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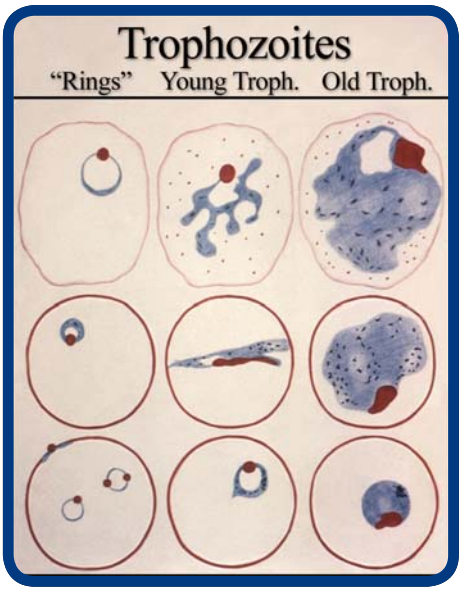
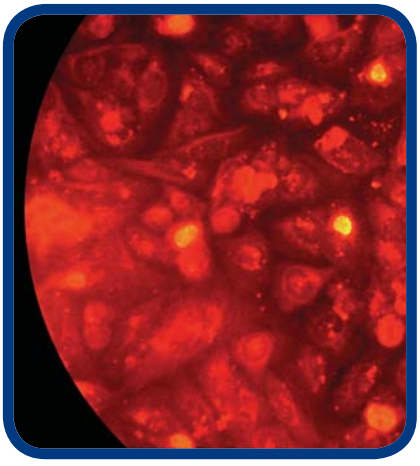
Department of Health and Ageing

Communicable Diseases Intelligence





Quarterly report

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Front cover: Surveillance Section, Australian Government Department of Health and Ageing.
Images sourced from the Centers for Disease Control and Prevention Public Health Image Library, courtesy of the Centers for Disease Control and Prevention, Atlanta, Georgia.
Clockwise from top left: Various forms that the developing malarial parasite undergoes prior to its schizont stage, Stephen Glenn, CDC; Female *Aedes aegypti* mosquito, James Gathany; Administration of hepatitis B vaccine, Jim Gathany; photomicrograph depicts HeLa cells infected with Type-A *Chlamydia trachomatis, Joe Miller, CDC*.

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Communicable Diseases Network Australia National Arbovirus and Malaria Advisory Committee annual report, 2005–06

Conan Liu,[1] Cheryl Johansen,[2] Nina Kurucz,[3] Peter Whelan[3]

Abstract

This report describes the epidemiology of mosquito-borne disease in Australia for the mosquito-borne disease season 1 July 2005 to 30 June 2006, in which the second largest number of notifications since 1995–96 was reported. Ross River virus (RRV) infections (66%), Barmah Forest virus (BFV) infections (23%) and malaria (9%) were the most common mosquito-borne diseases reported in 2005–06. National RRV notifications were the fifth largest on record. The Northern Territory had the highest rate of RRV notifications and the peak notification rate (in January 2006) was the third highest since 2000. National BFV notification rates were the highest on record. The Northern Territory also reported the highest BFV notification rate this season, peaking in February–March 2006, which was the highest reported BFV notification rate on record. BFV notification rates were significantly higher in teenagers compared to previous seasons. There were 731 notifications of malaria in 2005–06 of which none was reported as locally acquired. This was the third highest reporting period for malaria notifications since 2000. In contrast to previous years in which *Plasmodium vivax* was the predominant species, *Plasmodium falciparum* was reported as the infecting species in 45 per cent of the malaria notifications and *Plasmodium vivax* for 42 per cent of cases. Young adults in the 20–24 year age group had the highest number of cases and children in the 5–9 year age group accounted for 22 per cent of notifications. There were two cases of Kunjin virus (KUNV) infection and one case of Murray Valley encephalitis virus (MVEV) infection reported in 2005–06, all from Western Australia. Sentinel chicken surveillance data for flaviviruses and sentinel pig surveillance data for Japanese encephalitis virus are reported. There were 200 notifications of dengue virus (DENV) infection in 2005–06, of which 46 per cent (n=92) was reported as having been acquired overseas. Dengue serotypes 2 and 3 were detected in two outbreaks of locally-acquired dengue in Queensland this season. *Commun Dis Intell* 2006;30:411–429.

Keywords: arbovirus; Barmah Forest virus, dengue, disease surveillance; epidemiology, flavivirus, Japanese encephalitis, Kunjin, malaria, mosquitoes, Murray Valley encephalitis virus, Ross River virus

Introduction

This report describes the epidemiology of nationally notifiable mosquito-borne disease in Australia for the season 1 July 2005 to 30 June 2006, which was the second largest season since 1995–96.

The eight notifiable mosquito-borne diseases under national surveillance include the alphaviruses (Barmah Forest virus and Ross River virus), the flaviviruses (dengue, Japanese encephalitis, Kunjin, Murray Valley encephalitis and flavivirus not elsewhere classified), and malaria.

Alphaviruses are ribonucleic acid (RNA) viruses which cause disease epidemics characterised by fever, rash and polyarthritis. In Australia, Barmah Forest virus (BFV) infection and Ross River virus (RRV) infection are the alphaviruses of major public health significance. There is a variety of mosquito vectors which facilitate the transmission of these viruses in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[1,2] At this time, the alphavirus chikungunya virus is not notifiable and has not become established in Australia despite its

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increased activity in southern Asia and the Indian Ocean over the past year, and its occasional diagnosis in returning travellers.

Flaviviruses are single-stranded RNA viruses, some of which are associated with epidemic encephalitis in various regions of the world. In Australia, the flaviviruses of public health importance are the dengue viruses (DENV) with frequent seasonal outbreaks,[3–6] Japanese encephalitis virus (JEV) with occasional outbreaks,[7–12] and sporadic cases of infection with Murray Valley encephalitis virus (MVEV) or Kunjin virus (KUNV).[13] The International Committee for Taxonomy of Viruses refers to Kunjin as a strain of West Nile virus (WNV)[14] and the Australian Kunjin strains are phylogenetically located in the WNV lineage 1, clade B.[15]

Malaria is caused by infection with a protozoan blood parasite from the genus *Plasmodium* that has been transmitted by a species of mosquito from the genus *Anopheles*. Malaria was eradicated from Australia in 1981 and Australia was certified malaria-free by the World Health Organization (WHO) in 1983,[16] but the region of northern Australia above 19°S latitude in particular, remains receptive to malaria transmission. Since 1981, malaria acquired in Australia has been rare, but there have been several documented reports of outbreaks[17,18] and sporadic introduced cases[19,20] in Queensland, malaria acquired in the Torres Strait,[21] and the artificial induction of malaria by blood transfusion.[22]

Methods

Eight nationally notifiable mosquito-borne diseases were analysed for the seasonal period 1 July 2005 to 30 June 2006. Historical data from 2000, and in some cases from 1991, are also shown for comparison. Data were extracted by diagnosis date from the National Notifiable Diseases Surveillance System (NNDSS) on 18 August 2006. Hospitalisation data where available, were extracted from the online National Hospital Morbidity database, Australian Institute of Health and Welfare.[23]

Epidemic curves by state or territory were produced for each of the eight diseases. Notifiable mosquito-borne disease activity is shown compared with a five-year mean for the same period by jurisdiction. The number of notifications and annual or annualised notification rates for locally acquired mosquito-borne disease were calculated using the December population estimates from the Australian Bureau of Statistics (ABS). Age- and sex-specific notification rates were calculated using age and sex population estimates for each jurisdiction.

The geographical distribution of selected diseases was mapped using ArcGIS (ESRI, Redlands, CA, USA). Maps were based on the postcode of residence of each notification aggregated to the appropriate Statistical Division, and rates were calculated using the number of notifications (numerator) divided by the estimated 2005 ABS populations for each division (denominator).

The timeliness of reporting notifiable mosquito-borne diseases was calculated by quantifying the time lag from onset of disease to the public health unit notification received date. Where onset date was not supplied, the earliest date of specimen date, or notification date was used.

Sentinel chicken surveillance data for flaviviruses and sentinel pig surveillance data for Japanese encephalitis virus are reported.

Results

Alphaviruses

During this reporting period, there were 8,387 notifications of mosquito-borne disease (MBD) reported in Australia, which was twice the number of notifications reported for the last season. Overall, total MBD notifications during 2005–06 were the second highest on record and associated with increases in BFV and RRV notifications. The highest season on record was observed in 1995–96 with approximately 9,210 notifications of MBD reported.

RRV infections accounted for 66 per cent (n=5,515) of notifications with an onset in 2005–06, along with BFV infections (23%, n=1,895) and malaria (9%, n=731) (Figure 1).

Figure 1. Notifications of select mosquito-borne diseases, Australia, 1 July 2000 to 30 June 2006, by season of onset

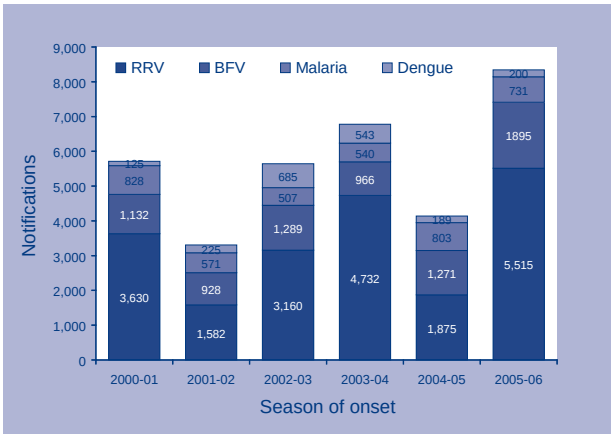
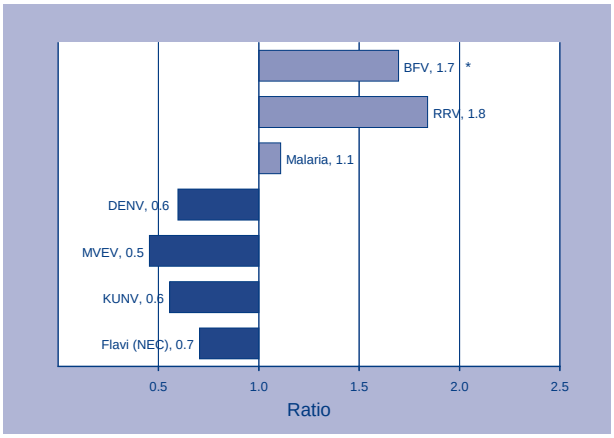


Figure 2 shows the ratio of BFV, RRV and malaria notifications for 2005–06 compared with the five-year mean. BFV notifications during 2005–06 exceeded two standard deviations above the five-year mean. Contributing factors to unusually high BFV notifications include an increase in vector numbers during spring, more active surveillance and clinical testing for BFV in some jurisdictions and the dual reporting of BFV and RRV notifications by some jurisdictions. An analysis of this is contained in the dual reporting of BFV and RRV section.

Figure 2. Ratio of 2005–06 notifiable mosquito-borne disease activity to mean of previous five years

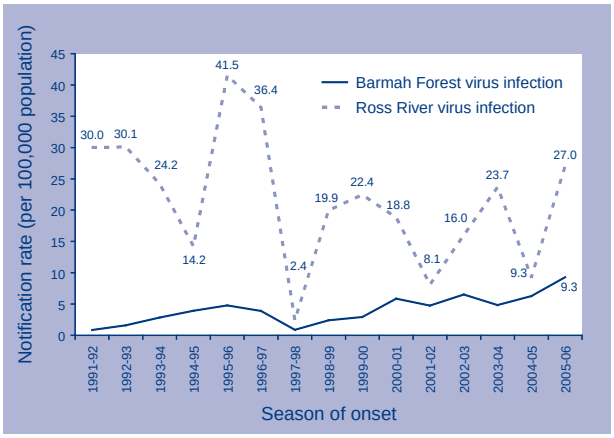


* Above 2 standard deviations.

No Japanese encephalitis virus infection cases in 2005-06.

The national notification rate for BFV in 2005–06 was 9.3 cases per 100,000 population (Figure 3) which was the highest notification rate for BFV since the commencement of the nationalised reporting of BFV in 1991. The national notification rate for RRV was 27 cases per 100,000 population, the highest reported notification rate for RRV since 1996–97.

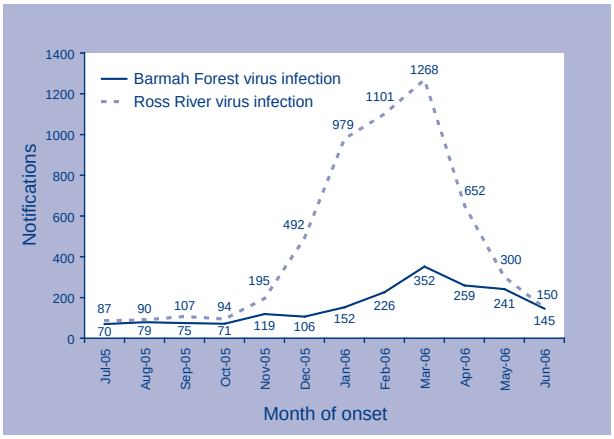
Figure 3. Crude annual rate of Barmah Forest virus and Ross River virus infections notifications, Australia, 1 July 1991 to 30 June 2006, by season of onset



These high notification rates were associated with a wet spring in 2005 in inland and coastal areas which produced favourable conditions for mosquito breeding.

During the 2005–06 season, the highest number of notifications for BFV and RRV was received in March (Figure 4). BFV notifications peaked earlier than in 2005 (April–May). The peak of RRV notifications (n=1268) in March 2006 was more than two and a half times the peak in March 2005.

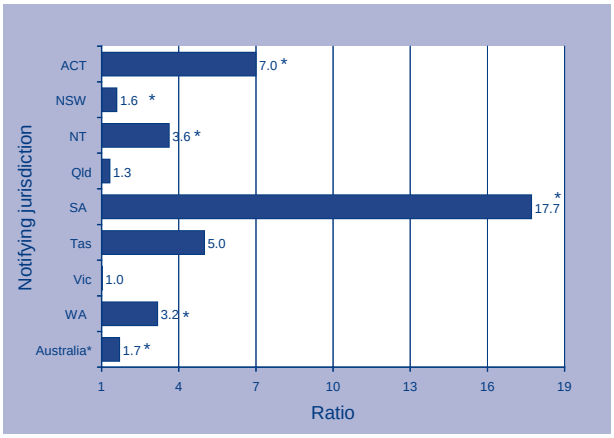
Figure 4. Barmah Forest virus infection and Ross River virus infections notifications, Australia, 1 July 2005 to 30 June 2006, by month of onset



Barmah Forest virus infections

During 2005–06, all jurisdictions except Victoria reported BFV activity above the five-year mean (Figure 5). Notifications of BFV from the Australian Capital Territory, New South Wales, the Northern

Figure 5. Ratio of Barmah Forest virus infection notifications to mean of previous five years, Australia, 1 July 2005 to 30 June 2006, by state or territory



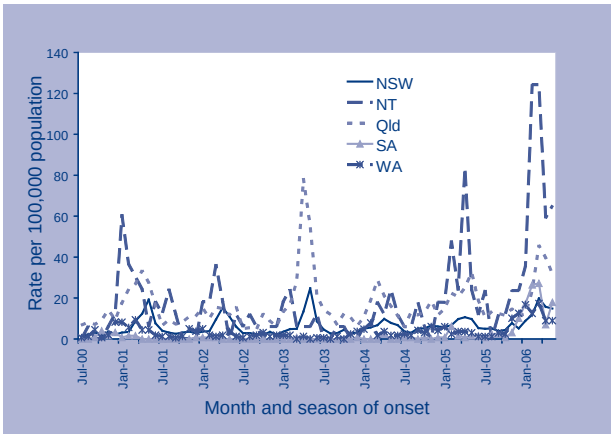
* Above 2 standard deviations.

Territory, South Australia and Western Australia, exceeded two standard deviations above the five-year mean. South Australia notified more than 17 times its five-year mean. This increase may be attributable to a change in surveillance practice from December 2005, when the Communicable Disease Control Branch, South Australia recommended that clinicians consider testing for both BFV and RRV if an arbovirus infection was suspected (Jane Raupach, personal communication).

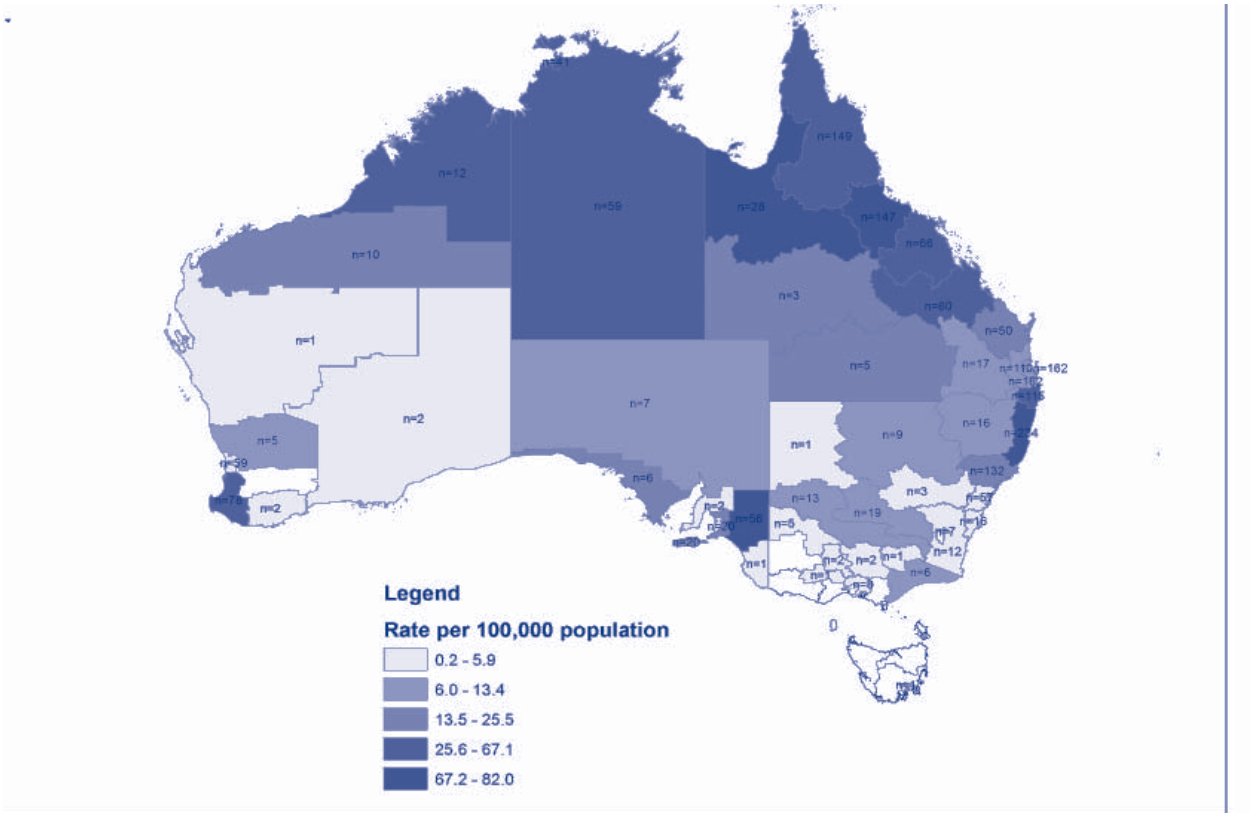
The highest rates of BFV notifications in Australia during 2005–06 were reported from Northwest Queensland (81.9 notifications per 100,000 population) and the Murray Lands in South Australia (81.5 notifications per 100,000 population) (Map 1). The majority of cases in the Murray Lands region occurred in the Coorong lower lakes area. The increase in BFV incidence for 2005–06 in this wetlands area was associated with the high abundance of *Aedes camptorhynchus* mosquitoes (Craig Williams, unpublished data). There is also evidence that elevated Culex australicus and Culex globocoxitus mosquito abundance in the Coorong lower lakes area during the three months preceding an outbreak is predictive of BFV activity, suggestive of the possible involvement of these vectors in an amplification cycle. Moderately high rates of BFV notifications were reported from Northern Queensland (71.5 notifications per 100,000 population) and from the Mid-North Coast area of New South Wales (79.3 notifications per 100,000 population). New South Wales reported the largest documented outbreak of BFV in Australia during this season.[24]

All states and territories reported peak notification rates of BFV in March 2006 (Figure 6). The Northern Territory notified the highest rate of BFV on record (124.3 cases per 100,000 population) during February and March 2006. Queensland reported

Figure 6. Annualised notification rate for Barmah Forest virus infections, select jurisdictions, 1 July 2000 to 30 June 2006, by state or territory



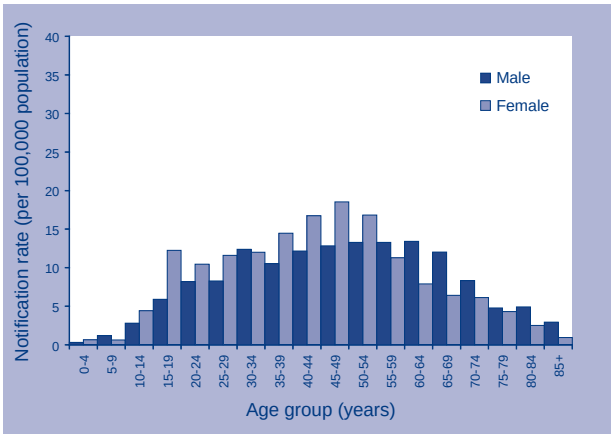
Map 1. Notifications and notification rates of Barmah Forest virus infection, Australia, 1 July 2005 to 30 June 06, by Statistical Division of residence



the second highest rate of BFV (45.7 cases per 100,000 population). South Australia also reported its highest ever BFV notification rate during March 2006 (27.2 cases per 100,000 population).

The national notification rate of BFV was highest in the 45–49 year age group (Figure 7). The highest notification rates were in females in the 45–49 year age group (18.5 cases per 100,000 population) and males in the 60–64 year age group (13.4 cases per 100,000 population). Notification rates were generally higher in females in the age range 15–54 years than males in the equivalent age groups.

Figure 7. Notification rate for Barmah Forest virus infections, Australia, 1 July 2005 to 30 June 2006, by age group and sex



In general, the age and sex distribution pattern for New South Wales (Figure 8) was similar to the pattern observed for the whole of Australia (Figure 7). Queensland (Figure 9) had higher age- and sex-specific notification rates than New South Wales, with males tending to have higher notification rates

Figure 8. Notification rate for Barmah Forest virus infections, New South Wales, 1 July 2005 to 30 June 2006, by age group and sex

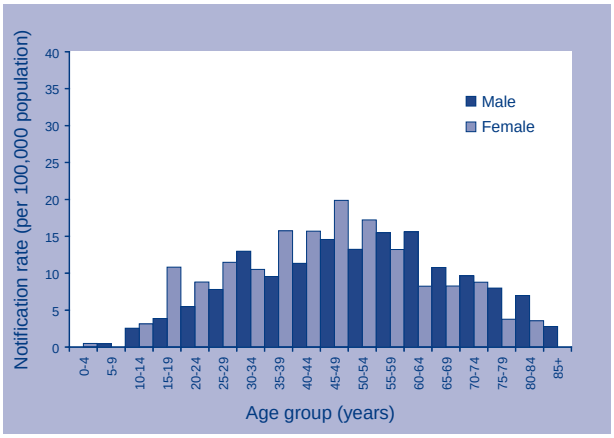
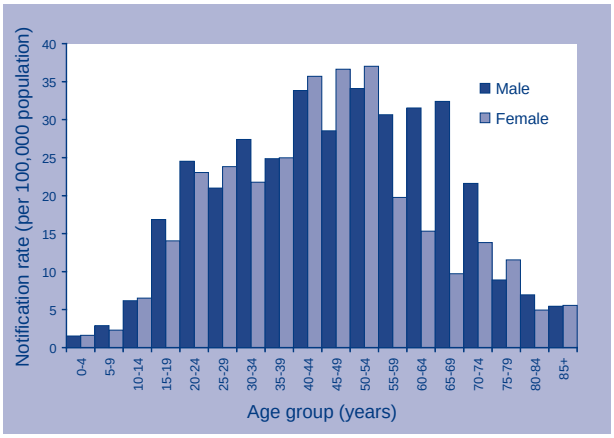
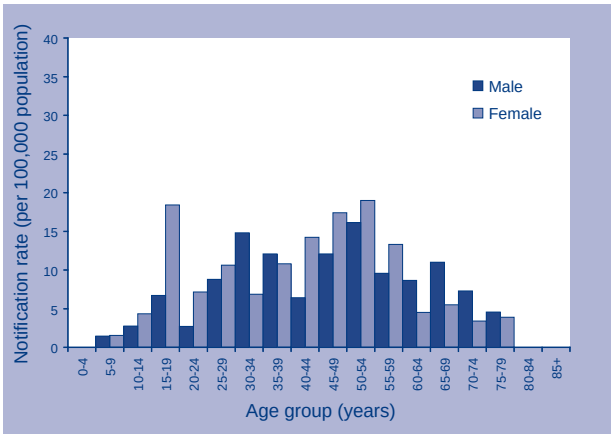


Figure 9. Notification rate for Barmah Forest virus infections, Queensland, 1 July 2005 to 30 June 2006, by age group and sex



than females in the 40–54 year age range. Western Australia (Figure 10) and the Northern Territory (Figure 11) displayed different age and sex distribution patterns of BFV notifications, with characteristically higher female-specific notification rates for many age groups. It is likely that small population sizes in age- and sex-specific age groups in these jurisdictions have affected the reporting rates (as small changes in the numerator lead to large changes in the overall rate).

Figure 10. Notification rate for Barmah Forest virus infections, Western Australia, 1 July 2005 to 30 June 2006, by age group and sex



In Western Australia, notifications in females in the 50–54 year age group (n=13) had the highest BFV age- and sex-specific notification rate (Figure 10). The Northern Territory reported high female notification rates in the 35–39 year (n=10, 123.6 cases per 100,000 population), the 45–49 year (n=9, 131.9 cases per 100,000 population), and the 60–74 year age groups (Figure 11). The highest age-specific BFV notification rate in the Northern

Figure 11. Notification rate for Barmah Forest virus infections, Northern Territory, 1 July 2005 to 30 June 2006, by age group and sex

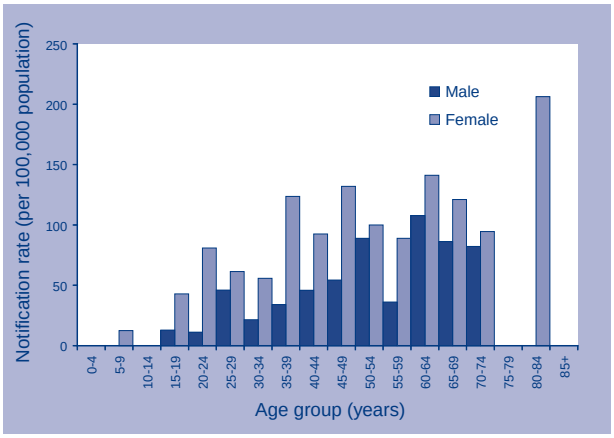
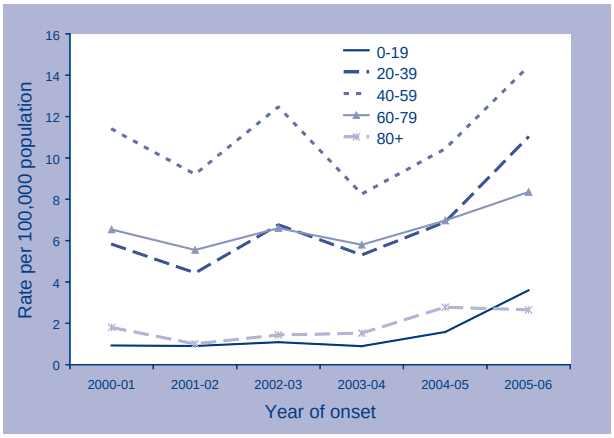


Figure 12. Trends in Barmah Forest virus infection notification rates, Australia, 1 July 2005 to 30 June 2006, by age group



Territory was observed in the 60–64 year age group (n=4 males, 4 females, 122.2 cases per 100,000 population). An extremely high female notification rate (206.2 cases per 100,000 population) in the 80–84 year age group was attributable to one notification reported in a relatively small age- and sex-specific population size (485 persons).

The age-specific rate of BFV notifications in 2005–06 changed significantly with a shift toward higher rates in children, teenagers and young adults when compared to the previous five seasons (Figure 12). The age-specific rate of BFV infection in the 0–19 year age range (3.6 notifications per 100,000 population) was over three times the five-year average (1.1 notifications per 100,000 population).

Timeliness of Barmah Forest virus infection notifications

The timeliness of BFV notifications is shown in Figure 13. In 2005–06, it took 8.5 days for 10 per cent of BFV notifications (n=1,238) and 106 days for 90 per cent of BFV notifications (n=1,886) from disease onset to be notified to public health units. The fastest notification times for the 10–50 percentiles were observed in 2002–03 and 2003–04.

Figure 13. Timeliness of Barmah Forest virus infection notifications, Australia, 1 July 2000 to 30 June 2006, by percentile

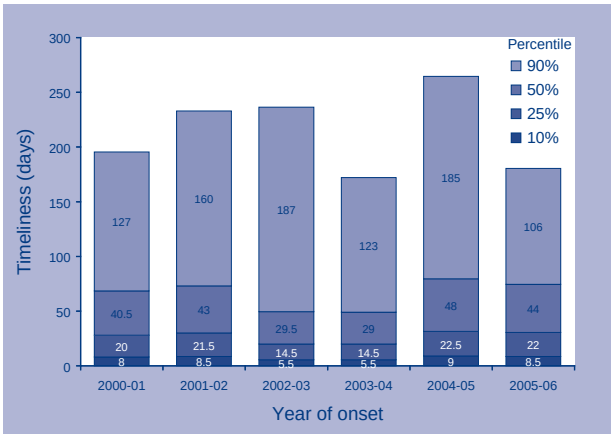
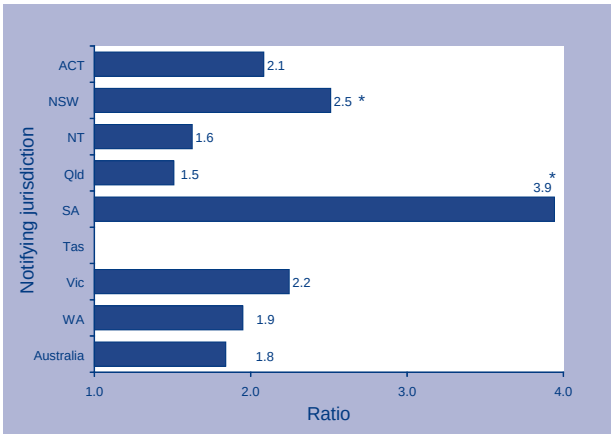


Figure 14. Ratio of Ross River virus infection notifications to mean of previous five years, Australia, 1 July 2005 to 30 June 2006, by state or territory



* Above 2 standard deviations.

Ross River virus infections

Historically, peaks in RRV notifications occur every four years (Figure 3). During 2005–06, all jurisdictions except Tasmania reported RRV activity above the five-year mean for their jurisdiction (Figure 14). Notifications of RRV from New South Wales and South Australia exceeded two standard deviations from the five-year mean for these jurisdictions. The peak notification rates for the Northern Territory (455.6 cases per 100,000 population) and South Australia (89.5 cases per 100,000 population) were

reported in January 2006 (Figure 15). Queensland (45.7 cases per 100,000 population), South Australia (27.2 cases per 100,000 population), New South Wales (19.8 cases per 100,000 population) and Western Australia (17.3 cases per 100,000 population) reported peak notification rates in March 2006.

The highest rates of RRV notifications in Australia during 2005–06 were reported from the Kimberley region of Western Australia (218.2 notifications per 100,000 population), the Eyre region (193.3 notifications per 100,000 population) and the Murray Lands region (157.1 notifications per 100,000 population) of South Australia (Map 2). Moderately high rates of RRV notification were reported throughout northern Australia, and from the Southwest region of Western Australia.

The rate of national notifications for RRV was highest in the 35–39 year age group (Figure 16). Females in the 45–49 year age group (52.3 cases per 100,000

Figure 15. Annualised notification rates for Ross River virus infection, select jurisdictions, 1 July 2000 to 30 June 2006, by state or territory

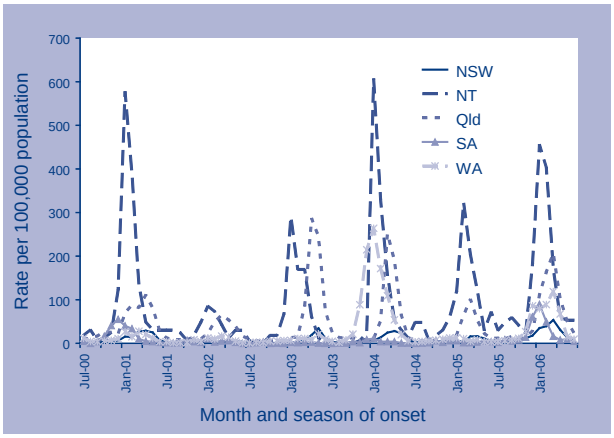
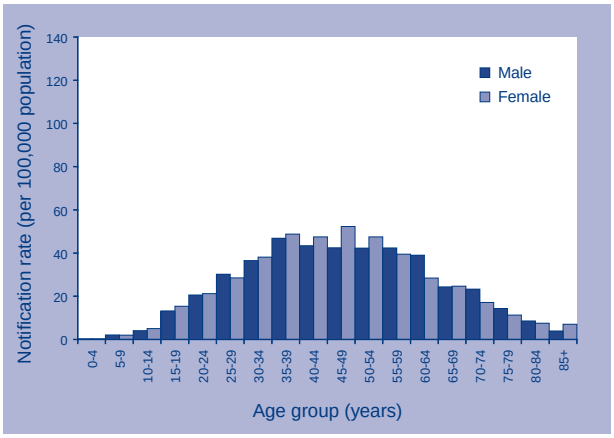
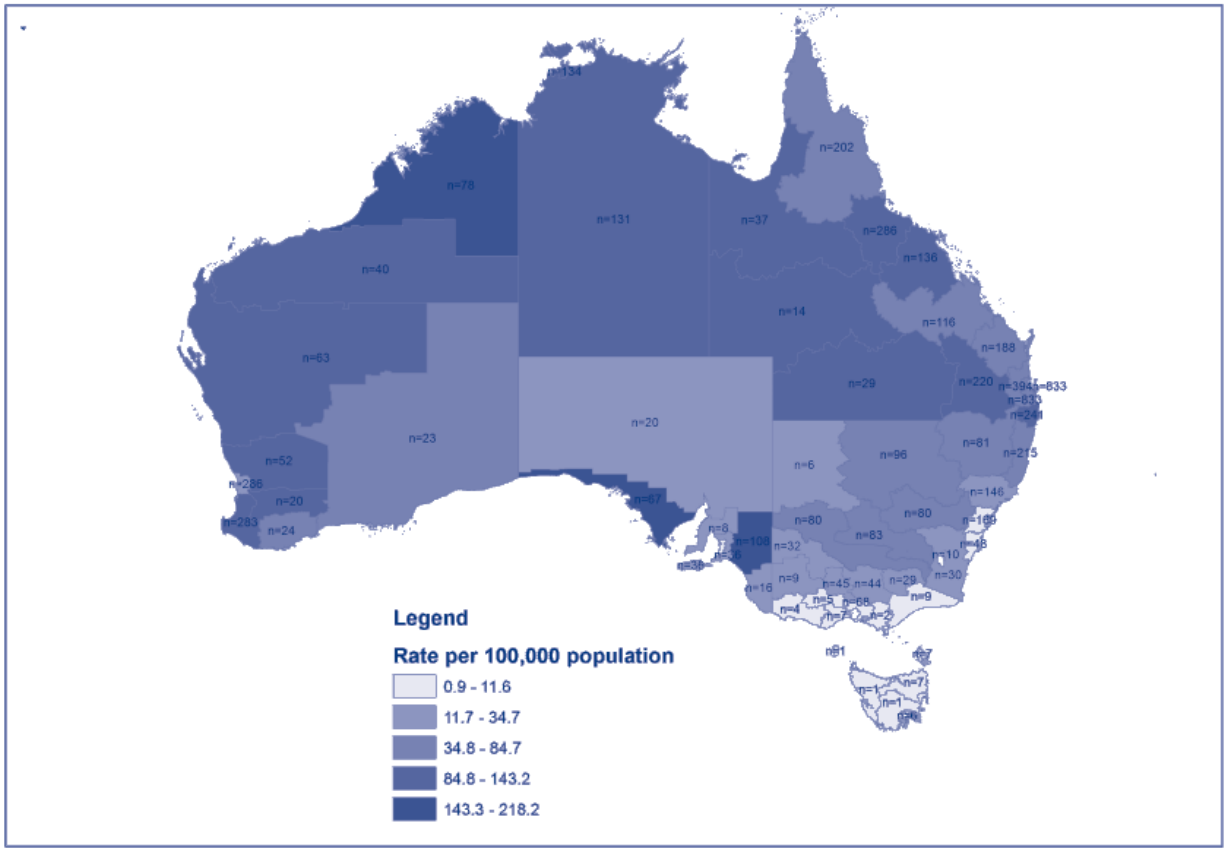


Figure 16. Notification rate for Ross River virus infections, Australia, 1 July 2005 to 30 June 2006, by age group and sex



Map 2. Notifications and notification rates of Ross River virus infections, Australia, 2005–06, by Statistical Division of residence



population) and males in 35–39 year age group (46.9 cases per 100,000 population) had the highest national age- and sex-specific notification rates.

In general, New South Wales (Figure 17) and Western Australia (Figure 18) showed similar age and sex distribution patterns to Australia. Age- and sex-specific notification rates of RRV from Western Australia were approximately twice the RRV notification rates from New South Wales.

Figure 17. Notification rate for Ross River virus infections, New South Wales, 1 July 2005 to 30 June 2006, by age group and sex

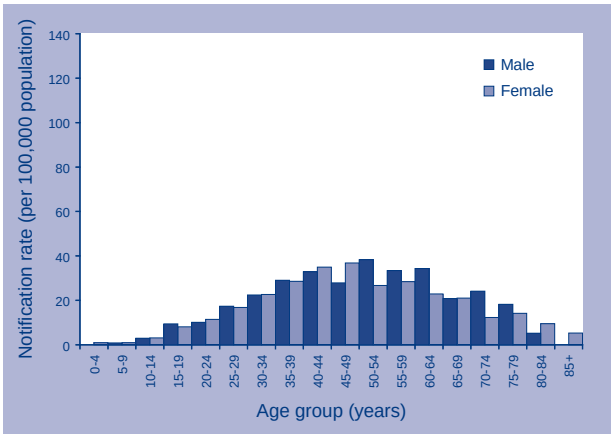
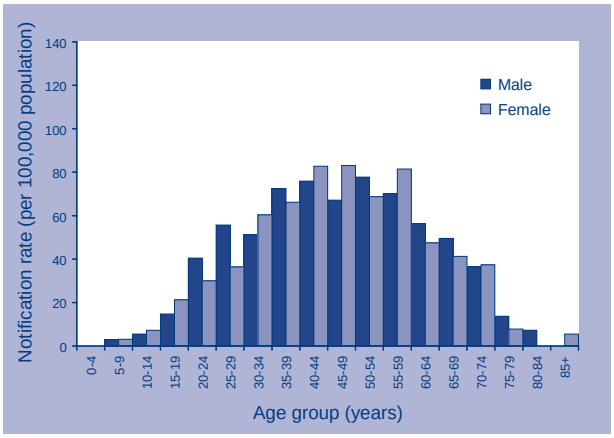
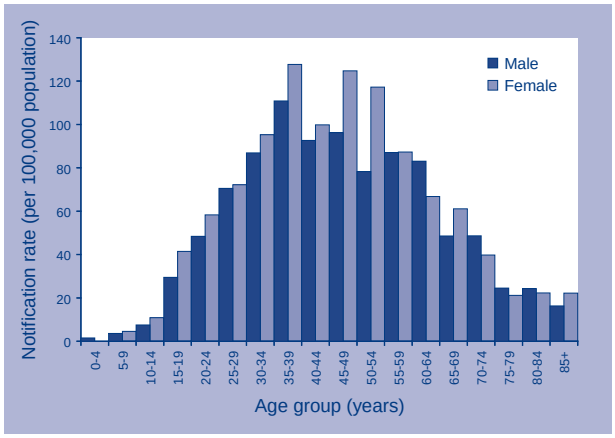


Figure 18. Notification rate for Ross River virus infections, Western Australia, 1 July 2005 to 30 June 2006, by age group and sex



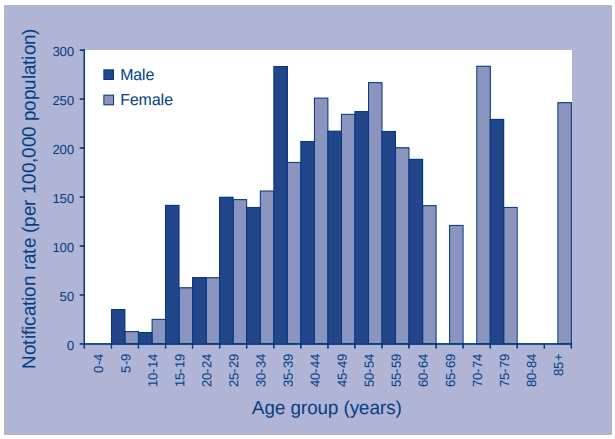
Notifications of RRV from Queensland (Figure 19) were highest in the 35–39 year age group (119.4 cases per 100,000 population). Notifications and notification rates in female-specific age groups were noticeably higher than male cohorts in Queensland for the 35–39 year age group (n=184, 127.7 cases per 100,000 population), 45–49 year age group (n=187, 124.7 cases per 100,000 population) and 50–54 year age group (n=152, 117.2 cases per 100,000 population).

Figure 19. Notification rate for Ross River virus infections, Queensland, 1 July 2005 to 30 June 2006, by age group and sex



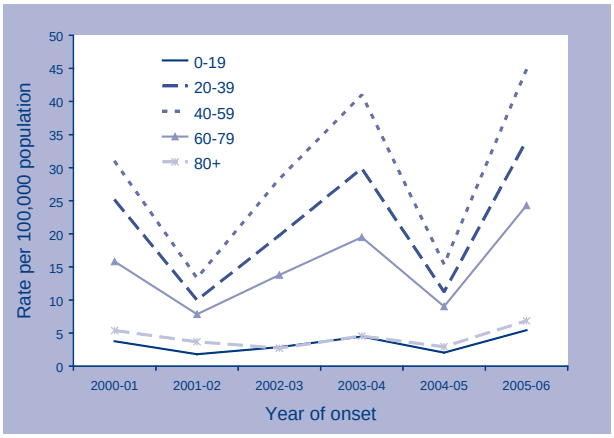
The Northern Territory overall had very high rates of RRV notifications, with very high age- and sex-specific peaks, some of which are due to small increases in the number of cases leading to large changes in the overall rate in this small population (Figure 20). Males in the 15–19 year age group had extremely high RRV notifications and notification rates (n=11, 141.5 cases per 100,000 population) when compared to either younger males or similarly aged females. Another high age specific peak was observed in the male 35–39 year age group (n=25, 283.3 cases per 100,000 population).

Figure 20. Notification rate for Ross River virus infections, Northern Territory, 1 July 2005 to 30 June 2006, by age group and sex



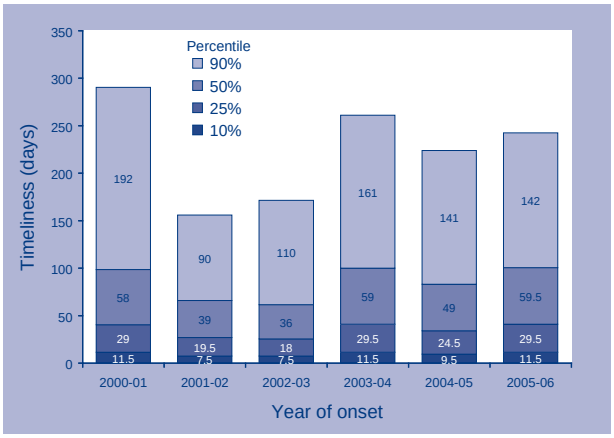
The age-specific rates for RRV notifications observed during this season were the highest on record since 2000. The overall age groups of the susceptible populations have not changed since 2000 (Figure 21).

Figure 21. Trends in Ross River virus infections notification rates, Australia, 1 July 2005 to 30 June 2006, by age group



The timeliness of RRV notifications is shown in (Figure 22). For this MBD season, 11.5 days elapsed between onset of disease and for 10 per cent of RRV notifications (n=4,031) to be reported to public health units. The fastest reporting of RRV notifications was observed in 2001–02 and 2002–03 for all percentiles.

Figure 22. Timeliness of Ross River virus infections notifications, Australia, 1 July 2000 to 30 June 2006, by percentile



Dual reporting of Barmah Forest virus infections and Ross River virus infections notifications

During 2005–06, South Australia reported that it was observing a larger proportion of BFV notifications which were also positive for RRV. These were also notified resulting in a dual national notification of BFV and RRV. Further investigations by the National Arbovirus and Malaria Advisory Committee (NAMAC) and the Public Health Laboratory Network (PHLN) have revealed that the dual notification of BFV and RRV may be linked to the cross-reactivity of sera in both in-house and commercial assay kits. PHLN has advised that caution should be exercised when interpreting the current reporting and notification of BFV in Australia, and it is considering revising the laboratory case definition by increasing the cut-off titre to improve the specificity.

Data in the NNDSS is de-identified. However, it is possible to estimate what proportion of BFV notifications have a RRV notification (and vice-versa) by matching notifications with the same date of birth, sex, residential postcode and onset date. The incidence of a true dual BFV and RRV infection is unknown although it has been estimated as a rare event (NAMAC members, personal communication). If the dual BFV notifications are excluded from epidemiological analyses, only BFV notifications from Queensland in 2005–06 (n=478) drop below the activity of the previous five-year mean (570), but the overall BFV activity for Australia remains above the five-year mean.

Barmah Forest virus infection notifications with a corresponding Ross River virus infection notification

Table 1 shows that in 2005–06, 12 per cent (n=465 of 3,894) of national BFV notifications correspond to a RRV notification with the same demographic and geographic attributes. By this method the frequency of dual notifications appears to have increased steadily since 2000. Some jurisdictions such as the Northern Territory, Queensland, and Western Australia are reporting increasingly higher numbers of dual BFV and RRV notifications each reporting year. In 2000–01, Victoria reported that 14 per cent of BFV notifications had a corresponding RRV notification, but in subsequent years there have been no further dual notifications from Victoria. South Australia has only recently been reporting dual notification after a hiatus of dual notifications since 2000–01, perhaps marking a change in public health laboratory practice. It is not known what proportion of these jurisdictional trends in reporting dual notifications are related to increasingly greater dependence on a specific type or brand of serological test.

Ross River virus infection notifications with a corresponding Barmah Forest virus infection notification

Table 2 shows nationally that only 3 per cent of RRV notifications have a corresponding BFV notification, but this has risen steadily from 2000–01. Some jurisdictions in particular such as New South Wales, the Northern Territory, Queensland, and South Australia are notifying more cases of dual RRV and BFV notifications each year. Western Australia and Victoria are reporting the dual RRV and BFV notifications in the same proportions each year.

Table 1. Number and proportion of Barmah Forest virus infections with corresponding Ross River virus infection, Australia, 1 July 2000 to 30 June 2006, by year of onset and state or territory

State or territory	2000–01			2001–02			2002–03			2003–04			2004–05			2005–06		
	dual n	BFV n	BFV % dual	dual n	BFV n	BFV % dual	dual n	BFV n	BFV % dual	dual n	BFV n	BFV % dual	dual n	BFV n	BFV % dual	dual n	BFV n	BFV % dual
ACT	0	2	0	0	0	0	0	1	0	0	2	0	0	2	0	0	7	0
NSW	9	375	2	3	378	1	15	423	4	13	438	3	9	375	2	62	626	10
NT	2	33	6	1	25	4	0	18	0	5	44	11	2	33	6	8	98	8
Qld	19	602	3	17	418	4	86	805	11	53	676	8	19	602	3	326	2,398	14
SA	2	17	12	0	4	0	0	1	0	2	20	10	2	17	12	24	289	8
Tas	0	1	0	0	0	0	0	0	0	0	0	0	0	1	0	0	2	0
Vic	3	21	14	0	58	0	0	16	0	0	22	0	3	21	14	1	73	1
WA	5	81	6	2	45	4	2	25	8	2	69	3	5	81	6	44	401	11
Total	40	1,132	4	23	928	2	103	1,289	8	75	1,271	6	40	1,132	4	465	3,894	12

Table 2. Number and proportion of Ross River virus infections with corresponding Barmah Forest virus infection, Australia, 1 July 2000 to 30 June 2006, by year of onset and state or territory

State or territory	2000–01			2001–02			2002–03			2003–04			2004–05			2005–06		
	dual n	RRV n	RRV % dual	dual n	RRV n	RRV % dual	dual n	RRV n	RRV % dual	dual n	RRV n	RRV % dual	dual n	RRV n	RRV % dual	dual n	RRV n	RRV % dual
ACT	0	14	0	0	0	0	0	0	0	0	4	0	0	14	0	0	10	0
NSW	9	773	1	3	217	1	15	453	3	13	441	3	9	773	1	62	1,275	5
NT	2	233	1	1	71	1	0	134	0	5	180	3	2	233	1	8	267	3
Qld	19	1,717	1	17	944	2	86	2,391	4	53	1,013	5	19	1,717	1	326	7,561	4
SA	2	271	1	0	57	0	0	20	0	2	50	4	2	271	1	24	773	3
Tas	0	11	0	0	120	0	0	2	0	0	5	0	0	11	0	0	46	0
Vic	3	375	1	0	43	0	0	14	0	0	38	0	3	375	1	1	594	0
WA	5	236	2	2	130	2	2	146	1	2	144	1	5	236	2	44	3,254	1
Total	40	3,630	1	23	1,582	1	103	3,160	3	75	1,875	4	40	3,630	1	465	13,780	3

Chikungunya virus infections

An outbreak of chikungunya virus (CHIKV) occurred in the south-east Indian Ocean region in early 2006, with an estimated two-thirds of the human population of Reunion Island infected. Travellers returning from outbreak areas have been diagnosed in Europe, Canada, the Caribbean, South America and the United States of America (USA).[25]

In March 2006, Victoria reported to NAMAC and the Communicable Diseases Network Australia, a case of CHIKV in a 59-year-old male visitor from Mauritius associated with the Commonwealth Games (Rodney Moran, personal communication). The case arrived in Australia on 7 March 2006, with an onset of illness on 10 March 2006 (lethargy, fever, arthralgia, myalgia, rash, headache, ankle arthritis). He was admitted to hospital on 12 March, improved and discharged 15 March 2006.

In New South Wales, there were three confirmed cases of CHIKV associated with attendance at the Commonwealth Games all reported from one laboratory. All three were acquired in Mauritius (Linda Hueston, personal communication).

Flaviviruses

The Sentinel Chicken Programme is a surveillance network involving New South Wales, the Northern Territory, Victoria and Western Australia, and is designed to detect flavivirus activity (including the endemic arboviruses MVEV and KUNV).[26] Table 3 shows notifications of flavivirus from 1 July 2005 to 30 June 2006, by state or territory.

Northern Territory

The Northern Territory sentinel chicken program commenced in January 1992 and replaced an earlier program run by the Australian Quarantine and Inspection Service (AQIS). Sentinel chicken flocks in the Northern Territory are maintained, bled and analysed for flavivirus in a combined program between the Northern Territory Department of Health and Community Services, the Northern Territory Department of Business Industry and Resource Development (DBIRD), and volunteers.

Map 3 shows that the sentinel chicken flocks are presently at Darwin urban (Leanyer), Darwin rural (Howard Springs), Beatrice Hill (Coastal Plains Research Station), Kakadu (Jabiru), Katherine, Nhulunbuy, Tennant Creek, Alyangula, Nathan River and Alice Springs (Ilparpa and Arid Zone Research Station). DBIRD officers or volunteers usually bleed flocks once a month and the samples are sent to the Northern Territory Department of Business Industry and Resource Development for specific testing for MVEV and KUNV. Sometimes for operational reasons, chickens are not bled during a schedule month and hence seroconversion shown in the next bleed could have occurred in the previous month. When chickens from a flock show new antibodies to MVEV during a prime risk period, a media warning is issued for the region for the risk period. These warnings advise the public of the need to take added precautions to avoid mosquito bites.

Chickens are replaced at least annually and more frequently if birds die or if large proportions seroconvert. They are well positioned to detect flavivirus

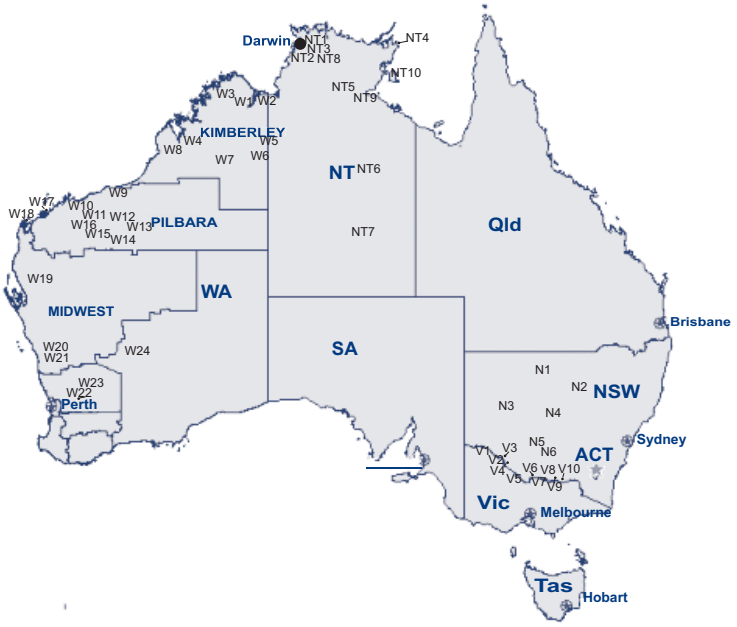
Table 3. Number and rate of flavivirus notifications, 1 July 2005 to 30 June 2006, Australia, by state or territory

State or territory	DENV		Flavivirus NEC		KUNV		MVEV	
	n	Rate	n	Rate	n	Rate	n	Rate
ACT	7	NA	0	NA	0	0	0	0
NSW	54	NA	2	NA	0	0	0	0
NT	16	NA	0	NA	0	0	0	0
Qld	79	NA	28	NA	0	0	0	0
SA	10	NA	0	NA	0	0	0	0
Tas	0	NA	0	NA	0	0	0	0
Vic	14	NA	13	NA	0	0	0	0
WA	20	NA	0	NA	2	0.1	1	0
Australia	200	NA	43	NA	2	0.01	1	<0.01

* Rate per 100,000 population.

NA Not applicable, rates not calculated since, most cases of dengue (outside Queensland) and flavivirus NEC were acquired overseas or had an unknown country of acquisition.

Map 3. Sentinel chicken testing sites, Australia 2005–06



Northern Territory

NT1. Howard Springs
NT2. Beatrice Hill Farm
NT3. Leanye
NT4. Gove
NT5. Katherine
NT6. Tennant Creek
NT7. Alice Springs (2)
NT8. Jabiru
NT9. Nathan River

New South Wales

N1. Bourke
N2. Macquarie Marshes
N3. Menindee
N4. Lake Cargellico
N5. Griffith
N6. Leeton

Victoria

V1. Mildura
V2. Robinvale
V3. Tooleybuc
V4. Swan Hill
V5. Kerang
V6. Barmah
V7. Toolamba
V8. Barooga
V9. Retheralen

Western Australia

W1. Wyndham
W2. Kununurra
W3. Kalumburu
W4. Derby (2)
W5. Sally Malay Mine
W6. Halls Creek
W7. Fitzroy Crossing
W8. Broome (2)
W9. Port Hedland
W11. Harding Dam (2)
W12. Marble Bar
W13. Nullagine
W14. Newman (2)
W15. Tom Price
W16. Pannawonica
W17. Onslow
W18. Exmouth
W19. Carnarvon
W21. Dongara
W22. Gingin
W23. York
W24. Leonora

activity near the principal towns of the Northern Territory and hence provide timely and accurate indication of risk to people in those towns.

In the 2005–06 season, MVEV activity was detected in Howard Springs in June, Leanyer in May, Adelaide River in March (probably February seroconversion) and June, Katherine in June, Tennant Creek in June (probably May seroconversion), Jabiru in May and Nathan River in June.

The MVEV total seroconversions this year (n=15) was similar to last year (n=13), with most seroconversion this year (n=4) occurring in the Adelaide River, followed by the Nathan River flock (n=3). Most seroconversion this year occurred in June (n=8) while the long-term seroconversion peak occurs in May closely followed by March and then February. The high number of seroconversions and the number of flocks seroconverting in June is most likely due to the extended wet season in the Northern Territory in 2005–06.

There were no seroconversions in the two Alice Springs flocks, most probably due to the below average summer rainfall and low vector numbers. In addition, the successful effluent swamp drainage and better effluent management from nearby sewage facilities in the Ilparpa area, have led to an overall reduction in vector numbers near the Alice Springs outskirts during summer.

There were also no seroconversions in the Nhulunbuy and the Alyangula flock. However, the Alyangula flock only commenced in April 2006.

No human cases of MVEV disease were reported in the Northern Territory in 2005–06 and the last reported case was in March 2005 when a 3-year-old boy from a community in Arnhem Land had a relatively mild illness and made a complete recovery.

Kunjin virus activity was present throughout the Northern Territory, with seroconversion to KUNV in Darwin (Howard Springs) in May; Darwin (Leanyer) in April; Adelaide River in May and June; Katherine in April, May and June; and in Tennant Creek in April.

There has been a trend over the last 10 years to increasing numbers of seroconversions to KUNV, with this year's total (n=13) higher than last year (n=12) and the highest since the program started in 1992. Most seroconversions occurred in the Katherine (n=6) and Adelaide River (n=4) flocks. Seroconversions mostly occurred this year in May (n=6), while the long term peak is also in May, followed at a substantially reduced level in April.

The Northern Territory did not report any human cases of KUNV infection this year. The last reported KUNV case from the Northern Territory was in a 23- year-old female from Alice Springs in May 2001.

Western Australia

Sentinel chicken flocks in Western Australia are maintained, bled and analysed for specific antibodies to MVEV and KUNV in a combined program between The University of Western Australia, Western Australian Department of Health, local governments and community volunteers. Twenty-eight sentinel chicken flocks were located at major towns and communities in the Kimberley, Pilbara, Gascoyne, Goldfields, Midwest and Central Coastal regions of Western Australia. Environmental health officers or trained volunteers took blood samples from the chickens each fortnight from December to June (the major MVEV 'risk' season) and monthly at other times. Samples were tested by the Arbovirus Surveillance and Research Laboratory at the University of Western Australia. Sometimes for operational reasons, chickens were not bled fortnightly and a seroconversion detected in the next bleed may have occurred earlier.

Rainfall was generally above average for most of the 2005–06 wet season in the Kimberley, Pilbara and Interior regions. There was extensive flooding in the Kimberley, Pilbara, Gascoyne and Interior regions in March and/or April 2006.

More than 3,000 serum samples from the 28 flocks located in Western Australia were tested for antibodies to flaviviruses during 2005–06. Seroconversions to flaviviruses were detected in 5.5 per cent of the samples. Overall, flavivirus activity was very high during the 2005–06 season, and the majority of seroconversions were due to infection with MVEV. The first seroconversions were detected at Sally Malay, Wyndham and Derby in February 2006. Activity rapidly increased to include all major towns in the Kimberley region and continued through to June 2006 (and into the 2006–07 season). The first flavivirus seroconversion in the Pilbara region was a MVEV infection in a sentinel chicken at Tom Price in March 2006. During the ensuing months, MVEV activity was detected at all locations in the Pilbara except Port Hedland. However, KUNV activity was subsequently detected at Port Hedland in July 2006. One seroconversion to MVEV was detected in the Carnarvon sentinel chicken flock in June 2006. This is the furthest south that MVEV has been detected since August 2000, when there was an extensive and prolonged period of MVEV associated with Cyclone Steve.[27] Unidentified flavivirus infections were detected at a number of locations in the Kimberley and Pilbara regions, and are possibly due to activity of other flaviviruses that are occasionally isolated from mosquitoes collected in northern Western Australia.

Three flavivirus human cases (Table 3) were reported from Western Australia during the 2005–06 season (Dr David Smith, PathWest, personal communication). The first case was reported in a 27-year-old woman from Broome who acquired a KUNV infection just prior to March 2006. There was one case of KUNV infection (with polyarthralgia) in a Kununurra resident in April 2006. One case of encephalitic MVEV infection requiring hospitalisation was diagnosed in an 8-year-old female from Broome in June 2006. Seroconversions to MVEV or KUNV in sentinel chickens provided advance warning of flavivirus activity in these regions.

The West Australian Department of Health initially issued health warnings on 24 February 2006, of increased risk of MVEV to residents and visitors to the north or east Kimberley region, following seroconversions to MVEV in the north-east Kimberley region. Additional warnings were issued on 7 April after heavy rainfall in the Pilbara region and the detection of MVEV; on 1 May following increased MVEV activity in the Pilbara, the detection of KUNV at Exmouth and continued heavy rainfall in the Pilbara and Gascoyne; and on 15 June after MVEV was detected in the sentinel chicken flock at Carnarvon. The warnings advised residents and travellers to the regions of the increased risk of disease and the need to take precautions to avoid mosquito bites.

New South Wales

Samples from six sentinel chicken sites were tested weekly for KUNV and MVEV antibodies in New South Wales from mid-October 2005 to the end of April 2006. There were no seroconversions to MVEV or KUNV during this period. There were no human cases reported from New South Wales for either MVEV or KUNV. The last reported case of KUNV from New South Wales was notified in May 2001 in a 58-year-old female from Griffith. There have been no recorded cases of MVEV to date in NNDSS from New South Wales.

Victoria

Samples from sentinel chicken flocks located throughout northern inland Victoria (10 sites along the Murray River, Map 3) were tested weekly for flavivirus antibodies from 1 November 2005 to early March 2006. No KUNV or MVEV activity was detected in any of the samples. There were no human cases of KUNV or MVEV reported from Victoria during 2005–06. The last reported case of

KUNV infection in Victoria was in October 2004. There have been no recorded cases of MVEV in NNDSS from Victoria.

Queensland

There were no sentinel chicken flocks in Queensland during 2005–06 although flocks were maintained in 2002–03. There were no cases of KUNV or MVEV reported by Queensland during 2005–06. The last reported KUNV cases from Queensland were three sporadic cases notified in July 2004, December 2004, and February 2005. The last reported MVEV case from Queensland was in a 3-year-old boy from Mount Isa in 2001.

Japanese encephalitis virus infections

Japanese encephalitis virus appears nearly annually in the Torres Strait in far northern Queensland, and threatens to invade the Australian mainland. Surveillance has involved the use of sentinel pigs that develop detectable viraemia and antibody titres to JEV.

The use of sentinel pigs on Badu Island was discontinued in 2006 on the recommendation by Queensland Health, who concluded that JEV incursions during the wet season will be an annual occurrence as far south as the central islands of the Torres Strait. In March 2006, an annual Northern Australia Quarantine Strategy (NAQS) survey of the Torres Strait and Northern Peninsula Area domestic livestock, detected JEV activity on Moa Island from blood samples obtained from juvenile domestic pigs.

AQIS, through the NAQS program, conducted monitoring for JEV for the 2006 wet season using sentinel pigs at Injinoo airport, Northern Peninsula Area, Cape York. The five sentinel pigs did not seroconvert and there was no evidence of transmission of JEV to the mainland in 2006 (based on results of testing at Queensland Health Scientific Services and the CSIRO Australian Animal Health Laboratory).

The use of pigs as a sentinel system poses a public health risk because pigs are amplifying hosts for JEV. A remote mosquito trap system does not have this risk and mosquito trapping on the Cape York peninsula has resulted in the first report of a mosquito isolate of JEV from the Australian mainland.[28] Ritchie (unpublished data) evaluated and compared a remote mosquito trap system with pigs for the surveillance of JEV on Badu and Moa islands in the Torres Strait and at Bamaga in northern Cape York Peninsula from 2002–2005. JEV was detected in mosquito collections each year but not for each trap type and no JEV was detected in trapped mosquitoes before detection in sentinel pigs. A remote mosquito trap system, employing stand alone traps and polymerase chain reaction for viral antigen detection was found to be a safe, economical way to detect arbovirus activity in these remote areas.

There were no human cases of JEV in Australia during 2005–06. The last reported JEV case was in February 2004, when Queensland notified that a 66-year-old male acquired JEV from Papua New Guinea. There have been nine other cases of JEV reported to NNDSS since 1995, although JEV was not nationally notifiable until 2001. Four of these notifications were reported in Torres Strait islanders from the Badu Island community, two of which were fatal (1995). The other locally-acquired JEV case was reported in a resident from the mouth of the Mitchell River, Cape York Peninsula, Queensland in 1998. The remaining four cases were reported as acquired from overseas countries.

Flavivirus infections (not elsewhere classified)

There were 43 flavivirus (not elsewhere classified or NEC) notifications during the 2005–06 season, of which 13 were acquired overseas. The source of acquisition was unknown for the remaining notifications.

Queensland reported 28 flavivirus (NEC) notifications; Victoria (n=13) and New South Wales (n=2). Of the 28 flavivirus infection (NEC) notifications from Queensland, there were six Kokobera and one Stratford virus. The flavivirus infection (NEC) notifications from Victoria and New South Wales were of unknown type.

Dengue virus infections

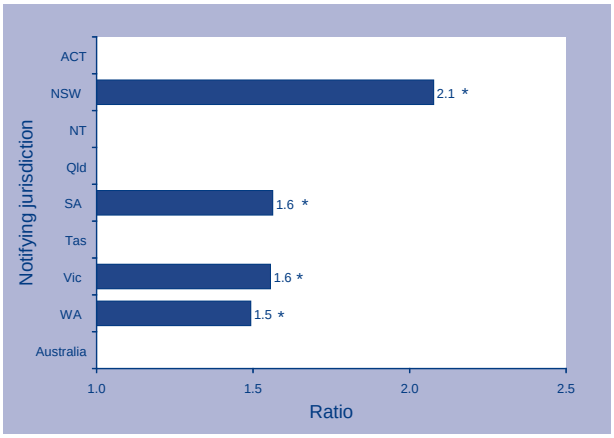
There were 200 notifications of DENV infections during the 2005–06 season. Table 4 shows that the cases were mainly from Queensland (n=79, 40%), New South Wales (n=54, 27%) and Western Australia (n=20, 10%). Notifications from New

Table 4. Locally acquired and imported dengue notifications, Australia, 1 July 2005 to 30 June 2006, by state or territory

Notifying jurisdiction	n	%
ACT	7	4
NSW	54	27
NT	16	8
Qld	79	40
SA	10	5
Tas	0	0
Vic	14	7
WA	20	10
Total	200	100

South Wales, South Australia, Victoria and Western Australia exceeded the five-year mean in each jurisdiction (Figure 23). Of the national DENV notifications notified, 46 per cent (n=92) were reported as imported, 43 per cent (n=86) were locally-acquired and 11 per cent (n=22) had an unknown source of acquisition.

Figure 23. Ratio of locally acquired and imported dengue notifications to mean of previous five years, Australia, 1 July 2005 to 30 June 2006, by state or territory

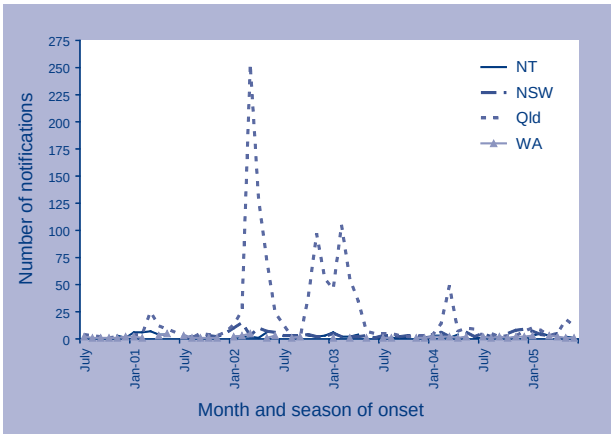


* Above 2 standard deviations.

Figure 24 shows that the number of DENV notifications received during the 2005–06 season was much lower than the previous three seasons. New South Wales, South Australia, Victoria and Western Australia reported DENV activity above the five-year mean.

There were two outbreaks of locally acquired DENV in Queensland during this season (Jeffrey Hanna, Scott Ritchie, personal communication). There were eight cases of DENV serotype 3 in Townsville from December 2005 – February 2006. In the Cairns and Gordonvale area, there were 19 cases of DENV serotype 2 up to 30 June 2006 with a peak in cases in May 2006 (Figure 24). The outbreak was finally controlled in September 2006 with a total of 29 cases (Jeffrey Hanna, Scott Ritchie, personal communication).

Figure 24. Dengue notifications (locally-acquired and imported cases), select jurisdictions, 1 July 2005 to 30 June 2006, by month and season of onset



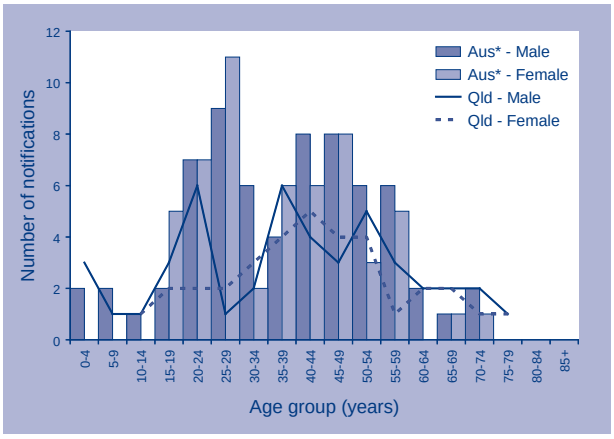
Of the 200 DENV notifications, dengue serotype 2 was reported in 16 per cent (n=31) of cases (Table 5). There were 15 cases of serotype 3 infection, 7 cases of serotype 1, and 5 cases of serotype 4. Serotype information was either not stated or unavailable for 71 per cent of the notifications (n=142).

Table 5. Dengue notifications (locally-acquired and imported cases), Australia, 1 July 2005 to 30 June 2006, by serotype

Serotype	n	%
Serotype 1	7	4
Serotype 2	31	16
Serotype 3	15	8
Serotype 4	5	3
Not typed/unknown	142	71
Total	200	100

Figure 25 shows that imported DENV notifications in Australia were most frequently reported in the 25–29 year age group (n=20, 17%) whereas in locally acquired cases from Queensland, this age group was rarely affected (n=3, 4%), with many of the locally acquired cases occurring in the 20–24 year age group (n=8, 10%) and the 35–54 year age groups (n=35, 44%).

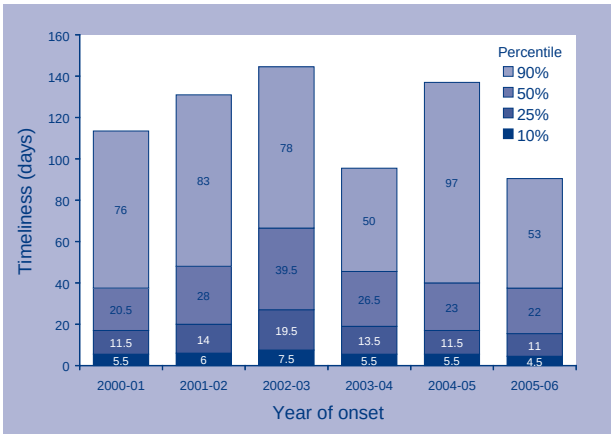
Figure 25. Dengue notifications, Queensland and Australia, 1 July 2005 to 30 June 2006, by age group and sex



* Excludes Queensland.

The timeliness of reporting DENV notifications is shown in Figure 26. During 2005–06, it took 4.5 days to notify 10 per cent of DENV notifications and 53 days to notify 90 per cent of notifications from onset of disease to receipt of notification by the public health unit. The late notification (50% >22 days) indicates that the risk of further transmission in areas with *Aedes aegypti* is high, particularly as the DENV replicates within the mosquito during an incubation period of 10–12 days.

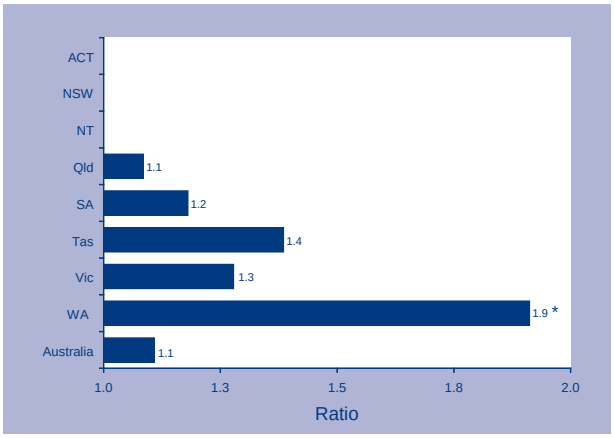
Figure 26. Timeliness of dengue notifications, Australia, 1 July 2000 to 30 June 2006, by percentile



Malaria

There were 731 notifications of malaria in Australia in the period 1 July 2005 to 30 June 2006. No reports of locally-acquired malaria were notified during the reporting period. Queensland reported the majority of cases (n=309, Table 6). Western Australia reported malaria notifications which exceeded two standard deviations above its five-year average (Figure 27). Victoria reported that a large number of malaria notifications in the period 2005–06 were acquired in Papua New Guinea (Rodney Moran, personal communication).

Figure 27. Ratio of malaria notifications to mean of previous five years, Australia, 1 July 2005 to 30 June 2006, by state or territory



* Above 2 standard deviations.

Figure 28 shows that the 2005–06 reporting period was the third largest for malaria notifications since 2000–01.

Figure 28. Number of notifications of malaria, Australia, 2000 to 2006, by year of onset

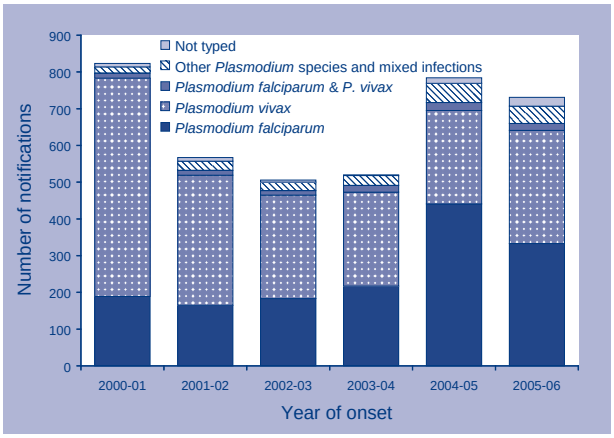


Table 6. Malaria notifications in Australia, 1 July 2000 to 30 June 2006, by parasite type

Infecting species	Year of onset						Last 5 year mean	Ratio 05–06/ 5 year mean
	2000–01	2001–02	2002–03	2003–04	2004–05	2005–06		
Plasmodium falciparum	189	165	183	217	441	333	239	1.4
Plasmodium vivax	594	354	282	256	254	308	348	0.9
Plasmodium falciparum and *P. vivax*	14	13	12	18	22	19	16	1.2
Other *Plasmodium* species and mixed infections	17	25	23	27	52	47	29	1.6
Not typed	9	10	6	2	15	24	8	2.9
Total	823	567	506	520	784	731		

Overall, malaria notifications were highest in the young adult 20–24 year age group (Figure 29). This trend was also observed in years prior to 2004–05 (Figure 30).

Figure 29. Number of imported malaria notifications, Australia, 1 July 2005 to 30 June 2006, by age group and sex

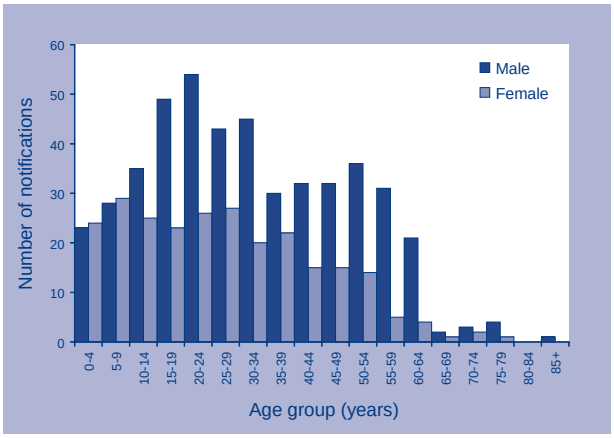
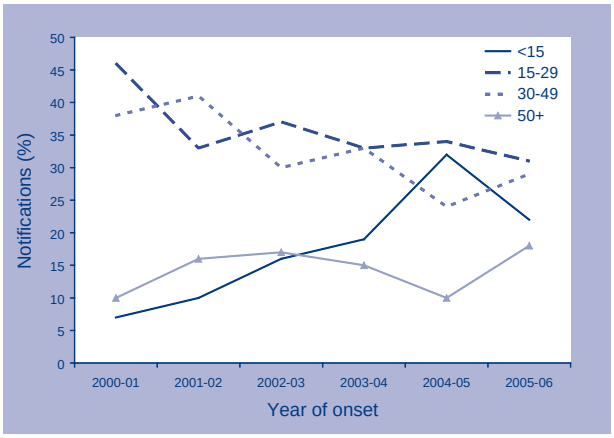


Figure 30. Trends in the age distribution of malaria notifications, Australia, 1 July 2000 to 30 June 2006, by age group



In the period 2005–06, the proportion of children under the age of 15 years notified with malaria was 22 per cent (n=164), but this was not as high as observed in 2004–05 when malaria notifications in this age group accounted for almost one third of all notifications for the first time since 1998. The trend in younger children represented in notifications has been discussed elsewhere and is related to refugee arrivals.[29]

More male notifications (n=471) than female notifications (n=253) were reported. Males in the 20–24 year age group were the largest reported sex-specific cohort whereas the largest reported numbers of female notifications were observed in the 5–9 year age group.

The infecting *Plasmodium* species was reported for 97 per cent of malaria notifications in 2005–06 (Table 6). The majority of the 731 malaria notifications were due to *P. falciparum* (45%, n=333) and *P. vivax* (42%, n=308) while other *Plasmodium* species or mixed *Plasmodium* species infections accounted for 6 per cent (n=47).

Figure 31 shows that in 2005–06 the proportion of notifications due to *P. falciparum* malaria (45%) decreased slightly from last year (56%) but was still 1.4 times the five-year mean for the same species. The number of hospital separations due to *P. falciparum* malaria increased significantly in 2004–05 (Figure 32), and this increase was most probably associated with the arrival of refugees from Sub-Saharan Africa, particularly children.[29]

Figure 31. Trends in malaria notifications, by infecting species and year of onset

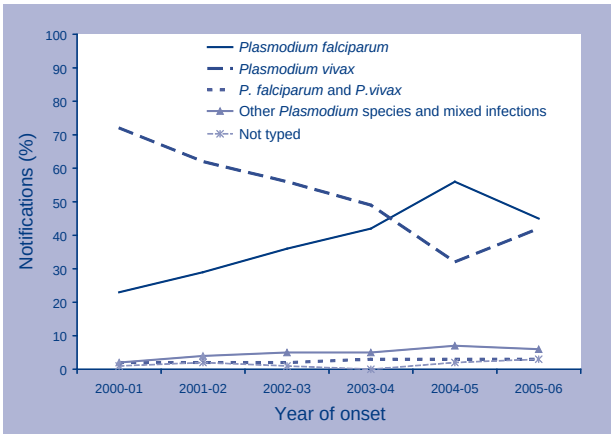
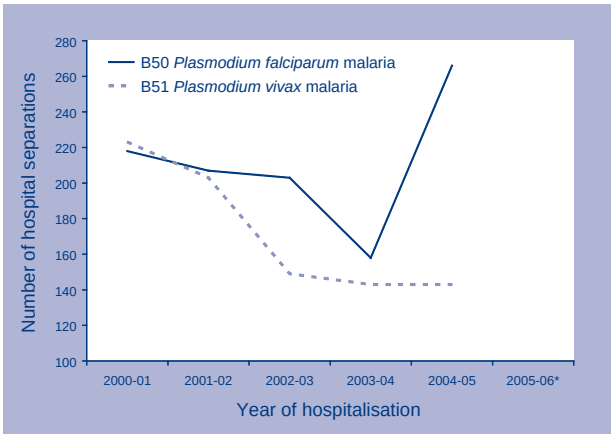
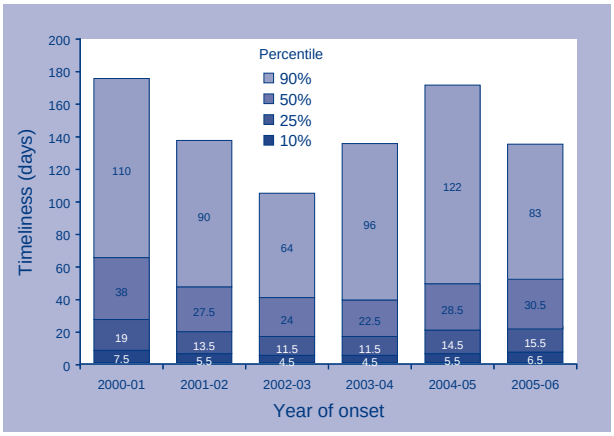


Figure 32. Hospital separations, by principal diagnosis and year of hospitalisation



The timeliness of malaria notifications is shown in Figure 33. In 2005–06, it took 6.5 days for 10 per cent of notifications to reach public health units. The timeliness of malaria notifications was generally better in 2002–03 and 2003–04 for all percentiles.

Figure 33. Timeliness of malaria notifications, Australia, 1 July 2000 to 30 June 2006, by percentile



* Data from 2005-06 not available at time of writing.

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

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Surveillance of antibiotic resistance in *Neisseria gonorrhoeae* in the WHO Western Pacific Region, 2005

The WHO Western Pacific Gonococcal Antimicrobial Surveillance Programme

Abstract

The World Health Organization Western Pacific Region Gonococcal Antimicrobial Surveillance Programme examined about 8,700 isolates of *Neisseria gonorrhoeae* from 15 countries for resistance to antibiotics in 2005. High to very high rates of resistance to penicillins and quinolones persisted in most centres. Increasing numbers of gonococci with decreased susceptibility to third generation cephalosporins were found in several countries. There were infrequent instances of spectinomycin resistance. *Commun Dis Intell* 2006;30:430–433.

Keywords: disease surveillance, Neisseria gonorrhoeae, *Western Pacific Region*

Introduction

Antimicrobial resistance in *Neisseria gonorrhoeae* has been a major problem in the World Health Organization (WHO) Western Pacific Region (WPR), and has impacted adversely on treatment and control of gonococcal disease for many years. The WPR Gonococcal Antimicrobial Surveillance Programme (WPR GASP) has monitored this resistance in gonococci at a regional level from 1992 onwards.[1] High levels of penicillin resistance had emerged in the 1970s and the WPR GASP has also reported on a progressive increase in quinolone resistance in gonococci in the region[1,2] as well as resistance to other established agents for the treatment of gonorrhoea. In recent years, increasing numbers of gonococci with decreased susceptibility to oral and injectable third generation cephalosporins have been detected, particularly in Japan.[3,4]

Laboratory assessment of *in vitro* resistance to antibiotics in *N. gonorrhoeae* is undertaken with the aim of optimising treatments, and thereby preventing the complications of gonococcal disease and assisting in its control.[5] The WHO recommends that a standard or programmatic treatment regimen should be altered once resistance is found in 5 per cent or more of isolates from a general population.[5,6] This report provides an analysis of antimicrobial resistance in *N. gonorrhoeae* in the WHO WPR derived from the results of the WPR GASP surveillance for 2005.

Methods

The methods used by the WHO WPR GASP have been published[1] and provide full details of the source of isolates, sample populations, laboratory test methods and quality assurance programs used to generate data. These methods were unaltered in 2005.

Results

About 8,700 gonococcal isolates were examined for susceptibility to one or more antibiotics in 15 participating countries in 2005.

Quinolone antibiotics

Table 1 shows the distribution of quinolone-resistant *N. gonorrhoeae* (QRNG) in 13 countries that examined a total of 8,233 isolates in 2005. The proportion of QRNG found in isolates tested ranged from 2 per cent in New Caledonia to nearly 100 per cent in the Hong Kong SAR and China. QRNG represented 20 per cent or more of all gonococci tested in the centres other than New Caledonia and 50 per cent or more isolates were QRNG in Brunei, Japan, Laos, Korea, the Philippines, Singapore and Vietnam. When compared to earlier data, these rates were in general higher than in previous years. Most isolates with altered quinolone susceptibility had ciprofloxacin MICs that are associated with high rates of treatment failure (≥1 mg/L).

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Table 1. Quinolone resistance strains of *Neisseria gonorrhoeae* isolated in 13 countries in the WHO WPR, 2005

Country	Tested	Less susceptible		Resistant		All QRNG	
		n	%	n	%	n	%
Australia	3,886	77	2	1,113	28.6	1,190	30.6
Brunei	116	14	12	64	55.2	78	67.2
China	1,442	47	3.2	1,384	96	1,431	99.2
Hong Kong SAR	1,887	67	3.6	1,794	95.1	1,871	98.7
Japan	26	1	3.8	20	76.9	21	80.8
Korea	48	5	10.4	39	81.2	44	91.7
Lao PDR	29					19	65.5
Malaysia	17	3	17.6	2	11.7	5	29.3
New Caledonia	55	0		1	1.8	1	1.8
New Zealand	310	0	0	60	19.4	60	19.4
Philippines	94					37	39.3
Singapore	160	13	8.1	95	59.4	108	67.5
Vietnam	163	35	21.5	96	58.9	131	80.4
Total	8,233	262		4,668		4,996	

Cephalosporins

Strains with some decrease in susceptibility to third generation cephalosporins were again detected in isolates from Australia, Brunei, China and Malaysia and were particularly prominent in China. Because of some methodological differences in testing, MIC values are not directly comparable between different centres, but values ranged up to 0.25 mg/L.

Spectinomycin

Only very small numbers of spectinomycin resistant gonococci have been reported in recent years in WPR GASP surveys. Of the 6,000 isolates tested in eight countries in 2005, three isolates in China and one each in Malaysia and the Philippines were spectinomycin resistant.

Penicillins

Resistance to penicillins has been widespread and at high levels for many years in the WPR, and may be the result of penicillinase production (PPNG) or aggregation of a number of chromosomally mediated mechanisms (CMRNG). These mechanisms may co-exist in the one strain. Table 2 shows the penicillin susceptibility of 8,722 gonococci in 15 WHO WPR centres. Once again penicillin resistance was widespread and found in a high proportion of isolates in most centres. The highest rates of resistance to the penicillins were found in China and Laos. In previous reports, some Pacific Island states have consistently reported low levels of penicillin resistance, but there has been a shift towards resistance in recent years. In Fiji, for example, 16 per cent of 328 gonococci were penicillin resistant in 2005 with 10 per cent of all isolates PPNG. In 2003, only three per cent of isolates were penicillin resistant and in 2004, 6.4 per cent of isolates were penicillin resistant.

Tetracyclines

These antibiotics are still widely available in the WPR. About 7,800 isolates were examined for one particular form of resistance, namely, high-level plasmid-mediated form (TRNG), in 10 countries in 2005 (Table 3). The highest rates of TRNG were reported from Singapore (78%) and Hong Kong (42%). Only low numbers were present in Malaysia and New Caledonia. Low proportions of TRNG (at or around 10%) were found in Japan, Korea, Australia and Vietnam, and slightly higher rates in China (28%) and the Philippines (30%).

Discussion

Despite limitations, surveys of this kind provide the best available indication of antimicrobial resistance (AMR) in *N. gonorrhoeae* in the WHO WPR. Additionally, they have a particular value in being able to follow trends in AMR over time. Little comfort can be taken from this latest report of resistance rates in *N. gonorrhoeae* in the WHO WPR. High rates of resistance to cheaper oral agents such as the quinolones have continued and the few situations where penicillins remained a viable treatment, such as some Pacific Island centres, are showing sustained upward trends in resistance to this group of antibiotics.

Table 2. Penicillin resistance strains of *Neisseria gonorrhoeae* isolated in 15 countries in the WHO WPR, 2005

Country	Tested	PPNG		CMRNG		All Pen R	
		n	%	n	%	n	%
Australia	3,886	410	10.5	738	19	1,148	29.5
Brunei	194					111	57.2
China	1,474	556	37.7	289			
China SH/GD*	298	126	42.2	163	54.7	289	96.9
Fiji	328	33	10	20	6	53	16
Hong Kong SAR	1,887	586	31	529	28	1115	59
Japan	26	2	7.7	11	42.3	13	50
Korea	48	7	14.6	22	45.8	29	60.4
Lao PDR	29	16	55	13	45	29	100
Malaysia	17	4	23.5	2	11.7	6	35.2
New Caledonia	55	1	1.8	0	0	1	1.8
New Zealand	310	6	1.9	28	9	34	10.9
Philippines	94					64	68
Singapore	160	81	50.6	16	10	97	60.6
Tonga	48					10	20.8
Vietnam	166	57	34.3	1	0.6	58	34.9
Total	8,722†	1,885		1,832		3,057	

* A sample of 298 gonococci examined for both lactamase production and chromosomal resistance in Shanghai and Guandong; elsewhere isolates were examined for penicillinase production only.

† Total excludes the 298 gonococci examined in Shanghai and Guandong.

Table 3. High-level tetracycline resistance in strains of *Neisseria gonorrhoeae* isolated in 10 countries in the WHO WPR, 2005

Country	Tested	n	%
Australia	3,886	534	13.7
China	1,442	405	28.1
Hong Kong SAR	1,887	791	41.9
Japan	26	2	7.7
Korea	48	5	10.4
Malaysia	17	1	5.8
New Caledonia	55	1	1.8
Philippines	94	28	29.7
Singapore	160	124	77.5
Vietnam	162	16	9.9
Total	7,777	1,907	

Decreased susceptibility to alternative agents is also a concern. One group of antibiotics now widely used is the third generation cephalosporins, either as an oral preparation such as cefixime or cefdinir or in the form of the injectable ceftriaxone. At different times Australia, Cambodia, Brunei, China, Japan, Korea, Malaysia, New Zealand, Papua New Guinea, Singapore and Tonga have reported the presence of gonococci with decreased susceptibility to these agents. This reduced susceptibility to later generation cephalosporins was associated with the presence of a number of mosaic *penA* genes,[7] but this is probably not the full explanation of this phenomenon.[8,9] Those gonococci with this decreased susceptibility to cephalosporins are often multi-resistant, and exhibit high-level quinolone resistance as well as resistance to other beta-lactam antibiotics. These strains have now spread beyond the WHO WPR.[10,11]

The current situation is one where the provision of effective treatment for individuals and disease control efforts are compromised by high and increasing levels of AMR. This has considerable health and economic consequences in the WHO WPR. The combination of high disease rates and general problems of antibiotic resistance means that control of gonorrhoea is unlikely to be achieved in the immediate future unless concerted long-term efforts are applied.

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National Rotavirus Surveillance Program annual report, 2005–06

Carl D Kirkwood,[1] David Cannan,[2] Nada Bogdanovic-Sakran,[3] Ruth F Bishop,[4] Graeme L Barnes[5] and the National Rotavirus Surveillance Group[6]

Abstract

The National Rotavirus Reference Centre together with collaborating laboratories Australia-wide has conducted rotavirus surveillance since June 1999. This report describes the serotypes of rotavirus strains responsible for the hospitalisation of children with acute gastroenteritis during the period 1 July 2005 to 30 June 2006. Eight hundred and forty-eight faecal samples from across Australia were examined using monoclonal antibody immunoassays, reverse transcription-polymerase chain reaction and polyacrylamide gel analysis. Serotype G1 was the dominant serotype nationally, representing 40.2 per cent of all strains, followed by serotype G4 (22.6%), serotype G9 (15.1%) and serotype G3 (14.7%). Genotype G12 strains were identified for the first time in Australia. As in previous years, there was substantial geographic variation in the prevalence of rotavirus serotypes. *Commun Dis Intell* 2006;30:434–438.

Keywords: disease surveillance; rotavirus

Introduction

Group A rotaviruses are the single most important cause of severe gastroenteritis in young children worldwide. An estimated 500,000 children die annually of severe diarrhoea, however few of these deaths occur in developed countries.[1] Rotavirus induced disease accounts for up to 50 per cent of childhood hospitalisations for diarrhoea, with 10,000 Australian children hospitalised each year,[2] costing an estimated $26 million in direct costs. Clinical trials of two live oral rotavirus vaccines, Rotarix® (GlaxoSmithKline) and RotaTeq® (Merck) conducted in over 60,000 children worldwide have shown that both vaccines are highly efficacious in prevention of severe diarrhoea and hospitalisation due to rotavirus infections.[3,4] As a result, these vaccines have been licensed in Australia and many other countries throughout the world during 2006.

National epidemiological surveillance of rotavirus strains remains an important component in rotavirus vaccine evaluation and implementation programs. During the past five years, the national surveillance program has reported the emergence of serotype G9 strains as the dominant serotype nationally, and reported the re-emergence by serotype G1 as the dominant type since the 2003–2004 rotavirus season.[5,6] The changing pattern of dominant serotypes, together with the multiple types identified in the Australian population each year, highlights the diversity of rotavirus strains capable of causing disease in children.

The surveillance and characterisation of rotavirus strains causing annual epidemics of severe diarrhoea in young children in Australia continues to be undertaken by the National Rotavirus Reference Centre in Melbourne, together with nine collaborating laboratories. In this report we describe the

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results for the period 1 July 2005 to 30 June 2006, and identify the geographic distribution of the predominant rotavirus serotypes.

Methods

Rotavirus positive specimens detected by enzyme immunoassay (EIA) or latex agglutination in collaborating laboratories were collected, stored frozen and forwarded to Melbourne, together with relevant age and sex details. Specimens were then serotyped using an in-house monoclonal antibody (MAb) based serotyping EIA. The EIA employed a panel of MAbs specific for the major glycoprotein VP7 of the outer capsid of the five major group A human rotavirus serotypes (G1, G2, G3, G4 and G9).[7] Strains which could not be assigned a G serotype were genotyped by reverse transcription/polymerase chain reaction, using serotype specific oligonucleotide primers.[8] Polyacrylamide gel electrophoresis (PAGE) was used to classify rotavirus strains genetically into electropherotypes, and to examine the extent of sharing of the same electropherotype between collaborating centres.

Results

Number of isolates

A total of 1,011 specimens were received for analysis from Melbourne and the collaborating centres in New South Wales, the Northern Territory, South Australia, and Western Australia. Eight hundred and forty-eight specimens were confirmed as rotavirus positive using our in-house EIA assay. Specimens containing insufficient specimen for testing, or specimens that were not confirmed to be positive for rotavirus (n=163) were not analysed further.

Age distribution

The overall age distribution of children with acute rotavirus gastroenteritis is depicted in the Figure. In the reporting period, 43.7 per cent of cases were from infants 12 months of age or less, 30.6 per cent were from patients 13–24 months of age, and 10.7 per cent were from patients 25–36 months of age. Overall, 85 per cent of samples were from children aged three years or less, and 92.9 per cent were from children aged five years or less. The male to female ratio was 1:1.

Figure. Cases of rotavirus, Australia, 1 July 2005 to 30 June 2006, by age group

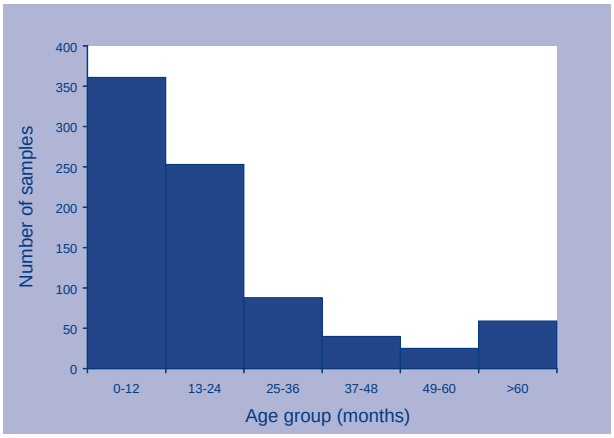

Table. Rotavirus G serotypes, Australia, 1 July 2005 to 30 June 2006

Centre	Total number	Serotype													
		G1		G2		G3		G4		G9		mix		NR	
		%	n	%	n	%	n	%	n	%	n	%	n	%	n
Melbourne	132	79.5	105	2.3	3	5.3	7	0.8	1	8.3	11	0.8	1	3.0	4
Sydney (POW)	52	58	29	3.8	2	0	0	5.8	3	34.6	18	0	0	0	0
Sydney (Westmead)	80	61.3	49	5.0	4	3.7	3	2.5	2	7.5	6	0	0	20	
Perth	76	40.8	31	0	0	2.6	2	54.2	41	1.3	1	0	0	1.3	1
PathWest WA	171	32.2	55	1.2	2	2.3	4	49.7	85	6.4	11	0.6	1	7.6	13
Alice Springs	133	3.0	4	0	0	69.2	92	23.3	31	2.3	3	1.5	2	0.7	1
Darwin	41	19.5	8		0	12.2	5	2.4	1	63.4	26	0	0	2.4	1
Darwin (Western Pathology)	70	0	0	0	0	17.1	12	0	0	71.4	50	2.9	2	8.6	6
Adelaide	93	64.5	60	0	0	0	0	30.1	28	2.2	2	0	0	3.2	3
Total	848	40.2	341	1.3	11	14.7	125	22.6	192	15.1	128	0.7	6	5.3	45

An additional 163 specimens were omitted from analysis due to insufficient sample, or specimen was not confirmed to be rotavirus positive.

* Eleven samples were identified as genotype G12.

Serotype distribution

The rotavirus serotypes identified in Australia from 1 July 2005 to 30 June 2006 are shown in the Table. Serotype G1 was the most common, representing 40.2 per cent of all specimens. It was the dominant strain in the 3 southern state capital cities of Melbourne, Sydney, and Adelaide, and was the second most common type in Western Australia. Serotype G4 was the second most common serotype nationally, and represented 22.6 per cent of specimens. It was identified in eight of the nine collaborating centres but was the dominant type only in Western Australia. Nationally, serotype G9 and G3 represented 15.1 per cent and 14.7 per cent of all specimens, respectively. Serotype G9 strains were found in all centres, and were the dominant type in Darwin. Similarly, serotype G3 strains were found in seven of the nine centres, and were dominant in Alice Springs. Only nine serotype G2 strains were each identified in three centres during the study, and represented less 1.3 per cent of the total strains identified. Interestingly, 11 samples from The Children's Hospital at Westmead in Sydney were found to be genotype G12.

Less than one per cent of the rotavirus samples contained multiple serotypes, and in 4.0 per cent of the samples a serotype was not identified. The latter could be samples with virus numbers below the detection limits of our assays, or could have contained inhibitors present in extracted RNA which prevent the function of the enzymes used in RT and/or PCR steps. It is unlikely that these represent unusual serotypes not identified using standard methods, since none of the non-typeable isolates exhibited unusual PAGE patterns. Future studies will include further characterisation of the genes encoding the outer capsid proteins of these strains.

Discussion

National rotavirus surveillance from 1 July 2005 to 30 June 2006 found that serotype G1 was the dominant serotype nationally, comprising 40.2 per cent of all strains. It was identified in all centres, and continues to be the dominant type along the Eastern seaboard, in Melbourne and Sydney and Adelaide. Serotype G1 has been the dominant type nationally for all except two years since 1999.[9–10] It was only the emergence of serotype G9 during 2001–03, which replaced G1 as the dominant serotype in Australia. Serotype G1 continues to be reported as the dominant type in epidemiological studies conducted throughout the world.[11,12]

The survey highlights the diversity of serotypes that cause disease in Australian children. Serotype G4, G9 and G3 strains were each the dominant serotype in one location (Western Australia, Darwin and Alice Springs), and comprised 22.6 per cent, 15.1 per cent and 14.7 per cent of all strains nationally.

Of significance was the emergence of serotype G4 strains as the dominant type in Western Australia, and second most predominant serotype in two centres (Adelaide and Alice Springs). The emergence of serotype G4 strains follows three previous annual reports which showed that G4 strains represented only a minor type nationally (<2% overall). The last time serotype G4 strains represented an important serotype in Australia was 2001, when it was identified in 9.7 per cent of strains nationally.[13]

While serotype G3 remained a significant cause of acute gastroenteritis in Alice Springs in this survey, its prevalence throughout Australia has declined rapidly, from a prevalence of 36.6 per cent in 2004–05 to 14.7 per cent nationally this year. The predicted eastward spread of G3 strains to Adelaide, Sydney and Melbourne did not occur during the 2005–06 reporting period and this may explain the decline in G3 predominance.

The prevalence of serotype G9 has slightly increased during the current survey, being present in all collaborating centres for the first time since the 2003–04 report, and represented 15.1 per cent of all strains nationally. This is an increase from the previous survey, however, whether serotype G9 will become the dominant type nationally as it was in 2001–03 remains to be determined.

The identification of genotype G12 strains represents the first report of strains belonging to this type in Australia. Genotype G12 strains were first identified in the Philippines in 1990, and have subsequently been identified in a variety of countries such as Japan, Malaysia, Italy, the United States of America and India as a single isolate or minor proportion.[14–16] Whether this genotype continues to emerge is unclear. In Australia, it is likely that genotype G12 will be similar to genotype G6 and G8 which represent rare strains, identified as single infections or small outbreaks. However, continued surveillance for these rare genotypes is important to understand whether they emerge as important types.

The rotavirus serotyping results from this survey, together with those of previous years, highlights the changes in the prevalence of rotavirus strains across Australia. In addition, the identification of genotype G12 further highlights the diversity of strains capable of causing severe disease in Australian children. Therefore, given the recent licensure of two rotavirus vaccines in Australia, understanding the fluctuations in rotavirus serotypes using multi-centre national surveillance will provide valuable insight into vaccine efficacy.

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Western Australia

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Supplementary report: surveillance of adverse events following immunisation among children aged <7 years in Australia, 1 January to 30 June 2006

Glenda Lawrence,[1] Ian Boyd[2]

Keywords: AEFI, adverse events, vaccines, surveillance, immunisation, vaccine safety

This report summarises national passive surveillance data collated in the Adverse Drug Reactions Advisory Committee (ADRAC) database at 30 September 2006 for adverse events following immunisation (AEFI) reported for children aged less than 7 years who received vaccines between 1 January and 30 June 2006.[1–3] It is the first full reporting period for AEFI data following the introduction of a new National Immunisation Program (NIP) schedule, which commenced on 1 November 2005. From that date, varicella vaccine was introduced for children at 18 months of age and inactivated poliovirus vaccines (IPV) replaced oral poliovirus vaccine (OPV) for children at 2, 4 and 6 months and 4 years of age. All children in Australia receive IPV in combined vaccines containing diphtheria-tetanus-acellular pertussis (DTPa) antigens (i.e DTPa-IPV; quadrivalent). In some states and territories, combined vaccines also include hepatitis B (HepB) (i.e. DTPa-IPV-HepB; pentavalent) or both HepB and *Haemophilus* influenzae type b (Hib) (i.e. DTPa-IPV-HepB-Hib; hexavalent).[4] As a result of these changes, DTPa and DTPa-HepB vaccines are no longer included in the NIP schedule.

Average annual population-based AEFI reporting rates were calculated using mid-2005 population estimates. Reporting rates per 100,000 doses were calculated for vaccines that are funded by the NIP using denominator data from the Australian Childhood Immunisation Register (ACIR).

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The data reported here are provisional only. It is important to note that an AEFI is defined as a medical event that is temporally associated with immunisation but not necessarily causally associated with immunisation. Readers are referred to previous reports for a description of the national AEFI passive surveillance system[1] methods used to analyse the data[1–3] and information regarding limitations and interpretation of the data.[2] Often several vaccines and reaction codes are listed in an AEFI record so the number of both vaccines and reaction codes will exceed the total number of AEFI records. When several vaccines are administered at the same time, the reported AEFI is usually assigned a causality rating of 'possible' unless the AEFI is an injection site reaction and the vaccine name and site are known. In that situation, a causality rating of 'certain' will be assigned to the report.

1 January to 30 June 2006

There were a total of 240 AEFI records (28.9 per 100,000 population) for children aged <7 years for vaccines administered in the first six months of 2006. This was a nine per cent decrease on the 265 records (29.7 per 100,000 population) for the corresponding six month period in 2005. Thirty-three per cent (n=79) of records were for children aged <1 year, 14 per cent (n=33) for children aged 1 to < 2 years and 53 per cent (n=128) for children aged 2 to <7 years, similar to 2005. The male to female ratio was 1.2:1.

Of the 240 records analysed, 32 (13%) had outcomes defined as 'serious' (i.e. recovery with sequelae, hospitalisation, life-threatening event or death), and was higher than previously reported for children aged <7 years (average of 8% for 2000–2005; range 7–13%). Serious or potentially life-threatening AEFIs reported included apnoea or respiratory depression (n=11), bradycardia (n=10), seizure (n=7) and hypotonic-hyporesponsive episode (HHE; n=10). There were no reports of anaphylaxis or death. The most common reaction categories were injection site reaction (n=140; 58%), fever (n=38; 16%) and allergic reaction (n=31; 13%).

One or more of the vaccines on the current NIP schedule, shown in the Table, was recorded as suspected of involvement in the reported adverse event for 232 of the 240 records analysed. Eight records listed only non-NIP vaccines as suspected of involvement in the reported AEFI. The nine vaccines listed in these AEFI records were influenza (n=5), BCG (n=1), hepatitis A (n=1), combined hepatitis A and B (n=1) and pneumococcal polysaccharide (n=1) vaccines.

The AEFI reporting rates per 100,000 vaccine doses recorded on the ACIR were similar to those in 2005 for most of the vaccines that have been included in the NIP schedule for some time, including meningococcal C conjugate vaccine (MenCCV), seven-valent pneumococcal conjugate vaccine (7vPCV) and Hib vaccine (Table). The apparent increase in the reporting rate for Hib-HepB vaccine may be related to reporting of AEFIs for the newer quadrivalent DTP-IPV vaccine among children aged <1 year as the two vaccines are both given at 2 and 4 months of age.[4]

Reporting rates for the different DTPa-IPV combination vaccines varied by vaccine type (Table). The reporting rate for pentavalent vaccine is likely to be inaccurate due to the small number of reports and some under-reporting to the ACIR of doses administered. The reporting rate for quadrivalent DTPa-IPV includes reports for children aged <1 year who were scheduled to receive the vaccine at 2, 4, and 6 months of age (reporting rate of 20.3 per 100,000 doses) and reports for children aged 4 years (reporting rate of 88 per 100,000 doses).

While the number of AEFI reports for children aged <1 year and 2 to <7 years was similar in 2006 and 2005, AEFI reporting rates per 100,000 vaccine doses increased for children in these two age groups (Table). The reporting rate for AEFIs defined as serious also increased from 0.6 in 2005 to 1.8 in 2006. Reasons for these changes are discussed below and relate mainly to a reduction in the denominator following the introduction of multivalent vaccines in November 2005.

DTPa-IPV combination vaccines

There were a total of 69 AEFI records for children aged <1 year where a DTPa-IPV quadrivalent, pentavalent or hexavalent combination vaccine was listed as suspected of involvement in the reported AEFI and the vaccine was administered during January to June 2006 (hexavalent = 32; pentavalent = 4; quadrivalent = 33). The overall reporting rate for these three vaccines was 18.9 AEFI records per 100,000 doses for children aged <1 year. This is slightly higher than the reporting rates in 2005 for DTPa and DTPa-HepB vaccines among children aged <1 year in 2005 (12.7 and 14.0 per 100,000 doses, respectively).

Of the 69 records, DTPa-IPV combination vaccines were listed as the only suspected vaccine for 9 (13%) while 62 (90%) had causality ratings of 'possible' and 20 (29%) records listed outcomes defined as 'serious'. The most frequently reported adverse events

Table. Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* children aged less than 7 years, ADRAC database, January to June 2006

Vaccine†	AEFI records* (n)	Vaccine doses‡ (n)	Reporting rate per 100,000 doses§	
			Jan–June 2006	Jan–June 2005
Haemophilus influenzae type b	10	51,768	19.3	18.1
Haemophilus influenzae type b-hepatitis B	45	203,482	22.1	15.4
Measles-mumps-rubella	54	253,134	21.3	23.2
Meningococcal C conjugate	23	138,626	16.6	17.9
Pneumococcal conjugate	62	390,975	15.9	15.6
Diphtheria-tetanus-pertussis-IPV	126	311,608	46.9	–
Pentavalent (DTPa-IPV-HepB)	4	8,643	46.3	–
Hexavalent (DTPa-IPV-HepB-Hib)	32	174,596	18.3	–
Varicella	15	109,093	13.7	–
Age group				
<1 year	76	907,593	8.4	6.4
1 to <2 years	32	444,313	7.2	7.5
2 to <7 years	124	290,019	42.8	26.3
AEFI category†				
Total	232	1,641,925	14.1	10.5
'Certain' or 'probable' causality rating	100	1,641,925	6.1	4.6
'Serious' outcome	29	1,641,925	1.8	0.6

* Number of AEFI records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 30 June 2006. More than one vaccine may be coded as 'suspected' if several were administered at the same time.

† Records where at least one of the nine vaccines shown in the table was suspected of involvement in the reported adverse event. AEFI category includes all records (i.e. total), those assigned 'certain' or 'probable' causality ratings, and those with outcomes defined as 'serious'. Causality ratings were assigned using the criteria described previously.[1,2] A 'serious' outcome is defined as recovery with sequelae, hospitalisation, life-threatening event or death.[1,2]

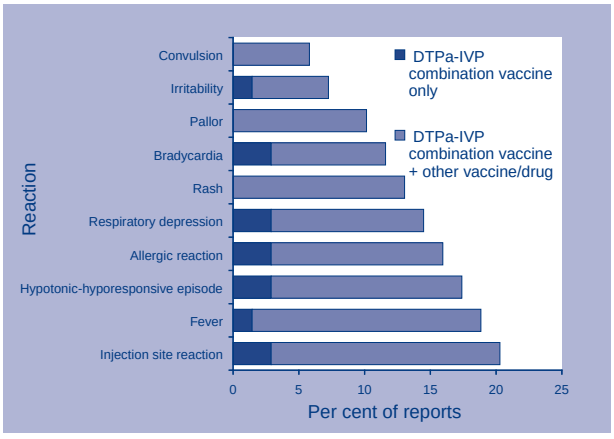
‡ Number of vaccine doses recorded on the Australian Childhood Immunisation Register (ACIR) and administered between 1 January and 30 June 2006.

§ The estimated AEFI reporting rate per 100,000 vaccine doses recorded on the ACIR.

following DTPa-IPV combination vaccines, administered alone or with other vaccines, were injection site reaction (n=14), fever (n=13) and allergic reaction (n=11) (Figure 1). More severe AEFIs included apnoea/respiratory depression (n=8), bradycardia (n=10), convulsion (n=4) and HHE (n=10). Reporting rates for these AEFIs were similar for both quadrivalent and hexavalent vaccines, except for HHE (4.8 per 100,000 doses of quadrivalent vaccine *cf* 0.6 per 100,000 doses of hexavalent vaccine, including reports where other vaccines were co-administered with either quadrivalent or hexavalent vaccine).

For children aged 2 to <7 years, there were 107 AEFI records that listed quadrivalent DTPa-IPV as a suspected vaccine (88 records per 100,000 doses). Of these, 73 records (68%) listed the vaccine as the only suspected vaccine while 67 (63%) had causality ratings of 'certain' or 'probable' and 7 (7%) listed outcomes defined as serious. Ninety-per cent

Figure 1. Frequently reported adverse events following immunisation with DTPa-IPV quadrivalent, pentavalent and hexavalent combination vaccines, children aged <1 year, ADRAC database, 1 January to 30 June 2006 (n=69 records)

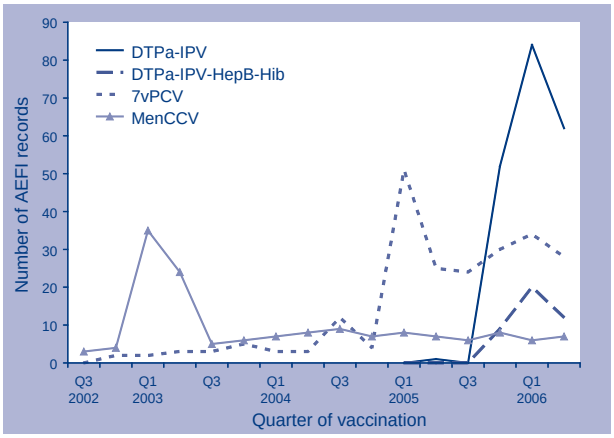


(n=97) of records listed injection site reaction. This is a reporting rate of 80 injection site reactions per 100,000 doses, the same as seen over the past three years for children receiving a fifth dose of a DTPa-containing vaccine.[3]

Discussion

The total number of AEFI records and population-based reporting rates were slightly lower (by 9%) for January to June 2006 compared with the same period in 2005. The increase in summary and age group reporting rates per 100,000 vaccine doses (Table) is largely related to two factors. First, there has been a reduction in the total number of vaccine doses administered to children aged <1 year and 2 to <7 years following removal of OPV from the NIP schedule and the introduction of IPV-containing multi-antigen vaccines from 1 November 2005. This reduced the total number of doses administered by approximately one third (i.e. approximately 800,000 doses) compared with the first six months of 2005, while there has been little change in the number of AEFI reports to ADRAC. Second, there appears to have been increased reporting by providers following the introduction of new vaccines into the NIP in November 2005, as has been observed following the introduction of other vaccines in recent years (Figure 2). It is expected that reporting of AEFIs for DTPa-IPV combination vaccines will decline from the peaks seen in the first quarter of 2006 and stabilise over time, as has occurred with both MenCCV and 7vPCV.

Figure 2. Reports of adverse events following immunisation, ADRAC database, 1 July 2002 to 30 June 2006, for vaccines recently introduced into the National Immunisation Program*



* Meningococcal C conjugate vaccine (MenCCV) was introduced into the NIP on 1 January 2003, 7-valent pneumococcal conjugate vaccine (7vPCV) on 1 January 2005, and both DTPa-IPV and DTPa-IPV-HepB-Hib combination vaccines on 1 November 2005.

The largest change in this reporting period has been the increase in the proportion of AEFI records with outcomes defined as serious. This increased from seven per cent of records in 2005 (a period following the introduction of 7vPCV) to 13 per cent for the first six months of 2006 after the introduction of the DTPa-IPV combination vaccines. Serious AEFIs reported for these vaccines were predominantly in children aged <1 year and included HHE, apnoea or respiratory depression and bradycardia. Importantly, there were no reports of anaphylaxis or death. The total number of serious reports is low and may represent increased awareness among providers following published reports from Germany that suggested an increased risk of sudden unexpected death in children aged <2 years after receipt of a hexavalent vaccine marketed in Germany.[5,6] It is important to note that (i) a large case-control epidemiological study conducted in Germany found no link between the use of hexavalent vaccines and sudden unexpected death in children;[7] (ii) the Global Advisory Committee on Vaccine Safety, convened by the World Health Organization, concluded that hexavalent vaccines are safe[8] and (iii) the specific vaccine in question is not used in Australia.

Observed differences in reporting rates of HHE following receipt of quadrivalent DTPa-IPV compared with hexavalent vaccine may be due to different AEFI surveillance practices in the states where the quadrivalent vaccine is used (i.e. South Australia, Victoria and Queensland) compared with other jurisdictions.[2]

A number of the reports to ADRAC of apnoea and bradycardia following quadrivalent and hexavalent vaccines occurred in very pre-term infants who received vaccines at eight weeks of age whilst in a hospital setting, as recommended in the *Australian Immunisation Handbook*.[9] Cardio-respiratory events including apnoea and bradycardia are known AEFIs in hospitalised pre-term infants and are managed appropriately in a hospital setting.[10,11] The benefits of immunisation in pre-term infants continues to outweigh the small risk of these manageable adverse events.

Conclusion

This report further demonstrates that changes to the NIP are reflected in the national passive AEFI surveillance data.[2,3] Changes in AEFI reporting rates for vaccines administered in the first six months of 2006, compared with same period for 2005, correspond in time with the replacement of OPV and DTPa vaccines with DTPa-IPV combination vaccines from 1 November 2005. Although serious AEFIs increased as a proportion of total reports compared with the previous reporting period, the majority of AEFIs reported to ADRAC were mild transient

events. Close monitoring of passive AEFI surveillance data for DTPa-IPV combination and other vaccines administered to children continues through ADRAC, the Therapeutic Goods Administration and state and territory health departments.

Acknowledgements

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An outbreak of *Salmonella* Typhimurium phage type 64 gastroenteritis linked to catered luncheons in Adelaide, South Australia, June 2005

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Abstract

Salmonella sp. **are important causes of foodborne illness, with restaurants and catered functions being commonly reported settings for outbreaks. In June 2005 an investigation commenced following reports of gastrointestinal illness in attendees at luncheons catered by an Adelaide café, as well as persons eating at the café itself. The investigation sought to determine the existence of an outbreak, identify a source and method of transmission and implement public health measures to prevent further cases. Lists of luncheon attendees were obtained from function organisers. A retrospective cohort study was commenced using a structured questionnaire developed from the café's menu listings. A suspected case was defined as a person developing two or more gastrointestinal symptoms after attending a luncheon catered by the café. A case series investigation was used for café diners. Of the 102 respondents, 61 (60%) met the case definition with 32 subsequently confirmed as *Salmonella* Typhimurium phage type 64 (STM 64) infections. Of the 61 cases, 59 (96%) reported eating a bread roll. STM 64 was detected in raw defrosted chicken recovered from the café's kitchen. This suggested cross-contamination from the chicken to one or more ingredients common to the bread rolls was the route of infection. To prevent further cases, perishable goods were discarded, the café was closed, the premises cleaned, then restrictions were placed on the types of foods served. This investigation's findings highlight the importance of safe food handling and hand hygiene in commercial food preparation.** ***Commun Dis Intell*** **2006;30:443–448.**

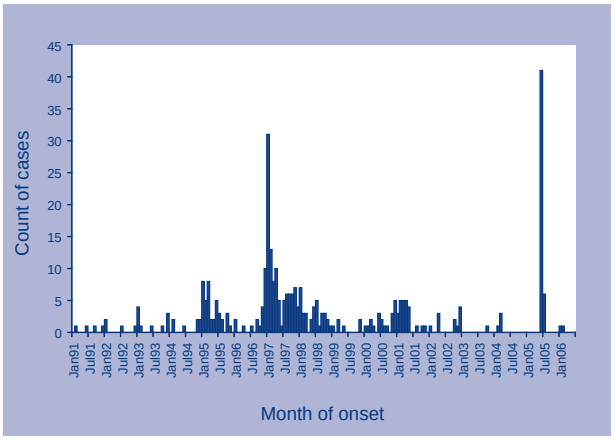
Keywords: Salmonella *Typhimurium, outbreaks, food handling, cross contamination, chicken*

Introduction

Since the commencement of national surveillance, *Salmonella* Typhimurium phage type 64 (STM64) has been presePnt in South Australia at a low endemic level (Figure 1). South Australia and other states began to show increased notifications of this phage type during the mid-1990s with annual reporting in Australia peaking at 374 cases in 1997.[1] Notifications exceeding 50 cases per year continued in some states until 2002 but since that time this phage type has largely disappeared from most jurisdictions, with the exception of South Australia.

Salmonellae are a frequent cause of foodborne illness, with restaurants and catered functions often reported as the settings for outbreaks.[2] The few

Figure 1. Number of notified cases of *Salmonella* Typhimurium phage type 64, South Australia, 1 January 1991 to 30 June 2006, by month of onset



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reports of outbreaks of STM64 infections in Australia include an investigation in 1997 linked to a restaurant in South Australia[3] where 16 of 19 symptomatic diners were diagnosed with STM64 infection. A number of food items, including chicken, tested positive for STM64. The investigation concluded that fried ice-cream was the cause of the illness, with the chicken the likely source of the contaminant. In the same year a joint study involving the Victorian Department of Human Services and the South Australian Health Commission[4] was also undertaken using hypothesis generating questionnaires. The descriptive epidemiology and subsequent case control analysis revealed no food items with a significant odds ratio.

The most recent outbreak was reported on 30 June 2005 when the Communicable Disease Control Branch (CDCB) in Adelaide was advised by a senior hospital clinician that a number of staff members who had attended a lunchtime meeting on 27 June had developed a gastrointestinal illness. Food for the meeting had been prepared by an Adelaide café. The following day further reports were received from local government and a university, advising of illness in members of the public, staff and students who had eaten at the café or attended other luncheons catered for by the same café. Additional cases were also reported via routine notification and when interviewed were found to have eaten at the implicated café.

Methods

Epidemiological investigation

The investigators sought to confirm the existence of an outbreak linked to the café, prevent further cases and to determine the source and method of transmission of the infection. The café owner provided details of catered events for the period 23 June to 4 July including contact details for organisers, number of attendees, menus and descriptions of food served. Luncheon organisers were contacted and asked to supply contact details for attendees. A retrospective cohort study was commenced using a food and illness questionnaire specific to the catered luncheons. This was administered via telephone, direct post or email. People who reported becoming ill after eating at the café itself (community cases) were not included in the cohort study because of their potentially different food exposures. Community case details were collected using standard hypothesis generating food and illness questionnaires. Descriptive analysis of the community case series was performed using Microsoft Excel.

Cohort study questionnaires were entered into Epi Info Version 6 before transfer to Microsoft Excel for descriptive analysis. Stata Version 8.2 was used for the analytical epidemiology. Univariate analysis involved the calculation of relative risks, in conjunction with a two-tailed Fischer's exact test and 95 per cent confidence intervals (CI).

A case was defined as either a person diagnosed with STM64 or a person developing two or more gastrointestinal symptoms after eating at the café or eating at luncheons, held between 27 and 30 June, where food had been supplied by the café.

Environmental health investigation

Café investigation

The first of several inspections of the café was conducted by Environmental Health Officers from the local City Council and the Environmental Health Branch of the Department of Health on 1 July 2005. The kitchen was assessed for appropriate hygiene and sanitation standards, cooking practices were observed and enquiries were made about the supply and storage of foods. A number of food samples and environmental swabs were taken. To conclude the investigation, experimental bread rolls were prepared for microbiological testing to confirm that the clean-up and disposal of foods by the café was effective in eliminating *Salmonella* from bread products.

It was found that the descriptions of bread rolls provided at the catered functions did not accurately match what some attendees had reported eating. This occurred because café staff would use different combinations of ingredients in order to provide greater variety at catered events. As a result meals eaten by community cases were examined in considerable detail as it was felt that food ordered from a set menu would be more true to description. A chicken caesar roll was identified and environmental health staff thoroughly investigated its preparation to identify ingredients that may have been contaminated or steps in the process where cross-contamination could have occurred.

Chicken wholesaler and chicken producer

The South Australian Department of Primary Industries and Resources (PIRSA) provided assistance to determine the source of chicken used in the café and to help organise the collection of any further microbiological evidence.

Laboratory investigations

Phage typing of both human and non-human *Salmonella* isolates was performed by the Australian Salmonella Reference Centre at the Institute of Medical and Veterinary Science (IMVS). In addition, a range of food and environmental samples were also submitted for testing to the Food and Environmental Laboratory at the IMVS for standard

plate counts and *Salmonella* culturing in addition to examination for organisms such as *Escherichia coli* and *Bacillus cereus*. A number of experimental bread rolls with assorted fillings were also made up and submitted for testing.

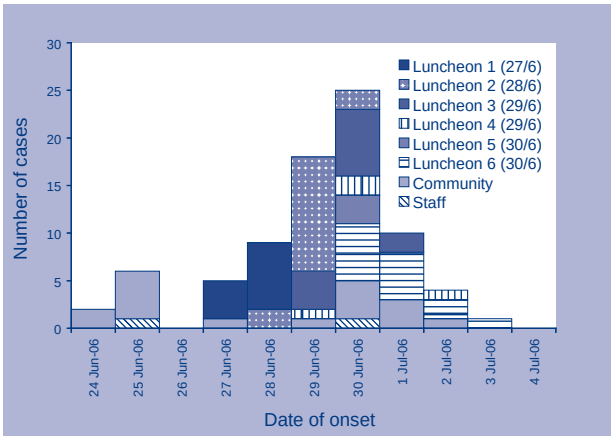
Results

The investigation comprised a cohort study of attendees at functions catered by the café and investigation of people who had eaten at the café (community cases), including ill staff members.

Epidemiological investigation – cohort study

A total of 119 persons were identified as probable attendees at one of six catered luncheons. Following questionnaire administration (either via direct interview, email or regular post) 102 responses were received (86%) from 49 female and 53 male respondents. The average age of respondents (n=99) was 38 years (range 21–63 years). Included among the 102 respondents to the questionnaire were 61 cases of gastroenteritis, providing an overall attack rate of 60 per cent. Among these cases 32 persons were confirmed as STM64 positive. Dates of onset for cases ranged from 26 June to 3 July. The epidemic curve (Figure 2) shows the distribution of cases by specific function in addition to community cases and staff.

Figure 2. Epidemic curve showing community and cohort (catered lunch) identified cases, by date of onset and individual luncheon



Symptoms included diarrhoea (97%), abdominal pain (97%), fever (92%), chills (90%), headache (49%), vomiting (48%), myalgia (20%) and nausea (8%). Four cases reported bloody diarrhoea. Where data were complete (n=55) incubation periods ranged from 2 hours to 81 hours, with a mean of approximately 20.5 hours (median: 16 hours). At the time that questionnaires were completed 64 per cent of cases (n=39) reported still being unwell. For the 19 cases with resolved symptoms, the median self-reported duration of illness was four days (range 2–10 days). Four cases provided no details on resolution of their symptoms.

A variety of food platters in different combinations were served at the six luncheons. Bread rolls (with assorted fillings) and fruit platters were common to all six functions. Cheeses, hot finger foods and Mediterranean style platters were also supplied by the café to some of the luncheons. Analysis of these foods showed 79 per cent of respondents (n=81) reported eating a bread roll (see Table). Fifty-nine of these persons became ill, giving a food-specific attack rate for bread rolls of 73 per cent. Eating a bread roll with any meat or fish filling showed a low but significant association with becoming ill (RR 1.75, 95% CI 1.09, 2.81 p=<0.01). Ham (RR 1.63, 95% CI 1.15, 2.30 p=0.01), smoked salmon (RR 1.57 95% CI 1.19, 2.07 p=0.02) and tuna filled rolls (RR 1.57, 95% CI 1.17, 2.11 p=0.04) all showed a low but statistically significant association with their consumption and illness. For other foods served at the luncheons no association with illness was shown.

Community cases

The median age of community cases was 25 years (range 18–59 years) and included 13 females and six males. Fourteen of the 19 community cases were positive for STM64. This included two staff members, neither of whom was involved in food preparation while they were ill. Dates of onset ranged from 24 June to 1 July, with symptom prevalence for those supplying illness details being: diarrhoea (100%), abdominal pain (94%), fever (82%), vomiting (71%), nausea (53%) and bloody diarrhoea (12%). The median incubation period was 16 hours (range 4–36 hours). At the time food history questionnaires were completed, 10 cases reported resolution of symptoms, with the duration of illness between 6–14 days.

Environmental findings

Café investigation

Prior to the arrival of investigators the café kitchen had been cleaned and all previously prepared food items and most other perishable goods had been discarded. As a consequence of this, investigators were restricted in their ability to hypothesise and test potential means of cross contamination. During the initial inspection on 1 July enquiries were made about catering arrangements and supply of food, including sources of chicken, perishable goods such as fruit and vegetables and other packaged foods used by the café. General hygiene and food handling practices were considered to be good. Remaining food items regarded as being potentially hazardous

Table 1. Food specific attack rates and relative risks for function attendees who reported eating and not eating selected food items

Food item	Persons who ate item			Persons who did not eat item			Relative risk	95% CI	*p* value
	Number ill	Total number	Attack rate (%)	Number ill	Total number	Attack rate (%)			
Any filled roll	59	81	73	0	11	–	–	–	–
Any meat filled roll	43	58	74	11	15	73	1.75	1.09, 2.81	<0.01
Chicken roll	18	23	78	28	52	54	1.45	1.04, 2.02	0.07
Ham roll	18	21	86	20	38	53	1.63	1.15, 2.30	0.01
Bacon roll	5	6	83	33	54	61	1.36	0.90, 2.07	0.40
Bacon & egg roll*	5	5	100	29	51	57	1.76	1.38, 2.23	–
Salami roll	6	8	75	33	53	62	1.20	0.76, 1.89	0.70
Beef roll	4	5	80	34	55	62	1.29	0.80, 2.10	0.64
Turkey roll*	4	4	100	41	68	60	1.66	1.37, 2.01	–
Smoked salmon roll	15	17	88	36	64	56	1.57	1.19, 2.07	0.02
Tuna roll	11	12	92	28	68	41	1.57	1.17, 2.11	0.04
Any vegetable filled roll	9	17	53	30	42	71	0.74	0.45, 1.21	0.23
Salad roll	6	9	67	33	49	67	0.99	0.60, 1.63	1.00
Roasted vegetable roll	7	16	44	41	61	67	0.65	0.36, 1.17	0.15
Mediterranean vegetable roll	2	4	50	34	64	53	0.79	0.29, 2.16	0.63
Egg roll*	5	5	100	35	57	61	1.62	1.33, 2.00	–

* *P* value not reported due to small numbers of subjects reporting these exposures.

were shown to be under temperature control following testing. The storage of raw food was closely examined, in particular the separation of chicken from other raw or ready to eat foods in the cold room. There were no identified safety issues with raw chicken, and all other foods were adequately covered and protected from contamination in the cold room, freezers and dry storage.

Hand washing and toilet facilities were clean and appropriately fitted. Previous routine inspections by local government had not identified any other concerns relating to kitchen hygiene, sanitation or food handling practices. Cleaning and sanitising of utensils, equipment and work surfaces was very good. The kitchen used colour coded cutting boards with staff displaying a good understanding of washing and sanitising practices.

Staff demonstrating preparation of chicken for cooking were seen to handle raw chicken meat immediately prior to seasoning it with pinches of salt and pepper kept in takeaway containers on the preparation bench. The same salt and pepper was then used for seasoning bread rolls as well as being used as an ingredient in guacamole. The guacamole was stored in plastic containers that sat on a kitchen bench during roll preparation and peak trading. The same knife used to spread guacamole onto bread rolls was also reported by the café as being used to spread butter onto hamburger buns, with hamburgers being identified as a food item eaten by a number of the community cases. It was also found that some of the bread rolls served at the luncheons did not match the descriptions of what was supplied for that particular event.

Laboratory findings

STM64 positive faecal specimens were obtained from 32 cohort and 14 community cases. STM64 was also cultured from a 25 g sample of raw defrosted chicken tenderloin obtained at the time of the first kitchen inspection. *Campylobacter jejuni* was also present in this specimen and in a faecal specimen provided by a staff member with a dual infection. An experimental bread roll filled with turkey, lettuce, cucumber, cheese, sprouts, cranberry sauce and dressing was positive for STM64. All other food and environmental samples were negative for STM64, although low levels of *Bacillus cereus* and *E. coli* were detected in samples of fried rice and cooked beef.

Chicken wholesaler and chicken producer

As the chicken meat sample was positive for STM64 the decision was made to undertake a trace back of chicken to identify contamination along the food chain. An Adelaide poultry wholesaler supplied the café with fresh chicken which was ordered as required. The wholesaler reported supplying the café with breast fillets while the café stated they received both breast and thigh fillets (tenderloins). The chicken was usually purchased in 5 kg lots, separated at the café and then frozen. It would then be removed from the freezer and allowed to thaw in a cool room prior to cooking. The local wholesaler sourced their chicken from an interstate processing facility. At the request of PIRSA the processor undertook a review of quality assurance data with no evidence of this phage type being found in the plant. PIRSA then sought assistance from the interstate producer to undertake drag swabbing of sheds from the farm where the chickens were raised.

Public health action

Public health action was taken to eliminate potential sources of infection and to reduce potential transmission of foodborne pathogens. Before inspection of the café by health department and local government investigators the café was cleaned with any remaining prepared food items and most perishable goods being discarded. The café voluntarily decided to close for additional cleaning and did not trade over the weekend. Restrictions were then placed on the types of food served. No sandwich items were permitted to be made and rolls for upcoming catered functions were brought in ready-made from an independent supplier. Only hot meals such as pasta dishes, schnitzels and fish and chips, in addition to plain salads, were to be prepared and served on the premises. Staff members were instructed on safe food handling practices and other issues relating to food safety by environmental health officers. In order to recommence catering and sandwich making the café was required to make up an assortment of menu-listed bread rolls for additional laboratory testing. Following the positive laboratory finding in the initial batch of experimental rolls the café was required to submit another two batches of rolls for testing. After both batches had tested negative for *Salmonella,* the café was permitted to recommence catering and making sandwiches and rolls. Traceback of chicken to suppliers, processors and farms was instituted, with the aim of identifying a primary source of contamination.

Discussion

The high food-specific attack rate associated with eating bread rolls, food items common to all functions, coupled with the low relative risks for individual roll types, suggests contamination of an ingredient common to many of the rolls served at both the luncheons and the café itself. The STM64 infections were likely acquired from eating bread rolls cross-contaminated from raw chicken, possibly via black pepper or guacamole.

Although the exact mechanism of cross-contamination in this outbreak may only be surmised, environmental health staff did identify a possible mechanism whereby *Salmonella* on raw chicken could have cross-contaminated a ready-to-eat food, reinforcing the need for restaurants and caterers to both comprehend and to comply with the requirements of the Australia New Zealand Food Standards Code.[5]

Although salmonellae will not grow in food with low water activity (a_w), they are able to survive for long periods in low a_w foods such as black pepper, gelatine and chocolate.[6] While the link between guacamole and chicken is less direct than that of the pepper it remains plausible. Guacamole could certainly act as a growth medium and sat in tubs on the kitchen bench at an ambient temperature during roll preparation and peak trading. This may have assisted the growth of any *Salmonella* added to the guacamole via contaminated black pepper.

A similar mechanism could explain detection of STM64 in the test bread roll. While enumeration of the salmonellae was not reported the numbers of organisms in the 25 g sample was thought to have been low, as following detection of the organism the remainder of the roll was divided and its individual ingredients tested separately, with no *Salmonella* detected.

The rapid onset of symptoms and duration of illness is suggestive of the ingestion of large numbers of *Salmonella*. This could not be confirmed as, with the exception of the positive chicken sample recovered from the cafe freezer, most perishable ingredients used in the rolls had been discarded prior to investigators inspecting the café. Furthermore, no specimens were recovered from any of the luncheons. Investigators did not collect data on number of bread rolls consumed by cases and thus no proxy measure of dose based on a relationship between incubation period and number of bread rolls eaten could be calculated.

Limitations

The respondent's memory and reporting of exposures may potentially lead to non-differential misclassification in this study. This issue is further clouded by the generic descriptions of bread rolls not matching what had been served at the luncheons. This could not be controlled in the cohort study and made it difficult to ascribe any epidemiological association

with contamination of a specific bread roll or bread roll component. However, if an ingredient common to many of the bread rolls had been contaminated as suggested, such bias would have had little effect on the conclusion of the investigation, apart from generating relative risks for individual rolls that are closer to the null than the actual effect.

This study has highlighted the importance of understanding and adhering to good food handling techniques in commercial kitchen and catering settings. Although the exact mechanism of contamination remains unknown our findings would indicate a breakdown in safe food handling and preparation as the likely cause. It was unfortunate that samples from the implicated farms where not obtained. The involvement of interstate health authorities may have helped in this matter.

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Change of address

Due to internal staff movements, the mailing address for Communicable Diseases Intelligence has changed.

Please note that correspondence should now be sent to:

Communicable Diseases Intelligence
Office of Health Protection
Australian Government Department of Health and Ageing
MDP 6
GPO Box 4898
CANBERRA ACT 2601

The facsimile number remains the same at: (02) 6289 7100

A multi-jurisdiction outbreak of *Salmonella* Typhimurium phage type 135 associated with purchasing chicken meat from a supermarket chain

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Abstract

A multi-jurisdiction case control study was conducted after an increase of *Salmonella* Typhimurium phage type 135 notifications (a local designated subgroup) was observed throughout Australia. Hypothesis generating interviews conducted in three jurisdictions identified consumption of chicken, eggs, beef and bagged carrots as common among cases and that a high proportion of cases (>80%) reported purchasing their groceries from a particular supermarket chain (Supermarket A). We conducted a case control study to test whether *S.* Typhimurium 135 infections were associated with these food items and the purchasing of these products from Supermarket A. The study comprised 61 cases and 173 controls. Cases were younger than controls (p=0.003) and their distribution by jurisdiction was also significantly different (p<0.001). In multivariate analysis, cases had significantly higher odds of having eaten chicken purchased from Supermarket A (OR=3.2, 95% CI 1.2,9.0) or having eaten chicken from a fast food outlet (OR=2.8, 95% CI 1.0,7.7) compared to controls. Two positive *S.* Typhimurium 135 results were obtained through a chicken sampling survey conducted at four Supermarket A stores in Victoria. The results of this study were presented to industry and retail representatives, which facilitated better communication between these groups. *Commun Dis Intell* 2006;30:449–455.

Keywords: Salmonella *Typhimurium, outbreak, chicken, case-control study*

Introduction

In September 2005, Tasmania, Victoria and New South Wales reported increased notifications of a locally designated strain of *S.* Typhimurium 135 (*S.* Typhimurium 135a). Nationally, the number of notifications in November 2005 was six times higher than in November 2004 (unpublished data: National Notifiable Diseases Surveillance System, Australian Government Department of Health and Ageing). In the period October to December 2005, Tasmania investigated four point source outbreaks of *S.* Typhimurium 135a associated with egg-based dishes prepared by bakeries, restaurants and caterers. These eggs were all sourced from the same egg farm. However, Tasmania also observed an increase in sporadic cases of *S.* Typhimurium 135a that were not associated with a defined point source outbreak. In addition, South Australia, New South Wales, Queensland and the Australian Capital Territory also received notifications during this time, although not above expected levels.

Previously in Australia, *S.* Typhimurium 135 and *S.* Typhimurium 135a had been associated with chicken,[1,2] raw egg products,[3–6] bakery products,[7]

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pork-filled bread rolls from Asian bakeries,[8–10] and a commercial fruit juice.[11] *S.* Typhimurium 135a is a sub-type of *S.* Typhimurium 135 that is used in Australia to more confidently identify epidemiological links between cases, but is not recognised internationally. Initially, the increase in cases was observed for *S.* Typhimurium 135, yet after further typing the focus became *S.* Typhimurium 135a. The National Enteric Pathogen Surveillance System, a voluntary system which collects data on human and non-human enteric pathogens in Australia,[12] reported isolating *S.* Typhimurium 135a from chicken meat samples from New South Wales and Victoria in October 2005. The Australian *Salmonella* Reference Centre also reported *S*. Typhimurium 135a in chicken meat samples from New South Wales (n=20), Queensland (n=19) and South Australia (n=10) in September 2005.[13]

Hypothesis generating interviews with people infected with *S.* Typhimurium 135a were conducted independently in Victoria, Tasmania and New South Wales to identify risk factors for illness. The food exposures most commonly reported by cases were chicken, eggs, beef and bagged carrots. A high proportion of cases (>80%) also reported purchasing their groceries from a particular supermarket chain (Supermarket A). As a result, OzFoodNet, a collaborative initiative of Australia's state and territory health authorities that aims to investigate and understand foodborne disease at a national level, coordinated a multi-state investigation into *S.* Typhimurium 135a infections. Given the similarities in food frequencies for a concurrent investigation of *S.* Typhimurium 44 cases, both phage types were included in the case control study.

The aim of this study was to determine whether there was an association between infection with *S.* Typhimurium 135a and *S.* Typhimurium 44 and the food products identified, in order to prevent further infections and inform future food safety measures. In this paper we report on the *S.* Typhimurium 135a component of the case control study.

Methods

Epidemiological investigation

We conducted a case control study to test whether *S.* Typhimurium 135a infections were associated with eggs, chicken, chicken products, bagged carrots and minced beef, and the purchasing of these products from Supermarket A. The investigation was conducted in the context of a public health intervention as per state and territory legislation. Consent was obtained from participants after being advised that participation in the study was voluntary and responses were confidential.

Study population

A case was defined as a resident of New South Wales, the Australian Capital Territory, South Australia, Victoria or Tasmania who had *S.* Typhimurium 135a isolated from a faecal specimen and notified to the respective jurisdiction between 2 November and 23 December 2005. Queensland also reported cases, but due to the notification rate not exceeding the expected threshold and competing demands, did not recruit cases into this study. Cases were excluded if they were co-infected with another enteric pathogen, returned from overseas travel within four days of onset of illness, were not the primary case in the household or were not interviewed within 40 days of specimen collection. Cases were also excluded if they had been part of the point-source outbreak investigations in Tasmania.

To identify controls we used progressive digit dialling, whereby phone numbers either side of each case's phone number were telephoned sequentially until an eligible household was contacted. The person in the household with the next birthday was invited to participate in the study. Attempts were made to recruit two controls for every case. Controls were excluded if they had returned from overseas travel in the previous four days or if a household member had diarrhoea in the previous two weeks. To compare another method of control selection, South Australia recruited an additional two controls per case using their control bank, a list of people that had participated in a previous health study that were willing to be contacted again.

Data collection

Cases and controls were interviewed over the telephone using a tailored questionnaire developed by OzFoodNet epidemiologists. Up to six attempts were made to contact each case and control phone numbers were called six times before a new number was attempted. Interviews were conducted on weekdays between 12 and 23 December, during the day and in the evenings. The interview included questions about food items consumed in the four days prior to onset of illness for cases or in the four days prior to interview for controls. Each jurisdiction was responsible for interviewing cases that resided in their jurisdiction and two controls per case. Completed interview data were entered by each jurisdiction onto a national NetEpi Case Manager (New South Wales Health) database, a web-based reporting system for which data entry can be conducted at multiple sites. OzFoodNet Central was responsible for maintaining the NetEpi system and downloading the data for analysis. No identifying information was entered onto the NetEpi system.

Data analysis

We obtained data from the National Notifiable Diseases Surveillance System to review the descriptive epidemiology of all cases of *S.* Typhimurium 135a. Statistical analysis was conducted using Microsoft Excel and Intercooled STATA version 8.[14] Demographics were compared using Pearson's chi-squared test for trend. Food exposures were compared using odds ratios with 95 per cent confidence intervals. Controls selected for both *S.* Typhimurium 135a and *S.* Typhimurium 44 were included in the analysis. A maximum-likelihood logistic regression model was constructed to analyse the association between chicken consumption and illness. This was adjusted for age and state of residence as well as those food items significantly associated with cases at the univariate level ($p<0.05$).

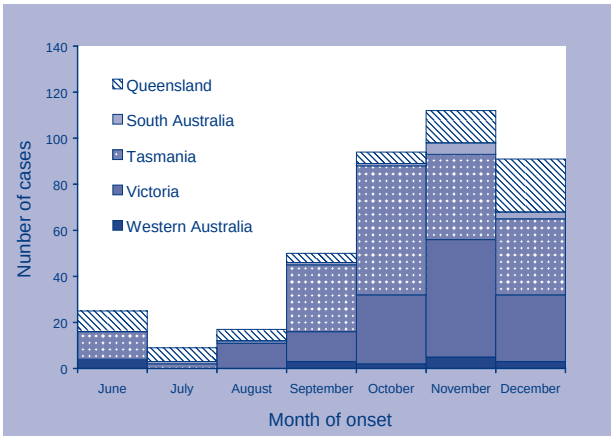
Environmental investigation

The Victorian Communicable Disease Control Unit conducted a food sampling survey, whereby chicken samples were obtained from four Supermarket A stores where cases reported purchasing chicken. Local government environmental health officers were requested to purchase chicken wings, a whole chicken and chicken breasts from the fresh pre-packaged section of the supermarket. These were then sent to the Microbiological Diagnostic Unit for *Salmonella* testing using standard methods of analysis.

Results

The epidemic curve (Figure 1) shows the increase in notifications of *S.* Typhimurium 135a from September 2005, which continued through the study period (December 2005). Most cases were from Victoria and Tasmania (point source outbreak cases included), with cases from New South Wales and Queensland increasing from October and November respectively.

Figure 1. Notifications of *S.* Typhimurium 135a, June to December 2005, by state or territory



The case control study was conducted between 12 and 23 December 2005 and comprised 61 cases and 173 controls (including those selected for the *S.* Typhimurium 44 study). From all jurisdictions 98 cases were enrolled in the study. Of these, 23 (23%) were ineligible and one did not have a phone number. Of the remaining 74 eligible cases, 11 (15%) were unable to be contacted; two (3%) refused and 61 (82%) completed the interview. For control recruitment using progressive digit dialling, calls were made to 729 individual phone numbers. Of these 293 (40%) were ineligible phone numbers (faxes or business numbers) and 19 (3%) were contacted but ineligible for the study. Of the 417 remaining, 200 (48%) were unable to be contacted, 70 (18%) refused and 147 (35%) completed the interview. South Australia, using their control bank, made an additional 40 calls, of which four (10%) were to ineligible phone numbers and three (7.5%) were contacted but ineligible. Of the remaining 33, six (18%) were unable to be contacted; one refused; and 26 (79%) completed an interview.

Symptoms reported by cases included diarrhoea (100%), cramps (88%), fever (86%), nausea (71%), headache (63%), vomiting (57%), muscle and body aches (49%) and blood in the stool (42%). The median duration of illness was 8 days (range 3–21 days) and 10 cases (17%) were hospitalised. The median age of cases was 23 years compared to 45 years for controls. The proportion of cases and controls by age group and by jurisdiction was significantly different ($p=0.002$ and $p<0.001$ respectively). There was no difference by sex (Table 1).

Table 1. Characteristics of cases and controls

Characteristic	Cases n	Cases %	Controls n	Controls %	p value†
Total	61	100	173	100	
Age group*					
1–9	22	36.1	23	13.3	0.002
10–19	6	9.8	10	5.8	
20–39	14	23.0	39	22.5	
40–59	11	18.0	51	29.5	
60+	8	13.1	47	27.2	
State					
ACT	1	1.6	5	2.9	<0.001
NSW	8	13.1	39	22.5	
Qld	0	0.0	21	12.1	
SA	6	9.8	51	29.5	
Tas	16	26.2	18	10.4	
Vic	30	49.2	39	22.5	
Sex*					
Females	25	41.0	74	42.8	0.802
Males	35	57.4	96	55.5	

* These variables may not add up to the total due to missing responses.

† P values calculated using Pearson's chi-squared test for trend.

In univariate analysis, when compared to controls, cases had significantly higher odds of having consumed chicken eaten outside the home; chicken eaten at a fast food chicken outlet; kebabs eaten at own home; other meat products purchased from Supermarket A; mince purchased from Supermarket A and; lamb purchased from Supermarket A (Table 2). Cases had significantly lower odds compared to controls of consuming lettuce, tomatoes, berries and carrots, as well as a combined fruit and vegetable variable.

Figure 2 schematically shows the different hypotheses tested for the association between illness and the consumption of chicken. Different combinations of chicken consumption categories were used to create the multivariate models, which were adjusted for age, state of residence, and lamb, mince and fruit and vegetable consumption (Table 3). After adjusting for these factors, cases had significantly higher odds of having eaten chicken purchased from Supermarket A (groups 6 and 8 compared to all others, OR=3.2) or having eaten chicken from a fast food outlet (OR=2.8) compared to controls. The odds of fruit and vegetable consumption were independently and significantly lower for cases compared with controls in all four multivariate models.

When selection of controls was restricted to controls selected for *S.* Typhimurium 135a cases only, the univariate results were similar, except that the odds ratios for any and meat and mince purchased at Supermarket A increased (data not shown). A similar increase in odds ratios was observed in multivariate analysis for chicken consumption category 1 (chicken versus no chicken) and category 3 (chicken eaten at home only, Supermarket A purchased versus other purchased), although they were not significant. The odds ratio for category 4 (home only and both at home and out, Supermarket A purchased versus all others) decreased.

When selection of cases was restricted to those from Victorian and Tasmanian cases only (the jurisdictions where most cases were reported), the univariate odds ratio for chicken purchased at Supermarket A became significantly associated with illness (OR=2.4, 95% CI=1.0,5.7). All the odds ratios decreased in the multivariate analysis, with wider confidence intervals and none were significant.

Environmental results

There were seven positive *Salmonella* results from the chicken sampling survey of Supermarket A in Victoria. A breast sample from one store and a thigh sample from another were positive for *Salmonella* Typhimurium 135a. Four samples, three breast and one drumstick from four different stores were positive for *Salmonella* Sofia and two, a thigh and wing sample from the one store, were positive for *Salmonella* Infantis.

Discussion

The results from this case control study suggest there was an association between infection with *S.* Typhimurium 135a and chicken consumption, in

Figure 2. Schema of hypotheses tested for association between chicken consumption and illness

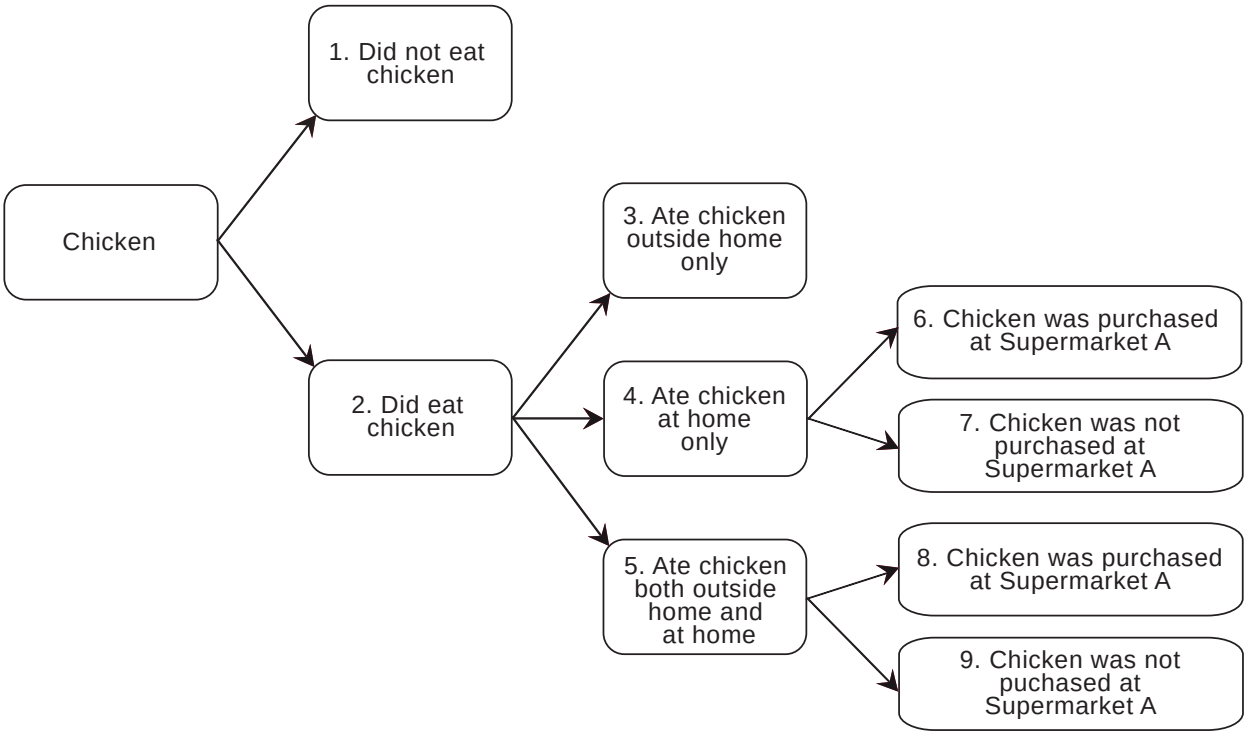


Table 2. Univariate analysis of exposure to selected foods amongst cases and controls

Exposure	Cases		Controls		Odds ratio	95% CI	P value
	Exposed /total*	%	Exposed/ total*	%			
Chicken							
Chicken	42/52	80.8	117/168	69.6	1.8	0.8-4.4	0.12
Chicken eaten outside home	30/50	60.0	72/168	42.9	2.0	1.0-4.0	0.02
Fast food chicken outlet	12/61	25.0	18/172	14.0	2.5	1.0-6.3	0.03
A la carte restaurant	4/61	8.7	9/172	7.1	1.3	0.3-4.8	0.70
Meal at another house	4/61	8.5	6/172	4.7	1.9	0.4-8.5	0.31
BBQ from supermarket	9/61	18.8	25/172	19.4	1.0	0.4-2.4	0.97
Chicken eaten at own home	25/51	49.0	63/166	38.0	1.6	0.8-3.1	0.16
Purchased at Supermarket A	13/61	52.0	22/173	12.7	1.9	0.8-4.2	0.11
Whole	3/51	5.9	10/168	8.8	1.0	0.2-4.1	1.00
Breast	13/51	25.5	35/168	20.8	1.4	0.6-3.2	0.46
Pieces	6/51	11.8	23/168	13.7	0.8	0.2-2.3	0.61
Kebab	4/49	8.2	2/168	1.2	8.1	1.1-91.6	0.01
Value added	3/50	6.0	5/169	3.0	2.1	0.3-11.7	0.30
Other meat							
Other meat total	46/54	85.2	148/170	87.1	0.9	0.3-2.4	0.76
Purchased at Supermarket A	19/61	31.1	31/172	18.0	2.1	1.0-4.2	0.03
Mince	19/47	40.4	45/163	27.6	1.8	0.8-3.7	0.07
Purchased at Supermarket A	9/61	17.3	10/163	6.1	2.8	1.0-8.2	0.03
Lamb	14/46	30.4	34/166	20.5	1.7	0.7-3.7	0.15
Purchased at Supermarket A	8/61	13.1	4/173	2.3	6.4	1.6-29.8	0.00
Pork	5/48	10.4	30/162	18.5	0.5	0.1-1.4	0.17
Hamburger	1/50	2.0	10/163	6.1	0.3	0.0-2.3	0.24
Ham	18/50	36.0	68/165	41.2	0.8	0.4-1.6	0.52
Salami	2/49	3.9	20/162	12.3	0.3	0.0-1.3	0.10
Eggs							
Any eggs	32/54	59.3	99/166	59.6	1.0	0.5-2.0	0.97
Eggs eaten outside the home	6/55	10.9	21/166	12.7	0.8	0.3-2.3	0.70
Eggs eaten at own home	25/53	47.2	88/167	52.7	0.8	0.4-1.6	0.50
Any raw egg product	21/60	35.0	53/173	30.6	1.0	0.5-2.1	0.47
Fruit and vegetables							
Lettuce	28/52	53.8	128/170	75.3	0.4	0.2-0.8	0.00
Tomatoes	24/53	45.3	123/169	72.8	0.3	0.2-0.6	0.00
Cucumber	23/55	41.8	80/166	48.2	0.8	0.4-1.5	0.39
Sprouts	2/59	3.4	11/168	6.5	0.4	0.0-2.1	0.29
Strawberries	18/52	34.6	61/169	36.1	1	0.5-2.0	0.97
Berries	2/57	3.5	26/168	15.5	0.2	0.0-0.9	0.02
Carrots	24/58	41.4	100/169	59.2	0.5	0.3-0.9	0.02
Apples	26/58	44.8	93/173	53.8	0.7	0.4-1.3	0.25
Kiwi	7/58	12.1	26/168	15.5	0.7	0.2-1.8	0.43
Any fruit/vegetables	50/60	83.3	166/173	96.0	0.2	0.1-0.7	0.00
Juice	18/58	31.0	54/168	32.1	1	0.5-1.9	0.88
Combined Supermarket A†	34/61	55.7	86-172	50.0	1.3	0.47-2.4	0.44

CI Confidence interval.

* Totals include all cases and controls in study, excluding those that answered, 'Don't know/unsure.' For example, the total for 'Chicken eaten outside home' includes those that said 'no' to having eaten chicken.

† Includes those purchasing fruit, vegetables, chicken and any meat from Supermarket A.

Table 3. Univariate and multivariate analysis of exposure to various chicken categories amongst cases and controls

Chicken category	Cases (%)	Controls (%)	Univariate analysis OR	Univariate analysis 95% CI	Adjusted analysis* OR	Adjusted analysis* 95% CI
Chicken vs no chicken (2 vs 1)	80.8	69.6	1.8	0.8–4.3	1.2	0.5–3.0
Out only chicken, from a chicken fast food outlet	25.0	14.0	2.5	1.0–6.3	2.8	1.0–7.7
Home only chicken, Supermarket A purchased vs other purchased (6 vs 7)	9.8	8.6	2.1	0.7–5.9	2.2	0.6–8.6
Home only and both at home and out, Supermarket A purchased vs all others (6 and 8 vs all other categories)	21.3	12.7	2.2	0.9–5.1	3.2	1.2–9.0

OR=Odds ratio, CI=Confidence interval

* Maximum likelihood logistic regression adjusted by age, state of residence, lamb, mince and fruit and vegetable consumption.

particular chicken purchased from Supermarket A. Cases were three times more likely to have eaten chicken purchased at Supermarket A and three times as likely to have eaten chicken purchased at a chicken fast food outlet. Analysis using controls selected for *S.* Typhimurium 135a cases only, and that restricted to Victoria and Tasmania, also supported the association between chicken and illness. The corroborating evidence of the two positive *S.* Typhimurium 135a chicken samples purchased at Supermarket A identified by cases is consistent with the findings of the case control study.

This study has several limitations. There were a high proportion of controls unable to be contacted and those recruited were significantly different to cases in respect of age and jurisdiction of residence. Controls were used from all jurisdictions in the combined *S*. Typhimurium 135a and *S*. Typhimurium 44 investigation regardless of whether there were cases in these jurisdictions, yet not all jurisdictions recruited two controls per case. These issues suggest that the controls may be an unrepresentative sample of the general population, which may have affected the results, although it is difficult to speculate in which direction bias may have occurred. Also, as the greatest increase in cases occurred in Victoria, and *S*. Typhimurium 135a cases from other states may represent background sporadic cases unrelated to the outbreak, using cases from other states might have reduced the power of the study to detect an association between exposure and illness.

Factors for *S*. Typhimurium 135a contamination or infection of retail chicken in this outbreak are unclear. It is plausible that the *Salmonella* infection of chicken that led to this outbreak occurred at the farm level. Poultry are exposed to *Salmonella* via sources such as feed or through environmental contamination and when introduced, *Salmonella* can spread rapidly throughout the flock.[15] Anecdotal evidence indicated that several farms, particularly in Victoria, had outbreaks of *S.* Typhimurium 135a in chickens during September and November 2005 that occurred concurrently with mice plagues.

It is also unclear why Supermarket A was more strongly associated with illness than other chicken retailers. The link may be due to the supermarkets purchasing chicken from affected suppliers. Discussions with food safety and retail specialists did not identify any hypotheses that would adequately explain this finding.

Salmonellosis outbreaks resulting from the consumption of chicken comprised 13 per cent of all outbreaks investigated in Australia from 1995 to 2000.[16] The incidence of human salmonellosis has increased in most industrialised countries in the 1980s and 1990s.[17] Sweden is an exception as authorities introduced voluntary testing for *Salmonella* and destruction of positive flocks between 1970 and 1984, after which the practice became mandatory. As a result the prevalence of *Salmonella* infection in chickens was reduced to 0.2–0.7 per cent in 1994 with a corresponding low prevalence of domestically acquired salmonellosis in humans.[18] The results of our investigations were communicated to industry, regulatory and retail representatives to improve longer-term objectives of reducing illness associated with chicken meat.

The results of the case control study indicated that cases were less likely to have reported eating fruit and vegetables in both the univariate and all the multivariate models. This was a consistent finding in this study, even when the results were adjusted for by age. Reasons for this protective effect of fruit and vegetables are unclear, although a possible explanation is that frequent consumers of fruit and vegetables are generally healthier and therefore less likely to become ill after eating contaminated chicken.

This outbreak demonstrates the importance of multi-jurisdiction cooperation and coordination for outbreak investigations. It was conducted over a short period of time, with most interviews completed within two weeks. Having a central web-based database for data entry allowed for efficient data analysis as current data could be downloaded directly on a daily basis. This combined effort from staff in most jurisdictions in Australia, co-ordinated by the OzFoodNet central office, should be repeated for future studies of this nature.

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Two years of enhanced surveillance of sexually-transmitted chlamydia in South East Queensland

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Abstract

The *National Sexually Transmissible Infections Strategy 2005–2008,* released in 2005, lists exploring and addressing barriers to enhanced data collection for chlamydia surveillance among the actions required for chlamydia control and prevention. This study describes a method of enhanced surveillance of sexually transmitted chlamydia notifications undertaken in South East Queensland, and the epidemiology and management of chlamydia over the study period. The service providers of a random sample of chlamydia notifications meeting preset inclusion criteria were faxed an information package and questionnaire. Telephone follow-up was initiated for non-responders. The first year of data were compared to the second year of data. The overall response rate was 93.2 per cent. Males were more likely than females to be tested because of symptoms in the first year of the study, but not the second. Females were 5.2 times (95% CI 2.43, 10.91) more likely to be screened on the suggestion of the service provider than males. The positivity rate among those tested for sexually transmitted chlamydia increased across the study period. An information package and questionnaire faxed to notifying clinicians is a simple and effective means of conducting enhanced surveillance of sexually transmitted chlamydia. An increase in the screening of males may be contributing to the increasing rate of notifications. An increasing positivity rate among all those tested for chlamydia may be due to more prevalent disease, or more focused testing of high risk groups. *Commun Dis Intell* 2006;30:456–461.

Keywords: Chlamydia, *sexually transmissible infections*

Introduction

Chlamydia is the most frequently notified infection in Australia, at a rate of approximately 180 notifications per 100,000 population in 2004,[1] increased from 152 notifications per 100,000 population in 2003.[2] In addition, the significant potential complications of infection for both women and men clearly demonstrate it as a disease of public health importance.

The *National Sexually Transmissible Infections Strategy 2005–2008,*[1] and the announcement of funding for a pilot screening program for chlamydia targeted at women aged 18–30 years[3] are key developments in chlamydia control in Australia. The former listed exploring and addressing barriers to enhanced data collection for chlamydia surveillance among the actions for chlamydia control and prevention. In this paper, we describe a simple method of enhanced surveillance that achieved a good response rate enabling us to explore the epidemiology and management of sexually transmitted chlamydia notifications in the jurisdiction of Southern Area Population Health Services – Brisbane Southside (SAPHS – BS) in South East Queensland (Figure), with an estimated resident population of 988,584 as at 30 June 2004.[4]

Methods

All notifications of *Chlamydia trachomatis* to SAPHS – BS between 1 February 2003 and 31 January 2005, were assessed against inclusion/exclusion criteria. The criteria were: the notification must occur within the study period; the residential address of the case must fall within the SAPHS – BS area or where no residential address is listed, the health service provider must operate within the SAPHS – BS area; and infections of the eye were excluded.

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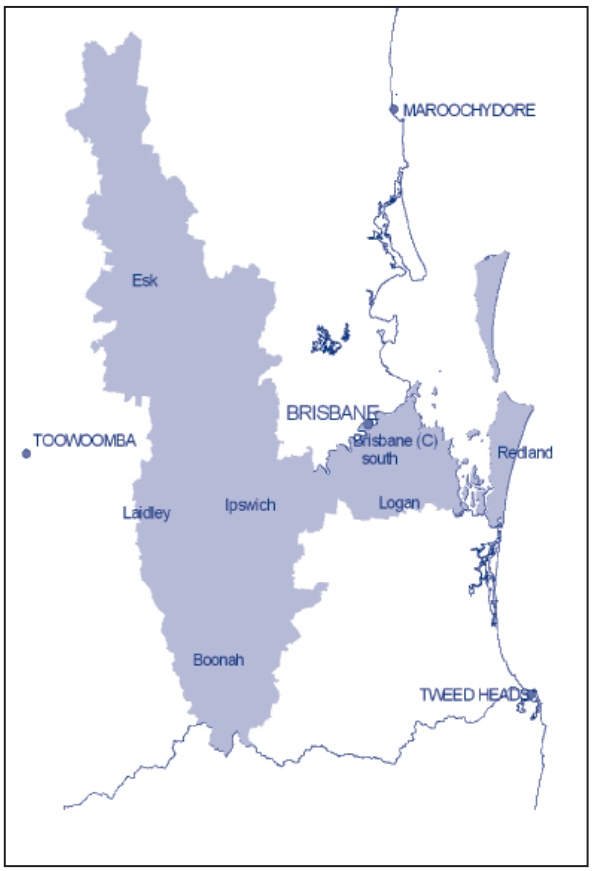
Figure. The geographical area covered by Southern Area Population Health Services – Brisbane Southside



Using a random number table, one out of every 10 eligible notifications was chosen for inclusion in the study and assigned a code. After the case's assigned code was communicated to the health service, the clinician who ordered the investigation was faxed an information package and one page de-identified questionnaire. The information package was developed with Princess Alexandra Sexual Health (PASH) based on a package used for gonorrhoea enhanced surveillance that was shown to improve clinician management practices in a local before-and-after study.[5] It included a patient information sheet, a contact tracing letter pro forma, and guidelines for the management of uncomplicated chlamydia. The questionnaire collected demographics; the reason for testing; sites tested; treatment; investigation for other sexually transmitted infections; contact tracing; and the suspected source of infection.

Four weeks were allowed for the return of the questionnaire by fax or post from the clinician. Up to three reminder phone calls were then made at approximately fortnightly intervals to the health service.

Data were analysed using SPSS for Windows version 11.5 and Epi Info 6. The notifications from the first year of the study (1 February 2003 to 31 January 2004 – period 1) were compared to the notifications from the second year of the study (1 February 2004 to 31 January 2005 – period 2) using the chi-squared test of significance or Mantel-Haenzel test where appropriate. This time comparison served to highlight any possible trends.

The study sample was compared to all notifications meeting inclusion/exclusion criteria, recorded in the Notifiable Conditions (NOCS) database for each study period. Hospital and private laboratories servicing the SAPHS -BS area were asked to provide the total number of requests received for urogenital chlamydia testing and the number of positive tests, using the same inclusion/exclusion criteria as the study.

This study of the epidemiology of a notifiable disease was undertaken under the provisions of the *Queensland Health Act 1937.*

Results

The response rate was 93.2 per cent (289 case report forms received out of 310).

The demographics of the cases for the two periods of the study approximated those of all 'genital' and 'unspecified' chlamydia notifications to the SAPHS –BS for the study period (Table 1). The proportion of females aged 18 to 30 years (the target group for the national pilot screening program) was similar over the two periods (71% and 77% of females for periods 1 and 2 respectively; p=0.41). This group accounted for 43 per cent and 47 per cent of all notifications for periods 1 and 2 respectively.

The male:female ratio did not change during the study. Six per cent of cases were identified as Indigenous in both study periods, while Indigenous status was either unknown or not answered in 22 per cent of cases in period 1 and 28 per cent of cases in period 2. For both study periods, 86 per cent of cases were known to speak English at home, approximately one per cent of cases were identified as working in the sex industry, and approximately four per cent of women were pregnant at the time of investigation for chlamydia.

The reasons for testing over the two study periods are given in Table 2. There was no change across the study in the proportion of cases tested because of symptoms (p=0.22). However, males were more likely than females to be tested because of symptoms in period 1 (p=0.005), but not period 2 (p=0.43). Urethral discharge or dysuria was the most common symptom for males for both time periods, whereas no particular symptom was dominant among females (data not shown).

Table 1. Comparability of all chlamydia notifications to SAPHS – BS meeting the study criteria and the study sample notifications

Period 1	All notifications	Study sample
Number	1,328	126 (incl. 9 non responders)
Age range (years)	13–64	14–62
Median age (years)	22	22
Males (%)	38	40
Females (%)	62	60
Identified as Indigenous (%)	6	6
Co-infected with gonorrhoea (%)	2	2
Period 2	**All notifications**	**Study sample**
Number	1,812	184 (incl. 12 non responders)
Age range (years)	6–70	14–53
Median age (years)	22	23
Males (%)	37	39
Females (%)	63	61
Identified as Indigenous (%)	5	6
Co-infected with gonorrhoea (%)	1	1

Table 2. The proportion of the sample giving particular reasons for testing for chlamydia*

Reason for testing	Period 1						Period 2					
	All		Males		Females		All		Males		Females	
	%	n	%	n	%	n	%	n	%	n	%	n
Contact with chlamydia infection	16	19	21	10	13	9	23	39	28	19	20	20
Screening test requested by patient	20	23	15	7	23	16	18	30	25	17	13	13
Screening test suggested by doctor	18	21	4	2	27	19	24	40	7	5	34	35
Antenatal screening test	3	3	–	–	4	3	0	0	–	–	0	0
Symptomatic	41	49	58	27	32	22	34	59	38	26	32	33
Other	2	2	2	1	1	1	1	2	2	1	1	1
Total	100	117	100	47	100	70	100	170	100	68	100	102

* Excludes 2 cases in period 2 for whom the reason for testing was not given.

Females were 5.2 times (95% CI 2.43, 10.91) more likely to be screened on the suggestion of the service provider than males. A greater proportion of both males and females appeared to be tested because of contact with someone found to have chlamydia in period 2 compared to period 1, but the difference was not statistically significant (p=0.42 for males and p=0.25 for females).

The reason for attendance was not associated with age group for either period (p=0.06 and p=0.76 respectively).

The specimens most commonly collected in both periods were cervical or vaginal swabs and urine samples. Approximately 6 per cent of diagnoses in each period were made from swabs of the urethra or rectum. Sixty-three per cent and 62 per cent of the cases had positive urinary polymerase chain reaction results in periods 1 and 2 respectively.

Azithromycin was prescribed as treatment for chlamydia in 80 per cent of cases in period 1 and 69 per cent of cases in period 2 (p=0.09). Fifteen per cent of cases were prescribed doxycycline in period 1 compared to 20 per cent in period 2 (p=0.22). Four per cent of cases in period 1 and 5 per cent of cases in period 2 had a second treatment prescribed: those specified were either azithromycin or doxycycline.

Fifty-six per cent of study cases in period 1, and 69 per cent of study cases in period 2 had testing requested for gonorrhoea (p=0.02). A small proportion tested positive (Table 1), while a further 5 per cent in period 1 and 8 per cent in period 2 had test results pending at the time of enhanced surveillance. Among all chlamydia notifications meeting the study inclusion criteria, 2 per cent in period 1 and 1 per cent in period 2 were also notified for urogenital gonorrhoea within a two month timeframe (Table 1).

There were no significant differences in the proportion of cases tested for each of HIV, hepatitis B, and syphilis between period 1 and period 2. These proportions ranged from 54 per cent to 61 per cent. Seventeen and 18 per cent of cases in periods 1 and 2 respectively had tests requested for hepatitis C. Co-infection was uncommon.

Contact tracing was initiated in 85 per cent of cases in period 1 and 81 per cent of cases in period 2, almost always (94% of these cases) by the cases themselves. In period 1, 88 per cent of cases listed a heterosexual relationship as the likely source of infection, while the gender of the contact was unknown to the service provider in 9 per cent of cases. In period 2, 86 per cent of cases listed a heterosexual relationship as the likely source of infection, and for 13 per cent, the gender of the contact was unknown to the service provider. Seven per cent of cases in period 1, and 2 per cent of cases in period 2 reported that it was likely they had acquired the infection overseas.

Males were 2.3 times (95% CI 1.53, 3.5) as likely as females to have identified a casual partner as the likely source of infection. For the majority of females in both periods (61% and 60%), their regular partner was thought to be the source of infection (vs males RR 1.8 – 95% CI 1.34, 2.37). For 20 per cent and 25 per cent (periods 1 and 2 respectively) of females and 24 per cent and 28 per cent (periods 1 and 2 respectively) of males the most likely source of infection was unknown to the service provider.

There was an increase in the positivity rate of chlamydia tests across the study period for each laboratory, although not all of these were statistically significant (Table 3).

Discussion

A faxed information package and questionnaire to notifying clinicians is a simple and effective means of conducting enhanced surveillance of sexually transmitted chlamydia. Factors that may have contributed to the high response rate included reminder phone calls,[6] the short length of the questionnaire,[7] provision of clinical information with the questionnaire,[8] faxing the questionnaire,[9] and collaboration between population health and a sexual health service. A metropolitan public health unit in New South Wales found that posting questionnaires to notifying service providers, with one posted reminder for non-responders, was also effective, with a response rate of 88 per cent.[10] The Victorian Department of Human Services distributes questionnaires to notifying clinicians via pathology laboratories.[11] The response rate in 2001 was 58 per cent. An earlier study in Victoria achieved an 85 per cent response rate using mainly telephone survey methods with multiple attempts to contact diagnosing providers.[12]

Enhanced surveillance in South East Queensland confirms the public health importance of sexually transmitted chlamydia infection in young adults in this area, and lends support to the target age group

Table 3. Tests requested for urogenital chlamydia, number positive and positivity rate 1 February 2003 to 31 January 2005, by laboratory for the SAPHS – BS area

	Period 1	Period 2	p value
Mater			
Positives	63	104	
Number	1,293	1,603	0.08
% positive	4.9	6.5	
Sullivan Nicolaides Pathology*			
Positives	274	479	
Number	5,473	7,766	0.003
% positive	5.0	6.2	
QML Pathology			
Positives	766	974	
Number	15,156	16,494	0.002
% positive	5.1	5.9	
Queensland Health Pathology and Scientific Services			
Positives	314	300	
Number	4,866	4,160	0.18
% positive	6.5	7.2	
Total†			
Positives	1,495	1,857	
Number	28,347	30,023	<0.001
% positive	5.3	6.2	

* Data given for period 1 is for 1 May 2003 to 31 January 2004 as number of positives for all of period 1 not available.

† Includes calculated data (positivity rate for 1 May 2003 to 31 January 2004 for Sullivan and Nicolaides applied to number of tests requested for all of period 1 for this laboratory to calculate total number and positives).

of the national screening pilot, with over 70 per cent of female notifications in those aged 18 to 30 years.

It is possible that a trend towards increased screening of males is responsible for at least part of the detected increase in chlamydia notifications in South East Queensland, although this requires verification in a longer study. Some of this apparent increase in screening may have been influenced by the information package sent to providers with the enhanced surveillance questionnaire. It is also acknowledged that enhanced surveillance does not account for those who were tested for chlamydia, but had a negative result. Enhanced surveillance of chlamydia notifications between 1997 and 2001 in Victoria also demonstrated a decrease in the proportion of men tested because of symptoms.[11]

Over both periods of the study, providers were more likely to recommend screening to females than males. This practice may account for some of the difference in notification rates between the sexes.

The positivity rate among those tested for sexually transmitted chlamydia increased across the study period. If indicative of a trend, this may indicate more prevalent disease, or more focused testing of high risk groups. A study in New South Wales[13] also found an increase in positivity rate with time, while a Victorian study[14] did not.

The increase in testing for gonorrhoea over the study period, if indicative of a trend, could have been due to a number of factors. These may have included awareness of the availability of gonorrhoea and chlamydia testing on the same specimen, the information package, and patient request.

As the study was limited to two years in length, further investigation into the reasons for the increasing notification rate, and the possible identified trends of increasing positivity and testing for gonorrhoea could be valuable.

While most providers reported that contact tracing was being undertaken, usually by the cases themselves, the effectiveness of that contact tracing was not investigated by this study. Contact tracing has been recognised as an area in need of further research.[1]

The demographic variables examined and the proportions of cases co-infected with gonorrhoea were comparable for the study sample and all chlamydia notifications to SAPHS – BS, suggesting the study methods achieved a representative sample. Thus, surveying the notifying providers of a 10 per cent random sample of notifications may prove a repeatable and cost-effective enhanced surveillance technique for sexually transmitted chlamydia.

As demonstrated by this study, effective enhanced surveillance can provide information on clinicians' investigative and management practices, as well as the epidemiology of chlamydia in the local population. This sort of information could be valuable to focus chlamydia control efforts at a local level.

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Communicable Disease Conference 2007

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The theme of the CDC Conference 2007 is *From Outbreaks to Pandemics in the Region: Building Our Capacity to Respond.* The proposed date and location for the CDC Conference 2007 is 15–16 March 2007 at Rydges Lakeside in Canberra.

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More information on the CDC Conference 2007 is available from the CDC Conference 2007 website (www.diseases.consec.com.au)

Community-acquired methicillin-resistant *Staphylococcus aureus* in Central Australia

Claire L Stevens,[1] Anna Ralph,[2] James ET McLeod,[3] Malcolm I McDonald[4]

Abstract

To date, there has been scant information about the burden of methicillin-resistant *Staphylococcus aureus* infections in Central Australia. Our aims were to determine the proportion of *Staphylococcus aureus* infections due to methicillin-resistant strains in Central Australia, to characterise resistance to non-beta lactam antibiotics and to correlate findings with available demographic information. We retrospectively reviewed *S. aureus* isolates identified by the Microbiology Laboratory of the Pathology Department, Alice Springs Hospital between September 2005 and February 2006. Multi-resistance was defined as resistance to three or more non-beta lactam antibiotics. We identified the recovery site and extended antibiotic resistance profile of each isolate. Demographic data included place of residence, discharge diagnosis and ethnicity. There were 524 *S. aureus* isolates: 417 (79.6%) methicillin-sensitive *S. aureus*, 104 (19.7%) non-multi-resistant MRSA (nmrMRSA) and 3 (0.7%) multi-resistant MRSA (mrMRSA). MRSA accounted for 7/22 (32%) invasive infections and 91/474 (19.2%) cases of staphylococcal skin infections. Aboriginal people comprised 89 per cent (93/104) of patients with nmrMRSA; 57 per cent lived in remote communities, 21 per cent in suburban Alice Springs, and 18 per cent in Alice Springs Town Camps. Six per cent (6/104) of nmrMRSA were hospital-acquired. Of the nmrMRSA isolates, 57 per cent (59/104) were resistant to erythromycin and 7 per cent (7/104) to fusidic acid. All MRSA isolates were susceptible to co-trimoxazole. In conclusion, Central Australia has high rates of community-acquired nmrMRSA and low rates of multi-resistant MRSA. Erythromycin resistance in *S. aureus* is also common. These findings should prompt the review of antimicrobial prescribing guidelines for the region, especially for treatment of skin and soft tissue infections. *Commun Dis Intell* 2006;30:462–466.

Keywords: Staphylococcus aureus, *methicillin resistance, community-acquired infection, Central Australia, Aboriginal*

Introduction

Methicillin-resistant (beta-lactam resistant) *Staphylococcus aureus* (MRSA) is increasingly recognised in non-health care settings around Australia, and Aboriginal Australians are among those most at risk.[1,2] A recent longitudinal study documented an increase of hospitalised patients with community-acquired MRSA from 4.7 per cent in 2000 to 7.3 per cent in 2004; the rise was especially marked in Darwin where the proportional increase was from 5 per cent to 20 per cent.[3] Community-acquired MRSA made up 23 per cent of *S. aureus* isolates from pyoderma lesions and throat swabs in a recent study conducted in remote Aboriginal communities in the Top End of the Northern Territory.[4] In addition, a recent cross-sectional survey in a remote Queensland Aboriginal community reported that 15 per cent of children were found to carry MRSA, although the numbers were small.[5] It has been projected by one authority that methicillin resistance may eventually become as ubiquitous in *S. aureus* as penicillin resistance did several decades ago.[6] This trend has important implications for empirical antibiotic prescribing and infection control measures in hospitals, urban settings and remote communities. Johnson and others, in a recent editorial in the *Medical Journal of Australia*, proposed management guidelines that include routine collection of local data, microbiological culture and antimicrobial susceptibility testing in settings where *S. aureus* are important pathogens.[7]

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There are no published data from Central Australia, although anecdotal reports indicate increasing rates of MRSA infection. Alice Springs Hospital serves a population of 51,000 people living in a region of approximately 1 million square kilometres, and encompassing southern and central Northern Territory, northern South Australia, and eastern Western Australia. This retrospective study is based on laboratory isolates collected over a six month period. It provides the first documentation of the MRSA burden in Central Australia, both community-acquired and health care-acquired. The findings have implications for hospital and community antimicrobial prescribing guidelines.

Methods

We reviewed the laboratory data for *S. aureus* isolates identified by the Microbiology Laboratory of the Pathology Department, Alice Springs Hospital (ASH) between September 2005 and February 2006. Colonies of gram-positive cocci were identified as *S. aureus* if they were catalase positive and tested positive for production of coagulase using Staphaurex® (Oxoid). The specimen site (blood, sterile body fluid, respiratory specimen, wound swab or screening swab) was recorded. As methicillin-sensitive *S. aureus* (MSSA) is not reported from screening swabs, these were excluded. Antimicrobial susceptibility testing was performed using a disc diffusion method in accordance with Clinical and Laboratory Standards Institute (CLSI) Methods.[8] The isolates were reported as MSSA or MRSA based upon the diameter of the zone of inhibition around an oxacillin 1 microgram Disc (Oxoid).

We also determined susceptibility to ciprofloxacin, erythromycin, flucloxacillin, fusidic acid, gentamicin, tetracycline and trimethoprim-sulphamethoxazole (co-trimoxazole) for all MRSA isolates using recommended CLSI methods. Clindamycin susceptibility was inferred when the isolate was resistant to erythromycin. This was based on data from the Top End of the Northern Territory,[4] acknowledging that clindamycin resistance might be over-estimated when due to the *msrA* mechanism rather than the *erm*-mediated mechanism.[9]

S. aureus was defined as non-multi-resistant (nmrMRSA) if resistant to methicillin and less than three other classes of non-beta lactam antibiotic.[10] Infections were classified as community or health care-acquired by the ASH Infection Control Unit; health care-acquired infection was defined as infection acquired after greater than 48 hours of hospitalisation, or within four weeks after discharge and due to an organism acquired during hospitalisation. Community-acquired infections were those considered to be present on admission to hospital and not able to be linked to previous admission to hospital.

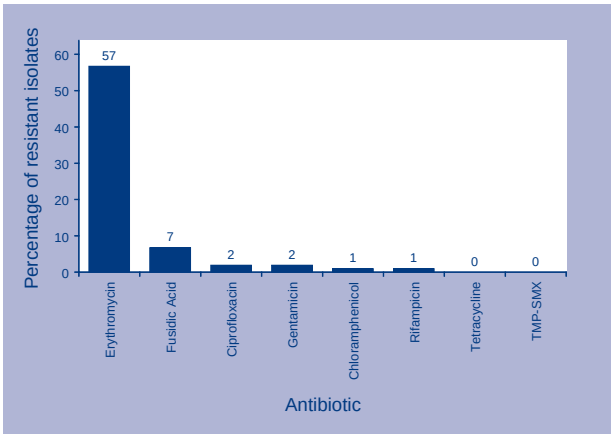
For each patient, we recorded the discharge diagnosis (coded as per the International Classification of Diseases), ethnicity (Aboriginal or non-Aboriginal), and place of residence, as noted in the medical records and/or electronic coding system. Proportions were compared using the Chi-squared test.

Results

A search of laboratory and clinical records produced 524 *S. aureus* isolates: 417 (79.6%) MSSA, 104 (19.7%) nmrMRSA and 3 (0.7%) mrMRSA. An additional 56 MRSA isolates (49 nmrMRSA and 7 mrMRSA) were detected on 'screening swabs'; these were not included in the study. Six per cent of nmrMRSA infections (6 of 104) and one of three mrMRSA infections were coded by the ASH Infection Control Unit as being hospital-acquired. However, this is likely to be an under-estimate because the ASH Infection Control Unit did not include outpatients and dialysis patients in the hospital-acquired group.

Susceptibility testing demonstrated that all nmrMRSA and mrMRSA isolates were susceptible to tetracyclines and co-trimoxazole. The three mrMRSA isolates qualified as multi-resistant on the basis of resistance to gentamicin, erythromycin and fusidic acid in addition to beta-lactam antibiotics. Of the nmrMRSA isolates, 59 (57%) were resistant to erythromycin, and 7 (6.7%) were resistant to fusidic acid. Antibiogram data for nmrMRSA isolates are shown in Figure 1.

Figure 1. Proportion (%) of antibiotic resistance in nmrMRSA isolates

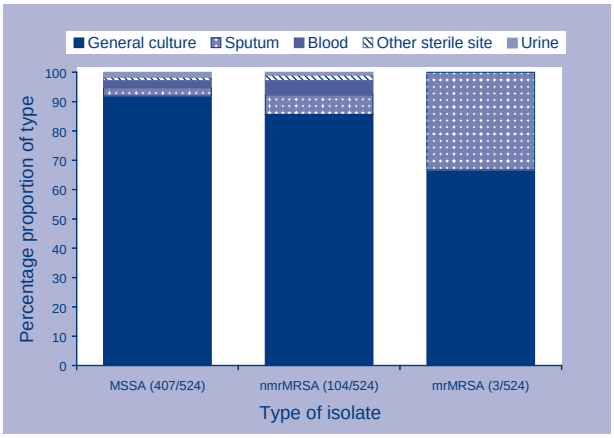


TMP-SMX = trimethoprim-sulphamethoxazole (co-trimoxazole)

Invasive staphylococcal infections (blood or other sterile site) accounted for 22 (5%) isolates; there were 15 cases of bacteraemia (10 MSSA, 5 nmrMRSA) and 9 infections at other sterile sites (5 MSSA, 2 nmrMRSA). Thus, 7 of 22 (31.8%) invasive *S. aureus* infections were due to nmrMRSA compared with 97 of 502 (19.4%) non-invasive infec-

tions (P=0.15). Skin swabs were the most common specimens received by the laboratory (Figure 2); 91 of 474 (19.2%) skin swabs yielded MRSA. The laboratory also had many specimens labelled 'general' category. When we reviewed their origin, 90 of 91 were found to be skin swabs. Thus, 'general' isolates have been counted with the skin isolates.

Figure 2. Site of infection as a proportion of total number of infections of each *Staphylococcus aureus* type, Northern Territory



Upon review of the clinical records of people with nmrMRSA, staphylococcal infection was included in the coded diagnoses on 58 of 104 (56%) occasions and appeared to be incidental to the primary diagnosis (not recorded as a significant finding) in the remainder. The most common staphylococcal infections were skin and soft tissue (Table) and 70 of 104 (67%) patients with nmrMRSA infection required hospital admission.

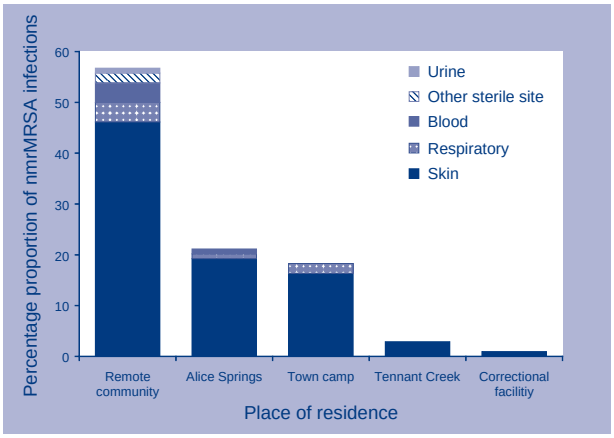
Table. Discharge diagnoses for patients with nmrMRSA

Diagnosis	n	%
Laceration or wound	20	19.2
Abscess	14	13.5
Surgical wound including compound fracture	8	7.7
Diabetic leg ulcer	3	2.9
Cellulitis	3	2.9
Burn	2	1.9
Pneumonia	2	1.9
Exacerbation of bronchiectasis	2	1.9
Other	4	3.8
Non-infective diagnosis only	46	44.2
Total	104	100

Eighty-nine per cent of patients from whom nmrMRSA was isolated were Aboriginal. The three mrMRSA isolates were from non-Aboriginal people. During the six month study period, Aboriginal people accounted for 80 per cent of admissions to ASH (unpublished data from ASH Separations data for the period including September 2005 to February 2006).

As shown in Figure 3, nmrMRSA was most commonly recovered from people who lived in remote communities - 59 of 104 (57%), followed by Alice Springs suburban residences - 22 (21%), Alice Springs town camps - 19 (18%), Tennant Creek - 3 (3%) and the Correctional Facility - 1 (1%). The three mrMRSA infections were in patients who resided in suburban Alice Springs.

Figure 3. Place of abode of patients with nmrMRSA infection



Discussion

Community-acquired non-multi-resistant MRSA infection in Central Australia has now become a major public health concern. Rates of MRSA infection greatly exceed those of the rest of Australia with the exception of the Top End of the Northern Territory,[3] Northern Queensland[5] and certain defined urban populations.[1] In Central Australia the burden of MRSA skin and soft tissue infection is largely borne by the Aboriginal population, especially people from remote communities. Moreover, the Aboriginal population is much more likely to develop life-threatening invasive disease as a result of skin and soft tissue infection than the non-Aboriginal population, and the outcome is worse.[2]

It is important to determine whether nmrMRSA was recently imported into Central Australia or arose *de novo* with local *S. aureus* strains acquiring the *mec* gene that encodes the low-affinity penicillin binding protein responsible for beta–lactam resistance (PBP2a). Knowing the source will, to some extent,

dictate the public health control measures required. Fortunately, *S. aureus* carry their 'pedigree' with them, written in the nucleotide sequences of seven basic housekeeping genes, and their lineage can be revealed using the molecular technique of multilocus sequence typing.[11] The *mec* genes and related genes (SCC*mec*) can also be typed to determine whether they are likely to be of community origin. It would be important to determine whether Central Australian strains have genes for the Panton-Valentine leukocidin, an important marker of skin infection and propensity to cause necrotising pneumonia.[6] It could be that in Central Australia we are experiencing an outbreak of MRSA, imported from Western Australia or the Top End, An alternative scenario is that we are witnessing an outbreak of an imported *mec* gene that is finding its way into long-established community strains of MSSA. A third scenario is that, in certain settings and perhaps promoted by local antimicrobial prescribing patterns, a *mec* gene crosses from a local non-*aureus* staphylococcus (such as *S. sciuri* on household pets – particularly dogs) into *S. aureus*. *S. sciuri* has previously been shown to be a plausible extra-species source of *mecA* for *S. aureus*.[12] Close contact between animals (especially dogs) and humans in Indigenous communities could potentially facilitate this process. In addition, nmrMRSA strains appear to have greater aptitude for establishing skin colonisation, displacing mrMRSA in hospital and other settings.[6] This could explain the relatively low rate of mrMRSA infection in ASH. Geographical or social isolation cannot realistically be invoked as the explanation because there is frequent traffic of patients and staff to intra– and inter-state hospitals.

High apparent rates of community acquisition indicate that attempts to contain MRSA need to be largely community-based. Examples of effective community interventions include 'Healthy Skin' programs, such as those employed in the Top End of the Northern Territory,[13] and installation of more swimming pools. Community pools have been associated with reduction in skin (and ear) infections.[14] Antimicrobial prescribing practices in remote communities could also be reviewed and modified if required.

Infection control precautions need reinforcement within hospital settings. We identified problems with classification of cases as health-care related or community-acquired MRSA. We also noted that isolation and contact precautions were instituted for only 8 of 24 inpatients with nmrMRSA infection during a recent two month surveillance period. Renewed enthusiasm of public health policy planners and health practitioners regarding regional infection control strategies would be welcomed.

The lack of efficacy of beta-lactams for up to one third of *S. aureus* infections in Central Australia contrasts with the preservation of beta-lactam susceptibility in other common local organisms: locally-acquired *Streptococcus pneumoniae, Haemophilus influenzae* and *Neisseria gonorrhoea* are almost universally beta-lactam-susceptible. There is also a high burden of beta-haemolytic streptococcal infection, especially pyoderma, and the post-streptococcal sequelae of rheumatic fever and glomerulonephritis. These factors lead to widespread dependence upon beta-lactam antibiotics in community and hospital antibiotic protocols. The most commonly used guideline in the region is the Central Australian Rural Practitioners Association Standard Treatment Manual[15] and it advocates intramuscular benzathine penicillin G for treatment of skin sores. A key rationale for treating skin sores is to prevent harmful post-streptococcal sequelae rather than because of local pathology. However, staphylococcal and streptococcal skin lesions are frequently indistinguishable; the two pathogens are often found in the same lesion.[3] Our data, and those of McDonald *et al*, indicate that beta-lactam therapy may no longer be effective for around 20 per cent of skin sores in Northern Territory Aboriginal communities, and the pathogenic potential of nmrMRSA is clearly evident.

Difficulties in achieving timely specimen collection, transportation to a laboratory, and follow up of patients in isolated and mobile populations, render strategies based on culture results problematic. Overuse of agents active against MRSA will lead to rising resistance to such agents. Bearing in mind these considerations, the authors support ongoing use of a beta-lactam agent as empirical therapy for non-severe suspected staphylococcal infections, but in keeping with other Australian guidelines.[7] We also advocate collection and follow-up of swab specimens from skin and soft tissue infections, and use of an agent active against MRSA, for empirical treatment of severe suspected staphylococcal infections, or infection in a patient known to be colonised or infected with MRSA.

Erythromycin and clindamycin cannot be relied upon empirically as non-beta lactam alternatives, as 57 per cent of nmrMRSA isolates demonstrated erythromycin resistance. Macrolide resistance may be driven by the widespread use of this antibiotic class in Central Australia for highly prevalent respiratory tract infections, genital *Chlamydia* and trachoma (including occasional mass community treatments with azithromycin). The data presented here show that MRSA isolates in Central Australia are reliably susceptible to co-trimoxazole, a potential alternative for treating non-severe infections. However clinical efficacy demonstrated by trial data is lacking. Fusidic acid susceptibility must be confirmed before using this agent because resist-

ance was identified in seven per cent of our isolates. Topical use of fusidic acid is likely to promote resistance and is discouraged.[16] Randomised controlled trials comparing antibiotic treatment regimens for nmrMRSA, are eagerly awaited. A planned prospective clinical study with molecular typing of Central Australia isolates should further our understanding of *S. aureus* infection in this region.

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OzFoodNet quarterly report, 1 July to 30 September 2006

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigation of outbreaks of gastrointestinal illness and clusters of disease potentially related to food occurring in Australia from 1 July to 30 September 2006.

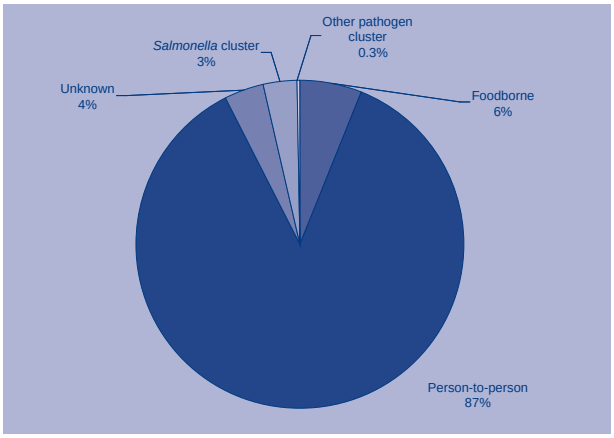
Data were received from OzFoodNet representatives in all Australian states and territories and a sentinel site in the Hunter/New England region of New South Wales. The data in this report are provisional and subject to change, as the results of outbreak investigations can take months to finalise.

During the third quarter of 2006, OzFoodNet sites reported 379 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports are delayed, meaning that these figures significantly under-represent the true burden of these infections. In total, these outbreaks affected 7,457 people of which there were 199 hospitalised and 15 died. The majority (87%, n=328) of outbreaks resulted from infections suspected to be spread from person-to-person (Figure). Of the outbreaks in institutions, 192 were in aged care facilities, 77 were in hospitals, 60 were in child care facilities and four were in various other institutional settings. Norovirus was identified as a cause of illness in 103 of the outbreaks in aged care facilities and was suspected in many more.

Foodborne disease outbreaks

There were 23 outbreaks during the third quarter of 2006 where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table). These outbreaks affected

Figure. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, 1 July to 30 September 2006



176 people and resulted in 20 people being admitted to hospital. There were no deaths. This compares with 30 outbreaks for the third quarter of 2005 and 22 outbreaks in the second quarter of 2006.

Salmonella was responsible for eight outbreaks during the quarter, with *Salmonella* Typhimurium being the most common serotype. *S.* Typhimurium 170/108 was responsible for two outbreaks, and *S.* Typhimurium 135a, *S.* Typhimurium 135, and *S.* Typhimurium 8 were each responsible for one outbreak each. The other *Salmonella* serotypes causing outbreaks were *S.* Kiambu (2 outbreaks) and *S.* Potsdam (1 outbreak). *Clostridium perfringens* intoxication and ciguatera fish poisoning were each responsible for two outbreaks. There was also an outbreak of methaemoglobinaemia associated with the consumption of a food additive powder and an outbreak caused by *Campylobacter*. The remaining nine outbreaks were caused by unknown aetiological agents.

Nine outbreaks reported in the quarter were associated with food prepared by restaurants, four with takeaway food premises, three by commercial caterers and three where food was prepared in private

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All data are reported using the date the report was received by the health agency.

Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* July to September 2006

State or territory	Month of outbreak	Setting prepared	Infection/illness	Number affected	Evidence	Responsible vehicle
NSW	July	Restaurant	Unknown	5	D	Unknown
		Commercial caterer	Unknown	5	D	Suspected catering food
		Restaurant	Unknown	4	D	Chinese buffet
		Restaurant	Unknown	2	D	Unknown
		Child care	*Salmonella* Potsdam	4	D	Pikelets made from whole eggs
		Takeaway	*Salmonella* Typhimurium 170	4	M	Eggs
		Takeaway	*Salmonella* Typhimurium 135A	2	D	Suspect eggs
	September	Restaurant	Unknown	7	D	Pasta, pizza
		Imported food	Sodium nitrite	6	D	Powder additive
		Commercial manufactured food	*Salmonella* Typhimurium 170	2	D	Suspect dips
NT	September	Private residence	Ciguatera toxin	14	D	Mother-in-law fish
Qld	August	Restaurant	*Salmonella* Typhimurium 135	6	D	Suspected chicken teriyaki sushi rolls
	July	Restaurant	*Clostridium perfringens*	13	M	Chicken & lamb guvec
		Restaurant	Unknown	6	D	Unknown
		Takeaway	Unknown	4	D	Suspected beef/ lamb component of doner kebab
		Private residence	Ciguatera toxin	2	D	Spanish mackerel
	September	Private residence	*Salmonella* Typhimurium 8	7	D	Unknown
		Takeaway	*Clostridium perfringens*	6	D	Lamb korma
Vic	August	Aged care facility	*Campylobacter*	13	D	Unknown
		Commercial caterer	Unknown	7	D	Sandwiches
	September	Commercial caterer	Unknown	19	D	Unknown
WA	September	Restaurant	*Salmonella* Kiambu	35	D	Unknown
		Restaurant	*Salmonella* Kiambu	3	D	Unknown

* No foodborne outbreaks were reported in the Australian Capital Territory, South Australia or Tasmania during the quarter.

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

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

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

residences. Single foodborne disease outbreaks were associated with food prepared by a commercial food manufacturer, an aged care home, and a child care facility. One outbreak was also associated with imported food.

To investigate these outbreaks, sites conducted five cohort studies and descriptive data were collected for the remaining 18 outbreaks. Investigators obtained microbiological evidence linking a food vehicle to illness in two outbreaks. For the remaining 21 outbreaks, investigators obtained descriptive epidemiological evidence implicating the food vehicle or suggesting foodborne transmission.

Queensland reported seven outbreaks of foodborne disease during the quarter. Four were attributed to foods being left to cool at room temperature before refrigeration. *C. perfringens* was identified as the agent responsible for illness in at least 13 people after a restaurant meal of chicken and lamb guvec. *C. perfringens* was detected in both clinical and

food samples. The guvec dishes were cooked in large batches in 40 litre pots and left to cool at room temperature for approximately eight hours before being placed into smaller containers and stored in a cold room. In another outbreak, six people from two unconnected groups were ill after consuming a common dish of lamb korma at the same restaurant on the same night. *C. perfringens* was detected in faecal specimens, but no leftover food samples were available for testing. A third outbreak of four cases who consumed takeaway lamb and beef kebabs was also suspected to be due to *C. perfringens*. The fourth outbreak possibly caused by storage of food at incorrect temperatures involved six people infected with *S.* Typhimurium 135 following consumption of sushi rolls from the same venue over a two day period.

New South Wales reported 10 outbreaks of foodborne disease during the quarter. *S.* Potsdam was identified in four children who attend the same child care facility. The onset of illness for the children were three days apart. Children were located in the toddlers (1 case) and general (3 cases) buildings, which have separate dining areas, sleeping areas, toilets and playground facilities. All four children attended child care on a Friday. Meals prepared at the facility were deemed low risk. All four children were involved in preparing pikelets (beating batter) and then consuming them after they were cooked. Raw eggs were an ingredient of the pikelet batter. Staff members reported that none of the children licked the beaters or stuck fingers in raw batter. However, the pikelets may have been undercooked, with the inside of some pikelets reported to be quite runny.

In late September, New South Wales investigated two clusters involving a total of six cases of methaemoglobinaemia that were associated with the consumption of Goldfish brand nutre powder. Laboratory testing of nutre powder showed that it was 100% sodium nitrite. Consumption of sodium nitrite converts haemoglobin to methaemoglobin, which is unable to bind with oxygen, resulting in hypoxia and has been previously associated with outbreaks.[1,2] In both clusters the nutre powder had been purchased from Asian grocery stores and added to food as a flavour enhancer. The product was imported from China and distributed to multiple states in Australia. Enhanced surveillance in all other jurisdictions did not identify any further cases. Food Standards Australia New Zealand coordinated a national consumer level recall of Goldfish brand nutre powder.

Victoria investigated three outbreaks during the quarter including an outbreak of 13 cases of illness in an aged care facility. Three residents were confirmed with *Campylobacter* infection. A staff member was also ill but likely to have been a secondary case as her onset was 5 days after the last case in a resident. A food source was suspected as the cause of this outbreak, but a specific food was unable to be identified during the investigation. A viral illness was suspected to have caused two outbreaks associated with commercially catered functions. One outbreak was suspected to have been associated with the consumption of sandwiches, but a food source was not identified for the other incident.

Western Australia reported two outbreaks caused by *S.* Kiambu that were probably related. The cases from both outbreaks had onset dates within a two week period and genetic patterns of the *S.* Kiambu isolates were indistinguishable from each other. One outbreak occurred in a cafe where three confirmed cases had eaten. A high risk food consumed by these cases was raw egg mayonnaise. Four confirmed cases were associated with dining at another restaurant venue. A cohort study of patrons at this restaurant venue found that 31 of 149 people who ate at the restaurant had become ill; four staff members also tested positive for *S*. Kiambu. There was no statistical association with illness and any of the menu items and *Salmonella* was not detected in food or environmental samples from the venue. The source of infection for either outbreak was not determined. The *S.* Kiambu isolates were sensitive to a wide range of antibiotics.

The Northern Territory reported one outbreak of ciguatera fish poisoning that affected 14 people. All were members of the same family and ate parts of one 'mother-in-law' fish, the common name for slate sweetlips (*Diagramma labiosum*), during an evening meal. Hospital treatment was required by four of the cases.[3]

South Australia, Tasmania and the Australian Capital Territory did not report any foodborne outbreaks occurring in the third quarter of 2006.

Enhanced hepatitis A surveillance and response

In early July, Queensland was notified of a laboratory-confirmed hepatitis A virus (HAV) infection in an 18-year-old female food handler in a smallgoods processing plant packing pre-cooked, ready-to-eat meats. The case worked during the 25 day infectious period before onset of symptoms. Although the risk of contaminating handled meat with HAV was considered low, the company voluntarily recalled potentially affected products. OzFoodNet enhanced surveillance for hepatitis A infections across all states and territories on behalf of the Communicable Disease Network Australia. Enhanced surveillance for hepatitis A did not detect any associated cases. From 26 June to 18 September 2006, 50 cases of HAV were notified to the National Notifiable

Diseases Surveillance System. The mean age of cases was 32 years (range 5 to 79 years) with a 1:1 male to female ratio. Thirty-seven of the 50 notified HAV cases were investigated. Nine were overseas-acquired infections and a variety of other potential risk factors were identified. The number of cases reported nationally during this period was less than the historical averages. No outbreak investigation was required and enhanced surveillance was ceased on 18 September 2006.

In mid-August, New South Wales reported a case of hepatitis A that occurred in a person who had worked as a food handler in a school tuckshop. This case was identified as part of an investigation of a group of people that had contracted their illness while visiting Fiji. The case reported having worked for one day at the school tuckshop, whilst infectious, preparing ready-to-eat foods which presented an opportunity for hepatitis A transmission. Normal human immunoglobulin (NHIG) was offered to any member of the school community who may have consumed ready-to-eat foods prepared by the case at the tuckshop. NHIG is effective in preventing hepatitis A if given within two weeks of being exposed to the virus.[4] The school provided a letter to parents and students including fact sheets about hepatitis A and NHIG, and sought consent for students at risk to be passively immunised. Two clinics were held at the school where information and NHIG were given to 568 students, teachers and tuckshop workers. No secondary cases of hepatitis A were identified in relation to this incident.

Comments

New South Wales and Queensland initiated major public health responses to incidents of potential hepatitis A contamination of food. The need for a public health response after a case of hepatitis A in a traveller to Fiji highlights the importance of appropriate pre-travel vaccination for people travelling to countries where hepatitis A is endemic.[5] This is especially important for food handlers. It is important to educate both travellers and general practitioners about the value of individually targeted travel advice and appropriate prophylaxis or referral of patients to a travel health clinic.

Acknowledgements

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Communicable diseases surveillance

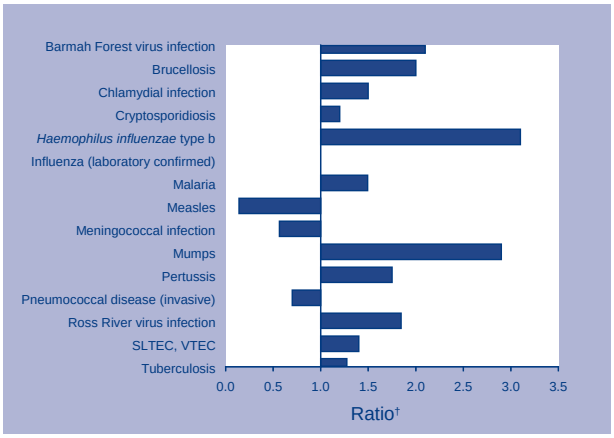
Highlights for 3rd quarter, 2006

Communicable diseases surveillance highlights report on data from various sources, including the National Notifiable Diseases Surveillance System (NNDSS) and several disease specific surveillance systems that provide regular reports to Communicable Diseases Intelligence. These national data collections are complemented by intelligence provided by state and territory communicable disease epidemiologists and data managers. This additional information has enabled the reporting of more informative highlights each quarter.

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. NNDSS collates data on notifiable communicable diseases from state and territory health departments. The Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme which collates information on laboratory diagnosis of communicable diseases. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', while data from the LabVISE scheme are referred to as 'laboratory reports'.

Figure 1 shows the changes in selected disease notifications with an onset in the third quarter of 2006, compared with the five-year mean for the same period. The following diseases were above the five-year mean: cryptosporidiosis, SLTEC/VTEC, chlamydial infection, gonococcal infection, *Haemophilus influenzae* type b infection, mumps, pertussis, Barmah Forest virus infection, malaria, Ross River virus infection, brucellosis, legionellosis and tuberculosis. Diseases for which the number of notifications was below the five-year mean for the same period include measles, meningococcal infection and invasive pneumococcal disease.

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 July to 30 September 2006 with historical data*



* Selected diseases are chosen each quarter according to current activity. Five year averages and the ratios of notifications in the reporting period in the five year mean should be interpreted with caution. Changes in surveillance practice, diagnostic techniques and reporting, may contribute to increases or decreases in the total notifications received over a five year period. Ratios are to be taken as a crude measure of current disease activity and may reflect changes in reporting rather than changes in disease activity.

† Ratio of current quarter total to mean of corresponding quarter for the previous five years.

‡ Some Victorian data for this period may be incomplete.

Gastrointestinal diseases

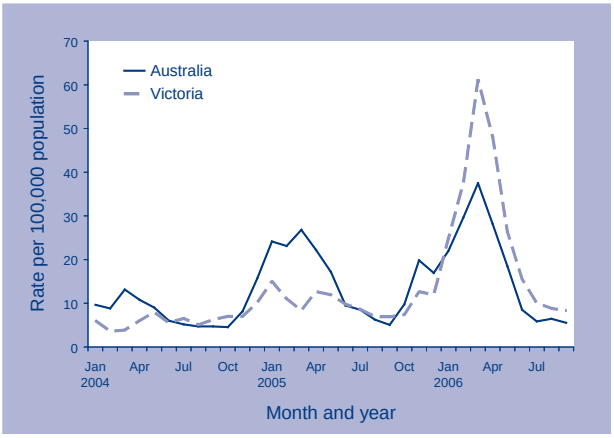
Cryptosporidiosis

There were 300 notifications of cryptosporidiosis between 1 July and 30 September 2006, which was 1.2 times the five-year mean for the third quarter. Nearly half of all cases were reported in Victoria (114 cases). This was a higher proportion than in the third quarter of 2005, when Victorian cases of cryptosporidiosis accounted for 94 of 336 notifications (28%).

There was a large decrease in the number of cryptosporidiosis notifications between the second and third quarters of 2006 (from 933 to 300); however, this is in line with the usual seasonal pattern (for example in 2005, notifications were 828 and 336 for the second and third quarters respectively) (Figure 2).

Three-quarters of the notifications had information on the infecting species, and all 224 of these were identified as *Cryptosporidium parvum,* which is the most important species in human disease (both the human and bovine genotypes).[1] Infection can be transmitted through contaminated food or water, through person-to-person or animal-to-person contact, or contact with contaminated environmental sources.

Figure 2. Cryptosporidiosis notification rates, January 2004 to September 2006, Australia and Victoria



Shiga-like toxin producing *Escherichia colil* verotoxin producing *E. coli*

There were 16 notifications of SLTEC/VTEC in the third quarter of 2006, which was 1.4 times the five-year mean. Half of the cases were from South Australia. Three cases from Queensland had serotype information, and these were all *E. coli* O111.

Sexually transmissible infections

Chlamydial infection

There were 11,343 notifications of chlamydial infection between 1 July and 30 September 2006, which was 1.5 times the five-year mean. More cases were reported in males (59%) than females. Over one-third (37%) of all chlamydial infections were in people aged 20–24 years.

The number of reported infections was less than in the previous quarter (down from 11,567) however, this was substantially higher than in the corresponding quarter of 2005 (a 12% increase from 10,146).

Vaccine preventable diseases

Haemophilus influenzae type b infection

There were 15 notifications of *Haemophilus influenzae* type b (Hib) infection between 1 July and 30 September 2006, which was 3.1 times the five-year mean. Nearly half of these cases (7) were from New South Wales and two-thirds of cases were in females. Three cases were in infants aged less than one year, with an additional four cases in children aged 1–5 years.

Indigenous status was recorded for 14 of the 15 cases; six notifications were in Indigenous people, including the three infants aged less than one year.

Routine vaccination against Hib became available in Australia in 1993. Vaccination status was available for all of the nine cases who were eligible for Hib immunisation; six cases were fully vaccinated for age (three Indigenous and three non-Indigenous cases).

Mumps

There were 90 notifications of mumps in the period 1 July to 30 September 2006, which was 2.9 times the five-year mean. Of these cases, 65 occurred in New South Wales. Half of all cases (46) occurred in people aged 25–34 years, and the median age of onset was 28 years. There were more cases among females (56%) than males.

Mumps rates were equivalent to 1.8 cases per 100,000 population per annum (ranging from no cases in Victoria or Tasmania to 5.8 cases per 100,000 population in the Northern Territory).

Vaccination status was known for 71 of the 90 notifications (79%); 12 cases were fully vaccinated for age and six partially vaccinated. Only two cases had received two doses of vaccine. Overall, 53 cases (59%) were not vaccinated.

The highest notification rate was 7.2 cases per 100,000 population in people aged 25–29 years. Mumps vaccine became available in Australia in 1980 for children aged 12–15 months, and was combined with the measles vaccine in 1982. As a result, few people in the 25–29 years age group would have received childhood vaccination against the mumps virus.

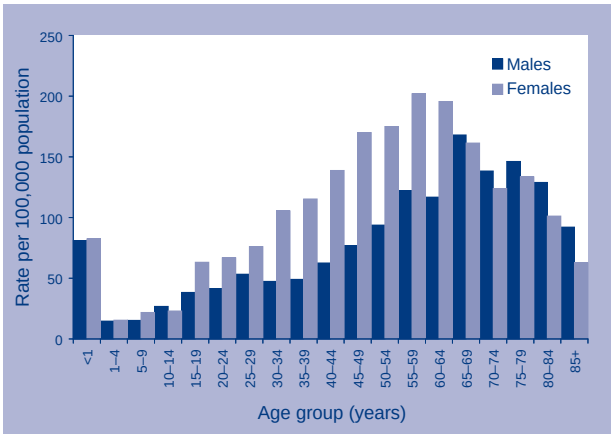
In the last few years, both the United States of America (USA) and the United Kingdom (UK) have reported increased mumps activity. In both regions, the majority of cases have occurred in college and university aged persons. In the USA for the period 1 January to 7 October 2006, the highest age-specific rate was among those aged 18–24 years.[2] Interestingly, in clusters occurring in August, a majority of cases had received two doses of the MMR vaccine. In the UK in 2004–2005, 79 per cent of confirmed cases of mumps were among those aged 15–24 years, a cohort which generally had not been eligible for routine mumps vaccination.[3]

Pertussis

There were 4,536 notifications of pertussis for the third quarter of 2006, which was 1.8 times the five-year mean. Of these, 53 cases (1.2%) were among infants aged less than one year, nearly half (49%)

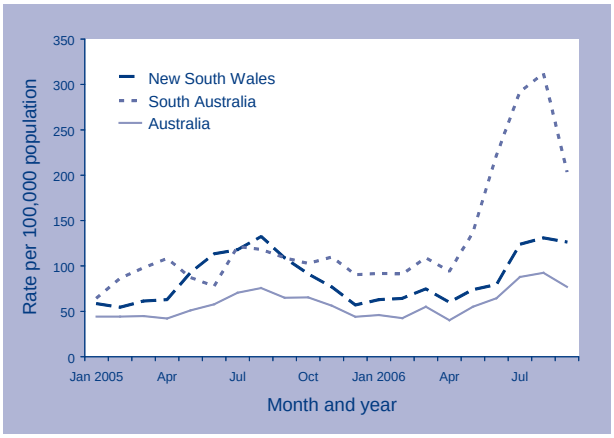
of whom were fully vaccinated for age. The average age at onset was 47 years, with the greatest number of notifications in people aged 55–59 years. The highest notification rate, however, was seen in those aged 65–69 years (165 cases per 100,000 population, annualised) (Figure 3).

Figure 3. Pertussis notification rates, 1 July to 30 September 2006, Australia, by age and sex



The overall rate of pertussis notifications was 88 cases per 100,000 population, ranging from 265 in South Australia to nine in Tasmania. The majority of pertussis notifications came from New South Wales (48%) and South Australia (23%) (Figure 4). Nearly two-thirds (62%) of notifications were for females.

Figure 4. Pertussis notification rates, January 2005 to September 2006, Australia, New South Wales and South Australia



Data from the NNDSS show that the age distribution of pertussis notifications changed between 2000 and 2006. In 2001, 41.5 per cent of notifications occurred in children aged less than 15 years, compared to 6.2 per cent in 2006 (The National Pertussis Report to CDNA, 12 August 2006). This observation is consistent with recent publications indicating that the epidemiology of pertussis is changing. Within Australia, the Hunter New England Area in New South Wales has reported a change in the age distribution of pertussis notifications over the period 1998 to 2005.[4] An increasing incidence of pertussis in adults has also been reported in other countries, including the USA and Germany.[5,6]

Part of the increase in notifications of pertussis may be due to false positive serology test results. In late September 2006, batches of PanBio Bordetella Pertussis IgA Elisa test kits were recalled because the cut-off determination point was set too low resulting in false positive results.[7]

Varicella

This report includes notifications of varicella infection for the first time. Varicella infection has become or is in the process of becoming notifiable in all jurisdictions except New South Wales. The primary purpose of surveillance of varicella infection is to monitor the impact of varicella immunisation, which was funded from November 2005 for all infants at 18 months of age and children 10 to 13 years who have not had chickenpox infection. These notifications include clinical reports from general practitioners which may or may not have been laboratory confirmed, and laboratory notifications with and without clinical information.

There were 1,752 notifications of varicella infection across Australia for the third quarter of 2006. These comprised 161 cases of chickenpox, 168 cases of zoster and 834 cases of laboratory-confirmed varicella zoster virus infection of unknown clinical diagnosis. These notifications are a small proportion of cases as surveillance in reporting jurisdictions is not yet fully implemented. No notifications were received from Victoria or the Australian Capital Territory.

Vectorborne diseases

Barmah Forest virus and Ross River virus infections

There were 353 notifications of Barmah Forest virus (BFV) infection and 338 notifications of Ross River virus (RRV) infection in the third quarter of 2006, which was 2.1 times the five-year mean for each disease. The majority of notifications came from Queensland (45% BFV and 42% RRV) and New South Wales (32% BFV and 23% RRV). While only 28 BFV notifications and 34 RRV notifications came from the Northern Territory, the annualised rates were substantially higher than in other jurisdictions at 54.4 cases per 100,000 population for BFV (com-

pared to 15.8 in Queensland and 6.6 in New South Wales) and 66.1 cases per 100,000 population for RRV (compared to 16.2 in Queensland and 5.2 in New South Wales).

Barmah Forest virus infection was reported more often for females than males (189 notifications versus 154). For both males and females, notification rates peaked for those aged 40–49 years (9.9 and 12.2 cases per 100,000 population respectively).

A similar number of notifications of RRV infection were reported for both males and females (191 and 197 notifications respectively). Notification rates peaked in women aged 40–49 years and also showed a lesser peak for those aged 70–79 years (12.7 and 10.1 cases per 100,000 population, respectively). Similarly, rates for males peaked in those aged 50–59 years and 70–79 years (11.6 and 9.3 cases per 100,000 population respectively).

Figures 5 and 6 show infection rates for BFV and RRV from 2004. Infection rates for both viruses are highest in the Northern Territory, and rates in Queensland are consistently above the national rate. Ross River virus infection rates peak in summer; in the Northern Territory the peak is seen around December while in Queensland the peak is seen around February. Barmah Forest virus infection rates tend to peak later (around March). Trend data indicate that current BFV infection rates in the Northern Territory are much higher than usually seen in September: 53.3 cases per 100,000 population in 2006 compared to 11.8 and 18.0 for 2005 and 2004 respectively. Similarly, Ross River virus infection rates in the Northern Territory appeared to increase earlier in 2006 than in previous years (100.6 cases per 100,000 population in September 2006 compared with 59.2 and 6.0 for 2005 and 2004 respectively).

The number of cases of RRV infection reported by NSW Health doubled from 582 in 2005 to 1,199 in 2006.[8] Cases of Barmah Forest virus infection also increased from 448 in 2005 to 634 in 2006 (an increase of 42%).[9] Notifications for both diseases peaked in March of 2006, with 299 cases of RRV and 110 cases of BFV. Notifications decreased over winter, as is the usual pattern.

Malaria

There were 208 notifications of malaria in the period 1 July to 30 September 2006, which was 1.5 times the five-year mean. Notifications peaked for people in the 20–24 year age group (7.7 cases per 100,000 population, annualised), and overall more males than females acquired the disease (148 cases versus 59).

Figure 5. Barmah Forest virus infection notification rates, January 2004 to September 2006, Australia, New South Wales, the Northern Territory and Queensland

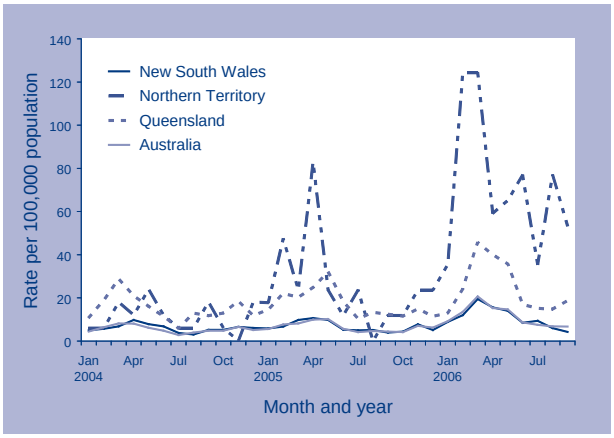
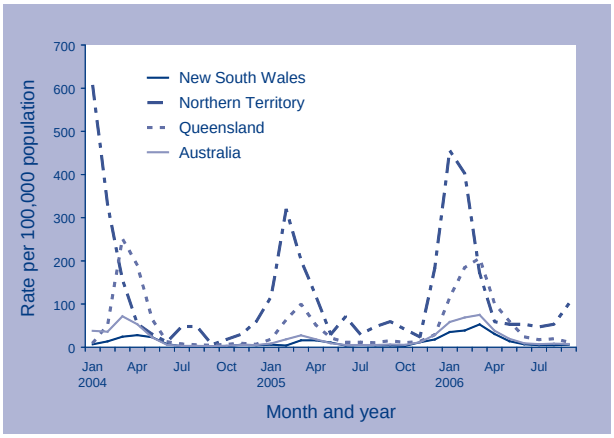


Figure 6. Ross River virus infection notification rates, January 2004 to September 2006, Australia, New South Wales, the Northern Territory and Queensland



Detailed place of acquisition information was available for 45 of the 61 cases notified in Queensland. Of these, 26 cases were acquired in Papua New Guinea.

Just over one-third of malaria notifications had the infecting organism identified; 45 were *Plasmodium falciparum*, 24 *P. vivax*, 2 *P. ovale*, 1 *P. malariae* and 7 mixed.

Zoonoses

Brucellosis

There were 14 notifications for cases of brucellosis between 1 July and 30 September 2006, which was 2.0 times the five-year mean. Ten of these cases were from Queensland.

Six cases had information on the infecting organism; three were due to *Brucella suis* and three due to *B. melitensis*.

Other bacterial infections

Legionellosis

There were 79 notifications of legionellosis for the third quarter of 2006, which was 1.2 times the five-year mean. Two-thirds of these cases were in males, and the median age at diagnosis was 65 years.

The infecting organisms responsible for these cases were *Legionella longbeachae* (42 cases), *L. pneumophila* (30 cases), *L. bozemanii* (1 case), 1 *L. micdadei* (1 case) and unspecified *Legionella* (5 cases).

There were three deaths due to legionellosis during this reporting period, all in Western Australia and attributed to *L. longbeachae*, in a 63-year-old male, a 75-year-old male and a 76-year-old female. While Western Australia reported an increased number of cases in September, all of these were sporadic.

Meningococcal infection

There were 111 notifications for cases of meningococcal infection in the third quarter of 2006—equivalent to 2.2 cases per 100,000 population per year—which was 0.6 times the five-year mean. Just over two-thirds of meningococcal infections were serogroup type B (76 cases, 68%), 8 cases (7%) were type C and 15 (14%) were of unknown type.

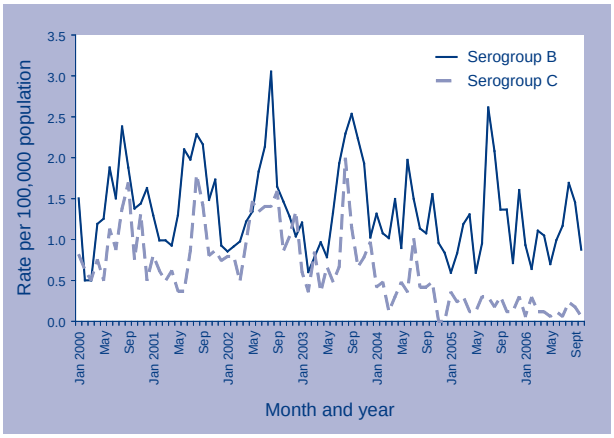
Nearly one-third of all cases (32%) were in children aged less than 5 years, and overall almost three-quarters of cases (72%) were in people aged less than 25 years.

There were three deaths all due to meningococcal type B infection during this period: in a 4-month-old female in New South Wales, a 2-year-old male in Queensland and a 19-year-old male in Victoria.

Serogroups B and C are the most common types of meningococcal infection in Australia.[10] It is important to note that vaccination against meningococcal type C – but not type B – is currently available in Australia (one type of the vaccine also protects against serogroups A, W135 and Y, however it not used for routine vaccination).

Cases of meningococcal type C have been decreasing over the last few years; there were 72 notifications in the third quarter of 2002, decreasing to eight for the current quarter. Routine vaccination against meningococcal type C for those aged 12 months or 15 years was introduced in 2003; catch-up vaccinations were available in 2003 to those aged 16–17 years. Figure 7 shows that since the introduction of the vaccination program, rates of meningococcal type C have declined, while the fluctuating pattern meningococcal type B infection rates has not shown much change.

Figure 7. Meningococcal infection rates for serogroups B and C, 2000 to 2006



Tuberculosis

There were 333 notifications of tuberculosis in the third quarter of 2006, equivalent to 6.5 cases per 100,000 population per annum, which was 1.3 times the five-year third-quarter mean.

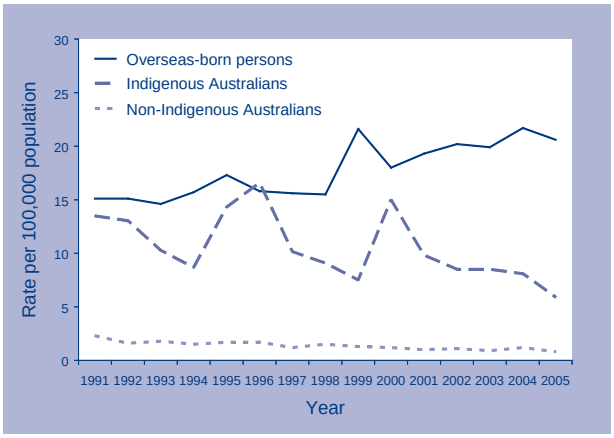
The average age at onset was 44 years, and the greatest number of notifications was for people aged 25–29 years (49 cases). Notification rates peaked in those aged 70–79 years (18.7 cases per 100,000 population). A total of 11 cases were among Indigenous people (3.3%) and 300 in non-Indigenous people (90%); Indigenous status was unknown for 22 cases (6.6%).

The majority of cases came from New South Wales (36%) and Victoria (34%). In 2005, 86 per cent of tuberculosis cases were in people who were overseas born (Paul Roche, personal communication). In Victoria, an increasing number of tuberculosis cases were in recently arrived refugees. While a number of cases were found through screening, a considerable number were found through clinical presentation, often soon after arrival in Australia. These were mostly new cases, rather than relapse (Lynne Brown, personal communication).

There were two deaths due to tuberculosis in this reporting period: a 49-year-old female in Victoria and a 79-year-old male in Tasmania.

Figure 8 shows tuberculosis notification rates from 1991 to 2005. Rates for non-Indigenous Australians have declined over this time, from 2.3 cases per 100,000 in 1991 to 0.8 in 2005. Rates for Indigenous Australians were subject to greater fluctuations, however overall also showed a decline, from 13.5 cases per 100,000 in 1991 to 5.9 in 2005. Conversely, notification rates in overseas-born persons have shown a steady increase from 15.1 cases per 100,000 in 1991 to 20.6 in 2005.

Figure 8. Tuberculosis notification rates by ethnicity, 1991 to 2005



Acknowledgments

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Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 35,834 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 July and 30 September 2006 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 6,360 reports re`ceived by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 July to 30 September 2006 (Tables 4 and 5).

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis B (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (incident)	All jurisdictions except Qld
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except NSW
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
SLTEC, VTEC	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Smallpox	All jurisdictions
Tularemia	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 (all)	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years or unspecified duration	All jurisdictions
Syphilis - congenital	All jurisdictions

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 infection (chickenpox)	All jurisdictions except NSW
Varicella infection (unspecified)	All jurisdictions except NSW
Varicella zoster infection	All jurisdictions except NSW
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Flavivirus infection (NEC)†	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis virus	All jurisdictions
Kunjin virus	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssaviruses unspecified	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Laboratory confirmed influenza is not notifiable in South Australia but reports are forwarded to NNDSS.

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

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 July to 30 September 2006, by date of onset*

Disease	State or territory								Total 3rd quarter 2006†	Total 2nd quarter 2006	Total 3rd quarter 2005	Last 5 years mean 3rd quarter	Year to date 2006	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	5	7	2	9	3	2	22	12	62	82	61	87.2	213	265.4	0.7
Hepatitis B (unspecified)	14	820	54	284	75	14	381	205	1,847	1,524	1,662	1,695.4	4,837	4,957.4	1.1
Hepatitis C (incident)	2	5	0	0	10	3	37	23	80	110	93	127.6	317	379.6	0.6
Hepatitis C (unspecified)	41	1,600	55	786	107	43	693	317	3,642	2,818	3,112	3,656.2	9,661	11,169.6	1.0
Hepatitis D	0	5	0	1	0	0	4	0	10	6	16	9.6	26	21.0	1.0
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	1	0.2	0	1.3	0.0
Campylobacteriosis§	87	NN	70	987	701	130	1,492	406	3,873	3,359	3,794	3,698.2	10,938	11,164.4	1.0
Cryptosporidiosis	2	43	2	73	13	3	114	50	300	933	336	249.4	2,743	1,763.6	1.2
Haemolytic uraemic syndrome	0	0	0	0	0	0	0	0	0	0	4	3.0	5	8.6	0.0
Hepatitis A	0	23	2	8	2	0	9	12	56	64	85	94.4	226	304.0	0.6
Hepatitis E	0	2	0	0	0	0	3	1	6	4	5	5.0	17	17.2	1.2
Listeriosis	0	7	0	1	2	0	3	1	14	7	9	12.8	46	47.0	1.1
Salmonellosis (NEC)	25	305	68	365	92	16	228	141	1,240	1,859	1,312	1,167.4	6,160	5,705.2	1.1
Shigellosis	0	10	19	17	9	1	11	30	97	137	152	114.8	421	428.8	0.8
SLTEC, VTEC‖	0	0	2	5	8	0	0	1	16	17	16	11.8	49	43.0	1.4
Typhoid	0	7	1	0	0	0	3	1	12	21	10	13.8	52	51.4	0.9
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	0	1.4	0	3.4	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Tularemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 July to 30 September 2006, by date of onset,* *continued*

Disease	State or territory								Total 3rd quarter 2006†	Total 2nd quarter 2006	Total 3rd quarter 2005	Last 5 years mean 3rd quarter	Year to date 2006	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	199	2,821	451	2,947	716	241	2,454	1,514	11,343	11,567	10,146	7,633.2	34,790	23,001.2	1.5
Donovanosis	0	0	0	0	0	0	0	0	0	2	3	4.2	2	13.8	0.0
Gonococcal infection	7	369	395	380	86	3	264	384	1,888	2,371	1,910	1,673.2	6,655	5,244.4	1.1
Syphilis (all)	4	205	48	93	10	6	161	29	556	637	560	521.2	1,813	1,563.8	1.1
Syphilis < two years duration	1	21	31	32	0	0	65	11	161	189	159	164.5	515	467.0	1.0
Syphilis > two years or unspecified duration	3	184	17	61	10	6	96	18	395	448	401	411.0	1,298	1,199.0	1.0
Syphilis - congenital	0	0	1	0	0	0	0	0	1	6	1	3.8	11	11.8	0.3
Vaccine preventable disease															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.2	NA
Haemophilus influenzae type b	0	7	0	4	0	0	1	3	15	2	4	4.8	19	17.2	3.1
Influenza (laboratory confirmed)	19	433	16	1,202	66	33	262	129	2,160	371	2,971	2,139.6	2,710	2,670.4	1.0
Measles	0	1	0	0	0	0	1	0	2	95	1	14.2	114	49.2	0.1
Mumps	1	65	3	8	12	0	0	1	90	90	78	31.0	223	95.2	2.9
Pertussis	79	2,170	25	687	1,028	11	466	70	4,536	2,727	3,579	2,463.4	9,699	5,463.4	1.8
Pneumococcal disease (invasive)	2	207	21	119	43	16	79	43	530	405	645	811.2	1,141	1,641.4	0.7
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Rubella	0	13	0	6	0	0	4	1	24	16	8	33.6	46	94.4	0.7
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	1	0.4	0	1.0	0.0
Tetanus	0	0	0	0	0	0	0	0	0	0	0	0.2	1	2.6	0.0
Varicella infection (chickenpox)	0	NN	91	145	152	7	0	55	450	161	0	NA	730	NA	NA
Varicella infection (unspecified)	0	NN	2	904	65	9	0	8	988	834	0	NA	2,677	NA	NA
Varicella zoster infection	0	NN	27	82	177	13	0	15	314	168	0	NA	641	NA	NA
Vectorborne diseases															
Barmah Forest virus infection	1	112	28	159	30	0	3	20	353	651	224	165.2	1,738	954.6	2.1
Dengue	1	16	4	14	0	0	0	3	38	56	34	33.2	152	280.8	1.1
Flavivirus infection (NEC)	0	0	0	4	0	0	0	0	4	10	8	13.8	29	51.6	0.3
Japanese encephalitis virus	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.4	NA
Kunjin virus‖	0	0	0	0	0	0	0	0	0	3	0	0.2	4	6.6	0.0
Malaria	2	38	20	61	14	6	36	31	208	207	162	134.6	631	496.4	1.5
Murray Valley encephalitis virus	0	0	0	0	0	0	0	0	0	1	0	0.2	1	2.2	0.0
Ross River virus infection	3	88	34	163	35	0	7	58	388	1,135	284	182.6	4,944	2,651.8	2.1

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 July to 30 September 2006, by date of onset,* *continued*

Disease	State or territory								Total 3rd quarter 2006†	Total 2nd quarter 2006	Total 3rd quarter 2005	Last 5 years mean 3rd quarter	Year to date 2006	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	1	0.0	NA
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Brucellosis	0	3	0	10	0	0	0	1	14	5	7	7.0	33	21.4	2.0
Leptospirosis	0	1	0	13	1	0	3	2	20	58	25	28.4	128	139.4	0.7
Lyssavirus unspecified	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	NA
Ornithosis	0	16	0	0	0	1	16	0	33	40	42	56.0	116	145.8	0.6
Q fever	0	43	1	42	7	0	7	0	100	88	74	122.8	289	430.6	0.8
Other bacterial infections															
Legionellosis	0	8	1	20	19	0	9	22	79	73	83	68.0	252	234.2	1.2
Leprosy	0	0	0	0	0	0	0	1	1	2	2	1.6	4	6.2	0.6
Meningococcal infection**	3	42	1	26	5	0	27	7	111	68	141	190.8	254	418.4	0.6
Tuberculosis	4	119	10	42	19	2	113	24	333	278	270	251.0	905	729.6	1.3
Total	501	9,611	1,454	9,667	3,507	560	6,913	3,621	35,834	33,069	31,931	27,537.0	106,544	82,770.5	1.3

* Date of onset = the true onset. If this is not available, the 'date of onset' is equivalent to the earliest of two dates: (i) specimen date of collection, or (ii) the date of notification to the public health unit. Hepatitis B and C unspecified were analysed by the date of notification.

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

‡ Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter. Note: Ratios for syphilis <2 years; syphilis >2 years or unspecified duration are based on 2 years data.

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

‖ Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/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 which excludes ocular specimens, and Western Australia which excludes ocular and perinatal infections.

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

NN Not notifiable.

NEC Not elsewhere classified.

Table 3. Notification rates of diseases, 1 July to 30 September 2006, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis B (incident)	6.1	0.4	3.9	0.9	0.8	1.6	1.7	2.3	1.2
Hepatitis B (unspecified)	17.1	48.1	104.9	28.1	19.3	11.5	30.0	40.1	35.9
Hepatitis C (incident)	2.4	0.3	0.0	0.0	2.6	2.5	2.9	4.5	1.6
Hepatitis C (unspecified)	50.0	93.9	106.8	77.9	27.6	35.2	54.6	62.1	70.9
Hepatitis D	0.0	0.3	0.0	0.1	0.0	0.0	0.3	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	106.1	NN	136.0	97.8	180.6	106.4	117.5	79.5	75.4
Cryptosporidiosis	2.4	2.5	3.9	7.2	3.3	2.5	9.0	9.8	5.8
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis A	0.0	1.3	3.9	0.8	0.5	0.0	0.7	2.3	1.1
Hepatitis E	0.0	0.1	0.0	0.0	0.0	0.0	0.2	0.2	0.1
Listeriosis	0.0	0.4	0.0	0.1	0.5	0.0	0.2	0.2	0.3
Salmonellosis (NEC)	30.5	17.9	132.1	36.2	23.7	13.1	18.0	27.6	24.1
Shigellosis	0.0	0.6	36.9	1.7	2.3	0.8	0.9	5.9	1.9
SLTEC, VTEC‡	0.0	0.0	3.9	0.5	2.1	0.0	0.0	0.2	0.3
Typhoid	0.0	0.4	1.9	0.0	0.0	0.0	0.2	0.2	0.2
Quarantinable diseases									
Cholera	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
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tularemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmissible infections									
Chlamydial infection§	242.6	165.5	876.1	292.1	184.5	197.3	193.3	296.5	220.8
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	8.5	21.7	767.3	37.7	22.2	2.5	20.8	75.2	36.7
Syphilis (all)	4.9	12.0	93.2	9.2	2.6	4.9	12.7	5.7	10.8
Syphilis < 2 years duration	1.2	1.2	60.2	3.2	0.0	0.0	5.1	2.2	3.1
Syphilis > 2 years or unspecified duration	3.7	10.8	33.0	6.0	2.6	4.9	7.6	3.5	7.7
Syphilis - congenital	0.0	0.0	1.9	0.0	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.4	0.0	0.4	0.0	0.0	0.1	0.6	0.3
Influenza (laboratory confirmed)	23.2	25.4	31.1	119.1	17.0	27.0	20.6	25.3	42.0
Measles	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Mumps	1.2	3.8	5.8	0.8	3.1	0.0	0.0	0.2	1.8
Pertussis	96.3	127.3	48.6	68.1	264.9	9.0	36.7	13.7	88.3
Pneumococcal disease (invasive)	2.4	12.1	40.8	11.8	11.1	13.1	6.2	8.4	10.3

Table 3. Notification rates of diseases, 1 July to 30 September 2006, by state or territory. (Annualised rate per 100,000 population), *continued*

Disease*	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, *continued*									
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.8	0.0	0.6	0.0	0.0	0.3	0.2	0.5
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Varicella infection (chickenpox)	0.0	NN	176.8	14.4	39.2	5.7	0.0	10.8	8.8
Varicella infection (unspecified)	0.0	NN	3.9	89.6	16.7	7.4	0.0	1.6	19.2
Varicella zoster infection	0.0	NN	52.4	8.1	45.6	10.6	0.0	2.9	6.1
Vectorborne diseases									
Barmah Forest virus infection	1.2	6.6	54.4	15.8	7.7	0.0	0.2	3.9	6.9
Dengue	1.2	0.9	7.8	1.4	0.0	0.0	0.0	0.6	0.7
Flavivirus infection (NEC)	0.0	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.1
Japanese encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	2.4	2.2	38.9	6.0	3.6	4.9	2.8	6.1	4.0
Murray Valley encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	3.7	5.2	66.0	16.2	9.0	0.0	0.6	11.4	7.6
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.2	0.0	1.0	0.0	0.0	0.0	0.2	0.3
Leptospirosis	0.0	0.1	0.0	1.3	0.3	0.0	0.2	0.4	0.4
Lyssavirus unspecified	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.9	0.0	0.0	0.0	0.8	1.3	0.0	0.6
Q fever	0.0	2.5	1.9	4.2	1.8	0.0	0.6	0.0	1.9
Other bacterial infections									
Legionellosis	0.0	0.5	1.9	2.0	4.9	0.0	0.7	4.3	1.5
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Meningococcal infection‖	3.7	2.5	1.9	2.6	1.3	0.0	2.1	1.4	2.2
Tuberculosis	4.9	7.0	19.4	4.2	4.9	1.6	8.9	4.7	6.5

* Rates are subject to retrospective revision.

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

‡ Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/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 which excludes ocular specimens, and Western Australia which excludes ocular and perinatal infections.

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

NN Not notifiable.

NEC Not elsewhere classified.

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 July to 30 September 2006, and total reports for the year†

	State or territory								This period 2006	This period 2005	Year to date 2006	Year to date 2005
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	–	–	1	–	–	1	–	2	1	54	4
Mumps virus	–	–	–	–	2	–	–	–	2	7	25	25
Rubella virus	–	–	–	3	–	–	1	1	5	4	13	11
Hepatitis viruses												
Hepatitis A virus	–	1	1	4	–	–	–	2	8	21	24	38
Hepatitis D virus	–	–	–	–	–	–	1	–	1	6	5	12
Hepatitis E virus	–	–	–	–	–	–	1	–	1	2	5	11
Arboviruses												
Ross River virus	–	2	8	18	8	–	–	8	44	51	1,021	333
Barmah Forest virus	–	–	–	11	26	–	–	–	37	28	266	158
Flavivirus (unspecified)	–	–	–	3	–	–	1	–	4	9	43	29
Adenoviruses												
Adenovirus type 1	–	–	–	–	–	–	2	–	2	5	3	6
Adenovirus not typed/ pending	2	116	–	14	56	–	42	–	230	213	490	498
Herpes viruses												
Cytomegalovirus	–	65	1	17	102	2	30	3	220	318	738	729
Varicella-zoster virus	1	40	–	134	98	5	9	–	287	381	897	1,111
Epstein-Barr virus	–	2	26	89	123	1	1	123	365	562	1,183	1,543
Other DNA viruses												
Molluscum contagiosum	–	–	–	–	–	–	1	–	1	–	1	–
Poxvirus group not typed	–	–	–	–	–	–	2	–	2	1	2	2
Parvovirus	–	2	–	34	19	–	6	–	61	44	149	123
Picornavirus family												
Coxsackievirus A9	–	6	–	–	–	–	–	–	6	–	11	2
Echovirus type 3	–	2	–	–	–	–	–	–	2	–	2	–
Echovirus type 8	–	1	–	–	–	–	–	–	1	–	1	–
Echovirus type 18	–	1	–	–	–	–	–	–	1	3	2	13
Rhinovirus (all types)	–	95	–	–	2	1	1	1	100	75	142	246
Enterovirus not typed/ pending	–	8	–	1	–	2	7	–	18	76	94	141
Picornavirus not typed	–	–	–	–	–	1	–	–	1	–	2	1
Ortho/paramyxoviruses												
Influenza A virus	2	58	–	61	24	4	79	–	228	527	300	647
Influenza A virus H3N2	–	1	–	–	–	–	–	–	1	2	1	2
Influenza B virus	–	33	–	14	68	–	7	–	122	148	166	230
Parainfluenza virus type 1	–	1	–	–	14	–	1	–	16	17	74	46
Parainfluenza virus type 2	–	3	–	–	2	–	–	–	5	13	12	46
Parainfluenza virus type 3	1	44	–	11	18	–	15	–	89	175	114	274
Respiratory syncytial virus	–	536	–	65	298	20	267	–	1,186	718	1,747	1,550
Other RNA viruses												
Rotavirus	3	366	–	–	184	47	137	1	738	760	869	999
Norwalk agent	–	18	–	–	–	–	411	–	429	68	1,110	163

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 July to 30 September 2006, and total reports for the year,† *continued*

	State or territory								This period 2006	This period 2005	Year to date 2006	Year to date 2005
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other												
Chlamydia trachomatis not typed	3	279	–	307	341	5	6	–	941	1,239	3,412	3,778
Chlamydia pneumoniae	–	–	–	–	–	–	1	–	1	2	1	6
Chlamydia psittaci	–	2	–	–	–	–	15	–	17	8	43	38
Chlamydia species	–	1	–	–	–	–	–	–	1	–	2	–
Mycoplasma pneumoniae	–	7	2	99	109	4	39	34	294	425	901	927
Mycoplasma hominis	–	10	–	–	–	–	–	–	10	2	20	4
Coxiella burnetii (Q fever)	–	3	–	6	8	1	5	–	23	34	93	121
Rickettsia tsutsugamushi	–	–	–	–	2	–	–	–	2	27	23	46
Rickettsia - spotted fever group	–	–	–	–	18	1	–	–	19	81	85	178
Streptococcus group A	–	–	–	65	–	–	–	–	65	189	329	431
Yersinia enterocolitica	–	1	–	–	–	–	–	–	1	–	5	6
Brucella species	–	2	–	–	–	–	–	–	2	6	5	9
Bordetella pertussis	1	15	–	38	498	–	–	–	552	416	1,224	1,167
Legionella pneumophila	–	1	–	–	5	–	–	–	6	3	25	17
Legionella longbeachae	–	–	–	–	4	–	1	–	5	18	15	37
Cryptococcus species	–	1	–	1	2	–	–	–	4	4	19	29
Leptospira species	–	–	–	3	2	–	–	–	5	7	16	23
Treponema pallidum	–	56	1	61	74	–	1	–	193	254	688	835
Entamoeba histolytica	–	–	–	1	–	–	–	–	1	4	1	12
Toxoplasma gondii	–	1	–	–	–	–	2	–	3	11	36	31
Total	13	1,780	39	1,061	2,107	94	1,093	173	6,360	6,965	16,509	16,688

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

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

– No data received this period.

Table 5. Virology and serology reports by laboratories for the reporting period 1 July to 30 September 2006*

State or territory	Laboratory	July 2006	August 2006	September 2006	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	129	172	152	453
	New Children's Hospital, Westmead	262	202	148	612
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	45	36	44	125
	South West Area Pathology Service, Liverpool	209	194	176	579
Queensland	Queensland Medical Laboratory, West End	277	420	404	1,101
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	2,105	–	–	2,105
Tasmania	Northern Tasmanian Pathology Service, Launceston	32	39	23	94
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	67	70	23	160
	Royal Children's Hospital, Melbourne	139	170	127	436
	Victorian Infectious Diseases Reference Laboratory, Fairfield	243	88	158	489
Western Australia	PathCentre Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	35	101	70	206
Total		3,543	1,492	1,325	6,360

* The complete list of laboratories reporting for the 12 months, January to December 2006, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– No data received this period.

Additional reports

Australian Sentinel Practice Research Network

The Research and Health Promotion Unit of the Royal Australian College of General Practitioners operates the Australian Sentinel Practice Research Network (ASPREN). ASPREN is a network of general practitioners who report presentations of defined medical conditions each week. The aim of ASPREN is to provide an indicator of the burden of disease in the primary health setting and to detect trends in consultation rates.

There are currently about 40 general practitioners participating in the network from all states and territories. Seventy-five per cent of these are in metropolitan areas and the remainder are rural based. Between 3,000 and 4,000 consultations are recorded each week.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published.

In 2006, six conditions are being monitored, four of which are related to communicable diseases. These include influenza, gastroenteritis, varicella and shingles. Definitions of these conditions were published in Commun Dis Intell *2006;30:158.*

Data from 1 January to 30 September 2006 compared with 2005 are shown as the rate per 1,000 consultations in Figures 9 and 10.

Figure 9. Consultation rates for gastroenteritis, ASPREN, 1 January to 30 September 2006, by week of report

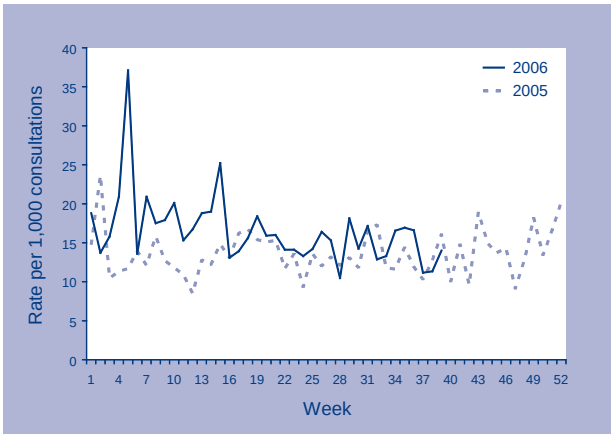
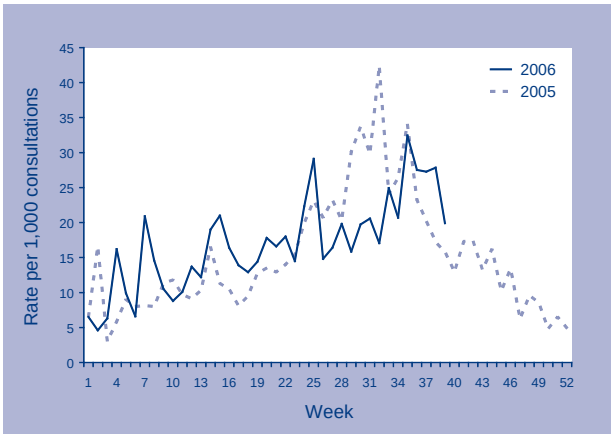


Figure 10. Consultation rates for influenza-like illness, ASPREN, 1 January to 30 September 2006, by week of report



Childhood immunisation coverage

Tables 6, 7 and 8 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 of age for the cohort born between 1 April and 30 June 2005, at 24 months of age for the cohort born between 1 April and 30 June 2004, and at 6 years of age for the cohort born between 1 April and 30 June 2000 according to the Australian Standard Vaccination Schedule.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, *published in* Commun Dis Intell *2006;30:157 and for a full description of the methodology used by the Register see* Commun Dis Intell *1998;22:36-37.*

Commentary on the trends in ACIR data is provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS). For further information please contact the NCIRS at telephone: +61 2 9845 1435, Email: brynleyh@chw.edu.au.

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia increased marginally by 0.1 percentage points to 90.8 per cent (Table 6), whilst there were no important changes in coverage for all individual vaccines due at 12 months of age. There were no significant movements in coverage for individual vaccines by jurisdiction.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia decreased marginally from the last quarter by 0.2 percentage points to 92.2 per cent (Table 7). There were no significant changes in coverage in any jurisdiction for 'fully immunised' coverage or for coverage for individual vaccines. It is notable that the estimate for 'fully immunised' at 24 months of age has been higher than the 12 months coverage estimate since the 18 month DTPa booster was no longer required from September, 2003.

It is also notable that, for the two vaccines where no further doses are due between 6 months and 24 months of age (DTP and polio), coverage at the national level was 95.1 per cent and 95.0 per cent respectively at 24 months versus 91.9 and 91.8 per cent at 12 months. This suggests that delayed notification or delayed vaccination is making an important contribution to the coverage estimates at 12 months of age and that the 'fully immunised' estimate in particular is likely to be a minimum estimate.

Table 8 shows immunisation coverage estimates for 'fully immunised' and for individual vaccines at six years of age for Australia and by state or territory. Surprisingly, 'fully immunised' coverage for Australia increased significantly by 3.5 percentage points and is now at the highest level ever recorded since it was first reported in early 2003. Coverage increased significantly in almost all jurisdictions and for all individual vaccines except in the Northern Territory where it decreased by 2.5 percentage points. Tasmania, Queensland and the Australian Capital Territory experienced the most significant increases for 'fully immunised' coverage, 6, 4.4 and 4.4 percentage points respectively. A possible factor in this increase in coverage at 6 years of age is the introduction of the multi-valent combination vaccine Infanrix-IPV onto the schedule that occurred in November 2005, reducing the number of vaccines to be recorded from three to two. Other factors which may have had an impact at the local level include promotional campaigns centred around childcare or school entry or data cleaning activities.

Figure 11 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 6 years, although the rate of increase has slowed over the past two years in all age groups. There have now been 12 consecutive quarters where 'fully immunised' coverage at 24 months has been greater than 'fully immunised' coverage at 12 months, following the removal of the requirement for the 18 month DTPa vaccine. Both measures have been above 90 per cent for this period. Currently, coverage for meningococcal C conjugate at 12 months and pneumococcal conjugate at 2, 4, and 6 months, is not included in the 12 or 24 months coverage data respectively.

Figure 11. Trends in vaccination coverage, Australia, 1997 to 2006, by age cohorts

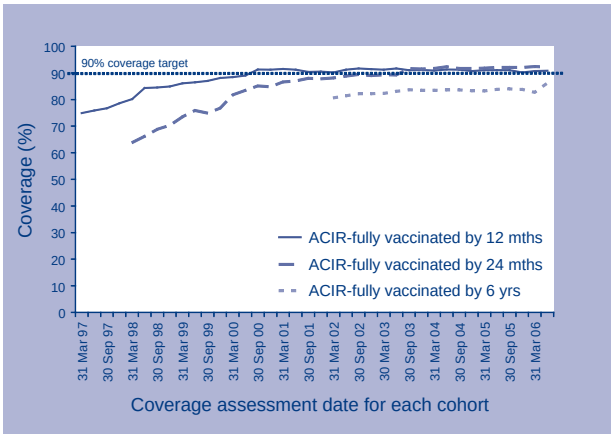


Table 6. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 April to 30 June 2005; assessment date 30 September 2006

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Number of children	1,036	22,738	944	14,415	4,481	1,495	16,140	6,663	67,912
Diphtheria, tetanus, pertussis (%)	91.3	91.7	91.3	91.9	91.8	94.3	92.7	90.1	91.9
Poliomyelitis (%)	91.1	91.6	91.1	91.9	91.6	94.1	92.6	90.1	91.8
Haemophilus influenzae type b (%)	94.7	94.5	95.8	94.1	94.1	96.1	94.7	93.6	94.4
Hepatitis B (%)	94.8	94.8	95.9	93.9	94.2	95.9	94.5	93.6	94.4
Fully immunised (%)	90.6	90.9	90.6	90.4	90.6	93.8	91.3	89.4	90.8
Change in fully immunised since last quarter (%)	-0.1	+0.8	-0.0	-0.4	-0.4	0.0	-0.5	+0.2	+0.1

Table 7. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 April to 30 June 2004; assessment date 30 September 2006*

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	983	20,991	884	13,039	4,257	1,338	15,157	6,519	63,168
Diphtheria, tetanus, pertussis (%)	95.8	94.7	96.6	94.6	95.3	96.0	96.0	94.7	95.1
Poliomyelitis (%)	95.9	94.6	96.2	94.5	95.2	96.0	96.0	94.6	95.0
Haemophilus influenzae type b (%)	94.7	93.0	95.1	93.5	93.9	94.7	94.7	92.9	93.7
Measles, mumps, rubella (%)	94.9	93.1	95.7	93.5	94.4	94.5	95.0	93.4	93.9
Hepatitis B(%)	95.8	95.5	97.3	95.3	95.9	96.3	96.6	95.1	95.8
Fully immunised (%)	93.8	91.4	94.6	91.6	92.7	93.8	93.7	91.2	92.2
Change in fully immunised since last quarter (%)	-0.4	-0.3	+0.1	-0.6	+0.5	+0.2	+0.1	-0.1	-0.2

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2005;29:434.

Table 8. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 April to 30 June 2000; assessment date 30 September 2006

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,082	22,382	870	13,573	4,597	1,562	15,821	6,606	66,493
Diphtheria, tetanus, pertussis (%)	88.9	87.0	82.9	87.0	85.2	89.2	89.6	81.9	87.0
Poliomyelitis (%)	88.6	87.0	83.9	87.2	85.3	89.5	89.7	82.0	87.1
Measles, mumps, rubella (%)	88.6	87.0	83.5	87.3	85.0	89.7	89.7	82.0	87.1
Fully immunised (%)[1]	87.6	86.2	82.2	86.2	84.5	88.6	89.0	80.7	86.2
Change in fully immunised since last quarter (%)	+4.4	+3.1	-2.5	+4.4	+2.2	+6.0	+3.9	+3.4	+3.5

Gonococcal surveillance

John Tapsall, The Prince of Wales Hospital, Randwick NSW 2031 for the Australian Gonococcal Surveillance Programme.

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see Commun Dis Intell *2006;30:157.*

NOTE. This report completes 25 years of continuous quarterly surveillance by the AGSP.

Reporting period 1 April to 30 June 2006

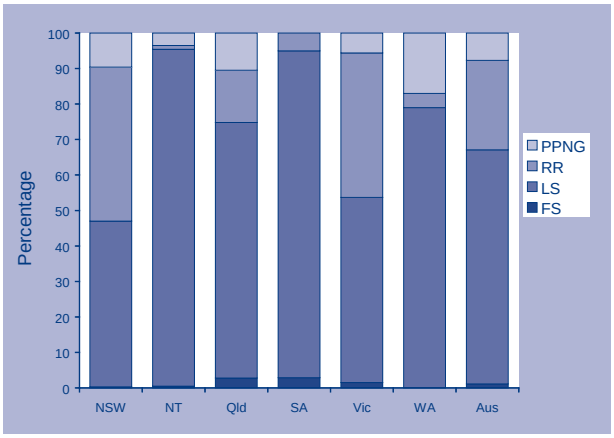
The AGSP laboratories received a total of 1,144 isolates in this quarter of which 1,107 underwent susceptibility testing. This was about 11 per cent more than the 1,028 gonococci reported for the same period in 2005. About 27 per cent of this total was from New South Wales, 23 per cent from Victoria, 16 per cent from the Northern Territory, 13 per cent from Queensland and 9 per cent from each of Western Australia and South Australia. Small numbers of isolates were also received from Tasmania and the Australian Capital Territory.

Penicillins

In this quarter 363 (32.8%) isolates examined were penicillin resistant by one or more mechanisms. Eighty-four (7.6%) were penicillinase-producing *Neisseria gonorrhoeae* (PPNG) and 279 (25.2%) resistant by chromosomal mechanisms, (CMRNG). While the number and proportion of PPNG was essentially unchanged from the equivalent period in 2005, the number and proportion of CMRNG increased from the 188 (18.7%) seen last year. The proportion of all strains resistant to the penicillins by any mechanism ranged from 4.5 per cent in the Northern Territory to 53 per cent in New South Wales. High rates of penicillin resistance were also found in Victoria (43%), Queensland (25%) and Western Australia (21%).

Figure 12 shows the proportions of gonococci fully sensitive (MIC ≤0.03 mg/L), less sensitive (MIC 0.06–0.5 mg/L), relatively resistant (MIC ≥1 mg/L) or else PPNG, aggregated for Australia and by state and territory. A high proportion those strains classified as PPNG or CMRNG fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

Figure 12. Categorisation of gonococci isolated in Australia, 1 April to 30 June 2006, by penicillin susceptibility and region



FS Fully sensitive to penicillin, MIC ≤0.03 mg/L.

LS Less sensitive to penicillin, MIC 0.06–0.5 mg/L.

RR Relatively resistant to penicillin, MIC ≥1 mg/L.

PPNG Penicillinase producing *Neisseria gonorrhoeae.*

In New South Wales and Victoria most of the penicillin resistance was due to CMRNG. In New South Wales (131, 43%) were CMRNG with 29 PPNG (9.6%) and in Victoria 109 (40%) were CMRNG and 15 (5.6%) PPNG. In Queensland 21 (14.7%) isolates were CMRNG and 15 (10.5%) were PPNG. In Western Australia PPNG were more prominent (17% of the 100 isolates) with 4 per cent CMRNG. All five resistant strains in South Australia were CMRNG. Six of the eight penicillin resistant isolates in the Northern Territory were from Darwin and five of these were PPNG. Both PPNG and CMRNG were reported from Tasmania and the Australian Capital Territory.

Ceftriaxone

Seven isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected, five in New South Wales and one each in Queensland and Western Australia.

Spectinomycin

All isolates were susceptible to this injectable agent.

Quinolone antibiotics

The total number and proportion, (373, 33.7%) of quinolone resistant *N. gonorrhoeae* (QRNG) continued to increase when compared with corresponding figures in the second quarter of recent years. In 2005, 307 (30%) QRNG were detected, in 2004 there were 172 (20%) and in 2003, 135 (14%). The majority of QRNG in the current period (367, 98%) exhibited higher-level resistance (ciprofloxacin MICs 1 mg/L or more). QRNG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥1 mg/L) groups.

QRNG were again widely distributed and were detected in all states and territories (Figure 13). The highest number (167) and proportion (55%) of QRNG were found in New South Wales. QRNG were also prominent in Victoria where 118 QRNG represented 44 per cent of isolates and in Queensland (47 QRNG, 33%). In South Australia there were 14 (13.7%) QRNG, in Western Australia 13 (13%) and six (3.4%) in the Northern Territory. Five QRNG were present in the Australian Capital Territory and three in Tasmania.

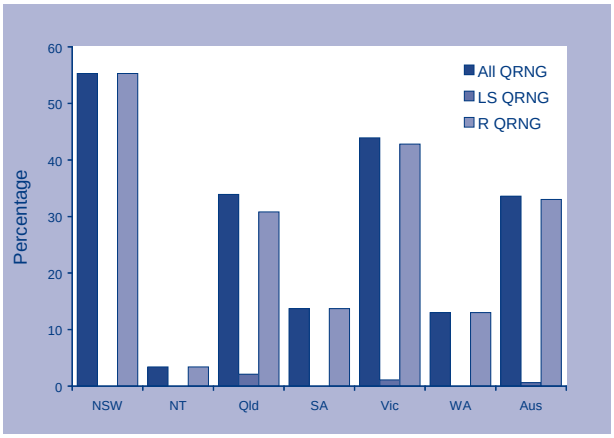
High level tetracycline resistance

The number (117) and proportion (10.6%) of high level tetracycline resistance (TRNG) detected were less than the 131 (13%) reported in this period of 2005. TRNG were found in all states and territories. The highest proportion of TRNG in any jurisdiction (34%) was in Western Australia.

Reference

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

Figure 13 The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 April to 30 June 2006, by jurisdiction



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.

R QRNG Ciprofloxacin MICs ≥1 mg/L.

Meningococcal surveillance

John Tapsall, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Meningococcal Surveillance Programme.

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of laboratory confirmed cases confirmed either by culture or by non-culture based techniques. Culture positive cases, where a Neisseria meningitidis *is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup, where known. A full analysis of laboratory confirmed cases of IMD is contained in the annual reports of the Programme, published in Communicable Diseases Intelligence. For more information see* Commun Dis Intell *2006;30:157.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 July to 30 September 2006, are included in this issue of Communicable Diseases Intelligence *(Table 9).*

Table 9. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 July to 30 September 2006, by jurisdiction and serogroup

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD	Q3	YTD
Australian Capital Territory	06			1	1	0	1	0		0		0		1	1
	05			2	3	1	3			0	1			3	7
	04			0	3	3	7							3	10
New South Wales	06			24	46	9	13	0	1	1	3	2	5	36	68
	05			27	60	4	13	0	3	4	7	2	3	37	86
	04			22	60	6	15	1	3	2	4	3	14	34	96
Northern Territory	06			1	3									1	3
	05			2	5	0	2			0				2	7
	04			0	5	0	0			0	1			0	6
Queensland	06	0	2	20	45	0	4			1	1			21	52
	05	0	0	13	34	5	7	0	0	0	0	0	0	18	45
	04	0	1	13	36	8	20	0	1	1	2	0	2	22	62
South Australia	06			3	9	0	0	0	1	1	1			4	11
	05			9	13	1	3							10	16
	04			2	11	1	1							3	12
Tasmania	06			0	3	0	1							0	4
	05			4	6	0	0							4	6
	04			3	6	5	5			0	1	1	3	9	15
Victoria	06			18	47	1	3	0	1	3	5	1	1	23	57
	05			26	41	3	6	1	1	1	3	2	3	33	55
	04			17	45	3	12	0	3	2	2	1	3	23	65
Western Australia	06			6	15	0	0	0	0	1	1			7	16
	05			20	29	0	0	0	2		0			20	31
	04			11	23	2	4			1	1			14	28
Total	06	0	2	73	169	10	22	0	3	7	10	3	6	93	212
	05	0	1	103	191	14	38	1	6	5	11	4	6	127	253
	04	0	1	68	189	28	64	1	7	6	11	5	22	108	294

Q3 = 3rd quarter.

YTD = Year to 30 September 2006.

HIV and AIDS surveillance

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

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell *2006;30:159.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 April to 30 June 2006, as reported to 30 September 2006, are included in this issue of Communicable Diseases Intelligence *(Tables 10 and 11).*

Table 10. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 April to 30 June 2006, by sex and state or territory of diagnosis

Sex		State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2006	This period 2005	YTD 2006	YTD 2005
HIV diagnoses	Female	0	7	0	3	0	0	7	3	20	21	59	47
	Male	1	58	0	19	2	0	72	14	166	247	387	451
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	1	65	0	22	2	0	79	17	186	268	447	498
AIDS diagnoses	Female	0	1	0	0	0	0	1	0	2	6	5	14
	Male	0	4	0	0	0	0	4	1	9	51	45	94
	Total*	0	5	0	0	0	0	5	1	11	57	50	108
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	0	2	2
	Male	0	5	0	0	0	0	3	1	9	14	20	28
	Total*	0	5	0	0	0	0	3	1	9	14	22	30

* Totals include people whose sex was reported as transgender.

Table 11. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 30 June 2006, and reported by 30 September 2006, by sex and state or territory

	Sex	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	32	841	18	253	94	8	351	192	1,789
	Male	258	13,258	128	2,645	898	96	5,130	1,182	23,595
	Not reported	0	231	0	0	0	0	22	0	253
	Total*	290	14,359	146	2,907	993	104	5,523	1,381	25,703
AIDS diagnoses	Female	10	246	3	68	32	4	106	37	506
	Male	93	5,336	42	1,011	395	50	1,943	419	9,289
	Total*	103	5,599	45	1,081	428	54	2,059	458	9,827
AIDS deaths	Female	7	136	1	41	20	2	60	24	291
	Male	73	3,567	26	654	274	32	1,391	292	6,309
	Total*	80	3,713	27	697	294	34	1,459	317	6,621

* Totals include people whose sex was reported as transgender.

National Enteric Pathogens Surveillance System

The National Enteric Pathogens Surveillance System (NEPSS) collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. Communicable Diseases Intelligence *NEPSS quarterly reports include only* Salmonella*. NEPSS receives reports of* Salmonella *isolates that have been serotyped and phage typed by the six* Salmonella *laboratories in Australia.* Salmonella *isolates are submitted to these laboratories for typing by primary diagnostic laboratories throughout Australia.*

A case is defined as the isolation of a Salmonella *from an Australian resident, either acquired locally or as a result of overseas travel, including isolates detected during immigrant and refugee screening. Second and subsequent identical isolates from an individual within six months are excluded, as are isolates from overseas visitors to Australia. The date of the case is the date the primary diagnostic laboratory isolated* Salmonella *from the clinical sample.*

Quarterly reports include historical quarterly mean counts. These should be interpreted cautiously as they may be affected by outbreaks and by surveillance artefacts such as newly recognised and incompletely typed Salmonella*.*

NEPSS may be contacted at the Microbiological Diagnostic Unit, Public Health Laboratory, Department of Microbiology and Immunology, The University of Melbourne; by telephone: +61 3 8344 5701, facsimile: +61 3 8344 7833 or email joanp@unimelb.edu.au

Scientists, diagnostic and reference laboratories contribute data to NEPSS, which is supported by state and territory health departments and the Australian Government Department of Health and Ageing.

Reports to the National Enteric Pathogens Surveillance System of Salmonella *infection for the period 1 July to 30 September 2006 are included in Tables 12 and 13. Data include cases reported and entered by 19 October 2006. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. For more information see* Commun Dis Intell *2006;30:159–160.*

There were 1,140 reports to NEPSS of human *Salmonella* infection in the third quarter of 2006, 31 per cent less than in second quarter of 2006. A winter nadir in reports of human salmonellosis is typical of seasonal trends in the incidence of salmonellosis in Australia. The second quarter count was five per cent less than the comparable third quarter of 2005 and around the ten-year historical mean for this period.

During the third quarter of 2006, the 25 most common *Salmonella* types in Australia accounted for 621 cases; 54 per cent of all reported human *Salmonella* infections. Eighteen of the 25 most common *Salmonella* infections in the third quarter of 2006 were also among the most commonly reported in preceding quarter. The combined count of *S.* Typhimurium phage type 135 and the similar phage-type 135a make these isolates by far the most common salmonellae in Australia, with most of these cases in the eastern mainland states and South Australia.

Other salmonellae manifesting increases over the recent historical average include *S.* Stanley (frequently acquired overseas), *S.* Kiambu (in Western Australia), *S.* Weltevreden (in Queensland) and *S.* Oranienberg and *S.* Potsdam (cases in various states and territories).

Acknowledgement: We thank scientists, contributing laboratories, state and territory health departments, and the Australian Government Department of Health and Ageing for their contributions to NEPSS.

Table 12. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 July to 30 September 2006, as reported to 19 October 2006

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	24	267	66	343	73	15	213	139	1,140
Total contributing *Salmonella* types	20	96	33	89	36	10	88	60	191

Table 13. Top 25 *Salmonella* types identified in Australia, 1 July to 30 September 2006, by state or territory

National rank	*Salmonella* type	State or territory								Total 3rd quarter 2006	Last 10 years mean 3rd quarter	Year to date 2006	Year to date 2005
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	S. Typhimurium PT 135	2	38	4	43	1	1	21	10	120	89	540	375
2	S. Saintpaul	1	5	4	28	2	0	3	6	49	46	317	322
3	S. Typhimurium PT 9	1	6	0	12	5	3	16	0	43	73	267	343
4	S. Stanley	2	9	0	6	1	0	10	3	31	17	73	53
5	S. Virchow PT 8	0	3	1	23	0	0	0	1	28	25	217	187
6	S. Typhimurium PT 170	0	14	0	5	0	1	4	1	25	25	270	405
7	S. Infantis	0	10	3	2	2	1	4	1	23	23	141	132
8	S. Birkenhead	0	12	0	10	0	0	0	0	22	23	219	154
9	S. Typhimurium RDNC	1	7	1	3	2	0	6	1	21	18	87	85
10	S. Typhimurium PT 197	0	2	0	15	1	0	3	0	21	15	84	490
11	S. Typhimurium PT 135a	0	1	0	0	20	0	0	0	21	3	42	16
12	S. Typhimurium PT 12	0	2	0	0	0	0	10	8	20	8	92	96
13	S. Oranienburg	1	0	0	7	0	0	6	5	19	7	131	32
14	S. Chester	0	3	1	7	1	0	3	3	18	22	119	143
15	S. Agona	1	7	0	5	1	1	1	2	18	13	56	52
16	S. Enteritidis PT 6a	0	2	0	1	1	0	4	10	18	8	36	74
17	S. Muenchen	0	1	6	3	3	0	1	3	17	17	121	113
18	S. Hvittingfoss	0	3	0	10	1	0	1	0	15	12	114	157
19	S. Kiambu	0	4	0	0	0	0	0	11	15	3	34	6
20	S. Weltevreden	0	2	3	9	0	0	0	0	14	9	76	43
21	S. Waycross	0	2	0	9	0	0	2	0	13	9	122	90
22	S. Potsdam	0	4	3	2	0	0	3	1	13	5	66	26
23	S. Newport	0	4	1	1	0	1	4	2	13	9	39	27
24	S. Anatum	0	2	1	6	1	0	1	1	12	12	89	53
25	S. Typhimurium U290	3	7	0	1	0	0	1	0	12	8	25	8

Overseas briefs

This material has been summarised from information provided by the World Health Organization (http://www.who.int) and by ProMED-mail (http://www.promedmail.org).

For the period 1 July to 30 September 2006

Avian influenza

From July to the end of September this year, WHO has reported 23 cases including 18 deaths from human influenza A (H5N1) in four countries: China, Indonesia, Iraq and Thailand.

Since the beginning of the outbreak in November 2003 to 30 September 2006, WHO has reported a worldwide total of 251 confirmed human H5N1 cases (with 148 deaths) in 10 countries.

China

The Chinese Ministry of Health confirmed two fatal H5N1 cases, one retrospectively, during the current reporting period.

The retrospective case is likely to be the index case of human H5N1 infection in mainland China in the current outbreak. The case, which dates back to November 2003, occurred in a 24-year-old man from the military who was based in Beijing. He developed symptoms on 25 November and died on 3 December 2003. As a result of the lengthy delay in diagnosing the cause of death, no history of poultry exposure was taken and the source of his H5N1 infection remains uncertain.

The second case occurred in a 62-year-old man from north-western China. The farmer developed symptoms on 19 June and died on 12 July 2006. No exposure history could be ascertained.

Confirmation of these two cases bought China's total confirmed human cases to 21 including 14 deaths since the beginning of the outbreak.

Indonesia

Human H5N1 cases continue to occur in Indonesia and there is no evidence of a decrease in the incidence of poultry H5N1 outbreaks with almost all Provinces reporting poultry outbreaks this year.

Between July and September 2006, the Indonesian Ministry of Health reported 17 confirmed H5N1 cases including 13 deaths. Confirmation brought Indonesia's total human cases to 68, including 52 deaths, since the beginning of the outbreak.

During August, a cluster of human H5N1 cases were reported in the remote sub-district of Cikelet, West Java Province. Prior to late June 2006, no mass poultry deaths were known to have occurred in the area. However, shortly after the purchase of chickens from an outside market and subsequent integration into local flocks, large numbers of chickens began dying in an outbreak that continued throughout July and into August. Three human cases from the area were confirmed positive for H5N1, two of whom died.

Additional deaths from respiratory illnesses were known to have occurred in July and August but no samples were taken. An investigation found no evidence of human-to-human transmission or that the virus was spreading more easily from birds to humans.

As a result of a change in the WHO H5N1 case definitions during the reporting period, WHO retrospectively confirmed three cases.

The first case from Banten Province occurred in an 8-year-old girl who developed symptoms on 24 June 2005 and died on 14 July 2005. She was part of a family cluster reported to WHO in July 2005 in which her father and sister also died.

The second case occurred in a 45-year-old male from Central Java Province. He developed symptoms on 25 November 2005 after exposure to diseased poultry but subsequently recovered.

The third case occurred in a 27-year-old male from West Sumatra Province. The case was identified during contact tracing of the man's sister who was confirmed H5N1 positive in May 2006. He developed mild symptoms after caring for his sister during her hospital stay.

The remaining 11 confirmed cases that occurred during this period, 10 of which were fatal, were localised to four provinces: East Java (n = 2), Jakarta (n = 4), West Java (n = 4) and South Sulawasi (n = 1). The non-fatal case occurred in a 6-year-old girl from West Java Province.

Iraq

The Iraqi Ministry of Health retrospectively confirmed the country's third case of avian influenza for the year on 19 September 2006. The case was a 3-year-old

boy who was hospitalised in Baghdad on 15 March 2006. He has since fully recovered. The other two fatal cases in Iraq occurred in January 2006.

Thailand

The Thai Ministry of Public Health reported three fatal cases between July and September this year, the only cases reported in 2006.

The first case was a 17-year-old man from Phichit Province in the country's north. He developed symptoms on 15 July, was hospitalised on 20 July and died on 24 July. This case had a history of previously handling poultry prior to his illness and at the same time as a confirmed H5N1 poultry outbreak in the province.

The second case occurred in a 27-year-old man from the central Uthai Thani Province. He developed symptoms on 24 July, was hospitalised on 30 July and died on 3 August. He also had exposure to dying household chickens.

The third case occurred in a 59-year-old farmer from Nong Bua Lam Phu Province in the north-east. He developed symptoms on 14 July, was hospitalised on 24 July and died on 10 August. Poultry outbreaks had been noted in the area. This patient was treated with oseltamivir while hospitalised. Repeated tests on upper respiratory tract samples (most after antiviral treatment) were negative for all influenza viruses by polymerase chain reaction. The H5N1 virus was isolated from lung samples taken at autopsy. This brings the confirmed cases in Thailand to 25 including 17 deaths.

Poultry outbreaks were confirmed in two Thai Provinces in late July, the first since November 2005.

[Source: WHO updates: 4, 14, 20 and 26 July; 7, 8, 9, 14, 17, 21, and 23 August; 8, 14, 19, 25, 27 and 28 September]

Chikungunya

WHO has reported 151 districts in 8 states/provinces in India have been affected by chikungunya fever from February to 10 October. More than 1.25 million suspect cases have been reported throughout the country. The outbreak is concentrated towards the south with 752,245 and 258,998 cases from the Karnataka and Maharashtra Provinces respectively. In some areas, reported attack rates have reached 45 per cent.

[Source: WHO update: 17 October]

Cholera and acute watery diarrhoeal syndrome

There was a sharp increase in cholera cases reported to the WHO in 2005 and this increased activity continues. During the reporting period, cholera outbreaks have occurred in 17 African countries: Angola, Chad, Côte d'Ivoire, Democratic Republic of the Congo, Ghana, Guinea, Kenya, Liberia, Mauritania, Niger, Nigeria, Senegal, Sudan, Togo, Tanzania, Uganda, and Zimbabwe.

All of these countries reported cholera outbreaks during 2005 with the exception of Angola and Sudan (neither had these countries reported outbreaks of acute watery diarrhoeal syndrome in 2005). However, neighbouring countries such the Democratic Republic of the Congo, have reported large cholera outbreaks in 2005 which are still continuing. Between 1 March and 17 September 2006, 9,565 cases including 196 deaths were reported in the Democratic Republic of the Congo.

Angola has reported 7,041 cases and 261 deaths between 24 June and 27 September 2006. The cumulative total as at 7 September was 53,537 cases and 2,187 deaths since 13 February [case fatality rate (CFR) = 4.0%]. This is the country's worst outbreak of water-borne disease in 20 years.

Sudan has reported 25,344 cases and 768 deaths between 28 January and 24 September 2006. The majority of the cases were from the first half of the year with 3,879 cases including 105 deaths reported between 19 July and 24 September. The last previously reported outbreak in Sudan occurred in 1996 with 1,800 cases. WHO has stated that the Sudanese outbreak was probably the cause of the current outbreak in Ethiopia, especially as the first Ethiopian cases occurred in Gambella in western Ethiopia (which borders Sudan) in April 2006.

As of 28 September, the Ethiopian Ministry of Health has reported a total of 22,101 cases and 219 deaths since the beginning of the outbreak (CFR = 1.0%). Five of Ethiopia's nine regions are affected with 79 per cent of the cases occurring in the Oromiya region. Ongoing flooding is a problem with the severely affected Amhara region reporting a CFR of 10 per cent. *Vibrio cholerae* 01 serotype Inaba has been confirmed in some areas.

Côte d'Ivoire has reported 372 cases including 8 deaths between 2 January and 23 July 2006 in contrast to 2005 when 39 cases including six deaths were reported for the entire year.

[Source: WHO Weekly Epidemiological Report, 4 August – 20 October; WHO update: 4 October]

Crimean-Congo haemorrhagic fever

Turkey experienced its largest-ever outbreak of Crimean-Congo haemorrhagic fever (CCHF) with 242 laboratory confirmed cases, including 20 deaths (CFR = 8.3%) between 1 January and 4 August 2006. From the period of 1 July to 4 August, 92 cases and nine deaths were reported. One of the fatal cases was a health care worker who acquired the infection while treating cases.

CCHF was initially characterised in the Crimea in 1944 however Turkey did not report its first cases until 2002. During 2005, Turkey reported 41 cases including one death in Yozgat Province. The most recent outbreak was reported in the Black Sea and Central Anatolia regions with six provinces affected. CCHF generally emerges in the summer months and is spread to humans via an infected tick bite although human-to-human transmission can also occur via exposure to blood of an infected patient.

The Southern Federal District of Russia also reported a rise in CCHF cases this year with 192 cases including five deaths reported throughout the area. During the 2005 outbreak, 127 cases including four deaths were reported in the same region.

[Source: WHO update: 8 August; ProMED, 20050729.2210, 29 July 2005; ProMED, 20060810.2242,10August;ProMED20060822.2359, 22 August]

Lassa fever

On 21 July 2006, Germany reported a case of Lassa fever imported from Sierra Leone in a 68-year-old man. The man, a Sierra Leone resident, became ill on 5 July and flew to Germany on 10 July. Although the risk of transmission to fellow passengers was low, the case initiated an international contact tracing exercise. No secondary cases have been reported. (The last reported imported case into Europe was three years ago in a soldier from the United Kingdom who had been serving in Sierra Leone.) [Lassa fever is considered endemic in West Africa from Nigeria to Senegal.]

Liberia, which neighbours Sierra Leone, experienced a Lassa fever outbreak between late May and September 2006 with 20 suspected/confirmed cases, including seven deaths. The cases occurred in the northern Nimba County, which shares borders with Côte d'Ivoire and Guinea.

[Source: WHO update: 25 July; ProMED, 20060724.2045, 24 July, ProMED, 20061001.2812, 1 October]

Pneumonic plague

A suspected pneumonic plague outbreak is occurring in Oriental province, in the north-eastern part of the Democratic Republic of the Congo. The outbreak began in mid-May 2006 and at 29 September was ongoing. Local authorities had reported 1,174 suspect cases including 50 deaths. Preliminary results from rapid diagnostic tests in the area found three of eight samples positive for pneumonic plague but final laboratory confirmation is still pending.

[Source: WHO update: 14 June, 13 October, 7 November]

Poliomyelitis – world update

As at 12 September 2006, four countries remain polio-endemic – Afghanistan, India, Nigeria and Pakistan. In addition to these countries, 12 countries have reported polio cases in 2006 due to importations – Kenya, Cameroon, Somalia, Yemen, Indonesia, Bangladesh, Ethiopia, Angola, Namibia, Niger, Nepal and the Democratic Republic of the Congo.

Endemic countries

In 2006, polio cases in northern Nigeria account for two-thirds of all global cases (803 of 1,228 cases). Five northern states account for 80 per cent of Nigeria's cases. Immunization Plus Days were held in September, with additional rounds planned for November and December in an effort to increase polio vaccination coverage of every child.

As at 3 October, India had reported 353 polio cases. Approximately half of the cases are concentrated in and around the Moradabad district in western Uttar Pradesh. The neighbouring state of Bihar has reported 20 cases. During 2005, only 66 confirmed cases were reported across the entire country. There is no indication that the current outbreak is being contained and the risk of further spread remains high. Polio originating from this area has been detected in a number of previously polio-free counties including Angola, Namibia, Democratic Republic of the Congo, Bangladesh and Nepal.

As at 12 September 2006, Pakistan has reported 17 cases since the beginning of the year, compared to 15 cases for the same period in 2005. Afghanistan has reported 26 cases, compared to four cases for the same period in 2005. Pakistan and Afghanistan continue to synchronise immunisation activities to increase coverage in the shared corridor of transmission.

Importation countries

Of the 12 countries that have had imported polio cases in 2006, eight countries have reported the date of onset of their most recent case between July and September 2006.

In Niger, 11 cases have been reported for the year to date with the date of onset of the most recent case being 23 August 2006. For the same period last year, six cases had been reported.

In Bangladesh, 15 cases have been reported for the year to date with date of onset of the most recent case, 22 August 2006. For the same period last year, no cases were reported. Bangladesh had not reported any cases of polio since 2000.

Cameroon has reported one case for the year with the date of onset of 22 August 2006 (compared to 1 case for the same period last year). Genetic testing is being undertaken on the case to determine the origin of the virus (Cameroon or Nigeria).

The Democratic Republic of the Congo has reported eight cases for the year with the date of onset of the most recent case on 13 August 2006. For the same period last year, no cases were reported. The Democratic Republic of the Congo has experienced two separate individual importations from Angola.

Nepal has reported two cases for the year with the date of onset of the most recent case on 1 August 2006 (compared to 1 case for the same period last year).

Ethiopia became re-infected with polio in December 2004. Since the beginning of 2006 15 cases have been reported. The onset date of the most recent case was 18 July 2006. Cases have been reported from four of Ethiopia's 11 regions.

Somalia became re-infected in 2005 after being polio-free for almost three years. Since the beginning of 2006 32 cases have been reported. The onset date of the most recent case was 5 September. Cases have been reported in 14 of Somalia's 19 regions.

Kenya had been polio-free for 22 years, reported its first polio case in September 2006. This was an imported case from neighbouring Somalia. The 3-year-old girl born in Kenya was living in a Somali refugee camp in the North Eastern Province bordering Somalia. Genetic sequencing indicates a virus of Nigerian origin, imported from Kismayo, Somalia.

Somalia, Ethiopia and Kenya simultaneously vaccinated more than 3.5 million children aged under five years in September 2006 in an effort to restrict polio transmission. Additional efforts have been made to engage nomadic leaders as half of the confirmed Somali cases this year are from nomadic populations.

[Source: Global Polio Eradication Initiative – monthly updates 4 July, 8 August and 12 September; ProMED, 20060912.2587, 12 September; 20061003.2830, 3 October; WHO update: 8 September, 19 October]

Tuberculosis – extreme drug resistance

South Africa is reporting the increasing spread of extreme-drug resistant tuberculosis (XDR-TB) with HIV-infected patients particularly vulnerable. Between January and March 2006, 53 people were identified with suspected XDR-TB, 52 of whom have since died. Since then, 106 cases have been reported (81 deaths) up to 4 September. The National Health Laboratory Services confirmed the new XDR-TB strain which was first identified in KwaZulu-Natal Province earlier this year, is now circulating through all nine South African Provinces.

[Source: ProMED 20060904.2518, 4 September; 20061019.3003, 19 October; WHO Weekly Epidemiological Record, 13 October]

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CDI reviewers 2006

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