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Annual reports

Annual report of the National Influenza Surveillance Scheme, 2006

Kathleen O'Brien, Ian G Barr

Abstract

Influenza surveillance in Australia is based on laboratory isolation of influenza viruses, sentinel general practitioner reports of influenza-like illness, and absenteeism data from a major national employer. In 2006, 3,130 cases of laboratory-confirmed influenza were reported to the National Notifiable Diseases Surveillance System, which was one-third lower than in 2005. The influenza season started in mid-June, with peak activity in late August. Influenza A was the predominant type notified (71%), however influenza B activity continued to increase as a proportion of reported cases. Reports of influenza-like illness from sentinel general practitioners showed a slow but steady increase throughout the first half of the year to peak in late August. In 2006, 657 influenza isolates from Australia were antigenically analysed: 402 were A(H3N2), 24 were A(H1N1) and 231 were influenza B viruses. Continued antigenic drift was seen with the A(H3N2) viruses from the previous reference strains (A/California/7/2004 and A/New York/55/2004) and drift was also noted in some of the A(H1N1) strains from the reference/vaccine strain A/New Caledonia/20/99, although very few A(H1N1) viruses were isolated in Australia in 2006. The B viruses isolated were predominately of the B/Victoria-lineage and similar to the reference/vaccine strain B/Malaysia/2506/2004. *Commun Dis Intell* 2007;31:167–179.

Keywords: influenza, surveillance, vaccine, influenza-like-illness, sentinel surveillance

Introduction

Influenza is an acute self-limiting viral disease of the upper respiratory tract. Influenza poses a major threat to worldwide public health because of its ability to spread rapidly through populations. Transmission is from person to person through infected respiratory droplets produced during coughing and sneezing.[1] The health and economic impact of influenza largely arise from related complications.

Influenza infections are seasonal in temperate climates, more commonly occurring in the colder months (June to September in the Southern Hemisphere and December to April in the Northern Hemisphere) but may occur year-round in tropical regions.

Annual influenza vaccination is recommended for people who are at increased risk of complications from the disease, such as those aged 65 years or older, and people with conditions such as cardiovascular disease and lung conditions which predispose them to severe influenza, and others with impaired immunity. Complications from influenza can result in increased hospitalisations and mortality. People who develop secondary bacterial pneumonia, for example, are at high risk of severe illness or death.[2]

There are 3 types of influenza—A, B and C—which are classified according to their distinct internal proteins.[3] Influenza type A is further characterised by 2 surface proteins: haemagglutinin (H) and neuraminidase (N). These proteins change constantly over time in order to avoid the hosts' immune system, a process that has been termed 'antigenic drift'. This is the main reason why seasonal influenza epidemics occur and vaccines need to be regularly updated. The ancestral hosts for influenza A viruses are aquatic birds, however, it has also been established in some mammals, such as humans and pigs. The natural host for types B and C is humans, although influenza C has been isolated from pigs.[4] Influenza C is more like the common cold in its effect, being less severe than influenza A or B.[5] Influenza types A and B are responsible for major outbreaks.[6]

Three pandemics occurred in the 20th century: the 1918–1919 'Spanish Flu' A(H1N1); the 1957 'Asian Flu' A(H2N2); and the 1968 'Hong Kong Flu' A(H3N2). The Spanish Flu is estimated to have caused as many as 40 million deaths worldwide, with unusually high mortality among young adults.[7] In Australia, 60% of deaths occurred in those aged 20–45 years. Mortality associated with the Asian and Hong Kong influenza pandemics was less severe, with the highest mortality rates being in the elderly and people with chronic diseases.[8]

Pandemics are caused by the introduction of a new influenza A subtype into the naïve human population, sometimes by the mixing of parts of avian viruses (at least the haemagglutinin) with

human or swine influenza viruses, a process termed 'antigenic shift'. Currently, there is concern that the avian A(H5N1) virus that has infected and killed millions of poultry in many countries will undergo such changes or naturally mutate to make it easily transmissible in humans and hence trigger a pandemic. While a complete understanding of this process is still lacking, it now appears that there are multiple changes that would be required to achieve this outcome. For example, scientists have experimentally added several human influenza genes from a recent A(H3N2) virus into to a current avian A(H5N1) virus and this reassorted virus was not more transmissible in mammalian animal models than the original avian A(H5N1) virus.[9]

An effective national surveillance system is essential for the control of seasonal epidemics and preparedness for potential pandemics, particularly as the timing and severity of a future pandemic cannot be predicted. Virological and epidemiological monitoring are important components of influenza surveillance.

The main objectives of influenza surveillance are:

- early detection of epidemics to enable the implementation of public health measures such as the vaccination of high risk groups, outbreak control campaigns and provision of clinical services;
- characterisation of the nature of the epidemic and evaluation of it's impact and associated public health measures; and
- isolation and antigenic characterisation of circulating influenza viruses to assist in the formulation of the following season's vaccine and to provide new vaccine strains.

Fortnightly influenza surveillance data were published throughout the 2006 season on the Communicable Diseases Australia website (http://www.health.gov.au/cda). This report is a summary of Australian surveillance information gathered throughout 2006, and includes a summary of international influenza activity.

Surveillance methods

Influenza surveillance in Australia is based on the following data sources:

- notifications of laboratory-confirmed influenza required by legislation in most states and territories, and reported to the National Notifiable Diseases Surveillance System (NNDSS);
- laboratory diagnosis including virus isolation and serology by laboratories participating in the Laboratory Virology and Serology Reporting Scheme (LabVISE);
- subtype and strain data of circulating influenza viruses provided by the World Health Organization (WHO) Collaborating Centre for Reference and Research on Influenza;
- consultation rates for influenza-like illness diagnosed by sentinel general practitioners;
- absenteeism data of workers from a national employer; and
- hospitalisations and mortality data.

National Notifiable Diseases Surveillance System

In all jurisdictions except South Australia, laboratory-confirmed influenza is a notifiable disease under state and territory legislation. Although influenza is not a notifiable condition in South Australia, laboratory reports are collected and sent to NNDSS. In this report, data are analysed by the date of onset, but when this was not available the earliest date from specimen collection date and notification date was used.

Laboratory surveillance

LabVISE is a national scheme of sentinel laboratories that reports influenza diagnoses throughout the year. In 2006, 11 laboratories from all jurisdictions except the Australian Capital Territory and the Northern Territory contributed to the scheme. Data were reported to LabVISE monthly and analysed by specimen collection date.

Sentinel general practitioner surveillance

Sentinel general practitioner surveillance schemes for influenza monitor the consultation rates for influenza-like illness (ILI). Sentinel surveillance schemes in Australia include: the Australian Sentinel Practice Research Network (ASPREN), which collects data at a national level as well as reporting for South Australia; the Queensland Influenza-like Illness Sentinel Surveillance in General Practice Program; the Victorian Influenza Surveillance Scheme; Western Australian sentinel general practices; and the Northern Territory Tropical Influenza Surveillance Scheme. ASPREN and the Northern Territory Tropical Influenza Surveillance Scheme report ILI rates throughout the year, while the other sentinel surveillance schemes report from May to October each year.

The national case definition of ILI is: presentation with fever, cough and fatigue. All sentinel surveillance schemes used the national case definition for ILI in 2006.

Absenteeism surveillance

Australia Post, a major nationwide employer, provided sick leave absenteeism data collected weekly throughout 2006. Absenteeism, defined as an absence due to illness for more than 3 consecutive days, was presented as a rate per 100 employees per week, on an average of 32,798 employees per week. It is important to note that this measures absenteeism from all illness, not just influenza.

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centres for Reference and Research on Influenza located in Australia, Japan, the United Kingdom and United States of America, are responsible for analysing influenza viruses collected through an international surveillance network involving 117 national laboratories in 88 countries. The Melbourne Centre analyses viruses received from Australia and from laboratories throughout Oceania, the Asian region and beyond. All virus isolates are analysed antigenically and a geographically and temporally representative number of viruses, together with any strains demonstrating uncharacteristic reactions during antigenic characterisation, are further analysed by genetic sequencing of the viral haemagglutinin gene and the neuraminidase gene. Together with serological and epidemiological data, this forms the basis from which WHO makes recommendations in February (for the Northern Hemisphere) and in September (for the Southern Hemisphere) for the vaccine formulation to be used in the following winter.

WHO vaccine formulation recommendations are made in the context of strains that are antigenically 'like' laboratory reference strains that are named according to a standard nomenclature for influenza viruses. For human isolates, this nomenclature is based on type, the place of isolation, sequential number and year of isolation, and for influenza A, the subtype of the H and N may also be included in brackets after the designation. An example of a human isolate is A/Sydney/5/97(H3N2), an influenza A(H3N2) virus that was the 5th sequential influenza A isolated in Sydney for the year in 1997. The WHO recommendations (available from: http://www.who.int/csr/disease/influenza/vaccinerecommendations/en/index.html) are then translated into actual virus strains acceptable to regulatory authorities and vaccine manufacturers by national and regional committees (such as the Australian Influenza Vaccine Committee, available from http://www.tga.gov.au/committee/aivc2007.htm)

Hospitalisations data

Data on hospital separations are available from the Australian Institute of Health and Welfare. These data are coded using the *International Statistical Classification of Diseases and Related Health Problems, 10th revision, Australian modification* (ICD-10-AM).

In this report, hospital separations for 2004–05 with a principal diagnosis of influenza due to identified influenza viruses (ICD-10-AM J10) and influenza where the virus was not identified (ICD-10-AM J11) are presented.

Mortality data

Mortality data are compiled by the Australian Bureau of Statistics from information provided by the state and territory Registrars of Births, Deaths and Marriages, and are coded using the tenth revision of the *International Classification of Diseases and Related Health Problems* (ICD-10). In this report, deaths for 2005 with an underlying cause of influenza and pneumonia (ICD-10 J10–J18) are presented.

Adult Vaccination Survey

The 2006 Adult Vaccination Survey was conducted in October 2006 and surveyed 8,022 Australians aged 18 years or over. The survey, which is the sixth national survey in its series, was conducted using computer-assisted telephone interview methodology. The survey included questions about influenza vaccination.

Results

National Notifiable Diseases Surveillance System

There were 3,130 notifications of laboratory-confirmed influenza reported to the National Notifiable Diseases Surveillance System in 2006, which is approximately two-thirds the number of cases reported in 2005 (Table 1). This equates to a rate of 15.2 notifications per 100,000 population.

Rates of laboratory-confirmed influenza in 2006 ranged from 5.7 notifications per 100,000 population in South Australia to 40.1 notifications per 100,000 population in Queensland.

The majority of influenza cases in 2006 were type A (71%) (Figure 1). One-quarter (25%) were type B and just over 1% of cases were notified as testing positive for both types A and B.

Notifications of laboratory-confirmed influenza started to increase in late May (week 21) and peaked in late August (week 34) (Figure 2). The influenza season started at a similar time in 2005, however the number of cases increased more rapidly than in 2006 and notifications peaked about 2 weeks earlier.

Table 1. Notifications of laboratory-confirmed influenza, Australia, 2004 to 2006, by type

Year	Influenza type (per cent of notifications)				Number of notifications
	Type A	Type B	Types A & B	Unknown	
2006	70.8	25.6	1.3	2.2	3,130
2005	73.2	22.7	1.4	2.6	4,564
2004	75.8	19.6	1.2	3.4	2,133

Source: National Notifiable Diseases Surveillance System

Figure 1. Notifications of laboratory-confirmed influenza, Australia, 2004 to 2006, by type and week of onset

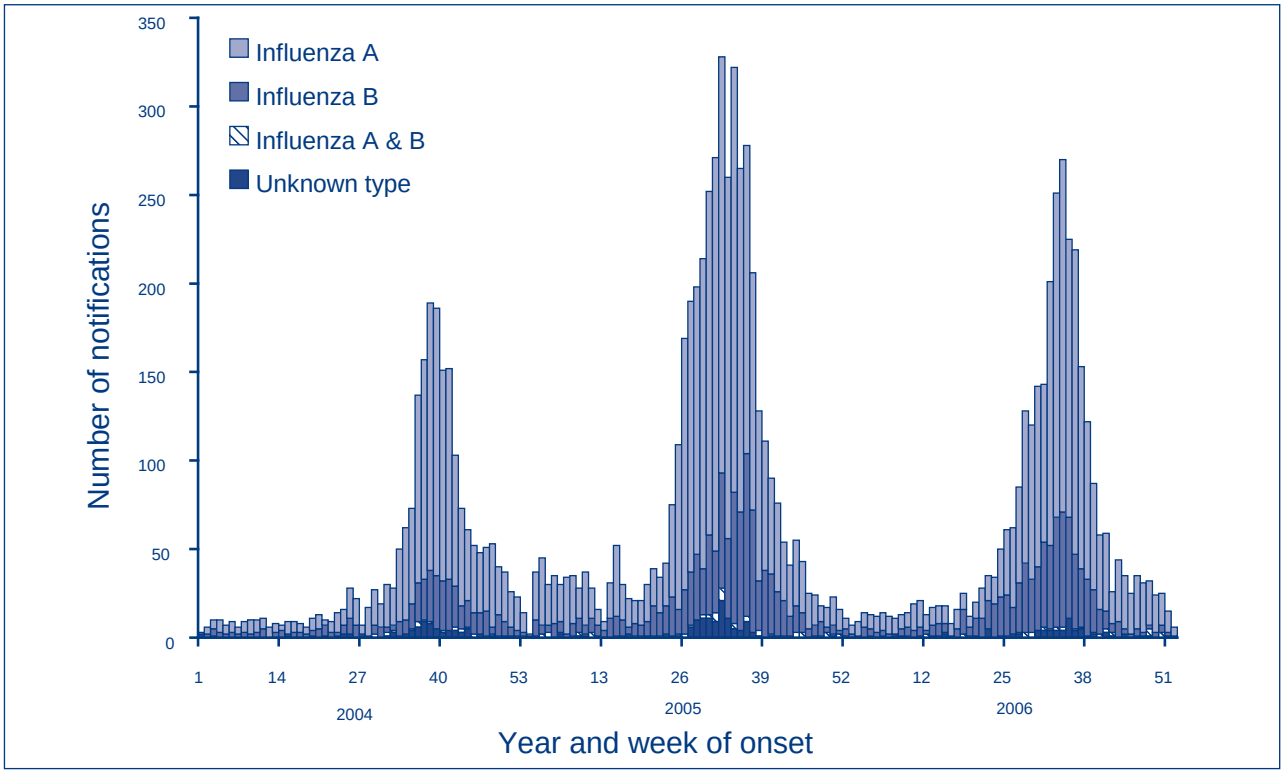
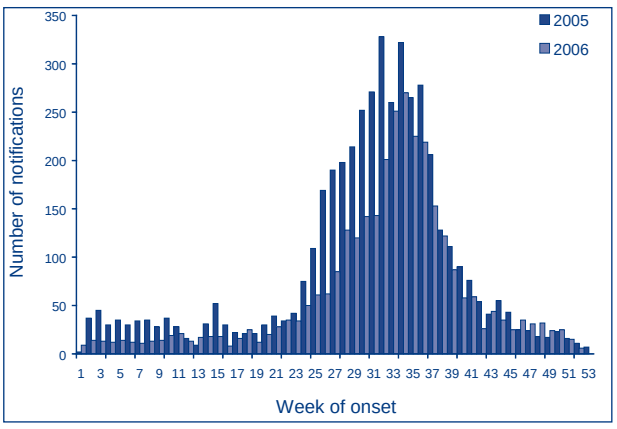


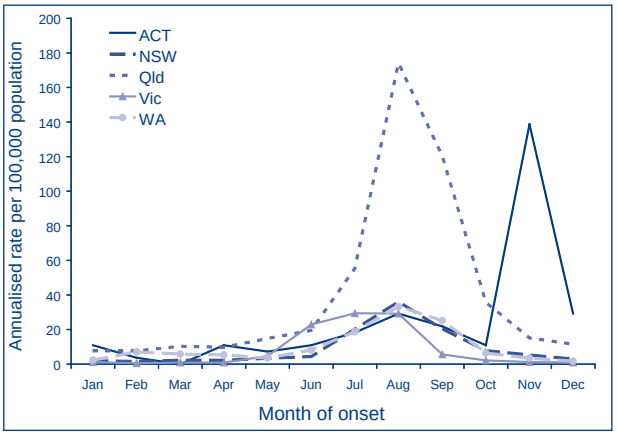
Figure 2. Notifications of laboratory-confirmed influenza, Australia, 2005 and 2006, by week of onset



Influenza notification rates for selected jurisdictions are shown in Figure 3. Most jurisdictions showed peaks in notification rates around August. Peak rates in Queensland in August were substantially higher than in other jurisdictions (174 notifications per 100,000 population in Queensland, annualised compared to 60 for all of Australia).

There was an unseasonal peak in influenza notifications in November in the Australian Capital Territory. Between 11 October and 6 December, 77 people (55 of 132 residents and 22 of 173 staff) in an aged care facility reported symptoms of influenza-like illness. Of these, 19 people (18 residents and 1 staff) were found to have laboratory-confirmed Influenza A infection.[10,11] Eight of these had been vaccinated with the 2006 influenza vaccine prior to the outbreak. Ten deaths of residents aged

Figure 3. Notification rates of laboratory-confirmed influenza, Australia, the Australian Capital Territory and Queensland, 2006, by month of onset

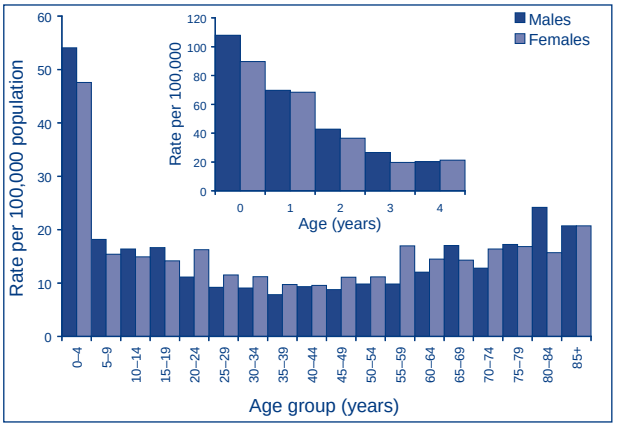


75–100 years were associated with the outbreak. Two of the residents who died had been fully vaccinated with the 2006 influenza vaccine.

Age-specific notification rates for laboratory-confirmed influenza reported to the NNDSS are shown in Figure 4. The highest notification rates were seen in those aged 0–4 years, with rates being around three times higher than for other age groups (51 cases per 100,000 population compared to an all-ages rate of 15 cases per 100,000 population). Figure 4 (insert) shows single-year age-specific rates for the 0–4 years age group. Within this group, rates showed a strong decline with increasing age; the notification rate for infants aged less than one year was 99 cases per 100,000 population, and in children aged 4 years was 21 cases per 100,000 population.

People aged 65 years and over are the target for influenza vaccination as they are at an increased risk of complications from influenza. Notification rates for people in this age group were 17 cases per 100,000 population for males and 16 cases per 100,000 population for females.

Figure 4. Notification rates of laboratory-confirmed influenza, NNDSS, Australia, 2006, by age and sex

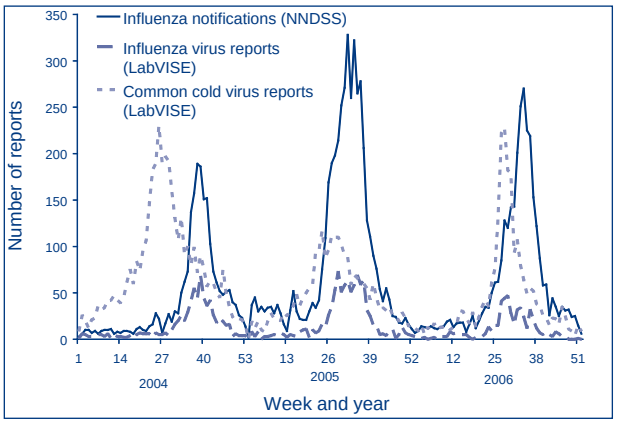


Notification rates were higher in males than females aged under 15 years, but higher for females aged 20–64 years. In infants aged less than one year, rates were higher for boys than for girls (108 cases per 100,000 population compared to 90 cases per 100,000 population).

Laboratory surveillance

A total of 510 laboratory diagnoses of influenza were reported by the 11 laboratories participating in LabVISE in 2006, around half the number reported in 2005 (967 diagnoses) (Figure 5). Two-thirds (66%) of influenza isolates in 2006 were type A, with the remaining isolates being type B. The number of influenza reports to LabVISE started increasing in late May, peaked in mid-July (week 29), and declined around September. LabVISE influenza reports peaked around the same time in 2005 and 2006, however started decreasing earlier in 2006.

Figure 5. Laboratory reports of influenza and common cold virus reports and notifications of laboratory-confirmed influenza reported to NNDSS, 2004 to 2006



Note: LabVISE reports are by week of specimen collection, and NNDSS reports are by week of onset.

Reports of common cold virus isolates (defined as respiratory syncytial virus, parainfluenza viruses and rhinoviruses) were also collected by LabVISE. In 2006, 2,322 reports of the common cold were reported to LabVISE, similar to the number reported in 2005 (2,511) (Figure 5). The number of common cold reports started increasing in early May, and peaked in mid-July (week 28), and declined around October (week 42). More than three-quarters (78%) of the common cold virus reports in 2006 were due to respiratory syncytial virus. Common cold reports peaked at similar times in 2005 and 2006, although

in 2005 reports started increasing earlier in the season and in 2006 the peak was double the number of reports for 2005.

Sentinel general practice surveillance

Australian Sentinel Practice Research Network

The ASPREN is a network of general practices that collect data on influenza-like illness. Sentinel practices contributing to ASPREN are located in all jurisdictions other than the Northern Territory. In 2006, an average of 27 general practices reported ILI cases to ASPREN at an average of 2,654 consultations per week (Table 2). The average number of participating practices and consultations has decreased since 2003.

In 2006, ILI reports to ASPREN started increasing in week 24, with peaks in weeks 25 (29.2 cases per 1,000 consultations, mid-June) and 35 (32.4 cases per 1,000 consultations, late August). In 2005, ILI reports peaked in early August (42.4 cases per 1,000 consultations) (Figure 6a).

State and territory general practice influenza surveillance programs

The Northern Territory collected information on ILI through the Tropical Influenza Surveillance System. Reports were received from 8 sentinel general practitioners (GPs) per week on average (range 1–12) with an average of 480 consultations per week (range 8–836). Reports of ILI peaked in early July (36 ILI cases per 1,000 consultations) with smaller peaks in mid-August (25 cases per 1,000), mid-September (29 cases per 1,000) and a late peak in late November/early December (26 cases per 1,000 consultations in week 49) (Figure 6b). The Northern Territory showed a different pattern to other jurisdictions due to it's tropical climate (cases are spread throughout the year).

Queensland reported information on consultations for ILI over the period June to October. Reports were received from 22 sentinel GPs per week on average (range 9–28) with an average of 2,797 consultations per week (range 1,218–3,638). A preliminary peak in ILI cases was seen in weeks 22 and 23 (17 ILI cases per 1,000 consultations) followed by a larger peak in week 34 (20 cases per 1,000) (Figure 6c).

South Australian ILI surveillance data are collected through ASPREN and were reported throughout 2006. On average, from late May to mid-October, 828 patients were seen by 8 doctors weekly, with 10 ILI cases being reported. Reporting of ILI cases started increasing in mid-May—with a spike in reports in mid-June—and started decreasing from mid-July (Figure 6d).

Victorian ILI surveillance data were reported by the Victorian Infectious Diseases Reference Laboratory for May to September 2006. On average, 23 general practices (range 19–25) reported 5,898 consultations per week (range 5,039–6,568). Consultations for ILI increased steadily from week 18 (1.8 ILI patients per 1,000 consultations) to an initial peak in early July (week 27, 9.1 ILI patients per 1,000) with a second peak in late August (week 33, 9.8 cases per 1,000) (Figure 6e).

Western Australian ILI surveillance data were reported by PathWest Laboratory Medicine WA for May to October 2006. On average, 11 practices (range 7–14) reported 54 ILI cases per week (range 14–121). Consultations for ILI increased steadily from initial reporting in early May (week 19) to peak in mid-July (week 29) with 82.1 ILI patients per 1,000 consultations (Figure 6f).

Table 3 summarises a comparison of rises and peaks in influenza reporting for the main data sources included in this report.

Absenteeism surveillance

Absenteeism surveillance is a non-specific index of influenza activity. In 2006, national absenteeism rates started increasing in early May (week 18) and peaked in early August (week 32) at 1.2% (Figure 7). This compares to an average absenteeism rate of 0.8% of employees per week during the reporting period (an average of 254 employees per week). Absenteeism rates declined again from early September (week 36), however remained relatively high until mid-December.

Table 2. General practices reporting influenza-like illness to the Australian Sentinel Practice Research Network, 2003 to 2006

	Reporting practices		Consultations	
Year	Average per week	Range per week	Average per week	Range per week
2006	27	11–39	2,654	934–3,999
2005	29	15–36	2,996	1,081–3,398
2004	31	11–42	3,321	1,224–4,219
2003	48	32–62	4,910	2,138–6,587

Figure 6. Consultation rates for influenza-like illness, 2005 and 2006, by sentinel surveillance scheme and week of report

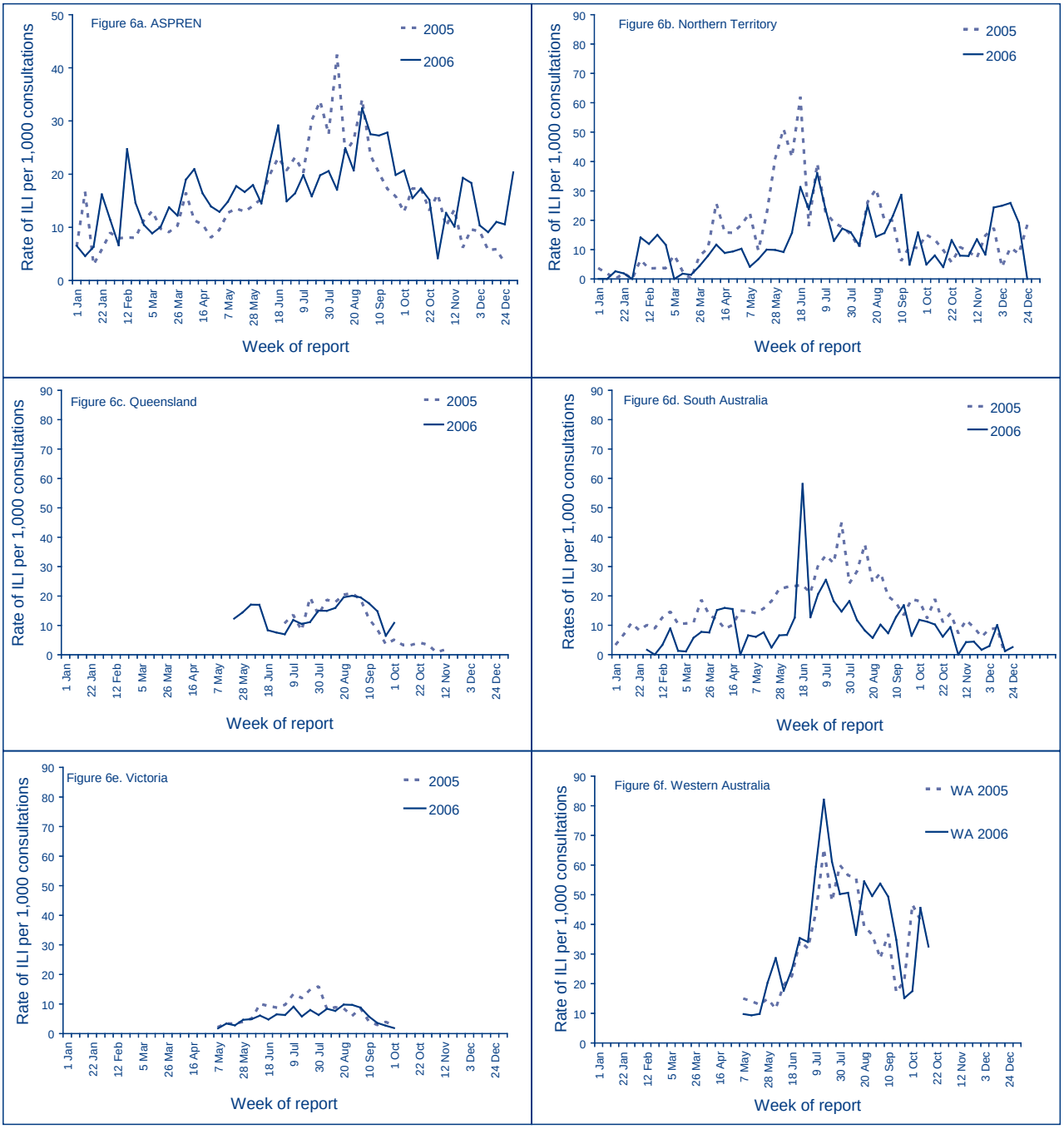


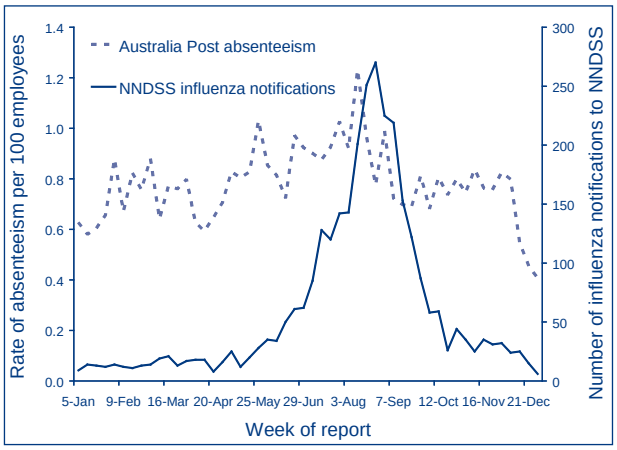
Table 3. Comparison of rises and peaks in influenza reporting, Australia, 2006

Surveillance system	Illness	Week of first rise	Week(s) of peak(s)
NNDSS	Laboratory-confirmed influenza notifications	24	34
LabVISE	Influenza reports	23	29
	All common cold virus reports	24	28
ASPREN	Influenza-like illness reports	24*	25 & 35
Australia Post absenteeism data	Absenteeism rates	18*	32

* ASPREN ILI cases and Australia Post absenteeism rates show reasonably steady increases throughout the first few months of the year.

A comparison of absenteeism rates and laboratory-confirmed influenza notified to the NNDSS show that while absenteeism rates were high when notifications peaked, absenteeism rates increased several weeks before notifications started increasing (Figure 7).

Figure 7. Absenteeism rates and influenza notification rates, 2006, by week of report



Adult Vaccination Survey

Results from the 2006 Adult Vaccination Survey will be published in the forthcoming report *2006 Adult Vaccination Survey: summary results*.[12] The target population for influenza vaccination is Australians aged 65 years or over. An estimated 77.5% of people in the target group were vaccinated against influenza in 2006. Coverage was slightly lower in 2006 than in 2004 (when it was 79.1%), however the difference was not statistically significant. The highest vaccination coverage was seen in South Australia (83.9%) and the lowest in the Northern Territory (63.3%). Vaccination rates were higher for women than for men (79.2% compared with 75.4%).

Hospitalisation and mortality data

There were a total of 1,378 hospital separations with a principal diagnosis of influenza in 2004–05 (the most recent national hospitalisations data); just over one-third (535 separations) were attributed to 'influenza due to identified influenza virus' (ICD-10-AM J10) and two-thirds (843 separations) to 'influenza, virus not identified' (ICD-10-AM J11).[13] Nearly one in 5 hospital separations (19%, 260 separations) for influenza were for people aged 65 years or over, including 50 separations for people aged 85 years or over. Females accounted for slightly more hospital separations than males (706 for females compared to 672 for males).

The most recent data on causes of death in Australia are for 2005. Influenza and pneumonia (ICD-10 codes J10–J18) were recorded as the underlying cause of death for 3,034 persons in 2005 (2.3% of all deaths).[14] More females than males died of influenza or pneumonia (1,703 females compared to 1,331 males); however the standardised death rate for males was higher than for females (15.8 versus 12.0). In 2004, 56% of influenza and pneumonia deaths were among people aged 85 years or over (whereas 31% of all deaths occurred among people in this age group).[15]

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centre for Reference and Research on Influenza received 657 isolates or clinical specimens from Australian laboratories in 2006 that yielded viable influenza viruses. This was the second lowest number of isolates received over the last 10 years (the lowest being in 2001). All of the 2006 viruses were analysed antigenically using the haemagglutination inhibition assay which identified 402 (61.2%) as A(H3N2) strains, 24 (3.6%) as A(H1N1) strains and 231 (35.2%) as influenza B strains. The 2006 Australian A(H3) viruses were mostly antigenically similar to the 2006 vaccine strain A/New York/55/2004 but a proportion of viruses had a reactivity pattern closer to A/Wisconsin/67/2005-like viruses and a significant proportion of viruses did not match either of these patterns (Table 4).

Consistent with the antigenic drift in the A(H3) isolates seen with ferret antisera, serological studies conducted with pre– and post-vaccination human sera from recipients of the 2006 vaccine containing the A/New York/55/2004 strain, showed a reduction in antibody titres to some 2006 A(H3) isolates. Antigenic analysis of the few Australian A(H1) strains that were isolated, showed that there was drift away from the 2006 vaccine strain A/New Caledonia/20/99, which was more pronounced than in recent years. Of the 231 influenza B viruses analysed, 93.5% were antigenically related to the 2006 vaccine strain B/Malaysia/2506/2004 (B/Victoria/2/87-lineage viruses) while the remaining 6.5% were closely related to B/Florida/7/2004 (B/Yamagata/16/88-lineage viruses), indicating a good match of the vaccine strain with the circulating B viruses.

Sequence analysis of the variable (HA1) region of the haemagglutinin gene was undertaken for 98 Australian 2006 strains [11 A(H1), 49A(H3) and 38 B] and for the neuraminidase gene, 46 Australian 2006 strains (7 H1, 25 H3, 14 B) were sequenced. The phylogenetic analysis of the 2006 A(H3) virus sequences (Figure 8) showed that most Australian viruses fell into one of 2 subgroups based on the HA1 domain of the

Table 4. Antigenic comparisons of influenza A(H3) viruses by the haemagglutination-inhibition test

Virus antigen	Ferret antiserum Reciprocal haemagglutination-inhibition titre		
	A/Wellington	A/California	A/Wisconsin
A/Wellington/1/2004	640	640	320
A/California/7/2004*	320	640	320
A/Wisconsin/67/2005	320	320	1,280
A/Brisbane/69/2006	320	640	1,280
A/Perth/30/2006	160	320	1,280
A/Auckland/119/2006	80	160	320
A/Sydney/17/2006	40	320	160
A/Victoria/178/2006	160	160	80
A/South Australia/4/2006	80	80	40

* An A/California/7/2004-like strain (A/New York/55/2004) was the H3 strain used in the 2006 Australian influenza vaccine.

Figure 8. Evolutionary relationships between influenza A(H3) haemagglutinins (HA1 region)

**2006 Australian influenza vaccine strain

Blue text indicates H3 virus groups circulating in 2006

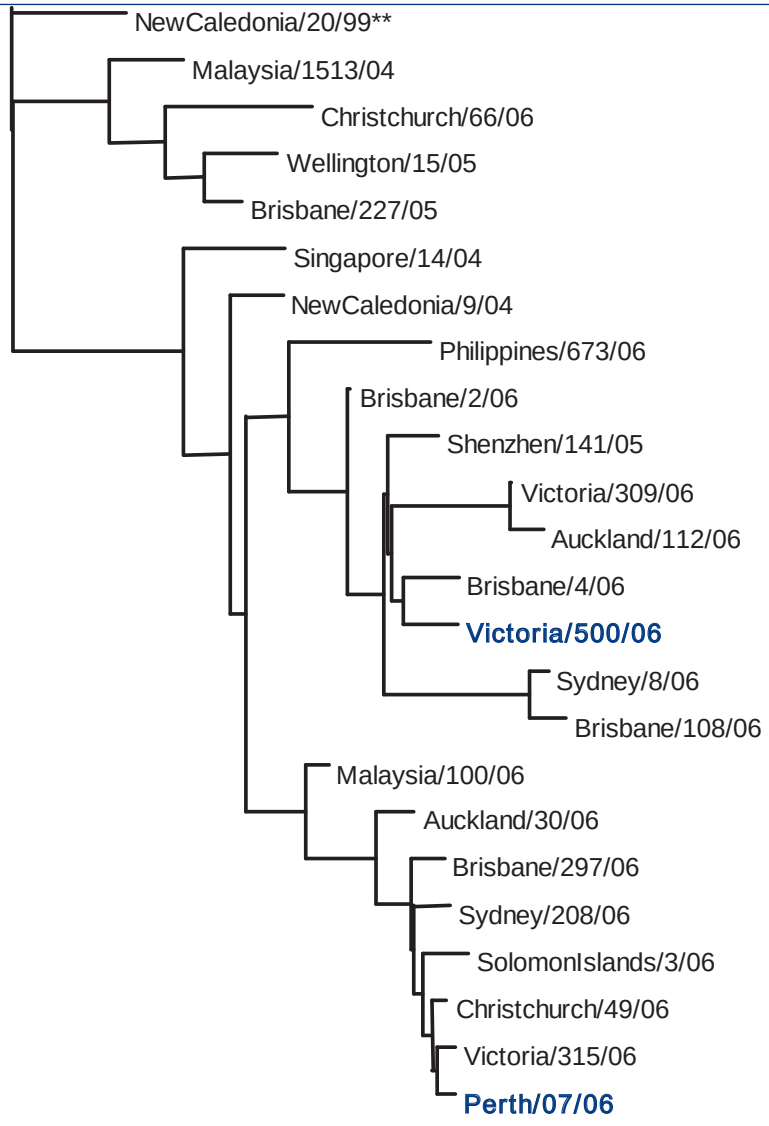
haemagglutinin gene. Both of these subgroups were closely related to the A/Wisconsin/67/2005 reference virus with one group similar to A/Victoria/503/2006 and the other group similar to A/Brisbane/9/2006. Viruses from the same cities and states fell into one of these 2 subgroups showing that both variants were co-circulating in Australia in 2006. The phylogenetic relationships of viruses A/Sydney/56/2006 and A/Sydney/65/2006 (a Canberra isolate) from influenza outbreaks in New South Wales and the Australian Capital Territory (Figure 8) showed that these were similar to other locally circulating A(H3) viruses.

Genetically, most of the A(H1) 2006 Australian viruses fell into 2 subgroups that were distinguishable from A/New Caledonia/20/99, one containing A/Perth/7/2006 (falling into the A/Malaysia/100/2006 group) and the another represented by the A/Victoria/500/2006 subgroup (Figure 9). The Australian 2006 influenza B isolates grouped into their respective lineages either the B/Victoria or B/Yamagata lineages with the B/Victoria-lineage, showing little change from the reference/vaccine strain B/Malaysia/2506/2004 (Figure 10).

International trends in influenza

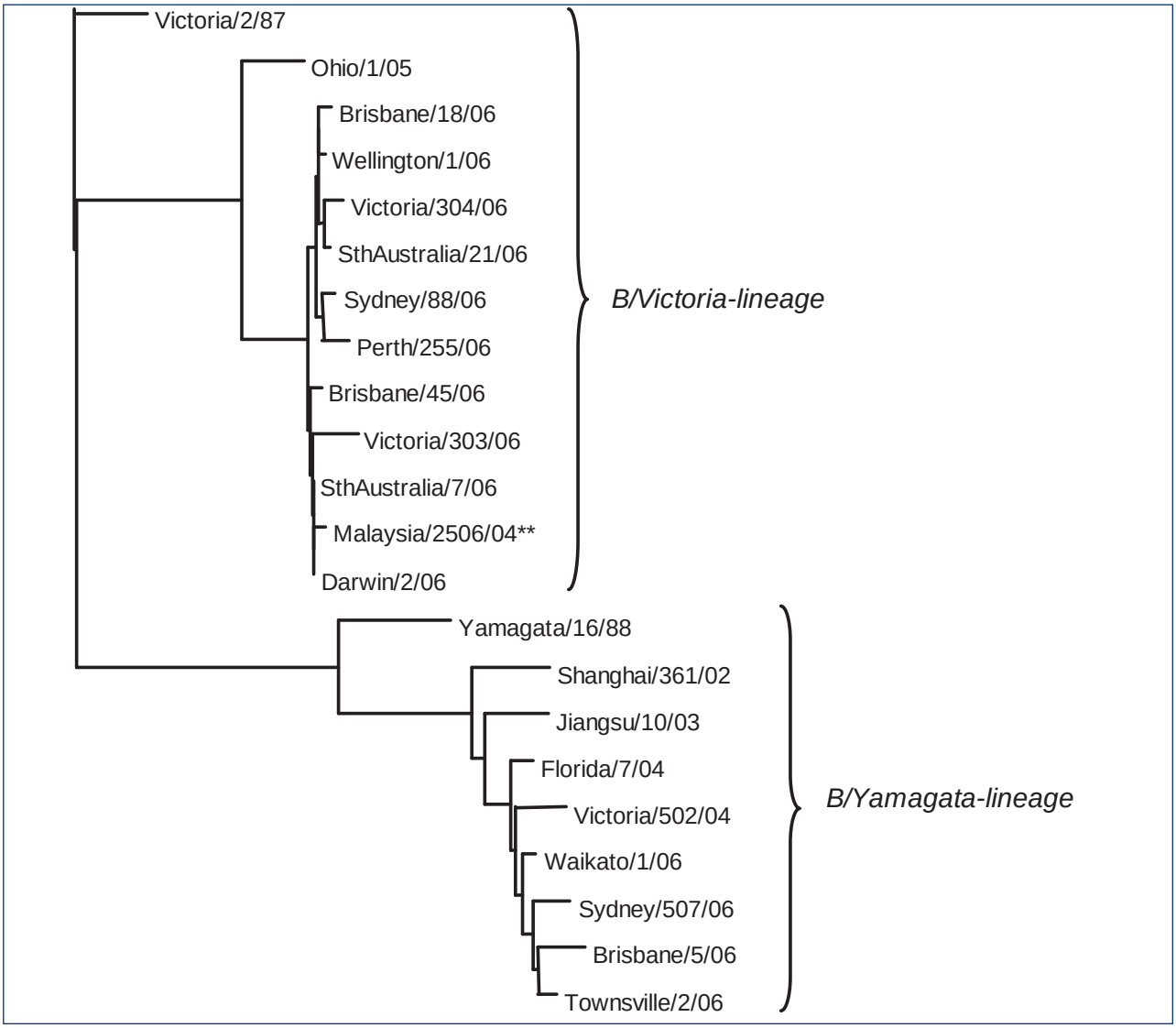
In 2006, global influenza activity was generally low compared to previous years. Influenza was detected in most countries although the number of influenza isolates obtained was markedly reduced compared

Figure 9. Evolutionary relationships between influenza A(H1) haemagglutinins (HA1 region)



**2006 Australian influenza vaccine strain
Blue text indicates H1 virus groups circulating in 2006

Figure 10. Evolutionary relationships between influenza B haemagglutinins (HA1 region)



to recent years. Influenza A(H1N1), A(H3N2) and influenza B co-circulated in many countries. In the Northern Hemisphere, outbreaks of A(H1N1) occurred in Hong Kong (SAR), Egypt, Japan, Thailand, Spain and the United States of America (USA). Outbreaks of A(H3N2) occurred in Egypt, Madagascar, Canada, USA, Japan, Kazakhstan, the Russian Federation, Slovenia and several European countries. Influenza B outbreaks occurred in Egypt, USA, China, Hong Kong (SAR), Republic of Korea, Uzbekistan and in a number of European countries early in 2006. In the Southern Hemisphere, influenza activity began in April and was generally mild to low. In South America influenza A(H1N1) viruses predominated with an outbreak reported in Brazil. Other outbreaks due to influenza A(H3N2) were reported in New Zealand and South Africa while elsewhere in Africa and Oceania influenza activity was low.

There were widespread outbreaks of A(H5N1) highly pathogenic avian influenza (HPAI) in chickens, ducks and other birds in many parts of the world in 2006, with the exception of the Americas and Oceania. According to the official WHO figures for 2006, 115 human infections with H5N1 occurred in 9 countries resulting in 79 deaths, which was more deaths than recorded in 2004 and 2005 combined (for details see the WHO avian influenza web site http://www.who.int/csr/disease/avian_influenza/en/). No H5N1 infections were detected in humans or in birds in Australia in 2006.

While the temporal pattern of the annual influenza season in New Zealand is broadly similar to Australia, outbreaks often begin earlier in the year. In 2006, the New Zealand consultation rates for ILI started to increase in late May and peaked at week 27 (first week of July) with a second smaller peak at week 33 (mid-August). Influenza hospitalisations peaked at

week 28. Of the 438 New Zealand isolates typed at the WHO Influenza Centre, the vast majority were A(H3) (82.2%) with 73 A(H1) viruses (16.7%) and very few influenza B viruses (only 5 isolates; 1.1%). The low level of influenza B viruses is in contrast to the previous season when influenza B viruses of the B/Victoria-lineage predominated and led to several relatively severe outbreaks. Overall influenza activity in New Zealand in 2006 was low and below levels seen in 2003–2005. The full report on the 2006 influenza season in New Zealand, produced by the Institute of Environmental Science and Research Limited, is available from: http://www.surv.esr.cri.nz/PDF_surveillance/Virology/FluAnnRpt/InfluenzaAnn2006.pdf.

Discussion

The 2006 Australian influenza season was mild in comparison to previous years and was predominantly due to influenza A infections.

Notifications to the NNDSS, influenza and common cold reports from LabVISE, and ILI reports to ASPREN all started increasing mid-way through the year (weeks 23 & 24) however LabVISE reports peaked earlier than notifications to NNDSS (weeks 28 & 29 for LabVISE; week 34 for NNDSS). The major peak for ASPREN reports came 1 week later than for NNDSS. Differences in reporting patterns from various sources may be due to factors such as timeliness of laboratory testing and reporting, or occurrence of other respiratory illnesses symptomatic of ILI. Absenteeism rates—which relate to absence for illness of any cause and so are non-specific—showed no apparent trend that could be attributed to increased influenza activity.

The influenza types reported via NNDSS for 2006 were 70.8% influenza A, 25.6% influenza B, 1.3% influenza A and B and 2.2% unknown type. The finding of 1.3% of notifications having both types of influenza was similar to previous years and is higher than would be expected as documented reports of dual infections are rare.[16] This may warrant further investigation in the future to confirm true dual influenza infections.

People aged 65 years or over are eligible for free annual influenza vaccination in Australia. Notification rates in 2006 for people in this age group were similar to those for people of all ages. While people aged 65 years or over are a target for immunisation, the highest age-specific rates of laboratory-confirmed influenza were seen in children aged under 5 years. Influenza vaccine can be given to children from 6 months of age, however those under 5 years are at increased risk of minor adverse events.[2]

While the 2006 influenza season was relatively mild, an outbreak of influenza A(H3N2) occurred in an aged care facility in the Australian Capital Territory in October. The outbreak control strategy included vaccination clinics, enhanced infection control and isolation of cases. Prophylactic treatment through administration of Oseltamivir was recommended to residents through their medical practitioners and provided to asymptomatic staff. The public health response also included laboratory investigation of suspect cases, social distancing and other measures to assist containment.

The majority of the Australian isolates (62.1%) analysed at the WHO Influenza Centre were A(H3N2) strains (similar to 2005) and a significant number of these strains showed a degree of heterogeneity based on their antigenic and genetic characteristics. Many strains were antigenically similar to the vaccine strain A/New York/55/2004 and to the newer reference strain A/Wisconsin/67/2005, while others appeared to have drifted somewhat from both of these viruses. Genetic analysis showed there were only minor changes in the HA1 region in the 2006 A(H3) viruses from the A/New York/55/2004 strain with most 2006 strains more like the A/Wisconsin/67/2005 reference virus. Very few influenza A(H1) strains were isolated in Australia in 2006 (24) but some of these also showed some drift away from the vaccine strain A/New Caledonia/20/99. Influenza B strains were almost exclusively of the B/Victoria/2/87-lineage, unlike recent years when both B/Victoria and B/Yamagata/16/88-like viruses have co-circulated in roughly even proportions.[17] The 2006 B/Victoria-like viruses were closely related both antigenically and genetically to the vaccine strain B/Malaysia/2506/2004. Influenza patterns in New Zealand were similar to Australia with low activity and mainly A(H3) strains of the A/Wisconsin/67/2005 type however there were very few influenza B viruses isolated in 2006 compared with Australia.

The WHO annual consultation on the composition of influenza vaccines for the Southern Hemisphere, 2007 took place in Geneva from 18–20 September 2006. The recommended composition of influenza virus vaccines for use in the 2007 Southern Hemisphere influenza season was:

an A/New Caledonia/20/99(H1N1)-like virus;

an A/Wisconsin/67/2005(H3N2)-like virus;

a B/Malaysia/2506/2004-like virus.

This recommendation has one change to the previous Southern Hemisphere vaccine for the 2006 influenza season, with the addition of a new A(H3) virus A/Wisconsin/67/2005 (replacing the vaccine

strain A/New York/55/2004). This recommendation was the same as the Northern Hemisphere recommendation for their 2006–07 influenza vaccine.

Preparation for a potential influenza pandemic is a high priority in Australia. In October 2006, the Australian Government Department of Health and Ageing (DoHA) coordinated and participated in Exercise Cumpston, Australia's largest ever health simulation exercise and one of the largest pandemic influenza exercises held in the world. The exercise tested Australia's preparedness for responding to pandemic influenza involving widespread human-to-human transmission of a new strain of the influenza virus. More information about the exercise and the Australian Health Management Plan for Pandemic Influenza can be found on the DoHA's website (http://www.health.gov.au/internet/wcms/publishing.nsf/Content/Pandemic+Influenza-1)

It is important to note that notifications of laboratory-confirmed influenza do not capture all cases of influenza in Australia. Those with mild disease may not present to a doctor, and those that do may not be referred for laboratory testing. Reporting of influenza-like illness by sentinel general practitioners is crucial for national influenza surveillance, as it provides an additional source of information and may provide an early warning system for increased influenza activity. Sentinel surveillance is currently being strengthened through ASPREN, who have undertaken a number of initiatives with jurisdictions to increase general practitioner recruitment and representativeness.

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Annual report of the Australian Gonococcal Surveillance Programme, 2006

The Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme (AGSP) monitors the antibiotic susceptibility of *Neisseria gonorrhoeae* isolated in all states and territories. In 2006 the *in vitro* susceptibility of 3,850 isolates of gonococci from public and private sector sources was determined by standardised methods. Different antibiotic susceptibility patterns were again seen in the various jurisdictions and regions. Resistance to the penicillins nationally was at 34% and, with the exception of the Northern Territory, ranged between 17% and 51%. Quinolone resistance in gonococci increased, especially in Queensland, with resistance to this agent found in all jurisdictions. Nationally, 38% of all isolates were ciprofloxacin-resistant, and most of this resistance was at high minimum inhibitory concentration (MIC) levels. With the exception of the Northern Territory excepted, proportions of quinolone resistant gonococci ranged between 16% and 54%. All isolates remained sensitive to spectinomycin. Less than 1% of isolates showed some decreased susceptibility to ceftriaxone. A high proportion of gonococci examined in larger urban centres were from male patients and rectal and pharyngeal isolates were common. In other centres and in rural Australia the male to female ratio of cases was lower, and most isolates were from the genital tract. *Commun Dis Intell* 2007;31:180–184.

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

Introduction

Antimicrobial resistance (AMR) is a major problem in *Neisseria gonorrhoeae* and treatment options have been severely limited by the increasing lack of efficacy of several major antibiotic groups. Strategies for treating and controlling gonorrhoea are based on use of single dose treatments that cure a minimum of 95% of cases.[1] Information on the most reliable treatment options are based on data derived from continuous monitoring of the susceptibility of gonococci to recommended antibiotics. This task has been undertaken by the Australian Gonococcal Surveillance Programme (AGSP) continuously since 1981.[2,3] The emergence and spread of penicillin and quinolone resistant gonococci has been closely followed. There are concerns about gonococcal isolates showing resistance to multiple antibiotics including decreased susceptibility to the third generation cephalosporin ceftriaxone, which is used extensively in Australia.[4] This analysis of AMR in *N. gonorrhoeae* in Australia is derived from data generated by the AGSP during the 2006 calendar year.

Methods

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

Gonococci, isolated in and referred to the participating laboratories, were examined for antibiotic susceptibility to the penicillins, quinolones, spectinomycin and third generation cephalosporins and for high-level resistance to the tetracyclines by a standardised methodology.[2,5] The AGSP also conducted a program-specific quality assurance (QA) program.[6] Antibiotic sensitivity data were submitted quarterly to a coordinating laboratory which collated the results and also conducted the QA program. Additionally, the AGSP received data on the sex of the patient and site of isolation of gonococcal strains. Where available, data on the geographic source of acquisition of antibiotic-resistant isolates were included in the analyses.

Results

Numbers of isolates

There were 3,937 gonococcal isolates referred to or isolated in AGSP laboratories in 2006, little different from the 3,980 examined in 2005. The source and site

of infection with these isolates are shown in the Table. One thousand one hundred and ninety-eight gonococci (30.4% of the Australian total) were isolated in New South Wales, 951 (24%) in Victoria, 565 (14.3%) in Queensland, 549 (13.9%) in the Northern Territory, 397 (10%) in Western Australia, and 244 (6.2%) in South Australia with small numbers in Tasmania (14) and the Australian Capital Territory (19). Three thousand eight hundred and fifty isolates remained viable for susceptibility testing.

Source of isolates

There were 3,315 strains of gonococci from men and 621 from women, with a male to female (M:F) ratio of 5.3:1, slightly higher than the 4.7:1 ratio for 2005. The number of strains from men increased by 27 and there was a corresponding decrease in the number of isolates from women. The M:F ratio was again high in New South Wales (12.6:1) and Victoria (11.3:1) where strains were more often obtained from urban populations. The lower ratios in Queensland (3.9:1) Western Australia (3.6:1), South Australia (2.5:1) and the Northern Territory (2:1) reflected the large non-urban component of gonococcal disease in those regions. Male rectal and pharyngeal isolates were most frequently found in Victoria (30% of isolates from men), and New South Wales (36%). About 1.5% of isolates are shown as being isolated from 'other' (46) or unknown (16) sites. These included 9 cases of disseminated gonococcal infection in men (0.3%) and 12 (1.9%) in women. There were 13 pharyngeal and 6 rectal isolates from women. Although not all infected sites were identified, isolates from urine samples were regarded as genital tract isolates. Most of the other unidentified isolates were probably from this source, although they were not so specified. There were small numbers of isolates from the eyes of both newborn and older infants and also adults, and from Bartholin's abscesses and infections ascending from the endocervix in women to pelvic organs.

Antibiotic susceptibility patterns

In 2006 the AGSP reference laboratories examined 3,850 gonococcal isolates for sensitivity to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics) and spectinomycin and for high level resistance to tetracycline (TRNG). As in past years the patterns of gonococcal antibiotic susceptibility differed between the various states and territories. For this reason data are presented by region as well as aggregated for Australia as a whole.

Penicillins

The categorisation of gonococci isolated in Australia in 2006 by penicillin minimum inhibitory concentration (MIC) is shown in Figure 1. Infections unlikely to respond to the penicillin group of antibiotics (penicillin, ampicillin, amoxycillin, with or without clavulanic acid) are those caused by gonococci shown as 'penicillinase-producing' *N. gonorrhoeae* (PPNG) and 'RR – relatively resistant'. Resistance in the PPNG group results from the production of beta-lactamase and in those 'relatively resistant' by the aggregation of chromosomally-controlled resistance mechanisms[1] – so-called CMRNG. Chromosomal resistance is defined by an MIC to penicillin of 1 mg/L or more.[1,5] (The minimal inhibitory concentration in mg/L is the least amount of antibiotic that inhibits *in vitro* growth under defined conditions.) Infections with gonococci classified as fully sensitive (FS, MIC ≤0.03 mg/L),

Source and number of gonococcal isolates, Australia, 2006, by sex, site and state or territory

Gender	Site	State or territory						
		NSW	NT	Qld	SA	Vic	WA	Aust*
Male	Urethra	698	360	370	145	601	289	2,482
	Rectal	255	1	53	10	159	10	495
	Pharynx	149	0	16	18	107	8	303
	Other/NS	8	4	11	2	7	3	35
	Total	1,110	365	450	175	874	310	3,315
Female	Cervix	79	175	110	65	64	80	575
	Other/NS	9	8	5	4	13	7	46
	Total	88	183	115	69	77	87	621
Unknown	Total	0	1	0	0	0	0	1
Total*		1,198	549	565	244	951	397	3,937

* Includes isolates from Tasmania (14) and the Australian Capital Territory (19).

NS Not stated.

The site of isolation and sex of some infected patients was not known.

less sensitive (LS, MIC 0.06–0.5 mg/L) would be expected to respond to standard penicillin treatments, although response to treatment may vary at different anatomical sites.

Figure 1. Penicillin resistance of gonococcal isolates, Australia, 2006, by state or territory

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

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

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

PPNG Penicillinase-producing *Neisseria gonorrhoeae.*

Nationally, 1,306 (34%) gonococci were penicillin resistant by one or more mechanisms in 2006, a further increase on the 1,148 (29.5%) resistant to this group of antibiotics in 2005 and the 770 (21.7%) resistant by any mechanism in 2004. Of these, 964 (25% of all isolates) were CMRNG and 342 (9%) PPNG. The proportion of penicillin-resistant gonococci of all gonococcal isolates in New South Wales was 51.4% (PPNG 8.6%, CMRNG 42.8%), Victoria 41.6% (PPNG 9.6%, CMRNG 32%), Queensland 27.6% (PPNG 13%, CMRNG 14.6%), Western Australia 19.2% (PPNG 11.2%, CMRNG 8%), and South Australia 17.3% (4.8% PPNG and 12.5% CMRNG). Two PPNG and 5 CMRMG were identified in the Australian Capital Territory, and in Tasmania there were 3 PPNG and 7 CMRNG. In the Northern Territory there were 21 PPNG and 4 CMRNG showing that 4.6% of strains were penicillin resistant (3.4% in 2005). Data on acquisition were available in 80 (23%) infections with PPNG. Thirty-four infections with PPNG were acquired locally and 46 by overseas contact. These contacts were principally in Western Pacific or South East Asian countries including China, India, Indonesia (Bali), Korea, the Philippines, Singapore and Thailand.

Ceftriaxone

From 2001 onwards, low numbers of isolates with slightly raised ceftriaxone MICs have been found in Australia. In 2002, there were 21 gonococci with ceftriaxone MICs more than 0.03 mg/L isolated nationally, 10 in 2003, 24 (0.7%) in 2004 and 48 (1.2%) in 2005. In 2006, there were 23 (0.6%) gonococci with ceftriaxone MICs in the range 0.06–0.25 mg/L. Fifteen of these were present in New South Wales (1.3% of New South Wales isolates) 1 (0.4%) in South Australia, 6 (1.1%) in Queensland and 1 (0.3%) in Western Australia. These isolates were generally also penicillin and quinolone resistant.

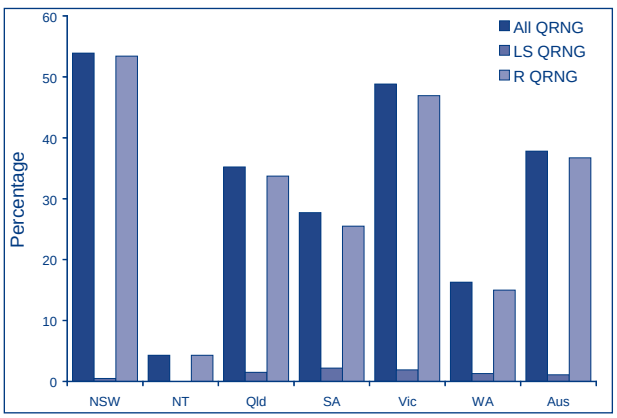
Spectinomycin

All isolates were again susceptible to this injectable antibiotic.

Quinolone antibiotics

Figure 2 shows the distribution of gonococci with altered susceptibility to quinolones nationally and by state or territory. Thus far, resistance to the quinolone antibiotics in *N. gonorrhoeae* is mediated only by chromosomal mechanisms so that incremental increases in MICs are observed. The AGSP uses ciprofloxacin as the representative quinolone and defines altered susceptibility as an MIC of 0.06 mg/L or more.[5] Treatment with currently recommended doses of 500 mg of ciprofloxacin is effective for strains with a lower level of resistance, viz. 0.06–0.5 mg/L, in about 90% of cases, but lower doses of the antibiotic will result in treatment failure more often. At higher levels of resistance i.e. an MIC of 1 mg/L or more, rates of failed treatment rise rapidly. Currently, gonococci with MICs up to 16 and 32 mg/L are being seen in Australia. At MIC levels of 4 mg/L or more, treatment failure, even with higher ciprofloxacin doses, approaches 100%.

Figure 2. Percentage of gonococcal isolates which were less sensitive to ciprofloxacin or with higher level ciprofloxacin resistance and all strains with altered quinolone susceptibility, Australia, 2006, by state or territory



LS QRNG MIC 0.06–0.5 mg/L.

R QRNG MIC 1 mg/L or more.

Nationally in 2006, 1,455 (37.8%) gonococci had some level of resistance to quinolones (QRNG), an increase over the 1,190 (30.6%) detected in 2005 and the 825 (23.3%) in 2004. Most of the QRNG (1,413 or 97%) had resistance at a higher level i.e. MICs ≥ 1mg/L and many of these had MIC levels of the order of 8–32 mg/L. A similar proportion had higher level resistance in 2005. The highest proportion of QRNG was seen in New South Wales where 635 QRNG were 53.9% of all isolates examined. In Victoria, there were 463 QRNG (48.8%), 193 (35.2%) in Queensland (double the number and proportion seen in 2005), 64 (27.8%) in South Australia, and 61 (16.3%) in Western Australia. In other jurisdictions the numbers of QRNG remained low (Northern Territory, 23; Tasmania, 7; the Australian Capital Territory, 9)

Information on acquisition of QRNG was available in 321 of the 1,455 cases (22%). Two hundred and sixty of these (81%) were acquired locally and 59 (19%) overseas from sources referred to under PPNG acquisition with contacts also reported in Brazil, Hong Kong, Korea, the Netherlands, Pakistan, Vietnam, the United Kingdom and the United States of America.

High-level tetracycline resistance

The spread of high-level tetracycline resistance in *N. gonorrhoeae* (TRNG) is examined as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea. There was an upsurge in TRNG isolation in 2002 when 11.4% of strains of this type were detected nationally with little further change in 2003. A further increase in TRNG numbers to 490 in 2004 saw them represent 13.8% of all gonococci. This proportion was unchanged in 2005 when 534 TRNG, were detected. In 2006, there were slightly fewer TRNG (12%).

TRNG were present in all states and territories with the highest proportions in Western Australia (105 TRNG, 28%) and Queensland (92, 16.8%). Lower proportions of TRNG were present in New South Wales (125, 10.6%), Victoria (97 TRNG, 10.2%) and South Australia (15, 6.5%). There were 24 (4.3% TRNG, found in the Northern Territory and 2 each in Tasmania and the Australian Capital Territory.

Discussion

The number and proportion of gonococci resistant to antibiotics used for treating gonorrhoea increased still further in 2006. A surge in resistant strains was noted in 2005,[4] but the increase in 2006 was relatively small. Resistance to both the penicillin and quinolone groups of antibiotics reached historical highs. Nationally, one third of gonococci were penicillin resistant by at least one mechanism and a slightly higher proportion was quinolone resistant. Figures 1 and 2 illustrate the need for disaggregated information rather than pooled national data in that significant differences in the rates of resistance can be seen in the various jurisdictions. Remote areas in some jurisdictions with high disease rates continue to be able to use penicillin-based treatments, but effective use of this cheap and acceptable treatment requires close monitoring of resistance patterns. In contrast, more than half the isolates from New South Wales and nearly half the isolates from Victoria were both penicillin and/or quinolone resistant. Significant increments in the proportion of quinolone resistant gonococci occurred in all jurisdictions and the MICs of these QRNG were also generally higher.

The presence of a number of gonococci with decreased susceptibility to ceftriaxone was again a concern. Although the numbers of these isolates still remains low at about 1% of all isolates tested, they are almost always also resistant to quinolones and penicillins. Data from regional surveys has confirmed the spread to countries in close proximity to Australia, of gonococci with decreased ceftriaxone susceptibility. Annual National Neisseria Network reports[4] have consistently emphasised that the local recommendation for a minimum dose of 250 mg of ceftriaxone is prudent given the presence of these isolates and the propensity for resistance to develop in *N. gonorrhoeae*. The mechanism of resistance to ceftriaxone in these isolates is not fully elucidated. Although alterations in the *penA* gene, including the presence of mosaic *penA* genes,[7] have been implicated, it is likely that other mechanisms may also be involved.[8] All gonococci tested in Australia in 2006, including those with altered cephalosporin susceptibility, were susceptible to spectinomycin. A low proportion of gonococci was also found to be resistant to azithromycin in 2006 and treatment failures have been recorded in Australia with 1 g doses of this antibiotic.[9] Although susceptibility to azithromycin is assessed in some jurisdictions, there are no firm parameters defined for this purpose.[9] Overseas, increasing resistance to azithromycin, widely used as an anti-chlamydial agent in conjunction with gonococcal treatment, has been reported.

These increasing and multiple problems with antimicrobial resistance in *N. gonorrhoeae* suggest a continuing need for surveillance of antimicrobial resistance in this organism. Standard treatment guidelines can be reliably based on the results of properly-conducted surveillance of antimicrobial resistance. This surveillance is still based on testing of gonococcal isolates. Despite challenges posed by the increasing use of non-culture based methods for the diagnosis of gonorrhoea, the number of gonococcal isolates available for testing in Australia

under the AGSP remains satisfactory for surveillance purposes. As a guide to interpretation of AGSP data, the WHO currently recommends that once resistance to an antibiotic has reached a level of 5% in a population, continuing use of that agent should be reconsidered.[1] A continuing commitment to maintenance of culture-based systems is still required to examine gonococci in sufficient numbers to detect resistance rates at the 5% level.[10]

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Annual report of the Australian Meningococcal Surveillance Programme, 2006

The Australian Meningococcal Surveillance Programme

Abstract

In 2006 there were 271 laboratory-confirmed cases of invasive meningococcal disease analysed by the National Neisseria Network, a nationwide network of reference laboratories. The phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility of 166 isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were determined and an additional 105 cases were confirmed by non-culture-based methods. Nationally, 217 (80%) confirmed cases were infected with serogroup B and 26 (9.6%) with serogroup C meningococci. The total number of confirmed cases was 74 (21%) fewer than the 345 cases identified in 2005. Numbers of cases decreased in all jurisdictions except Queensland. The age group showing the greatest decrease in numbers (by about one–third) was in those aged 25 years or more. A typical primary disease peak was observed in those aged 4 years or less with a lower secondary peak in adolescents and young adults. Serogroup B cases were 93% of all cases in those aged 4 years or less and 77% in those aged 15–24 years. The proportion of invasive disease represented by serogroup C disease was highest in the 20–24 years and 25–44 years age groups. The common phenotypes circulating in Australia were B:15:P1.7, B:4:P1.4, C:2a:P1.4 and C:2a:P1.5, but again with significant jurisdictional differences. No evidence of meningococcal capsular 'switching' was detected. About two thirds of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06–0.5 mg/L). All isolates remained susceptible to rifampicin and ciprofloxacin. *Commun Dis Intell* 2007;31:185–194.

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

Introduction

Invasive meningococcal disease (IMD) remains an infection of public health interest in Australia. Additionally, a publicly-funded program of selective vaccination with conjugate serogroup C meningococcal vaccine was completed in 2004 and the prospect of additional vaccines, e.g. porin-based vaccines for serogroup B meningococcal disease, increases the need for precise data on circulating meningococcal subtypes. The National Neisseria Network (NNN) is a national laboratory-based program for the examination of *Neisseria meningitidis* from cases of IMD and has operated since 1994 through the collaboration of reference laboratories in each jurisdiction. The NNN supplies information on the phenotype and/or the genotype of invasive meningococci, and their antibiotic susceptibility. These data are meant to supplement those from clinical notification schemes. The characteristics of the meningococci responsible for IMD are important both for individual patient management and to tailor the public health response.

Annual reports summarising data gathered since the inception of the program were published in *Communicable Diseases Intelligence*. The following report analyses the characteristics of meningococci isolated in the calendar year 2006 and follows the format used first for the 2004 annual report published in *Communicable Diseases Intelligence*[1] where data on all laboratory-confirmed cases were aggregated for analysis. Prior to 2004, data on IMD diagnosed by culture-based and non-culture methods were provided separately.

Methods

The NNN continues as a long-term collaborative program for the laboratory surveillance of the pathogenic Neisseria, *N. meningitidis* and *N. gonorrhoeae*. A network of reference laboratories in each state and territory (see acknowledgements) performs and gathers laboratory data on cases of IMD throughout Australia.

Isolate-based invasive meningococcal diseases cases

Each case confirmation was based upon isolation of a meningococcus from a normally sterile site and defined as IMD according to Public Health Laboratory Network criteria. Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was sought. The isolate-based subset of the program categorised cases on the basis of site of isolation of the organism. Where an isolate was grown from both blood and cerebrospinal fluid (CSF) cultures in the same patient, the case was classified as one of meningitis. It is recognised that the total number of

cases, and particularly the number of cases of meningitis e.g. where there was no lumbar puncture or else where lumbar puncture was delayed and the culture was sterile, is underestimated. However the above approach has been used since the beginning of this Programmme[2] and is continued for comparative purposes.

Phenotyping of invasive isolates of meningococci by serotyping and serosubtyping was based on the detection of outer membrane protein (porin) antigens using a standard set of monoclonal antibodies obtained from the National Institute for Public Health, The Netherlands. Increasingly, sequencing of products derived from amplification of the porin genes *porA* and *porB* has been used to supplement and supplant serotyping analyses based on the use of monoclonal antibodies. For the purposes of continuity and comparability, the typing data from both approaches has been unified in the accompanying tables by converting sequence data to the more familiar serotyping/serosubtyping nomenclature.

Antibiotic susceptibility was assessed by determining the minimal inhibitory concentration (MIC) to antibiotics used for therapeutic and prophylactic purposes. This Programme uses the following parameters to define the various levels of penicillin susceptibility or resistance when determined by a standardised agar plate dilution technique.[3,4]

Sensitive, MIC ≤ 0.03 mg/L.
Less sensitive, MIC 0.06–0.5 mg/L.
Relatively resistant MIC ≤ 1 mg/L.

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

Non-culture-based laboratory-confirmed cases

Additional laboratory confirmation of suspected cases of IMD was obtained by means of non-culture-based methods including nucleic acid amplification (NAA) and serological techniques. NAA testing is essentially by polymerase chain reaction (PCR) techniques[5] and has been progressively introduced in the different jurisdictions. Data from the results of these investigations were included for the first time in the 1999 report.[6] The serological results are based on results of tests performed using the methods and test criteria of the Manchester Public Health Laboratory Service reference laboratory, United Kingdom as assessed for Australian conditions.[7–9] Where age, sex and outcome data for patients with non-culture-based diagnoses are available, these were also recorded. The site of a sample of a positive NAA is also used to define the clinical syndrome. This separation is not possible for cases diagnosed serologically.

Results

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

Number of laboratory-confirmed cases

There were 271 instances of laboratory-confirmed cases of IMD in 2006 (Table 1) compared with 345 in 2005, 361 in 2004 and 494 in 2003. In 166 cases (61.2%), a positive culture was obtained with or without a positive non-culture-based test and 105 cases were confirmed by a non-culture-based method alone. The total number of all laboratory-confirmed cases decreased in most jurisdictions in 2006 when compared to 2005 data. The largest decreases in numbers were in New South Wales (to 84 from 112) and Western Australia (to 19 from 45). Smaller decreases were noted in other jurisdictions with the exception of Queensland where numbers detected increased

Table 1. Number of laboratory-confirmed cases of invasive meningococcal disease, Australia, 2006, by state or territory and serogroup

State or territory	Serogroup						Total
	B	C	A	Y	W135	NG*	
ACT	1	1					2
NSW	57	16		1	5	5	84
NT	3	0					3
Qld	58	4		1	1	4	68
SA	13	0		1	1		15
Tas	4	1					5
Vic	64	3		1	5	2	75
WA	17	1			1		19
Australia	217	26	0	4	13	11	271

* Not serogrouped.

to 68 from 58 after a fall in 2005. There were 2 less cases in the Australian Capital Territory, 3 less in the Northern Territory, 8 less in South Australia, 3 less in Tasmania and 4 less in Victoria.

There were also 2 instances of isolation of *N. meningitidis* from travellers returning to Australia from overseas—1 of serogroup A, apparently acquired in France, and 1 of serogroup B, acquired in Greece.

Seasonality

Fifty-seven cases occurred between 1 January and 31 March, 60 between 1 April and 30 June, 95 between 1 July and 30 September and 59 between 1 October and 31 December. A winter peak of meningococcal disease is usual.

Age distribution

Nationally, the peak incidence of meningococcal disease was again in those 4 years and under (Table 2, Figure 1). Those aged less than 1 year or in the 1–4 age group together accounted for 100 (37% of the total) cases in 2006. There were 110 cases confirmed in these age groups (32%) in 2005. A secondary disease peak is also usual in the adolescent and young adult age group. The total of 49 (18% of all confirmed cases) cases in those aged 15–19 years was much the same as the number and proportion of cases in this age group in 2005 (48, 17%), but remained less than the 61 (17%) cases seen in 2004 and the 89 (18%) cases seen in 2003. Those aged 15–24 years together accounted for 79 (29%) cases in 2006. There were 88 (23.4%) cases in 2005 and 96 (26.7%) cases in these combined age groups in 2004.

Figure 1. Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2006, by age group

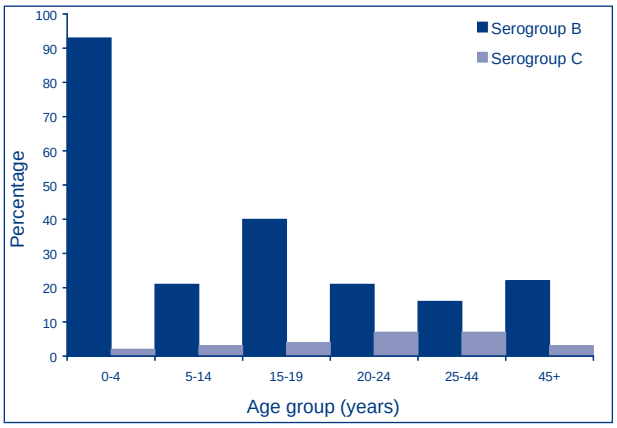
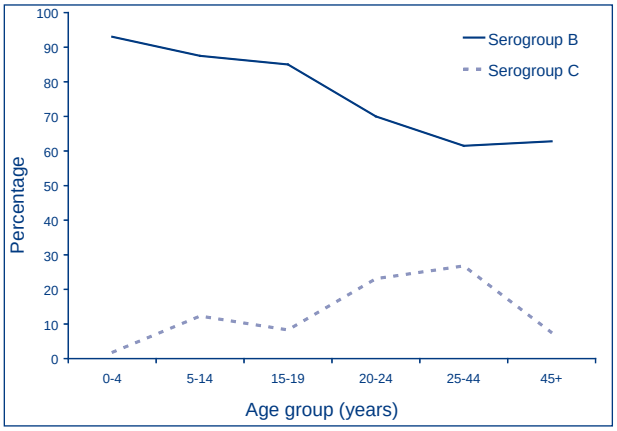


Figure 2. Serogroup B and C meningococcal disease as a percentage of cases of invasive meningococcal disease confirmed by all methods, Australia, 2006, by age group



Serogroup data

The serogroup of the meningococci causing disease was determined in 259 of the 271 laboratory-confirmed cases of IMD. Of these 259 where a serogroup was determined, 217 (83.8%) were serogroup B and 26 (10%) were serogroup C. In 2005, there were 251 (76.9%) confirmed cases of serogroup B and 50 (15.3%) of serogroup C and in 2004, 243 (73%) serogroup B cases and 71 (21%) serogroup C cases. In 2006, an additional 13 (5%) cases were W135 and 3 (1.2%) were serogroup Y.

The serogroup distribution varied with age (Figure 1) and jurisdiction (Table 2), as in previous years. Traditionally, serogroup B disease is concentrated in younger age groups with serogroup C infections increasing as a proportion of all isolates in adolescents and young adults (Figure 2).

Serogroup B meningococci predominated in all age groups in aggregated national data. Ninety-three (93%) of the total of 100 laboratory-confirmed IMD cases in those aged less than 4 years were serogroup B and 2 (2%) serogroup C. In those aged 5–14 years, 21 serogroup B meningococcal cultures represented 84% of the 25 confirmed cases and the 3 cases of serogroup C represented 12% of cases. There were 49 cases of IMD confirmed nationally in those aged 15–19 years in 2006 compared with 48 in 2005. These 49 cases comprised 40 (82%) serogroup B and 4 (8.2%) serogroup C—virtually identical numbers and proportions were reported in 2005. There were 30 instances of IMD in those aged 20–24 years in 2006. Of these, 21 (70%) were serogroup B and 7 (23%) were serogroup C. These numbers and proportions differ little from 2005 data for this age group when 22 (7%) infections with serogroup B and 8 (24%) with serogroup C meningococci were recorded out of a total of 33 cases. (In 2004, the number of infections, 35, and their distribution, 20 (57%) of serogroup B and 11 (35%) of serogroup C, was not too dissimilar).

Table 2. All laboratory-confirmed cases of invasive meningococcal disease, Australia, 2006, by age group, state or territory and serogroups B and C

State or territory	Serogroup	Age group										Total
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
ACT	B						1					1
	C						1					1
	Total						2					2
NSW	B	14	7	4	3	10	6	4	7	2		57
	C	1		2		2	5	4	1	1		16
	Total	15	9	6	3	14	13	9	11	4		84
NT	B	1	1	1								3
	C											0
	Total	1	1	1								3
Qld	B	16	17		1	15	5	4				58
	C	1				1		2				4
	Total	17	18	1	4	18	5	7	1			68
SA	B	2	2			1		2	2	1	3	13
	C											0
	Total	2	2			1		2	3	1	4	15
Tas	B		1	1				1	1			4
	C					1						1
	Total		1	1		1		1				4
Vic	B	12	12	8	2	13	6	4	6	1		64
	C						1	1		1		3
	Total	13	12	8	2	14	7	7	7	5		75
WA	B	7	1	1		1	3	1	1	1	1	17
	C				1							1
	Total	8	1	1	1	1	3	1	1	1	1	19
Australia	B	52	41	15	6	40	21	16	17	5	4	217
	C	2	0	2	1	4	7	7	1	2		26
	Other	2	3	1	0	5	2	4	6	4	1	28
	Total	56	44	18	7	49	30	27	24	11	5	271
	% of all	20.7	16.2	6.7	2.6	18	11	10	8.9	4	1.9	

NS Not stated. (Totals include cases due to other serogroups (n = 27) and cases where the serogroup was not determined (culture confirmed 0, NAA confirmed 8 and serology confirmed 3).

In older age groups (25 years and above), there were 62 laboratory-confirmed cases of IMD in 2006, of which 38 (61.2%) were serogroup B and 10 (16.1%) were serogroup C. The latter number is less than the 27 serogroup C cases seen in older age groups in 2005 and the 32 seen in 2004.

Table 3 shows a comparison of the number and proportion of serogroup B and C cases by age group from 2004 to 2006. There was no change in the number of serogroup B cases in those aged 4 years or less or in those aged 15–24 years, but a reduction in serogroup B numbers in those aged 5 to 14 years from 38 in 2005 to 21 in 2006. There has also been a substantial decline in the number of cases of IMD in those aged more than 25 years—from 101 to 61. This occurred for both serogroup B (to 38 from 51) and serogroup C (to 10 from 27) infection from 2005 to 2006.

Some pronounced differences in the jurisdictional distribution of serogroup B and C meningococcal have been noted previously in Australia. In recent years serogroup B infections predominated nationally and in all jurisdictions, but serogroup C disease was more prevalent in the eastern States and uncommonly encountered in South and Western Australia. The number of serogroup C cases nationally in 2006 was about half that for 2005. Serogroup B disease now accounts for more than 80% of IMD nation-

Table 3. A comparison of the number and proportion of serogroup B and serogroup C laboratory-confirmed cases, 2004 to 2006, by age group

Year	Serogroup	Age group									
		< 4 years	%	5–14 years	%	15–19 years	%	20–24 years	%	25+ years	%
2006	B	93	93.0	21	84	40	82	21	70	38	61.3
	C	2	2.0	3	12	4	8.2	7	23	10	16.1
	All	100		25		49		30		62	
2005	B	99	90.0	38	75	39	81.0	22	67	51	50.0
	C	6	5.5	5	10	4	8.0	8	24	27	27.0
	All	110		51		48		33		101	
2004	B	97	88.0	27	77	40	65.0	20	57	59	50.0
	C	6	5.5	5	14	17	28.0	11	31	32	27.0
	All	110		35		61		35		117	

ally with little jurisdictional variation in serogroup distribution. Numbers of serogroup C cases in most jurisdictions were low, but 16 of the 26 cases of serogroup C disease nationally occurred in New South Wales. Eleven of these 16 serogroup C cases in New South Wales were in those outside the 'vaccination' age group. In Queensland, while the total number of IMD cases increased, the numbers of serogroup C infections was low. Clusters of serogroup C disease were not uncommonly encountered in past years, but none were reported in 2006.

Phenotypes of invasive meningococcal isolates

Typically there is considerable heterogeneity in serogroup B meningococci and this was again the case in 2006 when the phenotype of invasive isolates, based on a determination of their serogroup, serotype and serosubtype, were analysed. The predominant serotypes/serosubtypes in each state and territory are shown in Table 4. Serogroup B meningococci are in general also more difficult to characterise by serological methods and a number could not be phenotyped. A total of 14 isolates of the B:4:P1.4 phenotype were identified in Victoria, New South Wales, Queensland and South Australia

Table 4. Common serotypes and serosubtypes of isolates from culture positive cases of *Neisseria meningitidis* infection, 2005, by state or territory

State or territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
ACT	4	1	1.7	1	2a	1	1.4	1
NSW	4	11	1.4	4	2a	8	1.5	7
			1.7	3			1.5,2	1
			1.15	1	nt	1	nst	1
			1.5	1				
			1.3,6	1				
			nst	1				
	2a	1	1.5	1				
	15	6	1.7	3				
			1.7,14,	1				
			nst	2				
	1	6	Various	4				
			nst	2				
	nt	17	nst	7				
			1.4	4				
			1..14	3				
			1.9	2				
			1.10	1				

Table 4. Common serotypes and serosubtypes of isolates from culture positive cases of *Neisseria meningitidis* infection, 2005, by state or territory, *continued*

State or territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
NT	nt	3	nst	3				
Qld	15	7	1.7	5	2a	1	1.5	1
			1.7,16	1	nt	2	1.5	1
			nst	1				
	1	4	Various	4			nst	1
	4	2	1.4	2				
	2a	1	1.5	1				
	nt	20	1.4	9				
			1.9	2				
			1.5	1				
			1.6	1				
			1.14	1				
			nst	6				
Tas	4,,7	2	nst	2	2a	1	1.4	1
	nt	1	nst	1				
Vic	4,7	14	1.19,15	5	2a	1	1.7,4	1
			1.22,14	3	2b	1	1.5,2	1
			1.5	3				
			1.7 (4)	2				
			1.18	1				
	15	5	1.7	5				
	19,1	7	1.18	4				
			1.22,14	3				
	19	3	Various	3				
	7	6	1.19	5				
			1.7,4	1				
WA	14	3	1.2,14	3	2a	1	nst	1
	15	1	1.2,14	1				
	1	1	nst	1				
	nt	2	1.19,15	1				
			nst	1				
SA	15	1	1.4	1				
	nt	7	1.14	3				
			nst	4				

nt Not serotypable.

nst Not serosubtypable.

in 2006. Numbers of isolates of this phenotype, circulating in New Zealand at high rates for many years, have declined in recent years in Australia. Historically, the other common phenotype circulating has been B:15:P1.7 and remained so in 2006. In 2006 nationally, a total of 16 examples were detected from the Australian Capital Territory, Victoria, New South Wales, Queensland and Western Australia. This distribution was also observed in 2005.

There is continuing interest in the presence of any serogroup B or serogroup C meningococci of serotypes that indicate the possibility of genetic recombination events, e.g. serogroup B isolates of serotype 2a or 2b. A single isolate of B:2a:P1.5 was found in New South Wales in 2006. Among serogroup C strains, phenotype C:2a:P1.4 is of particular interest. This phenotype has figured prominently in Victorian data in former years. In 2003 there were 29 cases while in

2004 there were 21 and in 2005 8 serogroup C isolates of this serotype/serosubtype were detected nationally. Eight isolates with this phenotype were seen nationally in 2006 and these were detected in the Australian Capital Territory, Victoria, New South Wales and Queensland, all in low numbers. All except one of the serotypeable serogroup C isolates was of serotype 2a. The most frequently detected 2a serosubtype, 1.5, was present only in New South Wales and Victoria.

Outcome data for invasive meningococcal disease for all laboratory-confirmed cases

Outcome data (survived or died) were available for 125 (46%) of the 271 laboratory-confirmed cases (Table 5). Nine deaths were recorded in this group (7.2%). Outcomes were available for 99 of 217 (45.6%) serogroup B infections and 12 of 26 (46%) serogroup C infections. There were 6 (6%) deaths from serogroup B infections and a single death attributable to serogroup C disease. The other 2 fatal cases were with infections due to W135 meningococci.

There were 3 deaths in 44 patients (6.8%) with meningitis, all due to serogroup B meningococci. Six deaths were recorded in 68 bacteraemic patients (8.8%). There were 58 cases of serogroup B meningococcal bacteraemia with 3 deaths (5%). The single fatality with serogroup C disease was in a group of 5 cases where outcomes were recorded and the 2 septicaemic fatalities due to W135 meningococci were recorded in 5 instances of bacteraemia with this serogroup.

Anatomical source of samples for laboratory-confirmed cases

Table 6 shows the source of clinical samples by which laboratory confirmation of IMD was obtained. Those diagnoses shown as culture positive may have had positive PCR and/or serology, those shown as PCR positive were culture negative with or without positive serology and those shown as serologically positive were culture and PCR negative. There were 48 isolates from CSF either alone or with a blood culture isolate and 116 from blood cultures alone. There were 2 other isolates from synovial fluid. The ratio of CSF isolates to blood culture isolates was 0.4:1. For PCR-based diagnoses, this ratio was 0.8:1.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

One hundred and sixty-seven isolates were available for determination of their susceptibility to penicillin. Using defined criteria, 113 isolates (67.6%) were less sensitive to penicillin in the MIC range 0.06–0.5 mg/L and 55 (32.4%) were fully sensitive

Table 6. Anatomical source of samples positive for a laboratory-confirmed case of invasive meningococcal disease, Australia, 2006

Specimen type	Isolate of meningococci	PCR positive*	Total
Blood	116	50	166
CSF +/– blood	48	42	90
Other†	2	5	7
Serology alone‡			8
Total	166	97	271

* Polymerase chain reaction (PCR) positive in the absence of a positive culture.

† Joint and tissue samples (5) or not stated (3).

‡ Serology positive in the absence of positive culture or polymerase chain reaction.

Table 5. Outcome data (survived, died) for laboratory-confirmed cases of invasive meningococcal disease, 2006, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	35	3	1	2	0	41
	Died	3	0	0	0	0	3
	Total	38	3	1	2	0	44
Septicaemia	Survived	58	4	1	3	2	68
	Died	3	1	0	2	0	6
	Total	61	5	1	5	2	74
All cases	Survived	93	11	2	5	5	115
	Died	6	1	0	2	0	9
	Total	99	12	2	7	5	125

NG Not groupable.

(≤ MIC 0.03 mg/L). These proportions are similar to those observed in recent years. Six isolates had MICs of 0.5 mg/L. Four of these were serogroup C isolates from New South Wales.

Other antibiotics

All isolates were fully susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins) and to ciprofloxacin. A single serogroup B strain from Queensland had a slightly elevated MIC for rifampicin of 1 mg/L.

Discussion

There has been a continuing decline in the number of laboratory-confirmed cases of IMD in Australia for several years[10] and this continued in 2006. Numbers declined in all states and territories with the exception of Queensland. Cultures were obtained from sterile sites in 166 cases, the lowest number of isolates available over the duration of the program that commenced in 1994. Non-culture-based diagnoses were used to confirm a further 105 (38.7%) cases IMD. This decrease in IMD has been attributable to all serogroups, but notably in serogroup C infections and the 26 cases of this serogroup identified nationally, accounted for 9.6% of all cases in 2006.

Traditionally, the distribution of cases of IMD in Australia showed major differences when considered by jurisdiction, age and serogroup of the infecting organism. Western and South Australia have long had a preponderance of serogroup B infections whereas Victoria, Tasmania and the Australian Capital Territory tended to have a greater proportion of serogroup C infections than New South Wales or Queensland. Differences between jurisdictions in serogroup distribution have become less in recent years with the relatively greater decline in serogroup C infections. Nationally in 2006, serogroup B infections were 8 times more common than serogroup C IMD. Only in New South Wales did the number of serogroup C cases detected reach double figures. Most of these 16 cases were in those outside the target age groups for vaccination programs with serogroup C conjugate vaccine. Only small numbers of infections due to serogroups Y and W135 were encountered.

Serogroup B infections were again more frequently encountered in younger age groups where there is a primary peak in IMD infection rates. In the earlier years of this Programme, serogroup C disease figured prominently in a secondary disease peak that occurred in adolescents and young adults in some jurisdictions. However, serogroup C infections were infrequently encountered in this and other age groups in 2006. Also of interest is the overall decline in numbers of IMD in those aged 25 years or more (Table 3). There has been a decrease in both serogroup B (by about one third) and serogroup C cases (by about two thirds) in this age group since 2004.

The NNN is not as well placed as others to analyse the effect of the national vaccination program with serogroup C conjugate vaccine on trends in IMD for reasons previously discussed.[10] These included differences over time in data collection and laboratory detection methods. Further, fluctuations in the rates of IMD can occur naturally or be influenced by rates of intercurrent viral infection. However, it is still possible to speculate that the decrease in serogroup C disease in older age groups may be the result of a 'herd' immunity effect whereby the number of circulating serogroup C meningococci is reduced by the vaccination program in younger age groups who may otherwise be responsible for the introduction of a pathogenic strain into family groups. This does not explain the accompanying, but lower, decrease in serogroup B disease.

The continuing absence of any substantial numbers of meningococci showing evidence of genetic recombination is reassuring and consistent with findings elsewhere. Some concerns have been expressed that the well established ability of *Neisseria meningitidis* to undergo substantial genetic reconfiguration by a number of mechanisms may pose threats to the longer term efficacy of monovalent capsular vaccines. Analysis of meningococcal subtypes and any evidence for the expansion of 'new' subtypes will continue as part of the NNN program.

Mortality data were assessable in only a proportion of cases and must be interpreted with caution. The NNN does not attempt collection of morbidity data associated with IMD.

NNN trend data show no major shifts in penicillin MICs in invasive isolates in Australia and penicillins remain a suitable treatment for IMD. The 4 New South Wales serogroup C isolates with penicillin MICs of 0.5 mg/L were of the same phenotype. However, this was the common serogroup C phenotype in New South Wales in 2006 and no case linkages were established. All isolates were susceptible to the third generation cephalosporins and to the prophylactic agents rifampicin and ciprofloxacin.

Acknowledgments

Isolates were received in the reference centres from many laboratories throughout Australia. The considerable time and effort involved in forwarding these strains is recognised and these efforts are greatly appreciated. These data could not have been provided without this assistance and the help of clinical colleagues and public health personnel.

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Creutzfeldt-Jakob disease: Australian surveillance update to March 2007

Genevieve M Klug, Alison Boyd, Victoria Lewis, Samantha L. Douglass, Helene Roberts, Rebecca Argent, Colin L Masters, Steven J Collins

Abstract

From October 1993, prospective, national surveillance of the rare class of neurodegenerative diseases known as transmissible spongiform encephalopathies (TSEs) has been performed by the Australian National Creutzfeldt-Jakob Disease Registry. Surveillance of TSEs prior to October 1993, involved the retrospective ascertainment of TSE cases from 1970 to 1993. In this report, surveillance data for 2006 are presented in detail and compared to cumulative national TSE ascertainment as well as international experience. The higher incidence of TSEs in 2006 is not without precedent and can be attributed to higher referrals and consequent post-mortem rates. *Commun Dis Intell* 2007;31:194–197.

Keywords: communicable diseases, cjd, Creutzfeldt-Jakob disease, transmissible spongiform encephalopathies

Introduction

Transmissible spongiform encephalopathies (TSEs) comprise a unique group of transmissible neurodegenerative disorders, including Creutzfeldt-Jakob disease (CJD), Gerstmann Sträussler-Sheinker syndrome, fatal familial insomnia and variant CJD (vCJD). Pathogenesis centres on a conformational change of the endogenous normal prion protein (PrPc) to a disease-associated conformer (PrPres), with subsequent accumulation in the brain, associated with neuronal damage, spongiform change and ultimately death. Precise mechanistic details concerning the molecular conversion of PrPc and

the subsequent pathogenetic pathways remain to be elucidated. Most human TSE cases arise sporadically with no plausible explanation (approximately 85%). Less commonly, TSEs are linked to a mutation in the prion protein gene (*PRNP*) (approximately 13%–15%) or arise through horizontal transmission (most commonly an iatrogenic event). Iatrogenic CJD has been associated with several different transmission routes, including neurosurgical procedures, parenteral hormone therapy and more recently for vCJD, through the transfusion of blood products in the United Kingdom (UK).[1,2]

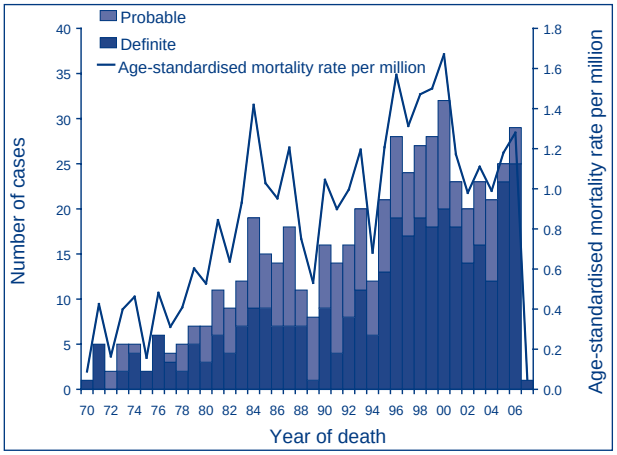
Australian National Creutzfeldt-Jakob disease Registry surveillance

After 13.5 years of Australian national surveillance, the Australian National Creutzfeldt-Jakob disease Registry (ANCJDR) has ascertained 1,178 cases of suspect TSE. Of these, 489 have been evaluated and classified as non-TSE on the basis of detailed follow-up of clinical histories and/or neuropathological examination. The remaining 689 cases have been classified as TSE according to internationally recognised case definitions[3] or are still under investigation (Table 1). As of 31 March 2007, the Registry has classified 344 cases as definite CJD, 203 cases as probable CJD and there are 11 possible cases. Definite cases are classified after confirmation of disease by neuropathological examination (usually post-mortem), whereas a probable case classification is determined by the clinical features and diagnostic investigation results that typify CJD. Investigation of the 131 incomplete cases is ongoing, with 86 of these patients still alive. The average, age-adjusted, CJD mortality rate for the entire 1970–2007 cohort is 0.86 deaths per million per year. For the prospective ascertainment period (1993– 2007), which provides a more accurate rate of CJD deaths in Australia, the mortality rate is 1.16 deaths per million per year.

TSE surveillance summary to 31 March 2007 with an emphasis on 2006

In 2006, increases in both notifications of suspect CJD cases (30% increase) and the number of confirmed CJD cases (21% increase) were observed compared with the previous year (Figure, Table 2). This annual number of referrals and CJD cases has been observed previously by the ANCJDR during 1999–2000 (Figure), a period that correlated with intense media attention regarding vCJD both domestically and internationally. Part of the explanation for the 2006 increase relates to increased referrals of suspect cases for cerebrospinal fluid 14-3-3 protein diagnostic testing offered through the ANCJDR and to a lesser extent through personal communications from family members and clinicians. These increased ante-mortem notifications enabled a greater number of autopsies to be conducted in this patient group, leading to the confirmation of a greater number of cases than usual, underscoring the importance of ongoing facilitation of post-mortem examinations.

Number and age-standardised mortality rate for ANCJDR definite and probable cases, 1970 to 31 March 2007



Mortality rates were calculated using the Australian Bureau of Statistics 2000 resident population estimates for Australia.

Table 1. Classification of cases on the Australian National Creutzfeldt-Jakob disease Registry, 1 January 1970 to 31 March 2007

Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total
Definite	303	36	5*	0	0	344
Probable	189	10	4	0	0	203
Possible	10	0	1	0	0	11
Incomplete	0	0	0	0	131†	131
Total	502	46	10	0	131	689

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

† Includes 86 living cases.

Table 2. Number of suspect cases notified and Creutzfeldt-Jakob disease outcomes, 1997 to 2007

Referral year	Suspect cases	CJD	% CJD
1997	69	29	42.0
1998	85	29	33.7
1999	102	33	32.0
2000	83	28	33.7
2001	58	26	44.8
2002	59	22	37.3
2003	54	22	40.7
2004	71	24	33.8
2005	66	23	34.8
2006	88	28	31.8
2007	21	1	4.7
Total	756	265	35.0

Fluctuating annual peaks in total CJD numbers is anticipated as a natural temporal variation in such a rare disease.

Three quarters of the cases notified in 2006 were ascertained via referrals for the cerebrospinal fluid 14-3-3 protein testing. The ANCJDR began offering 14-3-3 testing in September 1997, and since then the diagnostic test has been the initial referral source of 476 suspect CJD cases. Based on the total 1,178 referrals, since October 1993, the 14-3-3 testing has accounted for 40% of all referred suspect cases and establishes the diagnostic test as the most effective notification source of suspect CJD in Australia. The ANCJDR continues to provide this national diagnostic service.

The Australian CJD surveillance findings are congruous with those reported by international CJD surveillance units (notwithstanding the absence of a case of vCJD in Australia). In accordance with global experience, as reported previously, the large majority of definite and probable Australian cases (for the period 1970–2007) occurred sporadically (approximately 90%) with no obvious familial or iatrogenic association.[4] Familial cases (due to *PRNP* mutations) accounted for 8.4% of all Australian CJD cases and the remaining 1.5% of cases, were iatrogenic cases. For sporadic cases, the median age at death was 67 years (males, 66 years; females, 67 years). Fifty-four per cent of sporadic cases were female and their median duration of disease was 4 months. In men, slightly shorter disease duration was observed (median, 3 months). In comparison to sporadic CJD, genetically determined TSEs typically have a younger age at death (median, overall 59 years; males, 51 years; females, 62 years) and longer illness duration (median, overall 6 months; males, 4 months; females, 7.5 months) but equal sex ratio, identical to the sporadic CJD cohort. If compared to the total sporadic CJD cohort, the 2006 CJD cases had a similar age at death (overall, 64 years, males, 64 years, females 65.5 years), and duration (overall, 3 months, males, 2 months, females, 3 months) and the sex ratio was also consistent.

Due to the difficulty in differentiating sporadic CJD from some genetically determined forms of CJD, testing to detect mutations in *PRNP* is offered to the majority of patients with definite and probable CJD, but particularly when a family history of CJD exists, or when families are particularly interested in testing. Genetic testing is not systematically performed in Australia, in contrast to several European Union (EU) countries, where typically more than 50% of all CJD cases have undergone genetic testing.[5] Despite the non-routine genetic testing approach within Australia, the proportion of Australian TSE with an underlying *PRNP* mutation ascertained during the prospective surveillance period (1993–2007) was 10.2%, and is comparable with other EU countries consistently undertaking higher rates of genetic testing of CJD cases.[5] Over the last 12 months, at the time of publication, no cases of vCJD or further cases of iatrogenic CJD have been identified.

ANCJDR surveillance for vCJD

One of the primary ANCJDR activities is the surveillance of potential vCJD cases in Australia. The first identified case of vCJD was in the United Kingdom in 1995[6] and since this time the worldwide total of primary vCJD cases has reached 199, with 11 of these cases still alive (Table 3).[5] In the UK, three further symptomatic cases have been classified as secondary vCJD, with disease transmission almost certainly arising via the transfusion of blood products (non-leucodepleted red blood cells) from donors who developed the disease subsequent to donation. It is now evident that the primary disease epidemic has lessened in the UK,[7] yet in France the number of cases has increased and plateaued in 2005/2006. Despite the decline in the UK, there is still cause for concern regarding the potential for a prolonged 'tail' to the primary transmission epidemic based on differential genetic susceptibilities in the population leading to longer incubation periods, and the potential for secondary transmissions from sub-clinical carriers who may never develop disease but may transmit CJD in the health care setting.

To date, vCJD has not been identified in Australia. With the recognition of vCJD in Canada, the Republic of Ireland, Japan and the United States of America (USA), and given similarities in the reciprocal migration patterns and travel profiles of residents of Australia, Canada and the USA with

the UK, questions persist as to why this is the case. The number of Australians potentially exposed to contaminated UK meat products during the high risk period of 1980–1996 is large, and thus the likelihood of identification of at least 1 to 2 cases of vCJD in Australia is anticipated (personal communication, S Collins). Upon suspicion of this type of CJD, diagnostic testing on tonsil or brain biopsies to detect PrPres is offered by the ANCJDR and since 2000, on average, 1 Australian suspect vCJD case has been investigated every 2 years.

One possible explanation for the lack of identification of vCJD so far in Australia is clinical misdiagnosis or non-identification, but this is considered unlikely. Of all suspect TSE referrals between 1993 and 2006 where death is known to have occurred, 58% have undergone neuropathological examination. This large proportion of autopsied cases, accompanied by thorough ANCJDR screening of medico-demographic data and a comprehensive review of the available molecular-clinical profiles of Australian sporadic cases (where death occurred between 1992–2003 and appropriate tissue samples were available),[8] provides an underlying confidence that a case of vCJD has not been missed in Australia. The ANCJDR surveillance for vCJD continues to rely heavily on notifications and access to comprehensive clinical information. Variant CJD has a distinctly different clinical profile to classical CJD. Patients are typically younger at age of onset (12–74 years; median 26 years),[7] present with psychiatric and/or limb sensory symptoms, and have longer illness durations with later onset of dementia. Investigation findings also vary from classic CJD: the electroencephalograph is typically negative for periodic complexes; the MRI shows a characteristic high T2 signal in the pulvinar region of the thalamus; and the PrPres molecular pattern is distinctive. On brain biopsy or autopsy examination, neuropathological findings in the brain also distinguish vCJD and classical CJD.

Table 3. Worldwide number of variant Creutzfeldt-Jakob disease, to April 2007

Country	Total number of primary cases	Number alive
United Kingdom	162	6
France	22	2
Republic of Ireland	4	1
USA	3	0
Netherlands	2	0
Canada	1	0
Italy	1	0
Japan	1	0
Portugal	1	1
Saudi Arabia	1	1
Spain	1	0
Total	199	11

Adapted from: http://www.eurocjd.ed.ac.uk/results.htm

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Articles

Compliance with three simultaneous vaccinations due at the one visit at 12 months of age in Australia

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Abstract

The introduction of meningococcal C conjugate vaccine (Men C) into the National Immunisation Program Schedule in January 2003 was the first time that 3 simultaneous vaccine injections were recommended for all Australian children. This study aimed to assess the level of simultaneous vaccination at 12 months of age for 4 cohorts of Australian children. The percentage of children with all 3 vaccinations given simultaneously by jurisdiction increased for all states and territories across the 4 study cohorts, however some jurisdictions fared better than others. The percentage of children with all 3 vaccinations given simultaneously varied by the provider type of the Men C vaccine, being lower for general practitioner providers than other providers. Men C vaccine was the vaccine most commonly delayed. The percentage of children who received all 3 vaccinations simultaneously in Australia also varied by indigenous status, with Indigenous children more likely to receive immunisations simultaneously. The study suggests that some children in Australia are at risk of receiving Men C vaccine late, especially children in jurisdictions where general practitioners give the majority of vaccinations. *Commun Dis Intell* 2007;31:198–202.

Keywords: multiple injections, compliance, vaccination coverage, immunisation register, simultaneous vaccination

Introduction

The meningococcal C conjugate vaccine (Men C) was introduced into the National Immunisation Program (formerly the Australian Standard Vaccination Schedule) in January 2003. The vaccine is recommended at 12 months of age, the same age as the administration of the first dose of measles, mumps, and rubella (MMR) and the third dose of the *Haemophilus type B* vaccine (Hib). The introduction of Men C was the first time that 3 simultaneous injections were recommended for all Australian children at any schedule point.

Few studies have examined whether, and to what extent, vaccinations are delayed when more than two are due at the one time. Older studies from the United States of America, utilising attitudinal surveys of providers and analysis of medical record databases, are conflicting. Some report that providers were concerned about administering multiple vaccinations at one visit and were delaying vaccinations, whilst others reported almost complete compliance with multiple, simultaneous vaccinations.[1–6] A more recent Australian study from north Queensland has shown that 17% of non-Indigenous and 18% of Indigenous children did not receive the 3 injections, due by 12 months of age, at the one visit to an immunisation provider. Men C vaccine, the one most recently recommended, was most likely to be omitted.[7] These findings have implications for infant 7vPCV vaccination due at 2, 4 and 6 months of age introduced at the beginning of 2005 in Australia.

The aims of the current study were to determine the percentage of 4 consecutive cohorts of children in Australia who received the 3 vaccinations due at 12 months of age and of these, the proportion receiving 3 vaccinations simultaneously at the national level and by state or territory, indigenous status and provider type.

Methods

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases receives downloads of Australian Childhood Immunisation Register (ACIR) immunisation data from Medicare Australia (formerly the Health Insurance Commission) each quarter. We chose four 3-month wide birth cohorts, the first born 1 April to 30 June 2002, the second born 1 July to 30 September 2002, the third born 1 October to 31 December 2002, and the fourth born 1 January to 31 March 2003, as our study population. The vaccination records of all children born in these 4 cohorts in Australia and who were registered with Medicare were extracted from ACIR data as at 31 December 2004. This allowed the youngest children in the latest 3-month birth cohort (born 1/1/03 – 31/3/03) to reach 18 months of age at data analysis, giving at least 6 months to receive vac-

cinations scheduled at 12 months. The age of each child, the vaccine type and the dates of vaccinations were used to assess if and when children received the relevant 12-month vaccinations and if they received them simultaneously. The indigenous status field was used to assess any differences by indigenous status. This isn't a well completed section of the ACIR notification form in jurisdictions with smaller numbers of Indigenous children but completion has improved significantly in the past few years. The analysis was undertaken using the SAS software system.[8]

Results

There was little change over time in the percentage of children who received all 3 vaccinations by state or territory (regardless of whether administration was simultaneous or not) (Figure 1). A slight decrease from the third cohort to the fourth is most likely due to very late vaccinations not being recorded on the ACIR for children in the fourth cohort by the data assessment date. The Northern Territory (89.3% for the most recent cohort) had the greatest percentage of children with all 3 vaccinations received across all 4 cohorts, with Western Australia (75.6% for the most recent cohort) the lowest percentage. However, the percentage of children with all 3 vaccinations given simultaneously showed a steady increase in all states and territories across the 4 study cohorts (Figure 2). The Australian Capital Territory (86.2% for the most recent cohort), the Northern Territory (80.7%) and Victoria (79.9%) had the greatest percentage of children with all 3 vaccinations received simultaneously across all 4 cohorts. New South Wales (63.2%), Tasmania (63.0%), Western Australia (64.3%) and Queensland (65.2%) had the lowest percentage (Figure 2).

Analysis of the most recent cohort (born 1 January to 31 March 2003)

For more in-depth analysis, we examined the most recent cohort born 1 January 2003 to 31 March 2003. Of the 62,049 children in this cohort, 82.2% (n=51,004) children had a record on the ACIR of having received all 3 vaccinations due at 12 months of age. Of these 51,004, 15,701 (30.8% or 25% of the total cohort), did not receive the 3 vaccinations simultaneously. In this cohort, there were 11,045 children who did not receive all 3 vaccinations due at 12 months of age and were under-immunised. The vaccines these children were missing are shown in the Table. The large majority of children (54%) were missing a Men C vaccine only, with a further 20% missing all 3 vaccines.

The median age of receipt of Men C vaccine in the children who received the 3 vaccines simultaneously was 12.5 months, whilst the median age in the children who did not receive the 3 vaccines at the same time was 14 months.

Figure 1. Trends in the percentage of children with all three vaccinations, by state or territory

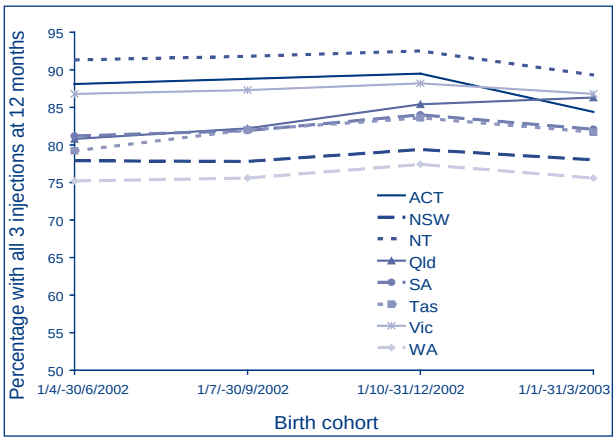
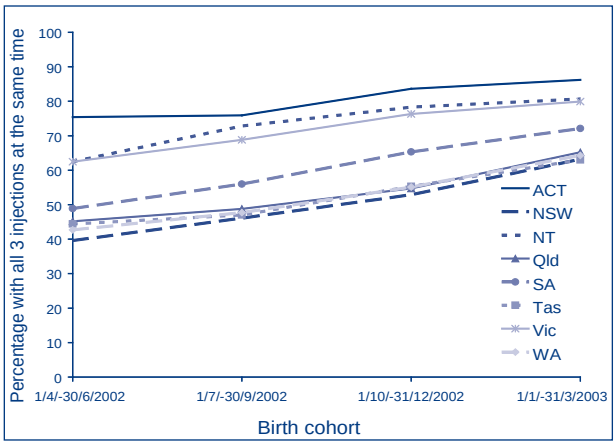


Figure 2. Trends in the percentage of children with all three vaccinations given simultaneously, by state or territory



The number and percentage of children who did not receive all three vaccinations due at 12 months of age, cohort born 1 January to 31 March 2003, by vaccine

Missing vaccines	Number	%
Men C only	5,963	54.0
All three vaccines	2,197	19.9
Men C and MMR	1,943	17.6
MMR only	594	5.4
Hib Only	168	1.5
Men C and Hib	96	0.9
MMR and Hib	84	0.8
Total	11,045	100.0

Men C Meningococcal conjugate vaccine
MMR Measles-mumps-rubella vaccine
Hib *Haemophilus type B* vaccine

The percentage of children who received all 3 vaccinations in Australia varied by indigenous status. Indigenous children (86.8%) were more likely to have received all vaccines than non-Indigenous children (82.0%) (Figure 3). Only in those jurisdictions with the lowest proportion of Indigenous children (the Australian Capital Territory, Tasmania and Victoria) did non-Indigenous children have a greater percentage of receiving all 3 vaccines than Indigenous children. A greater proportion of Indigenous (64.4%) versus non-Indigenous children (56.6%) also received vaccines simultaneously (Figure 4). The pattern varied by jurisdiction, with a smaller percentage of Indigenous children in the Northern Territory, South Australia, Tasmania and Victoria receiving vaccinations simultaneously than non-Indigenous children.

Figure 3. The percentage of children with all three vaccinations, cohort born 1 January to 31 March 2003, by state and indigenous status

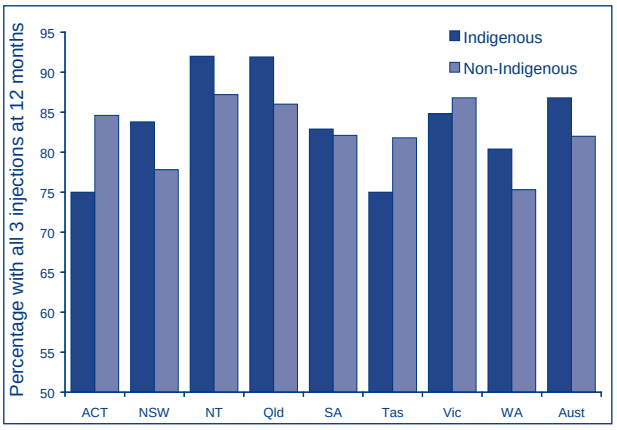


Figure 4. The percentage of children with all three vaccinations given simultaneously, cohort born 1 January to 31 March 2003, by state and indigenous status

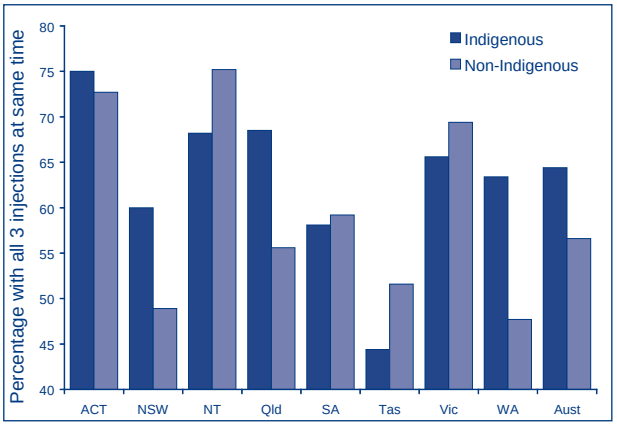
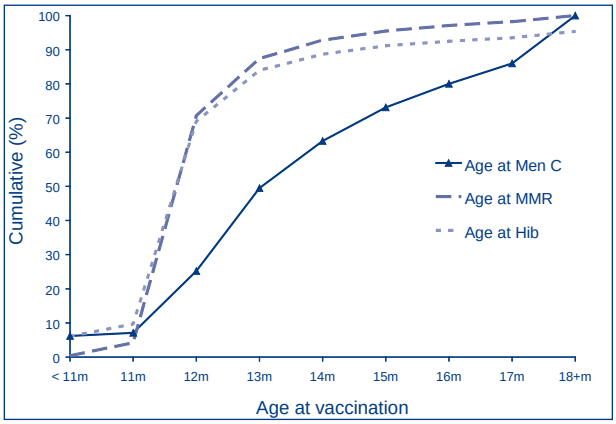


Figure 5 shows that, of the 3 vaccines given to children who did not receive them simultaneously, Men C was more likely to be delayed than MMR or Hib. Around 70% of children received the MMR and Hib vaccine at 12 months of age but only 25% received the Men C vaccine when it was due, with the delay in receipt of Men C vaccine continuing up to 18 months of age.

Figure 5. Age at vaccination for all three vaccines for children who did not receive the vaccinations simultaneously, cohort born 1 January to 31 March 2003



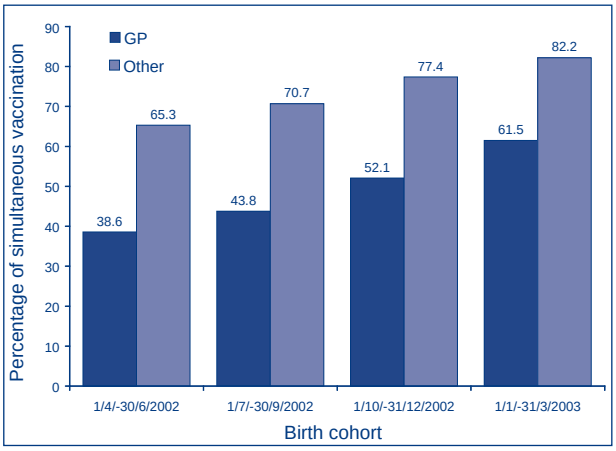
For all 4 study cohorts, the percentage of children with all 3 vaccinations given simultaneously varied by the provider type of the Men C vaccine with children who received their Men C from a general practitioner (GP) less likely to have received all 3 vaccines simultaneously, compared with children who received their Men C vaccine from another type of provider (Figure 6).

Discussion

This is the first study to examine the issue of simultaneous vaccination at 12 months of age in Australian children at the national level. A study undertaken in north Queensland in 2004 found that 83% of children had received the 3 injectable vaccinations simultaneously.[7] This figure is significantly higher than what we found for Queensland as a whole for the latest cohort studied (65%). However, simultaneous vaccination increased substantially over time for all jurisdictions, suggesting increasing acceptance by parents and providers.

Of more concern is the finding that the lowest levels of simultaneous vaccination occurred in the jurisdictions where general practitioners give the majority of immunisations: New South Wales, Queensland, Tasmania, South Australia and Western Australia. In fact, we found that GPs were less likely than other immunisation providers (by about 20 percentage

Figure 6. The percentage of children with all three vaccinations given simultaneously comparing GP versus Other, all four study cohorts, by provider type



GP' covers the following ACIR provider type categories: 'Division of General Practice', 'Medicare GP', and 'General Practice'

'Other' covers following ACIR provider type categories: 'Council', 'State Health', 'Flying Doctor', 'Community Health Centre', 'Community Nurse', 'Public Hospital', 'Private Hospital', 'Aboriginal Health Service', and 'Aboriginal Health Worker'

points) to give the 3 vaccines simultaneously. This suggests that GPs could be concerned about the logistics of administering multiple vaccination injections to young infants at the one visit and are delaying vaccinations to a later date.[2,5,6,9] State and council clinics, community health centres and Aboriginal health centres appear to have a greater degree of acceptance in giving 3 vaccines simultaneously. This could be related to differences in attending populations and their expectations, greater adherence to routine procedures or specific measures to allay parent's concerns regarding this.

Men C vaccine was the latest of the 3 vaccines, due by 12 months age, to be added to the National Immunisation Program, so it is no surprise that it was delayed the most, on average by up to 6 weeks, as in previous studies.[7] In fact, of those children who received all 3 vaccines due at 12 months of age but did not receive them all at the same time, 70% of them had received their MMR and Hib vaccines by up to 13 months of age but only 25% had received their Men C vaccine by this same age. Both the MMR and Hib vaccines are recognised by Australian immunisation providers and parents as highly effective and safe, despite the negative press MMR has received in the United Kingdom over the past 5 years. Men C vaccine is relatively new to Australian parents and its safety profile may not yet be well established. However, Men C is a vaccine for a disease that has attracted a significant amount of mainstream press over recent years, so it is surprising that parents and/or providers are choosing to delay its administration to young infants. In fact, Men C coverage for the most recent cohort in Western Australia, where there has been some publicity that the vaccine was not worthwhile, was the lowest nationally, by up to 13 percentage points when compared with all other jurisdictions. In this study, the median age of receipt of Men C vaccine in the children who had received the 3 vaccines simultaneously was 12.5 months, whilst the median age in the children who did not receive the 3 vaccines at the same time was 14 months. This was almost identical to that found in the north Queensland study, 12.3 and 14 months, respectively.[7]

In this study, Indigenous children were significantly more likely than non-Indigenous children to have received all 3 vaccinations due at 12 months of age, except in jurisdictions with a low proportion of Indigenous children. They were also more likely to have received all vaccine injections simultaneously, except for in the Northern Territory. This is different from the findings in north Queensland where the same percentage of Indigenous and non-Indigenous children received the 3 vaccines simultaneously.[7] However, this is not unexpected in light of the fact that community health centres and Aboriginal health centres appear to have a greater degree of acceptance in giving 3 vaccines simultaneously.

The findings of this study will have implications for those jurisdictions that have continued to use vaccines necessitating 3 simultaneous vaccinations at both the encounters due at 2, and 4 months after November 2005: the Northern Territory, Queensland, Victoria, and South Australia. Based on the results from the current study, this potential delay is more likely to occur in the jurisdictions where GPs give the majority of immunisations: Queensland and South Australia. As severe invasive pneumococcal disease notification rates are highest in children aged 1 year or less,[10] it is very important that delays in administering the 7vPCV vaccine to children due at 2, 4 and 6 months are minimised, and that providers and/or parents are informed about the consequences of delay. In particular, possible GP concerns over giving multiple injectable vaccines to young infants need to be addressed.

Limitations of this study are those common to all studies utilising data from the Australian Childhood Immunisation Register, most notably, the under-reporting of immunisation encounters. It is known that the ACIR underestimates coverage for scheduled vaccines by 3–5% because of failure of providers to report to it.[11] However, under-reporting is unlikely to affect the main findings of this study.

Another limitation of the study was our inability to determine why some parents or providers appear to delay the administration of Men C vaccine.

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Bordetella pertussis PCR positivity, following onset of illness in children under 5 years of age

Cheryn M Palmer, Brad McCall, Kari Jarvinen, Michael D Nissen

Abstract

Bordetella pertussis is a significant cause of respiratory illness and an ongoing public health problem. Pertussis polymerase chain reaction (PCR) testing has been widely utilised since 2001, especially in infants. Uncertainty exists as to how long PCR remains positive following symptom onset. Further information on the time frame for pertussis PCR testing would assist diagnosis, epidemiological research and disease control. The Brisbane Southside Population Health Unit (BSPHU) conducted a retrospective analysis of enhanced surveillance data from pertussis notifications between January 2001 and December 2005, in children less than 5 years of age, in the BSPHU reporting area with the aim to determine the possible range of duration of *Bordetella pertussis* PCR, from symptom onset for this age group.. Of 1,826 pertussis notifications to BSPHU between January 2001 and December 2005, 155 (8.5%) were children under 5 years of age, with 115 pertussis PCR positive results. Analysis indicated a range of PCR positivity from day one to day 31 from the onset of catarrhal symptoms with most (84%) being within 21 days from onset of catarrhal symptoms. The range of PCR positivity following onset of paroxysmal cough was from day one to day 38 with most (89%) being within 14 days from the onset of paroxysmal cough. This review of pertussis PCR data in young children showed that PCR positive results generally mirrored the understood length of infectivity with regard to both catarrhal symptoms and paroxysmal cough; namely that PCR positive results were obtained at least 21 days following onset of catarrhal symptoms and at least 14 days following onset of paroxysmal cough. *Commun Dis Intell* 2007;31:202–205.

Keywords: *Bordetella pertussis,* disease management, epidemiology, laboratory testing

Introduction

Bordetella pertussis infects the respiratory tract and the disease can have an insidious onset with catarrhal symptoms and later a paroxysmal cough.[1] Morbidity and mortality is greatest in young children, especially infants. Vaccination has reduced the incidence and mortality from pertussis infection, since the introduction of mass immunisation in 1942. However, pertussis continues to be a significant public health problem, with epidemics occurring every 3–4 years. While the majority of pertussis notifications occur in adult populations and vaccination uptake has been good among child populations; waning immunity and circulation of pertussis among adults continues to place younger children, especially under-vaccinated children, at risk of infection with potentially serious sequelae.[2,3,4]

Sensitive and specific laboratory investigations are vital for accurate diagnosis of pertussis. Available tests include bacterial culture, polymerase chain reaction (PCR) (nasopharyngeal aspiration or throat swabs) and serological assays (mainly to pertussis toxin). Culture can be fastidious and time consuming, while serology has several limitations, including the delay inherent in collecting paired sera to detect an antibody rise.[5] PCR is being used more frequently, especially in infants, and has a shorter turn-around time and higher sensitivity than culture.[5] Currently, there is no agreed inter-laboratory standardisation of PCR assays for *Bordetella pertussis*. In Queensland health laboratories, PCR methods for pertussis diagnosis have been utilised since 2000.[6,7]

Uncertainty exists as to how long PCR remains positive following symptom onset. A literature review, on the subject of pertussis PCR positivity, did not locate any publications addressing or resolving the issue of how long this test remains positive from the onset of infection. One Australian study in a boarding school outbreak, found PCR was more likely to be positive when specimens were collected within a week either side of symptom onset.[8] More definitive information on the time frame for pertussis PCR testing would assist diagnosis, epidemiological research and disease control.

Methods

In Queensland, pertussis PCR testing has been available since 1999–2000 and in wider use from 2001. Two PCR methods have been utilised for diagnosis of pertussis by Queensland Health; initially a PCR-Elisa-based method was utilised and from 2004 a real-time PCR assay has been in use.[6,7] Pathology laboratories in Queensland are mandated to notify population health units of definitive and suggestive evidence of pertussis infection; according to established notification criteria.[9] Definitive laboratory evidence is isolation on culture or detection by nucleic acid testing and suggestive laboratory evidence is based on serology. Pertussis notification data are recorded on the Notifiable Conditions System (NOCS) database; including demographic information and mode of diagnosis, whether culture, PCR or serological assay. The Brisbane Southside Population Health Unit (BSPHU) conducts enhanced surveillance of pertussis notifications in children less than 5 years of age. This surveillance includes collecting information on the date of onset of symptoms (catarrhal and paroxysmal cough), vaccination status, treatment received and contacts requiring prophylaxis.

Pertussis notification data from the BSPHU area between January 2001 and December 2005 were extracted from the NOCS database. These were further analysed with regard to PCR positive results in children less than 5 years of age. All data were entered into a purpose designed Excel® database for analysis.

Results

There were 1,826 pertussis notifications made to BSPHU between January 2001 and December 2005 and 155 (8.5%) of these notifications were in children under 5 years of age. Epidemics of pertussis infection occurred in 2002 and 2005 (Figure 1). Most notifications occurred in the youngest age groups. The proportion of notifications has increased in adult females, over the study period and the proportion of notifications in infants under 12 months increased in epidemic years.

Since 2001 information on pertussis notifications on the NOCS database has included the method of laboratory diagnosis. Most diagnoses were by serological assay (83%) but PCR use, almost exclusively in infants, had increased since 2001 (Figure 2).

Figure 1. Age distribution and annual trends pertussis notifications, Brisbane Southside Public Health Unit area, 2001 to 2005

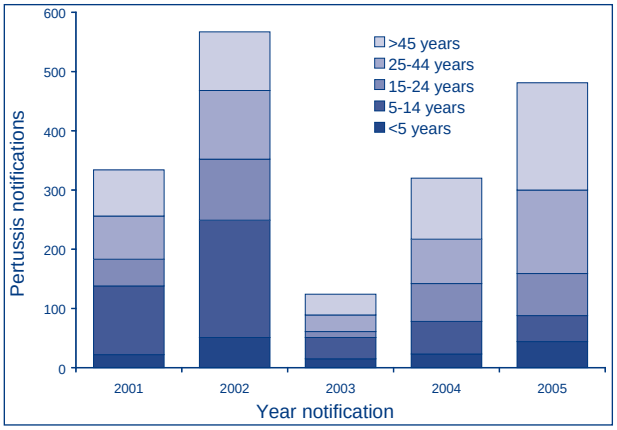


Figure 2. Age distribution of positive pertussis tests, Brisbane Southside Public Health Unit area, 2001 to 2005, by type

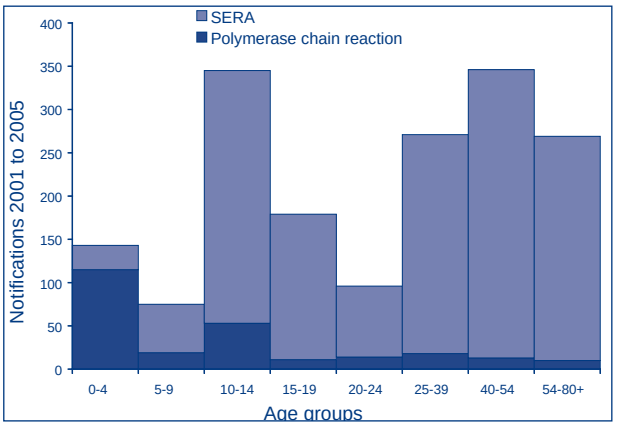
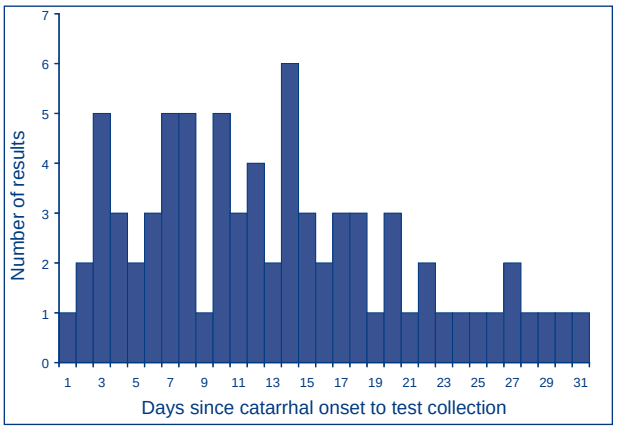
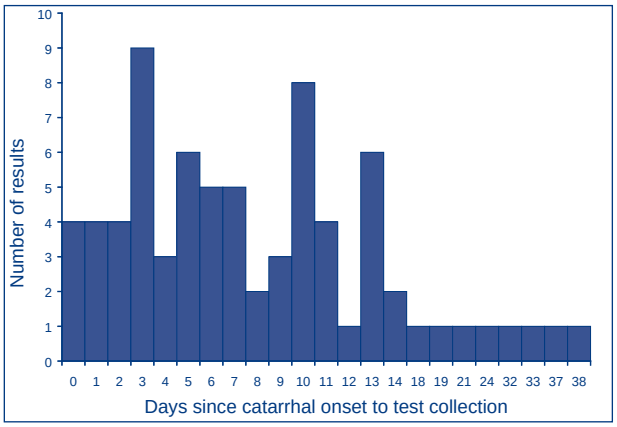


Figure 3. Polymerase chain reaction positive results by time (days) since catarrhal onset (<5 years age) N=75



Culture and antigen testing were infrequent. There were 115 PCR positive pertussis notifications during 2001 to 2005, in children aged under 5 years. Information was available on the date of onset of catarrhal symptoms in 75 (65%) of these notifications, the date of onset of paroxysmal cough in 74 (64 %) of these notifications and both pieces of information were available for 67 (58%) notifications. Analysis of these data indicated a range of PCR test positivity from day one to day 31 from the onset of catarrhal symptoms with most (84%) being within 21 days from onset of catarrhal symptoms (Figure 3).The range of PCR positivity following onset of paroxysmal cough was from day one to day 38 with most (89%) being within 14 days from the onset of paroxysmal cough (Figure 4).

Figure 4. Polymerase chain reaction positive results by time (days) since cough onset (<5 years age) N=74



Discussion

Over the last 5 years, pertussis notifications in the BSPHU have shown a shifting burden of illness toward older age groups and a recent increase in notifications in infants aged less than 12 months during epidemic years. The epidemiology of pertussis is evolving with a greater burden of disease among adults, including adults in contact with vulnerable under-vaccinated children. This shift in the age-related epidemiology of *Bordetella pertussis* has been noted across the western world, with increasing proportions of cases reported in adolescents and adults.[10,11] The limited duration of vaccine efficacy, complicated by changes in the methods and frequency of laboratory diagnosis may underlie the changing pattern. Interestingly, the cyclical pattern of pertussis epidemics has persisted since the pre-vaccine era to the present time.[11]

The review of pertussis PCR data in children under 5 years of age (between 2001 and 2005), showed that PCR positive results were associated with the length of infectivity with regard to both catarrhal symptoms and paroxysmal cough: PCR positive results were obtained at least 21 days following onset of catarrhal symptoms and at least 14 days following onset of paroxysmal cough. Positive results were infrequently recorded outside those time periods, with few positive results beyond 5 weeks from the onset of any symptoms.

There were several limitations to this study, including the retrospective nature and limitations of a selected cohort of pertussis positive notifications and a lack of detailed information about testing rates for pertussis. Results for children under 5 years of age may not be applicable to other age groups. The accuracy of data records relating to symptom onset may also be problematic, as it relies upon parent or care-giver recall.

Vaccination status would have been of interest but due to difficulties with data linkage was not possible. The lack of information in the medical literature and the preliminary findings of this study support the need for further investigation to determine the length of time PCR reliably remains positive from the onset of clinical illness.

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Pertussis epidemiology in Australia over the decade 1995–2005 – trends by region and age group

Helen E Quinn, Peter B McIntyre

Abstract

Important changes have occurred in the National Immunisation Program for pertussis during the decade 1995–2005, including the introduction of acellular pertussis vaccine for all doses, removal from the schedule of the booster dose at 18 months, and the introduction of a booster dose for adolescents. In addition, the coverage of pertussis vaccine at 12 and 24 months has substantially increased as recorded by Australian Bureau of Statistics surveys and the Australian Childhood Immunisation Register. There were 75,458 notifications nationally between 1995 and 2005, with little change in the annual number of notifications at the national level but with periodic epidemics, which varied among states and territories and dramatic changes in the age distribution of notified cases. Pertussis is well controlled in the 1–4 and 5–9 year age groups, and the highest annual notification rates continue to be in infants under 6 months of age. Adolescents aged 10–19 years had high notification rates in all states and territories, over this period, but 63% of notifications are now in the 20–59 year age range. Following the introduction of a fifth dose for adolescents, the current focus should be on protecting infants too young to be vaccinated and further defining the true morbidity of the disease in the elderly population. *Commun Dis Intell* 2007;31:205–215.

Keywords: pertussis, disease surveillance, immunisation

Introduction

Pertussis hospitalisations and deaths decreased dramatically in Australia following the introduction of mass immunisation in the 1950s, reaching record low levels in the 1970s and 1980s. However, particularly since measles control was achieved in 1998, notifications of pertussis have outnumbered those for any other vaccine preventable disease targeted by the National Immunisation Program (NIP).[1]

Important changes have occurred in the NIP for pertussis during the decade 1995–2005. From 1978, the Australian pertussis schedule, using a locally manufactured whole cell pertussis vaccine (DTPw), was 3 primary doses at 2, 4 and 6 months of age. A booster dose of DTPw at 18 months of age, which had previously been included in the schedule and was subsequently removed, was reintroduced in 1985. In 1994, a fifth dose at 4–5 years (school entry) was recommended. In 1997, acellular pertussis vaccines (DTPa) replaced DTPw for booster doses and from 1999 for all doses, although South Australia and the Northern Territory used DTPa for all doses from 1997. In September 2003, the spacing of the 5-dose schedule was changed, with the 18-month booster no longer recommended, based on evidence that 3 doses of acellular pertussis vaccine in the first year of life provide adequate protection until the age of 6 years.[2] From the beginning of 2004, an adolescent/adult formulated booster (dTpa) replaced the use of the adult diphtheria-tetanus vaccine (ADT) at 15–17 years of age. The latter change was prompted by evidence of a shift in pertussis notifications to these age groups following the introduction of the fifth dose of DTPa at 4–5 years.[1,3]

In addition, the coverage of pertussis vaccine at 12 and 24 months has substantially increased during this period. Coverage with three doses of DTP at 12–23 months of age was recorded at 86% during the 1995 Australian Bureau of Statistics survey,[4] rising to 92% for 3 doses of DTPa at 12 months and 95% for 3 doses of DTPa at 24 months, as assessed using the Australian Childhood Immunisation Register in December 2003.[1]

This paper summarises the age-specific trends in pertussis notifications over the 10 years (1995–2005) at the regional and national level in the context of the vaccine schedule and vaccine coverage changes during this period.

Methods

Notifications

In Australia, pertussis cases are notifiable under each state and territory Public Health Act. The criteria for notification can be a combination of clinical, epidemiological or laboratory evidence. Laboratory evidence includes culture of *Bordetella pertussis*, nucleic acid testing, serology or immunofluorescence assay.[5]

Disease notification data from the National Notifiable Diseases Surveillance System (NNDSS), for cases with an onset between 1 January 1995 and 31 December 2005, are included in this report. Notification data are presented and reported by date of onset. From 2000, the laboratory diagnosis method field in NNDSS was completed for more than 60% of notifications. Analysis using this field was performed for cases with an onset between 1 January 2000 and 31 December 2005. Only data from New South Wales, Queensland and the Northern Territory were included, as more than 70% of cases from other states and territories had an unknown diagnosis method recorded.

Population estimates

All rates were calculated using Australian Bureau of Statistics mid-year estimated resident populations, and are presented as annual rates or average annual rates per 100,000 total population, or population in age or geographical subgroups, as appropriate. Average annual rates were calculated by dividing the total number of cases for the period of investigation by the sum of the population for the same period.

Incidence rate ratios (IRR) were calculated to compare annual notification rates to state averages. Ninety-nine per cent confidence intervals (CI) and P values were calculated for each IRR using a standard procedure in EpiBasic (University of Aarhus, Denmark). Variables were explored for possible associations using a chi square test for trend, a P value of 0.05 was considered statistically significant.

Results

Secular trends

Pertussis is by far the most common vaccine preventable illness in Australia, with 75,458 notifications nationally between 1995 and 2005 (Table 1). Periodic epidemics of pertussis occur in Australia at intervals of 3–4 years, on a background of endemic circulation. In recent years the epidemic peaks have not been as evident in children aged less than 10 years. A seasonal pattern can be observed, with peak notifications in spring each year (Figure 1).

Regional variations in pertussis notification rates

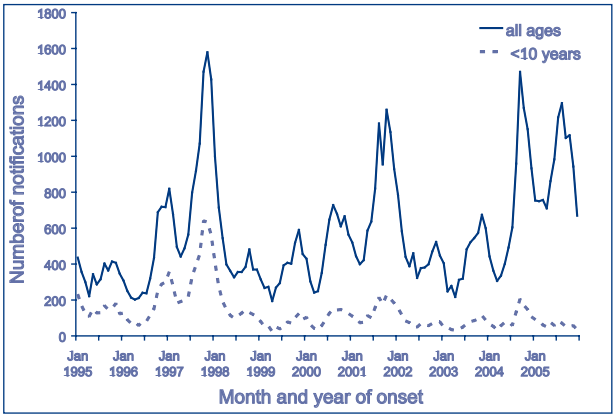
Table 1 shows crude national and jurisdictional notification rates over the decade 1995–2005 ordered by highest average rate from left to right. The national crude notification rate varied from 23.1 cases per 100,000 population in 1999 to 58.1 cases per

Table 1. Pertussis notification rates, Australia, 1995 to 2005, by state and territory

Year		State or territory								
		SA	NSW	ACT	Qld	Tas	WA	NT	Vic	Aus
1995		31.0	22.4	10.8	41.3	23.2	19.6	74.3*	9.0	23.2
1996		62.6	18.6	13.0	23.2	6.5	12.9	7.7	30.1*	24.8
1997		110.6*	67.7*	34.3	56.0*	23.6	67.1*	12.8	32.9*	58.1
1998		36.9	36.4	32.3	40.4	11.7	15.6	12.6	20.7	30.3
1999		15.2	22.0	26.6	27.5	135.3*	5.2	1.0	20.1	23.1
2000		39.1	56.7*	66.3*	15.1	30.3	5.0	4.6	15.1	31.2
2001		133.0*	64.1*	26.9	44.9*	22.0	11.9	75.8*	18.0	47.8
2002		31.1	30.2	17.1	50.0*	7.8	12.1	18.7	18.0	28.3
2003		15.2	42.5	112.6*	18.8	27.8	13.2	2.5	12.8	26.1
2004		65.2	52.8*	38.3	27.2	7.8	107.8*	13.6	17.8	43.9
2005		96.8*	86.2*	96.6*	44.7*	6.8	25.3	42.9*	23.0	55.1
Total	Number	9,587	32,605	1,514	13,919	1,433	5,575	511	10,314	75,458
	Average rate	63.9	45.8	43.6	39.2	30.2	30.0	24.1	21.8	39.6

* Incidence rate ratio significantly higher (P<0.05) than the state average.

Figure 1. Notifications of pertussis, Australia, 1995 to 2005, by month of onset



100,000 population in 1997, with an average annual national notification rate over the analysed period of 39.6 cases per 100,000 population.

Sixty-two per cent of all pertussis notifications occurred in New South Wales (n=32,605) and Queensland (n=13,919) in the 10 year period, although these states account for 53% of the Australian population. The highest average annual notification rate was recorded in South Australia (63.9 cases per 100,000 population; Table 1). All states and territories have had peak incidence rates significantly higher than the average annual national notification rate at various times since 1995.

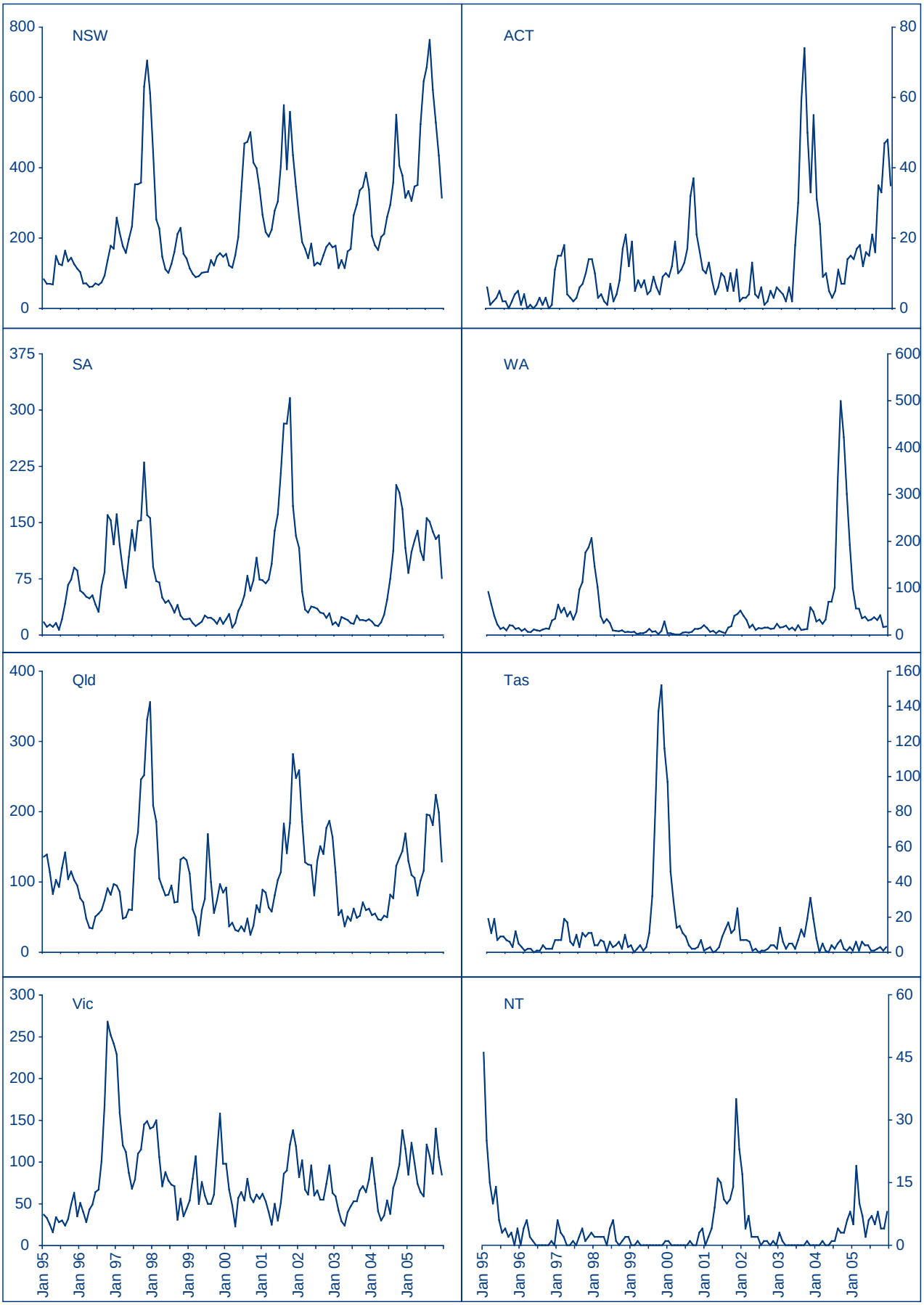
The epidemic cycles in New South Wales, Queensland and South Australia have followed a similar pattern, including double peaks during epidemics, however, the inter-epidemic period has varied. In the less populated and/or geographically isolated states and territories of Western Australia, the Northern Territory and Tasmania, epidemic cycles were spaced further apart (Figure 2).

Age distribution of cases

The age-specific pertussis incidence rates over the analysed period are shown in Figures 3a and 3b. Children less than 6 months of age had the highest annual notification rate in all of the analysed years. High rates were seen among children aged 5–9 years in the first years of the decade, with a peak notification rate in 1997 of 194 cases per 100,000 population. The notification rate in this age group has declined dramatically and remained at less than 50 cases per 100,000 population since 1999. High annual notification rates were recorded for children aged 10–19 years in 1997 and 2001 (114 and 128 cases per 100,000 population, respectively), corresponding to the national epidemic cycle. The notification rates for the 20–39, 40–59 and 60 years and over age groups have recently risen to record highs.

The majority of cases from year to year are now in the adult population (Figure 4). The proportion of adult cases has steadily increased, with 83% of pertussis notifications in persons aged over 20 years in 2005. Between 1995 and 2005 there was a significant downward trend in the proportion of cases occurring in the less than 1 year, 1–4 and 5–9 years age groups (P<0.001). During the same period there was a significant increasing trend in the proportion of cases occurring in the 10–19, 20–59 and 60 years and over age groups (P<0.001).

Figure 2. Pertussis notification patterns, Australia, 1995 to 2005, by state or territory



Note: Scales vary between jurisdictions.

Figure 3a. Age-specific incidence of pertussis for the age groups <6 month, 6–<12 months, 1–4, 5–9 and 10–19 years, Australia, 1995 to 2005, by age group

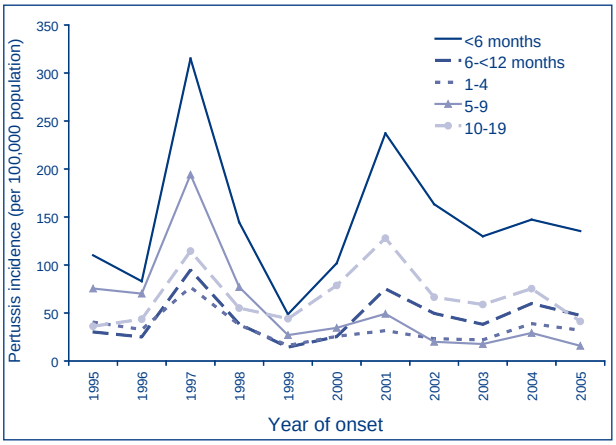
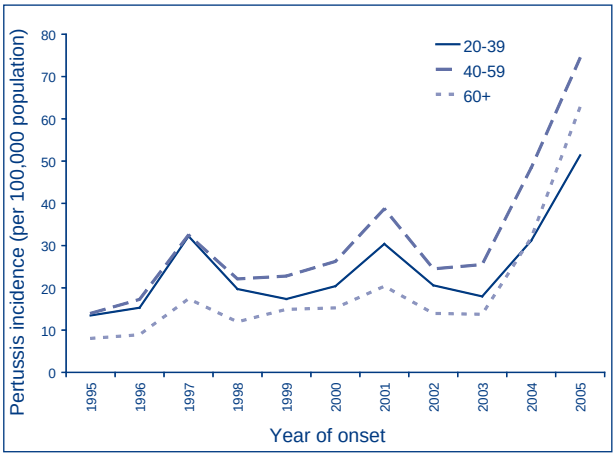
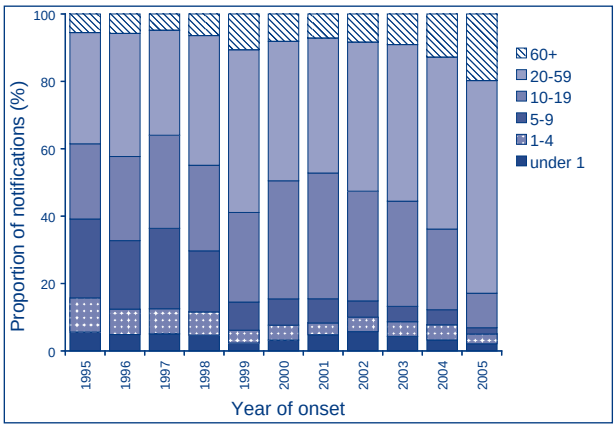


Figure 3b. Age-specific incidence of pertussis for the age groups 20–39, 40–59 and 60+ years, Australia, 1995 to 2005, by age group



As with the epidemic cycles of disease, the age distribution of cases varies among states and territories over time (Appendix 1 and Figure 5). In historical terms and with respect to other age groups, pertussis is relatively well controlled in the 1–4 and 5–9 year age groups; the exception to this being in Western Australia, where notification rates in these age groups were high in the 2004 epidemic year. High

Figure 4. Distribution of pertussis notifications, Australia, 1995 to 2005, by age group



annual notification rates in children under 1 year of age occur in all states and territories, and adolescents aged 10–19 years have also experienced high pertussis rates in all states and territories, relative to other age groups. In New South Wales and Western Australia, where whole of high school dTpa programs were conducted in 2004, the incidence in these age groups fell in 2005. During the period 1999–2003, combined pertussis incidence in New South Wales and Western Australia was 85.7 cases per 100,000 population. In 2005, this had decreased to 37.2 cases per 100,000 population. The increasing notification rate in the over 60 years age group observed nationally, is seen to varying degrees in all jurisdictions except the Northern Territory.

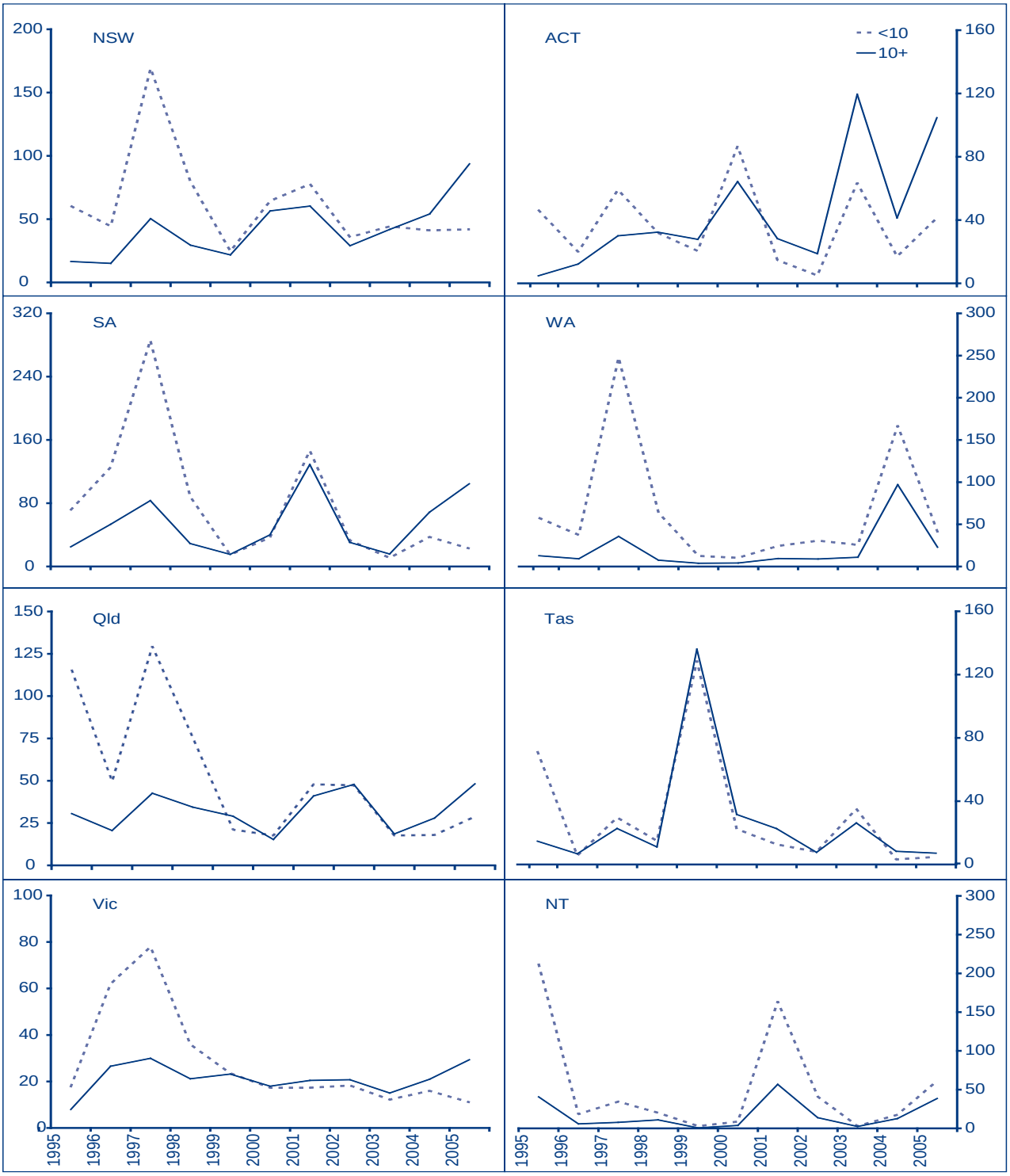
Method of diagnosis

Overall, serology was the predominant laboratory method for diagnosis of pertussis (74%), followed by nucleic acid testing (NAT; 12%). Diagnosis by serology increased from 41% of cases in 2000, to 65% of cases in 2005. NAT has been increasingly used as a method of diagnosis over time, and is the most common method for diagnosis in infants. The proportion of notifications diagnosed by NAT declines with increasing age, with serology the most prevalent diagnostic method over the age of 5 years. Culture is now used rarely, principally for diagnosis in infants less than 1 year of age (Table 2).

Table 2. Age-specific pertussis diagnostic methods for New South Wales, Queensland and the Northern Territory, 2000 to 2005, by age group

Diagnostic method	% by age group					
	<1	1–4	5–9	10–19	20–59	60+
Culture	9.6	3.1	1.9	1.3	0.9	0.9
Nucleic acid testing	59.7	39.1	21.3	11.1	7.1	4..3
Serology	8.7	26.4	52.5	73.8	81.3	88.0

Figure 5. Pertussis incidence for under 10-year-olds compared with over 10-year-olds, Australia, 1995 to 2005, by state or territory



Note: Scales vary between jurisdictions.

Discussion

This review highlights several aspects of the pattern of pertussis, as reflected in notification data, in Australia over the past decade; a time of significant changes in both diagnostic test availability and notification practice on the one hand and vaccination practice and vaccination coverage on the other.

Pertussis remains the most common vaccine preventable disease in Australia, with an average annual incidence rate of 39.6 cases per 100,000 population

and periodic epidemics. This review highlights for the first time the regional differences in epidemic patterns by magnitude and inter-epidemic period. South Australia (which only accounts for 8% of Australia's population), had a much higher average notification rate than any other state, 1.6 times the national average. The high notification rate in South Australia may in part be a result of greater case reporting, as the South Australian health department receives notifications from general practitioners as well as laboratories. In contrast, general practitioners in New South Wales are reluctant to notify pertussis without a laboratory confirmed diagnosis.[6] Differences among other states and territories with regard to use and availability of diagnostic tests, may have contributed to the variation in case ascertainment and notification rates. Unfortunately, there is insufficient detail on laboratory diagnosis methods in NNDSS to examine this in more detail but it is clear that serologic diagnosis is the primary method except in infants. While the national epidemiology of pertussis between 1995 and 2005 is similar to that of New South Wales, large outbreaks in other states or territories have influenced national trends at times, such as the 2004 peak in national notifications, which reflected an epidemic in Western Australia that year. Less populated and/or geographically isolated states and territories appear to be characterised by longer inter-epidemic periods and more intense epidemics than in other states. These notification patterns are synchronous with pertussis hospitalisations.[1,7]

The age-specific patterns of notification have reflected changes in vaccination practice, however the total number of notifications has not changed, with upward trends in older age groups not targeted for vaccination. Since 1999, notifications rates fell significantly among the 5–9 years age group reflecting the impact of the fifth dose of pertussis vaccine which was introduced nationally since 1994–1995.[8] High rates continue to occur in children aged less than 6 months who have received less than 3 vaccine doses, although there is a trend for less pronounced peaks to be seen over time, despite the increased availability of NAT as a diagnostic method for this age group. Both of these age distribution trends are consistent in all states and territories.

In 2005, there was a decrease in the national notification rate for the 10–19 year age group, which includes a highly susceptible cohort of adolescents in New South Wales, previously described,[9] and nationally.[8] New South Wales experienced a 43% decrease in the notification rate for the 10–19 year age group in 2005, following the implementation of a 'whole of high school' dTpa vaccination program in 2004.[10] As Australian school-based dTpa programs mature and successive cohorts are vaccinated in future years, pertussis in adolescents should be better controlled, as occurred in the 5–9 year age group following the introduction of the preschool booster.

The increasing adult burden of pertussis may be in part due to increasing awareness among clinicians, as well as waning vaccine induced immunity. The rise in adult cases raises several new issues and challenges both for diagnosis and for potential immunisation strategies. With respect to diagnosis, the majority of adult cases are diagnosed by serology, as older individuals often only present to a doctor after several weeks of coughing illness, when the probability of isolating *B. pertussis* is greatly reduced.[11] In Australia, serological diagnosis based on the detection of IgA antibodies to whole cell *B. pertussis* antigens has been available in some jurisdictions since the 1980s and nationally since 1993. Although insensitive, previous work suggested a specificity of 93%–98% in the presence of appropriate clinical symptoms.[12] However, specificity is likely to be considerably reduced by a change in diagnostic practice with a wider population of adults being tested, or by immunisation. The adult acellular pertussis vaccine trial (APERT) in the United States of America showed that IgA antibody responses to pertussis toxin (PT) after immunisation were predicted to remain above the threshold of detection for a minimum of 3.6 years.[13] This is in contrast to young children who do not produce an IgA antibody response after immunisation.[14] Within one year of vaccination, differentiating between antibody response due to immunisation and infection is particularly difficult and requires the use of a range of serologic criteria usually not available in a routine public health diagnostic setting.[15] It is unclear whether the response following adult immunisation with acellular pertussis vaccine observed with IgA-PT antibody assays, would be similar when using the current Australian IgA whole cell diagnostic assay. This requires urgent investigation, given the increasing reliance on serology for diagnosis of pertussis in Australia. It also stresses the need for serologically positive results to be notified only if the case has appropriate clinical symptoms, which can be challenging in the public health setting as it requires the active follow-up of all positive pertussis serology results.

The recent increase in the pertussis notification rate in the elderly warrants further investigation. Although less important than parents, grandparents can be a significant source of pertussis transmission to infants.[16] The level of morbidity from pertussis among the elderly is poorly defined and vaccines are currently not approved for use in persons aged over 65 years. However, 2 of 8 recorded pertussis deaths in the past 5 years have been in people aged 60 years or more.[1,17]

Although severe morbidity and mortality are less likely in adults, substantial morbidity still occurs[18,19,20] and increased circulation of pertussis can facilitate transmission to susceptible infants who are too young to be vaccinated.[16,21,22] Current adult pertussis immunisation recommendations in Australia aim at 'cocooning' infants by recommending immunisation in adults who are most likely to come into contact with them.[23] A universal adult immunisation strategy would be difficult to implement, as it is challenging to reach adults for immunisation.[24,25] The only current universal adult immunisation recommendations in Australia are a diphtheria-tetanus booster at 50 years of age and influenza (annually) and pneumococcal (5-yearly) vaccines from 65 years of age.[23]

The control of pertussis in all Australian states and territories remains a challenge despite a long history of immunisation and repeated modifications to the schedule. Immunisation has led to a change in the age distribution of cases, bringing about new issues with regard to diagnostic practices and future recommendations for immunisation. With the burden of disease now largely in adults, the current focus needs to be in protecting infants too young to be vaccinated and further defining the true morbidity of the disease in the elderly population.

Acknowledgements

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Appendix

Appendix 1. Age-specific pertussis notification rates, Australia 1995 to 2005, by state or territory

State or territory	Age group					
	<1	1–4	5–9	10–19	20–59	60+
ACT						
1995	135.0	16.4	53.1	2.1	5.6	3.5
1996	44.8	16.4	17.7	30.8	7.3	10.6
1997	70.8	46.2	66.8	47.3	28.0	17.7
1998	24.3	23.1	40.1	68.8	24.8	23.6
1999	71.5	5.8	22.3	49.5	22.6	26.6
2000	72.6	57.8	111.4	174.2	44.2	23.6
2001	74.3	12.5	4.8	58.9	24.2	14.0
2002	25.2	0.0	4.8	37.1	18.0	2.3
2003	124.6	62.4	52.4	421.2	70.1	21.0
2004	71.4	12.5	9.5	34.9	42.8	41.9
2005	23.8	37.4	47.7	69.8	105.1	139.8
Average	67.4	26.4	39.6	89.6	36.3	31.7
NSW						
1995	91.6	34.8	74.8	34.2	14.3	8.5
1996	80.6	29.1	49.5	25.3	14.4	8.4
1997	234.1	90.2	218.6	126.6	41.3	19.6
1998	139.9	43.4	96.7	59.7	26.3	15.6
1999	53.6	17.9	24.3	29.4	20.8	18.1
2000	134.3	46.2	65.2	144.3	43.0	26.1
2001	216.6	47.1	73.8	175.4	44.4	22.9
2002	115.3	30.9	24.4	67.4	24.8	12.8
2003	111.8	40.6	34.2	96.4	35.7	20.1
2004	99.6	46.2	25.8	72.9	53.2	42.6
2005	129.7	44.5	22.9	41.8	105.3	97.2
Average	128.1	42.8	64.8	79.0	39.2	27.5

Appendix 1. Age-specific pertussis notification rates, Australia 1995 to 2005, by state or territory, *continued*

State or territory	Age group					
	<1	1–4	5–9	10–19	20–59	60+
NT						
1995	384.2	161.9	215.4	46.7	42.2	0.0
1996	54.1	7.4	19.0	10.8	4.9	0.0
1997	56.5	35.2	29.3	16.7	5.2	9.7
1998	83.6	0.0	23.4	6.7	13.1	0.0
1999	28.3	0.0	0.0	3.3	0.0	0.0
2000	28.0	7.0	5.9	6.7	3.5	0.0
2001	762.9	71.9	108.2	171.2	33.4	6.7
2002	137.1	28.7	30.0	35.5	8.4	13.3
2003	26.9	0.0	0.0	0.0	3.3	0.0
2004	27.1	14.4	18.0	32.3	8.4	6.7
2005	0.0	8.2	3.1	2.9	6.2	12.9
Average	167.5	35.2	41.5	33.9	14.3	9.8
Qld						
1995	56.7	76.3	158.9	65.8	24.9	15.6
1996	25.0	25.4	74.1	47.9	16.5	7.6
1997	109.5	49.8	195.1	142.8	25.4	10.5
1998	67.8	42.2	102.9	88.2	26.2	13.4
1999	35.9	11.7	25.7	47.1	26.3	22.2
2000	31.3	10.7	20.6	35.1	11.3	10.9
2001	133.7	28.8	46.3	123.1	27.8	15.0
2002	186.5	33.8	32.5	128.9	34.5	22.8
2003	71.6	14.4	10.5	39.7	15.7	10.1
2004	63.3	14.4	12.3	48.6	24.8	20.6
2005	57.3	26.6	13.5	68.0	112.7	106.5
Average	81.4	30.1	61.2	76.3	25.4	18.8
SA						
1995	118.4	37.7	88.4	69.3	18.1	10.8
1996	109.8	55.3	182.7	140.0	42.4	19.7
1997	414.0	117.7	392.2	247.2	60.9	30.5
1998	152.7	45.3	110.0	84.6	20.4	14.5
1999	21.8	15.5	13.0	25.0	13.6	13.8
2000	66.7	28.4	37.0	101.1	31.8	20.0
2001	481.8	77.1	136.2	353.7	100.9	55.4
2002	159.6	18.2	20.8	51.4	28.9	21.1
2003	52.1	5.6	7.3	10.3	16.8	16.5
2004	97.5	35.0	28.1	83.2	66.8	63.6
2005	162.9	42.2	16.5	21.3	23.7	20.9
Average	157.6	42.4	94.6	112.2	47.0	34.9

Appendix 1. Age-specific pertussis notification rates, Australia 1995 to 2005, by state or territory, *continued*

State or territory	Age group					
	<1	1–4	5–9	10–19	20–59	60+
Tas						
1995	87.8	60.0	77.2	26.9	12.7	10.4
1996	15.0	0.0	8.3	9.9	5.6	7.8
1997	96.0	22.6	23.1	42.0	20.1	14.7
1998	17.1	3.8	23.1	7.2	12.6	9.8
1999	46.8	48.9	205.2	380.7	96.8	51.3
2000	50.8	18.8	20.2	57.9	29.9	14.7
2001	64.7	4.1	9.2	46.6	19.4	14.0
2002	68.5	0.0	3.1	10.2	6.6	8.6
2003	225.3	12.3	18.5	46.6	24.1	17.2
2004	34.5	0.0	0.0	4.4	7.0	15.0
2005	243.8	64.7	18.0	38.8	36.8	53.3
Average	63.9	16.8	36.4	57.7	21.9	15.9
Vic						
1995	69.4	8.9	19.1	13.5	5.6	4.2
1996	142.2	28.7	91.7	50.6	18.5	9.9
1997	117.6	49.5	92.5	60.0	20.2	14.2
1998	44.7	28.5	40.0	38.8	15.7	8.3
1999	35.2	17.8	25.8	47.1	16.5	7.6
2000	51.3	13.5	14.0	39.6	11.2	8.9
2001	70.0	7.8	14.7	39.7	14.1	10.4
2002	103.0	9.0	9.4	41.9	14.4	9.2
2003	68.1	6.6	5.9	27.5	10.6	8.1
2004	67.5	15.6	6.2	31.9	15.7	13.5
2005	112.4	26.8	14.9	67.2	42.6	50.7
Average	71.1	18.6	29.5	37.3	15.3	11.2
WA						
1995	135.2	48.1	50.0	27.0	11.5	3.0
1996	98.9	20.6	38.5	23.0	6.6	4.8
1997	513.1	145.9	276.5	93.8	26.0	15.0
1998	210.6	41.8	53.8	13.5	7.3	1.9
1999	23.8	9.7	12.7	8.6	3.2	1.9
2000	47.9	7.8	5.2	12.0	2.9	1.5
2001	112.9	21.1	10.5	28.3	6.4	3.5
2002	146.7	27.1	12.7	22.7	7.2	2.8
2003	113.8	24.1	11.2	30.1	8.1	4.4
2004	401.2	140.5	142.2	259.1	72.0	39.2
2005	12.9	16.8	6.2	21.4	25.2	26.3
Average	178.8	48.1	57.4	50.0	16.3	9.6

Epidemiology of *Leptospira weilii* serovar Topaz infections in Australia

Andrew T Slack, Meegan L Symonds, Michael F Dohnt, Bruce G Corney, Lee D Smythe

Abstract

Leptospirosis is a zoonotic disease with a worldwide distribution. *Leptospira weilii* serovar (sv.) Topaz is a newly described serovar first isolated in the far north of Queensland, Australia. The epidemiology of *L. weilii* sv. Topaz infections in Australia was characterised through the use of surveillance questionnaires and molecular studies. There have been 24 human and 2 animal (bovine and bandicoot) *L. weilii* sv. Topaz infections diagnosed since 1991. The majority of these infections have occurred in Far North Queensland, with the remaining infections occurring in South East Queensland and in Western Australia. The majority of patients with *L. weilii* sv. Topaz infections presented with classical leptospirosis symptoms including; fever, headaches, sweats, chills and myalgia. The occupations of human cases of *L. weilii* sv. Topaz infection included banana farming, dairy and beef cattle production and tourist related activities. Fluorescent amplified fragment length polymorphism (FAFLP) was performed on 15 *L. weilii* sv. Topaz isolates including 2 animal isolates. Clustering analysis grouped the 15 isolates into 5 main clades with 13 unique FAFLP profiles. A high level of relatedness was demonstrated between 2 animal and 2 human isolates. *Commun Dis Intell* 2007;31:216–222.

Keywords: epidemiology, *Leptospira weilii*, leptospirois, zoonoses

Introduction

Leptospirosis is caused by infection with spirochaetes of the genus *Leptospira*.[1] *Leptospira* are motile helical spirochaetes that metabolise long chain fatty acids as their sole carbon source.[1] The genus *Leptospira* contains 17 species as delineated by DNA-DNA hybridisation.[2–5] *Leptospira* are divided serologically into serovars of which there have been over 200 described. Leptospirosis is one of the world's most widespread zoonotic diseases with outbreaks reported worldwide in both humans and animals.[6,7,8] The organism enters humans through contact of abrasions or mucus membranes with urine or body fluids from an infected animal. This may occur directly or indirectly through contact with contaminated water or soil. Leptospirosis is more prevalent in tropical countries than temperate countries as the higher humidity, rainfall and temperature promote the survival of the organism in the environment.[9]

Leptospirosis was first reported in Australia in 1933. Since 1991, leptospirosis infections have been notified to the National Notifiable Diseases Surveillance System. From 1991 to 2005, there have been 7,629 cases notified in Australia.[10] The majority of cases have occurred in Queensland situated on the eastern seaboard of Australia.[11] Twenty-three *Leptospira* serovars have been identified in Australia including several which were first discovered in Australia.[11] *L. weilii* sv. Topaz was first isolated by Corney, et al. from a bovine source near the township of Topaz in Far North Queensland and is the second member of the *L. weilii* species found in Australia.[12] It was found to be a unique serovar by cross agglutinin absorption test (CAAT). The aim of this study is to present the descriptive and molecular epidemiology of *L. weilii* sv. Topaz in Australia.

Materials and methods

Serological identification of *L. weilii* sv. Topaz

Leptospira IgM-specific enzyme linked immunosorbent assay (ELISA, Panbio) was performed as a screening test by the submitting private and hospital laboratories. Positive ELISA samples were forwarded to the WHO/FAO/OIE Collaborating Centre for Reference and Research on Leptospirosis, Brisbane (WHO Reference Centre) for confirmation and serovar identification using the microscopic agglutination test (MAT). *L. weilii* sv. Topaz strain 94-79979/3 was used as the reference culture in the MAT for the detection of human antibodies against *L. weilii* sv. Topaz. Patients with a positive IgM ELISA supported by a single MAT titre of greater than or equal to 400, or a demonstrated fourfold rise or fall in MAT titres over paired specimens, were considered to meet the notification criteria for leptospirosis.

Culture identification

Leptospira culture from humans was performed as follows: 2–5 drops of uncoagulated whole blood was inoculated into Ellinghausen McCullough Johnson Harris (EMJH) media containing 0.5% agar. This was performed by the submitting laboratories and forwarded at room temperature to the WHO Reference Centre. Once at the WHO Reference Centre, the cultures were sub-cultured into EMJH media (containing no agarose) and incubated at 30°C for 6 weeks. The cultures would be inspected weekly for the growth of *Leptospira* using dark field

microscopy. Positive cultures were identified to a species level by sequencing of a partial fragment of the DNA gyrase sub-unit B gene (*gyrB*).[13] Serological identification of the isolates was performed by MAT using reference antisera covering the major serogroups of the genus *Leptospira*. Hyper-immune antiserum was prepared in rabbits using standard techniques.[14] Definitive serovar identification was performed by CAAT[15] using the *L. weilii* sv. Topaz strain 94-79979/3.

Surveillance questionnaires and descriptive epidemiology

Information regarding infection was collected from the patient's doctor using enhanced surveillance questionnaires. This provided information on symptoms, recreational activities, animal contacts, occupational data and hospitalisation. The data were entered into a Microsoft Access database and Microsoft Excel was used to conduct statistical analysis of these data.

Fluorescent amplified fragment length polymorphism

Cultures were prepared for DNA extraction by centrifugation of 1 mL of an actively growing *Leptospira* culture in EMJH at 12,500 g for 5 minutes. Genomic DNA was then extracted from the pellet using the ChargeSwitch gDNA mini bacterial kit (Invitrogen) as per manufacturers' instruction. FAFLP restriction digestion, ligation and amplification reactions was performed as previously described.[16,17]

Fragment sizing and data analysis

One μL of the 6 selective polymerase chain reaction products was mixed with 18.5 μL of HiDi formamide (Applied Biosystems) and 0.5 μL of Geneflo-625 (Chimerx) and denatured at 95°C for 5 minutes. The products were then loaded onto the ABI-310 capillary sequencer (Applied Biosystems) and were injected into a 47 cm capillary filled with performance-optimised polymer 4 (Applied Biosystems) at 15 kV for 12 seconds. The fragments were separated at 1 3kV for 35 minutes. The resulting electropherograms were manipulated using the Genotyper v2.5 software (Applied Biosystems) and the combined allele sizes were exported into an Excel spreadsheet. A previously described Excel macro[18] was used to convert the alleles into a binary sequence suitable for analysis using Bionumerics software (Applied maths). Each unique FAFLP binary pattern was assigned a letter code (e.g. A, B, C) to allow for easier referencing of the data.

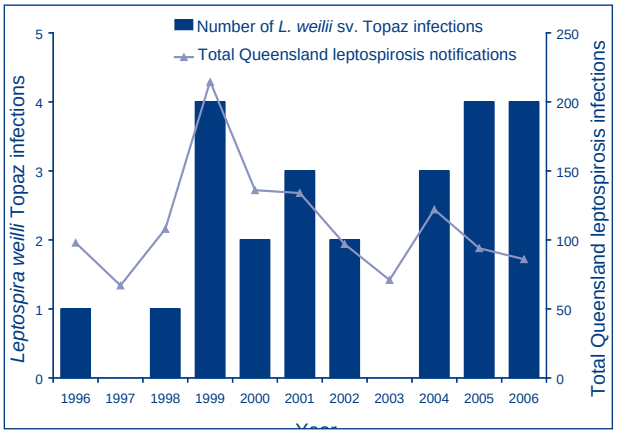
Results

Descriptive epidemiology of *Leptospira weilii* sv. Topaz

Since the initial isolation of *L. weilii* sv. Topaz in 1994,[12] there have been 26 additional cases of this serovar identified: 24 from human and 2 from animal sources (bovine and bandicoot). The first isolation of *L. weilii* sv. Topaz predates the identification of the type strain; 94-79970/3 having been isolated in 1991 and was identified at the time as a member of the Tarrasovi serogroup. Subsequently, examination of the isolate by CAAT using *L. weilii* sv. Topaz strain 94-7997/03, identified the isolate as serovar Topaz. Fifteen cases were diagnosed from *Leptospira* cultures taken from blood (human), urine (bovine) or from kidney tissue (bandicoot). The remaining 11 cases were diagnosed by MAT with titres ranging from 1 in 800 to 1 in 6,400 (Table 1).

Since 1994, *L. weilii* sv. Topaz infections in humans have been reported every year except in 1997 and 2003. Four cases were reported in 1999, 2005 and 2006 (up to August) (Figure 1). The majority of isolations occurred between January and June, with a single case reported in October, this is consistent with the seasonal trend of *Leptospira* infections noted by Slack, et al.[11]

Figure 1. Distribution of *Leptospira weilii* sv. Topaz cases from 1996 to August 2006 compared with the total Queensland leptospirosis notifications for the same period



The geographical distribution of *L. weilii* sv. Topaz in Australia has been dominantly in the far north of Queensland around the leptospirosis endemic areas extending south from Cairns to Innisfail and Tully, and west onto the eastern side of the Atherton

Table 1. *Leptospira weilii* serovar Topaz cases in Australia, 1991 to 2006

Year	Source	Laboratory details	Age	Sex	Serum MAT titre (against strain 94-79970/3)	Culture
1991	Human	LT 596*	Unknown	F	Not performed	Detected
1994	Bovine	94-79970/3* (type strain)	N/A	N/A	N/A	Detected
1996	Human	LT 762*	30	M	1600	Detected
1998	Human	LT 925*	34	M	Not performed	Detected
1999	Human	LT 952*	22	M	800	Detected
1999	Human	LT 969*	33	M	Not performed	Detected
1999	Human	LT 974*	21	M	Not performed	Detected
1999	Human	LT 981*	39	M	400	Detected
2000	Human	LT 1060*	58	M	1600	Detected
2000	Human	LT 1055*	37	M	Not performed	Detected
2001	Human	LT 1187*	14	M	Not performed	Detected
2001	Human	LT 1188*	33	M	800	Detected
2001	Human	LT 1191*	28	M	1600	Detected
2002	Bandicoot	LT 1412*	N/A	N/A	N/A	Detected
2002	Human	LT 1414*	48	M	Not performed	Detected
2004	Human	TSI 1†	62	M	800	Not performed
2004	Human	TSI 2†	23	M	800	Not performed
2004	Human	TSI 3†	40	M	800	Not performed
2005	Human	TSI 4†	28	M	3200	Not performed
2005	Human	TSI 5†	64	M	1,600	Not performed
2005	Human	TSI 6†	54	M	800	Not performed
2005	Human	TSI 7†	47	M	800	Not performed
2006	Human	TSI 8†	56	F	800	Not performed
2006	Human	TSI 9†	22	M	6400	Not performed
2006	Human	TSI 10†	24	M	800	Not performed
2006	Human	TSI 11†	21	M	1,600	Not performed

* Diagnosis of *Leptospira weilli* sv. Topaz made by identification of the *Leptospira* isolate from the blood culture

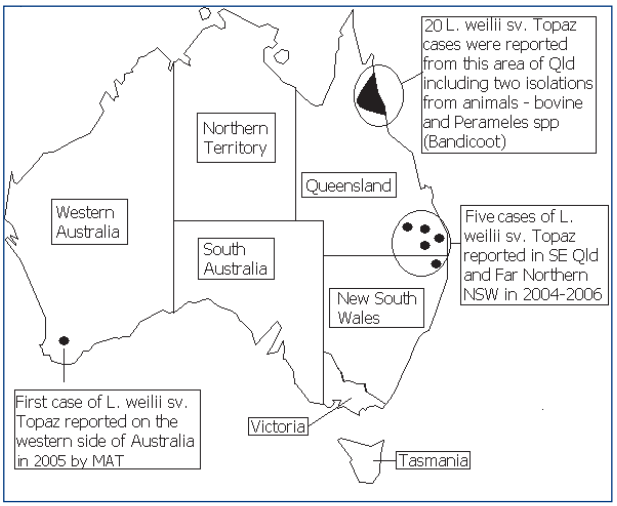
† Diagnosis of *Leptospira weilli* sv. Topaz made by serology using the microscopic agglutination test (MAT).

N/A Not applicable.

Tablelands (Figure 2). Twenty cases (77%) of *L. weilii* sv. Topaz have originated from this area; an area that has accounted for approximately 66% of Queensland leptospirosis notifications from 1998 to 2004 (Figure 2).[11] Five cases of *L. weilii* sv. Topaz were detected in South East Queensland and 1 case in northern New South Wales. The first detection of *L. weilii* sv. Topaz away from the eastern seaboard of Australia was a case in 2005 from Western Australia in which the patient had no history of recent travel to the eastern seaboard.

Of the 24 *L. weilii* sv. Topaz cases in humans, there was a male:female ratio of 11:1, which is consistent with the sex distribution of leptospirosis in previous studies.[11,20] The age of the patients ranged from 14 years to 64 years with a median age of 33 years. Twelve (50%) cases were reported to have been hospitalised with an average stay of 3 days. Patients with *L. weilii* sv. Topaz infections presented with classical symptoms of leptospirosis including; fever, headaches, sweats, chills and myalgia. Two patients presented with the more severe leptospirosis complications of pulmonary haemorrhage and aseptic meningitis (Table 2). The occupations of cases of *L. weilii* sv. Topaz infection included banana farming, dairy and beef cattle production and tourist related activities (Table 2). Contact with animals before infection was reported in the majority of cases, which is consistent with the rural occupa-

Figure 2. Geographical distribution of *Leptospira weilli* sv. Topaz cases, Australia, 1991 to 2006



tions and/or recreational exposure generally associated with *Leptospira* infections. The major animal contacts reported include rats or mice (this included native rodents such *Rattus fuscipes* or *R. sordidus* as well as the imported *R. rattus* and *Mus domesticus*), dogs, beef and dairy cattle (Table 2).

Molecular epidemiology of *Leptospria weilii* sv. Topaz

FALFP was performed on all 15 *L. weilii* sv. Topaz isolates including the 2 animal isolates (94-79970/3 and LT1412). There were 13 unique FAFLP profiles (designated A to M) amongst the 15 isolates tested. Clustering analysis of the FAFLP data was performed using the Jaccard coefficient (>50% mean) and unweighted pair group method with arithmetic mean (UPGMA) algorithm (Figure 3). There were 5 identifiable clades (designated i to v) within the dataset each containing between 2 and 4 isolates. Each clade contained isolates found over multiple years and showed clustering around the geographic area of isolation. For example clade ii and clade v contained isolates from the Cairns or Atherton tableland section of Far North Queensland whilst clade i and iv contained isolates from the more southern areas of Tully and Innisfail. An isolate from a native bandicoot species (LT1412) showed a high level of similarity to the human isolate; LT952. The type strain 94-7997/03 isolated from cattle, was found to share an identical FAFLP pattern with the human isolate; LT974.

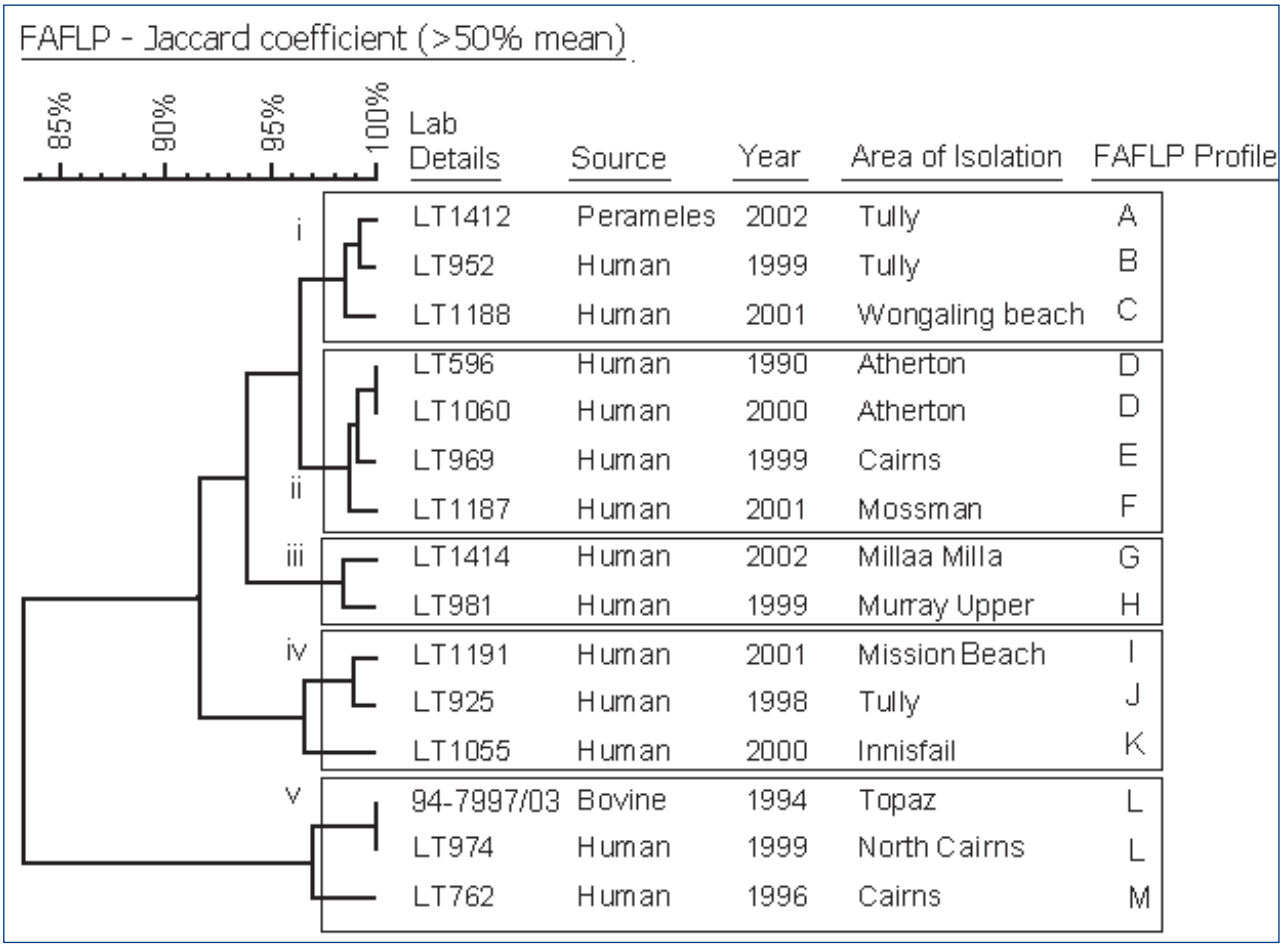
Table 2. Clinical and epidemiological characteristics of *Leptospira weilli sv.* Topaz infections

Clinical characteristics	Positive responses n=18	Percentage of positive responses
Fever	14	78
Headache	12	67
Arthralgia	10	56
Sweats	10	56
Chills	9	50
Myalgia	9	50
Nausea	7	39
Vomiting	6	33
Back pain	4	22
Respiratory symptoms	2	11
Asceptic meningitis	1	6
Conjunctival suffusion	1	6
Diarrhoea	1	6
Liver involvement	1	6
Pulmonary haemorrhage	1	6
Rash	1	6
Vision disturbance	1	6
Occupation	**Positive responses n=24**	**Percentage of positive response**
Banana farmer	4	17
Tourist operator/tourist	4	17
Dairy	3	13
Grazier	3	13
Labourer/tradesperson	2	8
Veterinarian	1	4
Not reported or unemployed	7	29
Animal	**Positive responses n=20**	**Percentage of positive responses**
Rats and mice	9	45
Dogs	8	40
Beef cattle	5	25
Dairy cattle	3	15
Feral pigs	3	15
Cats	2	10
Domestic pigs	1	5
Bandicoot	1	5
Sheep	1	5
No contact	1	5

Discussion

L. weilii sv. Topaz has been identified in both animal and human sources since its initial isolation in 1994 using both culture and serological methods (IgM ELISA combined with MAT) (Table 1). By utilising both methods initially, we were able to validate the specificity of the MAT in the diagnosis of *L. weilii* sv. Topaz infections. Diagnosis by MAT alone has been possible since the inclusion of *L. weilii* sv. Topaz in

Figure 3. Clustering analysis of *Leptospira weilii* sv. Topaz constructed from the FAFLP data using the Bionumerics software package



the WHO Reference Centre MAT panel since 2000. Initially, this testing was conducted by screening samples that had low *L. borgpetersenii* sv. Tarassovi titres (*L. weilii* sv. Topaz produces a serological cross-reaction with members of the Tarassovi serogroup, data not shown) for the presence of *L. weilii* sv. Topaz antibodies and in 2004 was added to the panel as a standalone serovar. Whilst the MAT has been criticised for a lack of specificity when determining the infecting *Leptospira* serovar,[19] we believe that its use in a well-defined leptospirosis environment such as Australia means that it can be sufficiently relied upon to determine the individual serovar involved.

The geographical distribution of *L. weilii* sv. Topaz in Australia suggests that there are 2 distinct pockets of this serovar in Queensland and northern New South Wales. However, the single Western Australia case of *L. weilii* sv. Topaz may indicate that this serovar may be much more widespread in Australia compared to the more geographical isolated *Leptospira* serovars such as *L. interrogans* sv. Zanoni.[11]

Since *L. weilii* sv. Topaz infection has been isolated from a native animal (bandicoot) this serovar may be indigenous to Australia.[11] Several *Leptospira* serovars in Australia are not found elsewhere in the world.[11] A recent unpublished study examining leptospirosis in Macropods (kangaroos), found that a significant proportion of the study animals had serological titres that would indicate exposure to *L. weilii* sv. Topaz. (personal communication, M Roberts and L Smythe).[11]

Overall, *L. weilii* sv. Topaz infections represent only 1.9% of the 1,288 reported leptospirosis infections (based upon Queensland data from 1996 to August 2006), however this figure is likely to be an under-estimate since assays for its identification have only recently (since 2000) been available at the WHO Reference Centre. To assess the geographic distribution of the serovar in Australia, it is recommended that all Australian laboratories performing leptospirosis testing include *L. weilii* sv. Topaz in their MAT panels or alternatively, that the samples with serovar Tarrasovi titres are forwarded to the WHO Reference Centre for confirmatory testing.

The epidemiology of *L. weilii* sv. Topaz infections is similar to that of other leptospirosis infections in Australia.[11] There is a higher likelihood of *Leptospira* infection in the period from January to May, as this is the peak rainfall period for Far North Queensland. The high rainfall combined with relatively high ambient temperatures provides ideal survival conditions for *Leptospira* in the environment, translating to a higher risk to humans.

Males of working age (18 to 60) are the most at risk. The median age of 33 years at infection with *L. weilli* sv. Topaz is consistent with other studies of leptospirosis in Australia.[11] The majority of *L.* weilli sv. Topaz patients report symptoms classically associated with leptospirosis. Banana farming and dairy/beef cattle farming appear to be the most at risk occupation groups. Both these occupations require contact directly and indirectly with animals and at times require close contact with contaminated soil and water. The other major occupation at risk is the tourist operator/tourist sector, accounting for 4 (17%) of the total number of *L. weilii* sv. Topaz infections (Table 2). A recently study by Slack, et al demonstrated that 17.8% (n=883) of *Leptospira* infections in Queensland were from recreational exposure.[11]

The isolation of *L. weilii* sv. Topaz from a bovine source may require further risk assessments to determine the risk posed to the domestic animal industries or workers in these industries in Australia. Additionally, further studies are required to determine what are the carriage rates and the level of disease caused by this serovar in Australian animal herds and wildlife.

FAFLP has been successfully used to examine the molecular epidemiology of *Leptospira* isolates.[16,17] Using FAFLP we were able to demonstrate a high level of relatedness amongst 2 animal and 2 human isolates (LT1414 and LT952, LT722 and LT974). These molecular tools allow links to be made between the carriage of this serovar in animals and human disease.

In conclusion, we have described both the descriptive and molecular epidemiology of *L. weilii* sv. Topaz in Australia. This research provides evidence for the presence of this serovar in native, domesticated animals and humans, however the role and burden of this serovar in animal health and disease needs to be further defined by additional research. Further research is needed to establish the prevalence of this serovar in Australian native fauna and animal industries.

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Q fever cases in the Northern Territory of Australia from 1991 to 2006

Anna Ralph, Peter Markey, Rosalie Schultz

Abstract

Q fever (infection with *Coxiella burnetii*) has been uncommon in Australia's Northern Territory, with no reported cases until 2002. Since then, twelve cases of Q fever have been reported, representing a much lower notification rate than in surrounding Australian states. Three cases were identified in Central Australia during 2006, prompting this review of clinical and epidemiological features of all notified Northern Territory cases. Three patients required Intensive Care admission, 1 died, 5 had moderately severe illness, 2 were treated as outpatients and 2 were excluded as unlikely Q fever cases on clinical grounds. Hospital stays were long (median length of stay 9.5 days), and diagnosis and definitive therapy were generally delayed. Although macrolides and quinolones have some reported efficacy against *C. burnetii,* 2 patients experienced prolonged fever (5 and 9 days respectively) despite azithromycin, and the fatality occurred in a patient treated with multiple antibiotics including ciprofloxacin. Four patients were Aboriginal, 3 were tested for HTLV-I and 2 were positive. The patient who died was diabetic. None had valvular heart disease. Greater awareness of acute and chronic manifestations of Q fever is required in the Northern Territory. Early institution of doxycyline in suspected cases is recommended, and more rapid diagnostic methods including polymerase chain reaction testing should be considered. Host risk factors for chronicity, which may be of particular importance in Indigenous patients, merit attention. Given the lack of occupational exposure in these cases, there seems little reason to change the current Northern Territory policy of opting out of the National Q Fever Vaccination Program. Recognised alternative exposures, such as non-occupational livestock and domestic animal contact, require consideration as local Q fever sources. *Commun Dis Intell* 2007;31:222–227.

Keywords: Disease surveillance, *Coxiella burnetii*, Northern Territory, Q fever

Introduction

Documented cases of infection with *Coxiella burnetii* (Q fever), a notifiable zoonotic disease, have been uncommon in the Northern Territory. *C. burnetii* is found in every country except New Zealand,[1] and in multiple animal hosts including wild and

domestic mammals, birds and ticks. Human cases are predominantly due to occupational exposure in livestock industries, but sporadic cases after minimal contact are increasingly recognised.[2] Under-diagnosis is common, despite exhortations to physicians to consider, investigate and treat Q fever where appropriate.

Acute Q fever ranges from asymptomatic to fulminant. A detailed review of Q fever in an Australian case series is provided by Spelman.[3] Asymptomatic infections (identified serologically or from skin testing) are estimated to represent the majority (60%) of cases; chronic manifestations (including endocarditis and osteoarticular infections) comprise around 0.2% of cases.[2,4] Up to 40% of patients with acute Q fever may develop endocarditis if they have pre-existing valvular disease.[5,6]

Q fever is notifiable in Australia if there is definitive laboratory evidence, or suggestive laboratory evidence with compatible clinical illness (Box). Australian national notification rates have fallen from 4.99 cases per 100,000 population in 1993[7] to 2.2 cases per 100,000 in 2004, probably due to the National Q Fever Management Program established in 2001,[8] with the majority of cases occurring in Queensland and New South Wales. The first notified Northern Territory case since commencement of electronic record keeping in 1991 was reported in 2002.[9] The report made note of several cases occurring over previous decades recalled by local physicians. It has been assumed that Q fever is uncommon in the Northern Territory, despite a moderately sized and growing pastoral industry with traffic of people and stock across territory/state borders.[10] Because of the lack of industry-associated cases, the Northern Territory has not been part of the National Q Fever Management Program.

When 3 cases of acute Q fever were identified in 2006 in Central Australia, a re-evaluation of the burden of Q fever throughout the Northern Territory was undertaken. We present the results of a retrospective review of epidemiological and clinical features of all notified Q fever cases in the Northern Territory and discuss host risk factors, treatment regimens and strategies for ensuring timely diagnosis.

Q fever case definition

A confirmed Q fever case requires:

EITHER

1. *Laboratory definitive evidence*

OR

2. *Laboratory suggestive evidence AND clinical evidence.*

Laboratory definitive evidence

1. *Detection of C. burnetii by nucleic acid testing*

OR

2. *Seroconversion or significant increase in antibody level to Phase II antigen in paired sera tested in parallel in absence of recent Q fever vaccination*

OR

3. *Detection of C. burnetii by culture (note this practice should be strongly discouraged except where appropriate facilities and training exist.)*

Laboratory suggestive evidence

Detection of specific IgM in the absence of recent Q fever vaccination.

Clinical evidence

A clinically compatible disease.

Methods

We identified all notified cases of Q fever from 1991 (commencement of electronic record keeping) to 2006, using the Northern Territory Centre for Disease Control (CDC) notifiable diseases database. Clinical, laboratory and radiological data were obtained from hospital charts, general practitioner, and CDC records. Information was recorded for each patient as follows:

1. indigenous status;
2. likely Q fever exposure;
3. illness severity graded as fatal, severe (requiring admission to Intensive Care), moderately-severe (requiring hospitalisation), moderate (outpatient, significant symptoms), mild (outpatient, mild symptoms) and asymptomatic;
4. clinical features (presence or absence of documented fever, fever duration, other clinical features noted in the medical file);
5. laboratory results including *C. burnetii* serology, liver function tests, platelet count and human T-lymphotrophic virus type 1 (HTLV-I) status;
6. chest radiograph; and
7. antibiotics administered.

Results

Twelve cases of Q fever were notified to the Northern Territory CDC between 1991 and 2006; 8 in Central Australia and 4 in the Top End. A further case diagnosed in late 2006 in the Top End was not included here. No cases were reported in the Top End prior to 2002, or in Central Australia prior to 2004 (Figure 1). After discussion with treating doctors and review of clinical notes, we excluded one Top End case and

one Central Australian case. The Top End patient remained in Intensive Care at the time of report with a protracted illness with auto-antibodies and multiple cross reactive serological results, but no Q fever-compatible illness. The Central Australian patient had atypical pneumonia without prolonged fever; Q fever Phase 1 and 2 antibodies were all elevated at low titre, and serology was also positive for *Bordetella pertussis* and *Mycoplasma pneumoniae*, the latter thought to be the more likely illness.

Of the remaining 10 cases, most were middle-aged males (Table 1). A clear exposure history was only evident in one instance (employee on live cattle export ship), but likely or possible exposures were able to be identified in each case. No clustering of cases was noted temporally or geographically. Four patients were Indigenous; 3 of these were tested for HTLV-1 and 2 were positive. One patient had diabetes and 1 had asthma. None were noted to have valvular heart disease and none was known to be pregnant.

Clinical and demographic features are summarised in Tables 1 and 2 and Figure 2. Diagnosis and definitive treatment was significantly delayed in all but one instance. Patients received multiple broad spectrum antibiotics, including macrolides (3 patients), beta lactams (3); cephalosporins (3); meropenem (2); vancomycin (2) and ciprofloxacin (1). Despite reported anti-*C. burnetii* activity of newer macrolides and quinolone antibiotics, the patient treated with ciprofloxacin died. The 2 patients whose antibiotic regimens included azithromycin had fever durations of 5 and 9 days respectively (compared with 1 day fever duration in the patient treated early with doxycycline, and a mean of 6.8 days in untreated or delayed treatment patients). Eight patients in total were treated with doxycycline, but this was often commenced after the resolution of clinical symptoms, when serological results had become available (28 days after illness onset in one instance). Invasive investigations included 3 lumbar punctures (all normal) and 1 liver biopsy (showing granulomatous hepatitis). Patients also had multiple other serological, microbiological and radiological investigations. The median hospital length-of-stay for the 8 hospitalised patients was 9.5 days (range 5 to 21).

Figure 1. Temporal distribution of Q fever cases in Central Australia and the Top End of the Northern Territory

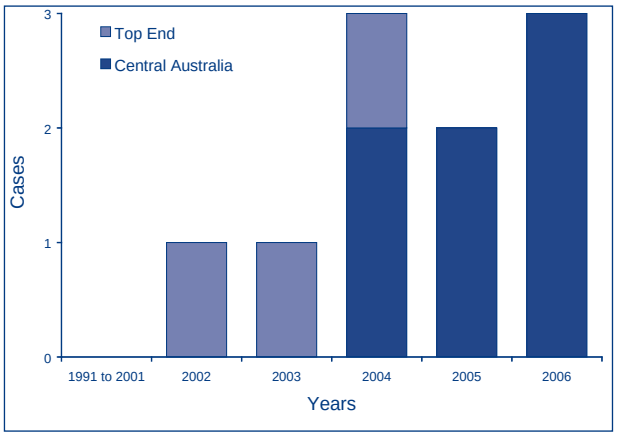


Table 1. Epidemiological and clinical data, Northern Territory Q fever cases, 1991 to 2006

Patient	Indigenous status	Age	Gender	Co-morbidities	Disease severity
Central Australian patients					
1	A	34	M	HTLV-1 positive	Moderate-severe
2	A	48	M	HTLV-1 positive, alcoholic liver disease, gastritis	Severe
3	A	37	M	Nil	Moderate-severe
4	A	27	F	Type 2 diabetes	Severe (died)
5	NA	43	M	Asthma	Moderate-severe
6	NA	48	M	Nil	Moderate
7	NA	29	M	Nil	Moderate
Top End patients					
8	NA	20	M	Nil	Moderate-severe
9†	NA	49	M	Nil	Severe
10	NA	62	F	Nil	Moderate-severe

A Aboriginal.

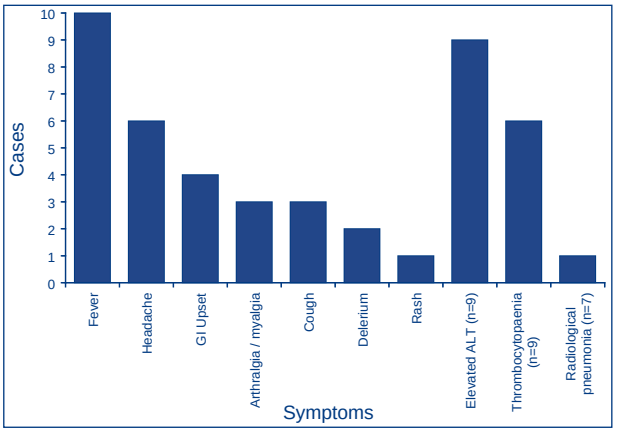
NA Non-Aboriginal.

† Patient described in case report, Reference 1.

Table 2. Possible exposure history, Northern Territory Q fever cases, 1991 to 2006

Possible exposure	Number of patients
Visited or lived in remote pastoral area	4
Non-occupational cattle slaughter	2
Minimal exposure evident, but drove in vicinity of cattle yards	2
Employee on live cattle export ship	1
Worked at cattle station	1

Figure 2. Symptom and laboratory abnormality frequency in 10 Q fever cases



GI Gastrointestinal.

ALT Alkaline phosphatase. Mean ALT=158.3 U/L.

Thrombocytopaenia range: 10 to 134 x 10^9/L.

The number of patients with results available for investigations are indicated in brackets.

Serological confirmation of Q fever was achieved using immunofluorescence assay in 9 instances, and a combination of enzyme-linked immunosorbent assay and complement fixation was done in the other instance. Five had paired serum samples demonstrating a rise in Phase 2 antibody titres, and 5 had significantly elevated Phase 2 antibodies on a single specimen. Q fever polymerase chain reaction (PCR) was not performed in any instance.

Discussion

Q fever reports from the Northern Territory since 1991 have all been acute Q fever diagnoses of at least moderate severity. The estimated financial cost to the health care system has been high due to multiple investigations, use of broad spectrum antibiotics and prolonged hospitalisation. These 10 notified cases probably represent a small proportion of all Q fever cases in the Northern Territory. European data indicate that hospitalised Q fever cases represent only 2%–4% of infected individuals.[2,4] Even though numbers are small, there is an impression that Q fever notification rates are rising in the Northern Territory. This may represent an increase in testing or a real increase, in contrast to the national figures.[8] During preparation of this manuscript, a further case of acute Q fever was notified in the Top End, bringing the number of confirmed cases in 2006 to four. Hence the notification rate for the Northern Territory in 2006 (population estimate 202,793[11]) was 2.0 cases per 100,000 population, compared with the most recently reported Australian national rate of 2.2 cases per 100 000 population.[8]

The clinical manifestations of Q fever are diverse; differences are thought to be attributable to (1) genetic differences in *C. burnetii* strains; (2) host factors such as age, sex, pregnancy, immunosuppression; (3) inoculum size; and (4) route of infection.[2,6] The clinical presentations of the patients described here are consistent with previously reported Australian Q fever cases, including the relatively uncommon finding of pneumonia or pneumonitis (1 of 10 patients) and the infrequent occurrence of rash (1 of 10 patients).[3] Although one of the 10 cases in this series died, death due to acute Q fever is uncommon, with 5 deaths recorded in Australia between 1982 to 1994.[2]

T-cell immunity is the primary mode of Q fever control by the infected host. Impaired T-cell immunity in HIV, cancers, lymphoma and pregnancy has been associated with failure to eradicate *C. burnetii*, and progression to chronic disease.[2,6,12] HTLV-1, which is endemic in Aboriginal Central Australians at up to 13.9% seroprevalence,[13] was positive in 2 patients in this series. While there are no previous reports of an association between HTLV-1 and Q fever, it is possible that the T-cell deficits associated with HTLV-1 may predispose to impaired clearance of intracellularly sequestered *C. burnetii* and a greater risk of chronic infection. The association of HTLV-1 and acute Q fever in the 2 patients reported is probably due to chance, but highlights the potential problems of the concurrence of endemic HTLV-I with Q fever. Since assiduous serological follow-up and prolonged therapy to reduce the risk of progression to chronic Q fever is recommended in other recognised risk groups (people with valvular heart disease or pregnancy)[6] after an episode of acute Q fever, such approaches may also be warranted in HTLV-1 positive individuals.

High rates of rheumatic heart disease in the Northern Territory[14,15] should also be cause for heightened Q fever awareness, since Q fever endocarditis is more likely to occur on previously damaged valves, especially if combined with T-cell immunosuppression.[2] A case of Q fever endocarditis is recalled from Alice Springs in the 1980s (Dr Nadarajah Rajabalendran, Alice Springs Hospital, personal communication), but details are unavailable.

Timely diagnosis and management of Q fever is required. This can be achieved through greater health staff awareness of this infection in the Northern Territory, with earlier testing and institution of effective antibiotic therapy. Q fever PCR tests are in development, which may facilitate early diagnosis. In an Australian cohort of 27 patients with acute Q fever, Q fever PCR assays (one or both of COM1 and IS1111 PCR) were positive in blood in 63% of patients overall, and in 89% of samples collected early in the illness prior to development of Phase 2 IgM antibody.[16]

Recommended first line treatment for acute Q fever is doxycyline 100 mg twice daily for 14 days,[17] or (debatably) co-trimoxazole 160 mg/800 mg twice daily in pregnant patients,[18] commenced within the first 3 days of illness to achieve reduction in fever duration.[2] The 2 patients described here who were treated from early in their illness with azithromycin remained febrile for 5 and 9 days compared with a mean of 6.8 days for untreated patients, and 1 day for the patient in whom doxycycline was administered early, suggesting lack of potency of azithromycin in these patients. This contrasts with a retrospective Greek study, where patients treated with clarithromycin were febrile for a mean of 3.9 days (only one day longer than those treated with doxycycline).[19] Heterogeneity in antibiotic resistance in different *C. burnetii* strains may explain such variable responses.[2] Because of the potential for late sequelae of *C. burnetii* infection, some recommend commencing treatment even after apparent symptom resolution if the initial opportunity for treatment was missed. Evidence for this approach in patients without risk factors for chronicity is lacking; nevertheless, all but 3 of the patients described here were treated in this manner as the diagnosis had not been made earlier in the course of the illness.

Even though Q fever might exist in the Northern Territory at higher rates than so far predicted, the lack of occupational exposure in this case series suggests that the current policy in the Northern Territory of opting out of the national vaccination program is appropriate, but this may require revision at a later date. The small inoculum required means that minimal exposure such as being in the vicinity of infected animals (as postulated for some patients in this series) may provide sufficient exposure for infection. Contact with other potential Q fever hosts (domestic dogs and cats, native bandicoots) may have been overlooked as possible exposures.

In conclusion, the occurrence or recognition of Q fever in the Northern Territory could be increasing, and under-diagnosis is likely. Small numbers prevent conclusions being drawn. Increased awareness, early testing and institution of effective anti-Q fever therapy may effect a decrease in morbidity and costs of the disease. While no cases of chronic Q fever were identified during this 15 year period, the risk factors for chronicity, especially in the Indigenous population, call for heightened awareness of this infection.

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Short reports

Reduction in invasive meningococcal disease in Queensland: A success for immunisation

Vicki G Slinko, Amy Sweeny

Abstract

Since 2003, the Australian government has funded a conjugate serogroup C meningococcal vaccine for those aged over 1 year and born since 1 January 1984. This summary of the epidemiology of invasive meningococcal disease (IMD) in Queensland assesses the effect that the vaccination program has had on IMD notifications. In Queensland, IMD cases are notified to the Notifiable Conditions System by clinicians and laboratories. Additional surveillance data are collected by population health units from relatives of the case, the case and medical practitioners. In 2005, Queensland recorded its lowest number of cases and lowest incidence of IMD since statewide surveillance began. This remained low in 2006. The serogroup C rate in Queensland also declined to its lowest in 2006. The pattern of age-specific incidence remains similar, though rates are lower in all but those aged less than 12 months. However, Indigenous rates are still twice non-Indigenous rates. The case fatality rate for IMD (all serogroups) has declined, possibly due to the reduced incidence of serogroup C and septicaemia cases. The program appears to have mostly achieved its aims of: reducing illness and death in the population at highest risk; inducing immunity in those who are vaccinated; and reducing the incidence of disease. However, there is considerable natural fluctuation in the rates of IMD and continued surveillance will be needed to monitor trends. *Commun Dis Intell* 2007;31:227–232.

Keywords: invasive meningococcal disease, Queensland, serogroup C, vaccination program, surveillance, notification, coverage, incidence, case fatality rate

Introduction

Meningococcal disease is an uncommon but important public health problem in Australia. The invasive form of the disease is a serious illness with a variable case fatality rate in industrialised countries ranging between 7% for meningitis and 19% for septicaemia.[1] Those known to be at highest risk of the disease are children aged less than five years (particularly infants), followed by adolescents and young adults.

The bacterium *Neisseria meningitidis* is usually carried asymptomatically in the back of the throat and nose. However, only a small number of people develop invasive disease, which appears most often as meningitis and/or septicaemia. Other localised manifestations include arthritis, pneumonia and conjunctivitis.

The factors leading to development of invasive meningococcal disease (IMD) are poorly understood, but risk factors include smoking, exposure to tobacco smoke and living in crowded conditions.[2]

In early 2003, a federally funded conjugate serogroup C meningococcal vaccination became part of the immunisation schedule in Queensland. All children aged more than 12 months and born since 1 January 1984 were eligible for the single dose vaccine. The program was introduced to:[3]

- reduce the illness and death in the population at highest risk of meningococcal disease;
- induce long term immunity in those who are vaccinated; and
- reduce the population incidence of disease.

This summary of the epidemiology of IMD in Queensland is to assess the effect that the vaccination program has had on the epidemiology of IMD.

Methods

IMD is immediately notifiable to Queensland Health under legislation, by both laboratories and clinicians. The data are maintained on the Notifiable Conditions Systems database and have been collated since 1993. In 1999, enhanced surveillance for IMD was established. Enhanced surveillance is conducted by communicable diseases staff of the population health units who also coordinate public health responses.

The criteria for notification of IMD are contained in Queensland Health *Guidelines for the Control of Communicable Diseases in the Community.*[4] This definition is compatible with the national guidelines,[5] though in Queensland nucleic acid testing is classified as laboratory definitive evidence rather than suggestive evidence. The Queensland IMD criteria were updated on 6 August 2005. The change was to make the case definition clearer but is unlikely to have changed the case detection rate. Cases are classified to the calendar year in which the date of onset occurs.

Figures for vaccination coverage come from the Vaccination Information and Vaccination Administration System, a Queensland Health register which maintains information on Queensland immunisation providers and vaccination events, and provides details to Australian Childhood Immunisation Register.

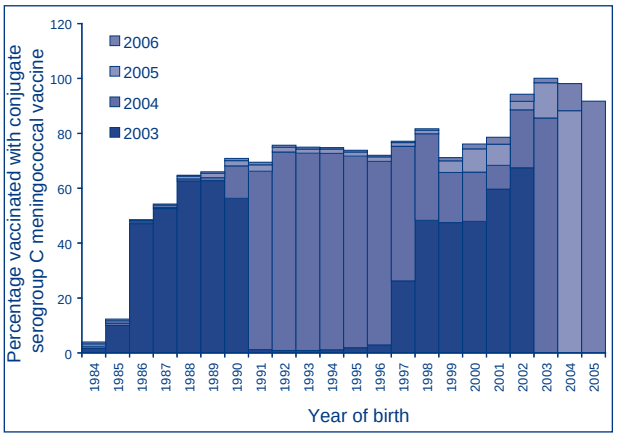
Denominators for notification rates each year use the Estimated Resident Population (ERP) from Australian Standard Geographical Classification.[6] The coverage percentages represent the number of persons in each annual birth cohort vaccinated divided by the 2006 ERP denominator for that birth cohort. The denominators for Indigenous calculations use high projections of Experimental Projections of Aboriginal and Torres Strait Islander Australians.[7]

Analysis was performed in Excel®[8] and Epi-Info version 3.3.2®.[9]

Results

High coverage against serogroup C IMD has been achieved for many at-risk populations in Queensland. Cohorts born since 2002, (children aged between 0–4 years in 2006), have coverage rates exceeding 90% (Figure 1). Persons born between 1988 and 2001 have achieved a coverage rate between 60% and 80% generally (Figure 1).

Figure 1. Per cent of coverage with conjugate serogroup C meningococcal vaccine, Queensland, date 2003 to 2006, by year of birth and vaccination



The notification rates for both serogroup C and non-serogroup C disease have declined since vaccine introduction (Figure 2). In 2005, the rate of IMD declined to its lowest (62 cases: 1.6/100,000 residents) since 1993. It remained low in 2006 (68 cases: 1.7/100,000). Only four cases of known serogroup C disease were reported in 2006.

The highest notification rate of IMD remains in those aged under 5 years (particularly infants) with a smaller peak of the disease in the teenage years (Table 1).

There has been little serogroup C disease in those aged < 1 year: 2 cases in 2003 and 1 in 2006. Since the vaccination program was introduced in 2003 there has been a gradual decline in the number of cases of serogroup C disease in those eligible for free vaccine: 2003 (14), 2004 (13), 2005 (4), 2006 (1). The

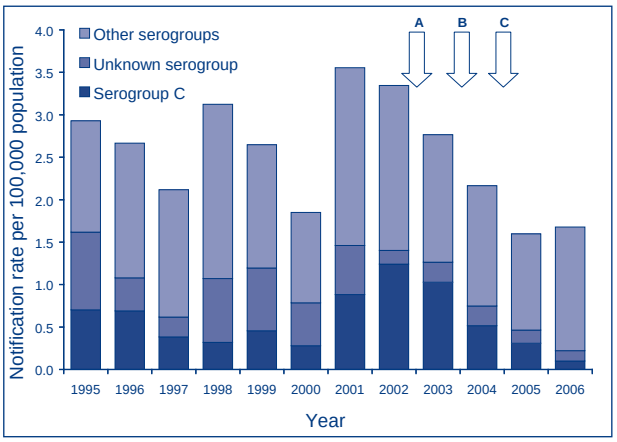
Table 1. Notification rate of invasive meningococcal disease, Queensland, 1995 to 2006, by age group

Age group	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006
<1	28.6	24.2	24.2	33	24.2	14.8	28.2	33.5	29.5	18.9	18.4	31.8
1–4	11.8	10.3	13.4	12.9	10.8	7.2	12	8	5.5	6.9	6	6.3
5–9	1.7	4.6	0.4	3.3	4.6	1.6	6.1	4.2	2.6	1.5	1.9	0.7
10–19	5.0	4.8	4.1	5.6	4.0	3.7	5.5	6.2	5.2	5.6	2.6	3.5
20–29	2.8	2.4	1.4	2.6	2.8	1.9	4.9	3.1	4.2	2.2*	1.3*	1.4*
30–39	1.2	0.4	0.4	0.8	0.4	0.4	0.7	2.2	1.8	0.9	0.4	0.9
40+	0.9	0.7	0.4	1.2	1.1	0.6	1	1.3	0.8	0.5	0.8	0.2
Total	2.8	2.6	2.1	3.2	2.8	1.9	3.6	3.3	2.8	2.2	1.6	1.7

Notification rates in shaded areas are for those groups eligible for free vaccine since the vaccination program began.

* Some individuals in these age groups will have been vaccinated in the catch up program.

Figure 2. Notification rate of serogroup C and invasive meningococcal disease, Queensland, 1995 to 2006



A Grades 8–12 school vaccination program introduced, vaccination of 1–5-year-olds and others not covered by continuing school program through usual providers.

B Grades 1–8 school vaccination program introduced.

C Vaccination of any others born on or after 1 January 1984, by usual providers.

Figure 3. Invasive meningococcal disease notifications, Queensland, 2000 to 2006, by month and year of onset

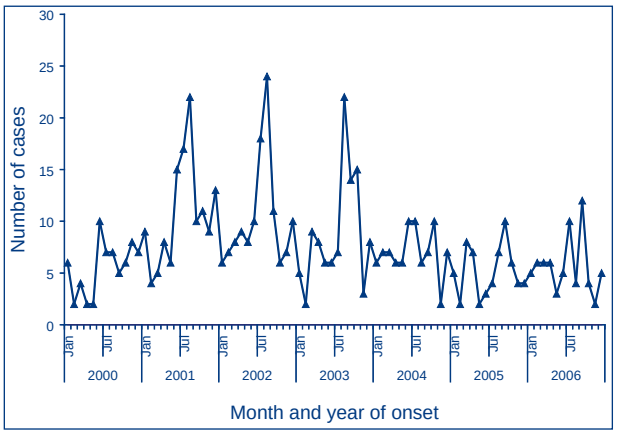
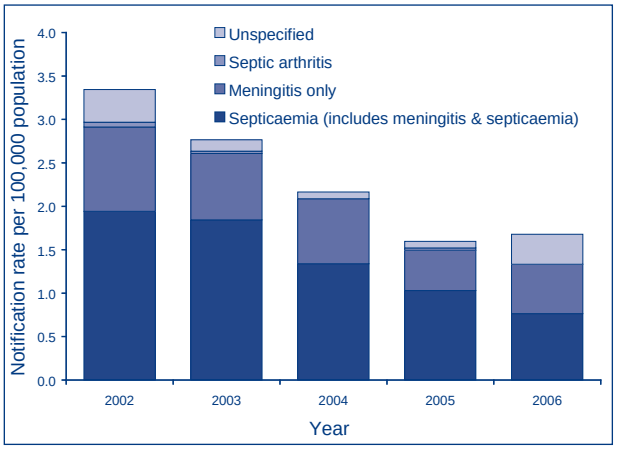


Figure 4. Notification rate of presentations of invasive meningococcal disease, Queensland, 2002 to 2006



vast majority of IMD in those eligible for free vaccine has been due to serogroup B disease (>90%). There have been no known vaccine failures.

The seasonality that was evident in previous years in IMD notifications (with a typical late-winter early-spring peak) is no longer obvious (Figure 3).

Types of clinical presentations since the vaccination program was introduced remain unchanged. The vast majority are meningitis and/or septicaemia. Since the introduction of the vaccination program, the number of presentations of septicaemia has been decreasing (Figure 4): 2002 (72), 2003 (70), 2004 (52), 2005 (40), 2006 (31).

The case fatality rate for IMD in Queensland is low (Table 2) and has fallen since the vaccination program. In Queensland, serogroup C disease is associated with nearly a fourfold greater risk of dying compared to serogroup B disease (Table 3, RR = 3.8; (95%CI 2.1, 7.2).

There continues to be a higher notification rate of IMD in Indigenous persons compared with non-Indigenous persons (Table 4). For the post-vaccination campaign period (2003–2006), Indigenous people were three times as likely to be notified with IMD compared to non-Indigenous (RR = 3.0; 95% CI 2.1, 4.3). There has not been a death of an Indigenous person from IMD since 2003.

Table 2. Invasive meningococcal disease case fatality, Queensland, 2000 to 2006

Year	Died	Total	Case fatality rate
2000	4	66	6.1
2001	11	129	8.5
2002	5	124	4.0
2003	9	105	8.6
2004	3	84	3.6
2005	3	62	4.8
2006	2	68	2.9
Total	37	638	5.8

Discussion

Case ascertainment may have improved due to advances in laboratory techniques. Nucleic acid testing/polymerase chain reaction has been used in Queensland since 1999 as laboratory definitive evidence of disease and the IgM test was introduced in 2000 (laboratory suggestive evidence). Improved case ascertainment would result in a higher rate of disease.

Table 3. Risk factors for dying of invasive meningococcal disease, Queensland, 2000 to 2006

Characteristic	Deaths (n=37)		All cases (n= 635)		Relative risk	95% confidence intervals
	n	%	n	%		
Sex						
M	21	57	346	54		
F	16	43	292	46	1.1	0.6, 2.1
Indigenous status						
Indigenous†	3	8	55	9		
Non-Indigenous†	33	92	536	91	0.9	0.3, 2.8
Serogroup						
C	21	57	163	26	3.8*	2.1, 7.2*
B	11	30	370	58	0.3*	0.2, 0.6*
Other/unknown	5	13	105	16	N/A	N/A

* Indicates statistical significance (α= 0.05).

† Indigenous status determined for 36 deaths, 591 cases.

Table 4. Risk of invasive meningococcal disease (all ages), Queensland, 2001 to 2006, by indigenous status

Vaccination program	Year	Indigenous cases	Non-Indigenous cases	Relative risk	95% confidence intervals
Before vaccination program	2001	7	118	1.5	0.7, 3.2
	2002	6	118	1.2	0.6, 2.8
During vaccination program	2003	13	92	3.4*	1.9, 6.0*
	2004	12	72	3.8*	2.1, 7.1*
	2005	6	56	2.4*	1.0, 5.5*
	2006	5	63	2.0	0.8, 5.1
	2003–2006	36	283	3.0*	2.1, 4.3*

* Indicates statistical significance (α= 0.05).

In 2005, the notification rate of IMD decreased to the lowest since Queensland-wide surveillance began and remained low in 2006. The notification rate of serogroup C disease is now at its lowest level recorded. Nonetheless, there has been considerable natural fluctuation in IMD.[10] The vaccination campaign and lower IMD notification rates follow a peak notification period in 2001–2002. Rates similar to those following the vaccination campaign were previously seen in 1997 and 2000. However, the steady declining trend since 2003 could be ascribed to the vaccination program.

One limitation to interpreting the change in serogroup-specific incidence is the change in the proportion of all IMD cases serogrouped over time. Before the vaccination campaign, up to 25% of IMD cases did not have serogroup information. This has improved since 2001.

The pattern of age-specific incidence of IMD remains unchanged. Those with the highest incidence (aged less than 12 months) are not eligible for free vaccine. The vast majority of the IMD in this age group is due to serogroup B. The small number of cases of serogroup C disease in those aged less than 1 year and in the whole population, may be due to reduced nasal carriage rates of *Neisseria meningitidis* serogroup C. There have been no recent studies of meningococcal nasal carriage in Australia to verify this hypothesis.

The rates of IMD in those eligible for free vaccine are currently some of the lowest recorded for Queensland. After the roll-out of the catch-up program from 2003, serogroup C disease has gradually been decreasing, with only 1 case recorded in those eligible for free vaccine in 2006. This was an overseas visitor working at an island resort, who may not have known about eligiblity or had access to vaccination. There is a continuing need to increase community awareness about the importance and benefits of vaccination for this disease.

The case fatality rate from IMD in Queensland has improved since the introduction of the vaccination program. The reduction in presentations with septicaemia and serogroup C disease (more likely to result in death) may account for this. The program appears to have achieved its aim of reducing illness and death in the population at highest risk of meningococcal disease.

Interestingly, the usual peak of notifications in winter[11] has not been evident after 2003. In 2000, before the vaccination program, there was no obvious winter peak of notifications and that year also had a low incidence of serogroup C disease.

Studies[12,13] suggest the meningococcal serogroup C vaccination program gives high short-term vaccine effectiveness and substantial herd immunity but the long term effectiveness remains unknown. There have been no known vaccine failures since the vaccination program was introduced in Queensland. Overall, the evidence suggests that the serogroup C meningococcal vaccination program has induced immunity for up to 4 years in those vaccinated.

The discrepancies between Indigenous and non-Indigenous health in Australia are well known. Though the relative risk of IMD in Indigenous persons has been decreasing recently, it remains at almost twice that of non-Indigenous persons. Obviously, further work needs to be done in this area, perhaps addressing risk factors for developing IMD.[2]

The meningococcal C vaccination program in Queensland has mostly been successful in achieving its aims. However, there needs to be continued vigilance with surveillance to evaluate longer term trends which can inform public health action and ensure sustained success.

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MENINGOCOCCAL SEPTICAEMIA AND A CASE OF CLINICALLY MILD ILLNESS

Danielle M Esler, Peter R Lewis

Introduction

The advent of new investigations for the detection of invasive meningococcal disease may lead to the diagnosis of milder forms of the infection which would previously have remained undiagnosed. In the context of mild disease there may be difficulty interpreting current guidelines and subsequently formulating an appropriate management and public health plan. This case study demonstrates the issues that may arise when positive serology results become available for a person with either partially, or un-treated mild invasive meningococcaemia.

Case study – a 28-year-old female

Day 1: Onset of mild headache, myalgia and arthralgia.

Day 3: The patient developed a petiechial rash (non-blanching) on lower limbs and trunk. She presented to the Emergency Department of her local hospital. In the hospital she was assessed as being systemically well with no fever, vomiting, photophobia or neck stiffness. Despite the absence of these symptoms she was given a differential diagnosis of meningococcal infection by the night medical registrar. He admitted her to hospital and over the next 12 hours had 2 g IV of ceftriaxone and 3 doses of IV penicillin. At this stage her white cell count was normal. Blood cultures, meningococcal polymerase chain reaction (PCR) and serology were ordered. The night registrar did not notify the public health unit.

Day 4: The patient was seen by a medical team. At this stage her rash had improved and she was still systemically well. Blood cultures and meningococcal PCR were found to be negative. The patient was discharged from hospital with a prescription for antihistamines and a presumed allergic reaction.

Day 12: The public health unit was notified that the patient's meningococcal serology was positive. The results were as follows:

- *Neisseria meningitides* IgM antibody positive to both outer membrane and capsular antigens (serogroup C);
- *Neisseria meningitides* IgG antibody negative.

The public health unit attempted to contact the admitting medical team and was able to speak with the patient's general practitioner. He told public health staff he had seen the patient 2 days prior for an unrelated minor procedure. During this visit she had seemed well and only briefly mentioned her hospital admission.

The patient was then contacted. She stated that she still had some resolving pain in her legs but felt otherwise well. She claimed her rash and headache had resolved. She denied ever having received a meningococcal vaccine. Public health staff advised her to attend the Emergency Department for clinical assessment and treatment of her meningococcal infection. The Emergency Department was contacted and advised of case details and a recommendation to treat the patient for invasive meningococcus.

Close household contacts were identified (her husband and 2 children) and prophylaxis administered accordingly.

The patient attended the Emergency Department and was seen by the staff specialist. She was assessed as clinically well and discharged with no treatment. She was asked to follow up with her general practitioner, the results of nasal and throat swabs which were taken in emergency. These swabs were negative for *N. menigitidis*.

Discussion

Based on an enzyme immunoassay, meningococcal capsular IgM serology has a sensitivity of 92% and specificity of 97%[1] between day 5 and day 20 after the onset of illness.

It may be positive if patients have recently been vaccinated.[1] In contrast, the assay for Outer Membrane Protein is less specific, being positive in some people with disseminated gonococcus. In view of both IgM assays being positive, and in the absence of vaccination this was almost certainly a case of meningococcal septicaemia though clinically a mild illness.

The NSW Notifiable Diseases Manual[2] states that:

> 'A confirmed case requires either:
>
> Laboratory definitive evidence,
>
> Or Laboratory suggestive evidence and clinical evidence.'

'High titre IgM or significant rise in IgM or IgG titres to outer membrane protein antigens of *N. meningitidis*' represents 'Laboratory suggestive evidence'[2] for invasive meningococcal infection. A time lag of 5–7 days between disease onset and IgM reaching diagnostic levels typically occurs.[3] 'Clinical evidence is then described as 'disease, which in the opinion of the treating clinician is compatible with invasive meningococcal disease.'[2] Similarly, the case definition in the national guidelines relies on 'disease which in the opinion of the treating clinician is compatible with invasive disease'[3]

The management of this patient (both during her initial admission and her second presentation to Emergency) was entirely dependent on the level of clinical suspicion for invasive meningococcus. She was discharged from hospital in the first instance because the treating team believed, in the absence of systemic symptoms, meningococcal infection was unlikely. Similarly, she was discharged from the Emergency Department after she attended at the request of Public Health staff. In this instance, after positive meningococcal serology was detected, with residual myalgias, and despite having been substantially under-treated for meningococcus, no further treatment was given. In both instances the treating doctors followed the guidelines. Their clinical suspicion for invasive meningococcus was low. Whether the treatment received by the patient was in her best interest however remains to be seen.

As this case demonstrates, serological testing for *Neiserria meningitidis* may lead to a delayed diagnosis of meningococcal disease in patients with mild disease. Such patients may not actually fulfil the case definition due to the mild nature of their illness. An outbreak of relatively mild invasive meningococcal infection, also confirmed by serology has been reported in the literature.[4] What needs to be quantified is the risk of people with mild disease progressing to more serious disease. The appropriate response of clinicians and public health personnel in such instances will remain unclear unless further evidence is generated allowing the guidelines to be updated.

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Quarterly reports

OzFoodNet quarterly report, 1 January to 31 March 2007

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 January to 31 March 2007.

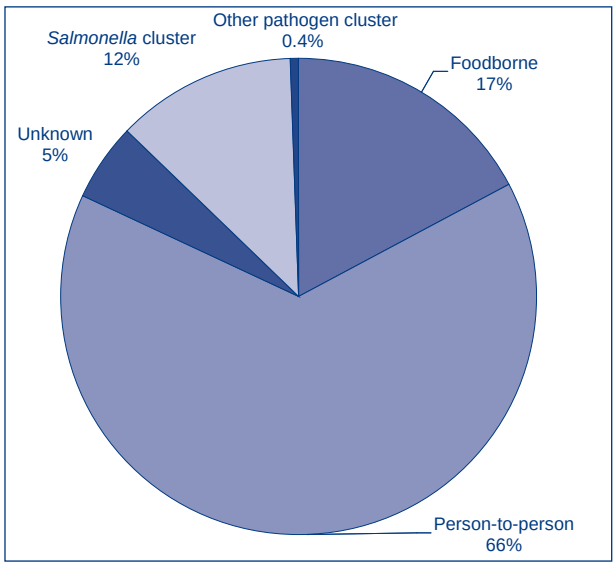
Data were received from OzFoodNet representatives in all Australian states and territories and a sentinel site in the Hunter/New England region of New South Wales. The data in this report are provisional and subject to change as the results of outbreak investigations can take months to finalise.

During the first quarter of 2007, OzFoodNet sites reported 234 outbreaks of enteric illness, including those transmitted by contaminated food. In total, these outbreaks affected 4,522 people, of which 239 were hospitalised and 6 died. The majority (65%, n=152) of outbreaks resulted from infections suspected to be spread by person-to-person transmission (Figure 1). Thirty-seven per cent (86/234) of outbreaks occurred in aged care facilities, 14% in child care centres and 12% (27/234) in hospitals. Norovirus was identified as a cause of illness in 36 outbreaks in aged care facilities. 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.

Foodborne disease outbreaks

There were 40 outbreaks during the first quarter of 2007 where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table). These outbreaks affected 861 people and resulted in 138 people being admitted to hospital. There were no deaths. This compares with 36 outbreaks in the first quarter of 2006 and 26 outbreaks in the fourth quarter of 2006.

Figure 1. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, 1 January to 31 March 2007



Salmonella was responsible for 18 outbreaks during the quarter, with *Salmonella* Typhimurium (16 outbreaks) being the most common serotype. *S.* Typhimurium 197 was responsible for 5 outbreaks; *S.* Typhimurium 44 four outbreaks; *S.* Typhimurium 9 three outbreaks; and *S.* Typhimurium 12, *S.* Typhimurium 135a, *S.* Typhimurium U302, *S.* Typhimurium U307 were each responsible for a single outbreak. Of these *S.* Typhimurium outbreaks, 12 were either confirmed or suspected to be associated with eating eggs or dishes containing eggs. The other *Salmonella* serotypes causing outbreaks during the quarter were *S.* Mbandaka and *S.* Saintpaul. There were 8 toxin-related outbreaks during the quarter including histamine poisoning (4 outbreaks), ciguatera fish poisoning (2 outbreaks), *Bacillus cereus* intoxication (1 outbreak) and *Clostridium perfringens* intoxication (1 outbreak). Norovirus was responsible for a single outbreak of foodborne disease. The remaining 13 outbreaks were caused by unknown aetiological agents.

Outbreaks of foodborne disease reported by OzFoodNet sites,* January to March 2007

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicles
NSW	January	Takeaway	Unknown	4	D	Suspected fried chicken
		Institution – other	Unknown	6	D	Unknown
		Restaurant	Unknown	9	D	Chicken stirfry or beef massaman
		National franchised fast food restaurant	Unknown	3	D	Unknown
	February	Restaurant	Unknown	4	D	Seafood platter
		Commercial manufactured food	Unknown	6	D	Berri blackcurrant play water
		Restaurant	Unknown	5	D	Unknown
		Restaurant	Histamine poisoning	2	D	Tuna steaks
		Grocery store/ delicatessen	Histamine poisoning	3	D	Tuna kebab steaks
		Takeaway	*Clostridium perfringens*	6	D	Hommus on a kebab
		Commercial caterer	*Bacillus cereus*	32	AM	Boiled gefilte fish (fish balls)
	March	Takeaway	*Salmonella* Typhimurium U302	34	D	Kebabs and crepes
		Takeaway	*Salmonella* Typhimurium 9	294	M	Vietnamese pork and chicken rolls
		Restaurant	*Salmonella* Typhimurium 12	7	D	Suspected marinated chicken dish, noodle dish, fried rice
Qld	January	Community	*Salmonella* Typhimurium 197	21	D	Suspected eggs
		Restaurant	*Salmonella* Typhimurium 197	3	D	Suspected eggs
	February	Private residence	Histamine poisoning	2	D	Tuna
		Primary produce	Ciguatoxin	2	D	Mackerel
		Camp	*Salmonella* Saintpaul	24	M	Bore water
		Restaurant	*Salmonella* Typhimurium 197	12	D	Suspected eggs
		Restaurant	*Salmonella* Typhimurium 197	6	D	Suspected eggs
		Restaurant	*Salmonella* Typhimurium 197	2	D	Suspected eggs
	March	Primary produce	Ciguatoxin	6	D	Mackerel
		Institution – other	Norovirus	45	A	Ham, salad, bread
SA	March	Restaurant	*Salmonella* Typhimurium 9	46	A	Multiple food items
Tas	January	Primary produce	Unknown	19	D	Suspected oysters
	March	Bakery	*Salmonella* Typhimurium 135a	20	D	Suspected eggs

Outbreaks of foodborne disease reported by OzFoodNet sites,* January to March 2007, *continued*

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicles
Vic	January	Private residence	*Salmonella* Typhimurium 44	4	M	Milkshake includes raw egg
		Restaurant	*Salmonella* Typhimurium 44	15	D	Caesar salad dressing includes raw egg
		Private residence	*Salmonella* Typhimurium 44	11	A	Trifle – includes raw egg
		Private residence	*Salmonella* Typhimurium 44	10	A	Tiramisu – includes raw egg
		Restaurant	Unknown	4	D	Unknown
		Takeaway	Unknown	17	A	Suspected meat curry
	March	Camp	Unknown	19	D	Suspected water
		Commercial caterer	Unknown	37	A	Suspected passionfruit coulis
		Restaurant	Unknown	10	A	Suspected feta cheese
		Camp	*Salmonella* Typhimurium 9	30	AM	Water
		Restaurant	Histamine poisoning	2	D	Tuna
WA	February	Restaurant	*Salmonella* Mbandaka	4	D	Unknown
	March	Restaurant	*Salmonella* Typhimurium U307	75	A	Caesar salad

* No foodborne outbreaks were reported in the Northern Territory 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.

Sixteen outbreaks reported in the quarter were associated with food prepared by restaurants, 5 from takeaway outlets, 4 in private residences, 3 from contaminated primary produce, 3 at camps, 2 by commercial caterers, and 2 associated with institutions. There were single outbreaks associated with food prepared by a bakery, a commercially manufactured food, community, a national franchised fast food restaurant and a grocery store.

To investigate these outbreaks OzFoodNet sites conducted 10 cohort studies, 3 case control studies, and collected descriptive data on 27 outbreaks. Investigators obtained analytical epidemiological evidence in 8 outbreaks, microbiological and analytical epidemiological evidence in 3 outbreaks and microbiological evidence alone in 3 outbreaks. For the remaining 26 outbreaks, investigators obtained descriptive epidemiological evidence implicating a food vehicle or suggesting foodborne transmission.

New South Wales reported 14 outbreaks of foodborne illness during the quarter. The aetiological agent was not identified in 7 of the outbreaks. In March, an outbreak of *S.* Typhimurium 9 due to contaminated Vietnamese style pork and chicken rolls affected 294 people and resulted in more than 100 people being hospitalised. Multiple food samples from the bakery were positive for *S.* Typhimurium 9. The response to this outbreak included a NSW Health media release alerting the public to the source of the outbreak and advice on what to do if you were ill. The NSW Food Authority restricted the bakery's production and sale of the implicated foods.

During March, 34 people were infected with *S.* Typhimurium U302 after eating kebabs and crepes prepared by a takeaway outlet. *S.* Typhimurium 12 was confirmed in 1 person from a group of 7 people ill following a meal of chicken and fried rice at a restaurant on Chinese New Years Eve.

There were 4 toxin-related outbreaks in New South Wales, including 2 outbreaks of histamine poisoning after meals of tuna; *Bacillus cereus* intoxication following consumption of boiled gefilte fish (fish balls); and *Clostridium perfringens* intoxication from kebab meals containing hommus.

Victoria reported 11 outbreaks of foodborne illness during the quarter. The aetiological agent was not identified in 5 of the outbreaks. Victoria experienced a state-wide increase in *S.* Typhimurium 44 infections during the quarter. There were 4 point source outbreaks associated with this community-

wide outbreak. The first was a group of 4 people who shared a raw-egg milkshake where the blender tested positive for *S.* Typhimurium 44. The second outbreak involved 15 cases who had eaten at the same restaurant and it was confirmed that the common food vehicle contained a dressing made with raw eggs. Eleven cases occurred in the third outbreak where the suspected source was a home-prepared trifle containing raw eggs and 10 people were ill after attending a function in a private residence where a home-prepared tiramisu made with raw eggs was served for dessert.

In Victoria, an outbreak of *S.* Typhimurium 9 affecting 30 children out of 55 adults and children who attended a school camp, was associated with drinking water. Water samples were positive for *S.* Typhimurium 9 and the camp was closed until drinking water met the standards specified in the Australian Drinking Water Quality Guidelines. Water was also suspected to have caused illness among 3 separate groups staying at another camp during late March. The untreated drinking water was being sourced from a nearby creek. Water samples at the camp were contaminated with *E. coli* and *Salmonella* (serotype pending). The camp was closed until a water treatment system was installed to meet the standards specified in the Australian Drinking Water Quality Guidelines.

One outbreak of histamine poisoning affected 2 people after a meal of tuna steaks. During the quarter, Victoria reported a single case of adult botulism. The same toxin that caused illness in the case was detected in the discarded remains of a ready-to-eat nachos meal consumed by the case during the incubation period. The case was severely ill requiring extensive hospital treatment including a long stay in Intensive Care Therapy. The manufacturer of the ready-to-eat nachos meal conducted a voluntary recall of the product as a precautionary health measure.

Queensland reported 10 outbreaks of foodborne disease for the quarter, 4 of which included restaurants and were part of a larger multi-state outbreak of *S.* Typhimurium 197 that began in November 2006. Queensland initiated a case control study to investigate the cause of illness. These outbreaks were all suspected to be associated with foods containing raw or undercooked eggs, or foods cross-contaminated by eggs from a farm where a specific *S.* Typhimurium 197 and its associated multiple-locus variable-number tandem-repeats analysis (MLVA) type was isolated. People infected with the matching outbreak MLVA strain were significantly more likely to have eaten a meal outside of the home in the 5 days before onset compared to other notified *Salmonella* cases chosen as controls (OR 11.2, 95%CI, 1.3–100). Cases with the outbreak strain were significantly more likely than controls to have attended a restaurant (OR 8.1, 95%CI, 1.8–35) and have eaten at a restaurant that was supplied eggs from the implicated farm (OR undefined). Environmental and other food samples from 3 of the 4 restaurants were all negative for *Salmonella*. However, samples taken from handwash basin, chopping board, preparation bench, food display unit lid and a tea towel in one restaurant were positive for the outbreak strain of *S.* Typhimurium 197.

S. Saintpaul caused illness among at least 24 children from 3 different schools who had attended a camp in Queensland during mid-February. Bore water was the only source of drinking water on site and was pumped to a storage tank and manually chlorinated. *S.* Saintpaul was detected in 1 of 5 water samples. The water tank and lines were cleaned and a more robust system of chlorination instituted.

An outbreak of norovirus among 45 people attending a Queensland training facility was epidemiologically associated with salad, ham, and bread. Queensland also reported 3 toxin-related outbreaks from fish consumption, including histamine poisoning after a meal of tuna imported from Indonesia. Two outbreaks of ciguatera fish poisoning, affecting 2 and 6 people respectively, occurred after meals of mackerel.

Western Australia reported 2 outbreaks of foodborne illness during the quarter. In March, *S.* Typhimurium U307 infected as many as 75 people after eating Caesar salad at a resort, prepared using a raw egg mayonnaise. The resort used eggs originating from Western Australia and Queensland. A comparison of *S.* Typhimurium U307 isolated from Western Australian cases and contemporaneous Queensland cases showed that cases from both states and an isolate from raw egg pulp in Queensland had the same MLVA type.

Investigators were unable to identify a food vehicle associated with an outbreak of *S.* Mbandaka during February. Three cases reported eating at the same café but had eaten different foods. A fourth locally acquired case had not eaten at the café. Isolates from all 4 cases had indistinguishable pulsed-field gel electrophoresis patterns.

Tasmania reported 2 outbreaks of foodborne illness during the quarter, including an outbreak of unknown aetiology affecting 5 groups of people following consumption of oysters. Eighteen people tested positive for *S.* Typhimurium 135a following consumption of salad rolls from a bakery in Tasmania. A further 2 people from the same community tested positive for *S.* Typhimurium 135a following consumption of eggs purchased from retailers who were supplied eggs from the same supplier

as that which supplied the bakery. The response to this outbreak included a government media release to warn the public that a very limited number of small egg producers had supplied cracked and dirty eggs potentially contaminated by *Salmonella*.

South Australia reported a single outbreak of foodborne illness during the quarter where *S.* Typhimurium 9 infected 46 patrons of a restaurant. Investigators identified multiple food items associated with illness, including any entrée (RR 3.6, 95%CI, 1.3–10), chicken salad (RR 1.7, 95%CI, 1.0–3.0), BBQ chicken (RR 1.8, 95%CI, 1.2–2.7) and chicken soup (RR 2.4, 95%CI, 1.5–3.7).

Northern Territory and the Australian Capital Territory did not report any foodborne outbreaks during the first quarter of 2007.

Cluster investigations

State and territory health departments conducted a substantial number of investigations into various clusters of *Salmonella* and other pathogens during the quarter. Jurisdictions reported investigating 29 clusters of various serotypes of *Salmonella*, some of which were related to point source outbreaks. In January, South Australia investigated a cluster of cases of Shiga toxin producing *E. coli* infections, predominantly serotype O157. OzFoodNet held several multi-jurisdictional teleconferences to discuss these cluster investigations and try to determine a source of infection. A common food source could not be identified despite an extensive investigation of these cases.

Comments

Overall, Australia experienced a 14% increase in reported *Salmonella* infection for the first 3 months of 2007 (3,500 cases) compared to the same period in 2006 (3,067 cases). The number of egg-associated outbreaks was considerably higher than in previous years. In response to this, health departments, departments of agriculture and food safety regulators have met with egg industry representatives and companies to discuss food safety. The Implementation Sub-Committee of the Food Regulation Standing Committee agreed to host a national egg food safety summit to discuss outbreaks. In addition, Food Standards Australia New Zealand is currently preparing a primary production standard for the egg industry, although this will not be finalised for some time.

This is the second consecutive quarterly report in which eggs have been identified as the most frequent cause of foodborne disease outbreaks. Between January and March 2007, 67% (12/18) of *Salmonella* outbreaks were suspected to be associated with dishes containing raw or undercooked eggs. This represented 30% (12/40) of all foodborne disease outbreaks during the quarter. During the last quarter of 2006, all outbreaks of *S.* Typhimurium were associated with dishes containing raw or undercooked eggs.[1]

The Queensland outbreak investigation associated with eggs contaminated by *S.* Typhimurium 197 was first identified in the last quarter of 2006. This investigation demonstrates the usefulness of MLVA as it allowed the Queensland investigation team to link multiple outbreaks and to specifically pinpoint the source of eggs on a farm.[2] The outbreak investigation lead to a series of public health actions including a voluntary recall of eggs by the egg company, a Queensland Health media release warning the public about *Salmonella* infections and how to safely use and consume eggs. Following these interventions in March 2007, there have been no further cases of *S.* Typhimurium 197 in Queensland linked to this egg farm (Figure 2). This investigation highlights the role of eggs in causing *Salmonella* infections and how contaminated produce from a single farm can result in community-wide outbreaks of human illness.[3]

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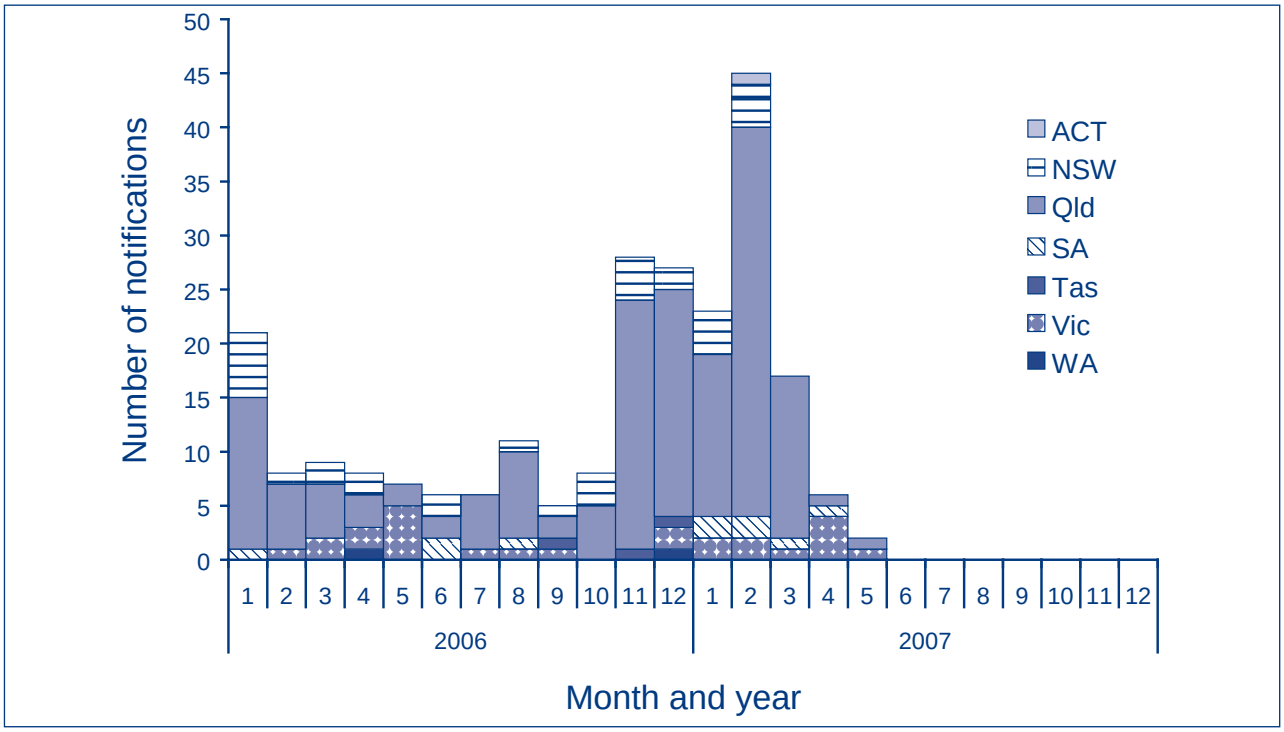
OzFoodNet thanks the investigators in the public health units and state and territory departments of health, as well as public health laboratories and local government environmental health officers who provided data used in this report. We would also like to thank laboratories conducting serotyping, phage typing and molecular analysis of *Salmonella* isolates for their work during the quarter.

The OzFoodNet Working Group is *(in alphabetical order)*: Robert Bell (Qld), Craig Dalton (Hunter New England), Gerard Fitzsimmons (DoHA), Kathleen Fullerton (DoHA), Robyn Gibbs (WA), Joy Gregory (Vic), Gillian Hall (NCEPH), Michelle Harlock (NT), Geoff Hogg (MDU), Martyn Kirk (DoHA), Karin Lalor (Vic), Meeyin Lam (NSW), Beth Lord (WA), Michelle McPherson (SA), Tony Merritt (Hunter New England), Sally Munnoch (Hunter New England), Jennie Musto (NSW), Lillian Mwanri (SA), Rhonda Owen (DoHA), Chris Oxenford (ACT), Raj Patil (DAFF), Nevada Pingault (WA), Jane Raupach (SA), Mark Salter (FSANZ), Minda Sarna (WA), Cameron Sault (TAS), Nicola Stephens (Tas), Russell Stafford (Qld), Hassan Vally (NCEPH), Tory Worgan (Hunter New England).

Figure 2. *Salmonella* Typhimurium 197 notifications reported to NNDSS, Australia, 2006–year to date, by month of diagnosis and state or territory



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Communicable diseases surveillance

Highlights for 1st quarter, 2007

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/or 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'.

Gastrointestinal diseases

Botulism

One case of botulism in a 25-year-old male was reported this quarter from Victoria. *Clostridium botulinum* toxin type A was isolated from the discarded residue of a pre-packaged nachos meal that the case had eaten, which was therefore suspected as the source of the case's illness. The Department of Human Services in Victoria triggered the National Food Incident Protocol as a precautionary measure and there was a voluntary recall of the food product (James Fielding, personal communication).

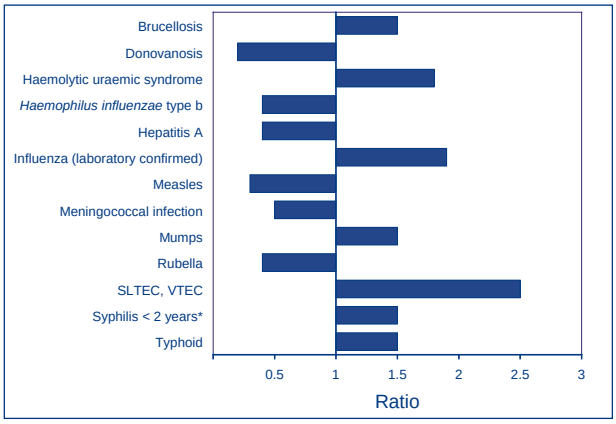
Botulism notifications in Australia are rare. Since 1998 there have been 13 cases of botulism notified nationally; 11 cases were intestinal (infant) botulism and 2 cases were adult botulism. The last adult case was notified in 1999.

Foodborne botulism has an incubation period of 12 to 36 hours and symptoms include blurry vision, lethargy and dizziness, followed by respiratory distress and paralysis. It cannot be transmitted from person to person.[1]

Typhoid

During the quarter there were 37 notifications of typhoid which was 1.5 times the 5-year mean for the same period. Notifications were from New South Wales (12), Victoria (10) and Western Australia (7) and 4 from South Australia, 3 from Queensland and 1 from Tasmania. There were 21 male and 16 female cases with an age range from 4 to 87 years. Notifications showed 32 cases acquired the infection overseas. One case was acquired locally by person-to-person transmission from recent refugee arrivals. The source of infection in the other 4 cases was unknown.

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 January to 31 March 2007 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.

Listeriosis

Fifteen notifications of listeriosis were received during the quarter, which was 80% of the 5-year mean for the same period. Those at highest risk of listeriosis are neonates, the elderly, immunocompromised individuals, pregnant women and alcoholic, cirrhotic or diabetic adults.[1]

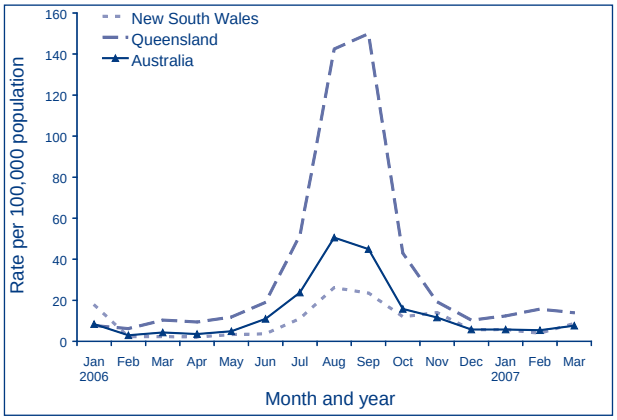
The majority of cases for this quarter were from New South Wales (5 cases), Victoria (4 cases), Queensland (3 cases) and 1 case each from South Australia, Tasmania and Western Australia. There were 5 male and 10 female cases. There was 1 neonate and the adults' ages ranged between 23 to 90 years. The highest rate of notifications was in the 80–84 year age group.

Vaccine preventable diseases

Influenza

There were 334 cases of laboratory-confirmed influenza in the first quarter of 2007. This was 1.9 times the 5-year mean for the same period. The majority of notifications were from New South Wales (32%) and Queensland (41%) (Figure 2). Seventy-eight per cent of the national laboratory-confirmed influenza notifications were type A, 15% type B and 7% unknown.

Figure 2. Notification rates of laboratory confirmed influenza, Queensland, New South Wales and Australia, 2006 to 2007



Measles

There were 4 cases of measles reported in the quarter. Cases were reported from New South Wales (2 cases), Queensland (1 case) and Victoria (1 case). Of the 4 cases, 3 were male and 1 was female. Two of the cases were aged 1 year or less and were unvaccinated, 1 child had travelled overseas and the other child was indigenous and had an unknown source. Two cases were aged between 30–34 years. Of the 2 adult cases 1 was unvaccinated and the vaccination history of the other was unknown. Both had recent overseas travel.

Meningococcal

There were 47 notifications of meningococcal infection during the quarter; half the 5-year mean for the same period. There were serogroup data available on 36 of the notified cases in the quarter. Thirty-one (66%) were serogroup B, 4 (9%) were serogroup C, 1 (2%) was W135 and in 11 (23%) the serogroup was unknown. Two meningococcal deaths were reported, 1 due to serogroup B and 1 to serogroup C. The 4 cases of serogroup C were aged between 24 –57 years and had not been vaccinated.

Zoonoses

Anthrax

One case of cutaneous anthrax was notified this quarter in a 35-year-old male knackery worker from Victoria. The case noticed a pimple on his left forearm 2 weeks after he gutted the first cow from the affected farm. Four days later the case was admitted to hospital, febrile and with a large necrotic lesion and extensive cellulitis extending from hand to the axilla. Penicillin-sensitive *Bacillus anthracis* was cultured. The case had contact with 2 cattle that were subsequently confirmed to have died from anthrax and was part of an outbreak among cattle from 10 farms in northern Victoria. An outbreak of anthrax among cattle occurred in the same area in 1997 (James Fielding, personal communication).

Human cases of anthrax are rare in Australia; only 2 other cases of anthrax have been notified since 1998 (1 case in 1998 from Queensland and 1 case in 2006 from New South Wales).

References

1. Heymann, D 2004, *Control of Communicable Diseases Manual,* 18th edition, American Public Health Association, Washington.

Acknowledgments

Thanks go to staff of the Surveillance Policy and Systems Section of the Australian Government Department of Health and Ageing and all our state and territory data managers.

Special thanks go to James Fielding for his contribution.

Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 36,988 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 January and 31 March 2007 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1. Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis 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 zoster (chickenpox)	All jurisdictions except NSW
Varicella zoster (shingles)	All jurisdictions except NSW
Varicella zoster (unspecified)	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 January to 31 March 2007, by date of onset*

Disease	State or territory								Total 1st quarter 2007†	Total 4th quarter 2006	Total 1st quarter 2006	Last 5 years mean 1st quarter	Year to date 2007	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	3	12	1	20	1	2	28	7	74	77	67	74	74	82.0	0.9
Hepatitis B (unspecified)	25	832	69	319	62	8	505	152	1,972	1,613	1,475	1,972	1,972	1,559.2	1.3
Hepatitis C (incident)	1	14	1	NN	7	5	21	19	68	88	129	68	68	116.0	0.6
Hepatitis C (unspecified)	64	1,558	65	807	77	63	735	298	3,667	3,035	3,183	3,667	3,667	3,512.0	1.0
Hepatitis D	0	3	0	3	0	0	2	0	8	5	10	8	8	6.0	1.3
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	1	0	1	1	0	1	1	0.4	2.5
Campylobacteriosis§	104	NN	70	1,075	946	223	1,861	545	4,824	4,390	3,733	4,824	4,824	4,119.6	1.2
Cryptosporidiosis	0	100	44	110	356	7	185	314	1,116	433	1,519	1,116	1,116	1,191.6	0.9
Haemolytic uraemic syndrome	1	4	0	1	1	0	0	0	7	8	5	7	7	4.0	1.8
Hepatitis A	2	23	2	6	2	0	9	2	46	54	106	46	46	112.0	0.4
Hepatitis E	0	4	0	0	0	0	2	0	6	6	8	6	6	8.0	0.8
Listeriosis	0	5	0	3	1	1	4	1	15	13	25	15	15	17.8	0.8
Salmonellosis (NEC)	38	1,028	181	894	386	116	546	267	3,456	2,099	3,066	3,456	3,456	2,833.8	1.2
Shigellosis	0	15	48	21	5	0	27	27	143	115	190	143	143	175.6	0.8
SLTEC, VTEC‖	1	3	2	4	24	0	6	0	40	21	16	40	40	16.2	2.5
Typhoid	0	12	0	3	4	1	10	7	37	21	20	37	37	25.0	1.5
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	3	0	0	0	0.8	0.0
Plague	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	NA
Smallpox	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	NA
Viral haemorrhagic fever	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	NA

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 January to 31 March 2007, by date of onset,* *continued*

Disease	State or territory								Total 1st quarter 2007†	Total 4th quarter 2006	Total 1st quarter 2006	Last 5 years mean 1st quarter	Year to date 2007	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	216	3,403	539	3,396	806	272	2,690	1,697	13,019	11,548	12,267	13,019	13,019	9,044.0	1.4
Donovanosis	0	0	0	1	0	0	0	0	1	1	0	1	1	4.2	0.2
Gonococcal infection	14	387	386	359	94	8	292	433	1,973	1,867	2,406	1,973	1,973	1,937.0	1.0
Syphilis (all)	12	356	42	89	8	9	203	29	748	719	626	748	748	560.6	1.3
Syphilis < two years duration	0	74	16	45	0	0	103	13	251	237	167	251	251	164.0	1.5
Syphilis >two years or unspecified duration	12	282	26	44	8	9	100	16	497	482	459	497	497	465.8	1.1
Syphilis - congenital	0	2	0	0	0	0	0	0	2	1	4	2	2	3.4	0.6
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0	NA
Haemophilus influenzae type b	0	0	0	1	0	0	1	0	2	7	2	2	2	5.4	0.4
Influenza (laboratory confirmed)	8	107	5	137	6	3	31	37	334	415	180	334	334	179.6	1.9
Measles	0	2	0	1	0	0	1	0	4	10	17	4	4	12.2	0.3
Mumps	0	23	2	12	2	0	1	5	45	42	43	45	45	31.0	1.5
Pertussis	18	343	4	327	80	5	196	21	994	1,279	2,428	994	994	1,708.2	0.6
Pneumococcal disease (invasive)	7	69	12	41	5	3	35	19	191	293	206	191	191	288.2	0.7
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0	NA
Rubella	1	2	0	2	0	0	2	1	8	12	6	8	8	21.8	0.4
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0
Tetanus	0	0	0	0	0	0	0	0	0	2	1	0	0	1.8	0.0
Varicella zoster (chickenpox)	NDP	NN	21	94	177	7	NDP	53	352	705	176	352	352	NA	NA
Varicella zoster (shingles)	NDP	NN	28	91	183	24	NDP	116	442	406	175	442	442	NA	NA
Varicella zoster (unspecified)	NDP	NN	0	776	92	6	NDP	194	1,068	920	862	1,068	1,068	NA	NA
Vectorborne diseases															
Barmah Forest virus infection	0	153	33	201	17	0	3	25	432	365	741	432	432	398.6	1.1
Dengue	0	31	5	36	4	0	4	11	91	36	57	91	91	159.6	0.6
Flavivirus infection (NEC)	0	0	0	9	0	0	1	0	10	4	15	10	10	20.8	0.5
Japanese encephalitis virus	0	0	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0
Kunjin virus	0	0	0	0	0	0	0	0	0	0	1	0	0	4.6	0.0
Malaria	4	29	5	61	3	6	32	19	159	152	212	159	159	204.4	0.8
Murray Valley encephalitis virus	0	0	0	0	0	0	0	0	0	0	0	0	0	1.0	0.0
Ross River virus infection	2	174	126	600	48	2	20	101	1,073	530	3,433	1,073	1,073	1,610.8	0.7

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2007, by date of onset,* *continued*

Disease	State or territory								Total 1st quarter 2007†	Total 4th quarter 2006	Total 1st quarter 2006	Last 5 years mean 1st quarter	Year to date 2007	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	1	0	1	0	1	1	1	0.2	5.0
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0	NA
Brucellosis	0	4	0	11	0	0	0	0	15	13	15	15	15	9.8	1.5
Leptospirosis	0	4	1	37	0	0	4	0	46	20	50	46	46	55.6	0.8
Lyssavirus unspecified	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0	NA
Ornithosis	0	9	0	0	1	0	12	0	22	39	43	22	22	40.4	0.5
Q fever	0	52	1	49	3	0	4	1	110	101	98	110	110	140.6	0.8
Other bacterial infections															
Legionellosis	1	31	0	13	4	1	5	11	66	94	101	66	66	83.6	0.8
Leprosy	0	1	0	0	0	1	0	0	2	0	1	2	2	2.8	0.7
Meningococcal infection**	1	18	3	14	1	0	7	3	47	68	75	47	47	90.0	0.5
Tuberculosis	1	96	11	34	9	1	89	19	260	338	289	260	260	260.6	1.0
Total	524	8,909	1,707	9,659	3,415	774	7,576	4,434	36,998	31,969	38,084	36,998	36,998	30,904.0	1.2

* 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 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, 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.

NDP No data provided

Table 3. Notification rates of diseases, 1 January to 31 March 2007, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis B (incident)	3.6	0.7	1.9	2.0	0.3	1.6	2.2	1.4	1.4
Hepatitis B (unspecified)	30.4	48.7	133.5	31.5	16.0	6.5	39.7	29.6	38.3
Hepatitis C (incident)	1.2	0.8	1.9	NN	1.8	4.1	1.6	3.7	1.6
Hepatitis C (unspecified)	77.9	91.3	125.8	79.6	19.8	51.5	57.7	58.1	71.2
Hepatitis D	0.0	0.2	0.0	0.3	0.0	0.0	0.2	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Campylobacteriosis†	126.5	NN	135.5	106.1	243.4	182.4	146.2	106.3	140.1
Cryptosporidiosis	0.0	5.9	85.2	10.9	91.6	5.7	14.5	61.2	21.7
Haemolytic uraemic syndrome	1.2	0.2	0.0	0.1	0.3	0.0	0.0	0.0	0.1
Hepatitis A	2.4	1.3	3.9	0.6	0.5	0.0	0.7	0.4	0.9
Hepatitis E	0.0	0.2	0.0	0.0	0.0	0.0	0.2	0.0	0.1
Listeriosis	0.0	0.3	0.0	0.3	0.3	0.8	0.3	0.2	0.3
Salmonellosis (NEC)	46.2	60.2	350.3	88.2	99.3	94.9	42.9	52.1	67.1
Shigellosis	0.0	0.9	92.9	2.1	1.3	0.0	2.1	5.3	2.8
SLTEC, VTEC‡	1.2	0.2	3.9	0.4	6.2	0.0	0.5	0.0	0.8
Typhoid	0.0	0.7	0.0	0.3	1.0	0.8	0.8	1.4	0.7
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§	262.8	199.4	1,043.1	335.1	207.4	222.5	211.3	331.0	252.7
Donovanosis	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	17.0	22.7	747.0	35.4	24.2	6.5	22.9	84.5	38.3
Syphilis (all)	14.6	20.9	81.3	8.8	2.1	7.4	15.9	5.7	14.5
Syphilis <2 years duration	0.0	4.3	31.0	4.4	0.0	0.0	8.1	2.5	4.9
Syphilis >2 years or unspecified duration	14.6	16.5	50.3	4.3	2.1	7.4	7.9	3.1	9.6
Syphilis - congenital	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.0	0.0
Influenza (laboratory confirmed)	9.7	6.3	9.7	13.5	1.5	2.5	2.4	7.2	6.5
Measles	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Mumps	0.0	1.3	3.9	1.2	0.5	0.0	0.1	1.0	0.9
Pertussis	21.9	20.1	7.7	32.3	20.6	4.1	15.4	4.1	19.3
Pneumococcal disease (invasive)	8.5	4.0	23.2	4.0	1.3	2.5	2.7	3.7	3.7
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3. Notification rates of diseases, 1 January to 31 March 2007, by state or territory. (Annualised rate per 100,000 population), *continued*

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, continued									
Rubella	1.2	0.1	0.0	0.2	0.0	0.0	0.2	0.2	0.2
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	NDP	NN	40.6	9.3	45.5	5.7	NDP	10.3	16.8
Varicella zoster (shingles)	NDP	NN	54.2	9.0	47.1	19.6	NDP	22.6	21.2
Varicella zoster (unspecified)	NDP	NN	0.0	76.6	23.7	4.9	NDP	37.8	51.1
Vectorborne diseases									
Barmah Forest virus infection	0.0	9.0	63.9	19.8	4.4	0.0	0.2	4.9	8.4
Dengue	0.0	1.8	9.7	3.6	1.0	0.0	0.3	2.1	1.8
Flavivirus infection (NEC)	0.0	0.0	0.0	0.9	0.0	0.0	0.1	0.0	0.2
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	4.9	1.7	9.7	6.0	0.8	4.9	2.5	3.7	3.1
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	2.4	10.2	243.8	59.2	12.3	1.6	1.6	19.7	20.8
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.1	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	1.1	0.0	0.0	0.0	0.0	0.3
Leptospirosis	0.0	0.2	1.9	3.7	0.0	0.0	0.3	0.0	0.9
Lyssavirus unspecified	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.5	0.0	0.0	0.3	0.0	0.9	0.0	0.4
Q fever	0.0	3.0	1.9	4.8	0.8	0.0	0.3	0.2	2.1
Other bacterial infections									
Legionellosis	1.2	1.8	0.0	1.3	1.0	0.8	0.4	2.1	1.3
Leprosy	0.0	0.1	0.0	0.0	0.0	0.8	0.0	0.0	0.0
Meningococcal infection‖	1.2	1.1	5.8	1.4	0.3	0.0	0.5	0.6	0.9
Tuberculosis	1.2	5.6	21.3	3.4	2.3	0.8	7.0	3.7	5.0

* 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, 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.

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NEC Not elsewhere classified.

NDP No data provided.

Laboratory Serology and Virology Reporting Scheme

There were 6,204 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 January to 31 March 2007 (Tables 4 and 5).

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 January to 31 March 2007, and total reports for the year†

	State or territory								This period 2007	This period 2006	Year to date 2007	Year to date 2006
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	1	–	2	2	–	1	–	6	11	6	11
Mumps virus	–	2	–	3	1	–	2	–	8	12	8	12
Rubella virus	–	–	1	5	1	–	–	–	7	2	7	2
Hepatitis viruses												
Hepatitis A virus	–	–	–	6	2	1	1	–	10	11	10	11
Hepatitis D virus	–	–	–	1	3	–	2	–	6	2	6	2
Arboviruses												
Ross River virus	–	1	26	226	43	–	–	3	299	788	299	788
Barmah Forest virus	–	8	3	113	15	–	1	–	140	144	140	144
Flavivirus (unspecified)	–	–	–	24	–	–	1	–	25	30	25	30
Adenoviruses												
Adenovirus not typed/ pending	–	62	–	17	60	–	19	–	158	155	158	155
Cytomegalovirus	–	26	–	64	166	1	19	–	276	325	276	325
Herpesviruses												
Varicella-zoster virus	4	96	–	429	148	1	12	–	690	352	690	352
Epstein-Barr virus	–	9	21	371	169	3	12	82	667	544	667	544
Other DNA viruses												
Poxvirus group not typed	–	–	–	–	–	–	1	–	1	–	1	–
Parvovirus	–	1	1	52	35	–	3	–	92	51	92	51
Picornavirus family												
Echovirus type 30	–	1	–	–	–	–	–	–	1	11	1	11
Rhinovirus (all types)	–	56	–	–	1	–	1	–	58	17	58	17
Enterovirus not typed/ pending	1	13	–	9	2	–	2	–	27	54	27	54
Ortho/paramyxoviruses												
Influenza A virus	–	5	2	35	3	–	2	–	47	30	47	30
Influenza B virus	–	–	–	1	4	–	–	–	5	6	5	6
Parainfluenza virus type 1	–	1	–	–	–	–	–	–	1	22	2	22
Parainfluenza virus type 2	–	2	–	3	–	–	–	–	5	1	5	1
Parainfluenza virus type 3	–	15	–	6	1	–	7	–	29	12	30	12
Respiratory syncytial virus	–	45	2	103	3	–	3	–	156	104	157	104
HTLV-1	–	–	–	–	2	–	–	–	2	4	2	4
Other RNA viruses												
Rotavirus	–	13	–	–	19	2	3	–	37	64	38	64
Norwalk agent	1	6	–	–	–	–	35	–	42	190	42	190

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 January to 31 March 2007, and total reports for the year,† *continued*

	State or territory								This period 2007	This period 2006	Year to date 2007	Year to date 2006
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other												
Chlamydia trachomatis not typed	1	138	–	1,373	595	7	4	–	2,118	1,475	2,127	1,475
Chlamydia psittaci	–	–	–	1	1	–	14	–	16	10	16	10
Mycoplasma pneumoniae	1	13	3	194	57	3	17	14	302	345	302	345
Mycoplasma hominis	–	3	–	–	–	–	–	–	3	10	3	10
Coxiella burnetii (Q fever)	–	1	–	18	5	–	–	–	24	47	24	47
Orentia tsutsugamushi	–	–	–	–	-	–	–	–	–	17	2	17
Rickettsia - spotted fever group	–	–	–	–	1	–	–	–	1	57	1	57
Streptococcus group A	–	10	3	198	–	1	20	–	232	137	232	137
Yersinia enterocolitica	–	1	–	–	–	–	–	–	1	3	1	3
Brucella species	–	–	–	1	–	–	–	–	1	2	1	2
Bordetella pertussis	1	6	–	93	62	–	13	–	175	429	175	429
Legionella pneumophila	–	1	–	–	–	–	1	–	2	8	2	8
Cryptococcus species	–	–	–	5	4	–	–	–	9	10	9	10
Leptospira species	–	–	–	23	–	–	–	–	23	7	23	7
Treponema pallidum	–	29	–	279	181	–	–	1	490	275	504	275
Entamoeba histolytica	–	–	1	1	–	–	–	–	2		2	
Toxoplasma gondii	–	–	–	5	2	–	1	–	8	16	8	16
Echinococcus granulosus	–	–	–	–	2	–	–	–	2	3	2	3
Total	9	565	63	3,661	1,590	19	197	100	6,204	5,793	6,231	5,793

* 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 January to 31 March 2007*

State or territory	Laboratory	January 2007	February 2007	March 2007	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	3	56	58	117
	New Children's Hospital, Westmead	63	45	59	167
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	18	19	16	53
	South West Area Pathology Service, Liverpool	24	7	–	31
Queensland	Queensland Medical Laboratory, West End	1,230	1,324	1,340	3,894
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	524	555	508	1,587
Tasmania	Northern Tasmanian Pathology Service, Launceston	9	6	1	16
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	–	–	–	–
	Royal Children's Hospital, Melbourne	46	46	15	107
	Victorian Infectious Diseases Reference Laboratory, Fairfield	45	40	1	86
Western Australia	PathCentre Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	41	2	103	146
Total		2,003	2,100	2,101	6,204

* The complete list of laboratories reporting for the 12 months, January to December 2007, 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

Childhood immunisation coverage

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

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 October and 31 December 2005, at 24 months of age for the cohort born between 1 October and 31 December 2004, and at 6 years of age for the cohort born between 1 October and 31 December 2000 according to the National Immunisation Program.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, *published in* Commun Dis Intell *2007;31:163–164 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 decreased marginally by 0.2 percentage points to 91.0% (Table 1), whilst there were no important changes in coverage for all individual vaccines due at 12 months of age. There were also no noteworthy 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 0.4 percentage points to 92.0% (Table 2). 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 2 vaccines where no further doses are due between 6 months and 24 months of age (DTP and polio), coverage at the national level was 94.8% and 94.8% respectively at 24 months versus 91.9% and 91.8% 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 3 shows immunisation coverage estimates for children 'fully immunised' and for individual vaccines at 6 years of age for Australia and by state or territory. For the second consecutive quarter, 'fully immunised' coverage for Australia, at 88%, remained at the highest level ever recorded since it was first reported in early 2003. There were no important changes in coverage for all individual vaccines due at 12 months of age and no noteworthy movements in coverage for individual vaccines by jurisdiction.

Table 1. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2005; assessment date 31 March 2007

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,145	22,544	827	13,626	4,320	1,551	16,486	6,573	67,072
Diphtheria, tetanus, pertussis (%)	92.2	91.7	91.7	91.9	91.6	92.7	92.6	90.5	91.9
Poliomyelitis (%)	92.1	91.5	91.5	91.9	91.6	92.8	92.5	90.5	91.8
Haemophilus influenzae type b (%)	94.9	94.6	94.9	93.9	94.1	95.9	94.8	94.2	94.5
Hepatitis B (%)	95.0	94.6	95.5	93.7	93.9	95.9	94.6	94.0	94.4
Fully immunised (%)	91.9	91.2	90.8	90.8	90.4	92.5	91.3	89.9	91.0
Change in fully immunised since last quarter (%)	0.0	-0.3	-1.5	+0.7	-0.7	-1.5	-0.4	-0.3	-0.2

Table 2. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2004; assessment date 31 March 2007

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,038	21,285	756	12,536	4,312	1,417	15,691	6,172	63,207
Diphtheria, tetanus, pertussis (%)	95.9	94.6	96.4	94.1	95.0	96.3	95.5	94.4	94.8
Poliomyelitis (%)	95.8	94.5	96.2	94.1	95.0	96.3	95.4	94.3	94.8
Haemophilus influenzae type b (%)	94.2	93.4	93.9	93.1	93.9	94.8	94.5	92.7	93.6
Measles, mumps, rubella (%)	94.1	93.1	95.1	93.1	94.3	94.9	94.8	92.7	93.7
Hepatitis B(%)	96.1	95.4	97.9	95.1	95.6	96.8	96.2	95.2	95.6
Fully immunised (%)	93.0	91.5	93.3	91.3	92.8	93.9	93.4	90.6	92.0
Change in fully immunised since last quarter (%)	-0.5	-0.6	-1.2	-0.5	+4	-0.6	-0.3	-0.2	-0.4

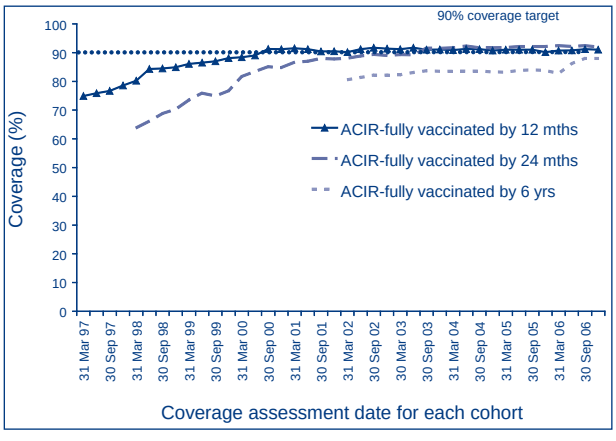
* The 12 months age data for this cohort was published in *Commun Dis Intell* 2006;31:266.

Table 3. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2000; assessment date 31 March 2007

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,073	22,321	839	13,504	4,444	1,597	16,157	6,573	66,508
Diphtheria, tetanus, pertussis (%)	90.0	89.0	87.4	88.0	86.8	91.5	91.5	84.2	88.8
Poliomyelitis (%)	89.9	88.9	88.0	88.1	86.7	91.6	91.6	84.4	88.9
Measles, mumps, rubella (%)	89.8	89.0	88.1	88.2	86.8	91.6	91.7	84.3	88.9
Fully immunised (%)[1]	88.6	88.1	86.3	87.2	85.9	90.7	91.0	83.2	88.0
Change in fully immunised since last quarter (%)	-0.8	+0.3	-1.8	-0.4	-0.7	+1.4	+0.9	-1.5	+0.1

Figure 3 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 2 years for all age groups. The recent increase in coverage at 6 years of age, described in the previous paragraph, is apparent in the Figure. It should be noted that currently, coverage for the vaccines added to the National Immunisation Program since 2003 (varicella at 18 months, meningococcal C conjugate at 12 months and pneumococcal conjugate at 2, 4, and 6 months) are not included in the 12 or 24 months coverage data.

Figure 3. Trends in vaccination coverage, Australia, 1997 to 31 December 2006, by age cohorts



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 *2005;29:91–92.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 October to 31 December 2006, as reported to 31 March 2007, are included in this issue of Communicable Diseases Intelligence *(Tables 4 and 5).*

Table 4. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 October to 31 December 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	18	2	8	4	0	11	0	43	19	141	92
	Male	0	95	1	47	15	1	61	11	231	216	850	868
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	0	114	3	55	19	1	72	11	275	235	994	961
AIDS diagnoses	Female	0	1	0	1	0	0	2	0	4	1	22	27
	Male	0	20	0	3	4	0	11	1	39	53	172	197
	Total*	0	21	0	4	4	0	13	1	43	54	196	224
AIDS deaths	Female	0	0	0	0	0	0	1	1	2	2	6	5
	Male	0	8	1	1	1	0	4	2	17	18	71	64
	Total*	0	8	1	1	1	0	5	3	19	20	79	69

* Totals include people whose sex was reported as transgender.

Table 5. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 31 December 2006, and reported by 31 March 2007, by sex and state or territory

	Sex	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	32	873	22	267	100	8	367	203	1,872
	Male	259	13,447	131	2,729	935	100	5,248	1,212	24,061
	Not reported	0	230	0	0	0	0	22	0	252
	Total*	291	14,579	153	3,005	1,036	108	5,659	1,422	26,253
AIDS diagnoses	Female	10	253	4	71	32	4	112	41	527
	Male	93	5,414	43	1,030	410	52	1,998	426	9,466
	Total*	103	5,684	47	1,103	443	56	2,122	469	10,027
AIDS deaths	Female	7	136	1	42	20	2	61	26	295
	Male	74	3,582	27	663	279	33	1,414	295	6,367
	Total*	81	3,729	28	707	299	35	1,484	322	6,685

* Totals include people whose sex was reported as transgender.

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 *2007;31:162.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 January to 31 March 2007, are included in this issue of Communicable Diseases Intelligence *(Table 6).*

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

Table 6. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2007, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD
Australian Capital Territory	07			1	1					1	1			2	2
	06													0	0
New South Wales	07			12	12	3	3					2	2	17	17
	06			9	9	1	1			1	1	4	4	14	14
Northern Territory	07			1	1									1	1
	06			1	1									1	1
Queensland	07			11	11									11	11
	06			15	15	1	1								16
South Australia	07			1	1									1	1
	06			3	3									3	3
Tasmania	07														0
	06			1	1	1	1							2	2
Victoria	07			6	6									6	6
	06			10	10	2	2	1	1	2	2			15	15
Western Australia	07			3	3									3	3
	06			5	5									5	5
Total	07			35	35	3	3			1	1	2	2	41	41
	06			44	44	5	5	1	1	3	3	4	4	57	57

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 January to 31 March 2007 are included in Tables 7 and 8. Data include cases reported and entered by 27 April 2007. 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 *2007;31:163–164.*

1 January to 31 March 2007

There were 2,527 reports to NEPSS of human *Salmonella* infection in the first quarter of 2007, 35% more than in fourth quarter of 2006. Data to date for this quarter are somewhat incomplete. Completion of phage typing and data-entry for the many recent *S*. Typhimurium isolates will result in at least 10% more cases, and a final count for the quarter somewhat above the historical average for this annual peak in human salmonellosis in Australia.

During the first quarter of 2007, the 25 most common *Salmonella* types in Australia accounted for 1,657 cases, 66% of all reported human *Salmonella* infections. Nineteen of the 25 most common *Salmonella* infections in the first quarter of 2006 were also among those most commonly reported in the preceding quarter.

The most notable feature of the current data is the large outbreak of *S*. Typhimurium phage type 44 in the south-eastern states and S. Typhimurium phage type 9 in South Australia and New South Wales.. Other increases and outbreaks include *S*. Mississippi and *S*. Typhimurium phage type 135 (in Tasmania), *S*. Typhimuium phage type 29 (in South Australia), *S*. Montevideo and *S*. Wangata (in New South Wales), *S*. Stanley, *S*. Newport, *S*. Cerro and *S*. London (in Victoria), *S*. Oslo in the Northern Territory, *S*. Typhimurium phage type 197 (in Queensland), *S*. Typhimurium phage type U302 (in New South Wales and Queensland), and *S*. Typhimurium phage type U307 (in Western Australia and Queensland).

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 7. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 January to 31 March 2007, as reported to 27 April 2007

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	32	644	95	713	173	110	534	226	2,527
Total contributing *Salmonella* types	23	107	35	109	59	18	113	78	228

Table 8. Top 25 *Salmonella* types identified in Australia, 1 January to 31 March 2007, by state or territory

National rank	*Salmonella* type	State or territory								Total 1st quarter 2007	Last 10 years mean 1st quarter	Year to date 2007	Year to date 2006
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium PT 135	2	102	0	46	2	25	34	14	225	247	225	262
2	*S.* Typhimurium PT 44	5	25	0	12	18	1	103	2	166	25	166	77
3	*S.* Typhimurium PT 9	1	78	0	19	19	2	40	4	163	182	163	158
4	*S.* Saintpaul	1	12	5	64	1	1	8	19	111	134	111	167
5	*S.* Typhimurium PT 170	4	60	0	7	0	1	33	0	105	96	105	167
6	*S.* Typhimurium PT 197	1	12	1	63	4	1	7	0	89	60	89	41
7	*S.* Virchow PT 8	0	6	5	67	1	0	2	0	81	104	81	114
8	*S.* Birkenhead	0	37	0	36	0	0	5	0	78	101	78	112
9	*S.* Mississippi	0	2	0	3	0	64	4	0	73	41	73	55
10	*S.* Chester	0	10	4	18	2	2	3	12	51	68	51	65
11	*S.* Muenchen	1	7	3	20	4	0	2	12	49	59	49	55
12	*S.* Infantis	0	10	2	5	10	0	16	3	46	49	46	70
13	*S.* Aberdeen	0	3	0	42	0	0	0	0	45	46	45	67
14	*S.* Typhimurium PT 12	0	21	0	1	2	2	11	3	40	35	40	40
15	*S.* Typhimurium PT 29	0	3	3	1	26	0	5	0	38	7	38	9
16	*S.* Typhimurium (PT pending)	1	1	0	0	1	2	31	1	37	0	37	0
17	*S.* Hvittingfoss	0	2	1	30	0	0	2	1	36	40	36	65
18	*S.* Typhimurium RDNC	2	15	0	15	1	0	3	0	36	30	36	37
19	*S.* Stanley	0	11	1	4	3	0	11	3	33	14	33	25
20	*S.* Typhimurium untypable	0	4	0	4	0	1	6	15	30	20	30	29
21	*S.* Montevideo	1	20	0	5	1	0	3	0	30	12	30	14
22	*S.* Waycross	0	14	0	13	0	0	0	0	27	46	27	60
23	*S.* Typhimurium PT U302	0	9	0	14	0	0	2	1	26	4	26	12
24	*S.* Oslo	0	1	21	1	0	0	1	0	24	4	24	1
25	*S.* Typhimurium PT U307	0	3	0	8	0	0	3	4	18	6	18	22

OVERSEAS BRIEFS

The Overseas briefs highlights disease outbreaks during the quarter that were of major public health significance world-wide or those that may have important implications for Australia and now includes an update on the global HIV epidemic.

Reporting period 1 January to 31 March 2007

Avian influenza

The number of cases of human H5N1 confirmed by the World Health Organization (WHO) during the first quarter of 2007 was similar to the same period in 2006. The WHO confirmed 39 cases (case-fatality rate, CFR 61.5%)[1] with dates of onset between 1 January and 31 March 2007, compared with 58 cases (CFR 55.2%)[2] during the same period in 2006. Indonesia continues to have the highest number of cases, with 19 confirmed cases (CFR 84.2%) between 1 January and 31 March 2007.[1]

There was no evidence of human-to-human transmission of avian influenza during the reporting period. The source of infection for many of the Indonesian cases is still being investigated due to their late confirmation (16 May 2007), but contact with backyard poultry has been established for 15 of the 20 cases from other countries (China, Egypt, Laos and Nigeria).[1]

Chikungunya

Between 1 January and 31 March 2007, continuing outbreaks of chikungunya fever were reported from India,[3] Indonesia[4] and Sri Lanka.[5] In addition, imported cases of chikungunya fever were reported from France (1 case)[6] and Malaysia (47 cases).[7] These cases were linked to India and Madagascar respectively, which are known to have endemic chikungunya. There were no reports of fatal cases of chikungunya fever during the quarter.

Cholera

During the reporting period, new and continuing outbreaks of cholera or watery diarrhoeal syndrome were reported from India, Malaysia, the Solomon Islands and the African nations of Angola, the Congo, Cote d'Ivore, the Democratic Republic of the Congo, Djibouti, Ethiopia, Kenya, Liberia, Namibia, Nigeria, Senegal, Sierra Leone, Somalia, the Sudan, Tanzania, Uganda, Zamibia and Zimbabwe.[8] Cholera is considered to be endemic in all of these areas.

Dengue fever

Health alerts were declared in the South American nations of Brazil, Paraguay and Bolivia during the reporting period after outbreaks of dengue fever in these and other South American countries. Argentina has also been affected but to a lesser extent.[9] Dengue fever is not uncommon in South America, but unusually high rainfall and warmer weather has provided favourable breeding conditions for mosquito populations.

Paraguay was the most affected country in the outbreaks. Between 1 January and 9 April 2007, the Pan American Health Organization (PAHO) reported 25,000 cases of dengue fever including 13 deaths from dengue haemorrhagic fever.[10] The most affected departments were Capital, Cordillera, Amambay, Central, Concepcion and Paraguari. Peak incidence occurred in epidemiological weeks 8 and 9 (18 February to 4 March 2007).[10]

Human immunodeficiency virus

This information is sourced from the annual update of Joint United Nations Programme on HIV/AIDS (UNAIDS)[11] unless otherwise indicated.

The prevalence and incidence of human immunodeficiency virus (HIV) and acquired immune deficiency syndrome (AIDS) world-wide are increasing. The Joint United Nations Programme on HIV/AIDS estimates that there were 39.5 million people living with HIV world-wide in 2006 including 4.3 million new infections, compared with 36.9 million cases including 3.9 million new infections in 2004.

Sixty-three per cent of the world-wide cases of HIV in 2006 were from sub-Saharan Africa according to UNAIDS. Swaziland has the highest adult prevalence (the prevalence in people aged 15 years or over) of HIV in the world (33.4%) followed by Botswana, Lesotho and Namibia (adult prevalence in each of these countries is 20%-24%). HIV prevalence varies between the 3 regions of sub-Saharan African. Southern Africa has the highest prevalence, with only Zimbabwe showing a trend of decreasing prevalence. In East Africa there was a general trend of stabilising or declining prevalence (to <10% in most countries) and West Africa had the lowest prevalence in sub-Saharan Africa (<5% in every nation).

UNAIDS estimates that 8.6 million people were living with HIV in Asia in 2006, including 960,000 new infections. South East Asia had higher levels of infection than the rest of the region. In South East

Asia, the highest risk factors for HIV infection were unprotected paid sex, unprotected sex between men who have sex with men and unsafe drug injecting practices. The number of people in Vietnam who were living with HIV doubled to 260,000 between 2000 and 2005, with 40,000 new cases each year, the majority of them injecting drug users or people who buy or sell sex. In Thailand however, where UNAIDS estimated that 580,000 people were living with HIV in 2005, the prevalence has stabilised and many new cases occurred in formerly low risk populations such as married women. In 2005, HIV prevalence in the Philippines remained at less than 0.1% and Indonesia had 170,000 people living with the infection.

In Oceania, UNAIDS estimates that 81,000 people were living with HIV in 2006 including 7,100 new infections. Three-quarters of these cases were in Papua New Guinea where adult prevalence was estimated to be 1.8%[11] compared with <0.1% in Australia.[12] HIV infection in Papua New Guinea seemed to be associated with heterosexual sex. HIV infections in women aged 15-29 years were twice as high as amongst men of the same age. According to UNAIDS, high rates of transactional sex, low levels of condom use, early sexual debut, widespread sexual violence against women, and frequent concurrent sexual partnerships have all contributed to the epidemic. Island nations such as Fiji, Kiribati, the Samoa, the Solomon Islands and Vanuatu have reported few cases (<300 cases each since reporting started), but risk factors exist which could lead to increasing incidence, with high proportions of men paying for sex and men having sex with men.

Measles

World-wide measles vaccination levels increased from 71% to 77% in the period 1999 to 2005, resulting in a 60% drop in the number of measles deaths.[13] A new target set by the WHO in partnership with other international agencies, aims to reduce measles deaths by 90% by 2010 compared with 1999 levels, primarily by ensuring a second dose of vaccine is received by all children shortly after the first.[14]

There are still major outbreaks of measles in some countries despite the progress that has been made in reducing measles deaths world-wide. During the first quarter of 2007, outbreaks of measles were reported from China, Japan, Kenya, Kuwait, North Korea, the Russian Federation, Scotland, Serbia and Spain.

In the month of February 2007, China reported 9,500 cases of measles, 68% more cases than were reported in the same period last year.[15] Measles outbreaks in the United States of America and Europe have been associated with the entry of adopted Chinese children into those areas.[16]

WHO Indonesia estimates that measles causes 30,000 deaths in children per year in Indonesia and that 1.2 million children in the country do not receive adequate measles immunisation.[17] The Government of Indonesia began a mass immunisation campaign on 20 February 2007 targeting 13 million children on the island of Java.[18]

The WHO estimates that in 2005, 86% of one-year-olds in China and 72% in Indonesia had received one dose of measles vaccine, which compares poorly with one-dose vaccination rates in Australia (94%) and Vietnam (95%).[19]

Meningococcal disease

The WHO states it is highly likely that a new epidemic wave of meningitis will emerge in Africa in the coming years.[20] The 2006 epidemic season saw a significant increase in meningitis outbreaks across the African meningitis belt, where outbreaks of meningococcal disease are common during the dry season (December to June).[21] In early 2007, the trend of an increasing number of outbreaks continued. Between January and March 2007, the WHO reported major outbreaks of meningococcal disease from Burkina Faso, the Sudan, and Uganda, with minor outbreaks occurring in Cote d'Ivore and the Democratic Republic of the Congo.

Burkina Faso had major outbreaks of meningococcal in 2006. More cases were reported in the first quarter of 2007 than previously reported for any full year since 1997.[22] Between 1 January and 8 April 2007, the WHO confirmed 22,255 cases (CFR 7%).[23] By 8 April 2007, 34 of 55 districts in the country had crossed the epidemic threshold (10 cases per 100,000 population).[23] Where antibiotics and hospital care are available, CFRs for meningococcal disease are expected to be between 8% and 15%,[24] so the CFR of the Burkina Faso cases is not unusual.

Mumps

Between early February and early April 2007, Canada reported 32 laboratory-confirmed cases of mumps from Nova Scotia.[25,26] Most cases were aged 19-30 years and many were students of one university.[27] Health authorities are concerned about the disease spreading to other areas when the students return home for the summer break.[28] This is the third outbreak in Nova Scotia in 3 years.

Poliomyelitis

Only Afghanistan, India, Nigeria and Pakistan have never interrupted wild poliovirus transmission.[29] However, case numbers this year in these endemic countries indicate progress has been made towards eradicating polio, with only half as many

cases reported between 1 January and 3 April 2007 (66 cases) as in the same period of 2006 (128 cases).[29] During this period, 14 cases of polio were reported from 3 re-infected countries: Somalia (5), Democratic Republic of the Congo (7) and Niger (2).[29]

One of the cases reported from India was a type 1 wild poliovirus case (the type most often associated with serious cases of paralysis[29]) from Bihar State where transmission of wild poliovirus type-one is ongoing. But worldwide in 2007, for the first time ever, the number of wild poliovirus type 1 cases is fewer than type 3 cases, demonstrating the effectiveness of the monovalent oral polio type 1 vaccine that is in widespread use.[30]

Rift Valley fever

Outbreaks of Rift Valley fever (RVF) were reported from Kenya, Somalia and Tanzania between mid-December 2006 and March 2007. The first of these outbreaks of RVF was reported from the Garissa district in Kenya's North East Province in mid-December 2006,[31] followed by Somalia in late December 2006 and Tanzania in February 2007.

Kenya and Somalia

The outbreaks of RFVF in Kenya and Somalia appear to have slowed, with no reports of new cases since mid-February 2007. Between 30 November and 12 March 2007, the WHO reported 684 cases (CFR 23%) from Kenya.[32] The North Eastern Province has been the most affected, with the Province's Garissa and Ijara districts reporting approximately 70% of the country's cases. Between 19 December 2006 and 20 February 2007, 114 cases (CFR 45%) of RVF were reported from 14 districts in the south of Somalia.[32] The security situation in the country made it difficult for health authorities to conduct surveillance and fully investigate cases. This may explain the higher CFR seen in Somalia compared with Kenya and Tanzania if only the most severe Somalian cases (those who had been hospitalised) were included in the case numbers. A lack of appropriate treatment might also have contributed to the higher CFR compared with other countries.

Tanzania

In early February 2007, the first Tanzanian cases of RVF in the current outbreak were reported from the areas of Arusha, Manyara and Tanga bordering Kenya. By mid-February, the WHO had reported 8 suspected cases from these areas. In mid-March the disease appeared to be spreading southwards in Tanzania, with 58 suspected cases (CFR 24%) reported from the Dodoma region, followed by 60 suspected cases from the Morogoro region by 23 March 2007.[33]

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A CELEBRATION OF THE LIFE OF PROFESSOR AILEEN PLANT

1948-2007

On 27 March 2007, while working in Jakarta, Professor Aileen Plant passed away suddenly. Professor Plant was one of the World Health Organisation's leading experts in outbreak investigation and a medical epidemiologist, as well as a professor of international health at Curtin University of Technology. Professor Plant also held the position of Deputy Chief Executive Officer of the Australian Biosecurity CRC for Emerging Infectious Diseases.

Within her extensive experience in outbreak investigation, her main interests were in the applied research and policy aspects of infectious disease control. She was passionate about her work and travelled extensively, often with great risk to herself, to help people and countries in need of her expertise.

It was her vast experience in the field of infectious disease control which prompted the WHO to invite her to join an expert investigation team being assembled in Vietnam to investigate the outbreak of a deadly virus, later to become known as SARS. Professor Plant and the medical investigation team in Hanoi worked tirelessly to identify the virus and to develop a method for its control, again at great risk to their own personal safety.

In recognition of her leadership during the SARS epidemic, Professor Plant was awarded the National Medal of Honour by the Vietnamese Government for her leadership of the SARS outbreak control program.

Professor Plant was a leader in her field and a person of great compassion. She will be sadly missed by her many colleagues and friends at Curtin and internationally.

A web site in tribute to Aileen has been established at: http://memorial.curtin.edu.au/aileenplant/index.cfm

Condolence messages can be read or added at: http://memorial.curtin.edu.au/aileenplant/messages.cfm

COMPOSITION OF THE AUSTRALIAN INFLUENZA VACCINE FOR THE 2007 SEASON

The Australian Influenza Vaccine Committee (AIVC) met on 4 October 2006, and agreed to adopt the September World Health Organization recommendations.

The AIVC decided that the influenza vaccine components for the 2007 Southern Hemisphere season should contain the following:

A (H1N1)	A/New Caledonia/20/99 (H1N1) - like strain. A/New Caledonia/20/99 (IVR-116) is recommended as a suitable vaccine strain.	15 µg HA per dose
A (H3N2)	A/Wisconsin/67/2005 (H3N2) - like strain. A/Wisconsin/67/2005 (NYMCX-161B) or A/Hiroshima/52/2005 (IVR-142) are also recommended as suitable vaccine strains.	15 µg HA per dose
B	B/Malaysia/2506/2004 - like strain.	15 µg HA per dose

National Serology Reference Laboratory, Australia

24th NRL Workshop on Serology

Radisson Resort, Palm Meadows Drive, Gold Coast, Queensland

23–26 October 2007

The NRL Workshop is an established feature of the Australian annual scientific meetings calendar. We invite you to participate in this important workshop. (All laboratory and scientists interested in quality and new developments). The Workshop offers opportunity for laboratory directors and scientists, manufacturers and distributors of diagnostic kits and reagents, regulators, quality assurers, students and others to interact and discuss advances and difficulties encountered in laboratory science.

The 2007 Workshop will build on the strengths developed in previous conferences. The poster/oral presentations format and breakout sessions will once again provide a relaxed but informative environment for exchanging information and ideas. Participants can expect to be part of a vibrant learning and networking experience, through the interactive sessions promoting discussion and debate on the pertinent issues surrounding the use of *in vitro* diagnostics.

Discussions will concentrate on clinical serology, performance driven quality control, measurement of uncertainty and the new regulatory framework for *in vitro* diagnostics. With the new IVD regulations validation of laboratory tests will assume new importance.

Important dates

Registration available On-line: From 2 April

Earlybird Registration closes: 2 July 2007

Abstract submission deadline: 1 August 2007

Authors notified of abstract acceptance ([two weeks following submission)

Late Registration closes: 1 October 2007

Registration cancellation deadline: 1 October 2007

Oral presentations to Secretariat deadline: 1 October 2007

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For further information: NRL website: www.nrl.gov.au – please check it regularly!

ERRATUM

Figure 19 in the article 'Australia's notifiable diseases status, 2005: Annual report of the National Notifiable Diseases Surveillance System', published in *Communicable Diseases Intelligence* (*Commun Dis Intell* 2007;31:27) was incorrect. The correct figure is reproduced below.

Figure 19. Notification rate for hepatitis A, Australia, 2005, by age group and sex