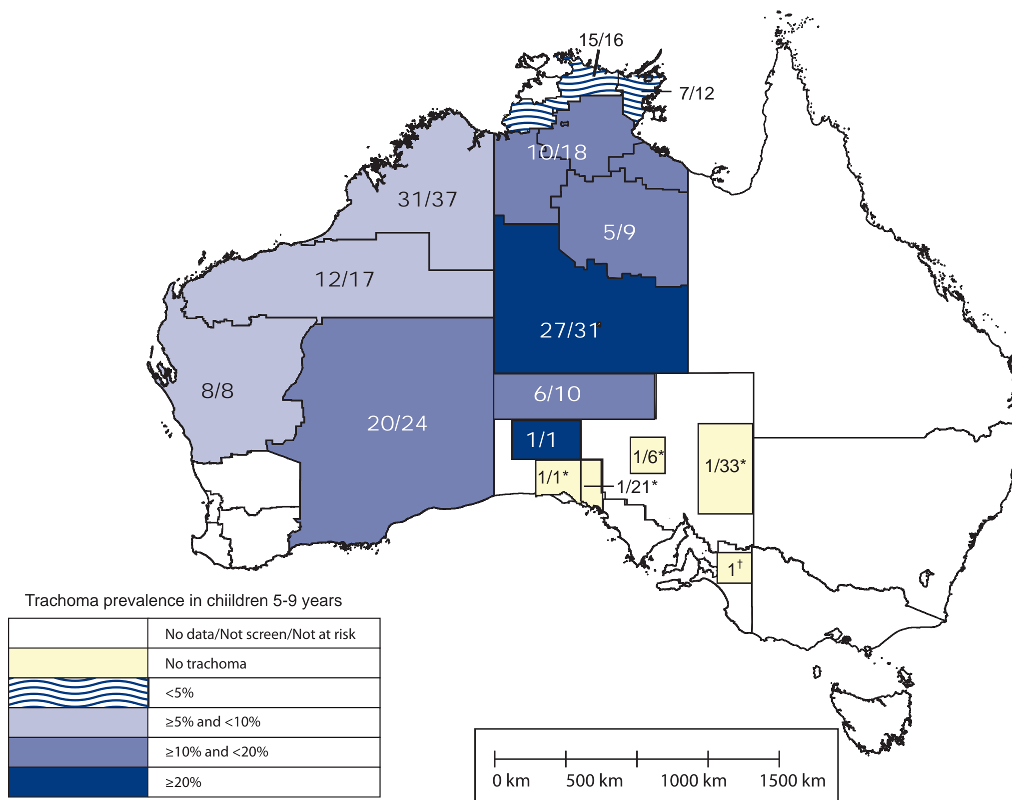
Screening coverage

Overall, 150 (63%) of 240 at-risk communities were screened for trachoma during the year 2010 (Table 1 and Figure 1). Within these communities, 6,762 (11.5%) of the estimated 58,429 resident children aged 1–14 years at risk of trachoma were screened. The proportion of children aged 1–14 years in at-risk communities who were screened was 45% for the Northern Territory, 37% for Western Australia and 3% for South Australia (Table 1). Compared with previous years, both the number of at-risk communities screened in the Northern Territory and Western Australia, and the proportion of children screened within these communities increased in 2010 (Figure 2). Screening coverage was greatest in the 5–9 year age group, at an average of 57% of children in at-risk communities (Table 1).

Clean face prevalence

In 2010, the prevalence of clean faces in screened populations was 80% overall, and among children aged 1–14 years it was 80% in the Northern Territory, 45% in South Australia and 81% in Western Australia (Table 1).

Figure 1: Number of at-risk communities screened and trachoma prevalence,* 2010



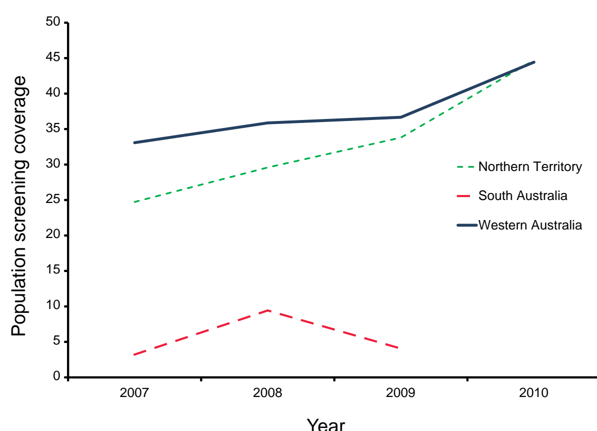
The numerator and denominator associated with each region refer to the number of communities screened and the number of at-risk communities within each region, respectively.

Colours denote the level of trachoma prevalence among children aged 5–9 years within the region.

* Prevalence is reported for children aged 5–9 years except in South Australia where data were only provided for the age grouping 1–14 years.

† Less than 10 children screened and number of communities at risk not known.

Figure 2: Population screening coverage* of children aged 5–9 years, 2007 to 2010, by year and jurisdiction



* Calculated as the number of children screened (in at-risk and not at-risk communities) in region containing at least one community at-risk divided by the estimated population of region.

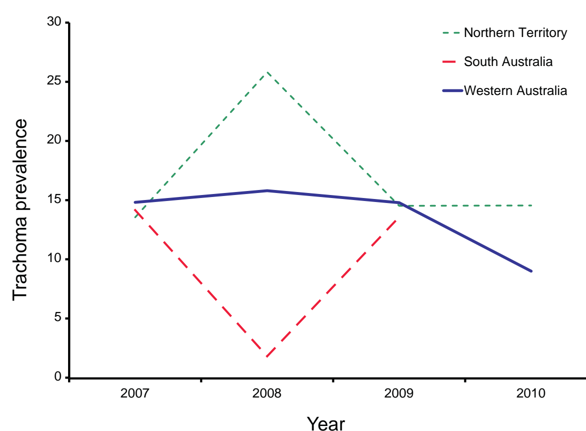
Trachoma prevalence

The prevalence of trachoma among children aged 1–14 years in at-risk communities who were screened was 11% (Table 1). In screened communities, 36% (52/146) had no trachoma detected, while 44% (64/146) had a prevalence of trachoma greater than 10% (Table 2). The prevalence of trachoma was 17% in South Australia, 12% in the Northern Territory and 9% in Western Australia (Table 1). In 2010 there was no significant change in the prevalence of trachoma among children aged 5–9 years screened in the Northern Territory (Figure 3), nor among the 1–14 year age group screened in South Australia, compared with the previous year. A significant decrease in trachoma prevalence was detected among communities screened every year from 2007 to 2010 in Western Australia (trend, $P<0.001$) (Figures 3 and 4). Data to examine time trends in trachoma prevalence were not available for South Australia. Five communities defined as potentially at-risk, but not designated at-risk, were screened for trachoma in 2010: one in the Northern Territory, one in South Australia, and three in Western Australia. Trachoma was found in all three of the Western Australian communities but not in the other two potentially at-risk communities.

Treatment coverage

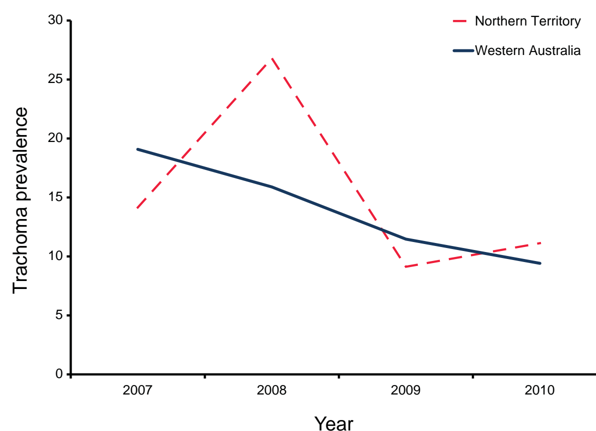
In the Northern Territory and Western Australia, cases requiring treatment were detected in 98 (73%) of the 135 communities that were screened. In 91 communities, both trachoma cases and their contacts were treated. Treatment coverage of cases and contacts was 64% in the Northern Territory, 90% in Western Australia and 70% across both jurisdictions combined. Data on treatment coverage were not available for South Australia (Table 3).

Figure 3: Trachoma prevalence in screened* children aged 5–9 years, 2007 to 2010, by year and jurisdiction



* Includes children in communities screened but not at risk.

Figure 4: Trachoma prevalence in communities consistently screened* 2007 to 2010, by year and jurisdiction



* Prevalence is for children aged 5–9 years in communities where more than 10 children were screened.

Trichiasis

A total of 1,036 adults (8.3%) of an estimated at-risk population of 12,557 were reported to have been screened across the Northern Territory, South Australia and Western Australia (Table 4). Nine cases of trichiasis were reported in the Northern Territory, 13 cases in South Australia and none in Western Australia, giving an overall prevalence among adults screened of 2%. No data were available regarding the extent of surgery for trichiasis in 2010.

Table 1: Trachoma screening coverage and prevalence and clean face prevalence, 2010, by state or territory

	Northern Territory				South Australia				Western Australia				Total			
Number of communities at risk	86				71				83				240			
Number of communities screened (% of all communities)	64 (74%)				11 (15%)				75 (90%)				150 (63%)			
Age group (years)	**1–4**	**5–9**	**10–14**	**1–14**	**1–4**	**5–9**	**10–14**	**1–14**	**1–4**	**5–9**	**10–14**	**1–14**	**1–4**	**5–9**	**10–14**	**1–14**
Estimated number of Aboriginal children at risk	2,843	3,705	3,395	9,943	818	1,029	1,125	2,972	1,724	2,300	1,980	6,004	5,384	7,034	6,500	18,918
Children examined for clean face	344	2,468	1,639	4,451	N/A	N/A	N/A	86	205	1570	501	2,276	N/A	N/A	N/A	6,813
Children with clean face	224	1,836	1,483	3,543	N/A	N/A	N/A	39	132	1241	461	1,834	N/A	N/A	N/A	5,416
Clean face prevalence	65%	74%	90%	80%	N/A	N/A	N/A	45%	64%	79%	92%	81%	N/A	N/A	N/A	79%
Children examined for trachoma	345	2,468	1,628	4,441	N/A	N/A	N/A	95	202	1,545	503	2,250	N/A	N/A	N/A	6,786
Trachoma screening coverage	12%	67%	48%	45%	N/A	N/A	N/A	3%	12%	67%	25%	37%	N/A	N/A	N/A	36%
Children with active trachoma	42	359	125	526	N/A	N/A	N/A	16	25	151	32	208	N/A	N/A	N/A	750
Active trachoma prevalence	12%	15%	8%	12%	N/A	N/A	N/A	17%	12%	10%	6%	9%	N/A	N/A	N/A	11%
Trachoma prevalence 1–9 years	14%				N/A				10%				13%*			
Trachoma prevalence 1–9 years (weighted by population)*	14%				N/A				11%				13%*			

* Calculated as the proportions of children with active trachoma in age groups 1-4 and 5-9 years, weighted by the estimated population sizes of each age group. This was undertaken in order to account for uneven coverage with respect to age groups.

Table 2: Number of communities, children aged 1–14 years, 2010, by trachoma prevalence range

Prevalence	At-risk communities							
	Northern Territory		South Australia		Western Australia		Total	
0%	15	23%	7	64%	30	42%	52	36%
>0% but <5%	9	14%	0	0%	5	7%	14	10%
≥5% but <10%	9	14%	0	0%	7	10%	16	11%
≥10%	31	48%	4	36%	29	41%	64	44%
Total	64		11		71		146	

Table 3: Trachoma treatment coverage 2010, by jurisdiction

	Northern Territory					Western Australia					Total				
Number of communities at risk	86					83					240				
Number of communities requiring treatment	50					48					98				
Age group (years)	**1–4**	**5–9**	**10–14**	**15+**	**All**	**1–4**	**5–9**	**10–14**	**15+**	**All**	**1–4**	**5–9**	**10–14**	**15+**	**All**
Active cases requiring treatment	42	360	125	N/A	527	25	151	32	N/A	208	67	511	157	N/A	735
Active cases who received treatment within 2 weeks	25	145	47		217	24	148	32		204	49	293	79		421
% Active cases received treatment					41%					98%					57%
Estimated contacts requiring treatment					4,257					1,181					5,438
Number of contacts who received treatment	370	505	381	1,595	2,851	97	271	149	537	1,054	467	776	530	2,132	3,905
Estimated overall treatment coverage					67%					89%					72%

N/A Not available

Table 4: Trichiasis screening coverage, prevalence and treatment among adults aged over 40 years, 2010, by jurisdiction

	Northern Territory		South Australia		Western Australia		Total	
	n	%	n	%	n	%	n	%
Estimated adult population of at-risk communities	6,509		2,297		3,751		12,557	
Number of communities at-risk	86		72		86		243	
Number of communities screened for trichiasis	18	21	12	38	14	17	34	17
Adults examined (% estimated adult population of at-risk communities)	221	3	438	19	377	10	1,036	8
Number with trichiasis (% of adults screened)	13	6	9	2	0		22	2
Offered ophthalmic consultation	12		0		0		12	
Surgery in past 12 months	1		0		1		1	

Discussion

The continuing presence of endemic trachoma in Aboriginal communities in Australia in 2010 highlights the need to prioritise its elimination. In 2010, a substantial amount of funding was provided to the jurisdictions by the Australian Government to increase activities to eliminate trachoma. This is reflected by some increase in community and population screening coverage, some reported increases in health promotion resources and programs, and in the number of personnel assigned to targeting trachoma elimination and promotion of hygiene and environmental improvements. It is anticipated that this should lead to a reduction in endemic trachoma in the next few years.

Screening coverage

Coverage can be measured as either the proportion of communities or the proportion of individuals screened. In 2010, community coverage levels were relatively high in the Northern Territory and Western Australia but low in South Australia. Individual screening coverage levels were low across all 3 jurisdictions. Interpretation of the coverage data is influenced by the accuracy of community population size estimates, the school-based approach to screening, and the designation of communities as 'at-risk'. Estimates of population sizes of communities were based on projections from census data. However, the estimates may not accurately reflect population sizes at the time of screening due to the small size of many communities and high mobility of many community members.

Most children were screened through school-based programs; consequently, screening rates were greater in the 5–9 and 10–14 year age groups than in the 1–4 year age group, even though the youngest group is recognised as being at highest risk of trachoma. In the 1–4 year age group, most children screened were at the upper end of the age range and attending preschools, kindergartens or play groups linked to the schools. Extending trachoma screening to other programs that target younger children would improve coverage in these important age ranges.

The designation of at-risk status does not appear to have been systematically reviewed in any jurisdiction. Data collected in Western Australia in 2010, as well as previous annual national trachoma reports and in the National Indigenous Eye Health Survey conducted in 2008, demonstrate that communities considered not-at-risk may in fact harbour endemic trachoma. The NTSRU and jurisdictional stakeholders should collaborate to establish a register of communities that includes the at-risk status and trachoma screening history of each community. This would provide guidance to jurisdictions regarding communities to be targeted for screened and to improve consistency in estimating and monitoring screening coverage over time.

Trachoma prevalence

Eleven per cent of all children screened across the jurisdictions were found to have trachoma, with varying rates of prevalence in at-risk communities. This demonstrates that Australia continues to have endemic levels of trachoma. The target set by both CDNA and the WHO is a community-level prevalence among children aged 1–9 years of less than 5%. Compared with previous years, the proportion of children with active trachoma decreased in Western Australia, remained stable in the Northern Territory, and increased in South Australia, although this estimate is based on small numbers of children screened. Among the at-risk communities that were screened annually from 2007 to 2010, there were clear decreasing trends in trachoma prevalence in Western Australia but not in the Northern Territory.

It is likely that the decrease in trachoma prevalence observed in at-risk Western Australia communities is real. The prevalence of clean faces has been at approximately the same levels in both Western Australia and the Northern Territory, as has the proportion of children screened. However, a greater proportion of communities designated as at-risk in Western Australia have been consistently screened compared with the Northern Territory. Furthermore, screening in Western Australia occurs within a shorter time period than in the Northern Territory, which also enables reductions in the interval from screening to treatment. Re-infection might occur more frequently in the Northern Territory, either in the interval between screening and treatment, or through contacts between people in screened and unscreened communities. This hypothesis requires further critical examination.

Trachoma treatment

The CDNA guidelines recommend treating active cases as well as their household contacts and community members when required, within 2 weeks of screening. Nationally, just over one-third of cases detected through screening, and their contacts, were treated according to this recommendation. In Western Australia, treatment coverage was 89% in 2010, an increase from 70% in 2009[5] and exceeding the WHO target of 80%. In the Northern Territory, 20% of cases and contacts were treated within the recommended time period. Treatment of active cases and their appropriate contacts, irrespective of the interval since screening, is also an important indicator of appropriate management. When treatment coverage is estimated irrespective of timing, coverage of contacts was 90% in Western Australia and 65% in the Northern Territory.

Data on active cases treated outside of the 2-week period following screening were not collected in 2010. The success in achieving treatment goals in Western Australia may be attributed to the method of program delivery, which involves screening and treatment all taking place over the same 2-week period across its regions. In the Northern Territory, unusually high rainfall during a normally dry weather season in 2010 contributed to some delays in treatment. South Australia did not provide data regarding treatment of cases or contacts. CDNA guidelines recommend different treatment strategies according to the prevalence and clustering of active cases. These guidelines have been interpreted differently by different stakeholders. For this report, a second method was used to estimate treatment coverage. This method leads to substantially lower treatment coverage estimates. Resolution of the inconsistencies in interpretation of the guidelines for treating contacts is required to ensure that best practice is followed.

Trichiasis

Screening coverage for trichiasis was low across all jurisdictions. Among adults aged 40 years or older, coverage was 3% in the Northern Territory, 19% in South Australia and 10% in Western Australia. The low levels suggest that current approaches to integrate trichiasis screening with other programs are not achieving their goal. Furthermore, it is not clear that the trichiasis screening is being optimally targeted, because communities that are currently at-risk for trachoma may not reflect adult populations exposed to trachoma as children, due to the changing risk status of the communities. Establishing a register of all remote communities may assist in establishing better records of communities that would have substantial adult populations affected by trichiasis.

Referral processes were reported to be functioning within 97% of communities in the Northern Territory and 45% of communities in Western Australia; however, this does not assess the effectiveness of the systems. Ophthalmic consultation and surgery reports do not reflect the extent of actual service delivery. Greater collaboration in developing data transfer processes with stakeholders and jurisdictions that provide ophthalmic consultations and trichiasis surgery is required.

Facial cleanliness

At a community level, lower levels of facial cleanliness are a recognised risk factor for trachoma.[1] For this reason, facial cleanliness is a major component of the SAFE strategy. The overall proportion of children screened who had clean faces remained stable, with 80% of children screened in the Northern Territory, 82% prevalence in Western Australia and 51% in South Australia having clean faces.

Measures of facial cleanliness may not be a true estimation of actual risk. This could be due to the clean face definition specified in the CDNA guidelines, 'absence of dirt or crusting on cheeks or forehead',[1] which does not align with actual risk of transmission. Risk of transmission may be more accurately reflected by the presence of ocular and nasal discharge.[6]

Data quality and surveillance systems

A number of issues must be addressed if the national trachoma surveillance system is to provide optimal support for control programs. These include the better definition of population denominators, designation of at-risk status for communities and the interpretation of the CDNA trachoma control guidelines. There are also issues of data quality to be addressed, particularly with regard to inconsistent and missing items. For example, counts by age group were not uniformly provided, and data were missing for numbers treated and for components of the SAFE strategy implemented.

The NTSRU will work with the National Trachoma Surveillance Reference Group and jurisdictions to address these issues. It will also undertake the development of a web-based data entry system and collaborate with jurisdictions and Aboriginal community controlled health organisations to facilitate the transfer of trachoma data from clinic-based health information systems to jurisdictional and national databases. These changes will reduce delays in data transfer and minimise human error in data transfer.

Particular attention is required for South Australia, where there has not previously been a systematic screening and treatment program. The data provided for the 2010 report show modest community coverage, low population coverage and inconsistent reporting of other variables. The establishment of a contract between the Department of Health and Ageing and the South Australian Government in late 2010 to conduct trachoma control activities should lead to a substantial improvement in program coverage and the quantity and quality of surveillance data from South Australia.

Recommendations for trachoma surveillance

Whilst improvements have occurred over the past 5 reporting years, data gaps and other limitations noted above prevent precise estimates of disease prevalence and program delivery and impact. To overcome these barriers, we recommend:

- the establishment of a web-based system that will allow efficient transfer of data between jurisdictions and the NTSRU, as well as the generation of reports in a timely manner;

- that jurisdictional data collection protocols and trachoma management guidelines are made consistent with the CDNA guidelines, ensuring that there is no ambiguity in the interpretation of the guidelines;
- the establishment of a systematic and accountable procedure for updating the designation of communities as at-risk or not-at-risk, including a register of communities;
- the extension of screening and reporting of trachoma to other Australian jurisdictions where communities may be at risk of trachoma; and
- that a review and formalisation of procedures (and agreements as needed) be conducted in the following areas:
  - estimation of denominators for population sizes of communities;
  - collection of antibiotic resistance data;
  - collection of environmental data;
  - collection of information on health promotion information, education and communication material and program activity; and
  - trichiasis screening processes and management, referral systems and related data collection including data pertaining to surgery for trichiasis.

These recommendations, along with greater collaboration within and between jurisdictions and communities, should enable greater monitoring and evaluation of trachoma control efforts during the important next few years over which Australia aims to move towards trachoma elimination.

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Annual report of the Australian Meningococcal Surveillance Programme, 2011

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Abstract

In 2011, there were 241 laboratory-confirmed cases of invasive meningococcal disease (IMD) analysed by the National Neisseria Network, which represented 100% of cases notified to the National Notifiable Diseases Surveillance System. One hundred and twenty-five isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were available for which the phenotypes (serogroup, serotype and serosubtype) and/or genotype and antibiotic susceptibility were determined. An additional 116 cases were confirmed by non-culture based methods (95 by nucleic acid amplification testing (NAAT) and 21 by serology), and where possible, serotyping was determined. Nationally, 179 (83.6%) laboratory-confirmed cases, where a serogroup was determined, were infected with serogroup B; 9 (4.2%) with serogroup C; 11 (5.2%) with serogroup W135 and 15 (7%) with serogroup Y meningococci. In 2011 there was a modest increase in the number of cases of IMD notified from that reported in 2010 (214). However, with the exception of 2010, this was the lowest number of laboratory confirmed IMD cases since surveillance data were recorded. Primary and secondary disease peaks were observed in those aged 4 years or less and in adolescents (15–19 years) and young adults respectively (20–24 years). There was also a disease peak observed in those aged 45–64 years. Serogroup B cases predominated in all age groups and jurisdictions. In 2011, the most common phenotype circulating in Australia was B:4:P1.7, corresponding to the *porA* genotype P1.7,2-4. Serogroup C cases were again numerically low, as were serogroups W135 and Y, however there was an increase in incidence of serogroup Y cases (7 in 2010, 15 in 2011). The proportion of isolates with decreased susceptibility to the penicillin group of antibiotics minimal inhibitory concentration (MIC) (0.06 to 0.5 mg/L) was 84.6% and 1 isolate exhibited relative resistance to penicillin (MIC = 1.0 mg/L). All isolates remained susceptible to ceftriaxone and ciprofloxacin. One isolate had reduced susceptibility to rifampicin (MIC = 0.5 mg/L). *Commun Dis Intell* 2012;36(3):E251–E262.

Keywords: antibiotic resistance; disease surveillance; meningococcal disease; *Neisseria meningitidis*

Introduction

The National Neisseria Network (NNN) is a long-term collaborative program for the laboratory surveillance of the pathogenic *Neisseria* species: *Neisseria meningitidis* and *Neisseria gonorrhoeae*. Since 1994 the NNN has operated through a network of reference laboratories in each state and territory to provide a national laboratory-based program for the examination of *N. meningitidis* from cases of invasive meningococcal disease (IMD).[1] The NNN supplies data on the phenotype and/or the genotype of invasive meningococci, and their antibiotic susceptibility, supplementing clinical notification data from the National Notifiable Diseases Surveillance System (NNDSS). The NNN receives samples for analysis from about 90% (range 85%–100% 2004–2011) of IMD cases notified to NNDSS.[2] The NNN annual reports are published in *Communicable Diseases Intelligence*.[3]

The characteristics of the meningococci responsible for IMD are important both for individual patient management and to tailor the public health response for outbreaks or case clusters locally and nationally. The introduction of publicly funded conjugate serogroup C meningococcal vaccine to the National Immunisation Program in 2003 (with a catch-up program for those aged 1–19 years that ran until May 2007) has seen a significant and sustained reduction in the number of cases of IMD evident after 2004.[2] However, IMD remains an issue of public health concern in Australia. The success of any further vaccine initiatives in Australia is dependent upon detailed analysis of the *N. meningitidis* isolates circulating locally. This report provides relevant details of cases of IMD confirmed by laboratory testing in Australia in 2011.

Methods

Isolate based invasive meningococcal disease cases

Case confirmation

Case confirmation was based upon isolation of, or positive nucleic acid amplification testing (NAAT) for, *N. meningitidis* from a normally sterile site; or by positive serology, and defined as IMD according to Public Health Laboratory Network criteria.[4]

Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was sought. The isolate-based subset of the program categorised cases on the basis of site of isolation of the organism. Where an isolate was grown from both blood and cerebrospinal fluid (CSF) cultures in the same patient, the case was classified as one of meningitis. It is recognised that the total number of cases, and particularly the number of cases of meningitis, is underestimated because no lumbar puncture was performed, or was delayed and the culture was sterile. However; the above approach has been used since the beginning of this program[1] and is continued for comparative purposes.

Phenotyping and genotyping

Phenotyping of invasive isolates of meningococci by serotyping and serosubtyping was based on the detection of outer membrane protein (porin) antigens using a standard set of monoclonal antibodies obtained from The Netherlands National Institute for Public Health. Increasingly, sequencing of products derived from amplification of the porin genes *porA, porB* and *FetA* (genotyping) is used to supplement and supplant meningococcal serotyping analyses based on the use of monoclonal antibodies.

Antibiotic susceptibility

Antibiotic susceptibility was assessed by determining the MIC to antibiotics used for therapeutic and prophylactic purposes. This program uses the following parameters to define the various levels of penicillin susceptibility or resistance when determined by a standardised agar plate dilution technique:[5]

Sensitive: MIC ≤ 0.03 mg/L

Less sensitive: MIC 0.06–0.5 mg/L

Relatively resistant: MIC ≥ 1 mg/L

Strains with MIC values that place them in the category of sensitive or less sensitive would be considered to be amenable to penicillin therapy when used in currently recommended doses. However precise the MIC, outcome correlations are difficult to obtain because of the nature of IMD.

Non-culture based laboratory-confirmed cases

Additional laboratory confirmation of suspected cases of IMD was obtained by means of non-culture based methods (NAAT and serology). NAAT testing is essentially by polymerase chain reaction (PCR) techniques[6] that demonstrate the presence of meningococcal-specific nucleic acid in appropriate samples and has been progressively introduced and updated in the different jurisdictions. Data from the results of these investigations were included for the first time in the 1999 report. The serological results are based on the demonstration of IgM antibody by enzyme immunoassay to *N. meningitidis* outer membrane protein using the methods and test criteria of the Health Protection Agency UK as assessed for Australian conditions.[7–10] Where age, sex and outcome data for patients with non-culture based diagnoses are available these were also recorded. The site of a sample of a positive NAAT is also used to define the clinical syndrome.

Results

Aggregated data on cases confirmed by culture and non-culture based methods

Number of laboratory confirmed cases

There were 241 laboratory confirmed cases of IMD in Australia in 2011 compared with 214 in 2010 and an average of 252 over the past 5 years (Table 1). In 2011, the number of laboratory confirmed cases of IMD was the same as the number of IMD notifi-

Table 1: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2011, by serogroup and state or territory

State or territory	Serogroup						Total
	B	C	Y	W135	NG	ND	
ACT	5	0	0	0	0	0	5
NSW	35	2	7	4	5	14	67
NT	1	0	0	0	0	0	1
Qld	50	3	3	1	0	4	61
SA	17	2	0	2	0	1	22
Tas	6	1	0	3	0	0	10
Vic	45	1	3	1	0	3	53
WA	20	0	2	0	0	0	22
Australia	179	9	15	11	5	22	241

NG Non-groupable

ND Non-determined, samples were examined by nucleic acid amplification test and serological methods.

cations to NNDSS.[2] In 125 cases (52%), a positive culture was obtained with or without a positive non-culture based test and 116 (48%) cases were confirmed by a non-culture based method alone.

The highest number of laboratory confirmed cases was from New South Wales (67 cases), which decreased from 77 cases in 2010. The number of laboratory confirmed cases in Queensland in 2011 increased to 61, from 47 in 2010. Victoria showed an increase to 53 cases from 38 cases in 2010. There were very slight increases from 2010 in Western Australia, South Australia and the Australian Capital Territory.

Seasonality

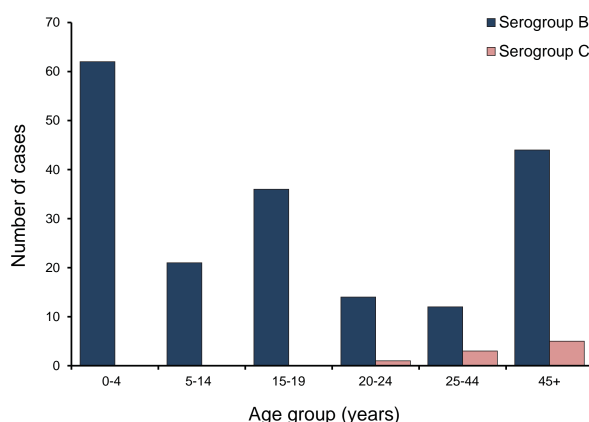
Fifty-seven cases occurred between 1 January and 31 March 2011, 56 between 1 April and 30 June, 73 between 1 July and 30 September and 55 between 1 October and 31 December. A winter peak of meningococcal disease is usual and the above pattern was also observed in 2007, 2008, 2009 and 2010.

Age distribution

Nationally, the peak incidence of meningococcal disease was in children aged less than 5 years, similar to previous years (Figure 1 and Table 2). Children aged less than 5 years accounted for 68 cases (28% of the total) in 2011, down from 33%–36% in 2007–2010.

A secondary disease peak was observed in previous years amongst adolescents and young adults aged 15–24 years. The 41 confirmed cases (17% of all cases) in those aged 15–19 years in 2011 was more than the number reported for 2010, but is similar to the range reported in this age group in the years 2007–2009 (19%–20%). There were 23 cases (9.5% of the total) 20–24 year age group, a marked decrease compared with the 22%–31% reported in this age group in the years 2007–2010.

Figure 1: Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2011, by age group



Serogroup data

The serogroup was determined in 219 of the 241 laboratory confirmed cases of IMD in 2011 (Table 1). Of these, 179 (84%) were serogroup B and 9 (4.2%) were serogroup C. The proportion of cases that were serogroup B was little changed from the proportion reported between 2006 and 2010 (85%–88%). There is a continuing decrease in the number of cases of serogroup C. In 2011 there were 11 cases (5.2%) of serogroup W135, which was little changed from 2010 (4.5%). There were 15 cases of serogroup Y (7%), which represented an increase from 2009–2010 (3.5%–4%). With the continuing low numbers of serogroup C infections, serogroup B meningococci predominated in all age groups and jurisdictional differences in serogroup distribution were not evident.

In 2011, total case numbers, and the number of cases due to serogroup B in those aged 14 years or less was similar to 2010, but was lower than the years 2004–2009 (Table 3), and there were no serogroup C cases. In people aged 15–19 years, the proportion of serogroup B cases decreased to 88% in 2011 from 94% in 2010. In people aged 20–24 years, the number of serogroup B cases (14) was similar to the period 2008–2010 (14–15) but there was a marked decrease in the proportion of serogroup B cases, from 80%–88% between 2007 and 2010 to 61% in 2011. There was an increase in the number of serogroup Y and W135 cases in this age group. In people aged 25 years or more, there was an increase in the number of serogroup B cases compared with 2010, but a continuing decline in the proportion of serogroup B cases. This may be in part be explained by an increase, in this age category for 2011, in the number of serological IMD diagnoses (and thus serogroup not determined). The reasons for this are unclear, however ease of blood collection at the point of care may have played a role. The proportion of serogroup C cases in this age group remained similar to the period 2006–2010.

Phenotypes of invasive meningococcal isolates

Serogroup B meningococci are typically of heterogeneous phenotypes. In 2011, the phenotypes of invasive isolates, based on a determination of their serogroup, serotype and serosubtype, were analysed for New South Wales, the Australian Capital Territory and Tasmania (Table 4). Serogroup B meningococci are in general more difficult to characterise by serological methods and a number could not be phenotyped. A total of 43 were serotyped. Twenty-nine of these were serogroup B, of which six were serotype 15 and four of these were serosubtype P1.7, which has been circulating in Australia for many years; seven were serotype 4, five (all from New South Wales) of which were serosubtype P1.7. Eight serogroup B were non-typeable.

Table 2: All laboratory confirmed cases of invasive meningococcal disease, Australia, 2011, by age, state or territory and serogroup

State or territory	Serogroup	Age group										Total
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
ACT	B	1	1	1	0	0	0	0	2	0	0	5
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	1	1	1	0	0	0	0	2	0	0	5
NSW	B	4	7	2	1	6	6	2	3	4	0	35
	C	0	0	0	0	0	0	1	0	1	0	2
	Total	5	12	2	3	9	11	11	8	6	0	67
NT	B	1	0	0	0	0	0	0	0	0	0	1
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	1	0	0	0	0	0	0	0	0	0	1
Qld	B	6	10	3	6	14	2	1	6	2	0	50
	C	0	0	0	0	0	1	2	0	0	0	3
	Total	6	10	3	6	17	4	4	6	5	0	61
SA	B	3	2	1	0	3	1	3	2	2	0	17
	C	0	0	0	0	0	0	0	2	0	0	2
	Total	3	2	1	0	3	1	3	6	3	0	22
Tas	B	1	0	0	0	1	1	1	0	2	0	6
	C	0	0	0	0	0	0	0	0	1	0	1
	Total	1	0	0	0	1	3	1	1	3	0	10
Vic	B	14	8	2	1	8	3	3	5	1	0	45
	C	0	0	0	0	0	0	0	1	0	0	1
	Total	14	8	2	1	9	3	6	8	1	0	53
WA	B	2	2	2	2	4	1	2	4	1	0	20
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	2	2	2	2	4	1	2	4	3	0	22
Australia	B	32	30	11	10	36	14	12	22	12	0	179
	C	0	0	0	0	0	1	3	3	2	0	9
	Total	33	35	11	13	41	23	27	35	21	0	241
	% B of within age group	97	85.7	100	76.9	87.8	60.9	44.4	62.9	57.1	0	83.6

Other: cases diagnosed by serology; or by nucleic acid amplification test where the serogroup was not determined.

Three serogroup C strains were phenotyped and two (both from New South Wales) were serotype 2a. This phenotype has predominated in serogroup C meningococci in Australia for many years. Of these two, one was phenotyped C:2a:P1.5, one C:2a strain was non-subtypeable. One serogroup C strain was non-typeable and non-subtypeable. There is continuing interest in the presence of any serogroup B or serogroup C meningococci of serotypes that indicate the possibility of genetic recombination events. Among serogroup C strains, phenotype C: 2a:P1.4 had been of particular interest where it figured prominently in Victorian data in previous years. For example, in 2003 there were 29 serogroup C isolates of this serotype/serosubtype detected nationally with 21 detected in 2004 and 8 detected in 2005. However, other than the two C:2a:P1.4 meningococcal isolates reported in New South Wales in 2010, no isolates with this phenotype or its equivalent genotype were seen in other jurisdictions in 2009 or 2011.

Genotyping data of invasive meningococcal samples (culture or nucleic acid amplification test products)

Sequencing products derived from amplification of the variable region *porA, porB* and *FetA* genes is used in an increasing number of jurisdictions in place of

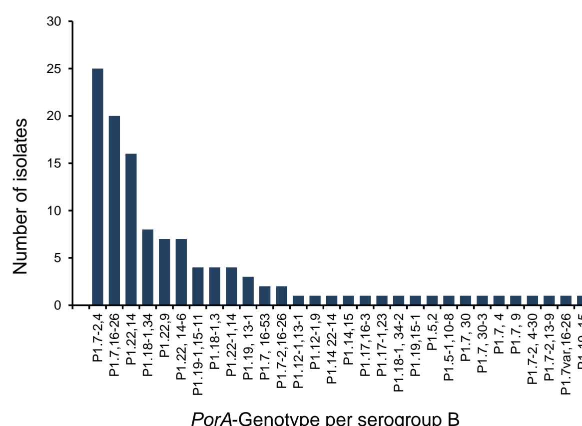
Table 3: A comparison of the number and proportion of serogroup B and serogroup C laboratory-confirmed cases, 2004 to 2011, by known cases

		Age group									
		< 5		5–14		15 –19		20–24		25+	
Year	Serogroup	n	%	n	%	n	%	n	%	n	%
2011	B	62	91	21	88	36	88	14	61	46	55
	C	0	0	0	0	0	0	1	4	8	10
	All*	68		24		41		23		83	
2010	B	61	85	19	76	29	94	15	88	39	60
	C	2	3	3	12	1	3	0	0	10	15
	All*	72		25		31		17		65	
2009	B	72	94	21	75	38	83	14	88	41	76
	C	2	2.6	3	11	1	2.2	1	6.3	4	7
	All*	77		28		46		16		55	
2008	B	82	89	23	96	42	91.3	15	83	57	85
	C	4	4.4	0	0	1	2.2	2	11.1	8	11
	All*	92		24		46		18		67	
2007	B	83	90	19	83	48	91	24	80	49	75
	C	4	4	0	0	2	4	3	10	8	12
	All	92		23		53		30		65	
2006	B	93	93	21	84	40	82	21	70	38	61
	C	2	2	3	12	4	8.2	7	23	10	16
	All	100		25		49		30		62	
2005	B	99	90	38	75	39	81	22	67	51	50
	C	6	5.5	510		4	8	8	24	27	27
	All	110		51		48		33		101	
2004	B	97	88	27	77	40	65	20	57	59	50
	C	6	5.5	5	14	17	28	11	31	32	27
	All	110		35		61		35		117	

* Cases where a serogroup was determined and patient's age was supplied.

serotyping using monoclonal antibodies. Since 2009, jurisdictions have moved to the use of genotyping. In 2011, genotyping data were available from all states and territories for 144/241 (60%) IMD cases. There was heterogeneity of typing data across jurisdictions with predominance of a few phenotypes or genotypes (Table 4). Figure 2 shows the collation of the national genotyping data of *porA* genotypes by number for all serogroup B confirmed cases of invasive meningococcal disease for 2011. The predominant *porA* genotypes include P1.7-2,4 (25 cases), P1.7,16-26 (20 cases), and P1.22,14-6 (16 cases). Figure 3 shows the collation of the national genotyping data of *porA* genotypes by number for serogroup C, Y and W135 in confirmed cases of invasive meningococcal disease for 2011.

Figure 2: Number of *porA*-genotypes* for serogroup B cases of invasive meningococcal disease, Australia, 2011



* Where genotype data were available

Table 4: Phenotypes (serotype, sero-subtype) and genotypes: *porB* variable region type, *porA* variable region, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease, 2011, by state or territory

State or territory	Sero-group	Phenotype				Genotype					
		Serotype	n	Sero-subtype	n	*porA*	n	*porB*	n	*FetA*	n
ACT	B	NT	1	NST	1	P1.7, 30	1				
		NT	1	P1.14, 22-14	1	P1.14 22-14	1				
		1	1	P1.6	1						
NSW	B	1	5	P1.14	2						
				P1.17	1	P1.17-1,23	1				
				P1.4	2						
		4	7	P1.15	1						
				P1.7	2						
				P1.7-2	3	P1.7-2,4	3				
				NT	1						
		15	6	P1.14	1						
				P1.7	4						
				NT	1						
		NT	5	P1.14	1	P1.22-1,14	1				
				P1.4	1						
				P1.9	2	P1.22,9	1	B,C,7,14b	1	F5-12	
				NT	1						
	C	2a	2	P1.5	1						
				NT	1						
	Y	14	2	P1.5	1						
				NT	1	P1.5-2,10-1	1				
		NT	4	P1.5,2	1						
				P1.6,3	1	P1.18-1,3	1				
				NT	2	P1.5-2,10-1	1				
	W135	1	1	P1.18	1	P1.18-1,16	1				
		NT	2	P1.4	1						
				P1.6,3	1						
NT											
Qld	B					P1-18-1,3	1			F1-5	1
						P1-19, 15	1			F5-2	1
						P1-19, 15-39	1			F5-1	1
						P1.12-1,13-1	1			F5-7	1
						P1.14,15	1			F1-64	1
						P1.18-1,34	3			F1-5	3
						P1.18-1, 34-2	1			F1-5	1
						P1.19, 13-1	3			F5-1	2
										ND	1
						P1.22,9	1			F1-55	1
						P1.22, 14	4			F4-7	1
										F5-9	3
						P1.22, 14-6	4			F1-5	1
										F5-5	1
										F5-88	1
										ND	1
						P1.7, 4	1			F1-5	1
						P1.7, 9	1			F3-20	1

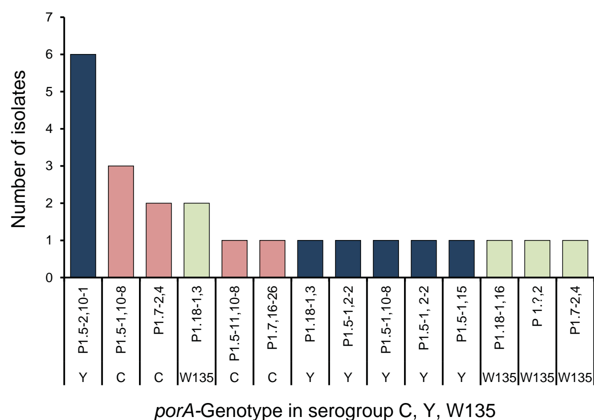
Table 4 *continued*: Phenotypes (serotype, sero-subtype) and genotypes: *porB* variable region type, *porA* variable region, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease, 2011, by state or territory

State or territory	Sero-group	Phenotype				Genotype					
		Serotype	n	Sero-subtype	n	*porA*	n	*porB*	n	*FetA*	n
Qld, *cont'd*						P1.7, 16-26	7			F2-9	1
										F3-3	5
										F5-19	1
						P1.7, 16-53	2			F3-3	2
						P1.7, 30-3	1			F1-2	1
						P1.7-2,4	11			F1-5	9
										F1-55	1
										F3-3	1
						P1.7-2, 4-30	1			F1-5	1
						P1.7-2,16-26	2			F3-3	2
	C					P1.5-1,10-8	2			F3-6	2
						P1.5-11,10-8	1			F3-6	1
	Y					P1.5-1, 2-2	1			F5-8	1
						P1.5-1,15	1			F5-1	1
						P1.5-2,10-1	1			F4-1	1
	W135					P1.18-1,3	1			F4-1	1
SA	B			P1.7-2,4	5	P1.7-2,4	5			F1-5	4
										F5-2	1
				P1.7,16-26	1	P1.7,16-26	1			F3-3	1
				P1.18-1,3	1	P1.18-1,3	1			F1-5	1
				P1.18-1,34	1	P1.18-1,34	1			F1-5	1
				P1.22,14	1	P1.22,14	1			F5-5	1
				P1.22,14-6	2	P1.22,14-6	2			F1-5	1
										F3-9	1
	C			P1.7-2,4	2	P1.7-2,4	2			F1-5	1
										F5-2	1
	W135			P 1.?,2	1	P 1.?,2	1			F1-1	1
Tas	B	A,A,A,Ba	1	P1.7var,16-26	1	P1.7var,16-26	1	A,A,A,Ba	1	F3-3	1
		19,Db,7c,14	1	P1.22,14	1	P1.22,14	1	19,Db,7c,14	1	F3-6	1
		NT	1	P1.22,14	1	P1.22,14	1	ND	1	NT	1
	C	4,B,7,14a	1	P1.7,16-26	1	P1.7,16-26	1	4,B,7,14a	1	F3-3	1
	W135	ND	1	P1.18-1,3	1	P1.18-1,3	1	ND	1	F3-6	1
Vic	B					P1.7-2,4	4	4,D,7,14a	1	F1-5	1
								19,Dvar,7b,14	1	F5-9	1
								ND	2	ND	2
						P1.7-2,16	1	New,Dvar,&b,Bvar	1	F3-3	1
						P1.7,16-26	8	A,A,A,Ba	5	F3-3	3
								ND	3	F3-3	3
						P1.7-2,13-9	1	ND	1	F1-5	1
						P1.12-1,9	1	4,D,7,14a	1	F1-5	1
						P1.17,16-3	1	19,Ab,7var,Aa	1	F5-5	1
						P1.18-1,3	2	ND	1	F3-9	1
								ND	1	ND	1
						P1.18-1,34	4	19,Ac,7a,1	2	F1-5	2

Table 4 *continued*: Phenotypes (serotype, sero-subtype) and genotypes: *porB* variable region type, *porA* variable region, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease, 2011, by state or territory

State or territory	Sero-group	Phenotype				Genotype					
		Serotype	n	Sero-subtype	n	*porA*	n	*porB*	n	*FetA*	n
Vic *cont'd*								4,D,7,14a(var)	1	F1-5	1
								ND	1	ND	1
						P1.19-1,15-11	4	B,C,7,14b	2	F5-1	2
								ND	2	F5-1	2
						P1.22,14	8	19,Ac,7a,1	3	F5-5	3
								ND	5	ND	5
						P1.22,9	5	B,C,7,14b	1	F1-55	1
								B,C,7,14b	1	F5-12	1
								new,Dvar,7b,Bvar	3	F5-12	3
						P1.22-1,14	1	4,B,7,14a	1	F3-9	1
						P1.5-1,10-8	1	4,D,7,14a	1	F1-5	1
	C					P1.5-1,10-8	1	ND	1	ND	1
	Y					P1.5-1,10-8	1	19,Db,7c,14	1	F1-3	1
						P1.5-2,10-1	2	19,Db,7c,14	1	F4-1	1
								ND	1	ND	1
	W135					P1.7-2,4	1	ND	1	F5-2	1
WA	B					P1.7,16-26	3			F3-3	2
										F3-6	1
						P1.7-2,4	2			F1-5	2
						P1.5,2	1			F1-7	1
						P1.19,15-1	1			F1-5	1
						P1.22-1,14	2			F4-1	1
										F5-2	1
						P1.22,14	1			F3-9	1
						P1.22,14-6	1			F3-9	1
	Y					P1.5-1,2-2	1			F5-8	1
						P1.5-2,10-1	1			F4-1	1

Figure 3: Number of *porA*-genotypes* for serogroup C, Y, W135 in cases of invasive meningococcal disease, Australia, 2011



* Where genotype data were available

Outcome data for invasive meningococcal disease for laboratory confirmed cases

Outcome data (survived or died) were available for only 25 (10%) of the 241 laboratory confirmed cases (Table 5). Five deaths were recorded in the 25 cases with outcome data available, all attributable to septicaemia. Four of these deaths were due to serogroup B infections and one to W135 infection. Outcome data were available for 18 of 179 cases with serogroup B infection and one of the 11 serogroup W135 infections, one of the 9 serogroup C infections and one of the 15 serogroup Y infections.

Anatomical source of samples for laboratory confirmed cases

There were 85 diagnoses of meningitis based on cultures or NAAT examination of CSF either alone

or with a positive blood sample; and 151 from blood samples (cultures or NAAT) alone (Table 6). Twenty-one cases were serologically positive where culture and NAAT were negative. Diagnoses made from sites other than blood, CSF or serum were brain tissue (2), lung tissue (1), pelvic fluid (1) and synovial fluid (1). Those diagnoses shown as culture positive may also have had positive NAAT and/or serology; however those shown as NAAT positive were culture negative with or without positive serology.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillins

Susceptibility to penicillin and other antibiotics was determined for one hundred and twenty-five meningococcal isolates of the 241 cases (52%). Using defined criteria, 108 isolates (86.4%) were less sensitive to penicillin in the MIC range 0.06–0.5 mg/L and (12.8%) fully sensitive (MIC 0.03 mg/L or less). One isolate (0.8%) was relatively resistant (MIC = 1.0 mg/L). The proportion of less sensitive strains (86.4%) is higher than that reported in 2007–2010 (range 67%–80%)

Other antibiotics

All isolates were fully susceptible to ceftriaxone and by extrapolation to other third generation cephalosporins. All isolates were fully susceptible to ciprofloxacin and there was 1 isolate with altered susceptibility to rifampicin (MIC = 0.5 mg/L).

Discussion

In 2011 there were 241 IMD cases laboratory confirmed by the NNN, representing 100% of notifications to the NNDSS.[2] There was a steady decrease in the number of notifications of IMD in Australia between 2003 and 2010, with numbers in 2010 less than half those in 2003 (558 and 214 cases respectively), but in 2011 there was a slight increase. The number of cases in 2011 was lower than the number of notified cases reported in any year from

Table 5: Outcome data for laboratory confirmed cases of invasive meningococcal disease, Australia, 2011, by syndrome and serogroup

		Serogroup					
Disease type	Outcome	B	C	Y	W135	NG	Total
Meningitis	Survived	4	0	0	1	0	5
	Died	0	0	0	0	0	0
	Unknown	70	3	3	2	4	82
	Total	74	3	3	3	4	87
Septicaemia	Survived	10	1	1	3	0	15
	Died	4	0	0	1	0	5
	Unknown	91	5	11	4	23	134
	Total	105	6	12	8	23	154
All cases	Survived	14	1	1	4	0	20
	Died	4	0	0	1	0	5
	Unknown	161	8	14	6	27	212
	Total	179	9	15	11	27	241

NG Serogroup not groupable or not determined.

Table 6: Anatomical source of samples positive for a laboratory confirmed case of invasive meningococcal disease, Australia, 2011

Specimen	Bacterial isolate	NAAT*	Serology†	Total
Blood	92	38	21	151
CSF +/– blood	31	54	0	85
Other‡	2	3	0	5
Total	125	95	21	241

* Nucleic acid amplification test (NAAT) positive in the absence of a positive culture.

† Serology positive in the absence of positive culture or NAAT.

‡ Joint, tissue and fluid samples (pelvic fluid (1), lung (1), brain tissue (2), joint fluid (1))

1991 to 2009 (range 259–687).[2] The proportion of IMD notifications with laboratory confirmation has increased from 88% to 100% since 2004.

The distribution of serogroup B IMD cases is essentially the same as that reported for 2006 to 2010. The proportion of serogroup C cases continues to decline as a result of the introduction of the serogroup C vaccine in 2003. There was an increase in the number of serogroup Y cases compared with the period 2006 to 2010. This will need to be monitored to determine if this is the beginning of an increasing trend.

There was a decrease in the number of culture confirmed cases from previous years with a corresponding increase in the number of NAAT confirmed cases. This may reflect the increasing availability of NAAT assays for diagnosis of IMD. Attention is drawn to earlier AMSP reports that explain the differences between the number of clinically notified cases and laboratory confirmed cases,[11] however in 2011, for the first time, there was a corresponding number of laboratory confirmed cases from the Australian Meningococcal Surveillance Programme data and NNDSS notifications. It should also be noted that surveillance systems rarely capture all cases in any given period so that small differences in the number of cases should be expected.

Only 9 serogroup C infections were identified nationally in 2011. Serogroup B disease accounted for 84% of all infections where a serogroup was determined. Low numbers of infections to serogroups Y and W135 is usual for Australia, however there was a proportional increase in serogroup Y disease in 2011 from 2010, which will continue to be monitored by the NNN surveillance program.

A primary peak in IMD infection rates was evident in younger age groups, as reported in previous years, with a secondary peak in adolescents and young adults. In people aged 25 years or more, there was a continuing decline in the proportion of serogroup B cases in 2011. This may be explained in part by an increase in the number of serological IMD diagnoses (where the serogroup is not able to be determined for serogroups B, Y and W135), therefore this must be interpreted with some caution.

The distribution of serogroup C disease was low across all age groups in 2010, with no reported cases in those aged less than 20 years. As in previous years, there were only a small number of serogroup C cases in those aged 25 years or more, which may reflect the secondary benefit of herd immunity accruing to the wider community following vaccination of those age groups where disease was formerly highly concentrated.[12]

As in previous years, phenotypic and genotypic data found no evidence of substantial numbers of cases of IMD caused by *N. meningitidis* that have undergone genetic recombination, although sporadic instances of this have been detected in Australia. There were some concerns expressed that the documented capacity for genetic reconfiguration within meningococci may lead to the emergence of new and invasive subtypes following extensive vaccine use.[12] Analysis of meningococcal subtypes and any evidence for the expansion of 'new' subtypes will continue as part of the NNN program. Mortality data were assessable in only a small proportion of cases (10%) and must be interpreted with caution. Four of the 5 fatal cases of IMD were associated with serogroup B infection, and one with serogroup W135. The NNN does not attempt collection of morbidity data associated with IMD.

The distribution of penicillin MICs in invasive isolates in 2011 showed that the proportion that were in the less sensitive category for penicillins was 86%. This was higher than the proportion reported in previous years. It should be emphasised that this shift from fully sensitive to less sensitive category does not affect clinical outcomes and penicillins remain a suitable treatment for IMD in Australia. All isolates were susceptible to the third generation cephalosporins and to the 'clearance' antibiotics rifampicin and ciprofloxacin with the exception of 1 isolate with decreased susceptibility to rifampicin from Queensland. Strains with decreased susceptibility to quinolone antibiotics have been the subject of on-going international interest following their first description from the Australian Meningococcal Surveillance Programme group in 2000.[13–16] There were no isolates with decreased susceptibility to quinolone antibiotics detected in 2011, compared with one in 2010, four in 2009; two in 2008; and one in 2007.

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Australian Paediatric Surveillance Unit annual report, 2011

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

Introduction

The Australian Paediatric Surveillance Unit (APSU) continues to facilitate national active surveillance of uncommon childhood conditions. In 2011, its 18th year of operation, a range of infectious, vaccine-preventable, mental health, congenital and genetic conditions, and injuries were studied. From 1994 to the end of 2011, the APSU had run a total of 52 surveillance studies. For many childhood conditions, the APSU provides the only mechanism for national data collection.[1]

In 2011, the APSU conducted national surveillance for acute flaccid paralysis (AFP), congenital cytomegalovirus (cCMV), congenital rubella, perinatal exposure to HIV and HIV infection, neonatal herpes simplex virus (HSV) infection, congenital neonatal varicella and severe complications of varicella. Surveillance for the severe complications of influenza was undertaken during the influenza season for the 4th year in a row.

Methods

Australian Paediatric Surveillance Unit

The APSU uses standardised protocols and case definitions, which are developed in collaboration with the study investigators who provide specialised expertise for each of the conditions studied. Detailed protocols and case definitions for all conditions under surveillance are available from the APSU web site (www.apsu.org.au)

In 2011, 1,375 paediatricians and other child health clinicians around Australia reported to the APSU; 84% via email or on-line. It is estimated that approximately 91% of paediatricians who have graduated as a Fellow of the Royal Australasian College of Physicians and are currently active in clinical paediatric practice within Australia are participating in the APSU. The APSU clinician database is continually updated to reflect changes in clinician details.[3]

Monthly report card return rates have remained at over 90% since 1994. The rate of returned report cards gives an estimate of participation.[2] Clinicians who report cases provide information on the child's demographics, clinical presentation, treatment and short-term outcome. The APSU is reliant on the study investigators for the clinical review of all data received and classification of notifications according to case definition criteria.[2]

It is important to note that complete ascertainment of cases by the APSU is unlikely. This is particularly relevant in remote communities where children have limited access to paediatricians, hospital admissions are brief or when children are not seen by a paediatrician. The APSU encourages the use of complementary data sources where available and reporting by a range of specialists to maximise case identification.[4,5]

Paediatric active enhanced disease surveillance

The Paediatric Active Enhanced Disease Surveillance (PAEDS) system is a joint initiative of the APSU and the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. This is a hospital-based surveillance system with ascertainment of cases of target conditions conducted by specialist surveillance nurses. The PAEDS system has been operating since 2007 in 4 tertiary paediatric hospitals around Australia (New South Wales, Victoria, South Australia and Western Australia), with Queensland joining the PAEDS network from July 2012. PAEDS complements surveillance for AFP where the surveillance nurses screen and investigate relevant admissions.[6]

Results

A total of 1,375 clinicians participated in the APSU surveillance during 2011. The report card return rate was 90% for 2011. The response rate was slightly lower than in previous years; this was in part due to administrative challenges faced whilst transitioning to the on-line reporting system for New South Wales clinicians as well as staff changes in the APSU. It is anticipated that the participation rate will return to previous high levels when the transition phase to on-line reporting is completed.

Acute flaccid paralysis

The World Health Organization (WHO) surveillance target of at least 1 per 100,000 children aged less than 15 years has once again been reached by combining data from the PAEDS surveillance system and the APSU. Following review by the Australian Polio Expert Panel there were 62 confirmed cases of AFP reported in 2011. The most common diagnoses

Table: Confirmed cases identified in 2011 and for the total study period, and reported rates per 100,000 of the relevant child population

Condition	Date study commenced	Questionnaire response rate (%)	Number of confirmed cases 2011	Reported rate for 2011 (per 100,000)	Number of confirmed cases for total study period	Reported rate for total study period (per 100,000 per annum)
Acute flaccid paralysis	Mar 1995	100	62*	1.4[†]	660	1.0[‡]
Congenital cytomegalovirus	Jan 1999	65	24	8.1[‡]	215	6.7[§]
Congenital rubella (with defects)	May 1993	Nil	Nil	Nil	51	0.1'
Perinatal exposure to HIV	May 1993	85	35	11.7[‡]	469	9.5[§]
HIV Infection	May 1993	NA	6	2.0[‡]	83	1.6[§]
Neonatal herpes simplex virus infection	Jan 1997	79	8	2.7[‡]	129	3.5[§]
Congenital varicella	May 2006	Nil	Nil	Nil	2	0.1[§]
Neonatal varicella	May 2006	Nil[‖]	Nil	Nil	18	1.3[§]
Severe complications of varicella	May 2006	67	2	0.1[†]	47	0.2[‡]
Severe complications of influenza[¶]	Influenza season each year since 2008	96	36	0.8[†]	215	1.3[‡]

* Includes all cases of acute flaccid paralysis reported via the APSU or PAEDS. All cases have been classified by the Polio Expert Panel as 'non-polio AFP' according to World Health Organization criteria.

† Based on population of children aged less than 15 years.

‡ Based on number of births.

§ Based on population of children aged less than 16 years.

|| Two notifications were received by the APSU, clinical data had not been returned at the time of submission.

¶ Influenza surveillance was conducted each year since 2008 during the influenza season, July to September except in the pandemic year (2009) when surveillance occurred from June to October.

All reported rates based on child population estimates published by the Australian Bureau of Statistics.[7]

All of the figures were correct at the time of submission and agreed by the chief investigators for each condition. As additional information becomes available cases may be reclassified for the current year and previous years.

of non-polio AFP was Gullain-Barré syndrome (29%), followed by transverse myelitis (19%) and acute disseminated encephalomyelitis (ADEM) (8%). Other diagnoses included conversion disorder, tick bite paralysis and Bell's palsy. Faecal specimen collection rates have remained low with only 42% of cases achieving the recommended 2 samples within 14 days of onset of paralysis, and only 31% of specimens being adequate for analysis by the National Polio Reference Laboratory. This is below the target of 80% achieving the recommended 2 samples set by the WHO.

Congenital cytomegalovirus

Since January 1999, 215 confirmed cases and 75 probable cases of cCMV have been reported to the APSU. A total of 24 confirmed cases were reported in 2011. Four infants received antiviral therapy and a further four were identified after developing hearing loss after the first year of life. This study continues to inform the debate about cytomegalovirus screening and treatment options for pregnant women and infants, as cCMV remains the most common infectious cause of congenital malformation in Australia. A detailed analysis of the cCMV data was published in the *Medical Journal of Australia* and showed that cCMV was under-diagnosed and patients were infrequently treated.[8] During the study period neonatal hearing screening was introduced for most Australian infants and resulted in an increase in the detection of hearing loss, from 19% of cCMV cases in the period 1999–2003 to 31% in 2004–2009.[8]

Congenital rubella with defects

There were no notifications of congenital rubella in 2011; the last reported case was notified to the APSU in 2009. This is clearly a reflection of the effectiveness of the vaccination program in Australia; however, we need to remain vigilant with regards to the potential for imported cases of rubella from people migrating to Australia from countries where rubella vaccination programs may not be well established.

Perinatal exposure to HIV and HIV infection

There were a total of 35 confirmed cases of perinatal exposure to HIV reported to the APSU during 2011. Six of the 35 children perinatally exposed to HIV acquired HIV infection. One of the six was the child of an Australian-born woman whose HIV infection was diagnosed antenatally. The other 5 children were born overseas in sub-Saharan Africa (3 children) or in South East Asia (2 children). Since May 1993, there have been a total of 469 cases of perinatal exposure to HIV infection reported to the APSU and 83 cases of HIV infection. Perinatal HIV infection remains a rare occurrence among children born to women whose HIV infection was diagnosed antenatally and who made use of interventions for minimising the risk of mother-to-child transmission.

Neonatal herpes simplex virus infection

In 2011, there were 8 confirmed and 2 probable cases (awaiting confirmation from clinicians) of neonatal HSV infection: 3 from Queensland, 2 each from New South Wales and Victoria, and 1 each from the Northern Territory, South Australia and Western Australia. HSV-1 remains the dominant serotype causing disease in this population (7/10). Three infants presented with localised disease to the skin, eye or mouth, 3 infants with encephalitis, and three with disseminated disease. Of note, there were no deaths reported at the time of notification. HSV continues to cause significant disease in the newborn period, predominately due to HSV-1 in Australia. There is a trend towards reduced mortality noted over the study period. Further surveillance of this important but uncommon condition is required to determine if this trend represents a significant change. From 2012, a new study with expanded scope to include HSV disease in the newborn and infants up to 1 year of age will commence to further define temporal trends in disease presentation and outcomes.

Severe complication of varicella infection

In 2011, 2 children hospitalised with severe complications of varicella were reported to the APSU. This was a significant decrease from 2010 with a total of 9 cases reported. Severe complications noted for these patients included; ataxia and bacteraemia. Both of the reported cases were in hospital for a total of 5 days with neither being admitted to the paediatric intensive care unit (PICU). The 2 children reported were unvaccinated and the infecting contacts were close family members (sibling and cousin).

Congenital and neonatal varicella

In 2011, there were no reported cases of congenital or neonatal varicella. A detailed analysis of surveillance data for the whole study period and a comparison with data collected by the APSU in 1994–1996 was recently published and demonstrated that the rates of congenital varicella decreased during the period 2006–2009, though this did not reach statistical significance.[9] However, a significant reduction in the incidence of neonatal varicella was demonstrated when compared with pre-vaccination data (1995–1997), supporting the effectiveness of the varicella vaccination program in Australia.[9]

Severe complications of influenza

In 2011, 36 children with severe complications of influenza were notified to the APSU. Their median age was 4.8 years (range 0.2 months–14.8 years). As in previous years, the main complication of influenza in 2011 was pneumonia, in 17 cases (47%). Fifteen (42%) required mechanical ventilation, 7 (19%) had encephalitis associated with seizures, and 7 (19%) had laboratory proven bacterial co-infection. Two children suffered myocarditis and rhabdomyolitis and 1 child was diagnosed with Guillain-Barré syndrome. Thirty-nine per cent of children (14) had chronic underlying conditions such as asthma or other chronic lung disease, immunocompromised, chronic heart disease and a variety of genetic conditions; only one of these children was vaccinated. None of the other children who had no underlying chronic conditions were vaccinated for influenza. Sixty-seven per cent (24) of children were admitted to PICU with a median stay of 7.5 days (range 1–58 days). Three children died.

Conclusions and future directions

The APSU continues to provide national surveillance data on a number of serious rare childhood diseases. The information collected by the APSU is extremely valuable to clinicians, policy makers and the wider community. For many conditions studied, the APSU is the only source of national data.

The APSU continues to inform public health policy and improve child health in Australia. This is evident from the results of the surveillance study of neonatal and congenital varicella, in which the rates of varicella (neonatal and congenital) infections have fallen dramatically since the introduction of the varicella vaccine to the National Immunisation

Program (NIP) in Australia.[6] These results support the continuation of this program and highlight the potential benefits of varicella vaccination in countries that do not have a vaccination policy in place.

In addition to providing ongoing national surveillance data the APSU is able to conduct seasonal surveillance and to effectively and rapidly respond to outbreaks or emerging diseases as demonstrated by the rapid response to the A(H1N1) 2009 influenza pandemic. Findings from the influenza study noted that although serious complications occurred in children who had an underlying chronic condition, very few had been vaccinated for influenza despite this being provided for under the NIP.

In children admitted to hospital with severe complications of influenza between 2008 and 2011, vaccination rates were low, with only 3% of reported cases vaccinated in 2011. This is particularly important with regards to children who have a pre-existing chronic disorder who are eligible for vaccination under the NIP.

In October 2011, the APSU commenced a new surveillance study for juvenile onset recurrent respiratory papillomatosis (JoRPP). This is a rare condition, which usually develops in childhood and is typically found in children aged less than 12 years, with the median age being 4 years. It is the most common cause of benign neoplasms of the larynx in children and is caused by human papillomavirus (HPV) infection, with HPV 6 and HPV 11 being the two most common causative genotypes.[10]

The APSU Biennial Research Report 2009–2010 was published in May this year and is available on the APSU web site (www.apsu.org.au).

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Notice to readers

Composition of Australian influenza vaccine for the 2013 season

The Australian Influenza Vaccine Committee (AIVC) met on 3 October 2012, and agreed to adopt the September World Health Organization recommendations.

The committee decided that the influenza vaccine components for the 2013 season should contain the following:

- A (H1N1): an A/California/7/2009 (H1N1) - like virus, 15 μg HA per dose
- A (H3N2): an A/Victoria/361/2011 (H3N2) - like virus, 15 μg HA per dose
- B: a B/Wisconsin/1/2010 - like virus, 15 μg HA per dose

For further information please see the Therapeutic Goods Administration web site (http://www.tga.gov.au/about/committees-aivc.htm).

Peer-reviewed articles

Review of strategies to enhance the uptake of seasonal influenza vaccination by Australian healthcare workers

Michael J Stuart

Abstract

Annual vaccination of healthcare workers (HCWs) against seasonal influenza is recommended by The Australian Immunisation Handbook to prevent personal morbidity and transmission to patients. There are limited data available concerning the uptake of this vaccination by Australian healthcare workers, and few studies have investigated the determinants of this uptake. This report therefore aims to review the seasonal influenza immunisation uptake rates of Australian HCWs, the determinants of these rates, and strategies to improve them. The Cumulative Index to Nursing and Allied Health Literature, PubMed and the Cochrane Library were searched for literature published online between January 2000 and May 2011. A manual search of the grey literature was also undertaken. Studies of influenza pandemic A(H1N1) 2009 immunisation were excluded. Eleven relevant studies were identified. The published data suggests that annual seasonal influenza immunisation rates among Australian HCWs are below recommended levels (range 22%–70%). Factors contributing to the decision to be immunised demonstrate only minor variations from those identified in international samples. There is little high quality evidence to support specific strategies and interventions to increase uptake of immunisation in HCWs. Further high quality research is needed to demonstrate the efficacy of strategies and interventions on HCW immunisation uptake, particularly in Australian samples, and if conventional interventions continue to prove ineffective, policy change to mandatory seasonal influenza immunisation should be considered. *Commun Dis Intell* 2012;36(3):E268–E276.

Keywords: influenza, vaccination, Australia, health personnel

Introduction

The recent pandemic A(H1N1) 2009 influenza and its public health response has significantly raised the public profile of both influenza and influenza vaccine in the context of an influenza pandemic. However this may not have significantly altered the attitude or behaviour of healthcare workers (HCWs) vaccination for seasonal influenza.[1] The overall burden of disease attributable to influenza remains difficult to quantify, and consistent data are generally unavailable for low and middle income countries.[2,3] Data from the United States of America (USA) estimated the burden of influenza related disease to be 334,185 hospitalisations and 41,008 deaths annually, with direct medical expenses of $10.4 billion, and a total economic burden of $87.1 billion each year.[4] Australian data also indicate a significant burden of disease attributable to seasonal influenza including, 18,404 hospitalisations, and up to 3,457 deaths per annum with a cost to the healthcare system of $115 million each year.[5,6] The influenza related hospitalisation estimates per 100,000 population are comparable between Australia and the USA; 94.2 and 88.4 respectively.[5]

Influenza vaccines are currently funded by the Australian Government for high risk groups including patients with medical comorbidities, Indigenous people and those aged over 65 years.[7] It has been demonstrated to be efficacious and cost effective in the latter group.[8,9] The cost effectiveness of lowering this threshold to 50 years of age has been debated, although there is currently insufficient evidence to recommend this.[10,11] There is some evidence that HCWs are at increased risk for influenza, and nosocomial infection with influenza has been reported in various healthcare settings.[12–14] Influenza vaccination for HCWs is frequently recommended in hospitals and other settings as a measure for reducing both nosocomial infection and staff absenteeism,[15,16] however compliance with these recommendations has been historically poor.[17–22]

Worldwide, many studies have attempted to identify the factors influencing the decision to be vaccinated, and many strategies have been trialled to enhance the uptake of influenza vaccination by HCWs.[23–27] Internationally, the most commonly cited factors predicting vaccination are desire for self-protection, belief in vaccine effectiveness, and previous receipt of vaccination. The most commonly cited barriers to vaccination were lack of knowledge about influenza virus infection and lack of convenient access to vaccine.[23–27] The disparity in healthcare systems, workplace environments and cultures between HCWs

from different countries is known to influence attitudes to influenza and influenza vaccination.[28] Therefore it is unclear whether the same barriers or enabling factors identified in these studies would translate to the Australian healthcare workforce and whether the strategies trialled in overseas hospitals are relevant to the Australian setting. This paper aims to review the literature as it applies to the Australian context by identifying the rates of influenza vaccination in Australian HCWs, the factors influencing the decision to be vaccinated, and the effectiveness of strategies that have been trialled to increase these rates.

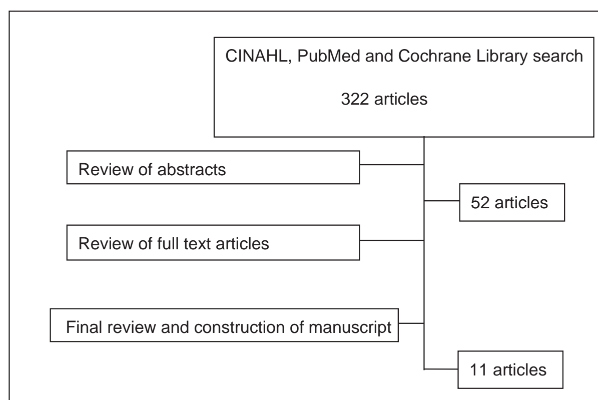
Methods

In this literature review the Cumulative Index to Nursing and Allied Health Literature (CINAHL), PubMed and the Cochrane Library were searched with the following terms: (Influenza OR flu OR orthomyxoviridae) AND (vaccin*(truncation) OR immunis*) AND Austral*. Human studies published online and in English between January 2000 and May 2011 were included. Initial searches yielded a total of 322 articles. After review of abstracts for relevance to the main aim of this review 52 articles were retrieved and studied. Studies of both hospital and non-hospital based healthcare workers were included. At both stages articles were excluded if they did not contain data from an Australian sample (n=289), were primarily concerned with pandemic A(H1N1) 2009 influenza vaccination (n=21), or if full text copies could not be retrieved (n=1). In addition, articles were obtained from an examination of the reference lists of several review articles. In total, 11 articles remained and were included in the final results (Tables 1–3). The Figure depicts this strategy. A manual search of the grey literature was also undertaken with the same search terms, which encompassed state health department web sites and the web sites of infection control interest groups. From this search only the Centre for Healthcare Related Infection Surveillance and Prevention (CHRISP) web site from Queensland Health provided relevant data and was included.

Results

The data on vaccination rates for Australian HCWs are limited (Table 1). These rates are highly variable (range 22%–70%) and predominantly from cross sectional surveys of self-reported vaccination. Notably, in the single relevant published study, HCWs employed in primary healthcare had higher rates of influenza vaccination than those employed in major metropolitan hospitals or aged care.[29] Although these searches did not identify any published results for the state of Queensland, the CHRISP reports on their web site that influenza vaccination coverage of government employed healthcare workers in

Figure: Study inclusion flowchart



* Figure includes non-clinical staff.

Queensland was 26% in 2006 and 60% in 2009.[16] Overall, these rates are comparable to those noted in HCWs overseas,[30–32] but still fall short of the 80% coverage recommended by the Centers for Disease Control and Prevention (CDC).[33] Studies of overseas samples often suggest that medical staff demonstrate the lowest uptake of seasonal influenza vaccination compared with other HCWs, however it is unclear whether this pattern holds for Australian HCWs also. It is concerning to note that Seale et al demonstrated that the lowest rates of coverage in their study were in the highest risk patient areas (15% in neonatal unit, 20% in intensive care).[34]

Table 2 summarises the literature detailing the enabling factors and barriers to seasonal influenza vaccination in Australian HCWs. Enabling factors were defined as reasons cited for obtaining influenza vaccination by personnel who were vaccinated in the last 12 months. Barriers to vaccination were defined as reasons cited by personnel who were not vaccinated in the last 12 months. Enabling factors and barriers were included in the table if they were cited by 20% or more of the study respondents.

The major enabling factors for seasonal influenza vaccination were consistent across studies; in all studies that included the question, the majority of HCWs replied that their key motivating factor in receiving vaccine was the desire to protect themselves, their friends, family, and patients.[21,34–37] Additionally, convenience was highlighted by several studies as a key enabling factor,[21,34] and lack of convenience as a key barrier to vaccination.[21] Reduction in sick leave and workplace guidelines were less frequently cited motivators.[21,34,35,37] Campos et al and Seale et al both determined that the perception of influenza as a serious illness was significantly associated with uptake of vaccination.[34,35]

Table 1: Baseline annual seasonal influenza vaccination coverage of Australian healthcare workers

Author/date	Aim	Design	Sample	Vaccine free of charge	Rate: medical staff	Rate: nursing staff	Rate: other clinical staff	Rate: total
Murray et al, 2002[17]	Investigate vaccination status and determinants at a Victorian hospital	Cross sectional survey. 87% response rate Vaccination self-report	Tertiary hospital staff: 245 Nursing, 101 Medical, 67 other	No	–	–	–	48%
Cooper et al, 2002[18]	Demonstrate the efficacy of a novel staff vaccination strategy in a Melbourne hospital	Unclear	Tertiary hospital staff: 1,362 distribution unclear	Yes	–	–	–	49%*
Halliday et al, 2003[36]	Investigate vaccination status and staff attitudes in aged care facilities in the Australian Capital Territory	Cross sectional survey. 65% response rate Vaccination self-report	Aged care facility staff: 381 Nursing	Some facilities	–	28%	–	28%
Bull et al, 2007[19]	Investigate vaccination rates in Victorian hospitals	Cross sectional survey. 70% response rate Vaccination self-report	>100 bed hospital staff: 5,411 Medical, 19,665 Nursing, 11,885 other	Not reported	29%	35%	50%	38%
Bellaard-Smith et al, 2008[20]	Investigate attitudes to influenza vaccination and strategies to increase uptake in a Victorian Health service	Unclear	Metropolitan health service staff: 10,000 distribution unclear	Yes	–	–	–	42.5%*
Kaufman et al, 2008[21]	Investigate vaccination status and determinants at Royal Darwin Hospital	Cross sectional survey: 62% response rate Vaccination self-report	Tertiary hospital staff: 150 Medical	Yes	28%	–	–	28%
Ballestas et al, 2009[22]	Trial and intervention to increase HCW influenza vaccination in South Metropolitan Perth	Review of vaccination consent forms and staff numbers from Health Corporate Network	Metropolitan health service staff: 11,000 staff	Yes	–	–	–	29%–51% across all hospitals*
Seale et al, 2010[34]	Investigate attitudes and beliefs toward vaccination in Sydney hospitals	Cross sectional survey: 74.5% response rate Vaccination self-report	Tertiary hospital staff: 281 Medical, 512 Nursing, 165 other	Yes	29%	19%	23%	22%
Osman et al, 2010[37]	Investigate enabling factors and barriers to influenza vaccination in a Melbourne emergency department	Cross sectional survey: 0% response rate Vaccination self-report	Tertiary hospital emergency department staff: 12 Medical, 39 Nursing	Yes	58.3%	56.4%	–	58.7%*
Ward et al, 2011[29]	Investigate vaccination rates in primary healthcare staff in New South Wales	Cross sectional survey: 36% response rate Vaccination self-report	Primary healthcare staff: 79 General practitioners, 60 Nursing	Some facilities	–	–	–	70%

Table 2: Enabling factors and barriers to seasonal influenza vaccination in Australian health care workers

Author/date	Objectives	Design	Sample	Major enabling factors (>20%)	Barriers (>20%)
Campos et al, 2002[35]	Investigate predictors of vaccination in a New South Wales hospital	Cross sectional survey: 80% response rate	200 bed hospital staff: 232 Nursing	Belief that influenza is a serious illness (60%)	Belief vaccine may cause flu (40%)
Halliday et al, 2003[36]	Investigate vaccination status and staff attitudes in aged care facilities in the Australian Capital Territory	Cross sectional survey. 65% response rate Vaccination self-report	Aged care facility staff: 381 Nursing	Protect self (68%), family/friends (57%), patients (73%) Vaccination recommended for healthcare workers (64%) Reminder system (28%)	Side effects (including flu) (42%) Poor efficacy (32%) Belief that HCW is at low risk of infection (30%) Unaware vaccination is recommended (26%)
Bellaard-Smith et al, 2008[20]	Investigate attitudes to influenza vaccination and strategies to increase uptake in a Victorian Health service	Semi-structured interview	Metropolitan health service staff: 20 who refused influenza vaccination. 7 Medical, 11 Nursing, 2 other	–	Belief that HCW is at low risk of infection (60%) Belief vaccine may cause flu (60%) Lack of information and evidence provided (50%)
Kaufman et al, 2008[21]	Investigate vaccination status and determinants at Royal Darwin Hospital	Cross sectional survey: 62% response rate	Tertiary hospital staff: 150 Medical	Protect self (90%), family/friends (57%), patients (71%) Convenience (57%) Reduced sick leave (43%) Peer pressure (26%) Reminder system (22%)	Unable to access conveniently (59%) Unaware of how to access (26%)
Ballestas et al, 2009[22]	Trial and intervention to increase HCW influenza vaccination in South Metropolitan Perth	Cross sectional survey. 29.7% response rate	Metropolitan health service staff: 201 Nursing	Belief that influenza is a serious illness (38%)	Belief that HCW is at low risk of infection (56%) Belief vaccine may cause flu (43%) Poor efficacy (22%)
Seale et al, 2010[34]	Investigate attitudes and beliefs toward vaccination in Sydney hospitals	Cross sectional survey: 74.5% response rate	Tertiary hospital staff: 281 Medical, 512 Nursing, 165 other	Protect self (76%), family/friends (82%), patients (77%) Convenience (91%) Reduced sick leave (67%)	Belief vaccine may cause flu (53%) Belief that HCW is at low risk of infection (33%)
Osman et al, 2010[37]	Investigate enabling factors and barriers to influenza vaccination in a Melbourne emergency department	Cross sectional survey: 90% response rate Vaccination self-report	Tertiary hospital emergency department staff: 12 Medical, 39 Nursing	Protect self (59%), family/friends (40%), patients (70%) Infection control campaign (31%)	Trust in or wish to challenge natural immunity (33%) Belief vaccine may cause flu (27%) Not concerned about flu (23%)

Barriers to vaccination included the perception that influenza was not a serious illness or that the HCW was at low risk of infection was held by more than 20% of respondents and considered a major barrier to vaccination in most studies.[20,22,34–37]

Further misconceptions also presented significant barriers to vaccination in many studies. Between 27%–60% of HCWs in each of the samples believed that the inactivated influenza vaccine provided by their hospital may cause influenza.[20,22,34–37] There was also a prevalent belief in several samples, albeit in a smaller percentage of responders, that the influenza vaccine has poor efficacy.[20,22,36] Additionally, a significant percentage of HCW who were not vaccinated claimed to be unaware of guidelines recommending HCW influenza vaccination, or how to access this.[20,21,36]

Only 3 studies were identified which documented a trial of an intervention to increase the uptake of seasonal influenza vaccination in Australian HCWs (Table 3). All of these studies described the use of a free, mobile vaccination clinic and educational or promotional materials as strategies to increase vaccination rates of HCWs. Both Cooper et al[18] and Bellaard-Smith et al[20] included the provision of an after-hours clinic to reach these staff, however it is unclear whether Ballestas et al[22] also provided this service. Furthermore, all studies demonstrated a positive effect of their interventions on vaccination rates.[18,20,22] Other strategies employed included: the vaccination of non-clinical staff to avoid confusion over eligibility;[18] the recruitment of senior staff as 'Flu Champions' to provide peer leadership;[22] incentives, educational sessions; and declination forms.[20]

Discussion

To the author's knowledge, this is the first paper to review strategies to enhance the uptake of influenza vaccination by Australian HCWs and the factors which influence this uptake. A recent review concluded that influenza vaccination rates of Australian hospital based HCWs are consistently low.[38] This review extends that finding to non-hospital based and non-clinical HCWs. Although the data for the Australian context are currently very limited, all available studies demonstrated an insufficient uptake of seasonal influenza vaccination by Australian HCWs in all settings.[17–22,29,34,36,37] The CDC recommends 80% coverage of HCWs to obtain the benefits of herd immunity to the vaccine strains, however 60% coverage of HCWs has also been demonstrated to be effective in reducing patient mortality in the United Kingdom.[39] This review found that vaccination rates amongst health-care workers fall short of these targets, with rates below 50% in the majority of included studies. This is comparable to the situation internationally; a systematic review by Hofmann et al demonstrated rates below 60% in all Canadian and European studies included, and only studies including promotional interventions reached above 60% coverage in the USA.[24]

This review has several limitations: the low response rates to several surveys and heterogeneity of study populations precludes further statistical meta-analysis of the data in Table 1, particularly with regard to comparisons between medical and nursing staff, or between primary care and major hospital staff. Additionally, comparisons with the international literature are complicated by the design of many Australian studies using closed questionnaires based upon factors identified in the international literature.

The majority of the key themes identified as enabling factors or barriers to vaccination in this review align closely with those described in overseas studies.[23–25] The most prevalent enabling factors in both Australian and international samples were a desire for protection of self, patients, family and friends, and convenience.[21,24,25,34,36,37] Additionally, in all studies of Australian HCWs, previous influenza vaccination was significantly associated with current vaccination or intention to be vaccinated.[20,22,34–37] This is also the most consistent predictor of influenza vaccination in international studies.[23–25] This suggests that intensive campaigns to increase uptake may provide recurring benefits in subsequent annual uptake.

The key barriers to vaccination identified in Australian and international samples were common misconceptions about influenza or influenza vaccination. The most prevalent included: the belief that the vaccination may cause influenza or influenza-like illness; the belief that influenza is not a serious illness, or the HCWs are at low risk of influenza virus infection; and doubts about the efficacy of the vaccine.[24,25] Any successful educational campaign must aim to address these issues.

Aside from the aforementioned similarities in identified enabling factors and barriers in Australian and international studies, this review has also identified several differences. Perhaps the most significant omission from Australian questionnaires is the fear of injections, which has been shown to account for up to 26% of vaccine declination in reviews of the international literature.[24,25] Future studies should ensure this is included in their questionnaires. Also, the reviews of international studies describing motivations for vaccination have found only one study in which the desire to protect patients is ranked as a more significant motivation than the desire for self-protection.[24,25] Conversely, three out of the seven surveys of Australian samples found that HCWs ranked patient protection as a greater motivator than self-protection.[34,36,37] This discrepancy is highly

Table 3: Strategies to enhance the uptake of seasonal influenza vaccination

Author/date	Objectives	Design	Intervention	Sample	Pre-intervention coverage	Post-intervention coverage
Cooper et al, 2002[18]	Demonstrate the efficacy of a novel staff vaccination strategy in a Melbourne hospital	Interventional study	Free of charge Mobile vaccination clinic After hours clinic Educational pamphlet Vaccination of non-clinical staff	Tertiary hospital staff: 1,362 distribution unclear	49%	81%
Bellaard-Smith et al, 2008[20]	Investigate attitudes to influenza vaccination and strategies to increase uptake in a Victorian health service	–	Free of charge Mobile vaccination clinic After hours clinic Educational pamphlet, posters, memos Opportunistic education sessions Reward for reaching target % Limited use of declination forms	Metropolitan health service staff: 10,000 distribution unclear	42.5%	57.5%
Ballestas et al, 2009[22]	Trial of intervention to increase HCW influenza vaccination in South Metropolitan Perth	Interventional study	Free of charge Mobile vaccination clinic Educational/promotional pamphlets, posters, emails Recruitment of senior staff	Metropolitan health service staff: 11,000 staff	29%–51%	48.8%–76.5%

relevant to advertising and promotional campaigns in Australian hospitals, suggesting that these campaigns should emphasise the protective effect of this vaccine on patients.

In the author's experience, many Australian healthcare settings and hospitals employ some form of seasonal influenza vaccination campaign. However this search of the literature demonstrated that these strategies have very seldom been published (Table 3). The report of Cooper et al was in the form of a letter to the editor and provided no description of the means of data collection employed in their study.[18] The study by Bellaard-Smith et al was primarily reporting on the results of a qualitative interview series as described in Table 2, and only incidentally reported on the intervention they utilised to increase HCW influenza vaccination.[20] Conversely, Bellestas et al reported their data collection procedures and the design of their study in significantly more detail.[22] However, there is a paucity of good quality evidence in comparison with the data that has been generated internationally, which includes a number of randomised controlled trials (Lam et al, 2010[27]). The systematic review by Lam et al determined that there remains a paucity of well-designed trials of interventions to increase influenza vaccination uptake, particularly those that dissect the benefits attributable to individual arms of each intervention. The available evidence suggested that educational and promotional campaigns alone were associated with minor or non-significant improvements in uptake, whereas multi-faceted interventions were associated with greater improvements.[27] In the absence of good quality evidence for each specific strategy, it is difficult to make evidence based recommendations for components to be included in an institutional vaccination campaign.

Interventions that have been trialled in Australian and international studies have most frequently included the provision of mobile clinics and a range of promotional and educational activities. These strategies clearly address the key barriers to vaccination, such as inconvenience and misconceptions about influenza and vaccination. When considering the provision of mobile clinics it is important to recognise the importance of after-hours clinics to cover staff on those shifts.[18,20] As a general principle, promotional and educational activities should be tailored to their target population and evolve as the needs of that population change. This requires an understanding of the knowledge and attitudes of that population and should be supported by standardised data collection at baseline and follow-up to monitor the effectiveness of this strategy. To this point, very few studies have reported utilising such an approach and this is suggested to represent a key deficiency in many interventions.[27] Additionally, the value of highly visible endorsement from senior clinical and management staff as part of a promotional campaign should not be understated.[22,27]

Other strategies that have been trialled include significant institutional policy changes. The introduction of mandatory declination forms, requiring non-vaccinated staff to wear protective masks during times of peak influenza activity, or simply introducing mandatory vaccination for all staff have all been trialled and resulted in a significant increase in uptake.[26,40,41] While these measures may potentially be viewed as draconian, strict adherence to the principles dictating that HCWs should at all times act in the best interests of their patients would mandate vaccination.[42,43] Some authors have suggested that institutional HCW vaccination rates should be reported publicly as a quality and safety metric.[44]

Influenza vaccination of HCWs is both efficacious in preventing inpatient mortality and cost effective,[39] yet annual seasonal influenza vaccination rates remain low. Because effective protection from influenza is dependent on annual vaccination, healthcare institutions worldwide will grapple with encouraging annual influenza vaccination uptake. Improvement of current strategies will require high quality research including randomised controlled trials in various healthcare settings, and samples specific to each culture and healthcare system. Only with such high quality evidence can cost effective and comprehensive vaccination campaigns be planned. Additionally, the development and inclusion of novel strategies such as non-binding declination forms into existing interventions may provide some benefit.[20,26] It is important to recognise that no combination of current strategies has been consistently documented to increase vaccination uptake above the 80% recommended by the CDC.[33] If conventional strategies prove unable to increase and maintain vaccination rates at sufficient levels then more significant policy changes may be required. The clear benefits of increasing HCW influenza vaccination rates should encourage a dialogue between staff, senior hospital management, and state health departments regarding a potential policy shift to mandatory seasonal influenza vaccination for HCWs.

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Measles with a possible 23 day incubation period

Tove L Fitzgerald, David N Durrheim, Tony D Merritt, Christopher Birch, Thomas Tran

Abstract

Measles virus (MV) eradication is biologically, technically and operationally feasible. An essential feature in understanding the chain of MV transmission is its incubation period, that is, the time from infection to the onset of symptoms. This period is important for determining the likely source of infection and directing public health measures to interrupt ongoing transmission. Long measles incubation periods have rarely been documented in the literature. We report on a previously healthy 11-year-old Australian boy who was confirmed with measles genotype D9 infection following travel in the Philippines. Epidemiological evidence supported an unusually long incubation period of at least 23 days and virological evidence was consistent with this finding. Although public health control measures such as post exposure prophylaxis, isolation and surveillance of susceptible individuals should continue to be based on the more common incubation period, a longer incubation period may occasionally explain an unexpected measles case. *Commun Dis Intell* 2012;36(3):E277–E280.

Keywords: measles, incubation period, elimination, epidemiology, genotyping

Introduction

Measles virus (MV) eradication is biologically, technically and operationally feasible as demonstrated in the Region of the Americas and countries in every other region.[1] A number of countries have already declared endemic measles eliminated, while others, including Australia, are presently gathering evidence to confirm interruption of endemic MV transmission.[2,3] In these countries, laboratory and/or epidemiological confirmation of every case is ideally required. This allows: the origin of cases as local or imported to be identified, particularly if genotyping is available; the size and duration of outbreaks to be determined; risk groups requiring public health action to be recognised; and existing public health activities to be reviewed.

An essential feature in understanding the chain of MV transmission is its incubation period, that is, the time from infection to onset of symptoms. This period is important for determining the likely source of infection and directing public health measures to interrupt ongoing transmission. Infectious disease and public health texts and global guidelines currently stipulate that the incubation period for measles infection is 7–18 days, but rarely as long as 19–21 days. This article reports on a measles case in a previously healthy child with a possible incubation period of at least 23 days.

Case presentation

An 11-year-old Philippines-born male with no previous significant medical history developed a cough on 19 May 2011, followed by fever (39°C) on 20 May. On 22 May he developed a typical generalised measles rash that began behind his ears before spreading to his face and trunk. Fever and cough remained present at rash onset.

On hospital paediatric review on 23 May he was febrile (39°C), had a red, blanching non-itching and non-tender rash covering his entire body, exhibited a persistent cough, had prominent coryza and conjunctivitis, and Koplik spots were noted on his buccal mucosa. After receiving 500 mg of paracetamol orally and having blood and urine collected, he was discharged home with a request to remain isolated. His MV serology was IgM positive/IgG not detected, thereby confirming the clinical diagnosis of measles infection. He made a full recovery.

Epidemiological investigation

The child was notified to the Hunter New England Population Health Unit on 23 May 2011. An epidemiological investigation was initiated the following day on the basis of the serological confirmation as measles, which is a notifiable disease under the *New South Wales Public Health Act 2010*. A detailed travel history, both international and domestic, was sought, exhaustive contact tracing was conducted in Australia, and his close (household) contacts were serologically investigated. Measles polymerase chain reaction (PCR) testing was only conducted for the case.

The child had travelled with three family members to the Philippines, returning by air to Sydney on 27 April. His travel companions included his mother (aged 45 years) and his 2 older brothers, aged 17 and 18 years. The family spent 2 weeks in the Philippines and returned to Sydney with a 24-hour stopover in Hong Kong, during which time they remained at the airport.

The family was unable to specifically identify a direct exposure to anyone with clinical measles during their visit to the Philippines, the stop-over in Hong Kong or in the period between returning to Australia and the onset of symptoms. The only close contacts the case had after returning to Australia were his mother, the 2 older brothers, a 16-year-old Australian-born step-sister, her 15-year-old friend, an aunt and her male partner, and his maternal grandmother. None of these individuals had experienced a febrile illness prior to the onset of symptoms in the case. Serological testing for MV-specific antibodies showed that all were IgM negative/IgG positive, suggesting previous infection or vaccination (Table 1).

Table 1. Measles virus antibodies of household contacts

Family member	IgM	IgG	Date of serology
18 year old brother	<1.0	113	24/05/2011
17 year old brother	Equivocal	109	24/05/2011
16 year old stepsister	<1.0	5	24/05/2011
Mother	<1.0	61	24/05/2011
Aunt	<1.0	29	25/05/2011
Partner of aunt	<1.0	4	25/05/2011
Friend of sister	<1.0	27	25/05/2011
Grandmother	<1.0	27	24/05/2011

The immunisation status of the case and his two Philippines-born brothers was not available on the electronic Australian immunisation register, and their mother had no physical records or memory of them being vaccinated in the Philippines as children or experiencing measles-like symptoms.

No measles cases had been identified in the Hunter New England health district in the 2 months prior to this case. There were 5 known cases of measles in New South Wales in the 23 days prior to the onset of symptoms. Two of these cases were identified as infected with measles genotype D4 and one of these cases had subsequently infected a quarantined sibling. Two cases in an adjoining health district were identified in a pair of siblings who had travelled to France and Italy. No typing was available for them, however, they and their family had not travelled out of their local area after returning to Australia on 1 May.

Laboratory investigation

SIEMENS Enzygnost™ assay was used to detect measles IgM and IgG in the case and his close contacts (Table 2). Urine and throat swab samples from the case were positive by real-time PCR (RT-PCR) for MV using Applied Biosystems® Taqman® Fast Universal PCR mastermix (2X) by Life Technologies. Molecular characterisation to determine the genotype of the virus involved sequencing a 450 nucleotide (nt) region of the viral nucleoprotein (N) gene as previously described.[4] The entire haemagglutinin (H) gene was also sequenced to enable a more detailed comparison with other measles strains circulating in 2011. Briefly, PCR products were purified using ExoSAP-IT PCR clean-up kit (GE Healthcare) according to the manufacturer's instructions. Purified PCR products were sequenced in the forward and reverse directions.[4] Nucleotide sequences were analysed on the Bio-Edit Sequence Alignment Editor software[5] and MV genotype determined using the National Center for Biotechnology Information (NCBI) on-line nucleotide blast program.

Table 2. SIEMENS Enzygnost™ assay values

Assay	Value (Index)	Interpretation
IgM	<0.5	Negative
	0.5–1.0	Equivocal
	>1.0	Positive
IgG	<1.0	Negative
	1.0–2.0	Equivocal
	>2.0	Positive

Case nucleotide sequences were submitted to Genbank, accession.number JX679861

Sequencing of the N gene identified the MV genotype as D9. The N gene nt sequences of 6 MVs from cases in the Philippines, five occurring during January and February 2011, and one occurring in April 2011, were compared with the case N gene sequence (Philippines N sequences provided by the National Reference Laboratory (NRL)-Measles, Research Institute for Tropical Medicine, Philippines). The 6 Philippine N gene sequences differed by a maximum of 3 nt from each other and from the local case.

Partial N gene and complete H gene nt sequences from the case and 3 D9 MV strains circulating during the first quarter of 2011 in New South Wales were also compared. One New South Wales MV strain differed by 1 nt in the N gene and 6 nts in the H gene to the case strain sequence. Two New South Wales strains had identical N and H sequences that differed from the case by 3 and 6 nts in the N and H gene, respectively.

Discussion

The epidemiological evidence supports a measles exposure in the Philippines as the source of this child's measles infection and the virological evidence is consistent with this hypothesis. All 29 genotyped measles cases from the Philippines between January 2010 and June 2011 were D9 strains (personal communication, Youngmee Jee, Western Pacific Regional Office of the World Health Organization). Comparison of N and H gene sequences of the case strain and strains from New South Wales (N and H sequences) and the Philippines (N gene only) were consistent with importation from the Philippines. As sequencing of MV genes other than N and H becomes more widely available, specific geographical location of individual cases' place of infection will be more readily possible.[6] Molecular sequencing methods have been identified as a research priority as we move towards measles elimination.[7]

The Hunter New England region of New South Wales, Australia, has a documented high quality enhanced measles surveillance system that meets the indicators recommended for elimination.[8] This suggests that general practitioners are unlikely to have missed other measles cases in the region if they presented for treatment. Although this scenario cannot be completely eliminated, undetected cases in the community would have likely resulted in additional cases due to the infectious nature of measles.

The case's 17-year-old brother had an equivocal IgM test (index 0.5–1.0) but a high IgG value. This suggests a previous infection but sequential IgG values may have assisted to confirm that he did not experience a subclinical case of measles and serve as the source of infection in Australia.

As the child's measles exposure most likely occurred in the Philippines or in transit no later than 27 April, the period to onset of symptoms on 19 May represents an incubation period of at least 23 days. Long measles incubation periods have rarely been documented in the literature, possibly in part because in measles endemic areas it can be difficult to ascertain the exact time of exposure. This complicates the accurate determination of incubation periods, and serial intervals or rash onset dates may then be used as a proxy for the incubation period. The incubation period is longer than the serial interval as the infectious period commences prior to symptom onset (personal communication, Professor Natasha Crowcroft, Health Canada).

An observational study in rural Kenya[9] demonstrated infrequent incubation periods of up to 24 days and observations on measles transmission in a fever hospital in 1931 documented infrequent incubation periods up to 25 days.[10]

The log-normal distribution of directly-transmitted infectious disease incubation periods (with the distribution dramatically skewed to the right) is well recognised.[11] The 99th percentile of the lognormal distribution for the measles incubation period is 22.3 days (95% CI 20.8–23.9) and thus an incubation period of 23 days should occasionally occur.[12,13] These unusually long incubation periods may reflect variations in infectious dose, pathogen replication time or degree of susceptibility.[14]

Conclusion

Measles elimination demands careful review and classification of each confirmed measles case as locally acquired, imported or import-related. Although public health control measures such as post exposure prophylaxis, isolation and surveillance of susceptible individuals should continue to be based on the common incubation period, a longer incubation period may occasionally explain an unexpected measles case.

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An outbreak of *Salmonella* Typhimurium phage type 135a gastroenteritis linked to eggs served at an Australian Capital Territory cafe

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Abstract

Salmonella is an important foodborne pathogen, with eggs and egg-containing foods being frequently implicated in causing outbreaks of disease. In April 2012, an investigation was commenced after a number of cases of salmonellosis were linked to a Canberra café. The investigation sought to identify the cause of illness and to introduce public health measures to prevent further disease. A case control study was undertaken using the café's booking list to identify potential cases and controls. A structured questionnaire was developed using the café's menu, with information collected via telephone interview or email. A case was defined as any person who ate at the implicated café on 25 April 2012 and subsequently developed gastroenteritis. A total of 20 cases and 22 controls were recruited into the study. All 20 cases had faecal cultures positive for *Salmonella* Typhimurium phage type 135a (STm 135a). Eating eggs Benedict (odds ratio 63.00, 95% confidence interval 6.08–2771.66 $P < 0.001$) was significantly associated with illness. While no microbiological evidence of STm 135a was obtained from foods sampled from the café, STm 135a was recovered from swabs taken from the kitchen environment. This report illustrates an ongoing trend in Australia, where raw and minimally cooked egg-containing foods are identified as the responsible vehicles in a high proportion of foodborne *Salmonella* outbreaks. *Commun Dis Intell* 2012;36(3):E281–E287.

Keywords: eggs, *Salmonella* Typhimurium, disease outbreak, foodborne disease, case control study

Introduction

Responding to outbreaks of salmonellosis linked to cafés and restaurants serving foods containing raw or minimally cooked egg is an increasingly common action undertaken by public health and food safety authorities in Australia.[1] In south-east Australian jurisdictions (including the Australian Capital Territory, New South Wales and Victoria), the most common agent causing such outbreaks has been *Salmonella enterica* serovar Typhimurium (STm).[1] Investigation of these outbreaks has shown that consuming raw or minimally cooked egg-containing foods, such as dressings like hollandaise sauce and desserts like tiramisu, is frequently associated with human illness.[2,3]

On 1 May 2012 the Communicable Disease Control (CDC) section of the ACT Health Protection Service was alerted to a higher than expected number of laboratory diagnoses of *Salmonella* infection. These cases were rapidly interviewed by CDC staff to determine if a common exposure could be identified. Seven cases reported eating breakfast at the same café on the ANZAC Day public holiday on 25 April 2012. An acute response team meeting was held and an investigation into an outbreak linked to breakfast served at the café on the ANZAC Day public holiday was commenced. The investigation sought to identify the cause of illness and to implement appropriate public health measures to prevent further cases.

Methods

Epidemiological investigation

Initial interviewing of *Salmonella* cases was conducted using a hypothesis generating questionnaire, which included a detailed three-day food history. The results of these interviews led to the formation of a hypothesis that illness was linked to one or more egg-containing dishes eaten at a Canberra café, with exposure having occurred on 25 April 2012.

A case control study was undertaken to test this hypothesis. Ethics approval was not sought as the investigation was conducted as part of a public health response. Cases were identified either via routine public health investigation of any laboratory-confirmed *Salmonella* infection or via interrogation of contacts listed on the café's booking list for 25 April 2012. Controls were also recruited from the booking list or through their nomination by other cases and/or controls. A structured questionnaire was developed based on the café's menu listing. The questionnaire sought to confirm symptoms, onset and exposure dates and times, in addition to providing detail on specific food and beverages consumed by cases and controls during the postulated day of exposure.

A clinical case was defined as any person who ate at the implicated café on 25 April 2012 and subsequently developed gastroenteritis (defined as diarrhoea with or without abdominal pain). Clinical cases were encouraged to provide a faecal sample to assist with the epidemiological investigation. A confirmed case was defined as per a clinical case, in addition to having a faecal sample positive for *Salmonella* Typhimurium Phage type 135a (STm 135a).

Data obtained from the structured questionnaire were entered into a Microsoft Excel® database before analysis using Stata® version 9. Case and control demographic details, such as gender, were compared using a Fisher's exact test, while age was compared using a student's *t*-test. An unmatched analysis was performed with crude odds ratios (OR) and associated 95% confidence intervals (CI) calculated for all food exposures. A multivariate logistic regression model was then constructed to adjust for potential confounding using food items that had a P value < 0.10 in the univariate analysis.

Environmental investigation

Environmental Health Officers (EHO) inspected the café on 1 May 2012, with the kitchen facilities and the food preparation procedures being reviewed. Copies of the café's booking list for 25 April 2012, which included patron contact names and telephone numbers, were obtained, while details on staff illness and absenteeism were also requested. Although no remnant food items remained from the likely date of exposure, a number of statutory food samples were taken, including whole shell eggs (n=60), an egg yolk mix, egg mayonnaise, lemon mayonnaise, chickpea dip and a café prepared barbeque sauce. Extensive environmental swabbing of the kitchen was also undertaken from sites including food display trays, sinks, a chopping board, a tap handle (for staff hand washing), refrigerators, a walk-in cool room, a display bench, the floor, and a variety of other kitchen implements, including scales, eggs rings, a knife and takeaway containers.

As initial case interviews had identified egg containing dishes as plausible food vehicles for infection, an extensive review of the preparation of raw or minimally cooked egg-containing foods was conducted. Particular emphasis was placed on the café's preparation and use of hollandaise sauce. Trace-back of egg supply and production was conducted, with the New South Wales Food Authority (NSWFA) and the NSW Ministry of Health both being contacted regarding a New South Wales-based grading facility thought to process the brand of eggs used by the café.

Advice regarding the safe handling of egg-containing foods was provided to café staff. In addition, a public health message describing the potential risk associated with use of raw eggs was incorporated into a routine correspondence with all Australian Capital Territory food businesses to alert them to the problem of salmonellosis and eggs.

A number of follow-up inspections of the café were conducted by EHO on 9 May and 30 May 2012, with additional swabbing performed at the latter visit. Sites tested included the cool room and refrigerator door handles, a food preparation bench, a cutting board and tongs.

Laboratory investigation

Stool samples were tested for enteric pathogens using standard laboratory methods. Food samples and environmental swabs taken from the restaurant were tested for the presence of *Salmonella* (and other pathogens), by the Australian Capital Territory Government Analytical Laboratory using standard food and environmental laboratory methods. *Salmonella* isolates were serotyped and identified by multi-locus variable number of tandem repeats analysis (MLVA) at the Microbiological Diagnostic Unit (MDU), University of Melbourne, Victoria or the New South Wales Enteric Reference Laboratory, Institute for Clinical Pathology and Medical Research (ICPMR), Sydney. Isolates that were received by the MDU initially underwent phage typing prior to MLVA results being specifically requested.

Results

Epidemiological findings

Descriptive epidemiology

A total of 20 cases were identified using *Salmonella* notification data or active case ascertainment via the café booking list (Figure). All cases confirmed that they ate at the café on 25 April 2012. The median incubation period was 13.5 hours (range 7.5 hours to 42 hours, interquartile range 4.5 hours). Symptom prevalence for cases was: diarrhoea (100%), abdominal pain (95%), fever (85%), headache (85%), nausea (80%), myalgia (60%), vomiting (50%), lethargy (45%) and bloody diarrhoea (30%). Nineteen cases (95%) consulted a doctor (either a general practitioner or a hospital emergency department) about their illness and 2 cases (10%) were hospitalised. The length of stay for the hospitalised cases was 3 days each. Detail on the duration of illness was unable to be reported as the majority of cases were still unwell at the time of data collection.

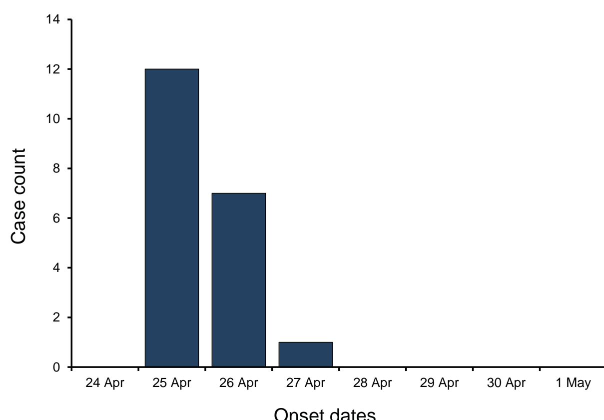
All 20 cases identified during the investigation were included in the case-control study. A total of 22 unmatched controls were enrolled in the study. Controls were either nominated by cases or recruited through the café booking list from

25 April 2012. The mean age of cases was 34 years (median = 31 years; range 19–62 years), with 65% being male. For the control group, the mean age was 33 years (median = 31 years; range 2–77 years) and 59% were females. The sex distribution did not differ significantly between cases and controls (Fisher's exact test, $P = 0.14$) and there was no significant difference in the mean ages between the groups ($t = –0.21$ $P = 0.84$).

Analytical epidemiology

The results of the univariate and multivariate analyses are shown in the Table. Eating eggs Benedict (OR 63.00, 95% CI 6.08–2771.66 $P < 0.001$) or any poached eggs (OR 8.19, 95% CI 1.57–53.60 $P = 0.003$) were both significantly associated with illness. However, only eggs Benedict (aOR 101.92, 95% CI 4.91–2112.97 $P = 0.003$) maintained a significant association with illness after adjustment via multivariate analysis.

Figure: Onset dates for cases of *Salmonella* Typhimurium PT 135a gastroenteritis linked to an Australian Capital Territory café, April 2012



Environmental findings

Inspection by EHO on 1 May showed that the café kitchen itself was generally clean and well maintained, with staff being able to demonstrate a good knowledge of food safety. Temperature checks for refrigerators were within accepted limits, with the exception of the main kitchen preparation fridge, which had a recorded temperature of 8.0 °C at the time of inspection. Hand washing facilities were however deemed unsatisfactory due to the absence of paper towels for hand drying. The need for additional cleaning in non-food preparation areas, as well as some minor structural repairs, was also reported. As a result of these findings, an improvement notice was issued. A second inspection on 9 May 2012 showed that the previously identified issues with temperature control, hand hygiene, cleaning and minor repairs had been remedied. A final visit to the café was conducted on 31 May 2012 to exclude on-going contamination following the isolation of the outbreak strain of *Salmonella* from environmental samples taken on 1 May 2012. No further issues were identified.

One staff member, a barista, was identified as being unwell with gastroenteritis at the time of the investigation. However this person's illness onset did not occur prior to other cases. Additionally, this staff member also reported eating eggs Benedict during a meal break on 25 April 2012. No other sick food handlers or staff members were identified as working during the exposure period.

Hollandaise sauce

The hollandaise sauce is made daily at the café and used only in breakfast items, specifically with eggs Benedict. In general, a single batch is made on weekdays and 2 batches are prepared on Saturdays and Sundays. The first batch is reported as always

Table: Odds ratios for selected breakfast items consumed by cases and controls

Foods consumed	Cases n	Cases %	Controls n	Controls %	Crude OR	95% CI	*P*-value	Multivariate analysis: Adjusted OR	Multivariate analysis: 95% CI	Multivariate analysis: *P*-value
Eggs Benedict	15	75	1	5	63.00	6.08–2771.66	<0.001	101.92	4.91–2112.97	0.003
Any poached eggs	17	85	9	41	8.19	1.57–53.60	0.003	1.25	0.08–19.99	0.87
Any eggs	20	100	15	68	undefined	2.21–undefined	0.006			
Any extras	9	45	4	18	3.68	0.76–19.90	0.06	5.38	0.40–72.06	0.20
Any eggs on toast	4	20	2	9	2.50	0.31–30.32	0.31			
Fried eggs	2	10	1	5	2.33	0.11–144.09	0.49			
Scrambled	1	5	0	0	–	–	0.28			
Poached eggs	1	5	1	5	1.11	0.01–90.83	0.95			
Breakfast burger	2	10	4	18	0.50	0.04–4.07	0.45			
Breakfast platter	1	5	1	4	1.11	0.01–90.83	0.95			

being used before the second is made, with any left-over sauce being discarded prior to the lunch menu commencing. On the ANZAC day public holiday, the breakfast menu, which included eggs Benedict, was available to customers throughout the day. On that particular day, two batches of hollandaise sauce were prepared due to high customer demand. The eggs poached for eggs Benedict are always served soft, unless otherwise requested by a customer. The hollandaise sauce is made in approximately 1 litre batches. It comprises 15 egg yolks, vinegar and clarified butter. The ingredients are whisked in a bowl over a pot of boiling water until the correct consistency is achieved. The chef estimated the process to take around 15 minutes. Once made the sauce is stored directly above the stove, on a shelf, in a stainless steel container. The chef stated that the sauce can remain on the shelf for up to 2.5 hours.

Trace-back of eggs

The café used free range eggs, which were delivered weekly by a local distributor. The café reported using approximately 1,800 to 2,500 eggs per week (i.e. 10–14 boxes, with each box containing 15 dozen eggs). All eggs were stored under refrigeration on receipt. The chef advised that the wholesaler usually supplied the same brand of eggs (packed at a New South Wales-based grading facility). However, on occasions different egg brands were received from the wholesaler. Details of the suspected grading facility were forwarded to New South Wales Food Authority and the NSW Ministry of Health to determine if this facility had been implicated in investigations in that jurisdiction. No batch details or leftover cartons from the delivery prior to the outbreak were available and New South Wales agencies reported the company had not been previously implicated in the supply of contaminated eggs. The café also ceased using the local distributor to supply eggs. The distributor was unable to be contacted.

Laboratory findings

Twenty stool samples were obtained from cases and all were positive for *Salmonella* Typhimurium (STm). Of the 14 samples received by MDU, all were phage typed as STm 135a. MLVA typing of these 14 isolates showed a common pattern, 03-13-11-10-523. For the 6 STm isolates typed at ICPMR, five shared a common MLVA pattern, 03-13-11-09-523. The 6th isolate was closely related, with a single digit difference at the second loci (MLVA 03-12-11-09-523). This isolate was also considered to be a case. The observed differences between the MDU and ICPMR generated MLVA patterns are most likely not real and relate to the latter laboratory's shift to the use of adjusted fragment sizes (personal communication Mary Valcanis and Karolina Dimovski). Positive environmental swabs taken from a refrigerator door handle and a walk-in cool room door handle on 1 May 2012 were also tested at MDU. Both were positive for STm 135a, with MLVA pattern 03-13-11-10-523. All other statutory food samples taken on 1 May were negative for *Salmonella*. Follow-up swabs taken from the kitchen on 30 May 2012 were negative also for *Salmonella*.

Discussion

The results of the epidemiological investigation support the hypothesis that the outbreak's probable cause was contaminated eggs used in the preparation of hollandaise sauce. Having eaten any poached eggs or eggs Benedict (which comprises both poached eggs and hollandaise sauce) showed significant associations with illness but after multivariate adjustment only the eggs Benedict remained significantly associated with illness. The findings and observations from the environmental investigation provide further support to the epidemiological evidence. On the day of exposure, the café reported a very high level of business, with in excess of 350 orders. This demand had an impact on the hollandaise sauce used that day, with staff needing to prepare an additional batch to meet demand. It is therefore plausible that a breakdown in food handling practices occurred, involving insufficient temperature during the hollandaise preparation. In addition, the description of the sauce being left above the stove during service, at likely ambient temperatures, could also have assisted the survival and potential proliferation of any bacteria present.

While the recovery of the outbreak strain on door handles indicates pathogen transfer via hands, it does not assist in determining whether the *Salmonella* may have been transferred from contaminated shell eggs (or some unknown source) to the hands of staff and then into the wider kitchen environment. It is possible that the eggs were free of bacteria prior to their arrival at the café and that the hollandaise sauce became contaminated during or after its preparation. Although STm 135a was not recovered from food items or eggs sampled from the café this is not an unsurprising finding as the café reported making fresh hollandaise sauce daily. Furthermore, the inherent delays between case exposure, illness onset and authorities identifying an outbreak and undertaking an inspection of the premises means that left over eggs, egg products, cartons and packaging from the time of exposure are unlikely to be recovered. Nevertheless, egg-associated outbreaks, including those both with and without positive food microbiology, are being identified with increasing frequency across Australia.[4–6]

The use of booking lists and recruitment of controls via known cases was an appropriate and practical strategy to use in response to this outbreak. The time from exposure to commencement of the analytical study was only 1 week, which may have assisted in reducing exposure misclassification. While a matched analysis was not performed for practical reasons, it is unlikely that the characteristics of cases and controls differed significantly, with whatever indefinable factors led cases to eat at the café likely applying to controls. This would have assisted with management of potential confounding and bias. The descriptive epidemiology lends support to this with no significance differences for gender and age between the 2 groups. The effect size and width of confidence intervals does however reveal some imprecision around the study's primary findings, a factor that can be difficult to control for in analysis conducted as part of a rapid public health response.

Although restaurants and cafés remain the most common settings for egg-associated outbreaks of salmonellosis,[7] more concerted efforts are required to reduce infection pressure at the primary production level. Arguably, if pathogen reduction is increased on farm, there should be a decrease in the sale of contaminated product, resulting in fewer cafés and restaurants being identified as settings for *Salmonella* outbreaks. With eggs being so frequently implicated as a cause of such outbreaks, the resulting impact of the new Food Standards Australia New Zealand *Primary Production and Processing Standard for Eggs* on this significant public health problem is eagerly awaited.

Conclusion

Egg-associated outbreaks of *Salmonella* are becoming an all too common problem for public health authorities across Australia, with this outbreak investigation contributing to the mounting body of evidence highlighting salmonellosis and its links to eggs. Effective control of this issue remains elusive. It requires a more concerted effort by public health and regulatory authorities to not only address issues with egg handling and use by food premises but also to improve consumer understanding of potential risk and to increase the egg industry's awareness of this as a public health issue.

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Policy and guidelines

The strategic plan for control of tuberculosis in Australia: 2011–2015

Key challenges, priorities and actions for tuberculosis (TB) control in Australia:

Challenges

- Maintaining the commitment to TB control in the face of low TB incidence in Australia
- Developing new strategies for TB control in a period of high rates of immigration, particularly from countries with high burdens of TB
- Managing increasing drug resistance
- Maintaining a workforce skilled in clinical, laboratory, and public health aspects of TB control
- Maintaining universal access to rapid and reliable diagnosis and treatment for TB

Priorities

- To provide a high standard of diagnosis of and treatment for TB
- To enhance surveillance for TB in groups at higher risk
- To reduce the disparities in TB rates among population sub-groups within Australia
- To minimise the development of drug resistance within Australia
- To ensure the continued provision of safe, timely laboratory diagnosis of TB
- To provide regional support and leadership for the elimination of TB

Key actions

- Develop a strategy for awareness campaigns for primary care and organisations representing high risk groups
- Develop a plan for recruitment, training and retention of the TB workforce
- Develop a national strategy for long term assured supply of quality TB diagnostics and medications
- Publish national practice guidelines for TB
- Increase engagement with regional partners in TB control, particularly with reference to the Western Province of Papua New Guinea
- Achieve real time national reporting of combined clinical and laboratory data

1. Executive summary

Australia is in the enviable position of having achieved and maintained one of the lowest rates of TB in the world. Despite this, TB continues to pose challenges, reflecting the ongoing global problem. These challenges include an increasing incidence of multi-drug-resistant TB (MDR-TB), the development of extensively drug-resistant TB (XDR-TB), the human immunodeficiency virus (HIV) pandemic, and immigration to Australia. Local challenges include the need to secure supplies of diagnostics and pharmaceuticals and an ageing workforce.

In the Australian-born population the rate of TB is very low and elimination is likely in the next 20 years. However, absolute numbers of TB notifications are increasing with 80%–90% of Australia's new cases occurring in arrivals from high burden countries,[1] including student and healthcare worker arrivals. It is predicted that by 2056 Australia's population will have risen from 22 to 35 million largely due to new arrivals.[2] The burden of TB in Australia will depend on future immigration policy, the control of TB in new arrivals, and the detection of TB as migrants age.

In order to maintain TB control in Australia we need to ensure that there is a continuing high standard of diagnosis and treatment. This will require the

current TB control infrastructure to be continued, including reporting mechanisms, laboratories, workforce and communication.

To further improve local TB control, we need to prioritise higher-risk groups; Aboriginal and Torres Strait Islander peoples and overseas-born persons. In addition, support for global TB control activities through continued engagement in regional TB control programs and by improving existing and developing new diagnostics, treatments and vaccinations is required.

2. Vision

Our vision is to eliminate TB within Australia.

3. Guiding principles

The guiding principles underpinning Australia's response to TB are:

- strong state and territory based TB programs, ensuring a close working relationship between public health, laboratory, and clinical services, with strategic advice provided by the National Tuberculosis Advisory Committee (NTAC);
- continued leadership provided by Australian Governments to facilitate national policy formulation, coordination, and implementation;
- early diagnosis and effective programmatic management of TB consistent with the World Health Organization (WHO) Stop TB Strategy;
- active case finding in high risk groups with prompt recognition and facilitation of management of comorbidities (e.g. HIV);
- removing barriers to effective and prompt diagnosis, management and treatment of persons with active TB to ensure efficient passive case finding;
- timely surveillance to monitor and evaluate TB control efforts;
- participation in global and regional TB control; and
- consistency with the Stop TB Strategy.

4. Governance: The NTAC, CDNA and AHPPC

In 1999, the Communicable Diseases Network Australia (CDNA), a sub-committee of the Australian Health Protection Principal Committee (AHPPC) endorsed the formation of NTAC. NTAC has two terms of reference:

1. To provide strategic, expert advice to CDNA on a coordinated national and international approach to TB control.

2. To develop and review nationally agreed strategic and implementation plans for the control of TB in Australia.

5. Goals, Objectives and Indicators

Goal	Objective	Indicator
To ensure sound tuberculosis (TB) control through rapid diagnosis, treatment and notification of TB.	Maintain awareness and education of all stakeholders, including professionals and local communities, of the continuing importance of TB control within Australia. Ensure accurate and timely diagnosis. Ensure timely access to appropriate treatment. Encourage communications amongst and between all stakeholders.	Proportion of cases with a diagnostic delay of greater than 1 month. Complete and current national guidelines. Proportion of TB notifications confirmed by microbiological laboratory diagnosis. Proportion of laboratories meeting recommended turn around time. Proportion of successful treatment of TB. Proportion of cases initially treated in Australia who relapse within 5 years of treatment. Proportion of culture-confirmed cases that undergo drug susceptibility testing.
To improve surveillance and reporting.	Ensure timely and accurate reporting of TB at all levels.	Proportion of TB cases with a recorded HIV status. Completeness of quarterly reporting. Publication of a combined notification and laboratory annual TB report by December of the following year. Annual reporting to WHO.
To eliminate TB in the Australian-born population.	Prevent the transmission of TB within Australia. Ensure prompt and effective contact tracing.	Incidence of TB in: • Indigenous Australian-born children/adults; • Australian-born non-indigenous children/adults. Number of cases of TB acquired within Australian health care institutions/ laboratories.
To reduce the difference in the incidence of TB between the overall Australian rate and specific higher risk groups.	Enhance the extent and effectiveness of special TB programs for high risk groups, including: • Active case finding, particularly in recent migrants; • Detection and management of TB infection. Work collaboratively with the Department of Immigration and Citizenship (DIAC).	Incidence of TB in Aboriginal and Torres Strait Islanders. Incidence and characteristics of TB in: • Overseas born persons; • Healthcare workers; • Irregular Maritime Arrivals.
To prevent the development and transmission of drug resistant TB in Australia.	Ensure prompt detection of drug resistance. Ensure effective case management of all cases of TB. Ensure good infection control practices in clinical and laboratory settings.	Time to identification of drug resistant TB. Incidence and characteristics of drug resistant TB acquired within Australia. Incidence and characteristics of drug resistant TB in migrants.
To assist global TB control activities.	Advocate and participate in actions towards TB control in the region. Contribute to the Western Pacific Region (WPR) Technical Advisory Group.	Incidence of TB in the region. Report Australia's participation in global control activities, annually.

6. Background

Tuberculosis a major global health problem

WHO estimates that there were 9.4 million incident cases, with 14 million prevalent cases of TB in 2009. There were 1.3 million deaths among HIV-negative people and 0.38 million deaths among HIV-positive people. There were 0.44 million incident cases of MDR-TB globally in 2008 and 0.15 million deaths from MDR-TB. It was estimated that in 2009 MDR-TB accounted for 3.3% of incident cases of TB. XDR-TB has now been confirmed in 58 countries.[3] TB is one of the world's most prevalent infectious killers.

TB continues to be a global problem for many reasons including:

- poverty and conflict;
- lack of political commitment;
- health system weaknesses, which affect the programmatic management of TB in both the public and private sectors;
- the lack of suitably trained and qualified human resources;
- the lack of quality assured drugs;
- the lack of standard infection control activities;
- migration (both in-country and between countries);
- the co-existence of TB with HIV;
- the increasing proportion of MDR-TB cases; and
- the recent emergence of XDR-TB.

The changing epidemiology of TB in our region and globally will impact on TB control in Australia in the years to come. In particular, the high prevalence of MDR-TB in some countries including the Asia–Pacific region will be reflected in TB cases in Australia in the future, and TB services need to plan for this development. The improvements in care of MDR-TB patients have made it possible to control the disease in over 50% of cases but this requires special multidisciplinary care by expert groups.

Tuberculosis in the Western Pacific Region

In October 2010, the Western Pacific Regional Office (WPRO) adopted the *Regional Strategy to Stop Tuberculosis in the Western Pacific (2011–2015)* with the goal of reducing by half the prevalence and mortality from all forms of TB by 2015. According to the latest WHO estimates, the WPR is not likely to achieve its goal. In 2007, there were an estimated 1.9 million incident TB cases and 0.3 million TB deaths in the WPR. China, the Philippines, Viet Nam and Cambodia together account for 93% of incident cases in the WPR.[4]

Particular challenges arise for Australia because of historical relationships with countries within the WPR and the special relationship with Papua New Guinea (PNG) across the Torres Strait. Traditional peoples on both sides of this narrow seaway have family and kinship relationships and are entitled to travel, with comparative ease, across the border.

The failure to control TB within neighbouring countries poses direct public health threats to Australia, as seen in the Treaty Zone between the outer Torres Strait Islands of Queensland and the various villages of the South Fly District of the Western Province of PNG. Transmission of MDR-TB has been recognised among PNG nationals accessing health care in the Torres Strait Islands within the treaty zone. From 2004 to 2007, 24 cases of MDR-TB were diagnosed among these visitors representing a substantial demand on human and financial resources.[5]

Tuberculosis in Australia

In the past three decades, Australia has achieved and maintained one of the lowest rates of TB in the world.

The majority (75%) of the Australian resident population are born in Australia.[6] This includes those born of immigrant parents. In this group, the rate of TB disease is very low, with new disease cases coming largely from the previously infected elderly population.

The present low rate of TB in Australia can be attributed to improved socioeconomic circumstances in the 20th century and the vision and sound public health programs of policy makers, clinicians and the political commitment of all Australian governments. They laid the framework for the very successful National TB Campaign following World War II.

The low rate of TB has been maintained despite large-scale immigration from countries, all with considerably higher TB rates than Australia. This is largely the result of effective pre-migration screening and the activities of specialised, multidisciplinary TB services in the States and Territories.

Prior to 1950, the incidence of active TB in Australia was over 45 per 100,000. During the 1950 to 1976 National Tuberculosis Campaign, there was a rapid and sustained decline in the notification rate of new cases.[7]

Since 1986, there has been a further decline in the rate of notified cases to a plateau of 4.8–5.9 per

100,000. In 2008, there were 1,210 cases (5.5 per 100,000) of TB reported in Australia. These rates are considerably lower than the global incidence of 137 per 100,000 in 2009 and continue to compare favourably with other developed countries.[8]

Mortality rates due to TB have also declined substantially from 10 per 100,000 in 1954 to less than 1 per 100,000 in 2008.

Although rates of TB in Australia have remained low, the absolute numbers of TB cases increased by 33% between 1998 and 2008, corresponding with Australia's migration policy and increasing population. Additionally, specific subgroups, such as Indigenous people and persons born overseas, still have rates many times those of non-Indigenous Australian-born persons.

Recently, the emergence of drug resistance has translated into a marked increase in cost per case managed. Despite this, TB services have not grown to meet changes in demand.

MDR and XDR-TB

The emergence worldwide of MDR-TB, and more recently XDR-TB,[9] has highlighted the importance of sustained TB control programs and systems of care to avoid the development of resistant strains, and to appropriately diagnose and manage the cases that arise.

In 2008, an estimated 440,000 cases of MDR-TB emerged globally. India and China carry the greatest estimated burden of MDR-TB, together accounting for almost 50% of the world's total cases. More than three quarters of the estimated cases of MDR-TB occur in previously untreated patients. The proportion of MDR-TB among new cases and previously treated cases of TB reported globally from 1994 through 2009 ranged from 0% to 28.3% and from 0% to 61.6%, respectively. The highest proportions of MDR-TB cases, and the most severe drug-resistance patterns, appeared in the countries of the former Soviet Union.

In 2009, there were 31 cases (2.9%) of MDR-TB identified in Australia from all isolates tested. This proportion was higher than in previous years, which had been around 1.5% of isolates tested. This highlights an increasing trend in MDR-TB case identification since drug susceptibility data collection began in 1986.

NTAC has identified that a large proportion of cases of MDR-TB occurring since 2006 were in PNG nationals who were provided with humanitarian treatment by and in Australia.

Recent documented difficulties in TB control in other high income nations with a low TB burden, and the concerns regarding TB control in countries in our region highlight the need for continued vigilance.[10,11]

The US Centers for Disease Control and Prevention (CDC) and WHO reported the results of an international survey that found 2% of more than 17,000 MDR-TB isolates collected between 2000 and 2004 were XDR-TB.[12] By the end of 2008, 58 countries had reported at least 1 case of XDR-TB. In Australia there has been one confirmed case of XDR-TB identified from a retrospective analysis of laboratory records since 2004 in accordance with the WHO case definition (2006). The financial and human resource cost of the management of this one case has been substantial.

7. Australia's priority populations

NTAC has identified the following groups as being at higher risk of TB than the rest of the population. These populations have rates of TB disease much greater than 6 per 100,000.

Persons in close contact with active disease

Special attention is paid to those in recent contact with infectious TB throughout Australia. Australia's excellent TB control activities identify transmission of TB early and treat latent infection to prevent ongoing transmission, but occasionally a large cluster of infected cases is found. Contact tracing requires extensive and prolonged public health action.

Indigenous Australians

The rate of TB in Indigenous Australians has decreased over the past 10 years but remains approximately 7 times the non-Indigenous rate. Rates increase with age and transmission of TB to infants and children still occurs. Indigenous Australians have higher rates of infection, disease, hospitalisation and mortality from TB than non-Indigenous Australians.[13] Observations from contact tracing and molecular testing have shown the clustering of cases in households, remote and town-camp communities and in regional areas. From 2000 to 2010 there were 24 cluster-linked cases identified in a network of Indigenous people from coastal northern New South Wales to southern Queensland.

Overseas-born persons

Immigrants from high burden countries have contributed to more than 80% of TB cases in Australia since 2000. The rates in middle income and less well resourced countries vary between 20 and 400 cases per 100,000 population. Most notified cases in

Australia have places of birth in the WPR and the South East Asia Region (SEAR). The rates of TB in overseas-born people have risen steadily over the past 10 years from 14.1 cases per 100,000 in 2000 to 20.4 cases per 100,000 in 2010. The age incidence rate in the overseas-born tends to show three peaks: one among children aged less than 5 years; a second among young adults (15–34 years); and a third peak in the over 65 year age group.

Secondary and tertiary students

Over 200,000 student visas were granted in 2009–10.[14] The majority of these students were from countries in the WPR and SEAR with a high burden of TB. Whilst students are screened for active TB prior to entry to Australia, after arrival they have a predictably higher rate of TB. When active disease develops in this group it can have a serious social and economic impact disrupting their education and contributing to great stress in the family off-shore. Additionally, the community and media take a special interest in such incidents.

Strengthened relationships with educational institutions are required to promote screening for TB infection and to detect active TB earlier.

Health care workers

Health care workers (HCW) have been identified recently as an important population sub-group amongst the overseas-born in Australia. In 2001, there were 17 cases of TB in overseas-born HCWs, rising to 83 cases in 2008. This rise is attributable to the increasing recruitment of HCWs from high burden countries. While there have been no recent reports of TB transmission to patients from HCW, the possibility of this occurring when there are potentially high rates of TB infection in the migrant workforce needs to be recognised. HCWs serve in a variety of health care settings in the community e.g. hospitals, community clinics and aged care facilities and provision of appropriate TB screening and follow up services for HCWs are essential.

Other potential risk groups

Elderly and immunosuppressed persons

The ageing population (those born before 1950) have a higher rate of TB infection and are at an increased risk of progressing to active disease due to therapeutic interventions or comorbidities that cause immunosuppression.

Likewise, people of any age referred for immunosuppressive treatment or for solid organ transplants are at an increased risk of TB infection or progression to disease from TB infection. Therefore, those on high dose corticosteroid treatment or tumour necrosis factor (TNF) inhibitors, now used widely for the management of rheumatoid arthritis, ankylosing spondylitis, psoriasis and inflammatory bowel disease and prospective transplant recipients, require appropriate screening and treatment for TB infection prior to commencing immunosuppression.

Tuberculosis and HIV co-infection

HIV infection has been globally recognised as an important risk factor for increased susceptibility to TB infection and the risk of developing active TB. Globally, TB is one of the major causes of death amongst people with HIV. HIV positive cases are also more likely to have extra-pulmonary disease than non-HIV infected cases.

To date, there has been a limited overlap between those with HIV infection and those with TB in Australia. HIV-TB co-infection is mainly found in overseas-born persons. In 2008, information on HIV testing status at the time of TB diagnosis was reported in only 83% of TB notifications nationally and of these, less than 1% (11 cases) were identified as being HIV positive. The results of HIV testing of all TB cases are still not available in Australia. This needs to be addressed.

Other risk groups

In contrast to previous and current overseas experience, the risk of TB disease in some groups in Australia, such as the homeless, prison residents and nursing home residents, is very low. The background TB infection rate in the first two groups is likely to be low, while the rate in the last group is likely to be higher. Regardless, all of these settings have the potential for isolated outbreaks of TB to occur.

8. Surveillance and laboratory services

Surveillance

The Commonwealth, together with NTAC, monitors the incidence of TB on a national basis using agreed enhanced data provided by State and Territory health authorities and laboratories, in conjunction with the National Notifiable Diseases Surveillance System (NNDSS).

The key elements of TB surveillance include:

- maintenance of the NNDSS and enhanced data systems; and
- reporting to WHO.

The Governments of Australia need to continue to maintain national TB surveillance in order to inform TB policy. This requires close working relationships

with the States and Territories and national bodies, including NTAC, the Department of Immigration and Citizenship, and the Public Health Laboratory Network (PHLN).

Laboratory services

State TB reference laboratories have responsibility for TB testing and antibiotic susceptibility testing. There are two Supranational TB Reference Laboratories in Australia.

The five state mycobacterium reference laboratories (MRLs) undertake the following functions:

- provision of basic TB diagnostic services in cooperation with other public and private laboratories;
- provision of specialised TB diagnostic services, such as mycobacterial identification, drug susceptibility testing, and rapid molecular detection of drug resistance;
- provision of molecular epidemiological typing by a nationally-approved method;
- provision of specialised diagnostic services for the investigation of clinically-significant non-tuberculous mycobacteria (NTM) infections;
- delivery of national quality assurance programs; and
- training of clinical, public health and laboratory personnel to maintain expertise in mycobacterial diagnostics in both the public and private sectors.

Like TB services in general, reference laboratories face the challenges of a workforce with diminishing expertise and a predicted increase in workload. Laboratory-specific challenges include the rising costs of providing a range of rapid molecular diagnostic tests. Compliance with progressively more stringent biosafety standards presents an additional challenge.

Expertise within the MRLs could be enhanced by the establishment of a mentoring scheme within Australia and by encouraging MRLs to support national TB laboratory services in neighbouring higher burden TB countries. TB laboratories must remain an integral part of the national and state TB control programs, and must be integrated into the programs' computerised data management systems. Finally, to meet the challenges listed above, the MRLs will require the continued support of federal and state governments so that the laboratories can remain an integral part of the nation's TB control program.

9. Workforce

TB care and control is an essential element of the health system, and like other parts of the system is increasingly vulnerable to constraints on human resources. In Australia, as in all low burden well resourced countries, the TB workforce is ageing, the distribution is uneven, the capacity diminishing and the knowledge and skills lost are not being replaced. Although TB prevalence continues to decrease, absolute numbers are increasing, and cases are becoming more complex, demanding more time and more skilled health workers. In line with the increasing sub-specialisation of health care, TB care and control is becoming a sub-specialty and TB disease is becoming an unfamiliar issue in an overcrowded undergraduate health curriculum.

Replacement, education and training of the workforce needs to be planned to enable continuing control of TB.

Strengthening the capacity of health and community services to respond effectively to TB will reduce the burden of this infection on Australia.

It is essential to:

- ensure that there is a sufficient pool of expertise in the future to maintain the necessary clinical, laboratory and public health activities for TB control in Australia; and
- maintain awareness in the general health workforce.

10. Abbreviations

AHPPC	Australian Health Protection Principal Committee
CDC	Centers for Disease Control and Prevention
CDNA	Communicable Diseases Network Australia
DIAC	Department of Immigration and Citizenship
HCW	health care workers
HIV	human immunodeficiency virus
LTBI	latent tuberculosis infection
MDR-TB	multi-drug-resistant tuberculosis
MRL	mycobacterium reference laboratories
NNDSS	National Notifiable Diseases Surveillance System
NTAC	National Tuberculosis Advisory Committee
NTM	non-tuberculous mycobacteria
PHLN	Public Health Laboratory Network
PNG	Papua New Guinea
SEAR	South East Asia Region
TB	tuberculosis
TNF	tumour necrosis factor
WHO	World Health Organization
WPR	Western Pacific Region
WPRO	Western Pacific Regional Office
XDR-TB	extensively drug-resistant tuberculosis

11. Definitions

In this plan the following definitions apply:

Active case finding is the deliberate search for TB disease or infection by means of clinical and radiographical examination, supplemented by tuberculin skin testing.

Case management is a system of healthcare delivery in which an individualised treatment plan for the patient is developed by a multidisciplinary team to achieve established patient care outcomes.

Contact refers to a person who has shared air with a person who has been notified with active disease.

Elimination of TB refers to less than one infectious (sputum smear positive) case per million in the general population.

Extensively drug-resistant tuberculosis is defined as MDR-TB plus resistance to any fluoroquinolone and at least one second-line injectable (amikacin, kanamycin or capreomycin).

Health care workers refer to all health care professionals, including trainees, students, and employees of health care establishments who have contact with patients.

Health undertaking is a medical service required for visa applicants who, through their medical examination for an Australian visa, are found to have evidence of exposure to TB or other diseases that may be of concern (such as hepatitis B).[15]

High risk groups refer to population segments with increased risk of exposure to TB

Immunosuppressed are persons whose immune response is inadequate and consequently their ability to fight infections is impaired.

Multi-drug-resistant tuberculosis is defined as TB caused by strains of *Mycobacterium tuberculosis* resistant to at least isoniazid and rifampicin.

Passive case finding involves detecting active TB disease among patients who present to medical services for diagnosis of symptoms.

Tuberculosis disease refers to an infectious disease caused by the *Mycobacterium tuberculosis* complex.

Tuberculosis (latent) infection refers to a subclinical infection with the tubercle bacilli without clinical, bacteriological or radiological features of disease.

Surveillance refers to the ongoing, systematic collection, analysis, interpretation, and dissemination of data regarding a health-related event for use in public health action.

12. Acknowledgment

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Quarterly reports

OzFoodNet quarterly report, 1 October to 31 December 2011

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. In each Australian state and territory, OzFoodNet epidemiologists investigate outbreaks of enteric infection. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, occurring in Australia from 1 October to 31 December 2011.

Data were received from OzFoodNet epidemiologists in all Australian states and territories. The data in this report are provisional and subject to change.

During the 4th quarter of 2011, OzFoodNet sites reported 322 outbreaks and clusters of enteric illness, including those transmitted by food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports may be delayed, meaning that these figures under-represent the true burden of enteric disease outbreaks. In total, these outbreaks affected 4,887 people, of whom 192 were hospitalised. There were 17 deaths reported during these outbreaks. The majority of outbreaks (70%, n=226) were due to person-to-person transmission (Table 1), with 57% (n=128) of these occurring in residential aged care facilities.

Foodborne/waterborne and suspected foodborne disease outbreaks

There were 36 outbreaks during this quarter where consumption of contaminated food or water was suspected or confirmed as the primary mode of transmission (Table 2). These outbreaks affected 453 people and resulted in 54 hospitalisations and 1 death. This compares with 34 outbreaks for the 3rd quarter of 2011[1] and a 5 year mean of 33 outbreaks for the 4th quarter between 2006 and 2010.

Salmonella enterica serotypes were identified as the aetiological agent for 9 outbreaks during this quarter (the majority were *S.* Typhimurium, refer to Table 2 for more detail). Of the remaining outbreaks, 5 (14%) were due to fish poisoning (2 ciguatera, 3 scombroid), 3 (8%) were due to *Clostridium perfringens*, and single outbreaks were due to *Bacillis cereus*, *Campylobacter*, and norovirus. In 16 outbreaks (33%), the aetiological agent remained unknown.

Fifteen outbreaks (42%) reported this quarter were associated with food prepared in restaurants. Further detail on food preparation settings associated with foodborne or suspected foodborne outbreaks is provided below in Table 3.

To investigate these outbreaks, sites conducted 9 cohort studies, 2 case control studies and collected descriptive case series data for 22 investigations. For 3 outbreaks no individual patient data were collected. The evidence used to implicate food vehicles included

Table 1. Outbreaks and clusters of gastrointestinal illness reported by OzFoodNet, 1 October to 31 December 2011, by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total
Foodborne/waterborne and suspected foodborne	36	11
Person-to-person	226	70
Unknown (*Salmonella* cluster)	14	4
Unknown (Other pathogen cluster)	4	1
Unknown	42	13
Total	322	100*

* Percentages do not add up due to rounding.

Table 2. Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites,* 1 October to 31 December 2011 (n=36)

State or territory	Month	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
Multi-jurisdictional	December	Cruise/airline	*S.* Typhimurium PT 135a	16	3	A	Unknown
ACT	October	Commercial caterer	Unknown	9	0	D	Suspected mixed sandwiches
ACT	December	Restaurant	*S.* Typhimurium PT 170 MLVA profile 03-09-07-14-523	41	7	M	Chicken Caesar roll containing raw egg mayonnaise
NSW	October	Bakery	Unknown	3	0	D	Unknown
NSW	October	Camp	Unknown	8	4	D	Suspect cooked pasta
NSW	November	Commercial caterer	Unknown	16	0	A	Suspect lamb curry
NSW	November	Restaurant	*S.* Typhimurium PT 9	3	1	D	Unknown
NSW	November	Restaurant	Unknown	12	0	D	Unknown
NSW	November	Restaurant	Unknown	34	0	D	Unknown
NSW	November	Restaurant	Scombroid fish poisoning	4	4	D	Fresh tuna salad
NSW	December	Private residence	Unknown	9	0	D	Unknown
NT	October	Takeaway	Unknown	3	0	D	Unknown
Qld	October	Restaurant	Unknown	3	0	D	Unknown
Qld	October	Restaurant	Norovirus	6	0	D	Unknown
Qld	November	Fair/festival/mobile service	*S.* Birkenhead	37	9	D	Unknown
Qld	November	Primary produce	Ciguatera fish poisoning	6	0	D	Spanish mackerel
Qld	November	Restaurant	Unknown	19	0	D	Unknown
Qld	November	Restaurant	Scombroid fish poisoning	3	3	D	Yellow-tail kingfish
Qld	December	National franchised fast food	Unknown	4	0	D	Unknown
Qld	December	Primary produce	Ciguatera fish poisoning	2	0	D	Coral trout
Qld	December	Restaurant	*S.* Typhimurium PT 197	25	2	D	Unknown
SA	November	Commercial caterer	*S.* Typhimurium PT 9	27	7	D	Multiple foods
Vic	October	Hospital	*C. perfringens*	4	0	D	Unknown
Vic	October	Hospital	*C. perfringens*	8	0	D	Unknown
Vic	October	Restaurant	Scombroid fish poisoning	3	0	M	Tuna
Vic	November	Commercial caterer	*C. perfringens*	17	0	D	Suspected roast beef
Vic	December	Private residence	*Campylobacter jejuni*	22	1	A	Suspected private drinking water supply (untreated rainwater)
Vic	December	Private residence	*S.* subsp I ser 4,5,12:i:- PT 193	4	1	D	Homemade pork salami

Table 2 *continued.* Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites*, 1 October to 31 December 2011 (n=36)

State or territory	Month	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
Vic	December	Private residence	*S.* Typhimurium PT 9	4	0	D	Raw egg chocolate mousse
Vic	December	Restaurant	*B. cereus*	12	0	M	Multiple foods
Vic	December	Restaurant	Unknown	4	1	A	Moroccan chicken salad
Vic	December	Restaurant	Unknown	14	0	A	Suspect mango sticky rice
Vic	December	Takeaway	*S.* Typhimurium PT 170	37	11	M	Pizza containing egg and raw egg chocolate mousse
WA	November	Private residence	Unknown	17	0	D	Chicken biriyani
WA	December	Commercial caterer	Unknown	10	0	D	Unknown
WA	December	Restaurant	Unknown	7	0	D	Unknown
Totals				453	54		

* No foodborne or suspected foodborne outbreaks were reported by Tasmania.

A Analytical epidemiological association between illness and 1 or more foods.

D Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission.

M Microbiological confirmation of agent in the suspected vehicle and cases.

MLVA Multi-locus variable number of tandem repeat analysis.

PT Phage type.

analytical evidence in 5 outbreaks and microbiological evidence in 4 outbreaks. Descriptive evidence alone was obtained in 27 outbreak investigations.

Table 3. Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet, 1 October to 31 December 2011, by food preparation setting

Food preparation setting	Outbreaks
Restaurant	15
Private residence	5
Commercial caterer	5
Takeaway	2
Primary produce	2
Hospital	2
Fair/festival/mobile service	1
National franchised fast food	1
Bakery	1
Camp	1
Cruise/airline	1
Total	36

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

Multi-jurisdictional

There was 1 reported multi-jurisdictional outbreak of foodborne or suspected foodborne illness during the quarter.

A multi-jurisdictional outbreak investigation (MJOI 2011-004) was initiated following reports of gastroenteritis in passengers (from New South Wales, Victoria, South Australia and Western Australia) and crew aboard a West Australian-owned ship cruising Papua New Guinea (PNG). There were 3 confirmed *S.* Typhimurium Phage Type (PT) 135a cases (1 case each from South Australia, Victoria and Western Australia). Fourteen of 31 people (7 passengers and 7 crew) who returned questionnaires and an additional 2 cases who did not compete the questionnaire, reported illness. Two crew members and 1 passenger were hospitalised. There was no clear association between illness and eating a particular food item. The majority of food consumed on the ship was supplied from Australia, mostly from Queensland. All meat was from Western Australia. Some produce from PNG was used on board, including eggs, pineapple, watermelon, mangoes, paw paw, cucumber, pumpkin, coconut and avocado. A number of sauces (including mayonnaise, hollandaise, anglaise) and desserts (ice cream, tiramisu) contained raw eggs. An inspection of the vessel was conducted, but no samples were collected. The food vehicle was not identified.

Australian Capital Territory

There were 2 reported outbreaks of foodborne or suspected foodborne illness during the quarter, of which one was due to *S.* Typhimurium and no pathogen could be identified for the other.

Description of key outbreaks

An investigation identified 41 cases of gastroenteritis, including 23 laboratory confirmed cases of salmonellosis (7 hospitalisations) linked to a restaurant. Typing of human isolates detected *S.* Typhimurium PT 170 / multi-locus variable number of tandem repeats analysis (MLVA) profile 03-09-07-14-523. Cases all reported having eaten a chicken Caesar roll, which contained a raw egg mayonnaise from the restaurant. Mayonnaise sampled from the restaurant and on-farm sampling of a New South Wales egg producer supplying eggs to the bakery also tested positive for *S.* Typhimurium PT 170 with an identical MLVA profile 03-09-07-14-523.

New South Wales

There were 8 reported outbreaks of foodborne or suspected foodborne illness during the quarter, of which one was due to *S.* Typhimurium, one was due to scombroid fish poisoning and no pathogen could be identified for the remaining outbreaks.

Description of key outbreaks

A public health unit was notified of the hospitalisation of four school children who had attended a 3 day camp. Eight of 111 students developed symptoms of vomiting and abdominal pain 4 hours (median) after consuming a meal of spaghetti bolognaise. The symptom profile for the cases was consistent with a foodborne illness caused by a pre-formed toxin. Other children, both from the same school and other schools who ate the meal, did not report symptoms. The environmental health investigation identified a number of breaches of the food safety regulations including the improper cooling of high risk foods, specifically a large container of spaghetti was reported to be at 22 degrees 3 hours after cooking. A warning letter was issued by the local council.

Northern Territory

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

Following a birthday party held at a private residence, 3 of 12 attendees experienced mild gastroenteritis.

Food was purchased from commercial premises and a supermarket. A cohort study was attempted but did not identify a food vehicle. A viral agent or toxin was thought to be responsible for disease, but no clinical or food specimens were obtained.

Queensland

There were 9 reported outbreaks of foodborne or suspected foodborne illness during the quarter, of which two were due to Ciguatera fish poisoning, and one each due to scombroid fish poisoning, norovirus, *S*. Birkenhead, and *S*. Typhimurium. For the remaining outbreaks, no pathogen could be identified.

Description of key outbreaks

Investigators were notified of an outbreak of gastrointestinal illness among a group of 9 people who had consumed a meal at a restaurant. Six of the 9 people developed symptoms of gastrointestinal illness, with anecdotal evidence suggesting that other patrons also experienced symptoms. No restaurant staff members reported any recent illness. Two faecal specimens collected during the investigation tested positive for norovirus. Salads were suspected as a possible food vehicle as all cases had consumed salad with their respective meals and environmental investigations revealed considerable use of bare hands whilst preparing and serving salads.

Authorities investigated an outbreak of salmonellosis among attendees (150–200) of a community event. A cohort investigation was conducted involving 48 attendees of whom 37 reported symptoms of gastrointestinal illness, with 9 hospitalised. *S*. Birkenhead was identified in 11 of these cases. Persons who had consumed a potato and pumpkin curry were at higher risk of illness, odds ratio (OR) 8.0 (95% CI: 1.4–47.2). One environmental sample tested positive for *S*. Birkenhead from the food processor used for preparing home-made lemonade. No food or environmental source of infection was identified during the investigation.

A large outbreak of *S*. Typhimurium PT 197 (MLVA profile 04-15-09-09-490) was identified among a group of 62 patrons (no staff reported illness) who had attended a dinner function at a community club. A cohort investigation identified that 25 of 55 persons experienced symptoms of gastrointestinal illness. Six cases were laboratory confirmed with *S*. Typhimurium PT 197 and 2 cases were hospitalised. Extensive environmental investigations of the kitchen area identified a number of deficiencies in food storage, food safety knowledge, and cleanliness. Environmental swabs and samples collected during this investigation were negative for bacterial pathogens. No food vehicle or source of infection could be identified from this investigation.

South Australia

There was 1 outbreak of foodborne or suspected foodborne illness investigated during the quarter.

Twenty-seven cases (74% female) of *S*. Typhimurium PT 9 were identified after an outbreak at a community event associated with food consumed from a temporary food stall. Foods consumed by cases included a variety of salads and vegetarian patties, although no common food item was identified. No pathogens were detected in 48 samples from an environmental inspection of the premises where the food was prepared. The company that produced the food also catered for other events and ran a retail outlet; 1 additional case who ate food from the retail outlet was identified.

Tasmania

There were no reported outbreaks of foodborne or suspected foodborne illness during the quarter.

Victoria

There were 10 reported outbreaks of foodborne or suspected foodborne illness of which three were due to *C. perfringens*, two were due to *S*. Typhimurium, and one each due to *Salmonella* sub-species I, *Campylobacter, B. cereus*, and scombroid fish poisoning. There was also 1 suspected waterborne outbreak during the quarter. For the remaining outbreaks no pathogen could be identified.

Description of key outbreaks

An outbreak of gastroenteritis occurred among attendees of a family reunion, where approximately 130 meals were supplied by a commercial caterer. Seventeen of 20 people interviewed reported illness, with 5 faecal specimens testing positive for *C. perfringens* enterotoxin. All of the interviewed cases reported eating the meat dishes, with 2 people specifically reporting only eating beef and those not ill either ate no meat at all or no beef specifically. Investigation of the caterer revealed that they were operating a business that was not registered under the *Food Act 1984* with the local council, and food preparation procedures were inappropriate.

An outbreak of *Campylobacter* occurred at a youth training camp involving 24 attendees and a host family of three. The accommodation was on a rural property and the main source of drinking water was a rainwater tank. Twenty-two of 26 people interviewed reported illness. A heavy rainfall event occurred over the weekend of the camp and this may have washed a large amount of debris into the rainwater tank resulting in contamination with *Campylobacter*, commonly found in bird faeces.

Statistical analysis of food and water exposures of camp attendees identified consumption of rainwater at Saturday breakfast, risk ratio (RR) 1.6 (95% CI 1.01–2.46), and Sunday breakfast, RR 1.6 (95% CI 1.01–2.46) as risk factors, supporting this hypothesis. Two rainwater tank samples, collected 10 and 18 days after the last day of the camp, and 3 food samples (frozen leftover food) were all negative for *Campylobacter*.

Routine follow-up of a single *Salmonella* case identified an outbreak among a group of family and friends who had attended a common dinner party. All 4 attendees consumed raw egg chocolate mousse at the dinner and all reported illness. Two cases were confirmed with *S.* Typhimurium PT 9 and trace-back of the eggs was initiated.

An outbreak of salmonellosis was detected after 4 of 6 family members became ill after eating a home prepared meal together. Homemade pork salami was suspected as the source (eaten only by ill cases) with the index case reporting that salami consumed during the meal had been processed differently to normal practices. Three of 4 cases and a sample of leftover salami were positive for *S.* subsp I ser 4,5,12:i:- PT 193.

An outbreak investigation commenced after routine surveillance detected a cluster of *Salmonella* cases residing in the same geographical area. The majority of cases reported eating from the same pizza and pasta takeaway food premises during their incubation period. Of 37 cases who had eaten at these premises, 36 reported eating either an 'Aussie' pizza or a pizza 'with the lot' (both contained egg). The remaining case reported eating a raw egg chocolate mousse from the venue. Several cases reported that the pizzas were 'soggy' and it is suspected that the pizzas, and specifically the egg on these pizzas, were not cooked thoroughly. The premises were closed on the day that the outbreak was linked to it. From over 80 different food and environmental samples, 5 were positive for *S.* Typhimurium PT 170, including 2 samples of egg pulp and a sample of raw egg chocolate mousse. Two other positive foods (grated cheese and tomato puree for pizzas) were likely to have been cross contaminated from the egg pulp during food preparation. One case had frozen leftover 'Aussie' pizza, which was sent for analysis and also found to be positive for *S.* Typhimurium PT 170. An investigation was conducted at the farm that supplied eggs to the food premises and all samples (eggs and environmental samples) were negative for *Salmonella*.

Western Australia

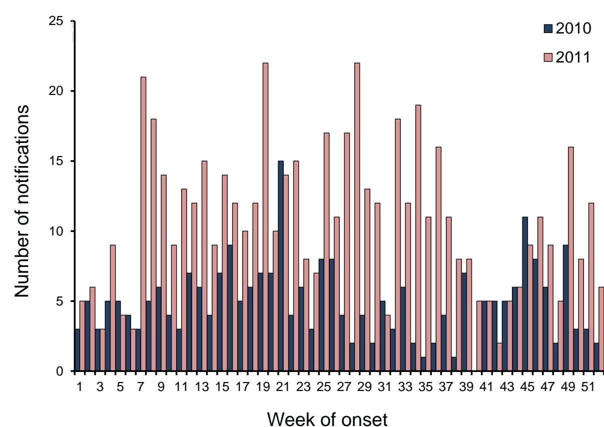
There were 3 reported outbreaks of foodborne or suspected foodborne illness during the quarter, for all of which no pathogen could be identified.

Investigations of note

An increase in notified cases of *Campylobacter* infection was identified in Queensland, in the Cairns (Figure 1) and Townsville (Figure 2) health service districts in the 2nd quarter 2011. The elevated numbers continued throughout the 3rd quarter and into the 4th quarter, though there was a downward trend in the last quarter. By contrast, the number of notified cases of *Campylobacter* in other jurisdictions of Queensland were not above expected levels based on historical data.

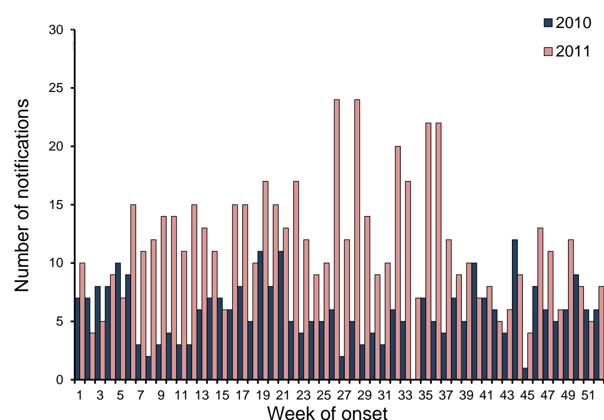
Safe Food Production Queensland and Queensland Health were involved in an extensive follow-up of the chicken meat industry and continue to work closely with a north Queensland abattoir and secondary processors in the supply chain. The implementation

Figure 1: Cairns and Hinterland health service districts *Campylobacter* counts, 2010 and 2011, by week*



* 4th Quarter 2011 runs from weeks 40 to 52

Figure 2: Townsville health service district *Campylobacter* counts, 2010 and 2011, by week*



* 4th quarter 2011 runs from weeks 40 to 52.

of several interventions at the abattoir in September 2011 including disposing of any viscerally contaminated chicken product detected after processing, the requirement to maintain tighter process and product controls, attention to maintenance of plant equipment and training of staff, contributed significantly to the decline in the number of notified cases of *Campylobacter* infection in north Queensland during the 4th quarter.

Comments

The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission, and in this quarter, 70% of outbreaks (n = 226) were transmitted via this route. The number of foodborne and suspected foodborne outbreaks this quarter (n=36) remained consistent with the same quarter of 2010. *S.* Typhimurium continues to be a leading cause of foodborne outbreaks in Australia, with 70% (7/10) of *Salmonella* being due to this serotype.

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

Outbreaks associated with raw or under-cooked egg products continued to be reported this quarter (n = 3). OzFoodNet continues to report on foodborne or suspected foodborne outbreaks associated with the consumption of dishes containing raw or under-cooked eggs, such as raw egg mayonnaise, chocolate mousse, tiramisu and dressings containing raw egg.[2]

A limitation of the outbreak data provided by OzFoodNet sites for this report was the potential for variation in categorisation of the features of outbreaks depending on circumstances and investigator interpretation. Changes in the number of foodborne outbreaks should be interpreted with caution due to the small number each quarter.

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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 54,806 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 April and 30 June 2012 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

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

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

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

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

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2012, by date of diagnosis

Disease	State or territory								Total 2nd quarter 2012	Total 1st quarter 2012	Total 2nd quarter 2011	Last 5 years mean 2nd quarter	Ratio	Year to date 2012	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Hepatitis B (newly acquired)*	0	6	3	9	2	1	10	3	34	51	45	62.0	0.5	85	125.6
Hepatitis B (unspecified)†	35	570	42	179	89	17	468	195	1,595	1,664	1,621	1,659.4	1.0	3,259	3,385.6
Hepatitis C (newly acquired)*,‡	2	8	0	NN	16	3	41	25	95	102	97	98.4	1.0	197	195.8
Hepatitis C (unspecified)†	37	797	33	555	89	61	508	225	2,305	2,588	2,462	2,704.6	0.9	4,893	5,488.2
Hepatitis D	0	1	0	0	0	0	4	1	6	6	12	10.4	0.6	12	19.8
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	1	0.2	0.0	0	0.6
Campylobacteriosis§	108	NN	39	939	442	164	1,217	359	3,268	4,797	4,023	3,713.4	0.9	8,065	8,254.4
Cryptosporidiosis	6	235	71	364	43	12	161	26	918	1,446	470	623.2	1.5	2,364	1,803.2
Haemolytic uraemic syndrome	0	4	0	1	0	0	0	0	5	5	1	3.6	1.4	10	8.8
Hepatitis A	0	9	1	6	0	0	14	6	36	45	32	74.6	0.5	81	143.8
Hepatitis E	1	3	0	1	0	0	3	0	8	15	9	9.4	0.9	23	22.0
Listeriosis	0	10	0	1	1	1	7	1	21	26	19	14.6	1.4	47	39.8
STEC, VTEC‖	2	5	0	2	12	1	1	0	23	36	16	16.4	1.4	59	49.2
Salmonellosis	62	575	119	617	223	47	556	264	2,463	3,956	2,601	2,401.8	1.0	6,419	6,101.2
Shigellosis	2	22	16	21	16	1	26	13	117	191	100	142.0	0.8	308	325.8
Typhoid	0	11	1	1	1	0	6	0	20	56	24	23.6	0.8	76	63.0
Quarantinable diseases															
Cholera	0	1	0	1	2	0	0	0	4	0	4	1.2	3.3	4	2.2
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	2	0.0	0.0	0	0.0

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2012, by date of diagnosis

Disease	State or territory								Total 2nd quarter 2012	Total 1st quarter 2012	Total 2nd quarter 2011	Last 5 years mean 2nd quarter	Ratio	Year to date 2012	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection[¶,**]	319	5,158	675	4,610	1,231	439	4,992	2,924	20,348	22,480	20,211	16,801.8	1.2	42,828	33,469.4
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	1.0
Gonococcal infection[**]	22	1,037	369	670	175	6	649	521	3,449	3,612	3,080	2,430.6	1.4	7,061	4,723.6
Syphilis – congenital[**]	0	0	0	1	0	0	0	0	1	0	0	1.2	0.8	1	2.8
Syphilis < 2 years duration[**]	2	98	6	88	27	5	122	20	368	360	300	340.2	1.1	728	682.6
Syphilis > 2 years or unspecified duration[**]	5	40	16	57	NN	8	127	22	275	308	299	329.2	0.8	583	661.2
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	4	0.8	0.0	0	0.8
Haemophilus influenzae type b	0	0	0	1	1	1	0	1	4	3	5	6.8	0.6	7	11.0
Influenza (laboratory confirmed)	133	2,399	150	1,704	1,399	59	802	694	7,340	1,265	4,124	4,269.6	1.7	8,605	5,146.8
Measles	0	22	0	3	0	0	5	1	31	9	30	16.6	1.9	40	58.2
Mumps	0	49	0	6	2	0	5	5	67	39	38	51.0	1.3	106	111.4
Pertussis	91	1,359	87	1,593	224	268	950	755	5,327	7,198	8,020	4,825.6	1.1	12,525	10,280.8
Pneumococcal disease (invasive)	5	184	23	81	36	12	99	61	501	231	550	437.8	1.1	732	646.2
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	1	2	0	3	0	0	1	1	8	13	14	11.4	0.7	21	23.4
Rubella – congenital	0	0	0	0	0	1	0	0	1	0	0	0.4	2.5	1	0.4
Tetanus	0	0	0	0	0	0	0	0	0	1	2	0.6	0.0	1	2.2
Varicella zoster (chickenpox)[††]	3	NN	34	59	98	9	178	72	453	399	406	364.4	1.2	852	699.0
Varicella zoster (shingles)[††]	13	NN	38	16	432	64	256	284	1,103	1,069	900	645.4	1.7	2,172	1,351.0
Varicella zoster (unspecified)[††]	29	NN	1	1,066	36	23	651	245	2,051	2,078	1,775	1,398.0	1.5	4,129	2,856.8
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	2	1	0	0	0	3	3	6	3.0	1.0	6	7.6
Barmah Forest virus infection	0	78	15	192	4	0	9	34	332	451	395	436.2	0.8	783	1,059.8
Dengue virus infection	5	65	10	85	14	3	87	143	412	689	122	153.0	2.7	1,101	510.0
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	1	0	0.0	0.0	1	0.0
Kunjin virus infection[‡‡]	0	0	0	0	0	0	0	0	0	0	1	0.4	0.0	0	1.0
Malaria	0	16	1	25	0	0	19	9	70	72	89	120.8	0.6	142	243.2
Murray Valley encephalitis virus infection[‡‡]	0	0	0	0	0	0	0	0	0	1	7	2.0	0.0	1	4.2
Ross River virus infection	5	217	62	553	59	3	107	204	1,210	2,293	990	1,367.0	0.9	3,503	3,412.2

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2012, by date of diagnosis

Disease	State or territory								Total 2nd quarter 2012	Total 1st quarter 2012	Total 2nd quarter 2011	Last 5 years mean 2nd quarter	Ratio	Year to date 2012	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.4
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Brucellosis	0	1	0	0	0	0	0	0	1	7	11	8.6	0.1	8	17.8
Leptospirosis	0	9	0	35	0	0	2	0	46	45	51	39.8	1.2	91	101.0
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	3	0	0	0	0	10	0	13	11	17	20.4	0.6	24	40.2
Q fever	0	22	1	45	3	0	3	1	75	102	77	88.0	0.9	177	183.8
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	1.0
Other bacterial infections															
Legionellosis	0	25	0	14	9	2	22	14	86	95	103	89.0	1.0	181	158.4
Leprosy	0	0	0	0	0	0	1	0	1	0	3	2.4	0.4	1	4.8
Meningococcal infection[§§]	0	23	3	12	13	2	9	6	68	32	57	59.8	1.1	100	107.2
Tuberculosis	5	69	7	35	17	2	74	35	244	288	280	267.6	0.9	532	555.4
Total	893	13,133	1,823	13,653	4,717	1,215	12,202	7,170	54,806	58,139	53,506			112,945	

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

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

‡ In Queensland, includes incident hepatitis cases.

§ Not notifiable in New South Wales.

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

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

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

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

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

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

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.

Table 3: Notification rates of diseases, 1 April to 30 June 2012, by state or territory. (Annualised rate per 100,000 population)

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)*	0.0	0.3	5.2	0.8	0.5	0.8	0.7	0.5	0.6
Hepatitis B (unspecified)†	38.3	31.2	72.9	15.6	21.5	13.3	33.3	33.2	28.2
Hepatitis C (newly acquired)*	2.2	0.4	0.0	NN	3.9	2.4	2.9	4.3	2.1
Hepatitis C (unspecified)†,‡	40.5	43.7	57.3	48.5	21.5	47.8	36.1	38.3	40.8
Hepatitis D	0.0	0.1	0.0	0.0	0.0	0.0	0.3	0.2	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis§	118.2	NN	67.7	82.0	106.7	128.5	86.6	61.1	85.3
Cryptosporidiosis	6.6	12.9	123.3	31.8	10.4	9.4	11.5	4.4	16.2
Haemolytic uraemic syndrome	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Hepatitis A	0.0	0.5	1.7	0.5	0.0	0.0	1.0	1.0	0.6
Hepatitis E	1.1	0.2	0.0	0.1	0.0	0.0	0.2	0.0	0.1
Listeriosis	0.0	0.5	0.0	0.1	0.2	0.8	0.5	0.2	0.4
STEC,VTEC‖	2.2	0.3	0.0	0.2	2.9	0.8	0.1	0.0	0.4
Salmonellosis	67.8	31.5	206.6	53.9	53.9	36.8	39.6	44.9	43.6
Shigellosis	2.2	1.2	27.8	1.8	3.9	0.8	1.9	2.2	2.1
Typhoid fever	0.0	0.6	1.7	0.1	0.2	0.0	0.4	0.0	0.4
Quarantinable diseases									
Cholera	0.0	0.1	0.0	0.1	0.5	0.0	0.0	0.0	0.1
Human pathogenic avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmitted infections									
Chlamydial infection¶,**	349.0	282.5	1,172.0	402.6	297.3	344.0	355.2	497.8	359.9
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	24.1	56.8	640.7	58.5	42.3	4.7	46.2	88.7	61.0
Syphilis – congenital**	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years duration**	2.2	5.4	10.4	7.7	6.5	3.9	8.7	3.4	6.5
Syphilis > 2 years or unspecified duration†,**	5.5	2.2	27.8	5.0	NN	6.3	9.0	3.7	5.2
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	0.0	0.1	0.2	0.8	0.0	0.2	0.1
Influenza (laboratory confirmed)	145.5	131.4	260.5	148.8	337.9	46.2	57.1	118.2	129.8
Measles	0.0	1.2	0.0	0.3	0.0	0.0	0.4	0.2	0.5
Mumps	0.0	2.7	0.0	0.5	0.5	0.0	0.4	0.9	1.2
Pertussis	99.6	74.4	151.1	139.1	54.1	210.0	67.6	128.5	94.2
Pneumococcal disease (invasive)	5.5	10.1	39.9	7.1	8.7	9.4	7.0	10.4	8.9
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	1.1	0.1	0.0	0.3	0.0	0.0	0.1	0.2	0.1
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.8	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 *continued*: Notification rates of diseases, 1 April to 30 June 2012, by state or territory. (Annualised rate per 100,000 population)

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Varicella zoster (chickenpox)	3.3	NN	59.0	5.2	23.7	7.1	12.7	12.3	11.8
Varicella zoster (shingles)	14.2	NN	66.0	1.4	104.3	50.1	18.2	48.4	28.8
Varicella zoster (unspecified)	31.7	NN	1.7	93.1	8.7	18.0	46.3	41.7	53.6
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.2	0.2	0.0	0.0	0.0	0.1
Barmah Forest virus infection	0.0	4.3	26.0	16.8	1.0	0.0	0.6	5.8	5.9
Dengue virus infection	5.5	3.6	17.4	7.4	3.4	2.4	6.2	24.3	7.3
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	0.0	0.9	1.7	2.2	0.0	0.0	1.4	1.5	1.2
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	5.5	11.9	107.7	48.3	14.2	2.4	7.6	34.7	21.4
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Leptospirosis	0.0	0.5	0.0	3.1	0.0	0.0	0.1	0.0	0.8
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.2	0.0	0.0	0.0	0.0	0.7	0.0	0.2
Q fever	0.0	1.2	1.7	3.9	0.7	0.0	0.2	0.2	1.3
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	0.0	1.4	0.0	1.2	2.2	1.6	1.6	2.4	1.5
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Meningococcal infection‡‡	0.0	1.3	5.2	1.0	3.1	1.6	0.6	1.0	1.2
Tuberculosis	5.5	3.8	12.2	3.1	4.1	1.6	5.3	6.0	4.3

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

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

‡ In Queensland, includes incident hepatitis C cases.

§ Not notifiable in New South Wales.

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

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

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

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

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

NEC Not elsewhere classified.

NN Not notifiable.

NDP No data provided.

Additional reports

Australian childhood immunisation coverage

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

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months, for 3-month birth cohorts of children at the stated ages between January and March 2012. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, pneumococcal conjugate, varicella, and meningococcal C conjugate vaccines, and is outlined in more detail below.

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

A full description of the basic methodology used can be found in CDI 1998;22(3):36-37.

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

The percentage of children 'fully immunised' by 12 months of age for Australia increased marginally from the previous quarter by 0.5 of a percentage point to 91.9% (Table 1). Important changes in coverage were seen only in the Northern Territory with coverage for DTP, polio, Hib vaccine and hepatitis B vaccine (Hep B) increasing by almost 2 percentage points. Coverage for DTP-containing vaccine, polio and 'fully immunised' for the Northern Territory are at their highest recorded levels for this age group.

The percentage of children 'fully immunised' by 24 months of age for Australia decreased marginally from the previous quarter by 0.4 of a percentage point to 92.3% (Table 2). Coverage for DTP-containing vaccine, polio, Hib vaccine and 'fully immunised' for the Northern Territory are at their highest recorded levels for this age group.

The percentage of children 'fully immunised' by 60 months of age for Australia increased from the previous quarter by 0.4 of a percentage point to 90.1% (Table 3). This continues the upward trend in coverage for this age milestone. Important changes in coverage were seen only in South Australia with coverage for DTP-containing vaccine, polio, and MMR vaccine increasing by almost 2 percentage points.

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

Table 1. Percentage of children immunised at 12 months of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2011; assessment date 30 June 2012

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,367	24,468	954	15,791	4,845	1,597	17,870	8,263	75,155
Diphtheria, tetanus, pertussis (%)	93.6	92.1	94.3	92.0	92.9	93.4	93.3	90.7	92.4
Poliomyelitis (%)	93.6	92.1	94.3	92.0	92.9	93.3	93.2	90.7	92.3
Haemophilus influenzae type b (%)	93.7	92.0	94.4	91.9	92.7	93.2	93.1	90.6	92.2
Hepatitis B (%)	93.2	91.9	94.2	91.8	92.7	93.2	92.8	90.3	92.0
Fully immunised (%)	93.1	91.7	94.2	91.7	92.6	93.1	92.7	90.1	91.9
Change in fully immunised since last quarter (%)	+0.5	+0.6	+1.9	+0.3	+0.7	+0.2	+0.7	+0.1	+0.5

Table 2. Percentage of children immunised at 24 months of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2010; assessment date 30 June 2012*

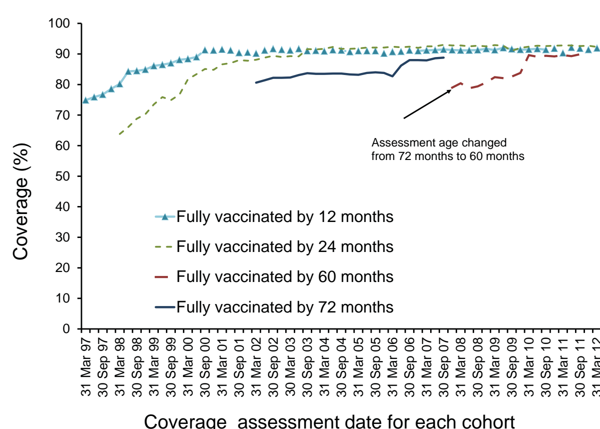
Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,339	24,421	922	16,109	4,955	1,525	18,235	8,231	75,737
Diphtheria, tetanus, pertussis (%)	95.1	94.5	97.3	94.3	94.1	95.1	95.1	93.0	94.5
Poliomyelitis (%)	95.0	94.4	97.3	94.3	94.1	95.0	95.0	92.9	94.4
Haemophilus influenzae type b (%)	95.2	94.9	97.3	94.4	94.4	95.7	95.2	93.4	94.7
Measles, mumps, rubella (%)	94.3	93.7	96.2	93.9	93.7	95.0	94.4	92.4	93.9
Hepatitis B (%)	94.3	94.0	97.2	93.9	93.8	94.9	94.7	92.4	94.0
Fully immunised (%)	92.8	92.1	95.7	92.6	92.2	93.6	93.0	90.1	92.3
Change in fully immunised since last quarter (%)	-0.8	-0.3	+1.3	-0.6	-0.3	-0.1	-0.4	-0.6	-0.4

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2011;35(1):49.

Table 3. Percentage of children immunised at 60 months of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2007; assessment date 30 June 2012

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,213	24,652	903	16,167	5,087	1,623	18,448	8,237	76,330
Diphtheria, tetanus, pertussis (%)	92.1	91.1	90.5	91.5	89.4	91.1	92.1	88.0	91.0
Poliomyelitis (%)	91.8	91.1	90.6	91.4	89.4	91.1	92.0	88.0	90.9
Measles, mumps, rubella (%)	91.2	91.0	90.4	91.4	89.0	91.3	92.0	88.1	90.9
Fully immunised (%)	90.9	90.6	90.4	91.0	88.8	90.8	91.6	87.6	90.5
Change in fully immunised since last quarter (%)	-0.3	+0.4	-0.4	+0.4	+1.9	-0.2	+0.2	+0.4	+0.4

Figure: Trends in vaccination coverage, Australia, 1997 to 31 March 2012, by age cohorts



over time for children aged 12 months, 24 months and 60 months (from December 2007). Coverage at 60 months of age is close to the coverage levels attained at 12 and 24 months.

Australian Sentinel Practices Research Network

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is funded by the Australian Government 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 influenza-like illness (ILI) testing was implemented, allowing for viral

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

The list of conditions is reviewed annually by the ASPREN management committee. In 2011, 4 conditions are being monitored. They include ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, *published in* Commun Dis Intell *2012;36(1):122.*

Reporting period 1 April to 30 June 2012

Sentinel practices contributing to ASPREN were located in all 8 jurisdictions in Australia. A total of 166 general practitioners contributed data to ASPREN in the 2nd quarter of 2012. Each week an average of 141 general practitioners provided information to ASPREN at an average of 13,081 (range 10,926–14,671) consultations per week and an average of 192 (range 92–312) notifications per week.

ILI rates reported from 1 April to 30 June 2012 averaged 9 cases per 1,000 consultations (range 3–19 cases per 1,000 consultations). This was higher compared with rates in the same reporting period in 2011, which averaged 7 cases per 1,000 consultations (range 4–13 cases per 1000 consultations) (Figure 1).

ILI swab testing continued during 2012. The most commonly reported virus during this reporting period was rhinovirus (15% of all swabs collected), with the second most common virus being influenza A (untyped) (14% of all swabs).

From the beginning of 2012 to the end of week 26, 42 cases of influenza had been detected, the majority of these being influenza A (untyped) (14% of all swabs), influenza B (7% of all swabs) and the remainder influenza A(H1N1)2009 (0.2% of all swabs) (Figure 2).

Figure 1: Consultation rates for influenza-like illness, ASPREN, 2011 and 2012, by year and week of report

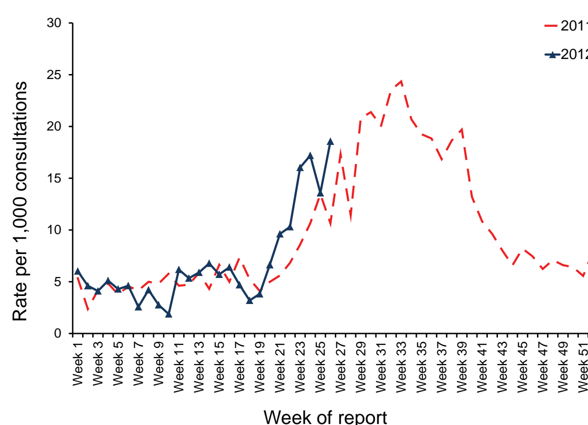
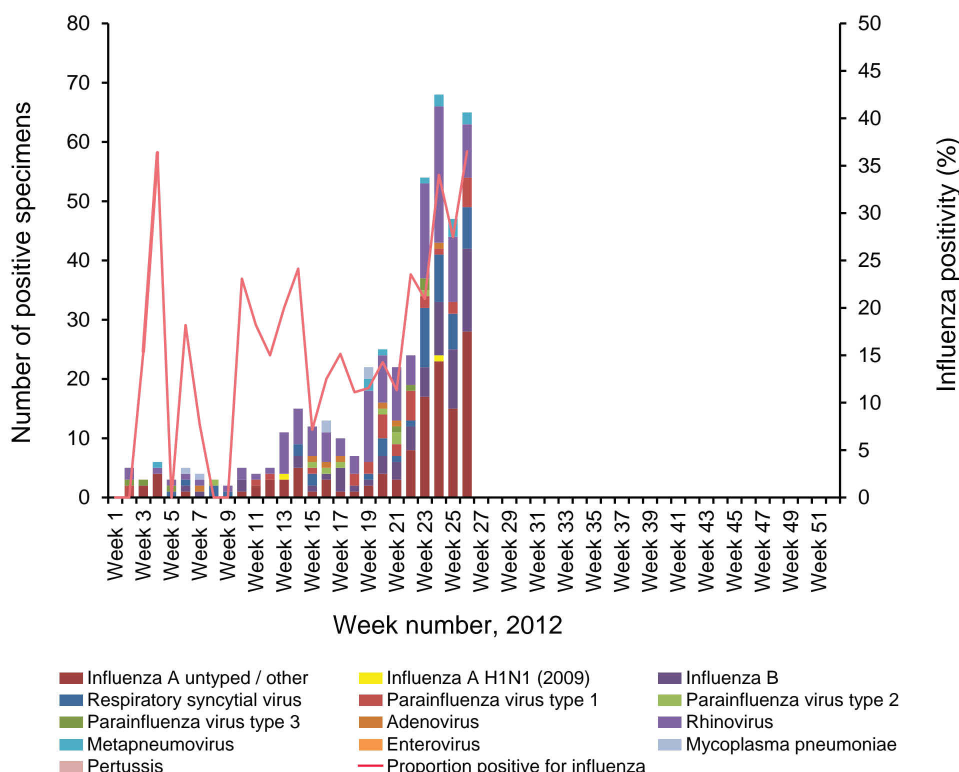


Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January to 30 June 2012, by week of report



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

Varicella infections were reported at a slightly lower rate for the second quarter of 2012 compared with the same period in 2011. From 1 April to 30 June 2012, recorded rates for chickenpox averaged 0.15 cases per 1,000 consultations (range 0–0.48 cases per 1,000 consultations, Figure 4).

In the 2nd quarter of 2012, reported rates for shingles averaged 0.8 cases per 1,000 consultations (range 0.56–1.23 cases per 1,000 consultations, Figure 5), slightly higher compared with the same reporting period in 2011, where the average shingles rate was 0.6 cases per 1,000 consultations (range 0.19–0.96 cases per 1,000 consultations).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 2011 and 2012, by year and week of report

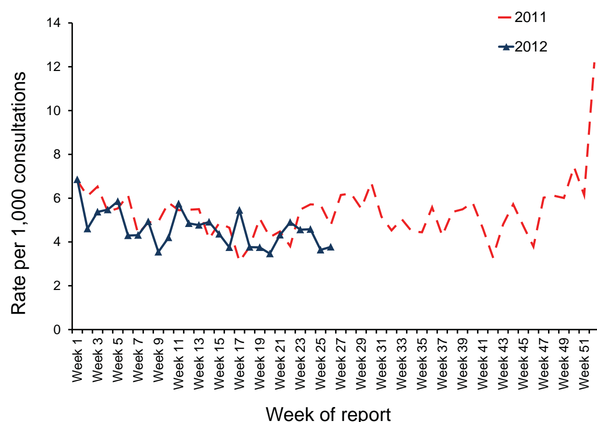


Figure 4: Consultation rates for chickenpox, ASPREN, 2011 and 2012, by year and week of report

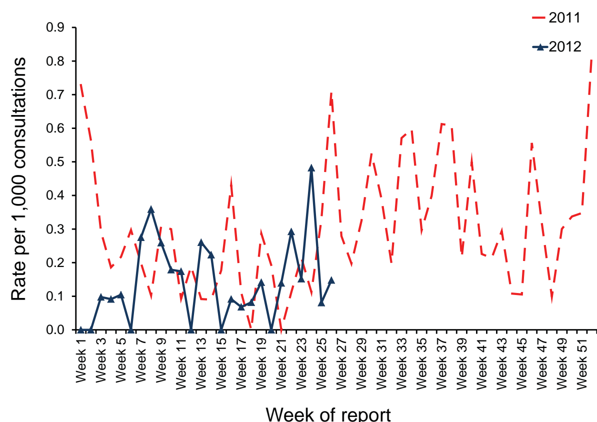
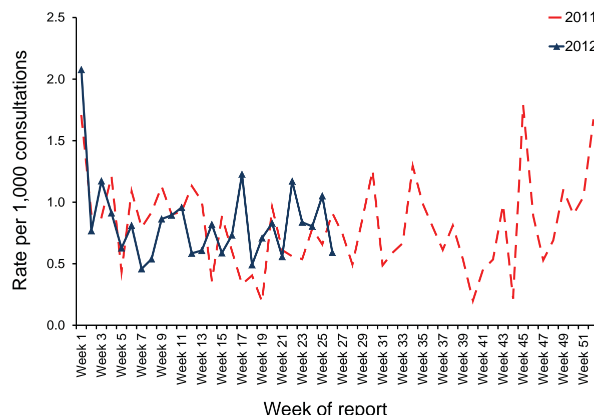


Figure 5: Consultation rates for shingles, ASPREN, 2011 and 2012, by year and week of report



Gonococcal surveillance

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

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the states and territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, which are current or potential agents used for the treatment of gonorrhoea. Azithromycin testing is now performed by all states and territories as it has a role as part of a dual therapy regimen in the treatment of gonorrhoea. When in vitro *resistance to a recommended agent is demonstrated in 5% or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatments.*[1] *Additional data are also provided on other antibiotics from time to time. 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. These data are reported in the AGSP Annual Report. 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. From the 2nd quarter of 2012 these data will be presented quarterly in tabulated form (Table 1), as well as in the AGSP annual report. Data for the 1st quarter of 2012 has been included in this report (Table 2) to complete presentation of the AGSP quarterly data in this format for 2012. For more information see* Commun Dis Intell *2012;36(1):121.*

Reporting period 1 April to 30 June 2012

Penicillin resistant *Neisseria gonorrhoeae* are defined as those isolates with an MIC to penicillin equal to or greater than 1.0 mg/L. Total penicillin resistance includes penicillinase producing *Neisseria gonorrhoea* (PPNG) and chromosomally mediated resistance to penicillin (CMRP).

Quinolone resistant *N. gonorrhoeae* are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L, and azithromycin resistance as those isolates with an MIC to azithromycin equal to or greater than 1.0 mg/L. In the Northern Territory there continues to be low levels of penicillin and ciprofloxacin resistance.

Isolates with ceftriaxone MIC values in the range 0.06–0.125 mg/L are reported as having decreased susceptibility. There has not been an isolate reported in Australia with an MIC >0.125 mg/L. The Figure presents AGSP data for the 1st and 2nd quarters for 2011 and 2012, by ceftriaxone MIC value for the first time to enable monitoring of shift in MIC values *N. gonorrhoeae* MIC values over time, in addition to reporting the proportion in the category of decreased susceptibility. A decrease in the proportion of isolates with a ceftriaxone MIC value of ≤ 0.008 mg/L is evident in 2012 compared with 2011, with increases in the higher MIC values demonstrating a right shift over these periods, which will continue to be monitored.

Reference

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

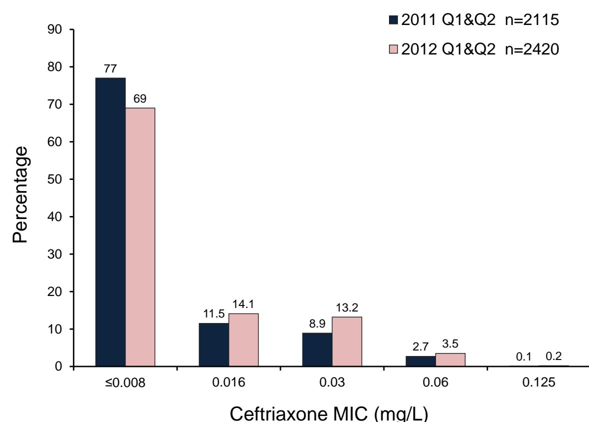
Table 1: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to ciprofloxacin, azithromycin and penicillin, Australia, 1 January to 31 March 2012, by state or territory

State or territory	Number of isolates tested	Decreased susceptibility Ceftriaxone		Resistance Ciprofloxacin		Resistance Azithromycin		Resistance Penicillin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	13	0	0.0	8	61.5	0	0.0	3	23.1
New South Wales	447	17	3.8	121	27.1	4	0.9	119	26.6
Northern Territory	77	0	0.0	1	1.3	1	1.3	1	1.3
Queensland	205	3	1.5	35	17.1	2	1.0	44	21.5
South Australia	27	0	0.0	8	29.6	8	29.6	12	44.4
Tasmania	1	1	100.0	1	100.0	0	0.0	0	0.0
Victoria	312	21	6.7	166	53.2	14	4.5	178	57.1
Western Australia	130	2	1.5	29	22.3	1	0.8	21	16.2
Australia	1,212	44	3.6	369	30.4	30	2.5	378	31.2

Table 2: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to ciprofloxacin, azithromycin and penicillin, Australia, 1 April to 30 June 2012, by state or territory

State or territory	Number of isolates tested	Decreased susceptibility Ceftriaxone		Resistance Ciprofloxacin		Resistance Azithromycin		Resistance Penicillin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	9	0	0.0	4	44.4	0	0.0	3	33.3
New South Wales	421	16	3.8	115	27.3	0	0.0	105	24.9
Northern Territory	82	0	0.0	0	0.0	0	0.0	1	1.2
Queensland	174	8	4.6	26	14.9	2	1.1	48	27.6
South Australia	44	0	0.0	7	15.9	0	0.0	7	15.9
Tasmania	4	0	0.0	1	25.0	0	0.0	3	75.0
Victoria	355	21	5.9	149	42.0	108	30.4	202	56.9
Western Australia	119	1	0.8	30	25.2	1	0.8	26	21.8
Australia	1,208	46	3.8	332	27.5	111	9.2	395	32.7

Figure: Distribution of ceftriaxone MIC values in gonococcal isolates tested at the Australian Gonococcal Surveillance Programme, 1 January 2011 to 30 June 2012



Meningococcal surveillance

Dr Monica M Lahra, 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 *2012;36(1):121.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 July to 30 September 2012 are included in this issue of Communicable Diseases Intelligence *(Table).*

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 July to 30 September 2012, by serogroup and state or territory

State or territory	Year	A Q3	A YTD	B Q3	B YTD	C Q3	C YTD	Y Q3	Y YTD	W135 Q3	W135 YTD	ND Q3	ND YTD	All Q3	All YTD
Australian Capital Territory	2012	0	0	0	1	0	0	0	0	0	0	0	0	0	1
	2011	0	0	0	5	0	0	0	0	0	0	0	0	0	5
New South Wales	2012	0	0	16	36	1	1	3	4	2	2	3	8	25	51
	2011	0	0	12	27	0	0	1	6	2	4	3	13	18	50
Northern Territory	2012	0	0	0	2	0	1	0	0	0	0	0	1	0	4
	2011	0	0	0	1	0	0	0	0	0	0	0	0	0	1
Queensland	2012	0	0	15	35	1	2	3	3	2	3	3	3	24	46
	2011	0	0	26	43	0	3	1	3	0	0	0	3	27	52
South Australia	2012	0	0	10	17	0	1	0	0	0	0	0	0	10	18
	2011	0	0	5	11	0	1	0	0	0	2	1	1	6	15
Tasmania	2012	0	0	2	3	0	0	1	1	0	0	0	1	3	5
	2011	0	0	4	6	0	1	0	0	1	3	0	0	5	10
Victoria	2012	0	0	8	21	0	0	2	4	0	0	0	0	10	25
	2011	0	0	11	35	0	0	1	1	1	1	0	3	13	40
Western Australia	2012	0	0	4	11	1	2	0	1	0	0	0	1	5	15
	2011	0	0	4	12	0	0	0	1	0	0	0	0	4	13
Total	2012	0	0	55	126	3	7	9	13	4	5	6	14	77	165
	2011	0	0	62	140	0	5	3	11	4	10	4	20	73	186

Announcements

Communicable Disease Control conference 2013

Emerging infections – setting the agenda for Australia

Abstract submissions now open

The Communicable Disease Control Conference Committee invites you to submit an abstract for the 2013 Conference taking place at the Hyatt Hotel Canberra on 19–20 March 2013 (Monday to Tuesday).

In 2013 the Communicable Disease Control Conference (CDC) will run back-to-back with the Australasian Society for Infectious Diseases (ASID) Annual Scientific Meeting being held on 20–23 March 2013 (Wednesday to Saturday).

This provides useful opportunities for delegates to increase their knowledge and understanding about issues relating to infectious and communicable disease control and prevention in Australia. Wednesday will see both conferences combined to offer international keynote speakers, from both CDC and ASID.

Abstract submission deadline: 25 January 2013

The call for abstracts is now open with a deadline of Friday 25 January 2013. Please ensure you refer to the abstract guidelines outlined on the conference website before you submit.

Step-by-step instructions are incorporated into the online abstract submission form. If you require assistance, or have any concerns about the abstract submission process, please contact the conference organisers at cdcconference@ashm.org.au

Please click the NEW ACCOUNT link at the bottom of the login page.

Submit an abstract

CDC Organising Committee contacts are:

Locked Mail Bag 5057
Darlinghurst NSW 1300
Phone: +61 2 8204 0770
Fax: +61 2 9212 4670
Email: cdcconference@ashm.org.au

Website: www.CDCconference.com.au

Conference deadlines

Abstract submission:

Friday 25 January 2013

Earlybird registration:

Friday 25 January 2013

Accommodation deadline:

Monday 18 February 2013

Standard registration:

Tuesday 12 March 2013