Three nursing home outbreaks of Norwalk-like virus in Brisbane in 1999

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Abstract

We report on three nursing home outbreaks of gastroenteritis in Brisbane in 1999. The presence of Norwalk-like virus (NLV) genogroup 2 was demonstrated by reverse transcription polymerase chain reaction (RT-PCR) in all three outbreaks. Common findings of these investigations were rapid spread of the illness within the institutions and difficulties in identifying a common source. Nursing home populations are vulnerable and it is important for each institution to have infection control policies in place so outbreaks can be managed promptly. This includes the exclusion of ill staff for 48 to 72 hours after recovery from illness. Genogrouping of NLV by RT-PCR can take several days so control measures will generally have to be instituted before results become available. *Commun Dis Intell* 2000;24:229-233.

Keywords: disease outbreaks, Norwalk-like virus, small round structured virus, gastroenteritis, nursing homes, polymerase chain reaction, infection control

Introduction

The Norwalk-like viruses (NLVs) - also known as small round structured viruses (SRSVs) - are a genetically diverse group of RNA viruses within the family *Caliciviridae*.[1] Two main groups of NLVs have been identified: genogroup 1 (Norwalk virus group) and genogroup 2 (Snow Mountain virus group).[2] Symptoms of infection with NLVs commonly include sudden onset of nausea and vomiting and/or diarrhoea, accompanied by abdominal cramps, headache and low grade

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fever.[3] The vomiting and diarrhoea are frequently explosive in nature.[4] Documented modes of transmission of NLVs are water, food (especially shellfish and salads), fomites and person to person spread.[5] Aerosol transmission has also been postulated.[6,7,8]

Outbreaks due to NLVs have been associated with various settings including hospitals, nursing homes, restaurants, catered events, schools, prisons and cruise ships; nursing homes are the setting most commonly identified.[1] An analysis of faecal samples from south-eastern Australia collected between 1980 and 1996 showed that the majority of incidents occurred in elderly people in nursing homes or hospitals.[2] This, together with increasing reports of NLV incidents in Australia and overseas,[1,2,9,10] demonstrates that NLVs represent a significant and previously under-recognised public health problem, particularly amongst the elderly and institutionalised. We describe here three outbreaks of gastroenteritis in long-stay nursing homes. The outbreaks were subsequently shown to be due to Norwalk-like virus.

Methods

Epidemiological investigation

A team from the public health unit conducted an investigation of each outbreak of gastroenteritis using a standard questionnaire to collect data on case characteristics, symptomatology and duration of illness. A case was defined as a person who developed nausea and/or vomiting and/or diarrhoea during the outbreak period. An illness and food-history questionnaire was administered and, where indicated, a kitchen hygiene audit conducted (including food and water sampling). Staff were requested to collect faecal or vomitus specimens from cases and to provide such specimens if they themselves were affected. Food, water and stool samples were delivered to Queensland Health Scientific Services for analysis.

Microbiological investigation

Food specimens were cultured for *Escherichia coli*, coagulase-positive staphylococci, *Clostridium perfringens*, *Vibrio parahaemolyticus*, *Bacillus cereus*, *Salmonella* spp, *Yersinia enterocolitica*, *Campylobacter* spp and *Listeria* spp. A standard plate count, coliform counts and *E. coli* counts were performed on water samples. Stools were examined for ova, cysts and parasites, and cultured for bacteria. Two faecal and one vomitus specimen from outbreak A were tested for staphylococcal and *Bacillus cereus* toxins. Reverse transcription polymerase chain reaction (RT-PCR) was used to test stools and vomitus for enteric viruses (rotavirus, astrovirus, Norwalk-like virus and adenovirus).

Results

Outbreak A

On 26 February 1999 the Brisbane North Public Health Unit was notified of an outbreak of gastroenteritis in nursing home A. From the date the outbreak started (18 February) both staff and residents were affected. The home had a hostel section where the more ambulant residents lived and a nursing home section for those requiring more care. Outbreak control measures already instituted at the time of notification included barrier nursing, hand-washing and restrictions on the movements of ill residents. In addition, the public health unit advised that affected staff should not return to work until 48 hours after cessation of symptoms.

The main outbreak features are summarised in the Table. Affected staff included domestic, administrative, catering and nursing personnel. An incubation period could be recognised for only one resident: he was absent from the nursing home when the outbreak started but developed symptoms within 48 hours of return.

Figure 1 shows the epidemic curve for Outbreak A. Most early cases occurred in the hostel section with subsequent spread to the nursing home. Analysis of food histories did not suggest any common food source and there were no reports of illness amongst food handlers just prior to the outbreak. Inspection of kitchen facilities did not reveal any major breaches of hygiene. No pathogens were identified in the food samples, and coliforms were not detected in the water samples. Stool cultures for bacterial pathogens and tests for staphylococcal toxin were negative. Sixteen specimens were submitted and Norwalk-like virus genogroup 2 was detected by PCR from two specimens from two different cases.

Outbreak B

On 14 April 1999, the Brisbane Southside Public Health Unit was notified of an outbreak of gastroenteritis in nursing home B that had started on 13 April and involved both staff and residents. Staff brought their own meals. Residents were provided with meals from a central kitchen at another nursing home. No other institutions serviced by the central kitchen had become affected and this kitchen had recently been audited and found to meet food hygiene standards. The outbreak lasted 3 days, with five cases (one staff, four residents) on the first day, five cases (two staff, three residents) on the second and eight cases (six staff, two residents) on the third day.

Both staff and residents became ill from the same date and one member of the nursing staff reported a gastrointestinal illness 4 days prior to the outbreak. Analysis of food histories

Contents, *continued*

Table. Main outbreak features

Outbreak		A		B		C	
Duration (days)		21		3		23	
Population (n)	Staff	278		105		67	
	Residents	517		70		112	
Affected (attack rate %)	Staff	30	11%	9	9%	36	58%
	Residents	136	26%	9	13%	29	26%
Median duration of illness (hours)	Staff	24		48		48	
	Residents	48		N/I*		N/I	
Median age (years)	Staff	45		45		43	
	Residents	87		90		N/I	
Symptoms (%)	Vomiting		57%		83%		67%
	Diarrhoea		79%		61%		78%
Outcomes (number of persons)	Hospitalised	1		0		3	
	Died	1		0		0	

* N/I = not interviewed

did not suggest any common food source. Four faecal and two vomitus specimens were submitted from five cases. *Staphylococcus aureus* was isolated from one faecal specimen but Norwalk-like virus genogroup 2 was detected by PCR in all six specimens.

Outbreak C

On 25 May 1999, the West Moreton Public Health Unit was notified of an outbreak of gastroenteritis in nursing home C. The outbreak had started on 7 May and also involved both staff and residents. Due to dementia and debility in a high proportion of residents, it was decided that only staff would be asked to complete a standardised questionnaire. Residents ate food prepared in the nursing home. Staff brought their own food.

Ill staff came from domestic, catering, managerial and nursing categories. Eleven cases (28%) were still symptomatic when they returned to work. Figure 2 shows the epidemic curve for staff cases. Norwalk-like virus genogroup 2 was detected by PCR in three of three faecal specimens submitted.

Discussion

The epidemic curve of outbreak A clearly demonstrates the spread of the outbreak within the nursing home. Although ill residents were restricted to their rooms, staff may have transmitted the virus while moving within the facility. In both outbreaks A and B, the rapid initial peak might suggest a point source but it may also reflect rapid person to person spread.

Interpretation of the epidemic curve in outbreak C is limited by the lack of known onset dates for resident cases and by the absence of confirmation of NLV in the first two cases. It was most probably a propagated source outbreak. The gap between the initial two and subsequent cases may be due to poor case ascertainment. Alternatively, these two cases may not have been part of the outbreak. The negative results of epidemiological and microbiological investigations

Figure 1. Norwalk-like virus outbreak A, February/ March 1999. Number of cases by onset date and location

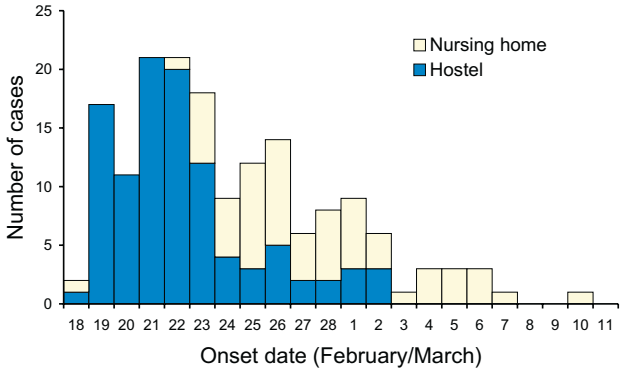
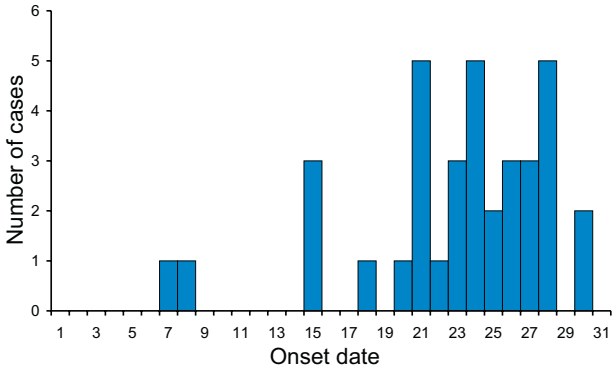


Figure 2. Norwalk-like virus outbreak C, May 1999. Number of cases by onset date



and the fact that staff and residents shared no food in any of these nursing homes suggest that food was an unlikely source of transmission.

It is particularly difficult to identify a source of infection in nursing home settings as many factors limit the quality of information available.[1] These may include inability of residents to provide accurate histories, uncooperative management fearing criticism from families of residents, lack of appropriate documentation, staff covering for sick colleagues being too busy to assist, and a lack of coordination between the various medical practitioners involved. In addition, late notification in two of the outbreaks described meant that secondary person to person transmission would have masked any common source.

The clinical and epidemiological features of all three outbreaks fit the criteria developed by Kaplan[11] for identifying NLV outbreaks, namely stool cultures negative for bacterial pathogens, mean or median duration of illness 12 to 60 hours, vomiting in 50% of cases, and a mean or median incubation period of 24 to 48 hours. Although *S. aureus* was isolated in one specimen, the significance of this is uncertain and the first three of the listed criteria were met in all three outbreaks. The incubation period could not be calculated due to lack of an obvious initial exposure.

In the outbreaks described here, the clinical and epidemiological features do not fit with other viral gastroenteritis agents of public health importance (rotaviruses, astroviruses, and certain serotypes of adenoviruses).[3,5] Rotaviruses can cause an illness lasting around 4 to 6 days in which vomiting, watery diarrhoea, fever and abdominal pain are prominent[3,5] but they usually affect young children. Astrovirus outbreaks are common in closed communities but the incubation period is 3 to 4 days, diarrhoea is more typical than vomiting and symptoms usually last 3 to 4 days.[3] Adenoviruses have a relatively long incubation period (3 to 10 days) and the illness lasts for a week or more.[5]

Substantial proportions of staff and residents were affected in these outbreaks. Attack rates in nursing homes can be as high as 50 to 70%.[7] Airborne transmission by aerosolised vomitus is suggested as a contributing factor to the rapid spread of NLVs.[4] In addition, spread of NLVs in nursing homes is facilitated by the enclosed environment, and by decreased personal hygiene related to patient factors such as immobility, incontinence and dementia. The risk of spread is augmented if staff return to work while still infectious or if staff members are rostered to work in unfamiliar areas of the nursing home. The early return of staff to work in nursing homes A and C may have prolonged these outbreaks.

NLVs cannot be grown in cell culture but can be identified by electron microscopy where sufficient amounts are present (1 million viruses/mL of stool). These levels of excretion usually only occur during the first 48 hours of diarrhoea.[5] Even when acute samples are obtained, only about 25% of samples will be positive.[12] PCR is the most sensitive diagnostic test available and can theoretically detect one virus particle. Although PCR has increased sensitivity to detect NLVs when specimens are collected late (more than 3 days) after onset of illness,[4] the success rate for detection is highest if the specimens (faeces and/or vomitus) are collected within 48 to 72 hours of illness onset. Specimens should be transported at 4°C if other enteric viruses are being considered (adenoviruses, astroviruses or rotaviruses), otherwise they can be frozen.

Genogroup 2 viruses were detected in all three outbreaks. Surveillance of outbreaks in Australia, the UK, the Netherlands and the US using PCR methodology has repeatedly found genotype 2 to be the predominant circulating strain.[1,2,4,9,10] Different primers are needed to detect the two genogroups and currently Queensland laboratories only report to this level. NLVs are genetically very varied and may sometimes fail to be detected because genetic mutations prevent the PCR primers used from binding.

The PCR results were not available for a minimum of 5 days in this series. Given adequate warning, the turnaround time for a confirmed result would be a minimum of 4 days from the receipt of specimens although provisional results could be available within 48 hours. Infection control measures clearly need to be instituted prior to obtaining results. In nursing home settings this is particularly important because poor mobility, chronic disease and diuretic therapy can both increase the initial susceptibility of residents to infection and also the risk of an adverse outcome.

Enteric disease control measures should be incorporated into the facility's infection control policies and outbreak management plans to ensure rapid and effective implementation. One particularly important measure when NLVs are suspected or confirmed is the exclusion of ill staff for 48 to 72 hours after recovery because excretion of the virus is minimal after this time.[3,5]

Recent studies suggest that viral shedding may persist for up to 10 to 14 days after recovery and can also occur in asymptomatic individuals, though the infectivity of the latter is uncertain.[13,14] Low levels of shedding may be important with NLV given the very small infectious dose (10 to 100 virions).[13] Exclusion of staff for prolonged periods is difficult to implement and of unproven benefit so it would appear reasonable to continue to recommend a 48 to 72 hour period of exclusion. However, staff returning to work should be scrupulous about hygiene.

Conclusion

Due to better diagnostic techniques, NLVs are now recognised as a major cause of epidemic non-bacterial gastroenteritis.[1,4,10] The highly susceptible population in nursing homes makes it imperative to have sound infection control policies in place for prompt management of NLV outbreaks.

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Typhoid and paratyphoid fever in south-eastern Sydney, 1992-1997

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Abstract

Notification records of typhoid and paratyphoid cases among residents of south-eastern Sydney during 1992-1997 were reviewed, with particular attention paid to identifying a source of infection and to completeness of follow up. Notifications comprised 30 cases of *Salmonella* Typhi, nine of *S.* Paratyphi A and five of *S.* Paratyphi B. These 44 cases had a median age of 20 years (range 2-62). Of the 39 cases with known country of birth, 30 were born overseas, predominantly in Asian countries. Of 39 cases with a known travel history, 33 were cases of overseas-acquired acute infection and two cases were asymptomatic chronic carriers. A source was identified in only one of four domestically acquired infections. Of eight household contacts in occupations posing a public health risk (seven food-handlers and one health-care worker), complete follow-up information was available for only five. Most cases were in overseas-born individuals who may have been infected when returning to their country of birth. Explicit follow-up protocols need to cover appropriate clinical management (including treatment of chronic carriage) and monitoring of those cases and contacts who could pose a public health risk. *Commun Dis Intell* 2000;24:233-236.

Keywords: typhoid, paratyphoid, surveillance, follow-up, travel, carrier

Introduction

Typhoid remains a disease of major importance worldwide although improvements in public health have made it an exotic disease in developed countries like Australia. Estimates of the global burden of typhoid suggest an annual incidence of 12.5 million cases with over three-quarters occurring in Africa and South East Asia.[1] In the United States the incidence has been stable at around 0.15-0.2 per 100,000 population since 1966, with an increasing proportion of cases being imported.[2,3]

A similar pattern is seen in Australia where 402 cases of typhoid and paratyphoid were notified to the National Notifiable Diseases Surveillance System (NNDSS) from 1992 to 1997, a crude annual incidence of 0.37 per 100,000 population (Communicable Diseases Network Australia New Zealand and NNDSS, personal communications). The vast majority of cases are acquired overseas; 66 (90%) of the 73 typhoid and paratyphoid cases reported with clinical details to the National Salmonella Surveillance Scheme in 1996 were known to have been acquired overseas.[4]

This report is a retrospective study of typhoid and paratyphoid notifications made to the South Eastern Sydney Public Health Unit (and its predecessors) from January 1992 to December 1997. We detail information on the source of

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infection and the results of the follow up of cases and contacts in sensitive occupations.

Methods

Cases of *Salmonella* Typhi and *S.* Paratyphi infection in south-eastern Sydney residents notified from 1992 to 1997 were identified from records held by the South Eastern Sydney Public Health Unit; these records include notifications passed on by other public health units. Records detail age (based on date of birth), sex, country of birth, history of overseas travel, date of onset of symptoms, date of notification, presumed source, contact screening and follow-up. This information had originally been obtained from each notification and from interviews with the case, the attending doctor and contacts as appropriate.

Cases were individuals from whom *S.* Typhi or *S.* Paratyphi had been isolated from a clinical specimen. The contact definitions and response guidelines used were those described in the May 1995 edition of the Infectious Diseases Manual issued to Public Health Units by New South Wales Health. These guidelines recommend screening of (i) household contacts of cases who have no history of overseas travel, in order to identify a source; and (ii) household contacts who work in sensitive occupations (i.e. food-handlers, health care workers with direct patient contact, and people who work in or attend child-care facilities).

The denominator population data for the calculation of rates were based on the Australian Bureau of Statistics 1996 Census of Population and Housing.

Results

Fifty-four culture-confirmed cases were notified by hospital and laboratory staff during 1992-1997. Seven visitors in transit and three cases living in other parts of Sydney were excluded from the analysis. This left 44 cases among local residents, 24 (55%) males and 20 (45%) females, including two chronic typhoid carriers found during contact screening of active cases. *S.* Typhi was isolated from 30 (68%) cases, *S.* Paratyphi A from nine (21%) and *S.* Paratyphi B from five (11%). Blood and/or stool cultures were the main source of the organism (Table 1). Cases ranged in age from 2 to 62 years, with a median age of 20 years. The highest incidence was in children of primary school age, with relatively high rates among teenagers, and lower rates in children under 5 years and among adults (Table 2). The overall annual incidence of active cases (i.e. excluding the two carriers detected on screening) was 0.94 per 100,000.

Of the 39 cases with known country of birth, nine were Australian born. Of those born overseas, 16 cases were Indonesian, six were from South Asia (Bangladesh, 3; Pakistan, 2; India, 1) and the remainder were born in other Asian countries (China, 2; Hong Kong, 1; Singapore, 1), the Middle East (Lebanon, 1; Saudi Arabia, 1), Chile (1) or Portugal (1). The age distribution of active cases according to place of birth is summarised in Table 2. University students were over-represented among overseas-born cases.

Source of infection

Presumed overseas-acquired

Information on previous overseas travel was available for 39 cases. Thirty-three cases were considered to have acquired their infection overseas. Symptoms were first evident between the date of arrival in Sydney and up to 9 months following arrival, with a median onset of 2 weeks

Table 1. Source of culture confirmation, typhoid and paratyphoid cases, south-eastern Sydney residents, 1992-97

Specimen	No.	%
Blood only	26	59.1
Stool only	12	27.3
Blood + stool	4	9.1
Others*	2	4.5
Total	44	100

* Liver abscess, 1; sternoclavicular joint aspirate, 1

Table 2. Active typhoid and paratyphoid cases, south-eastern Sydney residents, 1992-1997, by age and region of birth

Age group (y)	Born overseas	Australian born	Country of birth unknown	Total cases	Annual incidence/ 100,000*
0-4	3	0	0	3	1.2
5-9	5	1	1	7	2.9
10-14	2	3	0	5	2.1
15-19	6	0	0	6	2.2
20-29	5	3	1	9	1.1
30-39	1	1	1	3	0.4
40-49	4	1	1	6	0.9
50-59	1	0	1	2	0.4
60-69	1	0	0	1	0.3
Total	28	9	5	42	0.94

* Incidence based on estimated resident population, 1996 Census

after arrival. Of the overseas-acquired cases among those born overseas, there was concordance between country of birth and country of travel in 25 of 26 cases, suggesting that most infections were acquired during visits to relatives.

Two cases with apparently prolonged incubation periods are summarised below:-

Case 1: This 19-year-old woman was hospitalised in December 1996 after 3 days of fever and rigours. She was notified on the basis of serology and a blood-culture positive for *S.* Typhi. She had been in Australia for 9 months since her arrival from Saudi Arabia.

Case 2: *S.* Typhi was isolated from a liver abscess and stool of a 62-year-old Chilean man who had presented to hospital in June 1992 with a three-week history of pyrexia of unknown origin. He had been in Australia for five-and-a-half months since his last travel to Chile.

A further two cases were asymptomatic carriers found on screening contacts of symptomatic cases. Both were overseas-born males, one a 53-year-old Portuguese, and the other a 58-year-old from East Timor.

Domestically-acquired cases

Of the four cases with no history of overseas travel, one was the wife of a typhoid carrier, one child was presumed to have acquired the infection from relatives who had returned from a typhoid-endemic country, and in two other cases no source was found.

Cases in sensitive occupations

For the 22 of 29 adult cases for whom an occupation was recorded, only three were in sensitive occupations, and all were food-handlers. Following receipt of the notification, Public Health Unit staff ensured that the attending doctor of each case was aware of the criteria for return to work (i.e. two negative stool cultures at least 24 hours apart after cessation of antibiotic therapy). Copies of follow-up culture results were not available in the Public Health Unit's files.

Screening of contacts in sensitive occupations

Of the 103 household contacts identified, eight were in a sensitive occupation (seven food-handlers and one health care worker) (Table 3). The files of three food-handlers and the health care worker contained references to two negative stool cultures; a fourth food-handler was found to be a chronic typhoid carrier and was referred for specialist management. The files of the remaining three food-handlers contained no information on follow-up culture results.

Table 3. Follow up of typhoid and paratyphoid contacts in sensitive occupations, south-eastern Sydney, 1992-1997

Case	Case date	Occupation	Sex	Stool culture
1	Jun 1992	Health care worker	F	2 negative
2	Dec 1993	Food-handler	M	n/a*
3	Feb 1994	Waitress	F	2 negative
4	Apr 1995	Chicken shop proprietor	M	Chronic carrier 3 positive
5	Apr 1995	Food-handler	M	2 negative
6	Apr 1995	Food-handler	M	2 negative
7	May 1995	Restaurant supervisor	M	n/a
8	May 1995	Kitchenhand	F	n/a

* n/a: not available

Discussion

Under the New South Wales Public Health Act 1991, all salmonella infections are to be notified by pathology laboratories, whilst typhoid and paratyphoid are to be notified on a clinical basis by hospital chief executives or their delegates. Annual notifications fluctuated, with most cases (11) notified in 1995 and the fewest (3) in 1997. The 42 active cases - of which two thirds were of typhoid fever - equated to an annual incidence of 0.94 cases per 100,000. The mean age of 20 years was similar to that of 24 years reported from the United States[3] and 20 years from England.[5] Peak incidence was in the 5-9 year age group, with very few cases under 5 years, consistent with descriptions of typhoid fever in other populations.[6]

It was felt that 75% of cases were infected overseas, similar to the 72% of US cases reported in the period 1985-1994.[3] Two cases with apparently prolonged incubation after overseas residence may, in fact, have been infected in Australia from unknown sources. We were also unable to determine a source for a number of the domestically acquired cases, either as a result of incomplete follow up or insensitivity of screening methods. In their analysis of Birmingham cases, Braddick and colleagues found that urine cultures substantially increased the rate of detection of infected contacts,[5] and screening urine may be a valuable modification to the current New South Wales protocol.

When the public health records of three cases (all food-handlers) and eight household contacts (seven food-handlers and one health care worker) in sensitive occupations were reviewed, documentation of follow-up arrangements were found in most, but follow-up stool culture results were lacking in some. This highlights the importance of follow-up protocols covering identification of high-risk contacts, securing of stool specimens with culture results, referral arrangements, results of follow-up of any found to be carriers, and, most importantly, good documentation of all these steps.

The purpose of typhoid and paratyphoid surveillance is to minimise the risk to public health by identifying cases and carriers and guiding them to appropriate treatment, and by locating and eliminating any environmental sources. In particular, undetected carrier food-handlers pose a potential source for prolonged outbreaks of typhoid or paratyphoid fever.[7] Thorough investigation and follow up of new cases and their contacts is required if further transmission of typhoid and paratyphoid is to be avoided.

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A community outbreak of *Cryptosporidium* infection associated with a swimming pool complex

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Abstract

A case-control study was conducted to investigate the cause of a sudden increase in cases of cryptosporidiosis notified to the Brisbane Southside Public Health Unit from January to March 1998. Fifty-two eligible cases were identified over a three-week period early in 1998. Thirty-one of these cases and 21 control subjects participated in the study. Swimming in the 2 weeks before onset of illness was identified as a likely risk factor for cryptosporidiosis infection (OR 3.1, CI 0.8-12.6, P=0.06). Analysis of swimming pool attendance identified swimming at Pool Complex A as a significant risk factor for the acquisition of cryptosporidiosis (OR 8.9, CI 1.5-67.4, P=0.004). No other potential risk factors were significantly associated with illness. The detection of cryptosporidium oocysts in three of the four pools at Pool Complex A supported the findings of the case-control study. As a response to this outbreak, Queensland Health has developed a Code of Practice outlining measures for the control and prevention of future outbreaks of swimming pool-associated cryptosporidiosis and/or giardiasis. *Commun Dis Intell* 2000; 24:236-239

Keywords: gastroenteritis, cryptosporidiosis, giardiasis, oocysts, swimming pool, water-borne

Introduction

Cryptosporidium parvum, a protozoan parasite, was identified as a human pathogen in 1976 and has since been identified as the cause of outbreaks of gastroenteritis associated with drinking water and swimming pools.[1-4] It is transmitted mainly by ingestion of water or food contaminated with oocysts excreted in animal or human faeces. The infective dose of *C. parvum* has been shown to be as low as 30 oocysts and may even be lower.[5] The onset of clinical symptoms generally occurs within 2 to 12 days (average 7 days) of exposure. The main clinical symptom is diarrhoea, which may be profuse and watery, often accompanied by abdominal pain, fever, malaise and vomiting. The illness usually lasts between 1 and 3 weeks, and while it is generally self-limiting, may be prolonged or even fatal in immunocompromised individuals. Available treatment is limited to providing relief of symptoms.

Routine surveillance undertaken by the Brisbane Southside Public Health Unit (BSPHU) detected a sudden increase in human cryptosporidium notifications, particularly in bayside and eastern suburbs of Brisbane, in January and February 1998. During these months there were 104 reports compared with 37 notifications for January and February in 1997. A further 168 notifications were received for the month of March 1998 (Figure). An outbreak investigation was commenced by BSPHU on Friday 6 March 1998.

Methods

A case-control study was conducted using all cases notified to the BSPHU between 17 February and 9 March 1998. A case was defined as any person residing in the Redland Shire or the southern suburbs of Brisbane City who had an illness characterised by diarrhoea, vomiting or abdominal pain and had laboratory-confirmed *Cryptosporidium* oocysts detected in their stools during this period. Persons diagnosed before this period were not interviewed because of the potential for poor recall. Secondary household cases were excluded from the case-control study.

Cases were asked to nominate two control subjects who were of similar age (0-4, 5-12, 13-24, 25-39, 40+ years) and area of residence. Only one control per case was included in the study (the first control agreeing to participate and be interviewed). Controls were excluded if they had a history of enteric illness in the preceding 2 weeks. Whether these controls were infected with *Cryptosporidium* and had subclinical illness or were asymptomatic carriers was not ascertained.

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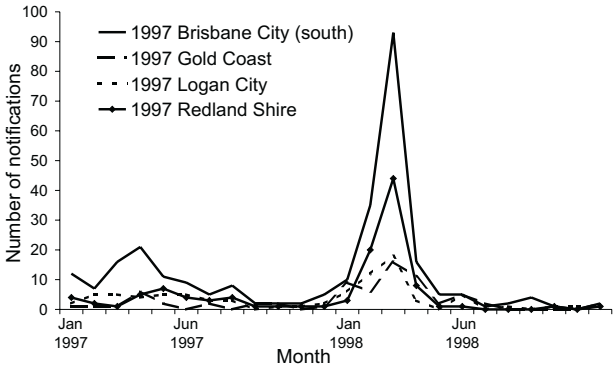
Figure. Cryptosporidium notifications, Queensland 1997-1998, by Local Government Area and month



Data were collected from cases and controls by telephone interview. A parent or guardian was interviewed if the case was aged 17 years or less. Cases were excluded from the study if three telephone contact attempts were unsuccessful. A standardised questionnaire sought information on basic demographics, symptoms and exposure to potential risk factors during the 2 weeks prior to onset of illness. Within 24 hours of the corresponding case interview, controls were interviewed about exposures during the two-week period immediately before their interview.

Potential risk factors included exposure to treated and untreated drinking water, consumption of unpasteurised milk products, contact with swimming pool or surface water (such as lakes, rivers), attendance at childcare facilities, contact with animals, and recent overseas travel.

Data were analysed with Epi Info v6.04b (univariate analysis)[6] and SPSS 7.5 (logistic regression).[6,7] Unmatched and matched odds ratios and 95% confidence intervals were calculated to determine associations between exposure factors and illness and tests of statistical significance applied to the data.

Environmental inspection of Pool Complex A was undertaken to obtain information on chlorination and filtration records and procedures, water quality results and records of faecal accidents.

Microbiological tests were conducted on water samples from the pools at Pool Complex A. The Complex consisted of four indoor swimming pools - a wading pool, 25 and 50 metre pools, and a diving pool. All pools had separate sand filtration systems, except the wading pool, which had a cartridge filter. A total of seven 10L water samples (surface and backwash samples) were collected from the swimming pools. Following a series of concentration steps, immunofluorescent microscopy was used for detection of oocysts in each 10L-water sample. Laboratory tests for oocyst viability and species identification were not available at the time of this investigation.

Table. Univariate analysis of the major potential risk factors for cryptosporidiosis

Risk factor (exposure during two weeks before illness or interview)	Cases		Controls		Odds ratio[1]	95% CI[2]	P value[3]
	Number (n=31)	%	Number (n=21)	%			
Male	16	51.6	10	47.6	1	0.3 - 3.4	0.96
Attended childcare centre	12	38.7	7	33.3	1.3	0.3 - 4.7	0.7
Overseas travel	0	0	0	0	-	-	-
Contact with pets	21	67.7	11	52.4	1.9	0.5 - 7.1	0.27
Contact with sick animals	1	3.2	0	0	Undef*	-	1
Contact with zoo animals	1	3.2	0	0	Undef	-	1
Contact with farm animals	0	0	2	9.5	Undef	-	0.16
Raw milk consumed	0	0	0	0	-	-	-
Untreated water consumed	4	12.9	1	4.8	3	0.3 - 76.6	0.64
Mains water consumed	30	96.8	21	100	Undef	-	1
Tank water consumed	1	3.2	0	0	Undef	-	1
Swimming	24	77.4	11	52.4	3.1	0.8 - 12.6	0.06
Swam at Pool Complex A	15	48.4	2	9.5	8.9	1.5 - 67.4	0.004

1. Unmatched odds ratio
2. 95% confidence interval
3. Mantel-Haenszel chi squared test or Fisher Exact test where appropriate,

* Undef = undefined value.

Results

Fifty-two cases of cryptosporidiosis were notified to the BSPHU during the period of the study. Sixty-two percent were aged 4 years or less (32 cases), with a median age of 2 years (range 4 months to 70 years). The male to female ratio was 1.1:1. Thirty-three were able to be contacted by telephone of whom 31 agreed to interview (median age was 3 years; range 1 to 58 years; male to female ratio was 1.1:1; 18 (58%) were aged 4 years or less). Twenty-one controls agreed to interview.

Symptoms reported by cases were diarrhoea (97%), abdominal cramps (94%), fever (65%), and vomiting (45%). Three (10%) reported blood in their stools. The median duration of symptoms was 11 days (range 3 to 24 days). No case was hospitalised.

The odds ratios for matched and unmatched analyses were similar, so the results of the unmatched analyses are presented in the Table. Age and geographic area were not confounding factors. Swimming during the 2 weeks before onset of illness was identified as a likely risk factor for cryptosporidiosis (OR=3.1, 95%CI 0.8-12.6, P=0.06). Analysis of attendance at particular pools identified that swimming at Pool Complex A was a significant risk factor for the acquisition of cryptosporidiosis (OR=8.9, 95%CI 1.5-67.4, P=0.004). This association remained statistically significant when adjusted for other potential confounding variables simultaneously using logistic regression.

Cryptosporidium oocysts were detected in water samples taken from the 25m, 50m, and wading pools. Surface sample counts varied between 13 and 62 oocysts per 10L sample. Backwash counts were 10 to 100 times higher. No *Cryptosporidium* oocysts were detected in diving pool water. *Giardia intestinalis* cysts were detected in surface water samples from the 50m and wading pools and in the backwash of the 50m pool. No records were kept of faecal accidents. Review of 1998 records of chlorination and water quality for each pool met the recommended standards. There was no evidence of filter malfunction preceding the onset of the outbreak.

Over the 3-week notification period from which eligible cases were identified, the notification rates were 7.2 and 20.4/100,000 persons for the Brisbane City South area and Redland Shire, respectively. The same 3-week notification rates for adjacent regions were 0.6/100,000 persons for Gold Coast City and 3.2/100,000 persons for Logan City (Figure).

Discussion

The findings of the case-control study suggest that a community outbreak of cryptosporidiosis occurred in association with swimming at Pool Complex A. The magnitude of this association, the consistency of these findings with other studies,[1-3] biological plausibility (oocysts resistant to typical pool chlorine levels and filtration methods),[8] and the temporal nature of infection following swimming, all support a causal association between swimming at Pool Complex A and developing cryptosporidiosis. In view of the small number of cases and controls it was not possible to determine with certainty which of the four pools in this complex were involved.

This outbreak occurred in the context of a large increase in notifications of cryptosporidiosis in the two local government areas neighbouring Pool Complex A. No other risk factors analysed in this study showed any significant association with cryptosporidiosis. The microbiological results of the water samples were limited by the non-availability of testing for speciation and viability. However, the finding of oocysts in water samples from the complex was considered to lend support to the epidemiological findings. As a result of these findings, Pool Complex A was closed for public use on 11 March 1998 for cleaning and disinfection.

The potential for information bias created by the different time exposure periods for cases and controls must be considered in this study. However, given the strength of the association between Pool Complex A and illness, it is unlikely that this effect could have been explained by information bias alone.

Despite the strong association, 16 of the 31 cases from the case-control study were not linked to that Pool Complex. Nine cases reported swimming at other pools, whilst seven cases did not report swimming in the 2 weeks before their illness. Other pools, person to person transmission, or other unrecognised exposures are possible sources of infection in these 16 cases.

Children too young to be toilet-trained frequently used the pool complex. The presence of *Giardia* cysts detected in the water samples supports the concept of faecal shedding in the pools. However, because *Giardia* is susceptible to chlorine levels normally found in pools, it is unlikely these were viable cysts.[8] Because *Cryptosporidium* oocysts are resistant to such chlorine levels[8] and are small in size (4-6 m), their inactivation and removal may not have been effected by the usual filtration and chlorination practices in place at Pool Complex A.

More than half the notified cases were aged less than 5 years. This may reflect a notification bias, as young children are more likely to attend a medical practitioner and to be tested than older children or adults. Furthermore, compared with older children or adults, young children are more likely to swallow pool water when swimming and increase their risk of becoming infected.

Following the identification of the outbreak, all four pools in the complex were treated with chlorine dioxide as a means of inactivating *Cryptosporidium* oocysts.[8] The pools were subsequently sampled and, following a series of negative results, the complex was reopened 14 days after its closure. No further cases linked to the complex were identified subsequent to its reopening.

Given the public health significance of this outbreak, a Code of Practice for the control of *Cryptosporidium* and *Giardia* in swimming pools, leisure pools, spas and hydrotherapy pools has been developed in Queensland.[10] The Code addresses issues relating to the maintenance of pools, disinfection procedures and preventive measures against future outbreaks of swimming pool-associated cryptosporidiosis and giardiasis. Routine screening of swimming pools for cryptosporidia and giardiae is not recommended. Protocols ensuring accurate recording and monitoring of chemical treatment and general pool maintenance - including contingency plans to deal with faecal accidents - are the mainstay of prevention strategies. However, testing is recommended when evidence suggests two or more cases of disease may be associated with a particular pool.

Acknowledgments

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Editorial statement. Preliminary reports of this work have appeared elsewhere.

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Notifications of vaccine preventable diseases in Australia. Quarterly report (January-March 2000)

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Abstract

Vaccine preventable disease notifications for Australia with disease onset dates between January and March 2000 are reviewed. During this quarter, numbers of notifications for *Haemophilus influenzae* type b disease and measles were the lowest ever recorded, while those for rubella were the lowest recorded since before the epidemic of spring 1992. These are promising trends that are likely to represent a true reduction in disease incidence. Numbers of pertussis notifications declined compared with the last quarter of 1999, but remain high, making up 88% of notifications for the vaccine preventable diseases reported here. *Commun Dis Intell* 2000:24:239-241

Keywords: vaccine, immunisation, Haemophilus influenzae type b, measles, pertussis, rubella, mumps, tetanus

Introduction

This is the first quarterly report on notification data for diseases targeted by the current standard childhood vaccination schedule (excluding hepatitis B). It includes notifications with disease onset dates between January and March 2000 that were notified by 23 May 2000. Comparisons were made with the first quarters of the previous five years, the last quarter of 1999 and historical data recorded on the current NNDSS database (established in 1991).

Vaccine preventable diseases (VPDs)

Invasive *Haemophilus influenzae* type b disease

There have been 10 or less notifications of *Haemophilus influenzae* type b (Hib) disease per month since 1995. In the first quarter of 2000 there were 3 cases, the lowest number recorded in any quarter. The cases were aged 7 and 10 months and 3 years with one case each from New South Wales, Queensland and Western Australia. The age specific data for invasive Hib disease in the age groups targeted for immunisation (children aged less than 10 years) are shown in Figures 1 and 2.

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Figure 1. *Haemophilus influenzae* type b disease notifications, 0 to 4 years age group, Australia, 1995-2000, by quarter

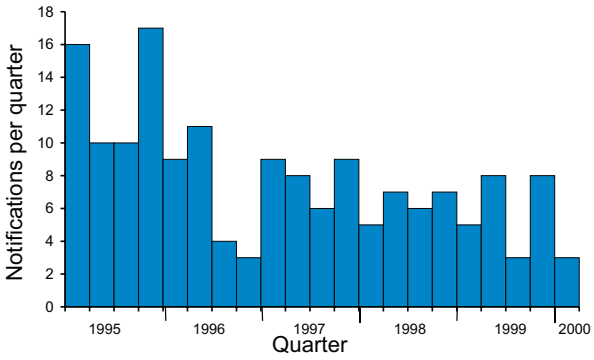
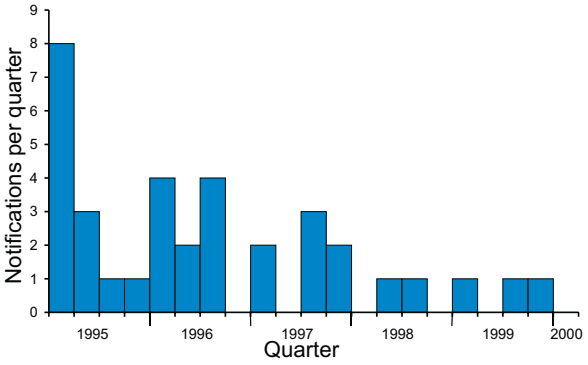


Figure 2. *Haemophilus influenzae* type b disease notifications, 5 to 9 year age group, Australia, 1995-2000, by quarter



Measles

Measles notifications for the first quarter of each year have been declining steadily over the past 6 years (Figure 3). There were 31 notifications of measles with an onset date in this quarter. This is the lowest number of notifications ever recorded for any quarter and is less than a third of that for the same quarter last year. Most cases (58%) were aged 15-29 years, with three cases each in the less than one year, 1-4 year and 5-14 year age groups. The remaining four cases were aged 30 to 49 years. There were more males than females (M:F ratio 1.6:1), especially in the adult (>15 years) groups (M:F ratio 2.7:1). A decline in cases from the same quarter last year was seen in all States and Territories except Queensland. Victoria showed the most dramatic decrease as this State experienced a measles outbreak at the beginning of 1999.

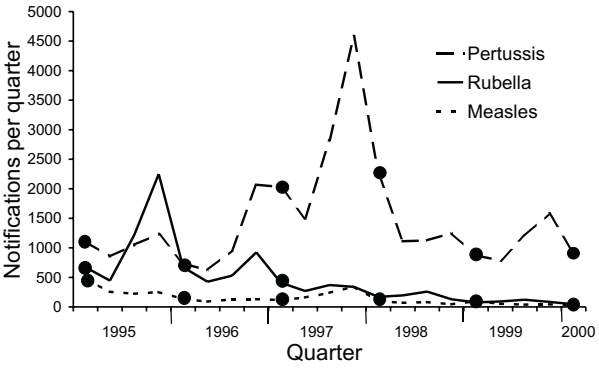
Pertussis

There were 915 notifications for pertussis this quarter, a slight increase overall from the same quarter last year (Figure 3). This increase was most evident in the 10-14 year age group (Figure 4) and in Tasmania, where high numbers of cases were reported for the second half of 1999 and first quarter of this year. However, total numbers this quarter are substantially lower than the peaks in the first quarters of 1997 and 1998 (Figure 3) and the 1,581 notifications for quarter 4 of 1999. Falls from the fourth quarter of 1999 were greatest in Tasmania and Victoria, while Western Australia had the lowest number of cases reported for a quarter since 1992 (seven cases). Most notified cases were aged 10 to 14 years (19%) and 26 (3%) were aged less than 6 months. Females predominated in almost all age groups and overall (M:F ratio 1:1.2).

Rubella

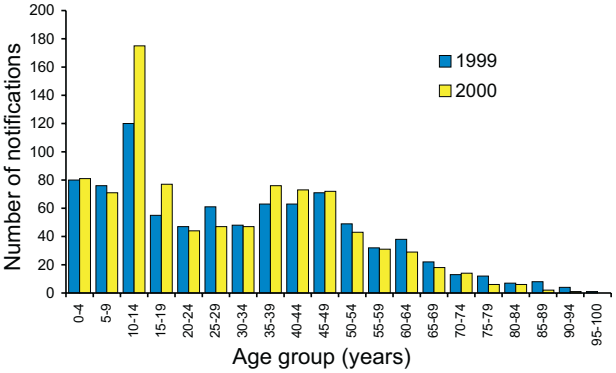
There were 46 cases of rubella notified this quarter (Figure 3). This is the lowest number of cases reported for any quarter since 1992. Equal numbers of males and females were notified overall. However in the 0-4 year age group, six of the seven cases were female, and males predominated in older age groups. Case numbers peaked in the 20-24 year age group (14 cases), with most cases (60%) aged 15-30 years. Queensland and Victoria recorded fewer than half the number of notifications than they had recorded in the last

Figure 3. Notifications of measles, pertussis and rubella, Australia, 1995-2000, by quarter[1]



1. ·indicates first quarter

Figure 4. Notifications of pertussis, Australia, first quarters of 1999 and 2000, by age group



quarter of 1999, while other States and Territories had similar numbers in these two periods. Compared with the first quarter of 1999, there were fewer cases in all jurisdictions except New South Wales.

Mumps

Numbers of mumps notifications have fluctuated between 28 and 59 per quarter over the past 6 years, with no overall trend either at a State and Territory level or nationally. The number reported this quarter (45 cases) is similar to that for the previous quarter (41 cases), but is higher than for the first quarter of last year (28 cases). All States and Territories except Queensland had similar or increased numbers of notifications compared with quarter 1 in 1999. Western Australia had the greatest increase (from 8 to 15 cases) and reported one third of the cases for this quarter. This is in contrast to the past 5 years in which Victoria reported most (29-51%) of the mumps notifications. Four of the 44 cases with a known age were aged less than 10 years, with the highest number (11 cases) reported in the 20-24 year age group. This is a change from previous years when cases aged less than 10 years predominated. There were slightly more males overall (M:F ratio 1.1:1) and in most age groups.

Other VPDs

There were no cases of polio or diphtheria and three cases of tetanus reported for this quarter. The tetanus cases were notified from New South Wales, Queensland and South Australia. One case was aged 50 years whilst the other two cases were aged at least 75 years; two were male. The number and age distribution of the tetanus cases is similar to that reported in each quarter of the previous 5 years where there were between one and four cases each quarter and 85% were aged at least 50 years.

The NCIRS was established by the National Centre for Disease Control, Commonwealth Department of Health and Aged Care. The Centre analyses, interprets, and evaluates national surveillance data on immunisation coverage and vaccine preventable diseases. NCIRS also identifies research priorities, and initiates and coordinates research on immunisation issues and the epidemiology of vaccine preventable diseases in Australia.

Editorial

Young adult measles vaccination

In 1997 the Federal Health Minister, the Hon Dr Michael Wooldridge MP announced a measles elimination program as a component of the 'Immunise Australia: Seven Point Plan'. The Measles Elimination Advisory Committee (MEAC) was charged with the task of delivering a national approach to measles elimination and now the goal of elimination is achievable.

Several milestones have been passed – the Measles Control Campaign (August to November 1998) and its subsequent evaluation by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (June 1999); the revision of the 'Guidelines for the control of measles outbreaks in Australia' by MEAC (July 2000),[1] the publication of the report 'Let's work together to beat measles' (August 2000)[2]and the 7th edition of the Australian Immunisation Handbook which includes revised recommendations for the use of Measles Mumps Rubella (MMR) vaccine.[3]

The Measles Control Campaign was a one off vaccination campaign to ensure that all children aged 5-12 years received their second dose of MMR vaccine. The campaign was in response to the change in the vaccination protocol that recommended the second dose of MMR be moved from 10-16 years of age to 4 years of age. Both the Measles Control Campaign and the two-dose MMR policy in place since 1994 appear to have raised levels of immunity in younger age groups to a point where sustained transmission of measles in unlikely to occur.

The history of measles vaccination scheduling and available seroprevalence data for Australia indicate that young adults are now the greatest risk with regard to measles transmission within Australia. Of cases that were notified during the measles outbreak in Victoria at the beginning of 1999, 84% were in the 1968 to 1981 birth cohort. Those in the 18 to 30 years age group were likely to have been unvaccinated or have received only one dose of MMR as part of the vaccination program that commenced in 1994. The first cohort to receive MMR in 1994 will be turning 16 or 17 years of age in 2000.

Other reasons why measles vaccination is important for young adults are

- They represent the 'adventurers' in the community. Young working holiday makers or tourists who are visiting measles endemic countries,
- Morbidity data suggests that adults have a higher risk of complications when they become infected,
- Recent experience from managing outbreaks has shown that this group incorrectly believes that they have immunity to measles infection, and
- That they readily confuse measles and rubella.
- The problems (and costs) involved in tracing measles contacts as illustrated by recent Australian cases.[4,5]

The recent decision by the Commonwealth Government to fund MMR vaccine for 18-30 year olds overcomes a significant barrier to reducing the risk of outbreaks of measles and, within young adult males particularly, rubella. The provision of 'free' vaccine will enable State and Territory Health Departments, General Practitioners and other vaccination providers to act upon the specific recommendation of the Guidelines "that, at all times, but particularly during a measles outbreak, centres such as general practices, medical centres, hospital emergency departments, university health services and sexual health clinics should be encouraged to identify individuals who may be susceptible to the measles virus from this at-risk group, and offer opportunistic MMR vaccination if not contraindicated."

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Revised list of diseases reportable to the National Notifiable Diseases Surveillance System (NNDSS)

On 8 February 2000 the Strategic Steering Committee of the Communicable Diseases Network Australia New Zealand agreed to a revised national list of diseases to be reported to the NNDSS. In the past, not all diseases reported to the NNDSS have been notifiable in all States and Territories. *Communicable Diseases Intelligence* will begin reporting on the revised list from 1 January 2001.

Notifiable Diseases list (as at February 2000)

AIDS
Anthrax
Arbovirus infections:
  Barmah Forest virus
  Dengue virus
  Japanese encephalitis virus
  Murray Valley encephalitis virus
  Ross River virus
  Kunjin virus
  Other arbovirus infections
Botulism (foodborne)
Brucellosis
Campylobacteriosis
Chlamydia trachomatis genital infection
Cholera
Cryptosporidiosis
Diphtheria
Donovanosis
Gonococcal infection
Haemolytic Uraemic Syndrome (HUS)
Haemophilus influenzae type b infection (invasive only)
Haemorrhagic fevers (quarantinable)
Hepatitis A
Hepatitis B
Hepatitis C
Hepatitis D
Hepatitis E
Hepatitis other
HIV infection
Influenza (laboratory confirmed)
Legionellosis
Leprosy
Leptospirosis
Listeriosis
Lyssavirus:
  Australian Bat lyssavirus
  Rabies
  Other
Malaria
Measles
Meningococcal infection
Mumps
Ornithosis (Psittacosis)
Pertussis
Plague
Poliomyelitis
Pneumococcal infection (invasive)
Q fever
Rabies (refer to Lyssavirus)
Rubella/congenital rubella
Salmonellosis
Shigellosis
Shiga-like toxin producing *E. coli*
  VTEC / SLTEC
Syphilis/congenital syphilis
Tetanus
Tuberculosis
Typhoid
Yellow fever

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 data by report date for information only. In Table 2 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 (*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 July, 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'.*

Three types of data are included in National Influenza Surveillance, 2000. These are sentinel general practitioner surveillance conducted by the Australian Sentinel Practice Research Network (ASPREN), the Department of Human Services (Victoria), the Department of Health (New South Wales) and the Tropical Influenza Surveillance Scheme, Territory Health Services (Northern Territory); laboratory surveillance data from the Communicable Diseases Intelligence *Virology and Serology Laboratory Reporting Scheme (LabVISE); and the World Health Organization Collaborating Centre for Influenza Reference and Research; and absenteeism surveillance conducted by Australia Post. Data from ASPREN are referred to as 'consultations' or 'encounters'. For further information about these schemes, see* Commun Dis Intell *2000;24:9-10.*

Figure 1 illustrates the July 2000 totals for selected diseases as ratios to the mean of their June to August levels for the previous 5 years.

Hepatitis B

There were 30 incident cases of hepatitis B infection in July 2000 - a notification rate of 1.9/100,000 population. Conversely, the notification rate of unspecified hepatitis B decreased from 30-40/100,000 population in previous years to 25.9/100,000 for this month.

Foodborne illness

There was an outbreak of *Salmonella* Ball in the Northern Territory in July 2000 but, to date, no common source has been identified. Eight cases this month is clearly higher than the background rate of *Salmonella* Ball in that region. The 'outbreak' cases have been restricted to eight in Darwin/Palmerston.

Shigella

Six of the 26 notifications in July 2000 were cases of *Shigella sonnei* biotype g in Victoria; five were in males aged between 20 and 40 years and one was of unspecified gender. Between 27 and 36 cases of *S. sonnei* biotype g are notified in Victoria each year. In the first six-month period of this year, there were 12 cases of locally acquired *S. sonnei* biotype g, which was similar to the expected number. However, the antibiogram of faecal isolates was identical to that of the recent Sydney outbreak strain associated with gay men (see Special Report below). To prevent the spread of shigellosis, the Department of Human Services distributed targeted preventive advice to local sex-on-premises venues and through the gay press. Unlike in the other States, there was no recognised association with gay men in the nine cases of shigellosis in Western Australia in July 2000.

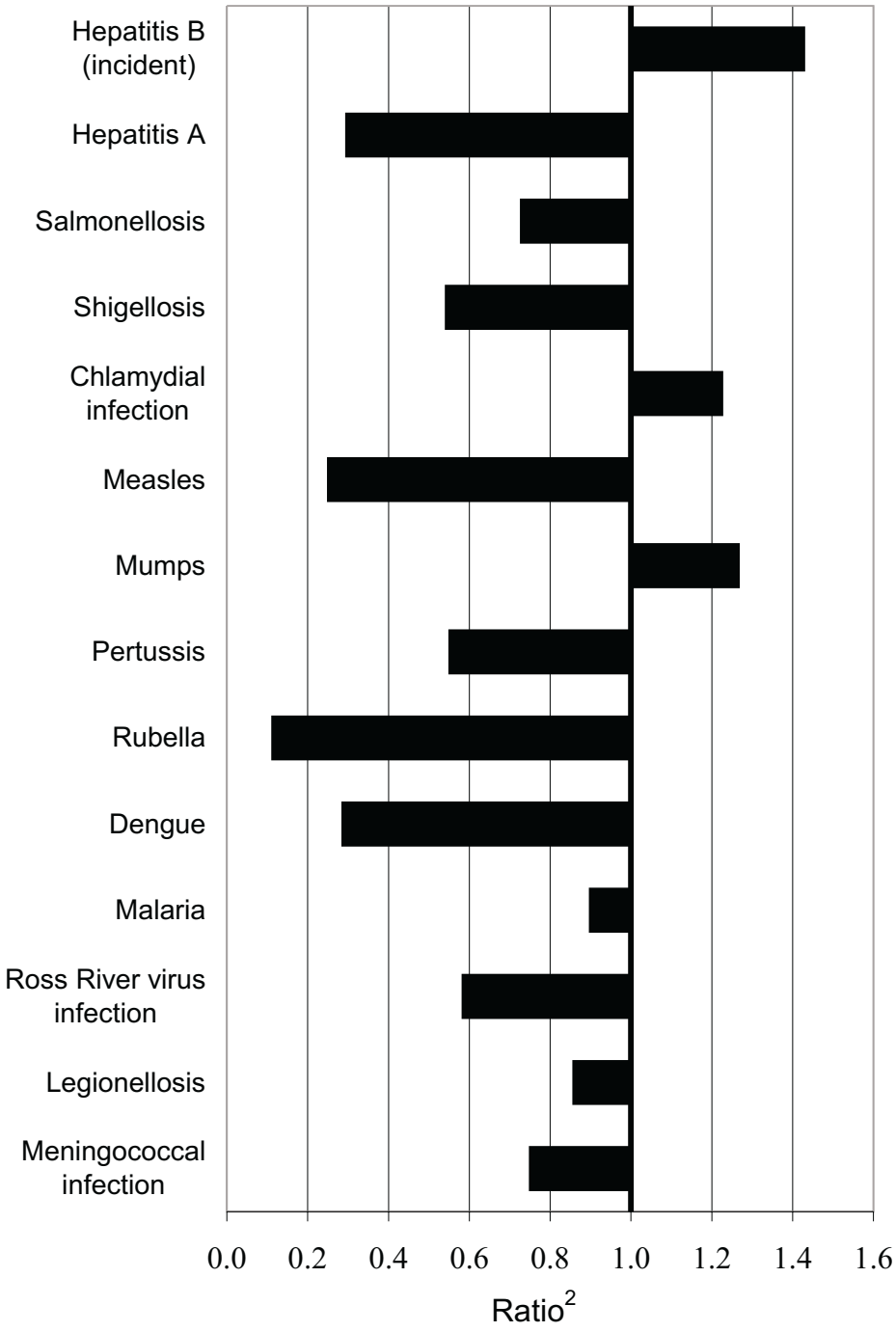
SLTEC/VTEC

There were two notifications in July 2000 from South Australia of Shiga-like toxin-producing *Escherichia coli* (SLTEC)/ verotoxigenic *Escherichia coli* (VTEC), one in a 15-year-old male and one in a 78-year-old female.

Typhoid

There were seven notifications of typhoid in July 2000 with five cases in New South Wales and two cases in Victoria. Two were associated with travel to Indonesia and five with

Figure 1. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 to 31 July 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 June to August data for the previous five years

travel to India. Two of these cases (a mother and baby) were linked; both travelled to India but it is unknown whether their exposure was there or whether household transmission occurred (the mother was asymptomatic).

Vaccine Preventable Diseases

All vaccine preventable diseases except mumps had fewer reports this month than for the 5-year-mean for July: there were no reports of diphtheria, *Haemophilus influenzae* type b, poliomyelitis and tetanus.

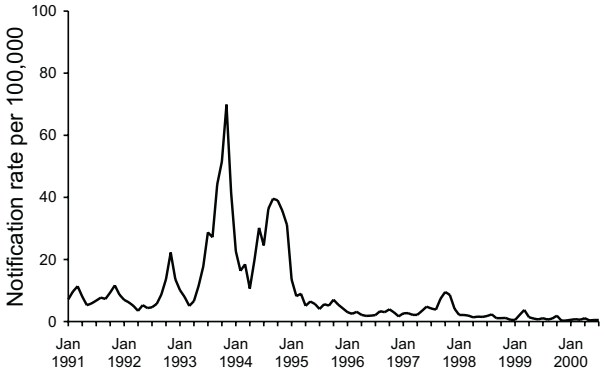
Mumps

The increase in the notification rate (1.2/100,000 population) for mumps was due to five reports in the Australian Capital Territory (19.2/100,000 population), two male and three female. There was no obvious epidemiological linkage between these five cases.

Measles

Measles cases continue to be at their lowest level since the national notification system began (Figure 2). Of the eleven cases in July 2000, five were reported in Victoria, two each in Western Australian and New South Wales and one each in South Australia and Queensland. Of the Victorian cases, four were linked to a receptionist at a doctor's surgery; all had measles virus of identical genotypes. The fifth case had no link to others and the virus was of a different genotype. Four of the five cases were in adults aged between 18 and 30 years; the remaining case was an infant less than 1-year-old. Of the Western Australian cases, one was a 25-year-old Thai postgraduate student, and one was a 24-year-old Malaysian postgraduate student, both studying in Perth, and both independently infected in their countries of origin before returning from holidays on separate flights via Singapore. These cases required extended follow-up of contacts as both went to general practices and public hospitals (one admitted) before the diagnosis was recognised. No secondary cases have been reported. One New South Wales case was a student whose siblings in Japan had measles. The student had measles on arrival and then travelled as part of a tour-group around Australia. Follow-up of potential cases is occurring. An unvaccinated individual in Queensland acquired measles locally.

Figure 2. Notification rate of measles, Australia, 1 January 1991 to 31 July 2000, by month of notification



Pertussis

The crude notification rate of pertussis in July 2000 was 14.5/100,000 population - less than the notification rates for July in previous years (20-60/100,000 population). Notification rates increased in New South Wales and the Australian Capital Territory (24.0/100,000 and 46.0/100,000 respectively; Figure 3).

No deaths have been reported so far this year in Australia. Preschool-aged children (1-4 years old) and infants (<1 year old) had the highest rates of reported disease (Figure 4). According to New South Wales Health press releases regarding the recent increase in pertussis year to date, to the end of July 1,370 cases were reported in New South Wales compared with 1,414 for all of 1999, 2,312 for all of 1998, and 4,251 for all of 1997. They advised that:

- All parents and doctors should ensure all children are fully immunised against pertussis (doses are due at 2, 4, 6, and 18 months, and at 4 years of age).
- Persons with symptoms of pertussis should seek medical diagnosis.
- Pertussis cases are infectious to others for up to 3 weeks after onset. Treatment with erythromycin given within 3 weeks of onset should render cases non-infectious after 5 days. Cases should not attend preschool or school (or other settings where there are susceptible persons, especially young children) while infectious.
- Pertussis can be prevented among household contacts of infectious cases with erythromycin.
- The treatment of choice for cases and their household contacts is erythromycin 40 to 50 mg/kg per day in 4 divided doses up to 1 gram per day for 10 days.
- Doctors, laboratories and hospitals should notify suspected cases to the local public health unit.

Figure 3. Notification rate of pertussis, New South Wales, Australian Capital Territory and Australia, 1 January 1991 to 31 July 2000, by month of notification

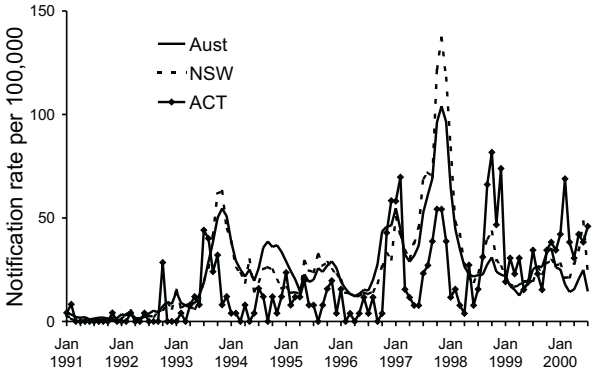
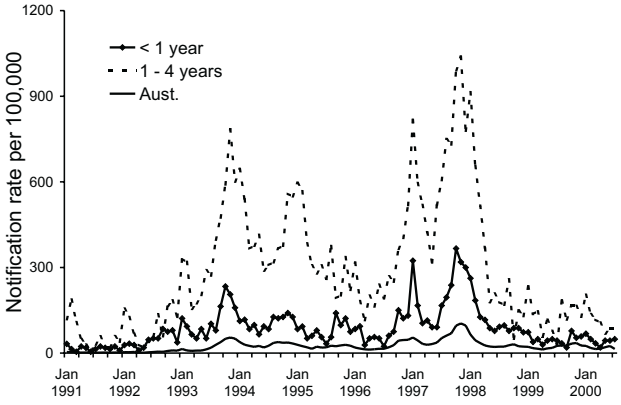


Figure 4. Notification rate of pertussis, Australia, 1 January 1991 to 31 July 2000, by month of notification



Encephalitis

The Health Department of Western Australia issued a press release warning people about the continued risk of mosquito-borne Australian Encephalitis in some northern regions of the State. This followed the worst recorded outbreak of the disease in Western Australia with 11 confirmed cases this season, two more than in the previous worst outbreak in 1993.

Legionellosis

In response to the increased number of legionella cases in Victoria, in June the Department of Human Services Working Party released a report entitled 'Legionnaires Disease: Managing the health risk associated with cooling towers'. The Victorian Government response to the report was released in July.

Meningococcal infections

There were 42 notifications of meningococcal infection in July 2000 - an incidence of 2.9/100,000 population (Figure 5). Of these cases, 17% were under 5 years of age, 21% were in the 5-14 year age group and 29% were in the 15-24 year age groups. The serogroups were available for 27 cases; of these 36%, 37% and 2% were serogroup B, C and W respectively. One sporadic case in Victoria involved an 18-year-old student for whom approximately 34 contacts were identified. The Communicable Disease Control Branch and Department of Human Services, Victoria, assessed the need for contacts to receive antibiotic chemoprophylaxis according to the National Health and Medical Research Council guidelines. Ten contacts were directed to metropolitan hospitals to receive medical assessment and antibiotic chemoprophylaxis.

Influenza

There were 183 laboratory reports of influenza for July 2000, a decrease from 687 in July 1999, but an increase from 111 in June 2000 (Figure 6). Of the laboratory reports received in July 2000 (weeks 27-30), 116 were influenza A and 55 were influenza B, with the weekly proportion of influenza B varying from 28% to 35% (Figure 7). The weekly percentage of influenza B has increased from the same period last year when it varied between 6% and 9%. Through the regular fortnightly teleconference of CDNANZ, the jurisdictions reported 82 laboratory confirmed influenza cases of which 48 were influenza A and 29 were influenza B.

Compared with June 2000, the percentage of Australia Post employees absent in July 2000 for 3 or more consecutive days was little changed (weeks 27-30, Figure 8). The Tropical influenza Surveillance Scheme (Northern Territory) reported the highest rate of influenza-like illness consultations (18/1,000 consultations) in July 2000 (weeks 27-30). In contrast, data from the Australian Sentinel Practice Research Network (ASPREN), New South Wales, Victoria and Northern Territory Sentinel Surveillance Schemes indicated that the influenza activities remained moderately low compared with last year (Figure 9).

Figure 5. Notification rate of meningococcal infection, Australia, 1 January 1991 to 31 July 2000, by month of notification

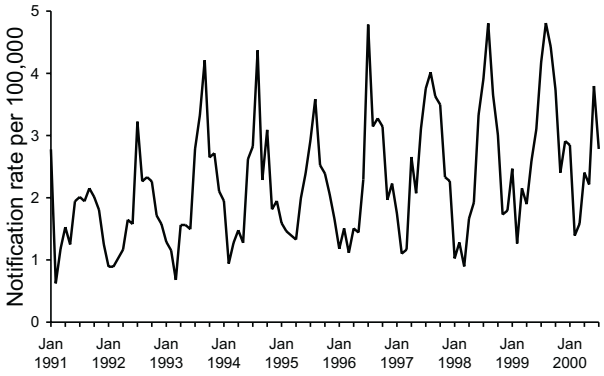


Figure 6. Laboratory reports of influenza, 1999 to 2000, by month of specimen collection

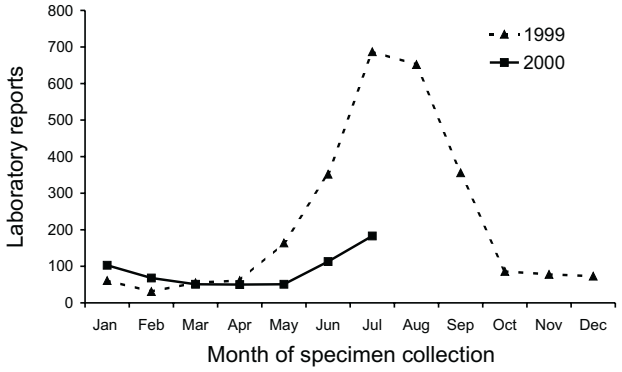


Figure 7. Laboratory reports of influenza, Australia, week 31 1999 to week 30 2000, by week of specimen collection

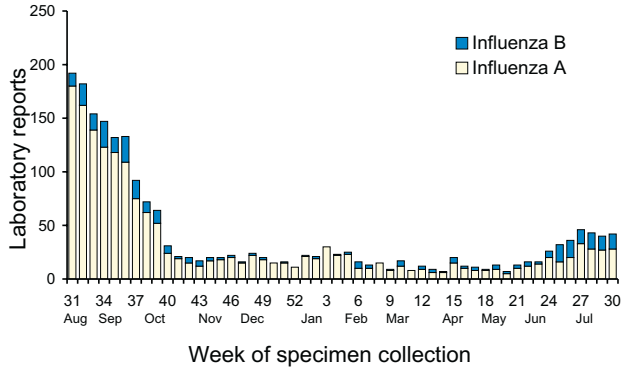


Figure 8. Absenteeism rates in Australia Post, 1999 and 2000 to July 31

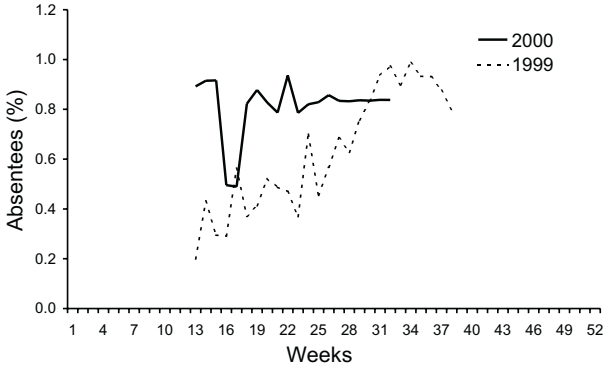
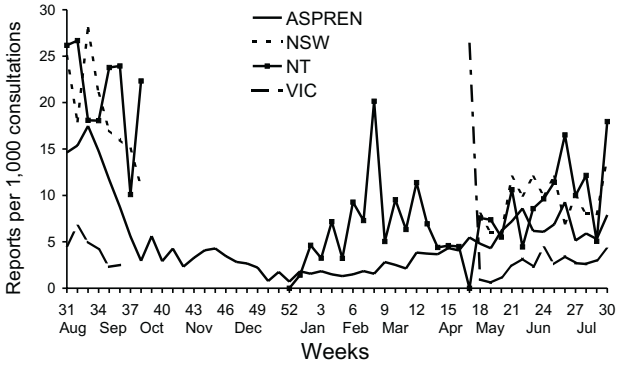


Figure 9. Sentinel general practitioner influenza consultation rates, week 31 1999 to week 30 2000, by scheme



Special report from Rob Menzies, Senior Surveillance Officer, New South Wales Health Department: Shigellosis outbreak among inner-Sydney men

Shigellosis is relatively uncommon in New South Wales, with fewer than five isolates received each month for serotyping by ICPMR laboratory, Westmead Hospital. In the last few weeks, New South Wales Health investigated an outbreak of shigellosis among inner-Sydney gay men.

Local doctors and laboratories reported that an increase in *Shigella sonnei* serotype g infections began in March 2000. Over 80 cases were identified from early March 2000 until mid-June 2000, compared to 21 cases in all of 1997. Over 90% of cases have been males mostly thought to be gay men between 20 and 40 years. Interviews with cases identified casual sex at sex-on-premises-venues as a likely risk factor for infection.

In response, New South Wales Health developed a prevention plan that included an education campaign among gay men, increased awareness among local doctors, and advice to sex-on-premises-venues on improving infection control. The number of reported cases has since declined.

Editorial comment. At present Shigellosis is not reportable in NSW but may be identified following notification of 'foodborne disease' or 'gastroenteritis in an institution'. This outbreak was brought to the attention of the NSW Public Health Unit by vigilant general practitioners and laboratory staff.

Tables

There were 5,064 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date in July 2000 (Table 1). Data by date of report for July 2000, are included in this issue of *Communicable Diseases Intelligence* (Table 3). The number of reports for selected diseases have been compared with a 5 year mean, calculated using June to August data for the previous 5 years (Figure 1).

There were 2,243 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 to 31 July 2000 (Tables 4 and 5).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 26 to 30, ending 30 July 2000, are included in this issue of *Communicable Diseases Intelligence* (Table 6).

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 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.*

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

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total July 2000[1]	Total June 2000[1]	Total July 1999[1]	Last 5 years mean	Year to date 2000	Last 5 years YTD mean	Ratio*
Bloodborne															
Hepatitis B (incident)	0	2	0	10	6	1	7	4	30	33	24	21	209	165	1.4
Hepatitis B (unspecified)[2]	1	99	0	62	6	3	180	58	409	698	765	570	4,541	4,082	0.7
Hepatitis C (incident)	0	2	0	-	3	0	3	7	15	31	25	15	245	109	1.0
Hepatitis C (unspecified)[2]	15	259	9	240	22	24	447	138	1,154	1,666	1,871	1,366	12,017	9,226	0.8
Hepatitis D	0	0	0	0	0	0	0	0	0	2	6	2	9	10	0.0
Gastrointestinal															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Campylobacterosis[3]	22	-	16	323	141	44	373	174	1,093	1,112	1,129	963	7,547	6,586	1.1
Haemolytic uraemic syndrome	NN	0	0	0	0	0	0	0	0	0	2	0	6	4	na
Hepatitis A	1	11	2	4	4	0	12	12	46	48	132	156	569	1,414	0.3
Hepatitis E	0	0	0	0	0	0	0	0	0	0	0	0	0	2	na
Listeriosis	0	0	0	0	0	0	3	0	3	6	3	4	46	37	0.8
Salmonellosis	4	17	22	72	23	2	57	57	254	390	356	349	3,980	4,401	0.7
Shigellosis[3]	1	-	2	3	1	0	10	9	26	40	45	48	297	435	0.5
SLTEC,VTEC[4]	NN	0	0	NN	2	0	0	NN	2	1	2	1	20	8	2.0
Typhoid	0	5	0	0	0	0	2	0	7	2	9	4	45	50	1.8
Yersiniosis[3]	0	-	0	8	0	0	0	0	8	3	12	15	47	151	0.5
Quarantinable															
Cholera	0	0	0	0	0	0	0	0	0	0	0	0	1	3	na
Plague	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Rabies	0	0	0	0	0	0	0	0	0	1	0	0	1	0	na
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Sexually transmissible															
Chancroid	0	0	0	0	0	0	0	0	0	0	0	0	0	1	na
Chlamydial infection[5]	14	136	68	367	78	24	188	118	993	1,363	1,249	810	9,434	5,770	1.2
Donovanosis	0	0	0	1	NN	0	0	0	1	2	3	3	11	27	0.3
Gonococcal infection[6]	1	35	83	76	14	0	58	76	343	508	411	368	3,716	2,768	0.9
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Syphilis[7]	2	34	18	59	0	2	0	6	121	147	172	141	1,017	1,016	0.9

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

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total July 2000[1]	Total June 2000[1]	Total July 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	na
Haemophilus influenzae type b	0	0	0	0	0	0	0	0	0	4	3	5	10	31	0.0
Measles	0	2	0	1	1	0	5	2	11	8	17	44	69	347	0.3
Mumps	5	3	0	0	1	1	6	3	19	22	19	15	129	99	1.3
Pertussis	12	128	0	20	16	4	47	2	229	405	401	416	2,143	2,791	0.6
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Rubella[8]	1	3	0	2	0	0	6	1	13	16	45	116	110	784	0.1
Tetanus	0	0	0	0	0	0	0	0	0	2	0	0	5	3	na
Vectorborne															
Arbovirus infection NEC	0	0	0	0	0	0	0	2	2	2	2	2	57	42	1.0
Barmah Forest virus infection	0	6	0	17	0	0	0	0	23	38	33	35	369	521	0.7
Dengue	0	0	0	2	0	0	0	0	2	7	1	7	195	111	0.3
Malaria	0	6	1	36	2	0	7	1	53	72	92	59	604	489	0.9
Ross River virus infection	1	15	1	29	3	1	6	14	70	180	81	120	3,493	4,315	0.6
Zoonoses															
Brucellosis	0	0	0	0	0	0	0	0	0	1	5	3	7	18	0.0
Hydatid infection	0	NN	0	0	0	0	0	1	1	0	4	5	18	23	0.2
Leptospirosis	0	1	0	4	0	0	1	0	6	13	15	13	147	126	0.5
Ornithosis	0	NN	0	NN	0	0	4	0	4	7	8	5	44	43	0.8
Q fever	0	3	0	19	0	0	3	3	28	30	36	45	279	312	0.6
Other															
Legionellosis	0	1	0	4	1	0	5	1	12	33	14	14	335	128	0.9
Leprosy	0	0	0	0	0	0	0	0	0	1	3	1	1	5	0.0
Meningococcal infection	0	18	0	6	5	0	8	5	42	60	66	56	270	229	0.8
Tuberculosis	2	9	0	0	0	0	26	7	44	49	94	84	475	603	0.5
Total	82	795	222	1,365	329	106	1,464	701	5,064	7,003	7,155	5,882	52,518	47,290	

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.

na Not applicable.

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

Table 2. Crude incidence of diseases by State or Territory, July 2000. (Rate per 100,000)

Disease[1]	State or Territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Bloodborne									
Hepatitis B (incident)	0.00	0.37	0.00	3.42	4.82	2.55	1.78	2.58	1.90
Hepatitis B (unspecified)[2]	3.83	18.53	0.00	21.18	4.82	7.66	45.84	37.40	25.88
Hepatitis C (incident)	0.00	0.37	0.00	-	2.41	0.00	0.76	4.51	0.95
Hepatitis C (unspecified)[2]	57.44	48.47	55.99	82.00	17.68	61.24	113.83	88.98	73.01
Hepatitis D	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Gastrointestinal									
Botulism	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Campylobacterosis[3]	84.25	-	99.54	110.35	113.32	112.28	94.99	112.20	69.15
Haemolytic uraemic syndrome	NN	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Hepatitis A	3.83	2.06	12.44	1.37	3.21	0.00	3.06	7.74	2.91
Hepatitis E	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Listeriosis	0.00	0.00	0.00	0.00	0.00	0.00	0.76	0.00	0.19
Salmonellosis	15.32	3.18	136.87	24.60	18.49	5.10	14.52	36.75	16.07
Shigellosis[3]	3.83	-	12.44	1.02	0.80	0.00	2.55	5.80	1.64
SLTEC,VTEC[4]	NN	0.00	0.00	NN	1.61	0.00	0.00	NN	0.13
Typhoid	0.00	0.94	0.00	0.00	0.00	0.00	0.51	0.00	0.44
Yersiniosis[3]	0.00	-	0.00	2.73	0.00	0.00	0.00	0.00	0.51
Quarantinable									
Cholera	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Plague	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Rabies	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Viral haemorrhagic fever	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Yellow fever	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Sexually transmissible									
Chancroid	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Chlamydial infection[5]	53.61	25.45	423.06	125.39	62.69	61.24	47.88	76.09	62.83
Donovanosis	0.00	0.00	0.00	0.34	-	0.00	0.00	0.00	0.06
Gonococcal infection[6]	3.83	6.55	516.38	25.97	11.25	0.00	14.77	49.01	21.70
Lymphogranuloma venereum	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Syphilis[7]	7.66	6.36	111.99	20.16	0.00	5.10	0.00	3.87	7.66
Vaccine preventable									
Diphtheria	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Haemophilus influenzae type b	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Measles	0.00	0.37	0.00	0.34	0.80	0.00	1.27	1.29	0.70
Mumps	19.15	0.56	0.00	0.00	0.80	2.55	1.53	1.93	1.20
Pertussis	45.96	23.96	0.00	6.83	12.86	10.21	11.97	1.29	14.49
Poliomyelitis	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Rubella[8]	3.83	0.56	0.00	0.68	0.00	0.00	1.53	0.64	0.82
Tetanus	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Vectorborne									
Arbovirus infection NEC	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1.29	0.13
Barmah Forest virus infection	0.00	1.12	0.00	5.81	0.00	0.00	0.00	0.00	1.46
Dengue	0.00	0.00	0.00	0.68	0.00	0.00	0.00	0.00	0.13
Malaria	0.00	1.12	6.22	12.30	1.61	0.00	1.78	0.64	3.35
Ross River virus infection	3.83	2.81	6.22	9.91	2.41	2.55	1.53	9.03	4.43

Table 2 (continued). Crude incidence of diseases by State or Territory, July 2000. (Rate per 100,000)

Disease[1]	State or Territory ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Zoonoses									
Brucellosis	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Hydatid infection	0.00	NN	0.00	0.00	0.00	0.00	0.00	0.64	0.06
Leptospirosis	0.00	0.19	0.00	1.37	0.00	0.00	0.25	0.00	0.38
Ornithosis	0.00	NN	0.00	NN	0.00	0.00	1.02	0.00	0.25
Q fever	0.00	0.56	0.00	6.49	0.00	0.00	0.76	1.93	1.77
Other									
Legionellosis	0.00	0.19	0.00	1.37	0.80	0.00	1.27	0.64	0.76
Leprosy	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Meningococcal infection	0.00	3.37	0.00	2.05	4.02	0.00	2.04	3.22	2.66
Tuberculosis	7.66	1.68	0.00	0.00	0.00	0.00	6.62	4.51	2.78
Total	314.03	148.79	1,381.16	466.35	264.42	270.49	372.82	452.01	320.39

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.

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

Disease[1]	State or Territory ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total this period	Year to date total
Bloodborne										
Hepatitis B (incident)	0	2	0	11	7	2	11	6	39	221
Hepatitis B (unspecified)[2]	2	176	0	61	24	4	182	73	522	4,764
Hepatitis C (incident)	1	3	0	-	7	0	4	10	25	264
Hepatitis C (unspecified)[2]	22	416	20	250	55	29	449	150	1,391	12,496
Hepatitis D	0	0	0	0	0	0	0	0	0	9
Gastrointestinal										
Botulism	0	0	0	0	0	0	0	0	0	0
Campylobacterosis[3]	31	-	21	309	169	42	376	201	1,149	7,654
Haemolytic uraemic syndrome	NN	0	0	0	0	0	0	0	0	6
Hepatitis A	1	16	3	3	4	0	12	14	53	606
Hepatitis E	0	0	0	0	0	0	0	0	0	0
Listeriosis	0	0	0	1	0	0	3	0	4	47
Salmonellosis	5	37	28	80	28	2	80	62	322	4,165
Shigellosis[3]	1	-	7	5	1	0	11	12	37	309
SLTEC,VTEC[4]	NN	0	0	NN	2	0	0	NN	2	23
Typhoid	0	4	0	0	0	0	2	0	6	50
Yersiniosis[3]	0	-	0	8	0	0	0	0	8	47

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

Disease[1]	State or Territory ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total this period	Year to date total
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]	15	207	101	399	124	30	263	155	1,294	9,695
Donovanosis	0	0	0	1	NN	0	0	1	2	12
Gonococcal infection[6]	0	53	132	109	45	0	63	100	502	3,806
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0
Syphilis[7]	2	59	17	72	4	2	0	13	169	1,074
Vaccine preventable										
Diphtheria	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	0	1	0	0	0	0	0	1	12
Measles	0	2	0	2	1	0	2	2	9	72
Mumps	7	5	0	0	3	1	7	3	26	133
Pertussis	21	309	0	28	28	4	62	6	458	2,407
Poliomyelitis	0	0	0	0	0	0	0	0	0	0
Rubella[8]	0	4	0	3	0	0	6	2	15	115
Tetanus	0	0	0	0	1	0	0	0	1	6
Vectorborne										
Arbovirus infection NEC	0	0	0	1	0	0	0	2	3	58
Barmah Forest virus infection	0	8	0	13	0	0	0	2	23	381
Dengue	0	0	8	2	0	0	0	0	10	216
Malaria	1	6	4	35	3	0	8	1	58	616
Ross River virus infection	2	31	1	39	3	0	10	25	111	3,698
Zoonoses										
Brucellosis	0	0	0	0	0	0	0	0	0	8
Hydatid infection	0	NN	0	0	0	0	0	1	1	18
Leptospirosis	0	1	0	1	0	0	1	0	3	150
Ornithosis	0	NN	0	NN	0	0	9	0	9	53
Q fever	0	5	0	25	0	0	3	4	37	294
Other										
Legionellosis	0	1	0	4	1	0	8	3	17	341
Leprosy	0	0	1	0	0	0	0	0	1	2
Meningococcal infection	0	25	0	4	5	0	11	6	51	277
Tuberculosis	3	17	5	22	0	0	29	10	86	578
Total	114	1,387	349	1,488	515	116	1,612	864	6,445	54,684

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 State or Territory[1] for the reporting period 1 to 31 July 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	0	0	0	0	0	0	1	2	3	6	29	136
Mumps virus	0	0	0	0	1	0	0	2	3	2	34	32
Rubella virus	0	1	0	0	1	0	0	1	3	92	25	141
Hepatitis viruses												
Hepatitis A virus	0	0	1	3	4	0	0	6	14	97	108	296
Arboviruses												
Ross River virus	0	1	1	8	2	0	1	12	25	174	1,089	1,219
Barmah Forest virus	0	1	0	4	0	0	0	0	5	40	109	156
Dengue not typed	0	0	0	0	0	0	0	2	2	0	166	33
Murray Valley encephalitis virus	0	0	0	0	0	0	0	1	1	0	19	2
Kunjin virus	0	0	0	0	0	0	0	1	1	0	4	5
Flavivirus (unspecified)	0	0	0	1	0	0	0	0	1	0	38	17
Adenoviruses												
Adenovirus type 40	0	0	0	0	0	0	0	6	6	11	75	44
Adenovirus type 41	0	0	0	0	0	0	0	1	1	0	1	0
Adenovirus not typed/pending	0	11	0	1	40	0	2	26	80	85	622	623
Herpes viruses												
Cytomegalovirus	1	11	1	11	30	2	11	4	71	121	691	724
Varicella-zoster virus	1	12	1	27	10	0	6	37	94	310	842	1,116
Epstein-Barr virus	0	6	1	25	43	0	4	27	106	480	1,312	1,666
Other DNA viruses												
Contagious pustular dermatitis (Orf virus)	0	0	0	0	0	0	0	1	1	0	7	6
Parvovirus	0	1	0	0	3	0	11	21	36	124	202	332
Picornavirus family												
Coxsackievirus A16	0	1	0	0	0	0	0	0	1	2	4	14
Rhinovirus (all types)	0	16	0	0	1	0	0	16	33	45	242	231
Enterovirus not typed/pending	0	0	0	2	0	0	10	24	36	75	576	459
Ortho/paramyxoviruses												
Influenza A virus	4	43	0	3	58	0	8	6	122	627	467	1,256
Influenza A virus H3N2	0	0	0	0	0	0	0	1	1	5	2	28
Influenza B virus	2	23	0	0	25	0	6	4	60	55	150	126
Parainfluenza virus type 1	0	1	0	2	17	0	0	9	29	7	209	30
Parainfluenza virus type 2	0	0	0	0	2	0	0	1	3	11	24	94
Parainfluenza virus type 3	0	3	0	0	7	0	0	10	20	116	129	324
Respiratory syncytial virus	4	164	0	28	74	10	62	463	805	955	2,068	2,000
Other RNA viruses												
Rotavirus	4	90	0	0	51	1	1	43	190	306	543	844
Other												
Chlamydia trachomatis not typed	2	25	6	71	35	1	3	74	217	855	1,954	2,404
Chlamydia psittaci	0	0	0	0	0	0	6	1	7	6	57	55
Mycoplasma pneumoniae	0	1	0	15	7	1	28	6	58	235	354	768
Coxiella burnetii (Q fever)	0	0	0	1	0	0	4	4	9	96	41	191
Streptococcus group A	0	2	8	14	0	0	14	0	38	207	219	281
Yersinia enterocolitica	0	1	0	0	0	0	0	0	1	0	9	8
Bordetella pertussis	0	4	0	3	5	0	29	2	43	383	317	705
Legionella pneumophila	0	0	0	0	0	0	11	1	12	0	26	15
Legionella longbeachae	0	0	0	0	1	0	0	1	2	1	37	20

Table 4 (continued). Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 31 July 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				
Cryptococcus species	0	0	0	0	1	0	0	0	1	0	9	6
Leptospira species	0	0	0	3	0	0	0	0	3	46	35	62
Treponema pallidum	0	1	20	33	40	0	0	1	95	343	462	509
Entamoeba histolytica	0	0	0	0	0	0	1	0	1	2	10	3
Toxoplasma gondii	0	0	0	0	1	0	0	0	1	0	8	5
Echinococcus granulosus	0	0	0	0	1	0	0	1	2	0	16	0
Total	18	419	39	255	460	15	219	818	2,243	5,920	13,341	16,986

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. From January 2000 data presented are for reports with report dates in the current period. Previously reports included all data received in that period.
3. Totals comprise data from all laboratories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

\- No data received this period.

Table 5. Virology and serology laboratory reports by contributing laboratories for the reporting period 1 to 31 July 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	176	195
	New Children's Hospital, Westmead	179	108
New South Wales	Repatriation General Hospital, Concord	-	-
	Royal Prince Alfred Hospital, Camperdown	58	78
	South West Area Pathology Service, Liverpool	-	-
Queensland	Queensland Medical Laboratory, West End	311	284
	Townsville General Hospital	7	13
South Australia	Institute of Medical and Veterinary Science, Adelaide	456	297
Tasmania	Northern Tasmanian Pathology Service, Launceston	14	18
	Royal Hobart Hospital, Hobart	-	-
Victoria	Monash Medical Centre, Melbourne	-	3
	Royal Children's Hospital, Melbourne	116	78
	Victorian Infectious Diseases Reference Laboratory, Fairfield	104	92
Western Australia	PathCentre Virology, Perth	389	246
	Princess Margaret Hospital, Perth	427	300
	Western Diagnostic Pathology	6	10
Total		2,243	1,722

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 6. Australian Sentinel Practice Research Network reports, weeks 26 to 30, 2000

Week number	26		27		28	
Week ending on	2 July 2000		9 July 2000		16 July 2000	
Doctors reporting	66		61		61	
Total encounters	7,544		7,392		7,423	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	70	9.3	38	5.1	44	5.9
Chickenpox	13	1.7	11	1.5	11	1.5
Gastroenteritis	51	6.8	60	8.1	71	9.6
Gastroenteritis with stool culture	6	0.8	11	1.5	10	1.3
ADT immunisations	36	4.8	30	4.1	24	3.2

Table 6 (continued). Australian Sentinel Practice Research Network reports, weeks 26 to 30, 2000

Week number	29		30	
Week ending on	23 July 2000		30 July 2000	
Doctors reporting	64		64	
Total encounters	8,115		7,882	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	44	5.4	63	8.0
Chickenpox	8	1.0	11	1.4
Gastroenteritis	64	7.9	70	8.9
Gastroenteritis with stool culture	10	1.2	14	1.8
ADT immunisations	23	2.8	32	4.1

Additional Reports

Gonococcal surveillance

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

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various States and Territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5% or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment (Anonymous. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37). Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented.

Reporting period 1 January to 31 March 2000

The AGSP laboratories examined a total of 938 isolates in this quarter, virtually the same number as in this period in 1999. About 38% of this total was from New South Wales, 22% from Victoria, 16% from Queensland, 11% from the Northern Territory, 8% from Western Australia and 4% from South Australia. There were few isolates from other centres.

Penicillins

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

About 22% of all isolates were penicillin-resistant by one or more mechanisms – 10% by penicillinase production and 12% by chromosomal mechanisms (CMRNG). The penicillin-resistant isolates comprised about half the isolates in South Australia and about a quarter of all isolates in New South Wales and Queensland, while about 15% of gonococci in Victoria and Western Australia were penicillin-resistant. In the Northern Territory, 2% of isolates were penicillin-resistant.

The number of PPNG isolated across Australia (91) increased slightly in this quarter compared with the corresponding period in 1999 (88). However the distribution of PPNG has altered. The highest proportion of PPNG was found in isolates from South Australia (24%), Queensland (15%) and Western Australia (14%) whereas the number (34, 14) and proportion (9.4%, 6.8%) of PPNG in New South Wales and Victoria respectively decreased. A single PPNG was isolated in the Northern Territory. Acquisition data on PPNG indicated a high rate of local acquisition throughout Australia. South-East Asian countries were the main source of external acquisition.

More isolates were resistant to the penicillins by separate chromosomal mechanisms (119). These CMRNG were especially prominent in New South Wales (21%) and South Australia (24%) with substantial proportions also in Queensland (8%) and Victoria (10%). Only one strain of this type was isolated in the Northern Territory.

Ceftriaxone and spectinomycin

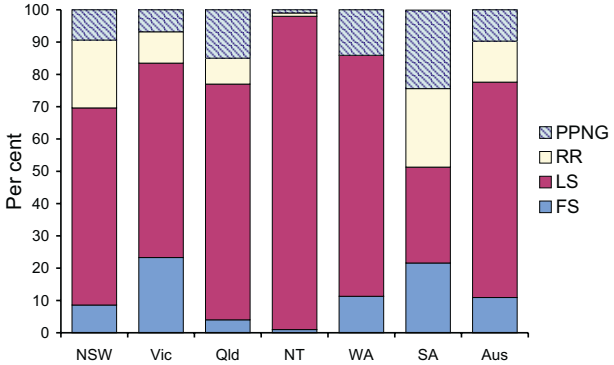
All isolates in Australia were again susceptible to these injectable agents, with the exception of one strain with decreased ceftriaxone susceptibility.

Quinolone antibiotics

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

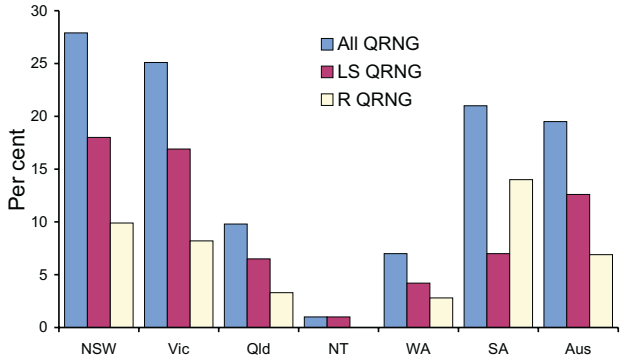
The total number (183) and proportion (20%) of all QRNG was again high and much increased over the first quarter of 1999 (106 isolates, 11%) (Figure 11). QRNG were present in all centres except Tasmania and the Australian Capital Territory. High rates were maintained in New South Wales (28%) and Victoria (25%) and together these regions accounted for 85% of QRNG isolated. QRNG were prominent also in South Australia (21% of isolates) and Queensland (10%). Of Western Australian isolates,

Figure 10. Gonococci isolated in Australia, 1 January to 31 March 2000, by penicillin-susceptibility and by region



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

Figure 11. Quinolone-resistance of *N. gonorrhoeae*, 1 January to 31 March 2000, Australia, by region



LS QRNG less sensitive quinolone-resistant *N. gonorrhoeae* (Ciprofloxacin MICs 0.06 to 0.5 mg/L)

R QRNG fully resistant quinolone-resistant *N. gonorrhoeae* (Ciprofloxacin MICs 1 mg/L)

7% were QRNG and a single QRNG was isolated in the Northern Territory. Thirty-six of the New South Wales and 17 of the Victorian QRNG exhibited high level resistance (MIC ciprofloxacin 1 mg/L) and higher level QRNG were also seen in Queensland, South Australia and Western Australia. Local acquisition became increasingly prominent and MICs ranged up to 16mg/L. However about two thirds of the QRNG were in the 'less sensitive' MIC range 0.06 to 0.5 mg/L and were found exclusively in males. Again the bulk of this group of isolates (101 of 118) was found in New South Wales and Victoria and infections with them were locally acquired.

High level tetracycline resistance (TRNG)

The number (89) and proportion (9.4%) of TRNG detected were similar to those noted for the first quarter of 1999. TRNG represented 19% of gonococci from South Australia, 14% of isolates from Queensland and Western Australia, 9% from New South Wales and 8% from Victoria. A single TRNG was isolated in the Northern Territory.

Adverse Events Following Immunisation Surveillance Scheme

Adverse Events data collected by both the Serious Adverse Events Following Vaccination Surveillance Scheme (SAEFVSS) for children and the Adverse Drug Reaction Scheme for children and adults are included in this report. This is a change from previous reports that have only included adverse events data collected by the SAEFVSS. Adverse events are classified as described in the Australian Immunisation Handbook 7th edition in which more details of the reporting of adverse events following immunisation can be found. (National Health and Medical Research Council. The Australian Immunisation Handbook. 7th ed. Canberra: Australian Government Publishing Services, 2000).

Acceptance of a report does not imply a causal relationship between the administration of the vaccine and the reported outcome, or that the report has been verified as to the accuracy of its contents.

It is estimated that 250,000 doses of vaccines are administered every month to Australian children under the age of 6 years.

Result for the reporting period 1 January to 30 June 2000

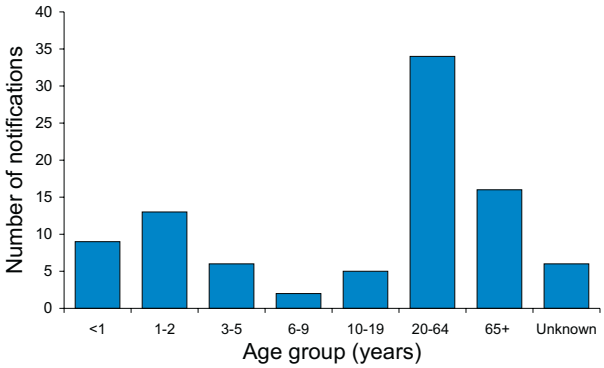
For this 6 months reporting period 164 notifications of adverse events following immunisation were received. These did not include notifications from the SAEFVSS in New South Wales. The most frequent sources were medical practitioners (38%), pharmaceutical companies (22%), and State/Territory Health Departments (20%), with the remainder from pharmacists (4%) and others (3%). Excluding nine notifications for which the reporting State/Territory was unknown, notifications for this period were received from the Australian Capital Territory (1%), New South Wales (22.5%), Northern Territory (8%), Queensland (14%), South Australia (13%), Tasmania (3%), Victoria (32%), and Western Australia (6%).

Of the 164 notifications, the assessed association with immunisation was certain (30%), probable (7%), possible (49%) and unknown (13%). Most certain associations were local reactions.

Most of the 164 notifications occurred in the 20 to 64 year age group (41%) followed by those under 10 years (32%) and those over 64 years (15%). Of the 53 notifications for those under 10 years, most were under 6 years, with 28% in those less than 1 year and 60% in those aged between 1 and 5 years (Figure 12). Most notifications were related to the administration of one vaccine only (78%).

For each of the 164 notifications, the severity was reported for 32% and the outcome for 72%. Of the 52 reports of severity, 65% required a doctor's visit, 29% needed hospitalisation and two were reported as 'life-threatening'. These two reports included a report from Queensland of thrombocytopenia within 1 week of MMR immunisation in a 15-month-old, and a report from Victoria of meningitis within 1 day of hepatitis B immunisation in a 13-year-old.

Figure 12. Notifications of adverse events following immunisation, 1 January to 30 June 2000, by age group



Of the 118 reports with a reported outcome, 65% had recovered, 33% had not recovered completely by the time of notification, and there were three deaths. The deaths, reported from Tasmania and Victoria, occurred within 1 day of the birth-dose of hepatitis B immunisation in a child of 2 days, within 1 week of immunisation with OPV plus DTP and Hib in a 15-month-old, and within 1 week of cholera immunisation in a 28-year-old. There were no notifications associated with OPV alone. The cholera vaccine was not the oral vaccine.

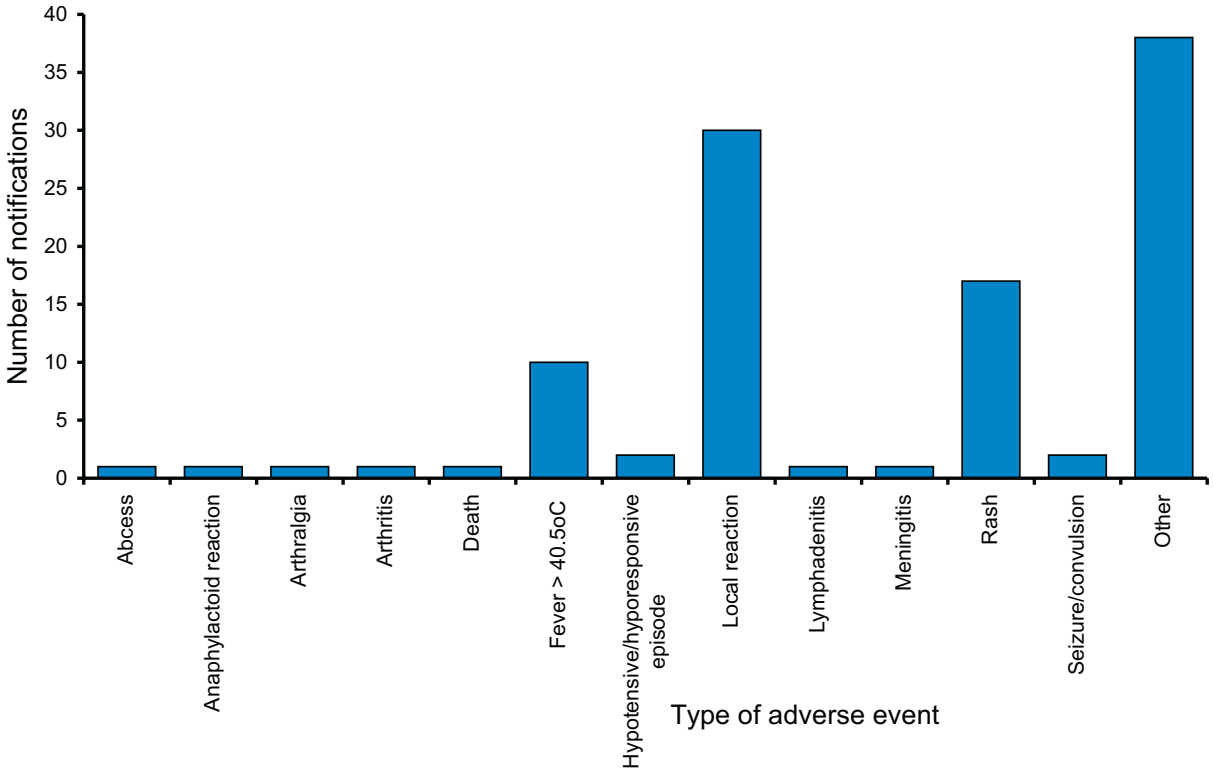
Each notification was associated with one or more adverse events. In total there were 184 adverse events reported for this period. The most frequently reported were other reactions (31%), local reaction (28%), rash (15%) and fever of over 40.5°C (9%) (Figure 13). Other reactions included headache, myalgia, gastrointestinal symptoms (such as nausea, vomiting and diarrhoea) and vasovagal type symptoms (such as hyperventilation, parasthesia and palpitations). The most serious adverse events notified included anaphylactoid reaction (1%), meningitis (0.5%), seizure or convulsion (2%) and thrombocytopenia (0.5%) and the three reported deaths (2%).

For 22% of encounters, more than one vaccine was administered. It was not possible to know which vaccine was associated with any adverse event other than local reactions; hence any adverse event was counted for each vaccine. In total there were 215 adverse events associated with the different vaccines administered. Most adverse events were reported following the administration of Influenza vaccines (22%), Diphtheria Tetanus Pertussis (DTP) vaccines (15%), and Measles Mumps Rubella (MMR) vaccine (8%). Adverse events associated with the DTP vaccines were mostly associated with the acellular DTP vaccines as whole cell DTP vaccines are no longer widely used. Most adverse events associated with Influenza, DTP, and MMR vaccines occurred when they were used alone.

Dose information was recorded for 60 administered vaccines. Most adverse events with DTP vaccines were associated with dose 4 (11/17, 65%); the dose was unspecified for an additional 15 reports. The dose was not recorded for any influenza vaccines but is likely to have been dose 1 as the recommendation for influenza immunisation mostly applies to adults over 65 years who require one dose each year. Adverse events with MMR vaccines were evenly divided between the first (4/9, 44%) and second (5/9, 56%) doses; the dose was unspecified for an additional nine reports.

Editorial statement. The Australian Immunisation Handbook (7th edition, Appendix 6) defines vaccination as '*the administration of a vaccine: if vaccination is successful it results in immunity*' and immunisation as '*the process of inducing immunity to an infectious agent by administering a vaccine*'. An Adverse Event Following Immunisation (AEFI) is defined by the Australian Immunisation Handbook (7th edition, page 22) as '*a serious uncommon or unexpected event following immunisation. Such an event may or may not be caused by the vaccine or may occur by chance after immunisation*'. The use of the term AEFI is a change (from Adverse Event Following Vaccination) and is consistent with World Health Organization terminology.

Figure 13. Notifications by reported adverse event following immunisation, 1 January to 30 June 2000, by type of adverse event



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 medical practitioner 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 March 2000, as reported to 30 June 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 March 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	0	0	2	0	0	2	2	6	9	20	18
	Male	0	8	1	12	5	0	15	2	43	68	157	161
	Sex not reported	0	1	0	0	0	0	0	0	1	0	3	0
	Total[1]	0	9	1	14	5	0	17	4	50	77	180	179
AIDS diagnoses	Female	0	0	0	0	0	0	0	0	0	4	7	5
	Male	0	3	0	1	1	0	5	1	11	12	43	34
	Total[1]	0	3	0	1	1	0	5	1	11	16	50	39
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	0	3	0
	Male	0	2	0	0	0	0	3	0	5	9	18	36
	Total[1]	0	2	0	0	0	0	3	0	5	9	21	37

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

Table 8. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 31 March 2000, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	26	607	11	155	61	5	214	118	1,197
	Male	223	10,987	110	2,002	679	79	3,907	920	18,907
	Sex not reported	0	249	0	0	0	0	24	0	273
	Total[1]	249	11,863	121	2,164	740	84	4,159	1,042	20,422
AIDS diagnoses	Female	9	188	1	48	25	3	69	26	369
	Male	86	4,648	35	824	347	44	1,624	351	7,959
	Total[1]	95	4,848	36	874	372	47	1,701	379	8,352
AIDS deaths	Female	4	113	0	32	15	2	49	16	231
	Male	66	3,172	24	567	231	29	1,273	248	5,610
	Total[1]	70	3,293	24	601	246	31	1,328	265	5,858

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

Letter to the Editor

Immunisation coverage estimates

Christine Selvey
Head Immunisation, Centre for Disease Control,
Northern Territory Health Services, Casuarina, NT 0810

To the Editor: The article by Hull and McIntyre[1] examines reasons why data from the Australian Childhood Immunisation Register (ACIR) may underestimate true immunisation coverage in Australia.

There are several anomalies in the data presented that are not addressed by the authors. Furthermore Hull and McIntyre present jurisdictional aggregate data from the ACIR that indicate low immunisation coverage in the Northern Territory (NT), but do not discuss reasons why the NT coverage rates do not reflect the true immunisation status of NT children.

The first anomaly is that the jurisdictions with the longest lag times (the time between the date of the immunisation and the date of processing by the Health Insurance Commission) for birth cohort 1, the Northern Territory (NT) and Queensland, have the lowest increase in coverage due to late notifications. This is counter-intuitive and requires further analysis on what the lag time is actually measuring.

The second anomaly within data presented by Hull and McIntyre is found in Table 5 of their article. This table examines coverage of measles, mumps and rubella vaccination for birth cohort 3 (born 1 July 1996 to 30 September 1996) assessed on 30 September 1998 and again on 30 June 1999. For the NT, the coverage between these two dates falls by 0.9 per cent. The authors do not comment on this inconsistency. The most likely explanation is that the two assessments are not performed on records of exactly the same children. That is, the children who were assessed as being resident in the NT in December 1998 are not the same children who were assessed as being resident in the NT in June 1999. Clearly this has major implications for interpretation of the jurisdictional data presented by Hull and McIntyre, particularly for the NT where the population has an extremely high level of interstate and overseas mobility, but also for other areas.

The third anomaly is that for birth cohort 1 (Table 1) the percentage coverage due to Immunisation History Form notifications (2.7%) for the NT is greater than the increase in coverage due to late notifications (1.2%). The Immunisation History Forms all represent late notifications, but the coverage rate has not increased by as much as the level of history forms. Again the authors did not address this inconsistency. I believe that the most likely explanation is the high rate of interstate migration of families who live in the NT.

I believe that the existence of these anomalies compromises the conclusions drawn by Hull and McIntyre and gives an incorrect impression of poor performance by NT immunisation service providers. Other inaccuracies in NT data are due to difficulties matching NT immunisation records with ACIR records generated by Medicare enrolments, and have consistently resulted in ACIR coverage rates that are well below those estimated by the NT Childhood Immunisation Database.[2,3]

For example, Thorman and Merianos[2] have estimated the NT coverage rate for full immunisation for the birth cohort 1 (1 January 1996 to 31 March 1996) to be 75%, which is significantly higher than the revised figure of 66.0% quoted in Table 1 of the article by Hull and McIntyre. Similarly, Thorman and Merianos estimated the coverage for MMR vaccine for this same birth cohort to be 91%, compared with the revised estimate of 71.4% from the ACIR. They based their estimates on the NT Childhood Immunisation Database (CID) and, despite the fact that all of the immunisations on the CID have been transmitted to the ACIR, a large gap in the two estimates of coverage remains.

The major reason that the ACIR estimates of immunisation coverage in the NT are inaccurate is that of poor matching of NT CID records with Medicare-generated ACIR records.[3] Most vaccine service providers in the NT do not access Medicare and so do not collect Medicare numbers. Immunisations transmitted to ACIR without a Medicare number are matched on name and date of birth and postcode, which results in a large proportion of records that do not match. Indigenous cultural practices, where name changes are common and the spelling of names is variable, compound the problem in a jurisdiction where 40% of the children aged 0-7 years are indigenous.

Another problem is that NT children have a high proportion of duplicate Medicare registrations. In a culture where children are highly mobile between different care givers and between different health care providers, and commonly have changes to their name and imprecise dates of birth, duplicate Medicare numbers are inevitable if health care providers are to be paid. Where there are duplicate (or triplicate) ACIR records, the immunisation data may sit in neither or one of the duplicate records, or be split between them.

These and other related problems are worse for the older cohorts on the ACIR than for the younger. The importance of reporting of a Medicare number with each immunisation notification has been stressed and, by May 2000, 81% of records on the NT CID contained a Medicare number. A process is nearing completion for all indigenous neonates born in NT public hospitals to be enrolled with Medicare prior to hospital discharge. In 1999 a major project was undertaken jointly by Territory Health Services, General Practice Divisions Northern Territory, the Commonwealth Department of Health and Aged Care and Medicare to "clean" ACIR data for NT children and put in place sustainable processes to address these issues. This project resulted in substantial increases in NT coverage rates as estimated by ACIR for the current cohorts, but there is considerable outstanding work to be done, especially for older cohorts.[3]

1. Hull BP, McIntyre PB. A re-evaluation of immunisation coverage estimates from the Australian Childhood Immunisation Register, *Commun Dis Intell* 2000;24:161-164.
2. Thorman S, Merianos A. Immunisation coverage of children 12-14 months in real time. *Northern Territory Disease Control Bulletin* 1997;4(4):1.
3. Merianos A. Northern Territory vaccination coverage statistics, ACIR third quarter assessments to 30 September 1997. *Northern Territory Disease Control Bulletin* 1998; 5(2):20.

MAE Conference 2001

"Charting New Directions: cutting-edge issues in applied epidemiology"

1-2 April 2001, Hyatt Hotel, Canberra

The 3rd Master of Applied Epidemiology (MAE) Conference will be held in conjunction with the Communicable Diseases Network Australia New Zealand (CDNANZ) Conference on 2-3 April, 2001, at the Hyatt Hotel in Canberra. Joint registration will be offered.

The MAE conference aims to promote discussion, innovation and collaborative action on cutting-edge issues in applied epidemiology within Australia and the Asia-Pacific region. The invited keynote speakers are:

Professor T McMichael, London School of Hygiene and Tropical Medicine/NCEPH
Dr Yvan Souares, Secretariat of the Pacific Community
Dr Dave Durrheim, Director of Disease Control, Mpumalanga Province, South Africa
Dr Tom Kiedrynski, Secretariat of the Pacific Community
Dr Taka Ohyama, FETP, Japan
Dr Rob Condon, WHO consultant, East Timor

For further MAE Conference information and application papers contact the MAE administration office, NCEPH, Phone (02) 6249 2790, Fax (02) 6249 0740, Email: MAE(DC) ros.hales@anu.edu.au or MAE(IH) elizabeth.lovell@anu.edu.au

Communicable Diseases Control Conference 2001

"Harnessing New Technologies"

2-3 April 2001, Hyatt Hotel, Canberra

The Communicable Diseases Network Australia New Zealand (CDNANZ) 2 day Communicable Diseases Control Conference will be held in conjunction with the MAE Conference of 1-2 April 2001.

The conference aims to promote evidence-based discussion around emerging disease control themes, including new laboratory technologies, epidemiological techniques, and information technologies. This conference is relevant to public health professionals and students working in all aspects of communicable disease control and may have particular relevance to rural and remote practitioners. The conference will aim to deliver recommendations for improvements in communicable disease control in Australia and New Zealand.

Abstracts for oral and poster presentations on all aspects of communicable disease control activities are now sought, especially those focussing on epidemiology, laboratory, prevention/control, and/or public health control aspects. Abstracts on the official submission form must reach Consec Conference Management, PO Box 3127, Belconnen Delivery Centre, ACT 2617 and also emailed to diseases@consec.com.au by 10 November 2000. Further information will be available shortly on www.health.gov.au/pubhlth/cdi/cdconf.htm

The CDNANZ/MAE Conference Dinner will be at the Hyatt Hotel on Sunday 1 April 2001

A half day workshop on the
Epi Info 2000 public domain epidemiological software

will be held on Wednesday 4 April, 2001, from 9.30am to 12.30pm at The Australian National University. Contact Ros Hales (ros.hales@anu.edu.au) to register your interest in attending.

In case you missed it

Meningococcal disease in Saudi Arabia

The Committee on Epidemic Diseases. Meningococcal disease caused by *Neisseria meningitidis* serogroup W135 among travellers returning from Saudi Arabia. *Epidemiological News Bulletin*. 2000;26(7):42-43.

In April 2000, four cases of meningococcal disease involving *Neisseria meningitidis* serotype W135 related to travel to Saudi Arabia were reported in Singapore. One case was a 45-year-old Australian who had worked in Saudi Arabia and arrived in Singapore on 30 March 2000. He had onset of fever, sore throat and generalised petechial rash on 2 April 2000. He developed severe complications, including necrotising gangrene of the extremities and died on 21 April 2000 from fulminant meningococcal septicaemia. *N. meningitidis* W135 was isolated from his blood culture.

Three of the cases involving *N. meningitidis* serogroup W135 were linked to individuals returning from the annual Haj pilgrimage in late March-early April 2000. Pilgrims going to the annual Haj (major pilgrimage) or Umrah (minor pilgrimage) are required by Saudi Arabia to be vaccinated against two serogroups (A and C) of *N. meningitidis*. As the bivalent vaccine does not protect against W135 serogroup, pilgrims should be protected with the quadrivalent vaccine against serogroups A, C, Y and W135.

vCJD in France

Oppenheim C, Brandel J-P, Hauw J-J, Deslys J-P, Fontaine B. MRI and the second French case of vCJD. *The Lancet* 2000;356:253.

The authors have confirmed the second case of variant CJD in France. The case was a 36-year-old woman with no history of travel abroad who presented with emotional and behavioural changes associated with severe depression. Seven months after onset of symptoms, a brain MRI led to the diagnosis of suspected CJD and after 14 months the patient died. The neuropathology was typical of vCJD and Western-blot analysis showed the presence of type 4 proteinase K resistant PrP confirming the diagnosis.

Canadian ban on blood donors linked to mad cow disease

Health Canada issued a directive on 30 August 2000 that people who have visited France between 1980 and 1996 for 6 months or more cannot donate blood. The alert is a precautionary measure against the spread of Creutzfeld Jacob disease, which is related to bovine spongiform encephalopathy. The same rule applies to people who lived in Britain during that period. Canadian Blood Services are reported as saying the action will only affect half of one per cent of all donors.

Fruit bats as a Nipah virus reservoir

Wayne Arnold, NY Times,15 Aug 2000 by-line (edited)

Nipah virus, a novel paramyxovirus, last year killed more than 100 people and compelled Malaysian authorities to slaughter roughly a million pigs. It is closely related to Hendra virus, which was isolated in 1994 after killing 14 horses and their trainer in Australia. As Hendra virus is carried by fruit bats, a similar link between Nipah virus and bats has been sought. Prof Kenneth Lam Sai Kit, of the Department of Microbiology at the University of Malaya, has confirmed that his research team successfully isolated Nipah virus from urine samples collected from the Island flying fox (*Pteropus hypomelaunus*), one of the many species of fruit bats that live throughout Southeast Asia.

Molecular analyses have confirmed that Nipah virus and Hendra virus are closely related, but different viruses. The N, C, P, V, M, F and G genes of Nipah virus have nucleotide sequence homologies ranging from 88% to 70%, and predicted amino acid homologies ranging from 92% to 67%, in comparison with Hendra virus. The intergenic regions and start/stop signals of the two viruses are identical. They are substantially different from, and have larger genomes than, previously known paramyxoviruses. Consequently, taxonomically these two bat viruses are likely to constitute a new genus in the family *Paramyxoviridae*.

It remains unclear exactly how the virus is transmitted from fruit bats to pigs. According to Professor Lam, as fruit trees in which bats forage for food are often near pigsties and Malaysia's pig farmers tend to live very close to their pigs, numerous opportunities exist for the rare crossover to occur. Because Nipah virus can also be found in saliva, it was possible that pigs become infected by eating fruit that bats had nibbled. How the virus moves from pigs to humans also remains unclear, but one possibility is that people inhale infected particles of saliva coughed up by Nipah virus-infected pigs.

Professor Lam's findings raise concern about the possibility that the bats, which are migratory, could carry the disease beyond Malaysia.

Overseas briefs

World Health Organization

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

Imported case of Lassa fever in The Netherlands - Update

The 48-year-old surgeon who was infected with Lassa fever virus while working in Kenema, Sierra Leone died on 25 July in Leiden University Hospital, The Netherlands, where he was being treated. He was initially treated for malaria in Sierra Leone on 11 July, and then travelled to The Netherlands (arrived 14 July) to visit relatives. He was admitted to the hospital on 15 July. Lassa fever was suspected when his condition worsened on 20 July and

treatment with ribavirin was started. On 22 July, the Bernard Nocht Institute for Tropical Medicine in Hamburg, Germany, reported that Lassa virus was detected in a blood sample sent for diagnostic testing.

While the risk of infection for those who travelled with this person from Freetown to Amsterdam is minimal, potential contacts are being traced and monitored in both The Netherlands as well as in Africa. As mentioned in the posting, this is the fourth case of Lassa fever imported into Europe this year: see previous reports. All four patients have died, although the death of one case was not the direct result of the acute infection.

Measles in Ireland

As of 29 July 2000, the National Disease Surveillance Centre, Ireland, has reported 1,376 cases of measles, including 2 deaths since 1 January 2000. Most of the cases occurred in the north Dublin city area. Control measures now being implemented include an intensification of the routine immunization programme, with increased advice given to parents in schools where cases of measles occurred and close follow-up of defaulters, and adjustments in the immunization schedule to better respond to the current epidemiological situation. For further information about the outbreak, visit the web site of the National Disease Surveillance Centre, Ireland, (http://www.ndsc.ie) as well as the article in the July 2000 issue of their online journal, EPI-INSIGHT (http://www.ndsc.ie/epi_insight.htm).

Meningococcal disease in Ethiopia

As of 17 August, a total of 855 cases and 19 deaths has been reported in Addis Ababa since the beginning of the current outbreak of meningococcal disease, which began in March 2000. *Neisseria meningitidis* serogroups A (90%) and C (10%) have been detected using latex agglutination tests in 311 of the patients. The age group most affected is <30 years. According to available data, no major outbreaks had been reported in Addis Ababa since 1989.

Ethiopia is in the African meningitis belt, and is regularly affected by both the endemic and epidemic forms of the disease. Outbreaks have been recorded since 1935. The most recent major outbreak affecting the whole country occurred in 1988-1989, with nearly 50,000 cases and 990 deaths, and an overall attack rate of 133 per 100,000. A major outbreak is anticipated in 1999-2000, and the regions of Amhara, Gambella and Tigray experienced an increase in the number of cases reported in March-April 2000.

Regional health authorities are conducting active surveillance in public and private health facilities, and committees are ensuring community surveillance. Cases are managed at hospital level by IV antibiotics (penicillin and chloramphenicol). The Ministry of Health has some stocks of oily chloramphenicol, but stock levels need to be carefully monitored. Mass immunization in Addis Ababa is under way with 1,000,000 people vaccinated to date, and external support may be needed depending on the evolution of outbreak.

Meningococcal disease in Rwanda

An outbreak of meningococcal disease in Kabgayi district (Gitarama prefecture) was confirmed on 10 August by a team comprising national medical personnel and WHO staff. *Neisseria meningitidis* serogroup A has been isolated. As of 22 August, 164 cases and 10 deaths had been reported since the beginning of the outbreak in mid-July. The outbreak has occurred in an area bordering the road between Rwanda and Burundi, 53 km from Kigali. Mass immunization of those affected or at risk (a population of 70,000) was started on 14 August. The national health authorities and WHO are monitoring the situation closely.

Yellow fever in Liberia

On 16 August 2000, the Ministry of Health (MOH), Liberia, confirmed an outbreak of yellow fever (YF) in Grand Cape Mount County. To date, the authorities have detected 29 cases meeting the case definition, including three deaths, originating from districts in the county. A report of one case in a third district has not yet been verified. Laboratory results have confirmed yellow fever IgM in one of five clinical samples sent to Institut Pasteur, Abidjan, Côte d'Ivoire, for testing; virus detection is now under way in this and the other samples.

These cases were detected following training in integrated surveillance for district surveillance officers in the county. As this training is introduced in the other counties, the outbreak may prove to be more extensive. Other districts are now beginning to detect suspected cases. All cases, which have been investigated, have occurred among unvaccinated persons. The most recent YF vaccination campaign in the area was conducted in 1999 but was limited to a single refugee camp. Most of the refugees who were vaccinated in that exercise have left the area. A nationwide YF vaccination campaign was conducted in 1995, but coverage was reported to be low, leaving much of the population susceptible.

The population in the Grand Cape Mount County lives in small villages or towns of approximately 5-10,000 people. There is a lot of movement across the border with Sierra Leone. The road between Grand Cape Mount County and the capital of Liberia, Monrovia, is in good condition and there is a considerable amount of traffic to and from Monrovia. If YF were introduced in Monrovia there would be 1.5 million people at risk.

In response to the outbreak, the MOH, Liberia, WHO, and health sector non-governmental organizations (NGOs) have agreed to:

- implement a mass vaccination campaign targeting the 150,000 population immediately at risk. WHO is providing vaccine and autodestruct syringes for this campaign;
- intensify vaccination and surveillance in the affected communities and other districts not yet known to be affected.

WHO is seeking to mobilize support for YF detection and response activities.

Acute haemorrhagic fever syndrome in Afghanistan - Update 2

A follow-up mission will leave for Herat, Afghanistan, on 27 August to train local health care workers in barrier nursing techniques and infection control procedures. The team consists of an infection control nurse and infectious diseases physician from Switzerland. They will be working with WHO, Afghanistan, national public health officials and United Nations organizations and non-governmental organizations (NGOs) involved in health services in the area.

ProMED-mail

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

vCJD iatrogenic dental transmission risk

Contributed by Paul N Goldwater, The Women's & Children's Hospital, North Adelaide.

The potential for dental transmission of the infectious prion protein of Creutzfeldt-Jakob Disease (CJD) and its (new) variant form (nvCJD) has not received wide attention. No doubt SEAC is exploring this possibility in its investigation of the Leicestershire cluster of cases. Whilst retrospective epidemiological studies have not revealed an association between dental treatment and CJD, there are reports of clusters of CJD cases possibly linked to dental procedures[1,2] or surgery for trigeminal neuralgia.[3]

Recent experimental data from an animal model warrants a fresh approach to this public health issue. Ingrosso et al,[4] using a hamster model, have demonstrated high levels of infectivity of scrapie prion in gingival and dental pulp tissues. Furthermore, successful transmission of the agent was achieved in all animals inoculated via the tooth pulp - a nerve-rich tissue. The implications of these findings, in the context of the evolving nvCJD epidemic in the wake of the epidemic of bovine spongiform encephalopathy (BSE), are obvious.

1. Will RG, Matthews WB. Evidence for case-to-case transmission of Creutzfeldt-Jakob disease. *J Neurol Neurosurg Psychiatry* 1982;45:235-238.
2. Arakawa K, Nagara H, Itoyama Y, Doh-ura K, Tomokane N, Tateishi J, Goto I. Clustering of three cases of Creutzfeldt-Jakob disease near Fukuoka City, Japan. *Acta Neurol Scand* 1991;84:445-447.
3. Matthews WB. Epidemiology of Creutzfeldt-Jakob disease in England and Wales. *J Neurol Neurosurg Psychiatry* 1975;38: 210-213.
4. Ingrosso L, Pisani F, Pocchiari M. Transmission of the 263K scrapie strain by the dental route. *J Gen Virol* 1999;80: 3043-3047.

Editorial note. SEAC is the Spongiform Encephalopathy Advisory Committee, which advises the UK government on the BSE and nvCJD outbreaks. The Committee estimated the risk of iatrogenic transmission through dental procedures to be real, but as yet hypothetical and not quantifiable. No special advice regarding the sterilization of dental instruments was issued by the Committee.

Pacific Public Health Surveillance Network

The Pacific Public Health Surveillance Network serves to disseminate information about communicable diseases in the Pacific region through Pacnet. Pacnet may be accessed, on registration, through the South Pacific Commission website (http://www.spc.org.nc).

Typhoid fever in Samoa

Dr Satupaitea Viali has provided a situational report, dated 9 August 2000, that typhoid fever is on the rise again in Samoa - with one death so far. This year from January to July there have been 122 cases of typhoid, 74 (61%) of which have been culture-proven. This contrasts with 30 to 40 cases per year of clinical and culture-proven typhoid fever reported for the previous 3 years.

Initially, patients usually presented to hospital in the 2nd to 4th week of their illness. With more public awareness and education, patients are now being seen earlier. The majority of the cases have been children under 15 years (~55%). Upolu is the most affected with 84% of the cases; most of these have been concentrated around the Apia region with around 62% of total culture-proven cases.

All patients have been investigated with 1-2 blood cultures, and stool cultures. Of the 74 culture-proven cases, two were diagnosed from stool culture alone, one from blood and stool culture, and the rest (96%) from blood culture. The medical unit's current management is such that if a patient was very sick intravenous chloramphenicol has been used first line, but for less sick patients Septrin or Ciprofloxacin has been given.

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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**

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