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Invasive pneumococcal disease in Australia, 2001

Paul Roche,¹ Vicki Krause², for the Enhanced Pneumococcal Surveillance Group of the Pneumococcal Working Party of the Communicable Diseases Network Australia³

Abstract

There were 1,681 cases of invasive pneumococcal disease (IPD) notified to the National Notifiable Diseases Surveillance System in Australia in 2001; a rate of 8.6 cases per 100,000 population. The notification rate varied between states and territories and by geographical region with the highest rates in the north of the country. Pneumococcal disease was reported most frequently in children aged less than 5 years (47.3 cases per 100,000 population). Enhanced surveillance for IPD was carried out in the Northern Territory, Western Australia, South Australia, Victoria, Tasmania and metropolitan areas of New South Wales, encompassing 72 per cent of the population and providing additional data on 86 per cent of all notified cases. Enhanced surveillance data revealed high rates of pneumococcal disease in Indigenous Australians. Rates of IPD in Indigenous children aged less than 5 years were as high as 483 cases per 100,000 population in the Northern Territory. The clinical presentation of IPD was most commonly pneumonia (56%) and bacteraemia (36%). There were 125 deaths attributed to IPD resulting in an overall case fatality rate of 8.6 per cent. More than half (54%) of all cases had a recognised risk factor for IPD. Eighty-six per cent of serotypes identified in non-Indigenous children compared with only 55% of serotypes in Indigenous children were in the 7-valent vaccine. Antibiotic susceptibility testing showed reduced susceptibility to penicillin in 12 per cent, and to third generation cephalosporins in 5 per cent of isolates. These are the first national data available on IPD in Australia and will assist in evaluating the impact of the newly introduced conjugate vaccine and guide overall pneumococcal vaccine strategies. *Commun Dis Intell* 2002;26:505–519.

Keywords: Streptococcus pneumoniae, antibiotic susceptibility, pneumococcal disease, surveillance, penicillin, cephalosporins

Introduction

Infection with *Streptococcus pneumoniae* is responsible for significant morbidity and mortality worldwide, especially in the very young, the elderly and those with predisposing risk factors. It is a leading cause of otitis media, pneumonia, bacteraemia, meningitis and a less frequent cause of other conditions including septic arthritis and mastoiditis. Invasive pneumococcal disease (IPD) is defined as a clinical condition in which *S. pneumoniae* is isolated from a normally sterile site, e.g. blood, cerebrospinal fluid or pleural fluid. IPD presents most commonly as pneumonia in adults and bacteraemia in children. The risk of disease is highest among people who are immunocom-

promised (e.g. due to HIV, asplenia) or have a chronic illness (e.g. chronic cardiovascular, pulmonary, renal or liver disease or diabetes).

In developed countries, the incidence rate of IPD is bimodal, with a peak in children under 2 years and another peak in adults over 65 years. The incidence rates can be many times higher in developing countries and in some populations of developed countries, including Australian and American indigenous people. Indigenous children from Central Australia have the highest rates in the world with the most recently reported incidence of 1,534 cases per 100,000 population.¹ Case fatality rates for IPD vary from 12–14 per cent depending on the age and the focus of the disease.²

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Since the 1970s, large outbreaks of severe pneumococcal disease caused by penicillin resistant organisms occurred in South Africa and Papua New Guinea and subsequently increased rates of penicillin resistance in pneumococci have been documented worldwide. In Australia, the rate of penicillin resistant pneumococci increased from one per cent in 1984 to 25 per cent in 1997. Reduced susceptibilities to other antimicrobials has also emerged in recent years with the rate of reduced susceptibility to third generation cephalosporins in Australia reaching 13 per cent in 1997.³ The emergence of multi-drug resistant pneumococci has been an important reason for the development of new pneumococcal vaccines.

Ninety serotypes of *S. pneumoniae*, which are unique in the polysaccharide composition of their capsules, have been described. Immunity to infection is thought to be serotype specific. Vaccines containing pneumococcal polysaccharides from a varying number of serotypes have been available for many years, with a 23-valent polysaccharide vaccine produced in 1983 being licensed in Australia in 1986 (Table 1). Polysaccharide vaccines have shown 50–80 per cent effectiveness in preventing invasive pneumococcal disease in immunocompetent adults,⁴ but are poorly effective in children.⁵ A vaccine in which polysaccharides from seven serotypes coupled to a protein carrier (mutated diphtheria toxoid) was developed to provide an effective vaccine for children and in a trial in the United States of America (USA) in infants aged 2 to 15 months of age demonstrated an efficacy of 93.9 per cent.⁶ This conjugate vaccine was licensed for use in Australia in January 2001 and the Australian Technical Advisory Group on Immunisation (ATAGI) recommended vaccination of children at high risk, commencing in July 2001 (Table 1).

The ATAGI group recommended that the impact of the new conjugate vaccine on IPD in Australia be monitored by means of national surveillance of all incident cases. The Communicable Diseases Network Australia agreed to make IPD a notifiable disease in all Australian jurisdictions from January 2001. In 2001, State and Territory health authorities made changes to legislation to make reporting of all cases of IPD mandatory in each jurisdiction. The Pneumococcal Working

Party of the Communicable Diseases Network Australia convened a working group to devise an appropriate surveillance dataset for IPD. This surveillance working group recommended that data be collected to establish baseline nationwide data on IPD and evaluate the impact of the new 7-valent conjugate vaccine and 23-valent polysaccharide vaccine on the clinical presentation, serotype and antibiotic resistance (to penicillin and third generation cephalosporins).

This paper reports on data from 2001 and combines National Notifiable Diseases Surveillance System (NNDSS) data with additional data collected on cases of pneumococcal disease in six jurisdictions.

Methods and materials

Case definition

A case of invasive pneumococcal disease was defined as the isolation from, or the detection in, blood, cerebrospinal fluid (CSF) or other sterile site, of *S. pneumoniae*.

National Notifiable Diseases Surveillance System

While IPD has been a notifiable disease in some States and Territories for several years, it became a notifiable disease in all Australian States and Territories only in 2001. This required changes to public health legislation in all States and Territories, resulting in different starting dates for collection of data from the individual jurisdictions. In some States and Territories, there was a retrospective collection of data for the whole year.

Data on IPD cases sent to the NNDSS included basic demographic data — age, sex and date of birth, residential postcode (except in the NT) and indigenous status — and the dates of onset, report and data transmission.

Enhanced data collections were available from prospective surveillance schemes in the Northern Territory, South Australia, Western Australia, Victoria and metropolitan New South Wales. Enhanced data for IPD for 2001 was collected retrospectively in Tasmania. The enhanced data set fields are shown in Table 2.

Table 1. Recommendations for pneumococcal vaccination, Australia, 2001

Vaccine	23-valent polysaccharide vaccine	7-valent conjugate vaccine
Pneumococcal serotypes	1, 2, 3, 4, 5, 6B, 7F, 8, 9N, 9V, 10A, 11A, 12F, 14, 15B, 17F, 18C, 19A, 19F, 20, 22F, 23F, 33F	4, 6B, 9V, 14, 18C, 19F, 23F
Date implemented	1998	July 2001
Target populations	<ul style="list-style-type: none"> All individuals aged 65 years and over Individuals with asplenia Immunocompromised patients Aboriginal and Torres Strait Islander people aged 50 years and over Immunocompetent individuals with chronic illness including chronic cardiac, renal or pulmonary disease, diabetes and alcohol-related problems 	<p>Tier 1: Indigenous children less than 5 years living in Central Australia</p> <p>Tier 2: Indigenous children aged less than 2 years particularly in rural and remote settings</p> <p>Tier 3: Indigenous children under 2 years living in other settings</p> <ul style="list-style-type: none"> Non-Indigenous children less than 2 years living in Central Australia Non-Indigenous children with conditions predisposing to pneumococcal infection
Data source	NHMRC Immunisation Handbook 7th edition, 2000	ATAGI recommendations, 2001

Table 2. Enhanced invasive pneumococcal disease surveillance data supplied by States and Territories used in this report

Data type	Data fields
Demographic	Date of birth Age Indigenous status: (Aboriginal, Torres Strait Islander, Aboriginal and Torres Strait Islander, other, unknown) Location (optional) Postcode
Risk factors	Premature birth (gestation less than 37 weeks) Congenital abnormality Anatomical or congenital asplenia Immunocompromised (e.g. HIV, lymphoma, transplant, multiple myeloma, nephrotic syndrome etc.) Chronic illness (e.g. cardiac disease, pulmonary disease, CSF leak, diabetes)
Clinical data	Clinical presentation (pneumonia, meningitis, bacteraemia, other, unknown) Date of onset Death due to IPD
Microbiology data	Specimen collection date Date laboratory results issued (report date) Date notification received Specimen type (blood, CSF, pleural fluid, joint fluid, other sterile site). Specimen culture positive or <i>S. pneumoniae</i> detected by nucleic acid tests Antibiotic susceptibility (penicillin, cefotaxime/ceftriaxone) Pneumococcal serotype
Vaccination history	Source of vaccination history (validated, not validated, information not collected) Pneumococcal vaccination dates, number of doses and type of vaccine Vaccination status: fully vaccinated for age, partially vaccinated for age, not vaccinated, not applicable, unknown

The rates presented in this report were calculated using population data produced by the Australian Bureau of Statistics (ABS). The Estimated Resident Population (ABS 3201.0) in each State and Territory and in Australia as a whole, as at 30 June 2001, was used as the denominator in rate calculations. Estimates of the Indigenous Australian population were based on projections from the 1996 census (ABS 3231.0). The ABS calculated projections based on assumptions about future births deaths and migrations in the Indigenous population and a 'low' and 'high' estimate were reported. The 'low' estimate has been used in this report, which is consistent with the reporting of other national communicable diseases.

Results

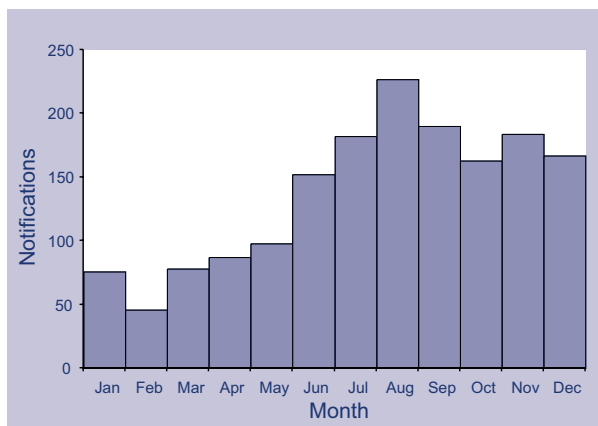
Notifications to the National Notifiable Diseases Surveillance System

There were 1,681 notifications of IPD to the NNDSS in 2001. The number of notifications and the notification rate per 100,000 population of IPD in Australian States and Territories are shown in Table 3. Since legislation to make IPD a notifiable disease came into force at different times during 2001, the number of cases in some jurisdictions may be under-estimated.

While the largest number of cases were found in New South Wales, Queensland and Victoria, the highest rates were found in the Northern Territory, which had a rate 5.6 times the national rate. Notifications of pneumococcal disease to NNDSS by month of report are shown in Figure 1. There was a peak of IPD in the second half of the year in late winter and early spring, with the largest numbers of notifications being in August 2001 (227 cases, Figure 1).

The geographical distribution of IPD by Statistical Division (Map), shows the rates of IPD in each Statistical Division shaded to indicate areas above >10% and <10% below the national rate (8.6 per 100,000 population). The highest rates occur in the Northern Territory, Far North Queensland and Western Australia. Areas of above average incidence were also noted in Queensland (South West, Wide Bay, Moreton and Brisbane), New South Wales (Hunter and the Central West), Victoria (Gippsland, Barwon and Loddon) and Tasmania (Northern and Mersey Lyall).

Figure 1. Notifications of invasive pneumococcal disease, Australia, 2001, by month of report



In Australia in 2001, IPD was largely a disease of the very young and very old. The highest rates of disease were among children aged less than 5 years (47.3 cases per 100,000 population) and adults aged more than 85 years (38.7 cases per 100,000 population, Figure 2). Among children aged less than 5 years, the highest rates of disease were in those aged 12 months (males 103 and female 91 cases per 100,000 population). Overall, there were more male cases and there was a male to female ratio of 1.2:1.

Figure 2. Notification rates of invasive pneumococcal disease, Australia, 2001, by age and sex

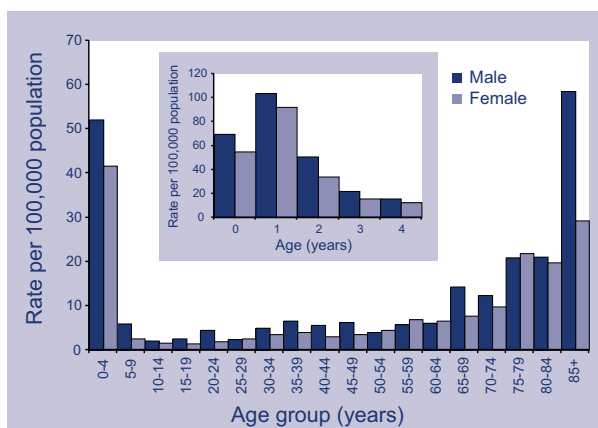
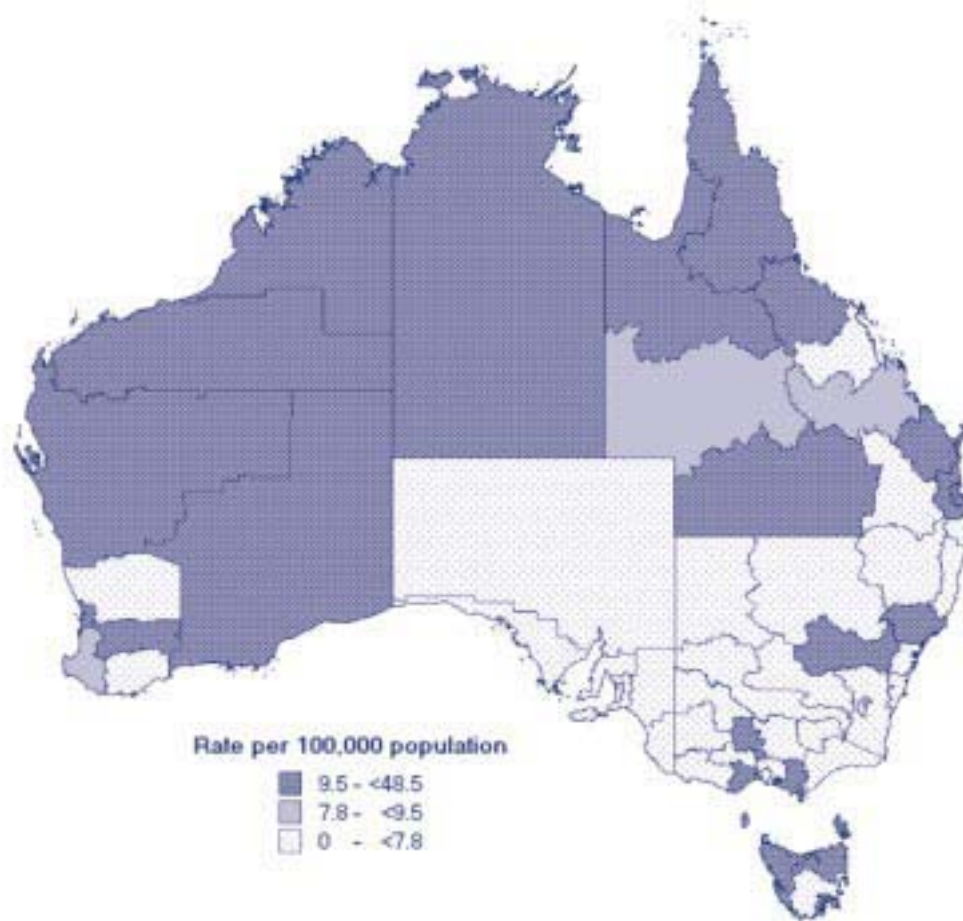


Table 3. Notifications and notification rate per 100,000 population, invasive pneumococcal disease, Australia, 2001*

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Notifications	18	434	97	425	114	61	327	205	1,681
Rate per 100,000 population	5.6	6.6	48.5	11.7	7.5	12.9	6.8	10.8	8.6

* Notifications to the NNDSS based on onset date between 1 January 2001 and 31 December 2001. Data received as at 6 September 2002 and subject to revision.

Map. Notification rates of invasive pneumococcal disease, Australia, 2001, by Statistical Division of residence



Enhanced surveillance for invasive pneumococcal disease in 2001

Additional data were available on cases from the Northern Territory, Victoria, South Australia, Western Australia, Tasmania and the metropolitan areas of New South Wales (from Newcastle in the north to Wollongong in the south). This enhanced surveillance covered 13,947,962 people or 71.8 per cent of the Australian population (based on mid-year 2001 population estimates).

The number of cases in the enhanced IPD datasets from most states and territories were similar to the total number of IPD notifications to NNDSS. In New South Wales, the total in the enhanced dataset, which covered only metropolitan areas of the State, was higher than the total number of notifications. The NNDSS total for New South Wales is an underestimate, probably because of delays in implementing legislation making IPD a notifiable disease in that jurisdiction. In all, enhanced data were available for 1,446 cases of pneumococcal disease or 86 per cent of the notified cases.

In the following analysis, we have combined the enhanced data from all jurisdictions to describe the epidemiology of invasive pneumococcal disease in Australia. This extrapolation should be interpreted with caution given that there were variations in data collection between jurisdictions in 2001 and data were not available for Queensland, the Australian Capital Territory or rural New South Wales.

Demographics

The demographic profile of cases reported in enhanced pneumococcal surveillance schemes is shown in Table 4. In the enhanced surveillance datasets there were more cases of IPD among males than females (national male to female ratio of 1.4:1). Children aged less than 5 years made up a significant proportion of cases (35%), although this varied by jurisdiction with 59 per

cent of cases in South Australia in this age group compared with 18 per cent in Tasmania (Table 4). These variations may reflect differences in clinical practice or in the ability to capture all cases, especially in the adult population, in those jurisdictions where this was the first year of notification (e.g. in South Australia).

Indigenous status was well reported in all jurisdictions although the accuracy of the data may be questioned due to the manner of acquisition (e.g. in New South Wales the status is obtained from medical records, rather than from individuals and may underestimate indigenous identification). Jurisdictions with large Indigenous populations, such as the Northern Territory reported more than two-thirds of IPD cases occurred in Indigenous people. The estimated rate of IPD in Indigenous Australians was 120 cases per 100,000 population in the Northern Territory and 60 cases per 100,000 population in Western Australia. Rates of IPD in Indigenous children, aged less than 5 years were 483 and 256 cases per 100,000 population in the Northern Territory and Western Australia respectively.

Clinical presentation

The clinical presentation of IPD was reported for 1,415/1,446 (98%) of cases. Clinical presentations were coded as pneumonia, meningitis, bacteraemia, other or unknown. Pneumonia was defined as blood culture positive for *S. pneumoniae* with consistent clinical and/or radiological signs of pneumonia. Meningitis was defined as CSF and/or blood culture positive with supportive CSF findings. Bacteraemia was defined as blood culture positive with no localising signs. 'Other' included detection of *S. pneumoniae* in pleural, peritoneal and joint fluid. More than one clinical presentation could be recorded for each case.

The clinical presentations reported by enhanced surveillance in 2001 are shown in Table 5.

Table 4. Demographic profile of invasive pneumococcal disease cases reported by enhanced surveillance systems, metropolitan New South Wales, the Northern Territory, South Australia, Tasmania, Victoria and Western Australia, 2001, by jurisdiction

Data		NSW (metro)	NT	SA	Tas	Vic	WA	Total
Number of records		643	99	116	62	322	204	1,446
Sex	Male:female	1.4:1	1.8:1	1.4:1	1.6:1	1.2:1	1.2:1	1.4:1
Age	<5 years	205 32%	33 33%	69 59%	11 18%	111 34%	83 41%	512 35%
	5 to 64 years	217 33%	61 61%	26 22%	29 47%	116 36%	80 39%	529 37%
	>65 years	221 35%	5 5%	21 18%	22 35%	95 30%	41 20%	405 28%
Indigenous status	Indigenous	9 1.4%	68 69%	3 3%	0 0%	2 0.6%	37 18%	119 8%
	Non-indigenous	634* 98.6%	30 30%	91 78%	47 76%	280 87%	160 78%	1,242 86%
	Unknown	0	1 1%	22 19%	15 24%	40 12%	7 4%	85 6%
	Estimated indigenous population 2001	121,142† (1.8%)	56,364 (28%)	24,313 (1.6%)	16,644 (3.5%)	24,586 (0.5%)	61,505 (3.2%)	427,094 (2.2%)

* Based on medical records

† The estimated indigenous population for New South Wales is the total for the State.

Table 5. Clinical presentations of invasive pneumococcal disease, metropolitan New South Wales, the Northern Territory, South Australia, Tasmania, Victoria and Western Australia, 2001, by jurisdiction

Data	No. of cases (% of cases)	NSW (metro)	NT	SA	Tas	Vic	WA	Total
Clinical presentation*	Pneumonia	365 57%	78 78%	49 42%	36 58%	157 49%	106 52%	791 56%
	Meningitis	40 6%	7 7%	7 6%	6 10%	13 4%	13 6%	86 6%
	Bacteraemia	223 35%	14 14%	58 50%	6 10%	138 43%	70 34%	509 36%
	Other	13 2%	1 1%	8 7%	3 5%	49 15%	9 4%	83 6%
	Unknown	2 0.3%	–	1 1%	13 21%	–	15 7%	31 2%

* Totals may exceed patient total and percentages exceed 100 per cent since patients may have had more than one type of clinical presentation.

Pneumonia was the most common clinical presentation, particularly among the elderly, while bacteraemia and meningitis were more common among children. The rate of pneumococcal pneumonia in the enhanced surveillance population was 5.7 cases per 100,000 population. The rate of pneumococcal bacteraemia was 3.6 cases per 100,000 population and pneumococcal meningitis was 0.6 per 100,000 population. The relative proportions of the clinical presentations of IPD in children aged less than 5 years were different in Indigenous and non-Indigenous children (Table 6).

Indigenous children presented with pneumococcal pneumonia more frequently than non-Indigenous children, while non-Indigenous children presented with bacteraemia more frequently than Indigenous children.

The case fatality rate by age group and Indigenous status is shown in Table 7. With the exception of South Australia, there was a higher case fatality rate in elderly patients with IPD, aged more than 65 years, than in children aged less than 5 years. The case fatality rate for Indigenous people with IPD was comparable to that in non-Indigenous people.

Risk factors for pneumococcal disease

Data on relevant risk factors were collected on 1,376/1,446 (95%) cases of IPD in enhanced surveillance systems. Overall, 749 (55%) cases

had a recognised risk factor for pneumococcal disease. The most common of these was chronic illness, which included chronic respiratory, cardiac and renal disease. Immunocompromising conditions such as long-term immunosuppressant use were common among IPD patients. Risk factor categories were defined by the national surveillance working party. Other risk factors were recorded but varied between jurisdictions. More than one risk factor could be recorded for each patient. The proportion of cases with an identified risk factor was significantly higher in cases aged 5 years and above (45%) compared with cases aged less than 5 years (15%, $\text{Chi}^2 = 68.5$, $p < 0.0001$). The proportions of patients in each age group with an identified risk factor varied widely between jurisdictions. The method of ascertainment of risk factor data varied from jurisdiction to jurisdiction with some interviewing cases directly and others dependent on medical records. The frequency of risk factors for IPD in Indigenous people and different age groups are shown in Table 8.

The rates of premature birth and chronic illness were significantly higher in Indigenous children with IPD compared with non-Indigenous children. Chronic illness was also more frequent in older Indigenous people with IPD than in non-Indigenous patients, but the proportion immunocompromised was higher in older non-Indigenous IPD cases than in Indigenous cases (Table 8).

Table 6. Clinical presentations of invasive pneumococcal disease in Indigenous and non-Indigenous children aged less than 5 years, Australia, 2001*

	Number of cases (%)		
	Indigenous (n=36)	Non-Indigenous (n=255)	Significance of difference [†]
Pneumonia	26 (72%)	81 (31%)	$p < 0.0001$
Meningitis	4 (11%)	19 (7%)	ns
Bacteraemia	7 (19%)	149 (58%)	$p < 0.0001$
Other invasive disease	0 (0%)	26 (10%)	$p < 0.05$

* Analysis did not include New South Wales

† Chi^2 with Yates correction

ns Not significant

Table 7. Case fatality rates for invasive pneumococcal disease, metropolitan New South Wales, the Northern Territory, South Australia, Tasmania, Victoria and Western Australia, 2001, by jurisdiction

Data	NSW (metro)	NT	SA	Tas	Vic	WA	Total
Total cases	643	99	116	62	322	204	1,446
Total deaths	75	3	9	3	19	16	125
Total case fatality rate (%)	11.6	3	7.7	4.8	5.9	7.8	8.6
Deaths in aged < 5y/ Total cases aged <5y (%)	1/205 0.5	0/33 0	1/69 1	0/11 0	0/111 0	3/83 3.6	5/512 1
Deaths in aged >65y/ Total cases aged >65y (%)	59/221 27	2/5 40	5/21 24	0/22 0	10/95 10.5	6/41 14.6	82/405 20
Deaths in Indigenous people Total Indigenous cases (%)	Nd	2/68 3	0/3 0	0/0 0	0/2 0	3/37 8	5/110 4.5
Death in non-Indigenous/ Total non-Indigenous + 'unknown' cases (%)	Nd	1/31 3	9/113 8	3/62 4.8	19/320 5.9	13/167 7.8	45/693 6.5

Nd Data not available

Table 8. The frequency of risk factors for invasive pneumococcal disease, Australia, 2001, by age group and Indigenous status*

	Cases aged less than 5 years			Cases aged 5 years or more		
	Indigenous (n=32)	Non-Indigenous (n=245)	Significance of difference [†]	Indigenous (n=63)	Non-Indigenous (n=399)	Significance of difference [†]
Premature birth	6 (19%)	10 (4%)	p<0.005	NA	NA	ns
Congenital abnormality	1 (3%)	21 (9%)	ns	0	5 (1%)	ns
Asplenia	0	0	–	0	3 (0.7%)	ns
Immunocompromised	2 (6%)	21 (8%)	ns	6 (10%)	81 (20%)	p<0.005
Chronic illness	12 (38%)	30 (12%)	p<0.001	41 (65%)	168 (42%)	p<0.005

* Analysis did not include New South Wales

† Chi² test with Yates correction

NA Not applicable

ns Not significant

Pneumococcal serotypes causing disease in Australia

The pneumococcal serotypes were identified in 1,179 (82%) of the 1,446 cases under enhanced surveillance in 2001. Overall, 75% (889/1,179) of serotypes were those in the 7-valent conjugate pneumococcal vaccine and 93% (1,097/1,179) were those in the 23-valent polysaccharide pneumococcal vaccine. The frequency of pneumococcal serotypes was analysed in the target group for the 7-valent vaccine (children aged less than 2 years) and the target group for the 23-valent vaccine (those aged more than 2 years, Table 9).

Overall, a large majority (126/154, 82%) of pneumococcal serotypes reported in children aged less than 2 years were covered by the 7-valent conjugate vaccine. Among all other age groups, 463/513 (90%) of pneumococcal isolates were serotypes covered by the 23-valent polysaccharide pneumococcal vaccine.

A significantly smaller proportion of serotypes in Indigenous children aged less than 2 years (12/22 55%), were serotypes contained in the 7-valent conjugate vaccine compared with serotypes isolated in non-Indigenous children (114/132, 86%, $p < 0.005$). Likewise, a significantly smaller proportion of isolates from Indigenous people aged more than 2 years with IPD (62/80 74%), were contained within the 23-valent pneumococcal vaccine, compared with isolates from non-Indigenous people (401/433, 93%, $p < 0.0001$, Table 9).

Vaccination status of IPD cases

Data on pneumococcal vaccination were available for only a minority of cases of IPD in 2001. No data were available from New South Wales and in only a minority of cases in Western Australia and Tasmania. Data from the Northern Territory and Victoria indicate that the majority of cases were not vaccinated (Table 10).

The majority of cases who had received pneumococcal vaccine had received the 23-valent polysaccharide vaccine, while a small number had received the 7-valent conjugate vaccine. Since vaccination with the conjugate vaccine commenced in Australia in July 2001 and was targeted at specific groups of children (Table 1), these data represent a baseline against which to compare data in future years when conjugate vaccination becomes more widespread.

Three cases of IPD in Victoria occurred in children aged less than 2 years who were fully vaccinated for age. Only one of the children was verified as having received the conjugate vaccine. This non-Indigenous child was 12 months of age at the time of disease onset and had *S. pneumoniae* serotype 14 isolated from blood culture. The child had biliary atresia. The other two Victorian children were aged 9 months and 17 months and the vaccine history was not verified. One had a serotype 14 isolated from blood culture and the other a serotype 19A also isolated from blood culture. No risk factors were identified in these two children.

Table 9. The proportion of pneumococcal serotypes isolated from cases of invasive pneumococcal disease, which were serotypes in the 7-valent and 23-valent pneumococcal vaccine, the Northern Territory, South Australia, Tasmania, Victoria and Western Australia, 2001, by age and Indigenous status*

	Cases aged less than 2 years with serotypes in 7-valent conjugate vaccine			Cases aged 2 years or more serotypes in 23-valent vaccine		
	Indigenous (n=32)	Non-Indigenous (n=245)	Significance of difference†	Indigenous (n=63)	Non-Indigenous (n=399)	Significance of difference†
Northern Territory	8/12 67%	12/12 100%	ns	38/53 72%	15/16 94%	ns
South Australia	2/2 100%	34/36 94%	ns	0/1 0%	50/54 93%	ns
Tasmania	0/0	3/5 60%	–	0/0 87%	26/30	–
Victoria	0/0	53/61 87%	–	2/2 100%	182/195 93%	ns
Western Australia	2/8 25%	12/18 67%	ns	22/24 92%	128/138 93%	ns
Total	12/22 55%	114/132 86%	p<0.005	62/80 76%	401/433 93%	p<0.0001

* Data for New South Wales not available

† Chi² test with Yates correction

ns Not significant

Table 10. Vaccination status of invasive pneumococcal disease cases, the Northern Territory, South Australia, Tasmania, Victoria and Western Australia, 2001, by age group and jurisdiction

Vaccination status	Invasive pneumococcal disease cases aged less than 2 years of age					Total
	NT	SA	Tas	Vic	WA	
Fully vaccinated for age	0	0	0	3	0	3
Partially vaccinated for age	1	2	0	1	0	4
Not vaccinated	43	45	2	67	5	163
Unknown	–	–	9	11	29	49
Vaccine						
7-valent	1	2	–	4	–	7
23-valent	0	0	–	0	–	0
Unknown	0	0	–	0	–	0
Vaccination status	Invasive pneumococcal disease cases aged 2 years of age or more					Total
	NT	SA	Tas	Vic	WA	
Fully vaccinated for age	18	4	1	24	1	48
Partially vaccinated for age	6	9	0	2	0	17
Not vaccinated	50	35	3	140	80	308
Unknown	0	17	36	73	56	182
Vaccine						
7-valent	1	0	0	0	0	1
23-valent	22	4	1	21	1	49
Unknown	1	9	0	5	0	15

Details of the 48 cases of IPD that occurred in individuals aged 2 years and over, who were reported as fully vaccinated are shown in Table 11.

Vaccine failure with the 23-valent vaccine, where polysaccharide vaccination was confirmed and disease was caused by one of the 23-valent vaccine serotypes was suggested in 26 cases. Surveillance of vaccine failures is continuing.

Antibiotic resistance in pneumococcal cases

Antibiotic susceptibilities of *S. pneumoniae* isolates from 1,245 patients were tested against penicillin and from 1,041 patients against third-generation cephalosporin (cefotaxime or ceftriaxone, Table 12).

Reduced susceptibility to penicillin was found in 147/1,245 (12%) of all isolates tested, with 38 (3%) isolates fully resistant and 109 (9%) isolates with 'intermediate' resistance (Table 13). There was a variable prevalence in penicillin resistance by jurisdiction with Western Australia reporting 17 per cent of isolates with reduced susceptibility and Tasmania reporting all isolates as fully susceptible. Reduced susceptibility to third-generation cephalosporins was found in 56 (5%) of all isolates tested. Only one per cent (10 isolates) were reported as 'fully resistant', while 46 (4%) had

intermediate resistance. All isolates from Tasmania were fully sensitive to the cephalosporins, while the Northern Territory reported 28 per cent of their isolates as having intermediate resistance.

The characteristics of cases with reduced susceptibility to antibiotics were analysed for all jurisdictions except metropolitan New South Wales. Pneumococcal serotypes associated with reduced penicillin susceptibility were also analysed. The results are shown in Table 13.

While the overall prevalence of penicillin resistance is low, there is evidence in some jurisdictions that penicillin resistance is more frequent in Indigenous cases and children. An analysis of patients with reduced susceptibility to third generation cephalosporins revealed that all such patients also had disease caused by vaccine serotypes with reduced susceptibility to penicillin. In the Northern Territory, 3/5 cases with reduced cephalosporin susceptibility were Indigenous children aged less than 5 years while 3/9 cases in Western Australia were Indigenous. Two of these were aged less than 5 years. All other cases with reduced cephalosporin susceptibility were non-Indigenous. One third (25/76) of the drug resistant isolates were serotype 9V, 21 per cent (16/76) were serotype 19F and 12% (9/76) were serotype 6B.

Table 11. Details of the 48 cases of invasive pneumococcal disease which occurred in recipients of the 23-valent pneumococcal vaccine, the Northern Territory, South Australia, Tasmania, Victoria and Western Australia, 2001, by jurisdiction*

	NT	SA	Tas	Vic	WA
Number	18	4	1	24	1
Age range (years)	>= 60 y	>=46y	85	>=24y	75
Indigenous	17/18	0/4	0/1	0/24	1/1
Risk factors present	15/18	3/4	0/1	18/18	0/1
23-valent vaccination confirmed	18/18	4/4	1/1	19/24	0/1
Serogroups (%) in 23-valent vaccine	10/17	4/4	No serotype information	16/17	1/1
Number of vaccine failure [†]	10	4	0	12	0

* Data not available for New South Wales

† Where polysaccharide vaccination was confirmed and disease was caused by a serotype in the 23-valent vaccine

Table 12. *S. pneumoniae* resistance to penicillin and third generation cephalosporins, metropolitan New South Wales, the Northern Territory, South Australia, Tasmania, Victoria and Western Australia, 2001, by jurisdiction

Antibiotic	Susceptibility	NSW	NT	SA	Tas	Vic	WA	Total
Penicillin	Resistant (n)	28	1	0	0	4	5	38
	(%)	4	1	0		3	3	3
	Intermediate (n)	53	8	15	0	7	26	109
	(%)	8	8	13		5	14	9
Susceptible (n)		552	89	99	63	138	158	1,098
	(%)	88	91	87	100	92	83	88
Total tested (n)		633	98	114	62	149	189	1,245
(%)								
Cefotaxime/ ceftriaxone	Resistant (n)	8	0	1	0	1	0	10
	(%)	1	0	1	0	1	0	1
	Intermediate (n)	30	5	2	0	0	9	46
	(%)	5	28	3	0	0	5	4
Susceptible (n)		588	13	63	59	82	180	985
	(%)	94	72	96	100	99	95	95
Total tested		626	18	66	59	83	189	1,041

Penicillin resistance was defined as 'fully resistant' (MIC > 1mg/L) or intermediate (MIC 0.1–1.0mg/L). Ceftriaxone resistance was defined as MIC >1mg/L or intermediate as MIC 0.1–1mg/L.

Table 13. Characteristics of invasive pneumococcal disease cases with reduced susceptibility to penicillin and cephalosporins, Australia*, 2001

	Reduced susceptibility	
	Penicillin	3rd generation cephalosporins
Total number of cases with reduced susceptibility [†]	66	18
No. aged less than 5 years with reduced susceptibility/ Total tested aged less than 5 years (%)	30/188 16%	6/153 4%
No. Indigenous cases with reduced susceptibility/ Total Indigenous tested	14/104 13%	6/43 14%
Proportion of serotypes in 7-valent vaccine	62/66 94%	18/18 100%
Proportion of serotypes in 23-valent vaccine	66/66 100%	18/18 100%
Proportion of cases vaccinated (all with 23-valent pneumococcal vaccine)	9/29 31%	4/9 44%

* Data not available for New South Wales

† Includes cases resistant and with intermediate susceptibility as defined above.

Discussion

This report is the first attempt to describe the epidemiology of invasive pneumococcal disease in Australia from a national perspective. The totals and rates described are likely to be underestimates as the capture of cases through the NNDSS was incomplete in this the first year that IPD was a nationally notifiable disease. In this early period of surveillance, there may have been a failure to report all diagnosed cases and to collect appropriate clinical specimens. It would appear that rates of pneumococcal disease in Australia were lower than in the USA in 2000 (20.7 cases per 100,000 population).⁷

Invasive pneumococcal disease in Australia is generally a disease of the very young and the very old and with a continuing high rate of disease in Indigenous children. There appears to be a geographical effect on disease incidence with the highest rates among Indigenous children in the inland desert areas of the country.¹ The clinical presentations of pneumococcal disease were typical of the age groups affected, however, pneumonia was a more common manifestation in Indigenous children than non-Indigenous children. The overall case fatality rate of 8.6 per cent represents a crude rate of 0.89 per 100,000 population. This estimate is higher than estimates of 0.3 fatalities per 100,000 population from the Australian Institute of Health and Welfare mortality database⁸ and well below the projected death rate for pneumococcal disease in the USA (2.3 per 100,000 population).⁷ Of importance, is the observation that case fatality rates were not significantly higher in Indigenous Australians, despite the high rates of disease and risk factors in that community.

More than half of all cases of pneumococcal disease in Australia occurs in people with recognised risk factors. The proportion of patients with risk factors is larger in older age groups. In the Northern Territory where a more comprehensive set of risk factors such as smoking (active or passive), previous pneumonia or IPD disease or excessive alcohol consumption was recorded, 83 per cent of cases were identified as having a risk factor. These data highlight that better strategies are needed to target and successfully immunise those with recognised risk factors. Some risk factors not included in the National Health and Medical Research Council guidelines,⁹ include smoking and excessive alcohol consumption. In some populations, universal immunisation may be the most effective method of disease control.

While a large proportion of pneumococcal serotypes causing disease in Australia are contained in the 7-valent and 23-valent vaccines, this proportion was significantly lower in Indigenous people. Among Indigenous children with pneumococcal disease aged less than 2 years, only 55 per cent had disease caused by serotypes in the 7-valent vaccine, while among older Indigenous people with IPD only 76 per cent had disease due to serotype of *S. pneumoniae* in the 23-valent vaccine. Cross-reactive immunity induced by vaccine serotypes has been noted to confer immunity to non-vaccine serotypes. Otitis media caused by serotype 6A was reduced by vaccination with the 7-valent conjugate vaccine which contains serotype 6B.¹⁰ The proportion of disease caused by non-vaccine serotypes of *S. pneumoniae* should also be closely monitored, especially in Indigenous communities.

In the USA, historical changes in pneumococcal serotype distribution over 70 years (1928 to 1978) have recently been analysed.¹¹ The authors found a significant decrease in the proportion of 'epidemic' pneumococcal serotypes 1, 2, 3 and 5, and an increase in serotypes contained in the 7-valent vaccine. This trend is thought to be explained by changes in antibiotic use, socioeconomic conditions, an ageing population and blood-culturing practices. As the 7-valent vaccine becomes more widely used, there may be strong selective pressure on the circulation of vaccine serotypes. Although replacement by non-vaccine serotypes in vaccine recipients of a 9-valent pneumococcal conjugate vaccine has been reported,¹² no increase in non-vaccine serotypes causing disease was observed in the 3.5 year 7-valent vaccine efficacy trial.⁶ Longer-term surveillance of pneumococcal serotypes is required to confirm these preliminary findings.

The level of reduced susceptibility to penicillin among pneumococcal isolates collected in this study (12%) is similar to that recorded for invasive isolates in Australia in 1997 (13%).³ The level of reduced susceptibility to ceftriaxone (5%) was also similar to that in the same study (6%). Changes in treatment practice over this period and differences in the sample population, site of isolation, and diagnostic methods between the two studies should be noted. The levels of antibiotic resistance in this study is also markedly lower than in the USA, where the proportion of penicillin resistant isolates increased between 1995 and 1998, from 21 per cent to 25 per cent, the proportion resistant to cefotaxime increased from 10 per cent to 14 per cent and multi-drug resistance increased from 9 per cent to 14 per cent.¹³

Antibiotic resistance in the pneumococci has been increasing worldwide³ and the development of multi-resistance (penicillin, macrolides, tetracyclines and cotrimoxazole) have posed a threat to treatment. Infections with penicillin resistant *S. pneumoniae* in Australia, have been shown to result in longer hospitalisation and longer resolution times, further resulting in higher treatment costs.¹⁴ Control of penicillin resistance among invasive pneumococcal isolates may be influenced by reducing the use of antibiotics which has been shown to reduce the carriage rates of resistant pneumococci.¹⁵ The higher rates of resistance among Indigenous children is a cause for concern, however most isolates with reduced antibiotic susceptibility in the present study were vaccine serotypes contained in the 7-valent vaccine and all were serotypes in the 23-valent vaccine. The impact of widespread vaccination is expected to be important in controlling the spread of drug resistant pneumococcal disease.

Although the pneumococcal vaccination history of the majority of cases reported in the enhanced surveillance was unknown, only a small number of cases were fully vaccinated for age. There was only one vaccine failure reported with the 7-valent vaccine during this period.

Generally, this report represents the epidemiology of pneumococcal disease on the eve of the introduction of the conjugate vaccine in Australia. In the coming years, it will be important to monitor the impact of the 7-valent conjugate vaccine on the epidemiology of pneumococcal disease in Australia. The vaccination schedule (Table 1) focuses on high-risk Indigenous children with the primary goal of reducing the incidence of disease in this group. Enhanced surveillance for pneumococcal disease in all Australian jurisdictions from July 2001, will measure changes in clinical presentation, serotype frequency and the prevalence of antibiotic resistance. Additionally, monitoring disease in those age groups recommended for the 23-valent vaccine will be important, as will the nationwide disease rates in other age groups to better guide 23-valent vaccine strategies and recommendations.

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Invasive pneumococcal disease in North Queensland, 2001

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Abstract

This report provides information on the 93 locally-acquired cases of invasive pneumococcal disease (IPD) notified in children and adults in north Queensland in 2001. Indigenous people represented 38 (41%) cases. Almost half (45) of all cases were in children under 15 years of age, 20 (44%) of these were in children less than 2 years of age and 20 (44%) in Indigenous children. Five severe cases of IPD occurred, all in non-Indigenous children under 2 years of age. Nine (10%) of the isolates from cases, mainly in young children, had some level of resistance to penicillin. Pneumococcal vaccination programs (including the Indigenous 'elderly and at-risk' adult program and the paediatric 'Indigenous and medically at-risk' conjugate vaccine program) are in place in Queensland although the vaccine is not currently funded for other at-risk groups. If vaccine recommendations had been adhered to in a timely fashion, two of the cases in children and one third (16) of the cases in adults that occurred in 2001 could potentially have been prevented. *Commun Dis Intell* 2002;26:520–524.

Keywords: pneumococcal disease, pneumococci, vaccination, antibiotic resistance

Introduction

The 23-valent pneumococcal polysaccharide vaccine (23vPPV) was included in a statewide vaccination program in Queensland for at-risk Indigenous adults that began in 1998 but the vaccine had been used in some parts of north Queensland since the mid-1990s.¹ The efficacy of the vaccine is 50 per cent to 80 per cent in 'at risk' individuals (i.e. the elderly and those with chronic diseases).^{2,3}

The 7-valent pneumococcal conjugate vaccine (7vPCV) was licensed for use in Australia in early 2001. The vaccine is approximately 97 per cent effective in preventing invasive disease caused by vaccine serotypes.⁴ A previous study in north Queensland indicated the serotypes in the 7-valent vaccine accounted for approximately 62 per cent and 88 per cent of the isolates from IPD cases in Indigenous and non-Indigenous children under 5 years of age respectively.⁵ This suggests the vaccine should prevent approximately 60 per cent and 85 per cent of cases of IPD in Indigenous and non-Indigenous children in the region. The vaccine became available in north Queensland for Indigenous children up to 2 years

of age and other medically at-risk children up to 5 years of age in the latter part of 2001. Because of the need for education and training, staggered implementation took place in different parts of the region from July to September 2001.

IPD has been a notifiable disease in Queensland since 1996. This report describes cases of IPD in north Queensland in 2001, and examines whether cases were preventable according to vaccine recommendations.

Methods

A case of IPD is defined as an acute systemic febrile illness and the isolation of *Streptococcus pneumoniae* from a normally sterile site. Laboratories routinely notify the Tropical Public Health Unit (TPHU) of any such isolation; TPHU staff follow-up each notification and administer a standardised questionnaire to each case or his or her guardian. The vaccination status of each case is checked, where applicable, on the statewide computerised immunisation database. Each invasive isolate is serotyped, and the antibiotic susceptibilities determined, by the Public Health Microbiology Laboratory, Queensland Health Scientific Services.

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Results

There were 97 cases of IPD notified in north Queensland in 2001. Three cases were acquired in Papua New Guinea and one case elsewhere in Queensland. Of the remaining 93 cases, 45 cases (48%) were in children <15 years of age, and 38 cases (41%) were in Indigenous people.

Of the 45 cases that occurred in children, 20 cases (44%) were in children under 2 years of age and 20 cases (44%) were in Indigenous children. Forty-four of the isolates from children were serotyped and in total 31 cases (70%) had serotypes that are included in 7vPCV. However, only 11 (55%) of the 20 Indigenous children had isolates with a serotype included in 7vPCV, much lower than in non-Indigenous children (83%). Table 1 illustrates the percentage of isolates in Indigenous and non-Indigenous children in the less than 2 years and 2–14 years age groups, that had a serotype included in 7vPCV.

All but one of the Indigenous children with IPD had either pneumonia or bacteraemia; there were no cases of pneumococcal meningitis in this group. The remaining Indigenous child, aged 5 years, had pneumococcal septic arthritis of a hip. There were 3 cases of pneumococcal meningitis among the non-Indigenous children. All three were less than 2 years of age and all the isolates had serotypes included in 7vPCV.

Although there were no deaths among the children, five of the cases could be classified as 'severe' based upon associated complications or sequelae (Table 2). The isolate from a 5-month-old child with severe pneumococcal pneumonia

had intermediate susceptibility to penicillin (MIC=1.0 mg/L) and was resistant to cotrimoxazole. Otherwise, the isolates from the remaining children with severe IPD were sensitive to penicillin and other antibiotics.

Based upon current recommendations (and taking the timing of implementation of the vaccination program into account), only two of the cases in children could have been prevented had 7vPCV been administered in a timely fashion. Neither a 13-month-old Indigenous child with an IPD onset in late October, nor a 2.75-year-old non-Indigenous child with leukaemia whose onset was in early September, had received any doses of 7vPCV.

Of the 48 cases that occurred in adults 15 years of age or older, 18 cases (38%) were Indigenous adults. Forty-four of the isolates from adults were serotyped. Thirty-six cases (82%) had serotypes included in 23vPPV: 14 (78%) of those from Indigenous adults and 22 (85%) of those from non-Indigenous adults (Table 1).

Fifteen cases (31%) occurred in adults who had previously received 23vPPV: 9 (50%) of the affected Indigenous adults and 6 (20%) of the non-Indigenous adults (Table 3). Of the 9 Indigenous adults, five were vaccine failures and the features of these cases are presented in Table 4. The remaining four episodes of IPD were caused by non-vaccine serotypes (16F x2, 23A & 38). Five of the 6 cases in vaccinated non-Indigenous adults were vaccine failures.

Table 1. Cases of invasive pneumococcal disease and number of isolates in each group with a serotype included in the age-appropriate pneumococcal vaccine

Age group	Indigenous cases	Non-Indigenous cases	Total cases
Child	6 6 (100%) isolates in 7vPCV	14 12 (86%) isolates in 7vPCV	20
	14 5 (36%) isolates in 7vPCV	11 8 (73%) isolates in 7vPCV*	25
Adult	18 14 (78%) isolates in 23vPPV	30 22 (85%) isolates in 23vPPV†	48

* 1 isolate not typed in this group.

† 4 isolates not typed in this group.

Table 2. Features of the severe cases of invasive pneumococcal disease in children in north Queensland, 2001

Age (months)	Ethnicity	Diagnosis	Complication/ sequelae	Length of hospital stay (days)	Serotype
5	Non-Indigenous	Pneumonia	Empyema	22	14
7	Non-Indigenous	Meningitis	Seizures (prolonged)	16	23F
12	Non-Indigenous	Pneumonia	Empyema	10	6A
13	Non-Indigenous	Pneumonia	Empyema	7	6A
18	Non-Indigenous	Meningitis	Deafness (profound)	10	14

Table 3. Vaccination status of adults with invasive pneumococcal disease in north Queensland, 2001

	Vaccinated		Unvaccinated	
	Vaccine failure	Non-vaccine serotype	Eligible	Non-eligible
Indigenous	5	4	5	4
Non-Indigenous	5	1	13	11
Total (%)	10 (21%)	5 (10%)	18* (38%)	15 (31%)

* 16 of these cases had serotypes included in 23vPPV

Table 4. Features of the previously vaccinated Indigenous adults who developed invasive pneumococcal disease caused by serotypes included in 23vPPV (i.e. vaccine failures), 2001

Age (years)	Sex	Interval since vaccination	Risk factors for IPD	Serotype
39.9	M	2.5 months	Diabetes, renal failure	23F
40.7	M	4.3 years	Alcohol abuse	11A
41.8	M	2.3 years	Alcohol abuse, liver disease	3
45.7	M	4.4 years	Diabetes, alcohol abuse	19A
52.0	M	5 months	Aged, alcohol abuse	19A

Over half (55%) of the affected adults that were unvaccinated were eligible for vaccine (Table 3) including 6 non-Indigenous adults aged over 65 years who had not been vaccinated according to recommendations. Most (89%) of the group who were eligible but had not been vaccinated had isolates with serotypes that were in 23vPPV. Therefore 16 (33%) of all cases of IPD in adults were potentially preventable had there been adherence to vaccine recommendations.

There were 3 deaths from IPD in the adult cases, a case-fatality of 6 per cent. All three were in unvaccinated non-Indigenous males: an immunosuppressed alcoholic 42-year-old with serotype 19F pneumococcal pneumonia, an 83-year-old with serotype 3 pneumococcal pneumonia and an 87-year-old with pneumococcal pneumonia (serotype unknown).

Nine (10%) of the 93 invasive isolates had some level of resistance to penicillin. One was fully resistant and eight had intermediate level resistance. Seven of these cases were in children, two of whom were Indigenous (Table 5). Of note, one child with intermediate level resistance required prolonged in-patient hospital care (22 days) because of severe pneumococcal pneumonia (complicated by empyema) and the child with the fully resistant isolate was the unvaccinated leukaemic child. The cases with isolates with some level of resistance to penicillin occurred throughout the year.

Discussion

Indigenous children were over-represented in the paediatric cases of IPD. Indigenous children constitute approximately 14 per cent of the total population under 15 years of age in north Queensland, yet 44 per cent of the total IPD cases in this population were in Indigenous children. There were two notable issues however, in relation to IPD and Indigenous children in 2001. Firstly, although severe disease has occurred not infrequently in previous years and 3 deaths were recorded in Far North Queensland over the previous 9 year period,⁵ there were no severe cases in Indigenous children in north Queensland during the surveillance period; virtually all had relatively mild pneumonia or bacteraemia.

Secondly, the current vaccine program will take several years to make a substantial impact on the incidence of IPD in Indigenous children unless there is considerable cross-protection to related serotypes not included in 7vPCV, and the vaccine markedly reduces nasopharyngeal carriage of pneumococci. This is because the pneumococcal vaccination program in Queensland targets Indigenous children under 2 years of age, while only 30 per cent of the Indigenous cases were aged under 2 years. Furthermore, only approximately 35 per cent of the isolates from Indigenous children aged 2 years or older are included in 7vPCV. Previous data collected on cases of IPD in Far North Queensland likewise showed only 53 per cent of cases in Indigenous children 2–4 years of age had isolates with serotypes included in 7vPCV.⁵

Table 5. Features of the cases of invasive pneumococcal disease in children caused by pneumococci with reduced susceptibility to penicillin, 2001

Month of onset	Age (years)	Ethnicity	Diagnosis	Length of hospital stay (days)	Serotype	Penicillin MIC* (mg/L)
Jan	3.2	Non-Indigenous	Bacteraemia	3	9V	1.0
Feb	2.75	Non-Indigenous	Pneumonia	3	14	1.0
April	2.1	Non-Indigenous	Bacteraemia	3	?	0.75
April	1.4	Indigenous	Pneumonia	1	6B	0.25
May	1.3	Indigenous	Bacteraemia	1	6B	0.5
Sept	2.75	Non-Indigenous	Bacteraemia	?	9V	2.0
Sept	0.4	Non-Indigenous	Pneumonia	22	14	1.0

* Intermediate level resistance is defined by a minimum inhibitory concentration (MIC) of 0.1–1.0 mg/L, whereas an isolate with an MIC \geq 2.0 mg/L is defined as being fully resistant to penicillin.

The intriguing feature of the cases of IPD in children was that all cases of pneumococcal meningitis and all the severe cases of IPD occurred in non-Indigenous children under 2 years of age. Indeed, five (36%) of the IPD cases in non-Indigenous children under 2 years were classified as severe. The serotypes of the isolates from the 3 meningitis cases in 2001, and from three of the severe cases are included in 7vPCV. The two remaining severe cases were caused by serotype 6A; it is likely that cross-protection from the closely related serotype 6B (included in 7vPCV) also prevents disease caused by serotype 6A.⁶ In other words, there may be a case for extending the 7vPCV vaccination program to non-Indigenous children under 2 years of age, not just those in identified risk groups.

Although at-risk Indigenous adults have been targeted for vaccination with 23vPPV in north Queensland for over 5 years, Indigenous adults were over-represented among the adult cases of IPD. Indigenous adults constitute approximately 7 per cent of the total population in north Queensland aged 15 years or older, yet 38 per cent of the total IPD cases in this population were in Indigenous adults. This over-representation is a reflection of several factors: the very high risk of infection in this population,^{1,7} the inevitable vaccine failures and the suboptimal uptake of the vaccine among eligible Indigenous adults.

It is of concern that about one third of all cases of IPD in adults in 2001 could potentially have been prevented had those eligible for vaccination according to current National Health and Medical Research Council criteria³ been vaccinated. Many of the unvaccinated cases were elderly, and presumably the remainder saw a general practitioner on a not infrequent basis. Clearly more has to be done to promote 23vPPV in the private sector; it could, for example, be made freely available to all Australian adults 65 years and over.

Most episodes of IPD caused by pneumococci with some level of resistance to penicillin were in young children. In this group three serotypes were involved, all of which (6B, 9V, 14) are included in 7vPCV. Two cases were of particular concern. One of the isolates with an intermediate level of resistance (MIC=1.0 mg/L) caused a

severe illness (pneumonia with empyema) that led to the longest in-patient hospital stay (22 days) recorded among children with IPD in 2001. The only episode of IPD caused by a fully penicillin resistant isolate (MIC=2.0 mg/L) occurred in an immunocompromised (i.e. leukaemic) child, who was unvaccinated.

Acknowledgments

Thanks are extended to the public health nurses, Tropical Public Health Unit Network, who undertook data collection. The Indigenous public health officers and immunisation nurses, Tropical Public Health Unit Network, are involved in the co-ordination and delivery of the Pneumococcal Immunisation Program in north Queensland.

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Tuberculosis notifications in Australia, 2001

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Abstract

In 2001, there were 997 cases of tuberculosis (TB) reported to the National Notifiable Diseases Surveillance System, of which, 967 were new cases of TB and 30 cases were relapses. The incidence rate of TB in Australia in 2001 was 5.1 cases per 100,000 population. The highest incidence of TB was reported in people born overseas (19.3 cases per 100,000 population), followed by Indigenous Australians (9.8 cases per 100,000 population). In contrast, the incidence rate of TB in the non-Indigenous Australian-born population was 1.0 cases per 100,000 population. This pattern of TB incidence rates amongst the sub-populations of Australia has been observed for over 10 years. Eighty-six per cent of TB cases completed treatment in 2001. Treatment was unsuccessful in 7 cases and only 22 cases defaulted. The National Tuberculosis Advisory Committee has published a National Strategic Plan with performance indicators to ensure that this enviable record of TB control is maintained and improved. *Commun Dis Intell* 2002;26:525–536.

Keywords: tuberculosis, surveillance, Mycobacterium tuberculosis

Introduction

Tuberculosis (TB) represents one of the most significant public health threats to the global population. In 2000, 3.7 million cases of TB were notified to the World Health Organization (WHO) Global Surveillance Programme, of which 42 per cent were sputum-smear positive,¹ however, these are underestimates of the global TB burden. The Western Pacific Region (WPR), of which Australia is a member, accounted for 22 per cent of all cases notified to the WHO in 2000. Four countries from the WPR were among the top 23 countries with a high TB burden. In contrast, Australia has one of the lowest incidence rates for TB in the world. There remain two sub-populations within Australia who have high incidence rates of TB; Indigenous Australians and Australian residents born overseas.

The targets for global TB control, set by the WHO, are to successfully treat 85 per cent of detected sputum smear-positive TB cases and to detect 70 per cent of all active TB cases. To meet the treatment target, the WHO has recommended

the Directly Observed Treatment — Short-course (DOTS) program. The five major components of the DOTS program are political commitment and resources, the use of microscopy to diagnose TB, standardised observed treatment for all patients with active TB, uninterrupted supplies of anti-TB drugs and a standardised reporting system for monitoring treatment and progress of TB patients.² The major principles that underpin the DOTS program guide the treatment of TB patients throughout Australia.

In order to minimise the burden and human impact of TB on the Australian population, the National TB Advisory Committee (NTAC) has prepared the *National Strategic Plan for TB Control in Australia Beyond 2000*, which was endorsed by the Communicable Diseases Network Australia.³ The Strategic Plan consists of three key elements: (1) case finding; (2) treatment; and (3) TB surveillance. Performance Indicators have been developed to allow regular review of the progress of the Strategic Plan. This annual report is the first to match national surveillance data to the Performance Indicators.

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In the past, TB notifications in Australia have been reported to the National Mycobacterial Surveillance System. Enhanced surveillance for TB notifications was commenced in 2001 as a part of the National Notifiable Diseases Surveillance System. Although the enhanced TB data set also makes provision for the reporting of drug susceptibility, the Australian Mycobacterium Laboratory Reference Network will publish its report on trends in multi-drug resistant TB in the next edition of *Communicable Diseases Intelligence*.

Methods

Data collection

Each jurisdiction in Australia has legislation that requires medical practitioners, public health laboratories and other health professionals to report cases of TB to the State or Territory health authority. Notifications of TB for 2001 were collated by jurisdictions and sent electronically to the Commonwealth Department of Health and Ageing. All records were in a de-identified format to ensure confidentiality. Data fields in the enhanced TB data set that are relevant to this report are listed in Table 1 with a brief description of each variable.

The National Tuberculosis Advisory Committee, as a sub-committee of Communicable Diseases Australia Network, was responsible for determining the dataset collected in 2001.

Table 1. Description of the data fields in the enhanced tuberculosis data set of the National Notifiable Diseases Surveillance System

Data field	Description
Country of birth	The country in which the patient was born
Microscopy	Whether acid fast bacilli were identified by microscopy from sputum or other body fluid at the time of diagnosis
Culture	Whether <i>M. tuberculosis</i> was identified by culture from sputum or other body fluid at the time of diagnosis
Nucleic acid testing	Whether <i>M. tuberculosis</i> was detected by nucleic acid testing at the time of diagnosis
Histology	Whether histological changes consistent with TB were observed at diagnosis
Pulmonary site	Details of pulmonary site involved
Extrapulmonary sites	Details of extrapulmonary diagnostic site
Selected risk factors	Selected risk factors including close contact with a TB patient, residing in a correctional facility, residing in an aged care facility, employed in an institution, employed in the health industry, HIV status or past residence in a high risk country
Anti-TB therapy	List of all of the anti-TB drugs given to the patient
New or relapse case	Whether the case was a new case (without previous treatment), relapse following full treatment in Australia, relapse following partial treatment in Australia, relapse following full treatment overseas or relapse following partial treatment overseas
TB outcomes	Whether the case was cured (bacteriologically confirmed), completed treatment, interrupted treatment (but still completed), died of TB, died of other cause, defaulter (failed to complete treatment), failure (completed treatment but failed to be cured), transferred out of Australia or still under treatment
Age	Age of patient at diagnosis
Indigenous status	Whether patient is self-identified Indigenous (Aboriginal and/or Torres Strait Islander) Australian or not
Sex	Male or female

Data processing and quality control

Data on all TB notifications reported in 2001 were received by September 2002. Each variable was examined for data completeness and only variables where data completeness was above 50 per cent for any given jurisdiction, were analysed. Data were also checked for validity, whereby any invalid entries were returned to the jurisdictions for review and correction.

Most cases of TB in Australia are reported to the surveillance system.⁴ Reasons for the high level of reporting include, the presence of an effective TB screening program, a high standard of health care for all TB patients, and specialised and multi-disciplinary TB services in each jurisdiction. The terms 'notification rate' and 'incidence rate', are used interchangeably throughout this report.

Case definition

In 2001, cases were either defined as new or relapsed. A new case required a diagnosis accepted by the Director of TB Control (or equivalent) in the relevant jurisdiction, based on laboratory or clinical evidence. Laboratory evidence includes either the isolation of *Mycobacterium tuberculosis* complex (*M. tuberculosis*, *M. bovis* or *M. africanum*) from a clinical specimen by culture; or nucleic acid testing indicating *M. tuberculosis* complex except where it is likely to be due to previously treated or inactive disease.

Clinical evidence is a diagnosis made by a clinician experienced in tuberculosis and includes clinical follow-up assessment.

A relapsed TB case was defined as a case of active tuberculosis diagnosed bacteriologically, radiologically or clinically, having been considered inactive or quiescent following previous treatment (as deemed appropriate by the State or Territory Director of Tuberculosis). Relapses refer to retreatment cases and some of these may be reinfections rather than a true relapse of prior disease.

Population estimates for 2001

The rates presented in this report were calculated using population data produced by the Australian Bureau of Statistics (ABS). The estimated resident population⁵ in each state and territory and in Australia as a whole, as at 30 June 2001, was used as the denominator in crude rate calculations.

Estimates of the Indigenous Australian population were based on projections from the 30 June 1996 census estimate of the Indigenous population in Australia.⁶ The ABS calculated the projections based on assumptions about future births, deaths and migrations in the Indigenous population and a 'low' and a 'high' estimate were reported. Throughout this report, the 'low' estimate has been used, which is consistent with previous annual reports for TB notifications in Australia.

Two different data sources were used to calculate incidence rates of TB in people born overseas, depending on data availability. The two data sources were preliminary results from the 2001 census and the estimated resident population in 2000 based on 1996 census results. Footnotes have been added to tables to indicate which data source was used. The estimated resident population of overseas-born people in 2000 (based on 1996 census data) was used as the denominator in rates in relevant analyses.

The population estimates of non-Indigenous Australian-born people were calculated by subtracting the Indigenous population estimate and the overseas-born population estimate from the total Australian population. Some notifications in this report may include people who were visitors or non-permanent residents of Australia during 2001. Therefore, some of the rates in this report may be overestimated.

Results

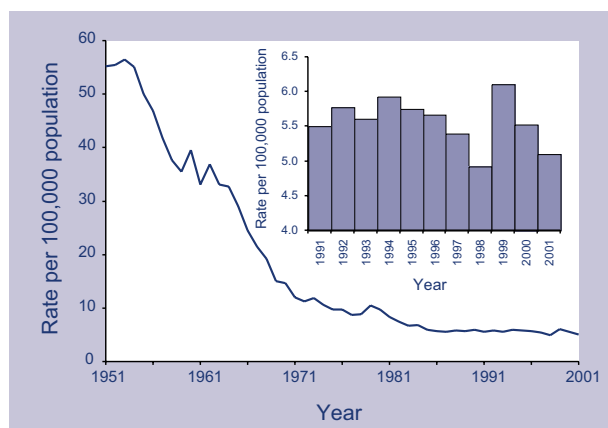
Data quality

In 2001, 18 of the 24 data fields relevant to the analysis from the enhanced TB data set were more than 50 per cent complete. Information on age and sex were reported for all TB notifications. Indigenous status was reported for 198 of the 204 (97%) people born in Australia and country of birth was recorded for 990 (99%) of all TB notifications. The site(s) of TB disease was reported for 945 cases (98%) and the method of diagnosis was reported for 992 (99%) of the cases for each method of diagnosis (i.e. culture, microscopy, histology and nucleic acid testing). The anti-TB drug regimen undertaken by cases was recorded for 960 cases (96%). Some of the data fields that were not well reported in 2001 include HIV status (4.2% complete), BCG vaccination status (53% complete) and sputum smear conversion at 3 months (23% complete).

TB notification rates

The number of cases of TB reported in Australia in 2001 was 997 (5.1 cases per 100,000 population). The notification rate of TB in 2001 was the second lowest rate on record (Figure 1). The national notification rate of TB has remained relatively stable since 1985, except for an increase in 1999, which was attributable to the number of TB cases amongst the East Timorese refugees who were evacuated to Darwin in the Northern Territory.

Figure 1. Incidence rates per 100,000 population for tuberculosis notifications, Australia, 1951 to 2001



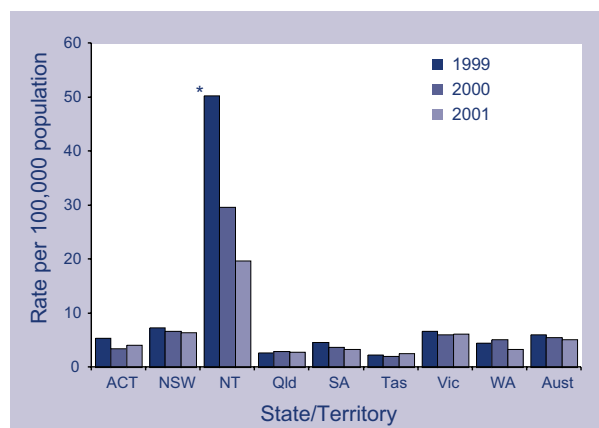
TB notifications by jurisdiction

New South Wales reported the most notifications (416 cases) of TB in 2001, however, the highest notification rate was recorded in the Northern Territory (19.7 cases per 100,000 population)

(Table 2). This rate was lower than the rate reported in 2000 (29.7 cases per 100,000 population) and 1999 (50.3 cases per 100,000 population) (Figure 2). The lowest notification rates in 2001 were reported in Tasmania (2.6 cases per 100,000 population) and Queensland (2.9 cases per 100,000 population).

Of the 997 cases reported in 2001, 967 (97%) were new cases of TB and 30 (3%) were relapsed cases (Table 2). Of the 30 relapsed cases reported in Australia, 14 relapsed after full treatment overseas, five had relapsed after full treatment in Australia, nine relapsed after partial treatment and two relapsed TB cases had unknown treatment histories.

Figure 2. Notifications rates for tuberculosis, Australia, 1999 to 2001, by State or Territory



* Includes the 61 cases of tuberculosis in East Timorese evacuees.

Table 2. Notifications of new and relapsed cases of tuberculosis and rates per 100,000 population, Australia, 2001, by State or Territory

State/Territory	New cases		Relapsed cases		Total cases	
	Number	Rate	Number	Rate	Number	Rate
Australian Capital Territory	13	4.1	0	0.0	13	4.1
New South Wales	397	6.1	19	0.3	416	6.4
Northern Territory	39	19.7	0	0.0	39	19.7
Queensland	100	2.8	6	0.2	106	2.9
South Australia	50	3.3	0	0.0	50	3.3
Tasmania	11	2.3	1	0.2	12	2.6
Victoria	296	6.1	2*	0.1	298	6.2
Western Australia	61	3.2	2	0.1	63	3.3
Australia	967	5.0	30	0.2	997	5.1

* Likely to be an underestimate as relapse status was poorly reported.

TB notifications in the Australian-born population

In 2001, 198 cases of TB occurred in the Australian-born population, of whom, 156 (79%) were non-Indigenous Australian-born and 42 (21%) were Indigenous Australian. There were 9 cases where Indigenous status or country of birth were unknown.

The highest notification rate of TB in the Australian-born population was reported in the Northern Territory (16.6 cases per 100,000 population) and the lowest rate was recorded in the Australian Capital Territory (0.4 cases per 100,000 population) (Table 3). The majority (25/42 cases; 59%) of cases in Indigenous Australians were reported in the Northern Territory (44.4 cases per 100,000 population), however, this is a considerable decrease from the 37 cases in 2000 (66.7 cases per 100,000 population). Queensland reported 9 cases of TB in the Indigenous population (7.6 cases per 100,000 population) and Victoria reported 3 cases (12.2 cases per 100,000 population).

The more populous states of New South Wales, Victoria and Queensland reported 67, 36 and 25 cases of TB, respectively, in the non-Indigenous Australian-born population, while the Northern Territory had 3 cases but the highest rate (2.7 cases per 100,000 population).

The incidence of TB in Indigenous Australians has fluctuated considerably over the past 10 years. In 2001 the incidence rate was 9.8 cases per 100,000 population, which is one of the lowest rates reported amongst this population since 1991. The non-Indigenous Australian-born population had the lowest incidence rate of TB (1.0 case per 100,000 population) and this rate has remained relatively stable over the past 10 years.

The rate of notifications of TB in 2001 was highest in overseas-born people (19.3 cases per 100,000 population), which was a slight increase from the rate in 2000 (18.0 cases per 100,000 population), but lower than the rate reported in 1999 (21.6 cases per 100,000 population) (Figure 3).

Figure 3. Trends of tuberculosis incidence rates, Australia, 1991 to 2001, by Indigenous status and country of birth

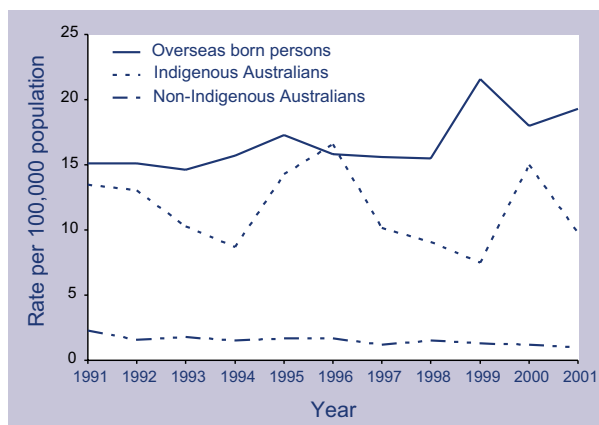


Table 3. Notifications of tuberculosis and incidence rates in Indigenous and non-Indigenous people born in Australia, 2001, by State or Territory

State/Territory	Indigenous Australian-born		Non-Indigenous Australian-born		Total Australian-born	
	Number	Rate	Number	Rate	Number	Rate
Australian Capital Territory	0	0.0	1	0.4	1	0.4
New South Wales	1	0.8	67	1.4	68	1.3
Northern Territory	25	44.4	3	2.7	28	16.6
Queensland	9	7.6	25	0.9	34	1.1
South Australia	1	4.1	7	0.6	8	0.7
Tasmania	0	0.0	7	1.7	7	1.6
Victoria	3	12.2	36	1.0	39	1.0
Western Australia	3	4.9	10	0.7	13	0.9
Australia	42	9.8	156	1.0	198	1.3

TB notifications in people born overseas

Of the 997 cases of TB reported in 2001, 790 cases (79%) were in people born overseas. Table 4 shows the number of TB notifications and incidence rate of TB based on the estimated Australian resident population for each country. Approximately 30 per cent (234/787 cases) of TB cases in people born overseas were in people from Vietnam (127 cases) and India (107 cases).

The incidence of TB amongst the resident Australian population was the highest in people from Somalia (593 cases per 100,000 resident population), Afghanistan (159 cases per 100,000 resident population) and India (112 cases per 100,000 resident population). Some caution is required when interpreting these results, as high rates may be attributable to temporary residents who may not be representative of the baseline resident population.

Table 4. Notifications of tuberculosis and estimated rate per 100,000 population for selected countries of birth, Australia, 2001

Country of birth	New cases	Relapsed cases	Total cases	Estimated Australian resident population by country of birth, 2001	Rate per 100,000 population in Australia by country of birth, 2001	WHO incidence rate (per 100,000 population) for country, 2000 [†]
Vietnam	125	2	127	154,833	82.0	115
India	104	3	107	95,455	112.1	111
Philippines	57	4	61	103,942	58.7	170
China [‡]	58	1	59	142,778	41.3	36
Indonesia	42	2	44	47,156	93.3	32
Korea [§]	27	3	30	38,958	77.0	47
Somalia	22	0	22	3,713	592.5	65
United Kingdom	20	2	22	1,083,318	2.0	10
Papua New Guinea	18	2	20	23,618	84.7	252
Afghanistan	18	0	18	11,297	159.3	33
New Zealand	16	0	16	355,765	4.5	9
Thailand	13	0	13	23,599	55.1	54
Italy	12	1	13	218,718	5.9	6
Cambodia	13	0	13	22,979	56.6	144
Overseas	760	27	787	4,087,928	19.3	
Australia	201	3	204	15,298,735	1.3	
Not stated	6	0	6			
Total	967	30	997	19,386,663	5.1	

Rates per 100,000 resident population should be interpreted with caution, as some of the cases are visitors to Australia who are not included in the census population.

* Country of birth for denominator is from the 2001 census.

† Rates from the World Health Organization 2002 Global tuberculosis report.

‡ China excludes SAR and Taiwan.

§ The notifications for Korea included both the Republic of Korea and the Democratic Peoples Republic of Korea.

|| The WHO figure quoted is for the Republic of Korea, as virtually all of Korean-born people in Australia are from the Republic of Korea.

TB notifications by age and sex

One of the key performance indicators of the National Strategic Plan is the incidence of TB among children aged less than 15 years. In 2001, there were a total of 34 cases of TB in individuals aged less than 15 years and the overall notification rate in this age range was 0.9 cases per 100,000 population. The notification rate was the highest in overseas-born children (6.0 cases per 100,000 population) when compared with Indigenous Australian children (2.4 cases per 100,000 population) and non-Indigenous Australian-born children (0.5 cases per 100,000 population) (Table 5).

The notification rates in Indigenous Australians were the highest in the age ranges 65–74 years (52.2 cases per 100,000 population) and 55–64 years (44.4 cases per 100,000 population). Amongst the non-Indigenous Australian-born population, the notification rates were highest in people aged 75+ years (5.8 cases per 100,000 population). The notification rates for people born overseas were the highest in the 15–24 year age range (28.5 cases per 100,000 population), 25–34 year age range (27.9 cases per 100,000 population) and the 75+ years age group (27.4 cases per 100,000 population).

The age- and sex-stratified incidence rates for TB in overseas-born and Australian-born (Indigenous and non-Indigenous) populations are shown in Figure 4. The pattern of distribution of TB cases by age group was quite different for overseas-born and Australian-born people. In the Australian-born population, there was a fairly stable rate of TB (approximately one case per 100,000 population) in people aged up to the 35–44 year age range for males and the 45–54 year age range for females, after which the incidence rate gradually increases. The highest rates of TB for the Australian-born population were in the 75+ age group for both males (8.5 cases per 100,000 population) and females (4.3 cases per 100,000 population). The overall rate of TB in Australian-born males was 1.6 cases per 100,000 population and 1.1 cases per 100,000 population in Australian-born females. The male:female ratio in Australian-born TB cases was 1.4:1.

Table 5. Notifications and estimated incidence rate of tuberculosis per 100,000 population, Australia, 2001, by age group, Indigenous status and country of birth

Age group (years)	Indigenous Australian-born		Non-Indigenous Australians Australian-born		Overseas-born	
	Number	Rate*	Number	Rate*	Number	Rate*
0–4	1	1.7	15	1.3	2	8.2
5–14	3	2.8	2	0.1	11	5.8
<i>Sub total for <15 years</i>	4	2.4	17	0.5	13	6.0
15–24	7	8.4	13	0.6	134	28.5
25–34	8	11.7	17	0.8	187	27.9
35–44	4	7.8	16	0.8	125	14.2
45–54	8	24.9	13	0.7	100	11.4
55–64	7	44.4	17	1.5	65	10.3
65–74	4	52.2	18	2.1	78	17.4
75+	0	0.0	45	5.8	88	27.4
Australia	42	9.8	156	1.0*	790	19.3*

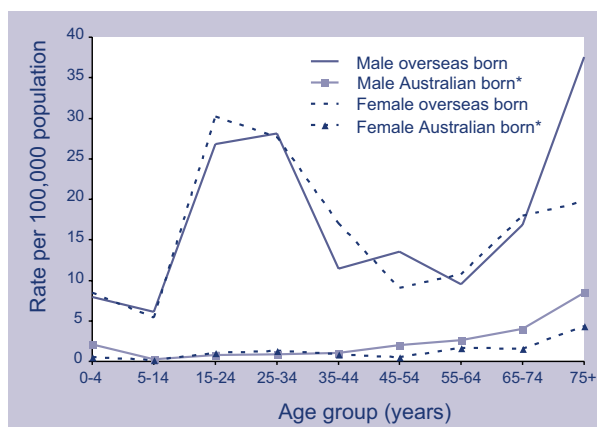
* The denominator used for total non-Indigenous Australian-born population is from the 2001 census, whilst age group breakdowns use denominator from estimated resident population in 2000 based on the 1996 census results.

Note: There were 9 cases where country of birth or Indigenous status were unknown.

The age- and sex-stratified incidence rates for TB in overseas-born and Australian-born (Indigenous and non-Indigenous) populations are shown in Figure 4. The pattern of distribution of TB cases by age group was quite different for overseas-born and Australian-born people. In the Australian-born population, there was a fairly stable rate of TB (approximately one case per 100,000 population) in people aged up to the 35–44 year age range for males and the 45–54 year age range for females, after which the incidence rate gradually increases. The highest rates of TB for the Australian-born population were in the 75+ age group for both males (8.5 cases per 100,000 population) and females (4.3 cases per 100,000 population). The overall rate of TB in Australian-born males was 1.6 cases per 100,000 population and 1.1 cases per 100,000 population in Australian-born females. The male:female ratio in Australian-born TB cases was 1.4:1.

The highest rate of TB in overseas-born females was in the 15–24 year age range (30.2 cases per 100,000 population), but decreased to 9.0 cases per 100,000 population in the 45–54 year age range and then increased again to 19.7 cases per 100,000 population in the 75+ year age group (Figure 4). The pattern of TB rates by age group was similar for overseas-born males. The highest rates were in the 15–24 year age range (26.8 cases per 100,000 population), 25–34 year age range (28.1 cases per 100,000 population) and the 75+ year age group (37.6 cases per 100,000 population). The overall male:female ratio of TB cases in the overseas-born population was 1:1 and the overall incidence rate for overseas-born males and females was 17.5 and 17.4 cases per 100,000 population, respectively.

Figure 4. Incidence rates of tuberculosis in Australian-born and overseas-born people, 2001, by age group and sex



* Includes Indigenous and non-Indigenous Australian-born populations

TB and HIV status

Information on HIV status was only provided for 42 of the 997 (4.2%) TB cases in Australia in 2001. Of the TB cases where HIV status was known, there were 5 people who were HIV positive, 4 of whom were born overseas and one born in Australia. The National Strategic Plan recommends that HIV status of all TB cases be collected. This is a goal which Australia is working towards in the future.

Sites of tuberculosis disease

In 2001, 558 (55%) of the TB cases had pulmonary disease. This was the only identified site of disease in 515 (92%) pulmonary cases of TB (Table 6). Forty-three cases reported the site of disease as 'pulmonary and other site of disease' where the other site of disease was not specified. Approximately 65 per cent of TB cases

Table 6. New and relapsed cases of tuberculosis in Australia, 2001, by site of disease

Site	New cases	Relapse cases	Total cases*	Total %
Pulmonary	538	20	558	55.0
Lymphatic	205	5	210	20.1
Pleural	74	0	74	7.3
Bone/joint	40	3	43	4.2
Peritoneal	15	0	15	1.5
Genitourinary	28	1	29	2.9
Miliary	15	1	16	1.6
Meningeal	3	0	3	0.3
Other	58	0	58	5.7
Unspecified	8	0	8	0.8

* The total number of cases do not add up to 997 as some cases had multiple sites of infection.

amongst both the Indigenous and non-Indigenous Australian-born populations had pulmonary TB as a site of disease. In contrast, 48 per cent of the overseas-born cases had pulmonary TB as a site of disease. The second most common site of disease in TB cases in 2001 was the lymphatic system (210 cases; 20.1%), followed by pleurae (74; 7.3%).

Antimicrobial therapy

The antimicrobial drug regimen given to cases was reported for 959 (96%) cases of TB. In 2001, there were 72 cases on a two drug regimen, 104 cases on a three drug regimen, 761 cases on a four drug regimen and 22 cases on a regimen of five or more antimicrobial TB drugs. Of the 761 cases on a four drug regimen, 754 cases (76%) were prescribed the four drug regimen of isoniazid, rifampicin, pyrazinamide and ethambutol, which is generally the standard short course treatment for active TB in those aged 8 years and older. Ethambutol is not recommended for use in young children where visual testing cannot be assured and of the 17 cases under 8 years of age, 14 had the three drug regimen of isoniazid, rifampicin and pyrazinamide.

Treatment outcomes

The outcome from treatment of TB was reported for 827 cases (83%). In 2001, 648 cases (65%) had completed treatment, of whom 60 (7%) were still undergoing treatment and 22 (2.7%) had returned overseas prior to treatment completion (Table 7). Of the remainder, satisfactory outcomes were reported for 641 cases (86%), comprising 65 cured (bacteriologically confirmed) and 576 people who completed treatment (no bacteriological confirmation). Only 3 cases (0.4%) interrupted treatment for TB. Adverse treatment outcomes (excluding deaths) were reported in 29 cases (4.3%); 7 failures and 22 defaulters.

Excluding the cases still under treatment, there was no difference in the proportion of cases who completed treatment amongst Indigenous Australians (78%), non-Indigenous Australian-born people (81%) nor people born overseas (88%). Seventy-two deaths were recorded during treatment in 2001, with 30 cases dying of causes other than TB. The remaining 42 deaths attributable to TB is likely to be an overestimation. New South Wales does not make any distinction between 'died of TB' and 'died of other causes' and all deaths are recorded as 'died of TB'. The case fatality rate was 4.2 per cent when including deaths from New South Wales and one per cent when deaths from New South Wales were excluded.

Table 7. Outcomes of treatment for tuberculosis, Australia, 2001, by Australian-born (Indigenous and non-Indigenous) and overseas-born individuals

Treatment outcomes	Indigenous Australian-born	Non-Indigenous Australian-born	Overseas-born	Unknown	Total
Cured (bacteriologically confirmed)	4	14	47		65
Completed treatment	25	87	460	4	576
Interrupted treatment	–	1	2		3
Died of TB*	2	13	26	1	42
Died of other cause	4	6	18	2	30
Defaulted†	2	1	19		22
Failed‡	–	3	4		7
Transferred out of Australia	–	–	22		22
Still under treatment	4	6	50		60
Unknown	1	25	142	2	170
Total	42	156	790	9	997

* This number is an overestimate, as New South Wales does not distinguish cause of death for tuberculosis data and each death recorded in this jurisdiction was reported as 'died of TB'.

† Defaulted means failed to complete treatment.

‡ Failed means treatment was completed but failed to be cured.

National Performance Indicators

At the 2002 meeting of the National Tuberculosis Advisory Committee, performance criteria were set against the National Performance Indicators. This annual report is the first to address these performance indicators (Table 8) and is therefore a baseline to work from in the future.

The performance criteria for people born overseas applies only to people who have been living in Australia for more than 5 years. The 'year of arrival' variable was used to estimate the number of years a person born overseas has

been living in Australia. It was assumed that any given person born overseas had been living in Australia since the year of arrival. Based on this assumption, of the 790 people with TB who were born overseas, 415 cases had been living in Australia for more than 5 years. The incidence rate for people born overseas who have been living in Australia for more than 5 years was 10.2 cases per 100,000 population and for less than 5 years was 7.6 cases per 100,000 population.

Table 8. National tuberculosis performance indicators, performance criteria and the current status of tuberculosis in Australia, 2001

National TB performance indicator	Performance criteria	2001
Annual incidence of TB (per 100,00 population)		%
Crude incidence		
Indigenous Australians	<1	9.8
Non-indigenous Australian-born	<1	1.0
Overseas-born persons*	§	10.3
Relapse cases initially treated in Australia	<2% of total treated cases	NA†
Incidence in children less than 15 years by risk groups:		
Indigenous Australian children	<0.1	2.4
Non-indigenous Australian-born children	<0.1	0.5
Overseas-born children*	§	6.0
Collection of HIV status in TB cases‡ (% of cases with data collected)	100% over next 3 years	4.2%
Treatment outcome measures (%)		
Sputum – smear positive cases that are sputum negative by the 3rd month	>90	NA†
Cases evaluated for outcomes	100	82.9
Cases that have treatment completed and are cured	>90	6.5
Cases recorded as treatment failures	<2	0.7

Note: Incidence is calculated using the number of newly diagnosed cases reported to the surveillance system.

* The performance criteria for overseas-born is applied to people who have been living in Australia for more than 5 years. The denominator for this rate is the total overseas-born population living in Australia in 2001.

† NA Not available (data incomplete).

‡ Incidence of HIV in TB cases was reported with consent from the individual (i.e. there was no data linkage).

§ Performance indicators currently under review

Discussion

Australia continues to report one of the lowest incidence rates of tuberculosis in the Western Pacific Region of the World Health Organization, and in the world.¹ In 2001, the incidence rate was 5.1 cases per 100,000 population, which was the second lowest rate ever recorded in Australia. The incidence of TB has remained between 5 and 6 cases per 100,000 population since the mid-1980s. The largest decrease in cases was observed in the Northern Territory. In late 1999, Darwin received 1,863 people evacuated from East Timor, 61 of whom had active TB,⁷ and in 2000, there was a large outbreak of TB in a remote community in the Northern Territory. Screening for TB in the people evacuated from East Timor and effective management of the outbreak has meant that to date, no further cases of TB have resulted from the evacuation nor the outbreak, thus allowing the number of cases to decrease in the Northern Territory in 2001.

In 2001, the rate of TB in Indigenous Australians was nearly 10 times higher than in non-Indigenous Australian-born people, with nearly 60 per cent of the cases occurring in the Northern Territory. Plant *et al.*⁸ have reported on some of the risk factors for TB in the Indigenous population, which include poor socio-economic status (reflected in overcrowding), co-morbidities (i.e. diabetes and renal disease), smoking, alcohol abuse and poor nutrition.

Incidence rates in Indigenous Australians should be interpreted with caution as the population fluctuates between years and identification of cases may be variable. Any small changes in the numerator when dealing with a small population can affect rates considerably. The highest rates of TB in Indigenous Australians were in older people aged 55–74 years, which was similar to the non-Indigenous Australian-born population. It is possible that the cases of TB in the ageing Indigenous and non-Indigenous population represent reactivation of previous TB infections. In contrast, the peak of TB in the overseas-born population occurred in persons aged 15–34 years, which is a pattern characteristic of areas where TB is endemic. Examination of the treatment outcomes also suggests that once Indigenous Australians gain access to anti-TB treatment, the proportion of people who complete treatment is the same as non-Indigenous Australians. The National Strategic

Plan has stated, as a goal, that the incidence of TB in the Indigenous Australian population should be the same as Australian-born non-Indigenous people. Addressing issues that affect TB transmission and assuring the basic standard of care is resourced will progress Australia towards achieving this goal.

The other sub-population in Australia in which the TB burden is high is those born overseas. In 2001, 40 per cent of all TB cases notified in Australia were in people born in Vietnam, India, the Philippines, China and Indonesia, all of which have been identified by WHO as high TB incidence countries.¹ Some of the possible reasons for the high rate of TB notifications in people born overseas are that people from high TB incidence countries are at much greater risk of exposure to TB in their country of birth and may have latent TB infection prior to arrival in Australia. Migration stress, co-morbidities and poor nutrition⁹ may also contribute to the rate of notifications amongst people born overseas, through the progression of latent TB to active TB.

A recent study conducted in Denmark,¹⁰ examined the incidence of TB in Somalian immigrants since the year of arrival. The annual incidence of TB in Somalian immigrants was found to decline only gradually over the first 7 years since arrival in Denmark. The authors concluded that the current Danish policy of screening only on arrival was not adequate for reducing the long term incidence of TB within this population. Australia has a policy of screening for TB before arrival. If a person has active TB that has been treated prior to arrival or non-active TB on a chest x-ray or on clinical review, they must sign a Health Undertaking, agreeing to contact the Health Undertaking Service upon arrival in Australia. The individual then reports to the State or Territory health authority for follow-up monitoring within the Australian health system.¹¹ This strategy at least allows migrants to become familiar with the health system and know places of contact for TB services. In 2001, approximately half of the people with TB amongst the overseas-born population had been living in Australia for more than 5 years. It is possible that some of these cases represent people with latent TB infections that were not found during the initial screening process¹⁰ or local transmission from other overseas-born people living in Australia. Australia needs to continue effective active surveillance of recent immigrants through Health Undertakings and ensure

inexpensive, friendly and culturally appropriate access to TB diagnosis and antimicrobial TB treatment. Further steps such as programs to diagnose latent TB infections and to provide treatment to prevent TB infection from progressing to active communicable TB may also help reduce the disease burden amongst migrants with TB in Australia.

The *National Strategic Plan for TB Control* will help Australia maintain its low incidence of TB and to assist neighbouring countries with a high TB burden. This annual report presented the performance criteria that will be used to help identify the areas where Australia is meeting the standards set in the performance indicators and areas where TB control is most needed. In 2001, Australia was close to achieving four of the performance criteria; the incidence rate in non-Indigenous Australian-born population (1.0 case per 100,000 population), the incidence rate in non-Indigenous Australian-born children less than 15 years of age (0.5 cases per 100,000 population), the total number of TB cases evaluated for treatment outcomes (83%) and the number of cases recorded as treatment failures (1%).

The areas that require more effective TB control and access to treatment are in Indigenous Australians and overseas-born people. Access to the best TB control practices (e.g. contact tracing, appropriate high risk group screening, treatment of those with latent TB infection and ensuring easy access to anti-TB treatment) will help Australia meet its targets. Improvements in surveillance and data collection will ensure that Australia can monitor its progress towards the goals set in the Strategic Plan in the future.

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Report of the Australian Rotavirus Surveillance Program, 2001/2002

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Abstract

The National Rotavirus Reference Centre together with collaborating laboratories Australia-wide has conducted rotavirus surveillance since June 1999. The serotypes of rotavirus strains that are responsible for the hospitalisation of children with acute gastroenteritis were determined for the period 1 June 2001 to 31 June 2002. We examined 754 rotavirus samples using a combination of monoclonal antibody immunoassay, reverse transcription-polymerase chain reaction, and Northern hybridisation. For the first time, serotype G9 strains were the most prevalent type nationally (40.4%) and found in 8 of the 9 centres. Serotype G1 strains were the second most prevalent type (38.9%), identified in 5 of the centres. These findings have important implications for vaccine development strategies which target serotypes G1-G4. *Commun Dis Intell* 2002;26:537-540.

Keywords: rotavirus; gastroenteritis

Introduction

There is wide acceptance of the need for a vaccine to prevent rotavirus disease in children under 5 years of age throughout the world. While there are few deaths in developed countries, there is considerable morbidity, with 10,000 Australian children hospitalised each year.¹ A major outbreak in the Northern Territory during the recent 12 month surveillance period has reinforced the seriousness of this infectious disease in the Australian community, especially in Aboriginal children. The impact on health services in Alice Springs was significant. During May 2001, 246 children with acute gastroenteritis presented to the Emergency Department of the Alice Springs Hospital, resulting in 145 being admitted, of whom 137 were confirmed as having rotavirus infection.²

Surveillance relies upon co-operation of microbiologists from major centres, and they continue to provide very valuable input into the system. Past experience has shown that Brisbane, Sydney, Melbourne, Adelaide and Hobart tend to have similar patterns of disease, while a high number of cases and unusual strains have been

identified in Western Australia. The Northern Territory has had very different epidemiology, with outbreaks at unpredictable times of the year and emergence of unusual strains, sometimes in conjunction with Western Australia. Surveillance has continued unchanged in Western Australia and the Northern Territory, but surveillance on the eastern seaboard has been limited to Melbourne.

Methods

Collaborating laboratories undertook rotavirus detection by enzyme immunoassay (EIA) or latex agglutination. Rotavirus positive specimens were collected, stored frozen and forwarded to the Royal Children's Hospital in Melbourne, together with relevant age and sex details.

Specimens were then tested using an in-house monoclonal antibody (MAb) based serotyping EIA. The EIA employed a panel of MAbs specific for the major glycoprotein VP7 of the outer capsid of the 5 major group A human rotavirus serotypes (G1, G2, G3, G4 and G9). Strains unable to be assigned a serotype were genotyped by reverse transcription/polymerase chain reaction

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(RT/PCR) using serotype specific oligonucleotide primers.³ Northern hybridisation analysis utilising G type specific DNA probes hybridised under stringent conditions was also employed to confirm serotype specificities.⁴ Polyacrylamide gel electrophoresis (PAGE) confirmed the sharing of the same electropherotype between collaborating centres.

Results

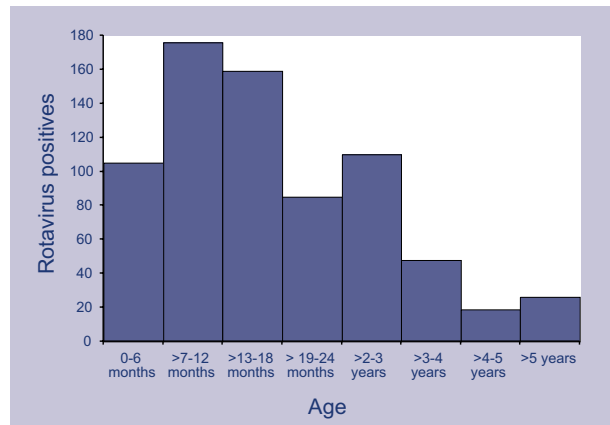
Number of isolates

A total of 847 specimens were received from the collaborating centres. Specimens containing insufficient specimen for testing or specimens that were not confirmed to be positive for rotavirus, were omitted from the serotyping data. A total of 754 positive specimens were analysed over a 13 month period from 1 June 2001 to 31 June 2002.

Age distribution

The age distribution of acute gastroenteritis cases were typical of rotavirus infection (Figure). In the reporting period, 39 per cent of cases were from infants 12 months of age or less, 33 per cent were from patients 13–24 months of age, and 15 per cent were from patients 25–36 months of age. Overall, 87 per cent of samples were from children 3 years or less, and 96 per cent were from children 5 years or less.

Figure. Age distribution of cases with rotavirus infection, Australia, 1 June 2001 to 31 June 2002



Serotype distribution

Rotavirus serotypes identified in Australia from 1 June 2001 to 31 June 2002 are shown in the Table. Serotype G9 was the most common nationally, representing 40.4 per cent of specimens. It was present in 8 of the 9 centres and was the dominant type in 6 of the centres. G1 was second most common, and represented 38.9 per cent of specimens. G1 was the dominant type in 2 locations (Melbourne and Perth) and was present in another 3 centres (Alice Springs, Darwin-Western Pathology, and WA PathCentre).

Table 1. Reports of rotavirus G serotypes in Australia, 1 June 2001 to 31 June 2002

Centre	Total number	Serotype percentage						
		G1	G2	G3	G4	G9	Mixed serotypes	No result*
Melbourne	166	47.6	4.2	0	7.2	13.3	5.4	22.3
Hobart	6	0.0	33.3	0	0.0	0.0	0.0	66.7
Perth	224	72.3	2.2	0	0.0	11.6	1.8	7.1
WA Pathcentre	94	37.2	0.0	0	1.1	40.4	4.3	17.0
Darwin	22	0.0	0.0	0	0.0	95.5	0.0	4.5
Darwin-Western Pathology	44	6.8	0.0	0	0.0	70.5	4.5	18.2
Alice Springs	118	2.5	0.0	0	0.0	80.5	5.9	11.1
Gove	30	0.0	0.0	0	0.0	86.7	0.0	13.3
Mt Isa	50	0.0	0.0	0	0.0	92.0	0.0	8.0
Total	754	38.9	1.9	0	1.7	40.4	3.4	13.7

* No result – unable to be serotyped with monoclonal antibodies or genotyped by RT/PCR.

Serotypes G2 and G4 each represented less than 2 per cent of the specimens. G2 was identified in 3 centres (Melbourne, Hobart and Perth), while G4 was identified in Melbourne and Western Australia. Serotype G3 was not identified during this surveillance period in any centre.

These serotyping results illustrate important differences between the distribution of serotypes in different parts of Australia. Serotype G9 was the dominant type in Central and northern Australia, while G1 was dominant in west and eastern Australia. Melbourne was the only centre where the four circulating serotypes were identified.

In the reporting period, 3.4 per cent of the rotavirus samples analysed contained multiple serotypes. The presence of mixed infections provides the opportunity for rotavirus to undergo reassortment, potentially resulting in new strains. In 13.7 per cent of the samples a serotype was unable to be assigned. These could represent unusual serotypes not identified using standard methods, or samples with low virus numbers which are below the detectable limits of our assays.

Discussion

National rotavirus surveillance from 1 June 2001 to 31 June, 2002 highlighted the emergence of serotype G9 as the nationally dominant serotype. This corresponded with the large outbreak of acute gastroenteritis (caused by rotavirus G9) that occurred in Alice Springs, where 145 children (137 were rotavirus positive) were hospitalised and several hundred more were affected during May 2001.² This outbreak continued to spread northward to Tenant Creek, Katherine, Darwin and Gove during June and July 2001. In addition, cases of acute gastroenteritis in several remote Western Australian, South Australian and Queensland locations were also attributed to this serotype. PAGE analysis of samples from each of these locations indicated that the same strain was responsible for the initial outbreak in Alice Springs and the subsequent spread.

Serotype G9 was first identified in 3 children with severe gastroenteritis during Australia-wide surveillance in 1997.⁵ Serotype G9 strains were not identified during 1998. During surveys conducted in 1999/2000 and 2000/2001,

serotype G9 was the second most prevalent serotype nationally, representing 10 per cent and 18.1 per cent respectively of specimens collected in those years.^{6,7} Serotype G9 strains have persisted since 1996 to 1997 in many countries including the United States of America, Bangladesh, and the United Kingdom, and their occurrence has now been documented in all continents.^{8,9,10}

For the first time since national rotavirus surveillance began in 1993, serotype G1 was not the dominant national type, being detected in 38.9 per cent of samples. Previously serotype G1 was the most prevalent serotype in Australia, representing 58 per cent and 49.5 per cent of specimens during 1999/2000 and 2000/2001. Serotype G1 was also dominant in a study conducted from 1993 to 1996.¹¹ The decline in the prevalence of serotype G1 strains around Australia can be attributed to the relative increase in the prevalence of serotype G9 strains.

The prevalence of serotype G4 increased in Melbourne from 5.2 per cent in 2000/2001 to 7.2 per cent. During the previous year (2000/2001), serotype G4 was the second most common type identified in Darwin and Sydney. Whether the Melbourne serotype G4 strains identified in 2001/2002 are related to those earlier strains from Darwin and Sydney requires further analysis. The decrease of serotype G2 in Melbourne (from 15.3% to 4.2%) continues the sporadic occurrence of this serotype from both a local and national perspective.

Ongoing surveillance of the seasonal variation in rotavirus is warranted. The identification of emerging serotypes highlights the continued evolution of rotavirus. Epidemiological knowledge of serotype prevalence will assist in the design of future vaccine strategies.

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Victoria

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Surveillance of antibiotic resistance in *Neisseria gonorrhoeae* in the WHO Western Pacific Region, 2001

The WHO Western Pacific Gonococcal Antimicrobial Surveillance Programme

Abstract

A long-term program of surveillance of antimicrobial resistance in *Neisseria gonorrhoeae* isolated in the World Health Organization's Western Pacific Region (WHO WPR GASP) continued in 2001. Seventeen focal points contributed data on about 10,000 gonococci. Resistance to quinolone and penicillin antibiotics remained widely dispersed and at high levels. Gonococci with decreased susceptibility to third generation cephalosporins were again observed in four centres. Spectinomycin resistance was infrequently encountered. Control of gonorrhoea in the WHO WPR is compromised by the further reduction in options for cheap and effective treatment of gonorrhoea. *Commun Dis Intell* 2002;26:541–545.

Keywords: surveillance; Neisseria gonorrhoeae; antimicrobial resistance; gonorrhoea; antibiotics; quinolones; penicillins; spectinomycin; cephalosporins

Introduction

The World Health Organization (WHO) estimates put the global number of cases of gonorrhoea at about 65 million each year with approximately half of these occurring in the South-East Asian and Western Pacific WHO regions. Control of this disease and its complications requires an integrated approach embracing efforts to reduce transmission through education and early case finding and includes as a key element, the provision of early and effective antibiotic treatment. Regrettably, the WHO Western Pacific Region (WPR) has an unfortunate record in terms of antibiotic resistance in *Neisseria gonorrhoeae*. The emergence and spread of penicillinase producing *Neisseria gonorrhoeae* (PPNG) in the 1970s was followed by the appearance of spectinomycin resistance for a period in the 1980s and by the currently well established quinolone resistance in the 1990s. The year 2000 report of this group documented some decreased susceptibility to third generation antibiotics. The gonococcus has thus become resistant to cheap and effective antibiotics in the WPR and this has significantly compromised both

individual and public health management of gonorrhoea. These resistant gonococci have now spread well beyond the WPR. Treatment of gonorrhoea is best given as a single dose treatment on initial diagnosis, and for this reason standardised treatment schedules have been established. It is therefore important to have accurate data on antimicrobial resistance in the gonococcus available in order to guide selection of an appropriate antibiotic treatment. Antibiotic resistance in gonococci can spread rapidly between countries, and infected travellers often present for treatment in countries distant from the place of contact. Thus for a number of reasons it is important to have regional as well as local data on antibiotic resistance available.

The WHO Western Pacific Region Gonococcal Antimicrobial Surveillance Programme (GASP) is a continuing program of susceptibility surveillance in the Region and has published surveillance data annually since 1992.¹ This report provides an analysis of surveillance of antimicrobial resistance in *N. gonorrhoeae* in the WHO WPR in 2001.

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Methods

The methods used by the WHO WPR GASP have been published² and provide full details of the source of isolates, sample populations, laboratory test methods and quality assurance programs used to generate data. These methods were unaltered in 2001. Most isolates were collected from symptomatic sexually transmitted disease clinic patients. As a guide to the interpretation of the following data, a WHO expert committee has recommended that treatment regimens be altered once resistance to a particular antibiotic reaches 5 per cent.³

Results

Approximately 10,781 gonococcal isolates were examined for susceptibility to one or more antibiotics in 17 participating countries (listed in the acknowledgments) in 2001.

Penicillins

Resistance to the penicillins remained widespread by both chromosomal (CMRNG) and plasmid mediated mechanisms (penicillinase producing *N. gonorrhoeae*). Table 1 provides details of CMRNG, PPNG and total penicillin resistance in 16 WPR countries. Very high rates of forms of penicillin resistance (CMRNG +/- PPNG) were recorded in Laos (96%), Korea (88%), the Philippines (86%) and China (85%). Singapore (58%), Malaysia (56%), Hong Kong (55%), Brunei (45%), Papua New Guinea (40%) and Vietnam (36%) also had high rates of penicillin resistance. Data from some Pacific Island states were again unavailable this year. In past years low rates of penicillin resistance were observed in these countries, and continued to be low in New Caledonia and Fiji. However, continuing surveillance is required if these agents are to be used. Other participants submitting data in 2001 (Australia, Japan and New Zealand) had rates of penicillin resistance between 9 and 29 per cent.

Table 1. Penicillin sensitivity of strains of *Neisseria gonorrhoeae* isolated in countries in the WHO Western Pacific Region in 2001

Country	Tested n	PPNG		CMRNG		All penicillin resistant	
		n	%	n	%	n	%
Australia	3,641	274	7.5	558	15.3	832	22.8
Brunei	57	21	36.8	5	8.7	36	45.6
China	748	219	21.3	438	58.5	657	85.8
Fiji	522	9	1.7	8	1.5	17	3.2
Hong Kong SAR	2,575	350	13.6	1,064	41.3	1,414	54.9
Japan	300	7	2.3	80	26.6	87	29.0
Korea	177	96	54.2	60	33.9	156	88.1
Laos	160	133	83.0	21	13.0	154	96.0
Malaysia	30	16	53.3	1	3.3	17	56.6
New Caledonia	57	0	–	0	–	0	–
New Zealand	765	29	3.8	39	5.0	68	8.9
Papua New Guinea	369	147	39.8	2	0.5	149	40.3
Philippines	399	268	67.1	76	19.0	344	86.1
Singapore	741	390	52.6	41	5.5	431	58.1
Tonga	32	2	6.3	4	12.5	6	18.8
Vietnam	166	59	35.5	1	0.6	60	36.1

PPNG Penicillinase producing *N. gonorrhoeae*

CMRNG Chromosomally mediated resistance in *N. gonorrhoeae*

Quinolones

Resistance to the quinolone antibiotics has been high and endemic in many parts of the WPR for many years. Additional data from Laos and Cambodia in 2001 confirmed the widespread extent of the problem in the WPR. Data from 14 WPR countries are shown in Table 2 and quinolone resistant strains (QRNG) are divided into 'less susceptible' and 'resistant' categories on the basis of susceptibility determinations. Thirteen of the 14 WPR countries which examined isolates for quinolone resistance detected QRNG in 2001, the exception being Papua New Guinea. Very high proportions of QRNG were detected in Cambodia, China, Hong Kong, Japan, Korea, the Philippines and Vietnam. There was again an upward shift in overall levels of resistance with most of the QRNG in the majority of countries having gonococci with high level resistance.

Cephalosporins

In the 2000 report, the presence of a small number of isolates with altered susceptibility to third generation cephalosporins was noted. Gonococci with this characteristic were again seen in low numbers in 2001 in Singapore, Brunei, China and Australia. Such strains have prevailed for a few years but are only now translating into treatment failure.⁴ A report of documented treatment failure with an oral third generation cephalosporin in the WPR has now been published.⁵ This is a finding of major significance as this group of antibiotics is crucially important in the treatment of gonorrhoea as resistance to other agents accelerates.

Spectinomycin

A small number of spectinomycin resistant strains were found in Cambodia (1 isolate) and China (3 isolates) with Vietnam reporting two isolates in the less sensitive to spectinomycin category. Only very occasional strains resistant to this injectable antibiotic have been found in recent WPR surveys.

Table 2. Quinolone resistance in strains of *Neisseria gonorrhoeae* isolated in countries in the WHO Western Pacific Region in 2001

Country	Tested	Less susceptible		Resistant		All QRNG*	
	n	n	%	n	%	n	%
Australia	3,641	149	4.1	489	13.4	638	17.5
Brunei	52	1	2.0	10	19.0	11	21.0
Cambodia	42	9	21.0	18	43.0	27	64.0
China	748	83	11.1	650	86.9	733	97.9
Hong Kong SAR	2,575	235	9.1	2,270	88.2	2,505	97.3
Japan	300	42	14.0	192	64.0	234	78.0
Korea	177	94	53.1	70	39.5	164	92.6
Laos	187	11	5.9	42	22.5	53	28.3
Malaysia	30	2	6.7	7	23.3	9	30.0
New Zealand	765	20	2.6	77	10.0	97	12.7
Papua New Guinea	96	0	–	0	–	0	–
Philippines	399	1	0.2	217	54.3	218	54.6
Singapore	741	35	4.7	207	27.9	242	32.6
Vietnam	168	33	19.6	71	42.3	104	61.9

* QRNG Quinolone resistant *Neisseria gonorrhoeae*

High-level tetracycline resistance

Although tetracyclines are not a recommended treatment for gonorrhoea, these agents are widely used and readily available in the WPR. One particular type of plasmid-mediated resistance gives rise to high-level tetracycline resistance (TRNG). About 7,583 gonococci were examined for high-level tetracycline resistance in 13 WPR countries in 2001 (Table 3). High rates of TRNG continue to be reported from Malaysia, Brunei, Singapore, Vietnam, China and now Laos with rates between 34 and 99 per cent. In other countries, rates of TRNG ranged between one and 17 per cent of strains examined.

Discussion

The data recorded in 2001 in the WPR provide no relief from concerns previously expressed in regard to increasing antibiotic resistance in gonococci. Any contemplated use of the

penicillins would be restricted to a few settings and would require prior validation of likely efficacy. The same approach would need to be applied to the quinolone group and their use has been discontinued in many WPR countries because of resistance. Selection of suitable alternative treatments is difficult given the cost of available antibiotics. The recognition of gonococci with altered susceptibility to third generation cephalosporins⁶ and now, leading to documented treatment failure, is also a matter of considerable concern. It remains to be seen if this treatment failure with oral third generation cephalosporins will extend to injectable agents such as ceftriaxone. The amount of ceftriaxone that can be given by injection exceeds by a considerable margin, that which is absorbed by administration of oral agents of the same class. Considerable attention will need to be given to any further emergence of gonococci with these attributes.

Table 3. High level tetracycline resistance in strains of *Neisseria gonorrhoeae* isolated in countries in the WHO Western Pacific Region in 2001

Country	Tested %	Less susceptible %	All TRNG* %
Australia	3,641	343	9.4
Brunei	47	23	49.0
China	748	256	34.2
Japan	300	4	1.3
Korea	177	2	1.1
Lao PDR	187	185	98.9
Malaysia	30	20	66.6
New Zealand	765	73	9.5
Papua New Guinea	369	56	15.2
Philippines	399	68	17.0
Singapore	741	499	67.3
Tonga	11	1	9.0
Vietnam	168	68	40.5

* TRNG Tetracycline resistant *Neisseria gonorrhoeae*

Acknowledgments

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Members of the Australian Gonococcal Surveillance Programme throughout Australia; Haji Mohamad Haji Kassim, Brunei Darussalam; Sar Borann, Cambodia; Ye Shunzhang and Su Xiaohong, Nanjing, China; Parmod Kumar and Sainimer Bavoro, Suva, Fiji; KM Kam, Hong Kong; Masatoshi Tanaka, Fukuoka, and Toshiro Kuroki, Yokohama Japan; K Lee and Y Chong, Seoul, Korea; Sithat Insisiengmay, Vientiane, Lao PDR; Rohani Yasin, Kuala Lumpur, Malaysia; B Garin, Noumea, New Caledonia; M Brokenshire, Auckland, New Zealand; M. Hombhanje, Port Moresby, Papua New Guinea; CC Carlos, Manila, Philippines; Cecilia Ngan and AE Ling, Singapore; Ane T Ika, Tonga; Le Thi Phuong, Hanoi, Vietnam.

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Errata

Sentinel Chicken Surveillance Programme in Australia, 1 July 2001 to 30 June 2002

The following corrections to *Commun Dis Intell* 2002;26:428–429 should be noted.

It was stated that there was no flavivirus activity in Queensland in the 2001/2002 season. This information was incorrect. Although there was no MVE activity detected in northern Queensland during this period the Public Health Virology Laboratory at the Queensland Health Scientific Services did detect flavivirus activity from mid-February to mid-April 2002. There were 4 seroconversions to KUN virus and 5 seroconversions to flaviviruses that could not be typed to MVE or KUN viruses. This information became available after the article was published.

Dr Annette Broom

Editorial: Diarrhoea associated with consumption of escolar (rudderfish)

The photographs of oilfish, escolar and rudderfish (Figures 1 to 4) were incorrectly credited. The photos were from the CSIRO Marine Research. We apologise for this error.

OzFoodNet: enhancing foodborne disease surveillance across Australia: quarterly report, April to June 2002

The OzFoodNet Working Group¹

Introduction

OzFoodNet is a collaborative network of epidemiologists, microbiologists and food safety specialists conducting applied epidemiological research into foodborne disease and improving existing surveillance mechanisms for foodborne disease. The Commonwealth Department of Health and Ageing established OzFoodNet in 2000 and the network has had representation on the Communicable Diseases Network Australia since 2001. All Australian jurisdictions participate in OzFoodNet.

This second quarterly report of OzFoodNet for 2002 summarises the incidence of foodborne disease in the 6 States of Australia and the Australian Capital Territory between April and June 2002. During the second quarter of 2002, OzFoodNet continued to collect data on the incidence of gastroenteritis and its causes around Australia. The New South Wales Health Department has enhanced surveillance in the Hunter Region, although data are reported for all of New South Wales where available. The Northern Territory participates as an observer, and data are only included where specified. All data are reported using the date the report was received by the health agency. Notifications received in the current quarter are compared against 4-year historical means for the years 1998 to 2001.

Notifications in the second quarter

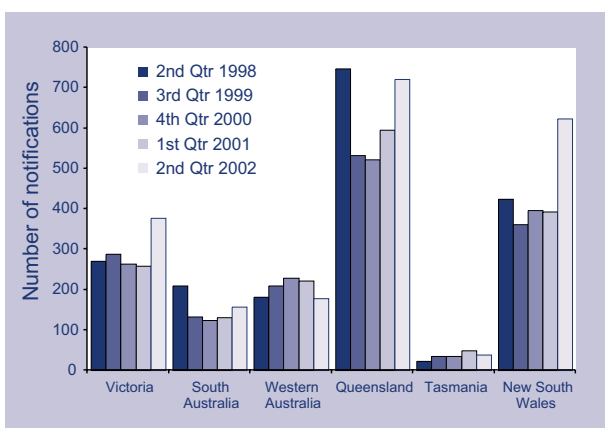
In the second quarter of 2002, there were 3,180 notifications of *Campylobacter* infection, which was slightly higher (7.4%) than the historical mean. South Australia had a 1.4 per cent decrease in the number of notifications, while the Australian Capital Territory experienced the largest increase of 24.4 per cent. The median age of cases in different sites was similar for the previous quarter and ranged from 27 to 30 years of age. Sites reported that the male to female ratio of cases ranged from 0.9–1.2:1.0. There were no reported outbreaks of *Campylobacter* infection during the quarter.

The incidence of salmonellosis in the second quarter 2002, was higher than in the previous 4 years in all OzFoodNet sites, except for Western Australia (Figure 1). Sites reported a total of 2,120 cases of salmonellosis during the second quarter of 2002, which represented a 26.5 per cent increase over the historical mean. All of New South Wales and the Australian Capital Territory combined, saw a marked increase in the number of notifications (58.1%) compared to the historical mean. OzFoodNet sites recorded that the median ages of cases ranged from 9 to 28 years. The lowest median ages of notified cases were recorded in Queensland (6.5 years), Tasmania (9 years) and Western Australia (11 years), while the remaining sites reported that the median age of notified cases was greater than 20 years. The male to female ratio ranged from 0.8–1.2:1.0.

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Figure 1. Notifications of salmonellosis in OzFoodNet sites during the second quarter in the years 1999 to 2002



During the quarter, there were four serovars that were among the most common in three or more states: *Salmonella* Typhimurium (phage types 9, 126, and 135), and *S. Saintpaul*. New South Wales experienced community-wide increases in several serovars, including 52 cases of *S. Bovismorbificans* phage type 24. In this quarter, *S. Typhimurium* 170 and *S. Heidelberg* continued to emerge as significant serovars in Queensland. The Victorian Department of Human Services reported continued emergence of *S. Typhimurium* 170. South Australia reported the emergence of two serovars that were previously uncommon in that State; *S. Typhimurium* 8 and *S. Typhimurium* 145. The OzFoodNet-Tasmania site reported a case of *S. Kalumburu*; a serovar that had not previously been isolated from humans. There was no obvious source for the infection. The Victorian site reported investigating four small clusters of *Salmonella*, including 3 cases of *S. Mississippi*. Two cases of *S. Mississippi* had travelled to Tasmania, and the third had eaten Tasmanian produce. Table 1 shows the five most common *Salmonella* infections reported to OzFoodNet during the quarter compared to the same quarter last year.

During the second quarter of 2002, the National Enteric Pathogen Surveillance Scheme reported that the five most common *Salmonella* infections nationally were *S. Typhimurium* 135 (168 cases), *S. Typhimurium* 9 (134 cases), *S. Typhimurium* 170 (122 cases), *S. Saintpaul* (99 cases), and *S. Typhimurium* 126 (72 cases). (Joan Powling, The University of Melbourne, personal communication, 18 July 2002).

State health departments received 17 notifications of listeriosis during the second quarter of 2002, which was 27 per cent higher than the historical mean for the previous four years (14 cases). All of these cases were reported in older people with severe immunocompromising conditions. The Queensland site reported 47 per

cent (8/17) of cases. Isolates from three of the five Queensland cases notified in April and May had indistinguishable Pulse Field Gel Electrophoresis patterns, although these patterns were also seen in isolates from two historical cases. There were no common foods identified in food histories of patients. The median age of cases from all sites ranged from 66 to 74 years, and the overall male to female ratio was 1.0:1.1. There were no materno-foetal infections reported during the quarter.

OzFoodNet sites reported 13 cases of enterohaemorrhagic *E. coli* infections during the quarter, which was a 58 per cent increase over the historical mean. The majority of cases were notified from South Australia (n=7) where enhanced surveillance is undertaken. Cases were also reported from New South Wales (n=2), Victoria (n=2), Western Australia (n=1), and Queensland (n=1). There were no common links identified between cases. No serotype was recorded for 62 per cent (8/13) of infections. Five were reported as *E. coli* O157 infections. The median ages of cases in different sites ranged from 39 to 68 years. Notifications were common among females (male to female ratio was 1.0:1.6), which was similar to the first quarter of 2002. There were three cases of haemolytic uraemic syndrome reported during the quarter, all of which were in New South Wales. Details of infecting *E. coli* serotype were not available.

OzFoodNet sites reported that during the quarter there were 8 cases of typhoid, which represented a 36 per cent decrease on the mean of the previous four years. Sites also reported 88 cases of shigellosis and 22 cases of yersiniosis, which represented decreases of 12 per cent and 14 per cent from the mean of the previous four years, respectively.

Foodborne disease outbreaks

During the second quarter of 2002, OzFoodNet sites reported 22 outbreaks of gastrointestinal infections with a probable food source, compared to 16 outbreaks for the second quarter of 2001. The outbreaks affected an estimated 642 people, of whom 26 were hospitalised (Table 2). There were no deaths associated with foodborne disease outbreaks during the period. Sites conducted nine retrospective cohort studies and four case control studies to investigate these outbreaks, with the remaining nine investigations relying on descriptive information. Forty-five per cent (10/22) of outbreaks for the quarter occurred in April. The majority of outbreaks (41%) occurred in conjunction with meals at restaurants, while 23 per cent occurred following functions or meals in homes. Nine of the outbreaks were of unknown aetiology.

Table 1. Number of notifications for the five most common *Salmonella* infections reported to OzFoodNet sites for the second quarter 2002 compared to second quarter 2001

OzFoodNet site	Top five <i>Salmonella</i> infections	Number of cases				
		2nd Qtr 2002	2nd Qtr 2001	YTD 2002	Total 2001	Ratio*
ACT	S. Typhimurium 135	5	0	7	2	–
	S. Typhimurium 9	3	0	15	10	–
	S. Typhimurium 126	1	0	1	4	–
Hunter	S. Agona	7	0	7	1	–
	S. Typhimurium 170	4	2	5	6	2.0
	S. Typhimurium 195	4	0	4	0	–
	S. Chester	3	0	4	1	–
	S. Javiana	3	0	3	0	–
NSW	S. Typhimurium 9	68	30	221	132	2.3
	S. Typhimurium 135	57	34	134	200	1.7
	S. Bovismorbificans phage type 24	52	0	53	1	–
	S. Typhimurium 126	40	8	55	96	5.0
	S. Typhimurium 170	36	8	92	35	4.5
Qld	S. Virchow 8	74	62	212	125	1.2
	S. Saintpaul	57	40	170	107	1.4
	S. Hvittingfoss	43	24	94	36	1.8
	S. Chester	31	18	60	49	1.7
	S. Birkenhead	30	33	86	95	0.9
SA	S. Typhimurium 8	49	0	53	3	–
	S. Typhimurium 145	16	0	17	0	–
	S. Typhimurium 126	9	25	27	110	0.4
	S. Saintpaul	7	0	9	5	–
	S. Typhimurium 108	7	5	19	31	1.4
Tas	S. Mississippi	26	30	59	102	0.9
	S. Typhimurium 135	4	1	12	4	4.0
	S. Typhimurium 4	1	2	1	1	0.5
	S. Typhimurium 195	1	0	1	0	–
	S. Typhimurium U290	1	0	2	0	–
Vic	S. Typhimurium 170	78	3	126	72	26.0
	S. Typhimurium 135	67	18	134	92	3.7
	S. Typhimurium 9	63	33	97	127	1.9
	S. Typhimurium 126	15	7	36	16	2.1
	S. Saintpaul	15	2	27	10	7.5
WA	S. Typhimurium 135	16	13	53	89	1.2
	S. Saintpaul	14	16	29	45	0.9
	S. Typhimurium 9	11	2	33	18	5.5
	S. Muenchen	10	7	18	26	1.4
	S. Typhimurium 141	8	5	10	9	1.6

* Ratio of cases for the second quarter 2002 to the second quarter of 2001

Table 2. Outbreaks reported by OzFoodNet sites, April to June 2002

State	Month of outbreak	Setting category	Agent responsible	Number affected	Evidence	Responsible vehicles
ACT	April	Miscellaneous	Suspected virus	192	D	Unknown
NSW (Hunter)	April	Takeaway	Unknown	5	D	Pizza with seafood topping
	May	Restaurant	Unknown	3	D	Suspected seafood
	May	Restaurant	Unknown	5	D	Unknown
	June	Restaurant	Unknown	4	D	Unknown
	June	Restaurant	Unknown	4	D	Suspected seafood
Qld	April	Home	Ciguatera fish poisoning	3	D	Grunter bream
	May	Restaurant	Unknown	7	D	Unknown
	May	Restaurant	Unknown	4	D	Unknown
	June	Home	<i>Bacillus cereus</i>	37	A, M	Rice
SA	April	Restaurant	<i>S. Typhimurium</i> 8	78	A, M	Caesar salad
	April	Community	<i>S. Typhimurium</i> 43	5	A	Sliced ham
	May	Home	<i>C. perfringens</i>	8	A, M	Potato pie
Vic	April	Home	<i>S. Typhimurium</i> 135	6	D, M	Home barbequed chicken
	April	Aged care/ health care	<i>S. Typhimurium</i> 9	18	D	Unknown
	April	Home	<i>S. Typhimurium</i> 170	6	M	Suspected eggs
	April	Child care centre	<i>Staphylococcus aureus</i>	7	M	Rice
	April	Cruise	<i>Clostridium perfringens</i>	18	D	Unknown
	May	Restaurant	Suspected <i>C. perfringens</i>	10	A	Pea and ham soup
	May	Restaurant	Norwalk virus	192	D	Unknown
	May	Community	<i>S. Typhimurium</i> U290	10	A	Cream filled cakes/pastries
	June	Community	<i>S. Typhimurium</i> 135	20	D	Vietnamese pork rolls

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

A Statistical association between illness and one or more foods.

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

* The number affected is calculated from the proportion of people interviewed who were ill, multiplied by the number of people exposed.

Seven outbreaks were due to *Salmonella* contamination. *Salmonella* Typhimurium 135 infection was responsible for two of the outbreaks, one of which was associated with barbecued chicken, and one associated with Vietnamese pork rolls. The 20 cases of *S. Typhimurium* 135 associated with the pork rolls were detected through surveillance. All cases reported eating products from the same premises on the same day. The pork rolls consisted of bread rolls with a raw egg mayonnaise, cooked meats, salad and pate. No specific problems with food safety were identified but the rolls were sometimes stored at room temperature. Vietnamese pork rolls have been responsible for large outbreaks in Australia, and were recently identified as the cause of an outbreak of *S. Typhimurium* 126 in New South Wales.^{1,2}

South Australia reported a large outbreak of *S. Typhimurium* 8 associated with Caesar salad from a restaurant. Several salad ingredients tested positive for *S. Typhimurium* phage type 8 including the salad dressing, anchovies and parmesan cheese. Victoria reported an outbreak due to *S. Typhimurium* U290 that was epidemiologically associated with consumption of cream and/or custard filled pastries from a bakery.

There were four outbreaks due to bacterial toxins following time-temperature abuse of the foods, including two due to *C. perfringens*. Queensland reported *Bacillus cereus* as the cause of an outbreak of gastrointestinal illness affecting 37 people. *Bacillus cereus* was identified in samples of rice. Epidemiological investigations found a significant association between the consumption of rice and illness. Rice had been cooked on the morning of the function and left at room temperature for the remainder of the day, prior to reheating for consumption.

Applied research

In the second quarter of 2002, OzFoodNet sites continued to recruit patients and controls for the national case control studies into *Campylobacter*, *Salmonella* Enteritidis, and *Listeria* infections. During the quarter, the Hunter-OzFoodNet site analysed the results of a *Campylobacter* case control study using typing data from nine phenotypic and genotypic methods. This typing comparison has relied on collaboration from many microbiology laboratories around Australia, which have imported data into a bionumerics database held at PathCentre in Western Australia. Preliminary results suggest that incorporating sub-typing of *Campylobacter* into epidemiological analysis reveals temporal clustering and specific risk factors for infection that were previously not recognised.

In the second quarter of 2002, 1,280 people were interviewed as part of the national OzFoodNet gastroenteritis survey. Overall 9.4 per cent of people experienced gastroenteritis compared to 12.2 per cent for the first quarter of 2002 (Table 3). There were noticeable differences by season in different jurisdictions, and the prevalence of gastroenteritis was highest in summer (Figure 2). During the quarter, residents of Tasmania reported the lowest crude proportion of people experiencing gastroenteritis in the previous month, and Victoria reported the highest. Nationally, the prevalence of gastroenteritis was highest for respondents interviewed in the month of April (9.8%). This is self-reported gastroenteritis and does not distinguish foodborne illness from other causes of gastroenteritis.

Table 3. Unweighted results of the national OzFoodNet gastroenteritis survey between January and March 2002 and April and June 2002, showing the number and proportion of respondents reporting an episode of gastroenteritis in the previous month

State or Territory	January – March 2002			April – June 2002		
	No. with gastroenteritis	No. interviewed	%	No. with gastroenteritis	No. interviewed	%
New South Wales*	40	255	15.7	13	183	7.1
Northern Territory	28	204	13.7	17	204	8.3
Queensland	22	203	10.8	14	126	11.1
South Australia	22	187	11.8	12	169	7.1
Tasmania	23	215	10.7	9	154	5.8
Victoria	26	229	11.4	34	233	14.6
Western Australia	22	206	10.7	21	211	10.0
Total	183	1,499	12.2	120	1,280	9.4

* Includes the Australian Capital Territory and an over-sample for the Hunter region of New South Wales

Figure 2. Unweighted results of the national OzFoodNet gastroenteritis survey showing the proportion of respondents reporting an episode of gastroenteritis in the previous month (n=5,196), September 2001 to June 2002



During the quarter, OzFoodNet held a workshop at the National Centre for Epidemiology and Population Health to develop the assumptions used in calculating the incidence of gastroenteritis due to food. These assumptions will use data from a variety of sources about the microbiological causes of gastroenteritis in the community.

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Editorial: Norwalk-like virus — issues for surveillance

Paul Roche,¹ John Marshall,² Jenean Spencer¹

Recent closures of hospital wards in Australia and Scotland due to Norwalk-like virus (NLV) infections have increasingly focussed attention on these agents as the cause of outbreaks of viral gastroenteritis.^{1,2,3} NLV has been identified as the leading cause of community-acquired gastroenteritis in a number of countries, including the Netherlands.⁴ Since the disease is mild, and specific surveillance for NLV has not been carried out, the actual incidence in the United Kingdom is considered to be more than 1,000 times greater than reported.⁵ Although associated with 'winter vomiting' in temperate climates,^{6,7} there is evidence for the virus causing gastroenteritis year-round. It has been suggested that there are differences in incidence year by year depending on the circulating strain.⁸ Data from Australia suggest that there is both a seasonal peak in NLV activity between late winter and early summer and a variation year by year, with peaks of activity noted in 1995 and 1996.⁹ Foodborne outbreaks of NLV are reported frequently through OzFoodNet.¹⁰ Large outbreaks of NLV gastroenteritis associated with eating oysters and with contaminated orange juice have been reported in Australia.^{11,12}

Australian hospitalisation data for 1998–99 and 1999–00 show 13,026 and 14,110 admissions for viral intestinal infections respectively.¹³ Although only 34 and 36 hospitalisations were identified as due to NLV, 5,526 and 9,133 were for unidentified viral agents in 1998–99 and 1999–00 respectively. Due to difficulties of diagnosis, many of the admissions for gastroenteritis with an unidentified cause could be due to NLV.

It has been hypothesised that NLV strains circulating in humans may represent a spill-over of those in animal reservoirs such as cattle,⁸ although there is evidence that these strains may be distinct.¹⁴ While foodborne transmission of NLV has been frequently documented,^{15,16}

transmission by water,¹⁷ environmental contamination,¹⁸ and aerosol¹⁹ have also been documented. While around 40 per cent of NLV disease in the United States of America (USA) is estimated to be foodborne,²⁰ more recent focus has been placed on person to person transmission.^{1,21}

Transmission from person to person, particularly in the setting of an aged care facility, has important implications for infection control procedures which are discussed in the article in this issue of *Communicable Diseases Intelligence*.¹ There is uncertainty about the duration of excretion of the virus in faeces after infection and whether the virus is shed by asymptomatic people. Following oral administration of NLV to healthy volunteers, 82 per cent were infected, two thirds of these were symptomatic and stool specimens remained positive for the virus for up to 7 days.²² Several reports have since appeared of high levels of NLV excreted up to 10 days after resolution of illness.²³ Another recent study of 99 subjects infected with NLV found 26 per cent were excreting the virus up to 3 weeks after the onset of illness.²⁴ The relatively long term excretion of NLV calls into question infection control guidelines which allow the return of staff to aged care facilities 24 to 48 hours after the cessation of symptoms.

While high rates of NLV infection are found in children early in life,²⁵ NLV is now also recognised as an important cause of gastroenteritis in the elderly.²⁶ Thus, much of the recent prominence of NLV may be due to increasing institutionalisation of the elderly. Among the elderly, immunocompromising conditions may also be important risk factors for NLV infection. Recent genetic work¹⁴ has opened the possibility that some NLV strains may be associated with particular settings such as aged care facilities.²⁶

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Until recently, diagnosis of NLV has been dependent on the identification of the virus particles by electron microscopy. Utilising published genomic sequences a number of investigators have developed reverse transcription (RT)-PCR methods for the detection and differentiation of NLV genogroups.^{27,28} These methods can detect fewer than 100 virus particles in 5µl faecal extract and are six times more sensitive than electron microscopy.²⁸ As these assays become standardised and more widely available, a more accurate assessment of the epidemiology of NLV can be expected.

Although NLV still can not be cultured in cell lines, vaccines based on Norwalk virus-like particles are being developed. The rationale behind such vaccine development is that since the NLV group is restricted to humans, vaccination would be effective in decreasing the disease burden. Although disease is mild and self-limiting in most cases, vaccines could be cost effective by reducing hospitalisations, medical consultations and time lost from work. Vaccines may be particularly useful in preventing disease in aged care facilities. However, the diversity within the genogroups of NLV is large enough to require the inclusion of multiple strains; the correlates of protective immunity are not known, nor is it known why natural infection fails to result in long lasting immunity.²⁵ There is no animal model of NLV disease in which candidate vaccines can be investigated. These factors represent considerable hurdles to vaccine development.

In the context of a high prevalence of gastroenteritis caused by NLV, which is increasingly accurately diagnosed and recognised as the cause of outbreaks, should this disease be included in communicable disease surveillance? What kinds of surveillance would be appropriate? A passive surveillance system would collect data on only a small fraction of cases, since the majority do not seek medical attention. An active surveillance system would be quickly overwhelmed by the sheer number of cases. A sentinel laboratory-based surveillance system, such as the Laboratory Virology and Serology Surveillance Scheme, would be well placed to provide data on the most significant cases, since the scheme includes many of the major hospital laboratories in the country. If there is evidence of NLV genotypes associated with different settings and temporal and geographic variation, genetic analysis of circulating strains would be useful.

Surveillance would also further our understanding of the epidemiology of NLV in Australia and identify viral strains which should be included in a future vaccine.

Acute gastroenteritis is a very common disease with estimates from recent surveys in Australia suggesting that the incidence is approximately one episode per person per year.¹⁰ For many years, acute gastroenteritis cases have been a 'diagnostic void' with a pathogen identified in less than 10 per cent of hospitalised acute gastroenteritis cases in the USA before 1970.²⁹ Improvements in diagnostic technology have identified various viral agents associated with acute gastroenteritis and it now appears that NLV infections represent a substantial proportion of acute gastroenteritis cases.⁴ The low infective dose of NLV (10 to 100 particles),⁷ and the multiple modes of transmission pose great challenges to disease control. We can expect to see further outbreaks of NLV gastroenteritis reported as diagnostic methods improve and are applied more widely. Improved understanding of the NLV virus and epidemiology will bring about new and effective tools of infection control and disease prevention.

Note: Norwalk-like viruses have recently been officially renamed the genus 'Norovirus'.³⁰

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Norwalk-like virus outbreak in Canberra: implications for infection control in aged care facilities

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Abstract

This paper reports on an outbreak of viral gastroenteritis in three institutions (two aged care facilities and one hospital) in Canberra during the winter of 2002. Norwalk-like virus genotype II was detected in samples from staff and/or residents in all three institutions. A case series investigation was conducted amongst both staff and residents. It is likely that the outbreaks in the three institutions were linked due to transfers of infected residents from one institution to another, early in the outbreak. A total of 281 cases were identified during the outbreak, which lasted 32 days. Attack rates in the three institutions were 46.3 per cent, 52.7 per cent and 55.2 per cent respectively. Person-to-person spread and/or airborne transmission were postulated as modes of transmission in all three institutions. Infection control practices in each of the aged care institutions were of an acceptable standard for accreditation, but were inadequate to control further spread of the outbreak within and between institutions. Outbreak management plans should be a part of the infection control standards for accreditation of aged care facilities. *Commun Dis Intell* 2002;26:555–561.

Keywords: Norwalk-like virus, outbreak, gastroenteritis, aged care facilities, hospitals, infection control

Introduction

Norwalk-like viruses (NLV), now also known as human caliciviruses, are a genetically diverse group of RNA viruses that are classified in the family *Caliciviridae*. There are three distinct genogroups of NLV, of which only genogroups I and II are pathogenic to humans.¹ The epidemiological characteristics of NLV illness include: incubation period of 24–48 hours; duration of illness between 12–60 hours; and greater than 50 per cent of cases reporting vomiting.² Other symptoms of NLV gastroenteritis include nausea, abdominal pain and diarrhoea.³ The main modes of transmission of NLV are person-to-person, foodborne and waterborne. Fankhauser *et al.*⁴ found that of the 233 outbreaks of confirmed NLV gastroenteritis investigated in the United States of America between July 1997 and June 2000, 57 per cent were foodborne, 16 per cent were person-to-person spread, 3 per cent were waterborne and 24 per cent had unknown modes of transmission. Airborne transmission of NLV has also been documented.^{5,6}

Nursing homes and hospitals have been common settings for outbreaks of NLV,^{7,8} due to the closed nature of these institutions and also because of the infectious nature of NLV. Attack rates from outbreaks of NLV in aged care facilities have been reported to be as high as 62 per cent in elderly residents in the Netherlands.⁹ Augustin *et al.*¹⁰ reported attack rates of 9 per cent and 11 per cent in outbreaks in two aged care facilities in Canada. Both facilities implemented infection control procedures such as increased surveillance, reinforcement of hand washing, keeping symptomatic residents in their rooms and relieving sick staff from their duties until 48 hours after the resolution of symptoms. Given these control measures, the outbreaks still lasted for 24–29 days. Outbreaks of NLV in aged care facilities in Australia have also been reported. The attack rates of three nursing home outbreaks of NLV reported in Brisbane ranged from 9 per cent to 58 per cent.⁷

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This paper details the public health response and describes the epidemiology of an outbreak of Norwalk-like virus, which occurred in two aged care facilities and a hospital in the Australian Capital Territory. The purpose of this investigation was to stop the spread of gastroenteritis in the three institutions, to identify the causative agent and identify the likely mode of transmission.

Methods

The outbreak occurred in three institutions. The environmental, epidemiological and microbiological investigation methods used in this outbreak are detailed below.

Environmental investigation

The kitchens and food handling practices in Institutions A and B (aged care facilities) were examined by Environmental Health Officers from the Australian Capital Territory Health Protection Service (ACT HPS). An infection control audit of these institutions was conducted separately by infection control officers from the ACT HPS. Institution C was a hospital with two dedicated infection control practitioners, who liaised with the ACT HPS to manage the outbreak.

Epidemiological investigation

An epidemiological investigation was conducted in each of the aged care facilities by the ACT HPS, starting at Institution A on 24 June 2002. A case was defined as a person who lived or worked at either Institution A or B and developed vomiting or diarrhoea after 1 June 2002. Staff were contacted by phone and asked to complete a standard questionnaire if they met the case definition. All staff were advised to contact the ACT HPS if they developed symptoms. The questionnaire collected information such as the date of onset, symptoms (diarrhoea, vomiting, nausea, stomach pain and fever), exposure details, food history and other locations of work. All staff who had gastrointestinal symptoms were asked to submit a stool sample for microbiological investigation.

Residents in Institutions A and B, who met the case definition were identified by staff. A questionnaire detailing symptoms and onset date was completed by senior nursing staff on behalf of each sick resident. Staff were also requested to collect stool samples from residents, where possible. Case notes of each resident were examined for evidence of vomiting or diarrhoea after 1 June 2002.

The case definition used in Institution C was a person who had vomiting or diarrhoea after 25 June for staff members who worked in any of the affected wards and after 1 June for patients admitted to any of the affected wards. The case definition was modified for staff as the number of people who had worked in the affected wards was too numerous and staff members were difficult to trace for interviews. Cases (staff and current patients) were identified in the affected wards by the infection control practitioners and details of onset date of illness and symptoms were provided. Stool samples were collected from some of the patients who met the case definition. Patients who had been in any of the affected wards since 1 June and had been discharged were followed up via telephone interviews for case ascertainment.

Microbiological investigation

Stool samples were collected from cases in each of the three institutions and were examined for parasites (*Cryptosporidium*, *Giardia*) and bacteria (*Salmonella*, *Shigella*, *Campylobacter* and *Yersinia*). Some of the stool samples were also tested for rotavirus. Samples collected within the first 24–48 hours of onset of illness were sent to the Institute of Clinical Pathology and Medical Research (ICPMR) or the Victorian Infectious Diseases Reference Laboratory (VIDRL) for testing of Norwalk-like viruses using reverse transcriptase polymerase chain reaction (RT-PCR).

Results

Description of facilities

The outbreak occurred in two aged care facilities and a hospital. Institution A was an aged care facility, where most of the residents required considerable contact with staff ('high care'). Showers and toilets were shared amongst residents and some rooms contained two beds. There was a shared dining room and several courtyards for use by all of the residents.

Institution B was an aged care hostel, where only half of the residents required a high level of care. One end of the hostel was designated for the residents who required a high level of care and the other end of the hostel was for residents who were largely independent and required minimal contact with staff ('low care'). There were two separate dining rooms, one for low care and the other for high care residents, however, residents mixed together regularly. Each resident had his or her own room and there was an ensuite bathroom in each of the rooms.

Institution C was a large public hospital. The outbreak was contained to the aged care ward and the oncology ward. Table 1 provides a summary of the institutions.

Description of investigations

The ACT HPS was notified on 24 June 2002 that 17 residents and 8 staff were sick with gastrointestinal illness at Institution A. An immediate environmental inspection was conducted but there was no evidence to suggest that the

kitchen was the source of the outbreak. The food hygiene and storage and food handling inspections were satisfactory. Before ACT HPS had conducted an infection control audit, a resident from Institution A was transferred to Institution B (on 24 June) and a sick resident who met the case definition of this outbreak was sent to Institution C (hospital) via an ambulance on 27 June. The epidemiological investigation revealed a total of 93 cases at Institution A, as shown in the epidemic curve in the Figure, of which, 52 cases were staff and 41 cases were residents. The overall attack rate in Institution A was 46.3 per cent, with a resident attack rate of 51.3 per cent and a staff attack rate of 43.0 per cent.

Figure. Number of cases of gastrointestinal illness by date of onset in three institutions, Australian Capital Territory, June to July 2002

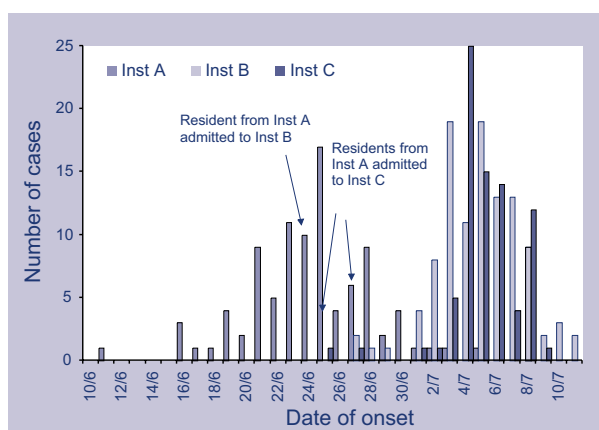


Table 1. Description of the three institutions involved in the outbreak

Institution	Level of care	No. of beds	Shared rooms	Shared toilet/ bathroom facilities	Shared dining areas
A	High care	92	Yes	Yes	Yes
B	High care	48	No	No	Yes
	Hostel	53	No	No	Yes
C	Aged care ward	21	Yes	Yes	Yes
	Oncology ward	23	Yes	Yes	Yes

As a result of the outbreak in Institution A, the ACT HPS contacted other aged care facilities in the Australian Capital Territory to determine if other facilities were experiencing elevated levels of gastrointestinal illness amongst residents or staff. On 3 July 2002, the Director at Institution B contacted ACT HPS to notify 8 cases of gastroenteritis overnight. Inspections of the sanitary condition of the kitchen and food hygiene practices were deemed satisfactory during a routine inspection on 1 July.

The resident who was transferred from Institution A on 24 June developed symptoms in Institution B on 27 June, which marked the beginning of the outbreak in Institution B (Figure). A staff member also developed symptoms on 27 June, however, this staff member was on work experience and was only present at Institution B on 24 and 25 June. This staff member had contact with the residents but was not a carer. A total of 108 cases were identified during the investigation with 56 residents and 52 staff members becoming ill with gastrointestinal illness in Institution B. The attack rate in staff (48.6%) was lower than in the residents (57.1%).

On investigation, a total of 80 cases were identified in Institution C and patients had a higher attack rate (66.1%) than staff (48.3%). As mentioned above, a sick resident from Institution A, who met the case definition, was transferred to the hospital via ambulance on 27 June. The ambulance officer developed symptoms on 29 June. The resident was admitted to the aged care ward and on 1 July another patient in this ward became ill (Figure).

Infection control audits

Infection control audits of Institution A and Institution B were conducted by infection control practitioners from the ACT HPS within one day of the outbreak being reported in Institution A and on the day of first report in Institution B. The infection control programs in Institutions A and B were of a standard to achieve Commonwealth accreditation in 2000. This accreditation process requires facilities to have an effective infection control program. The results of the audits suggest that some infection control measures were not in alignment with best practice, which is detailed in the National Health and Medical Research Council's *National Infection Control Guidelines*.¹¹ A summary of the results from the infection control audits in Institutions A and B are shown in Table 2.

Control measures were taken to address the issues identified in Table 2. The use of personal protective equipment for staff when working with sick residents, strict hand washing between contact with each resident, no new admissions or transfers to other aged care facilities and grouping sick residents away from well residents were among the infection control measures put in place. Follow-up audits found both facilities adhering to the recommendations.

One of the main issues that prolonged the outbreak within institutions A and B was the return to work of sick staff before they had recovered from the infection. At least 14 staff were identified who returned to work within the 48 hours period after cessation of symptoms.

Table 2. Summary of infection control issues identified in Institutions A and B

Infection control processes	Institution A	Institution B
High pressure hoses in pan room	Yes	Yes
Protective apparel in hose room	No	No
Knowledge on body fluid spills	No	No
Access to spill kits	Limited	Limited
Procedure for cleaning shower chairs	No	NA
Appropriate use of protective apparel when working with sick residents	No	No
Adherence to staff sickness procedures	No	No
Transfers between institutions during outbreak	Yes	Yes

NA Not applicable

Institution C had its own transmission-based infection control practices in place and a hospital wide infection control program. The two wards affected at the hospital were isolated immediately after the outbreak was identified and no new patients were admitted to either ward. The ACT HPS liaised with the infection control practitioners at the hospital to ensure that outbreak management procedures were being adhered to.

Patient outcomes

The characteristics of the outbreak and the cases are given in Table 3. The median duration of illness was not calculated as most cases were interviewed whilst they were still symptomatic. In addition, case notes for residents did not detail onset times nor duration of illness.

A total of four residents were hospitalised (in Institution C) during the outbreak, three from Institution A and one from Institution B. One resident from Institution A, with a history of a chronic neurological condition, died on 25 June 2002. There was a total of 49 GP consultations

for both staff and residents in both Institutions A and B.

Some secondary transmission of the virus to household members was also observed. From interviews with staff members, 6 cases were identified amongst family members of staff from Institution A. Secondary transmission to household contacts of staff from Institution B was observed and a total of 7 secondary cases were identified from staff interviews. In addition, anecdotal reports from sentinel GP practices in the Australian Capital Territory reported a higher number of consultations for gastrointestinal illness during the period of the outbreak, which may suggest that there was a high level of illness in the community at the time of the outbreak.

Forty-two stool samples were collected and all were negative for protozoal and bacterial pathogens. Of the 42 samples, 14 tested positive for Norwalk-like virus genotype II. As shown in Table 3, NLV genotype II was detected in each of the institutions, with the virus detected in 12 residents' samples and two staff samples.

Table 3. Characteristics of gastroenteritis outbreaks in three institutions, June to July 2002, Australian Capital Territory

Outbreak characteristics	Institution		
	A	B	C
Duration of outbreak (days)*	25	16	15
Number of cases			
Staff	52 [†]	52	43
Residents	41	56	37
Symptoms (%)			
Vomiting	68.8	75.0	66.7
Diarrhoea	78.5	83.0	86.3
Attack rate (%)			
Staff	43.0	48.6	48.3
Residents	51.3	57.1	66.1
Total	46.3	52.7	55.2
Number of stool samples tested	23	13	6
Number of NLV samples positive	2	8	4
Male:female ratio	1:3.9	1:5.4	NO
Median age (years)			
Staff	47.0	45.0	NO
Residents	84.0	87.5	NO
Outcomes			
GP consultation (% total cases)	27 (29.0)	22 (20.4)	NA
Hospitalised [‡] (% total cases)	3 (3.2)	1 (0.9)	NA
Died (% total cases)	1 (1.1)	0 (0)	0 (0)

* Duration of outbreak from day of first case to day of last case.

† Includes an ambulance officer who transported a resident from Institution A to Institution C.

‡ All hospital admissions were residents who were transferred to Institution C.

NO Information not obtained in questionnaire.

NA Not applicable.

Discussion

This investigation identified a widespread outbreak of gastrointestinal illness due to Norwalk-like virus in three institutions, probably as a result of a breakdown in infection control procedures or the transfer of ill residents to other facilities. Evidence suggestive of a link between the institutions includes: (a) identification of NLV genotype II from each institution; and (b) transfers from Institution A to Institutions B and C corresponding to the index cases in these institutions. The identification of NLV genotype II was observed in both staff and residents in both aged care facilities and in the patients from the hospital. However, NLV genotype II encompasses 10 distinct genetic clusters¹² and more specific genotyping data were not available to prove that the causative agents were genetically identical in each institution.

The environmental investigation conducted by Environmental Health Officers in the aged care facilities did not find deficiencies in kitchen hygiene or practices. The epidemic curve for each institution was not suggestive of a point source outbreak. The likely mode of transmission was person-to-person via faecal-oral route or by airborne transmission. Interviews with staff members identified several cases where person-to-person transmission could have occurred. For example, the staff who cared for the first sick residents at Institution B reported cleaning up vomit and diarrhoea and then became ill themselves 2 days later. Also, the resident transferred from Institution A to the hospital vomited in the ambulance on the way. The ambulance officer became sick with gastroenteritis 2 days later.

Outbreaks of gastroenteritis caused by NLV have been recorded throughout the world^{13,14} and in Australia.¹⁵ Genotype II NLV is the genetic group most commonly associated with outbreaks of gastroenteritis in nursing homes¹⁶ and hospitals⁹ and was found in all three institutions in the current investigation. Norwalk-like viruses can have a large detrimental impact on aged care facilities due to the highly susceptible population. The results from this investigation suggest that limitations in the infection control programs assisted in the spread of the illness within facilities and resident transfer between institutions led to the spread of NLV.

Infection control policies and practices were sub-optimal in the two aged care facilities. Some of the breakdowns in infection control include, the lack of protective apparel or improper use of protective apparel when present, improper use of spill kits and lack of policies for cleaning shower chairs between bathing each of the residents. It is therefore, imperative that aged care facilities have sound infection control policies and practices and it is recommended that outbreak management plans be incorporated into existing aged care accreditation standards. Outbreak management plans should include:

- (a) notification of hospitals where ill residents may be sent (so the hospital can implement infection control procedures);
- (b) provision to stop the transfer of residents between aged care facilities once an outbreak management plan has been activated; and
- (c) exclusion of sick staff from work duties until 48 hours after the cessation of symptoms.

Some broad principles of infection control used in public health care settings, including the use of Standard Precautions and strategies to educate staff and monitor compliance, should also be part of the framework to assess the effectiveness of infection control programs in residential care facilities.

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An outbreak of infections with a new *Salmonella* phage type linked to a symptomatic food handler

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Abstract

In December 2001, the South Australian Communicable Disease Control Branch investigated an outbreak of gastrointestinal illness linked to a Korean style restaurant in metropolitan Adelaide. Twenty-eight people were identified as having experienced gastrointestinal symptoms subsequent to dining at the restaurant between 9 and 12 December 2001. A case-control study implicated mango pudding dessert (OR 16.67 95% CI 2.03–177.04) and plain chicken (OR 10.67 95% CI 1.04–264.32). Nineteen cases and one food handler submitted faecal specimens that grew *Salmonella* Typhimurium 64var. Two samples of mango pudding and one sample of pickled Chinese cabbage also grew *Salmonella* Typhimurium 64var. The infected food handler reported an onset of illness 2 days before cases first reported eating at the restaurant. The food handler's only role was to prepare the mango pudding dessert in an area external to the restaurant's kitchen. Illness was strongly associated with consumption of a contaminated mango pudding dessert, with contamination most likely resulting from the symptomatic and culture positive food handler who prepared the dish. This outbreak demonstrates the importance of excluding symptomatic food handlers, and the need for appropriately informing and educating food handlers regarding safe food handling procedures. Restaurants with staff and management from non-English speaking backgrounds should be specifically targeted for education that is both culturally sensitive and language specific. *Commun Dis Intell* 2002;26:562–567.

Keywords: *Salmonella* Typhimurium, disease outbreak

Introduction

On 13 December 2001, a public health nurse at the South Australian Communicable Disease Control Branch (CDCB) detected a potential outbreak of acute gastrointestinal illness linked to a Korean restaurant in Adelaide. Three medical notifications had been received over a 2-day period, all of which had implicated the same restaurant (restaurant A) as the possible source of their illness. One case was a confirmed *Salmonella* infection and the remaining 2 cases were notified as cases of suspected food poisoning. Initial inquiries revealed that these cases belonged to two different groups who had dined at the restaurant on consecutive evenings. The restaurant implicated is a licensed 'self serve' restaurant, with facilities available for patrons to cook their own meat and seafood. It is located in metropolitan Adelaide, in an area well known for its range of multicultural restaurants.

An investigation was commenced to confirm the existence of an outbreak and the link to the restaurant to determine the source of the infection and to initiate public health measures to prevent further cases in the community.

Methods

Case series

A case series investigation was commenced and initial cases were defined as any person who was reported to the CDCB with a gastrointestinal illness subsequent to dining at the implicated restaurant. These cases were interviewed using open-ended questionnaires to obtain illness, travel, and seven day food histories to confirm the suspected association with the restaurant and other ill persons. A menu was requested from the restaurant, however, cases were asked for a brief description of menu items and beverages consumed at the restaurant prior to the menu being available.

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Despite requests made to the restaurant, a booking list was not available to assist in the process of identifying more cases. Reports from the restaurant suggested they rely primarily on passing trade and only rarely take telephone bookings. Therefore, in order to identify other patrons of the restaurant that were potentially exposed at the same time, cases were asked for names and contact details of all people with whom they dined.

From the initial case series investigation the hypothesis that illness was associated with the consumption of a food item at restaurant A was established.

Case control study

A case control study was initiated to test the hypothesis that illness was associated with the consumption of a specific food item at restaurant A. For this study, cases were identified from notifications of *Salmonella* infections and suspected food poisoning that were received by the CDCB and that implicated a restaurant as the possible source of their illness. A suspected case was defined as a person who had an onset of diarrhoea (defined as three or more loose bowel motions in a 24-hour period) within 3 days of eating a meal at the restaurant and was reported to the CDCB or to another person who dined at the restaurant. A case was confirmed if they had *Salmonella* Typhimurium phage type 64var (STM 64var) isolated from a stool specimen. Those who did not become ill after eating at the restaurant, as nominated by the cases, were used as unmatched controls.

A structured questionnaire was developed based on the set menu and preliminary findings. Telephone interviews were carried out with cases and controls using the structured questionnaire, and sought to determine the time of onset of symptoms and illness characteristics in cases, consumption of specific menu food items and beverages, as well as their own food handling practices, and cooking methods employed at the restaurant. Data analysis was conducted using Epi Info version 6 software, and both suspected and confirmed cases were included in the analysis.

Environmental investigation

Environmental Health Officers from the local council and the Environmental Health Branch of the Department of Human Services conducted

several inspections of the restaurant premises over a number of days. The first inspection was conducted 5 days after cases first reported eating at the restaurant. They assessed the kitchen for appropriate hygiene and sanitation practices. Food handlers were observed for appropriate food handling techniques, and information was sought using a specifically constructed staff questionnaire, regarding recent staff illness and overseas travel. Enquires were also made about the supply of food, including sources of meats, fruits and vegetables, and other packaged foods.

Laboratory investigation

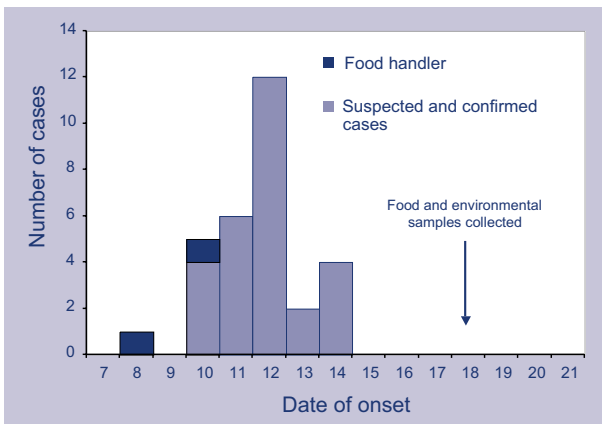
Nineteen cases and one food handler submitted faecal specimens to the Institute of Medical and Veterinary Science (IMVS) for microbiological testing including culture for *Salmonella*, *Shigella*, and *Campylobacter* species. Samples were also examined for parasites with direct and indirect microscopy. A range of food samples, including raw beef, fish, chicken, pork, mussels, capsicum, tomatoes, mushrooms, sweet and sour cabbage, spiced cucumber, lettuce, pickled Chinese cabbage (Kim Chi), and mango pudding were sent to the Food and Environmental Laboratories located at the IMVS for *Salmonella* culture. Food samples were also examined for total bacterial plate count, as well as for the presence of other specific organisms, namely *Escherichia coli* and faecal (thermotolerant) coliforms. Phage typing of both human and non-human *Salmonella* isolates was performed at the Australian Salmonella Reference Laboratory. Environmental swabs of kitchen and food preparation areas, and gloves used by food handlers were also collected and submitted for microbiological testing. Both food and environmental samples were collected 9 days after the first cases reported eating at the restaurant, with the sampled food items having been freshly prepared ready for serving on that day.

Results

In total, 28 cases were identified from notifications of *Salmonella* infection and suspected food poisoning that fulfilled the initial case definition. They reported eating at the restaurant over a period of 4 days, between 9 and 12 December 2001 (Figure). All reportedly dined in the evening, between 5.30 pm and 11.00 pm, on one of the 4 days. Of the 28 cases, 19 were

confirmed cases, having STM64var detected in a stool specimen. An additional STM 64var positive case linked to the restaurant was identified, however, this case resided interstate and could not be fully interviewed other than to confirm the link to the restaurant.

Figure. Number of cases of gastrointestinal illness categorised by date of onset



The cases belonged to 10 different groups of diners, comprising a total of 37 people. The number of persons in each group ranged from two to seven. The total number of diners at the restaurant over the 4-day period could not be accurately determined from restaurant records, however, it is conservatively estimated that over 240 persons had dined at the restaurant during the specified time period.

The average age of cases, where known, was 33 years (median: 28 years; range 13–63 years; $n=26$) and 57 per cent of cases were female. Eighty-nine per cent consulted a medical practitioner. Symptom prevalence for the 28 cases were: diarrhoea 100 per cent, vomiting 64 per cent, abdominal cramps 100 per cent, nausea 93 per cent, fever 96 per cent, lethargy 100 per cent. No cases reported bloody diarrhoea. The incubation period of each case ranged from approximately 5 hours to 53.5 hours. The average incubation period was 21 hours (median: 18 hours). At the time of interview, 15 cases reported that they were still unwell. Of the 13 whose illness had resolved, duration of illness ranged from 3 days to more than 5 days.

All 28 cases identified in the case series investigation fulfilled either the suspected or confirmed case definition and were enrolled in the case-

control study. Nine controls (as nominated by the cases) were enrolled in the study. The average age of the control group was 21 years (median: 28 years; range 3–51 years; $n=9$), and 66 per cent were males. The sex distribution did not differ significantly between the case and control groups (Fisher's exact two tailed test, $p=0.26$), however, the average age of each group was significantly different ($t=1.81$ $p=0.03$).

Exposure histories to approximately 50 foods, beverages, and condiments were obtained from the 28 cases and 9 controls. An additional three questions sought information regarding meat, poultry, and seafood that was cooked by the patrons themselves on a barbecue located at each table. Most cases and controls were interviewed as they were notified, with the time between exposure and interview ranging from 7 to 16 days. Several food items had undefined odds ratios, including the pickled Chinese cabbage, the sweet and sour cabbage, turkey, ox liver, and ox tongue. Indeed, many other foods had elevated ORs, including sweet bean curd, fish, squid, and cocktail sausage. However, the association of illness with these foods was not significant as the limits of the 95 per cent confidence interval for each food item included one. Additionally, the number of cases and controls that consumed each of these items were insufficient to explain the distribution of illness.

The consumption of 'plain' chicken was significantly associated with illness (crude OR 10.67 95% CI 1.04–264.32). Only 16 cases however, reported eating this dish. Two other chicken dishes, chili chicken and chili chicken wings, were not associated with illness. The consumption of any one of the three chicken dishes was not statistically significant (crude OR 6.67 95% CI 0.86–59.48). Eating undercooked meat or seafood, or using the same utensils (such as chopsticks or tongs) for cooking and then eating meats cooked on the barbecue, were not associated with illness.

Mango pudding was significantly associated with illness with a crude OR of 16.67 (95% CI 2.03–177.04) (Table). Twenty-five cases reported consuming between one and eight servings of the pudding. Three out of the 9 controls also reported eating the mango pudding dessert.

Table. Odds ratios and consumption distribution among cases and controls for some food items (Cases=28 Controls=9)

	Foods eaten		Foods not eaten		OR	95% CI
	Cases	Controls	Cases	Controls		
Plain chicken	16	1	12	8	10.67	1.04–264.32
Any chicken dish	25	5	3	4	6.67	0.86–59.48
Lamb	11	2	17	7	2.26	0.32–19.65
Kangaroo	5	1	23	8	1.74	0.14–46.36
Pickled chinese cabbage	8	0	20	9	?	?
Mango pudding	25	3	3	6	16.67	2.03–177.04
Sweet bean curd	13	1	15	8	6.93	0.68–171.14

Environmental findings

All foods at the restaurant were served in a buffet style where patrons 'served themselves'. There were several buffets with sections for hot dishes, salads and desserts. Raw meat and seafood were also selected from the buffet and cooked by patrons at small gas barbecues located on each table. Approximately one barbecue was available to every two patrons. All food items, apart from the mango pudding dessert, were prepared in the restaurant kitchen, including sauces and marinades.

Further investigation of the mango pudding dessert identified that it was the only food item not prepared in the kitchen. It was prepared in an area at the back of the restaurant where a small bench space and sink were available. The utensils used in its preparation were dedicated to this task. It was made fresh daily in individual servings. Ingredients included a dry crystal (gelatine) mixture, boiling water, and sliced fresh mangoes (layered on top). The freshly prepared dessert was placed directly into refrigeration until required by the customers.

Inspection of the kitchen and self-serve areas revealed major inadequacies. The general condition of the kitchen with respect to maintenance and upkeep was poor. The kitchen was inadequately cleaned, and there was no sanitation step for bench tops or items that did not go in the dishwasher. There were no dedicated hand washing facilities. Raw meats and vegetables were prepared in the same bench space due to the small size of the kitchen.

However, their preparation appeared suitably separated, with vegetables being prepared prior to meats, and chopping boards and utensils dedicated to each.

Two food handlers reported gastrointestinal illness in the week prior to the restaurant inspection. The first food handler reported an onset of illness the day before cases first dined at the restaurant (8 December). She continued to work until her symptoms resolved 6 days later. The primary role of this food handler was to prepare the mango pudding dessert and to work in the front area of the restaurant. She was not involved in the preparation of any other food items. A stool specimen submitted by this food handler 2 weeks after the onset of her illness was positive for STM 64var. The second food handler reported an onset of illness on 10 December. He continued to work for 3 days while symptomatic. He was not involved in the preparation of foods, but worked mainly in the self-serve area of the restaurant, topping up empty food containers. He did not submit a stool specimen for testing. Both food handlers reported symptoms of diarrhoea, abdominal pains and fever. Neither reported recent overseas travel.

Laboratory findings

Salmonella Typhimurium 64var was detected in 25 gm samples of both the mango pudding and the pickled Chinese cabbage. Two samples of the mango pudding had 46 and 110 colony-forming units (cfu) of *Salmonella* per gram,

respectively. The single sample of the pickled Chinese cabbage had less than 3 cfu per gram, which is at the limit of detection for *Salmonella*. Both of these food samples were of very poor microbiological quality, indicated by large numbers of coliforms (both thermo tolerant and faecal) and *Escherichia coli* organisms detected in the samples. The pH of both the mango pudding and the pickled Chinese cabbage was quite acidic, approximately 4.5 for each sample.

Control measures

Public health measures were instituted at the restaurant to eliminate the source and potential spread of infection, and to prevent any further outbreaks of foodborne illness. Initial control measures included advising sick food handlers to exclude themselves from work until 48 hours after the resolution of symptoms, and disposing of food items not subject to further cooking after preparation, such as salads, fruits, vegetables, sauces and desserts. The restaurant was closed to allow for food contact surfaces, food containers, and self-serve areas to be thoroughly cleaned and sanitised. Food handlers were advised to use a hand cream effective against *Salmonella* before starting work and after using the toilet. For the purpose of long-term prevention, recommendations were made regarding the ongoing use of sanitising practices, installation of dedicated hand washing facilities, and improvement in the general repair and condition of the kitchen area. Restaurant staff members were educated about appropriate food handling practices and other food safety issues by local council environmental health officers.

Discussion

The findings of this investigation show a clear association between the consumption of mango pudding and STM 64var infection. Epidemiological and microbiological evidence support this conclusion. Descriptive evidence shows a culture positive and symptomatic food handler to be a likely source of the contamination. This evidence includes the dedicated preparation of the implicated dish by this particular food handler, and onset of illness just prior to the commencement of this outbreak. Moreover, the infected food handler had freshly prepared the mango pudding on the day of sampling, and this was several days after the last cases reported eating at the restaurant.

Food handler contamination via direct hand contact is further suggested by the considerable handling of the mangoes in the preparation of the dessert. It has been demonstrated that salmonellae can survive on the fingertips for at least 3 hours,¹ and outbreaks of *Salmonella* associated with infected food handlers contaminating food items (not subject to further cooking) via direct hand contact have been documented.^{2,3} The fact that no dedicated hand washing facilities were available at the restaurant questions the personal hygiene practices of the implicated food handler. A recent survey of Australian food premises indicated that adequate facilities influenced the likelihood of good staff practice in regard to personal hygiene, with just under one in five (17%) Australian businesses lacking adequate hand washing facilities.⁴

Our results question the role of symptom-free non-typhoidal *Salmonella* excreting food handlers in foodborne disease outbreaks. In this outbreak, no further cases were notified subsequent to the resolution of the food handler's symptoms. This is despite evidence that the organism continued to be excreted without symptoms by the food handler for several weeks. This could suggest that the amount of bacteria carried in the convalescent stages of illness is insufficient to transmit illness and that simple food hygiene will sufficiently reduce any risk that exists. Equally, irrespective of the number of organisms contained in the faeces, hands may be more easily contaminated in the acute phase of illness because stools are loose and more frequent. Regardless, this demonstrates the importance of good hygiene practice and excluding food handlers who are actually ill.

This report documents the epidemiological, environmental, and microbiological features of an outbreak of a previously unrecognised *Salmonella* Typhimurim phage type. The phage type pattern of the *Salmonella* isolated in this outbreak did not conform to that of other known phage types. Despite this, its phage pattern was similar to that of phage type 64, having only one additional phage susceptibility. Thus, the Australian *Salmonella* Reference Laboratory termed the phage pattern of this isolate 64 'variant' or 'variety', reflecting its similarity to phage type 64 in phage pattern only.

The original source of this new phage type of *Salmonella* Typhimurium can only be surmised.

The emergence of new types of foodborne pathogens, including new types of *Salmonellae*, has been facilitated by changes in these pathogens over time, as well as increasingly centralised and concentrated food production, globalisation of the food supply, and increases in the populations at risk. It is possible that STM 64var was imported from overseas, or is the result of genetic variation or changes in the *Salmonella* Typhimurium organism. It is a continuing challenge to identify these new pathogens as they emerge, understand how they contaminate food and cause illness and define and implement best prevention strategies.

This outbreak demonstrates one way in which food premises can facilitate the spread of foodborne disease. It highlights the importance of appropriately informing and educating owners of food premises and their workers regarding safe food handling procedures, sanitation and hygiene. Changes to the South Australian Food Act will see the Food Safety Standards in the Australia New Zealand Food Standards code adopted into the legislation. These standards contain requirements relating to food safety practices, premises and equipment. More specifically, they address the responsibilities of food handlers and food premises with regards to illness and personal hygiene. A recent survey was conducted by the Food Standards Australia New Zealand (formerly Australian New Zealand Food Authority) to evaluate the impact of these changes.⁴ It indicated that between 10 and 20 per cent of Australian food businesses did not know correct food handling practices, and had poor practices and knowledge of washing and sanitising procedures. Personal hygiene and approaches to staff illness were areas identified as of most concern, with over half of the businesses saying it would be acceptable for a staff member with diarrhoea to undertake food handling tasks. While legislation plays an important role in preventing the spread of potential foodborne pathogens, it is necessary

that these regulations are properly interpreted and understood by those to whom they apply. This is particularly important for those food premises that have staff and management who are culturally and linguistically diverse. In light of this particular outbreak, the local council for the area in which the restaurant is located, has proactively contacted all Asian and multicultural restaurants in their district regarding safe food practices, and will continue educating these establishments about food hygiene on a regular basis.

Acknowledgements

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A statewide outbreak of *Salmonella* Bovismorbificans phage type 32 infection in Queensland

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Abstract

Between 30 May and 1 June 2001, 10 cases of *Salmonella* Bovismorbificans infection were reported to Public Health Services, Queensland Health. Investigations included enhanced surveillance, case interviews, a matched case control study, environmental audit and microbiological testing of faecal isolates (phage typing) and implicated food products. Forty-one cases of *S. Bovismorbificans* infection were detected, 36 cases were phage type 32. A matched case control study identified that illness was associated with consumption of food from 15 outlets of a fast food chain, Company A (matched odds ratio [MOR] 17.5, 95% CI 2.0–657.3, $p = 0.004$) and consumption of a particular product, Product X (MOR undefined, $p < 0.001$) in the week before onset of illness. Manufacturers of Product X ingredients were audited. Deficiencies were identified in equipment cleansing at the salad mixture processing plant (Manufacturer M). A swab of food residue behind the cutting wheel rim of the lettuce shredder was positive for *S. Bovismorbificans* phage type 32. This appears to be the first reported Australian outbreak of salmonellosis associated with a lettuce product. The investigations suggest that inadequate maintenance of cutting equipment to prepare lettuce ingredients for Product X by Manufacturer M was a key factor in this statewide outbreak. The statewide nature of this outbreak demonstrates the role of timely serovar identification of *Salmonella* isolates by a reference laboratory as an aid to outbreak identification, and the importance of adherence to appropriate food safety procedures in the manufacture and preparation of mass produced food items for the public. *Commun Dis Intell* 2002;26:568–573.

Keywords: *Salmonella Bovismorbificans*; outbreak; fast food; lettuce

Introduction

On 30 May 2001, the State Public Health Microbiology Laboratory reported 7 cases of *Salmonella* Bovismorbificans infection to the Foodborne Disease Epidemiologist, Queensland Health. A further 3 cases were reported in the next 48 hours. The 10 cases had occurred across several public health jurisdictions within Queensland. All 10 reported cases had a faecal collection date within a seven-day period. Previously, an average of 3 cases per month of *S. Bovismorbificans* infection were reported in Queensland between 1998 and 2000, with 8 cases reported in 2001 prior to 30 May. This paper describes the subsequent identification, investigation and control of a statewide outbreak of *S. Bovismorbificans* phage type 32 infection in Queensland during May to June 2001.

Methods

A statewide outbreak control team was formed with the aim of determining the cause of these infections and preventing further cases. All other Australian states and territories were notified of this outbreak and asked to review notifications of *S. Bovismorbificans* in their jurisdictions.

Selected hospitals, laboratories and clinicians were informed of the outbreak and were requested to collect faecal samples from all suspected cases of food poisoning. All *Salmonella* isolates were forwarded to the Public Health Microbiology Laboratory, Queensland Health Scientific Services for serovar identification. Initial cases of *S. Bovismorbificans* infection were interviewed by the Public Health Unit staff using a standardised questionnaire

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that sought information on environmental and occupational exposures and diet in the week before onset of illness.

Based on these interviews, a case control study was conducted using a telephone administered questionnaire that sought details on items consumed from fast food outlets in the week before onset of illness in the cases and the corresponding period in matched controls. A case was defined as any person notified to Queensland Health from 30 May 2001 with a *Salmonella* Bovismorbificans phage type 32 infection. Controls were obtained by asking the general practitioner of the case to nominate three patients matched by age group who had attended the practitioner within the last month and who did not have symptoms of gastrointestinal disease during that time. The case control study ceased on 17 June following publication of a media article on this date that described a *Salmonella* outbreak possibly linked to a fast-food chain.

Data were entered and analysed using Epi Info version 6.04d software.¹ Unmatched and matched odds ratios with 95 per cent confidence intervals and statistical significance tests were calculated to determine any associations between an exposure and illness. Analyses were performed using all cases including those who had been previously interviewed during the hypothesis-generating interviews. Further analyses were conducted using only those cases who had not previously been interviewed during the hypothesis-generating phase of the investigation (prospective cases only).

Manufacturers of implicated food products were audited. Food retention samples and environmental swabs collected from food suppliers and raw food products collected from farms, were submitted to the State Public Health Microbiology Laboratory. All *S. Bovismorbificans* isolates were forwarded to the Institute of Medical and Veterinary Science, Adelaide for phage typing.

Results

National surveillance data confirmed that the majority of cases of *S. Bovismorbificans* phage type 32 infections reported during the first 7 months of 2001 occurred in Queensland (Figure 1, Joan Powling, National Enteric Pathogens Surveillance Scheme Co-ordinator, Microbiological Diagnostic Unit, University of Melbourne,

personal communication). No cases occurred outside Queensland during the period of the outbreak. By 30 July 2001, 41 cases of *S. Bovismorbificans* infection were notified, 36 of which were phage type 32. Thirty-two (89%) of these cases were interviewed. The median age of these cases was 22.5 years (range 1–72 years) and the M:F ratio was 1:1. Reported symptoms included diarrhoea (94%), abdominal cramps (91%) and vomiting (34%). Fourteen (44%) cases reported bloody stools. Dates of illness onset are described in Figure 2. The median time interval between onset of patient symptoms and receipt of notification was 7 days (range 3–22 days). Six (19%) of the interviewed phage type 32 cases were hospitalised. There were no secondary household cases detected.

Figure 1. Reports of *Salmonella* Bovismorbificans phage type 32 to the National Enteric Pathogens Surveillance Scheme, 1997 to July 2001

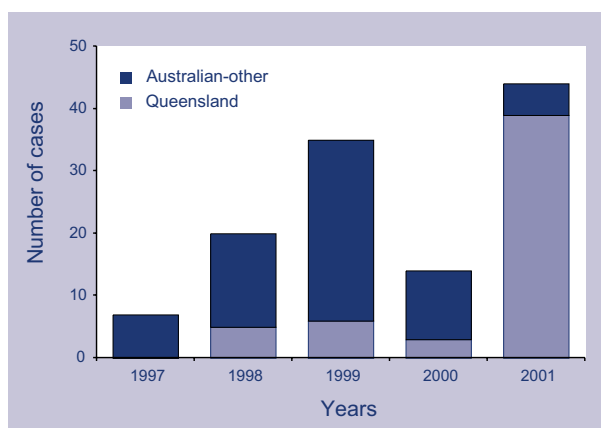
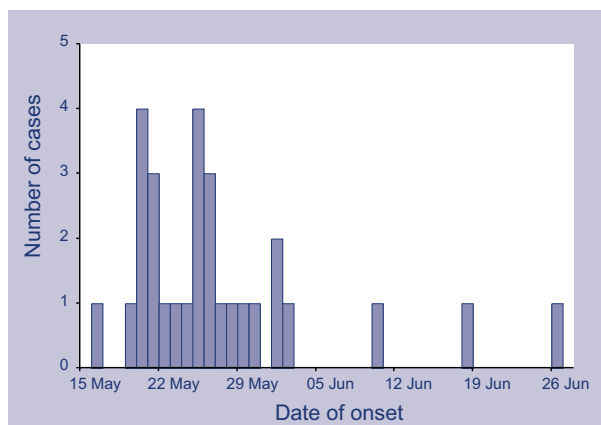


Figure 2. Notifications of *Salmonella* Bovismorbificans phage type 32, Queensland, by date of onset (n = 29)



Twenty cases (63%) reported having eaten at one of 15 outlets of a fast food chain (Company A) around the State in the week preceding their onset of illness. Most (95%) of these cases reported consuming Company A products between 13 and 30 May 2001, with one case consuming Company A products on 25 and 26 June 2001. Fourteen (70%) of these 20 cases reported consuming a particular product (Product X).

Twenty cases and 44 controls were included in the case control study. Among these 20 cases, the median age was 21.5 years (range 1–43 years), 50 per cent were aged between 20 and 39 years, and 55 per cent were female. Cases

were more likely than controls to have eaten at a Company A outlet (MOR 17.5, 95% CI 2.0–657.3, $p = 0.004$) and to have consumed Product X (MOR undefined, $p < 0.001$) in the week before their illness (Table 1). Findings were similar for cases that had not been previously interviewed with cases more likely to have eaten at a Company A outlet in the week before their illness (MOR 11.0, 95% CI 1.1 to 440.0, $P = 0.05$) and to have consumed Product X (MOR undefined, $P < 0.001$) (Table 2). There was no significant association between illness and consumption of items from any of eight other fast food chains in the week prior to onset of illness.

Table 1. Analysis of case control study of all *Salmonella Bovismorbificans* phage type 32 cases

Company/ product	Proportion exposed				Crude odds ratio	95% CI*	Matched odds ratio	95% CI	P value [†]
	Cases (n=20)		Controls (n=44)						
	n.	%	n	%					
Company A	14	70	13	30	5.6	1.5–21.2	17.5	2.0–657.3	0.004
Product U	0	0	2	5	0.0	0.0–9.6	?	–	0.90
Product V	0	0	1	2	0.0	0.0–42.8	?	–	0.72
Product W	0	0	2	5	0.0	0.0–10.1	?	–	0.80
Product X	11	55	0	0	? [‡]	9.5–?	?	–	<0.001
Product Y	2	10	0	0	?	0.4–?	?	–	0.10
Product Z	0	0	1	2	0.0	0.0–40.5	?	–	0.72
Company B	2	10	11	25	0.3	0.4–1.9	0.3	0.0–1.9	0.33
Company C	2	10	6	14	0.7	0.1–4.6	0.6	0.1–4.2	0.89
Company D	0	0	0	0	–	–	–	–	–
Company E	0	0	3	7	0.0	0.0–5.2	?	–	0.60
Company F	1	5	7	16	0.3	0.0–2.6	0.3	0.0–2.4	0.35
Company G	3	15	4	10	1.7	0.3–10.8	1.4	0.1–12.3	0.87
Company H	0	0	5	11	0.0	0.0–2.6	?	–	0.30
Company I	3	15	1	2	7.6	0.6–207.5	5.5	0.4–275.6	0.30

* 95% confidence interval

† Mantel Haenszel summary χ^2 test

‡ Odds ratios could not be calculated due to zero cell values

Table 2. Analysis of case control study of prospective *Salmonella Bovismorbificans* phage type 32 cases only

Company/ product	Proportion exposed				Crude odds ratio	95% CI*	Matched odds ratio	95% CI	P value [†]
	Cases (n=14)		Controls (n=33)						
	n.	%	n	%					
Company A	10	71	12	36	4.4	0.9–21.8	11.0	1.1–440.0	0.05
Product U	0	0	2	6	0.0	0.0–10.5	?	–	0.90
Product V	0	0	1	3	0.0	0.0–47.9	?	–	0.72
Product W	0	0	2	6	0.0	0.0–11.4	?	–	0.80
Product X	8	57	0	0	?	6.7–?	?	–	<0.001
Product Y	2	14	0	0	?	0.5–?	?	–	0.10
Product Z	0	0	1	3	0.0	0.0–44.3	?	–	0.72
Company B	2	14	9	28	0.4	0.1–2.7	0.4	0.0–2.9	0.56
Company C	1	7	4	12	0.6	0.0–6.5	0.6	0.0–5.9	1.00
Company D	0	0	0	0	–	–	–	–	–
Company E	0	0	2	6	0.0	0.0–10.5	?	–	0.90
Company F	1	7	3	9	0.8	0.0–10.0	0.9	0.0–18.2	0.61
Company G	2	14	3	9	1.6	0.2–14.6	1.4	0.1–12.3	0.87
Company H	0	0	4	12	0.0	0.0–3.8	?	–	0.42
Company I	2	14	0	0	?	0.5–?	?	–	0.21

Inspection of food safety standards at individual outlets identified no concerns. An audit of the manufacturer (Manufacturer M) of the salad mixture component used in Product X identified deficiencies in cleaning the equipment used for shredding the lettuce component of the salad mixture. A swab of food residue obtained from behind the cutting wheel rim of the lettuce shredder on 21 June was positive for *S. Bovismorbificans* phage type 32. Other environmental swabs, retention samples and source samples of Product X ingredients were negative for *Salmonella*. Epidemiological findings prompted Company A to obtain alternative supplies of salad mixture for their Product X on 20 June.

Discussion

Salmonella Bovismorbificans is a relatively common serovar with approximately 100 cases reported annually in Australia.^{2,3,4} There have been 9 outbreaks of *S. Bovismorbificans* infection recognised in Australia since 1989, associated with phage types 7, 13, 14, 21, 23, and 24 (Milka Karna-Marelj, Salmonella Reference Laboratory, Institute of Veterinary and Medical Science, personal communication). Outbreaks of *S. Bovismorbificans* in Sweden and Finland were associated with Australian alfalfa sprout seed.⁵ *Salmonella Bovismorbificans* phage type 32 has been reported in Australia only recently, with a national annual average of 19 cases between 1997 and 2000. It has been isolated from a variety of non-human sources, including ruminant animals and dogs.⁶

This outbreak of *S. Bovismorbificans* phage type 32 infection was strongly associated with eating food from Company A outlets and the consumption of Product X. The size of this outbreak is likely to be much larger than the 36 cases that were notified to Queensland Health. A recent population survey of diarrhoeal illness in Queensland found that 2.6 per cent of adults with acute diarrhoea during the preceding month had a faecal specimen submitted for pathology testing.⁷

All but one of the 20 cases of *S. Bovismorbificans* phage type 32 who indicated they had eaten Company A food had consumed it during a two and a half week period in May. Company A implemented a contingency plan on 20 June to obtain salad mixture for Product X from an interstate food manufacturer. The one case with an onset of 26 June may still be related to this outbreak on the basis of a prolonged incubation period and the case's reported regular consumption of Product X salad mixture. There were no cases notified to Queensland Health with an onset date after 26 June 2001. This supports the epidemiological evidence that Product X was the likely vehicle of transmission in this outbreak.

Based on the lack of cases appearing in other states or territories and the salad mixture being the only Product X component that was specific to Queensland, the outbreak control team suspected the salad mixture as a likely source of contamination for Product X. The manufacturer of the salad mixture (Manufacturer M) used a single lettuce shredder to prepare lettuce solely for the salad mixture for Product X. The detection of *S. Bovismorbificans* phage type 32 in food residue behind the rim of the julienne knife wheel of the shredder, supports the epidemiological evidence as to the likely vehicle of transmission in this outbreak. This evidence indicates that contaminated lettuce was the likely source of this pathogen and that the salad ingredient was responsible for the outbreak.

Six cases who ate at a Company A outlet had not consumed Product X. Four cases specified Product Y and 2 cases specified Product W. Company A confirmed that the salad mixture was also used as an ingredient of Product Y. It is difficult to explain the source of infection for the 2 cases who consumed Product W. Poor recall, cross-contamination of products, or sources other than Company A products are possible explanations.

Twelve (38%) cases of *S. Bovismorbificans* 32 infection had not eaten at Company A outlets in the week before their infection. A proportion of all cases during an outbreak will not identify the suspected exposure. Prolonged incubation periods, difficulties with recall of exposure history, or alternative unrecognised exposures may explain this finding.

The median time between onset of symptoms and notification to Queensland Health was 7 days. Cases and controls were interviewed about the fast food they had consumed in a seven-day period some 2 weeks before their interview. Consequently, recall bias is unlikely to have significantly influenced the outcome of the case control study. Questions were asked about food consumption from nine major fast food chains. There was no attempt to lead the cases or controls to choose a particular chain. The questions also specifically asked about all items on the menu thus offering the same non-leading information to both cases and controls. Interviewers were trained prior to implementation of the questionnaire and it is unlikely that they led the interviewees to their answers. Controls were obtained from GPs who were unaware of the hypothesis being tested and consequently selection bias should have minimal impact on these findings.

The similar demographic profile between all 32 cases investigated and the cases enrolled in the case control study, suggests that cases used in the case control study were representative of all notified cases. In addition, using retrospective cases in the case control study did not greatly bias the study results as shown by the similar findings when data were analysed using prospective cases only.

The environmental audit of Manufacturer M found that the cutting parts of the lettuce shredder were not being disassembled for daily cleaning as recommended in the instruction manual for the shredder. Trace back investigations did not reveal a source for the contamination of the lettuce and the manner by which the lettuce shredder became contaminated is unknown.

This appears to be the first reported outbreak in Australia of salmonellosis associated with a lettuce product. It demonstrates the importance of timely serovar identification of *Salmonella* isolates by a reference laboratory as an aid to outbreak identification. It illustrates the potential risk to public health created by the trend for

production of fresh food to be concentrated with larger food businesses, which can rapidly distribute the food to diverse geographic locations. Consequently, contamination of a single product may result in a major outbreak of foodborne illness because of the quantity of food produced and consumed.⁸ Such outbreaks may be difficult to identify and investigate. This outbreak also contains important lessons for manufacturers of fresh food products about adherence to instructions for the cleaning of food preparation equipment.

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Outbreak of staphylococcal enterotoxin food poisoning

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Coagulase positive staphylococci are generally difficult to grow in foodstuffs without substantial temperature abuse and foodborne outbreaks are uncommon. The following incident resulted in the first detection of staphylococcal enterotoxin in food in a Queensland outbreak and is the first reported outbreak of staphylococcal foodborne illness in Queensland since 1997 when 42 people in a Bundaberg nursing home became ill and subsequent faecal testing of a complainant isolated staphylococcal enterotoxin.¹

Eighteen elderly persons (from a party of 200) developed severe vomiting, diarrhoea and abdominal pain within 5 hours of consuming a pre-prepared meal of cold meat, salad and dessert at a club on 23 March 2000. Unconfirmed reports indicated that a total of approximately 50 guests (25% attack rate) were affected with many of these cases not being reported because of allegiance to the club. Two elderly females were hospitalised and had moderate and slight levels of coagulase positive staphylococci detected in faecal samples. Staphylococcal enterotoxin was detected in faecal and vomitus samples. An epidemiological and environmental investigation sought details of symptom history and exposure to potential sources of staphylococcal enterotoxin, including foods consumed.

The caterer advised that whole chickens were cooked at 200°C for 50 minutes by a butcher-delicatessen business on the morning of 22 March 2000. One batch of 18 was cooked at 10 am and placed into a hot box (for an estimated 3 hours) and another batch of 30 was cooked at 11.15 am and remained in the closed oven pending collection. A temperature check on the hot box yielded 45°C, a temperature at which bacterial growth will be supported.

The cooked chickens were collected at about 2 pm on that day and transported (40-50 minutes) in an iced esky to the luncheon venue. The temperature of the chickens (whether hot or cold) when collected is unclear. They were not transported in an approved refrigerated food vehicle as required by the Food Hygiene Regulations. The temperature within the esky is unknown and no records were kept of temperatures before, after or during transit. Outside temperatures reached approximately 28°C.

There is doubt as to whether the chickens were immediately refrigerated in a small cold room (3°C) upon arrival at the venue or placed on a food preparation bench at ambient temperature (approximately 27°C). Later that afternoon the caterer removed the chickens from the cold room and quartered them by hand. A common tea towel was used to dry hands. The chicken was consumed on the following day.

The Food Microbiology Laboratory at Queensland Health Scientific Services tested the food for coagulase positive staphylococci and found diagnostic levels of $>2.5 \times 10^6$ cfu/g in the 5 submitted samples. Using the TECRA Staphylococcal Enterotoxin Visual Immunoassay kit,² staphylococcal enterotoxin was detected in four out of five plated meals of chicken, ham, pasta and salad obtained on 24 March 2000. Further enterotoxin testing of individual food items indicated that the chicken was the most likely source of contamination. Pulsed Field Gel Electrophoresis demonstrated genetic relatedness between the food and human isolates.

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Environmental investigations concluded that improper storage temperatures post cooking and during transport were unacceptable in that the chicken was stored in the temperature danger zone (between 5°C–60°C) for a prolonged period increasing bacterial growth. Furthermore, the potential for cross-contamination was noted at the manufacturing premises due to food handlers handling both cooked and raw meats.

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Communicable Diseases Australia Website — new web address

The Communicable Diseases Australia Website is currently being redeveloped. The new look site will improve the navigational links and structure of the Website. We hope the redeveloped site will be operational early in the new year.

As part of the improvements to accessibility to our site we have shortened the url. This stage of the redevelopment has already been implemented so you can now access the site through the new web address: **<http://www.cda.gov.au/>**. This url replaces the path 'www.health.gov.au/pubhlth/cdi/'.

The previous longer addresses will still work until the new site is uploaded. The long addresses will no longer work once the site is physically moved. You may like to start updating any bookmarks (favourites) now.

There is no need to add the current home page name 'cdihtml.htm' to the web address.

Note: Some documents are external links from the Communicable Diseases Australia site. These document can be accessed through links from the Communicable Diseases Australia site or by using the 'www.health.gov.au/' address.

A farewell note

It is with regret that we farewell Ming Lin from the *Communicable Diseases Intelligence* Editorial Team. Ming joined the team in mid-1999 and has been a valuable member, particularly in regard to his expertise in data analysis. He has also contributed to the reports of national notifiable diseases and production of the influenza and tuberculosis annual reports.

Ming has taken a position with the Pharmaceutical Benefits Branch of the Department of Health and Ageing. We thank him for his hard work and contributions and wish him well for the future.

Surveillance of viral pathogens in Australia

For many years, a sentinel laboratory system, the Laboratory Virology and Serology Reporting Scheme (LabVISE) has been collecting data on viral pathogens of public health importance in Australia. In future editions of Communicable Diseases Intelligence, the editors will produce a series of articles focusing on the epidemiology of viruses and viral groups under surveillance through LabVISE which are of current public health interest.

Varicella-zoster virus

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Introduction

This article summarises current knowledge and some of the implications the introduction of universal varicella vaccination may have on the epidemiology of varicella in Australia. Appropriate surveillance strategies for this changing epidemiology are also discussed.

Varicella-zoster virus causes two distinct clinical diseases. Primary infection causes varicella or chickenpox in children and reactivation of infection causes herpes zoster (shingles) mostly among the elderly. The virus is a member of the herpesvirus family, restricted in its infective range to humans. Although chickenpox and shingles have been recognised for centuries, changes in population demographics, increasing numbers of people living with immuno-compromising conditions and the recent introduction of effective varicella vaccines could change the epidemiology of the diseases. The recent recommendation of the Australian Technical Advisory Group on Immunisation (ATAGI) to introduce universal childhood immunisation against varicella has highlighted the need to understand the epidemiology and develop surveillance strategies appropriate for Australia.

Chickenpox

Chickenpox is a ubiquitous and highly contagious infection in children, usually affecting 90 per cent of children before adolescence.¹ Before the introduction of a varicella vaccine in the United States of America (USA) in 1995, there were approximately 4 million cases per annum of which around 500,000 sought medical care, 10,000 required hospitalisation and there were 100 deaths.^{1,2} Varicella mortality declined

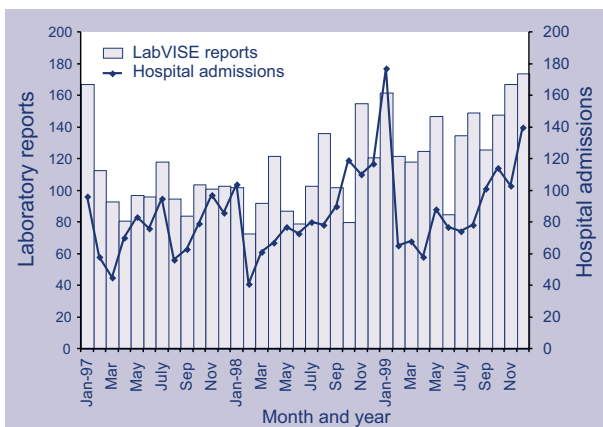
between 1970 and 1994 overall, but rates among adults increased. Adults had a risk 25 times greater and infants had a risk 4 times greater of dying from varicella than children aged 1–4 years.³ Although chickenpox is endemic in most populations, seasonal peaks may occur in late winter and early spring in temperate regions. Chickenpox may occur more frequently in adults in tropical regions than in temperate regions. Varicella infection during pregnancy may cause spontaneous abortion and intrauterine fetal infection may cause congenital abnormalities.⁴ The risk of fetal infection and damage is small but clinical manifestations may include growth retardation, skin lesions, skeletal hypoplasia, encephalopathy, eye abnormalities and structural or functional abnormalities of the gastrointestinal and genitourinary tract.

Varicella infection in the newborn varies in severity according to the timing of infection. When maternal infection occurs from 3 weeks to 5 days before delivery neonates have mild varicella disease because of protective maternal antibodies. However, if maternal varicella occurs between 5 days before to 2 days after delivery, and the virus is transmitted across the placenta, potentially severe neonatal varicella may occur, since there is no protective effect of maternal antibody. In the latter, disease develops at between 5 and 10 days of age. A case fatality rate of 20–30 per cent has been reported.⁴

In Australia, national surveillance data on the incidence of chickenpox have not been routinely collected. Sentinel surveillance data are available from laboratory reports collected through the Laboratory Virology and Serology Surveillance System (LabVISE) since 1982 and the Australian Sentinel Practice Network (ASPEN) between 1995 and 2001.

The vast majority of cases of chickenpox are well recognised by parents. A diagnosis, if made by a medical practitioner, is based on clinical signs and symptoms and does not rely on laboratory tests. Nonetheless, there have been 16,153 laboratory reports of varicella virus identification collected through LabVISE since 1982. Such reports do not distinguish between cases of chickenpox and cases of shingles. During this period, reports have been received from a varying number (13 to 26) laboratories located in all states and territories except the Northern Territory. Since this is a sentinel system, it is difficult to discern trends over time, however, a seasonal peak in laboratory reporting in January (Figure 1) is apparent. This peak coincides with a peak in hospitalisations for varicella in Australia (Australian Institute of Health and Welfare National Hospital Morbidity Databases).⁵

Figure 1. Laboratory reports of varicella-zoster virus to LabVISE and hospitalisations with a principal diagnosis of varicella, * Australia, 1997 to 1999



* ICD-10-AM codes: B01.0, B01.1, B01.2, B01.8, B01.9 and ICD-9-CM codes 052.0, 052.1, 052.7, 052.8, 052.9

Laboratory testing is more often performed in age groups in whom varicella infections are unusual. Only 758 (7%) of the 11,052 patients in whom varicella was confirmed by laboratory testing between 1991 and 2000 were aged less than 5 years (Figure 2).

The ASPREN surveillance system is a network of about 120 general practitioners, mostly located in metropolitan areas, who together record between 7,000 and 8,000 consultations per week of specified conditions. ASPREN data are reported as rates per 1,000 consultations.⁶ Between 1995 and 2001, cases of chickenpox, defined as 'an acute generalised viral disease with a sudden onset of slight fever, mild consti-

tutional symptoms and a skin eruption which is maculopapular for a few hours, vesicular for 3 to 4 days and leaves a granular rash' were reported. An examination of these data between 1999 and 2001 showed little evidence of a seasonal peak in reporting (Figure 3). The annual average rate for chickenpox was 1.6 cases per 1,000 consultations per week.

Figure 2. Laboratory reports of varicella-zoster virus to LabVISE, 1991 to 2000, by age and sex

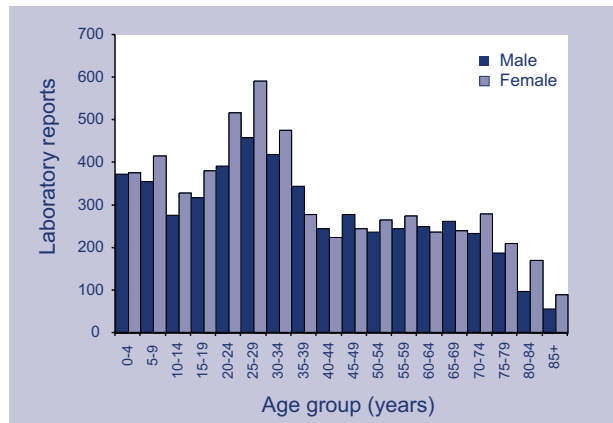
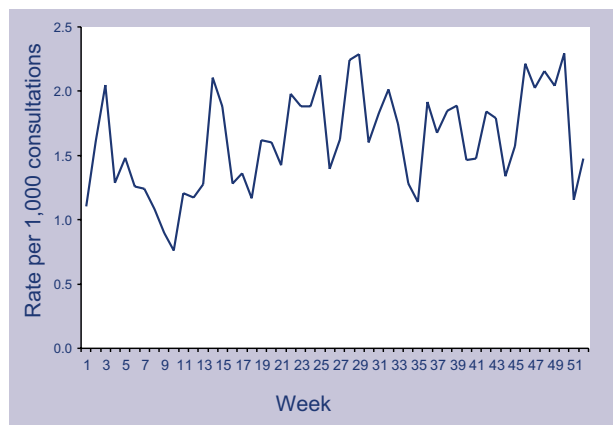


Figure 3. Average weekly consultations for chickenpox to sentinel practices (ASPREN), 1999 to 2001 combined, by week of consultation



There has been an average of 3–4 deaths each year since 1980.⁷ Fifteen of the 20 deaths due to varicella between 1998 and 2000 were in people aged 60 years or more.⁵ In the 2 years (July 1998 to July 2000), there were 3,725 hospitalisations, 60 per cent (2,241) of which had a principal diagnosis of varicella. Varicella infection required on average 7,823 hospital bed days per year with a median length of stay of 2 days. The highest rates of hospitalisation were in the 0–4 year age group and the length of stay was longest in the elderly. Of all varicella hospitalisations in this period, 3 per cent were cases of encephalitis and 8 per cent were cases of varicella pneumonia.⁵

A three-year survey, conducted by the Australian Paediatric Surveillance Unit (APSU) between 1995 and 1997, of more than 900 Australian paediatricians, detected 7 cases of congenital varicella, a rate of one per 107,000 pregnancies per year. Of these, 5 infants had congenital defects and 2 died.⁸ The APSU survey also detected 44 infants with neonatal varicella, a rate of one case per 17,000 pregnancies per year. Of these, only two had severe disease and there were no deaths.⁸

Herpes zoster infection (shingles)

After infection with varicella virus in childhood, the virus remains latent in one or more dorsal root ganglia. Latency is maintained by specific cell-mediated immunity, which is boosted periodically by exposure to people with acute varicella. The varicella virus is reactivated in the elderly as cellular immunity decreases, and the virus spreads via the sensory nerves into the dermis to produce the characteristic vesicular lesions of herpes zoster (or shingles).⁹ Individuals usually have single episodes of herpes zoster but an important sequela is post-herpetic neuralgia (PHN) which occurs in approximately 30 per cent of cases in the elderly.⁹ PHN is defined as localised pain persisting for at least 3 months after the acute inflammatory phase of zoster in the skin. Symptoms may include severe pain or sensations of burning or itching and are refractory to conventional analgesics. Symptoms may continue for years and the frequency and intractability of PHN increases with age.¹⁰

Herpes zoster is estimated to affect 20 per cent of the population, particularly the elderly. In the USA there are estimated to be 500,000 cases of herpes zoster infections per year, resulting in 1.5 million visits to physicians.¹ Data on shingles in Australia are limited. Data for 1999/00 indicate that there were 1,918 admissions to Australian hospitals for herpes zoster (ICD-AM-10 code B02). These were composed of 776 zoster infections without complications and 1,142 infections with complications. The most common complication was nervous system involvement other than encephalitis and meningitis (ICD-10AM code B02.3, n=646), which included polyneuropathy, trigeminal neuralgia and geniculate ganglionitis.¹¹ Recent evidence suggests that adults with contacts with children and therefore with chickenpox have a lowered risk of developing zoster infections. This protective effect is greatest in those adults with many social contacts with children outside the home including contacts with sick children.¹²

Varicella vaccines

A live attenuated varicella vaccine was developed in Japan in the 1970s and introduced into the infant immunisation schedule in the USA in 1995. Currently, in the USA, all children aged between 12 months and 13 years are given a single dose of the vaccine, while, if seronegative, those aged more than 13 years should receive two doses of vaccine, separated by 4 weeks. Side effects and adverse events from the vaccine have been monitored and the vaccine appears in general to be well tolerated.¹³ The vaccine virus strain can cause herpes zoster but does so at a significantly lower rate than the wild-type virus.¹⁴

Since the USA does not include varicella as a notifiable disease, the measurement of vaccine effectiveness has depended on case control studies and surveillance in sentinel sites. A case control study performed between 1997 and 2000, measured vaccine effectiveness at 85 per cent (95% CI 78–90%) for PCR-confirmed varicella and 97 per cent (95% CI 93–99%) against severe disease.¹⁵ Recent reports from sentinel surveillance sites established to measure varicella vaccination effectiveness,¹⁶ have shown declines in varicella cases of 71–84 per cent between 1995 to 2000 across all age groups, with the largest declines in children aged 1–4 years. Vaccine coverage by 2000 in these sentinel areas had reached between 74 and 84 per cent of the 19–35 month age group. Varicella vaccination of children with leukaemia¹⁷ and recipients of haemopoietic cell transplants,¹⁸ has been shown to protect against herpes zoster. Despite concerns of a rise in zoster, active surveillance for herpes zoster in the USA sentinel sites¹⁶ has not shown any change in herpes zoster incidence to date (JF Seward, personal communication).

The cost-effectiveness of varicella vaccination

The cost-effectiveness of introducing varicella vaccination in Australia using a variety of strategies has been examined.¹⁹ The study suggested that introduction of a vaccination program among infants would be the most cost-effective, but the possibility of a relative increase of disease among older children and adults means a 'catch up' program of vaccinating seronegative adolescents would have to be considered. There are considerable uncertainties about the duration of protective immunity induced by vaccination and models have assumed an undiminished effectiveness over 30

years, which may not be accurate. Varicella vaccination of infants in Australia has been estimated to avert 4.4 million cases, 3,500 hospitalisations and 30 fatalities over a 30-year period.¹⁹

Varicella vaccination and herpes zoster

More recent modelling of the impact of varicella vaccination has examined the effect on the incidence of herpes zoster (shingles). If reactivation of latent varicella infections is prevented by varicella immunity maintained by regular exposure to varicella, the reduction in circulating varicella virus might reduce immunity in the elderly and thereby increase the incidence of zoster. As noted above, zoster is a more serious illness than chickenpox with higher rates of hospitalisation and sequelae. Brisson and colleagues assessed the cost-effectiveness of introducing varicella vaccination in Canada²⁰ and concluded that if vaccination resulted in increases in zoster incidence, then vaccination became cost ineffective in the medium term. Subsequent modifications of their modelling led the authors to predict an epidemic of zoster affecting more than 50 per cent of those aged 10–44 years after the introduction of mass vaccination of children against varicella.²¹ This epidemic would consist of an estimated 21 million cases of varicella and result in 5,000 deaths. However, the incidence of herpes zoster would decrease as a larger proportion of the population becomes vaccinated and 30 to 50 years after the introduction of varicella vaccination, would fall below pre-vaccination levels.²¹

In the cost-effectiveness study of varicella vaccination in Australia, the authors did not include the potential increase in the incidence of zoster in their calculations.¹⁹ Neither this study nor the Canadian study²⁰ included the ultimate cost savings, from the eventual reduction in the incidence of herpes zoster to very low levels, by varicella vaccination. Despite the concerns about varicella vaccination and herpes zoster, the ATAGI has stood by their recommendation to give varicella vaccination to all 18-month-old children and to children aged 10–13 years without a history of varicella infection in Australia. The proposal will be considered by National Health and Medical Research Council in October 2002.

Surveillance of varicella disease in a new vaccine era

Given the expected changes in the epidemiology and control of varicella and zoster, what kinds of surveillance are needed in Australia? The large numbers of cases of chickenpox and the small proportion of cases who seek medical attention convinces most epidemiologists that making varicella a notifiable disease in Australia would be unworkable. However, to assess the impact of varicella vaccination, active sentinel surveillance such as that used in the USA, would be helpful.

In the USA, three counties have introduced active verification of every case using a standard case definition and the collection of vaccination history.¹⁶ Active surveillance is expensive and labour-intensive, but is important in measuring vaccine effectiveness and changes in varicella epidemiology, particularly to detect increased prevalence of infections in pregnant women and in adolescents and adults where morbidity is more severe. The incidence of zoster infections in adults would need to be monitored to determine whether there is an increase in zoster, a change in severity or in the age-specific attack rate. This information would be vital to determine whether the introduction of varicella vaccination or re-vaccination of adults would be required to boost varicella immunity in an era of declining natural infection.

Alternately, surveillance might be based on hospitalisation data, which would measure the vaccine's impact on severe disease. Australian hospitalisation data are, however, only available with a 12 to 18 months delay which limits the timeliness of this surveillance. Surveillance through the ASPREN sentinel general practice scheme would give some measure of the impact on severe disease in a timely manner. The representativeness of the populations under surveillance is, however, of concern and the scheme is not able to collect more than a minimum of data on each case. Specialised laboratory surveillance to detect whether post-vaccination cases of varicella or herpes zoster are due to wild type or vaccine strains of varicella would be valuable. This would require sophisticated genetic testing of clinical isolates, isolated from a representative population.

The impact of varicella vaccination on the incidence of herpes zoster will be directly addressed by an ongoing clinical trial of varicella vaccination of adults previously infected with

varicella.²² The study results, due in late 2004, will show whether or not varicella vaccination will prevent the development of herpes zoster in recipients. If varicella vaccination of the elderly boosts immunity to varicella and reduces the incidence of herpes zoster, vaccination of older age groups could be introduced. Control of both chickenpox and herpes zoster by a single vaccine would be an excellent public health outcome and highly cost-effective.

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Using the Australian Childhood Immunisation Register to track the transition from whole-cell to acellular pertussis vaccines

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Abstract

From 1997 to 1999, Australia changed from a whole-cell based pertussis vaccination program to an acellular one. This paper tracks the transition from whole-cell to acellular pertussis vaccines by calculating the number of whole cell (DTPw) and acellular (DTPa) pertussis vaccines recorded on the Australian Childhood Immunisation Register (ACIR) each month from January 1996 to August 2000. The number of combined diphtheria-tetanus (CDT) vaccines, recommended where DTP is contraindicated and for the fifth dose prior to 1994, was also calculated. The use of DTPa increased following its licensing in 1997, with a corresponding decrease in the use of DTPw. The increase was initially greatest in its use as a fourth and fifth dose, for which it was funded at a national level in 1997. Subsequently, a steep increase in its use for the first three doses followed in 1999, coinciding with it becoming free of charge for infants nationally. The use of CDT has decreased markedly since January 1996 and, since March 2000, fewer than 100 CDT vaccines per month were recorded on the ACIR, suggesting that this vaccine is not being inappropriately used. *Commun Dis Intell* 2002;26:581–583.

Keywords: acellular, pertussis, vaccines

Introduction

The Australian Childhood Immunisation Register (ACIR) is an important component of the *Immunise Australia Program*. It is administered and operated by the Health Insurance Commission and commenced operation on 1 January 1996.¹ The ACIR is best known for enabling the estimation of vaccination coverage and for allowing immunisation providers to check on the immunisation status of an individual child regardless of where that child was immunised. This paper describes another use of ACIR data — tracking changes in vaccine use — in this case the transition from whole-cell to acellular pertussis vaccines.

In terms of morbidity and mortality, pertussis is the most important vaccine preventable disease in Australia.² Complete vaccination of all children is the most important preventive measure for the control of pertussis.³ In the past, non-compliance with pertussis vaccination was often thought to be due to concerns about the side effects of

whole-cell vaccines. As a result, acellular vaccines have been developed. These new acellular vaccines have fewer side effects⁴ and appear to be efficacious,⁵ although they are more costly than the whole-cell vaccines.^{6,7} A three-component acellular pertussis vaccine combined with diphtheria and tetanus toxoids (DTPa), was licensed for use in Australia in 1997. The Commonwealth Government initially funded acellular vaccines in the national immunisation program for the fourth (18 month) and fifth (preschool) boosters, but since 1999 has also funded acellular vaccines to replace whole-cell (DTPw) vaccines in the primary vaccination course (at 2, 4 and 6 months of age). In South Australia and the Northern Territory, DTPa was funded for all doses in 1997. The fifth dose of DTP has only been included in the schedule since late 1994, when it replaced combined diphtheria-tetanus vaccine (CDT).⁸ Since 1994, CDT has only been recommended for the two absolute contraindications to DTP — unexplained encephalopathy within 7 days and anaphylaxis.⁸

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Methods

The number of CDT, DTPw and DTPa vaccines recorded on the ACIR each month from January 1996 to August 2000 was calculated using SAS.⁹ Prior to the licensing of DTPa, vaccination providers were not required to state the type of vaccine when sending vaccination encounter information to the ACIR. In the initial period after the licensing of DTPa, if the vaccine type (DTPa or DTPw) was not specified, it was assumed that DTPw had been given.

Results

Since early 1997 the use of DTPa increased, with a corresponding decrease in the use of DTPw (Figures 1–3). By August 2000, only 3 per cent of DTP vaccines were whole-cell vaccines. There was a steep increase in the use of DTPa in the primary course during 1999, and by April 1999 the number of DTPa vaccines given for the first three doses exceeded the number of DTPw vaccines (Figure 1). The number of acellular vaccines given as a fourth or fifth dose exceeded the number of whole-cell vaccines given for these doses by March 1998 (Figure 2), 13 months earlier than for the primary course. The number of DTP vaccines given as a fourth or fifth dose peaked in January each year.

Figure 1. Number of doses of DTPw and DTPa (doses 1–3) administered each month, January 1996 to August 2000

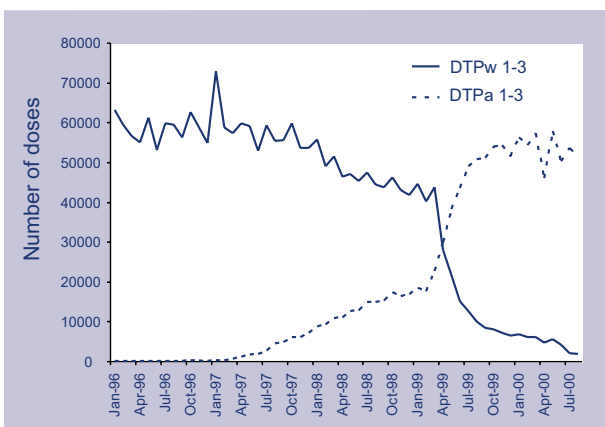
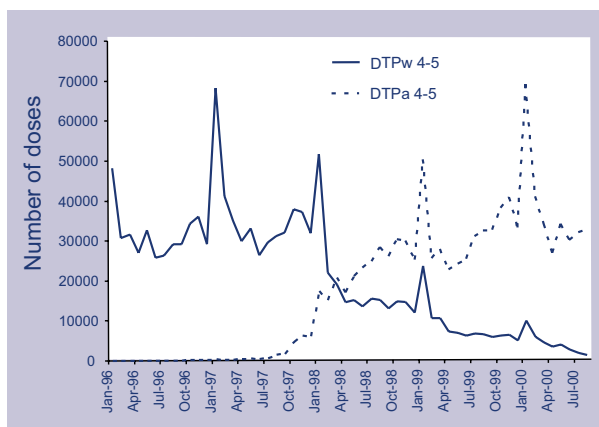


Figure 2. Number of doses of DTPw and DTPa (doses 4–5) administered each month, January 1996 to August 2000



The number of doses of CDT vaccines administered is negligible compared with the number of DTP vaccines (Figure 3). The use of CDT vaccines has decreased markedly since January 1996 (Figure 4). Of all the CDT vaccines administered in the time period examined, 30 per cent were given as a preschool booster dose. The reduction in the number of CDT vaccines given each year in January, particularly CDT 5, indicates that, although CDT use as the preschool booster continued well beyond 1994, its use decreased each year (Figure 4). By January 2000, only 173 doses of CDT 5 were recorded on the ACIR and fewer than 100 CDT vaccines per month were recorded since March 2000.

Figure 3. Number of doses of DTPw, DTPa and CDT (doses 1–5) administered by month, January 1996 to August 2000

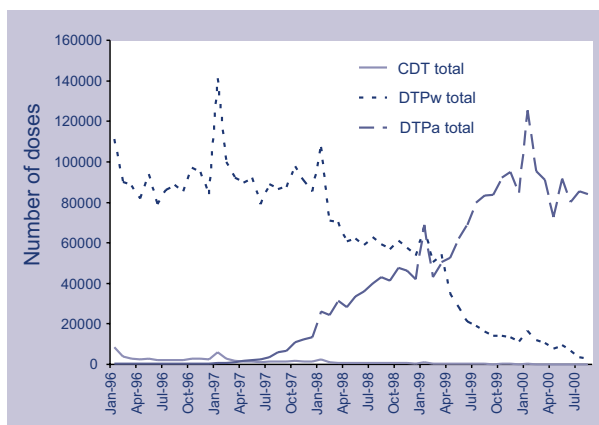
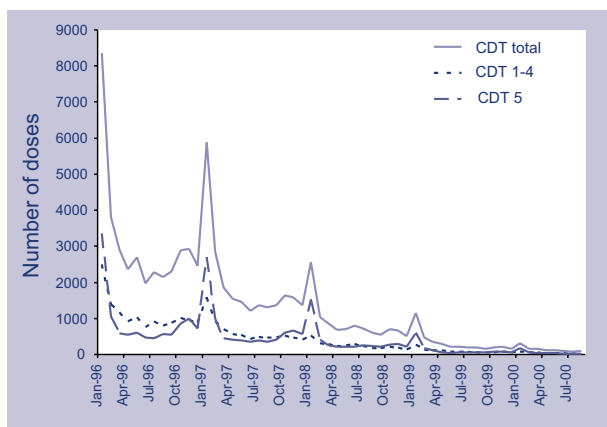


Figure 4. Number of doses of CDT (doses 1–5) administered by month, January 1996 to August 2000



Discussion

The use of DTPa exceeded the use of DTPw soon after it became available free of charge (in 1997 for DTPa 4–5, and in 1999 for DTPa 1–3) in all states and territories. The peak in the number of DTP vaccines given in January each year coincides with the timing of the preschool fifth dose. The use of DTPa in 1997, and possibly 1998, may have been underestimated if providers administering DTPa did not specify which vaccine they had used. This may explain why there does not appear to be any increase in the use of DTPa in January 1998, in spite of it being available free of charge for the preschool dose.

The very low numbers of CDT vaccines recorded on the ACIR suggest that this vaccine is not being inappropriately used. The decrease in CDT use since the introduction of acellular vaccines could be at least partly due to parents and providers who were concerned about side effects of whole cell vaccines being more willing to have their child vaccinated with an acellular vaccine.

It is not possible from these data to determine whether or not the introduction of DTPa improved vaccination coverage. Coverage figures for DTP 1–3 from the ACIR do suggest that coverage improved from March 1997 (the first birth cohort included on the ACIR), when 77 per cent of children aged 12 months were recorded as having received three doses,¹ to 90 per cent in September 2000.¹⁰ However, much of this increase is believed to be due to increased notification to the ACIR.² Any real increase in coverage could also be due to any one of the immunisation incentives schemes introduced during 1998 for both parents and vaccine providers.¹¹

From 1997 to 2000, pertussis vaccination in Australia changed from using entirely whole-cell vaccines to almost entirely acellular vaccines. Most of this transition occurred in 1998 and 1999. Since the ACIR commenced operation in January 1996, it has been used for purposes in addition to those for which it was intended. This paper has demonstrated yet another use for the ACIR data, which is important for evaluation of pertussis vaccine effectiveness, as any impact on pertussis cases from acellular vaccines could not be expected until the beginning of 2000.¹² Similar evaluations using ACIR data are also relevant to tracking the introduction by age and geographic area of other recently introduced vaccines such as conjugate pneumococcal vaccine.

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Infection control and public health aspects of a case of pertussis infection in a maternity health care worker

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Introduction

The potential for nosocomial outbreaks of pertussis is well recognised. Waning adult immunity to pertussis, failure to recognise the symptoms of adult pertussis infection and delayed introduction of control measures are important contributing factors.¹ This report describes the response to a case of pertussis infection diagnosed in a health care worker (HCW) in a busy antenatal/postnatal unit in a large metropolitan hospital and the results of interventions.

Background

In Australia between 1993 and 2000, there were 10 deaths from pertussis infection in infants under one year of age.² The serious sequelae of pertussis infection include pneumonia, hypoxic encephalopathy, seizures and death, with mortality in children under 6 months of age reported at 0.5 per cent.³ These serious outcomes often occur among children who are too young to be protected by vaccination.⁴ The age of children involved in the antenatal/postnatal setting places them at increased risk of the serious consequences of pertussis infection. Therefore, it is important to prevent exposure of young infants to pertussis, to identify potential exposures promptly and to carry out public health interventions when they occur. Key strategies include surveillance, awareness of the symptoms of pertussis in older children and adults (particularly among HCW and new parents), timely vaccination of infants and use of chemoprophylaxis when indicated. In very young infants the use of the standard agent for

chemoprophylaxis, erythromycin, is further complicated by an associated increased risk of Infantile Hypertrophic Pyloric Stenosis (IHPS).^{5,6,7}

On 7 June 2002, the Public Health Unit and the Director of Microbiology were separately notified of a positive serum pertussis IgA result in a HCW from an antenatal/postnatal unit of a large tertiary hospital. An incident control team consisting of microbiology, infection control, infectious diseases, maternity and public health unit staff, was formed to identify strategies to prevent further pertussis cases amongst staff or patients. The HCW provided a history of onset of illness on 17 May 2002 with non-productive paroxysmal cough since 22 May 2002. Symptoms were not relieved by regular nebulised salbutamol. The 4-year-old fully vaccinated child of the HCW was admitted to the children's ward of the same hospital on 5 June 2002 with a productive cough. Pertussis IgA serology collected from the child on 6 June 2002 was negative. However, the HCW had stayed overnight with the child in a shared hospital room with other paediatric patients.

The HCW had provided educational sessions in antenatal classes (ANC) and worked in the maternity ward during the infectious period of her illness (17 May 2002 to 6 June 2002). Decisions about contact definition for chemoprophylaxis were based on an assessment of the extent of exposure to the respiratory secretions of the case and the subsequent risk to the individual.⁸ The incident control team classified risk groups as:

1. Neonates potentially exposed to the respiratory secretions of the HCW during the infectious period.

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2. Mothers, partners and family members rooming with mothers on the maternity ward with exposure to the respiratory secretions of the HCW during the infectious period.
3. Pregnant women and partners who attended educational sessions at the ANC during the infectious period. In these people the onset of pertussis may have coincided with the delivery of their child or the immediate neonatal period.
4. Other HCWs with exposure to the respiratory secretions of the case (shared shifts, prolonged ward contact).
5. Paediatric patients (and family members rooming with the patients) sharing ward accommodation with the hospitalised child and HCW.

Intervention

The assistance of the patients' medical practitioners was sought in communicating the risk of pertussis exposure and the required intervention. Erythromycin chemoprophylaxis and information on pertussis infection was offered to all in categories one to five with the exception of one group of ANC attendees whose exposure was outside the incubation period of pertussis (more than 20 days). The latter group was provided with written information and advised to seek medical attention immediately should symptoms develop. Chemoprophylaxis was provided to parents, families and staff via the maternity ward. Parents of neonates were informed of the possible risk of IHPS in their infants and cautioned to seek medical advice should symptoms occur. All maternity staff were instructed to report the development of any upper respiratory tract symptoms during the next month and symptomatic staff were reassigned duties or excluded from patient contact until the results of investigations were finalised.

Results

Eight family groups had direct exposure to the HCW in the maternity ward. Seven families (including six neonates) were provided erythromycin by the hospital and one family received erythromycin from their private practitioner. Eighteen pregnant women and their partners were exposed to the HCW during the infectious period and were offered chemopro-

phylaxis. Six of these obtained it from the ward, 11 from their private practitioner and one couple declined prophylaxis. Ten pregnant women and their partners attended ANC during the infectious period but were only provided with written information because the incubation period had been exceeded. Twenty-three staff members were offered chemoprophylaxis, of which 14 took erythromycin, 2 took cotrimoxazole and 7 took roxithromycin because of a known prior adverse reaction to erythromycin. Six staff developed symptoms of respiratory tract infection. All six of these were negative on IgA serology and *Bordetella pertussis* PCR (n=4) and culture (n=1) of nasopharyngeal aspirate. One shared hospital room contact of the child and HCW received erythromycin.

No cases of pertussis infection have been reported in any of the people provided with chemoprophylaxis. No further cases of pertussis infection were identified among staff members. No health problems have been reported in the children who received erythromycin.

Discussion

This case of pertussis infection in a HCW demonstrates the importance of pertussis surveillance within high-risk health care settings such as maternity and paediatric units. The standard approach to the prevention of nosocomial transmission of pertussis includes early diagnosis, treatment and isolation (droplet precautions) of patients with clinical infection, investigation and treatment of all symptomatic staff with exclusion from contact with susceptible patients until they have received 5 days of antibiotic treatment, and post-exposure prophylaxis for all asymptomatic exposed employees.⁹ In this situation, the potential for cases to occur in exposed neonates, their parents, near term pregnant females and their partners, warranted the extension of chemoprophylaxis to this group. Four months later there has been no evidence of nosocomial transmission or complications associated with the use of erythromycin.

The introduction of an adult booster dose of pertussis vaccine has potential to prevent or reduce the impact of nosocomial pertussis infection in high risk health care settings.^{10,11} The extent to which the introduction of a booster dose of acellular pertussis vaccine for HCW in

these settings will prevent nosocomial outbreaks is unknown. Acellular pertussis booster vaccines may prevent cases arising in health care workers, but in the absence of evidence of the protective efficacy and duration of protection from adult acellular pertussis boosters, chemoprophylaxis of staff with erythromycin or alternatives such as azithromycin will remain a principal component of control measures.^{12,13} Above all, this incident confirms the requirement for education of health care staff on the resurgence of pertussis in the community and the recognition of pertussis in adults.

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An unusual reaction to rabies vaccine

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This report describes a previously undescribed reaction to human diploid cell rabies vaccine (HDCV). The vaccine is available for prophylaxis against Australian bat lyssavirus (ABL) infection both prior to and after possible exposure. Australian bat lyssavirus is the most recent lyssavirus to be discovered and has been linked to at least one death in Queensland. HDCV appears to be effective against the virus.¹

Case history

A 27-year-old man was scratched on the right shoulder on 22 March 1999 by an unidentified bat while he was at work on a construction site. He was assessed at his local hospital and, following consultation with the Public Health service, was given rabies immunoglobulin and one ampoule of HDCV. He was discharged and given instructions to return for further doses on days 3, 7, 14 and 28 following the first injection.

The vaccine administered to this patient was rabies strain Pitman-Moore/W 1381503-3M cultured on human diploid cells and inactivated by β -propiolactone. Each injection contains 2.5 IU of the virus. It is indicated for prophylaxis against ABL both prior to and subsequent to potential exposure and is administered intramuscularly.

He presented to the Mt Isa Base Hospital for the day three injection. He reported that following the first injection, he had experienced transient neurological symptoms for 30–60 minutes. These included blurred vision and occipital numbness and resolved spontaneously. He was given the day three vaccine, observed for 90 minutes and discharged. Visual acuity following injection was recorded on this occasion and was 6/36 in both eyes.

He presented for the third injection on 31 March 1999 and reported the same symptoms following the second injection as those that occurred after the first. However, on this occasion, the symptoms had lasted for nearly 2 days.

Approximately 30 minutes after the third injection, he developed blurred vision, light-headedness and occipital numbness, as well as tingling over the left parieto-temporal region.

Examination revealed a thin, fully orientated young man with stable haemodynamic parameters and a GCS of 15. Visual acuity was 6/60 in both eyes, correctable to 6/36 with pinhole. He had a left horizontal nystagmus and a positive Romberg's sign. Gait, coordination and the remaining neurological and general examination were normal, as well as fundoscopy.

At this point, advice was sought from the regional Public Health Unit as well as from a clinical microbiologist. While the literature at that time had reported no permanent neurological deficits linked to the use of HDCV, opinions were divided as to whether treatment should be continued. It was felt that the patient was at low risk of Australian bat lyssavirus exposure.

The available information was discussed at some length with the patient, who elected to continue the course of injections after considering all the information presented to him. The same symptoms developed after each subsequent injection, lasting 3 days after the final injection. When contacted by phone 6 weeks later, the patient reported no residual symptoms and was discharged from further follow-up.

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Discussion

The Australian bat lyssavirus is known to infect at least five bat species in Australia, as well as humans. The bat species include all four species of flying fox (megachiropterans) and one species of insectivorous bat (microchiropterans).¹ The current public health guidelines make the assumption that all bat species can potentially carry the virus.¹

HDCV was introduced over 20 years ago, following increasing concerns over the side effects of earlier vaccines. Vaccines prepared from neural tissue were associated with Guillain-Barré syndrome with an incidence of 1:1,600 to 1:32,000.² HDCV is associated with neurological complications in less than 1:150,000 cases. Four cases of neuroparalytic reactions have been reported worldwide, of which three exhibited features of Guillain-Barré.³ Neurological complications to this vaccine are therefore very rare and no permanent effects have been reported to date.⁴ It has been used in Australia since the 1996 death of a female patient in Queensland who developed encephalitis after a bat bite.¹

The neurological symptoms exhibited by the 27-year-old male patient were unusual in that the neurology was not focal and its onset was rapid following injection. Whilst the signs and symptoms are suggestive of an ocular Guillain-Barré syndrome, the rapid onset and resolution of these suggests otherwise. Given the lack of urticaria, angio-oedema and pruritus, an immune-complex mediated cause was considered unlikely. The pathophysiology of this presentation, therefore, remains unclear. The patient's antibody status prior to the first injection was unknown.

The issue of whether to continue the course of injections presented an interesting ethical dilemma, given that exposure to ABL was unlikely. In this instance, informed consent was obtained from the patient, after he considered all the information available at that time. He was aware that the possible incubation period of the virus remains unknown and based his decision on his own assessment of the risks and benefits of the two options presented to him.

Conclusion

We believe the reaction to HDCV as occurred in this patient has not been reported before. We obtained accurate and up to date information from several sources and this allowed the patient to decide the next course of action. We note the vital importance of adequate information when managing unusual cases, as well as the importance of patient autonomy in the decision-making process.

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A measles outbreak in the Whitsundays, Queensland: the shape of things to come?

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Abstract

This report describes a small outbreak of measles that occurred in the Whitsunday region, north Queensland, in July to August 2002. With one exception, all the cases were deliberately unvaccinated because their parents were conscientious objectors to vaccination. It is suggested that this pattern of measles outbreaks, with most cases being not preventable because of conscientious objection, will become increasingly recognised in the future. *Commun Dis Intell* 2002;26:589–591.

Keywords: measles, disease outbreak, surveillance, vaccination

Introduction

Epidemiological and virological studies provide compelling evidence that measles is no longer endemic in Australia, and that indigenous transmission ceased several years ago.^{1,2} As a consequence recent outbreaks of measles in Australia have several common characteristics: they are usually initiated by an imported case, they mainly involve young adults, and they can be of moderate size.^{3,4}

A recent outbreak of measles in north Queensland, although initiated by an imported case, had otherwise different features which may become more characteristic in measles outbreaks in the future.

Methods

Measles cases were defined according to national guidelines.⁵ Responses to cases, especially in health-care and child-care settings, were as described in the national guidelines.⁵

Measles virus RNA was detected in clinical samples by reverse-transcriptase polymerase chain reaction (PCR),² and genotyping was undertaken by comparison of the nucleotide sequence to reference strains.⁶

Results

The index case

In late July 2002 the Tropical Public Health Unit was notified by a general practitioner at Airlie Beach in the Whitsunday region, (approximately 140 km by road north of Mackay, north Queensland), of a 4.5-year-old child with a very typical measles presentation. The child, a local resident, was unvaccinated, as her parents were conscientious objectors to immunisation. There were no siblings. Measles RNA was detected by PCR on both a throat swab and urine taken on the first day of the rash.

The child went to a gym class, visited the local shopping complex and attended (full-time) the local preschool for 2 days, all whilst infectious. She had contact with approximately 20 children aged 6–14 years at the gym, and approximately 40 other children at the preschool.

The imported case

During the index case's exposure period a family of four was staying in the child's household. The family was from Europe and had apparently spent a week in Thailand before arriving in north Queensland.

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There was a 16-year-old female in the family; she became unwell 3 days after arriving in the Whitsunday region. She undertook several popular tourist activities locally whilst unwell, and went on several local shopping trips as well. She eventually saw a general practitioner 5 days after her onset, and because she felt worse presented to a hospital emergency department a day later. Although fever, conjunctivitis and a 'generalised dense, florid maculopapular rash' were documented in the hospital notes, measles was not considered in the diagnosis. No diagnostic tests were performed.

The family travelled by bus to Cairns 9 days after the onset of illness in the 16-year-old, then on to New Zealand by air. They were not able to be located for interview.

Other outbreak cases

A total of seven locally acquired cases, in two generations of transmission involving four families, were recognised (Figure). The 16-year-old visitor also apparently infected a 19-year-old female working in the local shopping complex, and a 14-year-old male from New South Wales visiting the Whitsundays where he too went shopping. Measles RNA was detected by PCR on samples taken from the 19-year-old, and serum from the 14-year-old was measles IgM positive. Neither teenager had been vaccinated as their respective parents were also conscientious objectors to vaccination.

The 4.5-year-old child infected an unvaccinated 11-month-old child who went to the same supermarket at the same time as the younger child. Although this case was not able to be laboratory confirmed as the mother declined venipuncture, he was classified as an epidemiologically linked case.⁵

The 19-year-old infected her 17-year-old sibling, despite the latter having received the appropriate volume (10 mL) of immunoglobulin intramuscularly on day six following exposure.⁵ This sibling's illness was also confirmed by PCR. The 14-year-old visitor infected his two siblings upon returning to New South Wales; measles RNA was detected in both siblings.

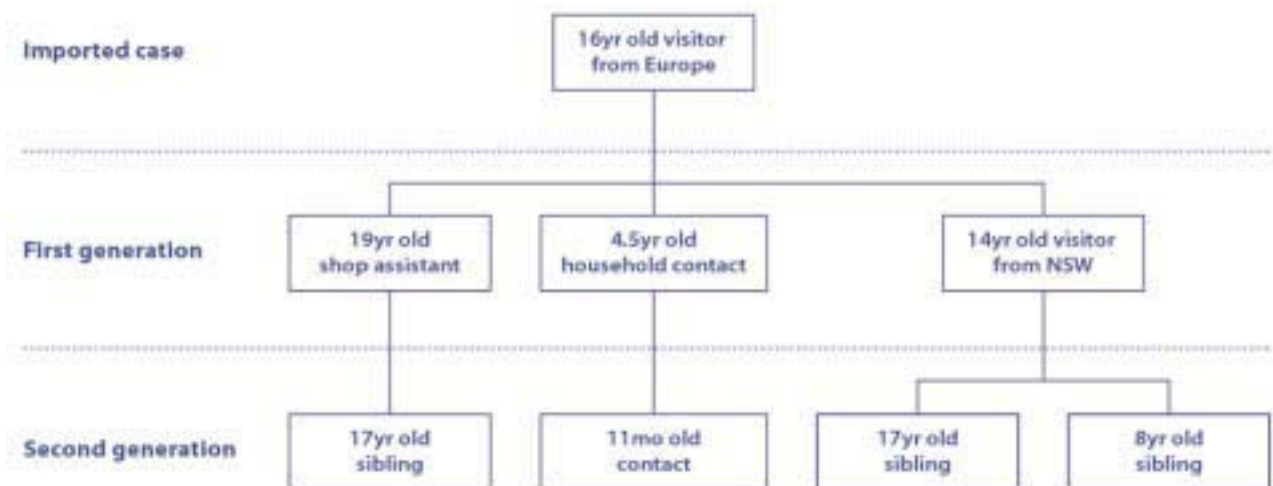
Genotyping

The infecting measles virus was identified as belonging to genotype D5. Sequencing showed that there was 100 per cent sequence homology in the viruses from the five PCR positive cases.

Discussion

Measles is now a rare disease in Australia,¹ indeed, the index was the first confirmed case in north Queensland since October 2000. This rarity has paradoxically created a problem for surveillance, and therefore control, in that many doctors are now not recognising the disease. Not only do younger doctors have little experience with the disease,⁷ but also (as happened with the imported case) some older doctors no longer recognise the clinical manifestations of the disease.

Figure. Schema of measles outbreak in the Whitsundays, Queensland, 2002



However, the prompt recognition of the index case enabled the recognition of the putative imported case. Indeed, had the 4.5-year-old child not been reported, the outbreak link to an imported case would probably never have been recognised. Genotyping identified the virus as genotype D5, which has recently been circulating in Thailand.⁸ This indicates that the visitor from Europe probably acquired measles during the week in Thailand en route to Australia.

This outbreak was small with only seven locally acquired cases despite the considerable potential for further transmission. This indicates that not only that the level of immunity in the region is high, but also that measles vaccine is very effective in preventing the disease. It also suggests that the prompt implementation of control measures⁵ can prevent further cases.

Recent outbreaks elsewhere in Australia have mainly involved young adults many of whom had somehow missed previously receiving two doses of measles vaccine.^{2,3} These cases are defined as being 'preventable'.⁵ Although all the locally-acquired cases in this outbreak were unvaccinated, with the exception of the 11-month-old infant who was too young to be vaccinated, the remaining 6 cases (from three separate families) were not preventable because their parents had actively rejected vaccination.

As vaccinated and therefore immune cohorts of children grow older, there will be fewer and fewer young adults who are susceptible to measles. In other words, the current pattern of measles outbreaks that mainly include preventable cases in young adults may eventually be superseded by a pattern of outbreaks that mainly include 'not preventable' cases in persons of all ages, whose parents were conscientious objectors to vaccination.

We suggest that this pattern, of outbreaks involving mainly deliberately unvaccinated persons of all ages, will become the predominant pattern in the near future. Indeed, the previous outbreak in north Queensland involved five unvaccinated siblings from the same family of conscientious objectors.⁷ These outbreaks are likely to be relatively small in size unless the measles virus finds a way into whole communities of deliberately unvaccinated individuals. Because of their small size, the probability of serious complications will be low thereby reinforcing any misconception that measles is a relatively benign illness.

Conscientious objection 'began with the first vaccinations, has not ceased, and probably never will'.⁹ Indeed, the prevalence of conscientious objection to vaccination could increase with time.¹⁰ However, there is no ready 'solution' to this 'problem',⁹ and certainly calls for compulsory vaccination would be inappropriate. Nevertheless, it is appropriate to ensure that children of parents who do not conscientiously object to vaccination are able to be vaccinated in as convenient and efficient a manner as possible so as to achieve the highest possible levels of vaccine coverage.¹¹

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Refining the public health response to primary meningococcal conjunctivitis

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Abstract

Primary meningococcal conjunctivitis (PMC) is accepted as an uncommon condition. This report describes two recent cases of PMC in newborn infants in a hospital nursery. In both cases the organisms identified were non-groupable strains of *N. meningitidis*, considered to be of low pathogenic potential. Both infants received systemic therapy and recovered without sequelae. The *Guidelines for the early clinical and public health management of meningococcal disease in Australia* recommend the notification of PMC to public health authorities and chemoprophylaxis of contacts. However, our 2 cases suggest that the guidelines should allow for an assessment of risk in determining the public health response. This assessment should include the severity of the conjunctivitis and the serogroup of the *N. meningitidis* isolate. *Commun Dis Intell* 2002;26:592–595.

Keywords: meningococcal conjunctivitis; Neisseria meningitidis

Introduction

Primary meningococcal conjunctivitis (PMC) is accepted as an uncommon condition, although the true incidence is unknown as most patients presenting with acute conjunctivitis receive antibiotic treatment empirically and recover without the collection of conjunctival exudate for culture. A review of 1,030 children with acute bacterial conjunctivitis, presenting to a hospital emergency department in Spain, found pure and abundant growths of *N. meningitidis* in conjunctival exudate in only 21 children (2% of cases).¹ A similar incidence of 2 per cent has been reported in a British paediatric accident

and emergency department.² Another series, however, suggests a lower figure, identifying *N. meningitidis* in only one case from 126 children presenting to an outpatient department, with acute conjunctivitis.³ Amongst 63 reported cases of PMC where serogrouping was performed (adult and paediatric cases) Barquet *et al*⁴ report that 34.9 per cent belonged to serogroup A, 44.4 per cent to serogroup B, 14.3 per cent to serogroup C, and 6.4 per cent were not groupable. In Australia however, serogroup A disease is rare, so the ability to generalise the results of this international review to our population must be questioned.

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Primary meningococcal conjunctivitis has been known to precede invasive disease. In a review of reported cases of PMC, Barquet *et al*¹ found that 17.8 per cent of cases developed systemic meningococcal disease. The risk of invasive disease in those treated initially with topical therapy alone was estimated to be 19 times greater than for those receiving systemic antibiotic treatment.¹ Consequently, systemic antibiotic therapy has been recommended for all patients with PMC.^{1,4,5}

In response to reports of PMC associated with invasive disease⁵ and a case report of invasive meningococcal disease in a contact of a child with PMC,⁴ Australian guidelines now require a public health response following the diagnosis of primary meningococcal conjunctivitis.⁶ Two recent cases of PMC in a hospital nursery where the identified organisms were non-groupable strains of *N. meningitidis*, are reported. These cases raise questions about the public health response, and highlight inconsistencies in the response to PMC compared to that required when meningococci are cultured from other non-sterile sites.

The Human Research and Ethics Committee of the South Eastern Sydney Area Health Service (Eastern Section) has approved publication of this paper.

Case 1

Case 1 was a male infant born by emergency caesarean section at 28 weeks gestation, and transferred to the Newborn Care Unit (NBCU) for management of respiratory distress. At 8 weeks of age, whilst still in the NBCU, he developed right eyelid swelling and erythema, with purulent discharge. An eye swab and blood for culture were collected, and the infant was commenced on intravenous cefotaxime (50 mg/kg q8h), flucloxacillin (50 mg/kg q12h), and topical chloramphenicol (q6h), whilst awaiting culture results. He was isolated and nursed in a single room. After 24 hours, his eye was much improved, with no discharge noted. Intravenous antibiotics were ceased after 48 hours, and oral cephalixin was commenced. Four days after the eye swab had been taken, the hospital laboratory advised that a light growth of a non-groupable *N. meningitidis* (sensitive to penicillin) had been isolated. Oral cephalixin was ceased, and intravenous cefotaxime was recommenced and administered for a total of 5 days. The public

health unit, assisted by the Newborn Care Unit's clinical staff and the hospital infection control staff, organised an information session for health care workers who had been involved in the care of the infant in the week prior to onset of his symptoms. The health care workers were treated as 'household-like' contacts, as the staff had been the main carers. The infant's parents and 8 staff members received rifampicin (600 mg twice daily for 2 days); one pregnant staff member received ceftriaxone (250 mg by intramuscular injection). The infant did not develop any sequelae or systemic meningococcal disease. He was discharged home on day 94 of life after resolution of his prematurity related problems.

Case 2

The second case was a full term male infant, born after normal vaginal delivery to a methadone dependent mother. The infant was admitted to the Newborn Care Unit for a brief period at birth for management of neonatal abstinence syndrome. He was transferred to the postnatal ward on day 2 of life. On day 4 he was noted to have a sticky left eye, which was managed with normal saline eye toilet. A swab from the left eye, taken on day 7 for Gram staining and culture, returned on day 10, a light growth of a non-groupable *N. meningitidis*, sensitive to penicillin. This isolate was phenotypically distinct from the isolate from Case 1. The conjunctivitis was considered mild, with redness of palpebral conjunctiva and a small amount of discharge. The infant was afebrile with no other constitutional symptoms or signs. He was commenced on intravenous cefotaxime (50 mg/kg q8h). The public health unit and the hospital infection control staff were notified, and the infant was nursed in isolation. Prophylaxis (rifampicin 600 mg twice daily for 2 days) was given to the infant's parents. As with Case 1, staff from the public health unit, assisted by clinicians and infection control staff, attended the unit to provide information to the health care workers who had been involved in the infant's care since his birth. Staff were advised that chemoprophylaxis was not warranted unless their contact with the infant had been close and prolonged. No nursing staff received prophylaxis. After 48 hours therapy the child's eyes were free of redness and discharge. Intravenous antibiotic therapy was ceased after 8 doses. There were no sequelae and the infant was discharged home on day 41 of life.

Discussion

Onset of PMC during the first week of life has been reported previously^{7,8,9} and it has been speculated that the source of the meningococcus in these cases has been the maternal genital tract.⁷ A recent case report of PMC adds weight to this theory, with strains identified from the conjunctival exudate of the newborn infant, the mother's endocervix and the mother's partner being of the same antigenic composition.¹⁰ Unfortunately, no swabs were able to be collected from the mother of the neonate (Case 2) to enable investigation of this possibility.

In slightly older infants, infection is likely to have been acquired after birth.⁷ Direct inoculation of *N. meningitidis* into the conjunctival sac from manual contact (e.g. rubbing the eyes) or through infectious airborne respiratory secretions have been suspected modes of infection.¹ Secondary seeding of the conjunctiva from the nasopharynx has been suggested by findings of identical strains (same serogroup, serotype and subtype) of meningococci isolated from the conjunctiva in PMC as from the nasopharynx.^{11 cited in 4} Indeed, from a microbiological perspective, the throat and conjunctiva of a newborn may be considered as essentially continuous mucosal surfaces, and the conjunctiva is often colonised by nasopharyngeal organisms when naso-lacrimal duct blockage occurs.

Systemic antibiotic therapy of PMC is recommended because topical therapy does not eliminate pharyngeal carriage or the risk of developing systemic meningococcal disease.¹ Out of 15 cases of invasive disease described by Barquet *et al*,¹ the serogroup of 13 strains was reported. Most were from serogroups A, B or C (A, n=3; B, n=6; C, n=2). Two cases were reported as non-groupable. However, one of the non-groupable cases was reported in 1936, raising the possibility of misidentification. The authors did not find any statistically significant difference between patients who developed systemic disease and those who did not develop systemic disease, in terms of serogroup or local (ocular) complications, but this may have been due to the small numbers in the study.

Strains of meningococci identified from deep isolates, such as blood or cerebrospinal fluid, are almost always encapsulated (with serogroups B and C accounting for most disease in Australia). In contrast, meningococci grown from the nasopharynx are more variably capsulated, and those with little or no capsular material most often represent colonisation or a

carrier state. Due to the lower pathogenicity of non-groupable meningococci and the absence of systemic signs or serious local disease (e.g. orbital cellulitis), Case 2 received a short course of parenteral treatment, with clinical resolution being reached by the conclusion of treatment.

The major rationale for chemoprophylaxis of contacts is that eradication of *N. meningitidis* from the nasopharynx of presumed carriers will prevent transmission to other (susceptible) people. Additionally, it may eliminate colonisation, and thus the risk of subsequent invasion in others also exposed to the carrier, or in those very close contacts exposed to the case after the onset of the illness, but prior to the commencement of antibiotic treatment.⁶ The virulence of the particular meningococci in cases of invasive meningococcal disease has been demonstrated through their ability to invade.

In the case of PMC, however, the Australian guidelines⁶ recommend that a public health response be mounted in the absence of invasive disease, and they do not allow for a differential response in the case of those meningococci whose virulence is well-recognised compared to the less pathogenic non-groupable organisms.

Unnecessary chemoprophylaxis of contacts increases the risk of bacterial resistance developing, and can eliminate non-virulent meningococci and other non-pathogenic bacteria (e.g. *Neisseria lactamica*) which have been shown in infants and young children to have an important role in induction of natural immunity to invasive meningococcal disease.¹² Further, as the public health response itself can create anxiety within the network of contacts and amongst those 'exposed' to the case, unnecessary alarm should be avoided wherever possible. A great deal of concern was evident amongst parents of infants placed in the same neonatal nursery as Case 1, and the event was reported in the media. Experience with Case 1 allowed a more rational approach from the hospital and public health unit when the second case was identified some weeks later. In this hospital nursery setting, the circle of close contacts was relatively small. However, situations exist where the routine public health response might include a greater number of contacts, such as the staff and children of a child-care facility, and the potential for inadvertent harm would be greater.⁶

The intent of the guidelines with respect to chemoprophylaxis following a case of PMC is to

prevent the rare, but nonetheless documented, secondary cases of invasive meningococcal disease.^{4,5} However, Australian guidelines for the management of meningococcal disease do not recommend a public health response when *N. meningitidis* is isolated coincidentally from other superficial sites (e.g. from oropharyngeal, genital or anal swabs).⁶ Further, in the absence of invasive disease, a public health response is not required when meningococci are isolated in the sputum (for example, in the case of pneumonia), despite the fact that transmission of *N. meningitidis* from cases of pneumonia has been reported.^{13,14} Clearly, there is a lack of consistency within the Australian guidelines on the public health response to the identification of *N. meningitidis*.

The existing guidelines recommending that PMC be notifiable to public health authorities should be supported, and the recommendation for chemoprophylaxis of contacts of cases of PMC should stand. However, the guidelines do not allow for an assessment of risk — the same response is required for cases of low pathogenic potential, as for cases of higher risk. We argue that rather than the routine implementation of chemoprophylaxis for contacts of all cases of PMC, the guidelines should allow for an assessment of risk. This assessment should include the severity of the conjunctivitis and the serogroup of the *N. meningitidis* isolate.

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A focal, rapidly-controlled outbreak of dengue fever in two suburbs in Townsville, North Queensland, 2001

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Abstract

In April–May 2001 an outbreak of dengue fever occurred in two suburbs in Townsville, north Queensland. This was the first outbreak in the Townsville region since a very large outbreak in 1992–1993. Notification delays resulted in late detection of the outbreak. Once recognised, control measures were implemented and rapid control was achieved. Dengue serotype 2 was the causative virus and 9 cases of dengue fever were documented. The approach to management of dengue fever outbreaks and vector control strategies have been improved and refined in the years since the 1992–1993 outbreak. These measures, in addition to favourable weather conditions, were likely to have contributed to the successful containment of this outbreak. *Commun Dis Intell* 2002;26:596–600.

Keywords: *Aedes aegypti*, dengue fever, disease outbreak

Introduction

In 1992–1993 an extensive and prolonged outbreak of dengue fever occurred in Townsville. Control measures appeared to have little impact on the progression of the outbreak and the disease spread to the nearby towns of Charters Towers and Hughenden. By the time the outbreak eventually subsided after 16 months, over 900 cases of dengue fever had been notified to Queensland Health.¹ Individual importations of dengue to Townsville (by viraemic travellers) were documented over the following 8 years but no outbreaks were recorded, despite some fairly intense 'risk' periods such as the return of over 2,000 military personnel from dengue-endemic East Timor in February 2000.²

On 15 May 2001 the Tropical Public Health Unit (TPHU) was notified of a case of dengue fever (diagnosed by IgM and IgG seroconversion by enzyme linked immunosorbent assay (EIA)) in a resident of Townsville who had no history of overseas travel. The specimen was referred for confirmatory testing to the Arbovirus Reference Laboratory, Queensland Health Pathology and Scientific Services and initial control measures

commenced. Two days later a second IgM positive case was notified to the TPHU. This case likewise had no travel history and the residential address was in the same suburb and in close proximity to the first case. The vector of dengue, *Aedes aegypti*, is present in north Queensland and is responsible for the spread of the virus from person to person. However, dengue fever is not endemic in north Queensland and transmission only occurs following importation of the virus (via a viraemic human) from a dengue-endemic area. The two reports of locally-acquired cases thus indicated importation of the virus and that subsequent transmission had occurred. This led to an outbreak being declared. This report describes the outbreak and discusses the likely factors that contributed to its rapid control.

Methods

On recognition of a local outbreak of dengue fever, enhanced surveillance for further cases was undertaken. On receipt of a notification of a suspected case, the patient was contacted by TPHU staff to collect information on their movements during their 'exposure' and

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'viraemic' periods. Information on movements during the 'exposure period' (i.e. 3 to 12 days prior to symptom onset) was required to determine where the infection may have been acquired. Details of the 'viraemic period' (one day prior to 12 days after symptom onset) were collected to determine where the person had been whilst infectious and this information was used to inform control measures.

An immediate retrospective investigation was also undertaken at a child day care centre that was situated in close proximity to the houses of the first two notified cases. The aim was to identify and arrange serological testing of those at the centre who had experienced non-specific febrile illness in the previous month. A non-specific febrile illness can be a common presentation of dengue fever in a child. The concern was that if unrecognised cases had occurred in the children at the centre, they could readily have acted as disseminators of virus into the community, as the centre attendees were drawn from many different suburbs of Townsville.

Specific intense control measures focussed on controlling the vector in the vicinity of case houses. The limited flight range of *Aedes aegypti* in the urban environment³ and its preference for breeding sites that include domestic receptacles such as containers, pot plant bases and roof gutters⁴ means premise-to-premise surveys are the appropriate means of control. Surveys were carried out within a 200 metre radius of the residence of the notified case (or at specific premises where cases had spent considerable amounts of time while viraemic), searching for and treating breeding sites if they could not be managed by removal of the source of water. Household members with recent febrile illness identified during these inspections were encouraged to seek medical attention or were followed up to ensure potential cases of dengue fever were not missed.

Broader measures to encourage mosquito control included provision of information to businesses in the outbreak area and media alerts. In addition, cryptic breeding sites such as underground drains and wells were located, inspected and treated.

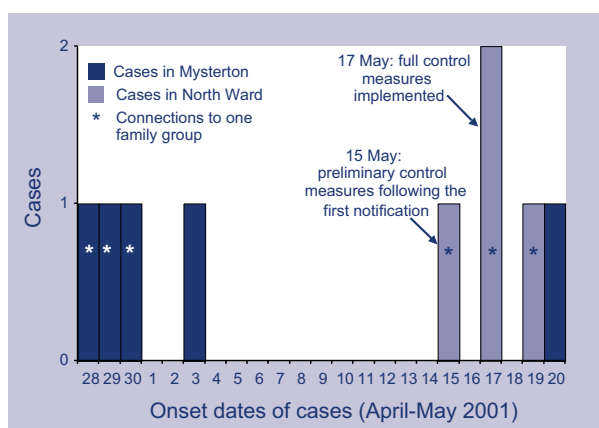
In an attempt to determine the 'index case' for the outbreak, householders living in the area where the outbreak was first recognised were asked about recent overseas travel to dengue-endemic countries. A laboratory search was also undertaken for patients with a suggestive haematological picture for dengue fever (thrombocytopenia and leukopenia) in the month prior to the onset of illness in the first case.

Initial dengue cases were confirmed by nucleic acid testing or demonstration of a dengue titre fourfold higher than other flavivirus titres by haemagglutination inhibition assay. Once the outbreak was confirmed, any IgM positive result by EIA was classified as a confirmed case.⁵

Results

The outbreak consisted of a total of 9 cases of dengue fever, occurring in two very distinct waves of transmission (Figure). A case with onset of disease on 30 April 2001 was the first case clinically diagnosed in the outbreak (2 cases with earlier onset dates were only diagnosed retrospectively). Although a General Practitioner (GP) diagnosed dengue fever in this case and also received a positive laboratory report on 9 May 2001, neither GP nor laboratory notified the TPHU immediately and an 8 day delay occurred before the result was received (on 17 May) from the laboratory. A case, notified by a laboratory on 15 May, with onset date of 3 May, was thus the first notification received. At the time of notification, 11 days had elapsed from the time this case first sought medical attention, and 4 days from collection of a blood specimen. The delays meant recognition of the outbreak and implementation of control measures were substantially delayed.

Figure. Epidemic curve for dengue fever outbreak in Townsville, 2001



One staff member and 3 children at the day care centre reported febrile illness during April and early May. The adult and two of the children were tested but results were negative. Recognising that children at the centre with unrecognised or subclinical illness could act as a source of dissemination of virus into the community, extensive measures were taken to prevent infection of children. This included provision of mosquito repellent devices and personal insect repellent for use at the centre. To ensure any possible cases were rapidly identified, parents were provided with information and asked to take their child to a GP immediately if a febrile illness developed.

The two recognised rounds of transmission occurred in late April to early May and again in mid-May (Figure) and involved two suburbs, Mysterton and North Ward. The probable link between these two suburbs was established. Three members of one family group living in Mysterton were infected in the first round of transmission. Following an afternoon visit to the residence of another family member in North Ward on 29 April (on which day all 3 cases would have been viraemic), mosquitoes at that residence were probably infected. A further 2 family members at the North Ward residence and another family member and a visitor in a neighbouring residence subsequently became ill.

As part of the mosquito control efforts, over 420 premises were inspected with 18 (4%) found to have *Aedes aegypti* breeding sites. Pot plant bases were the most common site of breeding (40% of all sites identified). Many of the residences had backyard wells which were known to be a common breeding site in the previous dengue fever outbreak in Charters Towers. Mosquito larvae or pupae were not present in any wells on inspection but all were treated as a precautionary measure. Internal spraying was conducted in 124 premises which represented 90 per cent of premises in which it was offered.

The outbreak was due to a dengue serotype 2 virus. The index case for the outbreak was not identified. No cases met the criteria for dengue haemorrhagic fever⁶ however, one patient required hospitalisation with significant haemorrhagic phenomena including haematemesis and epistaxis. The two identified cases in children were relatively mild. Both were diagnosed initially with respiratory illnesses. It was only subsequent to a parent being diagnosed with dengue fever that they were retrospectively tested and their illnesses confirmed as dengue fever.

Laboratory and GP notification delays meant a second round of transmission was inevitable. After recognition of the outbreak, transmission was halted completely. No further cases were notified more than one incubation period after control measures were implemented.

Discussion

In contrast to the last outbreak of dengue fever recorded in Townsville during 1992–1993, this outbreak was rapidly controlled. Several factors were likely to have contributed to this outcome. Townsville experienced its coldest May on record in 2001 with a minimum monthly average temperature of 14.4°C and several minimum recorded temperatures under 10°C.⁷ It is well recognised that cooler temperatures can affect the blood feeding activity of *Aedes aegypti*, prolong the extrinsic incubation period in the mosquito, and reduce adult mosquito longevity. All these factors will reduce the likelihood that transmission of infection will occur. In addition, no rainfall was recorded in Townsville in May.⁷ The impact of rainfall on dengue transmission is of lesser importance than for other mosquito-borne diseases as the vector predominantly breeds in and around homes in water-filled containers. The lack of rainfall, however, may have limited some potential breeding sites for the mosquito, such as containers in backyards that could collect rainwater.

Weather patterns may have assisted in control of the outbreak but outbreaks in north Queensland have in the past continued through the winter season^{1,5} and it is unlikely that weather was the single reason for control of the outbreak. Other factors such as overall vector density and viral virulence may also have contributed. Accurate comparisons with the previous outbreak cannot be made. The management of preventive and control aspects of dengue fever has been refined in north Queensland in the last decade. The first 'Dengue Fever Management Plan'⁸ was written after the extensive dengue fever outbreak in Townsville in 1992–1993. Further important control principles, recognised following a prolonged outbreak in Cairns and Port Douglas in 1997–1999,⁵ were incorporated into an updated 'Dengue Fever Management Plan for north Queensland, 2000–2005'.⁹ Implementation of these principles, including well-trained officers responding immediately to the notification of

dengue in a local resident, identifying 'dissemination' premises and searching for and treating cryptic breeding sites, almost certainly had an impact on the progress of the outbreak.

The index case for this outbreak was not identified despite extensive investigations. A possible source might have been a member of the Australian Defence Force as the outbreak coincided with the return of several hundred defence force personnel to Townsville from East Timor. However, no recent returnees were identified in the outbreak area.

The intense transmission of infection among one family (residing in three separate locations) was of interest. It is likely that infection of mosquitoes by the first generation of cases occurred during the afternoon the 3 cases spent at the residence of another family member. There was apparently sufficient opportunity for the infection of several mosquitoes, at least one of which reached the neighbouring residence of another family member. An alternative explanation was that infected mosquitoes were transported by car from one property to another. Transportation of mosquito vectors by passenger cars has been documented previously.³

A second round of transmission could have been prevented in this outbreak if the first diagnosed case had been promptly notified. Assuming an average incubation period of between 5 and 7 days,¹⁰ the cases that occurred as part of the second round of transmission were infected between 8 May and 15 May 2001. A positive dengue result was available on 9 May 2001. If mosquito control measures could have been implemented at that time these cases could potentially have been avoided.

Considerable delays in the notification of individual cases of dengue fever in north Queensland have been documented previously¹¹ and outbreaks associated with lack of timely notification of imported cases have occurred in the past.^{12,13} In areas outside of north Queensland and parts of central Queensland, there are no public health implications of cases of dengue fever. Local transmission cannot occur as *Aedes aegypti* are not present. Laboratories outside these regions often fail to appreciate the importance of notification of dengue cases for the prevention of outbreaks, when the patient resides in a region where *Aedes aegypti* are present.

A history of dengue fever is common among the population of north Queensland as a result of previous outbreaks in the region. Both the 1992–1993 Townsville outbreak and this outbreak were caused by the dengue serotype 2 virus. An epidemic due to another serotype would increase the likelihood of dengue haemorrhagic fever cases. Ongoing vigilance of clinicians and laboratories is required to ensure the risk of dengue fever to the north Queensland population is limited.

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An outbreak of Barmah Forest virus disease in Victoria

Jonathon Passmore,¹ Kerry Ann O'Grady,¹ Rodney Moran,¹ Elwyn Wishart²

Abstract

This report describes the epidemiological and clinical features of an outbreak of 47 cases of laboratory-confirmed Barmah Forest virus disease (BF disease) that occurred in Victoria between January and May 2002. Laboratory-confirmed cases were investigated, and information on travel history and clinical details was collected. Surveillance data from adult mosquito trappings and climatic conditions in the Wellington Shire were also reviewed. The response rate for interviews was 85 per cent (40/47). The most common symptoms reported by cases included arthralgia (95%), lethargy (90%) and maculopapular rash (72.5%). Transmission of BF disease in the Gippsland region was associated with unusually high numbers of *Ochlerotatus camptorhynchus* mosquitoes. This outbreak was of interest due to the fact that cases of BF disease outnumbered cases of Ross River virus disease (RR disease) in Victoria for the first time since data were available. Similar outbreaks of BF disease, in the absence of RR disease, occurred in Western Australia in 1993¹ and New South Wales in 1994/1995.² Although the majority of BF disease cases reported regular outdoor activity during which they could be exposed to mosquito populations, they infrequently take precautions to limit exposure. Further efforts need to be made to educate people of the importance of using repellents and other personal preventative measures. *Commun Dis Intell* 2002;26:600–604.

Keywords: Barmah Forest virus; surveillance; arbovirus; disease outbreak; *Ochlerotatus camptorhynchus*

Introduction

Barmah Forest virus (BF) is an alphavirus that was first isolated from mosquitoes trapped in the Barmah Forest of northern Victoria in 1974,³ but was only shown to be pathogenic to humans since 1988.⁴ BF is the causative agent of Barmah Forest virus disease (BF disease), which is similar to the epidemic polyarthritis caused by Ross River virus (RR).⁵

Since 1988, BF disease has been reported in Western Australia, Queensland, New South Wales, the Northern Territory and Victoria.⁶ In Victoria, outbreaks have been previously reported throughout the Murray Valley and the Gippsland areas.⁷

The Communicable Diseases Section of the Victorian Department of Human Services noticed a greater than expected number of cases of BF disease in February 2002, and this prompted health warnings to be sent out to General Practitioners and media warnings for the Gippsland area, advocating preventative measures to residents and visitors.

This report describes epidemiological and clinical features of the outbreak based on a survey of laboratory-confirmed cases. Adult mosquito surveillance data were also analysed to determine whether there was an association between vector abundance and disease incidence.

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Methods

Ethical clearance was not required for this investigation as it was part of routine surveillance of a notifiable disease.

The following case definition was used for this outbreak.

'Isolation of BF from clinical material; or detection of BF by nucleic acid amplification; or a significant rise in IgG to BF; or detection of BF-specific IgM'

Using a standard questionnaire, we collected clinical details and travel histories from each laboratory-confirmed case that could be contacted. Information was also obtained from treating physicians.

To confirm the diagnosis made by the commercial laboratories, sera from the first 7 patients was retested at the Victorian Infectious Diseases Reference Laboratory. The diagnosis between both laboratories was consistent and the same testing methods were used in both (PanBio ELISA). All other notifications received were not retested and were included as confirmed cases.

Adult mosquito trapping data from six shire councils were obtained from the Victorian Department of Natural Resources and Environment. Adult mosquitoes were collected using carbon dioxide baited light traps and these were set once a week at Mildura (4 sites), Moira (4 sites), Shepparton (4 sites), Swan Hill (4 sites), Wellington (4 sites) and Wodonga Shires (4 sites).

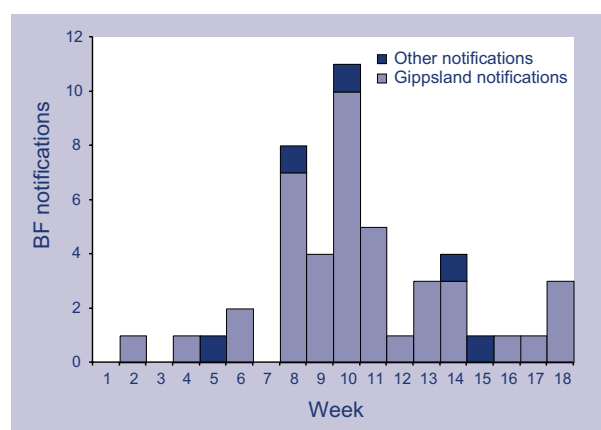
Mosquito trapping and counts have been carried out in four sites around Lake Wellington, Victoria, since 1991. Results are taken as representative of the Gippsland area (Wishart E, Department of Natural Resources and Environment, personal communication, May 2002). Trappings are seasonal and were carried out between 24 October 2001 and 8 April 2002.

Adult mosquitoes were identified to species and counted. Mosquito abundance was expressed as the mean number per trap. Rainfall data from each of the trapping sites and overall climate data for the Gippsland area were obtained from the Australian Bureau of Meteorology.

Results

Forty-seven cases of BF disease were notified to the Department of Human Services between 8 January and 1 May 2002 (Figure 1). Forty of the 47 patients were interviewed, a response rate of over 85 per cent. Cases consisted of 23 males and 24 females. Ages ranged from 17 to 74 years, with 51 per cent of cases aged between 20 and 49 years. One case was hospitalised.

Figure 1. Barmah Forest virus disease notifications, Victoria, January to May 2002, by notification date and link to Gippsland



* Week refers to the first 18 weeks of the year from 1 January 2002

Analysis of cases by patient's place of residence or travel history

Of the 47 cases, 38 lived in the Gippsland area; 34 within the East Gippsland Shire. Four cases resided in other regions of Victoria, but visited Gippsland between 10 January and 14 February 2002. Five cases had no link to Gippsland and were infected in other areas.

Clinical features

The most common symptoms experienced by cases included arthralgia, lethargy and maculopapular rash (Table 1).

Use of mosquito repellent and frequency of outdoor activities

A minority of cases (32.5% n=13) reported that they did use mosquito repellent regularly, with the remainder (67.5%, n=27) reporting no use, or occasional use. All cases that reported some use, said they applied repellent to all exposed areas of their body. Each case was questioned about extent of outdoor activity; 75 per cent participated in regular gardening (> 1/week), 25 per cent played golf at least once a week and 30 per cent went bushwalking (Table 2).

Table 1. Symptoms reported by cases (n=40)

Symptom	Number of patients	%
Arthralgia	38	95.0
Lethargy	36	90.0
Rash	29	72.5
Headaches	25	62.5
Myalgia	21	52.5
Fever	21	52.5
Chills	17	42.5

Ross River virus disease

Over the same time period, there were only 21 notifications of RR disease in Victoria, eight of which were notified from the Gippsland area. There were 326 notifications for the same time period in 2001. Figure 2 shows the comparison of BF notifications and RR notifications over the first 5 months of 2002.

Mosquito counts

The main vector species of interest in the Gippsland area is *Ochlerotatus camptorhynchus*, and counts over the 2001/2002 season compared to the average count, can be seen in Figure 3. For the overall season, the trapping count for *Oc. camptorhynchus* was 1.4 fold greater than the 1991 to 2002 standardised trapping average. For the 2001/2002 season, 88 per cent of all mosquito species trapped were *Oc. camptorhynchus*.

The five other trapping sites in Victoria are inland sites and so the main species of interest is *Culex annulirostris*. Figure 4 shows the below average population of this mosquito species seen in the Mildura Shire during 2001/2002. A similar pattern of low seasonal abundance compared to the average was also seen in other trapping shires in Victoria (Wodonga, Shepparton, Moira, Swan Hill, data not shown).

Table 2. Reported outdoor activity in interviewed cases (n=40)

Outdoor activity at least once/week	Number of patients	%
Gardening	30	75.0
Bushwalking	12	30.0
Golf	10	25.0
Camping	8	20.0
Fishing	7	17.5

Figure 2. Notifications of Barmah Forest virus disease and Ross River virus disease, Victoria, January to May 2002

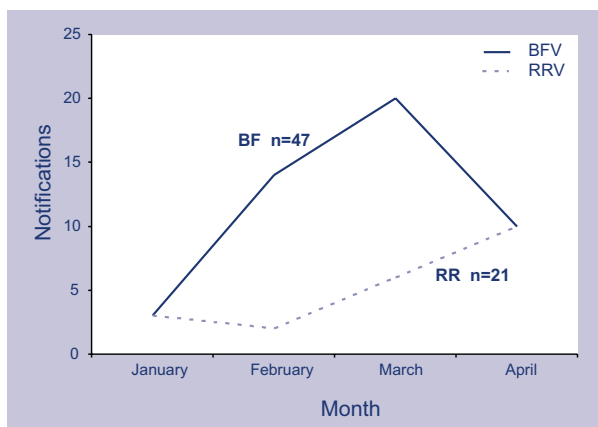
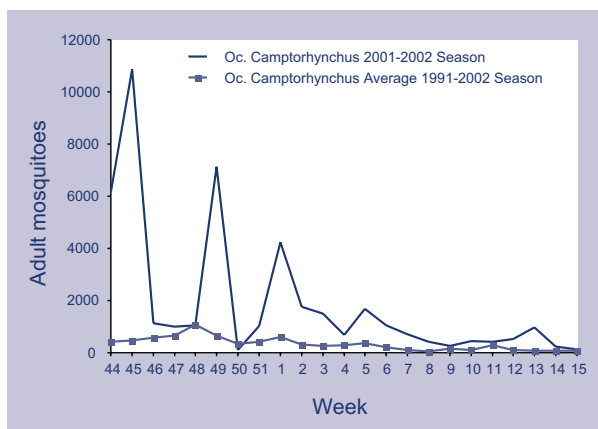


Figure 3. Mean weekly numbers of *Oc. camptorhynchus* per trap during the 2001/2002 season compared with overall mean for 1991/1992 to 2001/2002, Wellington Shire



Climate data

The Bureau of Meteorology data shows that the Gippsland area experienced a wetter than average spring to autumn months. Between October 2001 and March 2002, Gippsland received 1,046 mm of rainfall, with the average for these months being 746.75 mm, an increase of 40 per cent.

Rainfall data were also routinely collected from each mosquito trapping site and these figures were plotted against weekly mosquito counts. Figure 5 shows the relationship between rainfall and changes in mosquito density at the trapping sites in Wellington Shire.

Figure 4. Mean weekly numbers of *Cx. annulirostris* per trap during the 2001/2002 season compared with overall mean for 1992/1993 to 2000/2001, Mildura Shire

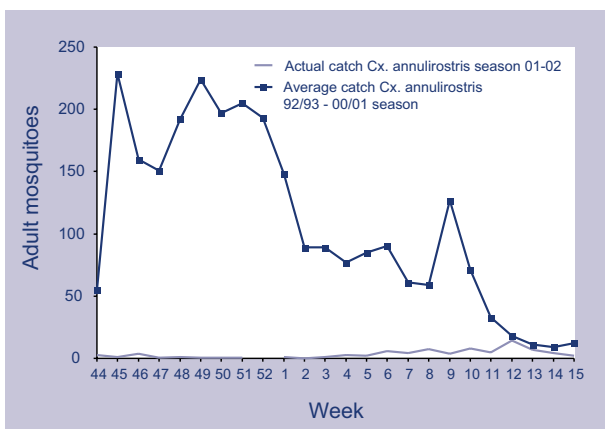
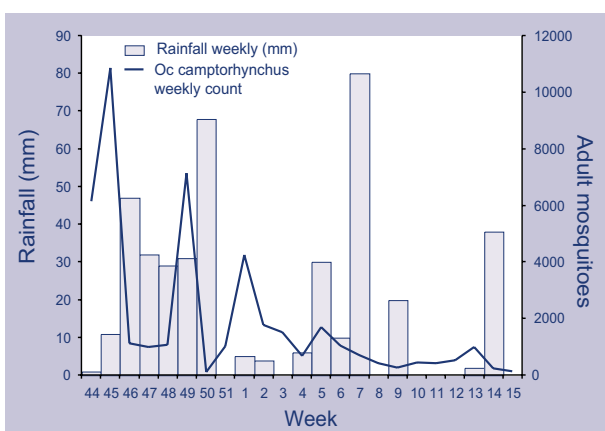


Figure 5. Relationship between mean weekly numbers of *Oc. camptorhynchus* per trap during the 2001/2002 season and weekly rainfall in Wellington Shire



Discussion

Between 1 January and 1 May 2002, 47 cases of BF disease were notified in Victoria. This figure is the highest number of cases in the first 5 months of the year since the RR and BF outbreak in the Loddon Mallee region of Victoria in 1993 (53 BF disease and 1,109 RR disease) (Notifiable Infectious Diseases Surveillance System Database. Communicable Diseases Section. Department of Human Services. Victoria, personal communication).

Comparisons against RR disease are of interest as this is the first time since data have been collected in Victoria, that BF notifications have outnumbered RR notifications. This same phenomenon has also been reported in the south-west of Western Australia in 1993¹ and in New South Wales in 1994-1995² and for the first 5 months of 2002 (Hogan D, New South Wales Health, personal communication, May 2002. and Communicable Disease Network Australia, National Notifiable Disease Surveillance System, personal communication, May 2002).

It is largely unknown why notifications of BF have been greater than RR, however the very dry summer experienced in the inland areas of Victoria appears to have greatly affected mosquito numbers, including the main vector species *Cx. annulirostris*. This in turn may have resulted in very little arbovirus amplification and transmission in the inland regions and therefore a reduction in the overall number of alphavirus notifications. This does not, however, explain the number of BF disease notifications from Gippsland or why very few RR disease notifications were received from this area. Gippsland experienced a wetter than average summer which resulted in an increase in the population of the salt marsh mosquito, *Oc. camptorhynchus*. As *Oc. camptorhynchus* is a vector for both BF⁸ and RR,⁹ one might expect to see a rise in BF notifications, but the low number of RR notification is not explained. However, it is interesting to note that the transmission of BF in the absence of RR has also been associated with large numbers of *Oc. camptorhynchus* in south-west Western Australia,¹ and with large numbers of *Oc. vigilax*, a closely related species from the same genus,

in New South Wales.² There is also laboratory evidence that RR transmission potential in *Oc. vigilax* may be reduced if the mosquitoes are exposed to high ambient temperatures.¹⁰

As theorised with previous outbreaks of BF in the absence of RR,¹ this incident may have been associated with lower immunity to BF compared to RR in both animal hosts and humans. Another possibility is that there was transmission of BF under conditions that were not conducive to the transmission of RR.¹

Relatively little is known about BF disease and its animal reservoirs. This lack of knowledge affects the management of this disease, and at present there is little that can be done other than reducing vector mosquito populations and educating the public on preventing exposure. When cases were being interviewed, some commented that exposure to mosquitoes was high. Despite this, information gathered from this outbreak suggests that residents infrequently take precautions to limit exposure to mosquitoes, and population complacency is a potential factor that may need to be addressed.

BF disease is a disease where prevention is critical in reducing the risk of being infected, and further efforts need to be made to educate people of the importance of using repellents that are effective against mosquitoes and taking other personal preventative measures. Further research is needed to determine what other methods could be employed to control this disease.

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The Australian Institute of Health and Welfare

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This paper presents the work of the Australian Institute of Health and Welfare (AIHW) and its role in communicable diseases information with a focus on communicable diseases. The AIHW has a number of datasets that can support investigations and research of communicable disease epidemiology. These datasets are managed under Commonwealth legislation and are accessible to external researchers where they meet privacy guidelines. This paper sets out more detail about these issues.

The Australian Institute of Health and Welfare

The Australian Institute of Health and Welfare was established as the Australian Institute of Health by an Act of Parliament in 1987, to report to the nation on the state of its health. In 1992, the Australian Institute of Health was expanded to embrace community service statistics becoming the Australian Institute of Health and Welfare. The AIHW origins can be traced back to antecedents including the School of Public Health and Tropical Medicine set up by the Commonwealth Department of Health at the University of Sydney in 1930. As an independent agency under the portfolio of Health and Ageing, the AIHW works with many government and non-government bodies across the nation to generate reliable, regular and current facts and figures on the health and welfare of Australians.

The AIHW's work is guided by a mission

To improve the health and well-being of Australians, we inform community discussion and decision-making through national leadership in developing and providing health and welfare statistics and information.

The AIHW's values of objectivity, independence, quality, respect, accessibility, client focus and people are reflected in all its work. These values, combined with an adherence to strict privacy and confidentiality standards, are enshrined in *The Australian Institute of Health and Welfare Act*

1987 and have positioned the organisation as a leader in the field of health and welfare statistics and information.

The scope of AIHW work can be summarised as:

- to identify and meet the information needs of governments and the community to enable them to make informed decisions to improve the health and welfare of all Australians;
- to provide authoritative and timely information and analysis to the Commonwealth, State and Territory governments and non-government clients through the collection, analysis and dissemination of national health, housing assistance and community services data; and
- to develop, maintain and promote, in conjunction with stakeholders, information standards for health, housing assistance and community services.

The AIHW's work is underpinned by expertise in all aspects of information technology, including database management, software development, information standards, data linkage and IT security.

The AIHW has both the skill and capacity to bring together the major interested parties to develop and promote standardised data definitions and collection methods, new national collections, the linking of separate national collections, and presentation of key summary statistics (or indicators). These skills give the AIHW the capacity to develop innovative approaches to information processing, information management, and data and information access within the varying limitations of the collaborating agencies specified by data providers. All major AIHW publications are available free of charge from its website (www.aihw.gov.au).

In summary, the AIHW role is to provide statistics and information on the nation's health and welfare to government and community bodies for use in policy development, discussions and decision making. However, the AIHW does not directly formulate health or welfare policy.

The AIHW's role in communicable disease reporting

The AIHW has four key functions which are relevant to communicable disease surveillance in Australia;

- Australia's Health and Australia's Welfare: flagship publications of the Institute;
- national data sets;
- national information standards and agreements; and
- protecting information.

Australia's Health and Australia's Welfare: flagship publications of the Institute

The AIHW has a legislative requirement to produce reports on the nation's health and welfare services each two years. *Australia's Health 2002* was the eighth biennial health report of the AIHW. It is the nation's authoritative source of statistics and information on patterns of health and illness, determinants of health, the supply and use of health services, and health service costs and performance. The sections on communicable disease provide information on acute respiratory infections; bloodborne diseases such as viral hepatitis and HIV/AIDS; non-HIV sexually transmitted infections; vaccine preventable diseases; and on topical problems such as meningococcal disease and Creutzfeldt-Jakob disease. The report can be found on the AIHW website at: <http://www.aihw.gov.au/publications/aus/ah02/index.html>.

Australia's Welfare 2001 was the fifth biennial report on welfare services by the AIHW. The publication draws together information in the fields of welfare services including expenditure, housing assistance, children's and family services, ageing and aged care services, and disability services, and well being.

National data sets

Hospital morbidity

The AIHW compiles the National Hospital Morbidity Database (NHMD) from data supplied by State and Territory health authorities. It is a collection of electronic, confidential summary records for admitted patients separated from public and private hospitals in Australia in the years 1993–94 to 2000–01 (ongoing). The NHMD uses the National Health Data Dictionary definitions, which ensures a high standard of data comparability.

The AIHW presents a summary of this collection in the annual report *Australian Hospital Statistics*. The report presents detailed information on the characteristics and hospital care of the six million people admitted to public and private hospitals each year, including their age, sex, diagnoses and the procedures they underwent. The report can be found at: <http://www.aihw.gov.au/publications/hse/ahs00-01/index.html>

Interactive cubes of data

Included on the AIHW website are interactive cubes of data from the National Hospital Morbidity Database, which allow users to specify tables and graphs. Each cube includes information on the number of separations (same day and overnight), patient days and average length of stay, by age group and sex and year of separation, for each diagnosis. The datacubes can be found at: <http://www.aihw.gov.au/hospitaldata/datacubes/index.html>

AIHW National Mortality database and the National Death Index

Mortality data are stored in several ways at the AIHW:

1. Long-term spreadsheets containing data on many specific causes of death by age and sex going back to 1907 (Available from the Mortality portal at: www.aihw.gov.au);
2. The AIHW National Mortality Database (unit record data from 1964 to 2001); and
3. National Death Index (unit record data from 1980 to 2002).

Information for the AIHW National Mortality Database is provided to the AIHW by the Registrars of Births, Deaths and Marriages and coded nationally by the Australian Bureau of Statistics (ABS). The database holds all registered deaths in Australia since 1964 (latest year is 2000). The information includes the disease or condition leading directly to death and the other contributing diseases or conditions, as well as demographic and administrative information.

The Registrars also provide the data for the National Death Index database. This database contains identified unit record data from 1980 to 2002 and can be used for up-to-date fact and cause of death case findings.

National information standards and agreements

Australia has an established structure to develop information for both the health and public health program portfolio. The AIHW understands and appreciates the information capture and management needs of its stakeholders.

National Health Information Agreement

The National Health Information Agreement operates under the auspice of Australian Health Ministers' Advisory Council; its signatories are the Commonwealth, State and Territory health authorities, the ABS and the AIHW. It operates through the National Health Information Management Group that comprises representatives of all the signatories.

National Health Information Management Group

The AIHW participates in the national health structure on the National Health Information Management Group and its National Health Data Committee. The AIHW produces the National Health Data Dictionary, which contains all national data definitions developed through the committee. The goal is national consistency and it is designed to improve the comparability of data across the health arena.

National Public Health Partnership

The National Public Health Partnership (NPHP) provides a formal structure for the Commonwealth, State and Territory Governments to come together to develop a joint Australian intergovernmental agenda for public health. The AIHW is a member of the NPHP, and its sub-committee, the National Public Health Information Working Group.

National Public Health Information Working Group

The National Public Health Information Working Group manages the information development portfolio of the of the NPHP. It publishes the National Public Health Information Development Plan which represents a comprehensive strategy for the development of public health information in Australia.

Protecting identified information

The *Australian Institute of Health and Welfare Act 1987* prescribes strict conditions to ensure the security of the data it manages. It provides for strict penalties (including imprisonment) for breaches of confidentiality. In particular, the Act prohibits the release of personal information to the police and courts. The AIHW has produced a suite of documents that provide guidelines for staff and researchers regarding the collection, storage, use and release of data collected under the AIHW Act.

Ethics

The Act provides for oversight of the AIHW data collections by the AIHW Ethics Committee. The AIHW is obliged to protect the confidentiality of the information it receives, to respect the privacy and sensitivity of those to whom it relates, to maintain high-level data security procedures and, where appropriate, to incorporate the requirements of its information providers in those procedures. Section 29 of the AIHW Act specifies confidentiality provisions. The Section allows for the release of identifiable information to specified third parties with the written permission of the information provider or with the formal approval of the AIHW Health and Welfare Ethics Committee.

Summary

This overview has described the AIHW role in Australia's health information which is highly relevant to communicable disease surveillance. The AIHW provides statistics and information on the nation's health and welfare within local, state, national and international settings. It has established an expertise in the collection, standards and dissemination of information. Given these qualities, the AIHW has a wealth of expertise and welcomes the use of its publications and data sources. Details of the Institute's work can be found on its website (www.aihw.gov.au).

Communicable Diseases Surveillance

Highlights for 3rd quarter, 2002

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 third quarter of 2002, compared with the 5-year third quarter mean. Disease notifications above or below the 5-year mean, plus- or minus- two standard deviations are marked with an asterisk. Diseases where the number of cases reported was two standard deviations above the mean of the same reporting period in the last 5 years in the current quarter were hepatitis E, shiga-like toxin producing *Escherichia coli* and ornithosis. The reports of Ross River virus infection, leptospirosis and tuberculosis were two standard deviations below the 5-year mean in this quarter. These and other disease trends are discussed below with additional commentary provided by State and Territory health authorities.

Due to the difficulties in data transmission in this quarter, Victorian data for sexually transmitted diseases, incident hepatitis B and C diseases, Ross River virus infection, dengue, tuberculosis and Barmah Forest virus infections were not updated and the numbers presented (Table 2) should be interpreted with caution.

Gastrointestinal disease

Campylobacteriosis

In all jurisdictions, there were fewer reports of campylobacteriosis in the third quarter (3,075) compared with the mean for the last five years (3,380, Table 2). Campylobacteriosis notifications are lowest in winter months and show a seasonal peak in spring and summer. Notifications of campylobacteriosis in Australia, which have increased steadily since 1991, may now be stabilising (Figure 2).

Figure 1. Selected¹ diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 July to 30 September 2002 with historical data²

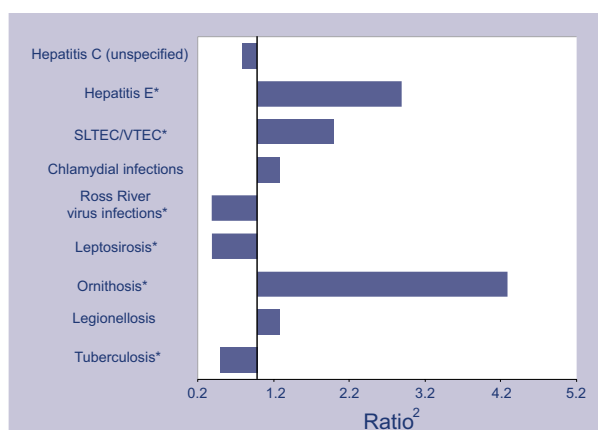
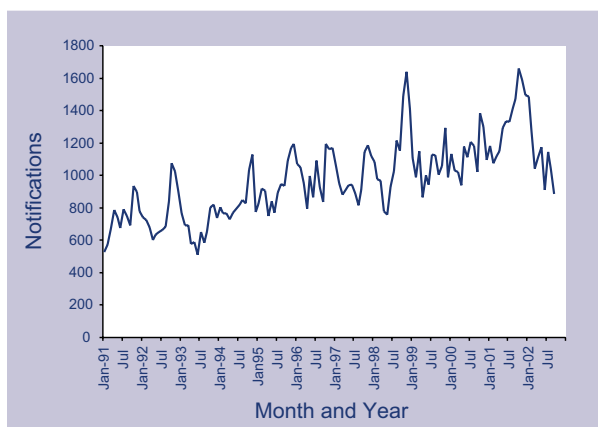


Figure 2. Trends in notifications of campylobacteriosis, Australia, 1991 to 2002

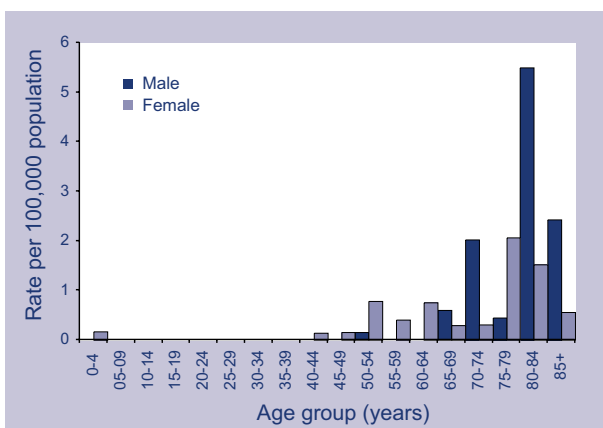


Despite the national totals of campylobacteriosis showing a decrease in this quarter, there was an increase in cases of campylobacteriosis in Far North Queensland. Interviews were conducted with 24 cases who had eaten chicken in the 7 days before onset of illness and 17/24 (71%) had eaten chicken in the 3 days before onset. Sixty-three per cent (15/24) had purchased fresh chicken to take home and cook. No other food, water or environmental exposures were common among cases. The results of quantitative testing of raw chicken samples and comparison of strains to isolates from cases remain to be assessed. Preliminary data suggests that approximately 80 per cent of raw chicken sold at retail outlets are contaminated with *Campylobacter*. The same poultry abattoir in North Queensland, was identified as a common link for cases of *Campylobacter* infection in 2000. This abattoir is the main supplier of chickens to region.

Listeriosis

Listeriosis is a serious bacterial disease caused by consumption of food contaminated with *Listeria monocytogenes*. The elderly and those with reduced immune function are at increased risk of infection and represent the majority of listeriosis cases in Australia (Figure 3).

Figure 3. Notification rates of listeriosis, Australia, 1 January to 30 September 2002, by age and sex



There was an increase in cases of listeriosis in Queensland during the quarter. Two cases reported consuming the same brand of smoked cod from South Africa and Argentina. Food samples tested positive for *Listeria*. As the cod was not marketed as a ready-to-eat food, there was no food recall, however, the importer was asked to clearly mark the food as requiring cooking before consumption.

In Western Australia, there was a recall of a feta cheese product contaminated with *Listeria*. The Communicable Disease Control Section of the Health Department of Western Australia advised that neither of the 2 cases of listeriosis reported in Western Australia in the quarter were associated with the consumption of this feta cheese product.

Salmonellosis

The number of notifications of salmonellosis was at a low level (101 cases) which is typical of winter months. There were 14 cases of *Salmonella* Hvittingfoss in Victoria, 11 of which were investigated as a possible cluster. One had a recent history of travel overseas, one was a breast-fed baby, and of the remaining nine, six had consumed cashews. There was, however, no common source or brand of cashews among the cases, no positive microbiology on samples, and no further cases. A case control study was not conducted and the source of the infections remains unknown.

Shiga-like toxin producing *Escherichia coli*

There were 12 cases of shiga-like toxin producing *Escherichia coli* (SLTEC/VTEC) infection notified to the NNDSS in the third quarter. This is twice the five-year mean for third quarter (Figure 1). The Communicable Diseases Control Branch of the South Australian Health Commission reported a cluster of SLTEC infections during the quarter. In South Australia all faeces with microscopic or macroscopic blood sent to microbiology laboratories are routinely screened by PCR for shiga toxin. Positive specimens are further screened using a multiplex PCR for multiple pathogenic genes. A cluster of four children (three females, one male: age range 1–7 years) and one female (aged 47 years) has been identified. Dates of onset ranged from 6 to 16 September 2002; four of the cases were residents of metropolitan Adelaide and one was from rural South Australia. The investigation has established a possible direct link between animal contact at a petting zoo and illness for 3 cases and an indirect link for a fourth. The multiplex PCR has detected the same pattern of genes in faeces from the 5 human cases and from a swab of the coat of one of the animals included in the environmental investigation. Shiga toxin positive *E. coli* O26 has been isolated from three of the human specimens. None of the cases developed haemolytic uraemic syndrome (HUS). Active surveillance of health services did not identify any other recent cases of HUS.

Other foodborne disease outbreaks

An outbreak of foodborne disease at a conference was reported from Western Australia in September 2002. An electronic questionnaire emailed to 533 delegates, of which 350 replied, identified 80 cases (23% attack rate). No pathogen was identified nor was a definitive link to any food established.

Quarantinable disease

Cholera

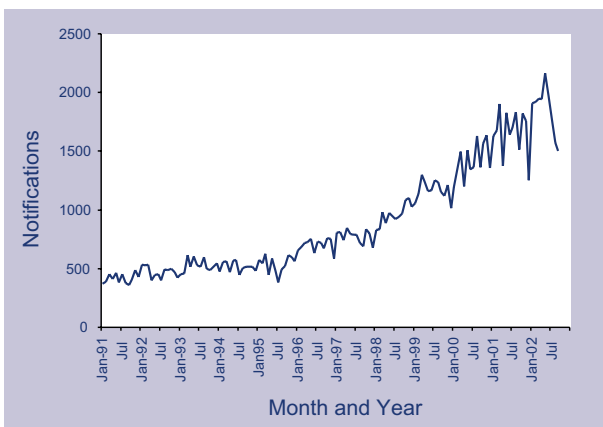
There were 3 cases of cholera reported to the NNDSS in the third quarter of 2002. One case, from South Australia, was subsequently identified as an infection with non-cholera *Vibrio* acquired in China. The other 2 cases were confirmed as *V. cholerae* O1, both acquired overseas (one in Pakistan and one in Vietnam).

Sexually transmitted infections

Chlamydial infection

There were 4,844 notifications of chlamydial infection in the third quarter of 2002, which was 30 per cent higher than the five-year mean (Figure 1). Chlamydial infections have been increasing in Australia since 1991 (Figure 4).

Figure 4. Trends in notifications of chlamydial infections, Australia, 1991 to 2002



Vaccine preventable diseases

Measles

There was a small outbreak of measles in North Queensland and New South Wales in July–August 2002. The outbreak was initiated by a 16-year-old visitor from Europe, who had travelled through

Thailand and resulted in 7 cases. The outbreak is described in more detail in a short report in this (Hanna).¹

One of the 7 cases was an unimmunised child from northern New South Wales, who subsequently infected two unimmunised siblings. The measles viral genotype in all cases in both States was identified as D5. This genotype has previously been identified as circulating in Thailand (Victorian Infectious Diseases Reference Laboratory annual report, 2001).

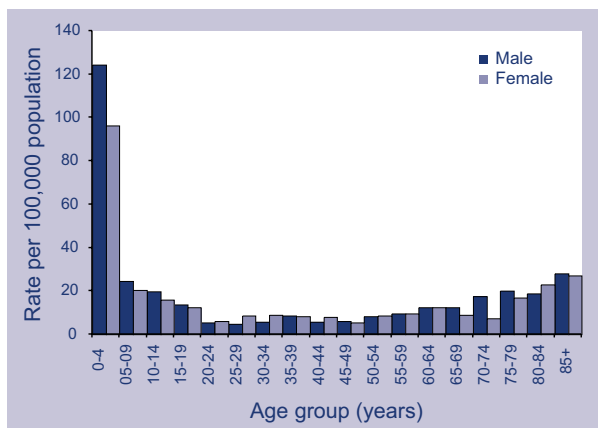
In August, a sailor from South-East Asia developed laboratory-confirmed measles within a few days of flying into Sydney. The public health unit assessed the risk to coworkers on the ship and among dock workers and recommended immunoglobulin to those who were susceptible. No further transmission was identified.

Two linked cases of measles occurred in Victoria in August in young adults without a history of travel. The first case, a nurse, may have been infected through contact with two other measles cases, neither of whom had a history of travel. Both cases were identified as measles genotype H1.

Influenza

There were 2,564 notifications of laboratory-confirmed influenza in the third quarter of 2002. The largest number of cases (944) and the highest rate (103.9 cases per 100,000 population) was in Queensland. The majority of notifications in the third quarter were influenza A; LabVISE data (Table 4) shows the ratio of influenza A:B isolations of 4.8:1. The majority of cases of laboratory-confirmed influenza cases were in children aged less than 5 years and in the elderly aged 65 years or more (Figure 5).

Figure 5. Notification rates of influenza, Australia, 1 January to 30 September 2002, by age and sex

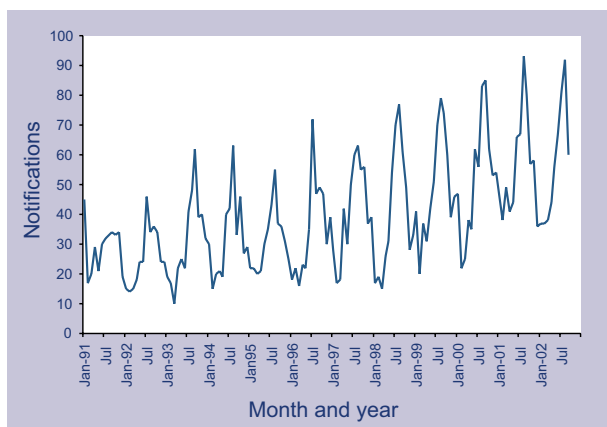


Other bacterial diseases

Meningococcal disease

There were 233 notifications of meningococcal disease in third quarter of 2002. The largest number of reports was from New South Wales (76 cases) and the highest rate was in Victoria (5.4 cases per 100,000 population). An analysis of meningococcal notification by month of onset, indicates a continuing increase since 1991 (Figure 6).

Figure 6. Trends in notifications of meningococcal infections, Australia, 1991 to 2002



There were three confirmed and one unconfirmed linked cases of meningococcal disease in a small Queensland rural community of 5,000 people. Two cases were identified as meningococcal group C and the third as a group Y. The group Y isolate did, however, show evidence of similarity with the group C cases on the basis of genetic typing. The cases were aged between 19 and 40 years. Tetravalent polysaccharide vaccine was offered to all people aged 18–40 years who lived or worked in the community and surrounds. In all, 2,300 vaccines were given. There were no further cases in the community and all the identified cases recovered.

A small cluster of meningococcal disease occurred in a child-care group in Victoria in July 2002. There were two confirmed cases of meningococcal serogroup C in children aged 4 and 5 years, both of whom attended the same day care centre. One child also attended a second child-care centre and conjugate meningococcal serogroup C vaccine was offered to children and staff at both centres.

Legionellosis

Public health authorities warned people to take care handling potting mix after a second death due to legionellosis disease in New South Wales in 2002. The death of a 77-year-old Sydney man in July 2002 follows that of a 78-year-old man in

January 2002. Other people infected with the illness in New South Wales this year have recovered. There were no deaths following *legionella* infection associated with potting mix in New South Wales last year, one case in 2000 and another single case in 1999. There were four deaths in 1998. *Legionella longbeachae* has been associated with lower respiratory infection in immunocompetent and immunocompromised individuals. The organism, especially in Australia, appears to be unique in being associated with pneumonia associated with exposure to soil. It has been found in soil and potting mixes from Australia but not from potting mix made in Europe. Importantly, the *legionellae* from the soil and the patient appeared to be closely related. Cases of *L. longbeachae* associated with potting mix have been described in the United States of America and Japan.

LabVISE

During the period July to September 2002, 12 participating laboratories (3 each in New South Wales, Western Australia and Victoria and one each in South Australia, Queensland and Tasmania), contributed 6,212 reports to LabVISE. Although there were no contributing laboratories in the Northern Territory, samples from this jurisdiction were included in reports from participating reference laboratories.

Of the 6,212 reports received, 4,532 (73%) were of viral infections and the remainder (1,680) were bacterial, spirochaete, fungal, protozoan or helminthic infections. Of the viral infections, reports of respiratory syncytial virus (1,302 reports) represented 28 per cent of all viral identifications and influenza virus (1,175 reports) represented a further 26 per cent of the viral pathogen total. This pattern of increased respiratory viral infections is typical of winter months. Among reports of non-viral pathogens, *Chlamydia trachomatis* (706 reports) represented 42 per cent of the total.

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Graham Tallis, Department of Human Services, Victoria

References

1. Hanna JN, Symons DJ, Lyon MJ. A measles outbreak in the Whitsundays, Queensland: the shape of things to come? *Commun Dis Intell* 2002;26:589–592.

Tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 22,527 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date between 1 July and 30 September 2002 (Table 2). The notification rate of diseases per 100,000 population for each State or Territory is presented in Table 3.

There were 6,212 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 July to 30 September 2002 (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 jurisdiction, except NT
Hepatitis C (incident)	All jurisdictions except Qld and NT
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Hepatitis NEC	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	
Cholera	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible diseases	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions except SA
Gonococcal infection	All jurisdictions
Syphilis	All jurisdictions

Disease	Data received from:*
Vaccine preventable diseases	
Diphtheria	All jurisdictions
<i>Haemophilus influenzae</i> type b	All jurisdictions
Influenza	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Tetanus	All jurisdictions
Vectorborne diseases	
Arbovirus infection NEC	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue	All jurisdictions
Japanese encephalitis	All jurisdictions
Kunjin	All jurisdictions except ACT [†]
Malaria	All jurisdictions
Murray Valley encephalitis	All jurisdictions [†]
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions except SA
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Ornithosis	All jurisdictions
Other lyssaviruses (NEC)	All jurisdictions
Q fever	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Jurisdictions may not yet be reporting a disease either because legislation has not yet made that disease notifiable in that jurisdiction, or because notification data for that disease are not yet being reported to the Commonwealth

† In the Australian Capital Territory, infections with Murray Valley encephalitis virus and kunjin virus are combined under Murray Valley encephalitis

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 July to 30 September 2002, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic [†]	WA	Total 3rd quarter 2002 ¹	Total 2nd quarter 2002 ¹	Total 3rd quarter 2001 ¹	Last five years mean 3rd quarter	Year to date 2002	Last five years YTD mean	Ratio [‡]
Bloodborne diseases															
Hepatitis B (incident)	0	27	4	12	4	5	2	3	57	122	119	91	276	254	0.6
Hepatitis B (unspecified)	26	863	NN	166	56	18	465	95	1,689	1,937	2,235	1,948	5,541	5,511	0.9
Hepatitis C (incident)	1	18	-	-	5	16	4	24	68	96	161	95	284	281	0.7
Hepatitis C (unspecified)	62	1,617	49	721	131	99	916	289	3,884	4,323	4,932	4,697	12,777	14,059	0.8
Hepatitis D	0	2	0	1	0	0	2	0	5	6	3	4	14	12	1.3
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0	0	2	-
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0.0
Campylobacteriosis ²	77	NN	63	889	623	127	884	412	3,075	3,199	4,212	3,380	10,074	9,541	0.9
Cryptosporidiosis	1	21	19	51	35	16	52	40	235	617	236	N/A	2,963	N/A	N/A
Haemolytic uraemic syndrome	0	1	0	0	0	0	0	0	1	3	1	2	7	8	0.5
Hepatitis A	0	25	18	8	3	1	13	11	79	81	149	359	306	1,357	0.2
Hepatitis E	1	3	0	0	0	0	0	0	4	6	1	1	11	7	2.9
Listeriosis	0	2	0	1	1	0	4	2	10	20	15	14	44	50	0.7
Salmonellosis	10	265	52	356	91	19	186	118	1,097	2,028	1,173	1,081	6,003	5,301	1.0
Shigellosis	0	22	19	19	4	1	16	20	101	130	126	120	379	460	0.8
SLTEC, VTEC ³	0	0	0	0	10	0	2	0	12	12	7	6	40	26	2.0
Typhoid	0	2	0	1	1	0	5	4	13	9	19	14	56	53	0.9
Quarantinable diseases															
Cholera	0	1	0	0	1	0	0	0	2	0	2	1	3	3	2.0
Plague	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
Viral haemorrhagic fever	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

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 July to 30 September 2002, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic [†]	WA	Total 3rd quarter 2002 ¹	Total 2nd quarter 2002 ¹	Total 3rd quarter 2001 ¹	Last five years mean 3rd quarter	Year to date 2002	Last five years YTD mean	Ratio [‡]
Sexually transmissible diseases															
Chlamydial infection	113	1,325	376	1,679	396	127	123	705	4,844	6,079	5,047	3,615	16,698	10,704	1.3
Donovanosis	0	0	1	2	NN	0	0	0	3	4	12	9	16	26	0.3
Gonococcal infection ⁴	5	276	435	223	43	3	79	311	1,375	1,685	1,518	1,323	4,630	4,233	1.0
Syphilis ⁵	2	167	140	105	1	3	43	30	491	480	369	425	1,343	1,217	1.2
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0
<i>Haemophilus influenzae</i> type b	0	0	0	1	1	0	0	1	3	11	4	10	25	28	0.3
Influenza	11	667	41	944	134	6	341	420	2,564	756	998	N/A	3,445	N/A	N/A
Measles	0	6	0	7	0	0	14	0	27	41	17	77	102	223	0.4
Mumps	0	7	0	0	3	0	1	4	15	20	20	48	54	144	0.3
Pertussis	6	331	3	411	82	4	127	38	1,002	1,175	2,962	2,037	3,984	4,743	0.5
Pneumococcal disease	8	281	25	204	42	19	158	90	827	577	657	N/A	1,701	N/A	N/A
Polio	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Rubella ⁶	1	11	0	64	0	1	0	1	78	52	70	182	186	466	0.4
Tetanus	0	0	0	0	0	0	0	0	0	0	0	1	2	4	0.0
Vectorborne diseases															
Arbovirus infection NEC	0	0	0	0	0	0	6	0	6	34	6	5	104	43	1.2
Barmah Forest virus infection	0	46	4	46	0	0	0	3	99	365	138	100	755	592	1.0
Dengue	2	13	3	10	2	0	0	4	34	67	48	29	188	186	1.2
Japanese encephalitis	0	0	0	0	0	0	0	0	0	0	0	N/A	0	N/A	N/A
Kunjin virus infection	-	0	0	0	0	0	0	0	0	0	0	N/A	0	N/A	N/A
Malaria	3	16	5	32	5	5	9	3	78	118	161	175	356	614	0.4
Murray Valley encephalitis	0	0	0	0	0	0	0	0	0	0	1	N/A	2	N/A	N/A
Ross River virus infection	0	15	1	66	6	0	1	3	92	567	158	212	1,326	3,828	0.4

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 July to 30 September 2002, by date of notification*

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic [†]	WA	Total 3rd quarter 2002 ¹	Total 2nd quarter 2002 ¹	Total 3rd quarter 2001 ¹	Last five years mean 3rd quarter	Year to date 2002	Last 5 years YTD mean	Ratio [‡]
Zoonoses															
Anthrax	0	0	0	0	NN	0	0	0	0	0	0	N/A	0	N/A	N/A
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	N/A	0	N/A	N/A
Brucellosis	0	1	0	6	0	0	1	0	8	8	4	13	29	25	0.6
Leptospirosis	0	5	0	7	0	2	0	0	14	44	38	36	128	170	0.4
Other lyssavirus	0	0	0	0	0	0	0	0	0	0	0	N/A	0	N/A	N/A
Ornithosis	0	59	2	1	1	0	9	1	73	66	29	17	158	56	4.3
Q fever	0	78	0	36	5	0	22	3	144	201	144	141	531	433	1.0
Other bacterial infections															
Legionellosis	1	7	1	9	15	0	13	13	59	96	60	47	215	216	1.3
Leprosy	0	0	0	0	0	0	0	0	0	0	1	2	3	4	0.0
Meningococcal infection	1	76	2	45	11	4	65	29	233	167	240	215	512	431	1.1
Tuberculosis	4	66	7	16	5	2	15	11	126	250	251	260	641	762	0.5
Total	335	6,322	1,270	6,139	1,717	478	3,578	2,688	22,527	25,452	26,344	20,793	75,912	66,074	1.1

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 because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

3. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).

4. Northern Territory, Queensland, South Australia, Victoria and Western Australia: includes gonococcal neonatal ophthalmia.

5. Includes congenital syphilis.

6. Includes congenital rubella.

* Date of notification = a composite of three dates: (i) the true onset date from a clinician, if available, (ii) the date the laboratory test was ordered, or (iii) the date reported to the public health authority.

† The number of notifications received from Victoria this quarter were fewer than expected due to technical difficulties in data transmission.

‡ Ratio = ratio of current quarter total to mean of the same reporting period over the last 5 years calculated as described above.

NA Not calculated as only notifiable for under 5 years.

NN Not notifiable

NEC Not elsewhere classified.

- Elsewhere classified.

Table 3. Notification rates of diseases by State or Territory, 1 July to 30 September 2002. (Rate per 100,000 population)

Disease	State or Territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic [†]	WA	
Bloodborne diseases									
Hepatitis B (incident)	0.0	1.6	8.0	1.3	1.1	4.2	0.2	0.6	1.2
Hepatitis B (unspecified)	32.1	52.2	NN	18.3	14.8	15.2	38.6	19.9	35.0
Hepatitis C (incident)	1.2	1.1	-	-	1.3	13.5	0.3	5.0	1.7
Hepatitis C (unspecified)	76.5	97.9	98.0	79.3	34.6	83.7	76.0	60.6	79.7
Hepatitis D	0.0	0.1	0.0	0.1	0.0	0.0	0.2	0.0	0.1
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis ²	95.0	-	126.0	97.8	164.5	107.4	73.3	86.5	95.5
Cryptosporidiosis	1.2	1.3	38.0	5.6	9.2	13.5	4.3	8.4	4.8
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	<0.1
Hepatitis A	0.0	1.5	36.0	0.9	0.8	0.8	1.1	2.3	1.6
Hepatitis E	1.2	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Listeriosis	0.0	0.1	0.0	0.1	0.3	0.0	0.3	0.4	0.2
Salmonellosis	12.3	16.0	104.0	39.2	24.0	16.1	15.4	24.8	22.5
Shigellosis	0.0	1.3	38.0	2.1	1.1	0.8	1.3	4.2	2.1
SLTEC, VTEC ³	0.0	0.0	0.0	0.0	2.6	0.0	0.2	0.0	0.2
Typhoid	0.0	0.1	0.0	0.1	0.3	0.0	0.4	0.8	0.3
Quarantinable diseases									
Cholera	0.0	0.1	0.0	0.0	0.3	0.0	0.0	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
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 diseases									
Chlamydial infection	139.4	80.2	751.9	184.8	104.6	107.4	10.2	147.9	99.4
Donovanosis	0.0	0.0	2.0	0.2	NN	0.0	0.0	0.0	0.1
Gonococcal infection ⁴	6.2	16.7	869.9	24.5	11.4	2.5	6.6	65.3	28.2
Syphilis ⁵	2.5	10.1	280.0	11.6	0.3	2.5	3.6	6.3	10.1
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
<i>Haemophilus influenzae</i> type b	0.0	0.0	0.0	0.1	0.3	0.0	0.0	0.2	0.1
Influenza	13.6	40.4	82.0	103.9	35.4	5.1	28.3	88.1	52.6
Measles	0.0	0.4	0.0	0.8	0.0	0.0	1.2	0.0	0.6
Mumps	0.0	0.4	0.0	0.0	0.8	0.0	0.1	0.8	0.3
Pertussis	7.4	20.0	6.0	45.2	21.7	3.4	10.5	8.0	20.6
Pneumococcal disease	9.9	17.0	50.0	22.4	11.1	16.1	13.1	18.9	17.0
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella ⁶	1.2	0.7	0.0	7.0	0.0	0.8	0.0	0.2	1.6
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 (continued). Notification rates of diseases by State or Territory, 1 July to 30 September 2002. (Rate per 100,000 population)

Disease	State or Territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic*	WA	
Vectorborne diseases									
Arbovirus infection NEC	0.0	0.0	0.0	0.0	0.0	0.0	0.5	0.0	0.1
Barmah Forest virus infection	0.0	2.8	8.0	5.1	0.0	0.0	0.0	0.6	2.0
Dengue	2.5	0.8	6.0	1.1	0.5	0.0	0.0	0.8	0.7
Japanese encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	-	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	3.7	1.0	10.0	3.5	1.3	4.2	0.7	0.6	1.6
Murray Valley encephalitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	0.0	0.9	2.0	7.3	1.6	0.0	0.1	0.6	1.9
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	NN	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.7	0.0	0.0	0.1	0.0	0.2
Leptospirosis	0.0	0.3	0.0	0.8	0.0	1.7	0.0	0.0	0.3
Other lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	3.6	4.0	0.1	0.3	0.0	0.7	0.2	1.5
Q fever	0.0	4.7	0.0	4.0	1.3	0.0	1.8	0.6	3.0
Other bacterial infections									
Legionellosis	1.2	0.4	2.0	1.0	4.0	0.0	1.1	2.7	1.2
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection	1.2	4.6	4.0	5.0	2.9	3.4	5.4	6.1	4.8
Tuberculosis	4.9	4.0	14.0	1.8	1.3	1.7	1.2	2.3	2.6

1. Rates are subject to retrospective revision.

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

3. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).

4. Northern Territory, Queensland, South Australia, Victoria and Western Australia: includes gonococcal neonatal ophthalmia.

5. Includes congenital syphilis.

6. Includes congenital rubella.

* The number of notifications received from Victoria this quarter were fewer than expected due to technical difficulties in data transmission.

NN Not notifiable

NEC Not elsewhere classified.

- Elsewhere classified.

Table 4. Virology and serology laboratory reports by State or Territory¹ for the reporting period 1 July to 30 September 2002, and total reports for the year²

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 3rd period 2002	This period 2001	Year to date 2002 ³	Year to date 2001
Measles, mumps, rubella												
Measles virus	-	-	-	-	-	-	6	-	6	8	15	104
Mumps virus	-	-	-	-	-	-	2	-	2	4	13	31
Rubella virus	-	-	-	17	1	-	2	1	21	21	69	51
Hepatitis viruses												
Hepatitis A virus	-	-	-	3	2	-	-	4	9	21	48	64
Hepatitis D virus	-	-	-	-	1	-	1	-	2	6	5	10
Hepatitis E virus	-	-	-	-	-	-	1	-	1	-	5	4
Arboviruses												
Ross River virus	-	-	-	9	-	1	2	3	15	30	362	808
Barmah Forest virus	-	1	-	21	2	-	1	1	26	20	155	240
Dengue not typed	-	-	-	-	-	-	-	8	8	34	152	185
Murray Valley encephalitis virus	-	-	-	-	-	-	-	1	1	1	6	7
Flavivirus (unspecified)	-	-	-	2	-	-	1	-	3	6	31	21
Adenoviruses												
Adenovirus type 40	-	-	-	-	-	-	-	8	8	8	29	43
Adenovirus not typed/ pending	-	47	-	12	67	-	5	59	190	314	557	766
Herpesviruses												
Cytomegalovirus	-	40	-	18	115	-	16	8	197	281	728	903
Varicella-zoster virus	-	26	-	125	28	4	10	109	302	371	1,198	1,280
Epstein-Barr virus	-	11	-	93	101	-	9	78	292	384	1,159	1,311
Other DNA viruses												
Contagious pustular dermatitis (Orf virus)	-	-	-	-	-	-	-	1	1	1	1	4
Molluscum contagiosum	-	-	-	-	-	-	-	3	3	1	15	10
Parvovirus	-	1	-	6	22	-	15	5	49	99	218	251
Poxvirus group not typed	-	-	-	-	-	-	1	-	1	2	5	2
Picornavirus family												
Coxsackievirus A9	-	1	-	-	-	-	-	-	1	-	2	2
Coxsackievirus B2	-	2	-	-	-	-	-	-	2	-	3	-
Echovirus type 6	-	1	-	-	-	-	-	1	2	1	58	2
Echovirus type 9	-	2	-	-	-	-	-	-	2	17	16	75
Echovirus type 13	-	1	-	-	-	-	-	-	1	12	15	28
Enterovirus — not typed	-	2	-	2	4	-	2	88	98	194	365	601
Poliovirus type 1 (uncharacterised)	-	4	-	-	-	-	-	-	4	8	18	17
Poliovirus type 2 (uncharacterised)	-	3	-	-	-	-	-	-	3	6	11	15
Poliovirus type 3 (uncharacterised)	-	1	-	-	-	-	-	-	1	5	3	7
Poliovirus not typed	-	1	-	-	-	-	-	-	1	-	1	-
Rhinovirus (all types)	-	46	-	-	5	-	-	36	87	119	280	319

Table 4 (continued). Virology and serology laboratory reports by State or Territory¹ for the reporting period 1 July to 30 September 2002, and total reports for the year²

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 3rd period 2002	This period 2001	Year to date 2002 ³	Year to date 2001
Ortho/paramyxoviruses												
Influenza A virus	-	289	-	150	187	-	37	310	973	543	1,359	702
Influenza B virus	-	30	-	16	68	-	3	85	202	75	425	136
Parainfluenza virus type 1	-	6	-	-	32	-	-	27	65	16	248	33
Parainfluenza virus type 2	-	2	-	1	5	-	-	6	14	20	59	45
Parainfluenza virus type 3	-	8	-	1	56	-	2	37	104	319	254	528
Respiratory syncytial virus	-	293	-	43	213	11	57	685	1,302	1,286	2,408	2,473
Other RNA viruses												
Astrovirus	-	-	-	-	-	-	1	-	1	-	1	1
HTLV-1	-	-	-	-	1	-	-	1	2	4	3	17
Rotavirus	-	202	-	-	95	11	60	113	481	679	867	1,302
Norwalk agent	-	-	-	-	-	-	49	-	49	19	186	130
Other												
<i>Borrelia burgdorferi</i>	-	-	-	-	-	-	-	1	1	-	1	-
<i>Brucella abortus</i>	-	-	-	-	-	-	-	1	1	-	2	1
<i>Brucella</i> species	-	-	-	1	-	-	-	-	1	1	4	3
<i>Chlamydia trachomatis</i> not typed	-	86	-	270	176	6	1	167	706	646	2,585	2,496
<i>Chlamydia psittaci</i>	-	-	-	-	-	-	10	2	12	12	40	58
<i>Chlamydia pneumoniae</i>	-	1	-	-	-	-	-	1	2	2	4	7
<i>Chlamydia</i> species	-	1	-	-	-	-	-	-	1	-	3	4
<i>Chlamydia</i> - typing pending	-	4	-	-	-	-	-	-	4	-	5	-
<i>Mycoplasma pneumoniae</i>	-	46	-	52	73	3	99	32	305	231	880	620
<i>Coxiella burnetii</i> (Q fever)	-	-	-	13	15	-	16	8	52	41	164	125
<i>Streptococcus</i> group A	-	-	-	71	-	-	21	-	92	75	302	262
<i>Yersinia enterocolitica</i>	-	2	-	1	-	-	-	-	3	2	7	4
<i>Bordetella pertussis</i>	-	7	-	43	46	-	46	16	158	470	723	925
<i>Legionella pneumophila</i>	-	-	-	-	-	-	20	-	20	18	66	48
<i>Legionella longbeachae</i>	-	2	-	-	1	-	7	5	15	8	37	19
<i>Legionella</i> species	-	-	-	-	-	-	4	-	4	5	9	12
<i>Cryptococcus</i> species	-	2	-	-	6	-	-	-	8	3	23	18
<i>Leptospira</i> species	-	-	-	2	-	-	-	-	2	6	18	33
<i>Treponema pallidum</i>	-	28	-	163	75	-	-	14	280	254	968	886
<i>Entamoeba histolytica</i>	-	-	-	1	-	-	-	3	4	-	19	9
<i>Toxoplasma gondii</i>	-	2	-	-	-	-	3	1	6	11	20	27
<i>Echinococcus granulosus</i>	-	-	-	-	-	-	1	2	3	4	23	17
Total	0	1,201	0	1,136	1,397	36	511	1,931	6,212	6,724	17,256	18,102

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
 2. From January 2000 data presented are for reports with report dates in the current period. Previously reports included all data received in that period.
 3. Totals comprise data from all laboratories. 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.
- No data received this period.

Table 5. Virology and serology laboratory reports by laboratories for the reporting period 1 July to 30 September 2002¹

	Laboratory	July 2002	August 2002	September 2002	Total this period
Australian Capital Territory	The Canberra Hospital	-	-	-	-
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	188	116	30	334
	New Children's Hospital, Westmead	234	152	-	386
	Royal Prince Alfred Hospital, Camperdown	-	-	-	-
	South West Area Pathology Service, Liverpool	214	186	81	481
Queensland	Queensland Medical Laboratory, West End	478	551	107	1136
	Townsville General Hospital	-	-	-	0
South Australia	Institute of Medical and Veterinary Science, Adelaide	757	640	-	1397
Tasmania	Northern Tasmanian Pathology Service, Launceston	27	9	-	36
Victoria	Monash Medical Centre, Melbourne	47	-	-	47
	Rickettsia Reference Laboratory, Geelong	-	-	-	0
	Royal Children's Hospital, Melbourne	149	56	10	215
	Victorian Infectious Diseases Reference Laboratory, Fairfield	152	97	-	249
Western Australia	PathCentre Virology, Perth	493	488	-	981
	Princess Margaret Hospital, Perth	473	339	79	891
	Western Diagnostic Pathology	-	35	24	59
Total		3,212	2,669	331	6,212

1. The complete list of laboratories reporting for the 12 months, January to December 2002, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

- Nil reports

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. The system provides the national surveillance of more than 50 communicable diseases or disease groups endorsed by the Communicable Diseases Network Australia and the National Public Health Partnership. Notifications of these diseases are made to State and Territory health authorities under the provisions of their respective public health legislation. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see Commun Dis Intell 2002;26:58.

LabVISE is a sentinel reporting scheme. Currently 15 laboratories contribute data on the laboratory identification of viruses and other organisms. This number may change throughout the year. Data are collated and published in Communicable Diseases Intelligence quarterly. These data should be interpreted with caution as the number and type of reports received is subject to a number of biases. For further information, see Commun Dis Intell 2002;26:61.

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 66 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 2002, 10 conditions are being monitored, six of which are related to communicable diseases. These include influenza, gastroenteritis and acute cough. Definitions of these conditions were published in *Commun Dis Intell* 2002;26:57.

Data to the end of September 2002 are shown as the rate per 1,000 consultations by week in Figures 7 to 9.

Figure 7. Consultation rates for influenza-like illness, ASPREN, 1 January to 30 September 2002 and 2001, by week of report.

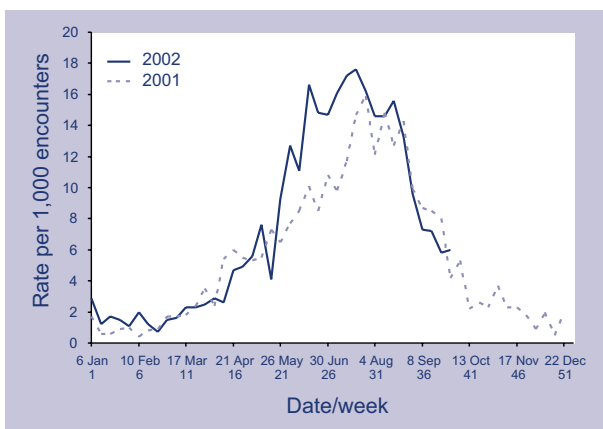


Figure 8. Consultation rates for gastroenteritis, ASPREN, 1 January to 30 September 2002, by week of report

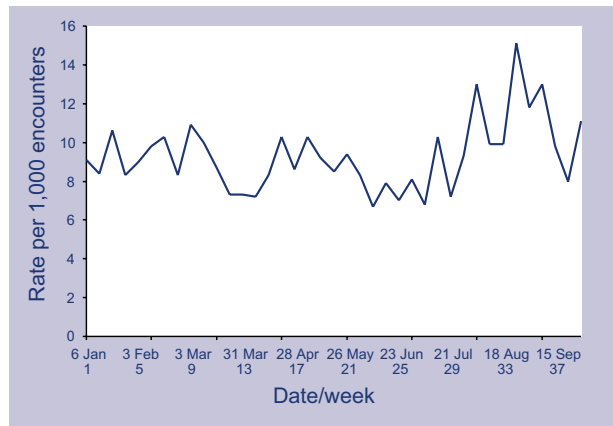
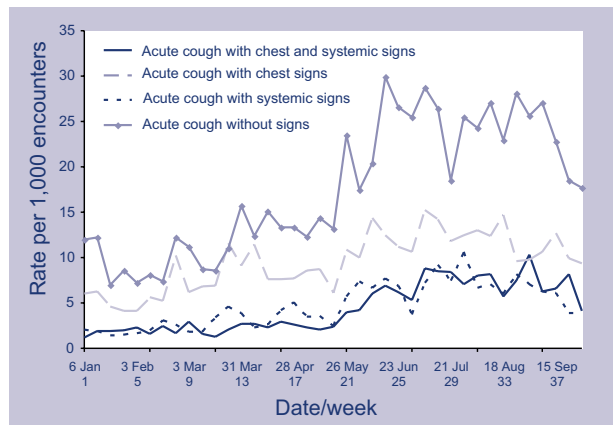


Figure 9. Consultation rates for acute cough, ASPREN, 1 January to 30 September 2002, by week of report



Gonococcal surveillance

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

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When *in vitro* resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.¹ Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see Commun Dis Intell 2002;26:61.

Reporting period 1 April to 30 June 2002

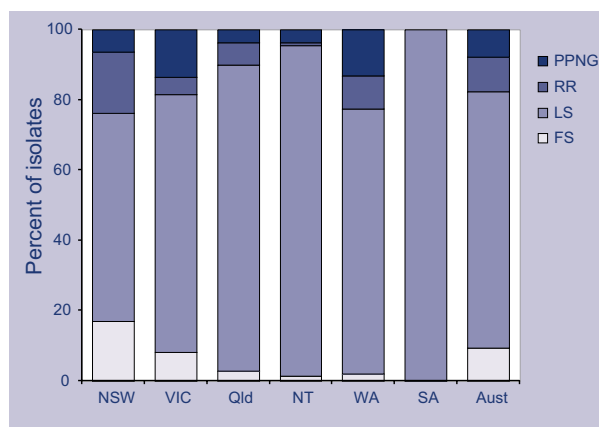
The Australian Gonococcal Surveillance Programme (AGSP) laboratories examined a total of 1,000 isolates in this quarter, an increase on the 858 recorded in the same period in 2001. About 40 per cent of this total was from New South Wales, 20 per cent from Victoria, 14 per cent from Queensland, 13 per cent from the Northern Territory, 10 per cent from Western Australia and 2 per cent from South Australia. Isolates from other centres were few. The progressive total of gonococci examined by the AGSP to 30 June is about 10 per cent higher than in 2001.

Penicillins

Figure 10 shows the proportions of gonococci fully sensitive (MIC \leq 0.03 mg/L), less sensitive (MIC 0.06 – 0.5 mg/L), relatively resistant (MIC \geq 1 mg/L) or else penicillinase producing (PPNG) aggregated for Australia and by State or Territory. A high proportion of strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

In this quarter about 17.7 per cent of all isolates were penicillin resistant by one or more mechanisms — 7.7 per cent PPNG and 10 per cent by chromosomal mechanisms (CMRNG). The proportion of penicillin resistant strains ranged from nil in South Australia to 24 per cent in New South Wales.

Figure 10. Categorisation of gonococci isolated in Australia, 1 April to 30 June 2002, by penicillin susceptibility and region



- FS fully sensitive to penicillin, MIC \leq 0.03 mg/L
 LS less sensitive to penicillin, MIC 0.06 – 0.5 mg/L
 RR relatively resistant to penicillin, MIC \geq 1 mg/L
 PPNG penicillinase producing *Neisseria gonorrhoeae*

The number of PPNG isolated across Australia (n=77) was higher in this quarter than in the corresponding period in 2001 (n=58). The highest proportion of PPNG was found in isolates from Victoria (13.6%) and Western Australia (13.2%). PPNG were present in all jurisdictions except South Australia including 5 (3.7%) in the Northern Territory. Data on geographic acquisition were available in 26 cases only, mostly from New South Wales and Western Australia. Local acquisition was prominent in both these States.

More isolates were resistant to the penicillins by separate chromosomal mechanisms (n=100, 10%). These CMRNG were especially prominent in New South Wales (71 CMRNG 17.4% of all isolates there), and Western Australia (9.2%). A single CMRNG was detected in the Northern Territory.

Ceftriaxone

Low numbers of isolates with decreased susceptibility to ceftriaxone (n=6, 1.5%) were present in New South Wales, but none were found elsewhere in Australia in this quarter. Treatment failure with cefixime, an oral third generation cephalosporin not available in Australia, has now been reported in Japan.²

Spectinomycin

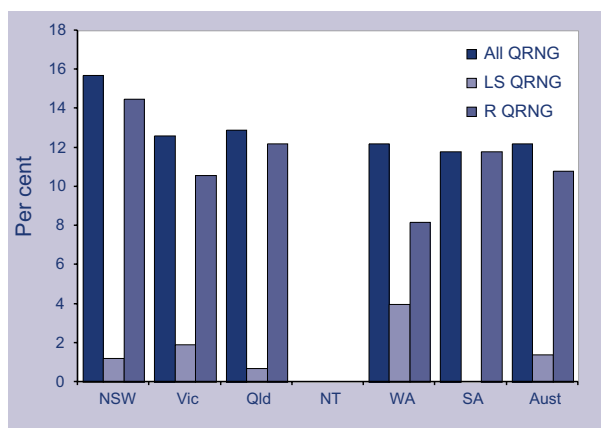
All isolates were susceptible to this injectable agent.

Quinolone antibiotics

Quinolone resistant *N. gonorrhoeae* (QRNG) are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06 – 0.5 mg/L) or resistant (MIC \geq 1 mg/L) categories.

The total number (n=122) of all QRNG was lower than in the corresponding period in 2001 (n=165), but similar in distribution to that seen in recent quarters (Figure 11). QRNG made up 12.2 per cent of all strains examined nationally and this proportion was lower than in 2001 (19%). QRNG were again widely distributed and represented between 12 per cent and 16 per cent in most jurisdictions. The exception was the Northern Territory where no QRNG were detected. In all jurisdictions, the high level resistance category of QRNG (MIC ciprofloxacin \geq 1 mg/L) predominated. Nationally, 108 of the 122 QRNG were in this category.

Figure 11. Distribution in Australia of *N. gonorrhoeae* displaying quinolone resistance, 1 April to 30 June 2002



LS QRNG Ciprofloxacin MICs 0.06 – 0.5 mg/L

R QRNG Ciprofloxacin MICs \geq 1 mg/L

High level tetracycline resistance

The number (122) and proportion (12.2%) of high level tetracycline resistance (TRNG) detected was double that of the same period in 2001. TRNG represented between 10 per cent and 15 per cent of isolates in Queensland, Victoria, New South Wales, and Western Australia and 4 per cent in the Northern Territory.

Reference

1. Management of sexually transmitted diseases. World Health Organization; Document WHO/GPA/TEM94.1 Rev.1 1997:37.
2. WHO Western Pacific Gonococcal Antimicrobial Surveillance Programme. Surveillance of antibiotic resistance in *Neisseria gonorrhoeae* in the WHO Western Pacific Region, 2001. *Commun Dis Intell* 2002;26:541–545.

Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit (APSU) conducts nationally based active surveillance of rare diseases of childhood, including specified communicable diseases and complications of rare communicable diseases in children. The primary objectives of the APSU are to document the number of Australian children under 15 years newly diagnosed with specified conditions, their geographic distribution, clinical features, current management and outcome. Contributors to the APSU are clinicians known to be working in paediatrics and child health in Australia. In 2001, over 1,000 clinicians participated in the surveillance of 15 conditions through the APSU, with an overall response rate of 98 per cent. For further information please contact the APSU on telephone: +61 2 9845 2200, e-mail: apsu@chw.edu.au.

The results for January to December 2001 are shown in Table 6.

Reporting period January to December 2001

About the APSU communicable diseases studies

Acute flaccid paralysis

Heath Kelly, Bruce Thorley, Kerri Anne Brussen, Jayne Antony, Elizabeth Elliott, Anne Morris

Acute flaccid paralysis (AFP) surveillance in children <15 commenced through the APSU in 1995. To the end of 2001 there were 232 confirmed cases of AFP. Based on these data, the estimated incidence was 0.9 (95% CI 0.8-1.1) per 100,000 children.

Congenital cytomegalovirus infection

William Rawlinson, Daniel Trincado, Gillian Scott, Sian Munro, Pamela Palasanthiran, Mark Ferson, David Smith, Geoff Higgins, Michael Catton, Alistair McGregor, Dominic Dwyer, Alisson Kesson

Congenital cytomegalovirus (CMV) surveillance in children <12 months of age commenced through the APSU in 1999. Between January 1999 and December 2001 there were 16 confirmed cases of CMV. The estimated incidence was 2.1 (95% CI 1.2-3.5) per 100,000 live births.

Congenital rubella

Margaret Burgess, Jill Forrest

Surveillance of newly diagnosed congenital rubella in children and adolescents aged <16 years commenced in 1993. Forty-two children with congenital rubella were identified through the APSU between May 1993 and December 2001. Twenty-seven of these children were born in Australia, 21 of which had defects attributable to congenital rubella. The estimated incidence of congenital rubella was 1.2 (95% CI 0.8-1.7) per 100,000 live births.

HIV infection, AIDS and perinatal exposure to HIV

Ann McDonald, John Kaldor, Michelle Good, John Ziegler

Between January 1997 and December 2001, 97 children with perinatally acquired HIV infection were reported through the APSU (72%), the National HIV/AIDS surveillance program (18%) or both sources (10%). The estimated incidence was 7.8 (95% CI 6.3-9.5) per 100,000 live births.

Table 6. Confirmed cases of communicable diseases reported to the Australian Paediatric Surveillance Unit January to December 2001*

Condition	Current reporting period 2001	Previous reporting period 2000
Acute flaccid paralysis	44	38
Congenital cytomegalovirus	6	10
Congenital rubella	0	0
Perinatal exposure to HIV	24	15
Neonatal herpes simplex virus infection	11	8

* Surveillance data are provisional and subject to revision

Neonatal herpes simplex virus infection

Cheryl Anne Jones, David Isaacs, Peter McIntyre, Tony Cunningham, Suzanne Garland

There were 43 confirmed cases of neonatal herpes simplex virus infection in infants <28 days of age between January 1997 and December 2001. The estimated incidence was 3.4 (95% CI 2.5-4.6) per 100,000 live births.

Hospitalised pertussis in infancy

Peter McIntyre, Elizabeth Elliott, Anne Morris, Greta Ridley, John Massie, Julie McEniery, Geoff Knight

Between January and December 2001, children <12 months of age admitted to hospital with pertussis were reported to the APSU. There were 140 confirmed cases of hospitalised pertussis in 2001. The estimated incidence was 56/100,000 (95%CI 47-66) per 100,000 live births.

HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory

(Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Cases of AIDS are notified through the State and Territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: <http://www.med.unsw.edu.au/nchechr>. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell 2002;26:59.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 April to 30 June 2002, as reported to 30 September 2002, are included in this issue of Communicable Diseases Intelligence (Tables 7 and 8).

Table 7. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 April to 30 June 2002, by sex and State or Territory of diagnosis

	Sex	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Totals for Australia			
										This period 2002	This period 2001	Year to date 2002	Year to date 2001
HIV diagnoses	Female	0	6	1	0	1	0	5	3	16	22	45	47
	Male	1	70	0	27	1	0	42	4	145	155	316	326
	Not reported	0	0	0	0	0	0	0	0	0	1	0	1
	Total ¹	1	76	1	27	2	0	47	7	161	178	364	375
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	3	7	7
	Male	0	11	0	4	3	0	7	1	26	36	71	69
	Total ¹	0	11	0	4	3	0	7	1	26	39	79	77
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	1	2	3
	Male	0	8	0	3	0	0	1	0	12	15	25	29
	Total ¹	0	8	0	3	0	0	1	0	12	16	27	32

1. Persons whose sex was reported as transgender are included in the totals.

Table 8. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 March 2002, by sex and State or Territory

	Sex	State or Territory								Australia
		ACT	NSW	NT	QLD	SA	TAS	VIC	WA	
HIV diagnoses	Female	28	685	12	185	77	5	264	142	1,398
	Male	233	11,728	113	2,201	739	84	4,302	994	20,394
	Not reported	0	237	0	0	0	0	24	0	261
	Total ¹	261	12,675	125	2,393	816	89	4,606	1,142	22,107
AIDS diagnoses	Female	9	208	0	53	29	3	81	31	414
	Male	88	4,853	38	900	372	45	1,746	387	8,429
	Total ¹	97	5,074	38	955	401	48	1,836	420	8,869
AIDS deaths	Female	4	122	0	35	18	2	57	19	257
	Male	71	3,356	25	596	244	30	1,319	267	5,908
	Total ¹	75	3,487	25	633	262	32	1,383	287	6,184

1. Persons whose sex was reported as transgender are included in the totals

Childhood immunisation coverage

Tables 9, 10 and 11 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children fully immunised at age 12 months for the cohort born between 1 April to 30 June 2001; at 24 months of age for the cohort born between 1 April to 30 June 2000; and at 6 years of age for the cohort born between 1 April to 30 June 1996, 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, E-mail: brynleyh@chw.edu.au.

Immunisation coverage for 'fully immunised' children at 12 months for Australia has increased from the last quarter by 1.0 percentage point to 91.2 per cent (Table 9). The change in 'fully immunised' coverage varied by state and territory but all jurisdictions experienced an increase in coverage. The Northern Territory (+2.8%) and Western Australia (+1.8%) experienced the greatest increases in

coverage. All other states experienced not insignificant increases in coverage over the quarter. Coverage is hovering around the 91 per cent level in almost all jurisdictions with the highest level in Tasmania (92.9%) and the lowest in Western Australia (90.3%). The most dramatic changes in coverage were evident in the Northern Territory where increases of more than 2 per cent occurred for almost all vaccines. The continued increase in coverage at 12 months of age for all jurisdictions and for all vaccines is very encouraging and indicates that coverage has perhaps not reached a plateau as first thought. For the first time, every jurisdiction has coverage greater than 90 per cent for 'fully immunised' and for all individual vaccines. The highest coverage for an individual vaccine at 12 months of age is for hepatitis B vaccine. National coverage for hepatitis B is almost 95 per cent and five jurisdictions have reached over 95 per cent coverage — New South Wales (95.2%), the Northern Territory (96.9%), Queensland (95.1%), South Australia (95.2%) and Tasmania (96.4%).

Coverage measured by 'fully immunised' at 24 months for Australia increased from the last quarter by 0.7 percentage points to 88.8 per cent (Table 10). Coverage increased from the previous quarter in all jurisdictions except for New South Wales and the Australian Capital Territory with the greatest increases in the Northern Territory (+2.2%) and Western Australia (+2.1%). However, only two jurisdictions

(Tasmania and South Australia) have achieved greater than 90 per cent coverage for 'fully immunised' at 24 months of age. Coverage for individual vaccines by 24 months for Australia, however, is much greater. Coverage for OPV is 94.7 per cent and 94.3 per cent for Hib suggesting that at least part of the lower figure for fully immunised may relate to data issues. At

the jurisdiction level, the most important changes in coverage occurred for the Hib vaccine. There were decreases in Hib coverage at 24 months of age in all jurisdictions. The decreases were not dramatic with the greatest decrease in the Australian Capital Territory (-1.9%), but are of concern as they were universal.

Table 9. Percentage of children immunised at 1 year of age, preliminary results by disease and State or Territory for the birth cohort 1 April to 30 June 2001; assessment date 30 September 2002

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	953	21,034	913	12,716	4,293	1,385	14,799	5,993	62,086
Diphtheria, tetanus, pertussis (%)	92.3	92.3	92.4	92.6	92.8	93.4	92.7	91.8	92.5
Poliomyelitis (%)	92.4	92.2	92.1	92.4	92.8	93.3	92.7	91.6	92.4
<i>Haemophilus influenzae</i> type b (%)	94.2	94.4	96.8	94.7	95.0	96.4	94.9	94.5	94.7
Hepatitis B (%)	94.5	95.2	96.9	95.1	95.2	96.4	94.4	94.0	94.9
Fully immunised (%)	90.8	91.0	91.3	91.4	91.8	92.9	91.3	90.3	91.2
Change in fully immunised since last quarter (%)	-1.0	+1.1	+2.7	+0.8	+0.9	+1.2	+0.6	+1.8	+1.0

Table 10. Proportion of children immunised at 2 years of age, preliminary results by disease and State or Territory for the birth cohort 1 April to 30 June 2000; assessment date 30 September 2002¹

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,114	21,839	905	12,417	4,461	1,473	15,264	6,168	63,641
Diphtheria, tetanus, pertussis (%)	89.4	90.1	88.2	91.9	91.4	93.7	91.6	89.5	90.9
Poliomyelitis (%)	93.1	94.1	96.1	94.7	95.5	96.1	95.5	94.1	94.7
<i>Haemophilus influenzae</i> type b (%)	92.8	94.0	94.8	94.5	95.0	95.5	94.7	93.2	94.3
Measles, mumps, rubella (%)	91.6	93.1	95.6	94.2	94.8	95.2	94.2	93.3	93.8
Hepatitis B (%)	-	-	-	-	-	-	-	-	-
Fully immunised (%)²	87.2	87.7	87.4	89.9	90.0	92.8	89.5	87.1	88.8
Change in fully immunised since last quarter (%)	-1.3	+0.8	+1.5	+1.1	+2.5	+3.2	+0.7	+0.8	+1.0

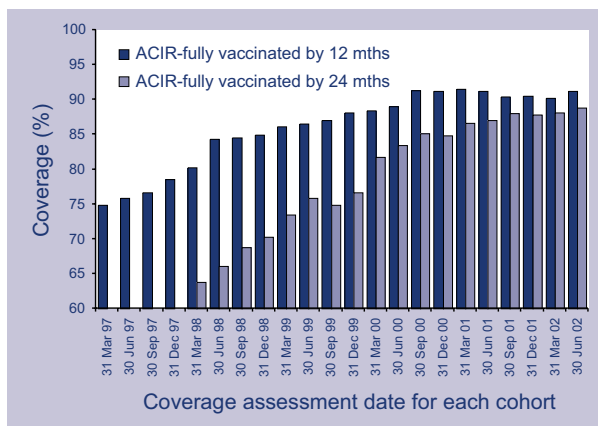
1. The 12 months age data for this cohort were published in *Commun Dis Intell* 2001;25:307.

2. These data relating to 2 year-old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

Table 11 shows immunisation coverage estimates for individual vaccines and for 'fully immunised' children at 6 years of age for Australia and by state or territory. These are the second set of officially published ACIR figures of immunisation coverage estimates for this age group. 'Fully immunised' coverage at 6 years of age for Australia increased from the last quarter by 0.8 percentage points to 81.4 per cent. The greatest increases in coverage occurred in the Northern Territory (+11.3%) and Tasmania (+4.8%) whilst two jurisdictions experienced small decreases in 'fully immunised' coverage for this age group, Queensland (-0.5%) and South Australia (-0.1%). National coverage by individual vaccine also increased from the last quarter for all vaccines for this age group but there were wide variations in the changes in coverage by jurisdiction. Both the Northern Territory and Tasmania experienced large increases in coverage for DTP, OPV and MMR coverage at 6 years of age, whilst coverage for these three vaccines decreased in Queensland and South Australia. The recent report published by NCIRS shows that true levels of coverage at 6 years of age are actually higher than reported here as late immunisation is still common (NCIRS, 2001).

Figure 12 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 and 24 months. However, the rate of increase in coverage is slowing with the curve beginning to flatten out and turn downward slightly for estimates at 12 months of age.

Figure 12. Trends in vaccination coverage, Australia, 1997 to 2002, by age cohorts



Reference

National Centre for Immunisation Research and Surveillance. Immunisation Coverage: Australia 2001. Report. Canberra: Department of Health and Aged Care, 2001. <http://www.health.gov.au/pubhlth/immunise/report.pdf>.

Acknowledgement: The Table figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Commonwealth Department of Health and Ageing. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the HIC: Telephone: +61 2 6124 6607.

Table 11. Proportion of children immunised at 6 years of age, preliminary results by disease and State or Territory for the birth cohort 1 April to 30 June 1996; assessment date 30 September 2002

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Number of children	1,023	22,081	15,740	13,011	4,758	6,759	1,549	822	65,743
Diphtheria, tetanus, pertussis (%)	85.5	83.4	85.8	83.9	84.5	81.8	85.9	85.2	84.1
Poliomyelitis (%)	85.4	83.4	86.3	84.4	84.8	82.1	86.1	86.7	84.4
<i>Haemophilus influenzae</i> type b (%)	-	-	-	-	-	-	-	-	-
Measles, mumps, rubella (%)	84.4	81.0	86.0	83.6	83.0	81.5	85.2	85.9	83.1
Hepatitis B(%)	-	-	-	-	-	-	-	-	-
Fully immunised (%)¹	83.5	79.1	84.4	82.1	81.7	79.3	84.5	83.3	81.4
Change in fully immunised since last quarter (%)	+2.2	+0.8	+11.3	-0.5	-0.1	+4.8	+1.1	+1.0	+0.8

1. These data relating to 6 year-old children should be considered as preliminary. The proportions shown as 'fully immunised' appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

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 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*.

Reported by Joan Powling (NEPSS Co-ordinator) and Mark Veitch (Public Health Physician), Microbiological Diagnostic Unit — Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne. For further information please contact NEPSS at the above address or on Telephone: +61 3 8344 5701, Facsimile: +61 3 9625 2689.

Reports to the National Enteric Pathogens Surveillance System of *Salmonella* infection for the period 1 July to 30 September 2002 are summarised in Tables 12 and 13. Data include cases reported and entered by 20 October 2002. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS.

1 July to 30 September 2002

The total number of reports to NEPSS of human *Salmonella* infection fell to 1,035 in the third quarter of 2002, 43 per cent less than the second quarter of 2002. This third quarter nadir is typical of the seasonal variation seen in human salmonellosis in Australia.

During the third quarter of 2002, the 25 most common *Salmonella* types in Australia accounted for 620 (60 percent) of all reported human infections.

S. Typhimurium phage type 135, *S. Typhimurium* phage type 9, *S. Saintpaul*, *S. Typhimurium* phage type 170, and *S. Typhimurium* phage type 126 were the most common salmonellae in both the second and third quarters of 2002, although the counts of all 5 serovars and phage types fell in the third quarter.

The most notable increase in reports was for *S. Enteritidis* phage type 4b. This phage type was first reported to NEPSS in 2001, and infections are typically associated with travel to Bali.

In contrast, *S. Virchow* phage type 8, *S. Bovismorbificans* phage type 24, and *S. Typhimurium* phage type 8 were common in the second quarter, but rare in the third quarter.

We thank contributing laboratories, scientists, Joan Powling (NEPSS Co-ordinator) and Mark Veitch (Public Health Physician), Microbiological Diagnostic Unit - Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne. For further information please contact NEPSS at the above address or on Telephone: +61 3 8344 5701, Facsimile: +61 3 9625 2689.

Table 11. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 July to 30 September 2002, as reported to 20 October 2002

Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total all <i>Salmonella</i> for quarter	9	254	32	296	93	19	211	121	1,035
Total contributing <i>Salmonella</i> types	8	87	22	93	52	12	69	51	207

Table 12. Top 25 *Salmonella* types identified in Australian States and Territories, 1 July to 30 September 2002

National rank	<i>Salmonella</i> type	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total 2nd quarter 2002	Last 10 years mean 2nd quarter	Year to date 2002	Year to date 2001	Total 2001
1	<i>S. Typhimurium</i> 135	2	28	0	7	4	6	24	11	82	68	531	470	636
2	<i>S. Typhimurium</i> 9	0	17	0	9	6	1	19	10	62	74	511	308	399
3	<i>S. Saintpaul</i>	0	6	2	32	2	1	10	5	58	40	320	210	289
4	<i>S. Typhimurium</i> 170	1	18	0	14	0	1	20	0	54	11	319	63	148
5	<i>S. Typhimurium</i> 126	0	3	0	4	6	0	15	6	34	16	177	220	313
6	<i>S. Enteritidis</i> 4b	0	12	0	0	0	1	9	11	33	<1	47	3	13
7	<i>S. Birkenhead</i>	0	10	0	16	1	0	2	0	29	20	200	180	253
8	<i>S. Infantis</i>	0	9	2	4	0	0	4	5	24	18	87	92	123
9	<i>S. Chester</i>	0	6	0	7	2	0	1	7	23	20	131	124	166
10	<i>S. Typhimurium</i> 197	1	12	0	6	0	0	4	0	23	<1	47	5	8
11	<i>S. Hittingfoss</i>	0	3	0	7	0	0	9	1	20	6	133	64	89
12	<i>S. Typhimurium</i> U290	0	4	1	1	0	0	12	2	20	<1	76	8	26
13	<i>S. Virchow</i> 8	0	3	0	16	0	0	0	0	19	13	249	188	245
14	<i>S. Aberdeen</i>	0	0	0	16	0	1	1	0	18	10	118	73	88
15	<i>S. Muenchen</i>	0	2	2	2	3	2	3	0	14	16	103	98	125
16	<i>S. Anatum</i>	0	3	2	5	0	0	1	3	14	12	66	52	58
17	<i>S. Agona</i>	1	5	2	1	1	1	2	0	13	15	66	37	56
18	<i>S. Havana</i>	0	1	0	3	5	0	0	3	12	10	30	34	46
19	<i>S. Singapore</i>	0	5	0	5	0	0	1	0	11	8	46	41	64
20	<i>S. Enteritidis</i> 26	0	3	0	5	0	0	3	0	11	4	36	11	24
21	<i>S. Waycross</i>	0	4	0	6	0	0	0	0	10	8	85	40	54
22	<i>S. Stanley</i>	1	1	0	3	1	0	2	2	10	12	42	80	107
23	<i>S. Typhimurium</i> 141	0	2	0	1	2	0	0	4	9	6	24	25	27
24	<i>S. Enteritidis</i> 6a	0	3	0	0	1	0	3	2	9	2	22	19	34
25	<i>S. Mgulani</i>	0	1	0	7	0	0	0	0	8	4	57	37	66

Overseas briefs

World Health Organization

This material has been summarised from information on the World Health Organization Internet site. A link to this site can be found under 'Other Australian and international communicable diseases sites' on the Communicable Diseases Australia website home page.

Influenza in Madagascar

An outbreak of influenza affected 5 out of 6 provinces in Madagascar in August 2002. The total number of reported cases (to 22 August) was 22,646 with 671 deaths. The epidemic of influenza was attributed to influenza A/Panama/2007/99-like (H3N2) viruses, the same strain that was associated with influenza epidemics worldwide during 2001-02. The observed increase in excess mortality compared to previous years appeared to be due to widespread transmission and not an especially virulent influenza strain. Most deaths occurred outside of health facilities and disproportionately affected young children.

The WHO Global response team recommended expanding the influenza sentinel surveillance system and implementing a standard case definition for influenza-like illness; improving case management and training for health care providers; and providing health education activities to inform the public about influenza and the need for those at high risk (young children, the elderly and those with chronic illness) to seek medical care if experiencing acute respiratory illness.

West Nile virus in the United States - update

As of 23 September 2002, the WHO Collaborating Centre for Arthropod Borne Viruses — Western Hemisphere, at the Centers for Disease Control and Prevention (CDC) has reported 1,963 human cases of the West Nile virus, with 94 deaths occurring in 32 states and the District of Columbia. During 2002, West Nile virus activity (evidence of infections in birds, humans, mosquitoes, and other animals — primarily horses) has been documented in 42 states and the District of Columbia.

For more information about this outbreak see the CDC web site at: www.cdc.org.

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 website home page.

Listeriosis in the United States of America

Source: NJ com, 27 September 2002 (edited)

Twenty cases of listeriosis and 5 deaths have occurred in New Jersey in the United States of America. Investigators have been unable to pinpoint the source of the sickness that has also struck people in 7 other states. Cases have also been reported in Pennsylvania, New York, Delaware, Connecticut, Maryland, Michigan, and Ohio. The Centers for Disease Control and Prevention have isolated and matched the strain of bacteria in 31 patients from five of the seven other states, suggesting that these patients acquired the illness from eating the same food.

Five of the 20 cases in New Jersey have been conclusively linked to the strain of *Listeria* identified from the outbreak. Throughout the United States, there are an estimated 2,500 cases of listeriosis each year, with about 500 reported deaths. Those at higher risk of getting listeriosis include the elderly, pregnant women, newborns, and adults with weakened immune systems, such as those with terminal cancer or AIDS.

E. coli O157: ground beef recall in the United States of America

Source: NY Times, 27 September 2002 (edited)

At least 56 people have become ill in the latest incident of ground beef suspected to be contaminated with a potentially deadly strain of *Escherichia coli* O157 bacteria. The outbreak in Wisconsin, Minnesota, and Illinois comes just days after the United States Department of Agriculture, under fire for allegedly failing to protect the public against *E. coli* O157:H7, declared 'war' on it and ordered all US beef plants to adopt new safeguards.

At latest count, symptoms among 52 people in Wisconsin, three in Minnesota, and one in Illinois have been linked to the contamination. Officials said 19 people in Wisconsin were admitted to hospital. "Heightened surveillance continues throughout Wisconsin."

First case of vCJD reported in Italy

Source: *Boston.com*, 27 September 2002 (edited)

A 25-year-old woman has been confirmed as Italy's first victim of the human form of mad cow disease, according to research published on 27 September 2002. The woman, who lives in Sicily, was hospitalised in November 2001 after suffering for 6 months with pain in her back and legs, a progressive disturbance in her walking, and unpleasant sensations when her skin was touched, said the report in the *Lancet* medical journal.¹

The ailing woman has never travelled to Britain or any other country with reported cases of mad cow disease. Following release of the report, Italy's Health Ministry reported that 73 cows have tested positive for bovine spongiform encephalopathy (BSE). Italy detected its first case of mad cow disease (BSE) last year after the European Union ordered mandatory tests on cattle older than 30 months destined for slaughter.

Reference

1. La Bella V, Collinge J, Pocchiari M, Piccoli F. Variant Creutzfeldt-Jakob disease in an Italian woman *Lancet* 2002;360:997–98.

vCJD tonsil and appendix survey

Source: *Eurosurveillance Weekly Issue 39*, 26 September 2002 (edited)

A paper published in the *British Medical Journal* has provided new information on the prevalence of preclinical variant Creutzfeldt-Jakob disease (vCJD) in the United Kingdom (UK).^{1,2} A distinctive feature of vCJD is the widespread distribution of an abnormal prion protein in peripheral lymphoid tissue,^{3,4} which may be detectable before any symptoms develop.⁵

The authors looked retrospectively for the presence of abnormal prion protein in 8,318 appendectomy and tonsillectomy samples and found one positive appendix. It is not known whether an asymptomatic person with detectable abnormal prion protein will go on to develop vCJD but, as discussed in the paper, 19 of 20 appendices removed at autopsy from patients with vCJD have shown accumulation of abnormal prion protein, as did the appendices removed from 2 patients prior to disease onset.⁵ Whilst the paper does report the first estimate of the prevalence of abnormal prion protein based on population testing, larger studies are needed to provide a more precise estimate.

References

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5. Variant Creutzfeldt-Jakob disease. *Lancet* 1998; 352:703–704.

vCJD update – United Kingdom

Source: *UK Department of health press release*, 2 September 2002

On 2 September 2002 the United Kingdom Department of Health issued the latest information about the numbers of known cases of Creutzfeldt-Jakob disease. This includes cases of variant Creutzfeldt-Jakob disease — the form of the disease thought to be linked to bovine spongiform encephalopathy. The following is a summary of vCJD cases:

Deaths from definite vCJD (confirmed): 92

Deaths from probable vCJD (without neuropathological confirmation): 22

Deaths from probable vCJD (neuropathological confirmation pending): 1

Number of deaths from definite or probable vCJD (as above): 115

Number of probable vCJD cases still alive: 12

Total number of definite or probable vCJD (dead and alive): 127

Smallpox vaccination strategies (USA)

Source: *NY Times*, 24 September 2002 (edited)

Federal health officials today instructed states to prepare to vaccinate every American in the event of a biological attack using smallpox, and issued a detailed plan showing how each state could quickly inoculate as many as one million people

in the first 10 days. Officials at the federal Centers for Disease Control and Prevention (CDC) said publicly for the first time that even one case of smallpox might result in a nationwide program of voluntary vaccinations.

Smallpox, which was eradicated worldwide two decades ago, is highly contagious and kills roughly a third of its victims, making it a potentially fearsome biological weapon. Officially, the virus is supposed to exist only in repositories in Moscow and the CDC's headquarters in Atlanta, but experts have long suspected that some nations harbor secret stocks of smallpox to use as a biological weapon.

The vaccine is one of the few that can work even if a person is already infected, and experts say it can protect people if given within 4 days of exposure to the virus. The guide says up to 75 million doses of the nation's vaccine stockpile could be shipped in a single day and 280 million doses, enough to cover every American, in 5 to 7 days.

The guidelines call for states to run 20 clinics 16 hours a day, an effort that the government estimates would require 4,680 public health workers and volunteers.

However, the plan does not address the vexing, and politically delicate issue of whether to vaccinate public health workers and emergency personnel before a terrorist attack. Many public health experts say the precautionary vaccinations are necessary, but the issue is complicated because the vaccine, made from a live virus, carries risks to patients with skin disorders and immune system deficiencies, including people with AIDS. Those who are vulnerable are endangered not only by being inoculated, but also by contact with others who have been inoculated.

The Center's previous smallpox preparedness plan revolved around a strategy in which public health workers would track down and vaccinate infected people and those who came into contact with them, working in concentric circles until the outbreak was contained.

The new document does not supplant the 'ring vaccination' plan, but the guide was undoubtedly influenced by recent studies showing that ring vaccination would not contain a large outbreak. Studies had found that if 1,000 people were infected in a large city like New York and ring vaccination was used, within 3 months there would be 300,000 cases of smallpox and 100,000 deaths, and the epidemic would not be contained. Mass vaccination, he said, would contain such an epidemic in 40 to 45 days, with 1,500 cases and 500 deaths.

Dengue update

Bangladesh

Source: The Daily Star Internet edition, 10 September 2002

In Bangladesh, a total of 4,955 people has been attacked with dengue so far. Of these, 45 have died and 178 are undergoing treatment.

Malaysia

Source: New Straits Times, 19 September 2002

The number of cases of dengue fever and dengue haemorrhagic fever in Malaysia rose from 19,429 with 52 deaths in 1997 to 16,263 cases and 43 deaths last year. Between January and August 2002, there were 17,341 cases of dengue fever and dengue haemorrhagic fever reported nationwide with 34 deaths. Health department officials attributed the increase in cases to a recent stretch of wet weather followed by a short dry spell.

Hong Kong

Source: BBC news Online, 22 September 2002

Hong Kong has reported its first locally-contracted case of dengue fever after a construction worker became stricken with the mosquito-borne disease. Inspectors have visited the construction site where the man worked on Ma Wan island, after blood samples taken from two of the man's co-workers showed they too had contracted the disease, but had recovered. The site is to be fumigated and the rest of the man's coworkers to be put under medical surveillance.

Although 10 cases of the fever have been previously reported in the territory, all the sufferers had contracted the disease while travelling abroad.

Texas

Source: Austin American Statesman, 22 September 2002

The Texas Department of Health confirmed seven of 24 suspected cases of dengue fever in Matamoros, Tamaulipas, Mexico. Officials in Brownsville fear the mosquito-borne illness could easily make its way across the Rio Grande to the city. So far, the disease has not crossed into Brownsville, where officials said it has been 3 years since a dengue case was reported. A bi-national spraying effort is ongoing in response to the West Nile virus outbreak and officials expect it to intensify as dengue concerns increase. On the Mexican side of the border, officials continue

to spray where the dengue cases were detected. Matamoros health department officials reported 6 confirmed classic dengue cases of 34 suspected cases in the city last year.

West Nile virus and acute flaccid paralysis

Source: *MMWR*, 20 September 2002

West Nile virus (WNV) infection can cause severe, potentially fatal neurologic illnesses including encephalitis and meningitis.^{1,2} Acute WNV infection also has been associated with acute flaccid paralysis (AFP) attributed to a peripheral demyelinating process (Guillain-Barré Syndrome [GBS]),³ or to an anterior myelitis.⁴ However, the exact etiology of AFP has not been assessed thoroughly with electrophysiologic, laboratory, and neuroimaging data. The *MMWR* report describes 6 cases of WNV-associated AFP in which clinical and electrophysiologic findings suggest a pathologic process involving anterior horn cells and motor axons similar to that seen in acute poliomyelitis. Clinicians should evaluate patients with AFP for evidence of WNV infection and conduct tests to differentiate GBS from other causes of AFP.

Reference

(See original *MMWR* report)

Anthrax in humans — USA

Source: *Palm Beach Post*, 16 September 2002 (edited)

FBI investigators believe photocopy machines helped spread anthrax throughout the American Media Inc. headquarters in 2001, before the building was quarantined. While testing the 3-story building for anthrax spores, investigators found that every copy machine in the building (more than 24 in all) tested positive for anthrax, according to a source familiar with the investigation. The anthrax is believed to have gotten into the copiers from reams of copy paper that had trapped airborne spores in the company's mail room, where the paper was stored. The FBI's theory helps explain for the first time the presence of anthrax throughout the 68,000-square-foot building. Once investigators realised the copy machines were contaminated, they traced the anthrax back to its point of origin: an open storage area in AMI's first-floor mailroom. Apparently, someone in the mailroom opened a letter containing anthrax, which dispersed the microscopic particles. The spores settled on the company's supply of copy paper.

Anthrax spores tend to stick to surfaces upon settling. The spores can detach from surfaces, but loosening the particles requires sufficient force. AMI employees unwittingly distributed the clinging spores throughout the building when taking reams of copy paper to every department in the building. When the copy paper was inserted into the machines and used to make copies, investigators believe, the spores dislodged and were 'aerosolised' into the atmosphere by the whirring fans and other moving parts of the high-speed copiers.

Meningococcal disease update

Burundi

Source: *WHO*, 12 September 2002

As of 2 September 2002, the number of cases of meningococcal disease reported in Muyinga province remains high at 50 cases per week. Additional cases have been reported in Cankuzo province as well as in Ruyigi province. WHO and UNICEF are providing 747,500 doses of vaccine for the vaccination campaign; the campaign will be supported by Medecins sans Frontieres (MSF) France in Muyinga province and by MSF Holland in Ruyigi province.

Rwanda

Source: *WHO*, 12 September 2002

As of 2 September 2002, a total of 636 cases of meningococcal disease and 83 deaths were reported in 8 out of 12 provinces in Rwanda. The WHO sub-regional intercountry advisor carried out an epidemiological assessment in the country. A consolidated appeal for 2 million doses of vaccine was launched by the Rwandan Ministry of Health, WHO, Medecins sans Frontieres and UNICEF to vaccinate populations in the areas at risk.

Tanzania

Source: *WHO*, 12 September 2002

A meningococcal vaccination campaign will begin early next week for refugees in the camps in the Kibondo district, Kibondo province as well as for those refugees registered for repatriation to the northern provinces in Burundi. It will be carried out by the International Rescue Committee, supported by the United Nations High Commissioner for Refugees. Surveillance is also being strengthened in Kasulo province, which borders on Kibondo province.

Refugees fleeing war in Burundi are the source of a bacterial meningitis outbreak in north-western Tanzania. State-run Radio Tanzania reported on 5 September 2002, that at least 24 people had died recently of meningitis. The radio station did not give the nationalities of the dead, but said a total of 117 cases were recorded in the past 2 weeks. At least one million Burundian refugees fleeing the almost 10-year-old war in their home country live in north-western Tanzania.

Nipah virus – Bangladesh

In May 2001, ProMED-mail posted a press report that described an outbreak of suspected Japanese encephalitis (JE) in a remote village in Bangladesh. The provisional diagnosis of JE was not confirmed and the outbreak was linked to a previous unexplained outbreak in North Bengal, India, inconclusively attributed to an atypical strain of measles virus.

In the outbreak there were 28 cases of acute neurological syndrome (progressive fever, malaise, headache, coma, and death) of which 9 were fatal. The cases were adults, with the majority of cases being male. The clinical diagnosis was viral encephalitis, but lumbar puncture and other investigations were not done and necropsies were not performed. Pigs lived in proximity to the villagers and JE was initially suspected, but had not been previously recognised as a problem in this area in Bangladesh and the sex, age, and case fatality rate were not consistent with JE. The epidemiological and clinical data provided to date suggested that the outbreak might have been the same disease that appeared in Siliguri, India, earlier in the year.

The Government of Bangladesh has made public that this cluster of encephalitis cases was linked to infection with Nipah virus or a closely related paramyxovirus. (Measles is also a paramyxovirus, so perhaps this explains the laboratory results, which led to the diagnosis of 'atypical measles' at the time. There is no mention of cough, which was a prominent symptom in both humans and pigs in the Nipah virus outbreak.)

Nipah virus is a newly discovered paramyxovirus responsible for a viral encephalitis outbreak in Malaysia in 1998–1999, which resulted in around 300 confirmed infections and a mortality of about 35 per cent in hospitalised cases. As in the Bangladesh outbreak, Japanese encephalitis was initially suspected, but the epidemiology was inconsistent with JE. A concurrent epidemic of respiratory illness occurred in pigs and over

1 million pigs were culled in outbreak control measures. Subsequent investigations suggested that fruit bats of the genus *Pteropus* were the probable virus reservoir. Genetic analysis revealed that Nipah virus is closely related to Hendra virus, another recently discovered paramyxovirus, associated with disease in horses and humans in Australia and also maintained in *Pteropus* fruit bats. Bengal and Bangladesh are within the distribution range of *Pteropus* fruit bats. Nipah and Hendra viruses are members of a previously unknown group of paramyxoviruses.

Vaccine-derived poliomyelitis – Madagascar

Source: *MMWR*, 19 July 2002

Surveillance for acute flaccid paralysis (AFP) in Madagascar has detected a cluster of 4 cases of paralytic poliomyelitis from which type 2 vaccine-derived polioviruses have been isolated. None of the children affected were fully vaccinated. Genetic sequencing studies of these vaccine-derived viruses indicate substantial genetic drift and recombination with non-polio enteroviruses. These findings are compatible with an outbreak of paralytic polio associated with a circulating vaccine-derived poliovirus (cVDPV), however, further investigation is required.

The 3 outbreaks of cVDPV described previously occurred in areas where routine oral polio vaccine (OPV) coverage is low, AFP surveillance is suboptimal, and supplementary vaccination activities have not been conducted for years.^{1,2} Vaccination coverage data suggest that during 1999, 37 per cent of children aged <1 year had received 3 doses of OPV. In 2001, the non-polio AFP rate of 0.3 case per 100,000 population aged <15 years was below the target level of 1.0.

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