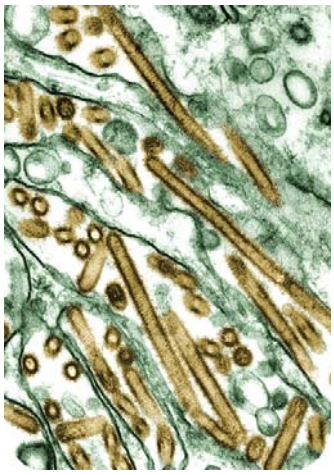
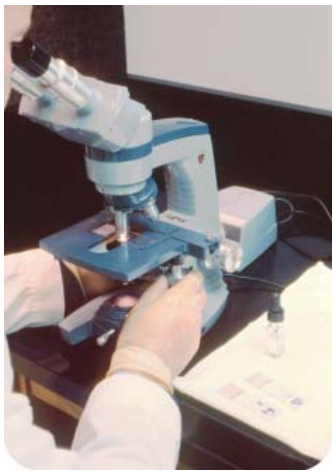
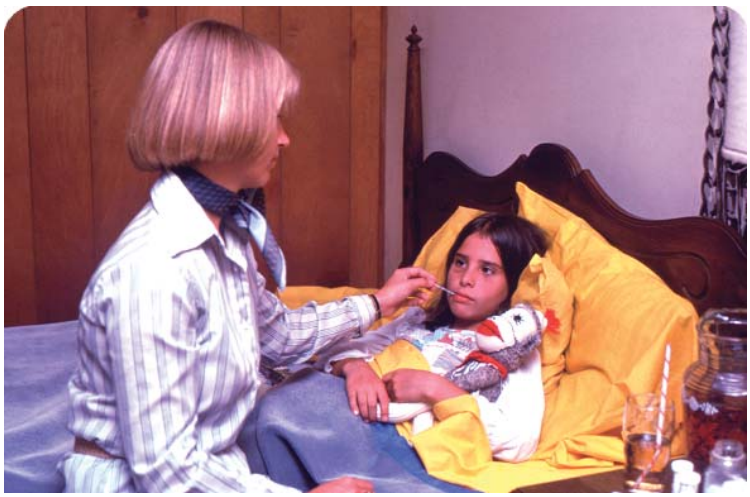
Australian Government
Department of Health and Ageing

Communicable Diseases Intelligence







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Images sourced from the Centers for Disease Control and Prevention Public Health Image Library, courtesy of the Centers for Disease Control and Prevention, Atlanta, Georgia.
Clockwise from top left: Colourised transmission electron micrograph of avian influenza A H5N1 viruses, C. Goldsmith; Microscopic examination of food, V Dowell; Sick child, Betty Partin.

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Contents

Annual report of the National Influenza Surveillance Scheme, 2004

Janet Li,[1] Alan Hampson,[2] Paul W Roche,[3] Keflemariam Yohannes,[3] Jenean D Spencer[3]

Abstract

The National Influenza Surveillance Scheme includes data on influenza-like illness from sentinel general practitioner practices, laboratory reports of influenza from National Notifiable Diseases Surveillance Scheme and absenteeism data from a national employer. In 2004, 2,116 cases of laboratory-confirmed influenza-like illness were reported, which was 41 per cent lower than the previous year. Peak activity was recorded in September, a month later than in 2003. Influenza A was again predominant while influenza B had an increased activity compared to the previous season. Four hundred and fifty-four isolates were antigenically analysed: 342 were A (H3N2 strain), 4 were A(H1N1 strain) strains and 108 were influenza B viruses. Further antigenic drift was seen in the A(H3N2) subtype with approximately one third of all isolates antigenically distinguishable from the A/Fujian/411/2002 reference strain. Vaccination coverage indicated that 79 per cent of Australians aged over 65 years received the 2004 influenza vaccine. *Commun Dis Intell* 2005;29:124–135.

Keywords: disease surveillance, influenza, vaccine

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 (peaking between June and September in the Southern Hemisphere and between December and 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 during 2003, influenza and pneumonia were the underlying causes of 3,566 deaths.[1]

There are three types of influenza—A, B and C—which are classified according to their antigenically distinct internal proteins. 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 viruses are successful human pathogens because of their ability to vary their two external proteins, haemagglutinin (H) and neuraminidase (N). 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 less likely it is that the virus will be recognised by immune cells primed by exposure to earlier infections or vaccines, and the greater the epidemic potential. 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 believed to occur

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in an intermediate host such as pigs. These 'shifts' result in the emergence of a new influenza virus. In the absence of immunity to these new viruses, there is a rapid spread of influenza with dramatically increased rates of morbidity and mortality.

Periodically, novel influenza viruses emerge and spread rapidly through susceptible populations, resulting in worldwide epidemics or pandemics.[4] 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. The outbreaks of influenza A(H5N1) virus in Asia in 2004/05 and the associated human 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/influenza/WHO_CDS_2005_29/en/). Virological and epidemiological monitoring are important components of influenza surveillance. The main objectives of virological and epidemiological surveillance of influenza 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 2004, the Communicable Diseases Australia website (http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-surveil-ozflu-flucurr.htm) published influenza surveillance data fortnightly during the influenza season. This annual influenza report is a summary of the surveillance information gathered by various systems in 2004.

The international influenza activity in 2004 and the relationship to Australian influenza activity are also included in this report.

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 nationally notifiable 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.

Australia also hosts one of the four WHO Collaborating Centres for Reference and Research on Influenza.

National Notifiable Diseases Surveillance System

In all jurisdictions with the exception of the Australian Capital Territory and South Australia, laboratory-confirmed influenza is a notifiable disease under state and territory legislature. In the Australian Capital Territory and South Australia, laboratory reports are also collected and sent to NNDSS although influenza is not a notifiable condition. In this report, data are analysed by the date of onset in order to present disease activity during the reporting period, but when this was not available either the specimen collection date or the notification date, whichever was the earlier, was used.

Laboratory surveillance

LabVISE is a national scheme of sentinel laboratories that reports influenza diagnosis all year round. In 2004, 17 laboratories from all jurisdictions except the Northern Territory contributed to the scheme. Data were reported monthly and were analysed by the specimen collection date.

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centres for Reference and Research on Influenza located in Australia, Japan, the United Kingdom and United States of America are responsible for analysing influenza viruses collected through an international surveillance network involving 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 HA and NA 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. Southern Hemisphere recommendation for 2005—http://www.who.int/wer/2004/wer7941/en/) 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/aivc2005.htm)

Sentinel general practitioner surveillance

Sentinel general practitioner surveillance schemes for influenza monitor the consultation rates for influenza-like illness (ILI). In Australia, there are five such schemes: the Australian Sentinel Practice Research Network (ASPREN) which collects data at a national level, the New South Wales Influenza Surveillance Scheme, the Victorian Influenza Surveillance Scheme, Western Australian sentinel general practices, and the Northern Territory Tropical Influenza Surveillance Scheme. In 2004, Queensland also joined the sentinel general practitioner surveillance schemes. 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.

In 2004, all sentinel surveillance schemes adopted the same case definition for ILI: presentation with fever, cough and fatigue. Victoria and Western Australia currently use this case definition, but it was used for the first time in 2004 by ASPREN, the Northern Territory and New South Wales. ASPREN used both old (definition 2) and new (definition 1) case definitions in their reporting in 2004.

Sentinel general practices contributing to the ASPREN scheme are mostly located in capital cities and larger regional centres on the east coast of Australia (Map). In 2004, an average of 47 (range 32–62) general practices reported ILI cases on an average of 4,962 (range 2,138–6,587) consultations per week.

The Northern Territory Tropical Influenza Surveillance reported cases of ILI as the rate per 1,000 consultations per week. Throughout the year, eight to fourteen centres reported to the surveillance system with an average of 866 (range 338–1,290) consultations per week.

In 2004, the New South Wales Influenza Surveillance program collected reports from New South Wales practitioners who are part of ASPREN and from five of the 17 Public Health Units (Southern New South Wales, New England, Wentworth, Central Sydney and South Eastern Sydney). Fifteen (range 2–21) general practitioners reported ILI cases weekly from May to October on an average of 1,854 (range 208–2,659) consultations per week.

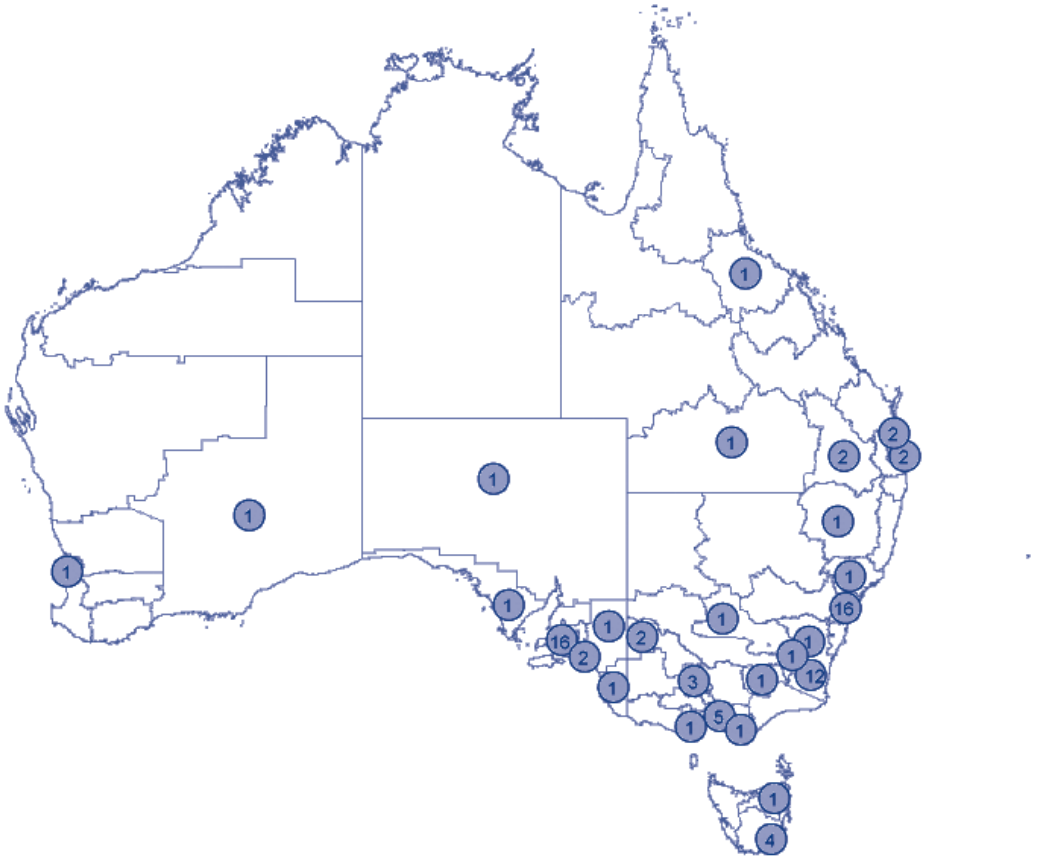
The Victorian Infectious Diseases Reference Laboratory, the WHO Collaborating Centre for Reference and Research on Influenza and the Victorian Department of Human Services contributed to the Victorian Influenza Surveillance Scheme. In 2004 the Victorian Influenza Surveillance Scheme also enlisted the Melbourne Locum Service. Overall, 76 general practitioners from metropolitan (15 sites) and rural (23 sites) regions were recruited to report ILI consultations between April and October. These practices reported on 6,995 (range 4,675–8,128) consultations per week. ILI was calculated as the rate per 1,000 consultations per week.

In Western Australia, 18 general practices, 14 in the metropolitan area (Perth) and four in rural regions (one each in Kalgoorlie, Busselton, Tom Price and Geraldton) participated in ILI surveillance from May to November. Data were reported, for the first time, as the rate of ILI per 1,000 consultations per week in 2004. Data collected prior to 2004 were reported as the number of cases per practice per week.

Table 1. Case definitions of influenza-like illness used in Australian sentinel practice schemes, 2004

Program	Case definition
Victorian Influenza Surveillance Scheme, Western Australian sentinel general practices, New South Wales State program, Northern Territory and ASPREN (definition 1)	Fever, cough, fatigue
ASPREN (definition 2)	(a) Viral culture or serological evidence of influenza virus infection; or (b) influenza epidemic, plus four of the criteria in (c) (c) Six of the following: sudden onset (within 12 hours); cough; rigors/chills; fever; prostration and weakness; myalgia; no significant respiratory symptoms other than redness of nasal mucous membranes and throat; influenza in close contacts.

Map. Geographic distribution of ASPREN sentinel general practice sites, Australia, 2004



In Queensland, 13 sentinel practices reported to the surveillance system during the influenza season. Three of these practices were in metropolitan area, while the rest were in rural and remote regions. The sentinel practices reported to the surveillance system with an average of 980 (range 129–1,521) consultations per week. ILI was reported as the rate per 1,000 consultations per week.

Absenteeism surveillance

Australia Post, a major nationwide employer provided sick leave absenteeism data collected weekly between March and December 2004. 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 32,973 employees per week in 2004.

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. Data for the 2003/04 financial year were not available at the time of writing this report.

Results

The influenza surveillance data presented here should be interpreted with caution. 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. As 2004 was the first time that all the sentinel schemes used the same case definition, comparisons of ILI between schemes was possible. However, it is difficult for some of the sentinel schemes to compare 2004 rates of ILI with previous years because of the adoption of a new case definition.

National Notifiable Diseases Surveillance System

In 2004, 2,116 laboratory confirmed cases of influenza were reported to NNDSS, which represents a 41 per cent decrease from the number of notifications in 2003. All jurisdictions reported laboratory-confirmed influenza to NNDSS, although Tasmania and the Australian Capital Territory reported very few cases. Tasmania's low reporting may have been due to their limited access to laboratory testing of influenza. In the Australian Capital Territory and South Australia, where influenza was not classified as a notifiable disease, the reporting of laboratory-confirmed influenza has never been complete.

Notifications of laboratory-confirmed influenza started to increase in late August; peaked in late September and started to decrease in late October (Figure 1). Compared to 2003, the influenza season started a month late; the magnitude of notifications was much smaller and spread out; the peak number of notifications was much lower; and the rise of the number of notifications was not as sharp as that observed in 2003.

A comparison of notification rates in each jurisdiction (with the exception of the Australian Capital Territory and Tasmania) is shown in Figure 2. The highest notification rate occurred in September in Queensland (9 cases per 100,000 population), the Northern Territory (6 cases per 100,000 population), and New South Wales (4 cases per 100,000 population).

Figure 1. Notifications of laboratory-confirmed influenza to the National Notifiable Diseases Surveillance System, Australia, 2003 and 2004, by week of onset

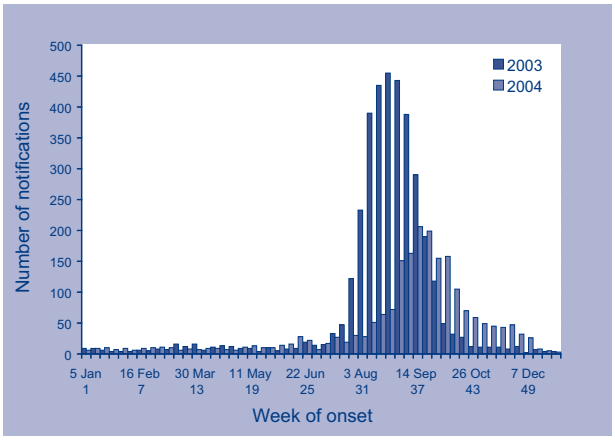
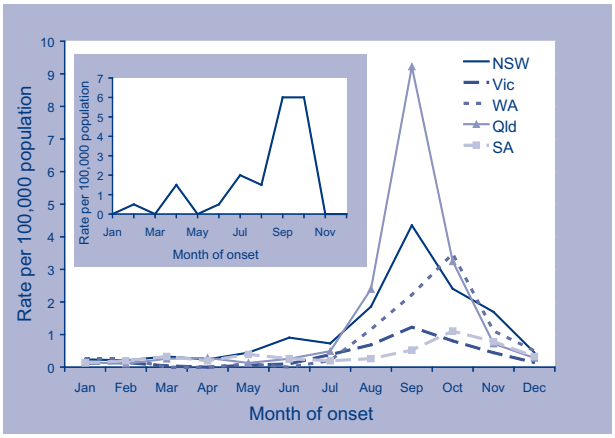


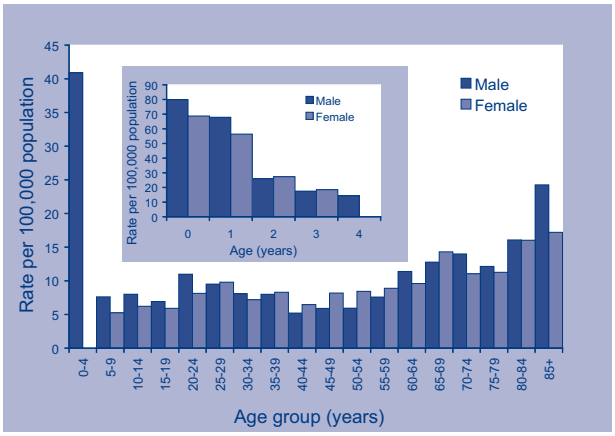
Figure 2. Notification rates of laboratory-confirmed influenza, Australia, 2004, by jurisdiction and month of onset



National age-specific notification rates are shown in Figure 3. The overall male to female ratio was 1:1. The 0–4 years age group had highest notification rate (39 cases per 100,000 population) – representing 23 per cent of all notifications.

Infants under the age of one composed 39 per cent of the notifications of the 0–4 years age group and had the highest notification rate at 75 cases per 100,000 population (Figure 3 insert). The 85 years and over age group had the second highest notification rate (19 cases per 100,000 population).

Figure 3. Notification rates of laboratory-confirmed influenza, Australia, 2004, by age and sex



Laboratory surveillance

A total of 706 laboratory diagnoses of influenza were reported to LabVISE participating laboratories, 69 per cent of which were influenza A (Figure 4). The overall influenza A to B ratio in 2004 was 2:1. The peak of influenza reports to LabVISE occurred in September, which was a week later than the peak influenza activity observed in the NNDSS surveillance data. This was because LabVISE data were analysed by the date of specimen collection while NNDSS data were analysed by date of onset.

WHO Collaborating Centre for Reference and Research on Influenza

The WHO Collaborating Centre for Reference and Research on Influenza received 454 isolates or clinical specimens from Australia that yielded viable influenza viruses (the lowest level for a decade) and they were all analysed antigenically. Of these viruses 342 (75.3%) were A(H3N2) strains, 108 (23.8%) influenza B and only four were A(H1N1) viruses. Sequence analysis of the variable (HA1) region of the haemagglutinin was undertaken for 51 strains (37 H3, 14 B) and of the neuraminidase for 31 strains (25 A and 6 B). Approximately one third of the Australian A(H3) viruses were genetically and antigenically distinguishable from the reference strain A/Fujian/411/2003 and 2004 vaccine strain A/Wyoming/3/2003 (Table 2, Figure 5), and were similar to a recent isolate A/Wellington/1/2004. Influenza A(H3N2) isolates from throughout the Asia-Pacific region displayed similar antigenic drift and the percentage of A/Wellington/1/2004-like strains increased as the year progressed.

Genetic analysis of the Australian A(H3) isolates demonstrated that, unlike the previous season when the neuraminidase antigen of most of the viruses was more closely related to that of strains circulating in the 2003 season than to that of the reference virus A/Fujian/411/2002 (Figure 6), the majority fell within the A/Fujian/411/2004 lineage with only a few strains (e.g. A/Victoria/520/2004 and A/Brisbane/59/2004) with a neuraminidase in the 2003 lineage. This is

Figure 4. Laboratory reports of influenza diagnoses reported to LabVISE, Australia, 2004, by type and month of specimen collection

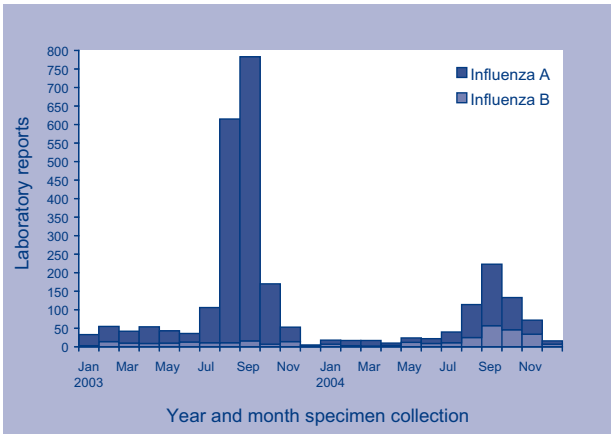


Table 2. Antigenic comparisons of influenza A(H3) viruses by the haemagglutination-inhibition test

Virus antigen	Ferret antiserum		
	Reciprocal haemagglutination-inhibition titre		
	A/Panama	A/Wyoming	A/Wellington
A/Panama/2007/99	640	40	160
A/Wyoming/3/2003*	230	2,560	640
A/Wellington/1/2004	160	320	1,280

* A/Wyoming/3/2003 was the A/Fujian/411/2002-like vaccine strain used in the 2004 vaccine.

suggestive of both a fresh introduction of A(H3) viruses from the Northern Hemisphere and some limited persistence of viruses from the previous season. Of the four A(H1) isolates all were A(H1N1) and these remained antigenically close to the reference and vaccine strain A/New Caledonia/20/99. Of the 108 influenza B viruses analysed the majority (83.3%) were antigenically and genetically closely related to the reference virus B/Shanghai/361/2002 (B/Sichuan/379/99 lineage) with 18 B/Hong Kong/330/2001-like strains (Figure 7).

Consistent with the antigenic drift in the A(H3) isolates demonstrated by ferret antisera (Table 2), serological studies conducted with pre– and post-vaccination human sera from recipients of vaccine containing the A/Fujian/411/2002-like strain showed a reduction in antibody titres to the recent A/Wellington/1/2004-like strains. While the Australian 2004 vaccine contained a B/Hong Kong/330/2001-like strain the majority of the influenza B isolates were B/Sichuan/379/99 lineage strains and the vaccine induced significantly reduced antibody levels to these viruses. Unpredictability

Figure 5. Evolutionary relationships between influenza A(H3) haemagglutinins (HA1 region)

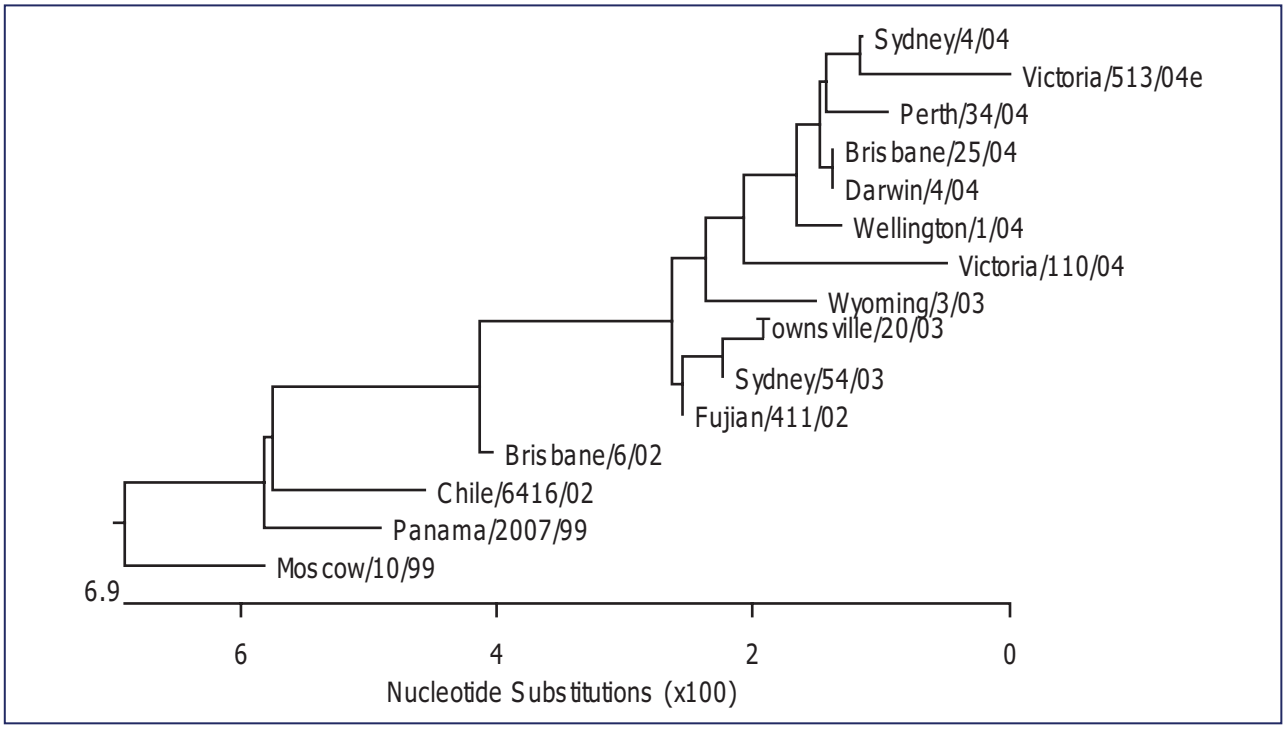


Figure 6. Evolutionary relationships between influenza N2 neuraminidases

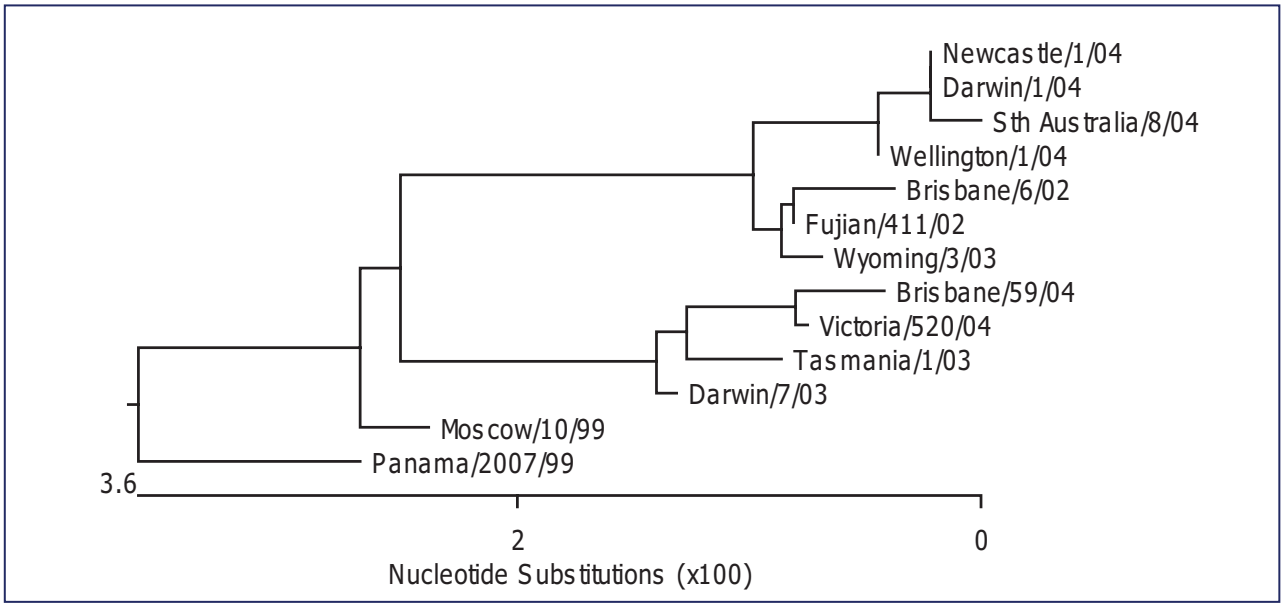
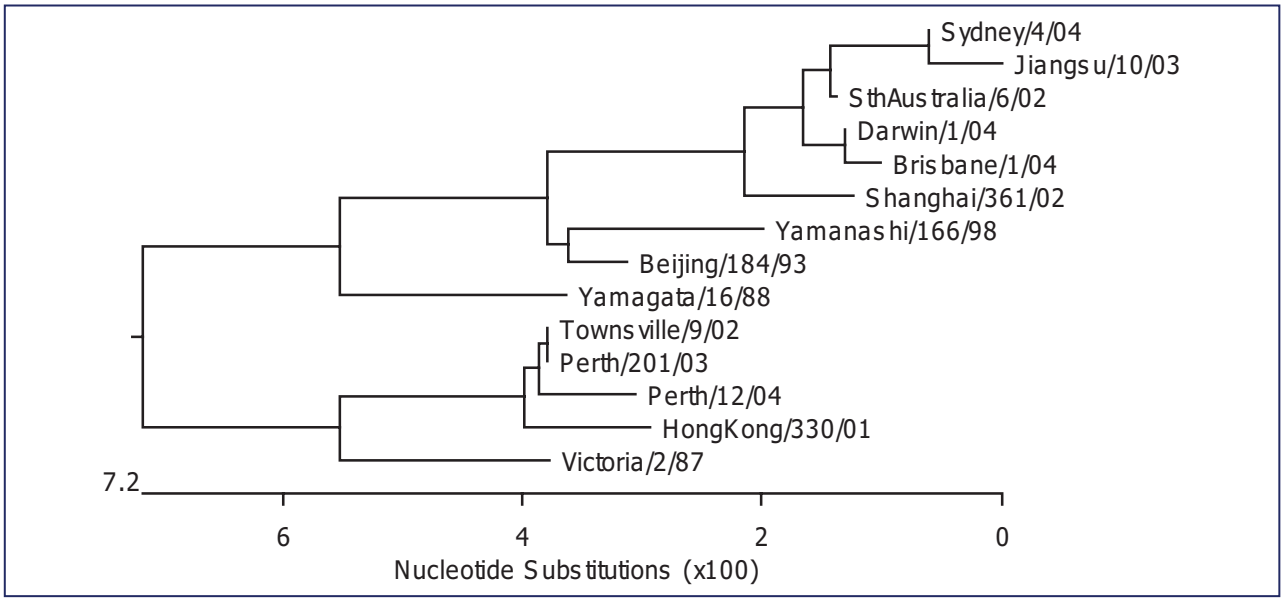


Figure 7. Evolutionary relationships between influenza B haemagglutinins (HA1 region)



regarding the predominance of the two type B lineages from year to year has been problematic for vaccine formulation in recent seasons.

Sentinel general practice surveillance

ASPREN data for 2004 is presented for both the old (case definition 2: cough, rigour or chills, fever, prostration and weakness, myalgia, redness of mucous membrane, with a sudden onset) and the new (case definition 1: fever, cough and fatigue) case definitions in comparison with 2003 data (Figure 8). According to case definition 1, ILI peaked twice in 2004: first in early July (20.3 ILI per 1,000 consultations), and again in mid-September (18.3 ILI per 1,000 consultations). The ILI rate of case definition 2 showed the same peaks also in early July (12.9 ILI per 1,000 consultations) and in mid-September (10.3 ILI per 1,000 consultations). As expected, the broader definition (case definition 1) identified more cases. The multiple peaks of ILI rates in the two definitions may reflect that rates of ILI peaked at different times in different jurisdictions.

The Northern Territory Tropical Influenza Surveillance Scheme data showed two peak ILI rates (Figure 9); one in week 12 (late March) (31 ILI per 1,000 consultations); and one in week 28 (mid-July) (27 ILI per 1,000 consultations). This is an established pattern of influenza in Australia's tropical north. In 2003, the highest ILI rate using the old case definition (ASPREN definition 2 in Table 1) was reported in week 36 (early September) with a rate of 39 ILI per 1,000 consultations.

Figure 8. ASPREN consultation rates for influenza-like illness, Australia, 2003 and 2004, by week of report

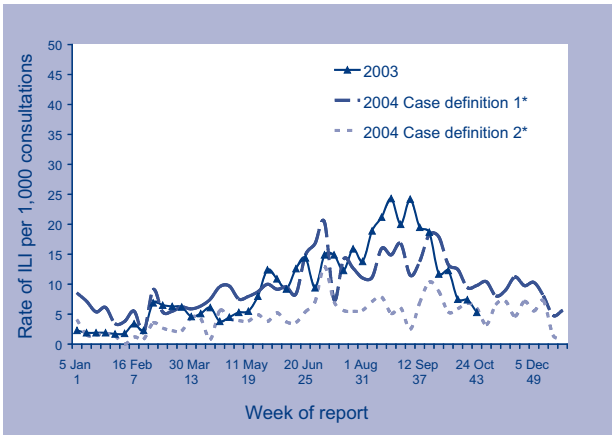
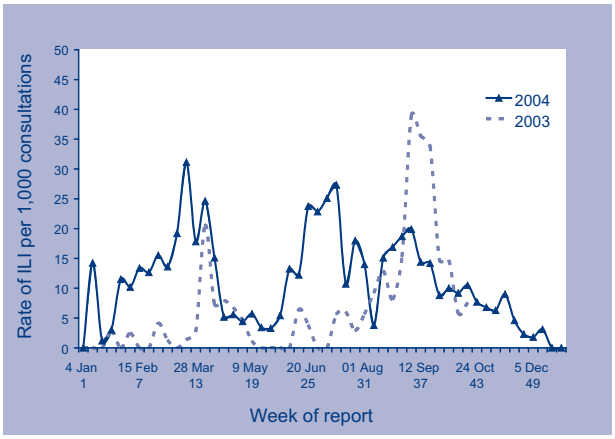
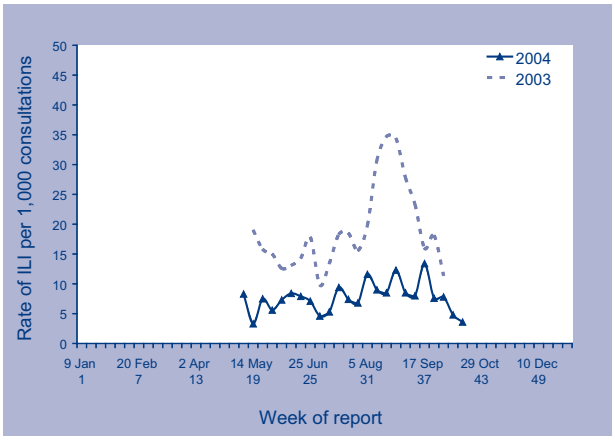


Figure 9. Consultation rates for influenza-like illness, Northern Territory, 2003 and 2004, by week of report



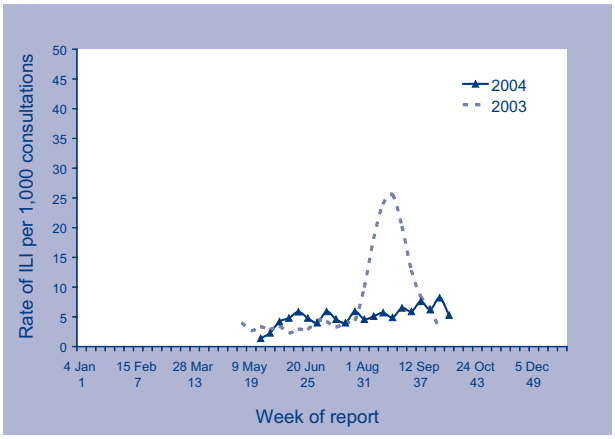
In New South Wales, ILI surveillance was conducted from May to October 2004. There was no obvious peak observed in 2004. The highest ILI rate (13 ILI per 1000 consultations) was reported in week 37 (mid-September) (Figure 10). In 2003, the peak rate using the old case definition (ASPREN definition 2 in Table 1) was reported in week 33 (late August) (35 ILI per 1,000 consultations).

Figure 10. Consultation rates for influenza-like illness, New South Wales, 2003 and 2004, by week of report



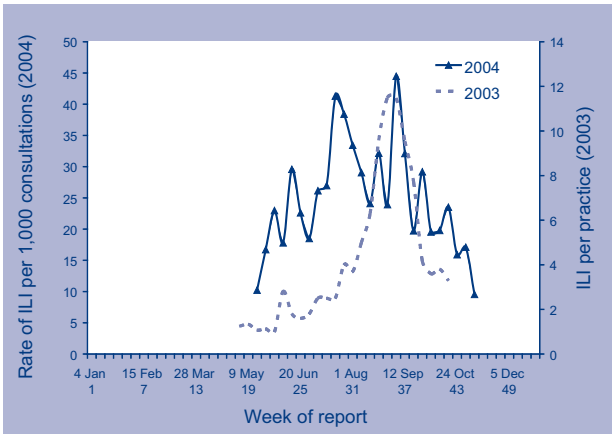
In Victoria, ILI surveillance was conducted from May to October 2004. Similar to the pattern observed in the New South Wales data, there was no obvious peak in 2004. The highest ILI rate (8 ILI per 1,000 consultations) was reported in week 39 (late September) (Figure 11). In 2003, the peak ILI rate (26 ILI per 1,000 consultations) occurred in week 34 (late August).

Figure 11. Consultation rates for influenza-like illness, Victoria, 2003 and 2004, by week of report



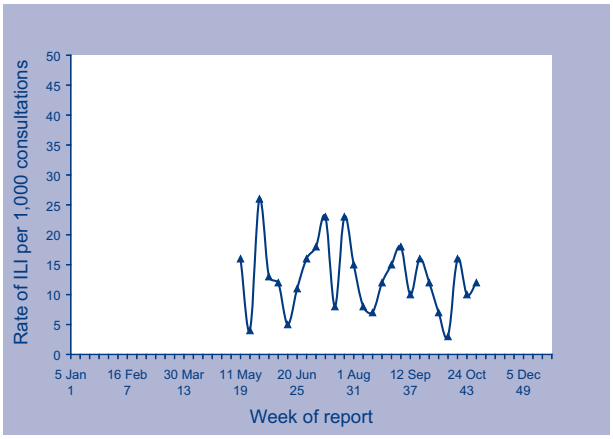
In Western Australia, the ILI rate peaked in week 36 of early September (44.5 ILI per 1,000 consultations). In 2003, the ILI was reported as the rate per practice and peaked in week 35 (Figure 12).

Figure 12. Consultation rates for influenza-like illness, Western Australia, 2003 and 2004, by week of report



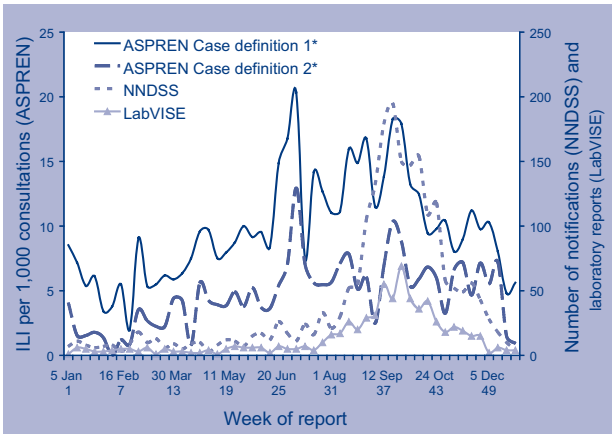
In Queensland, ILI surveillance was conducted from May to October 2004. There was no apparent trend during this influenza season. The highest rate (26 ILI per 1,000 consultations) was reported in week 21 (mid-May) (Figure 13).

Figure 13. Consultation rates for influenza-like illness, Queensland, 2004, by week of report



A comparison of the ASPREN, NNDSS and LabVISE reports is shown in Figure 14. The first peak of ILI in week 27 (early July) reflected by ASPREN was not seen in NNDSS or LabVISE however, the peak activity of ILI indicated by reports to ASPREN in the period between weeks 35 and 38 (29 August to 25 September) was reflected in laboratory reports of influenza to LabVISE and confirmed influenza notifications received by NNDSS.

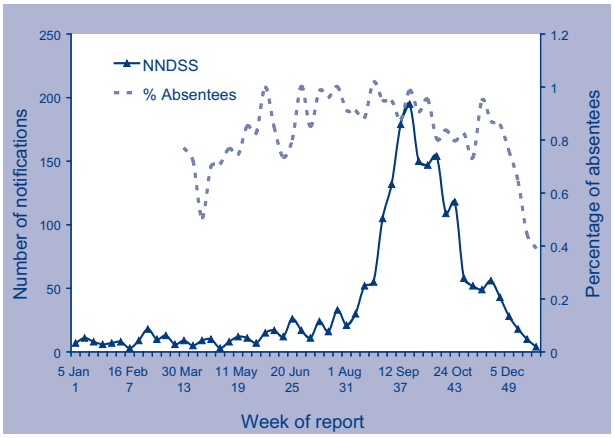
Figure 14. Laboratory reports to LabVISE, notifications to NNDSS and consultation rates in ASPREN of influenza, Australia, 2004, by week of report



Absenteeism surveillance

Absenteeism surveillance is a non-specific index of influenza activity. In 2004, there was no apparent trend observed in the absenteeism surveillance which could be attributed to increased influenza activity. The percentage of absentees started to increase in mid-May (0.9%), then remained steady until late November (Figure 15).

Figure 15. Rates of absenteeism and consultation rates of influenza-like illness, Australia, 2004, by week of report



World trends in influenza, 2004

In 2004, Influenza A (H3N2) viruses predominated in most parts of the world and were responsible for the majority of outbreaks. Influenza A (H1) and B viruses circulated at low levels. In the Southern Hemisphere, influenza activity was relatively mild. Outbreaks caused by influenza A (H3N2) were reported in South America and New Zealand. While the majority of influenza (H3N2) isolates were similar to A/Fujian/411/2002, an increasing proportion of isolates was distinguishable from the A/Wyoming/3/2003 vaccine virus and more closely related to A/Wellington/1/2004.[7] In some countries, influenza A (H1N1) and A (H1N2) viruses were isolated; the majority of these isolates were antigenically closely related to A/New Caledonia/20/99. In many countries, influenza B viruses were also isolated; the majority of recent isolates were closely related to B/Shanghai/361/2002 (B/Yamagata/16/88 lineage), while the reminder were more closely related to B/Hong Kong/330/2001 (B/Victoria/2/87 lineage).

In association with the widespread avian influenza among poultry in Asia and the localised outbreaks among poultry in Canada in 2004, human cases of influenza A (H5N1) and A (H7N3) were also reported. Between 1 January 2004 and 15 February 2005, 55 patients with influenza A (H5N1) were reported from Viet Nam, Thailand and Cambodia, 42 of whom died.[8] These cases were associated with outbreaks of highly pathogenic avian influenza (H5N1) in poultry. A recent study has shown evidence of a highly probable case of human-to-human transmission of a family cluster in Thailand.[9] In March 2004, two human cases of influenza A (H7N3) were associated with outbreaks of avian influenza A (H7N3) in poultry in British Columbia, Canada. There has been no evidence of human-to-human transmission of the H7N3 strain.[10]

The temporal pattern of influenza in New Zealand was similar to Australia with a late onset of the influenza season as compared to 2003. The New Zealand consultation rates for ILI started to increase in mid-August, and peaked in week 38 in late September.[11] New Zealand's influenza isolates in 2004 were predominately A strain (93%) with occasional B viruses (7%). The overwhelming majority of influenza A viruses were A(H3N2) strains and the WHO Collaborating Centre, Melbourne, reported that 61 per cent of the viruses characterised were A/Wellington/1/2004-like. Overall, influenza activity in New Zealand 2004 occurred at a moderate level compared to 2002 and 2003 seasons.

Discussion

The 2004 influenza activity in Australia was low compared with 2003, and the onset of the influenza season was about a month later than in 2003. Although influenza A remained the predominant virus type, there was increased activity of influenza B compared with 2003. The increase of influenza B activity was predicted as outbreaks tend to occur in alternate years.[12] Both influenza A and B peaked in the same month (September). While influenza activity was moderate in 2004, there were a number of outbreaks worth noting. There were two outbreaks of influenza in army camps, one in Victoria (A/Fujian (H3N2)-like) and

another in Queensland.[13] In September, 13 outbreaks of ILI were reported from residential institutions in New South Wales,[13] including 12 aged care facilities and one correctional centre. The outbreaks had high attack rates (up to 76% of residents and 42% of staff) and case fatality rates of 14 per cent.

The majority of influenza A isolates were characterised as influenza A(H3N2) subtype and approximately one third of these demonstrated detectable antigenic drift from the A/Fujian/411/2002-like strain used in the 2004 vaccine. Two separate lineages of influenza B continue to circulate worldwide and the predominant lineage varies from region to region and from season to season. In 2004, the Australian influenza B isolates were predominantly A/Shanghai/361/2002-like whereas the 2004 vaccine contained a B/Hong Kong/330/2001-like strain which induced relatively low levels of cross-reactive antibody. Few influenza A(H1) strains were observed and these showed no evidence of antigenic drift from the A/New Caledonia/20/99 strain incorporated in vaccines since 2000.

There was a higher rate of ILI using the national case definition (fever, cough and fatigue), compared with the old ASPREN case definition (definition 2 in Table 1). A Dutch study[14] has shown that the use of a less specific spectrum of symptoms such as fever, cough, acute onset and malaise had a higher predictive value than that of all the symptoms combined. An Australian study[15] also found that the combination of cough, fever and fatigue was both sensitive (43.5–75.1%) and specific (46.6–80.3%) with positive predictive values ranging from 23 to 60 per cent. These results are reassuring that our national case definition is simple and sensitive, and also predictive of laboratory-confirmed influenza.

Influenza-like illness peaked in the early spring in late September. As in previous years, the earliest reports of ILI were from the Northern Territory, signalling the beginning of the influenza season. Early reports of ILI were also received from Queensland. Laboratory reports of influenza showed slightly different time distribution compared with ILI data from ASPREN. The first peak of laboratory reports in week 33 (mid-August), was six weeks later than first peak seen in ASPREN data. This delay between general practitioner's reports and laboratory reports may reflect a delay in laboratory testing and reporting. Alternatively, the first peak of ILI described by ASPREN in week 27 (early July) may have been caused by viruses related to other respiratory illnesses, and therefore a parallel peak was not seen in NNDSS or LabVISE.

National absenteeism rates reported by Australian Post added little information to the other surveillance systems and remained relatively insensitive to the low levels of influenza activity in 2004. These data may prove more useful in a year of high levels of influenza activity.

Influenza vaccination of the elderly is an important public health action to reduce ILI-related deaths and morbidity. The National Health and Medical Research Council recommends annual influenza vaccination for all Australians aged 65 years or over. In 2004, the vaccination coverage of Australians aged over 65 years was 79 per cent which was higher than in 2003 (77%).[16] Compared to 2003, notification rates of influenza declined slightly (16 cases per 100,000 population in 2003; 14 cases per 100,000 population in 2004) in the over 65 years age group. There was a larger decrease (257 cases per 100,000 population in 2003; 75 cases per 100,000 population in 2004) in the rate among the under one year age group while rates in other age groups remained unchanged. Influenza vaccination can mitigate the impact of morbidity and mortality from annual influenza epidemics on the most susceptible populations.[17]

Preparation for an influenza pandemic is a high priority in Australia. As the threat of a potential pandemic is growing due to the rapid spread of avian influenza in Asia, a range of activities have been coordinated by the Australian Government to prepare for the challenge. The action plan for pandemic influenza is under revision in light of the current international situation. It focuses on the responsibilities of the Australian Government and the jurisdictions in the phases of an influenza pandemic. A range of surveillance activities at the national level, such as hospital surveillance, border surveillance and general practitioner surveillance has been planned. Other activities to enhance the pandemic influenza preparedness, such as rumour surveillance of influenza including avian influenza; stockpiling of antiviral drugs and personal protective equipment; and tracking of vaccine development are coordinated at the national level.

The recommended Australian influenza vaccine for 2005 is composed of an A/New Caledonia/20/99(H1N1)-like strain, an A/Wellington/1/2004 (H3N2)-like strain in place of A/Fujian/411/2002(H3N2)-like virus and a B/Shanghai/361/2002-like strain in place of B/Hong Kong/331/2001-like strains. The World Health Organization identified that there was a antigenic drift in the A(H3N2) viruses and predominance of influenza B viruses of the B/Shanghai/361/2002 lineage in the Northern Hemisphere in 2004. This antigenic drift has resulted in some concerns that vaccines containing an A/Fujian/411/2002(H3N2)-like virus, currently in use in the Northern Hemisphere, may provide sub-optimal protection.

Vaccination remains the most effective strategy of reducing the impact of influenza. Awareness among health care providers of current influenza activity is necessary for reducing the impact of influenza. As the threat of a potential pandemic influenza grows, the National Incident Room will continue its role of providing early warning of diseases including avian influenza and pandemic influenza. This information will add further value to the regular surveillance activity of the National Influenza Surveillance Scheme in the winter of 2005.

Acknowledgements

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Thanks also to staff of the Surveillance Section, in particular Stefan Stirzaker, for his support in Data Management of the NNDSS.

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Annual report of the Australian Gonococcal Surveillance Programme, 2004

The Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme monitors the antibiotic susceptibility of *Neisseria gonorrhoeae* isolated in all states and territories. In 2004, the *in vitro* susceptibility of 3,640 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 22 per cent and, with the exception of the Northern Territory, ranged between 12 and 30 per cent. Resistance to quinolones was found in all jurisdictions in a larger proportion of strains and with higher MICs. Nationally, 23 per cent of all isolates were ciprofloxacin-resistant, and most of this resistance was at high MIC levels. All isolates remained sensitive to spectinomycin. A small number of isolates showed some decreased susceptibility to ceftriaxone (MIC 0.06 mg/L or more) and were again concentrated in New South Wales. 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* 2005;29:136–141.

Keywords: antibiotic resistance; disease surveillance; Neisseria gonorrhoeae

Introduction

Antimicrobial resistance (AMR) in *Neisseria gonorrhoeae* is a continuing and increasing problem in Australia, impacting adversely on disease control. Standardised treatment regimens for gonorrhoea utilise single dose treatments that seek to cure 95 per cent or more of cases. Surveillance of AMR can provide data on susceptibility patterns and provide guidance regarding optimal choice of standard treatments.[1] The increase in AMR in gonococci in most jurisdictions has necessitated changes in programmatic treatments away from oral agents such as the penicillins and latterly, the quinolones. Where these agents are retained for treatment, continuous monitoring is therefore required to ensure their ongoing effectiveness. Increasingly, the injectable third generation cephalosporin ceftriaxone has been introduced for treatment of gonorrhoea, and another injectable antibiotic, spectinomycin, is also available. There are however increasing numbers of reports of gonococcal isolates showing resistance to multiple antibiotics including decreased susceptibility to third generation cephalosporins such as ceftriaxone.[2,3,4]

Monitoring of AMR in gonococci is conducted the Australian Gonococcal Surveillance Programme (AGSP) through a collaborative program conducted by reference laboratories in each state and territory. Data analysed by the AGSP have been published quarterly from 1981 and annual reports have appeared in *Communicable Diseases Intelligence* since 1996. This report is based on data obtained during the 2004 calendar year.

Methods

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

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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.[6] The AGSP also conducted a program-specific quality assurance (QA) program.[7] Antibiotic sensitivity data were submitted quarterly to a coordinating laboratory which collated the results and also conducted the QA program. Additionally, the AGSP received data on the sex of the patient and site of isolation of gonococcal strains. Where available, data on the geographic source of acquisition of antibiotic-resistant isolates were included in analyses.

Results

Numbers of isolates

There were 3,640 gonococcal isolates referred to or isolated in AGSP laboratories in 2004, slightly less than the 3,772 examined in 2003. The source and site of infection with these isolates are shown in the Table. One thousand one hundred and thirteen gonococci (30% of the Australian total) were isolated in New South Wales, 854 (23.4%) in Victoria, 621 (17%) in Queensland, 515 (14.1%) in the Northern Territory, 329 (9%) in Western Australia, and 166 (4.5%) in South Australia with small numbers in Tasmania (22) and the Australian Capital Territory (20). Of the total, 3,542 remained viable for susceptibility testing.

Nationally, 132 (5%) fewer isolates were received in 2004 than in 2003. The number of isolates fell by 66 in Victoria, 61 in South Australia, but only in small numbers elsewhere. Numbers in Tasmania and the Australian Capital Territory, although small, rose substantially from 2003 levels.

Source of isolates

There were 3,077 strains from men and 559 from women, with a male to female (M:F) ratio of 5.5:1, slightly less than the 5.8:1 ratio for 2003. The number of strains from men decreased by 137 but rose by seven from women. The M:F ratio was again high in New South Wales (13.3:1) and Victoria (11.9:1) where strains were more often obtained from urban populations. The lower ratios in Queensland (4.3:1) Western Australia (4.8:1), and the Northern Territory (1.3:1) reflected the large non-urban component of gonococcal disease in those regions. Male rectal and pharyngeal isolates were most frequently found in Victoria (31% of isolates from men), New South Wales (30%) and South Australia (20%) These percentages approximate those recorded in 2003 but also may reflect clinical sampling practices in those States. About three per cent of isolates are shown as being isolated from ‘other’ or unknown sites. These included six cases of disseminated gonococcal infection in men (0.2%) and seven (1.2%) 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

Table. Source and number of gonococcal isolates, Australia, 2004, by sex, anatomical site and state or territory

		State or territory						
	Site	NSW	NT	Qld	SA	Vic	WA	Aust
Male	Urethra	695	255	447	109	536	256	2,330
	Rectal	201	1	37	15	146	9	414
	Pharynx	118	1	14	13	102	4	253
	Other/NS	21	40	7	3	4	4	80
	Total	1,035	295	505	141	788	273	3,077
Female	Cervix	73	192	111	23	61	54	516
	Other/NS	5	24	5	25	5	2	43
	Total	78	216	116	25	66	56	559
Unknown	Total	0	4	0	0	0	0	4
Total*		1,113	515	621	166	854	329	3,640

* Includes isolates from Tasmania (22) and the Australian Capital Territory (20).

NS Not stated.

were probably from this source. There were a small number of isolates from the eyes (17) of both newborn and older infants and also adults, and from Bartholin's abscesses in women.

Antibiotic susceptibility patterns

In 2004, the AGSP reference laboratories examined 3,542 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 2004 by penicillin 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[8] – so-called CMRNG. Chromosomal resistance is defined by an MIC to penicillin of 1 mg/L or more. (The minimal inhibitory concentration in mg/L (MIC) 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.

Figure 1. Penicillin resistance of gonococcal isolates, Australia, 2004 by region

FS	Fully sensitive to penicillin, MIC ≥ 0.03 mg/L.
LS	Less sensitive to penicillin, MIC 0.06 – 0.5 mg/L.
RR	Relatively resistant to penicillin, MIC ≤ 1 mg/L.
PPNG	Penicillinase producing *Neisseria gonorrhoeae.*

Nationally, 770 (21.7%) gonococci were penicillin resistant by one or more mechanisms in 2004 and 377 (10.6%) were CMRNG and 393 (11.1%) were PPNG. The number and proportion of CMRNG increased sightly in 2004 compared to the 333 (9%) seen in 2003. The national increase in the number of PPNG in 2004 was more substantial, up from 306 (9%) in 2003.

Penicillin resistant gonococci were a particularly large proportion of gonococcal isolates in Victoria (30.8%; PPNG 10.3%, CMRNG 20.5%), Western Australia (30.4%; PPNG 18.8%, CMRNG 11.6%), and New South Wales (26.5%; PPNG 14.5%, CMRNG 11.9%). In South Australia and Queensland penicillin resistance was also high at 11.7 per cent and 12.1 per cent respectively. PPNG were prominent in Queensland (8.7%) and CMRNG were prominent in South Australia (7.1%). Three PPNG were identified in the Australian Capital Territory and four in Tasmania where three CMRNG were also identified. In the Northern Territory there were 19 PPNG and a single CMRNG giving a total of 4.2 per cent of strains being penicillin resistant. Thirteen of the PPNG were from Darwin and where acquisition data were available, all PPNG were acquired overseas. No penicillin resistant isolates were identified among gonococci isolated in Alice Springs. Data on acquisition were available in only 106 (27%) infections with PPNG. Fifty-one infections with PPNG were acquired locally and 55 by contact in South or South East Asian countries.

Ceftriaxone

Ceftriaxone is the third generation cephalosporin most used for treatment of gonorrhoea in Australia. The recommended dose for uncomplicated mucosal infection is 250 mg intramuscularly. To date there has been no instance of failure with ceftriaxone treatment attributable to decreased susceptibility described in Australia. Since 2001 however, low numbers of strains have been found with slightly raised ceftriaxone MICs. In 2002, there were 21 gonococci with ceftriaxone MICs > 0.03 mg/L isolated in Australia and 10 in 2003. In 2004, another 24 (0.7%) gonococci were seen with raised MICs. These were concentrated in New South Wales (22 of 24), with single isolates from the Northern Territory and Queensland. Isolates were usually also resistant to quinolones and penicillins, but spectinomycin sensitive. Recent Japanese data suggests that these strains are now more prevalent there, are multi-resistant and on occasion are associated with treatment failure with oral third generation agents not available in this country.[2,3,5]

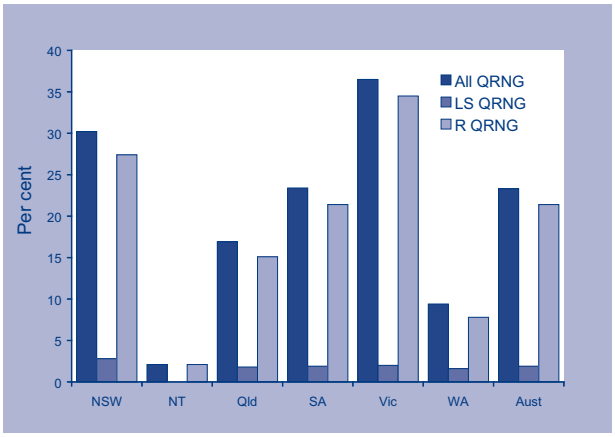
Spectinomycin

All isolates were susceptible. Resistance most often occurs as a result a single step ribosomal change.

Quinolone antibiotics

Figure 2 shows the distribution of gonococci with altered susceptibility to quinolones, by region. Thus far gonococcal resistance to the quinolone antibiotics is mediated only by chromosomal mechanisms so that incremental increases in MICs are observed. The AGSP uses ciprofloxacin as the representative quinolone and defines altered susceptibility as an MIC of 0.06 mg/L or more. 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 more often result in treatment failure. 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.

Figure 2. Percentage of gonococcal isolates less sensitive to ciprofloxacin or with higher level ciprofloxacin resistance and all strains with altered quinolone susceptibility, Australia, 2004, by region



LS QRNG MIC 0.06 – 0.5 mg/L.

R QRNG MIC 1 mg/L or more.

Nationally in 2004, 825 (23.3%) gonococci had some level of resistance to quinolones (QRNG). This represents a further substantial rise in quinolone resistance. In 2003, a total of 529 (14.4%) isolates were QRNG and in 2002 there were 389 (10%) QRNG detected. However, the volatility of these rates is demonstrated when the 638 gonococci (17.5%) QRNG seen in 2001 are considered. Most QRNG in 2004, (757, or 92% of all QRNG) had resistance at a higher level, i.e. MICs ≤ 1 mg/L.

The highest proportion of QRNG was seen in Victoria where the 309 QRNG represented 36 per cent of the total number examined. This is a further substantial increase in both the number (237) and proportion (26%) of QRNG seen in Victoria in 2003. In New South Wales, QRNG also increased markedly from 159 (14.4%) in 2003, to 331 (30%) in 2004. South Australia (36, 24%) and Queensland (103, 16.7%) also experienced increases in QRNG rates. In Western Australia, QRNG numbers were unchanged from 2003 and in other jurisdictions the numbers of QRNG were low (Northern Territory, 10; Tasmania, 3; Australian Capital Territory, 3).

Information on acquisition of QRNG was available in 179 of the 529 cases. One hundred and nine (60%) were acquired locally and the remainder overseas.

High level tetracycline resistance

The spread of high level tetracycline resistance in *N. gonorrhoeae* (TRNG) is examined as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea. There was an upsurge in TRNG isolation in 2002 when 11.4 per cent of strains of this type were detected nationally with little further change in 2003. There was a further increase in TRNG in 2004 to 490 (13.8% of all gonococci).

TRNG were present in all jurisdictions with the highest proportion in Western Australia (27%, 86 isolates) and New South Wales (18%, 195 isolates). Victoria (12.65%, 107) and Queensland (1.3%, 63) also had high rates. Lower numbers were found in South Australia (12) and the Northern Territory (19) and four isolates of TRNG were found in Tasmania and the Australian Capital Territory.

Discussion

The 2004 AGSP data show a further reduction in Australia of the susceptibility of gonococci to antibiotics used for treatment of gonorrhoea. Because of the pronounced regional differences in patterns of AMR in gonorrhoea in Australia, programmatic and standard treatment regimens are best derived from a consideration of local patterns of susceptibility rather than aggregated national data. As a guide to interpretation of AGSP data, the World Health Organization recommends that once resistance to an antibiotic has reached a level of five per cent in a population, continuing use of that agent should be reconsidered.

Penicillin resistance continued at a high rate in urban centres in 2004 and penicillin resistance in New South Wales, Victoria, South Australia, Queensland and Western Australia ranged between 12 and 30 per cent. Different mechanisms were responsible for these rates in different jurisdictions. In Western Australia and Queensland PPNG remained prominent and PPNG increased New South Wales. In Victoria and South Australia most penicillin resistance was chromosomal. PPNG were found in all jurisdictions including Tasmania, the Australian Capital Territory and the Northern Territory. In the latter jurisdiction, most PPNG seemed to be confined to the Darwin area and were in the main, imported infections.

Further increases in quinolone resistance were observed in 2004. QRNG were found in all centres, in higher numbers and proportions and with MICs in higher ranges. Local spread was also common. Jurisdictional rates of resistance ranged from two per cent in the Northern Territory to 36 per cent in Victoria where high rates of QRNG continue. In New South Wales the rate of QRNG increased substantially in 2004. Alternative treatments to quinolones should now be used in most settings in 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 Australia.

The AGSP has for several years observed and reported the presence of low numbers of gonococci showing some decreased susceptibility to ceftriaxone, and by inference other third generation cephalosporins. A similar observation was made in 2004 and most of the isolates with decreased susceptibility to ceftriaxone were found in New South Wales. Japanese isolates with raised MICs have been shown to possess mosaic penicillin-binding protein 2 (PBP-2) genes[5] possibly arising as a result of recombination events between *N. gonorrhoeae* and commensal *Neisseria*.[10] Only continued surveillance will reveal if further alterations resulting in still higher levels of resistance occur. Cephalosporin less susceptible isolates in Australia and elsewhere usually display resistance to multiple antibiotics.[4] All gonococci tested in Australia in 2004, including those with altered cephalosporin susceptibility, were susceptible to spectinomycin.

AMR surveillance conducted by the AGSP achieves consistently high technical standards through an on-going quality assurance program. For AMR surveillance it is necessary to obtain a sufficiently large and representative sample of isolates. The isolates obtained by the AGSP come from the public and private sector and are currently in sufficient numbers to detect resistance rates at the five per cent level. However the increasing use of non-culture based methods for the diagnosis of gonorrhoea decreases the number of gonococcal isolates available for testing. Thus a continuing commitment to maintenance of culture-based systems is required for the purposes of AMR surveillance, and alternative methods based on targeted culture of high-risk groups require exploration. Every effort should be made to obtain a gonococcal isolate from patients in whom treatment failure is suspected.

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Evaluation of the Australian Gonococcal Surveillance Programme

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Abstract

The Australian Gonococcal Surveillance Programme (AGSP) is a laboratory network that monitors the susceptibility of gonococcal isolates to antibiotics used in the treatment of infection. This report evaluates and reports on the simplicity, flexibility, sensitivity, representativeness, timeliness and acceptability of the AGSP. The World Health Organization's (WHO) Questionnaire for Assessment of Antimicrobial Resistance (AMR) National Networks was used in undertaking this evaluation and we report on the questionnaire's usefulness. The evaluation revealed that the AGSP was structurally simple, acceptable, timely and that the data were actively used by the stakeholders. However, the flexibility, representativeness and sensitivity of the AGSP are challenged by the increasing use of molecular based methods to diagnose gonococcal infections, as this is reducing the number of isolates available for testing. Despite this challenge, the AGSP has been able to identify differences in the antimicrobial susceptibility of gonococcal strains circulating in metropolitan and regional communities and the data generated are used to devise or modify standard treatment regimens for gonorrhoea. The functioning of the system can be improved by better availability of data through a dedicated website. Ideally, linkage of AGSP data to notification data would ensure that the AGSP is sensitive to and representative of the changes in gonococcal resistance amongst various sub-populations, although it will increase system complexity. The WHO questionnaire was found to be useful in undertaking the evaluation of the AGSP as it was simple and well-structured. However, the questionnaire needs to be expanded to include benchmarks that guide the assessment process. *Commun Dis Intell* 2005;29:142–148.

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

Introduction

Gonococcal infections are caused by the bacterium *Neisseria gonorrhoeae* and are almost always sexually transmitted. The disease is of public health importance as it impacts on sexual and reproductive health and amplifies the risk of HIV transmission by increasing the HIV viral load at the mucosa.[1]

Most effective treatments for gonococcal infections will eliminate the bacteria within 12 hours. The World Health Organization (WHO) recommends that an effective treatment should be a single dose of antibiotic that is prescribed on the patient's first presentation, where it should predictably cure at least 95 per cent of those treated.[2] To ensure that an antibiotic treatment is at least 95 per cent effective, public health practitioners need to monitor the rates of antibiotic resistance of the different gonococcal strains circulating in the community.

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A report released in 1999 by the Joint Expert Technical Advisory Committee on Antibiotic Resistance (JETACAR) led to the development of an Australia-wide strategy for the surveillance and management of antimicrobial resistance (AMR).[3,4] In the consultation phase of the strategy, existing AMR surveillance systems were to be evaluated. The aim of the evaluations was to develop longer term mechanisms to acquire national data on AMR. One of the first systems to be evaluated was the Australian Gonococcal Surveillance Programme (AGSP).

The AGSP is a network of laboratories that, among other activities, aims to enhance laboratory contributions to control gonococcal disease and monitors the susceptibility of gonococcal isolates to antibiotics used in the treatment of infection. The AGSP is a collaborative program, with a reference laboratory in each state and territory and a coordinating unit in Sydney. The network was established in 1979 and has been reporting on gonococcal AMR trends in Australia since 1981. The reference laboratories analyse isolates for AMR testing from any private or public laboratory or patient clinic submitting samples. The results for individual patients are reported back to the referring service and after analysis and comment, consolidated data are also reported to state and territory and national public health officials to monitor changes in gonococcal resistance circulating in the community.

This article reviews the process of gonococcal AMR surveillance in Australia that is undertaken by the AGSP. An evaluation of the simplicity, flexibility, sensitivity, representativeness, timeliness and acceptability of the system is presented. The article also describes the utility of the WHO's 'Questionnaire for Assessment of AMR National Networks' in evaluating the AGSP.[5] This may guide practitioners in future evaluations of AMR surveillance systems.

Methods

The guidelines for the evaluation of surveillance systems developed by the United States Centers for Disease Control and Prevention (CDC) were used in this assessment.[6] The attributes of the surveillance system simplicity, flexibility, sensitivity, representativeness, timeliness and acceptability were evaluated because of their importance to national AMR surveillance.

Simplicity was determined by assessing the flow of data in the system (collection, transmission, analysis and reporting). This also provided information about the timeliness of the system. Representativeness was evaluated by comparing the number of isolates tested by the AGSP with the total number of gonococcal notifications between 1995 and 2003. Sensitivity was assessed by examining whether the AGSP tests enough isolates to detect changes in AMR and flexibility was evaluated by identifying examples of the system's ability to adapt to changes in testing methods. Acceptability was assessed through survey-based consultation with AGSP stakeholders. Stakeholders surveyed included the AGSP reference laboratories (n=8); private and public referral laboratories (n=33); sexual health clinics (n=28); public health officials at the state and territory, and national level (n=21); and international stakeholders (WHO and the International Collaboration on Gonorrhoea, n=30).

The WHO Questionnaire for Assessment of AMR National Networks contains two components: a component assessing individual laboratory status with respect to infrastructure and capacity, and a component evaluating the overall functioning of the surveillance network. In Australia, the first component is dealt with by an existing system of laboratory accreditation (National Association of Testing Authorities) and it was excluded from the evaluation. The second component was assessed in the current evaluation to evaluate its application to Australian systems.

Results

Description of the Australian Gonococcal Surveillance Programme

The AGSP reference laboratories receive isolates from various public and private laboratories in their respective state or territory. Even though the number of contributing laboratories varies over time due to laboratory mergers and takeovers, the AGSP central coordinating unit in Sydney estimates that 85 per cent of laboratories in New South Wales contribute data, including private laboratories. Some sexual health clinics send samples for diagnosis and AMR testing directly to the AGSP reference laboratories.

The reference laboratories use a standard agar plate dilution methodology in AMR testing.[7] This was developed about 25 years ago and has been the basis for national studies on gonococcal AMR. An AGSP-specific quality assurance program which uses internationally validated reference cultures, is used to validate results.[8] Most isolates are tested and stored in the jurisdiction's reference laboratory.

The AGSP reference laboratories collect identifying information about the patient (name, date of birth, gender) and site of sample isolation. The AGSP also attempts to collect clinical notes and data on geographic acquisition, but these are rarely provided by the referring practitioner or laboratory. At a national level, the AGSP collects de-identified data including gender, state or territory for each isolate tested and site of sample isolation. There is good completion of the data fields collected at the national level, where

between 1996 and 2003 an average of only 0.5 per cent of isolates were missing these data (range 0.2% to 0.8%).

The state and territory AGSP reference laboratories maintain databases that have identifying information on each case in line-listing format. Each reference laboratory uses a different information technology system, depending on what is utilised by their hosting organisation. One concern with the current data flow is that there is duplication of data entry for the isolates received from the initial diagnostic laboratories, decreasing simplicity, acceptability and timeliness.

For the national data, the state and territory reference laboratories send aggregated data to the AGSP coordinating unit in a standard summary aggregated format, quarterly. The coordinating unit then collates and maintains the information in a Microsoft Access database. The states and territories sometimes delay sending the summary information due to the need to complete laboratory analyses of late-arriving referred isolates and to revise and clean their data e.g. of duplicate samples. Despite this, the national network has consistently reported on a quarterly and annual basis to the various stakeholders within six months of the end of the time period.

The AGSP disseminates information to a variety of stakeholders. At a local level, the reference laboratories circulate information to clinicians about recommended standard treatments through emails or by responding to queries. At a state and territory level, the AGSP produces updates on gonococcal AMR trends to public health units; sexually transmissible infections (STI) clinics; divisions of general practice; and laboratories through state and territory health bulletins. At the national and international levels, the AGSP produce quarterly and annual reports that are published in *Communicable Diseases Intelligence* (*CDI*). *CDI* is distributed to government agencies, public health practitioners, general practice and laboratories.

Usefulness of the Australian Gonococcal Surveillance Programme

The surveillance of gonococcal resistance patterns is essential for the establishment and modification of standardised treatment regimens for gonorrhoea and enables the economic and acceptable use of antimicrobials in this disease. One example of where the AGSP data were useful is in Western Australia, where regional and rural areas have different standard treatment regimens to those recommended in metropolitan areas. Despite widespread resistance to penicillins in many parts of Australia, the AGSP has demonstrated that most strains circulating in the regional and rural communities remain sensitive to penicillins. The penicillins (usually given orally as amoxycillin, often combined with clavulanic acid) are both cheaper and easier to administer than intramuscular ceftriaxone, which is the recommended treatment in metropolitan settings because of AMR. The Table shows cost comparisons of the two formulations based on standard purchasing procedures for individual patients. Although these costs would differ with bulk purchasing arrangements, they are indicative of the substantial price differentials between the two treatments.

In Western Australia, approximately 580 gonococcal infections per year are locally acquired and non-metropolitan (based on data from the last four years). Since the treatment regimen in Western Australia for this category of patients is penicillin, then the overall cost savings are substantial. There are also benefits at the patient level, where penicillin is a more acceptable drug than ceftriaxone since it is easy and pain-free to administer.

Evaluation

The evaluation of the system attributes and findings from the stakeholder surveys are presented below. The stakeholder survey was distributed to 90 national stakeholders and 30 international stakeholders. The response rate for the national stakeholders was 57 per cent (n=51) and 27 per cent (n=8) for the international stakeholders. One

Table. Comparison of drugs used in the treatment of gonococcal infections

Attribute	Penicillin (amoxycillin)	Ceftriaxone
Administration	Oral	Intramuscular
Cost*	$7.86 per course	$56.30 per course
Acceptability for patient	Few side effects, no associated administration equipment or expertise required	Very painful, additional cost of equipment and expertise needed to administer
Pharmaceutical Benefit Scheme*	Yes – easily accessible to clinicians	Restricted benefit

* Data obtained from MIMS Online, 2004[9]

potential reason for the low response rate was that questionnaires were sent to various individuals in most organisations, where co-workers may have collated their views and returned a single response or assigned a single person to respond on behalf of the group. The low response rate could also be attributed to the fact that email surveys are easily forgotten or ignored, although a reminder was sent after two weeks. The low response rate for the international stakeholders, most of whom were in the WHO system, may reflect the high turnover of technical staff in that organisation.

Simplicity

The AGSP is a relatively simple system as all the reference laboratories use standardised testing methods and the common Public Health Laboratory Network case definition. As Figure 1 shows, there are clear mechanisms for data flow through the system. The AGSP keeps the database fields to a minimum to help reduce the complexity of data collection and analysis.

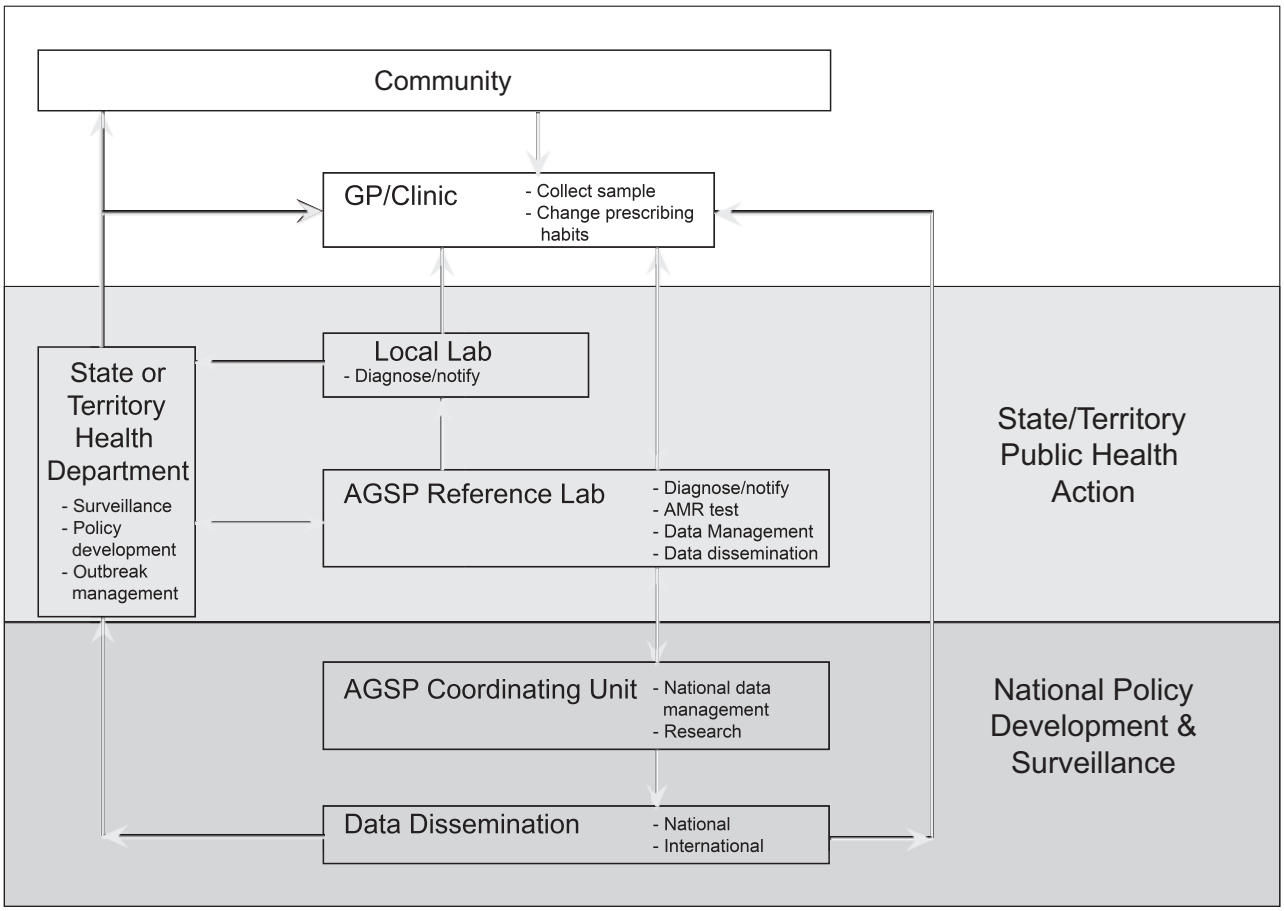
The majority (88%) of stakeholders responding to the survey thought that the AGSP was a simple system. Of those who disagreed, issues such as poorly defined jargon in annual reports and an ambiguous network structure were identified as concerns. One respondent noted, however, that the outputs of the system are too simple and that more detail in the annual reports is needed.

Flexibility

The network has had to adapt to some challenges. Since isolates currently need to be cultured for AMR testing, the introduction of molecular based methods to diagnose gonococcal infections has challenged the flexibility of the AGSP. Molecular based methods are cheaper and faster to use and have been eligible for rebates from Medicare since 1999. This is problematic for gonococcal AMR surveillance as it can decrease the number of isolates available for testing. To adapt to this advance in technology, the AGSP is in regular communication with public and private laboratories to remind them to forward any available isolates.

As more antimicrobials have been introduced for the treatment of gonococcal infections, the AGSP has adapted by introducing new controls and modifying quality assurance to include new resistance testing.

Figure 1. Flow chart of the Australian Gonococcal Surveillance Programme



Sensitivity

Sensitivity is an important attribute since the AGSP aims to detect changes in AMR over time so that public health action can be taken if resistance to a particular antimicrobial exceeds five per cent. Currently, the AGSP tests over 3,000 isolates each year nationally because the system is comprehensive and continuous. Even though this is a large number of isolates, it is difficult to assess whether this number is sufficient to detect significant changes in gonococcal AMR rates in sub-populations, such as men who have sex with men or rural populations. Detecting changes in sub-populations is only possible if clinical and risk factor information is made available to the AGSP laboratories, but this occurs infrequently and is beyond the capacity of laboratory-based systems to obtain, so little is known about these groups.

One measure of the system's sensitivity and usefulness is its ability to detect outbreaks of particular types of gonococcal infections distinct from issues of AMR. In 2002, the New South Wales reference laboratory noticed an increase in the number of disseminated gonococcal infections. The laboratory typed the isolates and found that they were identical. The reference laboratory informed the public health units, sexual health clinics and NSW Department of Health. The outbreak came to an end one year later. The AGSP also gave advice on patient treatment and follow-up in this outbreak, which may have reduced the infectivity period of cases.

Representativeness

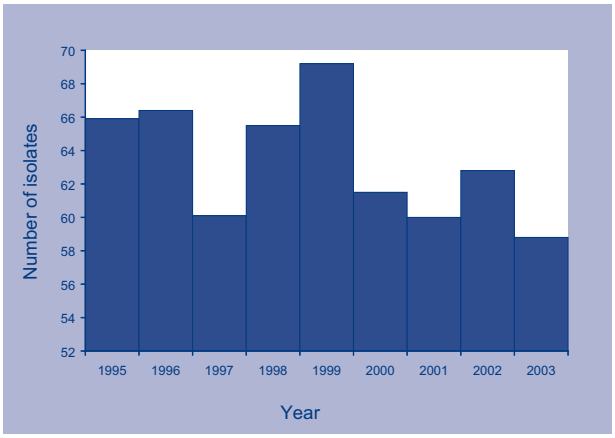
Between 1995 and 2003, the total isolates tested annually by AGSP averaged 63.1 per cent of the total gonococcal notifications in Australia (Figure 2). The representativeness of the system is affected by the increasing reliance on molecular based methods to diagnose gonococcal infections, which has decreased the number of isolates available for AMR testing. This is especially problematic in remote settings where the reliance on molecular based methods is increasing due to its cost efficiency. In addition, due to logistic issues of specimen collection and transport, molecular based testing is the only practical test method in remote communities. In time, this may compromise the ability to detect AMR patterns in these communities, and other ways of assessing the effectiveness of treatment regimens will need to be found.

Despite these challenges to representativeness, the AGSP has been able to show that a large proportion of the gonococcal infections contracted in larger cities is in homosexually active men where antibiotic resistant gonococci are observed, whilst gonococcal infections in rural settings are mostly transmitted heterosexually and gonococci are less antibiotic resistant.[11] To increase confidence in the inferences made about the patterns of resistance in specific sub-populations, some jurisdictions such as Western Australia attempt to integrate AMR data with clinical information. This includes anatomical site of infection (with rectal/pharyngeal gonorrhoea a surrogate for homosexual acquisition), overseas travel history and Indigenous status. The findings of these state health systems support the findings of the AGSP that gonococcal strains circulating in regional communities are less resistant than those circulating in metropolitan communities.

Timeliness

The timeliness of the national AGSP system is dependent not only on the efficiency of all the state and territory reference laboratories, but also on the timeliness of laboratories referring isolates for testing. Despite these constraints, the AGSP have continuously reported on a quarterly and annual basis in *CDI* within six months of the end of the reporting period. This is adequate for reporting on AMR trends.[2] Furthermore, the majority of stakeholders (78%) thought that the AGSP was a timely system.

Figure 2. Gonococcal isolates tested for antimicrobial resistance proportional to the annual notifications, 1995 to 2003



Acceptability

The acceptability of the system is high for the contributors to the system, where all reference laboratories have participated continually over the last 25 years. This is a very positive finding in the evaluation as it highlights the dedication of the individual laboratories to gonococcal AMR surveillance.

Ninety-two per cent of surveyed stakeholders believe that the AGSP contributes to the public health control of gonococcal infections. Of the 59 stakeholders responding to the evaluation survey, a large proportion used the AGSP data for patient treatment (n=30), treatment regimen change (n=28), outbreak detection and control (n=20), research (n=23) and disease prevention (n=17). The majority (83%) of stakeholders also noted that the outputs are well presented. Remarks made by the respondents included:

> 'I think the AGSP has functioned very well to date and has amassed a dataset that is the envy of the Western World because all contributors use a standard methodology to generate the data'.

On the other hand, users of the system noted the breakdown in the feedback of surveillance data as an issue:

> 'It would be nice to nominate a Public Health position in each region where the reports are routinely sent. It took me a year to get the first report and prior to that I would access the yearly reports from *CDI* which do not include as much details as I need' (State department public health officer).

This is currently a major concern in the surveillance activities of the AGSP. Even though report dissemination is timely for public health action, the evaluation found that some important public health stakeholders did not have access to the information. One potential mechanism of reaching this audience is through a website, where users of the system can access up-to-date reports, enhancing the accessibility and usefulness of the data. Forty-three (73%) respondents thought that a website would be a good idea. Respondents proposed that the website should contain the following information:

- network organisational chart and contact details of the reference laboratories;
- Public Health Laboratory Network case definition;
- protocols and surveillance data (e.g. local information on case clusters of disseminated gonococcal infections);
- mapping geographic distribution of AMR;
- risk factor data by geographic distribution (e.g. gender, sexual preference, travel, age, site of infection, Indigenous status);
- past reports and a general list of relevant articles, PowerPoint presentations produced by the reference laboratories and resources for medical students; and
- links to national and international websites relevant to the field.

The World Health Organization guidelines

The WHO's Questionnaire for Assessment of AMR National Networks was useful in evaluating the AGSP. The questionnaire recommended the collection of general information including population served by the network, years in operation and frequency of national data collation. However, the questionnaire did not provide quantitative or qualitative benchmarks to judge the adequacy of the network's activity levels on these attributes. One example was the question 'does the network receive regular and complete AMR data reports from all participating laboratories'? For this question, the evaluator does not have an estimate of acceptable levels of 'regular' and 'complete'.

The next section of the WHO questionnaire assessed quality control. This section was very useful for conducting the evaluation from a public health perspective as opposed to a laboratory perspective where it succinctly examined aspects of laboratory quality control that are relevant to AMR testing. Examples of this include whether the network has an external quality assurance program and whether the methodologies used at each laboratory enable comparability. For future evaluations of AMR systems, this section will be valuable for evaluators with little laboratory quality assurance knowledge.

The last section of the questionnaire considered the network's dissemination of information. Even though the questions were relevant, they were too general. For example, the question 'are the results of the AMR surveillance network regularly and effectively communicated to decision-makers in the Ministry of Health or other governing bodies?' needs clearer definitions of 'regular' and 'effective' to provide useful information.

Based on the current evaluation experience, it would have been useful if the questionnaire explored the integration of AMR networks with other public health surveillance systems or a national AMR strategy. Overall, until the questionnaire is expanded and benchmarks are provided to guide the assessment process, it is advisable to use the questionnaire in

conjunction with other tools such as WHO's surveillance standards for AMR or the CDC guidelines for the evaluation of surveillance systems.[2,6]

Conclusions and recommendations

The AGSP is a clearly defined laboratory based AMR surveillance system with stated objectives. It has been operating for many years using standardised methodologies including rigorous quality control components and has been meeting its goals. The system is connected with international programs such as the International Collaboration on Gonorrhoea and is regarded by many stakeholders as a model for good practice.

The strengths of the system are the quality of the methods used to generate the data, its high acceptability and its usefulness. The stakeholders currently utilise the outputs for a variety of purposes, including treatment of patients, control and research.

The major weaknesses of the system are the poor accessibility of the outputs to stakeholders and the technological challenges to its flexibility and representativeness. Information needs to be disseminated quickly and effectively to clinicians and public health practitioners in the states and territories to respond to the rapidly changing epidemiology of the disease. The increasing use of molecular methods for diagnosis may in time challenge the ability of the system to adequately monitor AMR and the ongoing representativeness of the system must be carefully monitored.

The problems identified by the evaluation would be improved by:

- reviewing the AGSP stakeholders in the states/territories and having points of contact within the health departments for communication of AGSP data; and
- establishing a website to enable ready access to AGSP data and information.

The dataset would undoubtedly be improved if state and territory health departments were able to enhance the dataset for gonococcal infections and to enable greater data linkage. This would be useful to allow reporting on the number of isolates by population density (rural/metropolitan) and age and gender and enable targeted health campaigns and to monitor gonococcal resistance in various subpopulations. However, these expanded objectives would increase the complexity of a currently simple and well functioning laboratory system which is currently meeting its remit under JETACAR recommendations for AMR surveillance.[3,4]

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Annual report of the Australian Meningococcal Surveillance Programme, 2004

The Australian Meningococcal Surveillance Programme

Abstract

This report by the National Neisseria Network, a nationwide collaborative laboratory programme, describes 361 laboratory-confirmed cases of meningococcal disease diagnosed in Australia in 2004. The phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility of 245 isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were determined and an additional 116 cases were confirmed by non-culture based methods. Nationally, the majority of cases were serogroup B (243 isolates, 68%) or serogroup C (71 isolates, 20%) meningococci. The total number of cases was 133 fewer than the 494 cases identified in 2003 and the number of confirmed cases decreased in all jurisdictions except Western Australia where the total was unchanged. There was a 15 per cent decrease in serogroup B infections, but a greater (45%), fall in the number of serogroup C cases. The age distribution of meningococcal disease showed a typical primary peak in those aged four years or less with a secondary peak in adolescents and young adults. Serogroup B cases were 88 per cent of all cases in those aged four years or less and 63 per cent in those aged 15–24 years age range. The proportion of all invasive disease represented by serogroup C disease was highest in the 15–24 years and older age groups. The common phenotypes circulating in Australia were B:4:P1.4 and C:2a:P1.4. However, significant jurisdictional differences in the serogroup and phenotypic distribution of meningococci was again evident and considerable heterogeneity of subtypes was noted. No evidence of sustained transmission of meningococci undergoing 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 to 0.5 mg/L). A single isolate was penicillin resistant at 1 mg/L. *Commun Dis Intell* 2005;29:149–158.

Keywords: disease surveillance; Neisseria meningitidis

Introduction

The National Neisseria Network (NNN) is a collaborative national programme of reference laboratories in each state and territory of Australia. It examines those aspects of microbiological laboratory medicine relevant to the public health control of invasive meningococcal disease (IMD), namely, diagnosis, antimicrobial resistance surveillance and organism typing, including both isolate-based and non-culture derived methodologies. The first reports from the Meningococcal Surveillance Programme, which began in 1994, relied solely on data derived from examination of isolates from culture-positive cases of IMD, in particular, their phenotype and antibiotic susceptibility. Increasingly, data have been derived from non-culture based methods, notably the genotype and diagnoses based on nucleic acid amplifications assays (NAA), and have been included in reports. The information is provided to supplement that from clinical notification schemes.

In 2003, a publicly funded program of vaccination of children and adolescents with serogroup C conjugate vaccine was commenced and generally was fully operational in 2004. This report analyses information gathered by the NNN on laboratory-confirmed cases of IMD in the calendar year 2004. The format departs from previous annual reports published in *Communicable Diseases Intelligence*[1–10] insofar as aggregated data on all laboratory-confirmed cases are now analysed together.

Methods

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

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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 the Public Health Laboratory Network definitions. Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was sought. The isolate-based subset of the program categorises cases on the basis of site of isolation of the organism. Where an isolate is grown from both blood and cerebrospinal fluids (CSF) cultures in the same patient, the case is classified as one of meningitis. It is recognised that the total number of cases and particularly the number of cases of meningitis e.g. where there was no lumbar puncture or else where lumbar puncture was delayed and the culture 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/resistance when determined by a standardised agar plate dilution technique:[11]

sensitive	MIC ≤ 0.03 mg/L;
sess 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 NAA and serological techniques. NAA testing is essentially by polymerase chain reaction (PCR) techniques[12] 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 annual 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.[13–15] 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 361 instances of laboratory-confirmed cases of IMD in 2004 (Table 1) compared with 494 cases in 2003. In 245 cases a positive culture was obtained with or without a positive non-culture based test and 116 cases were confirmed by a non-culture based method alone. The total number of all laboratory-confirmed cases fell in 2004 compared with 2003 in all jurisdictions except in Western Australia where identical numbers were recorded in each year. There were 47 fewer laboratory-confirmed cases in New South Wales in 2004, 38 fewer in Victoria, 21 fewer in Queensland, 17 fewer in South Australia, five fewer in the Northern Territory and three fewer in the Australian Capital Territory and Tasmania.

Seasonality

Eighty-four (23.5%) of cases occurred between 1 January and 31 March 2004, 99 (27.5%) between 1 April and 30 June, 109 (30%) between 1 July and 30 September and 69 (19%) between 1 October and 31 December 2004. A winter peak of meningococcal disease is usual.

Table 1. Number of laboratory-confirmed cases of invasive meningococcal disease, Australia, 2004, by state or territory and serogroup

State or territory	Serogroup						Total
	B	C	A	Y	W135	NG	
ACT	3	8					11
NSW	78	18		3	5	19	123
NT	5	1			1		7
Qld	50	19	1	2	2	1	75
SA	13	1					14
Tas	8	5			1	3	17
Vic	55	13		3	2	3	76
WA	31	6			1		38
Australia	243	71	1	8	12	26	361

NG Not groupable.

Age distribution

Nationally, the peak incidence of meningococcal disease was again in those aged four years and under (Table 2, Figure 1). Those aged less than one year or in the 1–4 years age group accounted for 49 (13.7%) and 62 (17.2%) cases respectively. The combined total of cases confirmed by all methods in these two groups (111) is less than that in 2003 (140). However, these two age groups together comprised a similar proportion of all cases in 2004 (30.9%) as in 2003 (28.3%). A secondary disease peak is also usual in the 15–19 years age group. The total of 61 cases (17% of all confirmed cases) in this age group in 2004 was less than the 89 (18%) seen in 2003. Those aged 15–24 years together accounted for 96 cases (26.7%).

Serogroup data

The serogroup of the meningococci causing disease was determined in 335 cases. Two hundred and forty-three (73%) were serogroup B, 71 (21%) serogroup C, one serogroup A, 8 (2%) serogroup Y and 12 (3.3%) serogroup W135. The serogroup was not determined in one culture confirmed case, in 10 of 101 cases confirmed by NAA or in any of the 12 serologically confirmed cases. In 2003, a total of 285 (58%) cases of serogroup B and 155 (31%) cases of serogroup C IMD were identified from a total of 494 laboratory-confirmed cases.

The serogroup distribution varied with age (Figure 2) and jurisdiction (Table 2), as in previous years. 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 1. Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2004, by age

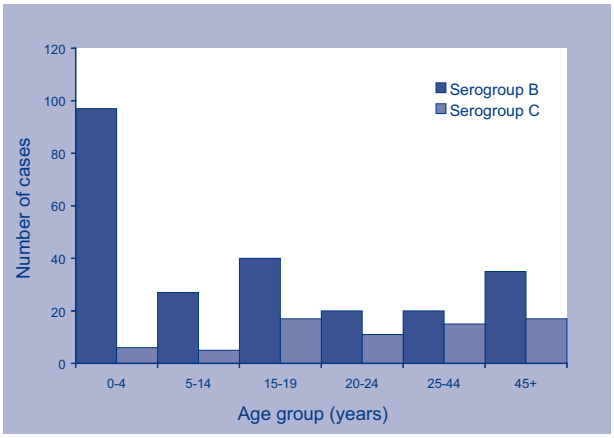


Figure 2. Serogroup B and C meningococcal disease as a percentage of cases of invasive meningococcal disease confirmed by all methods, Australia, 2004, by age

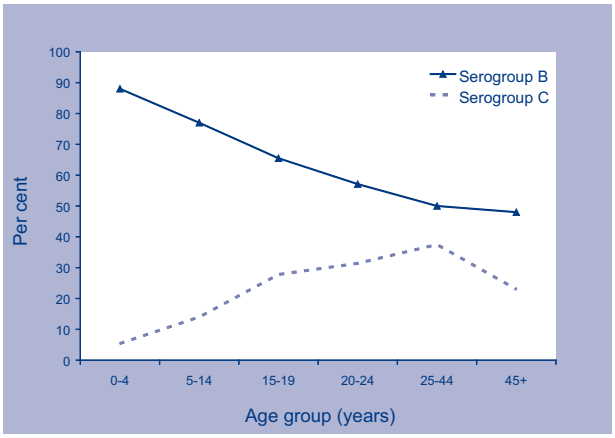


Table 2. All laboratory-confirmed cases of invasive meningococcal disease, Australia 2004, by age, jurisdiction and serogroups

State or territory	Serogroup	Age group										Total
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
ACT	B	1	0	0	0	1	0	0	0	0	1	3
	C	0	1	0	1	0	2	2	2	0	0	8
	Total	1	1	0	1	1	2	2	2	0	1	11
NSW	B	14	19	9	2	8	6	3	11	3	3	78
	C	0	1	0	0	3	3	6	5	0	0	18
	Total	15	24	10	3	13	13	15	23	4	3	123
NT	B	0	3	0	0	0	1	0	1	0	0	5
	C	0	0	0	0	0	0	0	1	0	0	1
	Total	0	3	0	0	0	1	0	3	0	0	7
Qld	B	8	11	3	3	11	4	5	2	3	0	50
	C	1	0	0	1	11	2	3	0	1	0	19
	Total	10	11	3	5	23	6	9	3	5	0	75
SA	B	3	1	0	0	2	0	4	3	0	0	13
	C	1	0	0	0	0	0	0	0	0	0	1
	Total	4	1	0	0	2	0	4	3	0	0	14
Tas	B	3	3	0	0	2	0	0	0	0	0	8
	C	0	0	0	1	2	0	1	1	0	0	5
	Total	3	3	0	1	4	0	1	4	1	0	17
Vic	B	10	8	5	2	8	7	5	5	5	0	55
	C	1	1	0	0	1	1	2	5	2	0	13
	Total	12	10	5	2	10	8	7	13	9	0	76
WA	B	4	9	2	1	8	2	3	1	1	0	31
	C	0	0	1	1	0	3	1	0	0	0	6
	Total	4	9	3	2	8	5	4	1	2	0	38
Australia	B	43	54	19	8	40	20	20	23	12	4	243
	C	3	3	1	4	17	11	15	14	3	0	71
	Other	3	4	1	2	4	4	5	15	6	0	47
	Total	49	62	21	14	61	35	42	52	21	4	361
	%	13.6	17.2	5.8	3.9	16.9	9.7	11.6	14.4	5.8	1.1	100

NS Not stated.

Totals include cases due to other serogroups (n = 21) and cases where the serogroup was not determined (culture confirmed 1, NAA confirmed 13 and serology confirmed 12).

In 2004, 97 (88%) 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. In those aged 5–14 years, 27 serogroup B meningococcal cultures represented 77 per cent of the 35 confirmed cases and the five cases of serogroup C represented 14 per cent. The 96 confirmed cases in those aged 15–24 years comprised 60 (63%) serogroup B and 28 (29%) serogroup C. Half of the 117 infections in older age groups were serogroup B and a quarter were serogroup C.

When data from 2004 and 2003 are compared, the number of both serogroup B and serogroup C cases in 2004 was lower in all the above age groups (Table 3). The decrease in serogroup C cases in 2004 when compared with 2003 was proportionally greater than the reduction in the number of serogroup B cases. Serogroup B infections thus represented a higher proportion of all cases in 2004 than in 2003.

Jurisdictional differences in the distribution of serogroup B and C meningococcal cases continued in 2004 (Table 1). Serogroup B infections predominated nationally and in all jurisdictions except the Australian Capital Territory where 8 of 11 confirmed cases were with serogroup C. In New South Wales, Queensland, South Australia, Tasmania and Victoria, the number and proportion of cases represented by serogroup C meningococci decreased. Substantial decreases in the number of serogroup C infections were noted in Victoria (from 47 in 2003 to 13 in 2004), New South Wales (44 to 18) and Queensland (37 to 19). Lower numbers of serogroup C cases were also seen in Tasmania (decrease in 2004 by 3), the Australian Capital Territory (by 2) and South Australia (by 1). The number of serogroup C cases increased by one in the Northern Territory and in Western Australia.

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

Outcome data (survived or died) were available for 225 (63%) of the 358 laboratory-confirmed cases (Table 4). Eighteen deaths were recorded in this group (8%) (Table 4). Outcomes were available for 160 of 243 (66%) serogroup B infections and 34 of 71 (48%) serogroup C infections. There were 13 (8.1%) deaths in serogroup B infections and 3 (8.8%) in serogroup C infections.

There were two deaths in 26 patients (7.7%) with meningitis; both of these patients were infected with a serogroup B strain. Fifteen deaths were recorded in 181 bacteraemic patients (8.3%). There were 135 cases of serogroup B meningococcal bacteraemia with 10 deaths (7.4%) and 30 cases were caused by serogroup C strains among whom three fatalities were recorded (10%). No fatalities were recorded with serogroup Y (5 cases), but there were two fatalities among the seven instances of serogroup W135 bacteraemia.

Table 3. A comparison of the number and proportion of serogroup B and serogroup C confirmed cases, 2004 and 2003, by age

Year	Serogroup	Age							
		< 4 years		5–14 years		15–24 years		25+ years	
		n	%	n	%	n	%	n	%
2004	B	97	88	27	77	60	63	59	50
	C	6	5.5	5	14	28	29	32	27
	All	110		35		96		117	
2003	B	116	83	33	53	69	48	66	47
	C	14	10	21	33	67	44	51	36
	All	140		63		151		140	

Table 4. Outcome data (survived, died) for laboratory-confirmed cases of invasive meningococcal disease, 2004, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	12	3	1	0	2	24
	Died	2	0	0	0	0	2
	Total	20	3	1	0	2	26
Septicaemia	Survived	125	27	4	5	5	166
	Died	10	3	0	2	0	15
	Total	135	30	4	7	5	181
All cases	Survived	147	31	5	5	19	207
	Died	13	3	0	2	0	18
	Total	160	34	5	7	19	225

NG Not groupable.

Phenotypes of invasive meningococcal isolates

Isolates continue to be phenotyped and considerable heterogeneity amongst invasive isolates was again evident. The predominant serotypes/serosubtypes in each state and territory are shown in Table 5. Serogroup B meningococci are in general more heterogeneous, but also more difficult to characterise by serological methods and a number could not be phenotyped. Nineteen isolates of the B:4:P1.4 phenotype were identified in Victoria, New South Wales, Queensland and Western Australia. Numbers 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 and 25 in 2003. Historically, the other common phenotype circulating has been B:15:P1.7 but only eight strains of this type were seen, five of them in New South Wales.

Table 5. Commonly isolated serotypes and serosubtypes and phenotypes of *Neisseria meningitidis* of interest, Australia, 2004, by state or territory

State/territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
ACT		0		0	2a	7	1.4	2
							1.5,2	1
							nst	4
NSW	4	16	1.4	8	2a	1	1.5	6
			1.14	3			1.5,2	0
			1.15	3			1.2	0
			nst	2			1.4	1
							nst	4
	15	7	1.7	5	14	`	1.14	1
			1.6	1	NT	3	1.5	2
			1.13	1			1.13	1
	nt	28	1.4	4				
			1.7	1				
			1.15	2				
			others	5				
			nst	16				
NT	14	2	nst	2	2a	1	1.5	1
Qld	4	4	1.4,(7)	2	2a	13	1.4	6
			1.14	2			1.5	5
	15	4	1.7,(1)	4			nst	2
	1	3	1.14	2	14	1	1.12,13	1
			1.2	1				
	nt	13	1.4	6				
			1.7	1				
			1.5,2	1				
			1.15	1				
			nst	3				
Tas	4	2	1.19,15	2	2a	4	1.4, 7–2	4
Vic	4	13	1.4	8	2a	8	1.4	7
			1.15	2			1.5,10	1
			1.16	1			1.14	1
	1	1	1.14	1	4	1	1.16	1
	15	6	various		nt	1	1.12,13	1
	2b	1	1.16	1				
	nt	18	1.15	8				
			1.14	4				
			nst	3				
WA	4	2	1.4	1	2a	1	1.4	1
			nst	1	nt	2	1.5	1
	15	1	nst	1			nst	1
	nt	1	1.16	1				

nt Not serotypable.

nst Not serosubtypable.

Of interest were any serogroup B meningococci of serotypes 2a or 2b. These serotypes are more often seen in serogroup C organisms, but in 2004, a single isolate only of phenotype B:2b:P1.16 was detected.

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 2004, 21 serogroup C isolates were of this serotype/serosubtype. In 2003, there were 29 isolates with this phenotype. This phenotype was detected in all jurisdictions except the Northern Territory and South Australia. Seven were found in Victoria, six in Queensland, four in Tasmania and two in the Australian Capital Territory with single examples in New South Wales and Western Australia. All except three of the typeable serogroup C isolates was of serotype 2a. Serotype 2b strains were not detected in serogroup C isolates.

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 53 isolates from CSF either alone or with a blood culture isolate and 186 from blood cultures alone. There were six other isolates from synovial fluid and tissues. Trends in relative rates of positive isolates have been followed in these reports (Figure 3). The ratio of CSF isolates to blood culture isolates was 0.28:1. For PCR based diagnosis, this ratio was 0.73:1. This probably reflects the capacity of PCR to amplify meningococcal DNA even after antibiotic treatment and/or delayed lumbar puncture.[16]

Table 6. Anatomical source of samples positive for a laboratory-confirmed case of invasive meningococcal disease, Australia, 2004

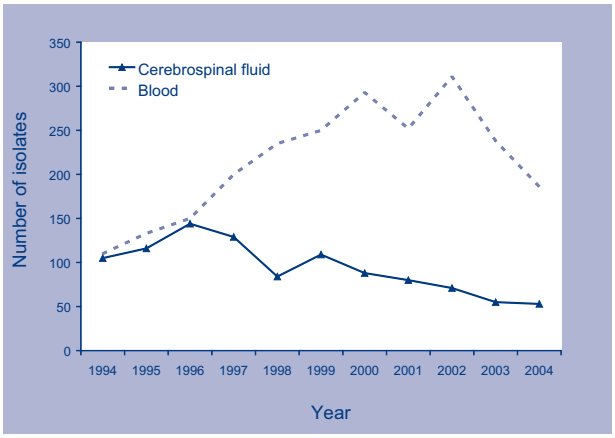
Specimen type	Isolate of MC	PCR positive*	Total
Blood	186	59	245
CSF +/– blood	53	43	96
Other†	6	2	8
Serology alone‡			12
Total	245	104	361

* 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 PCR.

Figure 3. Numbers of meningococcal isolates from cerebrospinal fluid and blood culture, 1994 to 2004



Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

Two hundred and thirty-eight isolates were available for determination of their susceptibility to penicillin. Using defined criteria, 90 strains (38%) were fully sensitive to penicillin and 147 (62%) less sensitive (MIC 0.06 to 0.5 mg/L). These proportions are similar to those observed in recent years. One isolate from a blood culture had an MIC of one mg/L and six isolates, from blood cultures (3), CSF (2) and joint fluids (1), had MICs of 0.5 mg/L.

Other antibiotics

All isolates were susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins) and to the prophylactic agents ciprofloxacin and rifampicin.

Discussion

There were 361 laboratory-confirmed cases of IMD in 2004, 245 (67.9%) by culture of *N. meningitidis* and 116 (32.1%) by non-culture based methods. The 245 isolates examined by NNN laboratories in the Australian Meningococcal Surveillance Programme (AMSP) in 2004 was the lowest number recorded since the 216 examined in the first year of the program in 1994. The annual numbers of isolates examined from 1997 to 2002 have ranged between 323 and 393 with 303 available in 2003.

This AMSP report is for the first time, based on a combined analysis of all cases of IMD confirmed by any recognised method. In earlier NNN reports, analyses were derived from culture confirmed cases, with NAA based data added from 1999. The increased contribution of non-culture-based

methods to IMD diagnosis means that a more comprehensive picture can be obtained if diagnoses by all test modalities are aggregated.

In 2004, the number of laboratory-confirmed cases of IMD was less in each jurisdiction than in 2003, except in Western Australia where 38 cases were identified in both years. Numerically, larger reductions in numbers were seen in New South Wales, Victoria, Queensland and South Australia. Serogroup B and serogroup C infections again predominated although numbers of both serogroups were less than those recorded in recent years. However the reduction in the number of serogroup C infections from 155 in 2003 to 71 in 2004 (54%) was proportionally greater than the decrease in serogroup B infections from 285 to 243 (15%). This was true for all age groups (Table 4) so that serogroup B infection accounted for a greater percentage of all IMD in 2004. NNN reports have consistently noted that the age distribution of IMD showed a primary peak in those aged four years or less and was predominantly with serogroup B meningococci while in a secondary peak in adolescents and young adults the proportion of serogroup C infections increased. This pattern was again observed in 2004. Serogroup C disease remained an important element of IMD in young adults and older age groups.

Analysis of the effect of the national vaccination program with serogroup C conjugate vaccine is beyond the scope of this report. Specific mention was made in the 2003 report of the caveats placed on AMSP data if used to assess disease rates and effects of vaccination campaigns.[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. Any assessment of the impact of the vaccination program on IMD rates will thus require a continuing and detailed analysis.

Preliminary data since the vaccination campaign indicate that some meningococci isolated show evidence of genetic recombination. Clonal complexes of meningococci responsible for IMD may express different capsular polysaccharides or recombination of *porA* and *porB* and other genes may occur. A number of strains that show these characteristics have been detected in Australia, but only in low numbers to date. However close attention should continue to be paid to analysis of meningococcal subtypes and any evidence of their clonal expansion thoroughly investigated.

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 one mg/L was detected in a single strain in 2004. NNN trend data show no recent 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.

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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Meningococcal vaccine failure in conjunction with an unusual meningococcal cluster in southern Tasmania

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Abstract

The following is a report of an unusual family cluster of group C invasive meningococcal disease in Tasmania. This unusual case cluster raises several important issues of public health significance regarding vaccine failure and nucleic acid amplification testing use in the setting of invasive meningococcal disease. *Commun Dis Intell* 2005;29:159–163.

Keywords: meningococcal; Neisseria meningitidis; *vaccine failure, nucleic acid amplification test*

Introduction

Although not common, invasive infection with *Neisseria meningitidis* can be devastating to affected patients and families, and, despite modern treatment, has a case-fatality risk of about nine per cent.[1]

In 2001/2002, there was an outbreak of group C meningococcal disease in Tasmania. In response to this outbreak, a subsidised state-wide polysaccharide ACW135Y vaccination campaign for persons aged 13–30 years was undertaken starting in mid-2002. This program finished in September 2004. The national conjugate group C program commenced in January 2003. At the time of this meningococcal cluster, both vaccination programs were in operation in Tasmania.

The following is a report of an unusual family cluster of group C invasive meningococcal disease.

Index case

In December 2003, a 3½-year-old male presented to the Department of Emergency Medicine (DEM) of a major private hospital in Hobart. He was vaccinated with Menjugate® vaccine eight months prior to presentation. His clinical history was of four days of a febrile illness with fevers to 40° C. Twenty-four hours prior to presentation he developed irritability, neck stiffness and non-blanching red/purple lesions on his lower limbs. When he presented to the DEM he had a temperature of 37.9° C, a purpuric rash on the lower limbs and nuchal rigidity. He was administered ceftriaxone 1-gram IV and benzylpenicillin 600 mg IV before transfer to the DEM of Royal Hobart Hospital, five minutes away by ambulance.

On presentation to the Royal Hobart Hospital he had a Glasgow Coma Score of 14, and a clinical presentation as above. He was given IV fluids and a further 400 mg of benzylpenicillin IV and transferred to the Intensive Care Unit. Investigations demonstrated a normal white cell count, a mild acidosis, and cloudy cerebrospinal fluid (CSF), with a positive latex agglutination for *Neisseria meningitidis.* CSF culture and throat swab were negative for *N. meningitidis* however blood cultures subsequently grew serogroup C *N. meningitidis* sensitive to ceftriaxone, penicillin and rifampicin.

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He continued to have high fevers for the next nine days, in spite of ceftriaxone one g bd IV and benzylpenicillin 900 mg IV every four hours. His temperature eventually settled and he was discharged without sequelae.

His immunisation status was confirmed with his local doctor and appropriate administration and cold chain procedures at the practice were confirmed. The patient subsequently underwent tests for immunological deficiency (including immunoglobulins and complement). These were normal. While the general tests for immunoglobulins were normal, the *Haemophilus influenza* type b IgG (Hib) serology test undertaken during clinical convalescence was less than 0.1 ug/ml, indicating suboptimal short term and long term protection.

This was despite the fact that he had on written evidence three Hib vaccines at two, five and eight months and a booster at 16 months of age.

The patient's three siblings, a 9-year-old male, a 7-year-old male and a 16-month-old female, two parents and two grandparents, were identified as contacts. The 16-month-old had been vaccinated against group C meningococcal disease four months previously. All received chemoprophylaxis with appropriate doses of rifampicin except for the 16-month-old sibling who received 250 mg of ceftriaxone IM. Chemoprophylaxis for the contacts was commenced the day of admission of the index case.

Case 2

Four days after presentation of the index case, the patient's 7-year-old sibling presented with a history of 12 hours of mild respiratory illness, with a 'croupy' cough, low grade fevers, nausea and a fine blanching macular rash spread over most of the back. The patient had completed a 2-day course of rifampicin for chemoprophylaxis two days earlier. Within 12 hours the sibling had developed neck stiffness and mild photophobia. He was assessed in the DEM of the Royal Hobart Hospital, where he was found to have a fever of 39.9 degrees Celsius, a tachycardia of 130, mild nuchal rigidity and mild photophobia. Blood was taken but a lumbar puncture was not performed. The patient was admitted to the paediatrics ward with a provisional diagnosis of upper respiratory tract illness, but a differential diagnosis of meningococcal disease was suggested. The patient was treated with ceftriaxone one gram IV daily and benzylpenicillin 600 mg bd. He had an elevated white cell count with a neutrophilia. His fever settled within 24 hours of antibiotic treatment. His blood cultures were negative but an in-house nucleic acid antigen test (NAAT) was positive for *N. meningitidis.* This child had not been immunised against meningococcal disease.

Case 3

Six days after presentation of the index case, the child's 9-year-old sibling presented with a febrile illness. This child had completed an appropriate 2-day course of rifampicin chemoprophylaxis for meningococcal disease four days earlier. The patient presented to the DEM of the Royal Hobart Hospital with a 24-hour history of fevers, neck stiffness, sore throat, dry cough and non-specific abdominal pain. No photophobia or rash was noted. Physical examination did not demonstrate rash, photophobia or nuchal rigidity. A provisional diagnosis of upper respiratory tract illness was made, however, in view of his contact with a known case of meningococcal disease, blood cultures and NAAT for *Neisseria meningitidis* were requested and a dose of ceftriaxone 1.5 grams IV and benzylpenicillin 1.8 grams IV were given. The patient was discharged home. Blood cultures were negative however the NAAT was positive for *N. meningitidis.* The patient was recalled four days later and received daily ceftriaxone 1.5 grams IV as an outpatient for five days.

At this point the parents and grandparents of the index case were given ciprofloxacin 500 mg as a single dose as further chemoprophylaxis. The immunised 16-month-old infant did not receive further chemoprophylaxis.

Further events

By this stage, the parents of the children were extremely concerned about the safety of the 16-month-old infant and themselves. They requested a blood test be performed on the remaining family members to see if they had evidence of meningococcal infection. The paediatrician who was caring for the children acquiesced.

NAAT performed on blood taken from the father was positive for *N. meningitidis.* The Public Health Unit was contacted to report the father as a case. The father was interviewed and was found to be clinically well. He reported no fever, rash, photophobia or neck stiffness. In view of this he was treated as a suspected, rather than confirmed, case of invasive meningococcal disease. He was referred to the Infectious Diseases Physician at the Royal Hobart Hospital who performed an assessment and found no clinical evidence of invasive meningococcal disease. In view of the positive NAAT result, and family cluster of invasive disease, he was commenced on daily ceftriaxone IM for three days.

The results of the molecular typing performed on the samples confirmed that the molecular type of all subjects was C: 14D4a:P1.5–2, 10–1. Typing was performed by the Melbourne Microbiological Diagnostic Unit.

Discussion

This unusual case cluster raises several important issues of public health significance regarding vaccine failure and NAAT use in the setting of invasive meningococcal disease.

Vaccine failure

This is the first report of a meningococcal serogroup C conjugate vaccine failure in Tasmania. Meningococcal group C vaccine is effective and vaccine failure is rare. Data from the United Kingdom (UK) confirm this and demonstrate that the impact of the introduction of meningococcal serogroup C conjugate vaccines in the UK has been extremely favourable where vaccine coverage has exceeded 80 per cent in all age groups targeted and up to the end of 2001, only 25 confirmed and one probable vaccine failure had been observed.[2]

In the UK, the definition of a vaccine failure to meningococcal disease is as follows:

- True vaccine failure – invasive meningococcal serogroup C disease meeting the case definition for definite serogroup C infection with onset more than 10 days after the last dose of vaccine scheduled for that age group.
- Probable vaccine failure – failure (i.e. probable serogroup C disease meeting the above vaccination criteria) where a person develops invasive serogroup C disease within 10 days of the last dose or before the last scheduled dose.[3]

Risk factors for vaccine failure are not clearly defined but include prematurity and low birth weight, a chromosomal abnormality or other genetic disorder, malignancy, any other underlying medical condition, known IgG deficiency or other immunological abnormality, hyposplenism and ethnic subgroups.[3,4] Subjects are classified as true or probable vaccine failures according to the above case definitions, regardless of the presence of risk factors. According to this definition, the index case in this case series constitutes a true vaccine failure. None of the above risk factors were identified as leading to his vaccine failure.

The phenotype of the organism is another factor that may influence vaccine efficacy. Meningococci have a number of surface antigens. The organism is classified into serogroups, types and subtypes based on the configuration of the surface antigens:

- serogroups – based on variants in the capsular polysaccharide;
- serotypes – based on the *PorB* outer membrane protein variants;
- serosubtypes – based on the *PorA* outer membrane protein variants.[5]

Conjugate vaccine works by priming the immune system to respond to capsular polysaccharide, not the cell wall antigens.[6] Newer vaccines in development are directed against *PorA* regions (in particular, vaccine against 'B' meningococcus), however, this is not relevant to this case.[7,8] The conjugate vaccine given to the index case ordinarily would have provided protection against the organism as the organism was a definite C capsular subgroup.

The index case was tested for complement and immunoglobulin levels, which were normal. The negative Hib serology results may indicate the presence of an IgG subclass deficiency, a possible explanation of this clinical event. However, the patient was not tested for IgG subclass antibodies. In future, if a case of meningococcal vaccine failure is reported, testing for IgG subclass antibodies is recommended.[3,9]

High attack rate

The attack rate for meningococcal disease among untreated household contacts varies between 4.2 and 27.7 per 1,000 subjects.[10,11,12] Chemoprophylaxis reduces the risk of subsequent cases by 89 per cent.[13] In this family, three out of seven household contacts were NAAT positive for testing for the invasive meningococcal strain. Two of the subjects had received rifampicin chemoprophylaxis prior to testing and the third had received both rifampicin and ciprofloxacin prior to testing. Rifampicin resistance has not been reported in Tasmania. In this case, the invasive *Neisseria* strain in the index case was proven sensitive to rifampicin. Rifampicin eliminates, in most instances, the nasopharyngeal carriage of *N. meningitidis* but it is recognised that it may not abort invasive disease if already incubating.[14]

The father of the index case was asymptomatic but NAAT positive. He may have been a case of nasopharyngeal carriage with transient bacteraemia rather than invasive disease. The population rate of nasopharyngeal colonisation with meningococci varies between 10 and 30 per cent.[15,16] Rates of nasopharyngeal colonisation with invasive strains of *N. meningitidis* are much lower than this.[17,18] There are little data on the use of NAAT in the screening of asymptomatic contacts of cases. As this case series illustrates, a positive NAAT result in the absence of clinical symptoms and a negative culture presents a clinical dilemma. A positive result in this setting may represent transient bacteraemia or could be the beginning of invasive meningococcal disease. Due to the precipitous nature of the illness, the clinician in this case was obligated to treat the patient as if he had incipient invasive disease even though it was more likely he did not. It may well be the case that transient and spontaneously resolving bacteraemia is not uncommon amongst this group.

Nucleic acid amplification testing issues

In this case cluster, three out of four subjects had a positive NAAT assay result in the absence of positive blood culture result (throat swabs were not collected). This phenomenon is well recognised. Antibiotic treatment prior to transport or admission to hospital has reduced the proportion of cases of invasive meningococcal disease from which *Neisseria meningitidis* can be isolated by standard microbiological techniques.[19] Identification of meningococci by NAAT is now a common method for detection of evidence of invasive meningococcal disease. The literature reports the sensitivity of the NAAT assay for culture-confirmed cases is between 91 and 98.5 per cent. The specificity of the test is between 76 and 96 per cent based on test results for patients from whom other bacteria were isolated, children with viral meningitis and afebrile negative controls.[20,21]

The NAAT in use at the Royal Hobart Hospital amplifies a region of the *N. meningitidis* insertion sequence, IS *1106*. This is an in-house assay that was adapted from that of Newcombe.[22] The literature reports the sensitivity and specificity of this NAAT assay as ranging from 83–100 per cent and 87–100 per cent respectively. This NAAT has been extensively validated in-house with local data suggesting sensitivity and specificity of 92 per cent and 94 per cent respectively. In addition, all NAAT positive samples were tested using a second genomic target (*PorA*) as part of the typing protocol. All three samples were positive in both NAAT assays. The three NAAT positive/culture negative cases reported here are therefore likely to be genuine.

This is supported in the literature. A study examining the meaning of a positive NAAT for *Neisseria meningitidis* in the presence of a negative culture found that NAAT improves diagnosis. In the study, 35 of 39 patients suspected to have meningococcal meningitis were microbiologically confirmed. Of these, 22 were culture and NAAT positive, three were microscopically and NAAT positive, one was only microscopically positive, and nine were positive only by NAAT. By using NAAT methodology, the number of confirmed diagnoses of meningococcal meningitis increased by 23 per cent compared with those obtained by microscopic observation and culture.[23]

Although the literature demonstrates that NAAT has certainly improved case ascertainment, especially where culture negative or post antibiotics, the evidence is limited to cases who actually had some sort of febrile illness which warranted the test in the first place. Studies of the sensitivity and specificity of NAAT in well subjects are lacking.

Conclusions

Invasive meningococcal disease continues to be an illness of considerable public health importance. According to the UK definition of vaccine failure, this constitutes a true vaccine failure. There was no evidence of hereditary immune deficiency in this case, however, if future cases occur, it would be prudent to perform IgG subclass antibodies as this IgG subclass deficiency could be responsible for failure to mount an immune response to the meningococcal conjugate vaccine. Group C conjugate vaccine failure is rare and efficacy of the vaccine is good.

The use of NAAT has improved diagnosis of invasive disease. Should the father in this case cluster have been tested? There is no easy answer to this. A disease such as invasive meningococcal disease does not always afford the clinician the luxury of time. By the time the signs and symptoms of disease have developed, the pathological processes that lead to death or disability may be well established. The advent of the NAAT test has altered the consequences of precautionary testing. Testing within the broader context in which the illness occurs is an issue still to be resolved, but would appear to remain generally appropriate that such tests only be performed when clinical symptoms warrant it.

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Reported foodborne illness and gastroenteritis in Australia: Annual report of the OzFoodNet network, 2004

The OzFoodNet Working Group

Abstract

In 2004, OzFoodNet sites recorded 24,313 notifications of eight potentially foodborne diseases, along with 118 outbreaks of foodborne disease. Overall, reports of both notifications and outbreaks were higher than previous years. The most common sporadic diseases were campylobacteriosis (15,640 cases) and salmonellosis (7,842 cases). Reports of sporadic cases of Shiga toxin-producing *Escherichia coli* were rare with only 46 cases, but there were two small clusters due to serotypes O157/O111 and O86. The 118 foodborne disease outbreaks affected 2,076 persons, of whom 5.6 per cent (116/2,076) were hospitalised and two people died. Foods prepared in restaurants and catering settings caused the most outbreaks and the most common agent was *Salmonella* Typhimurium. Outbreak investigations during 2004 implicated chicken, foods containing eggs, imported oysters and food handlers infected with norovirus. In addition to foodborne outbreaks, OzFoodNet sites reported 874 outbreaks that were spread from person-to-person affecting 25,363 people. Sites conducted 54 investigations into clusters of *Salmonella* and other pathogens where a source could not be identified. Surveillance of foodborne diseases continued to improve during 2004, with all jurisdictions contributing to national cluster reports and using analytical studies to investigate outbreaks. Ninety-eight per cent of *Salmonella* notifications on state and territory surveillance databases recorded complete information about serotype and phage type. Foodborne disease may cost Australia as much as $1.2 billion annually making it vital to intervene to prevent disease. *Commun Dis Intell* 2005;29:164–191.

Keywords: Campylobacter, *disease outbreak; disease surveillance; Enteritidis; foodborne disease;* Listeria; *norovirus;* Salmonella; Shigella; *typhoid;* Yersinia

Introduction

Foodborne disease is a major cause of morbidity and an occasional cause of mortality throughout the world.[1,2] Each year there are approximately 5.4 million cases (95% Credible Interval 4–6.9 million cases) of foodborne gastroenteritis in Australia, resulting in 80 deaths annually.[3] Approximately 32 per cent (95% Credible Interval 28%–38%) of all gastroenteritis in Australia is estimated to be foodborne.

Many countries conduct surveillance of potentially foodborne diseases to protect consumers and maintain a safe food supply.[4,5] The major aim of surveillance of diseases that arise from contaminated food is to detect outbreaks. Investigation of foodborne outbreaks have many clear public health benefits, as early intervention can remove contaminated product from the marketplace and contribute to policies to prevent further disease.[6] The public health benefits of surveillance and early outbreak detection have clear economic benefits.[7]

In addition, countries are increasingly using data from surveillance systems to support and monitor interventions in the food supply chain. One example is the use of the United States of America Centers for Disease Control and Prevention's FoodNet data to establish whether national disease targets set under the 'Healthy People 2010' initiative have been met (www.healthypeople.gov). FoodNet has been able to track the incidence of laboratory-diagnosed

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cases of foodborne disease that are not notifiable to State health departments.[8] Recent results from FoodNet indicate sustained declines in the incidence of several diseases, including salmonellosis and campylobacteriosis.[9] This is supported by findings in the United States of America meat industry that have recorded declining isolation of *Salmonella* and *Campylobacter* in meat processing plants.[10]

In 2000, the Australian Government Department of Health and Ageing (DoHA) established the OzFoodNet network to enhance surveillance for foodborne disease.[11] This built upon an 18-month trial of active surveillance in the Newcastle region of New South Wales that was modelled on the FoodNet surveillance system. The OzFoodNet network consists of epidemiologists specifically employed by each state and territory health department to conduct investigations and applied research into foodborne disease. The Network involves many different organisations, including the National Centre for Epidemiology and Population Health, and the Public Health Laboratory Network. OzFoodNet is a member of the Communicable Diseases Network Australia, which is Australia's peak body for communicable disease control.[12] The Australian Government Department of Health and Ageing funds OzFoodNet and convenes a committee to manage the Network, and a committee to review the scientific basis for various research projects.

This is the fourth annual report of OzFoodNet and covers data and activities for 2004.

Methods

Population under surveillance

In 2004, the coverage of OzFoodNet included all states and territories. The entire Australian population was estimated to be 20,111,297 persons in June 2004.[13] In addition, the Hunter Area Health Service had a separate OzFoodNet Site complementing foodborne disease surveillance across New South Wales. The Hunter site conducts thorough local investigation and provides a baseline for foodborne disease incidence in New South Wales. In June 2004, the population covered by the Hunter site was estimated to be 549,846 persons.

Data sources

Rates of notified infections

All Australian states and territories require doctors and/or pathology laboratories to notify patients with infectious diseases that are important to public health. Western Australia is the only jurisdiction where laboratory notification is not mandatory under legislation, although most laboratories still notify the health department by agreement. OzFoodNet aggregated and analysed data on patients notified with the following diseases or conditions, a proportion of which may be acquired from food:

- *Campylobacter* infections;
- *Salmonella* infections, including *Salmonella* Paratyphi A, B and C;
- *Listeria* infections;
- *Yersinia* infections;
- Shiga toxin-producing *Escherichia coli* infections and haemolytic uraemic syndrome;
- typhoid; and
- *Shigella* infections.

To compare disease to historical totals, OzFoodNet compared crude numbers and rates of notification to the mean of the previous six years. Where relevant, numbers and rates of notifications for specific sub-types of infecting organisms were compared to notifications for the previous year.

To calculate rates of notification the estimated resident populations for each jurisdiction for June 2004, or the specified year, were used.[13] Age specific rates for notified infections in each jurisdiction were calculated.

The date that notifications were received was used throughout this report to analyse notification data. These data are similar to those reported to the National Notifiable Diseases Surveillance System, but individual totals may vary with time and due to different approaches to analysis.

Gastrointestinal and foodborne disease outbreaks

OzFoodNet collected information on outbreaks of gastrointestinal disease, including foodborne illness, that occurred in Australia during 2004. An outbreak of foodborne disease was defined as an increase in the number of reports of a particular infection or illness associated with a common food or meal. The reports collate summary information about the setting where the outbreak occurred, where food was prepared, the month the outbreak occurred, the aetiological agent, the number of persons affected, the type of investigation conducted, the level of evidence obtained and the food vehicle responsible. To summarise the data, OzFoodNet categorised the outbreaks by aetiological agents, food vehicles and settings where the outbreak occurred. Data on outbreaks transmitted from infected persons, water, animals and cluster investigations were also summarised. The number of outbreaks and documented causes may vary from summaries published by different jurisdictions.

Risk factors for infection

To identify risk factors for foodborne infection in Australia, OzFoodNet reviewed summary data from outbreaks that occurred in 2004 and compared them to previous years. Data from several complementary OzFoodNet studies of foodborne illness in Australia were also examined.

Surveillance evaluation and enhancement

To identify areas where improvements to surveillance are critical, OzFoodNet compared the results of surveillance across different sites, including rates of reporting outbreaks, and investigation of clusters of *Salmonella*. To measure how well jurisdictions conducted surveillance for *Salmonella,* OzFoodNet examined the completeness of information contained on state and territory databases in 2004. The proportion of notifications with serotype and phage type information was compared with results for the previous three years.

Results

Rates of notified infections

In 2004, OzFoodNet sites reported 24,313 notifications of eight potentially foodborne diseases. This was a 9.0 per cent increase from the mean of 22,289 notifications for the previous six years. Reports for these eight diseases make up almost a quarter of notifications to the National Notifiable Diseases Surveillance System.[14] A summary of the number and rates of notifications by OzFoodNet sites is shown in Appendix 1.

Salmonella infections

In 2004, OzFoodNet sites reported 7,842 cases of *Salmonella* infection, which equated to a rate of 39.0 cases per 100,000 population. This rate of notifications represented an increase of 4.9 per cent over the mean rate for the previous six years (Figure 1). The rate of *Salmonella* notification in OzFoodNet sites ranged from 22.9 and 23.2 cases per 100,000 population in the Hunter and Victoria, respectively, to 195.1 cases per 100,000 population in the Northern Territory (Figure 2).

The notification rates of salmonellosis remained relatively constant over the last seven years. Overall, notification rates of salmonellosis for 2004 were increased in New South Wales (20.3%), the Australian Capital Territory (18.0%) and Queensland (9.9%) compared to historical means. Western Australia (-21.5%), Tasmania (-17.2%) and South Australia (-11.2%) recorded declines in the notification rate of *Salmonella*, with other jurisdictions recording similar rates to previous years (Figure 2).

OzFoodNet sites reported that the ratio of males to females was approximately 1:1.1. The highest age-specific rate of *Salmonella* infection was 221 cases per 100,000 population in males aged 0–4 years and 204 in females aged 0–4 years. Notification rates were also elevated in the 5–9 year age group with a secondary peak in the 20–29 year age range for males and females.

Rates of salmonellosis were highest in northern areas of Australia. The highest rate in Australia is consistently reported in the Kimberley region of Western Australia.[14] In the Northern Territory, the rate of *Salmonella* notifications was 283 per 100,000 population in Indigenous people compared to 137 per 100,000 population in non-Indigenous people.

During 2004, there were 629 notifications of *Salmonella* Typhimurium 170/108 and 585 notifications of *S.* Typhimurium 135 (including 135a) to OzFoodNet sites making these the most common *Salmonella* infections (Table 1). *S.* Typhimurium 170

Figure 1. Notifications and annual rates of *Salmonella* infections, Australia, 1998 to 2004

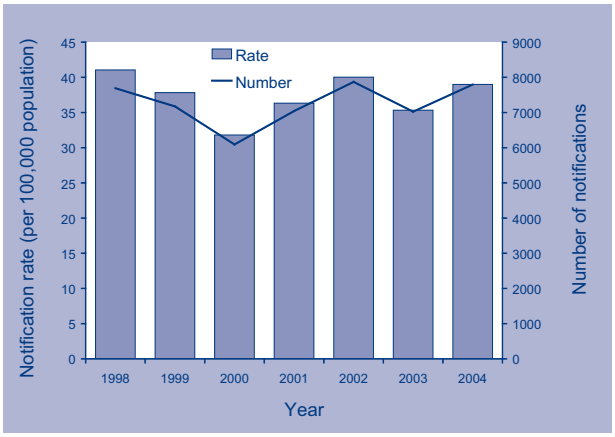
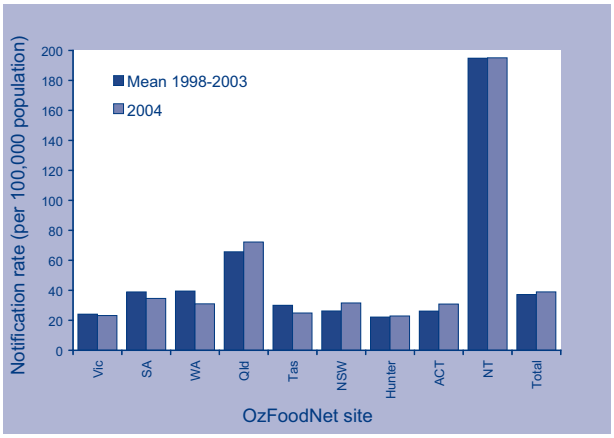


Figure 2. Notification rates of *Salmonella* infections for 2004 compared to mean rates for 1998 to 2003, by OzFoodNet site



and *S.* Typhimurium 108 are the same phage type and continued to emerge as a significant phage type around Australia, accounting for 10 outbreaks of foodborne disease during 2004. *S.* Typhimurium 197 emerged as a cause of significant disease, particularly in Queensland, where 56 per cent (141/251) of notifications of this phage type were reported. There were 383 cases of *S.* Saintpaul, making it the most common *Salmonella* serovar following *S.* Typhimurium.

Certain *Salmonella* serovars traditionally occupy localised niches in specific geographical areas in Australia. During 2004, *Salmonella* Birkenhead infections were the fourth most common serovar in New South Wales and the fifth most common in Queensland. The total number of cases in these two States (244 cases) was 38 per cent higher in 2004 compared to 2003. This elevated notification rate reflects an endemic focus of *S.* Birkenhead in northern New South Wales and south-eastern Queensland. In Tasmania, 53 per cent of reported cases of salmonellosis were *S.* Mississippi infections, equating to a rate of 13.2 cases per 100,000 population. The highest serovar specific rate in Australia was *S.* Ball in the Northern Territory, with a rate of 25.2 cases per 100,000 population. Reported rates of *S.* Saintpaul in the Northern Territory were 23.2 cases per 100,000 population.

Salmonella *Enteritidis*

S. Enteritidis is a serotype that can infect the internal contents of eggs via the oviducts of infected chickens, predominantly with one strain: *S.* Enteritidis phage type 4. People may become infected with this serotype after eating undercooked eggs. This phage type has caused major problems in the Northern Hemisphere where it has become established in commercial egg laying flocks. Australia is largely free of *S.* Enteritidis phage type 4 except in people infected overseas. OzFoodNet is currently investigating other phage types of *S.* Enteritidis that are acquired locally in Australia to determine risk factors for infection.

Table 1. Numbers, rates and proportions of the top 10 *Salmonella* infections, 2003 to 2004, by OzFoodNet site*

OzFoodNet site	*Salmonella* type (sero/phage type)	2004			2003		
		n	Rate†	Proportion (%)‡	n	Rate	Ratio§
Australian Capital Territory	Typhimurium 170	31	9.6	31.3	4	1.2	7.8
	Typhimurium 197	7	2.2	7.1	0	0.0	-
	Infantis	6	1.9	6.1	3	0.9	2.0
	Typhimurium 9	6	1.9	6.1	4	1.2	1.5
	Typhimurium 135	5	1.5	5.1	25	7.7	0.2
	Typhimurium 12	4	1.2	4.0	0	0.0	-
	Virchow 8	4	1.2	4.0	0	0.0	-
	Agona	3	0.9	3.0	0	0.0	-
	Chester	2	0.6	2.0	1	0.3	2.0
	Mbandaka	2	0.6	2.0	1	0.3	2.0
	Newport	2	0.6	2.0	0	0.0	-
	Stanley	2	0.6	2.0	0	0.0	-
	Subsp I ser 16:1,v:-	2	0.6	2.0	1	0.3	2.0
	Typhimurium 104L	2	0.6	2.0	2	0.6	1.0
	Typhimurium 12a	2	0.6	2.0	0	0.0	-
	Typhimurium U290	2	0.6	2.0	4	1.2	0.5
Hunter	Typhimurium 170	16	2.9	12.7	10	1.8	1.6
	Typhimurium 135	10	1.8	7.9	1	0.2	10.0
	Typhimurium 12	10	1.8	7.9	0	0.0	-
	Typhimurium 4	8	1.5	6.3	9	1.6	0.9
	Typhimurium 9	5	0.9	4.0	7	1.3	0.7
	Typhimurium U290	5	0.9	4.0	8	1.5	0.6
	Chester	5	0.9	4.0	3	0.5	1.7
	Potsdam	4	0.7	3.2	1	0.2	4.0
	Saintpaul	4	0.7	3.2	2	0.4	2.0
	Birkenhead	4	0.7	3.2	3	0.5	1.3

Table 1. Numbers, rates and proportions of the top 10 *Salmonella* infections, 2003 to 2004, by OzFoodNet site,* *continued*

OzFoodNet site	*Salmonella* type (sero/phage type)	2004			2003		
		n	Rate†	Proportion (%)‡	n	Rate	Ratio§
New South Wales	Typhimurium 170	333	4.9	15.7	232	3.5	1.4
	Typhimurium 12	170	2.5	8.0	38	0.6	4.5
	Typhimurium 135	140	2.1	6.6	135	2.0	1.0
	Typhimurium 9	108	1.6	5.1	131	2.0	0.8
	Birkenhead	77	1.1	3.6	68	1.0	1.1
	Typhimurium 4	66	1.0	3.1	34	0.5	1.9
	Infantis	53	0.8	2.5	86	1.3	0.6
	Typhimurium u290	46	0.7	2.2	30	0.4	1.5
	Typhimurium 197	43	0.6	2.0	66	1.0	0.7
	Virchow 8	40	0.6	1.9	27	0.4	1.5
Northern Territory	Ball	50	25.0	13.1	44	22.2	1.1
	Saintpaul	47	23.5	12.3	28	14.1	1.7
	Litchfield	15	7.5	3.9	9	4.5	1.7
	Muenchen	14	7.0	3.7	14	7.1	1.0
	Havana	13	6.5	3.4	11	5.5	1.2
	Anatum	12	6.0	3.1	22	11.1	0.5
	Chester	12	6.0	3.1	16	8.1	0.8
	Senftenberg	8	4.0	2.1	7	3.5	1.1
	Wandsworth	8	4.0	2.1	3	1.5	2.7
	Weltevreden	8	4.0	2.1	10	5.0	0.8
Queensland	Virchow 8	241	6.2	8.6	165	4.3	1.5
	Saintpaul	223	5.7	8.0	167	4.4	1.3
	Typhimurium 135	176	4.5	6.3	155	4.1	1.1
	Birkenhead+	163	4.2	5.8	109	2.9	1.5
	Typhimurium 197	141	3.6	5.0	90	2.4	1.6
	Aberdeen	114	2.9	4.1	75	2.0	1.5
	Hvittingfoss	110	2.8	3.9	72	1.9	1.5
	Waycross	94	2.4	3.4	50	1.3	1.9
	Chester	84	2.2	3.0	98	2.6	0.9
	Typhimurium 12a	53	1.4	1.9	1	0.0	53.0
South Australia	Typhimurium 108	70	4.6	13.4	32	2.1	2.2
	Typhimurium 9	46	3.0	8.8	28	1.8	1.6
	Typhimurium 135a	25	1.6	4.8	18	1.2	1.4
	Chester	21	1.4	4.0	24	1.6	0.9
	Typhimurium RDNC	20	1.3	3.8	12	0.8	1.7
	Typhimurium 135	19	1.2	3.6	17	1.1	1.1
	Infantis	17	1.1	3.2	20	1.3	0.9
	Singapore	17	1.1	3.2	9	0.6	1.9
	Typhimurium 8	17	1.1	3.2	2	0.1	8.5
	Typhimurium 126 var	17	1.1	3.2	0	0.0	-

Table 1. Numbers, rates and proportions of the top 10 *Salmonella* infections, 2003 to 2004, by OzFoodNet site,* *continued*

OzFoodNet site	*Salmonella* type (sero/phage type)	2004			2003		
		n	Rate†	Proportion (%)‡	n	Rate	Ratio§
Tasmania	Mississippi	63	13.1	52.5	70	14.7	0.9
	Typhimurium 9	4	0.8	3.3	7	1.5	0.6
	Typhimurium 12a	4	0.8	3.3	3	0.6	1.3
	Typhimurium 170	3	0.6	2.5	5	1.0	0.6
	Enteritidis 4b	2	0.4	1.7	0	0.0	-
	Enteritidis 6a	2	0.4	1.7	1	0.2	2.0
	Newport	2	0.4	1.7	2	0.4	1.0
	Paratyphi B bv Java Dundee	2	0.4	1.7	1	0.2	2.0
	Potsdam	2	0.4	1.7	0	0.0	-
	Saintpaul	2	0.4	1.7	5	1.0	0.4
	Typhimurium 135	2	0.4	1.7	6	1.3	0.3
	Typhimurium 141	2	0.4	1.7	0	0.0	-
	Typhimurium RDNC	2	0.4	1.7	0	0.0	-
	Virchow 6	2	0.4	1.7	0	0.0	-
Victoria	Typhimurium 9	145	2.9	12.8	159	3.2	0.9
	Typhimurium 170	137	2.8	12.1	125	2.5	1.1
	Typhimurium 135	88	1.8	7.8	233	4.7	0.4
	Typhimurium 197	59	1.2	5.2	21	0.4	2.8
	Infantis	43	0.9	3.8	54	1.1	0.8
	Typhimurium u290	36	0.7	3.2	88	1.8	0.4
	Typhimurium 126	28	0.6	2.5	18	0.4	1.6
	Virchow 8	26	0.5	2.3	9	0.2	2.9
	Typhimurium 12	23	0.5	2.0	19	0.4	1.2
	Stanley	21	0.4	1.9	19	0.4	1.1
	Typhimurium RDNC	21	0.4	1.9	11	0.2	1.9
Western Australia	Saintpaul	46	2.3	7.5	29	1.5	1.6
	Typhimurium 135a	45	2.3	7.3	42	2.2	1.1
	Typhimurium 135	29	1.5	4.7	30	1.5	1.0
	Chester	24	1.2	3.9	36	1.8	0.7
	Muenchen	23	1.2	3.7	28	1.4	0.8
	Enteritidis 6a	21	1.1	3.4	8	0.4	2.6
	Stanley	14	0.7	2.3	7	0.4	2.0
	Havana	13	0.7	2.1	10	0.5	1.3
	Senftenberg	13	0.7	2.1	15	0.8	0.9
	Typhimurium 9	13	0.7	2.1	20	1.0	0.7

* Where there were multiple tenth ranking *Salmonella* types all data have been shown, giving more than 10 categories for some sites.

† Rate per 100,000 population.

‡ Proportion of total *Salmonella* notified for this jurisdiction in 2004.

§ Ratio of the number of reported cases in 2004 compared to the number reported in 2003.

RDNC = 'Reactive but Does Not Conform' and represents phage type patterns that are not yet assigned.

During 2004, OzFoodNet sites reported 422 cases of *Salmonella* Enteritidis, which was higher than for 2003. The majority of cases were related to travel overseas (69%) or were of unknown travel history (18%) (Table 2). Relevant travel histories are difficult to obtain, as people have often travelled to several countries before visiting Australia. Asian countries were commonly mentioned, which reflects the frequency of Asia as a travel destination for Australians (Table 3). In the Asian region, cases of *S.* Enteritidis infection most commonly reported travelling to Bali (38%). Fifteen per cent of people acquiring their infection overseas reported travelling to Europe.

Table 2. Numbers of *Salmonella* Enteritidis infections acquired overseas and in Australia in 2004, by OzFoodNet site

OzFoodNet site	Overseas travel			Total
	Yes	No	Unknown	
Australian Capital Territory	2	0	0	2
New South Wales	43	4	50	97
Northern Territory	1	2	2	5
Queensland	51	40	21	112
South Australia	27	5	0	32
Tasmania	8	1	0	9
Victoria	84	2	0	86
Western Australia	77	0	2	79
Total	293	54	75	422

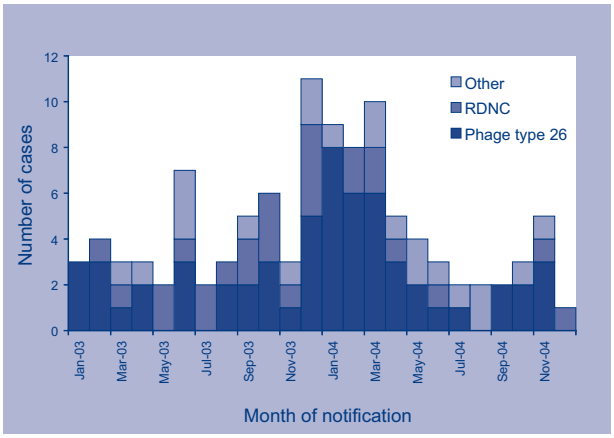
The most common phage types depended on the region that the person travelled to. For people travelling to Bali and Indonesia, the most common phage types were 6a, 5a, 4, and 4b. In Malaysia and Singapore the most common infecting phage types were 1 and 6a, with no phage type 4 reported. Thailand travellers were infected with the same phage types as Malaysian travellers, along with phage type 4. For travellers returning from Europe, phage types 1, 4, 5a and 6 were most common.

Overall, 13 per cent (54/422) of patients infected with *S.* Enteritidis acquired their infection in Australia (Figure 3). The median age of cases was 31 years (range 0–85 years) and the male to female ratio was 1.2:1. Thirty-six per cent (40/112) of *S.* Enteritidis infections in Queensland were locally acquired compared to Victoria where only two per cent (2/86) of infections were locally acquired. The majority of locally acquired infections in Queensland were due to phage type 26. There was a temporal clustering of cases of *S.* Enteritidis in December 2003—March 2004, although no common sources were identified. There were no locally acquired cases of *S.* Enteritidis in the Australian Capital Territory or Western Australia.

Salmonella *clustering*

In total, health departments conducted 81 investigations into clusters and point source outbreaks of salmonellosis during 2004. A source of infection was identified for 44 per cent (36/81) of these investigations.

Figure 3. Locally-acquired *Salmonella* Enteritidis infections, 2003 to 2004, by major phage type and month of notification



RDNC = 'Reactive but Does Not Conform' and represents phage type patterns that are not yet assigned.

Table 3. Numbers of *Salmonella* Enteritidis infections acquired overseas, 2004, by region of travel

Region of travel	Number of cases	Percentage
Africa	5	1.7
America	1	0.3
America/Europe	1	0.3
Asia/Americas	1	0.3
Asia/Europe	15	5.1
Asia-other	6	2.1
Bali	112	38.2
China	9	3.1
Europe	29	9.9
Hong Kong	9	3.1
Indian subcontinent	8	2.7
Indonesia	16	5.5
Malaysia	19	6.5
Middle East	8	2.7
Mixed Asia	9	3.1
Pacific	4	1.4
Phillipines	8	2.7
Singapore	16	5.5
Solomons	1	0.3
Thailand	13	4.4
Unknown	3	1.0
Total	293	100

Campylobacter *infections*

Campylobacteriosis is not notifiable in New South Wales, including the Hunter Health Area, and data for 2004 were unavailable. With this exception, in 2004 OzFoodNet sites reported 15,640 cases of *Campylobacter* infection, which equated to a rate of 117 cases per 100,000 population.

This rate represented a 4.6 per cent increase over the mean rate for the previous six years and represents a sustained increase in notifications (Figure 4). Notifications peaked in November with 1,666 reports, which was the highest for a single month in the previous six years. Victoria and Tasmania reported marked increases in the number of cases of 29 per cent and 17 per cent respectively, while South Australia (-18%) and the Northern Territory (-6%) reported decreases (Figure 5). All other jurisdictions reported minimal change from historical totals.

Figure 4. Notifications and annual rates of *Campylobacter* infections, Australia excluding New South Wales, 1998 to 2004

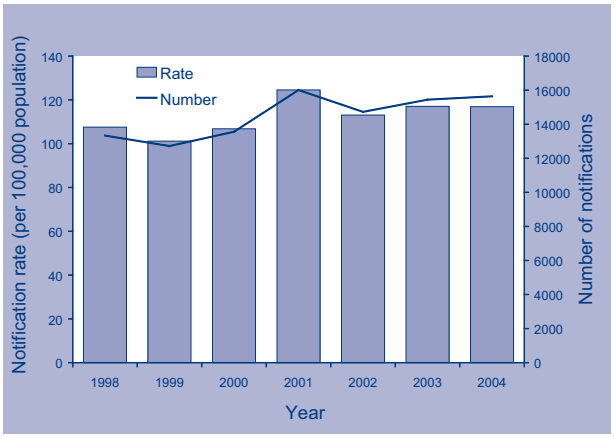
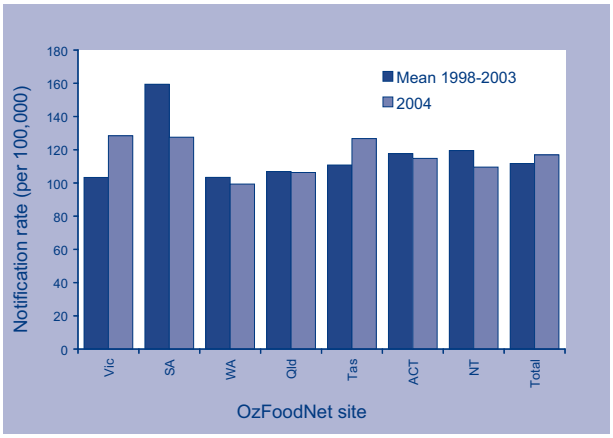


Figure 5. Notification rates of *Campylobacter* infections for 2004 compared to mean rates for 1998–2003, Australia excluding New South Wales, by OzFoodNet site



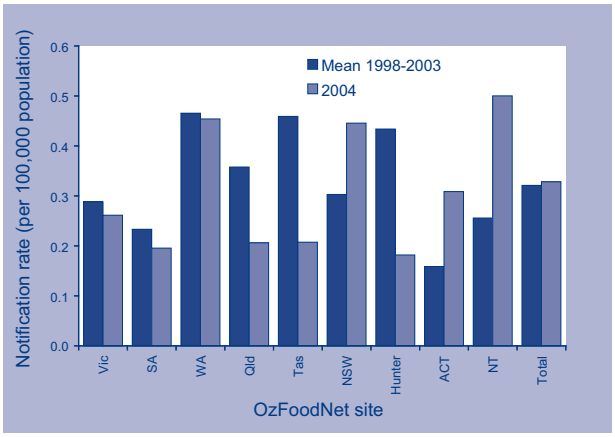
The overall ratio of infections in males and females was 1.2:1. The highest age specific rates were in children in the 0–4 year age group, with male and female children in this age group having rates of 261 and 188 per 100,000 population respectively. In the Northern Territory, the rate of campylobacteriosis in Indigenous peoples was 186 per 100,000 population compared to only 69 per 100,000 population in non-Indigenous persons.

During 2004, there were nine investigations of *Campylobacter* outbreaks affecting a total of 104 people. This was considerably higher than for 2003, where only one outbreak was investigated.

Listeria *infections*

OzFoodNet sites reported 66 cases of listeriosis in 2004, which represented a notification rate of 0.3 cases per 100,000 population (Figure 6). This was an increase of 6.5 per cent in the number of notifications compared to the historical mean. Western Australia and the Northern Territory both recorded 0.5 cases per 100,000 population. New South Wales recorded the greatest increase in notifications against historical averages with 30 notifications in 2004, which was 53 per cent higher than the six year mean. There was one small cluster of two cases in South Australia during 2004, although no source of infection was identified.

Figure 6. Notification rates of *Listeria* infections for 2004 compared to mean rates for 1998–2003, Australia, by OzFoodNet site

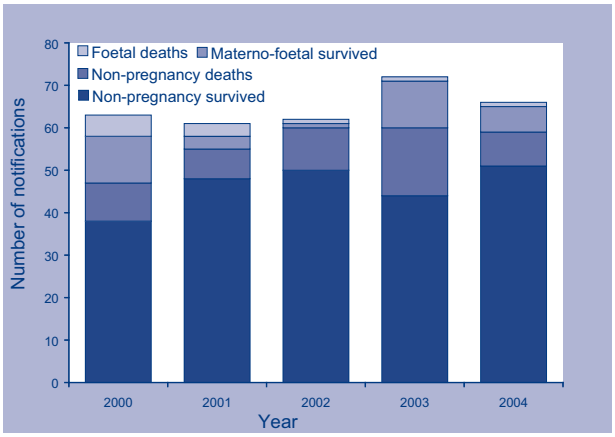


Eighty-nine per cent (59/66) of infections during 2004 were reported in persons who were either elderly and/or immunocompromised. Among cases of non-materno-foetal infections more males than females were notified, with the male to female ratio being 5.1:1. Sixty-one per cent (36/59) of cases were aged 65 years or greater. The highest age specific

rate of 5.3 cases per 100,000 population was in males over the age of 65 years. Fourteen per cent (8/59) of non-pregnancy associated cases died.

There were seven materno-foetal infections with one foetal death recorded. This equates to a rate of 2.7 cases of *Listeria* infections per 100,000 births.[15] A substantial decline in the number of materno-foetal infections occurred between 2000 and 2002, but numbers of infections rose again in 2003 and 2004 (Figure 7).

Figure 7. Notifications of *Listeria* showing non-pregnancy related infections and deaths and materno-foetal infections and deaths, Australia, 2000 to 2004

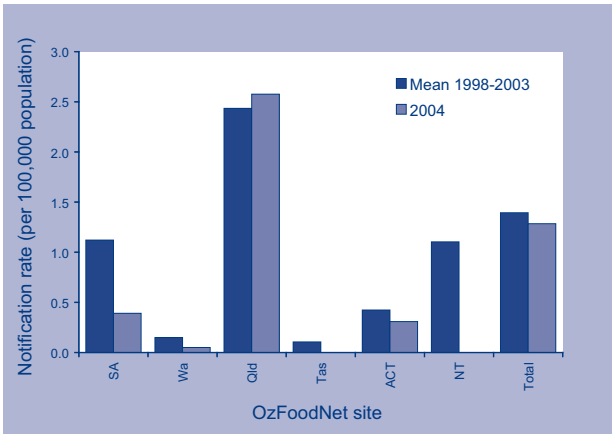


Yersinia *infections*

The Communicable Diseases Network Australia agreed to stop reporting notifications of *Yersinia* infections to the National Notifiable Diseases Surveillance System, as of January 2001. The main reason for this was the apparent decline in incidence and lack of identified outbreaks. In May 2001, the Victorian Government revised regulations governing reporting of infectious diseases, at which time they removed yersiniosis from the list of reportable conditions. *Yersinia* is also not notifiable in New South Wales. No other Australian jurisdictions have amended their legislation to remove yersiniosis from lists of reportable conditions.

In 2004, OzFoodNet sites reported 108 cases of yersiniosis, which equated to a rate of 1.3 notifications per 100,000 population (Figure 8). The overall rate declined 6.4 per cent from previous years, when adjusted for the absence of reporting from Victoria and New South Wales. Queensland reported 93 per cent (100/108) of all cases, which equated to a rate of 2.6 cases per 100,000 population. The rates of yersiniosis in Queensland decreased in the 1990s, but have steadily increased since 2002. In 2004 in Queensland, the median age of yersiniosis cases was 29 years (range <1–87 years) and notifications were similar in all three Queensland health zones. Biotype for *Yersinia enterocolitica* cases in Queensland was known for 96 cases, of which 48 per cent (46/96) were biotype 4 serotype O:3 and 25 per cent were biotype 1A serotype O:5. South Australia reported six cases of yersiniosis, while the Australian Capital Territory and Western Australia reported one case each.

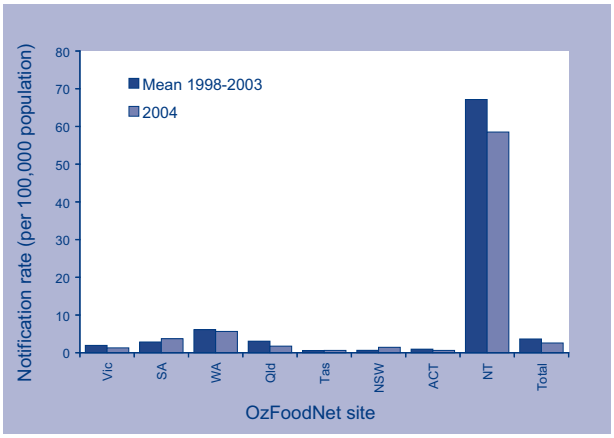
Figure 8. Notification rates of *Yersinia* infections for 2004 compared to mean rates for 1998–2003, Australia excluding Victoria and New South Wales, by OzFoodNet site



Shigella *infections*

OzFoodNet sites reported 520 cases of shigellosis during 2004, which equated to a notification rate of 2.6 cases per 100,000 population (Figure 9). This was a 29.1 per cent decrease in the notification rate compared with the six-year mean, after adjusting for the introduction of notifications from New South Wales in January 2001.

Figure 9. Notification rates of *Shigella* infections for 2004 compared to mean rates for 1998–2003, by OzFoodNet site



* Shigellosis became notifiable in New South Wales from 2001 onwards.

The highest rate of notification was in the Northern Territory (59 cases per 100,000 population), which was 20 times higher than the overall Australian rate. Within the Northern Territory, shigellosis was most commonly reported in the drier central regions and the rate in Alice Springs was 196 cases per 100,000 population. Eighty-one per cent (95/117) of notifications in the Northern Territory were in persons of Aboriginal or Torres Strait Island origin, which equated to a rate of 167 cases per 100,000 population. Only South Australia and Tasmania observed an increased rate compared to the six-year mean.

The male to female ratio of shigellosis cases was 0.8:1. The highest age specific rates were in males (11.6 cases per 100,000 population) and females (14.4 cases per 100,000 population) in the 0–4 year age group, with a secondary smaller peak in the 25–29 year age group for females. There were three reported outbreaks of shigellosis all of which were suspected to be spread from person-to-person. There were two outbreaks of *Shigella flexneri* 2a in the Northern Territory and one outbreak of *Shigella sonnei* biotype g in South Australia. In Australia, the majority of *Shigella* infections are thought to be due to person-to-person transmission, or are acquired overseas.

Typhoid

OzFoodNet sites reported 74 cases of typhoid infection during 2001, equating to an overall notification rate of 0.4 cases per 100,000 population (Figure 10). The number of notifications was similar to previous years. The highest rate was reported in New South Wales (0.6 cases per 100,000 population). Tasmania, the Northern Territory and the Hunter sites did not report any cases during 2004.

Where travel status was known, sites reported that 81 per cent (55/68) of cases of typhoid had recently travelled overseas (Table 4). Forty-seven per cent (26/55) of these cases had travelled to the Indian subcontinent and the predominant phage types of *S.* Typhi were E1a (11 cases) and E9 (4 cases). Thirteen cases had recently travelled to Indonesia and the predominant phage types were E2 and E9 with two cases each. Five of the cases infected with typhoid reported recent travel to Samoa and the predominant phage type was E1a (3 cases).

Figure 10. Notification rates of typhoid infections for 2002 compared to mean rates for 1998–2001, by OzFoodNet site

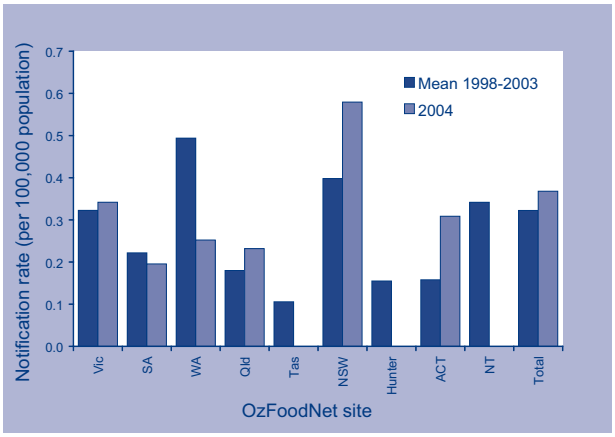


Table 4. Travel status and predominant phage types* for typhoid cases, Australia, 2004

Country	Number of cases	Predominant phage type (number of cases)
Albania	1	E9 (1)
Cambodia	4	E1a (2); E9 (1); Unknown (1)
China	1	O Variant (1)
El Salvador	1	A Degraded (1)
Indian Sub-continent	26	E1a (11); E9 (4); Degraded (3); O Variant (2); A degraded (1); Untypable (1); Unknown (4)
Indonesia	13	Untypable (3); E2 (2); E9 (2); E6 (1); D2 (1); Degraded (1); Unknown (3)
Jordan	1	Unknown (1)
Phillipines	2	Unknown (1); Degraded (1)
Samoa	5	E1a (3); E9 (1); E7 (1)
Sierra Leone	1	Unknown (1)
Locally acquired	13	E1a (5); E9 (2); Degraded (2); D2 (1); Untypable (1); Unknown (2)
Unknown	6	Unknown (5); Degraded (1)
Total	74	

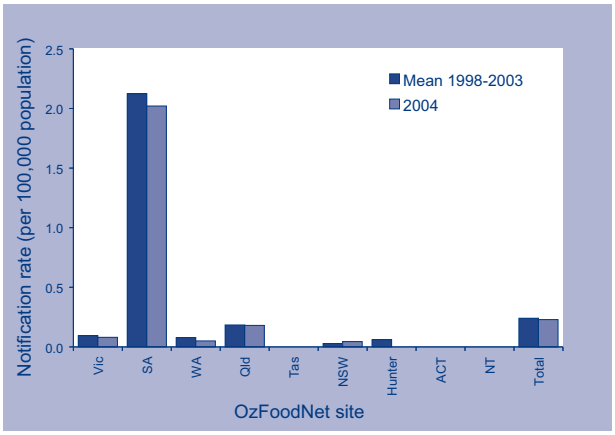
* Numbers in parentheses represent the number of cases infected by the phage type.

One of the locally acquired cases infected with phage type E1 reported that their brother had visited from Samoa. Eight of the locally-acquired cases were considered to be chronic carriers. There was one case of typhoid in a laboratory worker. Travel status was unknown for six cases.

Shiga toxin-producing Escherichia coli *infections*

OzFoodNet sites reported 46 cases of Shiga toxin-producing *E. coli* (STEC) infection during 2004 (Figure 11). This number does not include cases of haemolytic uraemic syndrome where a toxigenic *E. coli* was isolated. The notification rate of 0.2 cases per 100,000 population was a 0.5 per cent increase from the mean rate for the previous six years. South Australia (31 cases) reported the majority of cases, which represented a 3.1 per cent decrease over the historical mean for this State. The highest rate was in South Australia, which reported 2.0 notifications per 100,000 population (Table 5). The second highest number of cases was reported from Queensland, with 7 cases. There were no cases reported from Tasmania, the Hunter, the Australian Capital Territory or the Northern Territory during 2004.

Figure 11. Notification rates of Shiga toxin-producing *Esherichia coli* infections for 2004 compared to mean rates for 1998–2003, by OzFoodNet site



The male to female ratio of cases was 0.5:1 and the highest rates were in females aged between 5–9 years (0.8 cases per 100,000 population) and 20–24 years (0.7 cases per 100,000 population). The reason for the predominance of females amongst notified cases is unknown, but has been observed in previous years.

E. coli O157 was the most common serotype, accounting for 39 per cent of notifications. This serotype has been the most commonly reported for the last three years (Table 6). There were five cases of serotypes O111, four cases of serotype O26 and two cases of serotype O86. No serotype information was available for approximately one third of cases in South Australia due to the use of polymerase chain reaction (PCR) to diagnose infections.

Table 5. Infecting subtypes of Shiga toxin-producing *Escherichia coli*, Australia, 2004, by OzFoodNet site

State	Serotype						U/k	Total
	O111	O141	O157	O26	O5	O86		
NSW			1				2	3
Qld	1	1	1	1		2	1	7
SA	4		13*	2			12	31
Vic			2	1	1			4
WA			1					1
Total	5	1	18	4	1	2	14	46

* One case in South Australia was co-infected with *Escherichia coli* O113.

U/k Unknown.

Table 6. Infecting subtypes of Shiga toxin-producing *Escherichia coli*, Australia, 2002 to 2004

Organism type	2004	2003	2002
O111	5	8	0
O113	1*	0	2
O157	17	13	20
O26	4	0	6
O141	1	0	0
O5	1	1	0
O86	2	0	0
O128	0	0	1
O130	0	1	0
O28	0	1	1
O2	0	0	1
Unspecified	15	27	26
Untypeable	0	2	2
Total	46	53	59

* One case was co-infected with *Escherichia coli* O157.

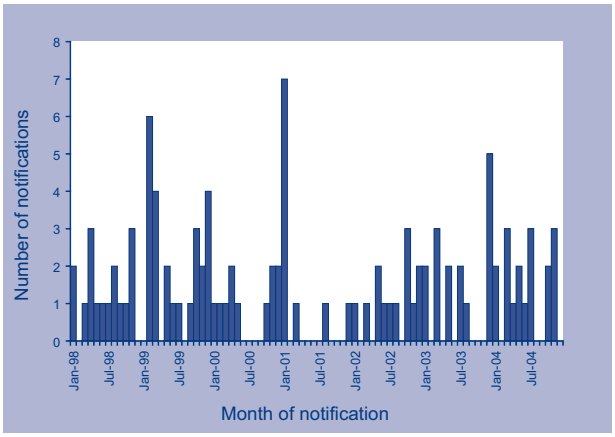
The marked difference in notification rates between states and territories is a result of the practices that pathology laboratories use to screen faecal specimens for toxin-producing *E. coli.* South Australia has the most intensive testing regime and test bloody stool for the presence of the genes coding for production of Shiga toxin. This is reflected in the higher notifications rates reported in South Australia.

All of the cases appeared to be sporadic, except for two small clusters in South Australia and Queensland. South Australia reported a cluster investigation into two cases of STEC; one with *E. coli* O111 and one with *E. coli* O157. Both cases had visited a common native animal petting zoo, however, stool samples of native animals were negative for STEC. Queensland reported an investigation into a cluster of cases of *E. coli* O86:H27 in a single town in January [see the report by A. Morgan, *et al* in this issue, pp 192–195]. There were four cases in total in two sets of siblings. Two children were diagnosed with haemolytic uraemic syndrome and are not included in this section. No source was identified for the Queensland cluster, although animal exposures were suspected as the cause.

Haemolytic uraemic syndrome

There were 17 cases of haemolytic uraemic syndrome reported during 2004, corresponding to an overall rate of 0.1 case per 100,000 population. New South Wales reported nine of these cases. Queensland reported three cases, South Australia two cases, while Victoria, Western Australia and the Northern Territory reported one case each (Figure 12). Cases occurred throughout the year and there was one investigation into a cluster of cases of *E. coli* O86 in Queensland, which involved two cases of haemolytic uraemic syndrome and two cases of Shiga toxin-producing *E. coli* [see pp192–195].

Figure 12. Numbers of notifications of haemolytic uraemic syndrome, Australia, 1998 to 2004, by month of notification

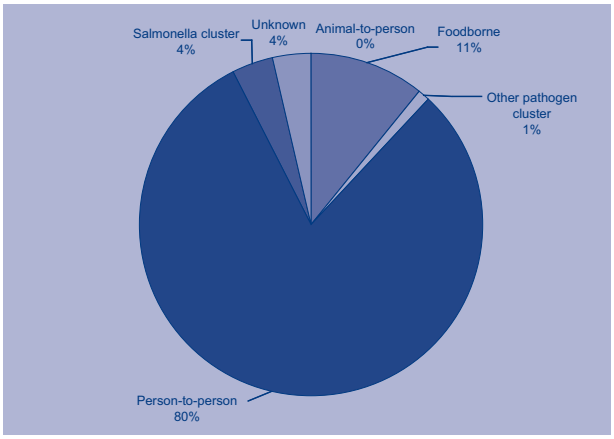


The male to female ratio was 0.4:1 and the highest rate of infection was in females in the 0–4 year age group (0.8 cases per 100,000 population). Details of specific toxigenic *E. coli* infections associated with haemolytic uraemic syndrome were not reported for 13 cases. Two cases were due to the O111 serotype and two were due to O86, while two cases had no STEC isolated.

Gastrointestinal and foodborne disease outbreaks

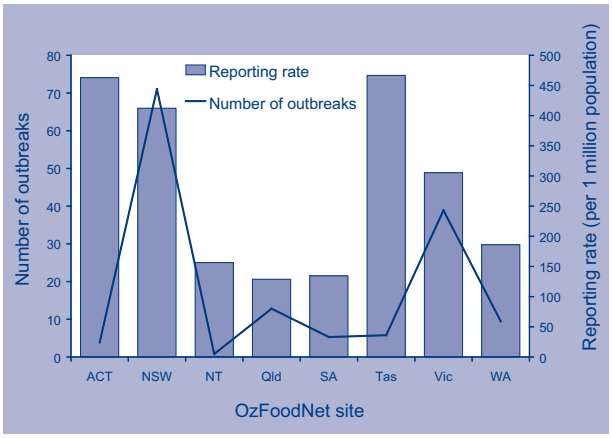
During 2004, OzFoodNet sites reported 1,085 outbreaks of gastrointestinal illness affecting 28,461 persons (Figure 13). One hundred and eighteen of the outbreaks were due to consumption of contaminated food or water giving an overall rate of 5.9 foodborne outbreaks per million population. During 2004, there was one outbreak of zoonotic origin that occurred throughout the year. This outbreak of antibiotic resistant *Salmonella* Paratyphi biovar Java was related to contact with tropical fish.

Figure 13. Foodborne and gastroenteritis outbreaks reported by OzFoodNet sites, Australia, 2004, by suspected mode of transmission (n=1,085 outbreaks)



The mode of transmission of the remaining outbreaks was either unknown or were most likely due to person-to-person transmission. Sites conducted 92 investigations into outbreaks or clusters where the mode of transmission was not determined, or a foodborne source was not identified. Person-to-person transmission was suspected as the cause of 874 outbreaks affecting 25,363 persons. The rates of non-foodborne outbreaks were reasonably consistent across different jurisdictions and ranged between 20.6 per million population in Queensland to 74.7 per million population in Tasmania (Figure 14). The majority of person-to-person outbreaks occurred in aged care facilities (593 outbreaks; 19,295 people affected) and hospitals (140 outbreaks;

Figure 14. Number and rates of non-foodborne gastroenteritis outbreaks,* Australia, 2004, by OzFoodNet site



* Includes outbreaks spread from person-to-person or of unknown mode of transmission and investigations of clusters of infections other than those caused by *Salmonella*, (*n*=924 outbreaks).

3,423 affected). Norovirus was confirmed as the aetiological agent for 398 outbreaks spread from person-to-person that affected 13,842 people.

Foodborne disease outbreaks

In 2004, 118 foodborne disease outbreaks affected 2,076 persons, resulting in 116 hospitalisations and two associated deaths (Table 7). A summary description of each outbreak is shown in Appendix 2.

New South Wales reported the largest number of outbreaks, which represented 36 per cent (43/118) of all outbreaks reported (Table 7). The reporting rates of foodborne outbreaks for different OzFoodNet sites ranged from 1.0 outbreaks per million population in Western Australia to 15.5 outbreaks per million population in the Australian Capital Territory. The majority of outbreaks occurred in summer and autumn (Figure 15).

Figure 15. Outbreaks of foodborne disease, Australia, 2001 to 2004, by selected aetiological agents

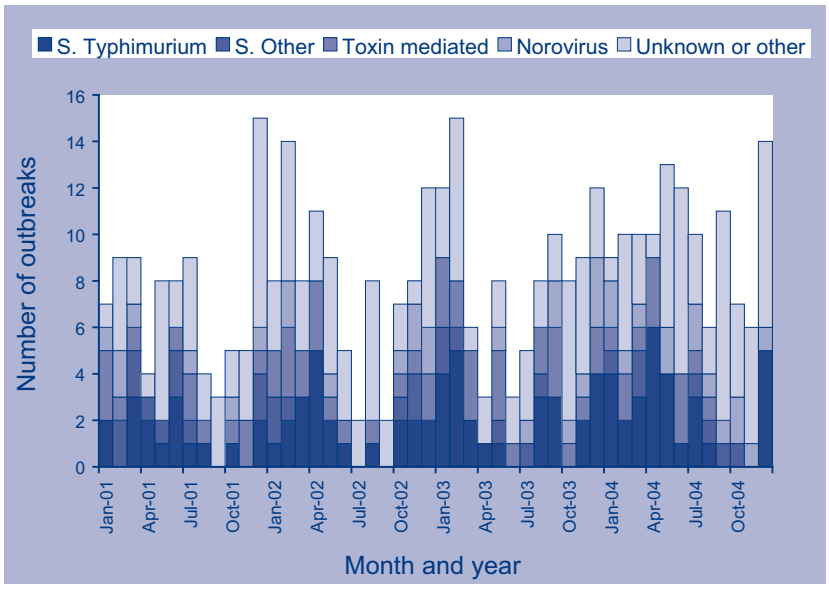


Table 7. Outbreaks of foodborne disease in Australia, 2004, by OzFoodNet site

State	Number of outbreaks	Outbreaks per million population	Mean number of cases per outbreak	Number affected	Hospitalised	Deaths
ACT	5	15.5	58.8	294	2	0
NSW	43	6.4	14.8	635	45	0
NT	2	10.1	7.0	14	2	0
Qld	27	7.1	9.4	254	20	0
SA	17	11.1	9.0	153	10	2
Tas	1	2.1	57.0	57	0	0
Vic	21	4.3	26.2	550	37	0
WA	2	1.0	59.5	119	0	0
Total	118	5.9	17.6	2,076	116	2

Aetiological agents

The most common agent responsible for foodborne disease outbreaks was *Salmonella*, which was responsible for 31 per cent (36/118) of outbreaks (Table 8). These outbreaks affected a total of 679 persons with a hospitalisation rate of 12 per cent (79/679). *S.* Typhimurium was responsible for 81 per cent (29/36) of *Salmonella* outbreaks. Norovirus caused 14 outbreaks of foodborne illness, with a low hospitalisation rate of only 0.4 per cent (2/500). There were eight outbreaks of 'suspected toxin' poisoning, which included seven outbreaks suspected to be due to *Clostridium pefringens* and one outbreak of suspected histamine poisoning due to 'butterfish' consumption.

In 2004, there were seven small outbreaks of ciguatera, all of which occurred in Queensland. There were five outbreaks of campylobacteriosis, which was more than in previous years. There were single outbreaks each of *Bacillus cereus*, rotavirus, mixed toxins from *B. cereus* and *Staphylococcus aureus* and *Listeria* infection. In an outbreak of listeriosis two cases died, although it is unclear whether *Listeria* infection was the major contributing factor to the deaths. Thirty-five per cent (41/118) of outbreaks were of unknown aetiology.

Food vehicles

There was a wide variety of foods implicated in outbreaks of foodborne disease during 2004 (Table 9), although investigators could not identify a vehicle for 47 per cent (55/118) of outbreaks. Contaminated fish was the most common food vehicle, seven of which caused ciguatera poisoning. Fish was responsible for nine per cent (10/118) of outbreaks, followed by seafood and mixed meat dishes each responsible for six outbreaks. Poultry, cakes, pizza, oysters and egg dishes were also common causes of outbreaks.

Outbreak settings

The most common settings where food was prepared was at restaurants and cafés (36%), followed by commercial caterers (14%), takeaway venues including nationally franchised fast food chains (13%), and private residences (11%) (Table 10). Contaminated primary produce was responsible for 7 (6%) outbreaks. Five outbreaks occurred in association with foods prepared in aged care facilities. Four outbreaks each were due to foods prepared in bakeries and in hospital settings. The settings where foods were consumed were similar to where it was prepared. Restaurants and cafés (36%) were the most common venues, followed by private residences (20%), catered functions (9%) and community settings (7%).

Investigative methods and levels of evidence

States and territories investigated 41 outbreaks using retrospective cohort studies and nine outbreaks using case control studies. Forty-four per cent (18/41) of outbreak investigations using cohort studies were of unknown aetiology. Twenty-four per cent (10/41) of investigations using cohort studies were *Salmonella* outbreaks. Fifty per cent of investigations of toxin and suspected toxin outbreaks used cohort studies. Fifty-six per cent (5/9) of outbreak investigations using case control studies were due to *Salmonella*. Sixty outbreaks relied on descriptive information to attribute a foodborne cause or identify a food vehicle, while investigators did not collect individual patient data for eight outbreaks.

Table 8. Aetiological agents responsible for foodborne disease outbreaks showing number of outbreaks and numbers of persons affected, Australia, 2004

Agent category	Number of outbreaks	Number of people affected	Number of people hospitalised	Mean size of outbreak
Bacillus cereus	1	6	0	6.0
Clostridium perfringens	3	128	1	42.7
Campylobacter	5	58	4	11.6
Ciguatoxin	7	24	3	3.4
Listeriosis	1	2	2	2.0
Norovirus	14	500	2	35.7
Rotavirus	1	14	0	14.0
Salmonella other	7	80	5	11.4
Salmonella Typhimurium	29	599	74	20.7
Suspected toxin	8	209	2	26.1
Mixed toxins	1	16	0	16.0
Unknown	41	440	23	10.7
Total	118	2,076	116	17.6

Table 9. Categories of food vehicles implicated in foodborne disease outbreaks, Australia, 2004

Food category	Number of outbreaks	Per cent	Number affected	Number hospitalised
Cakes	4	3.4	82	10
Custard	1	0.9	43	17
Dessert	1	0.9	4	0
Dips	1	0.9	14	0
Eggs	1	0.9	4	0
Fish	10	8.6	52	8
Mixed dish	5	4.3	63	1
Mixed meat dish	6	5.2	191	2
Oysters	4	3.4	35	1
Pizza	4	3.4	108	8
Pork	1	0.9	27	1
Poultry	6	5.1	188	3
Salad	1	0.9	28	3
Sandwiches	3	2.6	270	0
Seafood	6	5.2	45	10
Suspected eggs	2	1.7	19	6
Suspected poultry	2	1.7	24	2
Suspected red meat	1	0.9	5	5
Suspected water	1	0.9	7	0
Vegetable dish	1	0.9	6	0
Unknown	57	49.1	861	39
Total	118	100.0	2,076	116

Table 10. Categories of settings where food was prepared in association with foodborne disease outbreaks, Australia, 2004

Setting prepared	Number of outbreaks	Number affected	Number hospitalised
Aged care	5	75	4
Bakery	4	82	10
Café	2	17	3
Camp	1	5	0
Commercial caterer	16	683	15
Contaminated primary produce	7	58	9
Grocery store/delicatessen	2	30	0
Hospital	4	42	7
Institution	2	52	17
National franchised fast food	7	83	11
Private residence*	14	157	6
Restaurant	40	558	27
Takeaway	8	30	1
Other	1	27	1
Unknown	5	177	5
Total	118	2,076	116

* Includes one outbreak where food prepared included food prepared by takeaway stores.

To attribute the cause of the outbreak to a specific food vehicle, investigators obtained analytical evidence from epidemiological studies for 15 outbreaks. Sixty-six per cent (27/41) of cohort and 50 per cent (4/8) of case control studies did not identify a significant association between illness and a specific food vehicle. Microbiological evidence of contaminated food was found in 10 outbreaks, with a further four outbreak investigations obtaining both microbiological and analytical evidence. Investigators obtained analytical and/or microbiological evidence for 33 per cent (12/36) of *Salmonella* outbreaks (Appendix 2).

Large outbreaks (>50 persons affected)

Six outbreaks affected 50 persons or more in 2004. Two were due to norovirus, two due to *Salmonella*, one due to *C. perfringens,* and one due to suspected *C. perfringens* intoxication. The food for two of these outbreaks was prepared at commercial caterers, with a third using a mixture of food prepared at homes and takeaway food.

Outbreaks also resulted from the food prepared at a restaurant and a bakery. The sixth outbreak was a large community-wide outbreak of *Salmonella* Typhimurium 12 in New South Wales that was associated with chicken prepared in a variety of settings. A variety of foods were implicated in these large outbreaks, including: bakery products, chicken, sandwiches, and dishes containing chicken including pizza.

The two large outbreaks of norovirus were related to preparation of foods that required considerable handling. The food vehicles implicated in the two outbreaks were sandwiches containing salmon and egg fillings in one large outbreak involving a commercial caterer, and contaminated bakery products in the second outbreak. In both outbreaks, the investigation identified food handlers who had worked while ill with gastroenteritis.

One of the outbreaks of *Salmonella* was due to serotype Typhimurium phage type 9 at a pizza restaurant in Melbourne. Cases continued to occur after an initial cleaning of the facility. Several foods were positive for *S.* Typhimurium 9, along with swabs of food preparation areas. Cases occurring early in the outbreak were associated with pizza, whereas those occurring after the initial cleaning were associated with dishes containing chicken, including pasta, risotto and pizza. The cause of the outbreak was suspected to be due to cross contamination in the kitchen due to poor hygienic practices, including cooking chicken on trays in a pizza conveyor belt. After a second clean up of the restaurant there were no new cases or positive food samples.[16]

New South Wales conducted a major investigation into a state-wide increase of *Salmonella* Typhimurium 12 during February. A case series investigation of 40 cases interviewed with hypothesis generating questionnaires identified that cases reported a high consumption rate of fruit and vegetables when compared to previous studies. To examine this hypothesis, a case control study used community-based controls recruited randomly using the electronic white pages, and cases with other *Salmonella* Typhimurium phage types as controls. Forty-one cases—48 *S.* Typhimurium controls and 203 community controls—were recruited. Consuming chicken breast prepared in the home was identified as a risk factor for *S.* Typhimurium 12 infection (Odds Ratio 4.6, $p<0.1$). New South Wales reported 141 cases as part of this outbreak, making it the largest outbreak of salmonellosis during 2004.

Medium outbreaks (20–49 persons affected)

There were 22 outbreaks affecting between 20 and 49 persons. Seven of these outbreaks were due to *Salmonella,* including an outbreak of *Salmonella* Stanley in a school in Victoria. Food vehicles were only identified for three of these *Salmonella* outbreaks, which were custard, salad rolls containing red onion, and roast pork.

There were four outbreaks of norovirus, three of which occurred at restaurants and a fourth that occurred at a mass catered event. One norovirus outbreak in New South Wales affected 24 people following consumption of locally-grown oysters. No further illness was identified and norovirus was not detected in samples of oyster meat. There were two outbreaks of campylobacteriosis, one of which was suspected to be related to a barbecue meal at an aged care facility in Victoria, while the other was suspected to be due to a meal of chicken in New South Wales.

Victoria reported four outbreaks of suspected *C. perfringens* affecting between 20–49 people, only one of which could be confirmed by the presence of heavy growth of *C. perfringens* and the presence of toxin in stool samples. One of these outbreaks was in an aged care facility, while another was in a hospital. The other two suspected toxin related illnesses were associated with restaurants.

There were three outbreaks associated with imported foods with potential international implications. These outbreaks were all due to contaminated Individually Quick Frozen oysters. All outbreaks were small affecting a total of 11 people. Two occurred in Queensland and one in the Northern Territory. Investigations confirmed that these oys-

ters from Japan were the same as those implicated in December 2003 in outbreaks in the Northern Territory and Western Australia. Oysters from all outbreaks were tested but norovirus was not detected in any of these three batches. Investigation of the outbreak that occurred in Queensland in October 2004 implicated oyster meat from the same batch as the outbreak in the Northern Territory that occurred a year earlier, although it was a smaller size oyster.[17] This particular batch had reportedly been withdrawn from sale earlier in 2004, and had been shown by laboratory tests to be contaminated with norovirus.

Cluster investigations

A cluster is defined as an increase in infections that are epidemiologically related in time, place or person where investigators are unable to implicate a vehicle or determine a mode of transmission for the increase. An example is a temporal or geographic increase in the number of cases of a certain type of *Salmonella* serovar or phage type. Another example is a community-wide increase of cryptosporidiosis that extends over some weeks or months. In this report, there were a small number of outbreaks of different pathogens where the mode of transmission was unknown, that have been classified as a cluster.

During 2004, states and territories conducted 54 cluster investigations. These clusters affected 622 persons with 51 cases hospitalised. Seventy-eight per cent (42/54) of these investigations related to clusters of *Salmonella*. *Salmonella* clusters affected 473 persons with 46 cases hospitalised. *S.* Typhimurium was responsible for 55 per cent (23/42) of cluster investigations, with phage types 135/a (6 investigations) and 170/108 (5) being the most common. Of the remaining 19 investigations, there were 18 other different *Salmonella* serovars involved. There were 11 clusters due to pathogens other than *Salmonella*, with *Campylobacter*, *Cryptosporidium,* Shiga toxin-producing *E. coli* and *Shigella* causing two each and one investigation into a cluster of hepatitis A. No aetiology was identified for three cluster investigations.

OzFoodNet sites compared investigations into concurrent increases in several *Salmonella* serotypes that occurred across multiple jurisdictions. These included *Salmonella* serotypes Singapore, Typhimurium 12a, Typhimurium 170/108, and Paratyphi B biovar Java.

The cluster investigation into cases of *Salmonella* Paratyphi B biovar Java was part of a national case series to investigate the association with tropical fish aquariums. Eighteen cases infected with this multi-drug resistant serotype were investigated. In the month prior to illness, 85 per cent (11/13) of cases with aquarium/tanks had contact with sick or dead fish.

The true number of clusters investigated is difficult to determine, as the figures do not include all cluster investigations conducted in Public Health Units or local government areas. Jurisdictions have different definitions of 'cluster' and triggers for investigating clusters to fit with staff resources and local priorities.

Risk factors for infection

During 2004, OzFoodNet identified several important risk factors for foodborne illness as a result of outbreak investigations and from preliminary results of case control studies. These included risks due to the following foods and settings for foodborne disease.

Eggs

Sites continue to report outbreaks associated with the consumption of egg-based products, such as eggs, salad dressings, cakes and desserts. There were three outbreaks suspected to be caused by eggs, and a further three associated with desserts and cakes where eggs were suspected as the source of *Salmonella.* In one outbreak of *Salmonella* Typhimurium 126 infections in Victoria, illness was associated with consumption of one brand of organic eggs. In several of these outbreaks, investigators were unable to trace implicated eggs back to a single farm. There is a need to identify potential interventions, and a review of quality assurance in the industry may be appropriate. The restaurant and catering industries need to be made aware of the potential risks of using raw unpasteurised eggs in sauces, dressings and desserts.

Chicken and poultry

During 2004, outbreaks of poultry-associated salmonellosis continued to occur, including a major outbreak of *Salmonella* Typhimurium 12 in New South Wales. Poultry is consumed by approximately 80 per cent of people each week. To make our food supply safer, it is important to consider ways to reduce the burden of infections in the community due to consumption of poultry.

Oysters and fish

The four outbreaks associated with oysters during 2004 showed their potential to cause outbreaks of human illness. Three of these outbreaks were due to contaminated imported oyster meat from a single estuary system in Japan. In 2004, the Australian Quarantine and Inspection Service restricted the importation of these products from this growing area. Importation from certain growing areas in Korea were also restricted, as oysters from this area had caused norovirus outbreaks in New Zealand.

There were 10 outbreaks due to fish during 2004, making it the most common food vehicle. The majority of these were small outbreaks of ciguatera poisoning in Queensland. Many outbreaks of ciguatera relate to fish caught by amateur fishermen, but one of these outbreaks was associated with coral trout eaten at a restaurant. Ciguatera can be a severe illness and there is a continuing need to educate amateur fishermen about ciguatera including the risks associated with fishing known ciguatera areas and consuming large warm ocean fish.

Settings

There were several settings where food was prepared or consumed that were identified as high risk for foodborne disease, which included:

Bakeries

The four outbreaks occurring in bakeries in 2004 revealed the need for assessment of food safety issues in these premises. Three of the outbreaks were associated with cakes, some of which were filled with cream or custard. Two of the outbreaks were caused by *Salmonella* Typhimurium, while one was unknown and another was due to norovirus. Epidemiological investigation of these outbreaks often does not uncover the real source of contamination, as there is a time lag between food consumption and the recognition of the outbreak. Food safety agencies may need to consider the development of hazard reduction plans for these facilities to prevent further outbreaks.

Restaurants and catered events

Outbreaks in this sector constituted 49 per cent (58/118) of outbreaks. A variety of pathogens caused these outbreaks, including *Salmonella, C. perfringens,* norovirus and ciguatera. Outbreaks involving restaurants and commercial caterers are more readily recognised, as the meals are often served to large numbers of persons. A wide range of food vehicles were responsible for outbreaks in this sector. Clearly there is a need to continue to monitor the causes of outbreaks in restaurant and catering settings to identify potential gaps in food safety practices.

Hospitals and aged care

People resident in aged care facilities and patients in hospital are at particular risk for foodborne disease, which is shown by the nine outbreaks that occurred during 2004. Four of the outbreaks were suspected or confirmed *Clostridium perfringens* outbreaks, while two were due to *Campylobacter* infection, one due to *Salmonella* Typhimurium 126var, one due to *Listeria monocytogenes* O1 and one unknown. The majority of these outbreaks indicate problems with preparation and handling of foods for residents. The outcomes for patients in these settings are often more adverse, as these sub-populations are more susceptible to serious foodborne disease. The food supplied to hospital patients and persons in institutions should be comprehensively monitored. In addition, there is a need to ensure that patients at risk for infection should not be fed high-risk foods in hospitals.

Surveillance evaluation and enhancement

Continuous improvement of surveillance is important to ensure that foodborne illness is investigated rapidly and effectively. To improve surveillance it is necessary to evaluate and compare practices conducted at different sites.

National information sharing

In 2004, all jurisdictions contributed to a fortnightly national cluster report to identify foodborne illness occurring across state and territory boundaries. The cluster report supplemented information sharing on a closed list server, teleconferences and at quarterly face-to-face meetings.

Outbreak reporting and investigation

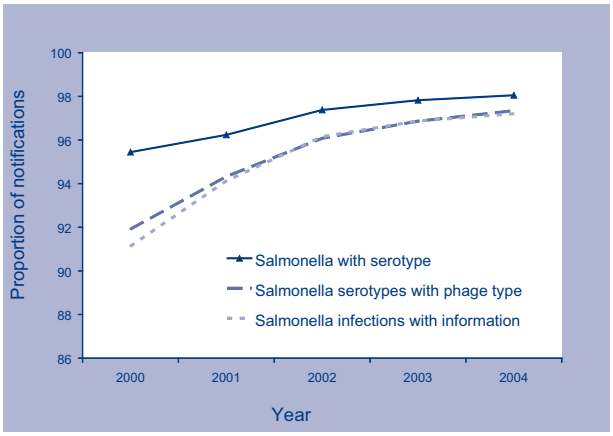
During 2004, the Australian Capital Territory site reported the highest rate of outbreaks of foodborne disease (15.5 outbreaks per 100,000 population). The rates of reporting foodborne *Salmonella* outbreaks ranged between 1.6–5.0 outbreaks per 100,000 population. New South Wales investigated the largest number of foodborne disease outbreaks (43 outbreaks; 6.4 per 100,000 population). Victoria and Queensland investigated 13 *Salmonella* clusters each, giving rates of 3.4 and 2.6 per million population respectively.

States and territories conducted 50 analytical studies (cohort or case control studies) to investigate foodborne disease outbreaks or clusters of suspected foodborne illness. Investigators used analytical studies for 42 per cent (50/118) of foodborne disease outbreaks, which was similar to previous years. Queensland conducted four case control studies to investigate outbreaks of foodborne infections during 2004, which was the most for any jurisdiction. Every jurisdiction reported conducting at least one cohort study. New South Wales conducted 40 per cent of all cohort studies.

Completeness of Salmonella *serotype and phage type reports*

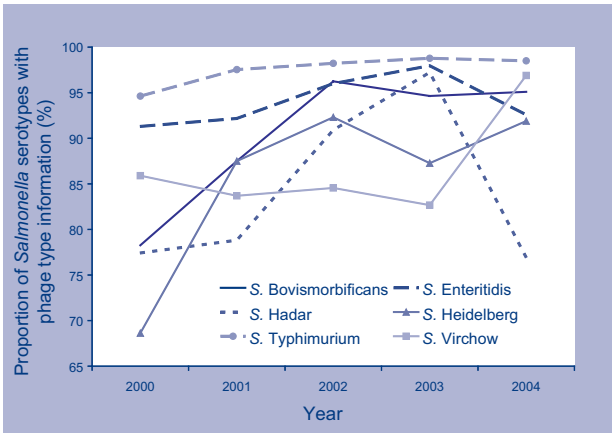
There was considerable improvement in the completeness of *Salmonella* available on state and territory surveillance databases between the years 2000 to 2004 (Figure 16). Overall 98.4 per cent (7,671/7,798) of *Salmonella* notifications on databases contained either serotype or phage type, which was an increase of 7.3 per cent from 2000 and 1.5 per cent from 2003.

Figure 16. Proportion of *Salmonella* infections notified to State and Territory health departments with serotype and phage type information available, Australia, 2000 to 2004



Only 76.9 per cent (39/48) of phage type information was reported for *S.* Hadar, which was a decline from the previous year (Figure 17). Phage typing information was available for 91.9 per cent (37/40) of *S.* Heidelberg and 92.6 per cent (418/449) of reports for *S.* Enteritidis in 2004. The largest increase in completeness between 2000 and 2004 was reported for *S.* Heidelberg (23.3%) and *S.* Bovismorbificans (17%).

Figure 17. Proportion of *Salmonella* infections for six serotypes notified to State and Territory health departments with phage type information available, Australia, 2000 to 2004



South Australia had the highest proportion of complete *Salmonella* notification (100%), while four sites reported 98 per cent or higher. Western Australia reported the lowest rate of completeness with 91.9 per cent. New South Wales reported the largest improvement with 19.1 per cent improvement, when compared to 2000 figures.

Discussion

This fourth annual report of OzFoodNet highlights the burden that foodborne illness places on the health system and community. The cost to Australia each year from foodborne disease may be as high as $AUD1.2 billion annually.[18] In recent years, Australia has experienced consistently increasing rates of notified infections, along with increasing numbers of foodborne outbreaks. In 2004, we observed a 28 per cent increase in reported foodborne outbreaks compared with 2003, which may be due in part to improved surveillance. However, there has not been any appreciable change in surveillance system for notifiable diseases. This is a concern, as there was a nine per cent increase in notifications of potentially foodborne diseases in this report when compared to historical averages.

The United States of America program—FoodNet—recently reported significant declines for 2004 in the incidence of human salmonellosis and campylobacteriosis, which were attributed to improvements in agricultural industry and reduction in isolation of *Salmonella* and *Campylobacter* in food processing plants.[9] Australia could consider gathering similar data on *Salmonella* and *Campylobacter* in animals and developing disease reduction targets to focus prevention efforts.

The major causes of foodborne disease in Australia during 2004 were similar to previous years, with fish, poultry, bakery products, seafood, and eggs being the major causes of outbreaks. While seafood and fish are responsible for large numbers of outbreaks they are usually small in size and are rarely associated with *Salmonella* and *Campylobacter* infections, which make up the majority of sporadic infections reported to health departments.[19] In contrast, poultry and eggs, are common causes of these sporadic infections. Risk of campylobacteriosis is strongly associated with consumption of under-cooked chicken in Australia and may be responsible for between 5–11 per cent of infections.[20] OzFoodNet sites reported several outbreaks associated with chicken during 2004.

The largest of these chicken-associated outbreaks was a community-wide outbreak of *Salmonella* Typhimurium 12 in New South Wales that affected 141 people. These community-wide increases occur commonly and are difficult to investigate due to poor patient recall of foods consumed and the high frequency of chicken consumption. Chicken meat is commonly contaminated with *Salmonella* and *Campylobacter* at retail sale.[21,22] While cooking readily kills these bacteria, reducing the concentration of bacteria on meat through control measures on farms or in processing plants could possibly lower the incidence of these diseases in the community. The *S.* Typhimurium 12 outbreak highlighted the potential of chicken as a vehicle of community outbreaks and the need to reduce contamination of raw meats through improved primary production and processing.

There were many outbreaks in 2004 where investigators were unable to confirm the aetiology. One reason for this is that surveillance of outbreaks is improving with smaller outbreaks being detected where it is difficult to confirm an aetiological agent. Another reason is the changing nature of laboratory tests. The availability of test kits for *C. perfringens* in Australia was limited in 2004, due to concerns about importation of *Clostridium* toxins. This made it difficult to confirm *Clostridium perfringens* as the cause of outbreaks, as traditional case definitions rely on confirming greater than 10^5 organisms in two or more faecal specimens or the demonstration of *C. perfringens* toxin.[23]

Similarly, epidemiologists were unable to identify a food vehicle in 63 per cent of outbreaks investigated using analytical epidemiological studies. In many of these outbreak investigations epidemiologists suspected a vehicle, but did not have the epidemiological, microbiological or traceback evidence to implicate a specific food. It can be difficult to effect and implement food recalls in these instances.

The outbreaks of norovirus associated with Individually Quick Frozen oyster meat were an example of where epidemiological and traceback evidence confirmed a food vehicle, but virological analysis was negative for norovirus. National discussions with food safety agencies identified the risks associated with these products, and Importers agreed to withdraw products from the marketplace. During 2004, the Northern Territory Health Services submitted oyster meat from an outbreak in the previous year to Environmental Science and Research in New Zealand, which confirmed contamination with norovirus (personal communication, Gail Greening, 3 May 2005). This positive test result, months after the initial investigation, vindicated various food safety agencies decisions to seek the withdrawal of these products. The small outbreak in Queensland in October several months after the original withdrawal showed how long-shelf life products can cause problems for public health agencies conducting food recalls. As a preventive measure, the Australian Quarantine and Inspection Service has implemented restrictions to imports of oyster meat from the specific harvest area where implicated oysters were harvested, and certain Korean oyster growing areas that have supplied products causing outbreaks in New Zealand.

Norovirus caused a considerable amount of gastroenteritis that was not foodborne in 2004. OzFoodNet sites reported a massive number of outbreaks spread from person-to-person, many of which occurred in aged care and hospital settings. Norovirus was responsible for 45 per cent of outbreaks spread from person-to-person and accounted for 13,739 cases. The genotype of these norovirus outbreaks was reported for very few of these outbreaks, as many were diagnosed using rapid enzyme-based detection test kits. It is likely that many of these outbreaks would have been due to the new variant of genotype II4 that affected Australia during 2004 and other countries internationally.[24] Epidemic clones of norovirus can affect multiple countries and cause widespread illness in community and institutional settings.[25] It is also likely that many of the person-to-person outbreaks of unknown aetiology would have been due to norovirus. The high burden of disease in the healthcare system is very costly and it is important for public health agencies to be able to identify interventions that are able to halt the spread of these outbreaks.[26]

OzFoodNet identified several risk factors for foodborne infections in 2004 based on the surveillance data and epidemiological studies. Many of these risk factors have been previously recognised, but may need to be considered again. Some of the risks require complex solutions, while others are far more simple, such as using pasteurised eggs in food service industries or avoiding consumption of shellfish and fish harvested from high-risk areas. While Australia does not have endemic *S.* Enteritidis 4 that contaminates the internal contents of eggs, there are clearly other subtypes of *Salmonella* that are associated with eggs. As noted in previous years, there is a need to identify the potential food safety failures in bakeries to prevent outbreaks of salmonellosis.[27]

It is important to recognise some of the many limitations of the data that OzFoodNet reports. Surveillance data are inherently biased and require careful interpretation. These biases include the higher likelihood that certain population groups will be tested, and different testing regimes in different states and territories, resulting in different rates of disease. In some jurisdictions, the rates of disease

are unstable due to small numbers of notifications and populations under surveillance. Importantly, some of the most common enteric pathogens are not notifiable, particularly norovirus, *Clostridium perfringens* and enteropathogenic *E. coli.* These organisms may be notified as the cause of outbreaks, but not as individual cases of disease. There can also be considerable variation in assigning causes to outbreaks depending on investigators and circumstances. States and territories are moving towards harmonising surveillance as much as possible to address some of these issues.

Surveillance of foodborne diseases in Australia has steadily improved in recent years, as shown by data on improving completeness of *Salmonella* subtyping on state and territory surveillance databases. OzFoodNet aims to continually improve surveillance and investigation practices regarding enteric and foodborne diseases. In 2004, the capacity of Australia to respond to multi-state foodborne disease outbreaks was examined in an external review. The review found that in recent years there had been significant improvements in capacity, but there was still room for improvement. Following the consultation, OzFoodNet conducted a trial of a web-based database for capturing individual patient data during multi-state outbreaks.[28] The trial was based on a mock outbreak of a fictitious *Salmonella* serotype—Mordor—amongst hobbits, wizards, ents and men after a hobbit's birthday party. The trial showed the benefits of using web-based databases for multi-site investigation of outbreaks. The issues highlighted in the review are common to many countries that investigate foodborne illness across multiple jurisdictions.[29]

The large burden of foodborne disease observed in 2004 is a concern for Australia. In future years, it may be appropriate to set targets for reducing foodborne disease in conjunction with other government agencies and industries. It is important that foodborne disease surveillance is able to assess whether food safety policies and campaigns are working. National surveillance of foodborne diseases has many benefits and provides long-term data to review causes of illness. Since OzFoodNet began surveillance in 2000, the network has collected information on the cause of approximately 400 outbreaks of foodborne disease. These data are becoming useful for reviewing less common, but important, causes of disease outbreaks. Ongoing efforts are needed to strengthen the robustness of these data and ensure that they continue to be useful to agencies developing food safety policy.

Acknowledgements

We would like to thank the many epidemiologists, project officers, interviewers and research assistants at each of the Sites who contributed to this report. We also acknowledge the work of various public health professionals and laboratory staff around Australia who interviewed patients, tested specimens, typed isolates and investigated outbreaks. The high quality of their work is the foundation of this report. OzFoodNet is an initiative of the Australian Government.

Appendix 1. Number of cases and rates per 100,000 population of potentially foodborne diseases reported to OzFoodNet sites, 2004

Infection or illness		ACT	Hunter	NSW	NT	Qld	SA	Tas	Vic	WA	Total
Campylobacter	cases	372	nn	nn	219	4,127	1,957	611	6,385	1,969	15,640
	rate	114.8	nn	nn	109.5	106.3	127.6	126.7	128.4	99.3	116.9
Haemolytic uraemic syndrome	cases	0	0	9	1	3	2	0	1	1	17
	rate	0.0	0.0	0.1	0.5	0.1	0.1	0.0	0.0	0.1	0.1
Listeria	cases	1	1	30	1	8	3	1	13	9	66
	rate	0.3	0.2	0.5	0.5	0.2	0.2	0.2	0.3	0.5	0.3
Salmonella†	cases	100	126	2,127	390	2,805	531	120	1,154	615	7,842
	rate	30.9	22.9	31.6	195.1	72.3	34.6	24.9	23.2	31.0	39.0
Shiga toxin producing *E. coli*	cases	0	0	3	0	7	31	0	4	1	46
	rate	0.0	0.0	0.0	0.0	0.3	2.0	0.0	0.1	0.1	0.2
Shigella	cases	2	–	97	117	68	57	3	64	112	520
	rate	0.6	–	1.4	58.5	1.8	3.7	0.6	1.3	5.7	2.6
Typhoid	cases	1	0	39	0	9	3	0	17	5	74
	rate	0.3	0.0	0.6	0.0	0.2	0.2	0.0	0.3	0.3	0.4
Yersinia	cases	1	nn	nn	0	100	6	0	nn	1	108
	rate	0.3	nn	nn	0.0	2.6	0.4	0.0	nn	0.1	1.3

nn not notifiable.

† Includes cases of *Salmonella* Paratyphi.

Appendix 2. Outbreak summary for OzFoodNet sites, 2004

State	Month of outbreak	Setting prepared	Agent category	Number affected	Hospitalised	Evidence*	Epidemiolgical study†	Food vehicle
ACT	April	Restaurant	Unknown	16	0	A	CCS	Suspected calamari
	April	Restaurant	*Salmonella* Typhimurium 197	12	2	M	C	Ling fish
	May	Commercial caterer	Norovirus	247	Unknown	A	C	Salmon and egg sandwiches
	May	Bakery	Unknown	7	0	D	D	Chocolate cake
	July	Grocery store/delicatessen	Unknown	12	0	D	D	Unknown
NSW	January	Hospital	Unknown	5	5	D	N	Suspected beef curry
	January	Takeaway	*Salmonella* Typhimurium 170	3	1	D	D	Suspected chicken
	January	Takeaway	Unknown	3	0	D	N	Unknown
	January	Restaurant	*Salmonella* Typhimurium 170	2	0	D	N	Tartare sauce, fish and chips
	February	Restaurant	Norovirus	32	0	D	C	Unknown
	February	Restaurant	Unknown	20	0	A	C	Bacon and mushroom dish
	February	Restaurant	Unknown	7	6	D	D	Fried rice, pippis
	February	Unknown	Unknown	6	0	D	C	Unknown
	February	Unknown	*Salmonella* Typhimurium 12	141	Unknown	A	CCS	Chicken
	March	Restaurant	*Salmonella* Typhimurium 170	17	1	M	D	Chinese food
	March	Private residence	Unknown	14	0	D	N	Unknown
	March	National franchised fast food	*Salmonella* Typhimurium U290	3	0	D	D	Fish cakes
	April	Restaurant	*Salmonella* Typhimurium 170	13	3	D	D	Chicken
	May	Institution	*Salmonella* Typhimurium 135	43	17	AM	C	Custard
	May	Other	*Salmonella* Typhimurium 170, RDNC	27	1	AM	CCS	Roast pork
	May	Grocery store/delicatessen	Unknown	18	0	D	D	Sandwiches
	May	National franchised fast food	Unknown	5	1	D	D	Suspected BBQ meat pizza
	May	Takeaway	Unknown	5	0	D	N	Takeaway chicken
	May	Restaurant	Unknown	3	0	D	C	Unknown
	June	Restaurant	Rotavirus	14	0	D	D	Dips (salsa, bean/guacamole)
	June	Restaurant	Unknown	8	0	D	N	Unknown
	June	Restaurant	Unknown	6	0	D	C	Unknown
	June	Restaurant	Unknown	6	0	D	N	Unknown
	June	Restaurant	Unknown	3	0	D	N	Chinese food
	July	Contaminated primary produce	Norovirus	24	1	A	C	Oysters
	August	Private residence	*Salmonella* Typhimurium U290	3	2	D	D	Chinese style minced fish balls

Appendix 2. Outbreak summary for OzFoodNet sites, 2004, *continued*

State	Month of outbreak	Setting prepared	Agent category	Number affected	Hospitalised	Evidence*	Epidemiolgical study†	Food vehicle
NSW cont'd	September	Restaurant	Unknown	13	0	D	C	Unknown
	September	unknown	Unknown	12	3	D	C	Unknown
	September	Restaurant	Unknown	11	0	D	C	Unknown
	September	Restaurant	Unknown	4	0	D	D	Unknown
	September	Takeaway	Unknown	4	0	D	D	Unknown
	October	Restaurant	Unknown	7	0	D	C	Cold chicken sandwiches
	October	Private residence	Unknown	5	0	D	D	Unknown
	October	Restaurant	*Salmonella* Chester	3	1	D	D	Unknown
	November	Commercial caterer	Unknown	33	0	D	C	Unknown
	November	Restaurant	Unknown	7	0	D	D	Unknown
	December	Commercial caterer	Unknown	42	0	D	C	Unknown
	December	Restaurant	*Campylobacter*	21	1	M	C	Suspected chicken
	December	Restaurant	Unknown	13	0	D	C	Unknown
	December	Restaurant	Unknown	12	0	D	C	Suspected bacon and ham
	December	Commercial caterer	Unknown	8	0	D	D	Unknown
	December	Takeaway	Unknown	6	0	D	D	Unknown
	December	Restaurant	*Salmonella* Typhimurium 135	3	2	M	D	Crab
NT	January	Café	*Salmonella* Typhimurium 108	9	2	D	D	Unknown
	May	Contaminated primary produce	Unknown	5	0	D	C	Oysters (frozen)
Qld	January	Contaminated primary produce	Norovirus	4	0	D	D	Oysters (frozen)
	January	Private residence	Ciguatoxin	2	2	D	D	Golden spotted trevally fish
	February	National franchised fast food	*Bacillus cereus*	6	0	M	D	Potato and gravy
	February	Restaurant	Ciguatoxin	4	1	D	D	Coral trout
	March	Restaurant	*Salmonella* Singapore	13	0	A	CCS	Sushi rolls
	March	Commercial caterer	Norovirus	8	0	D	C	Unknown
	March	Restaurant	Unknown	5	0	D	D	Sandwiches
	March	Restaurant	*Salmonella* Zanzibar Var 15+	5	0	D	D	Unknown
	March	Private residence	Ciguatoxin	2	0	D	D	Fish species unknown
	April	National franchised fast food	*Salmonella* Typhimurium 12a	41	10	D	CCS	Unknown
	April	Restaurant	Mixed toxins	16	0	M	D	Japanese rice balls,/omelette, chicken, fish
	April	Contaminated primary produce	Ciguatoxin	5	Unknown	D	D	Spanish mackerel/trevally

Appendix 2. Outbreak summary for OzFoodNet sites, 2004, *continued*

State	Month of outbreak	Setting prepared	Agent category	Number affected	Hospitalised	Evidence*	Epidemiolgical study†	Food vehicle
Qld, *cont*	June	Restaurant	Unknown	25	0	D	C	Buffet meal with cold salad
	June	Takeaway	Ciguatoxin	4	0	D	D	Grey mackerel
	June	Private residence	Ciguatoxin	3	0	D	D	Trevally
	July	Commercial caterer	Norovirus	26	0	D	CCS	Unknown
	July	Bakery	*Salmonella* Typhimurium 135a	5	5	D	D	Custard fruit tarts
	July	Private residence	Ciguatoxin	4	0	D	D	Grey mackerel
	August	National franchised fast food	Norovirus	7	0	D	D	Pizza
	August	National franchised fast food	*Clostridium perfringens*	6	0	M	D	Meat pizza
	August	Camp	Salmonella Virchow 8	5	0	D	D	Unknown
	September	Private residence	*Salmonella* Enteritidis 26	17	0	D	C	Unknown
	September	Restaurant	Norovirus	16	0	D	D	Unknown
	September	Takeaway	*Campylobacter*	2	0	D	D	Chicken kebab
	October	Private residence	Norovirus	13	1	D	CCS	Unknown
	October	Commercial caterer	Unknown	8	1	D	D	Unknown
	October	Contaminated primary produce	Norovirus	2	Unknown	D	D	Oysters (frozen)
SA	January	Private residence	*Salmonella* Saintpaul	4	0	A	C	Boiled eggs
	March	Bakery	*Salmonella* Typhimurium 108	13	5	AM	CCS	Cream cakes
	April	Restaurant	*Salmonella* Typhimurium 108	9	2	D	C	Unknown
	April	Private residence	*Salmonella* Typhimurium 108	8	Unknown	A	C	Lemon meringue & potato bake
	June	Restaurant	Unknown	9	Unknown	D	D	Unknown
	June	Takeaway	*Salmonella* Typhimurium 35	3	Unknown	D	D	Unknown
	July	Private residence	*Salmonella* Typhimurium 9	4	0	D	D	Home made icecream
	July	Restaurant	Unknown	4	0	D	C	Unknown
	July	Restaurant	*Campylobacter*	4	1	D	CCS	Unknown
	August	Restaurant	Unknown	8	0	D	C	Unknown
	September	National franchised fast food	Unknown	15	0	D	C	Pizza
	September	Hospital	Listeriosis	2	2	D	D	Unknown
	November	Institution	Norovirus II	9	0	D	C	Unknown
	December	Restaurant	Norovirus II	36	0	D	C	Unknown
	December	Aged care	*Salmonella* Typhimurium 126 var	13	0	D	D	Unknown
	December	Unknown	*Salmonella* Typhimurium 8	9	0	D	D	Unknown
	December	Private residence	*Salmonella* Typhimurium 126 var	3	0	D	D	Unknown

State	Month of outbreak	Setting prepared	Agent category	Number affected	Hospitalised	Evidence*	Epidemiolgical study†	Food vehicle
Tas	May	Bakery	Norovirus	57	0	D	C	Bakery products
Vic	January	Restaurant	*Salmonella* Typhimurium 9	90	7	M	D	Pizza, risotto and pasta
	February	Hospital	Suspected toxin	14	Unknown	D	D	Unknown
	February	Contaminated primary produce	*Salmonella* Typhimurium 126	11	5	D	D	Suspected eggs
	March	Restaurant	Suspected toxin	16	0	A	C	Suspected spaghetti bolognaise
	April	Unknown	*Salmonella* Typhimurium 9	9	2	D	D	Unknown
	May	Commercial caterer	*Salmonella* Typhimurium 12a	28	3	AM	C	Gourmet rolls/red onion
	May	Hospital	Suspected toxin	21	Unknown	D	D	Unknown
	May	Cafe	*Salmonella* Typhimurium 9	8	1	D	D	Suspected sauce - raw eggs
	June	Aged care	*Clostridium perfringens*	22	1	D	D	Unknown
	June	Aged care	Suspected toxin	9	1	D	D	Unknown
	July	Commercial caterer	*Salmonella* Stanley	33	4	D	C	Unknown
	July	Commercial caterer	*Salmonella* Typhimurium 126	21	1	D	C	Unknown
	August	Restaurant	Suspected toxin	45	0	D	C	Unknown
	September	Aged care	*Campylobacter*	24	2	D	D	Suspected barbecue
	October	Commercial caterer	Unknown	33	4	D	C	Unknown
	November	Commercial caterer	Suspected toxin	20	0	A	C	Chicken vol au vents
	November	Commercial caterer	Suspected toxin	9	0	D	D	“Butterfish” (rudderfish)
	November	Contaminated primary produce	Unknown	7	3	D	D	Redfin
	December	Private residence/takeaway	Suspected toxin	75	1	D	C	Unknown
	December	Commercial caterer	*Salmonella* Typhimurium 170	48	2	D	C	Unknown
	December	Aged care	*Campylobacter*	7	0	D	D	Suspected drinking water
WA	January	Commercial caterer	Norovirus	19	0	A	C	Prawns and cold meats
	April	Commercial caterer	*Clostridium perfringens*	100	0	M	D	Pasta meat sauce
Total				2,076	116			

* A=analytical epidemiological evidence; D=descriptive evidence: M=microbiological evidence.

† C=cohort study; CCS=case control study; D=descriptive study; N=individual patient data not collected.

RDNC = ‘Reactive but Does Not Conform’ and represents phage type patterns that are not yet assigned.

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A cluster of cases of haemolytic uraemic syndrome in north Queensland associated with a novel Shiga-like toxin-producing *Escherichia coli*

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Introduction

The Tropical Public Health Unit (TPHU) in Townsville was alerted on 16 March 2004 to the possibility of a cluster of cases of haemolytic uraemic syndrome (HUS). This followed the admission of an 18-month-old female from a small regional hospital to a hospital in Townsville. At the time of this child's admission, a report was also made (from the same hospital in Townsville) of another case of HUS, from the same small regional town, two months previously. Very few details were initially available on the previous case.

The public health responses comprised epidemiological and environmental investigations. The implications of the delay in notification of the previous case (hereafter referred to as the first case) on these investigations are discussed. In addition, this report describes the organism causing the HUS as a type of *Escherichia coli* not previously known to elaborate Shiga-like toxin (SLT) in human populations.

Method

Initial investigations by the TPHU included a follow-up of both cases. Identification of the first case was made through interviews with staff of the regional hospital, then following the clinical referral pathway to locate the case. After identification and location of the first case (a 10-year-old male), telephone interviews were conducted with the parents of both children using a standard questionnaire. The questionnaire included questions on symptoms, details of hospitalisation, attendance at school and educational facilities, identification of unwell siblings, travel, environmental exposures, and a food history for 10 days preceding the onset of the illness. In addition, the case report required information on laboratory criteria for the diagnosis of HUS as well as information on clinical presentations.

On identification of the original case and completion of a case report, it was noted that an incomplete examination of faecal samples had been undertaken during the original admission. Therefore, repeat samples from the case were collected, with a specific request for examination for Shiga-like toxins and SLT-producing organisms. At the same time, identification of a recent dysenteric illness in a sibling of the first case was noted, and faecal samples were also collected for this child.

A site visit was made to a remote cattle property, identified as a possible source of infection in the first case. Environmental samples (water, cattle faeces, cattle feed) were taken and referred to the Queensland Health Scientific Services, Brisbane, for testing.

A face-to-face meeting was arranged between the mothers of both cases in order to facilitate identification of any links between the two families. At this point the working hypothesis, based on interviews conducted, was that the first case had acquired his infection on the cattle station visited, with person-to-person transmission leading to the second case.

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Results

Epidemiological investigation

Interviews and case report questionnaires were carried out with the two families involved. The first case of HUS, the 10-year-old male, became unwell on 28 January 2004 and the second, the 18-month-old female, on 9 March 2004. Faecal samples taken at the time of admission of the second case were positive for a Shiga-like toxin in both faeces and culture, and an entero-haemorrhagic *E. coli* (EHEC), or Shiga-like toxin producing *E. coli* (STEC), 086:H27 was eventually identified.

Faecal samples taken from the first case during his admission were not specifically tested for the presence of a SLT, with an infection with *Salmonella* the only organism identified. Repeat samples, taken two months after the onset of the illness, were positive for the same SLT (*stx*-2) and the same STEC (*E. coli* 086:H27) as in the 18-month-old child.

In addition, from the histories taken it was noted the 5-year-old sibling of the first case had had a dysenteric illness (consistent with a haemorrhagic colitis) in February 2004, and had been admitted to another Queensland Hospital. Unlike his sibling, he did not develop HUS; he also did not have a complete faecal examination at the time of his admission. A repeat faecal sample collected from this child in late March was SLT (*stx*-2) positive and *E. coli* 086:H27 was isolated.

Similarly, the 18-month-old child with HUS had a 6-year-old sibling who had a recent vague history of abdominal pain. A faecal sample from this child was also SLT (stx-2) and *E. coli* 086:H27 positive.

Food histories taken from all family members did not identify any obvious potentially contaminated food items, and did not reveal any specific food items common to the two families. The first family affected had spent most of the month before the onset of illness in the first case at a remote cattle station in North Queensland. This stay involved working on a daily basis in close contact with cattle on the property, and included drinking untreated water from the property's supply. The second family had no link with the property and did not report close contact with any domestic animals.

However the face-to-face meeting between parents of both families revealed a common link between the families. The two siblings (who had tested positive for the SLT and *E. coli* 086:H27) of the HUS cases both attended the same class in the same pre-school.

Following these investigations, the working hypothesis was further refined. It appeared likely that the STEC had been acquired at the remote cattle station by the first HUS case, who then transmitted it during household contact to his sibling (who in turn developed a dysenteric illness). Transmission then probably occurred to the second family through the pre-school contact, then through household contact on to the 18-month-old (who developed HUS) from her sibling.

Environmental sampling

A visit to the remote cattle station, where the first case's family had spent most of the month preceding his illness, was undertaken by the TPHU team. Interviews were conducted with the station owner, and his family, in an attempt to identify particular high risk activities (for example, contact with cattle, drinking untreated water) undertaken by the first case during his stay, and to identify any other illness in contacts. Environmental samples were collected for testing from areas on the property where the first case had either spent some time or engaged in potentially risky activities.

No other individuals with a recent enteric illness of any sort were identified on the station. Because of his contact with cattle at the property, faecal samples were collected from the father of the first case; which was positive for a SLT (*stx*-1), but not the same STEC type as seen in the cases.

Samples taken from a number of water supplies (bore water at cattle yard, house kitchen, and shed bore supply) were negative for the SLT and the organism under investigation. In addition, samples of cattle faeces, chicken and duck faeces, cattle feed (molasses and coconut husk meal) were all also negative for the SLT and organism under investigation. One of the cattle had SLT-1 and, like the father, was eliminated from this investigation.

Discussion

An association between infection with Shiga-like producing *Escherichia coli* (so named because of their similarity to toxins produced by *Shigella*) and the post diarrhoeal haemolytic uraemic syndrome (HUS) was first described in 1983.[1] HUS is defined as a clinical syndrome made up of acute renal injury, thrombocytopenia and microangiopathic haemolytic anaemia.[1,2] The organism most commonly associated with this illness is *E. coli* 0157.[1] The epidemiology of *E. coli* O157 is now well described, although the pathophysiology of HUS is less well understood.[2] Since the initial descriptions of *E. coli* O157, other serotypes of Shiga-like toxin producing *E. coli* (STEC) have been noted to cause similar disease in humans.[3]

Infections with STEC have been described associated with the ingestion of both food and water contaminated with the organisms. Contaminated meat, particularly ground beef used in hamburgers, has resulted in multiple outbreaks of disease.[2] In South Australia in 1995, a large outbreak of HUS was linked to the consumption of contaminated sausage.[4] In this outbreak, the organism concerned was *E. coli* 0111:NM and a total of 23 cases of HUS, and 30 cases of haemorrhagic colitis, were described. Of the 23 cases of HUS one child died. A recent study in South Australia, in contrast, noted that ingestion of berries was a significant risk factor in the development of sporadic STEC infection.[5] Similarly, outbreaks have been described in relation to contaminated fresh produce including, for example, radish sprouts, lettuce, alfalfa sprouts, and unpasteurised apple juice.[1]

Reservoirs of infection in animals, particularly in cattle, have also been well described.[1] In Australia; up to 118 serotypes of STEC have been found in healthy cattle[6] though the importance of the diversity of serotypes described, in terms of human disease, is not clear.

Outbreaks of STEC infections following exposure to buildings contaminated with the organism after an agricultural show and following exposures in petting zoos, highlight the importance of these animal reservoirs as a source of infection.[7] In the United Kingdom a strong association between sporadic infections with STEC and contact with a farm environment has been demonstrated.[8]

In the cases detailed above, infection with a novel EHEC/STEC (O86:H27) is described. This is the first report of an O86 serotype producing SLT in humans (personal communication, Denise Murphy). In the four cases with proven STEC (with the serotype O86:H27) infection, two developed HUS, the most serious complication of such an infection (STEC are estimated to result in HUS in 5–8% of cases[2]). Whether this high rate of disease is the result of enhanced virulence of the organism, or a consequence of undetected mild or asymptomatic cases, is not clear.

Despite the negative findings of the environmental sampling, it remains likely that infection with STEC in the first case was the result of exposure to the organism on the cattle property visited, with person-to-person transmission leading to subsequent cases. The rate of person-to-person transmission of STEC is likely to be high, as only low numbers of the organism are required to cause disease.[2] The majority of family contacts of cases of HUS, for example, have evidence of infection with STEC as indicated by antibodies to the toxin.[2]

The delay in the notification of the first case of HUS meant that there was a delay in the public health response. These delays were the result of incomplete microbiological investigations of the first case's faecal sample, and the lack of awareness among the case's clinicians that HUS is a notifiable disease in Queensland. The *Salmonella* Chester, a non Shiga-like toxin-producing organism, isolated from the first case was almost certainly an incidental finding and in the context of HUS, should have raised the suspicion of a concurrent undetected infection with a SLT-producing organism.[9] If the first case had been promptly notified it is possible that further person-to-person transmission of the implicated STEC might have been prevented.

Acknowledgements

Environmental Health officers from the Tropical Public Health Unit were involved in sample collections.

Dr Karl Bettelheim from the Microbiological Diagnostic Unit, Melbourne, undertook the confirmation of the O serotype and H type.

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A report from the Communicable Diseases Network Australia January – March 2005

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.

Face-to-face meetings

In mid-March the full CDNA met in Adelaide, along with the CDNA Jurisdictional Executive Group (JEG). Further details of the meetings will be reported to *Communicable Diseases Intelligence* as outcomes are achieved. The major discussion points of these meetings were:

- revision of National Smallpox Guidelines;
- revision of the National Norovirus Guidelines;
- planning for the Communicable Diseases Control Conference 2-3 May 2005;
- Business Rules are being developed and will result in protocols for all aspects of the CDNA and its subcommittees (including appointment of the CDNA chair and making diseases nationally notifiable); and
- a workshop addressing the protocols around communicable diseases exposure on airlines and contact tracing to be conducted in Canberra in April 2005 was discussed.

These face-to-face meetings also provided an opportunity to conduct a joint meeting between the CDNA JEG and the National Immunisation Committee (a CDNA sub-committee). Outcomes of this meeting will be detailed in forthcoming reports to *Communicable Diseases Intelligence*. In summary the joint CDNA JEG/NIC meeting resolved to:

- re-establish the Measles Elimination Advisory Committee; and
- conduct varicella surveillance in support of the Australian Government's new varicella immunisation program.

Improving Indigenous Identifiers in Communicable Disease Reporting Project

The Improving Indigenous Identifiers in Communicable Disease Reporting Project (IIICDRP) report was finalised in late 2004 and is the result of over four years collaboration between the Australian Government Department of Health and Ageing (DoHA), the IIICDRP Steering Committee and various stakeholders. CDNA established a working group to consider implications of the recommendations of the IIICDRP report.

Introduction of new reporting format

A new reporting format, developed by the Surveillance Section of DoHA, was introduced to CDNA teleconferences. The new proforma is divided into two parts:

- disease specific tables (influenza notifications, meningococcal notifications, serogroup C meningococcal notifications, pertussis notifications,

vaccination status of pertussis notifications in children <1 year of age, vaccination status of pertussis notifications in children <1 year of age by state and territory, and measles notifications); and

- a table of all notifiable diseases, by state and territory, for the current reporting period.

State and territories will provide additional information to supplement the national report.

CDNA representatives to Australian Technical Advisory Group on Immunisation Rotavirus Working Party

In late 2004 the Australian Technical Advisory Group on Immunisation agreed to form the Rotavirus Working Party to review and report on the available data on the epidemiology and disease burden of rotaviral infections in Australia. Three members of CDNA will participate on the working party.

Advice on incoming refugees

In considering the public health implications for resettled refugees, CDNA sought and obtained a commitment from the Australian Government Department of Immigration and Indigenous Affairs to ensure notification of incoming refugees be provided to all jurisdictions prior to arrival. Details to be provided include country and camp of origin. This information will be useful in determining possible health management requirements of incoming refugees.

Communicable Diseases Network Australia: *Guidelines for the Control of Pertussis in Australia* (November 1997)

Advice to health professionals, April 2005

The Communicable Diseases Network Australia wishes to advise that the information contained in the publication *Guidelines for the Control of Pertussis in Australia* (November 1997) is no longer current.

Health practitioners are to consult the Australian Immunisation Handbook (section 3.16) for current information on the public health management of pertussis. The Handbook can be accessed on-line at http://www1.health.gov.au/immhandbook/

OzFoodNet: enhancing foodborne disease surveillance across Australia: Quarterly report, January to March 2005

Introduction

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

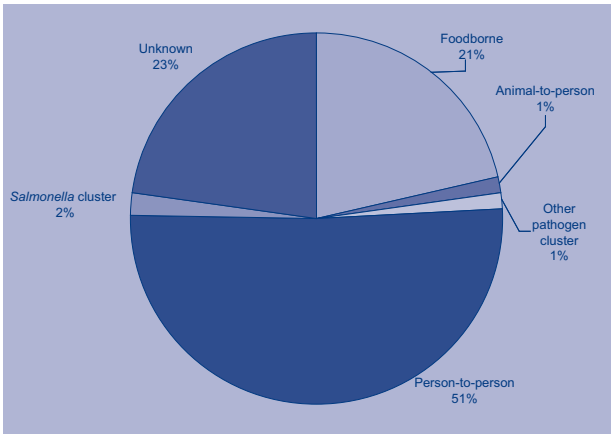
This report summarises the occurrence of foodborne disease outbreaks and cluster investigations between January and March 2005. Data were received from OzFoodNet representatives in all Australian states and territories and a sentinel site in the Hunter region of New South Wales. The data in this report are provisional and subject to change, as results of outbreak investigations can take months to finalise. 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 collected data used in this report.

Foodborne disease outbreaks

During the first quarter of 2005, OzFoodNet sites reported 145 outbreaks of foodborne or enteric illness. In total, these outbreaks affected 2,446 people and hospitalised 77 persons. Three deaths were reported. All three of the deaths occurred in aged care facilities. Two were associated with outbreaks of norovirus infection and one with *Salmonella* infection. As usual, the majority (51%, n=74) of outbreaks resulted from infections spread from person-to-person (Figure).

There were 31 outbreaks of illness where contaminated food was suspected or proven to be the primary mode of transmission. This compares with 24 outbreaks for the first quarter of the previous year and 25 outbreaks in the fourth quarter of 2004. *Salmonella* Typhimurium was the causative agent for seven outbreaks, while Ciguatera toxin was responsible for three outbreaks and *Campylobacter* for two. Of the remaining outbreaks, one each was caused by *Clostridium perfringens*, *Salmonella* Enteritidis 26 var, *Salmonella* Hessarek, *Salmonella* Saintpaul, and suspected scombroid poisoning. In one outbreak caused by *Salmonella*, there were multiple serotypes isolated from patients including: Chester, Muenchen and Subspecies 3b. An aetiological agent was not identified in 42 per cent (13/31) of the outbreaks.

Figure. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, January to March 2005



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

Ten of the outbreaks were associated with meals prepared in restaurants and another seven with food prepared in private residences. Five occurred in aged-care facilities and four were associated with food prepared by commercial caterers. Two outbreaks involved food prepared in takeaway food outlets. Single outbreaks occurred at a school camp, an institution and an undefined setting. Thirteen of the outbreaks occurred in January, six in February and 10 in March.

To investigate these outbreaks, sites conducted six cohort studies and two case control studies. For 21 outbreaks, only descriptive data were collected and in two outbreaks no individual case data was collected. In eight outbreaks, investigators obtained microbiological evidence linking a food vehicle to illness, and analytical epidemiological evidence in five outbreaks. For the remaining outbreaks, investigators obtained descriptive epidemiological evidence implicating the food vehicle or suggesting foodborne transmission.

In New South Wales there were eight outbreaks of foodborne illness reported during the quarter. One outbreak of *Campylobacter jejuni* in an institution affected two residents on a diet of pureed foods, although no food vehicle was identified. A second outbreak of *Salmonella* Saintpaul in a private residence affecting two people was suspected to be caused by cross contamination of salad by raw chicken. In the other six outbreaks no causative agent was identified. Four of these involved restaurants and two of the four followed the consumption of chicken. The suspected food vehicle was not identified in the other two outbreaks. These restaurant-related outbreaks affected between two and seven people each. The other two outbreaks of unknown aetiology involved a beef casserole prepared by a commercial caterer (13 cases) and food from a takeaway store (3 cases).

Victoria reported 14 outbreaks of foodborne disease, five of which were associated with different phage types of *Salmonella* Typhimurium. There was one large outbreak of *Salmonella* Typhimurium 197 at a Turkish restaurant. *S.* Typhimurium 197 was isolated from dips served at the restaurant. The results of this investigation are still being finalised. An outbreak of *S.* Typhimurium 12 following a barbeque at a private residence resulted in 15 people being ill. The specific food vehicle causing the outbreak was not identified.

Victoria reported two outbreaks of *S.* Typhimurium 9, one of which was associated with food prepared in a restaurant and the other was associated with a takeaway outlet. The restaurant outbreak affecting 13 people was caused by hollandaise sauce made with raw eggs. *S.* Typhimurium 9 was cultured from a sample of leftover hollandaise sauce. Hollandaise sauce at this restaurant also resulted in an outbreak of *S.* Typhimurium 9 one year earlier in May 2004. Trace back of the eggs revealed that the eggs were supplied by the same company for both outbreaks. The second Victorian outbreak of *S.* Typhimurium 9 was also caused by eggs from this company. In this outbreak affecting 14 people participating in a children's cooking class run by a commercial caterer, investigations implicated chocolate mousse made with raw eggs. Environmental investigations were undertaken on the farm supplying eggs and *Salmonella* Typhimurium 9 was cultured from a wash sample of dirty eggs.

Eggs were also suspected to be the cause of outbreaks of *S.* Enteritidis 26 var in an aged care facility and *S.* Typhimurium 126 var 4 in a private residence. The first affected seven residents and the eggs were traced back to a producer who had recently isolated *S.* Enteritidis 26 from one of their farms. In the second outbreak five people were ill after consuming chocolate mousse containing *S.* Typhimurium 126 var 4, although it was not possible to trace back the supply of eggs to a specific farm.

Victoria reported that 30 children and seven staff were ill with campylobacteriosis following a school camp. *Campylobacter* was isolated from the drinking water supply at the camp, although children also drank unpasturised milk and had contact with farm animals.

There were seven outbreaks in Victoria in which no causative agent was identified. Two of these occurred in aged care facilities and affected 30 and 11 residents respectively. Both were suspected to be caused by *Clostridium perfringens*. Three of the outbreaks were associated with commercial caterers, one with a restaurant and one with a takeaway outlet. Food vehicles associated with these outbreaks of unknown aetiology included chicken vol-au-vents, veal rolls, red curry, seafood platter, baked fish and hommus dip.

Queensland reported six outbreaks of foodborne illness, three of which were due to ciguatera fish poisoning. The fish responsible for the outbreaks of ciguatera were mackerel, black trevally and yellowtail kingfish. An outbreak of *Salmonella* Typhimurium 12 affected 10 people, although no food vehicle was identified. Thirty-six people were ill in an outbreak of *Clostridium perfringens* after a meal of braised steak and gravy at an aged care facility. *C. perfringens* was isolated from both the food eaten and residents' stools. In a second outbreak in an aged care facility, eight residents were ill with salmonellosis thought to be caused by a contaminated rainwater tank. Patients were infected with multiple serotypes of *Salmonella*, including Muenchen, Chester, and Subspecies 3b

over a three month period. Subspecies 3b was isolated from water from the facility's rainwater tank. The tank water was also heavily contaminated with *E. coli*. The source of the pathogens is unknown but may have been due to amphibians or reptiles.

South Australia reported an outbreak of *S.* Typhimurium 9 affecting 13 people following a restaurant meal. Shallot pancakes were the only food with a high, but non-significant, association with gastroenteritis.

Tasmania reported one outbreak of suspected scombroid poisoning in two people, thought to be due to yellowfin tuna in a nicoise salad served at a restaurant.

The Australian Capital Territory reported an outbreak of *S.* Hessarek caused by contaminated free range eggs. The eggs were served at a restaurant as poached eggs and as hollandaise sauce.

Comments

During the first quarter of 2005, there were five outbreaks suspected to be caused by contaminated eggs, which is a continuing concern for Australia.[1] Two of these outbreaks were related to hollandaise sauce prepared in restaurants in two different states. These outbreaks clearly highlight the importance of restaurants using pasteurised eggs in sauces and desserts.[2] It is a concern that one of the egg associated outbreaks during the quarter was an outbreak of *S.* Enteritidis in a Victorian aged care facility. Australia does not have *S.* Enteritidis endemic in egg laying flocks, so it is vital that public health agencies investigate infected patients, as they may represent sentinels for new emerging sources of this pathogen.[3]

During January to March 2005, there was an increase in *S.* Typhimurium 197 cases in New South Wales that occurred among people of Middle Eastern ethnicity. While this community-wide increase in infections occurred at the same time as the large outbreak in Melbourne, there was no apparent connection between the two outbreaks. The number of cases of *S.* Typhimurium 170/108 was also markedly increased in New South Wales during the first quarter of 2005. New South Wales investigated the increases of both of these phage types of *S.* Typhimurium using a case control study methodology exploring hypotheses developed during interviews of a series of infected patients. The hypotheses included consumption of poultry and red meats, although results of these studies were not finalised at the time of this report and are not reported in the summary of outbreaks above.

During the quarter, there were two outbreaks potentially associated with drinking water. The first was associated with an outbreak of campylobacteriosis on a school visit to a farm and the second with a prolonged outbreak of salmonellosis at an aged care facility. The aged care facility used rainwater tanks as the source of drinking water. The tanks may have been contaminated by animals on roof catchment areas. This outbreak and previous microbiological surveys illustrate that tanks may be unsuitable as a source of drinking water for aged care facilities.[4] Rainwater tanks have been implicated as a source of disease previously and they are difficult to clean and maintain.[5,6] Rainwater tanks may be a more common cause of disease than previously recognised.

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Table. Outbreaks of foodborne disease reported by OzFoodNet sites,* January to March 2005

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence	Responsible vehicle
ACT	Mar	Restaurant	*Salmonella* Hessarek	5	A	Hollandaise sauce
NSW	Jan	Restaurant	Unknown	2	D	Unknown
	Mar	Restaurant	Unknown	5	D	Chicken and fish green curry dishes
	Mar	Restaurant	Unknown	3	D	Chicken Caesar salad and chicken burger
	Mar	Caterer	Unknown	13	A	Beef casserole
	Jan	Home	*Campylobacter jejuni*	2	M	Unknown
	Feb	Institution	*Salmonella* Saintpaul	2	M	Unknown
	Mar	Restaurant	Unknown	7	D	Suspect vegetable filled naan or rice
	Mar	Takeaway	Unknown	3	D	Unknown
Qld	Jan	Home	Ciguatoxin	4	D	Mackerel
	Jan	Home	Ciguatoxin	2	D	Black trevally
	Jan	Aged care	*Clostridium perfringens*	36	M	Braised steak and gravy
	Feb	Other	*Salmonella* Typhimurium 12	10	D	Unknown
	Mar	Home	Ciguatoxin	2	D	Yellowtail kingfish
	Jan	n/a	*Salmonella* Chester, *Salmonella* Muenchen, *Salmonella* Subspecies 3b	8	M	Contaminated rainwater tank
SA	Feb	Restaurant	*Salmonella* Typhimirium 9	13	D	Unknown
Tas	Feb	Restaurant	Suspected scombroid poisoning	2	D	Yellowfin tuna
Vic	Jan	Home	Unknown	10	D	Unknown
	Jan	Aged care	*Salmonella* Enteritidis 26 var	7	D	Suspect eggs
	Jan	Aged care	Unknown	30	D	Unknown
	Jan	Home	*S.* Typhimurium 126 var 4	5	D	Suspect eggs
	Jan	Caterer	Unknown	29	A	Chicken vol-au-vents
	Jan	Restaurant	*Salmonella* Typhimurium 197	Pending	M	Dips
	Jan	Caterer	Unknown	40	A	Veal rolls/red curry
	Feb	Camp	*Campylobacter*	22	M	Suspected water
	Feb	Restaurant	Unknown	16	A	Seafood platter/baked fish/octopus
	Mar	Home	*Salmonella* Typhimurium 12	15	D	Unknown
	Mar	Takeaway	Unknown	6	M	Hommus dip
	Mar	Restaurant	*Salmonella* Typhimurium 9	13	M	Hollandaise sauce
	Mar	Aged care	Unknown	11	D	Unknown
	Mar	Caterer	*Salmonella* Typhimurium 9	14	D	Chocolate mousse

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.

* No foodborne outbreaks reported from Western Australia or the Northern Territory during the quarter.

Defining the risk of human exposure to Australian bat lyssavirus through potential non-bat animal infection

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Introduction

Human infection with Australian bat lyssavirus (ABLV) was first reported in November 1996, six months after the first identification of the virus in a flying fox in May 1996.[1,2,3] Only two human cases of ABLV infection have been described to date, although hundreds of potential human exposures to ABLV have been reported.[4,5] No cases of ABLV infection in other Australian mammals have been reported, although a number of animal exposures to ABLV positive bats have been investigated. The CSIRO Australian Animal Health Laboratories (AAHL) have undertaken some investigation into the effects of exposure to ABLV in terrestrial species, although this has been limited to preliminary studies in dogs and cats.[6] Experimentally infected animals showed mild transient behavioural changes within 2–3 weeks of exposure and seroconversion to ABLV within three months.[7] The natural end point of ABLV infection in dogs and cats is unknown.

Current public health guidance considers the risk of transmission of ABLV from a dog or cat to a person is very low.[6] Furthermore, owners are advised that although the animal has a remote possibility of being infected with ABLV, it should be observed closely for at least three months and to report any behavioural changes that occur.[6] We present two cases where the behaviour of dogs after potential exposure to ABLV posed significant questions for veterinary and public health authorities.

Case 1

On 16 January 2004, the Brisbane Southside Public Health Unit (BSPHU) was notified of an ABLV positive result on a black flying fox (*Pteropus alecto*). BSPHU investigations revealed that the bat was actually found on 2 January 2004, when the family pet, a 2-year-old desexed female Alsatian-Kelpie cross was observed barking at the bat on the ground and brought the bat to the owner's attention. The owner did not witness any direct contact between the dog and the bat. Initial reports (16 January) suggested that there were marks on the bat body that may have been attributable to attack by the dog. However, subsequent post mortem examination (19 January) found small multiple holes on both wing membranes (Figure), and no lacerations on the body or head that were consistent with dog bites.

Figure. Multiple small holes on patagia of bat in Case 1

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Seven days after the dog's potential exposure (9 January), the dog was relocated to the home of another family member. Up until 11 January the dog behaved normally. However, after that time the dog became more aggressive and bit the owner on 14 January and the owner's partner on 15 January. Although provocation could not be ruled out in one attack, all family members attested to the behavioural change in the dog, some 9 to 11 days after possible exposure to an ABLV positive flying fox. By 19 January 2004, the dog was reported to be well with no signs of persistent aggression. Consultation with national public health and veterinary authorities determined that the possibility of the dog being infectious with ABLV during the time of the attacks on humans could not be excluded and post exposure prophylaxis was provided to the owner and partner.

The dog was placed into quarantine in a secure, purpose-built facility at the Department of Primary Industries and Fisheries Animal Research Institute at Yeerongpilly, Brisbane. The dog's general health and behaviour were monitored daily, salivary samples were collected twice weekly, and serial blood samples were collected fortnightly over the six month quarantine period. The saliva swabs were subject to quantitative real time polymerase chain reaction (PCR) (Taqman™) analysis at Public Health Virology, Queensland Health Scientific Services (QHSS). Sera were forwarded to AAHL where they were screened for antibodies to ABLV by rapid fluorescent focus inhibition test. No salivary swabs were positive for ABLV antigen on PCR and no antibody titre to ABLV was detected. The dog's health and behaviour remained normal. At the end of the quarantine period, it was released to the owners.

Case 2

On 1 April 2004, the BSPHU was notified of a dog attack on a young child. The child had been attacked by one of two family pet dogs, an 18-month-old Rhodesian Ridgeback. In late January 2004, the bloodied remains of a bat had been found on the dog's bed outside the family home. The bat was not submitted for analysis. On 18 March, following a storm, the apparently well and unprovoked dog savagely attacked the young family member who required extensive surgery following the attack. The dog was previously well-behaved with no history of aggression according to current and previous owners and had been in the family for five months with no behavioural problems. The dog was taken to the local dog pound and destroyed. No comments were recorded about the physical condition of the dog and the remains were unavailable for examination. The veterinarian was informed of the possible exposure to the bat in January but noted the current Commonwealth advice that dogs were unlikely to be infected with ABLV. Subsequent contact by the family with Department of Primary Industry researchers led to the notification to the BSPHU.

After discussion with national public health authorities it was decided to offer the child a course of post exposure rabies vaccination including rabies immunoglobulin.

Discussion

Bats are the putative natural host of ABLV. Natural infections have been recorded in both megachiropteran (flying fox) and microchiropteran (insectivorous bat) species.[5] While the crude prevalence of ABLV infection, as determined by direct fluorescent antibody test on fresh brain impression smear, in free-living flying foxes is less than one per cent, in sick and injured flying foxes it is 6.5 per cent.[5] This positive association is a reflection of the clinical disease attributable to ABLV infection in bats. Because infected bats are debilitated, and typically found on the ground or roosting close to the ground, they have the highest probability of contact with concerned humans and inquisitive companion animals. Despite the absence of any recorded ABLV infection in terrestrial carnivores, a decision was made to quarantine and monitor the Case 1 dog based on its possible direct contact with an ABLV positive bat and the similarity between her reported behavioural change 9–11 days post-putative exposure and the transient behavioural change reported in experimentally infected dogs 2–3 weeks post inoculation.[7]

The absence of ABLV transmission in species other than bats is consistent with the view that lyssaviral infections are host-specific.[8] However, the two human cases tragically demonstrate that ABLV can infect non-bat species. While there is no historic or contemporary evidence of ABLV infection in dogs or cats, animal health authorities in Australia recognise at least a theoretical possibility of infection in these species, and recommend testing bats known or suspected to have had at-risk contact with companion animals.[6] In the last five years, 144 bats (115 with putative dog contact and 29 with putative cat contact) have been screened by direct fluorescent antibody test at the Animal Research Institute or QHSS. Of these, five bats with putative dog contact have tested positive. That is, five dogs have had a high risk of exposure to ABLV. Consistent with the risk minimisation approach, the Department of Primary Industries and Fisheries advised the owners of each of these dogs to place them under a 90 day home quarantine, in line with AUSVETPLAN recommendations.[9] None of the owners reported symptoms in their dogs consistent with ABLV infection, however

interpretation of these negative findings is limited by the small sample size (n= 5) and by the lack of certainty of exposure of any of the dogs.

Experimental ABLV infections in dogs undertaken at AAHL produced transient behavioural changes 2–3 weeks post-innoculation, but no observed clinical disease.[7] However, while the experimental studies appear to support the finding of absence of disease in dogs, interpretation of the experimental findings should be made with care. The study had several limitations: small numbers of young (possibly immunologically immature) animals were used; the inoculum was laboratory passaged and possibly attenuated; animals were observed for only three months post-innoculation and attempts to recover virus were limited to a single cell culture passage. Although virus could not be recovered from the brain of any of the ABLV challenged animals, attempts to isolate rabies virus from a control experimentally infected cat were also unsuccessful. However, the observed behavioural changes and the detection of anti-ABLV antibody in the cerebrospinal fluid of two of the inoculated dogs and one cat suggests infection reached the central nervous system. Thus there is some evidence to support the possibility of sub-clinical or mild clinical ABLV infection in dogs under experimental conditions.

While historic data on cross-species lyssaviral infection is limited (arguably due to the lack of availability of molecular techniques), there have been a number of recent reports of 'spill-over' of bat-variant rabies and European bat lyssavirus to terrestrial species.[10–16] Even more disturbing is the recent report of an outbreak of bat-variant rabies in skunks.[12,17] It is evident that lyssaviruses show a strong evolutionary association with bats – genotypes 2, 4, 5, 6 and 7 solely or predominantly infect bats and genotype 1 infects terrestrial carnivores and bats (the reservoir of genotype 3 is unknown). Further, it is now argued on the basis of molecular evidence that genotype 1 (terrestrial rabies) historically evolved from bat lyssaviruses.[18]

The case studies presented above highlight the uncertainty that still exists about the potential for ABLV to be transmitted to other non-human mammalian hosts. The available research did not satisfactorily resolve all the questions that were raised about the risk of bat-dog-human transmission in these two scenarios. Consequently, after extensive consultation, public health authorities recommended rabies post exposure prophylaxis to the affected persons. From a public health perspective, further studies in Australian domestic and wild carnivore species are necessary to more conclusively demonstrate the ability of ABLV to infect these species. This will enable public health officials to make more confident assessment of the risk of human ABLV infection associated with a bite from an exposed dog or cat.

Acknowledgments

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Communicable diseases surveillance

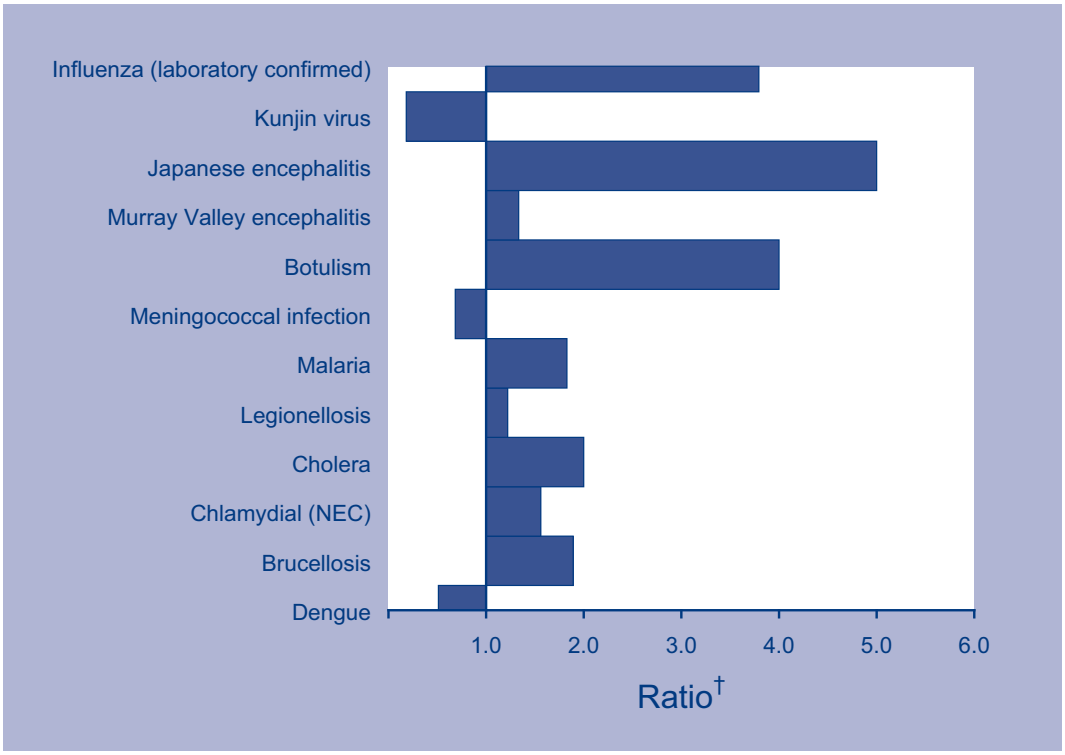
Highlights for 1st quarter, 2005

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 disease notifications with an onset in the first quarter of 2004 compared with a 5-year mean for the same period. The number of notifications received in the quarter was above the five year mean for influenza (laboratory-confirmed), Japanese encephalitis virus, Murray Valley encephalitis virus, botulism, malaria, legionellosis, cholera, chlamydial infection and brucellosis. The number of notifications received was below the five year mean for meningococcal infections, Kunjin virus and dengue.

Figure 1. Selected* diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 January to 31 March 2005 with historical data*



* Selected diseases are chosen each quarter according to current activity.

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

‡ Notifications above or below the 5-year mean plus or minus two standard deviations for the same period.

Gastrointestinal illnesses

Botulism

One case of botulism was reported in this quarter. The case was in a 4-month-old male in Queensland, who lived on a rural property with his parents and two siblings. The infant had only been breast-fed and did not have direct exposure to honey, a common risk factor for this disease as it can contain *Clostridium botulinum* spores.[1,2] Stool samples from the case tested positive for botulinum toxin A.

Quarantinable diseases

Cholera

Two cases of cholera were reported by Victoria in this quarter. The cases were in a 71-year-old female and a 45-year-old female, where both were infected with *Vibrio cholerae inaba*. The cases were in recent Liberian refugees who travelled from a camp in Tanzania. The cases were symptomatic in-flight and were hospitalised soon after arrival. Active surveillance for other cases was instigated by the Victorian health department, and factsheets were distributed to passengers on the same flight.

The cases were notified to the World Health Organization through the Australian Government Department of Health and Ageing's National Incident Room. To prevent further imported cases, the Communicable Diseases Network Australia is working with the Australian Department of Health and Ageing and the Department of Immigration, Multicultural and Indigenous Affairs to provide health screening and health advice to incoming refugees.

Vaccine preventable diseases

Influenza

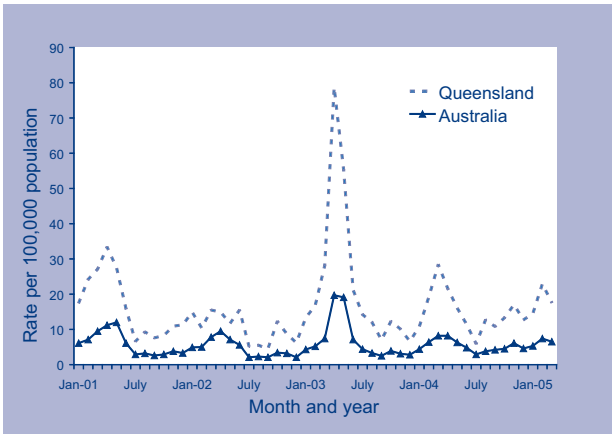
There were 378 cases of laboratory-confirmed influenza in the first quarter 2005. This was nearly four times the average number of cases for this time of year. Two hundred and seventy-six cases (73% of total reports) were from New South Wales. A number of these cases may have been diagnosed on the basis of a single high titre of antibodies to influenza virus using an enzyme immunoassay test and may not have had influenza. To increase the specificity of the surveillance, the national surveillance case definition for influenza is currently under review.

Vectorborne diseases

Barmah Forest virus infection

There were 327 notifications of Barmah Forest virus (BFV) infection in this quarter, of which 178 notifications (54%) were from Queensland. There has been a small increase in the rate of notifications in Queensland since July 2004. Rates of Barmah Forest virus are shown in Figure 2.

Figure 2. Notification rates of Barmah Forest virus infections, Queensland, compared to national rates, 1 January 2001 to 31 March 2005, by month of onset



New South Wales has noted an increase in BFV infection since 2000. The number of notifications has approximately doubled (from 195 cases in 2000 to 401 cases in 2004). The majority of these cases were identified in coastal areas.[3] It is difficult to determine the factors that have contributed to the increase in notifications in New South Wales as little is known about the natural cycle of BFV.[4] The increase in notifications could be a result of increased awareness of the clinical disease by doctors or artefacts in laboratory testing.[4]

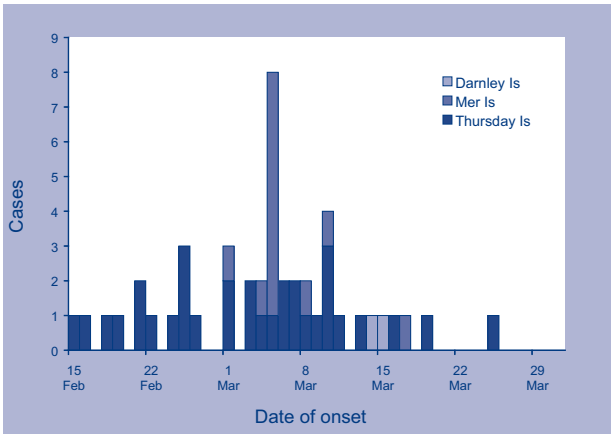
Japanese encephalitis virus

A single case of Japanese encephalitis virus (JEV) was reported from Queensland. The case was most likely exposed to JEV on Horn Island or in Port Moresby (Papua New Guinea) and had been vaccinated against JEV.

Dengue

There were 94 notifications of dengue during this quarter, which represents a fourfold increase compared to the previous quarter. In Queensland, where 69 per cent (65/94) of notified cases occurred, an outbreak of dengue type 4 was reported.[5] The outbreak started on Thursday Island, Torres Strait in February 2005. A Dengue Action Response Team was mobilised immediately to Thursday Island where the first case was a local resident without a travel history. So far, there have been 46 confirmed cases of whom two are epidemiologically linked and there are approximately 40 cases pending laboratory confirmation. The majority of the cases identified in the outbreak were on Thursday Island (n=32), however, there were also cases reported on Mer Island (n=12) and Darnley Island (n=2) (Figure 3).

Figure 3. Place of acquisition of Torres Strait dengue type 4 outbreak, 15 February to 31 March 2005, by date of onset



Malaria

There were 358 cases of malaria reported in the first quarter of 2005. The majority of the cases were reported by Queensland (n=139, 39%) and New South Wales (n=117, 33%). The notifications represent almost a twofold increase compared to the five year-to-date mean. The increase in malaria notifications can be attributed to the recent resettlement of refugees from both East and West Africa who had high rates of malaria infection. In the Northern Territory, public health precautions were taken to prevent the re-introduction of endemic malaria due to the refugee arrivals.[6] The refugees were screened immediately after arrival and parasitemic individuals were hospitalised until cured.

Of all notifications of malaria in this quarter, 57 per cent were male and 42 per cent were female. Of cases were the malaria species was known (n=200), most infection were due to *Plasmodium falciparum* (75%), *Plasmodium vivax* (14%) and *Plasmodium ovale* (4%). The other cases were infected by a combination of malaria species (7%).

Murray Valley encephalitis virus

Two cases of Murray Valley encephalitis virus (MVEV) were reported in this quarter. Both cases were detected in the high endemic period for MVEV, which is between March and May.[7] One case was in a 3-year-old boy from the Northern Territory who had travelled from Darwin to Katherine due to threats from Severe Tropical Cyclone Ingrid. The other case was in a 30-year-old male from Normanton in North Queensland. This is the second case of MVEV in Queensland in the last five years, where there was a case of MVEV in a 3-year-old Mount Isa boy in 2001.

Sentinel chicken seroconversions for MVEV were reported in Howard Springs and Katherine in February and March. Due to the recent human cases and the positive readings from sentinel chicken, Queensland and the Northern Territory health departments have issued media alerts to prevent further cases of MVEV.

Zoonoses

Brucellosis

Both Queensland and Victoria reported cases of brucellosis. The species in the Victorian case was *Brucella melitensis*, and was found in an abattoir worker. The Victorian Department of Primary Industries advised that the case was unlikely to have been acquired within the abattoir as *Brucella melitensis* is not detected in Australian stock. The case was probably acquired in Kenya prior to the person's arrival in Australia.

Thirteen cases were reported by Queensland. Most of the cases were male (n=11), where the average age was 33 years (range 20–51). Most of these cases were associated with farming or pig shooting. The *Brucella* species in three of the cases was identified as *Brucella suis*.

Scrub typhus

Fourteen soldiers in a base at Townsville, Queensland contracted scrub typhus in this quarter.[8] The cases were initially suspected to be leptospirosis, but serological tests led to the diagnosis of scrub typhus. The disease is spread to humans as a zoonosis by the bite of the larval stage of trombiculid mites. Previous clusters of scrub typhus have been documented in Queensland, including two clusters at military bases in 1996 and 1997.[9,10]

Other bacterial infections

Meningococcal infections

There were 75 notifications of meningococcal infection during the quarter, which was two-thirds the average number reported in the corresponding quarter over the previous five years. Of the 75 cases, meningococcal serogroup data was available on 64 cases. There were 41 serogroup B (64%), 14 serogroup C (22%) and two cases each of serogroup W135 (3%) and serogroup Y (3%).

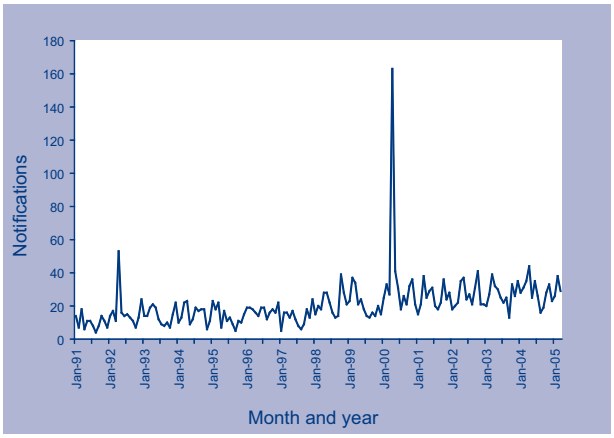
Legionellosis

There were 93 cases of legionellosis notified in the quarter, which was 20 per cent above the average reported in the corresponding quarter over the previous five years. Of cases where the *Legionella* species was known (n=87), 57 per cent were *Legionella pneumophila* (n=50), 41 per cent were *Legionella longbeachae* (n=36) and one per cent was *Legionella micdadei* (n=1).

A small outbreak of *Legionella pneumophila* occurred in Wollongong, a southern coastal city in New South Wales. The outbreak affected five people, ranging from 31 to 84 years of age. Cooling towers from three buildings in the central district of Wollongong tested positive for *Legionella pneumophila* and were cleaned to prevent further cases.

In 2003, the laboratory cut-off values for *Legionella* serology tests were revised in Victoria and South Australia to increase specificity. Tasmania uses laboratories in both Victoria and South Australia for serological diagnoses of legionellosis. This change should have decreased the number of cases notified had disease activity remained stable. However, there has been a slight but steady increase in notifications since 2000 (Figure 4). The Communicable Diseases Network Australia is monitoring the legionellosis trends and the Victorian health department has undertaken a health promotion campaign to inform the public about the disease.

Figure 4. Trends in notification of legionellosis, Australia, 1991 to 2005, by month of onset



With thanks to:

David Coleman (Health Department Tasmania)

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Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 30,752 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 January and 31 March 2005 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 4,127 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 January to 31 March 2005 (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 except NT
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 except ACT, Qld
Tularemia	All jurisdictions except Qld
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis (unspecified)	All jurisdictions
Syphilis < 2 years duration	All jurisdictions
Syphilis > 2 years 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
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 except ACT
Malaria	All jurisdictions
Murray Valley encephalitis virus‡	All jurisdictions except ACT
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 the Australian Capital Territory but reports are forwarded to NNDSS.

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

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

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2005, by date of onset*

Disease	State or territory								Total 1st quarter 2005[1]	Total 4th quarter 2004	Total 1st quarter 2004	Last 5 years mean 1st quarter	Year to date 2005	Last 5 years YTD mean	Ratio†
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis B (incident)	2	17	0	14	3	0	25	9	70	63	71	75.0	70	75.0	0.9
Hepatitis B (unspecified)	17	860	26	239	54	12	421	120	1,750	1,681	1,520	1,474.0	1,750	1,474.0	1.2
Hepatitis C (incident)	1	2	1	0	8	3	5	23	40	87	91	113.6	40	113.6	0.4
Hepatitis C (unspecified)	40	1,467	71	710	89	57	717	266	3,422	3,605	3,575	3,833.8	3,422	3,833.8	0.9
Hepatitis D	0	3	0	1	0	0	0	0	4	5	4	5.2	4	5.2	0.8
Gastrointestinal diseases															
Botulism	0	0	0	1	0	0	0	0	1	0	1	0.3	1	0.3	4.0
Campylobacteriosis[2]	118	NN	58	1,026	440	195	1,664	610	4,110	4,516	4,300	3,146.6	4,110	3,146.6	1.3
Cryptosporidiosis‡	4	134	39	719	58	1	140	60	1,155	449	528	794.5	1,155	794.5	1.5
Haemolytic uraemic syndrome	0	3	0	1	0	0	0	0	4	6	3	2.6	4	2.6	1.5
Hepatitis A	3	21	2	12	2	1	23	17	81	61	108	139.0	81	139.0	0.6
Hepatitis E	0	3	0	3	0	0	7	2	15	6	11	5.4	15	5.4	2.8
Listeriosis	0	8	0	1	1	0	1	2	13	14	16	16.6	13	16.6	0.8
Salmonellosis (NEC)	31	684	117	877	151	41	594	208	2,703	1,964	2,763	2,211.0	2,703	2,211.0	1.2
Shigellosis	3	40	57	27	11	1	33	51	223	144	153	131.4	223	131.4	1.7
SLTEC, VTEC[3]	0	2	0	3	6	0	1	1	13	14	14	14.0	13	14.0	0.9
Typhoid	0	16	0	1	1	0	5	1	24	16	30	25.6	24	25.6	0.9
Quarantinable diseases															
Cholera	0	0	0	0	0	0	2	0	2	0	1	1.0	2	1.0	2.0
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 2005, by date of onset,* ***continued***

Disease	State or territory								Total 1st quarter 2005[1]	Total 4th quarter 2004	Total 1st quarter 2004	Last 5 years mean 1st quarter	Year to date 2005	Last 5 years YTD mean	Ratio[†]
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection	172	2,801	362	2,340	556	211	2,148	1,256	9,859	9,120	9,222	5,169.0	9,859	5,169.0	1.9
Donovanosis	0	0	2	3	0	0	0	0	5	4	2	4.8	5	4.8	1.0
Gonococcal infection	7	349	417	377	67	9	261	468	1,955	1,757	1,822	1,437.4	1,955	1,437.4	1.4
Syphilis (unspecified)	4	0	0	0	2	0	0	0	6	3	71	261.2	6	261.2	0.0
Syphilis < two years duration	2	39	22	21	4	1	19	4	112	153	145	77.7	112	77.7	1.4
Syphilis > two years duration	0	215	23	45	0	5	91	26	405	435	533	278.0	405	278.0	1.5
Syphilis - congenital	0	2	1	0	0	0	0	0	3	3	2	2.0	3	2.0	1.5
Vaccine preventable disease															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0.2	0.0
Haemophilus influenzae type b	0	2	1	1	0	0	2	1	7	2	4	4.0	7	4.0	1.8
Influenza (laboratory confirmed)[‡]	3	276	1	29	12	3	29	25	378	764	110	79.0	378	79.0	4.8
Measles	0	4	0	0	1	1	1	0	7	25	16	26.0	7	26.0	0.3
Mumps	0	32	3	1	3	0	4	6	49	38	24	26.2	49	26.2	1.9
Pertussis	45	771	31	325	272	7	291	192	1,934	3,385	1,122	1,099.6	1,934	1,099.6	1.8
Pneumococcal disease (invasive)[‡]	10	74	10	41	17	8	54	26	240	461	326	232.0	240	232.0	1.0
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	0	2	0	2	0	0	3	0	7	14	8	35.8	7	35.8	0.2
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	1	0.4	0	0.4	0.0
Tetanus	0	0	0	0	0	0	0	0	0	1	2	1.0	0	1.0	0.0
Vectorborne diseases															
Barmah Forest virus infection	0	108	13	178	9	0	3	16	327	274	318	249.2	327	249.2	1.3
Dengue	1	10	9	65	1	0	3	5	94	19	236	117.6	94	117.6	0.8
Flavivirus infection (NEC)	0	3	0	8	0	0	0	0	11	6	43	25.0	11	25.0	0.4
Japanese encephalitis virus[‡]	0	0	0	1	0	0	0	0	1	1	1	0.2	1	0.2	5.0
Kunjin virus[‡]	0	0	0	1	0	0	0	0	1	2	8	3.3	1	3.3	0.3
Malaria	5	117	15	139	22	10	27	23	358	149	124	167.8	358	167.8	2.1
Murray Valley encephalitis virus[‡]	0	0	1	1	0	0	0	0	2	0	0	1.5	2	1.5	1.3
Ross River virus infection	3	115	107	542	12	0	11	42	832	237	2,443	1,395.4	832	1,395.4	0.6

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 January to 31 March 2005, by date of onset,* *continued*

Disease	State or territory								Total 1st quarter 2005[1]	Total 4th quarter 2004	Total 1st quarter 2004	Last 5 years mean 1st quarter	Year to date 2005	Last 5 years YTD mean	Ratio†
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax‡	0	0	0	0	0	0	0	0	0	0	0	0.0	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	13	0	0	1	0	14	17	6	6.6	14	6.6	2.1
Leptospirosis	0	14	1	21	0	0	1	0	37	26	69	60.0	37	60.0	0.6
Lyssavirus unspecified‡	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	20	0	1	0	0	9	1	31	54	73	33.6	31	33.6	0.9
Q fever	0	24	0	37	4	0	3	0	68	124	117	147.0	68	147.0	0.5
Other bacterial infections															
Legionellosis	0	27	1	12	17	2	23	11	93	80	93	65.8	93	65.8	1.4
Leprosy	0	0	0	2	0	0	0	2	4	0	2	1.6	4	1.6	2.5
Meningococcal infection	2	33	2	15	3	0	13	7	75	83	97	95.2	75	95.2	0.8
Tuberculosis	0	68	5	25	10	2	81	16	207	330	244	212.4	207	212.4	1.0
Total	473	8,366	1,398	7,881	1,836	585	6,716	3,497	30,752	30,201	30,473	23,306.0	30,752	23,306.0	1.3

1. 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.
2. Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/VTEC).

* 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.

† Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter.

‡ Notifiable from January 2001 only. Ratio and mean calculations are based the last three years.

NN Not notifiable.

NEC Not elsewhere classified.

Table 3. Notification rates of diseases by state or territory, 1 January to 31 March 2005. (Rate per 100,000 population)

Disease[1]	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis B (incident)	2.5	1.0	0.0	1.4	0.8	0.0	2.0	1.8	1.4
Hepatitis B (unspecified)	21.0	51.1	52.0	24.6	14.1	10.8	33.9	24.2	34.8
Hepatitis C (incident)	1.2	0.1	2.0	NN	2.1	0.0	0.4	4.6	0.8
Hepatitis C (unspecified)	49.4	87.2	142.1	73.2	23.2	51.4	57.7	53.7	68.1
Hepatitis D	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis[2]	145.7	NN	116.1	105.7	114.7	161.0	133.8	123.1	81.8
Cryptosporidiosis	4.9	8.0	78.0	74.1	15.1	0.8	11.3	12.1	23.0
Haemolytic uraemic syndrome	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Hepatitis A	3.7	1.2	4.0	1.2	0.5	0.8	1.9	3.4	1.6
Hepatitis E	0.0	0.2	0.0	0.3	0.0	0.0	0.6	0.4	0.3
Listeriosis	0.0	0.5	0.0	0.1	0.3	0.0	0.1	0.4	0.3
Salmonellosis (NEC)	38.3	40.6	234.1	90.4	39.4	34.0	47.8	42.0	53.8
Shigellosis	3.7	2.4	114.1	2.8	2.9	0.8	2.7	10.3	4.4
SLTEC, VTEC[3]	0.0	0.1	0.0	0.3	1.6	0.0	0.1	0.2	0.3
Typhoid	0.0	1.0	0.0	0.1	0.3	0.0	0.4	0.2	0.5
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	<0.1
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	212.3	166.4	724.3	241.1	145.0	185.8	172.8	253.5	196.1
Donovanosis	0.0	0.0	4.0	0.3	0.0	0.0	0.0	0.0	0.1
Gonococcal infection	8.6	20.7	834.4	38.8	17.5	7.5	21.0	94.4	38.9
Syphilis (unspecified)	4.9	0.0	0.0	0.0	0.5	0.0	0.0	0.0	0.1
Syphilis < 2 years duration	2.5	2.3	44.0	2.2	1.0	0.8	1.5	0.8	2.2
Syphilis > 2 years duration	0.0	12.8	46.0	4.6	0.0	4.1	7.3	5.2	8.1
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 by state or territory, 1 January to 31 March 2005. (Rate per 100,000 population) , *continued*

Disease[1]	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	2.0	0.1	0.0	0.0	0.2	0.2	0.1
Influenza (laboratory confirmed)	3.7	16.4	2.0	3.0	3.1	2.5	2.3	5.0	7.5
Measles	0.0	0.2	0.0	0.0	0.3	0.8	0.1	0.0	0.1
Mumps	0.0	1.9	6.0	0.1	0.8	0.0	0.3	1.2	1.0
Pertussis	55.6	45.8	62.0	33.5	70.9	5.8	23.4	38.7	38.5
Pneumococcal disease (invasive)	12.3	4.4	20.0	4.2	4.4	6.6	4.3	5.2	4.8
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.1	0.0	0.2	0.0	0.0	0.2	0.0	0.1
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	0.0	6.4	26.0	18.3	2.3	0.0	0.2	3.2	6.5
Dengue	1.2	0.6	18.0	6.7	0.3	0.0	0.2	1.0	1.9
Flavivirus infection (NEC)	0.0	0.2	0.0	0.8	0.0	0.0	0.0	0.0	0.2
Japanese encephalitis virus	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Kunjin virus	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Malaria	6.2	7.0	30.0	14.3	5.7	8.3	2.2	4.6	7.1
Murray Valley encephalitis virus	0.0	0.0	2.0	0.1	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	3.7	6.8	214.1	55.8	3.1	0.0	0.9	8.5	16.6
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	1.3	0.0	0.0	0.1	0.0	0.3
Leptospirosis	0.0	0.8	2.0	2.2	0.0	0.0	0.1	0.0	0.7
Lyssavirus unspecified	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	1.2	0.0	0.1	0.0	0.0	0.7	0.2	0.6
Q fever	0.0	1.4	0.0	3.8	1.0	0.0	0.2	0.0	1.4
Other bacterial infections									
Legionellosis	0.0	1.6	2.0	1.2	4.4	1.7	1.9	2.2	1.9
Leprosy	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.4	0.1
Meningococcal infection	2.5	2.0	4.0	1.5	0.8	0.0	1.0	1.4	1.5
Tuberculosis	0.0	4.0	10.0	2.6	2.6	1.7	6.5	3.2	4.1

1. Rates are subject to retrospective revision.
2. Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
3. Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (SLTEC/VTEC).

NN Not notifiable.

NEC Not elsewhere classified.

Table 4. Virology and serology laboratory reports by state or territory[1] for the reporting period 1 January to 31 March 2005, and total reports for the year[2]

	State or territory								This period 2005	This period 2004	Year to date 2005[3]	Year to date 2004
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Mealses, mumps, rubella												
Measles virus	–	–	–	1	1	–	–	–	2	7	2	7
Mumps virus	–	1	–	–	3	–	1	–	5	2	5	2
Rubella virus	–	–	–	1	–	–	1	2	4	6	4	6
Hepatitis viruses												
Hepatitis A virus	–	–	–	3	2	–	–	1	6	10	6	10
Hepatitis D virus	–	–	–	–	1	–	1	–	2	3	2	3
Hepatitis E virus	–	–	–	–	–	–	5	–	5	5	5	5
Arboviruses												
Ross River virus	–	5	23	161	16	–	2	1	208	431	208	431
Barmah Forest virus	–	1	–	43	10	–	–	–	54	85	54	85
Flavivirus (unspecified)	–	–	3	10	–	–	–	–	13	49	13	49
Adenoviruses												
Adenovirus not typed/ pending	–	15	–	16	53	–	1	–	85	171	85	171
Herpesviruses												
Herpes virus type 6	–	–	–	–	–	–	1	–	1	–	1	–
Cytomegalovirus	6	39	2	26	64	1	6	–	144	208	144	208
Varicella-zoster virus	2	30	3	213	103	–	3	1	355	450	355	450
Epstein-Barr virus	–	7	27	214	177	1	17	109	552	629	552	629
Other DNA viruses												
Parvovirus	–	4	–	30	10	1	9	–	54	61	54	61
Picornavirus family												
Echovirus type 7	–	2	–	–	–	–	–	–	2	–	2	–
Echovirus type 18	1	5	–	–	–	–	–	–	6	3	6	3
Echovirus type 30	–	6	–	–	1	–	–	–	7	2	7	2
Poliovirus type 1 (uncharacterised)	–	2	–	–	–	–	–	–	2	2	2	2
Poliovirus type 2 (uncharacterised)	–	3	–	–	–	–	–	–	3	2	3	2
Poliovirus type 3 (uncharacterised)	–	1	–	–	–	–	–	–	1	–	1	–
Rhinovirus (all types)	1	52	–	–	14	–	–	–	67	81	67	81
Enterovirus type 71 (BCR)	1	–	–	–	–	–	–	–	1	2	1	2
Enterovirus not typed/ pending	–	15	–	4	1	–	2	–	22	42	22	42
Ortho/paramyxoviruses												
Influenza A virus	–	1	–	5	15	–	1	–	22	40	22	40
Influenza B virus	–	–	–	3	21	1	2	–	27	12	27	12
Parainfluenza virus type 1	–	1	–	1	4	–	1	–	7	40	7	40
Parainfluenza virus type 2	–	1	–	–	5	–	–	–	6	2	6	2
Parainfluenza virus type 3	–	17	–	–	20	–	–	–	37	88	37	88
Respiratory syncytial virus	–	35	–	39	12	2	1	–	89	200	89	200
Other RNA viruses												
HTLV-1	–	–	–	–	1	–	1	–	2	2	2	2
Rotavirus	–	10	–	–	36	–	1	–	47	77	47	77
Norwalk agent	–	–	–	–	–	–	2	–	2	83	2	83

Table 4. Virology and serology laboratory reports by state or territory[1] for the reporting period 1 January to 31 March 2005, and total reports for the year,[2] *continued*

	State or territory								This period 2005	This period 2004	Year to date 2005[3]	Year to date 2004
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other												
Chlamydia trachomatis not typed	10	205	3	506	415	13	4	1	1,157	1,185	1,157	1,185
Chlamydia pneumoniae	–	1	–	–	–	–	2	–	3	2	3	2
Chlamydia psittaci	–	1	–	–	–	–	13	–	14	62	14	62
Mycoplasma pneumoniae	–	7	2	86	69	1	61	8	234	355	234	355
Mycoplasma hominis	–	1	–	–	–	–	–	–	1	1	1	1
Coxiella burnetii (Q fever)	–	2	–	6	25	–	1	–	34	48	34	48
Rickettsia prowazeki	–	–	–	–	22	–	–	–	22	–	22	–
Rickettsia tsutsugamushi	–	–	–	–	11	–	–	–	11	–	11	–
Rickettsia - Spotted fever group	–	–	–	–	47	2	–	–	49	–	49	–
Streptococcus group A	–	2	–	75	–	–	27	–	104	125	104	125
Yersinia enterocolitica	–	4	–	–	–	–	–	–	4	1	4	1
Brucella abortus	–	–	–	–	–	–	1	–	1	3	1	3
Brucella species	–	–	–	2	–	–	–	–	2	–	2	–
Bordetella pertussis	–	12	3	78	201	–	87	–	381	156	381	156
Legionella pneumophila	–	–	–	–	2	–	4	–	6	20	6	20
Legionella longbeachae	–	–	–	–	7	–	4	–	11	16	11	16
Cryptococcus species	–	–	–	4	6	–	–	–	10	13	10	13
Leptospira species	–	–	–	3	–	–	–	–	3	13	3	13
Treponema pallidum	1	9	1	114	98	–	–	–	223	328	223	328
Entamoeba histolytica	–	–	–	2	–	–	2	–	4	4	4	4
Toxoplasma gondii	–	1	–	2	3	–	2	–	8	14	8	14
Echinococcus granulosus	–	1	–	–	4	–	–	–	5	4	5	4
Total	22	499	67	1,648	1,480	22	266	123	4,127	5,145	4,127	5,145

1. State or territory of postcode, if reported, otherwise state or territory of reporting laboratory.
2. 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 2005*

State or territory	Laboratory	January 2005	February 2005	March 2005	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	62	88	129	279
	New Children's Hospital, Westmead	29	47	43	119
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	29	30	3	62
	South West Area Pathology Service, Liverpool	–	–	–	0
Queensland	Queensland Medical Laboratory, West End	603	543	596	1,742
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	436	463	578	1,477
Tasmania	Northern Tasmanian Pathology Service, Launceston	6	6	10	22
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Monash Medical Centre, Melbourne	9	–	–	9
	Royal Children's Hospital, Melbourne	46	54	39	139
	Victorian Infectious Diseases Reference Laboratory, Fairfield	48	37	24	109
Western Australia	PathCentre Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	35	58	76	169
Total		1,303	1,326	1,498	4,127

* The complete list of laboratories reporting for the 12 months, January to December 2005, 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 50 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 4,000 and 6,000 consultations are recorded each week.

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

In 2005, 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 2005. 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 *2005;29:91.*

Data from 1 January to 31 March 2005 are shown as the rate per 1,000 consultations in Figures 5 and 6.

Figure 5. Consultation rates for influenza-like illness, ASPREN, 1 January to 31 March 2005, by week of report

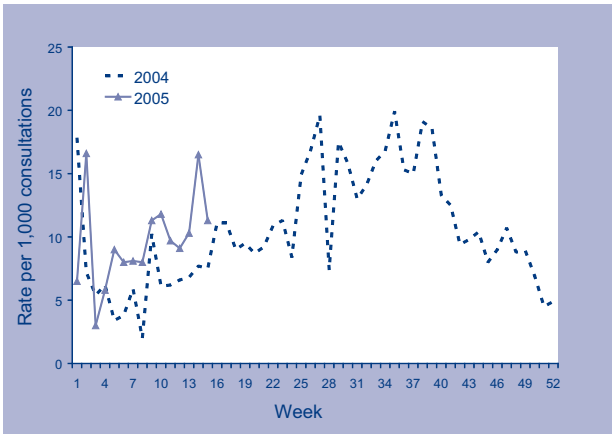
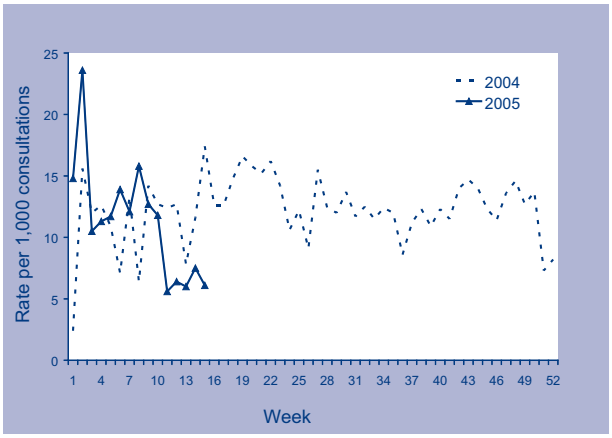


Figure 6. Consultation rates for gastroenteritis, ASPREN, 1 January to 31 March 2005, by week of report



Childhood immunisation coverage

Tables 6, 7 and 8 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at 12 months of age for the cohort born between 1 October and 31 December 2003, at 24 months of age for the cohort born between 1 October and 31 December 2002, and at 6 years of age for the cohort born between 1 October and 31 December 1998 according to the Australian Standard Vaccination Schedule.

A full description of the methodology used can be found in 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 1256, Email: brynleyh@chw.edu.au.

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia decreased marginally from the last quarter by 0.4 percentage points to 90.7 per cent (Table 6). There was a substantial decrease in 'fully immunised' coverage by state or territory in Western Australia, with a decrease of 2.5 percentage points, and a substantial increase of 2.4 per cent in the Northern Territory, whilst all other jurisdictions experienced very little significant change in coverage. As expected, Western Australia also had decreases in coverage for individual vaccines.

There was no change in coverage for children 'fully immunised' at 24 months of age for Australia, which remained at 91.7 per cent (Table 7). Coverage for individual vaccines remained largely unchanged in most jurisdictions with coverage greater than 95 per cent in almost all jurisdictions for all vaccines except *Haemophilus influenzae* type b.

Table 8 shows immunisation coverage estimates for 'fully immunised' and for individual vaccines at six years of age for Australia and by state or territory. 'Fully immunised' coverage at six years of age for Australia was largely unchanged overall, apart from small decreases in South Australia (-1.5%), Queensland (-1.0%) and in the Northern Territory (-1.6%), also reflected in individual vaccines. Coverage for vaccines assessed at six years is at or near 85 per cent in the most jurisdictions, but Western Australia and Queensland remain below the average.

Figure 7 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 18 months for all age groups. The figure shows that there have now been six 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 18 month DTPa vaccine. However, both measures have been above 90 per cent for this 18-month period and show levels of high coverage being maintained over a significant period of time.

Figure 7. Trends in vaccination coverage, Australia, 1997 to 2004, by age cohorts

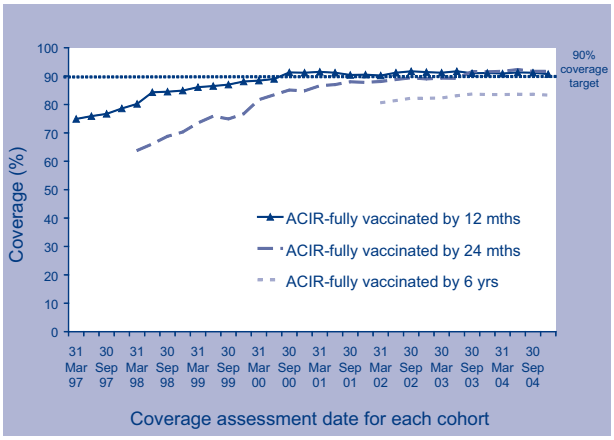


Table 6. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 October to 31 December 2003; assessment date 31 March 2005

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,061	21,546	857	12,581	4,338	1,487	15,793	6,086	63,749
Diphtheria, tetanus, pertussis (%)	93.4	92.1	92.9	92.2	92.0	94.6	92.6	90.3	92.2
Poliomyelitis (%)	93.4	92.0	92.3	92.0	91.8	94.2	92.5	90.2	92.0
Haemophilus influenzae type b (%)	95.9	94.2	97.2	94.2	95.1	95.6	94.4	94.1	94.4
Hepatitis B (%)	95.5	95.0	97.1	94.8	95.4	95.5	94.2	93.8	94.7
Fully immunised (%)	92.7	90.7	92.2	90.8	91.2	92.9	90.8	89.2	90.7
Change in fully immunised since last quarter (%)	-0.0	-0.0	+2.4	-0.6	+0.2	-0.1	-0.5	-2.5	-0.4

Table 7. 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 2002; assessment date 31 March 2005[5]

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,013	21,810	875	12,289	4,257	1,460	15,577	6,148	63,429
Diphtheria, tetanus, pertussis (%)	96.0	94.6	96.8	94.9	95.4	96.4	95.1	93.7	94.9
Poliomyelitis (%)	96.1	94.5	96.7	94.8	95.4	96.2	95.1	93.8	94.8
Haemophilus influenzae type b (%)	94.6	92.7	95.5	93.6	93.9	94.6	93.6	92.2	93.2
Measles, mumps, rubella (%)	94.5	92.8	96.2	93.6	94.3	94.8	94.0	92.2	93.4
Hepatitis B(%)	96.7	95.3	97.8	95.6	95.8	96.6	95.7	94.9	95.5
Fully immunised (%)	93.7	90.9	95.0	91.9	92.7	94.0	92.2	90.6	91.7
Change in fully immunised since last quarter (%)	+1.7	-0.2	+1.2	+0.3	-0.5	+1.1	-0.4	+0.7	0.0

* The 12 months age data for this cohort was published in *Commun Dis Intell* 2004;28:297.

Table 8. 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 1998; assessment date 31 March 2005

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,042	21,913	779	12,533	4,668	1,655	16,293	6,663	65,546
Diphtheria, tetanus, pertussis (%)	90.9	85.2	85.5	82.0	84.3	86.4	86.9	81.6	84.7
Poliomyelitis (%)	91.2	85.1	87.8	82.2	84.3	86.5	87.0	81.9	84.8
Measles, mumps, rubella (%)	90.1	84.9	88.3	82.1	84.1	85.6	87.1	81.4	84.6
Fully immunised (%)[1]	89.8	83.7	85.1	80.6	82.8	84.2	85.8	79.8	83.3
Change in fully immunised since last quarter (%)	+2.7	-0.0	-1.6	-1.0	-1.5	+0.9	+0.1	-0.8	-0.3

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 2005, are included in this issue of Communicable Diseases Intelligence *(Table 9)*

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.

Table 9. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2005, by jurisdiction and serogroup

Jurisdiction	Year	Serogroup							
		A	B	C	Y	W135	ND	All	
		Q1	Q1	Q1	Q1	Q1	Q1	Q1	YTD
Australian Capital Territory	05		1	1				2	2
	04		(4)	(2)				(6)	(6)
New South Wales	05		15	7	1		2	25	25
	04		(15)	(4)	(0)		(6)	(25)	(25)
Northern Territory	05		1	0				1	1
	04		(5)	(0)				(5)	(5)
Queensland	05		12	4				16	16
	04		(12)	(7)			(2)	(1)	(21)
South Australia	05		0	2				2	2
	04		(4)	(0)				(4)	(4)
Tasmania	05		0					0	0
	04		(2)				(2)	(4)	(4)
Victoria	05		7	1	0	2	1	11	11
	04		(10)	(4)	(2)	(0)	(2)	(17)	(17)
Western Australia	05		4	0	1			5	5
	04		(4)	(1)	(0)			(5)	(5)
Total	05		40	15	2	2	3	62	62
	04		(56)	(18)	(2)	(0)	(11)	(87)	(87)

* Numbers of laboratory-confirmed diagnoses of invasive meningococcal disease made in the same period in 2004 are shown in parentheses.

Q1 = 1st quarter.

YTD = Year to 31 March 2005.

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 *2004;28:99.*

HIV and AIDS diagnoses and deaths following AIDS reported for 1 October to 31 December 2004, as reported to 31 March 2005, are included in this issue of Communicable Diseases Intelligence *(Tables 10 and 11).*

Table 10. New diagnoses of HIV infection, new diagnoses of AIDS, and deaths following AIDS occurring in the period 1 October to 31 December 2004, by sex and state or territory of diagnoses

	Sex	State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2004	This period 2003	YTD 2004	YTD 2003
HIV diagnoses	Female	0	12	0	7	2	0	6	2	29	19	116	83
	Male	0	72	0	30	5	0	51	9	167	180	737	770
	Sex not reported	0	0	0	0	0	0	0	0	0	2	2	7
	Total*	0	84	0	37	7	0	57	11	196	201	856	861
AIDS diagnoses	Female	0	2	0	2	1	0	1	0	6	2	17	12
	Male	0	14	1	2	1	0	8	3	29	60	138	206
	Total*	0	16	1	4	2	0	9	3	35	62	156	219
AIDS deaths	Female	0	0	0	0	0	0	1	0	1	1	5	9
	Male	0	10	0	2	1	0	2	3	18	24	71	80
	Total	0	10	0	2	1	0	3	3	19	25	76	89

* Totals include people whose sex was reported as transgender.

Table 11. Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 31 December 2004 and reported by 31 March 2005, by sex and state or territory

	Sex	State or territory								Australia
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
HIV diagnoses	Female	31	781	18	230	84	8	312	164	1,628
	Male	246	12,663	123	2,436	833	89	4,768	1,097	22,255
	Not reported	0	238	0	0	0	0	22	0	260
	Total*	277	13,710	141	2,675	918	97	5,121	1,268	24,207
AIDS diagnoses	Female	9	225	2	63	31	4	97	35	466
	Male	92	5,165	42	976	387	48	1,873	410	8,993
	Total*	101	5,405	44	1,041	419	52	1,980	447	9,489
AIDS deaths	Female	6	128	1	41	20	2	59	23	280
	Male	71	3,506	26	638	269	32	1,369	287	6,198
	Total*	77	3,643	27	681	289	34	1,436	311	6,498

* Totals include people whose sex was reported as transgender.

National Enteric Pathogens Surveillance System

The National Enteric Pathogens Surveillance System (NEPSS) collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. These pathogens include Salmonella, E. coli, Vibrio, Yersinia, Plesiomonas, Aeromonas *and* Campylobacter. Communicable Diseases Intelligence *NEPSS quarterly reports include only* Salmonella.

Data are based on reports to NEPSS from Australian laboratories of laboratory-confirmed human infection with Salmonella. Salmonella *are identified to the level of serovar and, if applicable, phage-type. Infections apparently acquired overseas are included. Multiple isolations of a single* Salmonella *serovar/phage-type from one or more body sites during the same episode of illness are counted once only. The date of the case is the date the primary diagnostic laboratory isolated a* Salmonella *from the clinical sample.*

Note that the historical quarterly mean counts should be interpreted with caution, and are affected by surveillance artefacts such as newly recognised (such as S. Typhimurium 197 and S. Typhimurium U290) and incompletely typed Salmonella.

NEPSS is operated by the Microbiological Diagnostic Unit, Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne; and is overseen by a Steering Committee of state, territory and commonwealth stakeholders. NEPSS can be contacted at the above address or 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 2005 are included in Tables 12 and 13. Data include cases reported and entered by 21 April 2005. 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 *2005;29:93–94.*

First quarter 2005

The total number of reports to NEPSS of human Salmonella infection increased to 2,551 in the first quarter of 2005, 45 per cent more than in the fourth quarter of 2004. This count is six per cent less than the comparable first quarter of 2004 but similar to the historical mean for this time of year.

During the first quarter of 2005, the 25 most common Salmonella types in Australia accounted for 1,789 cases, 70 per cent of all reported human Salmonella infections. Twenty-one of the 25 most common Salmonella infections in the first quarter of 2005 were among the 25 most commonly reported in the last quarter of 2004.

S. Typhimurium phage type 197 was the most common cause of human salmonellosis this quarter, reflecting a large point-source outbreak in Victoria in early 2005 and steady increases in sporadic cases of this phage type in Queensland and New South Wales since 2002. An outbreak in New South Wales ensured the continued prominence of S. Typhimurium phage type 170/108, although counts of this strain have declined in Victoria and South Australia.

Reports of other common salmonellae with recent increases and counts that remain above historical averages include S. Saintpaul (in northern Australia), S. Aberdeen (particularly in Queensland), S. subsp I ser 16:l,v:- (in New South Wales) and S. Corvallis and S. Enteritidis 6a (both typically acquired overseas). S. Typhimurium phage types 9 and 135 remain common, although less so than in the mid to late 1990s.

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 2005, as reported to 21 April 2005

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all *Salmonella* for quarter	30	668	106	809	142	39	559	198	2,551
Total contributing *Salmonella* types	20	129	42	101	46	9	95	70	234

Table 14. Top 25 *Salmonella* types identified in Australia, 1 January to 31 March 2005, by state or territory

National rank	*Salmonella* type	State or territory								Total 1st quarter 2005	Last 10 years mean 1st quarter	Year to date 2005	Year to date 2004
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
1	*S.* Typhimurium 197	1	51	0	61	3	2	262	1	381	18	381	86
2	*S.* Typhimurium 170	5	128	0	10	1	0	16	3	163	69	163	197
3	*S.* Typhimurium 9	1	51	3	8	33	3	51	7	157	176	157	147
4	*S.* Saintpaul	2	9	17	102	3	0	6	12	151	125	151	132
5	*S.* Typhimurium 135	5	43	1	25	2	6	26	16	124	236	124	198
6	*S.* Virchow 8	0	7	1	85	2	0	3	1	99	85	99	143
7	*S.* Chester	0	13	1	38	3	0	2	14	71	66	71	78
8	*S.* Birkenhead	0	34	0	35	0	0	1	0	70	96	70	107
9	*S.* Aberdeen	0	6	2	56	0	0	1	0	65	39	65	34
10	*S.* Muenchen	1	15	6	23	3	0	4	12	64	58	64	45
11	*S.* Hvittingfoss	0	2	1	51	0	0	1	0	55	30	55	64
12	*S.* Typhimurium 12	0	17	3	15	2	0	9	8	54	27	54	140
13	*S.* Infantis	0	13	1	6	10	0	19	2	51	49	51	52
14	*S.* Waycross	0	13	0	28	0	0	3	0	44	44	44	59
15	*S.* Mississippi	0	2	0	0	1	23	4	2	32	36	32	41
16	*S.* Typhimurium RDNC	0	6	4	4	5	0	4	2	25	30	25	25
17	*S.* Typhimurium 4	0	20	0	2	1	0	2	0	25	23	25	29
18	*S.* subsp I ser 16:l,v:-	0	16	2	6	0	0	0	1	25	11	25	15
19	*S.* Corvallis	1	3	1	4	0	0	8	7	24	2	24	14
20	*S.* Singapore	0	6	0	5	0	0	3	5	19	21	19	40
21	*S.* Typhimurium 108	0	12	0	0	7	0	0	0	19	9	19	41
22	*S.* Enteritidis 6a	0	7	0	2	2	0	3	5	19	3	19	9
23	*S.* Anatum	0	3	0	11	0	0	1	3	18	32	18	46
24	*S.* Litchfield	0	2	9	4	0	0	0	3	18	13	18	14
25	*S.* Enteritidis 26	1	0	0	13	0	0	1	1	16	14	16	23

Overseas briefs

World Health Organization

This material has been summarised from information on the World Health Organization Internet site (www.who.int).

Avian influenza – situation in Viet Nam and Cambodia

29 March 2005

Viet Nam

The Ministry of Health in Viet Nam has confirmed three additional cases of human infection with H5N1 avian influenza. The cases concern a 5-year-old boy from the central province of Quang Binh, a 17-year-old girl from the northern province of Nam Dinh, and a 40-year-old woman from the northern province of Quang Ninh. The 17-year-old girl has died.

An earlier case has also been confirmed. These recently confirmed cases bring the total in Viet Nam since mid-December to 28 cases.

The current outbreak of human cases in Viet Nam has included several clusters, mostly in family members, of cases closely related in time and place. Thorough investigation of all such clusters is essential to determine possible changes in the behaviour of the virus and thus support assessment of the risk of an influenza pandemic.

There is currently no evidence that the H5N1 virus is spreading easily from person to person. Rapid sharing of information with the World Health Organization (WHO) on viruses from recent clusters of cases has become increasingly important. Analysis can determine whether any significant changes in the virus have taken place and provide further support for risk assessment.

Cambodia

The Ministry of Health in Cambodia has confirmed the country's second human case of avian influenza.

The 28-year-old man, developed symptoms on 17 March and was hospitalised in Phnom Penh on 21 March. He died on 22 March. The same day, laboratory tests confirmed that the man was infected with H5 avian influenza virus. The Cambodian government immediately launched an investigation to search for possible additional cases and identify possible sources of exposure to the virus. Numerous deaths among chickens in the area have been reported and samples taken from sick chickens have tested positive for avian influenza. The results from the investigation indicate the deceased man had contact with sick poultry.

An 18-year-old boy initially identified as an additional suspected case has tested negative for the avian influenza virus. Samples taken from 27 other people, including family contacts of the confirmed case and Phnom Penh medical staff involved in his care, have all tested negative for H5 avian influenza infection. Results from a further six people from Kampot Province have also tested negative for H5 influenza virus.

Cambodia's previous case, a 25-year-old woman who died in late January, was also from Kampot Province but lived in another district.

The majority of poultry in Cambodia are raised in small backyard flocks in rural areas, making surveillance for outbreaks especially challenging. A campaign to educate rural populations about the dangers of contact with dead or diseased poultry is being undertaken by the government, with support from WHO.

Marburg haemorrhagic fever in Angola

31 March 2005

As of 30 March 2005, 132 cases of Marburg haemorrhagic fever have been reported. These cases include 12 health care workers. Of the 132 cases, 127 have been fatal. This is the largest number of fatalities ever recorded during an outbreak of this rare, but extremely severe disease.

Mobile surveillance teams have been established and are investigating rumours of additional cases in Uige Province, which remains the epicentre of the outbreak.

WHO has despatched a further 500 kg of personal protective equipment and other supplies to assist in the immediate improvement of infection control in hospitals and the protection of front-line staff. To facilitate real-time coordination of response operations, WHO has despatched mobile communication field kits, which should greatly expedite the flow of information.

Dengue haemorrhagic fever in Timor-Leste

1 March 2005

As of 28 February, WHO has received reports of 336 hospitalised cases of dengue infection and 22 deaths. Two hundred and sixty-three of the 336 cases had clinical features compatible with dengue haemorrhagic fever (DHF) and the remaining 73 cases were diagnosed as suspected dengue fever (DF) using WHO standard case definitions.

Preliminary laboratory results have identified dengue 3 as the main circulating strain in this outbreak.

The Ministry of Health, with support from WHO, NIID, and USAID are carrying out vector control activities. Insecticide spraying has covered more than 2,000 households in high-risk areas, and additional spraying and larval control are underway in Dili and Baucau.

Health education activities are also being carried out to raise awareness of the disease and the need for appropriate prevention and control measures.

ProMED-mail

This material has been summarised from information provided by ProMED-mail (http://www.promedmail.org). A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia homepage.

Haemolytic uremic syndrome, petting zoo – USA – Florida

Source: WPTV 31 March 2005 (edited)

Five new cases of bacterial illness have been confirmed in children, who visited central Florida petting zoos, bringing the total number to 22 with 24 other infections suspected.

Some cases have tested positive for the *Escherichia coli* O157:H7 bacterium and others have developed a related kidney disease, haemolytic uremic syndrome. A suspected link between the outbreak and the death of a 12-year-old Pasco County girl has been ruled out. Tests came back negative for the *E. coli* strain.

Investigators suspect petting zoo animals rather than food vendors as the source of the problem. All of the victims visited the Central Florida Fair in Orange County or the Florida Strawberry Festival in Plant City earlier in March 2005.

HIV, multi-drug resistant – USA, New York City

Source: BMJ 2005;330:691 (edited)

Researchers have published clinical and sequencing details of a unique variant of human immunodeficiency virus (HIV) in a patient who is resistant to several classes of antiretroviral drugs and who rapidly developed AIDS, after media reports of a possible new 'super-strain' of the virus.

The patient, a man in his 40s from New York, was given a diagnosis of HIV-1 infection in December 2004 after he had exhibited fever, pharyngitis, weakness, and fatigue the previous month. Earlier he had tested negative for HIV-1 antibodies on several occasions between September 2000 and May 2003.

His illness progressed to AIDS within 20 months—and perhaps as little as 4 months—of infection. The case was first publicised at a press conference in New York in February 2005 because of the possible public health implications of a rapidly progressing strain of HIV with multiple drug resistance.

Detailed analysis of the virus showed that it was resistant to three of the four major classes of antiretroviral drugs: nucleoside reverse transcriptase inhibitors, non-nucleoside reverse transcriptase inhibitors, and protease inhibitors. Viruses usually become less virulent when they acquire drug resistance. However, the replication capacity of the patient's HIV-1 was 136 per cent in comparison with a median of 100 per cent derived from a large number of wild-type viruses. This indicated that, despite its multi-drug resistance, the virus replicated as well as most wild-type, drug-susceptible viruses.

Rapid progression to AIDS after acute HIV-1 infection has been described previously, as has the transmission of multi-drug-resistant viruses. However, one of the authors of the case report, said: 'The unique feature in this case is the convergence of two uncommon factors: the transmission of a multi-drug resistant HIV-1 variant and the extremely rapid clinical course to AIDS.'

Viral sequence analysis indicated that the patient's virus was subtype B, and the relative homogeneity of the viral population was consistent with early HIV-1 infection. Phylogenetic analysis of a nucleotide sequence from the viral *pol* gene and from 30 newly infected individuals and five reference HIV-1 strains showed that the viral sequence of the new case was unique. No match was found in the centre's sequence database.

Because of its unique features, this *pol* sequence is now being compared with those in other databases with the hope of finding a closely related HIV-1 that might provide an epidemiological link to this case. The rapid clinical course of the patient's illness could be explained by the properties of his unique HIV-1 variant.

Aedes aegypti*, other mosquitoes: name changes*

Source: ProMED-mail, 17 March 2005 (edited)

The so-called yellow fever mosquito, until recently known as *Aedes aegypti*, has recently been placed in the subgenus *Stegomyia*; and its name changed to *Aedes (Stegomyia) aegypti*.

Last year a detailed study on the phylogeny of the genus *Aedes* and another 45 aedine genera resulted in a reclassification of these mosquitoes. One of the more important changes is that the subgenus *Stegomyia* has been elevated to generic status, and so *Aedes aegypti* become *Stegomyia aegypti* (which can be abbreviated to *St. aegypti*) and *Ae. albopictus* becomes *Stegomyia albopicta.* Here the masculine name *albopictus* has to be feminised to agree with the genus name *Stegomyia*.

Many other former *Aedes* species, including vectors such as *africanus* (now *africana*), *bromeliae*, *polynesiensis* and *scutellaris*, are now in the genus *Stegomyia*.

References

The reference for these changes is:

Reinert JF, Harbach RE, Kitching IJ. Phylogeny and classification of *Aedini* (*Diptera: Culicidae*), based on morphological characters of all life stages. *Zoological J Linnean Society* 2004;142:289–368.

For those interested in Australasian mosquitoes a key reference is:

Reinert JF, Harbach RE. Generic and subgeneric status of aedine mosquito species (*Diptera: Culicidae: Aedini*) occurring in the Australasian Region. *Zootaxa* 2005;887:1–10.

Creutzfeldt-Jakob disease (new var.), United Kingdom update

Source: UK Department of Health, Monthly Creutzfeldt-Jakob Disease Statistics, Press release no. 2005/0161, 4 April 2005 [edited]

Definite and probable CJD cases in the UK

Deaths from definite vCJD (confirmed): 107

Deaths from probable vCJD (without neuropathological confirmation): 42

Deaths from probable vCJD (neuropathological confirmation pending): 0

Number of deaths from definite or probable vCJD (as above): 149

Number of probable vCJD cases still alive: 6

Number of definite or probable vCJD (dead and alive): 155

Influenza update – Northern Hemisphere

Source: World Health Organization (WHO), 10 February 2005 (edited)

Each year, the World Health Organization Global Influenza Program convenes meetings to analyse the global data on circulating influenza virus strains and make recommendations for the vaccine to be used for the coming influenza season. WHO also provides the vaccine manufacturing industry with prototype strains for the seasonal vaccine as well as materials to ensure and validate that global vaccine standards are met.

This year [2005], more than 10,000 influenza viruses from all continents were isolated and characterised by the WHO/National Influenza Centres. These laboratories, which are located in more than 80 countries, form the backbone of the global influenza surveillance program. Based on that assembled information, WHO has published its recommendations on the formulation of the influenza vaccine for the Northern Hemisphere.

WHO has recommended that vaccines to be used in the 2005–2006 season (Northern Hemisphere) should contain the following:

1. an A/New Caledonia/20/99(H1N1)-like virus;
2. an A/California/7/2004(H3N2)-like virus (candidate vaccine viruses are being developed);
3. a B/Shanghai/361/2002-like virus (the currently used vaccine viruses are B/Shanghai/361/2002, B/Jiangsu/10/2003 and B/Jilin/20/2003).

These recommendations are used by pharmaceutical manufacturers to update the composition of the influenza vaccines they produce. This annual adjustment is necessary to match the vaccine with the changing viruses expected to be circulating during the coming influenza season.

Recommendations for the composition of the vaccine to be used in the Southern Hemisphere for the 2006 season, will be made at a meeting in September 2005.

While influenza vaccine coverage has improved significantly in the last 10 years, the vaccine is not reaching everyone in the high risk categories. These categories, defined by WHO, include the elderly, those who are at increased risk because they have other respiratory or cardiovascular disease, and health care workers. However, influenza vaccine use in developing countries remains minimal to nonexistent.

Last year [2004], WHO's Member States set a goal of 60 per cent coverage for those in these high risk groups and 75 per cent coverage by 2010.

Since young children can develop severe disease, some countries have also started including vaccination of children as part of their national influenza policy. Vaccinating children may not only reduce their disease burden, but it may also reduce transmission to the elderly and others at increased risk.

Since the March 2005, the number of deaths from definite vCJD has increased by one, but the total number of deaths from definite or probable vCJD remains at 149 deaths. The number of probable vCJD cases still alive has increased from five to six. Therefore the overall total number of definite or probable vCJD cases (dead and alive) has increased from 154 to 155 cases.

Malaria, resistant – India

Source: Health India [edited] 7 January 2005

The malaria parasite is increasingly becoming resistant to chloroquine, and incidence of falciparum malaria is on the rise in India, suggesting a reappraisal of the treatment approach, so that a combination of drugs is used rather than only chloroquine.

A study at the Indian Institute of Science showed that 95 per cent of malaria cases were resistant to Chloroquine. Similar results were obtained from another study at the All India Institute of Medical Sciences, in which 90 per cent of malaria cases were found to be infected by the chloroquine-resistant parasite.

In India the share of deadly 'falciparum' malaria, which infects the brain and causes high mortality, is growing. While the ratio of 'vivax' malaria, which is usually easily treated with drugs, and 'falciparum' malaria used to be 85/15, the share of 'falciparum' malaria has now gone up to almost to half. In some tribal areas in Madhya Pradesh, falciparum malaria is contributing 75–90 per cent of malaria cases.