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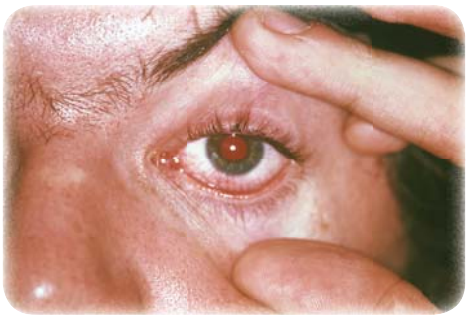
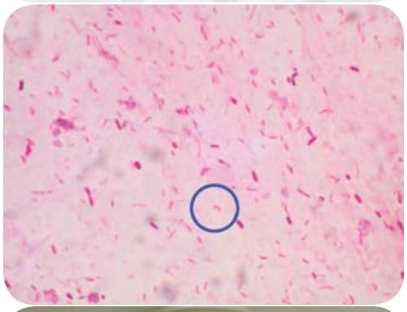
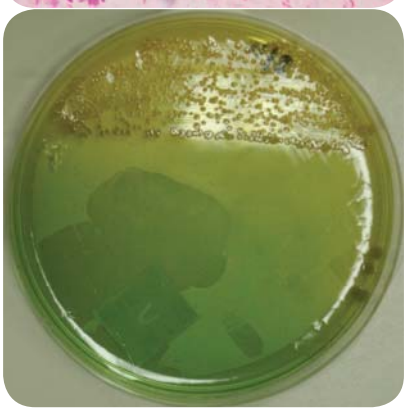
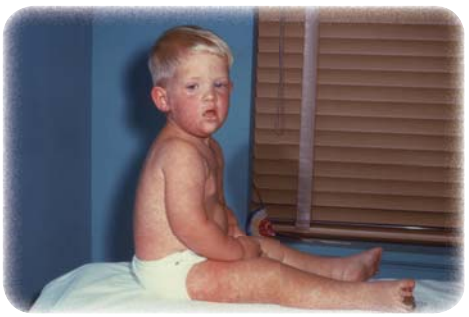
Department of Health and Ageing

Communicable Diseases Intelligence









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Front cover: Surveillance Section, Australian Government Department of Health and Ageing.
Left: Cholera vibrio photos courtesy of Luke Chen: stool sample; Cholera vibrio; selective medium showing typical yellow colonies.
Right: Reiter's syndrome usually begins as *Chlamydia trachoma,* Joe Miller, CDC; *Salmonella* Java can be acquired from aquariums; Child with measles, CDC. Images sourced from the Centers for Disease Control and Prevention Public Health Image Library, courtesy of the Centers for Disease Control and Prevention, Atlanta, Georgia.

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Contents

Cont'd next page

Annual report of the National Influenza Surveillance Scheme, 2005

Simon M Firestone,[1,2] Ian G Barr,[3] Paul W Roche,[1] John C Walker[1]

Abstract

Surveillance of influenza 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 2005, 4,575 cases of laboratory-confirmed influenza-like illness were reported, which was 115 per cent higher than in 2004. The influenza season started in the first week of June, with peak activity in early August, a month earlier than in 2004. Influenza A was the predominant type notified (73%), while influenza B activity continued to increase compared to previous years. During 2005, the influenza notification rate amongst persons aged over 65 years (22 cases per 100,000 population) was 70 per cent higher than the mean rate of the last four years. One thousand one hundred and seventy-four influenza isolates from Australia were antigenically analysed: 689 were A(H3N2), 210 were A(H1N1) strains and 275 were influenza B viruses. Continued antigenic drift was seen with the A(H3N2) viruses from the previous reference strains with approximately one quarter of isolates being distinguishable from A/Wellington/1/2004-like viruses and more closely matched to A/California/7/2004-like viruses. *Commun Dis Intell* **2006;30:189–200.**

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

Introduction

Influenza is a major threat to public health worldwide because of its ability to spread rapidly through populations, showing a greater severity in the very young, the frail elderly and people with chronic diseases.

Influenza is an acute self-limiting viral disease of the upper respiratory tract. The health and economic impact of influenza largely arise from related complications such as lower respiratory tract infections and exacerbation of cardiopulmonary and other chronic diseases. These complications result in excess hospitalisation and mortality.

Influenza infections are seasonal in temperate climates (June to September in the Southern Hemisphere and December to April in the Northern Hemisphere), but may occur throughout the year in tropical regions. The seasonal activity of influenza virus varies from year to year with some years marked by larger epidemics with higher morbidity and mortality. In Australia in 2004, influenza and pneumonia were the underlying causes of 3,381 deaths.[1]

The potential for an epidemic of influenza is dependent on the susceptibility of the population and the ability of the viruses to evolve. There are three types of influenza viruses, A, B and C. The ancestral hosts for influenza A viruses are aquatic birds, however, certain subtypes have become established in various mammals, including humans and pigs. Both influenza B and C are restricted to humans, although influenza C has been isolated from pigs.[2] Influenza C causes a less severe illness than either influenza A or B, more akin to the common cold.[3]

Influenza virus types are further subtyped by the antigenic properties of two surface proteins, haemagglutinin (H) and neuraminidase (N). Influenza viruses are successful human pathogens because of their ability to vary these two external proteins, thus evading the immune system. Mutations cause a gradual change in these proteins called 'antigenic drift', which results in annual epidemics of influenza. The greater the change in these proteins, the more likely it is that the virus will evade the immunity conferred by earlier infections or vaccinations, and the greater the epidemic potential.

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At irregular intervals, there are more dramatic changes in the viral proteins, called 'antigenic shift', which are a result of either direct introduction of avian influenza viruses into the human population or a reassortment between human and avian viruses which is postulated to occur in an intermediate host such as pigs. Periodically, these 'shifts' result in the emergence of a novel influenza virus that spreads rapidly through susceptible populations, resulting in pandemics (worldwide epidemics).[4] Unlike the seasonal epidemics of influenza, where attack rates depend on age, reflecting immunity conferred from previous infection, in pandemic influenza all age groups are susceptible.

Three pandemics occurred in the 20th century. The Spanish Flu (A/H1N1) pandemic of 1918–1919, is estimated to have caused at least 20 million deaths worldwide, with unusually high mortality among young adults.[5] Mortality associated with the 1957 'Asian Flu' (A/H2N2) and the 1968 'Hong Kong Flu' (A/H3N2) pandemics was less severe, with the highest mortality in the elderly and persons with chronic diseases.[6]

As it is impossible to predict when the next pandemic will occur or how severe the illness will be, an effective national surveillance system is essential for the control of seasonal epidemics and preparedness for potential pandemics. Since 2003, outbreaks of influenza A(H5N1) virus in multiple bird species in Asia, Europe and Africa and associated human and animal cases have raised serious concerns that this virus subtype may acquire the ability for person-to-person transmission and result in pandemic influenza (http://www.who.int/csr/disease/avian_influenza/en/index.html). 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 provisions of clinical services;
- characterisation of the nature of the epidemic;
- 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; and
- evaluation of the impact of the epidemic and associated public health measures.

In 2005, the Communicable Diseases Australia website (http://www.health.gov.au/cda) published influenza surveillance data fortnightly during the influenza season. This annual influenza report is a summary of the Australian surveillance information gathered by various systems in 2005, and includes a summary of related international influenza activity.

Surveillance methods

Surveillance of influenza in Australia is based on six sets of data:

- notifications of laboratory-confirmed influenza required by legislation in most states and territories, and notified 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
- hospitalisation 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 legislature. 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 in order to present disease occurrence during the reporting period, 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 report influenza diagnosis all year round. In 2005, 11 laboratories from all jurisdictions except Western Australia 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). In Australia, there are six such schemes: the Australian Sentinel Practice Research Network (ASPREN) which collects data at a national level, the New South Wales Influenza Surveillance Program, 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, including ASPREN, used the national case definition for ILI in 2005.

Absenteeism surveillance

Australia Post, a major nation-wide employer, provided sick leave absenteeism data collected weekly for 2005. Absenteeism, defined as an absence due to illness for at least three consecutive days, was presented as a rate per 100 employees per week, on an average of 32,938 employees per week.

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 the United States of America, are responsible for analysing influenza viruses collected through an international surveillance network involving over 100 national laboratories. 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 sample, together with any strains demonstrating uncharacteristic reactions during antigenic characterisation, are further analysed by genetic sequencing of the viral haemagglutinin antigen and, for a proportion of these, the neuraminidase antigen. Together with serological and epidemiological data, this forms the basis from which WHO makes recommendations in February (Northern Hemisphere) and September (Southern Hemisphere) for the formulation of vaccines 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; for influenza A the subtype of the haemagglutinin (H) and neuraminidase (N) proteins may be included in brackets after the designation. For avian/animal isolates the species yielding the isolate is also included. 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 (e.g. recommendations for 2006; http://www.who.int/csr/disease/influenza/vaccinerecommendations1/en/index.html) are then translated into actual virus strains acceptable to regulatory authorities and vaccine manufacturers by national and regional committees (e.g. the Australian Influenza Vaccine Committee http://www.tga.gov.au/committee/aivc2006.htm).

Hospitalisation data

The Australian Institute of Health and Welfare provided data on hospital separations in public and private hospitals. The number of separations with a primary diagnosis of influenza due to identified influenza viruses (ICD-10AM = J10) and influenza where the virus was not identified (ICD-10AM = J11) were reported for 2004. Data were not available for 2005 at the time of writing this report.

Adult Vaccination Survey

In past years, vaccination coverage rates for persons aged over 65 years were reported from the Adult Vaccination Survey (AVS). In 2005, the AVS was not conducted due to the anticipated effect of influenza vaccine supply problems on the results. Therefore coverage of the Influenza Vaccine Program for Older Australians was difficult to estimate. During 2005, numerous eligible persons aged over 65 years chose to buy their influenza vaccine through the Pharmaceutical Benefits Scheme (PBS) rather than wait for supplies to return to normal.

In 2004, the influenza vaccine was given to 79 per cent of Australians aged over 65 years.

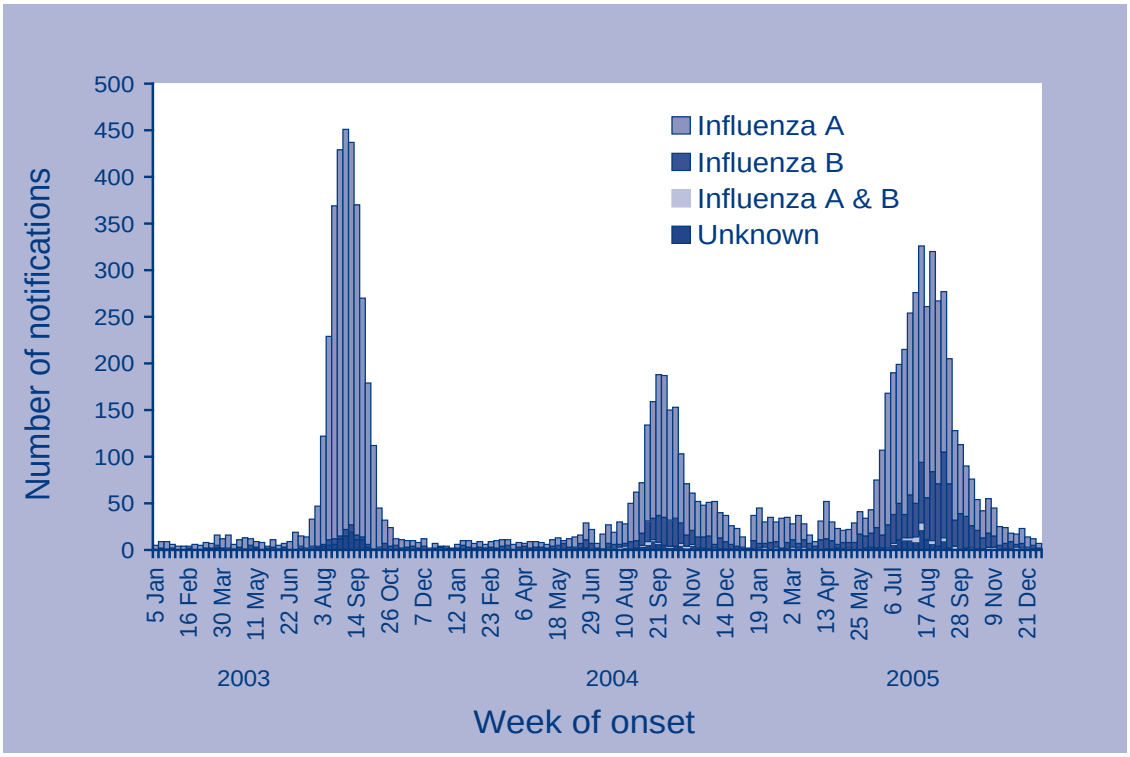
Results

Laboratory-confirmed influenza represents a small proportion of all influenza cases in the year and consequently the estimation of the circulating strains is based on a small sample, and should be interpreted with caution. As 2004 was the first time that all the sentinel schemes used the same case definition, this is the first report where comparisons of ILI rates, across all sentinel schemes, with the rates from the previous year, are possible.

National Notifiable Diseases Surveillance System

In 2005, 4,575 laboratory-confirmed cases of influenza were reported to NNDSS, which represents a 115 per cent increase from the number of notifications in 2004 (Figure 1). All jurisdictions reported laboratory-confirmed influenza to NNDSS, although Tasmania reported very few cases due to limited access to laboratory testing for influenza.

Figure 1. Notifications of laboratory-confirmed influenza to the National Notifiable Diseases Surveillance System, Australia, 2003 to 2005, by week of onset and type



In 2005, influenza A was the predominant type notified (73.1%), while influenza B activity continued to increase compared to previous years (Table 1).

Notifications of laboratory-confirmed influenza started to increase in early June (week 24) and peaked in early August (week 32) (Figure 2). The influenza season started nearly two months earlier than in 2004, and was characterised by a steady rise in the rate of notifications over 10 weeks.

A comparison of notification rates in each jurisdiction (with the exception of the Australian Capital Territory and Tasmania) is shown in Figure 3. In April, a small peak occurred in the influenza notification rate in the Northern Territory (3 cases per 100,000 population). Peak influenza notification rates occurred in July in Victoria (4 cases per 100,000 population), Western Australia (9 cases per 100,000 population), and South Australia (7 cases per 100,000 population) and in August in Queensland (17 cases per 100,000 population), the Northern Territory (10 cases per 100,000 population) and New South Wales (5 cases per 100,000 population).

Figure 2. Notifications of laboratory-confirmed influenza to the National Notifiable Diseases Surveillance System, Australia, 2004 and 2005 comparison, by week of onset

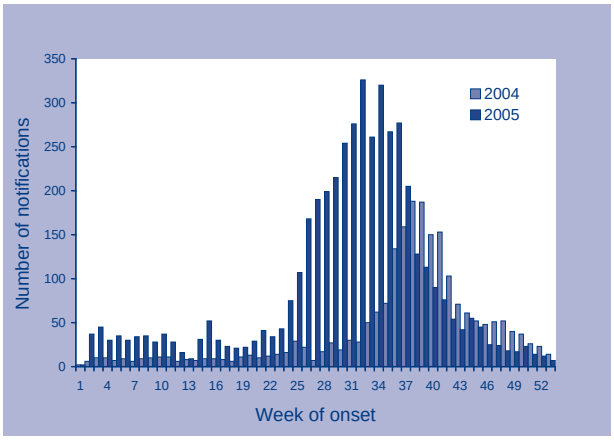


Figure 3. Notification rates of laboratory confirmed, Australia, 2005, by jurisdiction and month of onset

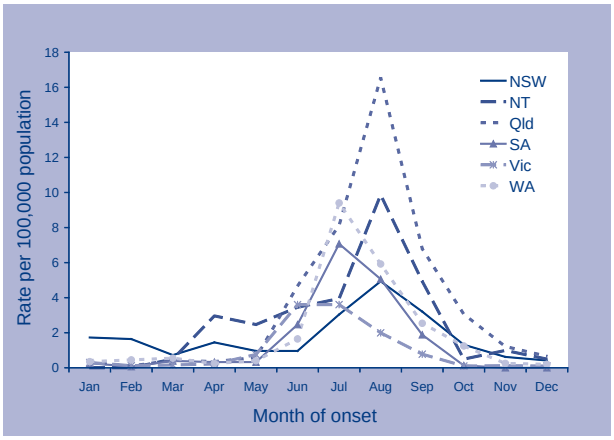
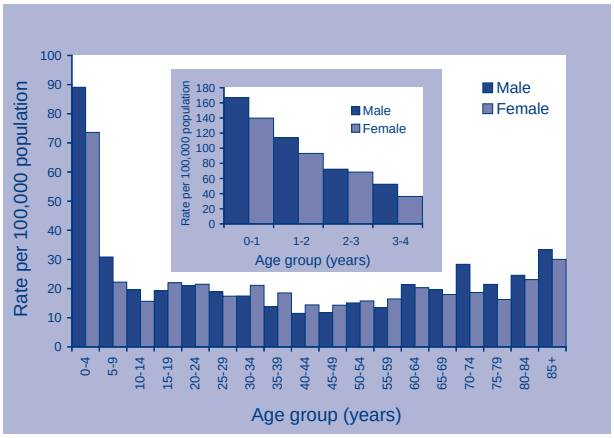


Table 1. Notifications of laboratory-confirmed influenza to the National Notifiable Diseases Surveillance System, Australia, 2003 to 2005, by type

Year	Proportion of notifications (%)				
	Influenza A	Influenza B	Influenza A & B	Unknown	Number of notifications
2005	73.1	22.8	1.4	2.7	4,575
2004	75.8	19.6	1.2	3.4	2,132
2003	93.5	3.6	0.0	2.9	3,483

National age-specific notification rates are shown in Figure 4. The overall male to female ratio was 1:1. The 0–4 year age group had highest notification rate (82 cases per 100,000 population), representing 23 per cent of all notifications.

Figure 4. Notification rates of laboratory-confirmed influenza, NNDSS, Australia, 2005, by age and sex



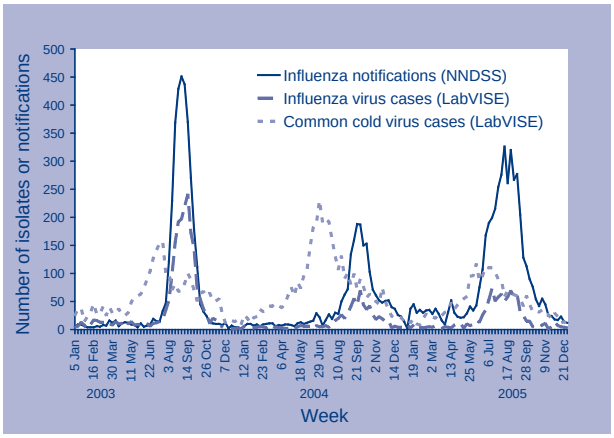
Within the 0–4 year age group, infants under the age of one composed 42 per cent of the notifications and had the highest notification rate at 154 cases per 100,000 population (Figure 4 insert). Notification rates among the 0–4 year age group were higher in 2005 (82 cases per 100,000 population) compared to 2004 (39 cases per 100,000 population). There was no difference between the notification rate in the 0–4 year age group during 2005 compared to the mean rate over the last four years.

Notification rates amongst persons aged over 65 years were higher in 2005 (22 cases per 100,000 population) compared to 2004 (14 per 100,000 population). The influenza notification rate amongst the over 65 year age group during 2005 was 1.7 times the mean rate over the last four years.

Laboratory surveillance

A total of 967 laboratory diagnoses of influenza were reported to LabVISE participating laboratories, which represents a 36 per cent increase from the number of reports in 2004 (Figure 5). In 2005, influenza A was the predominant type reported to LabVISE (73.4%). Reports of influenza to LabVISE started to rise from the middle of June (week 25) a week later than the rise in influenza activity observed in the NNDSS surveillance data. The number of influenza reports to LabVISE reached a plateau from early July until the first week of September.

Figure 5. Influenza and common cold virus cases reported to LabVISE compared to notifications of influenza to NNDSS, 2003 to 2005, by week of report



* LabVISE reports are by week of specimen collection. NNDSS reports are by week of onset.

A further 2,502 laboratory diagnoses of common cold viruses (respiratory syncytial virus, parainfluenza viruses, and rhinoviruses) were reported to LabVISE participating laboratories. Reports of common cold viral isolates to LabVISE started to rise from 13 May (week 15), nine weeks earlier than the rise in influenza activity observed in the NNDSS surveillance data. In 2005, respiratory syncytial virus was the predominant virus reported amongst LabVISE common cold viral isolates (67.0%). Diagnoses of respiratory syncytial virus cases rose from mid-April (week 16) and peaked in mid-June (week 24).

Sentinel general practice surveillance

Australian Sentinel Practice Research Network

The Australian Sentinel Practice Research Network (ASPREN) is a network of general practices, which collect data on ILI. Sentinel general practices contributing to the ASPREN scheme are mostly located in capital cities and larger regional centres on the east coast of Australia. In 2005, an average of 29 general practices reported ILI cases to ASPREN, on an average of 2,996 consultations per week (Table 2). The number of practices reporting to ASPREN and the number of consultations reported on have decreased over the last two years.

During 2005, a rise in reports of influenza-like illness to ASPREN was evident from mid-June (week 24), one week earlier than in 2004 (Figure 6a). In 2005, the peak ILI rate was observed in early August (week 32) at 42 cases per 1,000 consultations, which was over twice the peak rate in 2004.

State and territory general practice influenza surveillance programs

In New South Wales, ILI surveillance usually runs from May to September. In 2005, the season was extended for a further three weeks due to a small increase in influenza B notifications in the first week of October (Figure 6b). On average, ten general practitioners (range 2–15) reported ILI cases from 5 of 8 Area Health Services (Greater Western, Hunter/New England, Sydney South West, South East Sydney/Illawarra, and Sydney West). These sentinel practitioners reported on an average of 1,178 consultations per week (range 304-1,595). Peaks for ILI were evident in the New South Wales ILI data in early July and late August with peak rates of 33 and 42 ILI per 1000 consultations respectively (weeks 31 and 34). These peak rates were higher than the 2004 peak rate of 14 per 1000 consultations, but comparable to the 2003 peak rate of 35 ILI per 1,000 notifications.

Throughout 2005, the Northern Territory Tropical Influenza Surveillance Program received reports of ILI cases from an average of nine sentinel general practices (range 0–14), on an average of 596 consultations per week (range 0–944). The Northern Territory ILI data peaked in mid-June (62 ILI per 1,000 consultations, week 25) with smaller peaks in April and August (Figure 6c). In 2004, peaks were observed in March and July with peak rates of 31 and 27 ILI per 1,000 consultations respectively.

In Queensland, an average of 14 sentinel practices provided data per week (range 10–16), including three practices from northern Queensland and 13 practices from central and southern Queensland. The sentinel practices reported on an average of 2,015 consultations per week (range 1,460–2,349). Queensland's ILI surveillance commenced later in 2005 than in previous seasons due to difficulties recruiting general practitioners. The highest rate (22 ILI per 1,000 consultations) was reported in late August (week 35) (Figure 6d). The peak rate in 2004 was 26 cases of ILI per 1,000 consultations.

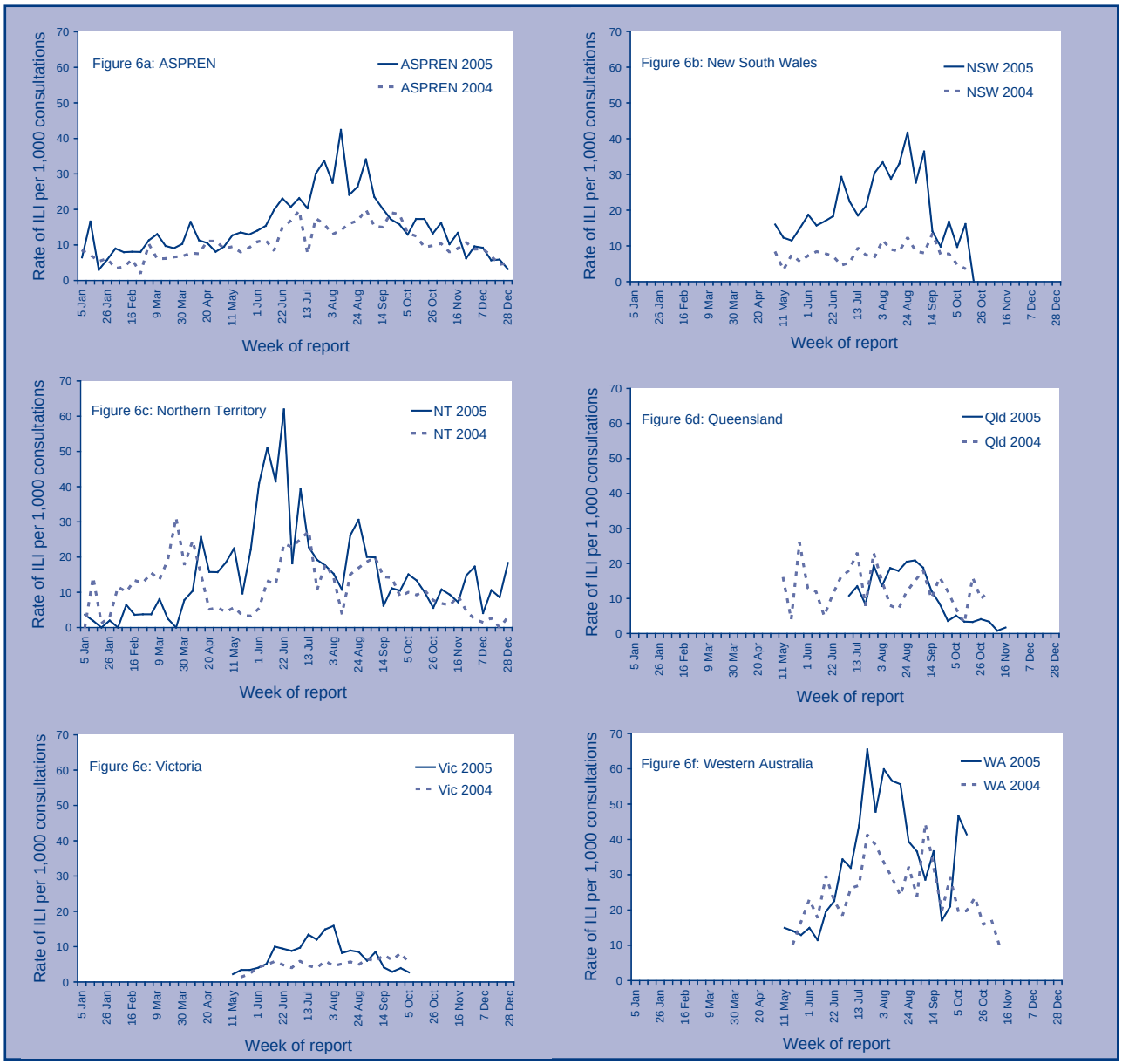
The Victorian Infectious Diseases Reference Laboratory, the WHO Collaborating Centre for Influenza and the Department of Human Services contribute to the Victorian Influenza Surveillance Scheme. On average, 28 general practitioners (range 20–37) were recruited to report ILI rates per 100 consultations per week between May and October. These practitioners reported on an average of 6,282 consultations per week (range 4,066–8,085). In Victoria in 2005, ILI rates started to rise in early June (week 23) and peaked in the first week of August (peak rate 16 cases per 1,000 consultations) (Figure 6e). This was double the peak rate of 2004, reported in late September.

During the 2005 winter season, 20 medical practices were involved in the influenza surveillance program in Western Australia. The majority (16) of the sentinel practices were based in the Perth metropolitan area. Country practices included Kalgoorlie (Goldfields), Busselton (Southwest), Tom Price (Pilbara) and Geraldton (Midwest). On average, 15 practices reported per week (range 10–18) on an average of 2,083 consultations per week (1,029–2,632). In Western Australia, the ILI rate per practice peaked in late July 2005 at 66 cases per 1,000 consultations (Figure 6f). This represents a 48 per cent increase compared to the peak in early September 2004. Influenza-like illness activity started to increase from mid-June 2005 (week 24).

Table 2. Number of general practices reporting influenza -like illness to the Australian Sentinel Practice Research Network, 2003 to 2005

Year	Reporting practices		Consultations	
	Average	Range	Average	Range
2005	29	15–36	2,996	1,081–3,698
2004	31	11–42	3,321	1,224–4,219
2003	48	32–62	4,910	2,138–6,587

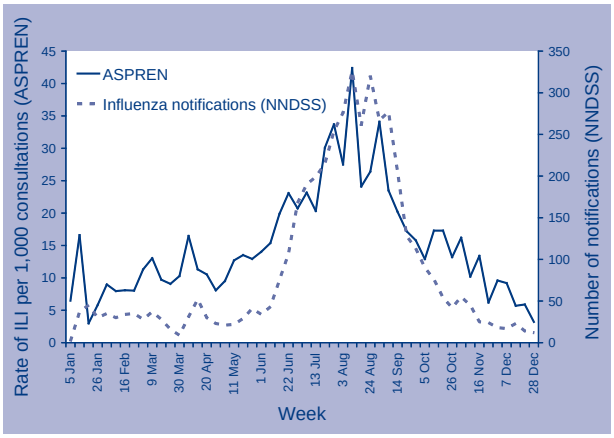
Figure 6a. Consultation rates for influenza-like-illness (ILI), ASPREN, 2003 to 2005, by sentinel surveillance scheme and week of report



A comparison of ASPREN influenza-like illness reports with influenza notification numbers reported to the NNDSS during 2005 is shown in Figure 7. ILI reports to ASPREN rose in mid-June (week 24) at the same time as the rise in influenza notifications to NNDSS. Furthermore, both surveillance systems peaked in the same week in early August (week 32).

As shown in Table 3, the mid-June (week 24) rise in laboratory-confirmed influenza notifications coincided with the rises observed in most ILI surveillance systems. In this report NNDSS notification data are presented by week of onset (diagnosis date), a reflection of actual disease occurrence in the population. When analysed by date of notification to the NNDSS, a reflection of recognition of disease occurrence by the system, epidemic curves shifted 10 days later. Therefore, three ILI surveillance sys-

Figure 7. Comparison of reports of influenza-like-illness to ASPREN with notification numbers from NNDSS, Australia, 2005, by week of report



* ASPREN reports are by week of report. NNDSS reports are by week of onset.

tems (ASPREN, the Victorian Influenza Surveillance Scheme, and Western Australian sentinel general practices) detected the start of the influenza season by 1 to 2 weeks before NNDSS.

Table 3. Comparison of rises and peaks in influenza surveillance system data, Australia, 2005

Surveillance system	Week of first rise	Week of peak
NNDSS		
Laboratory-confirmed influenza notifications*	24	32
LabVISE		
Influenza reports	25	28
All common cold virus reports	15	23
Respiratory syncytial virus reports	16	24
ASPREN		
Influenza-like illness (ILI) reports	24	32
State and territory GP ILI surveillance		
New South Wales	26	34
Northern Territory	15	25
Queensland	30	35
Victoria	23	31
Western Australia	24	29
Australia Post absenteeism data	20	24

* NNDSS notification data presented by week of onset (diagnosis date)

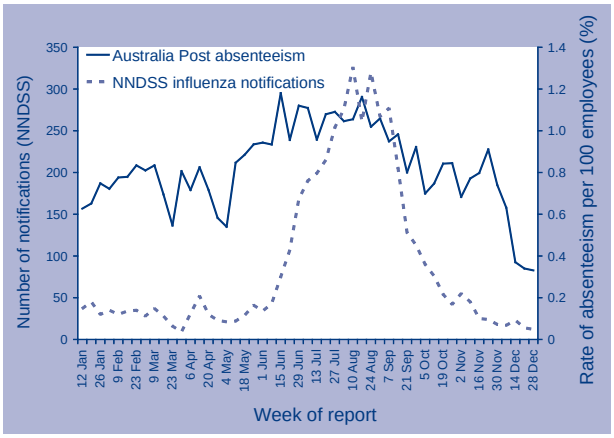
Absenteeism surveillance

Absenteeism surveillance is a non-specific index of influenza activity. In 2005, national absenteeism rates peaked in mid-June (week 24) at 1.2 per cent (n=389); an increase of 44 per cent from the average of 0.8 per cent absentees per week (n=271) during the reporting period. The absenteeism rates began to rise from week 20 (mid-May) and peaked on week 24, preceding the rise and peak influenza notification activity in NNDSS by four and eight weeks respectively (Figure 8).

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centre for Reference and Research on Influenza received 1,174 isolates or clinical specimens from Australian laboratories in 2005 that yielded viable influenza viruses (second only to 2002 for total number of isolates received). These were

Figure 8. Rates of absenteeism and notification rates of influenza, Australia, 2005, by week of report



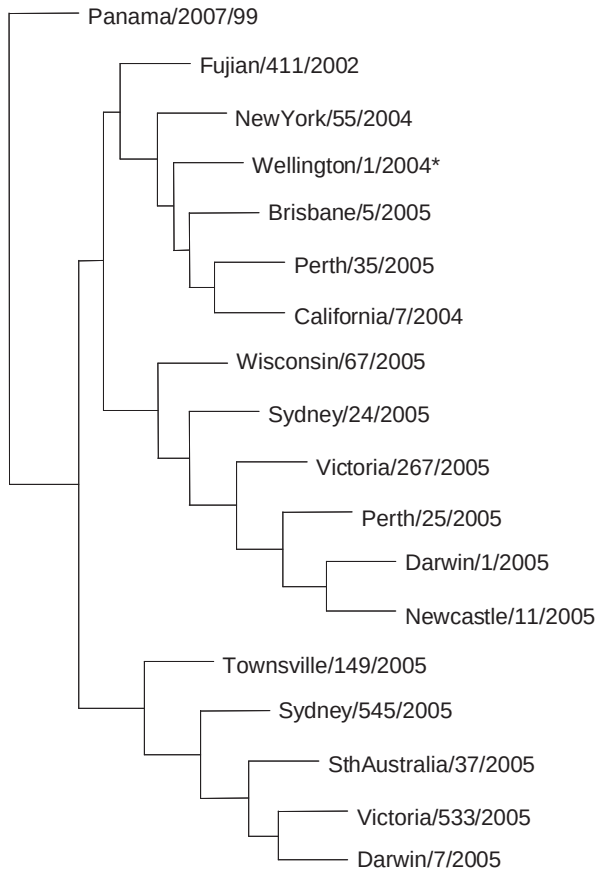
all analysed antigenically using the haemagglutination inhibition assay which identified 689 (58.7%) as A(H3N2) strains, 210 (17.9%) as A(H1N1) strains and 275 (23.4%) as influenza B strains. Sequence analysis of the variable (HA1) region of the haemagglutinin gene was undertaken for 76 strains (41 H3, 16 H1, 29 B) and of the neuraminidase gene for 21 strains (12 H3, 4 H1, 5 B). The 2005 Australian A(H3) viruses were mostly antigenically similar to the 2005 vaccine strain A/Wellington/1/2004 but a proportion of viruses had a reactivity pattern closer to A/California/7/2004-like viruses and a significant proportion of viruses did not match either of these patterns (Table 4).

Genetic analysis of the Australian A(H3) 2005 isolates (Figure 9) showed that most viruses fell into one of three subgroups based on the HA1 domain of the haemagglutinin gene. One group were similar to A/Victoria/533/2005, another were similar to A/California/7/2004 and a third group were similar to A/Darwin/1/2005 (with the latter group increasing in proportion towards the end of the 2005 season).

All of the 210 A(H1) isolates had an N1 type neuraminidase (i.e. A(H1N1)) as determined by an ELISA assay and these mostly remained antigenically close to the reference and vaccine strain A/New Caledonia/20/99. Genetically, most of the A(H1) 2005 Australian viruses fell into one subgroup that was distinguishable from A/New Caledonia/20/99 represented by A/Malaysia/1513/2004 (Figure 10).

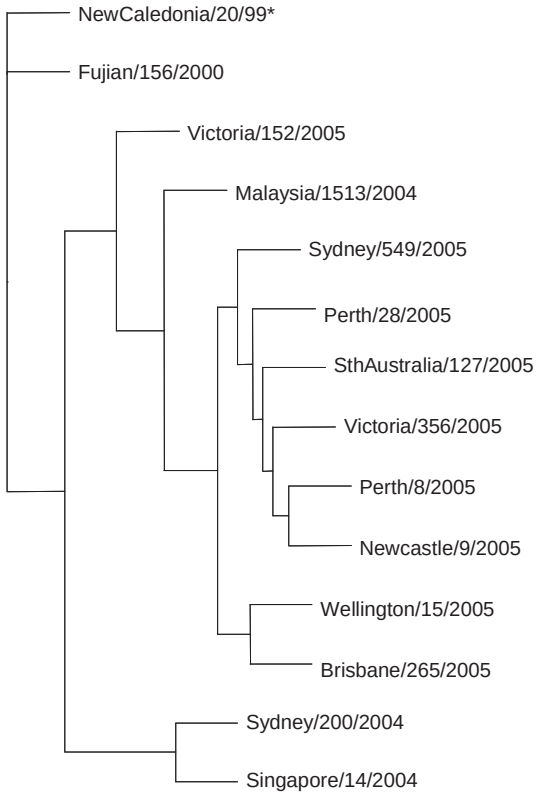
Of the 275 influenza B viruses analysed 51.3 per cent were antigenically and genetically closely related to the reference virus B/Shanghai/361/2002 (B/Yamagata/16/88-lineage) while the remaining 48.7 per cent were B/Hong Kong/330/2001-like or B/Malaysia/2506/2004-like strains (B/Victoria/2/87-lineage) (Figure 11).

Figure 9. Evolutionary relationships between influenza A(H3) haemagglutinins (HA1 region)



*2005 Australian influenza vaccine strain

Figure 10. Evolutionary relationships between influenza A(H1) haemagglutinins (HA1 region)



*2005 Australian influenza vaccine strain

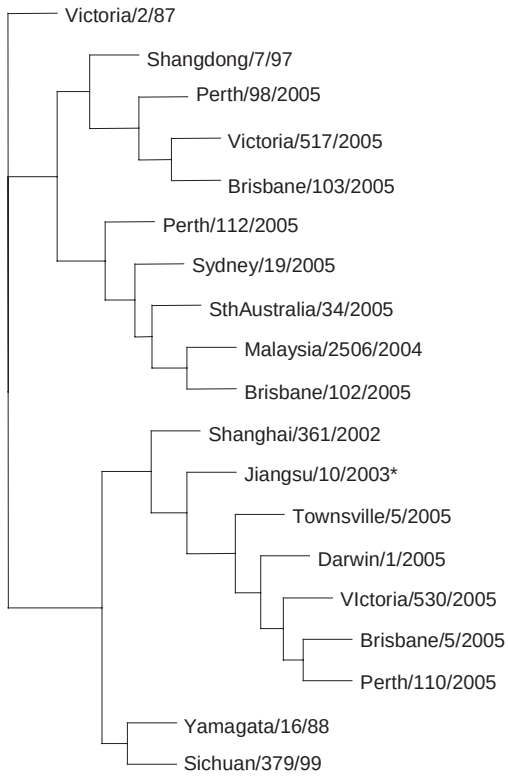
Consistent with the antigenic drift in the A(H3) isolates demonstrated with ferret antisera (Table 4), serological studies conducted with pre– and post-vaccination human sera from recipients of vaccine containing the A/Wellington/1/2004 strain showed a reduction in antibody titres to some 2005 A(H3) isolates. The Australian 2005 vaccine also contained a B/Shanghai/361/2002-like strain (B/Jiangsu/10/2003) which covered the 2005 influenza B isolates from this lineage fairly well but as expected had reduced titres to 2005 B viruses from the alternative lineage (B/Malaysia/2506/2004) which made up the other half of the isolates analysed. As both lineages have co-circulated in the past three years in Australia it has been difficult to predict which lineage will predominate from year to year and this has made selection of the B component of the vaccine formulation problematic in recent years.

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

Virus antigen	Ferret antiserum Reciprocal haemagglutination-inhibition titre		
	A/Fujian	A/Wellington	A/California
A/Fujian/411/2002	**160**	640	160
A/Wellington/1/2004*	320	**1,280**	1,280
A/California/7/2004	640	640	**1,280**
A/Brisbane/85/2005	40	40	160
A/Townsville/107/2005	320	1280	160
A/Sth/Aust/98/2005	160	640	80
A/Victoria/216/2005	320	640	640
A/Brisbane/49/2005	40	80	80

* A/Wellington/1/2004 strain was used in the 2005 vaccine.

Figure 11. Evolutionary relationships between influenza B haemagglutinins (HA1 region)



*2005 Australian influenza vaccine strain

International trends in influenza, 2005

During the period February to September 2005, influenza A(H1N1), A(H3N2) and B viruses circulated in many parts of the world. In the Northern Hemisphere, influenza A(H3N2) viruses predominated and caused most outbreaks including a severe and long outbreak in China. Influenza A(H1N1) viruses caused outbreaks in Eastern Europe and Asia between February and April. Influenza B viruses caused outbreaks in several countries in Africa, Asia and eastern Europe.

In the Southern Hemisphere, influenza activity began in April and increased during May in Oceania, and during June in South America. In Oceania and South America, influenza A(H3N2) and B viruses co-circulated and caused several outbreaks including an epidemic of influenza B in New Zealand. Influenza A(H1N1) also circulated at low levels in some countries.

The WHO annual consultation on the composition of influenza vaccines for the Southern Hemisphere, 2006 took place in Malta from 10 to 15 September 2005. The recommended composition of influenza virus vaccines for use in the 2006 Southern Hemisphere influenza season was:

— an A/New Caledonia/20/1999(H1N1)-like virus;

— an A/California/7/2004(H3N2)-like virus;

—a B/Malaysia/2506/2004-like virus.

This recommendation included two changes to the previous Southern Hemisphere vaccine for the 2005 influenza season, with the addition of a new A(H3) virus (replacing A/Wellington/1/2004 with A/California/7/2004 – the actual vaccine strain used will be A/New York/55/2004) and a new B virus (replacing B/Jiangsu/10/2003, a B/Yamagata-lineage virus with B/Malaysia/2506/2004, a B/Victoria lineage virus).

There were widespread outbreaks of A(H5N1) HPAI influenza in chickens, ducks and other birds throughout South East Asia in 2005. According to the official WHO figures 95 human infections with H5N1 occurred in five countries resulting in 41 deaths. No H5N1 infections were detected in birds or humans in Australia in 2005.

Whilst the temporal pattern of influenza in New Zealand is broadly similar to that in Australia, outbreaks often begin earlier. The New Zealand consultation rates for ILI started to increase in mid-May, a few weeks earlier than in 2003, and peaked in week 27 in late July. During the 2005 winter, New Zealand experienced large outbreaks of influenza type B in school-aged children. This epidemic was associated with significant morbidity with some schools in Auckland, and Wellington experiencing student absenteeism rates in excess of 20 per cent. During this epidemic, three children died suddenly from complications of infection with influenza B/Hong Kong/330/2001 (B/Victoria/2/87-lineage). According to WHO recommendations a B/Yamagata/16/88-lineage strain was included in the 2005 vaccine for Australia and New Zealand, in place of a B/Hong Kong/331/2001-like strain.

The WHO Influenza Centre typed 770 isolates from New Zealand in 2005. Of these the majority were B strains (87.6%) and were mainly of the B/Victoria/2/87-lineage (80%) with a minority (20%) being of the B/Yamagata/16/88-lineage. Of the remaining viruses analysed, 72 were A(H3) (9.2%) and 14 A(H1) (0.2%). Overall, influenza activity in New Zealand in 2005 was average and similar to 2003 but above the levels seen in the 2004 season (a full report on the 2005 influenza season in New Zealand is available from: http://www.surv.esr.cri.nz/PDF_surveillance/Virology/FluAnnRpt/InfluenzaAnn2005.pdf).

Discussion

The 2005 influenza season in Australia started earlier and was substantially larger than in 2004. Compared with recent seasons, the 2005 influenza activity was higher than normal, relatively early, and sustained in that it lasted for around four months. Influenza A was again the predominant type isolated in Australia, however, the proportion of diagnoses of influenza B increased for the third successive year.

The rise in reports of common cold viruses to LabVISE preceded the start of the influenza season by nine weeks. Respiratory syncytial virus was the predominant viral isolate amongst these common cold virus reports, and the rise in RSV reports preceded the influenza season by eight weeks. The LabVISE common cold data coincided with the rise observed in absenteeism rates during 2005 reported by Australia Post.

The majority of the Australian isolates (58.7%) analysed at the WHO Influenza Centre were A(H3N2) strains and a significant number of strains showed a degree of heterogeneity based on their antigenic and genetic characteristics. Many strains were antigenically closely matched with A/Wellington/1/2004 while others appeared to be more closely matched to A/California/7/2004-like viruses and a third group reacted poorly with reagents prepared against both of these viral lineages. These sub-groupings could also be seen from the haemagglutinin gene sequence analysis and an increasing proportion of low-reacting strains had the characteristic amino acid changes in the HA1 gene of S193F and D225N from other strains.

In Australia, like many other countries in 2005, the two lineages of influenza B viruses (B/Victoria/2/87 and B/Yamagata/16/88-) co-circulated. An almost even proportion of the two lineages circulated in Australia unlike in New Zealand where approximately 80 per cent of all B strains isolated were of the B/Victoria-lineage.

Influenza A(H1) viruses began to re-emerge in Australia in 2005 after very few viruses were isolated in the previous three years, and made up some 17.8 per cent of total influenza viruses. Interestingly while some genetic changes were evident in the circulating A(H1) strains, little change was seen in their antigenic characteristics when compared to the reference and vaccine A(H1N1) strain A/New Caledonia/20/99.

During 2005, notable influenza outbreaks occurred in New South Wales. Four of the New South Wales outbreaks were reported from aged care facilities and one outbreak from a school. Influenza B was isolated from patients in the first outbreak (in July) and influenza A in the remaining four outbreaks that occurred between August and September. Antiviral drugs (Oseltamvir) were used to control the outbreaks in the aged care facilities, and appeared to be successful. No deaths associated with these outbreaks were reported.

The rate of notifications amongst persons aged over 65 years was the highest recorded in the last five years. The vaccine coverage in this age group in 2005 was unknown. Limited influenza vaccine supply during 2005 may explain the higher than expected notification rates amongst this age group. Amongst vaccinated over 65-year-olds, vaccine effectiveness was expected to be high with regard to circulating influenza A strains, but less so for circulating influenza B strains. However, this age group had a lower proportion of influenza type B infections (18.8%) compared to the rest of the population (24.2%). Vaccine effectiveness may only explain a small proportion of the observed increase in influenza in the over 65 year age group during 2005. When available, the Australian Institute of Health and Welfare data on deaths due to influenza and pneumonia amongst the over 65 year age group during 2005 will clarify whether increased influenza infection rates led to increased mortality.

Preparation for an influenza pandemic is presently a high priority in Australia. Outbreaks of avian influenza ('bird flu') amongst poultry in neighbouring Asian countries, Europe and Africa during 2004 and 2005 have heightened the likelihood of an influenza pandemic occurring. In a pandemic, the priority groups and the timing of vaccination may be quite different from those during inter-pandemic periods. In addition, the number of vaccine doses and the optimal time for vaccination may differ. In June 2005, the National Influenza Pandemic Action Committee (NIPAC) released *The Australian Management Plan for Pandemic Influenza* (http://www.health.gov.au/internet/wcms/publishing.nsf/Content/phd-pandemic-influenza.htm). NIPAC has included guidelines for vaccine use during inter-pandemic and pandemic periods within the management plan (Section 3 and Annex 3), and will advise health authorities regarding priority groups, dosing schedules and timing of vaccination should a pandemic occur. The Australian Management Plan for Pandemic Influenza is expected to be revised during 2006.

Online references for influenza

Australian Government Department of Health and Ageing

National Influenza Surveillance Scheme: http://www.health.gov.au/internet/wcms/publishing.nsf/Content/Influenza-1

Pandemic influenza information: http://www.health.gov.au/internet/wcms/publishing.nsf/Content/phd-pandemic-influenza.htm

Influenza-like illness surveillance in Australia

NSW Health influenza surveillance report (May–October): http://www.health.nsw.gov.au/infect/diseases.html#I

South Australian information for general practitioners on influenza and RSV diagnoses: http://www.dh.sa.gov.au/pehs/notifiable-diseases-summary/flu-resp-intro.htm

Influenza data from New Zealand compiled by ESR Kenepuru Science Centre: http://www.surv.esr.cri.nz/virology/influenza_weekly_update.php

Additional Australian influenza data and information can be accessed at:

Melbourne WHO Collaborating Centre for Influenza: http://www.influenzacentre.org

CSL Limited influenza information website: http://www.flu.com.au/

National Institute of Clinical Sciences (NICS) influenza website: http://www.fightflu.com.au/

The Victorian Influenza Surveillance Scheme publishes fortnightly reports (May to October), available at: http://www.vidrl.org.au

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Augmentation of influenza surveillance with rapid antigen detection at the point-of-care: results of a pilot study in Tasmania, 2004

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Abstract

Tasmania contributes very few laboratory confirmed cases to Australia's national influenza surveillance statistics. In 2004, a study was conducted to pilot test sentinel syndromic surveillance for influenza-like illness supplemented by point-of-care testing using the Binax Now Flu A Test Kit and by viral culture, to assess the feasibility and acceptability of this method of surveillance. Overall, the goal of such a system would be to increase laboratory surveillance activity within Tasmania and increase the number of specimens sent for viral culture. Five sites participated in the study, including three public hospital emergency departments and two general practices. Despite being conducted during a period of low influenza activity, the pilot study demonstrated that augmentation of syndromic surveillance with point-of-care testing is both feasible and acceptable but is best conducted in the general practice setting. *Commun Dis Intell* 2006;30:201–204.

Keywords: influenza; laboratory diagnosis

Introduction

Influenza is a highly contagious, febrile, acute respiratory disease in humans. The incidence of influenza is estimated at 500 million cases annually, or one in every three of the world's population per year.[1] A definitive diagnosis of influenza requires laboratory confirmation, since clinical diagnosis on the basis of clinical symptoms is not sensitive and the predictive value of clinical diagnosis of influenza in the absence of an epidemic is only 30–40 per cent.[2]

Diagnostic tests for influenza fall into four categories: virus isolation; detection of viral nucleic acid; detection of viral proteins; and serological diagnosis. Viral isolation i.e. culture, is the current gold standard for laboratory diagnosis. Detection of viral nucleic acid is widely used for typing and subtyping influenza viruses. The advantage of nucleic acid amplification tests (NAAT) is their high sensitivity and specificity, more rapid turn-around time, an expanded range of specimen types suitable for testing, and the ability to detect viruses that are difficult to grow in cell culture.[3,4] However, NAAT is not universally available and significant time delays in centres with poor access to influenza NAAT-capable laboratories preclude its clinical usefulness, reducing the number of tests clinicians order.

Tests that detect viral proteins at the point-of-care are becoming more common. They are easy to perform and results are available in less than an hour. However, they are considerably less sensitive than culture or NAAT.[5] A positive test is useful, for both directing initiation of therapy in the clinician's office and making a positive diagnosis of influenza in patients with influenza-like clinical syndromes.[6] As the technology is continuing to advance, the test sensitivity is likely to improve.

In Tasmania, laboratories have very limited diagnostic capability for influenza. Specimens for culture, serology and NAAT are all sent interstate for processing. This creates substantial time delays before results become available. As a consequence, testing rates by Tasmanian clinicians for influenza are very low and Tasmania contributes very few laboratory confirmed cases to Australia's national influenza surveillance statistics.

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Public health surveillance for influenza is necessary to determine the distribution of illness, detect outbreaks, monitor changes in disease agents and facilitate planning.[7] Increasingly, non-traditional methods of surveillance are being utilised. Point-of-care testing trials for influenza have been conducted in Hawaii and Germany. These trials have demonstrated that integrating influenza rapid antigen testing into public health surveillance, by coupling rapid tests with cultures, improves testing rates by clinicians (as an immediate result is provided by the point-of-care test) and enhances influenza surveillance (as the culture specimen allows the reference centres to characterise circulating viral strains and to detect fully new variants).[8,2]

The aims of this study were to pilot test sentinel syndromic surveillance for influenza-like illness (ILI) in Tasmania, supplemented by point-of-care testing using the Binax Now Flu A Test Kit and by viral culture, to assess the feasibility and acceptability of this method of surveillance.

Methods

Surveillance was conducted between May and October 2004. The case definition adopted for ILI was: presentation with cough, fever (greater than or equal to 37.5 degrees Celsius) and fatigue.[9]

Five sites were recruited to the project. These were the Departments of Emergency Medicine (DEM) of the Royal Hobart Hospital, Launceston General Hospital and North West Regional Hospital, East Devonport Medical Centre and the After Hours Doctors Service at Derwent Park. The sites were selected to represent the geographical transport entry points into Tasmania. The Royal Hobart Hospital and After Hours Doctors Service are both in Hobart, in southern Tasmania. Domestic flights enter Hobart from Sydney, Brisbane and Melbourne. Cruise ships dock in Hobart, arriving from multiple international destinations. The Launceston General Hospital is in Launceston in northern Tasmania. Domestic flights from Sydney and Melbourne enter Launceston. The North West Regional Hospital is in Burnie in north-west Tasmania. The City Medical Practice is in Devonport, also in north-west Tasmania. East Devonport is the main entry point for domestic passenger ships arriving by sea into Tasmania.

A site co-ordinator was nominated for each sentinel site. Staff from the Tasmanian Department of Health and Human Services Communicable Diseases Prevention Unit (DHHS-CDPU) briefed the coordinator on the project and requirements for reporting. Medical staff at each site were trained in the use of the Binax Now Flu A Test Kit. They were briefed on the case definition for influenza and the reporting requirements for each patient in the study. The information collected included: the surveillance period dates, the number of patients in each reporting period meeting the case definition for ILI, the sex and age of each case, and whether or not the case had been vaccinated against influenza in 2004.

Data were entered into an Excel spreadsheet and descriptive statistical analysis undertaken using Excel 2000. At the conclusion of the project DHHS-CDPU staff administered purpose-designed surveys to each of the site coordinators, seeking information regarding the feasibility and acceptability of the surveillance system, and the clinical utility and ease of use of the point-of-care test.

Results

Influenza-like illness (ILI)

During the surveillance period, reports were received from all sites as per the reporting requirements of the study.

From all sites there was a total of 53 patients satisfying the study criteria for the clinical diagnosis of ILI, as defined by the case definition above. Of these, 40 (76%) were tested with the Binax Now Flu A Test Kit. No positive results for influenza were obtained using the Binax Now Flu A Test Kit and no clinical specimens were sent for culture.

Influenza vaccination status was recorded for 30 of the 53 cases of ILI (56%). Of these, 7 (23%) were recorded as having received influenza vaccine in 2004.

Survey results

Survey results were received from 3 of the 5 participating sites (response rate = 60%).

Procedural aspects of the surveillance method

The written information supplied to each site was considered adequate for the purposes of the project and available public health support was sufficient. Respondents indicated that the case definition of ILI was simple and easy to apply for the purposes of surveillance and to indicate which patients should receive a point-of-care test. The availability of a rapid test for influenza was regarded as an incentive to test patients. However, respondents did indicate that incentives for their involvement in surveillance activities would improve their participation and that without incentives it was unlikely they would continue to act as sentinels.

Feasibility and acceptability of the point-of-care test

The majority of respondents considered the Binax Now Flu A Test Kit easy to use. However, the time taken before results could be read (15 minutes) was an inconvenience in both DEM and general practice settings. Within the DEM it was not feasible to ensure that the quality of nasal specimens collected by different staff members remained of a consistently high quality. This was not identified as an issue in the general practice setting.

Discussion

Influenza activity in Australia during 2004 was low. A total of 2,116 cases of laboratory-confirmed influenza were reported via the National Influenza Surveillance Scheme, which was 41 per cent lower than the number of reported cases in 2003.[10] In Tasmania, there were two notified cases of laboratory-confirmed influenza A and one case of laboratory-confirmed influenza B during 2004. However, because ILI was present, pilot testing of sentinel surveillance could still be undertaken. In general, participants viewed the surveillance system favourably as an appropriate method of influenza surveillance in Tasmania.

There were multiple barriers to conducting surveillance for influenza in the DEM setting. It was not possible to ensure that all staff-members were identifying and recording patients who met the case definition of ILI or that testing for influenza A of all patients who met the case definition was occurring. Additionally, it was not possible to identify whether the staff-members using the Binax Now Flu A Test Kit were using the test or taking nasopharyngeal samples appropriately. It was felt that staff did not have the time to undertake sufficient education or training to ensure that the testing protocol was reliable between users. After the sample was acquired, it took 15 minutes before the result could be read. In a busy emergency department, this was seen as a barrier to the test's use. Staff did identify that lack of remuneration was a barrier for undertaking surveillance activities.

Surveillance in general practice was more acceptable and feasible. Staff could be appropriately trained and educated on the use of the test in this setting. The time taken before the result could be read was seen as only a small additional time burden. Case ascertainment was more complete and the protocols for the study were more rigorously applied. However, general practitioners also identified that the lack of remuneration for their participation was a barrier – particularly as the presence or absence of influenza did not necessarily alter their patient management.

Workforce capacity within the DHHS-CDPU was also an issue identified in this study. Frequent and regular communication with sentinel sites was necessary to ensure compliance with data reporting requirements. Data collection and analysis were also labour intensive. The workforce requirements within the DHHS-CDPU were significantly underestimated and future surveillance using this method will need to address this.

There are significant limitations with the point-of-care test itself. The sensitivity of the point-of-care test is only 65–77 per cent compared with culture or NAAT testing for known circulating influenza strains and is probably much lower for pandemic strains (point-of-care tests are unlikely to detect pandemic strains of influenza). However, in spite of this, clinical trials have demonstrated that a positive test is clinically useful and by coupling the point-of-care test with viral culture, a sufficiently sensitive test is utilised that is capable of detecting pandemic strains.[6] Many viruses cause ILI in patients and are not generally distinguishable from influenza on clinical grounds alone. These include respiratory syncytial virus, rhinovirus, human corona viruses, human metapneumovirus, adenovirus, picornavirus and parainfluenza virus.[3] Antigen detection is not useful for differentiating between these viruses.

The pilot study demonstrated that augmentation of syndromic surveillance with point-of-care testing is both feasible and acceptable. However, the setting in which this form of surveillance is best conducted is general practice. The procedural barriers within the DEM setting were far greater and the model can be more efficiently applied within the general practice setting, without sacrificing breadth of surveillance.

To adopt this form of surveillance within Tasmania, selected general practitioners could be targeted. Financial remuneration for service providers would almost certainly be necessary. Teams at each participating general practice (including practice nurses, practice managers and general practitioners) could be formed to conduct sentinel surveillance. This would ensure that continuity of surveillance is maintained and would improve data collection, timely reporting and minimise the time burden on the general practitioner by efficiently utilising practice support staff.

It is also foreseeable that this method of surveillance could be conducted by health personnel via Community Assessment and Information Centres that would become operational in the Tasmanian response to a pandemic influenza threat.

Overall, the goal of such a system would be to increase laboratory surveillance activity within Tasmania and increase the number of specimens sent for viral cul-

ture. This would establish an improved and enhanced method of influenza surveillance in Tasmania, which is an important inter-pandemic priority.

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Annual report of the Australian Gonococcal Surveillance Programme, 2005

Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme monitors the antibiotic susceptibility of *Neisseria gonorrhoeae* isolated in all States and Territories. In 2005 the *in vitro* susceptibility of 3,886 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 29.5 per cent and, with the exception of the Northern Territory, ranged between 14 and 47 per cent. Quinolone resistance in gonococci increased with resistance to this agent found in all jurisdictions and in a larger proportion of strains and with higher minimal inhibitory concentrations (MICs). Nationally, 30.6 per cent of all isolates were ciprofloxacin-resistant and most of this resistance was at high MIC levels. All isolates remained sensitive to spectinomycin. Slightly more than one per cent of isolates showed some decreased susceptibility to ceftriaxone (MIC 0.06 mg/L or more). 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* 2006;30:205–210.

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

Introduction

Antimicrobial resistance (AMR) surveillance in *Neisseria gonorrhoeae* has been undertaken by the Australian Gonococcal Surveillance Programme (AGSP) continuously since 1981 to provide data regarding the most reliable therapy for individual and programmatic treatment of gonococcal disease.[1,2] There has been a continuing and increasing problem of AMR in gonococci in Australia,[2,3] and this has compromised disease control efforts and necessitated changes to use of more expensive and/or injectable antibiotics. Standardised treatment regimens for gonorrhoea utilise single dose treatments that seek to cure 95 per cent or more of cases.[4] Where treatment with oral agents such as the penicillins and quinolones is retained, continuous monitoring is required to ensure their ongoing effectiveness. There are also increasing numbers of reports of gonococcal isolates showing resistance to multiple antibiotics including decreased susceptibility to the third generation cephalosporin ceftriaxone which is used extensively in Australia.[3,5–9] This analysis of AMR in *N. gonorrhoeae* in Australia is derived from data generated by the AGSP during the 2005 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 (laboratories are listed in the 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.[1,10] The AGSP also conducted a program-specific quality assurance (QA) program.[11] Antibiotic sensitivity data were submitted quarterly to a coordinating laboratory which collated the results

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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

Number of isolates

There were 3,980 gonococcal isolates referred to, or else isolated in AGSP laboratories in 2005, about nine per cent more than the 3,640 examined in 2004. The source and site of infection of these isolates are shown in Table 1. One thousand two hundred and eighteen gonococci (30.6% of the Australian total) were isolated in New South Wales, 837 (21%) in Victoria, 651 (16.4%) in Queensland, 646 (16.2%) in the Northern Territory, 403 (10%) in Western Australia, and 180 (4.5%) in South Australia with small numbers in Tasmania (23) and the Australian Capital Territory (22). Three thousand eight hundred and eighty-six isolates remained viable for susceptibility testing.

The increase in numbers of gonococci nationally (340, 9%) was mainly the result of more isolates from the Northern Territory (increased by 131), New South Wales (105) and Western Australia (73) with smaller increases in Queensland and South Australia with a slight decrease in numbers in Victoria. Numbers in Tasmania and the Australian Capital Territory, although small, approximated those obtained in 2004.

Source of isolates

There were 3,288 strains from men and 688 from women, with a male to female (M:F) ratio of 4.7:1, less than the 5.5:1 ratio for 2004. The number of strains from men increased by 111 and there were 129 more from women. Although lower than in 2004, the M:F ratio was again high in New South Wales (11.6:1) and Victoria (8.8:1) where strains were more often obtained from urban populations. The lower ratios in Queensland (3.6:1) Western Australia (4:1) and the Northern Territory (1.7:1) reflected the large non-urban component of gonococcal disease in those regions. Male rectal and pharyngeal isolates were most frequently found in Victoria (23% of isolates from men), New South Wales (36%) and South Australia (29%). These percentages are higher than those recorded in New South Wales and South Australia in 2004 and lower in Victoria, but also may reflect clinical sampling practices in those States. About 3.6 per cent of isolates are shown as being isolated from 'other' or unknown sites. These included nine cases of disseminated gonococcal infection in men (0.3%) and 10 (1.5%) in 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 a small number of isolates from the eyes (10) of both newborn and older infants and also adults, and from Bartholin's abscesses in women.

Table 1. Source and number of gonococcal isolates, Australia, 2005, by sex, site and state or territory

		State or territory						
	Site	NSW	NT	Qld	SA	Vic	WA	Aust
Male	Urethra	665	379	441	90	569	302	2,476
	Rectal	238	4	41	24	105	12	429
	Pharynx	171	1	15	13	70	6	282
	Other/NS	48	26	14	0	8	3	101
	Total	1,122	410	511	127	752	323	3,288
Female	Cervix	90	219	133	51	75	72	642
	Other/NS	5	15	7	1	10	8	46
	Total	95	234	140	52	85	80	688
Unknown	Total	1	2	0	1	0	0	4
Total*		1,218	646	651	180	837	403	3,980

* Includes isolates from Tasmania (23) and the Australian Capital Territory (22).

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

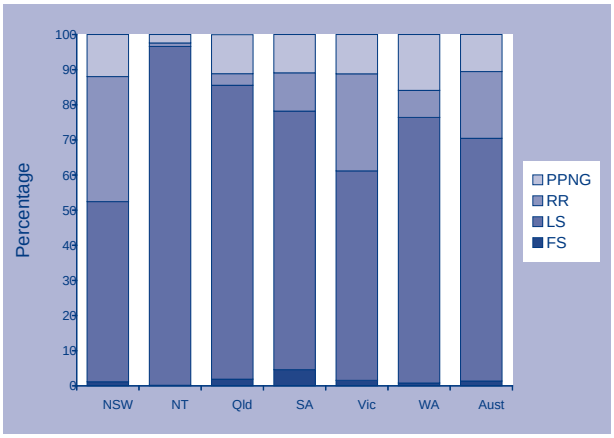
Antibiotic susceptibility patterns

In 2005 the AGSP reference laboratories examined 3,886 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 2005 by penicillin minimal 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 (CMRNG).[12] Chromosomal resistance is defined by an MIC to penicillin of 1 mg/L or more.[1,10] (The minimal inhibitory concentration in mg/L is the least amount of antibiotic which inhibits *in vitro* growth under defined conditions.) Infections with gonococci classified as fully sensitive (FS, MIC ≤ 0.03 mg/L), or less sensitive (LS, MIC 0.06–0.5 mg/L) would be expected to respond to standard penicillin treatments, although response to treatment may vary at different anatomical sites.

Figure 1. Penicillin resistance of gonococcal isolates, Australia, 2005, 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, 1,148 (29.5%) gonococci were penicillin-resistant by one or more mechanisms in 2005, an increase on the 770 (21.7%) resistant to this group of antibiotics in 2004. Of these, 738 (19%) were CMRNG, almost twice as many as in 2004 (377, 10.6%). This increase was accounted for mainly by an increase in CMRNG in New South Wales from 130 in 2004 to 432 in 2005. Nationally, another 410 (10.5%) were PPNG, the number and proportion of which was little different from 2004 (393, 11.1%). In 2004, there had been more PPNG than in 2003 while numbers of CMRNG had remained unchanged in that period.

Penicillin-resistant gonococci were particularly high as a proportion of gonococcal isolates in New South Wales (47.6%; PPNG 12%, CMRNG 35.6%), Victoria (38.6%; PPNG 11.2%, CMRNG 27.6%), Western Australia (23.6%; PPNG 15.9%, CMRNG 7.7%), and South Australia (21.8%, with equal proportions of PPNG and CMRNG). In Queensland, penicillin resistance was also high at 14.4 per cent with 11.1 per cent PPNG. Seven PPNG and one CMRMG were identified in the Australian Capital Territory and in Tasmania there was one PPNG and one CMRNG. In the Northern Territory there were 15 PPNG and six CMRNG resulting in 3.4 per cent of strains that were penicillin resistant (4.2% in 2004). Data on acquisition were available for 128 (31%) infections with PPNG. Sixty-six infections with PPNG were acquired locally and 62 by overseas contact. These contacts were principally in Western Pacific or South East Asian countries including China, Fiji, India, Indonesia (Bali), Malaysia, the Philippines, Thailand, and Vietnam but also through contact in Europe (Germany, Italy, Spain and the United Kingdom), and South Africa.

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 and 24 (0.7%) in 2004. In 2005, there were 48 (1.2%) gonococci with ceftriaxone MICs in the range 0.06 to 0.25 mg/L. Thirty-seven of these were present in New South Wales (3% of isolates there), six (0.7%) in Victoria, and five (0.8%) in Queensland. Forty-five of these 48 gonococci also displayed high-level quinolone resistance and, with the exceptions of one penicillin sensitive isolate and two PPNG, all were also CMRNG.

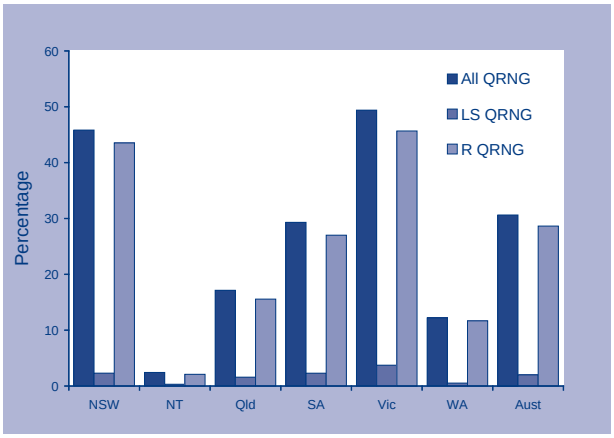
Spectinomycin

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 resistance as an MIC of 0.06 mg/L or more.[10] 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 per cent 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 per cent.

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, 2005, by state or territory



LS QRNG MIC 0.06–0.5 mg/L.

R QRNG MIC ≥ 1 mg/L.

Nationally in 2005, 1,190 (30.6%) gonococci had some level of resistance to quinolones (QRNG), a further increase over the 825 (23.3%) QRNG detected throughout Australia in 2004. In 2003, a total of 529 (14.4%) were QRNG and in 2002 there were 389 (10%) QRNG. Most of the QRNG (1,113, 93.5%) had resistance at a higher level i.e. MICs ≥ 1 mg/L. A similar proportion had higher-level resistance in 2004.

The highest proportion of QRNG was seen in Victoria where the 411 QRNG were 49.4 per cent of the total number examined. This was a further substantial increase in both the number (309) and proportion (36%) of QRNG seen in Victoria in 2004. There was also a considerable increase in QRNG in New South Wales where 555 (45.8%) QRNG were detected in 2005 compared to 331 (30%) in 2004. There were 51 (29.3%) QRNG detected in South Australia (36, 24% in 2004) and 108 (17.1%) in Queensland (103, 16.7% in 2004). In Western Australia, QRNG numbers increased slightly to 46 (12.2%) from the 30 (9.4%) in the previous year, and in other jurisdictions the numbers of QRNG remained low (Northern Territory 15; Tasmania 1; Australian Capital Territory 3).

Information on acquisition of QRNG was available in 377 of the 1,190 cases (31%). Three hundred and eighteen of these (84%) were acquired locally and 59 (16%) overseas from sources referred to under PPNG acquisition with contacts also reported in Canada, Hong Kong, Korea and the United States of America.

High-level tetracycline resistance

The spread of high-level tetracycline resistance in *N. gonorrhoeae* 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 per cent 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 per cent of all gonococci. This proportion was unchanged in 2005 when 534 TRNG were detected.

TRNG were present in all state and territories with the highest proportion in Victoria (201 TRNG, 24.2%) and Western Australia (78, 20.7%). Lower proportions of TRNG were present in South Australia (21, 12%), Queensland (74, 11.7%) and New South Wales (138, 11.4%). Lower numbers were found in the Northern Territory (17), Tasmania (4) and the Australian Capital Territory (1).

Discussion

Further notable and important changes in the susceptibility of *N. gonorrhoeae* to antibiotics used for the treatment of gonorrhoea occurred in 2005.

Penicillin resistance is of historic interest only in many parts of Australia, but is of considerable relevance in some non-urban settings with high disease rates where antibiotics of this group remain a cheap, effective and acceptable treatment. Of note was the significant increase in resistance medi-

ated by chromosomal mechanisms in New South Wales where both there and in Victoria CMRNG contributed markedly to the historically high levels of penicillin resistance recorded in this report. In other jurisdictions PPNG were a more prominent cause of penicillin resistance. Although incomplete, data on acquisition of PPNG recorded in 2005 show the continuing influence of resistant gonococci introduced into Australia from overseas and the wide diversity of the country of origin of these imported resistant strains.

Similarly, use of the quinolone group of antibiotics has been discontinued for the treatment of gonorrhoea in many parts of Australia for quite some time because of high levels of resistance. Quinolone resistance too is concentrated mainly in larger urban areas. Almost half of the isolates from Victoria and New South Wales and about a third of all gonococci nationally were resistant to ciprofloxacin and at MIC levels that have also increased substantially in recent years. Again however there were considerable jurisdictional differences in rates of quinolone resistance with the Northern Territory having a low (2.4%) proportion of quinolone resistant gonococci. Of particular relevance is the high rate of sustained endemic transmission of QRNG within Australia. QRNG are also widely distributed in countries close to Australia[9] and antibiotics other than quinolones should be used for gonococcal infection acquired outside the country.

The number of gonococci with decreased susceptibility to ceftriaxone increased again in 2005. Although the number of these isolates remains low at about one per cent of all isolates tested, these strains almost always also exhibit high-level resistance to quinolones and penicillins. These findings are consistent with recent Japanese data that suggests that these strains are increasingly prevalent there, are multi-resistant and on occasion are associated with treatment failure with oral third generation agents not available in this country.[5,6,8] Ceftriaxone is the third generation cephalosporin most used for treatment of gonorrhoea in Australia. The recommended dose of ceftriaxone for uncomplicated mucosal infection in Australia is 250 mg intramuscularly, higher than that in some other treatment schedules in place overseas. This local recommendation for a higher dose treatment would appear to be prudent given the increase in MICs to third generation cephalosporins observed in gonococci in Australia and in nearby countries. It is emphasised that to date there has been no instance of failure with ceftriaxone treatment attributable to decreased susceptibility described in Australia. All gonococci tested in Australia in 2005, including those with altered cephalosporin susceptibility, were susceptible to spectinomycin.

Surveillance of antimicrobial resistance is an essential component of local, regional and international efforts for control of gonorrhoea. Standard treatment guidelines can be reliably based on the results of properly conducted surveillance of antimicrobial resistance, but this in turn requires that the data derived be from epidemiologically as well as microbiologically sound monitoring systems.[13] As a guide to interpretation of AGSP data, the WHO currently recommends that once resistance to an antibiotic has reached a level of 5 per cent in a population, continuing use of that agent should be reconsidered. 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 per cent level and the sample should also be as representative as possible.[14] Despite 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.

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Annual report of the Australian Meningococcal Surveillance Programme, 2005

Australian Meningococcal Surveillance Programme

Abstract

In 2005 there were 345 laboratory-confirmed cases of invasive meningococcal disease (IMD) analysed by the National Neisseria Network, a nationwide network of reference laboratories. The phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility of 214 isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were determined and an additional 131 cases were confirmed by non-culture-based methods. Nationally, 251 (73%) confirmed cases were infected with serogroup B and 50 (14.5%) with serogroup C meningococci. The total number of confirmed cases was 16 fewer than the 361 cases identified in 2004. The number of cases decreased in Queensland, Tasmania, New South Wales and the Australian Capital Territory and increased slightly in Victoria, South Australia, Western Australia and the Northern Territory. The age distribution of IMD showed a typical primary peak in those aged four years or less with a lower secondary peak in adolescents and young adults. Serogroup B cases were 90 per cent of all cases in those aged four years or less and 75 per cent in those aged 15–24 years. The proportion of all invasive disease represented by serogroup C disease was highest in the 20–24 years and older age groups. The common phenotypes circulating in Australia were B:15:P1.7 and C:2a:P1.5. However significant jurisdictional differences in the serogroup and phenotypic distribution of meningococci were again evident and considerable heterogeneity of subtypes was noted. No evidence of meningococcal capsular 'switching' or genetic recombination was detected. About two thirds of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06–0.5 mg/L). A single isolate was penicillin resistant at 1 mg/L and another was rifampicin resistant. *Commun Dis Intell* 2006;30:211–221.

Keywords: disease surveillance; meningococcal disease; Neisseria meningitidis

Introduction

Laboratory confirmation of the clinical diagnosis of invasive meningococcal disease (IMD) is desirable, not only for the certainty provided for the management of the individual patient, but also to assist in the application of public health measures for disease control. The National Neisseria Network (NNN) is a collaborative national program of reference laboratories in each state and territory of Australia. NNN examines the recognition, antimicrobial resistance surveillance and typing of invasive meningococci, including both isolate-based and non-culture derived methodologies. The NNN began in 1994 and relied solely on data derived from examination of isolates from culture-positive cases of IMD. Subsequently this information has been complemented by non-culture based-methods.

A publicly funded program of vaccination of children and adolescents with serogroup C conjugate vaccine was commenced in 2003 and was fully operational in 2004. This report analyses information gathered by the NNN on laboratory-confirmed cases of IMD in the calendar year 2005. It follows the format used for the 2004 annual report published in *Communicable Diseases Intelligence*[1] in that data on all laboratory-confirmed cases was aggregated for analysis. Prior to 2004 data on IMD diagnosed by culture-based and non-culture methods were provided separately.[2–11]

Methods

The NNN is a long-term collaborative program for the laboratory surveillance of the pathogenic Neisseria, *N. meningitidis* and *N. gonorrhoeae.*[1–11] A network of reference laboratories in each state and territory (laboratories are listed in the acknowledgements) performs and gathers laboratory data on cases of IMD throughout Australia.

Isolate-based invasive meningococcal disease 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 definitions. Information on the site of infection, the age and sex of the patient and the outcome

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(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 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 sterile, is underestimated. However the above approach has been used since the beginning of this program 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 program uses the following parameters to define the various levels of penicillin susceptibility or resistance when determined by a standardised agar plate dilution technique.[12]

sensitive, MIC ≤ 0.03 mg/L.

less sensitive, MIC 0.06–0.5 mg/L.

relatively resistant MIC ≤ 1 mg/L.

Strains with MICs which 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[13] 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. The serological results are based on results of tests performed using the methods and test criteria of the Manchester PHLS reference laboratory, United Kingdom as assessed for Australian conditions.[14–16] 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 345 instances of laboratory-confirmed cases of IMD in 2005 (Table 1) compared with 361 in 2004 and 494 in 2003. In 214 cases a positive culture was obtained with or without a positive non-culture-based test and 131 cases were confirmed by a non-culture-based method alone. The total number of all laboratory-confirmed cases changed

Table 1. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2005, by state or territory and serogroup

State or territory	Serogroup						Total
	B	C	A	Y	W135	NG*	
ACT	4	3			2		9
NSW	69	19		3	9	12	112
NT	5	3					8
Qld	44	13		1			58
SA	18	3		1		1	23
Tas	9	1					10
Vic	61	7	1	3	3	5	80
WA	41	1		2		1	45
Australia	251	50	1	10	14	19	345

NG Not groupable

slightly in all jurisdictions in 2005 when compared to 2004 data. The largest decrease in numbers was in Queensland (to 58 from 75) with smaller numerical decreases in New South Wales (11 fewer cases), the Australian Capital Territory (2 less) and Tasmania (7 less). There were nine more cases in South Australia, seven in Western Australia, four in Victoria and one more in the Northern Territory.

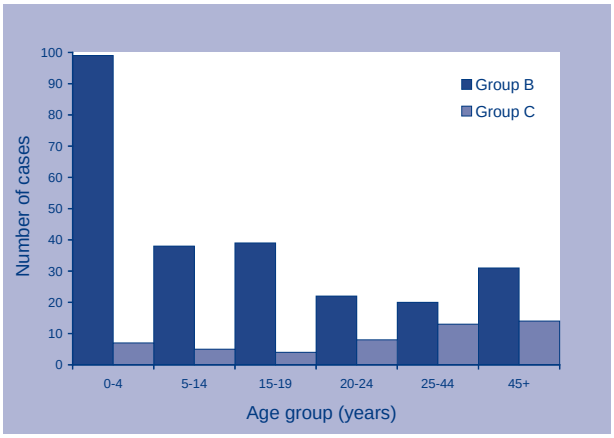
Seasonality

Sixty-one (17.6%) cases occurred between 1 January and 31 March, 63 (18.2%) between 1 April and 30 June, 126 (36.5%) between 1 July and 30 September and 95 (27.5%) 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 aged four years or less (Table 2, Figure 1). Those aged less than one year or in the 1–4 age group accounted for 47 (13.6%) and 63 (18.2%) cases respectively. These numbers are virtually identical to the total number and proportions recorded in these age groups in 2004. The combined total of cases confirmed by all methods in these two groups (110) was one less than that recorded in 2004 and this age grouping accounted for 31.8 per cent of all laboratory-confirmed cases, again a proportion little different from the 30.9 per cent reported in 2004. A secondary disease peak is also usual in the 15–19 years age group. The total of 48 cases (14% of all confirmed cases) in this age group in 2005 was less than the 61 (17%) cases seen in 2004 and the 89 (18%) seen in 2003. Those aged 15–24 years, together accounted for 88 (23.4%) cases (96 cases, 26.7%, in 2004).

Figure 1. Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2005, by age group

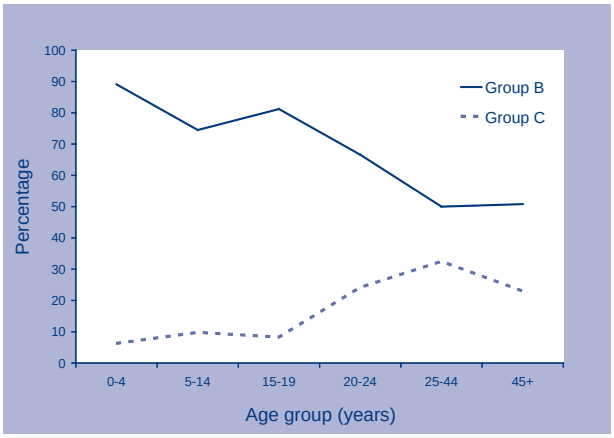


Serogroup data

The serogroup of the meningococci causing disease was determined in 326 of the 345 laboratory confirmed cases of IMD in 2005. Two hundred and fifty-one (76.9%) were of serogroup B, 50 (15.3%) of serogroup C, 1 of serogroup A, 10 (3%) of serogroup Y and 14 (4.3%) of serogroup W135. The serogroup was not determined in two of the 214 cases confirmed by culture, in 6 of 111 cases confirmed by NAA or in 11 of the 20 serologically-confirmed cases. In 2004, a total of 243 (73%) cases of serogroup B and 71 (21%) of serogroup C IMD were confirmed from a total of 361 laboratory-confirmed cases. The corresponding data for 2003 were a total of 285 (58%) cases of serogroup B and 155 (31%) of serogroup C IMD identified from a total of 494 laboratory-confirmed cases.

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).

Figure 2. Serogroup B and C meningococcal disease as a percentage of cases of invasive meningococcal disease confirmed by all methods, Australia, 2005, by age group



In 2005, serogroup B meningococci predominated in all age groups in aggregated national data. Ninety-nine (90%) of the total of 110 laboratory-confirmed IMD cases in those aged less than four years were serogroup B and 6 (5.5%) were serogroup C. These figures differ little from 2004 data. In 2004, 97 (88%) of the total of 111 laboratory-confirmed IMD cases in those aged less than 4 years were serogroup B and 6 (5.5%) were serogroup C.

Table 2. All laboratory confirmed cases of invasive meningococcal disease, Australia, 2005, by age, state or territory and serogroup

State or territory	Serogroup	Age group										Total
		< 1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
ACT	B	0	1	0	0	1	0	2	0	0	1	4
	C	0	0	0	0	1	2	0	0	0	0	3
	Total	0	2	0	0	2	2	2	1	0	0	9
NSW	B	12	13	4	5	11	8	7	3	5	1	69
	C	2	1	1	1	1	3	9	1	0	0	19
	Total	15	17	6	8	14	13	19	7	12	1	112
NT	B	2	1	0	0	0	0	2	0	0	0	5
	C	0	0	1	0	0	0	1	1	0	0	3
	Total	2	1	1	0	0	0	3	1	0	0	8
Qld	B	8	9	5	6	5	3	3	4	0	1	44
	C	0	2	0	0	2	2	0	7	0	0	13
	Total	8	11	5	6	7	5	3	11	1	1	58
SA	B	3	4	4	0	2	2	1	0	2	0	18
	C	0	0	1	0	0	0	1	1	0	0	3
	Total	3	4	6	0	2	2	2	1	3	0	23
Tas	B	2	3	3	0	1	0	0	0	0	0	9
	C	0	0	1	0	0	0	0	0	0	0	1
	Total	2	3	4	0	1	0	0	0	0	0	10
Vic	B	8	9	3	6	14	7	3	10	1	0	61
	C	0	1	0	0	0	1	2	2	1	0	7
	Total	8	10	6	6	14	10	8	14	4	0	80
WA	B	9	15	2	0	5	2	2	6	0	0	41
	C	0	0	0	0	0	0	0	1	0	0	1
	Total	9	15	3	0	7	2	2	7	0	0	45
Australia	B	44	55	21	17	39	22	20	23	8	2	251
	C	2	4	4	1	4	8	13	13	1	0	50
	Other	1	4	6	2	5	3	7	5	11	0	44
Total		47	63	31	20	48	33	40	41	20	2	345
% of all		13.6	18.2	9	5.8	13.9	9.5	11.5	11.9	5.8	0.8	

NS = not stated.

Totals include cases due to other serogroups (n = 25) and cases where the serogroup was not determined (culture confirmed 2, NAA confirmed 6 and serology confirmed 11).

In those aged 5 to 14 years, 38 serogroup B infections represented 74 per cent of the 51 confirmed cases and the 5 cases of serogroup C represented 10 per cent. The number of serogroup B cases in this age group increased from the 27 identified in 2004, but the serogroup C case numbers were unaltered.

There were 48 cases of IMD confirmed nationally in those aged 15–19 years in 2005 (61 in 2004). These comprised 39 (81%) serogroup B and 5 (10%) serogroup C cases. In 2004 there were 40 (67%) serogroup B cases and 17 (28%) serogroup C infections in this age group.

There were 33 instances of IMD in those aged 20–24 years in 2005, 22 (67%) with serogroup B and 8 (24%) with serogroup C meningococci. In 2004, the number of infections and their distribution was not dissimilar with 35 infections; 20 (57%) serogroup B and 11 (35%) serogroup C.

In older age groups (25 years and above), there were 101 laboratory-confirmed cases of IMD in 2005, of which half were serogroup B and a quarter serogroup C. Again these data were closely similar to data from 2004.

A comparison of data from 2005 and 2004 shows little change in serogroup B numbers, except for an increase from 27 to 38 cases in those aged 5–14 years and a smaller decrease in those aged 25 years or more (Table 3). Similarly, there was little further change in the number of serogroup C cases except in those aged 15–19 years. There were only four laboratory-confirmed cases of serogroup C IMD in this age group in 2005 compared with 17 in 2004 and 36 in 2003.

Jurisdictional differences in the distribution of serogroup B and C meningococcal cases continued in 2005 (Table 1). Serogroup B infections predominated nationally and in all jurisdictions. In New South Wales there was little change between 2004 and 2005 in the number and proportion of all serogroup B and C cases. In Queensland, while the total number of IMD cases decreased, the proportion of serogroup B and C infections was unchanged. In Victoria, the total number of cases was also little changed from 2004, but the number and proportion of serogroup B cases increased and that of serogroup C decreased further. In both the Australian Capital Territory and Tasmania there had been clusters of serogroup C cases in earlier years. The number of all cases remained low and in 2005 serogroup C cases declined in number. South Australia, Western Australia and the Northern Territory have had a marked preponderance of serogroup B cases for many years. There were three serogroup C cases of a total of 23 infections in South Australia in 2005 (a single case serogroup C case in 2004), a single case serogroup C infection of a total of 45 cases in Western Australia (6 in 2004) and three of eight cases were serogroup C in the Northern Territory (1 in 2004).

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

Outcome data (survived or died) were available for 163 (47%) of the 345 laboratory-confirmed cases (Table 4). Fifteen deaths were recorded in this group (9.2%). Outcomes were available for 117 of 251 (47%) serogroup B infections and 20 of 50 (40%) serogroup C infections. There were 10 (8.5%) deaths in serogroup B infections and 3 (15%) in serogroup C infections.

Table 3. A comparison of the number and proportion of serogroup B and serogroup C laboratory-confirmed cases of invasive meningococcal disease, 2004 and 2005, by age

Year	Serogroup	Age group (years)				
		< 4	5–14	15–19	20–24	25+
2005	B	99 (90%)	38 (75%)	39 (81%)	22 (67%)	51 (50%)
	C	6 (5.5%)	5 (10%)	4 (8%)	8 (24%)	27 (27%)
	All	110	51	48	33	101
2004	B	97 (88%)	27 (77%)	40 (65%)	20 (57%)	59 (50%)
	C	6 (5.5%)	5 (14%)	17 (28%)	11 (31%)	32 (27%)
	All	110	35	61	35	117

Table 4. Outcome data (survived, died) for laboratory-confirmed cases of invasive meningococcal disease, Australia, 2005, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	41	0	2	0	0	43
	Died	2	0	0	0	0	2
	Total	43	0	2	0	0	45
Septicaemia	Survived	66	17	4	5	13	105
	Died	8	3	1	1	0	13
	Total	74	20	5	6	13	118
All cases	Survived	107	17	6	5	13	148
	Died	10	3	1	1	0	15
	Total	117	20	7	6	13	163

NG Not groupable.

There were two deaths in 45 patients (4.4%) with meningitis; both of these patients were infected with a serogroup B strain. Thirteen deaths were recorded in 118 bacteraemic patients (11%). There were 74 cases of serogroup B meningococcal bacteraemia with 8 (10.8%) deaths and 20 cases were caused by serogroup C strains among whom three fatalities were recorded (15%). A single fatality was recorded in the seven serogroup Y cases and another among the six instances of serogroup W135 bacteraemia.

Phenotypes of invasive meningococcal isolates

Examination of the phenotype of invasive isolates by determination of their serogroup, serotype and serosubtype revealed considerable heterogeneity especially in the serogroup B isolates. The predominant serotypes/serosubtypes in each state and territory are shown in Table 5. Serogroup B meningococci are in general more difficult to characterise by serological methods and a number could not be phenotyped. A total of 15 isolates of the B:4:P1.4

Table 5. Common serotypes and serosubtypes of isolates from culture-positive cases of *Neisseria meningitidis* infection, Australia, 2005, by state or territory

State or territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
ACT	4	2	1.7	1	2a	3	1.4	2
			1.16	1			nst	1
	15	1	1.7	1				
NSW	4	15	1.4	7	2a	8	1.5	6
			1.14	2			1.4	1
			1.15	3			nst	1
			1.7	2				
			nst	1	nt	4	1.12	1
	15	6	1.7	4			1.12,13	1
			1.7,16	1			nst	2
			nst	1				
	1	5	1.14	3				
			nst	2				
	nt	17	1.4	3				
			1.7	2				
			1.15	1				
			1.9	2				
			nst	9				
NT	14	1	nst	1	nt	1	1.15	1
Qld	4	4	1.4	3	2a	11	1.4	1
			1.7	1			1.5	6
	15	6	1.7	4			1.5,2	2
			1.6	1			nst	2
			nst	1	nt	1	1.5,2	1
	1	4	1.14	3				
			nst	1				
	nt	18	1.4	5				
			1.14	3				
			1.12,13	1				
			1.13	1				
			nst	7				

Table 5. Common serotypes and serosubtypes of isolates from culture-positive cases of *Neisseria meningitidis* infection, Australia, 2005, by state or territory, *continued*

State or territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
SA	15	5	1.16	4	2a	1	1.5,2	1
			nst	1				
	14	1	nst	1				
	4	1	1.4	1				
	nt	4	1.14	4				
Tas	4	2	1.19,15	2				
Vic	4,7	7	1.4	3	2a	5	1.4	4
			others	3			1.5,2	1
			nst	1	2b	1	nst	1
	7	2	1.7,16	1				
			nst	1				
	15	8	1.7	6				
			1.7,16	2				
	17,7	4	nst	4				
	19	6	various	6				
	nt	3	1.3	2				
			nst	1				
WA	1	1	1.14	1				
	14	3	nst	3				
	15	1	1.7	1				
	nt	17	various	7				
			nst	10				

nt Not serotypeable.

nst Not serosubtypeable.

phenotype were identified in the Australian Capital Territory, Victoria, New South Wales, Queensland and South Australia in 2005. The number of isolates of this phenotype, circulating in New Zealand at high rates for many years, have declined in recent years in Australia. Forty-one meningococci of this phenotype were detected in 2002, 25 in 2003 and 19 in 2004. Historically, the other common phenotype circulating has been B:15:P1.7. In 2004, eight strains of this type were seen and were concentrated in New South Wales. In 2005, this was the commonest phenotype detected nationally with a total of 16 examples, detected in the Australian Capital Territory, Victoria, New South Wales, Queensland and Western Australia. There is continuing interest in the presence of any serogroup B meningococci of serotypes 2a or 2b but no serogroup B strains of these serotypes were detected in 2005.

Among serogroup C strains, phenotype C:2a:P1.4 is of particular interest. This phenotype has figured prominently in Victorian data in recent years. In 2003 there were 29 and in 2004, 21 serogroup C isolates of this serotype/serosubtype. Only eight were seen in 2005 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 were 2a. The most frequently detected 2a serosubtype, 1.5, was present only in New South Wales and Victoria.

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 51 isolates from CSF either alone or with a blood culture isolate and 160 from blood cultures alone. There were three other isolates from synovial fluid.

Table 6. Anatomical source of samples positive for a laboratory-confirmed case of invasive meningococcal disease, Australia, 2005

Specimen type	Isolate of meningococci	PCR positive*	Total
Blood	160	57	217
CSF +/– blood	51	53	104
Other†	3	1	4
Serology alone‡			20
Total	214	111	345

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

‡ Joint and tissue samples.

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

CSF Cerebrospinal fluid.

The ratio of CSF isolates to blood-culture isolates was 0.3:1. For PCR based diagnosis, this ratio was 0.93:1. This probably reflects the capacity of PCR to amplify meningococcal DNA even after antibiotic treatment and/or delayed lumbar puncture.[17]

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

Two hundred and six isolates were available for determination of their susceptibility to penicillin. Using defined criteria, a single isolate from CSF was resistant to penicillin at an MIC of 1 mg/L, 140 isolates (68%) were less sensitive to penicillin in the MIC range 0.06–0.5 mg/L and 65 (31.5%) fully sensitive (MIC 0.03 mg/L or less). These proportions are similar to those observed in recent years. Eleven isolates had MICs of 0.5 mg/L; blood cultures (5), CSF (5) and joint fluids (1).

Other antibiotics

All isolates were fully susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins) and to the prophylactic agents ciprofloxacin and rifampicin, with the exception of a single isolate with an MIC for rifampicin of 1 mg/L.

Discussion

There was a further decline, albeit slight, in the number of laboratory-confirmed cases of IMD in Australia in 2005. Numbers declined most in Queensland but also in Tasmania and the Australian Capital Territory where some case clusters had been seen in recent years. Cultures were obtained from sterile sites in 214 cases, the lowest number of isolates available since 1994. Non-culture based diagnoses were used to confirm 131 (38%) of cases IMD.

The distribution of cases of IMD in Australia shows major differences when considered by jurisdiction, age and serogroup of the infecting organism and these were again present in 2005. Western and South Australia have had a preponderance of serogroup B infections for many years, Victoria, Tasmania and the Australian Capital Territory until recently tended to have a greater proportion of serogroup C infections than New South Wales or Queensland. Nationally, serogroup B infections were five times more common than serogroup C IMD. This differential was greater in Western and South Australia as in previous years, and in 2005 also in Victoria and Tasmania. Nearly 40 per cent of all serogroup C disease was in New South Wales and another 25 per cent of the national total occurred in Queensland. The Australian Capital Territory and the Northern Territory had low numbers of cases but serogroup C infections remained prominent.

Serogroup B infections have been more frequently encountered in younger age groups where there is a primary peak in IMD infection rates, and in 2005, 90 per cent of 110 confirmed infections in those aged four years or less were with serogroup B. This proportion is little changed from 2004. Serogroup C infections were again infrequent in this age group (Table 3). NNN reports have consistently noted that in the usual secondary peak in IMD in adolescents and young adults, the proportion of serogroup C infections increased over those present in younger age groups. Table 3 shows that this pattern had changed in 2005 from 2004 and earlier years with both the number and proportion of serogroup C cases in the 15–19 years age group now lower. Although overall numbers of cases are low, the same proportional decrease from 2004 to 2005 was not evident in those aged 20–24 years or in those aged 25 years or more. However in those aged 20–24 years, the number of serogroup C infections had already declined from 35 in 2003 to 11 in 2004. There were 41 serogroup C cases identified in those aged 15–19 years in 2003.

The NNN is not as well placed as others to analyse the effect of the national vaccination program with serogroup C conjugate vaccine for reasons previously discussed.[10] These included differences between clinical and laboratory surveillance case definitions, the different rates of introduction and use of non-culture-based confirmatory tests over time and the influence of clinical practice on laboratory-based diagnosis. These concerns remain and fluctuations in the rates of IMD can occur naturally or be influenced by rates of intercurrent viral infection. The data available here and in previous reports will hopefully assist in this formal assessment.

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. While there is some evidence that some meningococci isolated and examined by the NNN in recent years show evidence of genetic recombination, these existing strains have not proliferated and no new combinations were encountered in 2005. Analysis of meningococcal subtypes and any evidence for the clonal 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.

A penicillin MIC of 1 mg/L was detected in a single isolate in 2005. NNN trend data show no major shifts in penicillin MICs of invasive strains. Penicillins remain a suitable treatment for IMD in Australia. All isolates were susceptible to the third generation cephalosporins and the prophylactic agents rifampicin and ciprofloxacin retained their high rate of *in vitro* efficacy.

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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Erratum

Australia's notifiable diseases status, 2002: annual report of the National Notifiable Diseases Surveillance System – *Commun Dis Intell* 2004;28:56

An error has been noted in the section of the 2002 annual report of the National Notifiable Diseases Surveillance System for 2002 in the section on Australian bat lyssaviruses. The statement in the last sentence of this section is incorrect and should read: 'There have been 630 bats detected with European bat lyssavirus in Europe between 1977 and 2000.'

Change of contact details for *Communicable Diseases Intelligence*

Please note that due to recent accommodation changes the contact details for *Communicable Diseases Intelligence* have changed, specifically the Mail Drop Point and facsimile numbers. The new contact details are as below:

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Multi-drug resistant *Salmonella* Java infections acquired from tropical fish aquariums, Australia, 2003–04

Jennie Musto,[1] Martyn Kirk,[2] Diane Lightfoot,[3] Barry G Combs,[4] Lillian Mwanri[4]

Abstract

Antibiotic resistant *Salmonella* infections are rare in Australia. We investigated an increase in multi-drug resistant *Salmonella* Paratyphi B biovar Java (*S.* Java) infections in Australia during 2003–04. Eighty-two per cent (18/22) of *S.* Java cases enrolled into the study reported that they had been in contact with aquariums housing fish during their incubation period. Seventy-two per cent (13/18) of cases were infected with strains that were resistant to ApSmTcCmSuSp (ampicillin, streptomycin, tetracycline, chloramphenicol, sulfonamides, spectinomycin). Case households commonly reported high risk behaviours, such as cleaning aquaria in sinks. Sixty-one per cent (11/18) of cases reported that fish in their aquarium had been sick or died in the week prior to their illness, and *S.* Java was isolated from the water or gravel of 5 cases. These antibiotic strains are being spread internationally and may become endemic in countries importing tropical fish or result in transfer of resistance to other more common *Salmonella* serotypes. *Commun Dis Intell* 2006;30:222–227.

Keywords: Salmonella, *paratyphi, antibiotics, aquariums, fish, reptiles, zoonoses*

Introduction

Salmonella enterica is an important cause of human illness throughout the world. While *Salmonella* is predominantly transmitted via contaminated food, infected animals are also important sources of infection. *Salmonella* infection in people who have been in contact with reptiles is well recognised.[1,2] Public health investigators have also recorded instances where patients infected with *S.* Paratyphi B biovar Java infection have had an aquarium housing tropical fish in their home.[3,4,5] In Australia, *Salmonella enterica* subspecies *enterica* serotype Paratyphi B biovar Java (*S.* Java) has been isolated from the faeces of ill people and water and gravel samples taken from their tropical fish tanks (D Lightfoot, Microbiological Diagnostic Unit, unpublished data).

During 2003, we observed an increase in human infections of *S.* Java acquired within Australia, and particularly strains that were resistant to several antibiotics. To determine the causes for this increase, we conducted a case series investigation of all infections reported to Australian health departments between March 2003 and April 2004.

Methods

The aim of this case series was to identify environmental and behavioural risk factors for *S.* Java infection acquired in Australia. A case of *S.* Java was defined as a person who had:

- a diarrhoeal illness;
- *S.* Java isolated from a stool culture;
- not travelled in the week prior to illness; and
- not been a case in a recognised outbreak.

Cases of *S.* Java infection were identified through state- and territory-based registers of patients infected with conditions reported under public health legislation.

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Epidemiologists conducted telephone interviews of cases using a standardised questionnaire. Investigators interviewed cases aged between 10 and 18 years old with guardian consent. A surrogate, usually a parent, was interviewed for cases under 10 years of age. The questionnaire collected information on the patient's illness and exposure to animals, particularly tropical fish and reptiles in the thirty days prior to illness. The presence and care of aquariums, along with hand washing practices after exposure to the aquarium was also recorded.

Where investigators identified that cases had prior contact with tropical freshwater fish, they visited the patient's home to sample water and gravel from the fish tank using a standardised protocol. After stirring the tank water, investigators collected 100 ml of tank-water in a sterile container, which was refrigerated and transported to the laboratory within six hours. Investigators also collected at least 25 grams of gravel from the bottom of the tank and fish food, which was transported to the laboratory in the same manner as the water. Water was tested for the presence of *Salmonella* using membrane filtration, while aquarium foods and gravel were tested using standard isolation methods for food.[6,7]

Pathology laboratories send all *Salmonella* isolates to a State reference laboratory for confirmation of species and serotype. Isolates of *S.* Java were sent to the Microbiological Diagnostic Unit public health laboratory at the University of Melbourne for phage typing and antibiotic susceptibility testing.[8] This included *Salmonella* isolated from aquarium environs. We assigned local codes for distinct phage type patterns where isolates of *S.* Java Reacted but Did Not Conform (RDNC) to phage types recognised by the Laboratory of Enteric Pathogens, Colindale. To distinguish these different strains, we assigned additional codes to these Australian RDNC isolates, such as 'Aus 3', 'Aus 4', etc. The method of susceptibility testing used was disk diffusion as described by the National Committee for Clinical Laboratory Standards and covered ampicillin (Ap), streptomycin (Sm), chloramphenicol (Cm), sulphathiazole (Su), trimethoprim (Tp), kanamycin (K), nalidixic acid (Na), spectinomycin (Sp), gentamicin (G), ciprofloxacin (Cp) and cefotaxime (Cf).[9]

Results

During the 14-month period from March 2003 to April 2004 there were 76 cases of *S*. Java notified to Australian health departments. Travel to South East Asia was recorded for 21 per cent (16/76) of cases. Twenty-seven per cent (21/76) of cases were excluded from the study, as they were unable to be contacted by telephone, or were contacted more than 30 days following the onset of their illness. The reason for non-recruitment was unspecified for 17 cases. We enrolled 22 cases of *S.* Java into the study that had acquired their infection in Australia. This represented 29 per cent (22/76) of all cases of *S*. Java notified in Australia during the period.

The median age of cases was three years old (range 4 months-48 years old) and the male to female ratio was 1:1.2. Cases reported diarrhoea (100%), fever (77%), abdominal cramps (86%), vomiting (68%), blood in stool (55%), headaches (55%) and myalgia (50%). The median duration of diarrhoea for cases was eight days with a range of 3–26 days. Thirty-seven per cent (8/22) of cases were hospitalised.

Eighty-two per cent (18/22) of cases reported that they had been in contact with aquariums housing fish during their incubation period. Of the remaining four cases, one case infected with *S.* Java that was fully sensitive to all antibiotics tested had acquired a diamond python in the two months prior to illness. The other three cases not reporting exposure to aquariums or pet fish during the month prior to their illness were also infected with sensitive strains (phage types: Battersea, 3b var, and untypable).

For the 18 cases reporting exposure to aquariums, there were 11 cases of S. Java RDNC Aus 3 infection, followed by two cases each of RDNC Aus 5 and Dundee var 2 (Table 1). There were single infections each due to 1 var 15, 3b var and RDNC Aus 6. In cases infected with *S*. Java RDNC Aus 3, 82% (9/11) reported exposure to tropical fish and patient isolates were resistant to ApSmTcCmSuSp. The other two cases infected with multi-drug resistant *S*. Java were siblings who kept goldfish. Thirteen cases were infected with strains that were resistant to ApSmTcCmSuSp.

Of cases reporting exposure to aquariums housing fish, 72 per cent (13/18) reported that the fish tank was kept in the main room of the house (Table 2). Three cases had aquariums present in the kitchen. All aquariums had been present in cases' homes for at least two months (median 5–12 months) prior to illness. Only two cases had more than one aquarium in their house.

Only one case reported being the person that regularly maintained the aquarium. Forty-four per cent (8/18) of cases reported changing the water for their aquarium more frequently than fortnightly. Fifty per cent (9/18) cases reported discarding aquarium water down their sink. Two cases discarding water down the sink reported using the kitchen sink, while the remainder used the sink in their laundry. Two households also reported cleaning their aquarium filter in the kitchen sink.

Table 1. Demographics of cases of *Salmonella* Java, antibiotic resistance profile and fish ownership

Age (years)	Sex	State	Month of isolation	Hospitalised (Y/N)	Duration of illness (days)	*Salmonella* Paratyphi B biovar Java phage type	Antibiotic resistance profile†	Warm water aquarium (Y/N)	Aquarium tested (Y/N)	Fish types
4	F	Qld	Jun-03	Y	8	RDNC Aus 5	Ap	N	N	Goldfish
48	F	Vic	Mar-03	N	7	RDNC Aus 3	ApSmTcCmSuSp	Y	Y	Tropical
4	**M**	**Qld**	**Apr-03**	**Y**	**8**	**RDNC Aus 3**	ApSmTcCmSuSp	N	N	Goldfish
0	**M**	**Qld**	**Jun-03**	**N**	**UK**	**RDNC Aus 3**	ApSmTcCmSuSp	N	N	Goldfish
28	F	Qld	May-03	N	8	3b var	ApSmTcCmSuSp	Y	N	Tropical
3	F	Qld	May-03	N	8	RDNC Aus 3	ApSmTcCmSuSp	Y	N	Tropical
12	M	Vic	May-03	N	UK	RDNC Aus 3	ApSmTcCmSuSp	Y	Y	Tropical
1	F	WA	Jun-03	N	26	RDNC Aus 3	ApSmTcCmSuSp	Y	Y	Tropical
15	F	Qld	Jul-03	N	3	RDNC Aus 3	ApSmTcCmSuSp	Y	N	Tropical
44	F	Vic	Jul-03	Y	17	RDNC Aus 3	ApSmTcCmSuSp	Y	Y	Tropical
48	F	NSW	Aug-03	N	6	1 var 15	ApSmTcCmSuSp	N	N	Goldfish
2	M	Qld	Feb-04	N	4	RDNC Aus 3	ApSmTcCmSuSp	Y	N	Tropical
1	F	NSW	Feb-04	Y	6	RDNC Aus 3	ApSmTcCmSuSp	Y	N	Tropical
2	M	Qld	Feb-04	Y	11	RDNC Aus 3	ApSmTcCmSuSp	Y	N	Tropical
0	M	Qld	Jun-03	Y	8	RDNC Aus 5	Sensitive	N	N	Goldfish
1	M	SA	Apr-04	Y	9	Dundee var 2	Sensitive	Y	N	Tropical
34	F	NT	Mar-03	N	7	RDNC Aus 6	Sm	N	N	Locally caught
1	M	NSW	Oct-03	Y	11	Dundee var 2	SmSuSp	Y	Y	Tropical

* Cases in bold are siblings.

† Ap=ampicillin, Sm=streptomycin, Tc=tetracycline, Cm=chloramphenicol, Su=sulphathiazole, and Sp=spectinomycin

Table 2. Exposures reported by cases of *Salmonella* Paratyphi B biovar Java who had recent exposure to aquariums housing fish.

Exposure	Cases reporting exposure	Proportion exposed (%)
Tank present in home more than 2 months	18	100
Tank in main living room	13	72
Fish tank in kitchen	3	17
Contact with tank water in previous week	11	61
Tank cleaned at least every two weeks	8	44
Water discarded down sink	9	50
Tropical fish (warm water)	12	67
New fish in previous month	5	28
Fish ill in previous month	7	39
Dead fish in month before illness	7	39

One case reported that their aquarium was leaking and another reported adding new rocks in the week before the case's illness. Sixty-one per cent (11/18) of cases reported contact with fish and/or aquarium water in the week before illness, defined as putting hands in water (n=6), feeding fish (n=4) or cleaning the aquarium (n=1). Hand washing with soap and running water following routine maintenance of the aquarium was reported by 78% (n=14) cases.

Cases reported keeping a variety of ornamental cold and warm water fish. Cold water fish—goldfish—were kept by five cases (two of whom were siblings). One case kept fish caught in a creek in the Northern Territory. The other cases kept a variety of tropical freshwater fish including tetras (44%), guppies (39%) and angel fish (28%). Both cold and warm water fish were associated with multi-drug resistant strains of *S*. Java (Fishers Exact Test p=0.176). No cases reported exposure to tropical saltwater fish. Sixty-one per cent (11/18) of cases reported that fish in their aquarium had been sick or died in the week before their illness. Only 28 per cent of cases reported adding new fish to their aquariums in the month before illness. One case reported adding a general purpose antibiotic to their aquarium water, but were unable to identify the specific type.

Aquarium water and gravel was sampled in five homes up to three months after the cases were diagnosed. All samples of water and gravel were positive for *S*. Java. The phage types of *S.* Java isolated from these aquarium environments were the same as those isolated from the cases and were resistant to ApSmTcCmSuSp. One of these case studies was published during the study period.[10] Fish food was sampled from one home one month after their illness and no *Salmonella* was detected. No fish were tested for *Salmonella*.

Discussion and conclusions

Our study indicates that the presence of fish aquariums containing tropical freshwater fish in the home is a risk factor for multi-drug resistant *S*. Java infection, particularly in children aged less than 5 years. This is a major concern, as Australia has very low rates of antibiotic resistant *Salmonella* infection (Unpublished Data, The Microbiological Diagnostic Unit Public Health Laboratory, Melbourne). A recent review of multi-drug resistant *S.* Java strains in the United Kingdom found that S. Java isolates (that were not associated with poultry in Europe) had an identical genetic mechanism of resistance to that of the epidemic clone of *S.* Typhimurium DT 104. These results suggest that there is either a common origin or the horizontal transfer of the resistance gene cluster.[11] *S.* Typhimurium DT 104 is not endemic in Australia, but is a major problem for animal industries in the northern hemisphere.[12,13] In the United Kingdom, a 'fish tank' strain was recently isolated from a calves, showing the potential for this strain to become established in primary production settings.[14] These strains of *S.* Java appear to have emerged in other countries, which is a cause for global concern.[14,15]

Pet ownership is common in Australia. In 2002, there were an estimated 12.2 million fish kept as pets.[16] The way that fish are maintained and transferred between tanks allows for *Salmonella* to travel from the country of origin of the tropical fish to the wholesalers, pet stores and finally, to the home. Domestic wastewater, a possible source of *Salmonella*, is used for the cultivation of fish in places in the world that export exotic fish.[17]

S. Java has been previously isolated from water used to import tropical fish from Singapore.[4,18] In Australia, the carriage water is discarded at the quarantining aquarium and the fish are netted into a

new tank environment, suggesting that the original carriage water from the country of origin is not the direct source of illness to humans in Australia. It is quite possible that aquarium tanks and environments used to quarantine tropical fish are a continual source of *S.* Java for new fish sold to the public.

Australian fish importers use antibiotics in fish tanks as a preventative measure against illness from aquatic fish pathogens. Tetras, the most common fish imported from Indonesia and kept by 44 per cent of cases in this investigation, are prophylactically treated in Australia by adding chloromycetin, tetracycline, metronidazole and sulphadiazine to the tank water. These are four of the six antibiotics that *S*. Java show decreased susceptibility to, and may encourage selection of resistant strains. Consideration should be given to the role prophylactic antibiotics may play in promoting the development of antibiotic resistant bacteria in quarantined fish.

Tropical fish can act as bacterial reservoirs and excrete *Salmonella* in their faeces, without displaying any physical symptoms of illness, which may result in the water or environment becoming contaminated.[4] Once in the home, tropical fish aquarium water is rarely emptied in full, allowing for the *Salmonella* to remain in the aquarium water, gravel and equipment even if fish carrying *Salmonella* are removed. In our survey, several cases or their carers' reported poor hygienic practices, including discarding aquarium water down kitchen sinks. There is a clear need to educate the public about the safety of pets held in aquariums, including those housing fish.

We expected to see that cases reported exposure to warm water tropical fish, but were surprised to find some cases only reporting exposure to cold water goldfish. Retail outlets at the point of sale should provide written information on the risk of infection with *Salmonella* and household aquariums with advice regarding hand washing and safe disposal of aquarium water.

Contact with aquarium water, particularly in children, was associated with illness. Pre-school aged children are often tasked with feeding fish, and may 'play' with aquarium water, and in this study are shown to be more likely to be infected than the main carers of the aquarium. This is probably because children do not wash their hands adequately, if at all, after contact with aquarium water. This study was limited by its' ability to assess the hand washing practices of the cases. The question sought information on hand washing following 'cleaning, feeding fish and touching the water'. As most cases were young children the respondents were parents, not cases, and described their own hygiene practices as the main carer of the aquarium. As such this question did not necessarily capture the hand washing practices of the case. Further investigation may be useful to identify specific behavioural exposures.

Salmonella can result in serious illness, particularly in children. In this study 55 per cent of cases reported bloody diarrhoea and 37 per cent required hospitalisation, which is a higher proportion than previous Australian case control studies of patients infected with antibiotic susceptible strains of *Salmonella enterica* (OzFoodNet, unpublished data). Previous studies have shown that people infected with multi-drug resistant strains have a higher likelihood of severe symptoms and hospitalisation.[19,20] Prevention of multi-drug resistant *S.* Java requires knowledge of its potential sources and adherence to good hygiene following contact with aquariums and fish. Australia needs to ensure that these multi-drug resistant strains do not become established or allow genetic transfer of resistance mechanisms to other more common *Salmonella* serotypes.[13] Consideration should be given to preventing the importation of ornamental fish that harbour antibiotic resistant bacteria.

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OzFoodNet: quarterly report, 1 January to 31 March 2006

Introduction

The Australian Government 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 between 1 January and 31 March 2006.

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

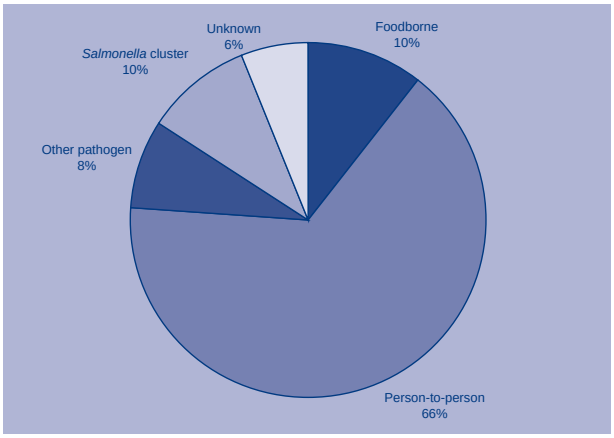
During the first quarter of 2006, OzFoodNet sites reported 248 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or reports are delayed, meaning that these figures significantly under-represent the true burden of these infections. In total, these outbreaks affected 5,092 people of which 72 people were hospitalised and three died. As has been the case in previous reports, the majority (66%, n=163) of outbreaks resulted from infections suspected to be spread from person-to-person (Figure). Fifty-one per cent of these person-to-person outbreaks occurred in aged care facilities, 15 per cent in hospitals and 13 per cent in child care centres.

Foodborne disease outbreaks

There were 26 outbreaks of illness where consumption of contaminated food was suspected or proven to be the primary mode of transmission. These outbreaks affected 263 people. This compares with 31 outbreaks for the first quarter of 2005 and 36 outbreaks in the fourth quarter of 2005.

Salmonella was responsible for nine outbreaks during the quarter, with *Salmonella* Typhimurium being the most common serotype. *S.* Typhimurium 170/108 was responsible for two outbreaks, *S.* Typhimurium 44, a mixed pathogen outbreak (*S.* Typhimurium 44/ *S.* Typhimurium U303), and *S.* Typhimurium 135 were each responsible for one outbreak. Other *Salmonella* serotypes causing single outbreaks were *S.* Anatum, *S.* London, *S.* Montevideo, and *S.* Saintpaul. Norovirus was responsible for five outbreaks, while scombroid poisoning and ciguatera fish poisoning were each associated with three outbreaks during the first quarter of 2006. No aetiological agent was identified for the remaining 6 (23%) outbreaks.

Figure. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, January to March 2006



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

Nine outbreaks reported in the quarter were associated with meals prepared by restaurants, six with meals prepared in private residences and four prepared by takeaway food premises. Single outbreaks were associated with food prepared in an aged care facility, bakery, camp setting, by a commercial caterer, and a national franchised fast food group. The setting where food was prepared was not identified or applicable to the remaining outbreaks. Eight of the outbreaks occurred in January, eleven in February and seven in March.

To investigate these 26 outbreaks, sites conducted six cohort studies. In 17 other outbreaks, only descriptive data were collected and individual patient data was not collected for another three outbreaks. Investigators obtained microbiological evidence linking a food to illness in seven outbreaks, and analytical epidemiological evidence in three outbreaks. For the remaining 16 outbreaks, investigators obtained descriptive epidemiological evidence implicating the food vehicle or suggesting foodborne transmission.

Victoria

In Victoria there were eight outbreaks of foodborne illness reported during the quarter. The aetiological agent was identified in seven of these outbreaks. *S.* Typhimurium 44 affected four people, all of whom were hospitalised. All cases had shared a milkshake containing raw egg and *Salmonella* was later isolated from the blender used to make the milkshake. *S*. Saintpaul affected 11 patrons of a sushi restaurant. Bean shoots from an open container in the restaurant were positive for *S*. Saintpaul, but subsequent testing of bean shoots from a sealed package were negative for *Salmonella.* Five people infected with *S*. London had eaten home-prepared salami which was positive for *S.* London.

Norovirus affected 41 people from four separate groups that had eaten food prepared by a commercial caterer. No specific food was identified as causing the illness. A food handler reported being ill prior to the event and worked while the were ill. In another outbreak, illness due to norovirus was reported in 6 of 19 groups that had eaten at a restaurant. No specific food was identified as causing illness but there were anecdotal reports of a food handler and a waiter being ill just after the event. Norovirus was isolated and likely responsible for illness in nine people from two separate groups that had eaten at a restaurant. A food handler reported illness in the week prior to the event but did not work while ill.

Two cases of scombroid fish poisoning were reported after a meal of kingfish in a restaurant. A histamine level of 3,450 mg/kg was reported in a sample of the fish. An inspection identified inappropriate thawing and cold storage of fish at the restaurant.

Five residents of an aged care facility were ill with gastroenteritis that was consistent with *Clostridium perfringens* intoxication.

Queensland

Queensland reported eight outbreaks of foodborne illness during the quarter. Three outbreaks were caused by ciguatera fish poisoning: two cases consumed ‘cod’ fish steaks from a fish caught during a fishing trip east of Gladstone; trevally fish fillets purchased for preparation at home caused two cases of illness; and four cases of illness in two separate groups was caused by Spanish mackerel which had been served at a restaurant and prepared at home. Two cases of scombroid fish poisoning were reported after a meal of blue fin tuna steaks purchased for preparation at home. Norovirus was the cause of an outbreak of foodborne gastroenteritis among 45 guests who attended a birthday function at a restaurant. There were several foods independently associated with illness but the epidemiological associations were weak. A commercial caterer was suspected as the cause of an outbreak of norovirus gastroenteritis in 66 attendees at a school camp. Norovirus was detected in the stools of two food handlers who were ill with gastroenteritis. Norovirus was also detected in stools from six students.

Queensland also reported an outbreak of suspected foodborne gastroenteritis involving as many as 80 to 100 guests of a birthday function. The foods were poorly handled prior to consumption although a source of illness was not conclusively identified.

New South Wales

New South Wales reported five foodborne outbreaks for the quarter. A chicken schnitzel in gravy from a takeaway restaurant was suspected to have caused illness in three people. Two cases of scombroid fish poisoning were reported after a meal of tuna steaks in a restaurant. An inspection identified inadequate refrigeration and other fish storage problems at the restaurant. In another outbreak, four cases of *S.* Typhimurium 170/108 were reported over a short time period from a small township. Three of the cases had consumed hamburgers or chicken burgers from the same takeaway shop in the three days prior to illness onset. An environmental investigation did not identify any areas of non-compliance with the Food Safety Standards.

S. Montevideo affected three people that had eaten hamburgers from a New South Wales takeaway food shop. Food and environmental sampling identified *S*. Montevideo from hamburger, eggs, and a food scraper. It is thought that eggs introduced the pathogen into the premises, and then cross-contaminated the hamburger. An investigation of the egg farm identified that a new egg sorting machine may have spread contamination from egg-to-egg. Swabs taken from a wire brush machine on the farm were positive for *S.* Montevideo.

In another outbreak, two people were ill 1–2 hours after a restaurant meal, although no common food was identified.

South Australia

South Australia reported three outbreaks of foodborne illness during the quarter. *S*. Typhimurium 170/108 affected seven people after they had eaten a homemade chocolate ice-cream and topping containing raw egg. *S.* Typhimurium 170/108 was later isolated from the ice-cream and topping. In another outbreak, *S*. Typhimurium 135 affected four members of a family after a meal of silverside. *S.* Anatum affected five people after eating beef burger with bacon and egg prepared at a hotel restaurant.

Australian Capital Territory

The Australian Capital Territory reported an outbreak of gastroenteritis of unknown aetiology in members (10 cases) from different families who attended a family function. A birthday cake was the most likely food to cause illness. The bakery that supplied the cake was inspected and found to have poor hygiene standards. Samples of ingredients taken during inspection were negative for pathogens.

Tasmania

Tasmania reported an outbreak of both *S.* Typhimurium 44 and *S.* Typhimurium U303 that affected nine people following a family dinner. The cases had eaten multiple dishes prepared by different members of the family but the majority found it difficult to recall what foods they had eaten.

Northern Territory and Western Australia

West Australia and the Northern Territory did not report any foodborne outbreaks occurring in the first quarter of 2006.

Clusters and multi-state investigations

During the quarter, OzFoodNet and jurisdictions investigated many clusters of infection that were localised to single states or territories or spread over several jurisdictions. The major multi-jurisdictional investigations occurring during the quarter were for clusters of cases of listeriosis, and *S.* Bovismorbificans.

In February, Western Australia identified an increase in the number of cases of *Listeria.* Other jurisdictions also reported moderate increases and OzFoodNet held several teleconferences to discuss possible hypotheses. Laboratories in different jurisdictions sent recent *Listeria* isolates to PathWest where Pulsed Field Gel Electrophoresis (PFGE) testing was conducted with eight isolates from Western Australia, three from Victoria and one from South Australia being identified. A group of three Western Australian isolates had indistinguishable PFGE patterns (Cluster A). Another group of two Western Australian isolates had PFGE patterns indistinguishable from each other (Cluster B). Two of the Victorian isolates had PFGE patterns indistinguishable from the South Australian isolate (Cluster C). Cluster A had a PFGE pattern indistinguishable from an isolate from a salami product that was collected one week prior to the onset of the first of these three cases. Consumption of this salami was not mentioned by the cases in a review of their food history however their food recall was poor. The concentration of *Listeria* in this salami product was < 3/g and the company is reviewing their production processes. A common food source was not identified in Clusters B and C. This investigation again highlights the importance of incorporating information from DNA-based testing methods into surveillance and cluster investigations in Australia.

The outbreak of *S.* Bovismorbificans mainly affected Queensland, the Australian Capital Territory, New South Wales and Victoria. The main phage types that were responsible for the increase were phage types 24, 14 and 13. OzFoodNet sites conducted hypothesis-generating interviews of several cases but did not find a common food-vehicle or exposure associated with this multi-state increase in illness.

In February, the NSW Health Department was notified of two cases of hepatitis A in 12-year-old boys whose most likely exposure was a camp that they had attended in Fiji. The camp organisers were contacted and provided with a letter for distribution to camp attendees informing of their possible exposure to hepatitis A. Twelve children and four adults, from Queensland, New South Wales and New Zealand, attended the camp. New Zealand and Fiji health authorities were informed of the outbreak and our investigation. Subsequently, two further cases of hepatitis A were diagnosed in children in New South

Wales. All four children were infected with hepatitis A and were not immunised. Whilst hepatitis A is endemic in Fiji, the source of infection remains unclear.

During the quarter, states and territories reported large increases in sporadic and outbreak related cases of cryptosporidiosis (see *CDI* Highlights p. 251). There were six outbreaks of cryptosporidiosis in February and 15 in March. Seventeen of these outbreaks were related to swimming pool exposure, while the remaining outbreaks were of unknown or person-to-person transmission, including one outbreak in a prison.

Western Australia health authorities completed an investigation into a large outbreak of *Salmonella* Oranienburg that occurred from November 2005 to February 2006. In total 126 cases were reported as part of the outbreak, compared to an average of 6–12 notifications annually in Western Australia. Initial hypothesis generating questionnaires failed to identify a food vehicle, but the male to female ratio was 1:2. Western Australia conducted a case control study that identified alfalfa sprouts to be strongly associated with illness caused by *S.* Oranienburg. Brands of alfalfa purchased by the cases were traced back to a single sprout production facility. *S*. Oranienburg was isolated from alfalfa taken from the residence of cases and at the production facility. The PFGE pattern of isolates from alfalfa sprouts and clinical cases were identical. The company issued a recall of a range of sprout products during February 2006.

Discussion

There was considerable activity during the first quarter of 2006, with overall notifications of *Salmonella* to the National Notifiable Diseases Surveillance System increased by 14 per cent when compared to the mean for the same time frame in the previous two years. There were several multi-jurisdictional investigations into *Salmonella* and *Listeria*, including the completion of the investigations into *Salmonella* Typhimurium 44 and 135 from the previous quarter.[1] The reasons for the generalised increase in salmonellosis were unknown, but some of the large ongoing salmonellosis outbreaks in eastern States and Western Australia would have contributed.

The outbreak of *Salmonella* Oranienburg in Western Australia resulted in a recall of alfalfa sprouts and is the largest reported outbreak of gastroenteritis associated with sprouts in Australia to date. Sprouts are a high risk food that have caused many outbreaks internationally.[2] The outbreak has identified a need to review the procedures associated with the growing, harvesting and production of sprouts in Australia. As a result of this outbreak, national food safety policy committees will consider ways to improve food safety for this industry sector.

Acknowledgements

We would like to thank 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 and acknowledge the work of all laboratories providing notifications of pathogens that can cause foodborne illness and in particular those laboratories that conduct serotyping and phage typing of *Salmonella*.

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Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* January to March 2006

State	Month of outbreak	Setting prepared	Infection/illness	Number affected	Evidence	Responsible vehicle
ACT	February	Bakery	Unknown	10	D	Birthday cake is most likely vehicle
NSW	January	Takeaway	*Salmonella* Typhimurium 170/108	3	D	Suspect chicken hamburger/ beef hamburger
	February	Takeaway	*Salmonella* Montevideo	3	M	Plain hamburger
		Takeaway	Unknown	3	D	Chicken schnitzel in gravy
		Restaurant	Scombroid	2	D	Tuna steaks
	March	Restaurant	Unknown	2	D	Sweet corn chicken soup or crumbed chicken
Qld	January	National franchised fast food	Unknown	24	A	Unknown
	February	Camp	Norovirus	66	D	Unknown
		Private residence	Scombroid	2	D	Blue fin tuna steaks
		Not applicable	Ciguatoxin	2	D	Cod
	March	Restaurant	Norovirus	15	A	Unknown
		Takeaway	Ciguatoxin	4	D	Spanish mackerel
		Restaurant	Unknown	8	A	Unknown
		Private residence	Ciguatoxin	2	D	Trevally fish
SA	January	Private residence	*Salmonella* Typhimurium 170/108	7	M	Home-made ice-cream topping containing raw egg
	February	Private residence	*Salmonella* Typhimurium 135	4	M	Silverside
		Restaurant	*Salmonella* Anatum	5	D	Beef burger with bacon and egg
Tas	January	Private residence	*Salmonella* Typhimurium 44 and U302	9	D	Unknown
Vic	January	Private residence	*Salmonella* Typhimurium 44	4	M	Milkshake containing raw egg
		Restaurant	Norovirus	15	D	Unknown
		Restaurant	Norovirus	9	D	Unknown
		Aged care facility	Unknown	5	D	Unknown
	February	Commercial caterer	Norovirus	41	D	Unknown
		Restaurant	Scombroid	2	M	Kingfish
	March	Restaurant	*Salmonella* Saintpaul	11	M	Suspected bean shoots
		Unknown	*Salmonella* London	5	M	Salami (non-commercial)

* No foodborne outbreaks were reported in West Australia or the Northern Territory 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.

Hypovolemic shock and metabolic acidosis in a refugee secondary to O1 serotype *Vibrio cholerae* enteritis

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A 71-year-old Burundian woman presented to Monash Medical Centre emergency department in March 2005. She described two days of lethargy, profuse diarrhoea and vomiting but denied fevers, or other symptoms. The following travel history was obtained. Upon receiving a Special Humanitarian Program visa to Australia, her family was translocated to an United Nations High Commissioner for Refugees (UNHCR) processing centre from a refugee camp in rural Tanzania, then onto Nairobi, Kenya. Her gastrointestinal symptoms commenced as they began their journey from Nairobi. The family travelled with 45 other refugees on a commercial flight to Australia and arrived 36 hours before presentation at the emergency centre. The patient's daughter-in-law developed similar, but milder, symptoms of diarrhoea during the flight and was also admitted to our hospital.

On examination, she was afebrile but tachycardic with blood pressure of 85/45 mmHg. Tissue turgor was markedly reduced, with dry mucous membranes and sunken eyes. The remainder of her examination was unremarkable. An ECG revealed sinus tachycardia and prolonged QTc interval and her biochemistry showed potassium of 2.5 mmol/L (3.5–5.0), creatinine of 0.32 mmol/L (0.04–0.12) and metabolic acidosis, with bicarbonate of 12.2 mmol/L (22–32).

Initial management was directed at treatment of her hypovolemic shock with intravenous fluid resuscitation, and potassium replacement. As suspicion for cholera was high, specific cholera culture from faecal specimen was performed. The Victorian Department of Human Services was notified with a presumptive diagnosis of cholera.

Curved Gram negative bacilli consistent with *Vibrio cholerae* were isolated from selective culture of faeces of both patients (Figures 1–3). Confirmation was performed biochemically and via enterotoxin

Figure 1. Photograph of patient's stool sample

Figure 2. Curved Gram negative rods – *Vibrio cholerae*

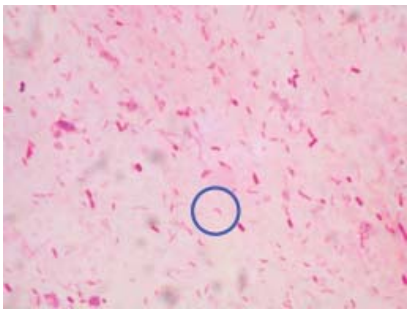
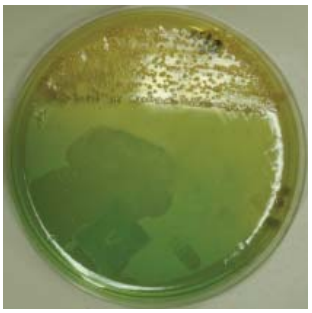


Figure 3. Selective medium (TCBS) showing typical yellow colonies



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polymerase chain reaction testing. The isolate was serotype 01, biotype non-El tor, non-classical and sub-serotype Ogawa. Close contacts were negative on screening.

Management continued with fluid and electrolyte repletion. Doxycycline was commenced. This patient was also diagnosed with intestinal hookworms, *Escherichia coli* cystitis, and penicillin-resistant *Streptococcus pneumoniae* bronchitis. Notably, the patient was HIV antibody negative. On day six, both patients were discharged. Prior to leaving, they celebrated their survival of this deadly disease by performing a traditional dance of life to thank the treating staff.

Of the 45 passengers who travelled with our patients, none reported any diarrhoeal symptoms. Four were subsequently diagnosed with *Plasmodium falciparum* malaria.

Cholera is a diarrhoeal disease caused by *Vibrio cholerae*. It is one of the few conditions requiring notification to the World Health Organization under the International Health Regulations.[1]

The mode of cholera transmission is via ingestion of contaminated food or water. The incubation period of cholera is short, with symptoms commencing within hours to five days after exposure. Most cases of infection are asymptomatic with less than 20 per cent of infections progressing to cholera gravis. This is characterised by profuse diarrhoea and vomiting, without fever. An excerpt from Dr John Snow's treatise describes the symptoms of the ensuing and often fatal dehydration: 'Loss of water from the blood…causes many of the symptoms of a true haemorrhage, as debility, faintness, and coldness',[2] Death can occur within hours due to complications such as hypokalemia, arrhythmias, hypoglycaemia, acute renal failure and metabolic acidosis.

Overall mortality if untreated is at least 50–70 per cent, and higher in pregnant women and children. Recognising the above, treatment for suspected cholera should begin before laboratory identification of the causative organism is available. The laboratory must be notified in suspected cholera for biosafety reasons and the requirement for selective medium to culture the organism.

Cholera is readily treatable (Box). The aim is to replace lost fluids and electrolytes promptly. Fluid repletion does not need to be parenteral unless the patient is intolerant of oral intake or severely dehydrated. Anti-diarrhoeals are not advised. Antibiotics are adjunctive to fluid replacement and have been shown to reduce the volume of diarrhoea and the duration of *Vibrio* excretion.[3] The latter effect is used to aid control of an outbreak. Tetracyclines are the drugs of choice, with quinolones and macrolides as alternatives in children, and pregnant women.[3,4]

Box. Key points in the management of cholera

Severe dehydration and frequent diarrhoea should always prompt the diagnosis of cholera.

Prompt fluid replacement is vital, death by dehydration can occur within hours.

Special medium must be requested for detection of *Vibrio* and other enteropathogens such as *Campylobacter, Aeromonas,* and *Yersinia*.

More than one infection may be present in a traveller.

Refugees are likely to have multiple untreated acute and chronic infections, take the opportunity to screen and vaccinate.

Despite the ease of treatment in sporadic cases, cholera remains a global threat and is one of the key indicators of social development. The World Health Organization estimates at least 5.5 million cases of cholera occur worldwide each year, with more than 100,000 deaths.[5] West Africa reported a recent large outbreak with 31,259 cases and 517 deaths, and spreading to Central Africa.[6] This is an underestimate due to the lack of surveillance data. While cholera no longer poses serious threat to countries with minimum standards of hygiene, it remains a challenge to countries where safe drinking water and adequate sanitation cannot be guaranteed. As well as high mortality, cholera causes significant socioeconomic loss, particularly in regions of endemicity in Asia, Central Europe and Africa.

Cholera is an uncommon diagnosis in Australia, with less than 10 cases reported annually in the last decade. The majority of cases were imported. In Victoria, from 1995–2003 there were five cases, acquired in Bali (2 cases), India, Vietnam and Indonesia or Singapore.

Cases related to rivers on the east coast of Australia have been reported.[7,8] Cholera should be suspected in travellers who develop severe diarrhoea and dehydration. The Victorian Health (Infectious Disease) Regulations 2001 require suspected cholera cases to be notified by telephone prior to laboratory confirmation.[9] Contacts of the index case are followed up for five days from the last exposure to the index patient. Chemoprophylaxis and vaccination are not recommended.

Australia has increased its refugee intake as part of UNHCR's Special Global Program (now averaging 13,120 annually). Refugees often come from

regions of poor sanitation. These factors place them at high risk of acute and chronic infectious diseases including cholera. Despite pre-migration screening, schistosomiasis, HIV, tuberculosis, and falciparum malaria are commonly diagnosed for the first time post-migration. As a result, post-migration screening for infectious diseases is recommended but may not occur.

Health providers can expect significantly increased presentations of untreated medical and psychological diseases in refugees. Familiarisation with imported conditions is vital to provision of appropriate health care to new immigrants. Diseases prevalent to Africa and the Middle East are particularly important as new intake from these areas now account for 65 per cent and 24 per cent of total respectively.[10]

Acknowledgements

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Prevalence and control of trachoma in Australia, 1997–2004

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Abstract

This study aimed to document the prevalence of active trachoma and trichiasis from 1997 to 2003 and from 1987 to 2004, respectively, and to provide an overview of trachoma control activities in Australia in 2004. Prevalence data were obtained from state, territory and regional population health units and unpublished surveys. Information about trachoma control programs and activities currently implemented in Australia was obtained through structured interviews with staff involved in trachoma control. Active trachoma prevalence in Aboriginal and Torres Strait Islander children, ranging from 0–40 per cent, were reported from the Eastern Goldfields, Midwest-Murchison and Kimberley Population Health Units in Western Australia and the Northern Territory's Centre for Disease Control. Large differences in trachoma prevalence were reported within and between different regions and from different years in the same region. Recent surveys of trichiasis in Kimberley and Central Australian Aboriginal and Torres Strait Islander adults demonstrated a prevalence of 9–12 per cent in inland, desert areas. In contrast with developing countries where active trachoma and trichiasis are more common among adult women than men, Australian surveys have identified equal prevalence in both sexes. Interpretation of trachoma prevalence and inter-regional/state/national comparisons were hampered by lack of a uniform method of data collection and analysis. Trachoma control programs were implemented consistently in some communities, and irregularly and/or in piecemeal fashion in others. Trachoma control programs led by regional population health units working in collaboration with primary health care services were more likely to be consistently implemented over long periods of time. Trachoma is a significant public health issue in some Aboriginal communities within Australia. The Communicable Diseases Network Australia has developed guidelines for the public health management of trachoma which provide recommendations on trachoma screening, control and data collection trachoma for affected states and territories. *Commun Dis Intell* 2006;30:236–247.

Keywords: trachoma, trichiasis, epidemiology, public health management, Aboriginal

Introduction

Trachoma is caused by *Chlamydia trachomatis* infection of the conjunctiva. Recurrent infection may result in conjunctival scarring, trichiasis, corneal opacification and blindness. Although trachoma had disappeared from most parts of remote Australia by the 1930s as housing, hygiene and living conditions improved, active trachoma in Aboriginal and Torres Strait Islander children, and trichiasis in Aboriginal and Torres Strait Islander adults, is still found in some regions.[1]

The World Health Organization (WHO) trachoma grading system and 'SAFE' intervention strategy have been implemented in some trachoma-endemic areas of Australia for many years.[2,3,4] The WHO simplified trachoma grading system classifies trachoma

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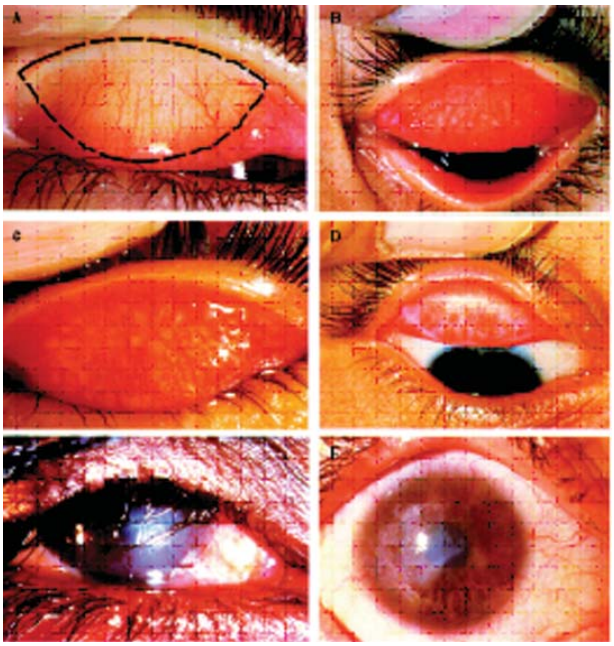
into five clinical stages: follicular trachomatous inflammation (TF); intense trachomatous inflammation (TI); trachomatous scarring (TS); trachomatous trichiasis (TT); and corneal opacity (CO) as shown in the Figure.[2] The SAFE acronym stands for ***S***urgery for trichiasis, community use of ***A***ntibiotics to reduce transmission of chlamydial infection, ***F***acial cleanliness, and ***E***nvironmental health improvements.[4] The WHO recommends that active trachoma (i.e. TF and/or TI) prevalence in children aged 1–10 years be used to determine the nature and coverage of public health interventions for trachoma within a community or population.[2] The WHO and its partners (including Australia) aim to eliminate avoidable blindness, including blinding trachoma, (VISION 2020 – the right to sight) by 2020.[5]

Figure. World Health Organization simplified trachoma grading classification system[2]



Source: World Health Organization.

(A) Normal everted upper tarsal conjunctiva.

(B) Trachomatous inflammation follicular (TF) presence of five or more follicles in the upper tarsal conjunctiva of at least 0·5 mm.

(C) Trachomatous inflammation intense (TI) pronounced inflammatory thickening of the tarsal conjunctiva that obscures more than half of the normal deep tarsal vessels.

(D) Trachomatous scarring (TS) presence of scarring in the tarsal conjunctiva.

(E) Trachomatous trichiasis (TT) at least one eyelash rubs on the eyeball or evidence of recent removal of in-turned eyelashes.

(F) Corneal opacity (CO) easily visible corneal opacity over the pupil.

Prevalence data for active trachoma until 1996, and for trichiasis until 1980, in many trachoma-endemic regions have already been published.[6,7,8] This paper aimed to document the prevalence of active trachoma and trichiasis from 1997 to 2003 and from 1987 to 2004, respectively, and to provide an overview of trachoma control activities in Australia in 2004.

Methods

Active trachoma and trichiasis prevalence

In June 2003, the Office for Aboriginal and Torres Strait Islander Health (OATSIH), a division of the Australian Government Department of Health and Ageing (DoHA), requested prevalence data on active trachoma in children collected after 1987, and on trichiasis in adults collected after 1980, from the Northern Territory, Queensland, New South Wales, South Australian and Western Australian health departments. Tasmania, Victoria and the Australian Capital Territory were not surveyed as trachoma had ceased to be a public health problem in these jurisdictions decades ago. Data were collected using a WHO survey proforma. If prevalence data for individual communities were reported, these were combined into regional datasets based on Australian Bureau of Statistics (ABS) census collection regions due to the small populations of many Aboriginal and Torres Strait Islander communities. Trachoma prevalent areas, whilst large geographically, are remote and sparsely populated.

Regional population data were obtained from the ABS Census of Population and Housing, or in the case of some regions in Western Australia, from the Western Australian Department of Education or the Western Australia Department of Health. It was not always possible to match population estimates with the age range screened. Where this was not possible the closest age range estimates are given. The regional population data presented are for Aboriginal and Torres Strait Islander children only, with the exception of the Murchison region of Western Australia where non- Aboriginal and Torres Strait Islander children were included.

Trachoma control programs

Information about trachoma control programs and activities currently implemented in Australia was obtained through structured interviews with 95 people conducted by DBM between July and August 2004. Interviewees included people involved in trachoma or communicable disease control at state, territory and regional population health unit and primary health care levels, and ophthalmologists and optometrists.

Interviews lasted 20 to 60 minutes depending on the interviewee's level of involvement in trachoma control programs/activities. Where available, trachoma control guidelines, staff education resources and reports of trachoma control activities were obtained from interviewees.

Results

Active trachoma prevalence in children, 1997–2003

The data presented in Table 1 were reported by the Eastern Goldfields, Midwest-Murchison and Kimberley Population Health Units of the Department of Health Western Australia and the Northern Territory Government's Centre for Disease Control.

Queensland Health reported minimal trachoma data collected through laboratory notifications. Active trachoma prevalence could not be ascertained from these data.

South Australian health authorities reported verbally that they had no data on trachoma. No response was received from NSW Department of Health.

As shown in Table 1, there was considerable variation in sampling methods, and in the proportion of the regional childhood population examined for active trachoma between different regions. Large differences in active trachoma prevalence between regions and in the same region between different years were observed. In addition (not shown in Table 1), there were large differences in active trachoma prevalence between towns and communities within the same region. For example, in the Kimberley in 2003 regional active trachoma prevalence was reported as 11 per cent. Trachoma is no longer endemic in some Kimberley towns and coastal communities, which therefore were not screened, and trachoma prevalence in screened schools and communities ranged from 5 per cent to 60 per cent.[13] Apart from the Eastern Goldfields where a small proportion of TI was reported, TF and TI prevalences were not reported separately.

Data on the Aboriginal and/or Torres Strait Islander status of children examined were not reported, except from the Murchison region.

Table 1. Active trachoma prevalence in Australia, 1997–2003*

Location	Age group surveyed	Month and year of survey	Estimated number of Indigenous children in region†	Number of children examined for active trachoma	Number of children examined with active trachoma	Active trachoma prevalence in children examined (%)
NT						
Barkly	5–15	2002	810§§,11	81	10	12
Barkly	5–15	2001	810§§,11	90	3	3
Barkly	5–15	1998	925§§,12	60	3	5
Barkly	5–15	1997	925§§,12	237	14	6
East Arnhem	5–15	2002	1,816§§,11	675	135	20
East Arnhem	5–15	2001	1,816§§,11	143	0	0
East Arnhem	5–15	1998	1,828§§,12	93	3	3
Katherine	5–15	2002	1,850§§,11	344	3	1
Katherine	5–15	2001	1,850§§,11	231	7	3
Katherine	5–15	2000	1,829§§,11	623	142	23
Katherine	5–15	1999	1,829§§,12	420	67	16
Katherine	5–15	1998	1,829§§,12	223	35	16
Katherine	5–15	1997	1,829§§,12	329	35	11
Darwin rural	5–15	2002	2,270§§,11	347	30	8
Darwin rural	5–15	2001	2,270§§,11	569	6	1
Darwin rural	5–15	2000	2,079§§,11	513	15	3
Darwin rural	5–15	1999	2,079§§,12	639	37	6
Darwin rural	5–15	1998	2,079§§,12	336	53	16
Alice Springs	5–15	Jun 1999	2,789§§,12	278	97	35
Alice Springs	5–15	1998	2,789§§,12	119	47	40

Table 1. Active trachoma prevalence in Australia, 1997–2003,* *continued*

Location	Age group surveyed	Month and year of survey	Estimated number of Indigenous children in region†	Number of children examined for active trachoma	Number of children examined with active trachoma	Active trachoma prevalence in children examined (%)
WA						
Eastern Goldfields	< 18‡	May-June 2003	> 2,028§,9	516	140‖	27
Murchison¶	1–10	March 2002	304**	129**	34	26
			266††	82††	3	4
Murchison¶	1–10	March 2001	304**	146**	14	10
			266††	92††	7	8
Murchison¶	1–10	March 2000	304**	126**	16	13
			266††	92††	0	0
Murchison¶	1–10	March 1999	304**	137**	10	7
			266††	129††	0	0
Murchison¶	1–10	March 1998	304**	127**	13	10
			266††	105††	2	2
Murchison¶	1–10	March 1997	304**	190**	41	22
			266††	131††	1	1
Kimberley‡‡	5–16+	Sept-Nov 2002	3,639[10]	1,670	178	11
Kimberley‡‡	5–16+	Sept-Nov 2001	3,693[10]	1,676	195	12

* Sources: Eastern Goldfields, Midwest-Murchison and Kimberley Population Health Units of the Western Australia Department of Health and the Northern Territory Government's Centre for Disease Control.

† Indigenous children only, except Murchison.

‡ Children attending school only.

§ 0–14 years. Also included in this survey area were some communities on the Nganyatjarra Lands (estimated number of children unknown).

‖ Includes 11 TI cases.

¶ Screening performed at pre-schools and schools in four remote towns and one remote boarding school.

** Indigenous

†† Non-Indigenous

‡‡ 28 schools surveyed.

§§ 5–14 years.

Trichiasis prevalence in adults, 1987 to 2004

No trichiasis data were available for 1981 to 1986. From 1987 to 2004, survey data on trichiasis prevalence were available from only the Kimberley, Central Australia and the Anangu Pitjantjatjara lands, as shown in Table 2. As with active trachoma, there were marked differences in trichiasis prevalence both between and within regions. No differences in trichiasis prevalence between men and women were observed.

In the Kimberley region, trichiasis prevalence was significantly higher in inland, desert areas than in those in close proximity to the sea or major rivers.[7,14] A survey by Landers, *et al* in 2003 indicated that trichiasis was still endemic among Aboriginal and Torres Strait Islander adults in Central Australia.[16] Many health professionals reported rarely or never seeing trichiasis but no recent data were available from areas outside the Kimberley and Central Australia.

Trachoma control

Notification of trachoma

Trachoma is not a nationally notifiable disease based on clinical diagnosis nor through laboratory notifications. It is also not notifiable based on clinical diagnosis in any State or Territory, except in Western Australia where notification has not been requested since 1993 because local experience demonstrated that it was neither an appropriate nor an effective surveillance strategy. In Queensland

Table 2. Trichiasis prevalence in Australia, 1987 to 2004

Location	Year	Population screened	Percentage of target population screened	Trichiasis prevalence (%)
Western Australia				
Kimberley (excluding Broome Shire)[14]	2004	Aboriginal and Torres Strait Islander 50+ years	20.0*	9.5† (Halls Creek Shire 12.5, Wyndham-East Kimberley Shire 6.0, Derby – West Kimberley Shire 4.3)
Kimberley[7]	1998	Aboriginal and Torres Strait Islander 50+ years	41.8‡	2.9† (Halls Creek Shire 11.0, Wyndham-East Kimberley Shire 1.8, Derby – West Kimberley Shire 1.7 Broome Shire 1.0)
Fitzroy Crossing[15]	1993	Not reported	Not reported	1.0
Northern Territory				
Central Australia[16]	2003	Aboriginal and Torres Strait Islander 40+ years, presenting to a general ophthalmology outreach clinic for symptoms/reasons unrelated to trachoma	100%	9.0†
Alice Springs/Barkly[17]	1987-90	Aboriginal and Torres Strait Islander, 40+ years	Not reported	5.2
Katherine[17]	1987-90	Aboriginal and Torres Strait Islander, 40+ years	Not reported, < 30 people screened	6.9
Darwin Rural[17]	1987-88	Aboriginal and Torres Strait Islander, 40+ years	Not reported, < 30 people screened	0
East Arnhem[17]	1987-89	Aboriginal and Torres Strait Islander, 40+ years	Not reported	13.0
South Australia				
A-P§ lands[18]	1999-2000	Aboriginal and Torres Strait Islander, all ages	75%	0.6 (5.2% in 50 + years age group)
A-P lands[19]	1989-90	Aboriginal and Torres Strait Islander, all ages	54.3%	2.9 (19.1% in 60+ years age group)

* 46.7 per cent, 10.7 per cent and 10.4 per cent in Halls Creek, Wyndham-East Kimberley and Derby-West Kimberley Shires, respectively.

† No difference in trichiasis prevalence between males and females.

‡ 74.3 per cent, 34.1 per cent, 66.3 per cent and 22.7 per cent in Halls Creek, Wyndham-East Kimberley and Derby-West Kimberley and Broome Shires, respectively.

§ Anangu Pitjantjatjara.

and the Northern Territory, laboratory notifications of *Chlamydia trachomatis* specify the site from which the isolate was obtained. This allows differentiation between ocular and genital *Chlamydia* infections but does not distinguish trachoma from *C. trachomatis* genital serovar ocular infections (i.e. inclusion conjunctivitis and paratrachoma).

Trachoma control programs/activities

Australian and WHO trachoma guidelines identified by this study are summarised in Table 3. The Northern Territory and several Western Australian regions had trachoma control programs and trachoma control guidelines. The *Central Australian Remote Practitioners Association (CARPA) guidelines* and *Therapeutic Guidelines: Antibiotic* were also used in clinical management of trachoma.[22,24] Few practitioners reported knowledge or use of the OATSIH *Specialist Eye Health Guidelines for use in Aboriginal and Torres Strait Islander Populations* or those developed by Couzos and Taylor.[21] All trachoma control guidelines commonly used in Australia are based on the 1993 WHO guidelines, and do not include changes recommended by the WHO in 2004.

Table 3. Comparison of the 'A', 'F' and 'E' components of the WHO and Australian trachoma control guidelines*

Source	Screening target group	Screening interval	Screening time	Consent process	Recommended treatment/interventions		
					Hyperendemic, prevalence > 20%	Endemic, prevalence 5–19%	Non-endemic, prevalence < 5%
WHO 1993[†,2]	Children 1–10 years	Annual	Not specified	Not specified	Topical antibiotic treatment to all community members Systemic antibiotics to severe cases Face-washing and environmental health improvement	Topical antibiotic treatment to all community members or to cases and their families Face-washing and environmental health improvement	Topical antibiotic treatment for cases only
WHO 2003[†,20]	Children 1–9 years	Every 3 years	Not specified	Not specified	If prevalence ≥ 10%: annual antibiotic treatment of all community members aged > 6 months until prevalence < 5%, aim for 80% coverage of eligible population; hygiene promotion and environmental improvement to achieve 80% of children with clean faces		If prevalence < 10%: antibiotic treatment of cases and their families
OATSIH, Australia 2001[‡,3]	Children 2–7 years	Not specified	Not specified	Not specified		Azithromycin to cases and their family/ household (i.e. people who live together or share a sleeping area), repeat treatment annually until active trachoma disappears If prevalence > 20%, it may be simpler to treat the entire community Promote environmental health and facial cleanliness	

Source	Screening target group	Screening interval	Screening time	Consent process	Recommended treatment/interventions		
					Hyperendemic, prevalence > 20%	Endemic, prevalence 5–19%	Non-endemic, prevalence < 5%
Aboriginal primary health care 2003[21]	Children 1–9 years	Not specified	Not specified	Not specified		As for OATSIH, above	
Therapeutic guidelines: antibiotic 2003[22]	–	–	–	–	Community-based treatment program using azithromycin	Azithromycin to cases and their household contacts	
Centre for Disease Control NT 1998†,[23]	School aged children in remote areas, as part of Healthy School Aged Kids Program	Annual if trachoma prevalence > 5%	Not specified	Written consent for screening Verbal consent from individual caregivers for treatment	Azithromycin to all children > 6 months of age and their female care-givers within 14 days after screening and at six months Health promotion	Azithromycin to cases and their household contacts	Azithromycin to cases only
CARPA 2003†,§,[24]	–	–	–	–		Azithromycin to cases and their household contacts within 14 days Erythromycin twice daily for 14 days if < 6 months Check with local CDC for who else needs treatment Encourage face and hand washing	
Kimberley Population Health Unit WA 2004†,[13]	School aged children and household members of school aged children with TF	Annual Cease screening if prevalence < 5% for 5 consecutive years	Sept, Oct, Nov	Varies from written individual consent to verbal community consent	Azithromycin to all school children with TF and their household contacts aged 5–16 years, and to all children aged 1–4 years Environmental health and health promotion	Azithromycin to all school children with TF and their household contacts aged 1–16 years found to have trachoma Environmental health and health promotion	Azithromycin to all school children with TF and their household contacts aged 1–16 years found to have trachoma Environmental health and health promotion

Source	Screening target group	Screening interval	Screening time	Consent process	Recommended treatment/interventions		
					Hyperendemic, prevalence > 20%	Endemic, prevalence 5–19%	Non-endemic, prevalence < 5%
Pilbara Population Health Unit WA 2004†,[25]	School children Children 6 months–5 years and school aged household members of children with > 5 trachoma follicles or TI Household members where more than 1 child has ≥ 1 trachoma follicle	Annual if trachoma prevalence > 5%	June, July	Written consent for screening and treatment of school aged children	As for Kimberley Population Health Unit	As for Kimberley Population Health Unit	As for Kimberley Population Health Unit
Mid-West and Murchison Population Health Unit WA 2003†,[27]	As for Pilbara Population Health Unit	Annual if trachoma prevalence > 5%	February, March – completed in one week	Written consent for screening Verbal consent from individual caregivers for treatment	Azithromycin to all community members > 6 kg Environmental health and health promotion	Azithromycin to children with TF and their family members. Environmental health and health promotion	
Goldfields Population Health Unit WA 2004†,[27]	As for Mid-West and Murchison Population Health Unit	Annual if trachoma prevalence > 5%	May, completed in one week	Community consent for screening Verbal consent from individual caregivers for treatment	As for Midwest Murchison Population Health Unit	As for Midwest Murchison Population Health Unit	

* ‘S’ component not included in comparison as it does not feature strongly in the Australian guidelines used by most staff working in trachoma control programs.

† Guidelines used by staff working in Australian trachoma control programs.

‡ Office of Aboriginal and Torres Strait Islander Health (OATSIH).

§ Central Australian Remote Practitioners Association (CARPA).

In the Northern Territory, trachoma control was incorporated into the school health program which targets children living in remote communities and includes immunisation and annual screening for trachoma and a variety of other medical conditions. The program did not apply in the capital or major towns. Population health staff provided support for program implementation at the request of primary health care staff. Implementation of the program occurred annually in some communities, and irregularly and/or in piecemeal fashion in others.

Despite the absence of a state-wide approach to trachoma control in Western Australia, trachoma control programs had been established in four regional population units since the late 1980s/early 1990s. Population health units assumed responsibility for clinical leadership (through the development and distribution of regional trachoma control program guidelines and the provision of staff education and resources for staff and community education) and coordinating a regional approach to program implementation. Primary health care staff were responsible for trachoma screening, treatment and community education. There was general consensus that a regional approach, led by the population health unit working in collaboration with primary health care services, was crucial to the success and sustainability of trachoma control. Within each region, trachoma control was implemented in school settings over a specific one week to two month period with the aim of reducing the pool of active infection by screening and treating as many people in as short a time period as possible to prevent reinfection from untreated individuals in neighbouring areas. Programs were scheduled to occur during, or as soon as possible before, the maximal fly breeding season and took into account logistical constraints such as school holidays/camps, cyclones, flooding and customary law.

No statewide, regional or local trachoma control programs were identified in Victoria, the Australian Capital Territory, Tasmania, Queensland, South Australia or New South Wales.

Since all trachoma control programs currently in operation in Australia target school aged children, there has been relatively little focus on blinding trachoma or the 'S' component of the SAFE strategy. Screening and treatment for trichiasis were systematically implemented only in the Kimberley region of Western Australia.

Discussion

Transmission of active trachoma and occurrence of trichiasis in Aboriginal and Torres Strait Islander communities is still a reality in 21st century Australia. Large differences in trachoma prevalence were reported from different regions and from different times within the same region. In contrast with developing countries where active trachoma and trichiasis are more common among adult women than men, Australian surveys have identified equal prevalence in both sexes.[3,7,14,16,28–30] There were no consistently implemented state or territory level trachoma control programs. Trachoma control programs led by regional population health units working in collaboration with primary health care services were more likely to be consistently implemented over long periods of time.

Although Table 1 reports regional active trachoma prevalence, lack of standardisation of numerators and denominators between different regions means that it cannot be assumed that these data accurately reflect active trachoma prevalence in children less than 10 years in all Aboriginal and Torres Strait Islander communities within these regions. Reasons for this include:

- In some regions non-Aboriginal and Torres Strait Islander children were included in the surveys and their results included in prevalence calculations. They usually contribute very few, if any cases to the numerator but were not included in the denominator (except where specified in the Murchison region of Western Australia). This may result in an overestimation of regional trachoma prevalence within Aboriginal and Torres Strait Islander populations.

- In most regions where trachoma is known to be endemic, large populations of Aboriginal and Torres Strait Islander people live in areas where trachoma is, or is believed to be, no longer endemic, e.g. Broome, Darwin and Alice Springs. These populations were not included in the denominator when calculating regional trachoma prevalence. This may result in overestimation of regional Aboriginal and Torres Strait Islander trachoma prevalence.

- Age groups targeted for screening tend to be school aged children, but are not uniform across regions. Thus prevalence calculations may include data from children aged less than 10 years, while children aged one to four years are likely to be under-represented, if represented at all.

- Screening coverage of the target populations is generally low, so observed prevalence may not truly reflect population prevalence. For example screening coverage was 65 per cent, 67 per cent and 63 per cent in 1999, 2000 and 2001 respectively in the Kimberley, 71 per cent and 77 per cent in 1995 and 1996 respectively in the Katherine region, and 24 per cent in Central Australia in 1998, 1999 and 2000.[31–34]

A similar lack of standardisation of survey methods and prevalence calculations also applies to trichiasis data.

Some health professionals suggest that trachoma is no longer as prevalent or severe as previously in many regions and that trachoma may be over diagnosed in Australia. This may be attributed to the fact that clinical signs of active trachoma are not specific to trachoma, genital *Chlamydia* is highly endemic in many trachoma-endemic areas and may be present in ocular swabs,[35] and trichiasis appears to be absent from some trachoma-endemic communities. However, a recent Northern Territory study identified *C. trachomatis* isolates corresponding to serovars Ba and C (i.e. trachoma, not genital, serovars) from ocular swabs.[36] Genotyping of ocular swabs from Kimberley children presenting with conjunctivitis also confirmed the presence of *C. trachomatis* serovars Ba and C (personal communication, Drs Michelle Porter and David Smith, PathWest). Furthermore, recent trichiasis prevalence surveys indicate that trichiasis is still present in Central Australia and parts of the Kimberley region.[7,14,16,18]

Throughout Australia there is anecdotal, but no published, evidence that TI and TS are decreasing, indicating that trachoma may be decreasing in intensity and therefore less likely to result in trichiasis in later life.[8] Thus, TI and TT prevalence data should be collected as a routine component of trachoma control.

National guidelines for the public health management of trachoma have been developed by the Communicable Disease Network Australia (Box).[37] These guidelines are consistent with the spirit of, but do not replicate, the WHO guidelines because high level evidence from randomised controlled trials or meta-analyses of randomised controlled trials exists only for the 'S' component of the SAFE strategy, and because it was considered important that Australian guidelines reflect Australian experiences of trachoma control.[28,38,39,40] As described in this paper, there is considerable trachoma control activity underway within Australia. The new Australian guidelines will promote consistent trachoma screening and control programs, and result in a strengthened national collaboration in surveillance and data collection.

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Box. Key recommendations from Guidelines for the public health management of trachoma[37]

	Recommendation
	Trachoma control should be the responsibility of government-run regional population health units and be organised on a regional basis where population mobility is high. Primary health care services should be involved in the detection and treatment of trachoma under the coordination of population health units. Trachoma control activities should be planned and implemented in consultation with community representatives and other key stakeholders. Areas with the highest number of persons with active trachoma and areas with the highest prevalence of active trachoma should be prioritised for trachoma control. Regional population health units should collect trachoma data in accordance with the minimum national trachoma dataset.
Surgery	Regional population health units, primary health care services and specialist eye health services need to decide, collaboratively, on the best way of identifying patients with trichiasis in their region and the best system to ensure that these patients have access to timely surgical referral and treatment. In regions where trachoma is endemic but trichiasis prevalence is unknown, the burden of trichiasis should be quantified. In areas where trachoma or trichiasis is or has been endemic, Aboriginal and Torres Strait Islander people aged 40–54 years should be screened every two years and those aged 55+ years should be screened annually for trichiasis as part of an adult health check Patients with trichiasis should be referred to an ophthalmologist for surgical intervention. Following trichiasis surgery, patients should be followed up annually so that recurrences can be detected promptly
Antibiotics	The minimum target group for active trachoma screening should be Indigenous children aged 5–9 years living in communities/towns where trachoma is endemic. In communities where trachoma is endemic, annual screening for active trachoma is recommended until active trachoma prevalence is < 5% for 5 consecutive years, after which annual screening should cease. All children found to have active trachoma (TF and/or TI) should be treated with single-dose azithromycin If ≥ 10% of screened Aboriginal and Torres Strait Islander children aged < 10 years have active trachoma and there is no obvious clustering of cases, single-dose azithromycin is recommended for all Aboriginal and Torres Strait Islander children in the community aged 6 months to 14 years *and* all household contacts aged 6 months or more. If ≥ 10% of screened Aboriginal and Torres Strait Islander children aged < 10 years have active trachoma *and* cases are obviously clustered within several households *and* health staff can easily identify all household contacts of cases, single-dose azithromycin[a] is recommended for all household contacts aged 6 months or more *only.* Community wide treatment is not required If < 10% of screened Aboriginal and Torres Strait Islander children aged < 10 years have active trachoma, single-dose azithromycin is recommended for all household contacts aged 6 months and over Antibiotic treatment of cases, household contacts and community members (when required) should be completed within two weeks of screening. In regions where population mobility is high, all screening and treatment activities within the region should be completed in as short a time frame as possible to minimise the likelihood re-infection and to achieve higher population coverage.
Facial cleanliness	Facial cleanliness in children should be promoted by including regular face-washing as part of a holistic personal hygiene program.
Environmental health	Environmental health, school and health promotion staff should be involved as key stakeholders when regional population health units and primary health care services plan and implement trachoma control activities so that 'F' and 'E' strategies appropriate to individual communities/ regions can be implemented.

A report from the Communicable Diseases Network Australia

1 January to 31 March 2006

The Communicable Diseases Network Australia (CDNA) consists of communicable disease authorities from various Australian Government agencies and state and territory health authorities, in addition to expert bodies and individuals in the specific areas of communicable disease epidemiology, clinical management, disease control and laboratory diagnosis. The CDNA provides national public health leadership and co-ordination on communicable disease surveillance, prevention and control, and offers strategic advice to governments and other key bodies on public health actions to minimise the impact of communicable diseases in Australia and the region.

During the first quarter of 2006, CDNA considered the following issues:

Pre-departure communicable diseases health screening protocol for refugees

In February, CDNA endorsed the *Pre-Departure Communicable Diseases Health Screening Protocol for Refugees Arriving from Africa* for use by the International Office of Migration which conducts pre-departure health screening on behalf of the Australian Government Department of Immigration and Multicultural Affairs. In March, this was followed by endorsement of *Pre-Departure Communicable Diseases Health Screening Protocol for Refugees Arriving from the Thai/Burmese Border*.

Australian Quarantine and Inspection Service work instruction – processing ill passengers and those requiring yellow fever certification

State and territory health authorities reported an increase in calls from the Australian Quarantine and Inspection Service (AQIS) regarding passengers arriving from overseas with diarrhoea. CDNA provided advice to the Australian Government Department of Health and Ageing officers liaising with the Australian Customs Service and AQIS.

HIV/AIDS, STI and hepatitis C health promotion programs

In March, CDNA endorsed *Infrastructure Benchmarks for the Design, Implementation and Evaluation of HIV/AIDS, STI and Hepatitis C Health Promotion Programs.* The benchmarks document was developed by the Inter-Governmental Committee on AIDS, Hepatitis C and Related Diseases.

Review of non-Medicare funded pathology services

CDNA approved Dr Rod Givney as their representative in a review of non-Medicare funded pathology services. The review is being undertaken by the Australian Government Department of Health and Ageing. This appointment was made in March and is current for the term of the review.

New Chair – National Tuberculosis Advisory Committee

Following his retirement as Chair of the National Tuberculosis Advisory Committee (NTAC), CDNA members thanked Dr Antic for his significant contributions over many years to the management, prevention and control of tuberculosis. CDNA endorsed Dr Vicki Krause as the new Chair of NTAC on 29 March 2006.

New Chair – Case Definitions Working Group

CDNA noted Dr Gary Dowse had resigned as Chair of the Case Definitions Working Group and thanked Gary for his contribution to the development of case definitions used within the National Notifiable Diseases Surveillance System. Working Group member Dr Mark Bartlett was endorsed as the new Chair of the Case Definitions Working Group on 29 March 2006.

Surveillance systems

CDNA continues to provide policy and implementation advice to the Biosecurity Surveillance System project team and the Australian Government Department of Health and Ageing. Issues surrounding surveillance, privacy, outbreak case reporting and user testing are focus points for CDNA members, in particular the CDNA jurisdictional members. CDNA is also providing policy and implementation advice regarding the Syndromic Surveillance System being developed by the Australian Government Department of Health and Ageing.

Pandemic influenza

Jurisdictional representatives from CDNA continue to be involved in planning for possible pandemic influenza through their membership of the National Influenza Pandemic Action Committee Jurisdictional Executive Group. Teleconference meetings have allowed the group to consider issues which will be directly relevant at the jurisdictional level if a pandemic were to occur. Issues such as communication strategies, antiviral availability and registers, social distancing and border screening are under consideration.

Office of Health Protection

In February 2006 the Australian Government established a new division within the Department of Health and Ageing – the Office of Health Protection (OHP). OHP will consolidate and build on the work already undertaken by the Government to manage communicable diseases and maintain Australia's biosecurity. In particular, the OHP will lead national health preparedness for a possible pandemic or other health emergency. CDNA secretariat services are resourced through OHP.

Note: Documents published by CDNA can be viewed at www.health.gov.au/cdna.

Letter to the Editor: The limitation of fever in case definitions for avian influenza and SARS

Two emerging infectious diseases, namely avian influenza and severe acute respiratory syndrome (SARS), have recently brought infectious diseases to the forefront of the public eye. Case definitions for both infections from most health authorities require a fever greater than or equal to 38º C (100.4º F) before the individual can even be considered as a suspect case.

One definition of fever is as follows:

'An early morning oral temperature of 37.2º C (99º F) or an oral temperature above 37.8º C (100º F) at other times of the day.'[1]

In other words, fever varies according to time of day and anatomical site. It also varies between individuals.

Fever is one of the most common symptoms and signs of illness recognised both by health professionals and lay people. Temperature is an integral part of the 'vital observations' measured in hospitalised patients. Yet, given its diurnal and anatomical variation at so many different anatomical sites, doctors have struggled to correctly define fever.[2]

The difficulties outlined above are relevant to its role in the case definitions for avian influenza and SARS. Firstly, health authorities should specify in their case definitions at what anatomical site the temperature is taken. This is because an oral temperature of 38º C is different to 38º C measured at other sites, such as the tympanic membrane, rectum, axilla and forehead. The anatomical site for measurement of temperature in hospitals will vary between countries. While some studies provide approximate conversion factors to relate temperature at one site to another, these can be highly variable and not necessarily reliable.[1] Furthermore, in pandemic situations, case definitions will be used by lay people to monitor themselves for signs of illness, making it even more important for health authorities to clarify how to identify fever.

The diurnal variation of fever highlights the limitations of a single temperature measurement to exclude cases. This has implications for border screening, where a single normal temperature measurement should not exclude the disease in someone who otherwise meets the case definition. In fact, there were SARS patients who had a normal temperature on admission to hospital.[3]

Fever can often be identified from the history alone through descriptions such as drenching sweats, 'hot and cold' episodes, shivers and severe uncontrolled shaking (rigors). Therefore, it is pleasing to see that the Australian Government has included 'fever…. **OR** *history of fever*' in its case definition for avian influenza.[4] However, it is not clear whether 'history of fever' only refers to a numeric temperature or to symptoms consistent with fever, and if it does refer to symptoms, what are they?

This in no way should belittle the concept of a case definition—it is an important epidemiological tool, which provides an objective way of identifying cases. However, it should be remembered that defining fever as a single measurement with no anatomical reference, might lead to confusion and failure to identify cases. The inclusion in a case definition of the specific symptoms attributed to fever and fever defined according to different anatomical sites, might overcome this difficulty.

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Communicable diseases surveillance

Highlights for 1st quarter, 2006

Communicable disease 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 or 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', and those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.

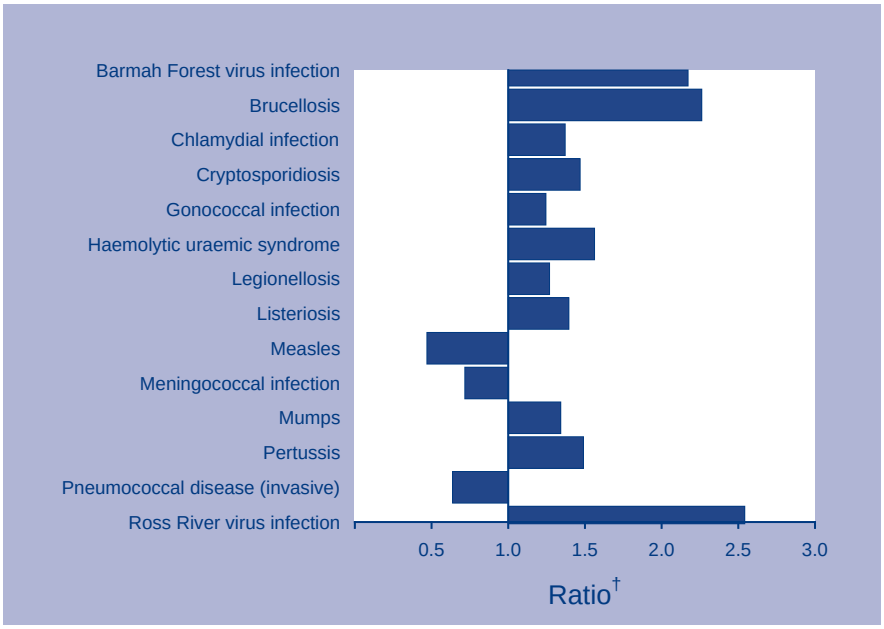
Figure 1 shows the changes in select disease notifications with an onset in the first quarter of 2006, compared with a five year mean for the same period. The following diseases were above the five year mean: cryptosporidiosis, haemolytic uraemic syndrome, listeriosis, chlamydial infection, gonococcal infection, mumps, pertussis, Barmah Forest virus infection, Ross River virus infection, brucellosis and legionellosis. Diseases for which the number of notifications was below the five year mean for the same period include hepatitis A, measles, pneumococcal disease and meningococcal infection.

Gastrointestinal illnesses

Cryptosporidiosis

There were 1,454 notifications of cryptosporidiosis during the quarter which was 1.5 times the five year mean for the same period. All jurisdictions reported cases but the majority were from New South Wales, Victoria and Queensland. This continued a trend reported in the last quarter. One thousand and

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 January to 31 March 2006 with historical data



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

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

twenty-two notifications (70%) had information on the infecting species and all were identified as *Cryptosporidium parvum* infection.

A report for the Communicable Diseases Network of Australia on the increases in cryptosporidiosis in this quarter concluded that seasonal increases in cryptosporidiosis had been higher and more prolonged in 2005 and 2006 compared to the previous four years. Cryptosporidiosis had increased in New South Wales and Victoria and declined in Queensland although this State continues to report a significant proportion of all cases. A relative increase was observed in the proportion of notifications in cases aged more than four years compared with those aged less than four years, particularly in Victoria (Rhonda Owen, personal communication).

Listeriosis

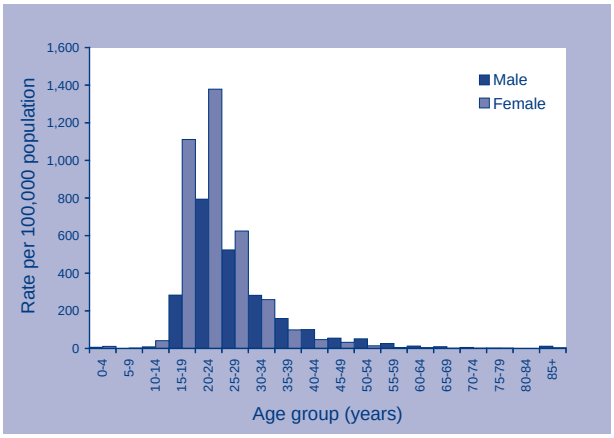
There were 24 cases of listeriosis reported to NNDSS in the first quarter which was 1.4 times the five year mean. Nine cases were from New South Wales and eight from Western Australia. The cases occurred in the elderly (19 of the 24 cases were aged 60 years or more) and in people with underlying medical conditions. OzFoodNet undertook investigations for a common food source (see OzFoodNet report).

Sexually transmissible infections

Chlamydial infections

There were 10,492 notifications of chlamydial infection in the quarter which was 1.4 times the five year mean. The highest rates of notification continue to be among young women (1,378 cases per 100,000 population) and men (794 cases per 100,000 population) (Figure 2).

Figure 2. Notification rates of chlamydial infections, Australia, 1 January to 31 March 2006, by age group and sex



Vaccine preventable diseases

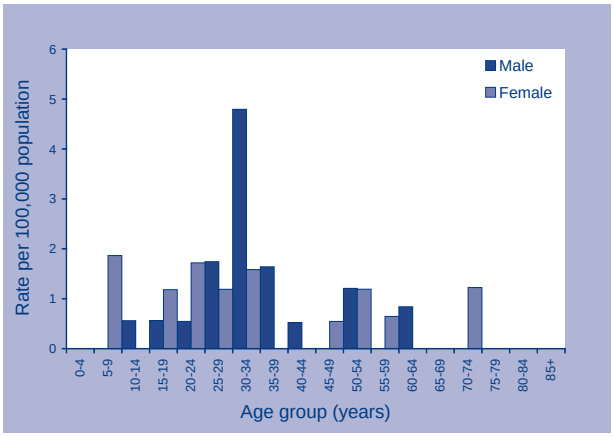
Measles

There were 11 cases of measles reported in the quarter. Cases were reported from New South Wales (5 cases), Victoria (4 cases) and one case each in South Australia and Western Australia. Of the 11 cases, seven were male and four female; three were aged less than five years and the remainder were aged between 20 and 30 years. All of the seven cases with vaccination status recorded were unvaccinated.

Mumps

There were 40 notifications of mumps in the quarter, which was 1.3 times the five year mean for the same period. There were 22 male and 18 female cases with an age range from 6 to 72 years (Figure 3).

Figure 3. Notification rates of mumps, Australia, 1 January to 31 March 2006, by age group and sex



The vaccination status data in the notifications showed that one case was partially vaccinated, 22 were not vaccinated while the vaccination status for the remaining 17 cases was unknown.

Pertussis

Two thousand two hundred and forty-eight pertussis notifications were received in the quarter which was 1.5 times the five year mean for the same period. Of the total number of notifications, 1,059 were reported by New South Wales and 467 were from Queensland. Of the 2,248 notifications 1.4 per cent (32 cases) were reported in infants aged less than one year. The highest rate of infection in females (100 cases per 100,000 population) occurred in the

50–54 year age group. The highest rate in males was 73 cases per 100,000 population in the 65–69 year age group.

Vectorborne diseases

Barmah Forest virus infection

There were 703 cases of Barmah Forest virus infection in the fourth quarter which was 2.2 times the five year mean for the same period. The majority of cases were from Queensland (265 cases) and New South Wales (265 cases). Nationally, the infection rate was 13.8 cases per 100,000 population, but it was higher in the Northern Territory at 96.7 cases per 100,000 population (49 cases) and Queensland with 26.7 cases per 100,000 population.

Ross River virus infection

This quarter, 3,228 notifications of Ross River virus infection were reported compared to 928 in the same period last year. The majority of cases were from Queensland (1,583) and New South Wales (680), while the notification rates were above the national rate in the Northern Territory, Queensland and Western Australia.

Other bacterial infections

Legionellosis

There were 98 cases of legionellosis reported in the first quarter, which was 1.3 times the five year mean. Twenty-nine cases were reported in both New South Wales and Victoria, which included cases in outbreaks in metropolitan Sydney and Melbourne respectively.

Meningococcal infection

There were 74 notifications of meningococcal infection in the quarter which was 70 per cent of the five year mean. Of the 74 cases, 44 (59%) were serogroup B, 7 (9%) were serogroup C, 3 were serogroup W135, 1 was serogroup Y and the serogroups of the remaining 17 cases was unknown. There were two deaths reported in the quarter, both in patients with serogroup B disease.

Of the serotype C cases, two were eligible for vaccination – one of these was a fully vaccinated 15-year-old, and one was a 5-year-old who was not vaccinated. The remaining cases were aged 24 to 74 years. Serogroup C disease continues to decline in Australia as a result of the National Meningococcal C Vaccination Program which commenced in 2003 and completed vaccination of all under 19-year-olds by the end of 2004.

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 34,999 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 January and 31 March 2006 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 5,543 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 January to 31 March 2006 (Tables 4 and 5).

Table 1. Reporting of notifiable diseases by jurisdiction

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

Disease	Data received from:
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)*	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
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 2006, by date of onset*

Disease	State or territory								Total 1st quarter 2006†	Total 4th quarter 2005	Total 1st quarter 2005	Last 5 years mean 1st quarter	Year to date 2006	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	1	13	3	14	0	2	18	11	62	53	75	88.0	62	87.6	0.7
Hepatitis B (unspecified)	22	657	36	243	73	8	401	96	1,536	1,660	1,798	1,652.0	1,536	1,652.4	0.9
Hepatitis C (incident)	0	12	0	0	15	2	28	27	84	79	88	132.0	84	132.2	0.6
Hepatitis C (unspecified)	51	1,634	73	803	112	82	697	273	3,725	3,573	3,473	3,986.0	3,725	3,986.4	0.9
Hepatitis D	0	2	0	3	0	0	0	0	5	6	4	5.0	5	4.8	1.0
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	1	1.0	0	0.5	0.0
Campylobacteriosis§	110	NN	54	907	429	135	1,302	573	3,510	4,979	4,177	4,068.0	3,510	4,067.6	0.9
Cryptosporidiosis‖	39	397	26	333	78	11	498	72	1,454	785	1,257	990.0	1,454	989.8	1.5
Haemolytic uraemic syndrome	0	4	0	0	1	0	0	0	5	8	4	3.0	5	3.2	1.6
Hepatitis A	2	35	13	17	3	1	15	7	93	68	82	112.0	93	112.2	0.8
Hepatitis E	1	2	0	0	0	0	2	0	5	2	17	8.0	5	7.6	0.7
Listeriosis	0	9	0	0	2	0	5	8	24	18	13	17.0	24	17.2	1.4
Salmonellosis (NEC)	36	719	93	1,167	195	81	464	270	3,025	2,461	2,720	2,702.0	3,025	2,701.8	1.1
Shigellosis	1	25	28	40	11	1	21	52	179	171	228	173.0	179	173.4	1.0
SLTEC, VTEC¶	0	3	0	2	10	0	0	0	15	24	15	16.0	15	16.2	0.9
Typhoid	3	7	1	1	1	0	6	3	22	9	23	28.0	22	28.0	0.8
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	2	1.0	0	1.0	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

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2006, by date of onset,* *continued*

Disease	State or territory								Total 1st quarter 2006†	Total 4th quarter 2005	Total 1st quarter 2005	Last 5 years mean 1st quarter	Year to date 2006	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection**	209	3,029	462	2,462	731	255	1,884	1,460	10,492	9,858	10,342	7,650.0	10,492	7,650.2	1.4
Donovanosis	0	0	0	0	0	0	0	0	0	3	5	6.0	0	5.6	0.0
Gonococcal infection	8	474	378	426	117	5	386	421	2,215	1,952	2,052	1,778.0	2,215	1,778.2	1.2
Syphilis (all)	4	251	55	72	12	6	92	45	528	490	538	300.0	537	300.0	1.8
Syphilis < two years duration	0	27	35	25	3	2	22	5	119	125	155	132.0	119	132.0	0.9
Syphilis > two years or unspecified duration	4	224	20	47	0	4	70	40	409	364	383	347.0	409	347.0	1.2
Syphilis - congenital	0	2	1	0	0	0	0	0	3	3	5	3.0	3	3.2	0.9
Vaccine preventable disease															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.2	0.0
Haemophilus influenzae type b	0	1	2	0	0	0	0	0	3	5	7	5.0	3	5.2	0.6
Influenza (laboratory confirmed)‖	4	32	8	86	3	7	10	25	175	417	384	153.0	175	153.2	1.1
Measles	0	5	0	0	1	0	4	1	11	2	5	23.0	11	23.4	0.5
Mumps	0	19	2	6	1	0	7	5	40	37	51	30.0	40	29.8	1.3
Pertussis	76	1,059	26	467	325	8	235	52	2,248	2,819	2,260	1,508.0	2,248	1,508.4	1.5
Pneumococcal disease (invasive)‖	3	60	9	36	12	5	43	15	183	333	268	287.0	183	287.2	0.6
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	0	4	0	2	2	0	0	0	8	3	6	32.0	8	32.2	0.2
Rubella - congenital	0	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	1	0	2.0	0	1.8	0.0
Vectorborne diseases															
Barmah Forest virus infection	6	220	49	265	86	0	9	68	703	296	364	324.0	703	323.6	2.2
Dengue	3	14	11	21	3	0	1	6	59	48	95	155.0	59	155.4	0.4
Flavivirus infection (NEC)	0	0	0	10	0	0	9	0	19	4	9	22.0	19	22.0	0.9
Japanese encephalitis virus‖	0	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	2	1	5.0	0	5.0	0.0
Malaria	5	31	11	91	8	4	27	33	210	140	349	211.0	210	211.4	1.0
Murray Valley encephalitis virus‖	0	0	0	0	0	0	0	1	1	0	2	2.0	1	2.0	0.5
Ross River virus infection	5	680	171	1,583	193	6	164	426	3,228	762	928	1,270.0	3,228	1,269.8	2.5

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2006, by date of onset,* *continued*

Disease	State or territory								Total 1st quarter 2006†	Total 4th quarter 2005	Total 1st quarter 2005	Last 5 years mean 1st quarter	Year to date 2006	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax‖	0	1	0	0	0	0	0	0	1	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	1	0	18	0	0	0	0	19	18	12	8.0	19	8.4	2.3
Leptospirosis	0	6	2	38	0	0	0	0	46	26	40	63.0	46	63.0	0.7
Lyssavirus unspecified‖	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	23	0	1	0	0	8	1	33	28	38	41.0	33	40.6	0.8
Q fever	0	36	0	36	4	0	9	3	88	79	82	160.0	88	159.6	0.6
Other bacterial infections															
Legionellosis	2	29	0	9	7	1	29	21	98	82	88	77.0	98	77.2	1.3
Leprosy	0	0	0	0	0	0	0	0	0	1	5	3.0	0	3.4	0.0
Meningococcal infection††	0	27	2	16	3	2	19	5	74	105	74	103.0	74	103.2	0.7
Tuberculosis	0	62	5	38	16	1	90	30	242	182	231	232.0	242	232.4	1.0
Total	595	9,836	1,576	9,285	2,457	629	6,575	4,055	34,999	32,081	32,756	28,914.0	35,008	28,917.1	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'.

‖ Notifiable from January 2001 only. Ratio and mean calculations are based on the last five years.

¶ 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.

Table 3. Notification rates of diseases, 1 January to 31 March 2006, by state or territory. (Rate per 100,000 population)

Disease*	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis B (incident)	1.2	0.8	5.9	1.4	0.0	1.6	1.4	2.2	1.2
Hepatitis B (unspecified)	27.1	38.8	71.0	24.5	18.9	6.6	31.9	19.1	30.2
Hepatitis C (incident)	0.0	0.7	0.0	0.0	3.9	1.6	2.2	5.4	1.7
Hepatitis C (unspecified)	62.7	96.5	144.0	81.0	29.1	67.6	55.5	54.3	73.3
Hepatitis D	0.0	0.1	0.0	0.3	0.0	0.0	0.0	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	135.3	NN	106.5	91.5	111.3	111.3	103.7	114.0	103.6
Cryptosporidiosis	48.0	23.4	51.3	33.6	20.2	9.1	39.7	14.3	28.6
Haemolytic uraemic syndrome	0.0	0.2	0.0	0.0	0.3	0.0	0.0	0.0	0.1
Hepatitis A	2.5	2.1	25.6	1.7	0.8	0.8	1.2	1.4	1.8
Hepatitis E	1.2	0.1	0.0	0.0	0.0	0.0	0.2	0.0	0.1
Listeriosis	0.0	0.5	0.0	0.0	0.5	0.0	0.4	1.6	0.5
Salmonellosis (NEC)	44.3	42.5	183.4	117.8	50.6	66.8	37.0	53.7	59.5
Shigellosis	1.2	1.5	55.2	4.0	2.9	0.8	1.7	10.3	3.5
SLTEC, VTEC‡	0.0	0.2	0.0	0.2	2.6	0.0	0.0	0.0	0.3
Typhoid	3.7	0.4	2.0	0.1	0.3	0.0	0.5	0.6	0.4
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§	257.1	178.9	911.3	248.4	189.6	210.2	150.0	290.5	206.5
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	9.8	28.0	745.6	43.0	30.3	4.1	30.7	83.8	43.6
Syphilis (all)	0.0	0.0	0.0	0.0	2.3	0.0	0.0	0.0	0.2
Syphilis < 2 years duration	0.0	1.6	69.0	2.5	0.8	1.6	1.8	1.0	2.3
Syphilis > 2 years or unspecified duration	4.9	13.2	39.4	4.7	0.0	3.3	5.6	8.0	8.0
Syphilis - congenital	0.0	0.1	2.0	0.0	0.0	0.0	0.0	0.0	0.1

Table 3. Notification rates of diseases, 1 January to 31 March 2006, by state or territory. (Rate per 100,000 population), *continued*

Disease*	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	3.9	0.0	0.0	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)	4.9	1.9	15.8	8.7	0.8	5.8	0.8	5.0	3.4
Measles	0.0	0.3	0.0	0.0	0.3	0.0	0.3	0.2	0.2
Mumps	0.0	1.1	3.9	0.6	0.3	0.0	0.6	1.0	0.8
Pertussis	93.5	62.5	51.3	47.1	84.3	6.6	18.7	10.3	44.2
Pneumococcal disease (invasive)	3.7	3.5	17.8	3.6	3.1	4.1	3.4	3.0	3.6
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.2	0.0	0.2	0.5	0.0	0.0	0.0	0.2
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vectorborne diseases									
Barmah Forest virus infection	7.4	13.0	96.7	26.7	22.3	0.0	0.7	13.5	13.8
Dengue	3.7	0.8	21.7	2.1	0.8	0.0	0.1	1.2	1.2
Flavivirus infection (NEC)	0.0	0.0	0.0	1.0	0.0	0.0	0.7	0.0	0.4
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	6.2	1.8	21.7	9.2	2.1	3.3	2.2	6.6	4.1
Murray Valley encephalitis virus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Ross River virus infection	6.2	40.2	337.3	159.7	50.1	4.9	13.1	84.8	63.5
Zoonoses									
Anthrax	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	1.8	0.0	0.0	0.0	0.0	0.4
Leptospirosis	0.0	0.4	3.9	3.8	0.0	0.0	0.0	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	1.4	0.0	0.1	0.0	0.0	0.6	0.2	0.6
Q fever	0.0	2.1	0.0	3.6	1.0	0.0	0.7	0.6	1.7
Other bacterial infections									
Legionellosis	2.5	1.7	0.0	0.9	1.8	0.8	2.3	4.2	1.9
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection‖	0.0	1.6	3.9	1.6	0.8	1.6	1.5	1.0	1.5
Tuberculosis	0.0	3.7	9.9	3.8	4.2	0.8	7.2	6.0	4.8

* 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.

NN Not notifiable.

NEC Not elsewhere classified.

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 January to 31 March 2006, and total reports for the year†

	State or territory								This period 2006	This period 2005	Year to date 2006	Year to date 2005
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	4	–	3	1	–	3	–	11	2	11	2
Mumps virus	–	–	1	3	2	–	6	–	12	6	12	6
Rubella virus	–	–	–	–	1	–	–	1	2	4	2	4
Hepatitis virus												
Hepatitis A virus	–	–	1	4	5	–	–	–	10	6	10	6
Hepatitis D virus	–	–	–	–	1	–	–	–	1	2	1	2
Hepatitis E virus	–	–	–	–	–	–	1	–	1	6	1	6
Arboviruses												
Ross River virus	–	29	23	487	186	1	21	36	783	208	783	208
Barmah Forest virus	–	4	–	43	93	–	–	–	140	54	140	54
Flavivirus (unspecified)	–	1	–	21	–	–	6	–	28	13	28	13
Adenoviruses												
Adenovirus not typed/ pending	6	33	–	16	62	–	2	–	119	113	119	113
Herpesviruses												
Herpes virus type 6	–	–	–	–	–	–	1	–	1	1	1	1
Cytomegalovirus	–	46	–	39	201	7	4	–	297	171	297	171
Varicella-zoster virus	1	30	–	236	67	1	9	–	344	364	344	364
Epstein-Barr virus	–	8	23	225	188	1	16	78	539	569	539	569
Other DNA viruses												
Parvovirus	–	3	–	11	31	–	5	–	50	55	50	55
Picornaviruses												
Coxsackievirus A9	–	2	–	–	–	–	–	–	2	1	2	1
Echovirus type 5	–	2	–	–	–	–	–	–	2	–	2	–
Echovirus type 22	–	2	–	–	–	–	–	–	2	–	2	–
Echovirus type 30	–	7	–	–	–	–	–	–	7	9	7	9
Rhinovirus (all types)	–	5	–	–	5	–	–	–	10	82	10	82
Enterovirus not typed/ pending	4	41	–	6	1	–	–	–	52	24	52	24
Ortho/ paramyxoviruses												
Influenza A virus	–	1	–	2	22	–	–	–	25	24	25	24
Influenza B virus	–	1	–	–	2	–	–	–	3	33	3	33
Parainfluenza virus type 1	–	7	–	–	4	–	–	–	11	10	11	10
Parainfluenza virus type 2	–	–	–	–	1	–	–	–	1	6	1	6
Parainfluenza virus type 3	–	3	–	–	7	–	1	–	11	46	11	46
Respiratory syncytial virus	–	12	–	53	6	1	3	–	75	112	75	112
Other RNA viruses												
HTLV-1	–	–	–	–	4	–	–	–	4	2	4	2
Rotavirus	–	7	–	–	32	10	3	–	52	69	52	69
Norwalk agent	–	–	–	–	–	–	181	–	181	15	181	15

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 January to 31 March 2006, and total reports for the year,† *continued*

	State or territory								This period 2006	This period 2005	Year to date 2006	Year to date 2005
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other												
Chlamydia trachomatis not typed	6	204	–	648	533	19	21	2	1,433	1,191	1,433	1,191
Chlamydia psittaci	–	–	–	–	–	–	7	–	7	14	7	14
Mycoplasma pneumoniae	1	6	4	137	105	11	51	20	335	258	335	258
Mycoplasma hominis	–	10	–	–	–	–	–	–	10	1	10	1
Coxiella burnetii (Q fever)	–	1	–	17	24	–	5	–	47	35	47	35
Rickettsia - spotted fever group	–	–	–	–	56	–	–	–	56	49	56	49
Streptococcus group A	–	5	–	101	–	–	31	–	137	104	137	104
Yersinia enterocolitica	–	2	–	1	–	–	–	–	3	4	3	4
Brucella species	–	–	–	2	–	–	–	–	2	2	2	2
Bordetella pertussis	1	6	3	49	301	1	62	–	423	386	423	386
Legionella pneumophila	–	3	–	–	3	–	1	–	7	7	7	7
Legionella longbeachae	–	–	–	–	5	–	1	–	6	12	6	12
Cryptococcus species	–	1	–	2	7	–	–	–	10	10	10	10
Leptospira species	–	2	–	1	4	–	–	–	7	3	7	3
Treponema pallidum	–	7	2	145	110	–	1	–	265	252	265	252
Toxoplasma gondii	–	3	–	4	4	3	2	–	16	10	16	10
Echinococcus granulosus	–	–	–	–	3	–	–	–	3	5	3	5
Total	19	498	57	2,256	2,077	55	444	137	5,543	4,350	5,543	4,350

* 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 2006*

State or territory	Laboratory	January 2006	February 2006	March 2006	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	116	118	153	387
	New Children's Hospital, Westmead	19	20		39
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	–	–	–	–
	South West Area Pathology Service, Liverpool	–	–	–	–
Queensland	Queensland Medical Laboratory, West End	794	769	802	2,365
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	700	645	753	2,098
Tasmania	Northern Tasmanian Pathology Service, Launceston	24	17	11	52
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	6			6
	Royal Children's Hospital, Melbourne	39	28	50	117
	Victorian Infectious Diseases Reference Laboratory, Fairfield	147	87	63	297
Western Australia	PathCentre Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	78	88	16	182
Total		1,923	1,772	1,848	5,543

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

– No data received this period.

Additional reports

Australian Sentinel Practice Research Network

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

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

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

In 2006, six conditions are being monitored, four of which are related to communicable diseases. These include influenza, gastroenteritis, varicella and shingles. There are two definitions for influenza for 2006. A patient may be coded once or twice depending on their symptoms. The definition for influenza 1 will include more individuals. Definitions of these conditions were published in Commun Dis Intell *2006;30:158.*

Data from 1 January to 31 March 2006 compared with 2005 are shown as the rate per 1,000 consultations in Figures 4, and 5.

Figure 4. Consultation rates for influenza-like illness, ASPREN, 1 January to 31 March 2006, by week of report

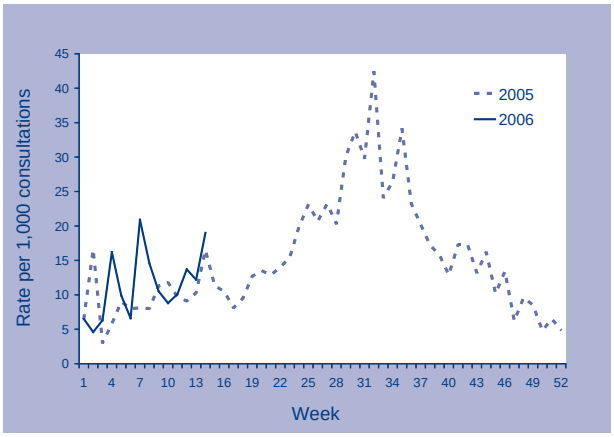
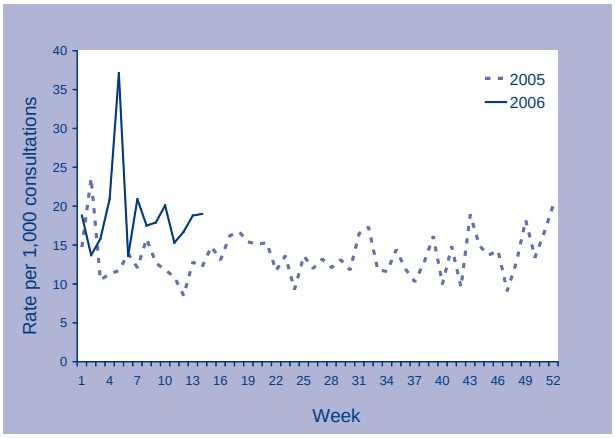


Figure 5. Consultation rates for gastroenteritis, ASPREN, 1 January to 31 March 2006, by week of report



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 *2005;29:93.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 January to 31 March 2006, are included in this issue of Communicable Diseases Intelligence *(Table 6).*

Table 6. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2006, by jurisdiction and serogroup

Jurisdiction	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	06													0	*0*
	05			1	*1*	1	*1*							2	*2*
	04					2	*2*							2	*2*
New South Wales	06			9	*9*	1	*1*			1	*1*	4	*4*	14	*14*
	05			15	*15*	7	*7*	1	*1*			1	*1*	24	24
	04			19	*19*	5	*5*	1	*1*	1	*1*	5	*5*	31	*31*
Northern Territory	06			1	*1*									1	*1*
	05			1	*1*									1	*1*
	04			3	*3*									3	*3*
Queensland	06			15	*15*	1	*1*							16	*16*
	05			10	*10*	5	*5*							15	*15*
	04			12	*12*	7	*7*					2	*2*	21	*21*
South Australia	06			3	*3*									3	*3*
	05					2	*2*							2	*2*
	04			4	*4*									4	4
Tasmania	06			1	*1*	1	*1*							2	*2*
	05													0	*0*
	04			2	2							2	*2*	4	*4*
Victoria	06			10	*10*	2	*2*	1	*1*	2	*2*			15	*15*
	05			7	*7*	1	*1*			2	*2*	1	*1*	11	*11*
	04			9	*9*	4	*4*	2	*2*			1	*1*	16	*16*
Western Australia	06			5	*5*									5	*5*
	05			5	5			1	*1*					6	*6*
	04			4	*4*	1	*1*							5	*5*
Total	06			44	*44*	5	*5*	1	*1*	3	*3*	4	*4*	57	*57*
	05			39	*39*	16	*16*	2	*2*	2	*2*	2	*2*	61	*61*
	04			53	*53*	19	*19*	3	*3*	1	*1*	10	*10*	86	*86*

Q1 = 1st quarter.

YTD = Year to 31 March 2006.

HIV and AIDS surveillance

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

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW

2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell *2005;29:91–92.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 October and 31 December 2005, as reported to 31 March 2006, are included in this issue of Communicable Diseases Intelligence *(Tables 7 and 8).*

Table 7. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 October to 31 December 2005, 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 2005	This period 2004	YTD 2005	YTD 2004
HIV diagnoses	Female	0	4	0	2	2	0	9	3	20	35	95	121
	Male	0	88	0	33	11	0	64	13	209	193	861	774
	Not reported	0	0	0	0	0	0	0	0	0	0	0	1
	Total*	0	92	0	35	13	0	73	16	229	228	956	897
AIDS diagnoses	Female	0	0	1	0	0	0	0	0	1	7	26	21
	Male	0	12	0	9	1	0	12	3	37	42	168	156
	Total*	0	12	1	9	1	0	12	3	38	49	194	179
AIDS deaths	Female	0	2	0	0	0	0	0	0	2	1	5	7
	Male	0	5	0	4	1	0	3	1	14	27	56	83
	Total*	0	7	0	4	1	0	3	1	16	28	61	90

* Totals include people whose sex was reported as transgender.

Table 8. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 31 December 2005, and reported by 31 March 2006, by sex and state or territory

	Sex	State or territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	30	819	18	244	89	8	341	182	1,731
	Male	252	13,096	125	2,592	881	95	4,993	1,157	23,191
	Not reported	0	231	0	0	0	0	22	0	253
	Total*	282	14,174	143	2,845	971	103	5,375	1,346	25,239
AIDS diagnoses	Female	10	244	3	68	31	4	105	36	501
	Male	92	5,296	41	1,010	393	50	1,925	418	9,225
	Total*	102	5,557	44	1,080	425	54	2,040	456	9,758
AIDS deaths	Female	7	134	1	41	20	2	59	24	288
	Male	71	3,552	26	652	273	32	1,385	292	6,283
	Total*	78	3,696	27	695	293	34	1,452	317	6,592

* Totals include people whose sex was reported as transgender.

Childhood immunisation coverage

Tables 9, 10 and 11 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 2004, at 24 months of age for the cohort born between 1 October and 31 December 2003, and at 6 years of age for the cohort born between 1 October and 31 December 1999 according to the Australian Standard Vaccination Schedule.

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

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

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia decreased for the first time in 12 months, a drop of 0.8 percentage points to 90.2 per cent (Table 9). Coverage for all individual vaccines due at 12 months of age decreased by 0.5–0.7 percentage points. The only significant movements in coverage for individual vaccines by jurisdiction was in Tasmania, where coverage for all four vaccines due at 12 months decreased by 1.6–2.2 percentage points.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia did not change from the last quarter, remaining at 92.1 per cent (Table 10). Similarly, there were no significant changes in coverage in any jurisdiction for 'fully immunised' coverage or for coverage for individual vaccines.

Table 9. 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 2004; assessment date 31 March 2006

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,019	21,277	774	12,317	4,263	1,408	15,517	6,012	62,587
Diphtheria, tetanus, pertussis (%)	92.9	91.7	91.9	91.4	91.8	92.8	92.1	90.8	91.7
Poliomyelitis (%)	92.8	91.6	91.7	91.4	91.7	92.8	92.0	90.7	91.6
Haemophilus influenzae type b (%)	94.9	93.5	96.4	93.6	94.4	93.4	94.1	93.7	93.8
Hepatitis B (%)	95.6	94.6	96.8	94.1	94.7	93.5	94.0	93.7	94.3
Fully immunised (%)	92.2	90.0	91.5	90.3	90.6	91.2	90.3	89.3	90.2
Change in fully immunised since last quarter (%)	-1.6	-0.6	+1.4	-0.8	-0.7	-2.2	-1.7	+0.6	-0.8

Table 10. 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 2003; assessment date 31 March 2006*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,086	21,739	849	12,867	4,424	1,501	15,926	6,269	64,661
Diphtheria, tetanus, pertussis (%)	95.8	95.0	96.8	94.7	95.1	97.0	95.8	93.6	95.1
Poliomyelitis (%)	95.5	94.9	96.7	94.7	95.1	97.1	95.7	93.6	95.0
Haemophilus influenzae type b (%)	93.7	93.1	95.1	93.5	93.9	95.1	94.4	91.6	93.5
Measles, mumps, rubella (%)	93.5	93.4	95.9	93.3	94.3	95.5	94.9	92.4	93.8
Hepatitis B(%)	96.2	95.9	97.5	95.3	96.1	97.8	96.5	94.9	95.9
Fully immunised (%)	92.1	91.6	94.4	91.8	92.7	94.4	93.2	90.1	92.1
Change in fully immunised since last quarter (%)	-2.7	-0.1	+1.2	-0.1	+1.7	-0.0	+0.8	-1.3	-0.0

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2005;29:219.

Table 11 shows immunisation coverage estimates for 'fully immunised' and for individual vaccines at 6 years of age for Australia by state or territory. This was largely unchanged in all jurisdictions except for Tasmania. Coverage for all vaccines due at 6 years of age in Tasmania decreased by 3 percentage points. However, Tasmania is not a large jurisdiction in terms of population and has experienced such changes in coverage, in both directions, on numerous occasions since coverage at 6 years of age was first reported in 2002.

Figure 6 shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 6 years, although the rate of increase has slowed over the past two years for all age groups. The Figure shows that there have now been 10 consecutive quarters where 'fully immunised' coverage at 24 months of age has been greater than 'fully immunised' coverage at 12 months of age, following the removal of the requirement for the 18-month DTPa vaccine. However, both measures have been above 90 per cent for this 27-month period and show levels of high coverage for the vaccines included have been maintained over a significant period of time. Currently, coverage for the more recent vaccines, 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 6. Trends in vaccination coverage, Australia, 1997 to 2005, by age cohorts

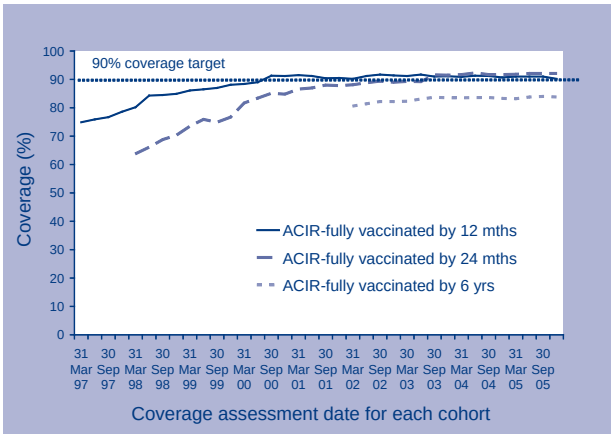


Table 11. Percentage of children immunised at 6 years of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 1999; assessment date 31 March 2006

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	984	21,547	789	12,942	4,563	1,573	15,913	6,612	64,923
Diphtheria, tetanus, pertussis (%)	87.8	85.2	82.8	83.1	83.5	84.5	88.0	80.7	84.9
Poliomyelitis (%)	88.8	85.0	83.4	83.2	83.6	84.6	87.9	80.3	84.8
Measles, mumps, rubella (%)	88.2	85.1	83.9	83.4	83.7	84.6	88.0	80.4	84.9
Fully immunised (%)[1]	87.0	84.1	82.0	81.8	82.6	83.6	87.1	79.1	83.8
Change in fully immunised since last quarter (%)	-1.2	-0.6	-1.1	+0.4	+0.8	-3.0	-0.2	-0.5	-0.2

National Enteric Pathogens Surveillance System

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

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

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

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

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

Reports to the National Enteric Pathogens Surveillance System of Salmonella *infection for the period 1 January to 31 March 2006 are included in Tables 12 and 13. Data include cases reported and entered by 24 April 2006. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. For more information see* Commun Dis Intell *2006;30:159–160.*

First quarter 2006

The total number of reports to NEPSS of human *Salmonella* infection rose to 2,876 in the first quarter of 2006, 31 per cent more than in fourth quarter of 2005. The first quarter count was seven per cent more than the comparable first quarter of 2005 and approximately 12 per cent greater than the ten-year historical mean for this period. Indeed, the 2,876 reports represent the second highest count for any quarter since at least 1991.

A wide range of salmonellae have contributed to this excess of cases, including those associated with outbreaks and unseasonable increases in *S.* Typhimurium phage type 135 (widespread), *S.* Typhimurium phage type 44 (Victoria and New South Wales), *S.* Oranienberg (Western Australia), and *S.* Bovismorbificans phage type 24 (eastern states). More modest recent increases have involved *S.* Birkenhead (northern New South Wales), *S.* Infantis (New South Wales and South Australia), *S.* Hvittingfoss (Victoria and Queensland), S. Anatum (South Australia), *S.* Potsdam (New South Wales), and *S.* Virchow phage type 25 var 1 (Queensland). The sustained elevation in disease due to the related *S.* Typhimurium phage types 170 and 108 continues.

During the first quarter of 2006, the 25 most common *Salmonella* types in Australia accounted for 1,888 cases, 66 per cent of all reported human *Salmonella* infections. Twenty-two of the 25 most common *Salmonella* infections in the first quarter of 2006 were also among the 25 most commonly reported in preceding quarter.

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

Table 12. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 January to 31 March 2006, as reported to 24 April 2006

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	37	741	91	993	180	81	473	280	2,876
Total contributing *Salmonella* types	22	146	41	134	53	10	103	72	253

Table 13. Top 25 *Salmonella* types identified in Australia, 1 January to 31 March 2006, by state or territory

National rank	*Salmonella* type	State or territory								Total 1st quarter 2006	Last 10 years mean 1st quarter	Year to date 2006	Year to date 2005
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium PT 135	5	79	0	52	10	14	72	24	255	233	255	129
2	*S.* Typhimurium PT 170	3	97	0	24	0	11	30	0	165	81	165	165
3	*S.* Saintpaul	1	14	8	93	2	0	15	23	156	129	156	157
4	*S.* Typhimurium PT 9	4	25	0	23	19	8	69	6	154	180	154	166
5	*S.* Birkenhead	1	46	0	63	0	0	3	0	113	96	113	71
6	*S.* Virchow PT 8	1	9	2	94	0	0	2	1	109	93	109	102
7	*S.* Oranienburg	1	5	0	3	2	0	2	69	82	17	82	13
8	*S.* Typhimurium PT 44	0	18	0	12	4	4	33	4	75	19	75	5
9	*S.* Infantis	2	29	3	8	13	0	7	2	64	46	64	52
10	*S.* Aberdeen	0	2	0	59	0	0	2	1	64	43	64	65
11	*S.* Hvittingfoss	1	3	2	40	0	0	17	0	63	35	63	55
12	*S.* Chester	0	11	1	36	2	0	3	9	62	67	62	74
13	*S.* Mississippi	2	3	0	5	1	39	3	2	55	38	55	31
14	*S.* Waycross	1	14	0	39	0	0	0	0	54	46	54	44
15	*S.* Muenchen	0	11	4	23	1	0	2	9	50	59	50	65
16	*S.* Anatum	0	4	3	13	18	0	7	5	50	32	50	19
17	*S.* Bovismorbificans PT 24	0	15	1	23	3	0	8	0	50	4	50	6
18	*S.* Typhimurium RDNC	1	12	1	5	6	1	12	4	42	30	42	31
19	*S.* Potsdam	2	15	0	17	3	0	3	1	41	20	41	9
20	*S.* Typhimurium PT 197	0	9	0	24	1	0	5	0	39	56	39	383
21	*S.* Typhimurium PT 12	0	17	0	4	4	0	4	8	37	32	37	56
22	*S.* Typhimurium untypable	0	4	0	2	0	1	13	8	28	20	28	15
23	*S.* Weltevreden	0	4	4	13	2	0	3	1	27	11	27	14
24	*S.* Virchow PT 25 var 1	0	1	0	25	0	0	1	0	27	0	27	6
25	*S.* Typhimurium PT 108	0	3	0	1	22	0	0	0	26	11	26	20

Overseas briefs

World Health Organization Disease Outbreak News

This material has been summarised from information provided by the World Health Organization (http://www.who.int).

Avian influenza

China

24 March 2006

The Ministry of Health in China has confirmed the country's 16th case of human infection with the H5N1 avian influenza virus. The case, which was fatal, occurred in a 29-year-old female migrant worker. She was hospitalised in Shanghai on 15 March with symptoms of pneumonia and died on 21 March.

This is the first case reported in Shanghai. Her source of infection is under investigation. No poultry outbreaks have been reported in the Shanghai area since February 2004. According to Chinese authorities, the woman's close contacts have been placed under medical observation.

To date, China has reported 16 cases of H5N1 infection, of which 11 have been fatal.

Cambodia

24 March 2006

The Ministry of Health in Cambodia has confirmed the country's fifth case of human infection with the H5N1 avian influenza virus. The case, which was fatal, occurred in a 3-year-old girl from Kampong Speu Province, west of Phnom Penh in the southern part of the country. The child developed fever on 14 March. Her condition deteriorated rapidly and she was hospitalised in Phnom Penh on 20 March. She died on 21 March. Samples from the girl tested positive for H5N1 infection at the Pasteur Institute in Cambodia.

A team of officials from the Ministry of Health and WHO have investigated the situation in the remote village where the child lived. Backyard poultry began dying in the village in February, and chicken deaths have continued. The child is known to have played with chickens, including some showing signs of illness.

The investigation found seven residents with fever but no respiratory symptoms. All had a history of recent contact with diseased birds or had been involved in caring for the child. Although none of these people presently shows symptoms compatible with H5N1 infection, all have been placed under medical observation as a precaution.

This is the fifth confirmed case in Cambodia and the first in almost a year. The four previous cases, all from the adjacent Kampot Province which borders Viet Nam, and all fatal, occurred from end-January 2005 through mid-April 2005.

Egypt

29 March 2006

The Ministry of Health in Egypt has confirmed the country's second fatal case of human infection with the H5N1 avian influenza virus. The death occurred in a 30-year-old woman from the Qaliubiya governorate near Cairo. She developed symptoms on 12 March following the home slaughter of chickens. She was hospitalised on 16 March and died on 27 March.

Tests conducted by the Cairo-based US Naval Medical Research Unit 3 (NAMRU-3) have confirmed an additional three cases. A 32-year-old man, who worked on a farm where poultry were recently culled, developed symptoms on 16 March and was hospitalised the same day. He has since recovered. A 17-year-old boy, whose father runs a poultry farm in the Gharbiya governorate in the Nile Delta, developed symptoms on 18 March and was hospitalised the following day. He has since recovered. The third case is an 18-year-old girl from the Kafr El-Sheikh governorate. She developed symptoms following the slaughter of sick backyard poultry. She was hospitalised on 25 March.

Health authorities have screened more than 350 people who were contacts of these patients or had a recent history of exposure to diseased birds. All test results have been negative for H5N1 infection.

Egypt has a large population of poultry, many of which are kept on roof terraces in close proximity to humans. H5N1 outbreaks in poultry have now been reported in 19 of the country's 26 governorates. Since the first outbreak was confirmed on 17 February, more than 25 million birds have died or been destroyed.

ProMED-mail

This material has been summarised from information provided by ProMED-mail (http://www.promedmail.org).

Anthrax, inhalation – USA

Source: Center for Infectious Disease Research and Policy, University of Minnesota, 28 February 2006 (edited)

Laboratory testing in the case of the New York City drum maker who recently contracted anthrax has supported the belief that he inhaled anthrax spores while working with contaminated animal hides, according to federal health officials. The Centers for Disease Control and Prevention (CDC) said that tests revealed *Bacillus anthracis* in the workplace, home, and van of the 44-year-old man. He fell ill with inhalational anthrax after performing in a concert in Mansfield, PA, on 16 February 2006. The test results 'are consistent with the hypothesis that the patient's exposure occurred while working on contaminated hides while making traditional drums,' CDC said in a notice sent through its Health Alert Network.

The man, who is the first known US anthrax case since 2001, remains in serious condition in a Pennsylvania hospital. He was reported to have made drums from goat hides imported from Africa.

'No cases of inhalation anthrax in the US have ever been associated with animal hide drums,' the agency said. The man's exposure 'occurred when he was making and finishing drums made from un-tanned animal hides and was not associated with playing finished drums. His exposure was similar to that experienced during industrial handling of hides.'

Poliomyelitis

Source: Eurosurveillance Weekly Release Surveillance Report, Vol 11, Issue 3, 9 March 2006 (edited)

The World Health Organization-coordinated global Poliomyelitis Eradication Initiative has made tremendous progress since its beginning in 1988. Polio became close to being eradicated in 2001 when the lowest annual case count ever, less than 500 worldwide, was reported. However, several setbacks, mainly due to sub-optimal vaccine coverage, have prevented this goal being reached.

From 2002 to 2005, 21 countries which were previously polio-free were affected by cases imported from what were the six remaining countries with endemic wild poliovirus (WPV) type 1 circulation (Afghanistan, Egypt, India, Niger, Nigeria, and Pakistan). Most of these were from Nigeria, while three importations were from India. For eight countries, imported WPV cases did not result in sustained transmission, but in the remaining 13, WPV caused multiple outbreaks. In 2005, 1,983 cases of poliomyelitis were reported worldwide, and 50 cases were caused by vaccine-derived polioviruses.

Large outbreaks in 2005

Three countries were affected by outbreaks of more than 100 WPV cases in 2005: Yemen, Indonesia and Somalia. Most outbreaks required multiple rounds of large-scale immunisation activities to control and stop transmission.

Yemen

Yemen confirmed its first detected case of imported WPV infection (onset February 2005) in late April 2005. By the end of 2005, 478 polio cases had been reported from all over the country. A National Immunisation Day (NID) had been conducted in mid-April 2005, before the index case was identified, in response to the threat of importation from Sudan. Six additional NIDs were conducted from May to December 2005.

Indonesia

To date, 303 cases caused by WPV have been reported from 10 provinces on Java and Sumatra. The first case in the current outbreak was reported with onset in March 2005. By the time the first response mass immunisation campaign was organised, 99 cases had been detected. Three full NIDS were carried out in August 2005. The most recent case onset was December 2005.

Nigeria

Cases began to increase in Nigeria in 2002 when immunisations with OPV were interrupted in some northern states because of an unjustified fear of harmful side-effects, and consequently, the number of polio cases increased in several parts of Nigeria. Type 1 WPV subsequently spread to 18 countries that had been polio-free for several years.

The virus is known to have been transmitted from Nigeria to Chad, onward to Sudan, and onward again to Saudi Arabia and Yemen. Only two cases were reported in Saudi Arabia, but the virus was transmitted onward from Saudi Arabia to Indonesia. Children under 15 years of age travelling to Saudi Arabia from countries where wild poliovirus has been reported must now be vaccinated against polio before entry into the country, whether or not they are visiting for the Hajj pilgrimage.

In early 2006, after intensive supplementary immunisation campaigns, outbreaks in most of the reinfected African countries are under control, with the exception of Somalia. These episodes have postponed the possible world WPV eradication date again by several years; according to the World Health Organization. Worldwide eradication cannot be declared until at least three years after the most recent isolation of WPV anywhere in the world. WHO now considers that endemic transmission of WPV has stopped in Egypt and Niger, leaving only four countries in the world with persistent indigenous WPV transmission: Afghanistan, India, Nigeria, and Pakistan.

Through impressive international and inter-agency collaboration, poliomyelitis has become a rare disease in the world. However, the proclaimed target, global eradication of wild-type poliovirus, has yet to be reached.

Somalia

To date, 185 cases have been confirmed, with the most recent onset being in November 2005. The first onset was July 2005, after importation from Yemen. Three NIDS have been carried out.

India

A major outbreak in 2002 was successfully contained, and in 2005, there were only 66 new cases. Continuing WPV circulation in India resulted in a single imported case in Lebanon in 2003, a few cases in Nepal and re-established circulation in Angola in 2005.

Hand, foot and mouth disease – Malaysia (Sarawak)

Source: Bloomberg.com, 10 March 2006 (edited)

Malaysian authorities said another child in Sarawak died two days ago from hand, foot and mouth disease, bringing the total to five since December 2005 in an outbreak the Health Ministry has called an epidemic.

Two of the deaths were caused by enterovirus 71, the more virulent type of the group of enteroviruses that cause hand, foot and mouth disease, the World Health Organization said, citing a Malaysian Health Ministry statement. All 488 kindergartens and nine primary schools in Sarawak state, on the island of Borneo, are closed for two weeks. Sarawak is trying to prevent a repeat of 1997, when 31 children died from the disease.

The nation of 26 million people is speeding up efforts to contain the disease, which had infected 3,269 children in Sarawak as of 8 March 2006. That's more than 10 times the number of cases last year [2005]. The cases tend to increase from February to June according to the Sarawak government's website.

Malaysia has outbreaks of hand, foot and mouth illness, characterised by fever, mouth sores and blisters, about every three years. Sarawak had 2,113 cases in 2003, and 3,560 in 2000. The disease is common in children, and in most cases, sufferers recover without medical treatment in 7 to 10 days.

Mumps virus, genotype G – USA (Iowa)

Source: ABC News, 15 March 2006 (edited)

State health officials have stated that they are concerned about a rare strain of mumps virus behind an outbreak of 60 mumps cases in Iowa. The director of the State University's Hygienic Laboratory, said the genotype G strain is infrequently seen in the United States. With the number jumping from 17 cases just two weeks ago, she predicted there could be more outbreaks this spring [2006].

The patients with confirmed cases of mumps range in age from 11 to 41 years, but half have been college students. The virus may have come from Europe, but a similar strain has been detected in New Jersey. The US Centres for Disease Control and Prevention is investigating the source.

Symptoms of mumps include fever, headache and swollen salivary glands at the jaw line, below and in front of the ears. Mumps is generally a mild illness for healthy people and is spread through coughing or sneezing. It can cause serious complications, including deafness in children and spontaneous abortion in pregnant women.

Mumps infection occurs worldwide, and humans are the only natural host for the virus. Various vaccine strains have been developed and have been effective in reducing the incidence of mumps worldwide. The virus is essentially monotypic, although there may be some mismatch between individual strains and vaccines. Despite the monotypic nature of the mumps virus, different genetic lineages of mumps virus exist and co-circulate globally. Genotypes A to J have been defined on the basis of the nucleotide sequence of the most variable gene, the SH gene. The different lineages are useful properties for tracking the spread of mumps virus, but there is no clear association of the different lineages with different clinical symptoms.

Chikungunya - Indian Ocean

India (Andhra Pradesh)

Source: New IndPress, 29 March 2006 (edited)

Chikungunya has affected about 8,000 people in 75 villages, but no deaths have been reported. According to the district medical and health department officials, the disease is spreading to other parts of the region, and they have taken up awareness campaigns to arrest its spread to new areas. A district medical and health officer said that chikungunya spreads through the tiger mosquito, which breeds in fresh water and bites during daytime. He advised people not to store drinking water for long periods and to keep the containers clean. He said chikungunya causes high fever coupled with severe body pains and could be treated with paracetamol (acetaminophen). The tiger mosquito usually does not survive beyond 30° Celsius, and with the soaring temperatures of summer, the incidence of the disease will come down.

Reunion

Source: Yahoo News, UK, 29 March 2006 (edited)

The disabling mosquito-borne disease, Chikungunya, which has affected more than a quarter of people living on the French Indian Ocean island of Reunion is now in sharp decline, the French Senate was told. New cases are down to 4,400 a week compared with 47,000 at the start of February 2006.

Overall, some 218,000 people out of a total population of 777,000 have contracted Chikungunya in the last year, of whom 155 have died directly or indirectly as a result of the disease.

The disease gets its name from a Swahili word meaning ‘that which bends up’ because of its arthritic-type symptoms that leave victims stooped. Most patients eventually recover. The French government has committed emergency health and economic aid worth over 90 million Euros to help Reunion fight the scourge but was criticised for failing to react quickly enough.