Tuberculosis notifications in Australia, 1998

National TB Advisory Committee (currently Ral Antic (Chair), John Carnie, Amanda Christensen, Margo Eyeson-Annan, Jag Gill, Anastasios Konstantinos, Vicki Krause, Mark Hurwitz, Avner Misrachi) for the Communicable Diseases Network Australia and New Zealand[1]

Abstract

Since the inception of the National Mycobacterial Surveillance System (NMSS) in 1991, annual crude notification rates for tuberculosis have remained stable at between 5 and 6 per 100,000 population. In 1998, there was a total of 923 TB notifications in Australia of which 884 were new TB cases, and 39 relapsed cases. The corresponding annual crude notification rate for new and relapsed TB was 4.72 and 0.21 per 100,000 respectively. Seventy-seven percent of notifications that had a country of birth reported were overseas born. In keeping with trends observed over recent reporting years, the populations for which notified TB rates are highest include the overseas born from high prevalence countries and Indigenous Australians. The lowest rates of disease have continued to be reported in the non-Indigenous, Australian born population. Surveillance reports over the last seven years indicate that the rate of disease in this population is gradually declining. ***Commun Dis Intell*** **2001;25:1-8.**

Keywords: tuberculosis, surveillance, indigenous, immigrants, mycobacterium

Introduction

The dominant global threat of tuberculosis (TB) to human health has been reaffirmed in a series of recent World Health Organization reports. Annually, over 2 million deaths worldwide were attributable to TB,[1] with 95 percent of these occurring in developing countries. It is estimated that there were over 8 million new cases of TB in 1998 worldwide with over 3.6 million reported to the WHO Global surveillance programme by 189 countries.[1] Of these TB notifications 39 per cent were reported to be managed under the WHO Directly Observed Treatment-Short course (DOTS) strategy for TB control[1] and 1.4 million of these

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notifications (40%) were new sputum-positive pulmonary cases.

The HIV pandemic continues to fuel the TB epidemic in many regions of the world, especially Asia and sub-Saharan Africa. Up to 40 per cent of AIDS deaths in Asia and sub-Saharan Africa are due to TB, and it is estimated that by the end of the century HIV will account for 1.5 million new TB cases per year that would otherwise have not occurred.[1]

The global burden of TB has been further exacerbated by poverty, natural disasters, conflict and political instability, all of which have served to thwart the development of health services in many countries, or have led to a progressive erosion of existing health infrastructures. Human migration, so often the consequence of these events, has created a social context in which the delivery of effective drug treatment is further compromised. Poorly supervised and inadequately treated TB is the basis for the emergent problem of multi-drug resistant TB (MDR-TB).

Of global TB case notifications in 1998, 59 per cent were from South-East Asia and the Western Pacific regions.[1] In the face of this major regional disease threat, Australia has maintained stable TB rates through effective pre-migration screening and the activities of specialised, multi-disciplinary TB services in the States and Territories.

The National Mycobacterial Surveillance System (NMSS), established in 1991, has enabled trends in the rates of active TB to be monitored over the last 7 years, has helped describe the epidemiology of TB in Australia, and has assisted in identifying high-risk groups for targeted control. Future enhancements to the existing system will serve to better inform policy makers, public health practitioners and clinicians on the outcomes achieved from TB control efforts.

Methods

Notifications reported to State and Territory health authorities are collated on an annual basis and referred to the NMSS in computerised format with all reports being de-identified beforehand. A core data field is shared with the National Notifiable Disease Surveillance System (NNDSS). Variables reported in this core field include a unique identifier for each notification, disease code (to differentiate *Mycobacterium* TB complex from atypical mycobacterial infections), postcode of residence, date of birth, sex, dates of disease onset and report, Indigenous status, and confirmation status of the report. A supplementary data set includes Indigenous status, country of birth, length of residence in Australia for overseas-born persons, species of the pathogen, principal site of disease, methods of diagnosis, antimicrobial therapy initiated at the time of notification, past BCG vaccination, HIV status and classification of TB as new or relapsed disease.

Tuberculosis (new case)

A case which has been confirmed by the identification of *Mycobacterium tuberculosis* (or *M. africanum* or *M. bovis*) by culture,

or

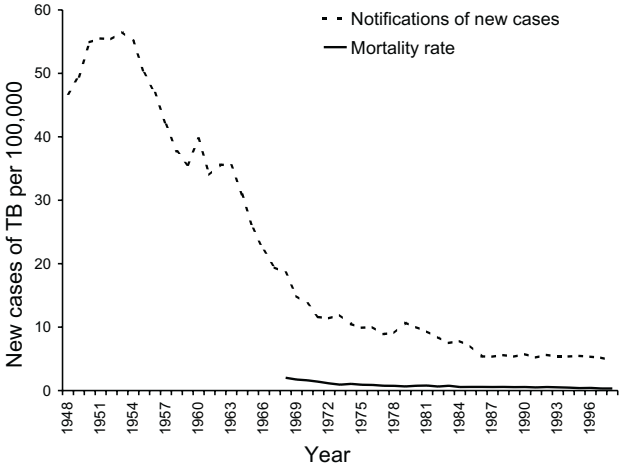
A case which has been diagnosed to be active clinically and which has been accepted as such by the State or Territory Director of Tuberculosis.

Tuberculosis (relapsed)

A case of active tuberculosis diagnosed again (bacteriologically, radiologically or clinically) having been considered inactive or quiescent following previous full treatment (as deemed appropriate by the State or Territory Director of Tuberculosis).

Mortality data for tuberculosis, and denominator population data for the calculation of rates, were obtained from the Australian Bureau of Statistics (ABS).[2] Denominator data for age and sex are based on mid-year population estimates for 1998. Resident population by Indigenous status and country of birth were based on estimates of the relevant populations as at 30 June 1998. The classification of countries adhered to the ABS standard classification of countries for social statistics.[2]

Figure 1. Incidence rates for new TB notifications (1948-1998) and crude TB mortality rates (1967-1998) per 100,000 population, Australia



Contents, *continued*

Table 1. Notifications of new and relapsed cases of tuberculosis, and rates per 100,000 population, Australia, 1986 to 1998 by year

	New cases		Relapsed cases		Total cases	
Year	Number	Rate	Number	Rate	Number	Rate
1986	863	5.39	43	0.27	906	5.66
1987	868	5.34	39	0.24	907	5.58
1988	925	5.60	29	0.18	954	5.77
1989	902	5.36	50	0.30	952	5.66
1990	979	5.74	37	0.22	1,016	5.95
1991	903	5.22	47	0.27	950	5.50
1992	983	5.62	28	0.16	1,011	5.78
1993	944	5.35	47	0.27	991	5.61
1994	996	5.58	61	0.34	1,057	5.93
1995	988	5.47	50	0.28	1,038	5.75
1996	983	5.37	54	0.29	1,037	5.66
1997	954	5.15	47	0.25	1,001	5.40
1998	884	4.72	39	0.21	923	4.92

Table 2. Notifications of new and relapsed cases of tuberculosis and rates per 100,000 population, Australia, 1998, by State and Territory

	New cases		Relapsed cases		Total cases	
State/Territory	Number	Rate	Number	Rate	Number	Crude rate
Australian Capital Territory	14	4.54	0	0.00	14	4.54
New South Wales	376	5.93	20	0.32	396	6.24
Northern Territory	30	15.79	1	0.53	31	16.32
Queensland	99	2.86	6	0.17	105	3.04
South Australia	51	3.43	2	0.13	53	3.56
Tasmania	7	1.48	1	0.21	8	1.70
Victoria	234	5.02	6	0.13	240	5.15
Western Australia	73	3.99	3	0.16	76	4.15
Total	884	4.72	39	0.21	923	4.92

Note: Only 5 cases were not residents in the State of notification.

Results

Notification rates – new and relapsed cases

In 1998, 923 cases of active tuberculosis were notified nationally (4.92 per 100,000); of those 884 (96%) were new cases and 39 were relapsed cases (Table 1). The corresponding crude annual incidence rate was 4.72 per 100,000 for new cases and 0.21 per 100,000 for relapsed cases (Figure 1).

Crude incidence rates vary widely between jurisdictions (Table 2) because of high incidence rates in overseas born persons in New South Wales and high incidence rates in Indigenous people in the Northern Territory. Since 1991, rates of TB have been less than 5 per 100,000 in Tasmania, Queensland, South Australia and Western Australia. In the Australian Capital Territory, rates have been less than 5 per 100,000 for all years except 1992 and 1995. The 2 most populous States, Victoria and New South Wales, have reported intermediate rates of between 5 and 8 per 100,000

Table 3. Notifications of new cases of tuberculosis and rates per 100,000 population, Australia, 1998, by age group and sex

Age group (years)	Males		Females		Total	
	Number	Rate	Number	Rate	Number	Rate
0-4	8	1.21	2	0.32	10	0.78
5-9	10	1.47	8	1.24	18	1.36
10-14	4	0.60	7	1.09	11	0.84
15-19	20	2.96	17	2.65	37	2.81
20-24	34	4.90	42	6.29	76	5.58
25-29	45	6.09	69	9.39	114	7.74
30-34	51	7.24	38	5.37	89	6.30
35-39	35	4.69	39	5.20	74	4.95
40-44	32	4.58	37	5.26	69	4.92
45-49	24	3.66	26	4.00	50	3.83
50-54	27	4.56	17	2.98	44	3.79
55-59	19	4.26	13	3.01	32	3.65
60-64	25	6.78	17	4.59	42	5.69
65-69	28	8.40	25	7.18	53	7.77
70-74	18	6.30	18	5.46	38	6.18
75-79	28	13.93	28	10.45	60	12.80
80-84	17	15.45	17	9.45	34	11.73
85+	14	20.21	17	10.74	31	13.62
Unknown	1		1		2	
Total	446	4.78	438	4.65	884	4.72

Table 4. Notifications of new and relapsed cases of tuberculosis in Australia, 1998, by site of disease

Site	New cases	Relapsed cases	Total cases	Total %
Pulmonary	518	29	547	59.3
Pleural	37	0	37	4.0
Lymph nodes	190	7	197	21.3
Bone/Joint	30	0	30	3.3
Genitourinary	41	1	42	4.6
Miliary	4	0	4	0.4
Meningeal	9	0	9	1.0
Peritoneal	16	0	16	1.7
Others	31	2	33	3.6
Unknown	8	0	8	0.8
Total	884	39	923	100

since 1991, and the Northern Territory has reported rates in excess of 15 per 100,000 over the same time period.

Age and sex

In 1998, sex was reported in all cases of notified TB. Information on age was available in over 99 per cent of cases with age data missing for only one male and one female (Table 3). Among the new TB cases males accounted for 446 (51%) and females for 438 (49%) of the notifications. The corresponding incidence rates for new disease in males and females was 4.78 and 4.65 per 100,000 population respectively. Ten (10) new cases of tuberculosis were notified in children under 5 years of age with a corresponding rate of 0.78 per 100,000 population.

For relapsed disease, females accounted for 21 (54%) cases and males for 18 (46%). Twenty-nine (74%) of the notifications were in persons aged over 40 years.

Principal sites of disease

A principal site of disease was reported for all but 8 cases of new TB and all cases of relapsed TB. Of the new cases, 518 (59%) had pulmonary and 190 (21.5%) had nodal disease (Table 4). Thirty-two percent (203 cases) of the new pulmonary cases were smear-positive.

Rates for pulmonary tuberculosis in Australian born persons was 0.9 per 100,000 population compared to overseas born (8.9 per 100,000 population) and Indigenes (5.6/100,000 population). The rate of extra-pulmonary tuberculosis in Australian born persons was 0.3 per 100,000 population compared with 7.2 per 100,000 population in overseas born persons and 3.3 per 100,000 population in Indigenous Australians.

Table 5. Initial drug regimen at time of notification of tuberculosis, Australia, 1998

Drug* regimen	New cases	Relapsed cases	Total
Six-drug regimen			
Iso+rif+pyr+etha+str+eth	1	0	1
Five-drug regimen			
iso+rif+pyr+etha+other	50	2	52
iso+rif+pyr+etha+str	2	1	3
iso+rif+pyr+etha+cyc	1	0	1
iso+rif+pyr+etha+pro	1	0	1
Four-drug regimen			
iso+rif+pyr+etha	644	27	671
iso+rif+pyr+other	8	0	8
iso+pyr+etha+str	1	0	1
iso+rif+etha+cyc	1	0	1
iso+rif+pyr+str	1	0	1
iso+rif+etha+other	1	0	1
rif+pyr+etha+str	1	0	1
Three-drug regimen			
iso+rif+pyr	59	3	62
iso+rif+etha	26	2	28
iso+pyr+etha	14	1	15
iso+etha+other	1	0	1
iso+etha+str	1	0	1
iso+rif+str	1	0	1
rif+pyr+etha	3	1	4
rif+etha+str	1	0	1
rif+pro+cyc	0	1	1
Two-drug regimen			
iso+rif	13	0	13
iso+pyr	2	0	2
iso+etha	2	0	2
etha+other	1	0	1
rif+etha	1	0	1
rif+pyr	1	0	1
Total	838	38	876

* Iso = isoniazid; rif = rifampicin; pyr = pyrazinamide; etha = ethambutol; str = streptomycin; eth = ethionamide; cyc = cycloserine.

Bacilles Calmette-Guérin (BCG) status

BCG vaccination status was not provided for 620 (67%) of the 923 TB notifications, 103 (11%) reported a history of BCG vaccination and 200 (22%) had not received a dose of BCG.

Antimicrobial therapy

The choice of antibiotic regimen started at the time of notification was reported in 876 (95%) cases of TB (Table 5). The most commonly prescribed combination was a four-drug combination of isoniazid, rifampicin, pyrazinamide and ethambutol in 671 (76.6%) cases (644 new cases and 27 relapsed cases). The next most common was the three-drug combination of isoniazid, rifampicin and pyrazinamide in 62 (7%) cases. Overall, a six-drug regimen was started in one (0.1%) case, a five-drug regimen in 57 (6.5%) cases, a four-drug regimen in 684 (78%) cases, a three-drug regimen in 114 (13. %) cases and a two-drug regimen in 20 (2.3%) cases. The reasons why patients were prescribed a two-drug regime included that they were children (3 cases), suspected of TB or preventative treatment (4 cases), medical complications (2 cases) or to complete treatment commenced overseas (1 case). No further information was available for the remaining 10 two-drug regimen treatments.

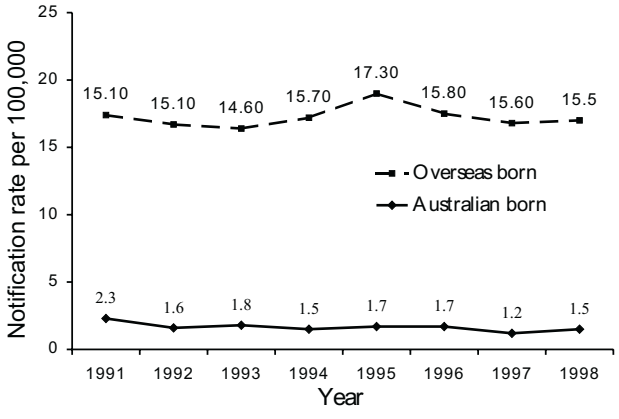
HIV status

HIV status was not provided in 864 (94%) of notified cases of TB. Of the 59 cases in which HIV status was reported, 4 were positive and 55 negative.

Country of birth

Most (77%) TB notifications were in people born overseas (710). The number of new TB cases reported in the Australian and overseas born populations was 204 (23%) and 680 (77%) respectively. The corresponding rate of new TB disease in the Australian and overseas born populations was 1.5 and 15.5 per 100,000 population respectively (Figure 2).

Figure 2. Incidence rates, new disease, in the Australian and overseas born, 1991-1998



The incidence rates of all TB notifications (new and relapsed) per 100,000 overseas born resident populations in Australia are shown in Figure 3. The countries of origin with the highest rates of TB include Vietnam (141 cases; 81.2 per 100,000); Indonesia (42 cases; 73.9 per 100,000); India (61 cases; 64.0 per 100,000); China (52 cases; 34.9 per 100,000); and the Philippines (74 cases; 64.7 per 100,000). Together these countries accounted for 370 (52%) notifications in the overseas born cases. The rates of TB, per 100,000, overseas born resident population in Australia for 1998 are presented together with World Health Organization case incidence rates for TB in the country of origin for the same year (Table 6). In some countries, such as Indonesia, the estimated rates are considered to be higher than those officially reported.

The length of time that overseas born persons had been resident in Australia was reported for 466 (66%) notifications. Of these 90 (19%) had been resident for less than one year 44 (9%) from one to less than 2 years, 58 (12%) from 2 to less than 5 years, 92 (19%) from 5 to less than 10 years and 182 (39%) for 10 years or more.

The age and sex distributions of Australian born and overseas born TB incidence rates are illustrated in Figure 4. The overseas born population show high age-specific rates in both young adults and the elderly, whereas in the Australian born population, there is a gradual increase in age-specific rates with advancing age.

Indigenous status

Indigenous status was reported for 202 (95%) of all notifications for people born in Australia. Indigenous Australians accounted for 39 TB cases in 1998, of which 1 was a relapsed case and 38 were new cases of TB. Twenty-five (64%) notifications of TB in Indigenous Australians were reported from the Northern Territory. The Australian Capital Territory, South Australia, Tasmania, and Victoria reported no Indigenous cases. The annual crude incidence rate of new disease per 100,000 Indigenous

Table 6. Total notifications of tuberculosis, Australia, 1998. Number and estimated rates per 100,000 for selected countries of birth *

Country of birth. Selected countries	New cases	Relapsed cases	Total cases	Estimated population by country of birth	Rate per 100,000 population in Australia, by country of birth	WHO 1998 incidence rate (per 100,000) in country birth
Vietnam	135	6	141	173,549	81.2	112.7
Philippines	68	6	74	114,304	64.7	219.2
India	60	1	61	95,259	64.0	115.0
China	50	2	52	149,101	34.9	36.4
Indonesia	41	1	42	56,798	73.9	19.6
Hong Kong	23	2	25	55,256	45.2	115.2
UK and Ireland	18	2	20	1,168,986	1.7	9.6
Sri Lanka	15	0	15	55,240	27.2	38.1
Italy	13	0	13	247,519	5.3	10.0
Greece	12	0	12	140,955	8.5	10.2
Former Yugoslavia	10	1	11	203,488	5.4	28.5
New Zealand	7	0	7	342,705	2.0	9.7
Turkey	6	0	6	31,428	19.1	34.9
Malaysia	6	0	6	89,527	6.7	65.9
Poland	6	0	6	70,639	8.5	34.4
Fiji	4	0	4	38,889	10.3	20.9
Singapore	4	0	4	28,772	13.9	61.0
Germany	4	0	4	122,690	3.3	12.7
USSR	3	0	3	55,344	5.4	82.4
Total born overseas	680	30	710	4,383,760	16.2	
Australian born	204	9	213	14,364,044	1.5	
Total	884	39	923	18,747,804	4.9	4.9

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

Figure 3. Incidence rates, by country of birth, per 100,000 resident population in Australia, 1998

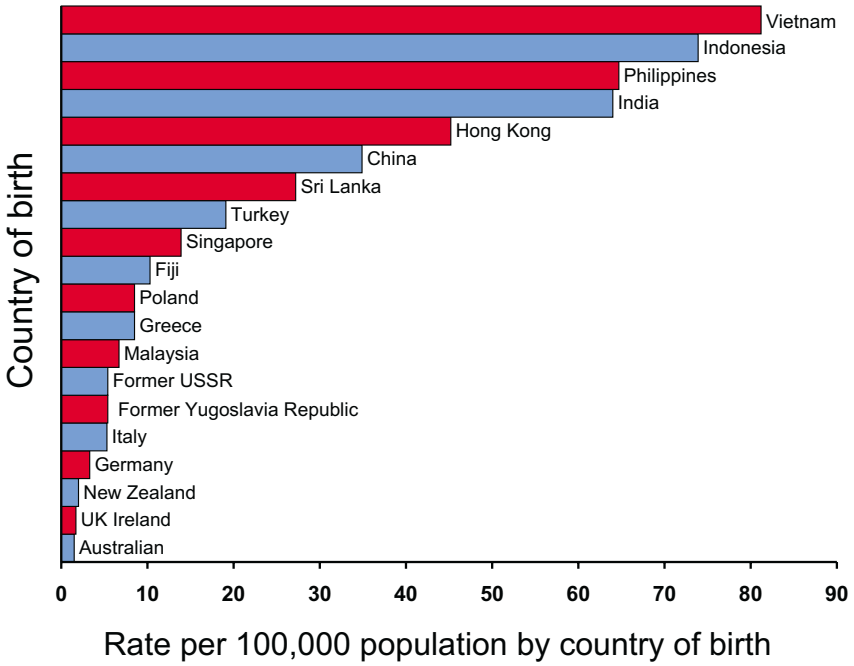


Figure 4. Age specific TB incidence rates in Australian born and overseas born individuals per 100,000 resident population

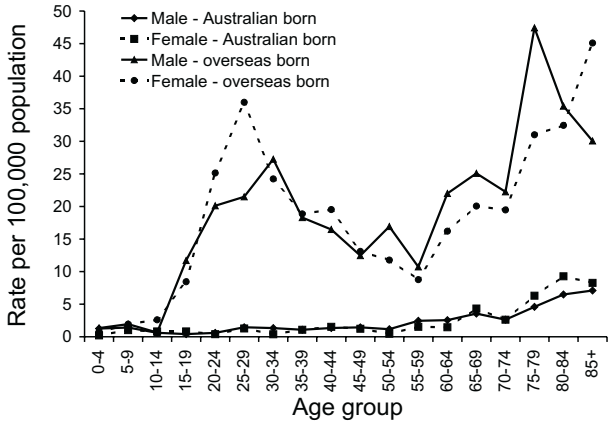
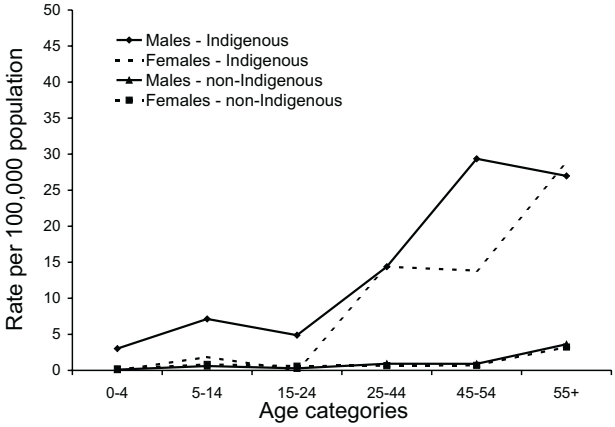


Figure 5. Age specific TB incidence rates in Indigenous Australian born and non-Indigenous Australian born



population was between 8.8 and 9.4 based on upper and lower Indigenous population estimates for the year. Relapse rates were 0.23 and 0.25 per 100,000 population based on the same estimates. The comparative TB rate of new disease in the Australian born, non-Indigenous population was 1.2 per 100,000 population.

Twenty-two notifications were in males and 16 in females. Six (16%) of the Indigenous notifications were aged over 60 years, and 6 cases were aged less than 14 years.

The age and sex incidence rates for Indigenous and non-Indigenous Australian born persons are illustrated in Figure 5. Both show increase in age-specific rates with advancing age, with the Indigenous rates being up to thirty times higher in boys under the age of 5 years and in men aged 45-55 years compared with those of their non-Indigenous counterparts.

Mortality

In 1998, the Australian Bureau of Statistics[3] reported 62 deaths for which TB was the underlying cause. The crude mortality rate was 0.33 per 100,000, which is the same as the lowest rate for TB in 30 years reported in 1997 (0.33 per 100,000 population). Of these deaths, 41 (66%) were in males and 21 in females. Fifty-four (89%) occurred in persons over the age of 60 years, and 2 TB deaths were registered in persons under 40 years of age (both males; one in the 10 to 14 year age group and one in the 20 to 24 year age group).

Discussion

Australia continues to report one of the lowest TB rates in the world. Other developed countries that have reported

rates of 5 per 100,000 or less in 1998 include Sweden, Malta and Norway.[1] From 1986 to 1997 annual crude incidence rates for TB in Australia stabilised at between 5 and 6 per 100,000[4-10] and in 1998 dropped below 5 per 100,000 population.

Over half (52%) of all TB notifications in the overseas born in 1998 were from India, Indonesia, China, the Philippines and Vietnam. WHO has indicated that these 5 countries account for more than 52 per cent of all new TB cases notified annually throughout the world.[1]

While the proportion of overseas born cases represented in annual TB notifications has increased over the last decade, the rates of TB have not. In 1986, 60 per cent of all annual notifications were overseas born, compared to 70 per cent in 1990, 75 per cent in 1996 and 77 per cent in 1998.[4-10] For all years, with the exception of 1995, rates in the overseas born have been between 15 and 17 per 100,000. In the Australian born population there has been a decline in the proportion of all TB notifications as well as a progressive decline in incidence rates, from 2.8 per 100,000 in 1986 to a low of 1.5 per 100,000 in 1998.

Over the last 7 years, rates of TB have been 10 to 15 fold higher in Indigenous Australians compared with the non-Indigenous, Australian born population.[4-10] Reporting accurately on trends in this group has been made difficult by the shifts in the census denominator estimates for this population, and also because of the inconsistent reporting of Indigenous status by some jurisdictions. Among the risk factors for TB in Indigenous Australians are poor socioeconomic status, diabetes, renal disease, smoking, alcohol abuse, and poor nutrition.[11]

There are few indications that the global TB threat is abating, which reinforces the need for all nations to remain vigilant. Having a surveillance system in place that can accurately report on trends and important changes in the epidemiology of TB alerts public health authorities and policy makers to emerging problems and facilitates appropriate action.

Acknowledgments

The members of the Communicable Diseases Network Australia New Zealand, together with the State and Territory Directors of Tuberculosis and other Health Department personnel in the States and Territories who are involved in compiling the individual data sets, are thanked for their cooperation with this surveillance initiative. Special thanks is offered to Louise Carter and Joyce Della in the Australian Capital Territory, Rob Menzies and Mohammed Habib in New South Wales, Lyn Barclay and Mary Verus in the Northern Territory, Patrick Derhy in Queensland, Richard Stapledon and Kylie Van Roekel in South Australia, David Coleman in Tasmania, Martyn Kirk and Trevor Lauer in Victoria, and Sing Pang, Gary Dowse and Jag Atrie in Western Australia. In addition, a note of appreciation is extended to the many physicians and medical practitioners and nurses who contribute to the collection of data relating to TB.

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Changes to the Editorial Team

A member of the Editorial team with major input into the Communicable Diseases Surveillance section of, and various other reports in, *CDI* over the last 14 months (Ms Margo Eyeson-Annan) has left to join the Epidemiology and Surveillance Branch of NSW Health. We wish Margo well in her new position and thank her for her expert contribution to *CDI* in her time in the Department. We welcome Dr Paul Roche and Ms Kelly Collison to the team while Dr Ming Lin and Mr Mark Bullock take a few months' leave. Dr Jenny Thomson, a regular contributor to *CDI*, has re-located to the Melbourne Office of the Commonwealth Department of Health and Aged Care and we wish her well in her endeavours there.

A primary school outbreak of pharyngoconjunctival fever caused by adenovirus type 3

Dave Harley,[1] Bruce Harrower,[2] Michael Lyon,[2] Alan Dick[1]

Abstract

High rates of absenteeism in a North Queensland primary school, due to eye irritation, fever, headache, and stomach pain, were reported to the Tropical Public Health Unit in October 2000. Subsequent investigation demonstrated that the symptoms were due to adenovirus infection. Symptoms were consistent with a diagnosis of pharyngoconjunctival fever. At the height of the outbreak, about 40 per cent of students were absent. There was a strong association between the development of symptoms, and having been swimming on a recent school camp. Adenovirus could not be isolated from swimming pool water from the resort where the camp had been held. However, when inspected the swimming pool was not adequately chlorinated or maintained. It is probable that adenovirus infection was transmitted via swimming pool water at the school camp, and the outbreak might have been avoided by higher standards of swimming pool maintenance. *Commun Dis Intell* 2001;25:9-12.

Keywords: adenovirus, pharyngoconjunctival fever, school, swimming pool

Introduction

Adenovirus infections of humans are ubiquitous, and with other less common manifestations, cause coryza and pharyngitis in infants, upper respiratory infections, pharyngoconjunctival fever, diarrhoea and haemorrhagic cystitis in children, acute respiratory disease and pneumonia in young adults, and epidemic keratoconjunctivitis in adults. There are 47 known serotypes of human adenoviruses. No clear role in causing human disease is known for over half of these; for those that do cause disease the serotype of the virus and age of the host are important in determining clinical presentation.[1] Acute respiratory disease in military recruits, mostly caused by adenovirus type 4 and less often adenovirus type 7, can cause considerable morbidity.[2] In children adenovirus type 7 may cause community outbreaks, with respiratory symptoms and fever, sometimes causing severe disease and possibly death.[3]

Pharyngoconjunctival fever is a syndrome of pharyngitis, conjunctivitis, fever,[2] rhinitis and cervical adenitis.[1] It usually affects children and young adults, and is most commonly caused by serotype 3 or 7, although others including serotypes 1, 4 and 14 have also been implicated.[2] The syndrome may occur sporadically and can cause outbreaks in families and other closed populations, but it is best recognised as causing outbreaks centred around 'summer camps', especially those with swimming pools and small lakes.[2] In a large outbreak of 80 cases acquired during a swimming contest in Greece in 1995, adenovirus was detected in swimming pool water using PCR thus demonstrating the role of swimming pool water as a source of infection.[4]

We describe an outbreak of pharyngoconjunctival fever due to adenovirus type 3 that occurred in north Queensland after a school camp during October 2000.

Materials and methods

The Tropical Public Health Unit was contacted by the principal of a north Queensland primary school on 19 October 2000, because of concern regarding absenteeism caused by an outbreak of eye irritation, fever, headache and stomach pain. Subsequently, 3 visits were made to the school, on 20 October, 1 November, and 8 November 2000. These were to review students clinically and collect viral culture specimens and acute sera, to collect convalescent sera, and to complete a questionnaire on symptomatology and for a case-control study, respectively. Samples were collected from the swimming pool suspected of being the source of the infection on 9 November 2000.

One of us (DH) reviewed the students clinically. A history was taken and examination performed. Throat and eye swabs were collected. Virus isolation was performed as follows: samples were inoculated into MRC-5, LLC-MK2, BGMK, RD and MDCK cells in Dulbecco's MEM. All cultures were passaged every 7 days for 3 weeks. After each passage cells were screened for respiratory viruses by fluorescent antibody tests using the Bartels Respiratory Kit. The isolates were typed by DNA Restriction Analysis.[5] Serological testing was performed using complement fixation. Swimming pool water was tested for adenovirus by using hollow-fibre ultrafiltration and nested RT-PCR.[6,7]

Students completed a questionnaire on symptoms, attendance at the camp and activities while there. The data were then entered into an Access database, subsequent analysis performed using the Statistical Package for the Social Sciences (SPSS), and graphs produced using Excel. Data on absenteeism were also collected from the school rolls. Data were only studied for those days when complete rolls were available: information on preschool absenteeism

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was not analysed because these students do not attend school every day.

Results

Description of the school and the school camp

The school is located on the Atherton Tablelands, approximately 50km north-west of Cairns. At the time of the outbreak 53 students were enrolled at the school (Table 1).

Children at the school were aged between 4 and 12, with a median of 9, years. Twenty-eight (53%) were female.

Between 11 and 13 October 2000, children from the school attended a camp at a coastal resort in Far North Queensland. The camp was at an educational facility with a large saltwater swimming pool. Only children from grades 3 to 7 were able to attend the school camp, and of the 36 children in these grades, 30 attended the camp.

Clinical case descriptions and laboratory investigations

Seven children, all of whom had attended the school camp, were reviewed on 20 October 2000, and symptom information was supplemented from a questionnaire completed on 8 November 2000 (Table 2). Physical findings are not shown, but when these were measured the children had fever and tachycardia. One 12-year-old female (F, 12y; Table 2) had obvious conjunctivitis of the left eye and one 12-year-old male (M2, 12y; Table 2) cervical lymphadenopathy. None had physical signs of lower respiratory tract infection. Cardiovascular examination was normal in all, and none had hepato- or splenomegaly. None had a rash. The joints of one 9-year-old child (F, 9y; Table 2) who complained of arthralgia were examined, but no objective signs of arthritis were found. Three of 4 children who had paired sera collected showed a diagnostic CF titre rise to adenovirus. Adenovirus 3 was isolated by MRC-5 culture from all of 2 eye swabs and 4 throat swabs collected.

Table 1. Students by grade, adenovirus 3 outbreak, north Queensland, 2000

Grade	Number of students
Preschool	5
Year 1	7
Year 2	5
Year 3	3
Year 4	10
Year 5	9
Year 6	6
Year 7	8
Total	53

The outbreak

The greatest rate of absenteeism among the 48 students in grades 1 to 7 occurred on 20 October 2000, when nearly 40 per cent were absent. The highest rates of absenteeism for students who had been on the school camp were on 19 and 20 October 2000. For those who had not been on the school camp peak absenteeism, just under 40 per cent, occurred on 31 October 2000 and 3 November 2000 (Figure 1). The mean (range) number of days absent for the period shown in Figure 1 was 2.9 (0-9), 3.1 (0-11), and 3.0 (0-11) days, for those who did and those who did not attend the camp, and grades 1 to 7, respectively.

Table 2. Symptoms and laboratory findings, adenovirus 3 outbreak, north Queensland, 2000

Gender and age (years)	Onset date (Oct)	Symptoms	CF titres		Viral culture results (collected 20 October)
			20 October	1 November	
F, 8y	16	Sore throat, fever, diarrhoea, headache, nausea, anorexia, jaw ache	-	-	Not done
M, 9y	18	Sore throat, fever, diarrhoea, headache, vomiting, anorexia	< 8	< 8	Throat and left eye swabs. Adenovirus 3 detected
M1, 12y	18	Sore throat, fever, diarrhoea, headache, vomiting	< 8	> 512	Throat swab. Adenovirus 3 detected
F, 9y	17	Sore throat, fever, rhinorrhoea, otalgia, headache, nausea, vomiting, anorexia, back pain, muscle and joint pain	-	-	Not done
M2, 12y	17	Itchy, red eyes, sore throat, fever, rhinorrhoea, diarrhoea, headache, vomiting, anorexia	< 8	128	Throat swab. Adenovirus 3 detected
M, 11y	18	Itchy, red eyes, sore throat, fever, rhinorrhoea, diarrhoea, otalgia, headache, nausea, vomiting, anorexia, myalgia, slight cough	< 8	> 512	Throat swab. Adenovirus 3 detected
F, 12y	17	Itchy, red eyes, fever, headache, nausea	-	-	Left eye swab. Adenovirus 3 detected

Figure 1. School absenteeism, adenovirus type 3 outbreak, north Queensland, 2000

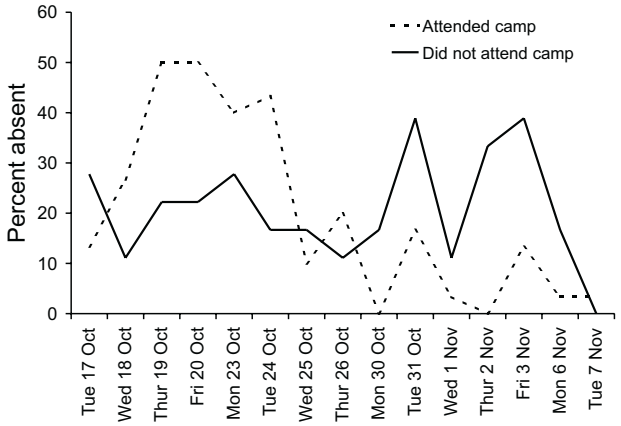
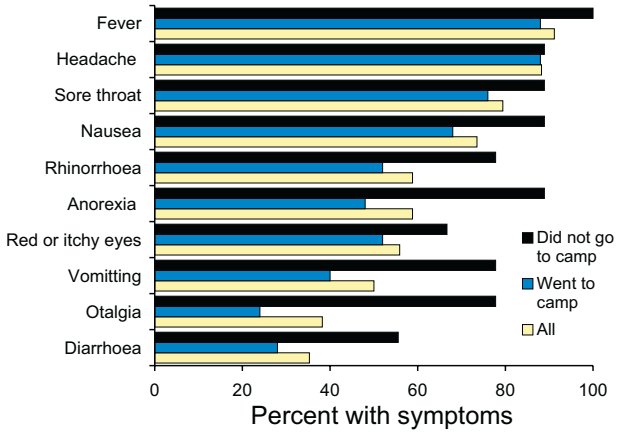


Figure 2. Symptoms, adenovirus type 3 outbreak, north Queensland, 2000



Thirty-four students were unwell in the period 17 to 23 October 2000, suggesting an incubation period of 6 to 9 days following the school camp;[2] of these, 25 (74%) had been on the school camp. Assuming primary cases were acquired at the camp, the other 9 cases were presumably secondary cases, and may have been acquired within households from older siblings who had attended the camp. Fever, headache and sore throat were the commonest symptoms among these 34 students (Figure 2).

There were only 5 students who stated they became unwell after the incubation period as defined above; 4 had a fever, 4 a headache, 3 a sore throat, 4 nausea, 3 rhinorrhoea, 1 anorexia, 3 red or itchy eyes, 1 vomiting, 3 otalgia and 1 diarrhoea.

The likelihood of having been unwell within the incubation period (i.e. 6 to 9 days[2]) was significantly increased for students who had been swimming at the school camp (odds ratio = 9.0, 95% confidence interval: 2.4-33.9). The likelihood of having one or more of red, itchy or watery eyes, fever, or a sore throat was also significantly increased (odds ratio = 5.5, 95% confidence interval: 1.6-18.4).

A one-hundred-litre sample of swimming pool water collected on 9 November 2000 was tested by PCR for adenovirus DNA; none was detected. The residual chlorine concentration when the pool was sampled at approximately 11.00 am, was 1.0 ppm. Faecal coliforms were cultured from swabs taken from among algae on the steps of the swimming pool.

Discussion

The study demonstrates that pharyngoconjunctival fever caused by adenovirus 3 was the cause of a primary school outbreak that resulted in high rates of absenteeism in October 2000. While not conclusive, there is a strong link between the resort education centre swimming pool that the school had recently used, and pharyngoconjunctival fever. The later peak of absenteeism for students who had not attended the camp suggests that secondary cases were acquired after the return of students from the camp, either within households or the school community. The degree of morbidity caused was considerable, and the rate of absenteeism resulted in appreciable disruption to the school routine.

While adenovirus was not isolated from the swimming pool water, there was evidence that the pool was not appropriately maintained. For a commercial swimming pool in this geographical location an inadequate chlorine residual of 1.0 ppm was being used, and this was only being checked once per day in the cool of the morning. It is likely that on a hot day, with a large number of swimmers, all the available chlorine in the pool would be exhausted within 2 hours of testing. Because of the extreme and prolonged heat that can be experienced in Far North Queensland, and also the dilution caused by high rainfall, 2.0 ppm chlorine residual would be appropriate. These levels should be checked at least 5 times per day to ensure the desired levels are being maintained.

Faecal coliforms were detected in patches of algae on the swimming pool steps, suggesting the pool was not being adequately cleaned. While no legislation currently exists to enforce water quality and operational standards of Queensland swimming pools, recommended standards are available.[8] Transmission of adenovirus via swimming pool water, probably as a result of inadequate chlorination, has been demonstrated in another outbreak[4] and the evidence presented here points to the swimming pool as the source of infection. It is likely that the outbreak could have been avoided through better standards of swimming pool maintenance.

Acknowledgments

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Measles outbreak among young adults in Victoria

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Abstract

An outbreak of laboratory confirmed measles involving 30 young adults and an infant is reported. Of the young adult cases, 17 (57%) were hospitalised. After a trip to India, the primary case returned to Sydney early in January 2001 and then visited Melbourne infecting several individuals. Secondary spread appears to have occurred at a nightclub. On the basis of RNA typing the measles strain involved is of genotype 'D'. *Commun Dis Intell* 25;2001:12.

Keywords: measles, vaccine, surveillance, overseas travel

The Communicable Diseases Section at the Victorian Department of Human Services was advised in mid-afternoon 1 February 2001 of a suspected case of measles in a 19-year-old female who had been admitted to the Royal Melbourne Hospital. The following day 2 more cases (a 28-year-old male and a 22-year-old male) were reported. As of 16 February 2001, 31 laboratory-confirmed cases had been identified. All but one of these (a 10-month-old female) were aged between 15 and 34 years. The previous measles outbreak in Victoria affected a similar age group.[1]

Young Australian adults (born between January 1970 and December 1983) are at greater risk of measles infection because either they have not had a wild measles virus infection or have not been adequately immunised. Measles-containing vaccines were first available in Australia in 1968. The two-dose Measles-Mumps-Rubella vaccination program was introduced in 1994, so very few of those within the affected age group are likely to have received two doses of a measles-containing vaccine.[2] As with the 1999 outbreak, the hospitalisation rate among this group has been high. Seventeen cases (57%) have been hospitalised.

The primary case in the current outbreak was a 19-year-old male with no documented history of previous vaccination who had returned to Sydney on 4 January 2001 after holidaying in India. He visited Melbourne from 17 to 20 January 2001 during his infectious period. Onset of rash in the primary case was 20 January. Onset of rash in the remaining cases was between 28 January and 13 February. Eight cases have been directly linked to the primary case including the 10-month-old child who had attended the same restaurant on the same night as the primary case. The 3 most recent cases (rash onset 9 February to 13 February 2001) appear to represent the next wave of transmission; 2 of these are known to have attended the same nightclub on the same night.

The Victorian Infectious Diseases Reference Laboratory has harvested measles virus RNA from 13 of the cases. Initial genotyping of the virus strain places it within genotype "D"; subsequent analysis will allow more specific genotyping. Virus culture in B95a cells has yielded at least 5 isolates and is ongoing.

We encourage both clinicians and laboratories to continue to notify on suspicion of measles, and NOT to delay notification pending laboratory confirmation of the diagnosis.

Acknowledgments

We gratefully acknowledge the cooperation we have received in this investigation from the patients, nursing staff, clinicians and pathology collection centres. In particular, we wish to thank staff at the Victorian Infectious Diseases Reference Laboratory, our phlebotomist, Debbie Gercovich, and Patrick Maywood, NSW Health.

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Varicella vaccine in post-exposure prophylaxis

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Abstract

Evidence concerning the effectiveness of Oka-based varicella vaccines when administered following exposure to varicella zoster virus in domestic and hospital settings is reviewed. The evidence appears to support post-exposure use of Oka-derived varicella vaccines in children within 3 days of rash onset in the index case. Despite vaccination, a small proportion will develop mild, but infectious, chickenpox, especially if they have been exposed in the household setting. Controlled studies of post-exposure prophylaxis in adults using both Varilrix and Varivax II are still needed. The applicability of this approach to disease control in health care facilities and in community settings warrants wider discussion. *Commun Dis Intell* 2001;25:13-15.

Keywords: varicella, zoster, chickenpox, post-exposure prophylaxis, vaccine

Background

Two live, attenuated, varicella vaccines derived from the Oka strain of varicella-zoster virus (VZV) Varilrix (Glaxo Smith-Kline Beecham) and Varivax II (Merck/CSL) received Australian marketing approval during 2000. The indication for their use is the prevention of chickenpox in healthy individuals of 12 months of age or older. This paper reviews evidence concerning the effectiveness of Oka-based vaccines when administered following exposure to VZV. Use of the vaccines in Australia for post-exposure prophylaxis is not currently approved by the National Health and Medical Research Council; this is reflected in the 2000 edition of the *Australian Immunisation Handbook* and approved product information.

Theoretical basis for post-exposure prophylaxis

The development and use of Oka-based vaccines was first reported by Takahashi and colleagues in Japan in 1974.[1] Since that time several reports have referred to post-exposure prophylaxis in hospital or household settings using experimental or production lots of vaccine of varying composition and infectivity/virus dose. The theoretical basis for post-exposure prophylaxis relates to the ability of Oka-derived vaccines to induce cell-mediated and antibody responses within 5 to 7 days[2,3] and the relatively prolonged incubation period of 10 to 18 days of primary VZV infection, ie chickenpox. The pathogenesis of chickenpox follows the general scheme for viral exanthems proposed by Fenner.[4] Respiratory tract inoculation allows initial viral replication in regional lymph nodes. Primary viraemia leads to replication in the liver and spleen, and secondary viraemia disseminates virus (within infected mononuclear cells) to various organs including the skin leading to the characteristic rash. An immune response mounted prior to the secondary viraemia may abort or ameliorate clinical disease.

Post-exposure prophylaxis in the hospital setting

In their original report, Takahashi and colleagues described 23 children seronegative by complement fixation titre who received vaccine with 500 plaque forming units (PFU) of infectious virus 'immediately' (not otherwise defined) after diagnosis of the index case. Two children developed low-grade fever and a mild vesicular rash attributed to the vaccine.[1]

Katsushima and colleagues first reported in 1982 administration of experimental vaccines containing between 250 and 3,000 PFU to children with no history of chickenpox after hospital exposure and extended their findings in a later report. None of 149 children receiving 250-3,000 PFU within 3 days of exposure and none of 15 who received 1000 PFU at 100 hours (ie 4 days, rather than 5) became ill.[5,6]

A further Japanese report found that 41 of 46 child contacts given 'emergency vaccination' with 300-2,000 PFU were protected from disease, but gave no details of timing.[7]

Post-exposure prophylaxis in the household setting

Asano and colleagues reported a controlled trial using experimental vaccines of varying infectious doses in which none of 17 seronegative children vaccinated within 3 days of onset of chickenpox rash in a sibling developed illness compared with 19 of 19 unvaccinated household contacts who developed chickenpox.[8] The same group reported their experience with 43 child contacts in the absence of controls: 4 of 10 given 300-600 PFU, but all of 30 given 800-15,000 PFU within 3 days of household exposure were protected, whilst none of 3 given 1,500-4,000 PFU at 5 days were protected (no subjects were vaccinated at 4 days).[9] Naganuma and colleagues also found that 30 of 40 sibling contacts were protected by 'emergency vaccination' with 300-2,000 PFU, but details of vaccination timing were not provided.[7]

An extensive US dose-ranging study incorporated a small randomised double blind, placebo-controlled trial of post-exposure prophylaxis using a pre-production 4,350 PFU Oka-Merck vaccine.[10] Of 13 placebo recipients, 12 developed chickenpox. In the active group, only one of 10 vaccinated within 3 days of exposure developed mild chickenpox

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(20 lesions) whilst all 3 vaccinated at 4 to 5 days developed mild disease.[10]

Only 2 English-language reports evaluating standard production lots of vaccine for this purpose have been published, both US studies of the licensed Oka/Merck vaccine containing no less than 1350 PFU. In the first, 10 children without a history of chickenpox were vaccinated within 3 days of rash onset in a sibling; of these, 5 developed mild illness with 5 to 83 skin lesions and 5 remained well.[11]

In the most recent study, residents of a women's refuge with a negative chickenpox history were offered vaccination within 3 days of onset of rash in a mother and her child. The authors argued that this setting was equivalent to a household. None of 25 adults and only 2 of 42 children developed illness. However, the 2 children with chickenpox were members of the family of (and were housed in the same room as) the index case and both developed a brief, afebrile illness characterised by fewer than 20 lesions.[12]

Summary of results

The studies described above lend credence to the view that post-exposure prophylaxis with Oka-derived varicella vaccine is feasible. In relation to the hospital setting, one uncontrolled study of experimental vaccine batches suggested that vaccination within 3 days (and possibly within 4 days) prevented illness.[6]

More, and more recent, data are available regarding vaccination of children following household exposure. A controlled study demonstrated that early, Japanese experimental vaccine batches were protective when given within 3 days of exposure,[8] as did a double-blind, randomised, control trial of Oka/Merck vaccine containing three times the infectious virus of the current vaccines.[10] The only 2 studies using licensed vaccine, both uncontrolled, showed protection against moderate-to-severe disease, but some occurrence of mild disease (mostly fewer than 20 lesions) following household exposure.[11,12]

Reliable data are not available in relation to contact with zoster, a far less infectious condition than chickenpox, although in theory prompt post-exposure prophylaxis should also offer protection.

There is minimal and conflicting evidence regarding protection when vaccine is received 4 to 5 days after exposure.[6,9,10] In interpreting the data, it should be borne in mind that all of the Japanese studies refer to days after exposure, whilst the US studies refer to days after onset of lesions. However, it is probable that all studies counted the first day of exposure as the day of onset of the varicella rash. Variation in vaccination effectiveness between studies may be explained by the more intense exposure in the household setting and the greater likelihood of exposure to the index case in the 2 days before rash onset.

Caveats and discussion

Scientific issues affecting the applicability of these studies include the small numbers of subjects, variety of vaccine formulations and infectivity, and paucity of data relevant to current commercial vaccines (and absence of data relating to the 2,000 PFU Varilrix vaccine). Most studies used inadequate assessment of susceptibility, based on history; a negative history, especially in adults,[13] is poorly predictive of VZV susceptibility. The study in the women's refuge found that no vaccinated adult contacts developed chickenpox,[12] but it is likely that the majority were immune, and in the absence of a control group the true effect of vaccination cannot be determined.

Subjects in all other studies were children, rather than adolescents or adults in whom 2 doses of varicella vaccine are recommended for reliable pre-exposure protection. Thus, the feasibility and effectiveness of post-exposure prophylaxis in those aged 13 years and older is unknown.

The final consideration is the interpretation of, and response to, the individual who develops a papular or vesicular rash following post-exposure varicella vaccination. A mild rash occurs 7 to 28 days after vaccination in approximately 5 per cent of recipients, so that a rash developing within a week of vaccination is most likely to be a result of natural infection. However, the individual is considered potentially infectious whether the rash is vaccine-induced or associated with natural VZV infection, and should be isolated from non-immune contacts in accordance with accepted practice for the particular setting. Vesicular fluid should be collected and sent to a virology laboratory for virus strain identification by polymerase chain reaction.

Conclusions

Theoretical considerations and experimental evidence appear to support the post-exposure use in children of Oka-derived varicella vaccines within 3 days of rash onset in the index case. Despite vaccination, a small proportion will develop mild, but infectious, chickenpox, especially if they have been exposed in the household setting. Controlled studies of post-exposure prophylaxis in adults using both Varilrix and Varivax II are still needed, whilst the applicability of this approach to disease control in health care facilities and in community settings warrants wider discussion.

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Onshore catering increases the risk of diarrhoeal illness amongst cruise ship passengers

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Abstract

Of 134 Queensland passengers on a cruise, 91 (67.9%) people reported various illnesses including 41 (30.6%) who reported diarrhoeal symptoms. Queensland passengers who ate while onshore at non-Australian ports were significantly more at risk of developing diarrhoeal symptoms than those who did not. Passengers were particularly at risk when they ate onshore while undertaking a tour compared with those who did not undertake this tour. Travellers should be warned of the possibility of contracting diarrhoeal illness from onshore catering. *Commun Dis Intell* 2001;25:15-17.

Keywords: diarrhoea, onshore catering, cruise ship, food-borne

Introduction

In May 1999, passengers and crew aboard a cruise ship visiting various non-Australian ports reported a range of gastrointestinal, respiratory or febrile symptoms. Of the 949 passengers, 138 (14.5%) were from Queensland. Although 12 people from other States were diagnosed with typhoid fever (CDN report),[1] there were no confirmed cases amongst the Queensland passengers. Possible associations between specific exposures and subsequent illness were investigated by the Queensland Health Department. We describe the findings relating specifically to reports of diarrhoeal illness.

Methods

A list of cruise passengers resident in Queensland was obtained. All were contacted and invited to take part in the study. For passengers aged less than 12 years, the respondent was a parent who accompanied them on the cruise. A questionnaire administered by telephone in June 1999 sought information on disease symptoms before and after joining the cruise, contacts with other ill passengers, activities both on-board and onshore, and food and water consumption. Respondents reporting ongoing symptoms at the time of interview were advised to seek medical attention. Those with ongoing diarrhoeal symptoms were advised to provide at least 2 stools for laboratory examination to exclude the possibility that they posed a risk to others. Dates of symptom onset and duration were also collected. We defined self-reported diarrhoeal illness as diarrhoea +/- other symptoms reported (Table 1).

Queensland Health was not able to assess environmental conditions on board ship. A subset of passengers was asked to report on sanitation, swimming pools, and overall hygiene.

Epi Info version 6.04b (CDC)[2] was used for data entry and analyses. Associations between exposures of interest and gastrointestinal symptoms were calculated using relative risk analyses. Either uncorrected ^{2} or two-tailed Fisher exact p values were used where appropriate.

Results

Of the 138 Queensland passengers, 134 (97.1%) were contacted and agreed to take part in the study. Sixty-eight (50.7%) were male. The median age was 58 years; ages ranged from 1 to 92 years. The cruise commenced in Australia on 8 May 1999 (Port 1; see Figure).

Diarrhoeal illness

There were no reports of diarrhoea prior to boarding ship. Forty-one (30.6%) respondents (Table 1) reported diarrhoeal symptoms during and after the cruise.

The first reported onset of diarrhoea and vomiting in the Queensland group was on 13 May, one day after boarding at Port 2 (Figure). Four further cases with diarrhoeal symptoms occurred on 14 May at Port 3, outside Australian waters.

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Table 1. Forty-one cruise passengers experiencing diarrhoeal symptoms

Symptoms*	Number	Frequency (%)
Diarrhoea +/- other gastrointestinal symptoms*	25	61
Diarrhoea + gastrointestinal* and other symptoms†	16	39

* Gastrointestinal symptoms were abdominal pain, vomiting, nausea, lethargy, fever, and headache.

† Other symptoms were ear, nose, cough and throat symptoms.

Subsequently, the ship docked at 5 more non-Australian ports and endured a cyclone (22 to 23 May 1999).

Passengers who ate onshore at non-Australian ports or during the onshore tour were more than three times more likely to report a subsequent diarrhoeal illness than passengers who went onshore at one or more ports but who did not eat at any of them. (Relative risk 3.6; 95% CI 1.8 – 7.3; p < 0.0001;Table 2). This includes passengers who ate during an onshore tour from Port 3 on day 7. These passengers were more than four times more likely to report subsequent diarrhoeal illness after this tour compared with those who did not participate. (Relative risk 4.6 95% CI 3.1 – 6.8; p < 0.0001;Table 2).

Only one passenger who reported diarrhoeal symptoms subsequent to participating in the tour did not eat the food provided during this or at any other onshore venue. His symptoms developed 13 days after this tour and 5 days after he complained of sanitation problems in his cabin following the cyclone.

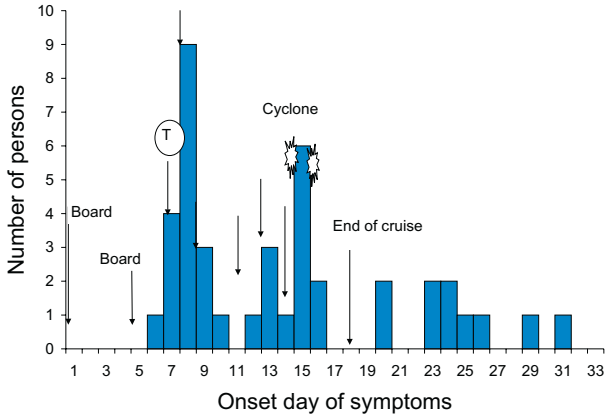
Laboratory investigations

Ten passengers who had ongoing gastrointestinal symptoms at the time of interview were advised to provide a stool for faecal examination for parasitic and bacterial pathogens. No reports of significant isolates were received.

Environmental conditions

A subset of 62 /134 (46.3%) passengers was asked about sanitation on board the ship. Of this subset, 32 (51.6%) said toilets in their own cabins and toilets on the decks that they had used were overflowing and failed to flush during the cruise. During the cyclone on days 15 and 16 of the cruise, 18 of these 32 passengers also reported that toilets in their own cabins overflowed, there were smells in their cabins

Figure. Onset of diarrhoea amongst Queensland cruise-ship passengers, 13 May to 7 June 1999



* Day 1–start of cruise 8 May 1999. Day 18– cruise ended 25 May 1999. Day 33 – interviews commenced 9 June 1999. Arrows indicate ports. T = onshore tour as well as disembarkation at this port. Cyclone occurred 22-23 May 1999.

and that they suspected sewage to have seeped onto the carpets in their cabins.

Of the 32 passengers who said they had experienced sanitation problems, 17 (53.1%) reported diarrhoeal symptoms during the cruise compared with 6 of the 30 (20%) passengers who said they had not experienced any sanitation problems on the cruise (Relative risk 2.7; 95% CI 1.2-5.8; p = 0.007). Although 3 passengers reported symptoms before they reported problems with their own cabin toilets during the cyclone, it is unclear whether they and others who complained were exposed to sanitation problems prior to developing their symptoms. However, all but 3 of those who reported diarrhoeal symptoms and all but 2 who did not report diarrhoeal symptoms also reported other possible risk factors, namely eating onshore at ports including the tour, contact with other sick people and/or swimming.

Eighteen (13.4%) reported swimming on board the ship as well as onshore. Of these, 6 (33.3%) reported diarrhoeal symptoms, but most (4/6) also reported other exposures.

Fifteen (11.2%) reported having close contact with passengers who they knew were ill on the cruise. This includes 7 who said this contact was prior to developing

Table 2. Diarrhoeal illness in Queensland cruise ship passengers

Activity	Exposed: ill/total	Unexposed: ill/total	Relative risk	95% CI	P value
Ate onshore at non-Australian ports or during the tour*	27/54	8/58†	3.6	1.8 – 7.3	<0.0001
Ate during an onshore tour at Port 3‡	14/15	23/113§	4.6	3.1 – 6.8	<0.0001

* Missing data on 22 passengers who did not report or who could not recall when they developed the diarrhoeal symptoms they reported and/ or who could not recall whether they ate onshore or where they ate onshore.

† Did not eat or drink when they were onshore at any non-Australian port (Ports 3 to8; Figure) or during the onshore tour during this cruise.

‡ Missing data on 5 passengers who did not report or who could not recall when they developed the diarrhoeal symptoms they reported and/ or who could not recall whether they participated in this particular tour.

§ Did not participate in this tour

symptoms themselves. However, most (5/7) also reported exposure to other risk factors such as eating at ports or during the onshore tour, and experiencing sanitation problems and swimming. Only one case reported symptoms prior to engaging in any identified exposures.

Discussion

Risk factors identified by our study for diarrhoeal symptoms on this cruise included eating or drinking onshore at ports (especially during an onshore tour) and experiencing problems with on-board sanitation. However, most passengers reported exposure to a number of risk factors and so results must be interpreted with caution. The onshore tour occurred at the beginning of the cruise prior to the advent of other activities. However, most passengers reported participating in a range of activities as the cruise progressed so that it was not possible to determine if any individual port was independently associated with symptoms.

Passengers suffered a range of symptoms on this cruise, although, apart from the *Salmonella typhi* isolates, the clinical and microbiological diagnoses were undetermined (viral cultures were not performed). Some of those interviewed reported more severe symptoms than those reported by others. This raises the possibility that agents other than *S. typhi* were also involved. Those who complained of vomiting and nausea alone could have been suffering from seasickness. A recent paper describes a similar occurrence of concurrent outbreaks of gastroenteritis and typhoid fever on a floating restaurant.[3] This paper also illustrates the difficulties in determining whether 2 syndromes had the same aetiology.[3]

We found that interviewers did not consistently ask about swimming in pools on board and onshore, and not all activities provided during the cruise were identified. We are therefore unable to draw any inference about a possible association between swimming and diarrhoeal illness. Additionally, we are unable to determine the role of person to person transmission because these data were incomplete including information on the temporal relationship between such exposure and symptoms. This cohort of Queenslanders was part of the larger cohort of all cruise passengers and crew, and the effect of exposures on these individuals and their activities are undetermined.

The use of onshore caterers for off-ship excursions has previously been reported as a risk factor for gastrointestinal illness amongst cruise ship passengers.[4] A cruise-associated diarrhoeal disease outbreak was defined as the occurrence of diarrhoeal symptoms among at least 3 per cent of passengers on a cruise.[4]

Reports of gastrointestinal symptoms aboard cruise ships are common.[5,6,7,8] Identified risk factors include inadequate treatment of water aboard ship and the need for strict hygienic control for food handlers and food preparation areas. This study was unable to investigate such risks.

Limitations of this study

More than 75 per cent of the passengers were interviewed more than 2 weeks and up to a month after the onset of their symptoms. Those who were ill may have been more likely to recall problems with sanitation and attribute their symptoms to particular events, especially as legal proceedings had already commenced by the time of interview. Few cases had ongoing symptoms when interviewed and many had taken antibiotics. Environmental investigations of the ship were not possible. Anecdotal reports regarding illness in the ship's crew did not indicate whether this related to food handlers. Lack of consistency of approach amongst interviewers resulted in less detailed data in some instances.

Conclusions

It has been estimated that one third of outbreaks on cruises might be prevented if onshore caterers are not used for off-ship excursions.[4] We support this conclusion, although the results of our study need to be interpreted with caution, as we cannot exclude person to person transmission, on-board sanitation and other activities as being responsible for at least some of the illness. Standard public health messages regarding care in food consumption outside Australia[9] should be reinforced.

Acknowledgments

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Communicable Diseases Surveillance

Presentation of NNDSS data

In the March 2000 issue an additional summary table was introduced. Table 1 presents 'date of notification' data, which is a composite of three components: (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 unit. Table 2 presents the crude incidence of diseases by State or Territory for the current reporting month. Table 3 presents data by report date for information only. In Table 3 the report date is the date the public health unit received the report.

Table 1 now includes the following summary columns: total current month 2000 data; the totals for previous month 2000 and corresponding month 1999; a 5-year mean which is calculated using previous, corresponding and following month data for the previous 5 years (*MMWR Morb Mortal Wkly Rep*, 2000:49;139-146); year to date (YTD) figures; the mean for the year to date figures for the previous 5 years; and the ratio of the current month to the mean of the last 5 years.

Highlights for December, 2000

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 who have recently formed a Data Management Network. This additional information has enabled the reporting of more informative highlights each month.*

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia New Zealand, and the CDI *Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme. In this report, data from the NNDSS are referred to as 'notifications' or 'cases', whereas those from ASPREN are referred to as 'consultations' or 'encounters' while data from the LabVISE scheme are referred to as 'laboratory reports'.*

In December 2000, compared with the 5-year mean, reports of incident hepatitis C (ratio 1.3), chlamydial infection (1.2), Barmah Forest virus (1.1) and meningococcal infection (ratio 1.5) increased (Figure 1, Table 1).

Gastrointestinal illness

Campylobacter notifications fell to 885 cases in December from 1299 cases in the previous month with a rate of 83.7/100,000 population but overall the trend in notifications is increasing. *Salmonella* notifications were lower than in the previous month with a rate of 32.1/100,000 population. Tasmania had the highest rate for *Campylobacter* (148/100,000 population) and the Northern Territory the highest for *Salmonella* (178/100,000 population).

Five cases of typhoid have been reported, four males in the 15 to 44 years age group and a 40-year-old female: four acquired the disease overseas, two from India, one from Indonesia (see Dr David Peacock's additional report, p32) and one from an unreported country. The New South Wales case had no history of overseas travel.

Shiga-toxin producing *Escherichia coli* was detected in South Australia in a 79-year-old male.

There were 4 cases of haemolytic uranic syndrome in December, two male children aged 1 year and 2 years from Queensland and Western Australia respectively, and two females aged 58 years and 83 years both from New South Wales.

Chlamydial infection

There were 1056 notifications of chlamydial infection in December 2000, a notification rate of 66.1/100,000 population – an increase from previous years. Of these cases, 84 per cent were in the 15 to 34 years age range; the male:female ratio was 0.7:1. The Northern Territory continues to have the highest rate for chlamydial infection (337.7/100,000 population).

Vaccine preventable diseases

Apart from one report of tetanus, vaccine preventable disease reports for December were below those for the previous month and for the 5-year mean. The tetanus case, a 17-year-old male, was reported from New South Wales: his immunisation status was unknown.

Pertussis notifications were down compared with the previous month (397 cases with a rate of 24.9/100,000 population compared with 622 cases with a rate of 39/100,000 population). Since August 2000 (when the national rate for the year peaked at 46/100,000 population) the rates for all States except the Northern Territory have decreased. The rate for the Northern Territory increased from the previous month (18.7 per 100,000 population) to 24.9 per 100,000 population in December 2000.

Measles cases continue to be at their lowest level since the national notification system began. Five cases were reported in December 2000, all in New South Wales (see: Outbreak Report, p19). They included four partially vaccinated children (two boys aged 1 and 5 years and two

girls aged 3 and 4 years) and a 31-year-old unimmunised male.

Of the *Haemophilus influenza* type b cases, three were children (a 1-year-old girl, and two boys aged 3 and 6 years) and one a 16-year-old female. The 16-year-old had not been immunised. The immunisation status of the 3-year-old was not stated and that of the other two cases unknown.

Legionellosis

There were 17 notifications of legionellosis in December 2000, a notification rate of 1.1/100,000 population. One case in Queensland was *Legionella pneumophila* and two in South Australia *Legionella longbeachae*. For the other cases information on the *Legionella* species involved was not available.

Meningococcal infections

There were 48 notifications of meningococcal infection in December 2000, a notification rate of 3.0/100,000 population. Of these cases, 40 per cent were under 5 years of age and 31 per cent were in the 5 to 19 years age group. The serogroups were available for 19 cases; these were serogroup B (68%) and serogroup C (32%).

Vectorborne diseases

In December there were fewer Barmah Forest virus infection notifications compared with November (51 cases with a rate of 3.2/100,000 population compared with 66 cases with a rate of 4.1/100,000 population) but these were slightly higher than the 5-year mean (48 cases). The majority (27 cases, 9.1/100,000 population) were from Queensland.

Ross River Virus notifications remain unchanged (159 cases with a rate of 10.0/100,000 population compared with November 2000, 164 cases with a rate of 10.3/100,000 population) the highest rate being in the Northern Territory (104.4/100,000 population). Most cases were reported from Queensland (62) and South Australia (51).

Outbreak report: measles cluster in south-eastern Sydney with transmission in a general practice waiting room.

Contributed by the Infectious Diseases Team and Director,
South Eastern Sydney Public Health Unit, Zetland, NSW

South Eastern Sydney Public Health Unit recently investigated a cluster of 5 cases of measles with onset in December 2000. The index case was a 31-year-old male who presented to his doctor with a rash and fever. The diagnosis was confirmed by a positive measles IgM assay at a private pathology laboratory. The illness was thought to have been acquired through occupational exposure in another health area. The second and third cases were siblings aged 18 months and 3 years who were thought to have become infected after a few minutes' contact with the index case in the waiting room of a local general practice. Measles was subsequently transmitted to 2 other children, aged 5 and 4 years, who were childcare contacts of the second case. All 4 affected children had documentation of one MMR vaccination at 1 year of age whilst the 5-year-old had documentation of a second MMR vaccine dose at 4 to 5 years of age. In addition to the index case, laboratory confirmation was obtained for 3 of the 4 subsequent cases by the Serology and Virology Laboratories, SEALS Randwick. In 2 cases, measles IgM was detected, and in 2 cases measles antigen was detected in throat swabs by direct immunofluorescence.

Tables

There were 5,585 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date in December 2000 (Table 1). The crude incidence of diseases per 100,000 population for each State or Territory (Table 2) was included for the first time in the August 2000 issue of *Commun Dis Intell*. Data by date of report for December 2000, are included in this issue of *Commun Dis Intell* (Table 3). Figure 1 illustrates, for selected diseases, the December 2000 totals as ratios to the mean of their November to January levels for the previous 5 years.

There were 1,247 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 to 31 December 2000 (Tables 4 and 5).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 48 to 52, ending 31 December 2000, are included in this issue of *Commun Dis Intell* (Table 6).

Table 1. Notifications of diseases received by State and Territory health authorities in the period 1 to 31 December 2000, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total December 2000[1]	Total November 2000[1]	Total December 1999[1]	Last 5 years mean	Year to date 2000	Last 5 years YTD mean	Ratio*
Bloodborne															
Hepatitis B (incident)	0	3	0	6	0	0	10	4	23	35	31	23	413	276	1.0
Hepatitis B (unspecified)[2]	2	200	0	66	12	1	162	40	483	791	537	525	8,631	6,833	0.9
Hepatitis C (incident)	1	5	0	-	12	0	4	4	26	27	30	20	494	205	1.3
Hepatitis C (unspecified)[2]	11	455	13	216	46	14	390	88	1,233	1,641	1,453	1,332	20,672	15,841	0.9
Hepatitis D	0	1	0	0	0	0	1	0	2	10	1	1	27	20	1.8
Gastrointestinal															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0.0
Campylobacterosis[3]	25	-	21	262	133	58	275	111	885	1,299	983	1,188	13,480	12,220	0.7
Haemolytic uraemic syndrome	0	2	0	1	0	0	0	1	4	1	1	1	14	n/a	-
Hepatitis A	0	8	1	11	1	0	11	4	36	44	102	168	811	2,165	0.2
Hepatitis E	0	0	0	0	0	0	0	NN	0	0	0	0	1	4	0.0
Listeriosis	0	5	0	0	2	0	0	0	7	3	5	6	66	64	1.2
Salmonellosis	4	135	29	143	37	13	97	55	513	532	534	622	6,081	6,714	0.8
Shigellosis[3]	1	-	6	8	3	0	22	4	44	38	36	55	488	669	0.8
SLTEC,VTEC[4]	0	0	0	NN	1	0	0	NN	1	1	6	3	33	n/a	-
Typhoid	0	1	1	0	0	0	1	2	5	3	7	7	65	74	0.7
Yersiniosis[3]	0	-	0	4	0	0	2	0	6	3	7	20	73	232	0.3
Quarantinable															
Cholera	0	0	0	0	0	0	0	0	0	0	0	0	1	4	-
Plague	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	-
Viral haemorrhagic fever	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	-
Sexually transmissible															
Chancroid	0	0	0	0	0	0	0	0	0	0	0	0	0	1	-
Chlamydial infection[5]	16	236	55	291	151	8	151	148	1,056	1,656	1,009	864	17,718	9,897	1.2
Donovanosis	0	0	0	0	NN	0	0	0	0	0	0	4	12	45	0.0
Gonococcal infection[6]	3	55	73	67	11	0	47	42	298	415	416	408	6,047	4,649	0.7
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Syphilis[7]	1	38	7	36	2	0	0	5	89	187	107	120	1,932	1,666	0.7

Table 1 continued. Notifications of diseases received by State and Territory health authorities in the period 1 to 31 December 2000, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total December 2000[1]	Total November 2000[1]	Total December 1999[1]	Last 5 years mean	Year to date 2000	Last 5 years YTD mean	Ratio*
Vaccine preventable															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Haemophilus influenzae type b	0	1	1	1	0	0	0	1	4	6	3	4	32	51	1.0
Measles	0	5	0	0	0	0	0	0	5	10	4	39	109	610	0.1
Mumps	0	2	1	0	2	0	3	1	9	12	14	13	208	168	0.7
Pertussis	6	224	4	54	52	1	50	6	397	622	424	665	5,772	5,942	0.6
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	-
Rubella[8]	0	14	0	2	1	0	4	1	22	51	17	193	314	1,931	0.1
Tetanus	0	1	0	0	0	0	0	0	1	0	0	1	8	5	-
Vectorborne															
Arbovirus infection NEC	0	0	0	0	0	0	0	0	0	0	0	5	64	56	0.0
Barmah Forest virus infection	0	10	2	27	2	0	5	5	51	66	43	48	622	698	1.1
Dengue	0	0	0	0	1	0	0	0	1	3	26	31	210	195	0.0
Malaria	2	11	2	9	5	0	8	4	41	47	65	59	951	724	0.7
Ross River virus infection	1	9	17	62	51	0	12	7	159	164	290	337	4,137	4,946	0.5
Zoonoses															
Brucellosis	0	0	0	2	0	0	0	0	2	4	3	4	26	40	0.5
Hydatid infection	0	NN	0	1	0	0	2	0	3	3	4	3	27	45	0.9
Leptospirosis	0	5	3	7	1	0	2	0	18	22	20	18	244	204	1.0
Ornithosis	0	NN	0	NN	0	0	8	0	8	14	7	10	98	91	0.8
Q fever	0	6	0	22	1	0	2	0	31	50	45	43	519	537	0.7
Other															
Legionellosis	0	0	0	1	2	0	12	2	17	34	16	18	469	207	0.9
Leprosy	0	0	0	0	0	0	0	0	0	1	1	1	4	8	0.0
Meningococcal infection	0	15	2	8	4	2	15	2	48	52	46	32	601	461	1.5
Tuberculosis	1	24	0	1	0	0	29	2	57	68	92	90	960	1,058	0.6
Total	74	1,471	238	1,308	533	97	1,325	539	5,585	7,915	6,385	6,755	92,436	79,558	

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. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
5. WA: genital only.
6. NT, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.
7. Includes congenital syphilis.
8. Includes congenital rubella

\# Date of notification = a composite of three components: (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 unit.

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

* Ratio = ratio of current month total to mean of last 5 years calculated as described above.

n/a Not calculated as only notifiable for under 5 years.

Table 2. Crude incidence of diseases by State or Territory, 1 to 31 December 2000. (Rate per 100,000 population)

Disease[1]	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Bloodborne									
Hepatitis B (incident)	0.0	0.6	0.0	2.0	0.0	0.0	2.5	2.5	1.4
Hepatitis B (unspecified)[2]	7.6	37.1	0.0	22.2	9.6	2.6	40.8	25.5	30.3
Hepatitis C (incident)	3.8	0.9	0.0	-	9.6	0.0	1.0	2.5	2.0
Hepatitis C (unspecified)[2]	42.0	84.5	79.8	72.7	36.9	35.7	98.2	56.1	77.2
Hepatitis D	0.0	0.2	0.0	0.0	0.0	0.0	0.3	0.0	0.1
Gastrointestinal									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacterosis[3]	95.5	-	128.9	88.2	106.6	148.0	69.2	70.7	83.7
Haemolytic uraemic syndrome	0.0	0.4	0.0	0.3	0.0	0.0	0.0	0.6	0.3
Hepatitis A	0.0	1.5	6.1	3.7	0.8	0.0	2.8	2.5	2.3
Hepatitis E	0.0	0.0	0.0	0.0	0.0	0.0	0.0	NN	0.0
Listeriosis	0.0	0.9	0.0	0.0	1.6	0.0	0.0	0.0	0.4
Salmonellosis	15.3	25.1	178.0	48.1	29.6	33.2	24.4	35.0	32.1
Shigellosis[3]	3.8	-	36.8	2.7	2.4	0.0	5.5	2.5	4.2
SLTEC,VTEC[4]	0.0	0.0	0.0	NN	0.8	0.0	0.0	NN	0.1
Typhoid	0.0	0.2	6.1	0.0	0.0	0.0	0.3	1.3	0.3
Yersiniosis[3]	0.0	-	0.0	1.3	0.0	0.0	0.5	0.0	0.6
Quarantinable									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
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									
Chancroid	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Chlamydial infection[5]	61.1	43.8	337.7	97.9	121.0	20.4	38.0	94.3	66.1
Donovanosis	0.0	0.0	0.0	0.0	NN	0.0	0.0	0.0	0.0
Gonococcal infection[6]	11.5	10.2	448.2	22.5	8.8	0.0	11.8	26.8	18.7
Lymphogranuloma venereum	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis[7]	3.8	7.1	43.0	12.1	1.6	0.0	0.0	3.2	5.6
Vaccine preventable									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.2	6.1	0.3	0.0	0.0	0.0	0.6	0.3
Measles	0.0	0.9	0.0	0.0	0.0	0.0	0.0	0.0	0.3
Mumps	0.0	0.4	6.1	0.0	1.6	0.0	0.8	0.6	0.6
Pertussis	22.9	41.6	24.6	18.2	41.7	2.6	12.6	3.8	24.9
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella[8]	0.0	2.6	0.0	0.7	0.8	0.0	1.0	0.6	1.4
Tetanus	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1
Vectorborne									
Arbovirus infection NEC	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Barmah Forest virus infection	0.0	1.9	12.3	9.1	1.6	0.0	1.3	3.2	3.2
Dengue	0.0	0.0	0.0	0.0	0.8	0.0	0.0	0.0	0.1
Malaria	7.6	2.0	12.3	3.0	4.0	0.0	2.0	2.5	2.6
Ross River virus infection	3.8	1.7	104.4	20.9	40.9	0.0	3.0	4.5	10.0

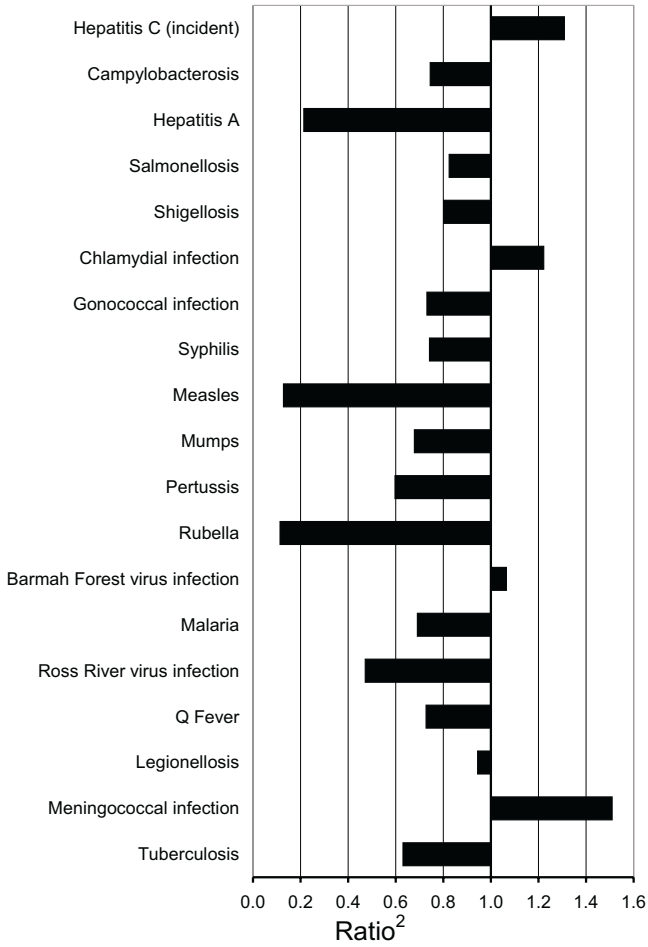
Table 2 (continued). Crude incidence of diseases by State or Territory, 1 to 31 December 2000. (Rate per 100,000 population)

Disease[1]	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Zoonoses									
Brucellosis	0.0	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.1
Hydatid infection	0.0	NN	0.0	0.3	0.0	0.0	0.5	0.0	0.3
Leptospirosis	0.0	0.9	18.4	2.4	0.8	0.0	0.5	0.0	1.1
Ornithosis	0.0	NN	0.0	NN	0.0	0.0	2.0	0.0	1.1
Q fever	0.0	1.1	0.0	7.4	0.8	0.0	0.5	0.0	1.9
Other									
Legionellosis	0.0	0.0	0.0	0.3	1.6	0.0	3.0	1.3	1.1
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection	0.0	2.8	12.3	2.7	3.2	5.1	3.8	1.3	3.0
Tuberculosis	3.8	4.5	0.0	0.3	0.0	0.0	7.3	1.3	3.6
Total	282.8	273.1	1,461.1	440.1	427.1	247.5	333.6	343.3	349.8

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. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
5. WA: genital only.
6. NT, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.
7. Includes congenital syphilis.
8. Includes congenital rubella.

NN Not Notifiable.
NEC Not Elsewhere Classified.
\- Elsewhere Classified.

Figure 1. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 to 31 December 2000 with historical data[2]



1. Selected diseases are chosen each calendar month according to current activity
2. Ratio of current month total to mean of November to January data for the previous five years

Table 3. Notifications of diseases received by State and Territory health authorities in the period 1 to 31 December 2000, by date of report*

Disease[1]	State or Territory								Total this period	Year to date total
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Bloodborne										
Hepatitis B (incident)	0	1	1	6	2	0	11	5	26	418
Hepatitis B (unspecified)[2]	2	260	0	74	16	1	163	48	564	8,831
Hepatitis C (incident)	1	7	0	-	10	0	1	5	24	503
Hepatitis C (unspecified)[2]	9	622	15	245	48	19	387	128	1,473	20,929
Hepatitis D	0	1	0	0	0	0	1	0	2	27
Gastrointestinal										
Botulism	0	0	0	0	0	0	0	0	0	2
Campylobacterosis[3]	22	-	12	230	141	49	293	148	895	13,518
Haemolytic uraemic syndrome	0	0	0	0	1	0	0	1	2	11
Hepatitis A	0	6	2	9	1	0	14	6	38	838
Hepatitis E	0	0	0	0	0	0	0	NN	0	1
Listeriosis	0	3	0	0	2	0	0	0	5	65
Salmonellosis	4	146	34	108	28	16	97	76	509	6,130
Shigellosis[3]	1	-	8	7	3	0	27	7	53	488
SLTEC,VTEC[4]	0	0	0	NN	2	0	0	NN	2	36
Typhoid	0	2	0	0	0	0	3	2	7	68
Yersiniosis[3]	0	-	0	4	0	0	2	0	6	74
Quarantinable										
Cholera	0	0	0	0	0	0	0	0	0	1
Plague	0	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0
Yellow fever	0	0	0	0	0	0	0	0	0	0
Sexually transmissible										
Chancroid	0	0	0	0	0	0	0	0	0	0
Chlamydial infection[5]	16	303	64	340	157	13	205	199	1,297	17,765
Donovanosis	0	0	0	0	NN	0	0	0	0	13
Gonococcal infection[6]	3	67	59	59	18	0	48	65	319	6,088
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0
Syphilis[7]	1	45	5	58	1	0	1	7	118	1,990
Vaccine preventable										
Diphtheria	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	0	2	1	0	0	0	1	4	32
Measles	0	6	0	0	0	0	0	0	6	110
Mumps	0	1	1	0	2	0	4	1	9	211
Pertussis	5	350	3	47	101	1	57	17	581	5,876
Poliomyelitis	0	0	0	0	0	0	0	0	0	0
Rubella[8]	0	16	0	4	2	0	5	2	29	315
Tetanus	0	1	0	0	0	0	0	0	1	9
Vectorborne										
Arbovirus infection NEC	0	0	0	0	0	0	0	0	0	65
Barmah Forest virus infection	0	12	1	35	2	0	4	8	62	633
Dengue	0	0	0	1	0	0	0	0	1	231
Malaria	0	10	3	15	4	0	10	6	48	974
Ross River virus infection	1	11	6	53	59	0	12	16	158	4,288

Table 3 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 to 31 December 2000, by date of report*

Disease[1]	State or Territory								Total this period	Year to date total
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Zoonoses										
Brucellosis	0	0	0	4	0	0	0	0	4	26
Hydatid infection	0	NN	0	0	0	0	3	0	3	28
Leptospirosis	0	2	2	3	2	0	6	0	15	245
Ornithosis	0	NN	0	NN	0	1	16	0	17	104
Q fever	0	6	0	21	0	0	6	0	33	536
Other										
Legionellosis	0	4	1	2	3	0	22	2	34	474
Leprosy	0	0	0	0	0	0	0	0	0	5
Meningococcal infection	0	20	2	5	4	3	13	4	51	603
Tuberculosis	3	37	2	3	0	0	30	7	82	1,051
Total	68	1,939	223	1,334	609	103	1,441	761	6,478	93,613

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. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with Shiga-like toxin (verotoxin) producing *E. coli* (SLTEC/VTEC).
5. WA: genital only.
6. NT, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.
7. Includes congenital syphilis.
8. Includes congenital rubella.

* Date of report is the date the public health unit received the report.

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 4. Virology and serology laboratory reports by contributing laboratories for the reporting period 1 to 31 December 2000[1]

State or Territory	Laboratory	This period	Total this period[2]
Australian Capital Territory	The Canberra Hospital	-	-
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	71	72
	New Children's Hospital, Westmead	36	81
New South Wales	Repatriation General Hospital, Concord	-	-
	Royal Prince Alfred Hospital, Camperdown	28	21
	South West Area Pathology Service, Liverpool	-	152
Queensland	Queensland Medical Laboratory, West End	330	973
	Townsville General Hospital	15	20
South Australia	Institute of Medical and Veterinary Science, Adelaide	497	1,042
Tasmania	Northern Tasmanian Pathology Service, Launceston	12	11
	Royal Hobart Hospital, Hobart	-	-
Victoria	Monash Medical Centre, Melbourne	-	-
	Royal Children's Hospital, Melbourne	89	117
	Victorian Infectious Diseases Reference Laboratory, Fairfield	110	209
Western Australia	PathCentre Virology, Perth	-	-
	Princess Margaret Hospital, Perth	32	38
	Western Diagnostic Pathology	27	-
Total		1,247	2,736

1. The complete list of laboratories reporting for the 12 months, January to December 2000, will appear in every report from January 2000 regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.
2. Total reports include both reports for the current period and outstanding reports to date.

\- Nil reports

Table 5. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 31 December 2000, and total reports for the year[2]

	State or Territory[1]								This period 2000	This period 1999	Year to date 2000[3]	Year to date 1999
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	1	-	-	-	1	-	-	-	2	3	44	172
Rubella virus	-	2	-	1	2	-	-	-	5	5	47	145
Hepatitis viruses												
Hepatitis A virus	-	-	-	2	1	-	-	-	3	24	137	375
Hepatitis D virus	-	1	-	-	-	-	-	-	1	1	9	8
Arboviruses												
Ross River virus	-	1	8	11	35	-	-	3	58	129	1,250	1,423
Barmah Forest virus	-	1	1	10	2	-	-	-	14	22	158	180
Adenoviruses												
Adenovirus not typed/pending	1	8	-	6	44	-	9	5	73	122	982	1,128
Herpes viruses												
Cytomegalovirus	-	14	-	6	46	-	16	4	86	145	1,262	1,216
Varicella-zoster virus	2	8	2	65	22	-	21	2	122	174	1,296	1,658
Epstein-Barr virus	-	3	3	32	51	-	14	14	117	205	1,852	2,194
Other DNA viruses												
Parvovirus	-	1	2	20	12	-	1	-	36	25	350	437
Picornavirus family												
Coxsackievirus B1	-	-	-	-	1	-	2	-	3		4	1
Coxsackievirus B4	-	-	-	-	-	-	4	-	4	2	15	3
Coxsackievirus B5	-	-	-	-	-	-	1	-	1		5	7
Poliovirus type 1 (uncharacterised)	-	2	-	-	-	-	-	-	2		18	26
Poliovirus type 2 (uncharacterised)	-	1	-	-	-	-	-	-	1	2	8	16
Rhinovirus (all types)	2	21	-	-	4	-	-	-	27	51	376	501
Enterovirus type 71 (BCR)	-	1	-	-	-	-	-	-	1	4	2	15
Enterovirus not typed/pending	-	1	-	4	1	3	19	-	28	48	711	752
Ortho/paramyxoviruses												
Influenza A virus	2	4	-	-	26	-	2	3	37	70	1,366	1,898
Influenza B virus	2	-	-	1	12	-	-	1	16	4	550	279
Parainfluenza virus type 1	-	-	-	-	1	-	-	-	1	6	230	44
Parainfluenza virus type 3	-	14	-	10	19	-	1	8	52	62	459	803
Respiratory syncytial virus	-	5	-	7	16	-	-	1	29	77	2,689	3,054
Other RNA viruses												
Rotavirus	-	8	-	-	52	4	14	8	86	167	1,720	2,244
Norwalk agent	-	-	-	-	1	-	16	-	17	4	75	59
Other												
Chlamydia trachomatis not typed	4	31	7	48	55	1	8	1	155	322	2,855	3,289
Chlamydia psittaci	-	-	-	-	-	-	7	-	7	5	100	78
Mycoplasma pneumoniae	-	1	1	17	17	2	21	5	64	92	646	1,125
Coxiella burnetii (Q fever)	-	-	-	7	-	-	3	-	10	14	90	221
Rickettsia - Spotted fever group	-	-	-	-	-	4	-	-	4		6	1
Streptococcus group A	-	2	3	11	-	-	2	-	18	47	348	368
Yersinia enterocolitica	-	1	-	-	-	-	-	-	1		14	10
Bordetella pertussis	-	8	-	11	27	-	30	-	76	76	665	845

Table 5 (continued). Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 31 December 2000, and total reports for the year[2]

	State or Territory[1]								This period 2000	This period 1999	Year to date 2000[3]	Year to date 1999
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Legionella pneumophila	-	-	-	-	-	-	3	-	3		42	17
Legionella longbeachae	-	-	-	-	1	-	-	-	1	12	53	51
Cryptococcus species	-	-	-	-	3	-	-	-	3		18	9
Leptospira species	-	-	-	5	2	-	-	-	7	4	59	55
Treponema pallidum	-	2	14	16	44	-	-	-	76	89	855	774
Total	14	141	41	290	498	14	194	55	1,247	2,013	21,366	25,481

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 6. Australian Sentinel Practice Research Network reports, weeks 48 to 52, 2000*

Week number	48		49		50		51	
Week ending on	3 December 2000		10 December 2000		17 December 2000		24 December 2000	
Doctors reporting	56		52		56		52	
Total encounters	6,484		6,709		6,877		6,274	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	13	2.0	9	1.3	18	2.6	4	0.6
Chickenpox	16	2.5	11	1.6	9	1.3	5	0.8
Gastroenteritis	56	8.6	55	8.2	61	8.9	72	11.5
Gastroenteritis with stool culture	7	1. 1	9	1.3	11	1.6	7	1.1
ADT immunisations	28	4.3	25	3.7	26	3.8	23	3.7

* **Editorial note:** For week 52 ending 31 December 2000 there were 4 reports of influenza and 7 of chickenpox. The other conditions are no longer reported.

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. The system coordinates the national surveillance of close to 50 communicable diseases or disease groups endorsed by the Communicable Diseases Network Australia New Zealand 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 legislations. De-identified core unit data are supplied fortnightly for collation, analysis and dissemination. For further information, see Commun Dis Intell *2000;24:6-7.*

LabVISE is a sentinel reporting scheme. Currently 17 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 *monthly. 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 *2000;24:10.*

ASPREN currently comprises about 120 general practitioners from throughout the country, not all of whom report each week. Between 7,000 and 8,000 consultations are reported each week, with special attention to 14 conditions chosen for sentinel surveillance in 2000. Communicable Diseases Intelligence *reports the consultation rates for five of these. For further information, including case definitions, see* Commun Dis Intell *2000;24:7-8.*

Additional Reports

Australian encephalitis: Chicken Surveillance Programme

Sentinel chicken flocks are used to monitor flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin which cause the potentially fatal disease Australian encephalitis in humans. Currently 29 flocks are maintained in the north of Western Australia, eight in the Northern Territory, nine in New South Wales and ten in Victoria. The flocks in Western Australia and the Northern Territory are tested year round but those in New South Wales and Victoria are tested only from November to March, during the main risk season.

Results are coordinated by the Arbovirus Laboratory in Perth and reported bimonthly. For more information and details of the location of sentinel chicken sites see Commun Dis Intell *2000;24:8-9.*

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2. Veterinary Research Institute, Victoria
3. Virology Department, Westmead Hospital, New South Wales
4. Department of Microbiology, The University of Queensland
5. Berrimah Agricultural Research Centre, Northern Territory
6. PathCentre, Western Australia
7. Department of Health and Community Services, Northern Territory

Sentinel chicken serology was carried out for 27 of the 29 flocks in Western Australia in November and December 2000. There were no seroconversions to flaviviruses in November. However, 10 seroconversions to flaviviruses were detected in December, 5 from Derby (Curtin Airbase) in the Kimberley 5 from Ophthalmia dam near Newman in the Pilbara. At Derby, 3 of the chickens were positive for MVE antibodies, 1 for MVE and Kunjin antibodies and 1 for Kunjin alone. All 5 of the seroconversions detected at the dam near Newman were to MVE virus. This activity was unusual as it occurred at the beginning of the wet season. As a result of these findings the Health Department of Western Australia issued a health warning to residents living in these areas warning of the increased risk of infection with MVE virus.

A new sentinel chicken flock was established at Gapuwiyak (north-east of Darwin) in the Northern Territory in November 2000. Serum samples from all of the 8 Northern Territory sentinel chicken flocks were tested in our laboratory in November and December 2000. There were no seroconversions to flaviviruses during this period. The October seroconversion to MVE virus at Beatrice Hill Farm, east of Darwin, was confirmed in the November bleed.

The MVE sentinel chicken surveillance programs in New South Wales and Victoria re-commenced in November 2000. Serum samples were tested in November and December 2000 but no flavivirus antibodies were detected.

Rotavirus Surveillance

The National Rotavirus Reference Centre (NRRC) undertakes surveillance and characterisation of rotavirus strains causing annual epidemics of severe diarrhoea in young children throughout Australia.

There are currently fourteen laboratories contributing data and rotavirus specimens for the characterisation of representative rotavirus serotypes.

The NRRC is happy to give and receive notifications of rotavirus outbreaks Australia-wide. The NRRC can be contacted at the Murdoch Childrens Research Institute, Department of Gastroenterology and Clinical Nutrition, Royal Children's Hospital, Flemington Road, Parkville, Victoria 3052. Telephone: (03) 9345 5069, Facsimile: (03) 9345 6240,
Email: masendyp@cryptic.rch.unimelb.edu.au. For more information see Commun Dis Intell *2000;24:10.*

June to November, 2000

Rotavirus specimens have been received from Northern West Western Australia, Perth, Adelaide, Hobart, Melbourne, West Sydney, Brisbane, Townsville, Alice Springs, Gove and Darwin for the time period 1 June to 30 November 2000.

Over 600 specimens have been included in a monoclonal antibody based serotyping enzyme immunoassay (EIA). The results show that G9 rotaviruses, which were the second most important serotype in 1999/2000, were not as widespread in the first part of the 2000/2001 period. They were detected only in Brisbane, Sydney and Melbourne, having been previously identified in most other centres. The epidemiological significance of G9 rotaviruses in Australia remains unclear and further monitoring is warranted.

Serotype G2 viruses appeared in most of the centres studied, including Perth, Adelaide, Melbourne, Hobart, West Sydney, Gove and Darwin. Molecular techniques including reverse transcriptase/PCR and Northern hybridisation were required for the detection of serotype G2 viruses because the currently used G2 serotyping monoclonal antibody (MAb) derived in 1986 did not recognise these strains. Sequence analysis of the circulating G2 strains identified an amino acid change at the same position where variant viruses unreactive with this MAb also showed an amino acid substitution. This change in the virus may have implications for future vaccine development.

The collection period June to December 2000 was notable for the first 'sighting' of serotype G4 rotaviruses in the Northern Territory's 'Top End'. These G4 viruses were first detected from a child in Darwin in August 2000 and subsequently from children on Elcho Island in East Arnhem Land. Children from 10 communities in East Arnhem Land were admitted to the Gove District Hospital in Nhulunbuy and were all found to be serotype G4. Further analysis by RNA polyacrylamide gel electrophoresis and reverse transcriptase/PCR showed the children were all infected with the same epidemic strain. The outbreak was confined to the top end (as at 11 January, 2001) and appeared to be limited to the period August to October 2000.

Rotavirus collection continues, and the National Rotavirus Reference Centre welcomes any notifications of rotavirus outbreaks.

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 and related diseases in Australia Annual Surveillance Report. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: (02) 9332 4648. Facsimile: (02) 9332 1837.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 to 31 August 2000, as reported to 30 November 2000, are included in this issue of Commun Dis Intell *(Tables 7 and 8).*

Table 7. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 31 August 2000, by sex and State or Territory of diagnosis

										Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2000	This period 1999	Year to date 2000	Year to date 1999
HIV diagnoses	Female	0	4	0	1	0	0	2	0	7	8	54	52
	Male	1	22	0	3	4	0	15	0	45	74	424	443
	Sex not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total[1]	1	26	0	4	4	0	17	0	52	82	480	495
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	3	9	12
	Male	0	3	0	0	0	0	2	0	5	23	103	103
	Total[1]	0	3	0	0	0	0	2	0	5	26	112	115
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	0	5	3
	Male	0	5	0	1	0	0	3	0	9	7	75	75
	Total[1]	0	5	0	0	0	0	3	0	9	7	80	79

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 August 2000, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	28	620	9	161	62	5	223	119	1,227
	Male	227	11,130	108	2,034	686	78	3,969	925	19,157
	Sex not reported	0	245	0	0	0	0	24	0	269
	Total[1]	255	12,016	117	2,202	748	83	4,230	1,049	20,700
AIDS diagnoses	Female	9	188	0	49	25	3	72	26	372
	Male	87	4,691	35	832	347	45	1,648	356	8,041
	Total[1]	96	4,891	35	883	372	48	1,728	384	8,437
AIDS deaths	Female	4	114	0	32	15	2	50	17	234
	Male	67	3,226	24	572	231	29	1,283	252	5,684
	Total[1]	71	3,348	24	606	246	31	1,339	270	5,935

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

Childhood Immunisation Coverage

Tables 9 and 10 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 July and 30 September 1999 and at 24 months of age for the cohort born between 1 July and 30 September 1998, according to the Australian Standard Vaccination Schedule.

A full description of the methodology used can be found in Commun Dis Intell *1998;22:36-37.*

Table 9. Percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 July to 30 September 1999; assessment date 31 December 2000

	State or Territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,094	22,277	786	12,801	4,600	1,547	15,869	6,241	65,215
Diphtheria, Tetanus, Pertussis (%)	93.3	91.1	91.0	92.4	92.5	92.5	92.5	90.8	91.8
Poliomyelitis (%)	93.1	91.0	91.0	92.3	92.4	92.4	92.4	90.8	91.8
Haemophilus influenzae type b (%)	93.1	90.9	93.1	92.6	92.4	91.7	92.4	90.6	91.7
Fully immunised (%)	92.7	90.5	89.2	92.0	92.2	91.6	92.1	90.2	91.3
Change in fully immunised since last quarter (%)	+1.0	+2.7	+8.7	+1.7	+2.2	+1.8	+1.9	+2.5	+2.3

Table 10. Proportion of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 July to 30 September 1998; assessment date 31 December 2000[1]

	State or Territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,119	22,021	853	12,712	4,691	1,636	15,949	6,386	65,367
Diphtheria, Tetanus, Pertussis (%)	90.1	88.0	84.1	91.2	92.3	91.6	90.4	88.0	89.6
Poliomyelitis (%)	93.0	91.3	91.7	93.5	94.4	94.4	93.5	91.8	92.7
Haemophilus influenzae type b (%)	89.0	87.9	87.7	91.3	91.9	91.6	90.5	88.2	89.6
Measles, Mumps, Rubella (%)	93.1	90.7	89.8	93.1	94.0	94.4	93.5	91.7	92.3
Fully immunised (%)[2]	87.8	81.9	80.0	88.3	88.6	88.6	86.4	83.1	85.1
Change in fully immunised since last quarter (%)	-0.2	+1.2	+2.9	+0.9	+3.2	+4.0	+2.3	+1.5	+1.7

1. The 12 months age data for this cohort was published in *Commun Dis Intell* 2000;24:42.
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.

Acknowledgment: These figures were provided by the Health Insurance Commission (HIC), to specifications provided by the Commonwealth Department of Health and Aged Care. For further information on these figures or data on the Australian Childhood Immunisation Register please contact the Immunisation Section of the HIC: Telephone 02 6124 6607.

Bulletin Board

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The Communicable Diseases Network Australia New Zealand (CDNANZ)
Communicable Diseases Control Conference 2001
2-3 April 2001
Hyatt Hotel, Canberra, Australian Capital Territory
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http://www.health.gov.au/pubhlth/cdi/cdconf.htm

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Fax: +49 89 33 6038
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Website: http://www.istm.org/istm_c7.html

Institute for Microbiology of Medical Faculty of Masaryk University & St Anna's Faculty Hospital
10th Tomasek Days
Annual conference of young microbiologists
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Brno, Czechia
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10-13 June 2001
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Website: http://www.ymk.gu.se/eng/x2001.htm

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Website: http://www.cbsm.uwa.edu.au/ASM2001/

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Contributions to the Bulletin Board are invited from those organisations with forthcoming events relevant to communicable disease control.

Case report: typhoid in the Northern Territory

Contributed by Dr David Peacock

Our recent typhoid case was a 42-year-old Indonesian oil-rig worker. He was a medical evacuation from the rig when he became unwell. He had recently spent some leave back home in Indonesia and that is where it is believed he contracted the disease. He was not a food-handler and there have been no reports of any other illness on the rig or supporting vessels.

He was diagnosed in Darwin Hospital and treated appropriately. He has now returned home.

CDI publication dates for 2001

The *CDI* team would like to remind readers that from 2001 *CDI* will be published quarterly instead of monthly. The publication dates for the remainder of this year are 30 April, 31 July and 31 October. We look forward to your continued readership.

Website

http://www.health.gov.au/pubhlth/cdi/cdihtml.htm

Contributions

Contributions covering any aspects of communicable diseases are invited. All contributions are subject to the normal refereeing process. **Instructions to authors can be found in *Commun Dis Intell* 2000;24:5**.

Subscriptions and contacts

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