Annual report of the Australian Meningococcal Surveillance Programme, 1999

The Australian Meningococcal Surveillance Programme[1]

Abstract

The National Neisseria Network has undertaken meningococcal isolate surveillance by means of a collaborative laboratory based initiative since 1994. The phenotype (serogroup, serotype and serosubtype) and antibiotic susceptibility of 368 isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were determined in 1999. Ninety percent of the invasive isolates were either serogroup B or C. Serogroup B strains predominated in all States and Territories and were isolated from sporadic cases of invasive disease. Serogroup B phenotypes were generally diverse, but in New South Wales phenotype B:4:P1.4(7) became more prominent. The number of serogroup C isolates increased significantly in Victoria and remained prominent in New South Wales, especially in adolescents and adults. Phenotype C:2a:P1.2, infrequently isolated prior to 1999, was the most frequently encountered serogroup C phenotype. A number of infections with a phenotype new to Australia, C:2a:P1.4(7), were noted in Victoria and to a lesser extent in New South Wales. Phenotype C:2a:P1.5 was less frequently encountered than in previous years. About three-quarters of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06 to 0.5 mg/L). Three isolates showed reduced susceptibility to rifampicin. Data relating to 92 laboratory-confirmed but culture-negative cases were included in this report. Some differences in the patterns of disease were revealed when culture-based and non-culture-based data were compared. *Commun Dis Intell* 2000;24:181-189.

Keywords: meningococci, surveillance, invasive, phenotype, genotyping, diagnosis, antibiotic susceptibility

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Introduction

Invasive meningococcal diseases remained a focus of public health and general attention in 1999. Invasive meningococcal disease (IMD) presents mainly as septicaemia and/or meningitis and occasionally as single organ disease such as arthritis. Manifestations of IMD may range from the mild and even subclinical to the rapidly progressive and fatal. While some of the reasons for these different responses have been elucidated, many remain unknown. however, both the host response and the outcome of disease in an individual patient and the patterns of the infection within a community may be materially altered by the characteristics of the infecting organism.[1,2]

The public health response to IMD is also influenced by a number of the features of the subtypes of the meningococci involved. These features may be used to confirm or exclude the presence of an outbreak or cluster of cases suspected on clinical grounds, and to influence the public health response to such an outbreak. For example, vaccines are available for some serogroups of meningococci but not for others and the presence of different subtypes of meningococci excludes case clustering if this is suspected epidemiologically.

A national programme for the examination of isolates of *Neisseria meningitidis* from cases of IMD was commenced in 1994 through the collaboration of reference laboratories in each State and Territory. This laboratory-based activity is designed to supplement data from existent clinical notification schemes by adding information on the phenotype (the serogroup, the serotype and subserotype), and on occasion the genotype, and the antibiotic susceptibility of invasive isolates to clinical data.

Annual reports summarising data gathered since the inception of the programme have been published in *Communicable Diseases Intelligence (CDI)*.[3-7] The following report analyses the characteristics of meningococci isolated in the calendar year 1999. Non-culture based laboratory testing, based on nucleic acid based amplification assays and serology, is increasingly used to confirm IMD.[9] This report includes some data from IMD confirmed by these means.

Methods

The National Neisseria Network (NNN) is a collaborative programme for the laboratory surveillance of the pathogenic Neisseria, *N. meningitidis* and *N. gonorrhoeae*.[3-8] A network of reference laboratories in each State and Territory (see acknowledgments) performs meningococcal isolate surveillance.

Isolate-based surveillance

Each case was based upon isolation of a meningococcus from a normally sterile site. Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was sought. The isolate surveillance subset of the programme categorises cases on the basis of site of isolation of the organism. Where an isolate is grown from both blood and CSF cultures in the same patient, the case is classified as one of meningitis. It is recognised that the total number of cases - and particularly the number of cases of meningitis (e.g. where there was no lumbar puncture or else where lumbar puncture was delayed and the culture sterile) - was underestimated. However, the above approach has been used since the beginning of this programme and is continued for comparative purposes.

Phenotyping of invasive isolates of meningococci by serotyping and serosubtyping was based on the detection of outer membrane protein antigens using a standard set of monoclonal antibodies obtained from the National Institute for Public Health (RIVM), The Netherlands.

Antibiotic susceptibility was assessed by determining the minimal inhibitory concentration (MIC) to antibiotics used for therapeutic and prophylactic purposes. This programme uses the following parameters to define the various levels of penicillin susceptibility/resistance when determined by a standardised agar plate dilution technique:[8]

sensitive MIC 0.03 mg/L

less sensitive MIC 0.06 - 0.5 mg/L

relatively resistant MIC 1 mg/L

Contents, *continued*

Strains with MICs that place them in the category of 'sensitive' or 'less sensitive' would be considered to be amenable to penicillin therapy when used in currently recommended doses.

Non-culture-based laboratory-confirmed cases

Additional laboratory confirmation of suspected cases of IMD is increasingly available by means of non-culture based methods such as nucleic acid based amplification assays (NAA) and serological techniques. NAA testing is essentially by use of polymerase chain reaction (PCR) techniques.[9] Data arising from these investigations are included for the first time in this 1999 report. The serological results are based on tests performed using the methods and test criteria of the Manchester PHLS reference laboratory, UK.[10,11] Demographic data on non-culture based cases were obtained by telecommunication with the laboratory or public health unit involved. Where age, sex and outcome data for patients with non-culture based diagnoses are available these are also recorded. The site of a sample of a positive PCR test is also used to define the clinical syndrome. This separation is not possible for cases diagnosed serologically.

Results

Numbers of isolates from culture-confirmed cases

In total 368 invasive isolates of meningococci were examined in 1999. There were 122 isolates from patients whose infections were acquired in New South Wales (33% of all isolates), 94 (26%) from Victoria, 66 (18%) from Queensland, 39 (11%) from Western Australia, 24 (6%) from South Australia, 11 (3%) from Tasmania, 7 (2%) from the Northern Territory and 5 (1%) from the Australian Capital Territory (Table 1).

Seasonality

Fifty-eight (16%) of cases occurred between 1 January and 31 March, 69 (19%) between 1 April and 30 June, 142 (39%) between 1 July and 30 September and 95 (26%) between 1 October and 31 December. A winter peak of meningococcal disease is usual.

Age group

The age distribution of patients infected with invasive isolates in each State and Territory is shown in Table 2. Nationally, the peak incidence of meningococcal disease occurred in those 4 years and under. Those aged less than 1 year or in the 1-4 year age group accounted for 15% and 19% of cases respectively. A secondary peak was noted in the 15-19 year age group when 59 cases accounting for 16% of the total were recorded. A further 35 cases (9.5%) occurred in those aged 20-24. Victoria differed from the national pattern in that the number of cases of invasive disease in those aged 15-24 (30) was higher than for those aged 4 years or less (20).

Serogroup, serotype and serosubtype (phenotype) distribution

The distribution of the isolates by serogroup is shown in Table 1. Nationally, 232 serogroup B isolates represented 63% of all strains, the same proportion as in 1997 and 1998. The 120 serogroup C strains (33%) were more than the number (81) and proportion (25%) detected in 1998. The number (9) and proportion (2.5%) of serogroup Y strains in 1999 was half that recorded in 1998. Six serogroup W135 meningococci were also identified. One was not viable and not serogrouped. No serogroup A isolates were encountered in 1999.

Some important differences in the distribution of serogroups were evident when data were disaggregated by region. Serogroup B predominated in national data (63%) and in all jurisdictions. When examined regionally, Western Australia (85% of isolates), the Australian Capital Territory (80%), Tasmania (73%), South Australia (70%) and Queensland (69%) had high proportions of serogroup B strains. In New South Wales the 70 group B strains accounted for 57% of isolates, in Victoria serogroup B isolates were 53% of the total and in the Northern Territory 57% of 7 strains were serogroup B. Group B disease comprised unlinked and apparently sporadic cases.

A substantial increase in serogroup C infections occurred in Victoria in 1999 in that 42 isolates (45% of the total) were group C. In 1998 there were 7 (17.5%) serogroup C strains in Victoria. Serogroup C isolates were less prominent in

Table 1. ***Neisseria meningitidis*** **isolates, Australia, 1999, by State or Territory and serogroup**

State/ Territory	Serogroup											Total	
	B		C		A	Y		W135		NG*			
	n	%	n	%	n	n	%	n	%	n	%	n	%
ACT	4	80.0	1	20.0	0	0		0		0		5	1
NSW	70	57.4	45	36.9	0	5	4.1	2	1.6	0		122	33
NT	4	57.2	3	41.8	0	0		0		0		7	2
Qld	46	69.6	15	22.7	0	2	3.0	2	3.0	1	1.5	66	18
SA	17	70.0	6	25.0	0	1	5.0	0		0		24	6
Tas	8	72.7	2	18.2	0	0		1	9.1	0		11	3
Vic	50	53.1	42	44.7	0	1	1.1	1	1.1	0		94	26
WA	33	84.6	6	15.3	0	0		0		0		39	11
Total	232	63.0	120	33.0	0	9	2.5	6	1.5	1	0.5	368	100

* NG = not viable for serogrouping

Table 2. ***Neisseria meningitidis*** **isolates, Australia, 1999, by State or Territory and age group**

	Age group (years)										
	<1	1-4	5-9	10-14	15-19	20-24	25-44	45-64	65+	NS*	All
ACT	0	1	0	0	2	0	2	0	0	0	5
NSW	27	20	9	6	15	11	14	11	8	1	122
NT	2	2	0	0	1	1	1	0	0	0	7
Qld	9	16	6	1	10	8	7	6	3		66
SA	1	5	1	4	3	2	4	1	2	1	24
Tas	2	1	2	0	0	0	3	1	0	2	11
Vic	8	12	5	6	21	9	12	10	6	5	94
WA	5	13	5	0	7	4	0	4	1	0	39
Total n	54	70	28	17	59	35	43	33	20	9	368
%	15	19	7.5	4.5	16	9.5	11.5	9	5.5	2.5	100

* Not stated

New South Wales in 1999 where 45 strains accounted for 37% of all isolates. Eighty-seven group C meningococci or 73% of all serogroup C strains isolated in Australia were from infections in New South Wales and Victoria. In the Northern Territory 3 of the 7 strains were group C. Numbers and/or proportions of group C strains were lower in other States and Territories. There were 15 group C isolates (23%) in Queensland, 2 in Tasmania, 6 (25%) in South Australia, 6 (15%) in Western Australia and a single isolate in the Australian Capital Territory. No clusters of serogroup C infection were identified.

Serogroup distribution was again age associated (Figures 1a-c). Serogroup B strains predominated in younger age groups (less than 14 years) in all centres and in all age groups for centres other than New South Wales and Victoria. In these States group C meningococci were seen more often than in other centres, and represented the highest proportion of any serogroup in those aged between 15 and 44 years. In Victoria this trend was also present in those aged over 45 years. This differential distribution of serogroups by age has been noted previously in these reports.

There was again considerable phenotypic heterogeneity amongst invasive isolates as determined by serotyping and serosubtyping. The predominant serotypes/serosubtypes in each State and Territory are shown in Table 3. Serogroup B meningococci are more difficult to characterise by serological methods and a number could not be phenotyped. B:4:P1.4(7) strains predominated in New South Wales and were also present in Queensland and Victoria. B:15:P1.7 strains were also seen in New South Wales, Queensland, Victoria, Western Australia and South Australia.

Figure 1b. Serogroup B and C infections, Victoria, 1999, by age

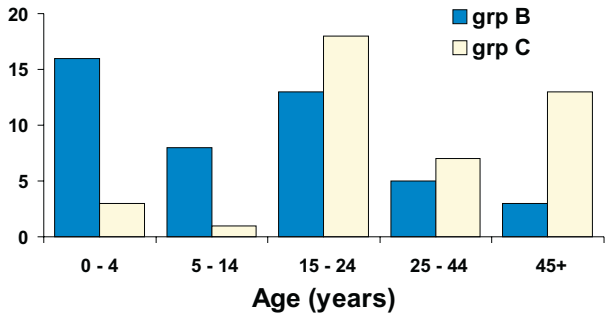


Figure 1a. Serogroup B and C infections, centres other than Victoria and New South Wales, 1999, by age

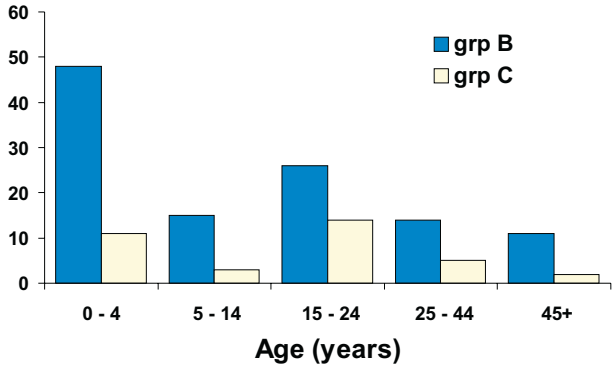


Figure 1c. Serogroup B and C infections, New South Wales, 1999, by age group

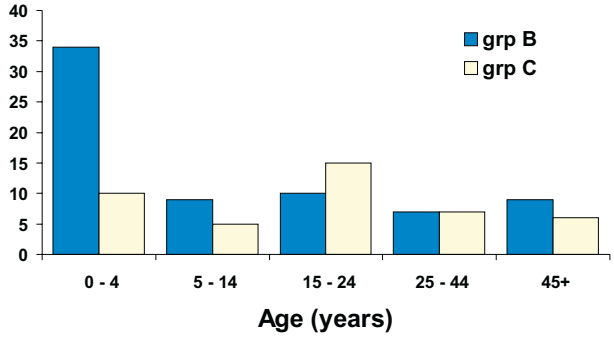


Table 3. Commonly isolated serotypes and serosubtypes and phenotypes of *N. meningitidis* of interest, Australia, 1997 to 1999, by State and Territory.

	Serogroup B			Serogroup C		
State/Territory	Serotype:serosubtype	n =	('97,'98)*	Serotype:serosubtype	n =	('97,'98)*
Qld	4:P1.4(7)	5	(4, 3)	2b:P1.5,2	2	(3, 0)
	NT:P1.4	6	(3, 9)	2a:P1.5	1	(4, 1)
	15:P1.7	5	(1, 3)	2a:P1.5,2	1	(0, 3)
	2b:P1.10	0	(1,2)	2b:P1.2	1	(0, 3)
NSW	4:P1.4(7)	28	(5, 17)	2a:P1.5	11	(23, 39)
	NT:nst**	10	(8, 13)	2b:P1.5,2	2	(6, 8)
	2b:P1.10	1	(4, 11)	2a:P1.5,2	7	(8, 3)
	15:P1.7	7	(1, 7)	2a:P1.2	9	
Vic	NT:P1.4	14	(11, 8)	2a:P1.2	13	
	15:P1.7(16)	6	(1, 3)	2a:P1.4(7)	10	
	4:P1.4	4	(2, 3)	2b:P1.2	1	(2, 1)
	2b:P1.10	1	(1, 2)	2a:P1.5	2	(1, 1)
SA	15:P1.7	1	(3)	2b:P1.5,2	1	(2)
	4:nst	1	(3)	2a:P1.5,2	1	
	4 :P1.4	0	(1)	2a:nst	1	
	NT:nst	5				
Tas	NT:nst	2		2b:P1.2	1	(1, 2)
ACT	Single isolate only	1				
NT	2b:nst	1	(5)	2a:P1.5	1	
	NT:nst	2		2a:P1.5,2	1	
WA	NT:P1.4	9		2a:nst	4	

* The numbers of isolates of each phenotype in 1997 and 1998 are shown in parentheses

** nst = non serosubtype tested

There was less heterogeneity amongst serogroup C meningococci. All isolates were either serotype 2a or 2b. Two phenotypes present in 1999 in New South Wales and Victoria that are worthy of note were C:2a:P1.2 and C:2a:P1.4(7). The former phenotype was uncommonly encountered in previous years but the 22 isolates of this phenotype in 1999 represented 19% of all serogroup C strains. The latter phenotype was especially prominent in Victoria. The other group C serosubtypes present were either P1.5, P1.5,2 or P1.2. There were 16 serogroup C strains of phenotype 2a:P1.5, 14% of all group C strains phenotyped. Most of these were found in New South Wales, but the number and proportion of this phenotype was much less than in 1998 (28 strains, 54%). Strains of this phenotype were also isolated in Queensland, the Northern Territory and Victoria.

Site of isolation

There were 111 isolates from CSF either alone or with a blood culture isolate and 251 from blood cultures alone. There were six isolates from synovial fluid.

Outcome data for cases with sterile site isolates for 1999

Outcome data (survived or died) were available for 320 patients (87%). Twenty-nine deaths were recorded (9.1%) (Table 4). Outcomes were available in 87% of serogroup B infections and 86% of serogroup C infections. There were 13 (6.4%) deaths in serogroup B infections and 16 (14.9%) in serogroup C infections ($p < 0.05$). Where outcomes were known, there were 11 deaths in 96 patients (11.4%) with meningitis. Six patients were infected with serogroup B and 5 with serogroup C strains. Eighteen deaths were recorded in 220 bacteraemic patients (8.2 %). There were 129 cases of serogroup B meningococcal bacteraemia with 7 deaths and another 82 cases were caused by serogroup C strains among which 11 fatalities were recorded. No fatalities were recorded with serogroup Y or W135 infections.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

Three hundred and fifty two isolates of the 368 strains were tested for their susceptibility to penicillin. A single isolate was regarded as resistant with an MIC of 1 mg/L. Using defined criteria, 90 strains (25.5%) were fully sensitive to penicillin and 261 (74%) less sensitive (MIC 0.06 to 0.5 mg/L). These proportions are virtually the same as for 1998. Only three isolates had MICs of 0.5 mg/L.

Table 4. Outcome of 316 meningitic and septicaemic cases of meningococcal infection, Australia, 1999, by serogroup.

		Serogroup				Total	
Disease Type	Outcome	B	C	Y	W135	n =	%
Meningitis	Survived	66	18	0	1	85	88.5
	Died	6	5	0	0	11	11.5
	Total	72	23	0	1	96	
Septicaemia	Survived	122	71	4	5	202	91.8
	Died	7	11	0	0	18	8.2
	Total	129	82	4	5	220	
All cases*	Total	203	107	4	6	320	
	Died	13	16	0	0	29	9.1

* Includes 2 serogroup B and 2 serogroup C strains from joint aspirates from patients who survived.

Other antibiotics.

All 352 isolates which were tested for susceptibility to ceftriaxone (and by extrapolation to other third generation cephalosporins) were susceptible to these therapeutic agents. Three meningococci had raised MICs to the prophylactic antibiotic rifampicin (MICs of 1 mg/L or more, including one with a MIC >100mg/L). Sulphonamide testing was not performed.

Numbers and sources of non-culture diagnoses of IMD in 1999

There were 92 diagnoses of invasive meningococcal disease in 1999 where PCR and/or serology were positive in the absence of positive cultures (Table 5). In 13 cases both serology and PCR testing were performed and both tests were positive. however, it was more usual to have available samples suitable for testing by only one of the above techniques. Thus there were 41 cases where PCR testing in isolation was positive and 38 cases where serology testing was positive.

With PCR testing it was also possible to categorise the disease type by source of specimen in a manner similar to that used for culture-positive cases (Table 5). Of the 54 cases positive by PCR, 36 were from CSF and 18 from blood. This is a distinct difference from the distribution of culture-based diagnosis. Culture-based diagnosis of blood yielded 2.5 times the number of cultures derived from CSF.

Table 5. Source of non-culture based diagnosis of invasive meningococcal disease 1999

All non culture based diagnoses	92
PCR and serology positive	13
PCR positive alone	41
CSF PCR positive (including those with positive serology)	36
Blood PCR positive (including those with positive serology)	18
Serology positive alone	38

With PCR based diagnosis the ratio of blood to CSF positive was 0.5:1.

Serogroup and age distribution of non-culture based IMD

In addition to diagnostic PCR, this technique can also be used to ascertain whether serogroup B or C meningococci were involved in the disease process. (At present this is not available for serogroups other than B or C). There were 54 cases where a PCR-based diagnosis was made and in 49 of these the serogroup was also determined as B or C (Table 6).

For those cases diagnosed by serology alone, age distribution was different with most diagnoses – 35 of 38 in those aged 10 years or more (Table 7). This reflects in part the difficulty in obtaining serum samples from young children. The categorisation of IMD by site of organism capture cannot be determined with serology. Additionally, serogroup determination is not possible.

Outcome data for IMD based on non-culture based diagnosis

Non-culture based diagnosis is currently less well established than that for IMD based on positive culture. For IMD diagnosed by PCR based tests, the outcome is known in 24 cases. All 9 diagnosed by PCR on blood survived (3 each of serogroup B and C and 3 where no serogroup was determined). Of a further 15 patients with PCR positive on a CSF sample, 12 patients survived (8 group B, 3 group C, 1 undetermined serogroup) and 3 died (one each with serogroup B and C and one with an undetermined serogroup). One death and 11 survivors were recorded amongst the 38 cases diagnosed serologically.

Discussion

The total 368 isolates examined by NNN laboratories in the Australian Meningococcal Surveillance Programme in 1999 was the highest since the inception of the programme in 1994. The number of isolates examined each year by the NNN increased each year from 1994 until 1998 when it decreased slightly. The numbers of isolates examined between 1997 and 1999 ranged between 323 and 368 i.e. small aggregate differences only. Importantly however,

Table 6. Invasive meningococcal disease diagnosed by polymerase chain reaction, Australia, 1999, by serogroup and age group

Serogroup	Age group (years)									Total
	<1	1-4	5-9	10-14	15-19	20-24	25-44	45-64	64+	
B	6	10	0	2	10	3	5	1	0	37
C	0	2	1	1	5	0	3	0	0	12
U*	0	3	0	1	0	0	0	1	0	5
All	6	15	1	4	15	3	8	2	0	54

* U = undetermined

Table 7. Cases of invasive meningococcal disease diagnosed by serology alone, Australia, 1999, by age group

Age group (years)	Cases
<1	0
1-4	2
5-9	1
10-14	4
15-19	11
20-24	7
25-44	7
45-64	4
>65	2
Total	38

greater differences in the annual number of isolates are revealed when data are examined by jurisdiction. The number of isolates available in Victoria in 1999 (94) was more than twice the 41 examined in 1998. In Queensland the number of isolates decreased from 81 to 66. Isolate numbers in other centres varied little from 1998 totals. The number of isolates available for examination will always be less than the number of clinically notified cases because clinical surveillance case definitions include culture-negative cases.

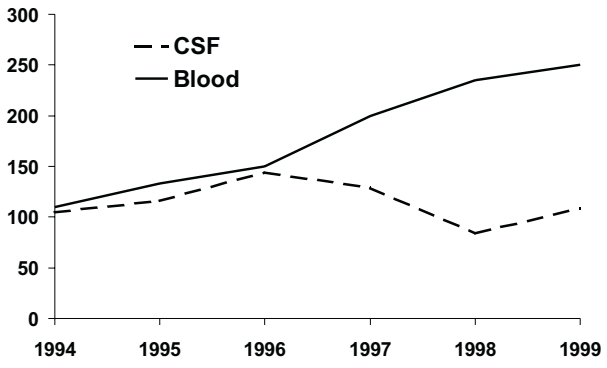
Eighty-six clinical cases were confirmed only by non-culture based laboratory examinations in 1999. These procedures include NAA assays using PCR and/or serological examination. These cases were included separately in this report. Some of the PCR techniques in use can provide additional data on the serogroup of the isolate. It is anticipated that laboratory confirmation of invasive meningococcal disease by non-culture based methods will continue to increase. In general serologically diagnosed disease is usually milder – patients survive to have serological tests. One corollary of this is that serological tests diagnose some previously unrecognised but milder IMD syndromes. NNN laboratories may be contacted for advice regarding these tests.

The ratio of cases of meningitis to bacteraemia was 0.44:1 in 1999 in culture confirmed cases, continuing a trend noted first in 1997 (Figure 2). From 1994 to 1996, the ratio of cases of meningitis to bacteraemia was close to 1:1 in NNN isolate-based data. In 1997 this ratio decreased to 0.6:1 and in 1998 further declined to 0.36:1. NNN cases are based on the site of isolation of the organism and thus tend to overestimate the number of bacteraemic cases for several reasons.[7] NNN case definitions have remained constant to allow year-to-year comparisons. The PCR-based diagnostic data included in this report has a markedly different disease ratio, with meningitis featuring prominently in disease syndromes. Again however, there is an inherent bias in these data in that PCR was initially only performed on CSF samples and sensitivity of PCR techniques in blood samples is less than that for CSF.

The predominant disease pattern throughout the country remained sporadic infection with serogroup B meningococci. The proportion of serogroup C cases in aggregated data in 1999 increased to 32.5% from 25% in 1998. however, the numbers of isolates from the more populous States introduce some distortions. The increase in 1999 was due to increased numbers of serogroup C cases in Victoria - from 7 in 1998 to 42 in 1999. Serogroup C infections have been prominent in New South Wales from 1996 onwards, but until 1999 were infrequently encountered in other States and Territories. In New South Wales in 1998 and 1999, serogroup C strains have declined as a proportion of all isolates tested. Serogroup C cases were also sporadic in 1999. No serogroup A meningococci were isolated in 1999. The number and proportion of cases of serogroup Y infection decreased in 1999.

Children aged 4 years or less remained the group most frequently infected. A secondary incidence peak in young adults and adolescents was again present. Serogroup C disease occurred more often in the young adult age group

Figure 2. Numbers of meningococcal isolates from CSF and blood culture, 1994 to 1999



(Figures 1a – c) as previously noted in NNN reports, and was responsible for an upsurge in adult cases in Victoria. This picture of serogroup B and C disease occurring as sporadic cases is typical of the pattern of meningococcal disease in developed countries. Occasional clusters of cases of serogroup C infection have been noted in recent years but were not seen in 1999.

Phenotyping data obtained on the basis of serotyping and serosubtyping was again available from all centres in 1999. These data reinforce the considerable differences that exist in meningococcal subtypes causing IMD in different jurisdictions. The heterogeneity of serogroup B isolates present in Australia was once more evident (Table 3). Of interest amongst the group B strains were phenotypes B:4:P1.4(7) and B:15:P1.7 associated with hyperendemic disease in New Zealand and Europe respectively. B:4:P1.4(7) strains were encountered in higher numbers in 1999 and in New South Wales this phenotype represented about 23% of all isolates. The distribution of phenotype B:15:P:1.7 was little changed from 1998.

Earlier reports noted the appearance and spread of the phenotypes C:2a:P1.5 and C:2a:P1.5,2, particularly in New South Wales. These phenotypes, although present in small numbers in several States and Territories in 1999, declined in prominence. Of particular interest was the emergence in Victoria of a phenotype C:2a:P1.4(7) that appears to be an example of 'swapping' of outer membrane protein genes. This phenotype was also seen in a small number of cases in New South Wales. Additionally phenotype B:4:P1.5 was recognised. While it remains speculative how these events arose, it emphasises the considerable recombination potential of meningococci. Also more prominent in 1999 was the number of isolates of phenotype C:2a:P1.2 in both New South Wales and Victoria, again illustrating the temporal and geographic variation in meningococcal subtypes that occurs in Australia.

The overall mortality recorded in 304 assessable culture-positive cases was 9.4%, similar to the 9% observed in 1998. A higher mortality rate was again observed with serogroup C cases. Although serogroup C strains have been associated with increased mortality overseas, other factors, such as age, and time from onset to presentation and treatment - on which data were not available - may also explain this difference.

Continuing interest has been shown in the decrease in susceptibility of meningococci to penicillin in many parts of the world. Further, other isolates have occasionally been shown to be resistant to other antibiotics that are used currently for either therapeutic or prophylactic purposes in meningococcal disease. This programme therefore includes

routine examination of the antibiotic susceptibility of invasive isolates as part of its surveillance. Trend data indicate that since 1994 there has been an increase in the proportion of invasive meningococci showing some decrease in penicillin susceptibility. In 1994, 52% of strains were in the 'less sensitive' range (MIC 0.06 - 0.5 mg/L). In 1995, 155 (63%) of 247 strains tested were 'less sensitive'. The proportion of less sensitive isolates increased further to 74% of 297 isolates in 1996. This proportion remained unchanged in 1997 (73%) and no further change was recorded in 1998 or 1999. The isolation of a meningococcus with a MIC in the less sensitive range does not mean that therapeutic failure will occur, but the increase in the number and proportion of strains in this category is rather an epidemiological marker of the slow progression to resistance. A single isolate with an MIC of 1 mg/L was found in 1999. All isolates were fully sensitive to third generation cephalosporins. Chloramphenicol testing is no longer performed in this programme.

The definition of what constitutes 'resistance' to the prophylactic agent rifampicin varies. This programme has chosen to monitor the number of isolates with MICs of 1 mg/L or more. There were three isolates with rifampicin MICs of 1 mg/L or more in 1999. All isolates were quinolone susceptible.

The NNN programme has examined a total of about 1,800 strains from all States and Territories since 1994. It is a continuing, long-term collaborative study that has evolved to accommodate the situation that exists in Australia. As such it has assisted in clarifying and expanding information on invasive meningococcal isolates in Australia. It is emphasised that this is an independent laboratory based and structured programme designed to augment those data which should properly be collected separately by clinically based surveillance systems. The nature and high public recognition of meningococcal disease suggests that the efforts of this programme should continue. For further details the relevant NNN member should be contacted (see acknowledgments for contact numbers).

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Isolates were received in the reference centres from many laboratories throughout Australia. The considerable time and effort involved in forwarding these strains is recognised and these efforts are greatly appreciated. These data could not have been provided without this assistance and the help of clinical colleagues and Public Health personnel.

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The National Neisseria Network 1979 – 200?

The National Neisseria Network[1]

Keywords: gonococci, meningococci, surveillance, antibiotic resistance, methods, quality assurance

Introduction

The federal nature of the Australian health system means that the different jurisdictions administer and deliver services appropriate to the diverse needs of populations of varied composition and density distributed over a continent. The different approaches that have developed as a consequence of this approach pose some problems in relation to defining and dealing with issues of public health microbiology. A number of laboratory-based programmes have emerged that examine specific issues of surveillance of infectious disease across the 'jurisdictional divide'. For example, significant data have been gathered by mycobacterial and enteric pathogen programmes over many years, to name but two. This article briefly describes the origins and aims, organisation and structure, and past, present and future functions of another of these programmes, the National Neisseria Network (NNN).

The NNN undertakes laboratory-based surveillance of isolates of the two pathogenic Neisseria, *Neisseria gonorrhoeae* and *Neisseria meningitidis*. It may seem incongruous to some that surveillance of these two pathogens would be dealt with in a single network given the quite different public health responses required for gonorrhoea and invasive meningococcal disease (IMD). however, there is considerable overlap in the laboratory procedures and approaches to the two organisms - *N. gonorrhoeae* is essentially a highly evolved meningococcus. The commonality of laboratory approaches is finite however, so that two separate systems exist within the NNN, namely, the Australian Gonococcal Surveillance Programme (AGSP) and its meningococcal equivalent the Australian Meningococcal Surveillance Programme (AMSP). The two systems will be described separately.

The Australian Gonococcal Surveillance Programme (AGSP)

Background

The AGSP is a programme of long-term continuous surveillance of the susceptibility of gonococci to antibiotics used in the treatment of gonorrhoea. It is a collaborative network of reference laboratories in each State and Territory which use an agreed methodology to determine the quantitative susceptibility (minimal inhibitory concentration – MIC) of gonococci to a core group of antibiotics.

Why gonococcal susceptibility surveillance?

The necessity for such a programme is now firmly established. Effective antibiotic treatment of gonorrhoea is one pillar by which control of gonococcal disease may be achieved. Appropriate treatment quickly renders patients non-infectious decreasing both the transmissibility of the disease and the duration of infectiousness of the individual. In terms of disease control there are thus direct benefits from use of proper treatment. Additionally the well-recognised complications of gonorrhoea – infertility, pelvic inflammatory disease, ophthalmia, foetal loss, disseminated infection – are significantly reduced by early and appropriate treatment. It is important also to remember that HIV transmission is significantly amplified in the presence of gonorrhoea. Males with HIV and gonorrhoea have greatly increased HIV loads in seminal fluids compared to controls with HIV but not gonorrhoea, but this load returns to the level found in controls once proper treatment is effected. Those with gonorrhoea without HIV are also more susceptible to HIV infection because the target cells for HIV are recruited to the inflammatory process initiated by gonococcal infection. Again this susceptibility is removed by effective treatment resolving the inflammatory infiltrate. There are thus very cogent reasons why gonorrhoea should now be actively diagnosed and effectively treated. Treatment of gonorrhoea is by single dose antibiotic treatment at first diagnosis – well before any susceptibility testing of individual isolates can be performed. Empiric treatment is thus used, but is directed not by testing of individual isolates on an emerging basis, but rather by determining the pattern of susceptibility of prevalent gonococcal isolates. This is ascertained by obtaining a suitable sample of isolates, measuring the in vitro susceptibility of the gonococci so obtained and, on this basis, establishing a suitable antibiotic treatment regimen. There is a strong correlation between in vitro susceptibility determinations (MICs) and likely outcome of treatment in gonorrhoea. It is usually necessary to discontinue a treatment regimen once 5% of isolates are resistant to that agent.

Gonococcal resistance to antibiotics can be quite volatile. Australians travel frequently in our region (where antibiotic resistant gonococci are highly prevalent) and introduce resistant isolates into local transmission chains. Surveillance of antibiotic resistance in gonococci should thus be designed not only to monitor patterns of resistance but also be able to detect emergence of new forms of resistance and the spread of these resistant strains. The spread of antibiotic resistant gonococci is by no means inevitable as antibiotic resistance is but one of many factors which determine the 'success' of a subset of gonococci in establishing themselves within a community. Determining the pattern of introduction of antimicrobial resistant gonococci (AMRGC) can assist in planning and control of the spread of such organisms.

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Origins and outcomes of the AGSP

This programme arose form a perceived need to have available in Australia reliable and comparable data on patterns of AMRGC. Data were being generated differently in a number of centres and could not be integrated or compared. In 1979 staff from a group of laboratories agreed to explore a common approach to gonococcal susceptibility testing with a view to describing a standard method for determining MICs. Over an 18-month period, systems were assessed and the AGSP method of MIC determination was agreed and introduced.[1] No one laboratory was able to examine all isolates available for testing in Australia. A networked approach was thus developed whereby the standard test method was used in each centre and an extensive and comprehensive quality assurance programme established.[2] Data on gonococcal susceptibility were first reported in *Communicable Diseases Intelligence (CDI)* in 1981[3] and quarterly reports have been published continuously since then. Initially, susceptibility to the penicillins was examined, but other antibiotics were introduced as their use warranted. Intermittent reviews of AGSP susceptibility data were reported[1,4] but in recent years *CDI* has provided a vehicle for regular publication of annual reports of the AGSP.[5-7,10]

In addition to the information on AMRGC, it became apparent that the AGSP could contribute meaningful data on trends in gonococcal disease in Australia. Although the AGSP did not obtain all gonococci from notified cases, it established a broadly based and stable sample from which site specific and trend data on gonococcal disease could also be derived. This 'secondary' benefit saw data published on this topic,[8] incorporated in other data sets[9] and included in AGSP annual reports[5-7,10] to complement more extensive information from clinical notification systems.

Current status

The 1999 report of the AGSP has recently appeared in *CDI*[10] and reinforced the need for continuing surveillance of AMRGC. This latest report described changing patterns of AMRGC in major urban centres and worrying levels of decreased susceptibility to agents used for treating gonorrhoea in rural and northern Australia where rates of disease are excessive. In the major centres of Sydney and Melbourne for practical purposes there is no suitable oral therapy for the treatment of gonorrhoea. Isolates with altered susceptibility to injectable third generation cephalosporins have now been detected. In northern Australia the proportion of isolates resistant to the penicillins, which is the mainstay of treatment, is reaching critical levels. Under these circumstances susceptibility surveillance should not just be maintained but enhanced.

Future

Newer diagnostic systems using non-culture based tests have been adopted widely in Australia. Those in use are nucleic acid amplification assays (NAA), with polymerase chain reaction (PCR) methodology the most widely employed. These tests have allowed the possibility of an aetiological diagnosis in STD syndromes in patients in remote regions where circumstances prevented culture-based assessment. This improved diagnostic capability has materially assisted in enhanced disease control,[11] but presupposes that treatment modalities will remain effective. In urban practice, use of NAA tests has increased following inclusion of a rebate for NAA testing in STDs in the pathology services table.

This change in diagnostic practice will mean that fewer isolates are available for susceptibility testing. Ironically this is occurring at a time when antibiotic resistance patterns are changing in both urban and rural areas and there is a need for enhanced susceptibility surveillance. At present the sample of isolates available to the AGSP is sufficient for its primary purpose of susceptibility surveillance. The AGSP has discussed strategies whereby this sample base can be maintained despite the use of PCR for diagnosis; for example, culture of urine samples positive on PCR testing. This of course imposes an extra cost on the health system. It should perhaps be remembered the NAA testing offers no real increase in sensitivity when proper culturing can be performed. Thus while NAA testing has significant advantages in outreach situations, culture-based examination should be maintained if not actively pursued in clinic-based practice.

Data on trends in disease patterns from AGSP sources will be progressively devalued if and when the isolate sample base is altered through introduction of NAA testing. Again this comes at a time when rates of gonococcal disease are increasing and precise definition of the subpopulations where this phenomenon is occurring is required.

The AGSP has been a successful model of laboratory-based surveillance with considerable public health relevance. It links antimicrobial resistance directly to disease control and health outcomes in a condition that is of major public health importance and which is highly transmissible. For over a decade, the AGSP methodology has been successfully adapted for use in World Health Organization programmes in about 30 countries in our region. This too is a benefit to Australia as gonococci do not recognise territorial boundaries and this knowledge of regional susceptibility patterns helps determine our treatment strategies.

The Australian Meningococcal Surveillance Programme (AMSP)

Background

Despite the high public profile of invasive meningococcal disease, laboratory data on invasive meningococcal isolates found in Australia was at best piecemeal until 1994 when the AMSP was formed. The AMSP laboratories used an approach similar to that adopted for the AGSP, namely, jurisdiction-based, collaborative and consensus-based methodologies, programme specific Quality Assurance and pooling of systematically generated and comparable data. Again the laboratory data were seen as complementary to the existing formal notification schemes.

The emphasis placed on laboratory-based meningococcal surveillance is tailored to the needs of disease control and concentrates on meningococcal strain characterisation and differentiation. Although the AMSP is laboratory-based it does obtain clinical data which have enabled it to provide other information of relevance e.g. the NNN reports provide data on serogroup linked to age group. Antibiotic resistance is not as well developed in meningococci as in gonococci, but the same principles of susceptibility surveillance developed for gonococci were readily applicable to meningococci.

Approach of the AMSP to strain characterisation and differentiation in relation to public health

Characterisation and differentiation of meningococci (typing) from cases of IMD undertaken for public health reasons is to confirm or to exclude a suspected outbreak or cluster of cases and to define the meningococcal population circulating at any one time. Various phenotypic and genotypic techniques are available and are employed for different purposes at different times.

Currently all isolates are phenotyped by NNN laboratories by determining the serogroup as soon as practicable after receipt and then the serotype and serosubtype using standard monoclonal reagents. Serotyping and serosubtyping is performed by batching of isolates and testing at regular intervals – less frequently in low incidence periods and more frequently in the winter/spring. Serotyping and subserotyping is NOT *routinely* performed on an emerging basis, as it is wasteful of reagents that are no longer produced. These techniques can however, be rapidly employed if an epidemiological link between cases is established or suspected clinically and can quickly exclude the presence of clustering of cases.

Many meningococcal strains cannot be typed by serological methods and reagent stocks are finite. Genotyping (molecular) procedures are thus now supplanting phenotyping (serotyping) methods. Those available include pulsed field gel electrophoresis (PFGE), *porA/porB* sequencing and MLST. These techniques are used for different purposes eg PFGE and *porA* sequencing are used for short-term studies of strain relatedness and MLST for longer-term 'population' studies of meningococci. PFGE methods are not uniform – there are significant variations in choice of cutting enzymes, and pulse and ramp times, but PFGE patterns are usually considered of short-term significance in differentiating suspected clusters under local conditions. The non-clonal nature of serogroup B meningococci, for example, means that comparisons of PFGE patterns are not suitable for distinguishing invasive meningococci separated temporally and/or geographically across Australia.

Similarly *porA/porB* typing is increasingly available and can also be applied for short-term examination of possible clusters but is not suited to longitudinal genotyping studies. A global standard nomenclature for *porA* sequencing is being developed meaning that greater comparability of strains may be achieved by this means.

As it examines more stable parts of the genome, MLST is at the moment a technique more appropriately used for studies of meningococcal populations.

The application and development of these techniques in Australia is under constant review by the NNN.

It should also be remembered that the presence of isolates with an indistinguishable phenotype (serogroup, serotype and serosubtype) and/or genotype *does not* of itself establish a true epidemiological link; the latter should properly be established by clinical public health procedures. That is the possibility of outbreaks or clusters of cases is raised on clinical epidemiological grounds and confirmed or excluded by application of the typing techniques described here. Using phenotyping data without *prior* clinical epidemiological analysis to define case clustering is to place the cart before the horse.

Diagnostic advances

The laboratory diagnosis of IMD depends on the demonstration of *N. meningitidis*, or detection of its polysaccharide antigen or DNA in samples from normally sterile sites, or positive serology. As with gonorrhoea, non-culture based diagnosis is making an increasingly important contribution to confirmation of IMD. In meningococcal disease, non-culture based diagnosis becomes increasingly important as 'treat first, diagnose later' management options are followed. Also relevant is an evident reluctance to undertake lumbar puncture in cases of suspected meningitis. This produces a bias in data from culture-based cases. The AMSP attempts to capture PCR and serologically based diagnoses, and includes these in its analyses. however, this becomes more difficult as technological innovations become more widely used.

Some outcomes of AMSP surveillance

Prior to 1994 there was not even a comprehensive knowledge of serogroup distribution of IMD isolates in Australia. Currently, national serogroup data on IMD are available to public health bodies in each jurisdiction on a fortnightly basis. Since its inception the AMSP has provided data on the epidemiology of IMD in Australia previously or still otherwise unavailable. It has determined that most IMD in Australia, like that in most industrialised nations, is sporadic and due principally to serogroup B and C meningococci. Importantly it has revealed significant regional variation in the proportion of these two serogroups and monitored the changing patterns in serogroup distributions in the past several years. Age related distribution of disease by serogroup is specifically included in reports. Some clusters of serogroup C disease have occurred in recent years, but no instances of serogroup A infections have been seen for some time. Particular subtypes of serogroups B and C have been responsible for outbreaks and clusters of disease and for hyperendemic disease. Changes in the antibiotic susceptibility of IMD isolates to penicillin have been recorded and the frequency of isolation of isolates resistant to agents used for prophylaxis of IMD in Australia monitored. Data are reported annually in *CDI*.[12-16]

Concluding remarks

In both surveillance systems that monitor isolates of the pathogenic *Neisseria*, a comprehensive amount of relevant data has been obtained, analysed and reported over many years. Benefits other than these published data, often intangible, also accrue to the participants e.g. commonality of methodology, method development and shared experience. The total output of the NNN is seemingly greater than the sum of its parts. NNN labs are often consulted formally and informally in relation to *Neisseria* infections in their jurisdiction.

Some essential features of networks of this kind are that publication and recognition accrues to the network itself and decisions are based on consensus and agreement. This collaborative system is the antithesis of the competition-based approach currently fashionable but at least, in this instance, it has the benefit of a proven track record as justification for its continuation.

The NNN and its members have always been delighted to work with other interested parties. Individual jurisdictions

have forged strong links between clinical and laboratory systems using models best suited to their needs. The NNN has anticipated a universal approach by maintaining jurisdictional independence of its participants while at the same time combining and analysing national laboratory data. We would hope that any attempts to provide additional insights into clinical and public health aspects of IMD in Australia would see fit to include the NNN as full partner and use the experience already gained to enhance this process.

Acknowledgments

A large number of people have given generously of their time and expertise over many years to the NNN. Past and present members of the NNN are listed in the acknowledgments sections of NNN publications. It is pertinent to remember some who have retired or else moved to new fields of endeavour but who made substantial contributions in establishing the network and these include Yvonne Cossins, Justin Raby, Joc Forsyth, and Greg Handke.

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Notes from the North

Contributed by Dr Jan Savage, Northern Territory Health Services, Casuarina

Surveillance of gonococcal antibiotic susceptibility patterns is fundamental to prevention and control of gonorrhoea. In Northern Australia the rates of gonorrhoea are high and to date the overwhelming majority has been sensitive to penicillin. The advent of highly sensitive and robust polymerase chain reaction (PCR) testing has lead to increased diagnosis and treatment of gonorrhoea, at the same time, fewer specimens are being sent to laboratories for culture (and antibiotic susceptibility testing).

The effect of this was recently highlighted after reports of increased numbers of penicillin resistant gonorrhoea in Far North Queensland (FNQ) were received. There is considerable travel between the Northern Territory and FNQ residents, with the potential for wide transmission of resistant strains.

The NT response to this included:

1. Confirmation that notified cases of PPNG had remained steady, but that cases diagnosed by culture had decreased by 400%;
2. Identification that there are deficiencies in the surveillance system at all levels and development of methods to improve this;
3. Establishment of a Gonococcal Advisory Group (GAG) to examine and advise on issues of diagnosis, surveillance, management etc. Members of this group include infectious diseases clinicians, public health practitioners, representatives from all laboratories and Assoc Prof John Tapsall;
4. Investigation of the establishment of sentinel surveillance sites;
5. Education of clinicians of the importance of requesting culture as well as PCR on all specimens.

Current testing methods and penicillin resistance in *Neisseria gonorrhoeae* in North Queensland

Contributed by David Bradford, Director of Sexual Health, Cairns District

Prevalence of gonorrhoea has always been high in rural and remote Indigenous communities and until fairly recently there was little sign that any intervention had much impact. Most medical intervention occurs in primary health care centres, and not in specialist clinics, so its efficacy depends on both the health care seeking behaviour of those affected by sexually transmitted infections and the ability of generalist staff to recognise symptoms and signs and to adhere to standard management guidelines. The cultural significance of shame factors together with the reluctance to seek treatment for embarrassing and only minimally symptomatic genital conditions in affected communities militate against good control. Another important consideration is the fact that much early gonococcal infection is asymptomatic, so those affected with asymptomatic disease will go undetected unless specific screening interventions are in place and opportunistic testing of sexually active people is proactively encouraged by primary health care staff.

The recent availability of non-invasive nucleic acid amplification tests has meant that, for the first time, there is an opportunity to effectively screen asymptomatic people for gonococcal infection. With rapid follow-up of positive results, and appropriate treatment, it is possible some impression can be made on the high prevalence rates. It is an easy concept to promote amongst generalist staff – that painful, embarrassing and invasive tests need no longer deter people from being tested. It is a sad irony that just as this dream seemed capable of being achieved - with very high PCR testing rates being recorded in North Queensland - penicillin resistance in the form of beta-lactamase producing strains of *N. gonorrhoeae* suddenly appeared in indigenous communities.

Thus, those responsible for gonococcal control in northern Australia face a difficult dilemma at the present time. On the one hand they want to continue to promote screening and opportunistic testing of asymptomatic sexually active people attending primary care clinics using non-invasive DNA amplification tests - and to keep this message high on the agenda of generalist staff. On the other hand, they want to maintain surveillance of penicillin and other antibiotic resistance in local gonococcal strains by encouraging generalist staff to take appropriate specimens for culture and sensitivity testing. It is always difficult to promote mixed messages!

Practical steps which have been taken to date are:

- Queensland Health has officially changed standard drug protocols for treatment of gonorrhoea so that first-line treatment is now ceftriaxone throughout the State;
- primary health care staff are being encouraged to continue opportunistic screening using PCR tests in asymptomatic people;
- primary health care staff are also being encouraged to take swabs for culture and sensitivity on all those who present with symptoms, and on women who have vaginal speculum examinations for any reason.

Annual report of the Rotavirus Surveillance Programme, 1999/2000

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Abstract

The National Rotavirus Reference Centre has conducted rotavirus surveillance by means of a collaborative laboratory based initiative started in June 1999. The serotypes of rotaviruses that lead to the hospitalisation of children with acute diarrhoea were determined from June 1999 to May 2000. We examined 1126 rotavirus specimens using a combination of monoclonal antibody immunoassay, reverse transcription-polymerase chain reaction, and hybridisation. The four most common serotypes G1-G4 were represented. More than 50% of isolates tested were serotype G1, with serotype G1 being represented in most centres Australia-wide. Serotype G9 rotaviruses were identified for the first time in Australia, and were second in importance with 10% of samples tested. The significant presence of G9 viruses throughout Australia suggests the emergence of a new serotype and has implications for current rotavirus vaccine strategies that target serotypes G1-G4. *Commun Dis Intell* 2000;24:195-198.

Keywords: rotavirus, surveillance, infants, serotypes, vaccine, gastroenteritis

Introduction

Rotaviruses are the most important cause of severe gastroenteritis in young children worldwide. The pathogen is believed to be responsible for the annual admission of up to 10,000 children to hospitals nationwide.[1] A national rotavirus surveillance programme was commenced in June 1999 to undertake the surveillance and characterisation of rotavirus strains causing annual epidemics of severe diarrhoea in young children throughout Australia. The programme was designed to monitor the antigenic variation of rotaviruses prior to and after the anticipated rotavirus vaccine release in Australia. The study was reliant on the cooperation and participation of sentinel laboratories from all States and the Northern Territory. It was designed to supplement data from existing notification schemes, by reporting the serotypes circulating in Australian urban centres. The following report covers the period June 1999 to May 2000.

Methods

A network of laboratories from each State and the Northern Territory undertake rotavirus detection by enzyme immunoassay (EIA) or latex agglutination. Rotavirus-positive specimens were collected, stored frozen and forwarded to the Royal Children's Hospital (RCH) in Melbourne, together with relevant age and sex details. Representative faecal specimens were then tested using an in-house monoclonal antibody (MAb) based serotyping EIA. The EIA incorporates a panel of MAbs specific for the common group A human rotavirus serotypes (serotypes G1, G2, G3, and G4). Specimens with an absorbance value greater than 0.2 were considered positive for that serotype. Northern hybridisation analysis with G type specific DNA probes using stringent hybridisation conditions was also employed to confirm serotype specificities. Selected strains unable to be assigned a serotype, were genotyped by reverse transcriptase/ polymerase chain reaction using serotype specific primers.

Results

Number of isolates

In all 1545 rotavirus positives were sent to the Royal Children's Hospital. Specimens received from New South Wales made up 30.5% of all specimens received (Newcastle, Narrabri and Sydney). Twenty-five percent of specimens were received from Western Australia (Perth and northern Western Australia), 16% from Victoria (Melbourne and Horsham), 14% from South Australia (Adelaide), 7% from Queensland (Brisbane and Townsville), 7% from the Northern Territory (Darwin, Gove and Alice Springs) and 0.5% from Tasmania (Hobart). Representative specimens were incorporated into the serotyping EIA (1126 specimens). Specimens that were not confirmed to be positive for rotavirus, or had insufficient specimen for testing, were excluded.

Seasonal occurrence

The overall nationwide rotavirus reports peaked in August (Figure 1). The peak month of activity varied between centres, with temperate regions experiencing a noticeable winter peak between July and October. The rotavirus seasons of combined centres in Victoria, Tasmania and New South Wales peaked in August, followed by Queensland in September (Figure 2). Western Australia shared its rotavirus peak with Alice Springs and Adelaide in October. The tropical Northern Territory rotavirus season peaked in January 2000.

Age group

The age distribution of rotavirus-positive patients showed the peak incidence occurred in children aged between 1 and 2 years (Figure 3). The male:female ratio was 1.18:1.

Serotype distribution

The serotypes circulating in Australia from June 1999 to May 2000 are shown in Figure 4. Serotype G1 accounted for

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Figure 1. Rotavirus reports, Australia, June 1999 to May 2000, by month

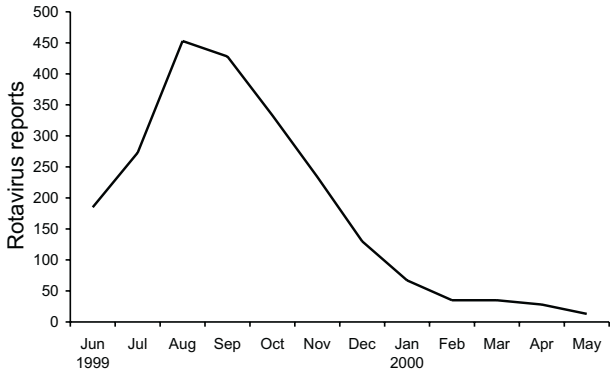


Figure 2. Seasonal peaks of rotavirus reports, Australia by region, June 1999 to May 2000, by month

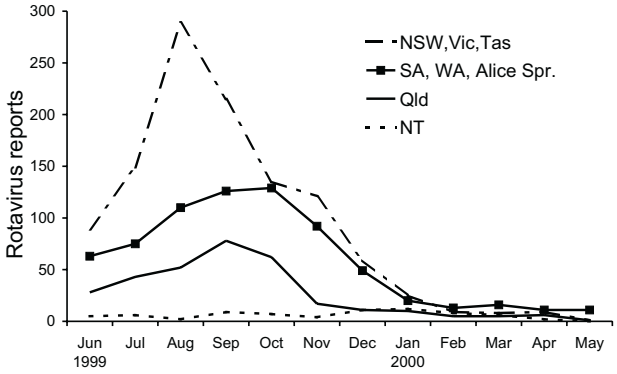
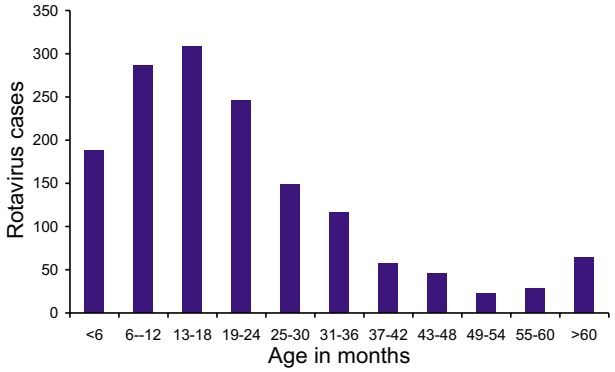
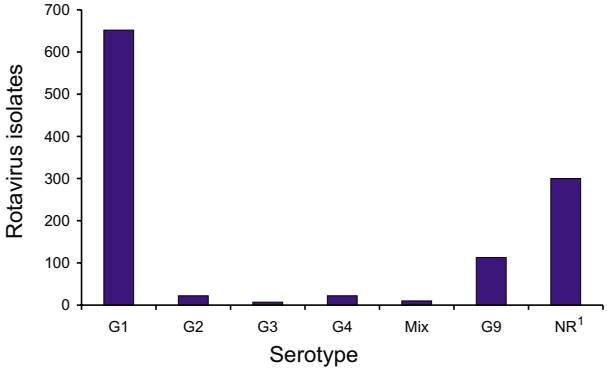


Figure 3. Rotavirus positive children, Australia, June 1999 to May 2000, by age group in months



58% of specimens nationally, and was responsible for more than 60% of rotavirus serotypes in some centres; Hobart (100%), Darwin (80%), Northern Western Australia (69%) and Perth (80%) (Table 1). Second in importance to G1 were G9 rotaviruses (10%). The G9 rotaviruses appeared in Sydney and Melbourne in June 1999 and were present in 12 of the 18 centres studied (all centres except Darwin, Townsville, Horsham, Gove, northern Western Australia

Figure 4. Rotavirus isolates, Australia, June 1999 to May 2000, by serotype



1. NR = non-reactive

and Hobart). Serotype G2, G3 and G4 viruses accounted for less than 5% of all serotypes detected. Serotype G2 viruses were detected along the east coast of Australia, Narrabri, Adelaide, Perth and northern Western Australia. Serotype G3 viruses were present in Melbourne and Adelaide only. Serotype G4 viruses were found in all States except Tasmania and the Northern Territory. A serotype could not be assigned to 27% of specimens. Specimens containing mixtures of rotavirus G types were detected in less than 1% of samples tested.

Discussion

The surveillance study was marked by the appearance of G9 rotaviruses in Australia for the first time.[2] The virus was responsible for at least ten percent of acute diarrhoea rotaviral hospital admissions. Retrospective analysis showed two G9 viruses were present in Perth and Melbourne in 1997. The subsequent spread of G9 viruses appears to be almost nationwide with the increase in numbers making G9 viruses second in importance to G1 viruses. G9 viruses were found in all States except Tasmania. G9 viruses have been reported in India (1993-1994),[3] Bangladesh (1987-1997),[4] Malawi (1997-1998),[5] the USA (1996-1997)[6] and the UK (1996).[7] The rapid emergence of G9 as a major infecting serotype has important implications for rotavirus vaccine strategies. Current candidate vaccines target only serotype G1-G4 infections. Ongoing surveillance is warranted to obtain a clearer picture of the importance of the spread of G9 viruses.

Serotype G1 viruses were the most prevalent serotype in Australia during the sampling period. This G1 dominance was consistent with recent studies undertaken in Australia (1993-1996) (Personal communication, Professor Ruth Bishop, Department of Gastroenterology, Royal Children's Hospital. Parkville Victoria), the UK (1996)[7] and the USA (1996-1997)[6]. The virus was present in all centres tested except Townsville and Narrabri.

Centres that showed evidence of more than one serotype generally had larger populations. There appeared to be more serotypes circulating and presenting as hospital admissions in the bigger centres. Smaller population centres such as Hobart, Townsville and Darwin had only one serotype detected and appear to have insufficient population size to sustain multiple G types. Serotype G2, G3

Table 1. Rotavirus positive specimens, Australia, June 1999 to May 2000 by typing centre and serotype

	Serotype							
Centre	G1	G2	G3	G4	G9	Mix	NR[1]	Total
Brisbane	31	2		2	5	1	34	75
Townsville		3					16	19
South-eastern Sydney	123			1	31		52	207
Western Sydney	46	1		2	20	1	23	93
Newcastle	2			2	7		5	16
Narrabri		2			2		19	23
Melbourne[2]	117	1	5	8	26	5	72	234
Melbourne[3]	3						1	4
Horsham	6			2				8
Hobart	5							5
Adelaide	52	5	2	2	3	2	11	77
Perth	199	4		3	7	1	32	246
WA PathCentre	18	4					4	26
Darwin[4]	22						2	24
Darwin[5]	6						5	11
Gove	2						1	3
Alice Springs	20				12		23	55
Total	652	22	7	22	113	10	300	1,126

1. NR = Non reacting to G1, G2, G3, G4 and G9 monoclonal antibodies.
2. Royal Children's Hospital
3. Southern Cross Pathology
4. Royal Darwin Hospital
5. Western Diagnostic Pathology

and G4 viruses were present in small numbers and appeared sporadically.

Antigenic similarities between G4 and G9 viruses were noted. There were a number of specimens that reacted to both G4 and G9 serotyping MAbs. The use of Northern hybridisation and RT/PCR analysis clarified any serological cross reactivities.

Several specimens were unable to be assigned a serotype by EIA. These non-reactive (NR) specimens, generally contained minimal amounts of viral capsid protein and were unable to be detected by the serotyping MAbs. Electrophoretic analysis of the RNA from some of them showed that the viruses circulating in Townsville, Brisbane and Narrabri all shared the same electrophoretic profile. RT/PCR typing of limited numbers showed the samples were serotype G2. Type G2 viruses were under represented in the serotyping EIA suggesting genetic changes in the epitopes targeted by G2-specific MAbs. Specimens received from remote regions of northern Western Australia shared serotype similarities with specimens collected from Perth children. This coexistence of virus serotypes in geographically diverse locations suggests rapid spread of virus strains across the country. The incorporation of remote locations into the study helped to gain a better insight into the pattern of spread.

Ongoing surveillance of seasonal rotavirus serotype patterns is warranted, in particular to monitor the spread of new or emerging serotypes. Such information will influence the strategy for development of second and third generation rotavirus vaccines, and will show whether Australia's requirements differ from those of other parts of the world. This is particularly important now that the first G1-G4 targeted vaccine has been withdrawn from sale in the USA due to its apparent association with intussusception - a form of bowel obstruction.

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Legionnaires' disease outbreak: Victoria's largest identified outbreak

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Abstract

This paper describes and analyses some aspects of an outbreak of Legionnaires' disease in Victoria, commencing in late October 1998. In all, 18 cases caused by *Legionella pneumophila* serogroup 1 were notified within 10 days making this the largest outbreak in Victoria reported to that date. All cases had epidemiological links to an industrial estate in a northern Melbourne suburb. Extensive environmental sampling revealed *Legionella* bacteria in five cooling towers. Molecular sub-typing techniques were used to compare clinical and environmental isolates. Isolates from one tower had a pulsed-field gel electrophoresis pattern that was indistinguishable from clinical isolates from eight cases. Control of outbreaks caused by *Legionella* bacteria requires rapid, coordinated responses to linked cases of disease. The *Legionella* urinary antigen test facilitated a rapid public health response, and culture and molecular sub-typing of clinical specimens assisted in developing epidemiological links. *Commun Dis Intell* 2000;24:199-202.

Keywords: legionellosis, surveillance, urinary antigen, cooling towers, environmental health

Introduction

Legionnaires' disease is caused by *Legionella* bacteria, which are gram-negative intracellular pathogens. The typical presentation is a severe community-acquired or nosocomial pneumonia.[1] European and North American studies have estimated that *Legionella* species may cause between 2% and 15% of all community-acquired pneumonia requiring hospitalisation.[2] In Australia, from national data on notified cases, the rate is approximately 1 per 100,000 persons (Communicable Diseases Network Australia New Zealand National Notifiable Diseases Surveillance System; personal communication). The case fatality rate has been reported as ranging from 5 to 30% depending on underlying risk factors of patients.[3] The disease is a public health priority, since it is potentially preventable through ongoing identification and treatment of environmental sources, and can be treated effectively with antibiotics if diagnosed promptly.

Legionnaires' disease has been notifiable in Victoria by doctors and laboratories since 1979. The definitive laboratory diagnosis for the disease is culture from respiratory specimens on selective culture media.[1] The other methods routinely in use in Victoria are serological testing and the *Legionella pneumophila* serogroup 1 (LP1) urinary antigen test. Most outbreaks of Legionnaires' disease in Australia have been due to LP1.[4-10] Between 1995 and 1998, LP1 comprised 82% of all notifications of Legionnaires' disease in Victoria (unpublished observations).

In Victoria, the case definition for Legionnaires' disease is: - a clinically compatible illness (pneumonia) and at least one of the following: (1) culture isolation of *Legionella* species; (2) fourfold rise in immunofluorescence (IFA) titre in paired sera to at least 128; (3) stable high titre (>512) IFA in convalescent serum; (4) demonstration of *Legionella* species antigens in urine or other specimens.[11] In late October 1998, over 2 days, the Communicable Diseases Section of the Victorian Department of Human Services (DHS) received three notifications of Legionnaires' disease due to LP1. The cases were men who lived or worked in the vicinity of Thomastown, a northern Melbourne suburb. The close temporal and geographical clustering of cases suggested that the three cases were related. An outbreak investigation was initiated to prevent further transmission, to identify undiagnosed cases, and to determine the source(s) of the outbreak.

Methods

We undertook telephone interviews of cases or of their next of kin, using a standardised case questionnaire. All were asked about possible risk factors for infection with LP1 in the 10-day period prior to onset of illness. Results from the case questionnaires were used to direct environmental investigations. Workplaces of cases in employment were contacted to determine whether there were cooling towers at the workplace or in the immediate vicinity.

Enhanced community and hospital surveillance for LP1 was undertaken concurrently with the investigation. This involved media releases directed at the general public, letter-drops to local residents, health alerts distributed by pathology companies to general practitioners, and hospital alerts to local and major regional hospitals. Further

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measures included alerts to local Divisions of General Practice, and workplace illness surveillance for both any workplace with a confirmed case, and all workplaces in the industrial estate where the outbreak appeared to be centred. When a diagnosis of LP1 was made by the *Legionella* urinary antigen test, the clinician was contacted and urged to pursue culture confirmation if possible.

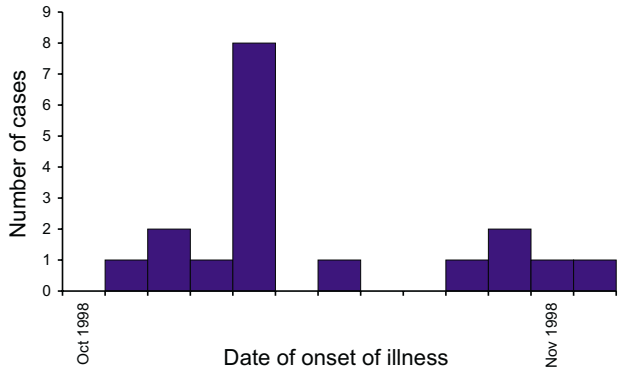
Officers of the Environmental Health Unit of the DHS inspected suspected areas and identified potential exposure sources with the focus on cooling towers. Cooling towers identified in the suspected area were inspected, water samples collected for testing, and owner/managers ordered to organise immediately for the towers to be treated with biocidal agents. Compliance with the Health (Infectious Diseases) Regulations[12] relating to routine tower maintenance was assessed, and water-testing results were reviewed.

Methods for isolation of *Legionella* from sputum, for LP1 urinary antigen analysis, and subtyping by pulsed field gel electrophoresis are described elsewhere.[1]

Results

In all 18 epidemiologically linked cases satisfying the case definition were identified. Figure 1 is the epidemic curve for the outbreak. The first case was confirmed on 29 October on the basis of the urinary antigen test. This case was culture confirmed on 30 October, but the next positive clinical isolate was only obtained on 2 November, although additional cases had been confirmed by the urinary antigen test on 30 October.

Figure 1. Legionnaires' disease cases, Melbourne, Victoria, 21 October to 2 November 1998, by date of onset of illness

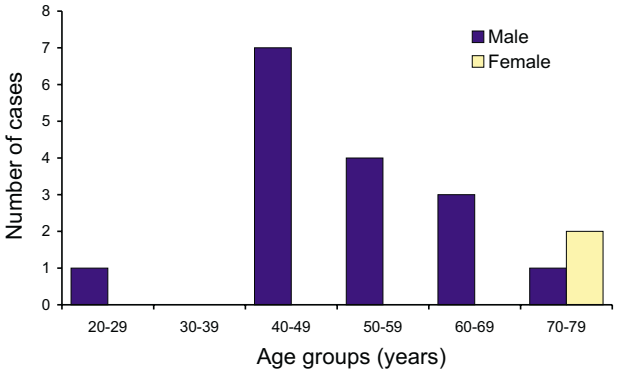


Case demographics

The age and sex distribution of cases is shown in Figure 2. Of the cases, 16/18 (89%) were male (male/female ratio = 8:1), with the median age for males being 50 years. Of the males, 13/16 (81%) were employed full-time, two were retired and one was on sickness benefits. The two females were retirees aged 71 and 74 years (for cases of Legionnaires' disease in Victoria, the median female age between 1995 and 1998 was 61 years).

Of the cases, 15/18 (83%) were hospitalised, with 2/15 (13%) hospitalised cases ventilated in intensive care. There were no fatalities. The major risk factors for illness were being a working male aged 40-70 years who smoked (14/18

Figure 2. Legionnaires' disease cases, Melbourne, Victoria, 21 October to 2 November 1998, by age group and sex



(78%) were regular smokers) and had epidemiological links to the implicated area through either work, residential address or travel on the major arterial road. From the epidemic curve, it can be estimated that the period of likely exposure for most cases was the third week of October 1998.

Of the cases, 16/18 (89%) were initially notified on the basis of a positive *Legionella* urinary antigen result, with 7/16 (44%) of these subsequently being culture-confirmed. For seven of the urinary antigen-positive cases, sputum culture was negative for *Legionellae* or other respiratory pathogens. Sputum was not available for testing from the two other urinary antigen-positive cases. For the seven urinary antigen- and sputum-positive cases (all hospitalised), the median time from hospital admission until sputum collection was 1 day (range 0-1 days). For the six urinary antigen-positive but sputum-negative cases hospitalised, the median time from hospital admission until sputum collection was 2 days (range 1-7 days).

Three suspected cases identified through active case finding in local hospitals had negative urinary antigen tests initially, but these proved positive after the initial urine specimens were concentrated and retested. These three cases were subsequently confirmed by culture.

One non-hospitalised case was initially notified from a positive sputum isolate; this case had a negative urinary antigen result on urine collected 3 days after onset of illness. One hospitalised case did not have sputum collected (and was negative by urinary antigen test on urine collected 6 days after onset of illness) but had a high titre acute serology result. For the 16 urinary antigen positive cases, there was a median time of 5 days from onset of illness until urine was collected (range 1-9 days).

Environmental investigations

Twenty-three premises were inspected and their compliance with the Health (Infectious Diseases) Regulations 1990[12] assessed. Most premises were found to have complied with the Regulations. A minority had only partially complied with the regulations, while some were found to have not complied with any aspect of them. Only one of the premises was found to have had recent routine biocidal treatment of its cooling tower; this had been done the day before DHS testing. Premises with lack of evidence of compliance with the regulations were informed of their

regulatory responsibilities and educated by DHS on the optimisation of their cooling tower maintenance practices. These premises were subsequently attended to ensure that appropriate practices for maintenance had been instituted.

After sampling, towers in the implicated area were treated with biocidal agents. Decontamination was carried out on cooling towers where *Legionella* bacteria were isolated. In total, of the 65 cooling towers sampled, five tested positive for LP1. Some of these LP1-positive cooling towers were compliant with the minimum requirements for maintenance, emphasising there may be a need for higher levels of maintenance for particular sites.

All clinical and environmental isolates of LP1 were subtyped by pulsed-field gel electrophoresis (PFGE). A total of eight clinical isolates and five isolates from different cooling towers were analysed. All eight clinical isolates were indistinguishable by PFGE, and generated an electrophoretic pattern not previously reported in Victoria. The same novel pattern was seen in an isolate from one of the cooling towers sampled. This tower had not been maintained in accordance with the Regulations. Isolates from other cooling towers demonstrated electrophoretic patterns different from that of the outbreak strain. One isolate from a cooling tower did not survive to be able to be submitted for subtyping.

Discussion

Availability and use of the *Legionella* urinary antigen test allowed earlier outbreak identification, and earlier confirmation of most suspected cases of Legionnaires' disease, If *Legionella* urinary antigen testing had not been available, the outbreak would not have been identified until 4 days later (on 2 November). The initial case was culture-confirmed on 30 October, but the next positive clinical isolate was not until 2 November. Until that time, DHS would have been investigating the initial case as a sporadic case, rather than an outbreak. This means that the extensive measures specifically undertaken in this outbreak would have been delayed. These included the extensive environmental inspections and sampling undertaken, and the range of alerts to the general public, doctors and hospitals initiated on the 30 October.

Furthermore, the use of the urinary antigen test meant that we were able to have laboratory confirmation on some cases on the day they first presented to doctors. This greatly enhanced the early stages of the investigation, when we were confronted by a number of hospitalised patients with pneumonia which could have been due to any of a range of pathogens. This relative ease of obtaining prompt confirmation of cases contrasts with experiences reported from an outbreak of LP1 in Sydney in 1992, where direct immunofluorescent staining (DFA) was used as a rapid diagnostic tool.[13]

Of urinary antigen-positive cases, 7/16 (44%) were subsequently culture-confirmed. It was noted that, in contrast with those with negative sputum cultures, the hospitalised cases with culture confirmation had sputum collected soon after admission. Once cases were hospitalised and had commenced antimicrobial therapy - and the longer they were on that therapy - the less likely was *Legionella* to be cultured from their sputum. Thus to optimise culture-confirmation of cases, sputum should be collected as soon as possible from suspected cases of Legionnaires' disease

It is important to recognise the limitations of the urinary antigen test. It is valid only for LP1 and moreover, although a positive test is considered to be almost 100% specific, a negative test does not exclude *Legionella* infection.[14] However, concentration of urine specimens by ultrafiltration increases test sensitivity without loss of specificity.[15] This proved helpful in this outbreak since three sputum positive cases, initially testing negative on routine urinary antigen testing, were positive after urinary concentration.

For this outbreak, 9/16 (56%) of the urinary antigen-positive cases had urine collected 5 days or less after the onset of illness, which indicates the urinary antigen test is useful in diagnosis in the very early stages of illness. One study has reported that antigen was detectable in some cases as early as 2 days after onset of illness.[16] Another has reported that urine samples collected within the first 5 days of the disease may be negative.[17] The two cases in this outbreak with negative urinary antigen results had urine collected for testing 3 and 6 days after the onset of illness. Urine may continue to test antigen-positive for many weeks after onset of illness. It has been reported that 10% of culture-confirmed cases of Legionnaires' disease are still urinary antigen test-positive after 60 days.[1] Another study reported that most cases were urinary antigen-positive between 3 and 5 weeks after illness onset.[18]

Our results suggest that, because urine antigen testing was used, most cases were identified earlier and treated appropriately; alternatively less severe cases were identified which may not otherwise have been correctly diagnosed.

Although an alternate source was not excluded, a possible source of the outbreak was the cooling tower where the LP1 isolate with a PFGE pattern indistinguishable from the clinical isolates was found. This tower had not been maintained in accordance with the Regulations. The latter require regular cleaning and disinfection of towers, monthly microbiological monitoring by Total Bacteria Counts, and maintenance of documentation to confirm these activities.[12] The role of the cooling tower with the LP1 isolate which did not survive to permit PFGE analysis is uncertain.

As a result of our experience from this outbreak, we recommend the following practices in the investigation of cases of Legionnaires' disease. Clinicians should be encouraged to use the *Legionella* urinary antigen test. The test should be used in conjunction with culture of respiratory specimens (to allow molecular sub-typing) and collection of serological specimens (in case urine and sputum tests are negative, and to exclude alternate causes of atypical pneumonia). If the initial urinary antigen test is collected in the first week of illness and is negative for a suspected case, it is worth repeating the test in the second week of illness. Opportunities exist for collaboration between clinicians, public health practitioners and diagnostic laboratories to develop algorithms for determining which urinary specimens should be concentrated to improve sensitivity. Used appropriately, the urinary antigen test can be a valuable tool in the investigation of sporadic cases or outbreaks in jurisdictions where there is a high proportion of cases due to LP1.

We recommend that cooling towers in Victoria be maintained in accordance with the Regulations.[12] Appropriate cooling tower design and maintenance has been shown[19] to minimise the risk of proliferation of *Legionella* bacteria in cooling towers: this may reduce the risk of the latter

becoming sources of infection for sporadic cases or even outbreaks of Legionnaires' disease.

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Editorial Statement. The October/November 1998 outbreak reported by the authors was the largest recorded outbreak in Victoria at the time their article was first submitted to *Commun Dis Intell* (29 October 1999).

Hepatitis A in south-eastern Sydney 1997-1999: continuing concerns for gay men and an outbreak among illicit drug users

Valerie C Delpech,[1] Sarah V Thackway,[2] Lorraine Young,[1] Giulietta Pontivivo,[1] Elizabeth Smedley,[1] Keira Morgan,[1] Mark J Ferson[1]

Abstract

The incidence of hepatitis A virus (HAV) in south-eastern Sydney is one of the highest in Australia with large outbreaks previously associated with male-to-male sexual contact. We report HAV notification trends over the period 1 June 1997 to 31 May 1999 for this location. In the first twelve-month period, 233 cases were notified (crude rate 30.5/100,000 per year) with a peak incidence of 110/100,000 in males aged 20-39 years. Over 60% of male cases reported male-to-male sexual contact. The notification rate (crude rate 15.5/100,000) and proportion of males (61%) was considerably lower in the following twelve month period with 118 cases notified. Less than a third of males reported male-to-male sexual contact. An outbreak (n = 45) of HAV among illicit drug users and their contacts was detected in December 1998. The transmission of HAV remains endemic in south-eastern Sydney. Vaccination among high-risk groups remains an important preventative strategy. *Commun Dis Intell* 2000;24:203-206

Keywords: hepatitis A virus, illicit drug use, male-to-male sexual contact, surveillance

Introduction

South-eastern Sydney has one of the highest incidences of hepatitis A virus (HAV) infection in Australia.[1] Large HAV outbreaks associated with male-to-male sexual contact have occurred in inner and eastern suburbs of Sydney. Peak notification rates of 520/100,000/year in 20-29 year old males and 405/100,000/year in 30-34 year males were recorded during two recent outbreaks in 1991-2 and 1995-6 respectively.[1,2]

Cases of HAV among injecting drug users (IDUs) have also been reported in south-eastern Sydney.[1,3] In the 1994-5 outbreak, one quarter of all HAV cases notified to the South East Sydney Public Health Unit (SESPHU) reported a recent history of injecting drug use.[3] Outbreaks of HAV among IDUs have also been documented in a number of countries including the United States,[4-8] Canada,[9] Norway,[10,11] Finland[12] and Sweden.[13] More recently, Queensland health authorities described several linked outbreaks of HAV among illicit drug users.[14] Of the 800 cases notified in Queensland during 1997, a quarter was associated with drug use.

In New South Wales, hepatitis A is notified to the local public health unit by doctors on clinical suspicion and by laboratories on detection of anti-HAV IgM. Case investigation and public health follow up are conducted by public health and clinical staff on all confirmed and suspected cases. A confirmed case of hepatitis A is defined as a person with a laboratory report of anti-HAV IgM in serum with symptoms of acute hepatitis A or epidemiologically linked to a case confirmed serologically. Details of confirmed cases are recorded onto the New South Wales Notifiable Diseases Database (NDD). Additional information collected through case investigation, including potential source/s of infection, risk factors and exposure, is recorded onto a discrete SESPHU hepatitis database.

In this article, we review notifications of HAV in south-eastern Sydney over a two year period (June 1997 to May 1999) and report on an outbreak among illicit drug users detected in December 1998.

Methods

Methods of data collection and the information contained in the SESPHU hepatitis database have been described previously.[1] Cases of HAV infection notified to the SESPHU with an onset date between 1 June 1997 and 31 May 1999 were extracted from NDD and the SESPHU hepatitis database. Data analysis was conducted using Epi Info 6. Australian Bureau of Statistics census data were used to estimate populations for south-eastern Sydney. Illicit drug use was defined as the use of illicit drugs (including injecting drugs) within the previous two months.

Results

Over the two-year period, 354 cases were notified to the SESPHU (Figure 1). Distinct patterns of notifications were noted involving two twelve-month periods (June 1997 to May 1998 and June 1998 to May 1999). Each period will be discussed separately.

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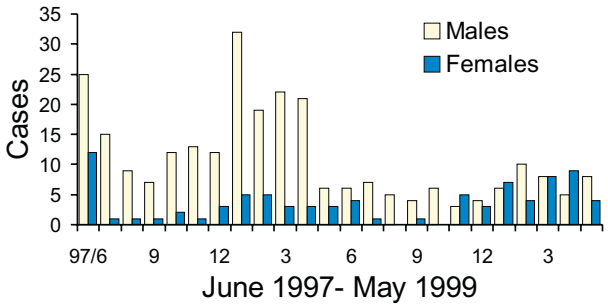
Figure 1. Hepatitis A notifications, South East Sydney Health Service Area, June 1997 to May 1999



June 1997 - May 1998

Between June 1997 and May 1998, 236 cases of HAV were reported (crude rate 30.9/100,000 per year) with incidence peaks in the months of June 1997 (n = 37) and January 1998 (n = 37) (Table 1). Twenty-seven (12%) reported contact with a person who had a clinical history and/or a diagnosis of HAV and 12% had a history of recent overseas travel.

Adult males accounted for 82% of cases, representing a rate of 50.8/100,000 (compared to 11.2/100,000 in women). Male cases were aged between 12-81 years (mean 32 years). However, most (78.2%) males were aged between 20-39 years with an age-specific rate of 110.1/100,000. Male-to-male sexual contact was reported in 61% of male cases with 83% residing in inner and eastern Sydney. Only four (2%) cases reported injecting drug use.

June 1998 - May 1999

During the 1998-99 period, the notification rate was considerably lower than the previous twelve months with 118 cases notified (crude rate 15.5/100,000) (Table 2). Cases ranged between 1-76 years in age (mean 33 years) and resided predominantly in inner and eastern Sydney (71.2%). Males accounted for 61% (rate 19.0/100,000) and less than a third (29%) reported male-to-male sexual contact.

An increase of HAV cases among the illicit drug users and their contacts was detected in December 1998. Over the ensuing six months (1 December to 31 May 1999), 45 of the 76 (59%) HAV notifications reported illicit drug use or had contact with an illicit drug user. A small but continuing number of cases was reported each week with no peak in notifications.

Demographic and risk factor information on cases associated with the outbreak is detailed in Table 3. The majority (69%) were residents of eastern Sydney and, in particular, the Kings Cross area (Area 2, Figure 2). The male: female ratio was 1: 1.02 and the mean age was 28 years (range 7-72).

Thirty-one (69%) of the 45 cases used illicit drugs: 12 (27%) reported smoking marijuana and 29 (64%) reported injecting drug use. Nine cases (20%) reported sex work, and four males (13% of all male cases) reported male-to-male sexual contact. Two of the cases had recently been in or visited a detention centre. No case had travelled overseas in the two months prior to illness.

Table 1. Hepatitis A cases and rates,[1] south-eastern Sydney residents, June 1997 to May 1998, by age and sex

	Males		Females		Total	
Age-group	Number	Rate	Number	Rate	Number	Rate
<5	0	0.0	1	4.8	1	2.3
5-19	11	16.9	6	9.6	17	13.3
20-39	151	110.1	28	21.2	179	66.5
40-59	30	30.9	7	7.4	37	19.3
60+	1	1.7	1	1.4	2	1.5
Total	193	50.8	43	11.2	236	30.9

1. Rates per 100,000 persons

Table 2. Hepatitis A cases and rates,[1] south-eastern Sydney residents, June 1998 to May 1999, by age and sex

	Males		Females		Total	
Age-group	Number	Rate	Number	Rate	Number	Rate
<5	0	0.0	2	9.6	2	4.7
5-19	1	1.5	11	17.7	12	9.4
20-39	50	36.5	24	18.1	74	27.5
40-59	17	17.5	6	6.4	23	12.0
60+	4	6.9	3	4.1	7	5.3
Total	72	19.0	46	12.0	118	15.5

1. Rates per 100,000 persons

Table 3. Outbreak of Hepatitis A among illicit drug users and their contacts, south-eastern Sydney, December 1998 to May 1999, n = 45

Demographic/ Risk Factor	Number of cases	%[1]
Males[2]	23	51
Resident of eastern Sydney	31	69
Illicit drug use	31	69
Injecting drug use	29	64
Prison	2	4
Known contact with a person with HAV	3	7
Sex work	9	20
History of travel	0	0
Eating at community food vans	7	16
Male to male sexual contact	4	13

1. % of all male cases
2. Age: mean = 28y, median = 27y

Discussion

Hepatitis A remains endemic in south-eastern Sydney, with gay men continuing to be at particularly high risk of contracting the illness. More recently, HAV infection rates have increased among illicit drug users and their contacts. The outbreak was first identified in late 1998 and was predominately reported within the Kings Cross area, containing one of Australia's largest populations of IDUs. Unlike the 1994-1995 epidemic, there was no apparent increase of HAV among persons reporting risk factors other than illicit drug use over the same period and only four cases reported male-to-male sexual contact.

Epidemics of HAV among illicit drug users have also recently been noted in northern New South Wales (personal communication Marianne Trent, Infectious Diseases Clinical Nurse Consultant, Northern Rivers Institute of Health and Research), Queensland[14] and other Australian capital cities.[15] While HAV outbreaks among illicit drug users have been reported nationally and internationally,[1-15] the route of transmission in most cases remains unclear and is probably multifactorial.[16-19]

Possible transmission routes of HAV infection associated with drug use include injection or ingestion of contaminated drugs[4,6,18] and direct or indirect person-to-person contact, such as behaviours related to sharing needles, sexual contact or poor personal hygiene.[1,5] Various injected drugs have been associated with HAV outbreaks, including heroin, amphetamines, and cocaine.[6] However, HAV has purported to have been transmitted through non-injecting drug use, including smoking marijuana.[6,8] While there have been reports of parenteral transmission of HAV,[19] the relatively short viraemic phase of HAV infection means that parenteral transmission of HAV is unlikely to have been a common mechanism in cases in injecting drug users.[6]

Further epidemiological investigation of the south-eastern Sydney outbreak is being undertaken in an attempt to identify risk factors for HAV among illicit drug users and ascertain potential sources of transmission that may be amenable to preventative measures. Vaccination against hepatitis A among high-risk groups remains an important preventative strategy.

Acknowledgements

The SESPHU would like to acknowledge the assistance of Kirketon Road Centre staff, in particular Ingrid van Beek and Craig Rodgers, in identifying the outbreak and subsequent investigations.

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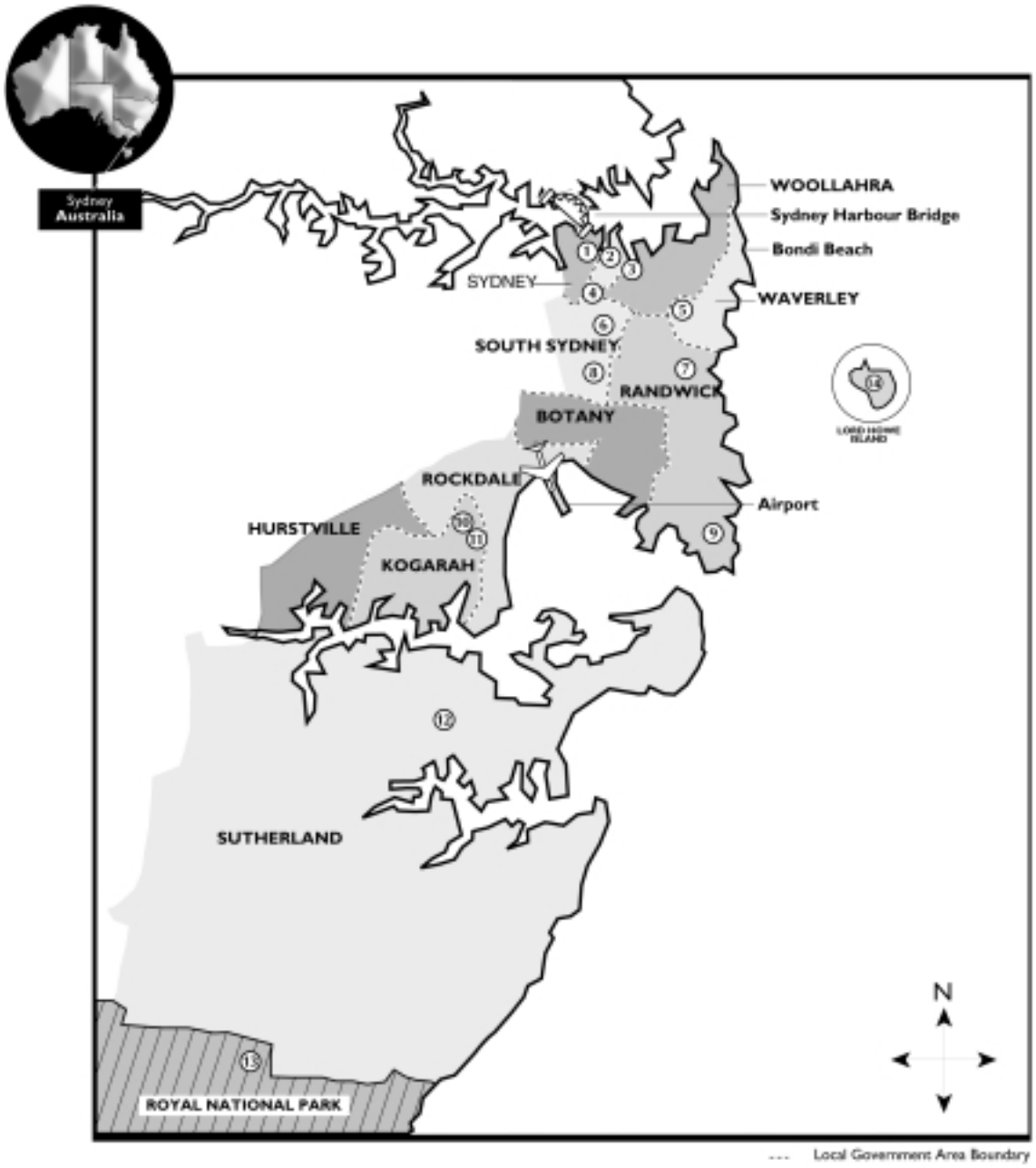
Figure 2. South East Sydney Health Service Area



LEGEND

1. Sydney & Sydney Eye Hospital
2. Kirketon Road Centre
3. St Vincent's Hospital
4. Albion Street Centre
5. War Memorial Hospital
6. Langton Centre
7. Prince of Wales Hospital, Sydney Children's Hospital & Royal Hospital for Women
8. Royal South Sydney Community Health Complex
9. Prince Henry Hospital
10. St George Hospital
11. Calvary Hospital
12. Sutherland Hospital
13. Garrawarra Centre for Aged Care
14. Gower Wilson Memorial Hospital

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The laboratory containment of wild poliovirus in Australia

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Keywords: poliovirus, containment, survey, inventory, infectious material, eradication

In September 1988, the thirty-ninth session of the World Health Organization's (WHO) Regional Committee for the Western Pacific adopted a resolution calling for the eradication of poliomyelitis in the Region by the year 2000.[1] Australia, along with other countries of the Western Pacific Region, is now moving rapidly towards the certification of poliomyelitis eradication. If all countries within the Region provide evidence consistent with the absence of indigenous wild poliovirus for three consecutive years under conditions of high quality surveillance, the Region can be certified as poliomyelitis-free.

The last case of indigenous acquired poliomyelitis, an 18 month old girl from Cambodia, occurred in the Region in March 1997.[2] Over three years have passed and it is therefore anticipated that the WHO will declare the Western Pacific Region free of poliomyelitis in the second half of 2000.

Because circulation of wild poliovirus in the region has been interrupted, the only known sources of wild poliovirus remaining are within the region's laboratories. These laboratories may store specimens from known poliomyelitis cases or store other materials that are potentially infected with wild poliovirus (Box 1). Therefore, the task of polio-myelitis eradication will not be complete until all known and potential sources of poliovirus are properly contained.

The Commonwealth Department of Health and Aged Care, with the assistance of the Victorian Infectious Diseases Reference Laboratory (VIDRL), is conducting a national laboratory survey that targets all Australian diagnostic, biological, environmental, reference, research, teaching, manufacturing and regulatory laboratories. The national survey is the next stage in Australia's efforts to eradicate poliovirus and aims to identify and produce an inventory of all wild poliovirus stocks and potentially infectious materials that are stored in Australian laboratories.

The development of a wild poliovirus inventory is part of the first phase of a larger world wide containment process that consists of three phases (Box 2). Phase two will commence one year after the last case of poliomyelitis is detected and phase three will commence at least three years after the global certification of polio eradication. In phase three oral polio vaccine will no longer be used.

In February 2000 VIDRL appointed a national coordinator of laboratory poliovirus containment. The role of the national coordinator includes writing a national plan for laboratory containment, coordinating the implementation of the plan and preparing the final national inventory for submission to the WHO Regional Office.

A national workshop on wild poliovirus containment

Six weeks following the appointment of the national coordinator, a workshop was held to discuss the concept and logistics of a national survey. Senior microbiologists were invited from each Australian State and Territory and, with the exception of two, all were able to attend. These microbiologists also represented many of the different classes of laboratories that could store poliovirus. The State/Territory representatives who were unable to attend provided comment on the outcome of the workshop.

Participants at the workshop discussed the optimal approach for identifying and communicating with laboratories and institutions, potential problems associated with identifying and locating infectious material, and specific technical problems relating to the containment and disposal of relevant material. In particular, a clear communication strategy was identified as critical for the smooth running of the national survey. Having reviewed the WHO national laboratory survey information pack and survey form, participants expressed concern with the complexity of the laboratory survey and speculated that laboratories may not be keen to participate. It was therefore decided to conduct two pilot surveys to evaluate the effectiveness of the standard documentation. A summary of the pilot surveys is outlined below.

At the conclusion of the workshop, a national advisory committee of six members was formed. Members of this committee included representatives from private diagnostic, research, reference, environmental regulatory laboratories

Box 1. Biological materials potentially infectious for wild poliovirus

1. **Materials known to be infected with wild poliovirus**	1.**Materials *potentially* infected with wild poliovirus**
• **Clinical/diagnostic specimens:** throat, faecal, blood, CSF, or unfixed autopsy specimens from polio cases.	• Materials stored in a manner known to preserve virus survival.
• **Environmental specimens:** water or sewage samples + polio (controls).	• AND collected in a place and during a time when wild poliovirus was circulating.
• **Research material isolates:** genetic material, cell lines, infected animals.	

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Box 2. Phases of laboratory containment of wild poliovirus

Phase	Actions	Timeline
I. Pre-regional certification of polio eradication	Safe handling of all wild poliovirus (or potentially infectious) materials in PC2/polio laboratory or above. Option to destroy wild poliovirus (or potentially infectious) materials or transfer to regional repository. Substitute Sabin vaccine or non-polio enteroviruses for wild polioviruses where possible.	Year 2000
II. Pre-global certification of polio free status	Increased containment in PC3/polio laboratory or above. Destroy wild poliovirus (or potentially infectious) materials or transfer to regional repository.	One year after the last case of polio reported globally.
III. Pre-cessation of OPV immunisation	Maximum containment in PC4/polio laboratory. Sabin polioviruses to be handled in PC3/polio laboratory.	At least three years after global certification of polio eradication.

Box 3. Members of the National Advisory Committee

- Dr David Anderson, Macfarlane Burnet Centre for Medical Research
- Dr John Andrew, Gribbles Pathology
- Professor Lyn Gilbert, Westmead Hospital
- Dr Gary Grohmann, Therapeutic Goods Administration
- Mr Greg Sam, Department of Health and Aged Care
- Dr Greg Smith, Queensland Health and Scientific Services

Box 4. State/Territory representatives

ACT	Dr Gary Grohmann, Therapeutic Goods Administration
NSW	Professor Lyn Gilbert, Westmead Hospital Dr Dominic Dwyer, Westmead Hospital
NT	Dr Gary Lum, Territory Health Services
QLD	Dr Greg Smith, Queensland Health and Scientific Services Dr Ted Gardner, Department of Natural Resources
SA	Dr Geoff Higgins, Institute of Medical and Veterinary Science
TAS	Dr Jan Williamson, Royal Hobart Hospital
VIC	Dr David Anderson, Macfarlane Burnet Centre for Medical Research Dr John Andrew, Gribbles Pathology
WA	Dr Gerry Harnett, PathCentre

and the Commonwealth Government (Box 3). The national advisory committee continues to work closely with the national coordinator to develop and refine the national survey. In addition to the national advisory committee, representatives from each State and Territory have been appointed to act as local spokespersons and facilitators for the containment process (Box 4).

A national plan for containment

Following the workshop, a draft national containment plan was developed and circulated to members of the national advisory committee. The plan outlined the progress of Australia's containment efforts and plans for implementing the national survey and development of a national inventory. The national plan is summarised in Box 5.

Developing a database of laboratories and institutions that manage laboratories

Before the national survey can be conducted it is necessary to identify every laboratory that could store poliovirus or material that may be infected with wild poliovirus (Box 1). Therefore, a database of laboratories and institutions that manage laboratories has been created. As of July 2000, the database included more than 700 diagnostic, reference,

Box 5. National Plan for Poliovirus Containment - Key Components

- **Coordination** - all States and the Commonwealth represented.
- **National workshop** - to develop a national plan.
- **National Advisory Committee** - to assist with refinement of the national plan.
- **State/Territory representatives** - to support the process at the State level.
- **National database of laboratories /organisations/institutions** - to compile a database of all institutions that may store wild poliovirus or biological materials potentially infectious for wild poliovirus.
- **Pilot surveys** - to evaluate the effectiveness of the planned approach to contacting and surveying institutions on the national database.
- **National laboratory screening survey** - to determine specific institutions that may store wild poliovirus or biological materials potentially infectious for wild poliovirus. These institutions will be surveyed a second time in order to develop a database of all wild poliovirus or biological materials potentially infectious for wild poliovirus.
- **National laboratory freezer clean up** - to encourage all laboratories to identify all biological material in storage and consider option to destroy wild poliovirus (or potentially infectious) materials or transfer to the regional repository.
- **New "enteroviruses for old" scheme** - exchange uncharacterised enteroviruses or non-Sabin polioviruses used for teaching, challenge or control work for prototype enteroviruses supplied by VIDRL.
- **National inventory** - a database of all wild polioviruses or biological materials potentially infectious for wild poliovirus in Australia.

research, regulatory, environmental and manufacturing laboratories, as well as all Australian hospitals, universities and independent research institutes.

Refining the survey form

As discussed above, the participants at the national workshop identified problems with the information pack and national survey form; therefore, it was decided to undertake two pilot surveys. The first survey was conducted during April 2000 and involved surveying a reference laboratory, two teaching hospitals and two universities. The second pilot survey was undertaken in May 2000 targeting the institutions of the national advisory committee members.

The first pilot study used the documentation provided by the Western Pacific Regional Office of WHO.[3] Interviews with managers contacted in the first pilot survey indicated that the documentation was too involved; documentation for the second pilot survey was therefore simplified.

The results of the pilot surveys confirmed some of the problems anticipated by the participants of the national containment workshop. In particular, both the lack of enthusiasm by laboratory staff to participate, and the difficulty for staff in larger institutions to survey all their laboratories within the nominated four-week period, were highlighted. The feedback collected from both surveys was used to modify further the background material, survey form and reporting mechanism that will be used for the national survey.

Implementing the national survey

The lack of compliance with the pilot surveys indicated that a direct and active survey method would be required to ensure an adequate survey response. With the assistance of the Department of Health and Aged Care's Social Marketing Unit, a marketing strategy for the national laboratory survey was developed.

It is planned that in August 2000, a letter and short questionnaire will be posted to all laboratories and institutions on the national database. The aim of the first mail out is to inform laboratories of the national survey and enable VIDRL to identify all relevant laboratories in Australia that could store poliovirus or potentially infectious material. Laboratories identified from the questionnaire will be contacted by telephone and sent an information pack and national laboratory survey form. During this time a telephone information line will operate to answer any queries related to the national survey. The information line will also follow up laboratories that do not return the national laboratory survey form in the nominated time frame.

The information pack will outline what laboratories need to do to complete the national survey. In short, laboratories will be asked to conduct a search for poliovirus infectious or potentially infectious materials and complete the national laboratory survey form, providing details of any poliovirus stocks or potentially infectious material.

The information collected from the laboratory survey form will be used to develop the final national inventory. Once complete the national inventory will finalise Australia's contribution to Phase I of the certification of global polio eradication.

At the completion of phase I, laboratories that store wild poliovirus must comply with PC2/polio containment conditions. These enhanced physical containment level 2 conditions are equivalent to Australian/New Zealand Standard AS/NZS 2243.3:1995[4] with the following added precautions for handling wild polioviruses:

- all staff are to be vaccinated against poliomyelitis; and
- an inventory and dedicated storage area for wild poliovirus stocks is to be established; and
- the substitution of wild poliovirus with attenuated vaccine polioviruses, inactivated antigens or non-polio enteroviruses is to be undertaken.

It is therefore recommended that potentially infectious materials be destroyed if no longer required and only viruses that are readily identifiable by molecular methods be used.

Prior to the activation of Phase II, laboratories whose collections are included in the national inventory will be

contacted to ascertain their proposed containment policy. These laboratories will be required to:

- destroy their polioviruses and poliovirus potentially infectious materials; or
- work with their poliovirus material in a PC3/polio containment laboratory; or
- transfer their poliovirus material to the WHO Western Pacific regional repository at the National Institute of Infectious Diseases, Tokyo, Japan.

Phase III will commence when immunisation using oral polio vaccine ceases. At this stage wild poliovirus must be handled in PC4 containment laboratories and Sabin vaccine virus in PC3/polio containment.

The final results of the national laboratory survey will be published later in 2000. For further information regarding the National Survey please contact the National Coordinator of Laboratory Poliovirus Containment, Nittita Prasopa-Plaizier, on (03) 9342 2603.

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New publication

The first comprehensive report on the recent epidemiology of vaccine preventable diseases and vaccination coverage in Australia is now available.

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) has prepared a report entitled *Vaccine Preventable Diseases and Vaccination Coverage in Australia, 1993–1998* which provides a comprehensive national picture of the recent epidemiology of vaccine preventable diseases (VPDs). The report reviews notifications (1993-8), hospitalisations (1993/4-1997/8), and deaths (1993-7) for eight diseases on the routine childhood vaccination schedule (diphtheria, *Haemophilus influenzae* type b, measles, mumps, pertussis, poliomyelitis, rubella, tetanus), and four other diseases potentially preventable by childhood vaccination (hepatitis A, acute hepatitis B, invasive pneumococcal disease, varicella). It also examines vaccination coverage data from the Australian Childhood Immunisation Register (ACIR) and Australian Bureau of Statistics (ABS) for the same period.

The report brings together for the first time all three national sources of routinely collected data about vaccine preventable diseases, for all age groups in Australia, together with information about vaccination coverage. It is a valuable resource for health professionals providing evidence of the impact of changes in vaccination practice over the six years, particularly notable for measles and *Haemophilus influenzae* type b, and a baseline against which further changes can be measured.

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Measles in health care facilities: some salutary lessons

Jeffrey Hanna,[1] Ann Richards,[1] Dallas Young,[1,2] Susan Hills,[3] Jan Humphreys[3]

Keywords: measles, overseas travel, nosocomial transmission, vaccine, immunoglobulin

Measles transmission occurs readily in health care facilities such as general practices and hospitals. It can not only lead to measles in other patients at the facilities but also to measles in medical and nursing staff. In the recent (1999) measles outbreak in Victoria, four of the 62 cases were health care workers.[1] A further obvious concern in health care facilities is that those exposed may include immuno-compromised patients or very young children, two groups in whom measles can be very severe.

The reasons for the ready transmission are:

- many patients with measles present to GPs and hospitals for diagnosis and management,
- patients with measles are highly infectious; the infectious period extends from 5 days before the appearance of the rash until 4 days after the onset of rash;
- many younger doctors have never seen measles;
- many doctors are not aware of the need for a prompt public health response and do not notify a case of measles until it has been confirmed by a laboratory; and
- the lack of clear vaccination policies for health care providers.

A recent outbreak of measles in north Queensland highlighted some of the above issues, and provides useful lessons for the future.

The outbreak

Five children, all from the same north Queensland family, were confirmed as having measles on Monday 15 May. Not one of the children, whose ages ranged from 2.8 to 11.5 years, had been vaccinated.

Two of the children, a boy and a girl, had travelled to the United Kingdom in early April on an evangelical tour. En route they stayed overnight in a hotel in Colombo, Sri Lanka. The tour included 10 nights in Scotland, 7 in England and 2 in Wales. The children on the tour stayed, as billets, in private homes.

The boy (9.3 years) first became unwell in England in mid-April, 12 days after leaving Sri Lanka. The rash appeared three days later on Good Friday (21 April) while in England. He was seen by two GPs that day and although both made a clinical diagnosis of measles neither requested any tests and neither gave any specific advice (about isolation and other precautions) to the adults caring for the boy on the tour.

There were 8 children (aged 9-16 years) and 5 adults in the Australian party on the tour. Apart from the north Queensland children, all the remainder of the party remained well. (Incidentally, the church involved is not anti-vaccination; it was a parental decision, not based on a religious belief, not to have the children vaccinated).

An English teenager, who was in contact with the north Queensland boy in mid-April, became unwell at about the end of the month. He had fever, vomiting, sore throat, cough and a 'very red rash'. He was diagnosed by a GP as having scarlet fever and was prescribed antibiotics.

The tour party returned to Australia in late April. The boy was by then well but on the day of arrival back in their home city in north Queensland the girl (11.5 years) was noted by her parents to be unwell. She was taken to a local medical centre where the contact with measles was elicited at the consultation, 'fever and malaise' noted, and a diagnosis of possible measles made. The child was seen again at the medical centre two days later. A florid rash appeared the next day (3 May) and a clinical diagnosis of measles was made on 5 May at her third visit to the centre. No diagnostic tests were ordered, no specific instructions were given to the family and the clinical diagnosis was not notified.

All three younger siblings (2.8, 5.75 and 7.6 years) became unwell about 8-9 May. On the weekend on 6-7 May the family travelled from their home city to another north Queensland city, where they stayed at a caravan park, and back.

On 10 May the father and two boys (the boy infected while overseas and the 5.75 year old, the latter of whom was unwell) again travelled to the other city. En route the three stayed in a small rural town. The ill boy was seen on the evening of 12 May by a GP in the town. The boy developed a rash the next day (13 May). He presented that evening to the emergency department of the city's public hospital and was admitted to an isolation bay 5 hours later. He was notified to the Tropical Public Health Unit (TPHU) 36 hours after being admitted; his measles IgM was positive. Subsequent studies done on PCR-positive throat swabs that had been collected from all four children who became unwell in Australia showed that the infecting measles virus was the 'Sri Lankan genotype' (personal communication, Dr Heath Kelly, Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria).

Meanwhile the youngest sibling had been seen at the medical centre in her home city on 14 May. A clinical diagnosis of measles was made, but again the consulting doctor failed to request diagnostic tests, to give her mother specific instructions (eg. about keeping the child in isolation) and to notify the clinical diagnosis.

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Fortunately not one of the five children attended either day-care or school during their infectious periods, and their mother has tended to keep them at home for much of this time. Nevertheless, they had had a few visitors and had on occasion been locally out of the house.

Within 24 hours of receiving the notification, the TPHU requested the two medical practices and the public hospital to identify, as a matter of urgency, all those who were in the practices and emergency department from one hour before until two hours after the measles cases were seen. (The measles virus remains viable in the environment for up to 2 hours). Each individual had to be assessed to determine whether they were susceptible to measles; children 1-4 years with one documented dose of MMR, those over 4 or born during or after 1970 with two documented doses of MMR, and those born before 1970 are considered to be immune ie. not susceptible.[2]

Susceptible persons over 9 months of age were to be offered an immediate dose of MMR if it was less than 3 days after the exposure to measles at the health care facility, otherwise if 3-7 days post-exposure they were to be offered immunoglobulin. Clearly not only was this exercise extremely demanding in the city general practice and in the hospital, but also it could not 'capture' every person who had been exposed. At the public hospital for example, over 80 people had to be contacted (including staff, patients, those accompanying the patients etc) and 29 doses of MMR and one dose of immunoglobulin were given. With one exception, all those requiring MMR or immunoglobulin were susceptible 20-29 year olds.

Other GPs in the two cities were informed, via the Divisions, about the outbreak as were other local emergency departments. They were asked to ensure that any possible measles case be promptly triaged and not left in waiting rooms, to request measles IgM serology (even if the patient was a young child) and to immediately notify TPHU (without waiting for results to become available).

Meanwhile, TPHU had to undertake contact tracing elsewhere in the two cities (eg. at the caravan park) and in the rural town. This contact tracing, and arranging for susceptible contacts to be given either MMR or immunoglobulin required the commitment of five TPHU personnel (physicians, nurses, indigenous public health worker) full-time for about seven days.

Over the next three weeks 7 possible cases of measles were notified to TPHU; one was indeed a case (imported from Malaysia, and not related to the local outbreak) but the remaining six were eventually proven not to be measles. The outbreak was declared over three weeks after the onset of the last child in the family.

Lessons learned

A history of close contact with a measles case, especially in an unvaccinated person, must make health care providers consider measles as a likely diagnosis in an unwell patient with a fever.

A history of overseas travel must make health care providers consider measles as a possible diagnosis in a patient with a febrile illness and a morbilliform rash. Measles still in widespread circulation in many countries; Sri Lanka, the Netherlands and East Timor have had recent large outbreaks.

Health care providers should always notify a case of measles upon clinical suspicion. This enables TPHU to recommend to the provider the most appropriate tests, and to make recommendations about necessary interventions, exclusions etc.

Health care providers must be aware that their facilities can serve as effective sites for measles transmission, and can precipitate or aggravate measles transmission in the community. A prompt notification from the general practice on 5 May (10 days before the date of the actual notification) would have saved the practice (and probably the other practice and the hospital) a lot of unnecessary work.

Health care providers have a duty-of-care to ensure that they do not put patients at unnecessary risk. Therefore they are obliged to recall patients, to assess their susceptibility and to provide either MMR or immunoglobulin in an attempt to abort any incubating measles infection inadvertently acquired at their facilities.

A verbal history of prior doses of MMR, (or of prior measles), is very unreliable, particularly in adolescents and young adults. We know of two circumstances in this outbreak where a verbal history was shown to be incorrect. Attempts must be made to obtain documentation of prior vaccination; if proof is not available, a person should be considered to be susceptible and managed accordingly.

TPHU learned just how valuable PCR tests can be, particularly early in the illness before measles IgM antibodies have developed. Throat swabs were PCR-positive in four, and urine PCR-positive in two, of the children. This enabled genotyping to be performed.

Finally, TPHU once again learned just how important Divisions are for the rapid dissemination of information to local GPs.

References

1. Lambert S, Lynch P, Morgan M, et al. Measles outbreak – young adults at high risk. *Vic Infect Dis Bull* 1999;2: 21-22.
2. Measles Elimination Advisory Committee. Guidelines for the control of measles outbreaks in Australia. 2nd ed Canberra: Department of Health and Aged Care (in press).

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.

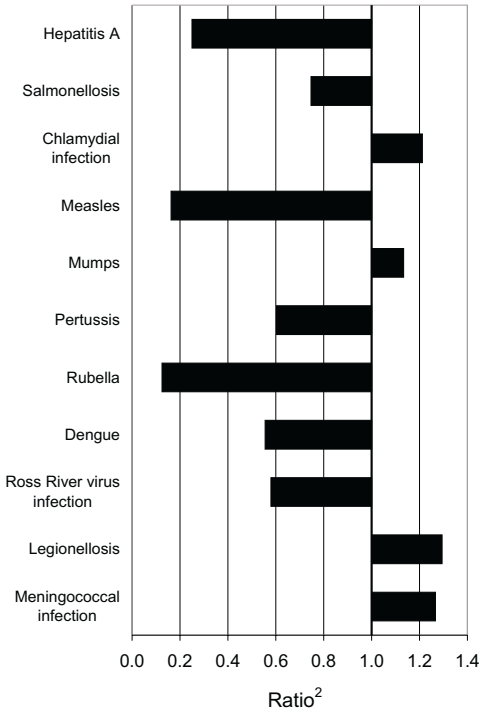
Highlights for June 2000

Communicable Diseases Surveillance consists of data from various sources. The National Notifiable Diseases Surveillance System (NNDSS) is conducted under the auspices of the Communicable Diseases Network Australia New Zealand. 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 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 (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.*

Compared with the 5-year mean, in June 2000 there was an increase in the number of cases of chlamydial infection (ratio 1.2), legionellosis (ratio 1.3) and meningococcal infection (ratio 1.3) (Figure 1).

Figure 1. Selected[1] diseases from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 to 30 June 2000 with historical data[2]



Chlamydia

There were 996 notifications of chlamydial infection in June 2000 - a notification rate of 63.02/100,000 population (Figure 2); the Northern Territory and Queensland contributed most to the increase in notifications for this month (447.94/100,000 and 125.39/100,000 respectively). Part of this increase can be explained through the use of PCR urine-testing technology and screening programs in several States.

Encephalitis

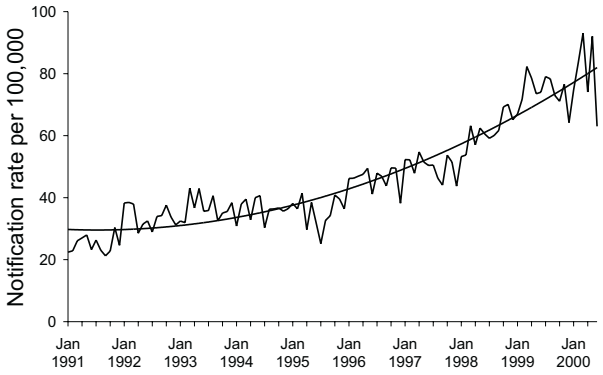
A renewed warning was issued in June 2000 by the Northern Territory government regarding Australian Encephalitis in the top end. There have been no further cases since May 2000 in Western Australia or the Northern Territory.

Foodborne illness outbreaks

There is currently an increase in *Salmonella* Typhimurium phage-type 9 in Victoria. A common source of infections has not been identified. Two family clusters of 4 and 5 cases each have been identified one of which was thought to be related to a home cooked meal of chicken.

1. Selected diseases are chosen each calendar month according to current activity

Figure 2. Notification rate of chlamydial infection, Australia, 1 January 1991 to 30 June 2000, by month of notification



Legionellosis

There were 22 notifications of legionellosis in June 2000 - a notification rate of 1.39/100,000 population. In Victoria there were three confirmed cases and one unconfirmed case of *L. pneumophila* serogroup1 associated with a spa pool at a football club. The other cases were sporadic with no obvious sources.

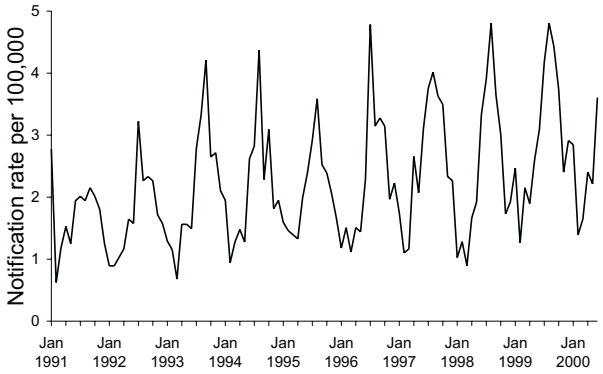
Listeria

There were three cases of listeria in Western Australia. One case was a pregnant woman resulting in the still birth of her baby, who also tested positive, and another was a woman with end stage renal failure who was on haemodialysis. The Health Department of Western Australia issued a reminder to pregnant women, the elderly and people with lowered immunity to take special care to avoid food that may contain the bacterium *Listeria*. The reminder followed a recent state-wide survey of sandwiches served at cafes, bakeries and lunchbars, which found *Listeria monocytogenes* present in 11 of 228 samples.

Meningococcal infections

There were 57 notifications of meningococcal infection in June 2000 - a notification rate of 3.61/100,000 population (Figure 3). Of these cases 33% were under 5 years of age and 39% were in the 15-24 age range. The serogroups were available for 41 cases; of these 56%, 42% and 2% were serogroup B, C and Y respectively.

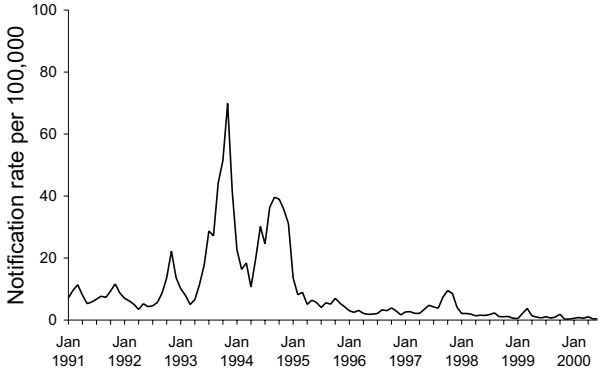
Figure 3. Notification rates of meningococcal infections, Australia, 1 January 1991 to 30 June 2000, by month of notification



Vaccine preventable diseases

All vaccine preventable diseases (except mumps) had fewer cases this month compared with the 5-year mean for June. There were two male and two female cases of *Haemophilus influenzae* type b reported in June, an increase in the number of notifications from May (one case). Of the June cases, one was a child aged less than 1 year, and the others were aged 33, 40 and 84 years; the immunisation status of all was unknown. For measles, the last 3 months have had the lowest number of notifications since the national notification system began (Figure 4). Of the seven cases in June, two were in children under 5 years and one was confirmed as having been imported from the United Kingdom.

Figure 4. Notification rate of measles, Australia, 1 January 1991 to 30 June 2000, by month of notification



Influenza

The New South Wales sentinel surveillance network reported the highest rate of consultations for influenza (10/1,000 patients). There were 113 laboratory reports for June 2000 - a decrease from 352 in June 1999 (Figure 5). Of the laboratory reports received in June 2000 (weeks 23-26), 70 were Influenza A and 40 were Influenza B with the percentage of influenza B increasing from 13% in week 23 to 44% in week 26 (Figure 6). Compared with June 1999, the percentage of Australia Post employees absent in June 2000 (weeks 23-26) for three or more consecutive days in 1 week increased (Figure 7). However, to date in 2000, there were fewer reports from laboratories of influenza - and from ASPREN of influenza-like illness - compared with 1999 (Figures 5 and 8).

Figure 5. Laboratory reports of influenza, 1999 to 2000, by month of specimen collection

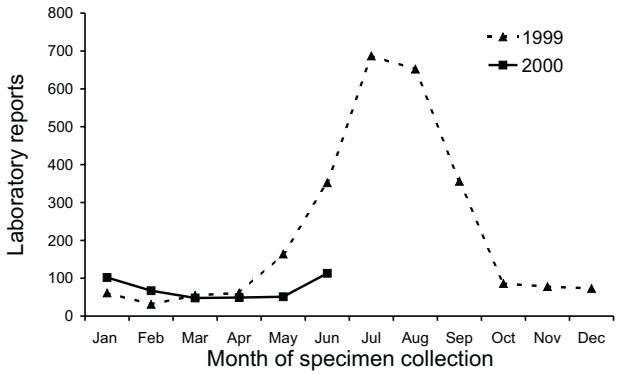


Figure 6. Laboratory reports of influenza, Australia, week 1 1999 to week 26 2000, by week of specimen collection

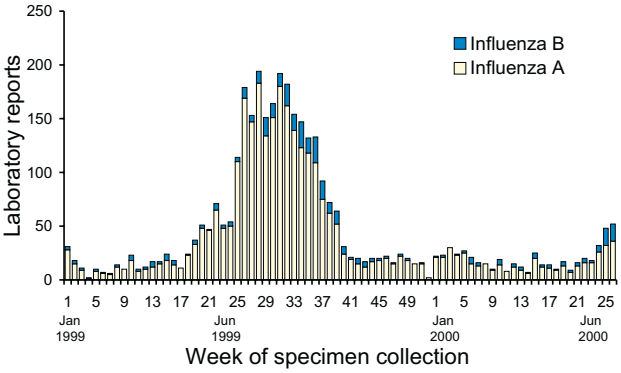


Figure 7. Absenteeism rates in Australia Post, 1999 and 2000 to June 30

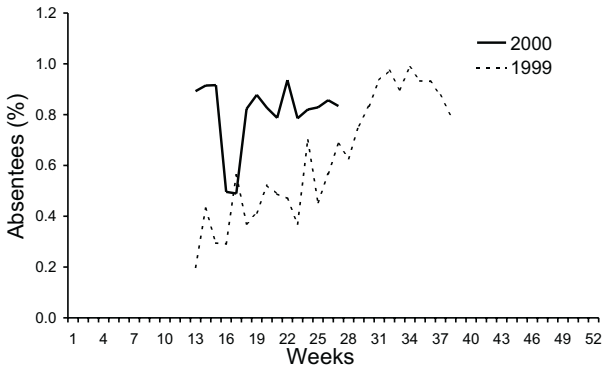
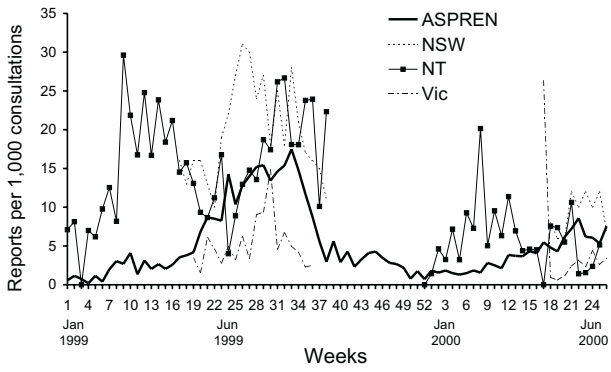


Figure 8. Sentinel general practitioner influenza consultation rates, week 1 1999 to week 26 2000, by scheme[1]



1. Week 26, 2000, ASPREN data are for Australia other than the Northern Territory

Tables

There were 5,419 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification date in June 2000 (Table 1). Data by date of report for June 2000, are included in this issue of *Communicable Diseases Intelligence* (Table 2). The number of reports for selected diseases[1] have been compared with a 5 year mean, calculated using May to July data for the previous 5 years (Figure 1).

There were 2,132 reports received by the *CDI* Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 to 30 June 2000 (Tables 3 and 4).

The Australian Sentinel Practice Research Network (ASPREN) data for weeks 22 to 25, ending 25 June 2000, are included in this issue of *Communicable Diseases Intelligence* (Table 5).

Table 1. Notifications of diseases received by State and Territory health authorities in the period 1 to 30 June 2000, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total June 2000[1]	Total May 2000[1]	Total June 1999[1]	Last 5 years mean	Year to date 2000	Last 5 years YTD mean	Ratio*
Bloodborne															
Hepatitis B (incident)	0	5	0	4	0	4	5	4	22	31	17	22	155	144	1.0
Hepatitis B (unspecified)[2]	0	246	0	61	0	5	159	59	530	713	779	586	3,995	3,474	0.9
Hepatitis C (incident)	0	4	0	-	3	1	4	3	15	21	16	15	161	95	1.0
Hepatitis C (unspecified)[2]	7	466	8	260	40	23	458	111	1,373	1,815	1,850	1,363	10,721	7,867	1.0
Hepatitis D	0	0	0	1	0	0	0	0	1	0	1	2	8	8	0.5
Gastrointestinal															
Botulism	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0.0
Campylobacterosis[3]	21	-	18	325	107	31	307	136	945	1,189	942	931	6,286	5,586	1.0
Haemolytic uraemic syndrome	NN	0	0	0	0	0	0	0	0	0	0	2	6	4	0.0
Hepatitis A	0	11	1	7	1	1	10	11	42	81	104	168	519	1,256	0.3
Hepatitis E	0	0	0	0	0	0	0	0	0	0	2	2	0	2	0.0
Listeriosis	0	1	0	0	0	0	0	3	4	5	4	4	42	33	1.0
Salmonellosis	6	43	12	87	29	6	86	44	313	639	375	419	3,577	4,057	0.7
Shigellosis[3]	0	-	6	3	3	0	11	6	29	57	37	54	264	377	0.5
SLTEC,VTEC[4]	NN	0	0	NN	1	0	0	NN	1	1	3	2	18	7	0.5
Typhoid	0	2	0	0	0	0	0	0	2	8	5	5	39	44	0.4
Yersiniosis[3]	1	-	0	2	0	0	0	0	3	8	9	17	44	134	0.2
Quarantinable															
Cholera	0	0	0	0	0	0	0	0	0	0	0	1	1	2	0.0
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	0	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	174	72	367	39	33	156	141	996	1,455	1,170	822	7,595	4,958	1.2
Donovanosis	0	0	0	1	NN	0	0	0	1	1	0	4	9	24	0.3
Gonococcal infection[6]	0	50	83	79	13	2	75	81	383	613	439	385	3,078	2,411	1.0
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Syphilis[7]	0	25	4	54	1	1	0	7	92	177	176	146	835	865	0.6

Table 1 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 to 30 June 2000, by date of notification[#]

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total June 2000[1]	Total May 2000[1]	Total June 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	1	1	2	0	0	0	0	4	1	5	5	10	28	0.8
Measles	0	0	0	2	1	0	4	0	7	6	11	43	61	306	0.2
Mumps	4	8	0	0	1	1	2	1	17	27	15	15	104	84	1.1
Pertussis	5	112	0	29	19	9	46	0	220	300	296	365	1,677	2,371	0.6
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0	0	0	na
Rubella[8]	1	2	0	1	0	0	8	0	12	18	28	96	93	686	0.1
Tetanus	0	0	0	0	0	0	1	0	1	0	1	1	4	3	1.0
Vectorborne															
Arbovirus infection NEC	0	0	0	1	0	0	0	0	1	10	4	3	58	40	0.3
Barmah Forest virus infection	0	9	0	22	0	0	1	0	32	75	41	52	340	488	0.6
Dengue	0	0	5	0	0	0	0	0	5	9	6	9	189	105	0.6
Malaria	2	21	3	27	1	0	6	1	61	134	41	61	540	420	1.0
Ross River virus infection	3	38	5	48	5	0	13	44	156	572	170	269	3,375	4,229	0.6
Zoonoses															
Brucellosis	0	0	0	1	0	0	0	0	1	1	1	3	7	15	0.3
Hydatid infection	0	NN	0	0	1	0	0	0	1	5	3	4	20	17	0.3
Leptospirosis	0	3	0	8	0	0	0	0	11	43	15	17	140	111	0.6
Ornithosis	0	NN	0	NN	0	0	0	1	1	6	3	6	37	39	0.2
Q fever	0	5	0	17	0	0	1	0	23	39	53	49	246	268	0.5
Other															
Legionellosis	0	3	0	8	1	0	8	2	22	41	18	17	310	113	1.3
Leprosy	0	0	0	1	0	0	0	0	1	0	0	1	1	4	1.0
Meningococcal infection	0	28	0	7	4	3	11	4	57	35	49	45	223	169	1.3
Tuberculosis	0	7	0	2	0	0	24	1	34	40	79	83	378	496	0.4
Total	64	1,264	218	1,427	270	120	1,396	660	5,419	8,176	6,768	6,082	45,167	41,341	

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. Notifications of diseases received by State and Territory health authorities in the period 1 to 30 June 2000, by date of report*

Disease[1]	State or Territory								Total this period	Year to date total
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Bloodborne										
Hepatitis B (incident)	0	10	0	4	2	3	9	6	34	169
Hepatitis B (unspecified)[2]	0	366	0	66	23	6	177	74	712	4,259
Hepatitis C (incident)	0	8	0	-	7	1	5	4	25	190
Hepatitis C (unspecified)[2]	10	696	11	277	82	23	459	154	1,712	11,113
Hepatitis D	0	1	0	1	0	0	0	0	2	9
Gastrointestinal										
Botulism	0	0	0	0	0	0	0	0	0	0
Campylobacterosis[3]	22	-	17	356	134	29	419	156	1,133	6,504
Haemolytic uraemic syndrome	NN	0	0	0	0	0	0	0	0	6
Hepatitis A	0	14	2	13	1	1	18	13	62	554
Hepatitis E	0	0	0	0	0	0	0	0	0	0
Listeriosis	0	1	0	0	0	0	0	4	5	43
Salmonellosis	10	81	19	119	34	6	98	49	416	3,850
Shigellosis[3]	1	-	10	6	3	0	12	10	42	273
SLTEC,VTEC[4]	NN	0	0	NN	1	0	0	NN	1	21
Typhoid	0	3	0	0	0	0	0	1	4	44
Yersiniosis[3]	1	-	0	3	0	0	0	0	4	45
Quarantinable										
Cholera	0	0	0	0	0	0	0	0	0	1
Plague	0	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0
Yellow fever	0	0	0	0	0	0	0	0	0	0
Sexually transmissible										
Chancroid	0	0	0	0	0	0	0	0	0	0
Chlamydial infection[5]	16	253	89	390	75	33	238	206	1,300	7,899
Donovanosis	0	0	0	1	NN	0	0	0	1	10
Gonococcal infection[6]	1	78	96	90	37	3	81	108	494	3,134
Lymphogranuloma venereum	0	0	0	0	0	0	0	0	0	0
Syphilis[7]	0	48	12	61	0	1	1	9	132	910
Vaccine preventable										
Diphtheria	0	0	0	0	0	0	0	0	0	0
Haemophilus influenzae type b	0	1	0	4	0	0	0	0	5	11
Measles	0	0	0	3	1	0	4	0	8	63
Mumps	3	17	0	0	0	1	3	2	26	107
Pertussis	12	257	0	33	36	9	54	3	404	1,950
Poliomyelitis	0	0	0	0	0	0	0	0	0	0
Rubella[8]	1	3	0	3	0	0	9	0	16	100
Tetanus	0	0	0	0	0	0	1	0	1	5
Vectorborne										
Arbovirus infection NEC	0	0	1	2	0	1	0	1	5	55
Barmah Forest virus infection	0	14	1	27	0	0	1	2	45	358
Dengue	0	1	4	4	0	0	0	0	9	206
Malaria	2	22	6	41	3	1	12	1	88	558
Ross River virus infection	3	75	7	60	13	2	18	111	289	3,587

Table 2 (continued). Notifications of diseases received by State and Territory health authorities in the period 1 to 30 June 2000, by date of report*

Disease[1]	State or Territory								Total this period	Year to date total
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA		
Zoonoses										
Brucellosis	0	0	0	1	0	0	0	0	1	8
Hydatid infection	0	NN	0	0	1	0	0	1	2	20
Leptospirosis	0	5	0	16	0	0	0	0	21	147
Ornithosis	0	NN	0	NN	0	0	4	2	6	49
Q fever	0	5	0	17	0	1	1	0	24	260
Other										
Legionellosis	0	5	0	11	3	0	24	5	48	322
Leprosy	0	0	0	1	0	0	0	0	1	1
Meningococcal infection	0	26	1	8	4	3	10	4	56	226
Tuberculosis	0	21	5	10	0	1	24	1	62	491
Total	82	2,011	281	1,628	460	125	1,682	927	7,196	47,558

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.
2. Unspecified numbers should be interpreted with some caution as the magnitude may be a reflection of the numbers of tests being carried out.
3. Not reported for NSW because it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.
4. Infections with Shiga-like toxin (verotoxin) producing *E.* coli (SLTEC/VTEC).
5. WA: genital only.
6. NT, Qld, SA , Vic and WA: includes gonococcal neonatal ophthalmia.
7. Includes congenital syphilis.
8. Includes congenital rubella.

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

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 3. Virology and serology laboratory reports by contributing laboratories for the reporting period 1 to 30 June 2000[1]

State or Territory	Laboratory	Reports this period	Total this period[2]
Australian Capital Territory	The Canberra Hospital	9	0
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	162	175
	New Children's Hospital, Westmead	138	167
New South Wales	Repatriation General Hospital, Concord	0	0
	Royal Prince Alfred Hospital, Camperdown	55	58
	South West Area Pathology Service, Liverpool	0	0
Queensland	Queensland Medical Laboratory, West End	429	365
	Townsville General Hospital	0	0
South Australia	Institute of Medical and Veterinary Science, Adelaide	412	458
Tasmania	Northern Tasmanian Pathology Service, Launceston	7	19
	Royal Hobart Hospital, Hobart	0	0
Victoria	Monash Medical Centre, Melbourne	0	3
	Royal Children's Hospital, Melbourne	154	208
	Victorian Infectious Diseases Reference Laboratory, Fairfield	171	213
Western Australia	PathCentre Virology, Perth	484	465
	Princess Margaret Hospital, Perth	100	374
	Western Diagnostic Pathology	11	6
Total		2,132	2,511

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.

Table 4. Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 30 June 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	1	0	3	1	5	3	26	130
Mumps virus	0	0	0	0	0	0	0	2	2	3	31	30
Rubella virus	0	0	0	0	1	0	1	1	3	10	22	49
Hepatitis viruses												
Hepatitis A virus	0	1	0	0	2	0	0	6	9	26	94	199
Hepatitis D virus	0	0	0	1	0	0	0	0	1	0	3	4
Arboviruses												
Ross River virus	0	1	2	17	8	0	2	45	75	72	1,064	1,045
Barmah Forest virus	0	0	0	9	0	0	0	0	9	8	104	116
Dengue not typed	0	0	0	0	0	0	0	3	3	2	164	33
Kunjin virus	0	0	0	0	0	0	0	1	1	1	3	5
Flavivirus (unspecified)	0	0	0	2	0	0	0	0	2	1	37	17
Adenoviruses												
Adenovirus type 1	0	0	0	0	1	0	0	0	1	0	3	6
Adenovirus type 2	0	0	0	0	0	0	1	0	1	1	5	7
Adenovirus type 19	0	0	0	0	0	0	1	0	1	0	6	0
Adenovirus type 37	0	0	0	0	0	0	1	0	1	0	9	9
Adenovirus type 40	0	0	0	0	0	0	0	9	9	11	68	33
Adenovirus not typed/pending	0	12	1	2	44	0	13	20	92	97	541	538
Herpes viruses												
Cytomegalovirus	3	10	0	8	33	0	17	11	82	95	615	603
Varicella-zoster virus	1	3	0	20	7	0	24	44	99	100	745	806
Epstein-Barr virus	3	13	4	145	65	0	5	34	269	115	1,206	1,186
Other DNA viruses												
Molluscum contagiosum	0	0	0	0	0	0	0	1	1	2	9	8
Parvovirus	0	0	0	0	4	0	8	12	24	39	166	208
Picornavirus family												
Echovirus type 30	0	0	0	0	0	0	3	0	3	0	102	6
Rhinovirus (all types)	0	9	0	0	0	0	1	26	36	42	208	186
Enterovirus not typed/pending	0	0	2	2	0	0	18	38	60	88	576	385
Ortho/paramyxoviruses												
Influenza A virus	3	13	0	1	43	0	5	8	73	322	339	629
Influenza A virus H3N2	0	0	0	0	0	0	1	0	1	14	1	23
Influenza B virus	0	10	0	0	15	0	8	6	39	16	90	71
Parainfluenza virus type 1	0	3	0	2	14	0	6	6	31	5	179	23
Parainfluenza virus type 2	0	1	0	0	0	0	1	3	5	28	21	83
Parainfluenza virus type 3	0	0	0	0	5	0	0	4	9	38	108	208
Respiratory syncytial virus	4	187	0	29	36	4	139	154	553	495	1,259	1,045
Other RNA viruses												
HTLV-1	0	0	0	0	1	0	0	0	1	2	3	6
Rotavirus	0	53	0	0	29	0	6	52	140	200	349	538
Other												
Chlamydia trachomatis not typed	3	38	9	60	31	3	4	84	232	290	1,735	1,549
Chlamydia pneumoniae	1	0	0	0	0	0	0	0	1	0	1	0
Chlamydia psittaci	0	0	0	0	0	0	6	2	8	14	49	49
Chlamydia species	0	1	0	0	0	0	0	0	1	2	6	11
Mycoplasma pneumoniae	1	3	3	18	11	0	18	3	57	80	295	533
Mycoplasma hominis	0	5	0	0	0	0	0	0	5	0	6	4

Table 4 (continued). Virology and serology laboratory reports by State or Territory[1] for the reporting period 1 to 30 June 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				
Rickettsia spp - other	0	0	0	0	0	0	0	3	3	0	6	5
Streptococcus group A	0	4	2	11	0	0	4	0	21	46	181	74
Yersinia enterocolitica	0	3	0	0	0	0	0	0	3	1	8	8
Bordetella pertussis	3	2	0	8	8	1	21	6	49	46	274	322
Legionella pneumophila	0	0	0	1	0	0	0	0	1	1	14	15
Legionella longbeachae	0	0	0	0	3	0	0	3	6	2	35	19
Cryptococcus species	0	2	0	0	1	0	0	0	3	0	8	6
Leptospira species	0	0	0	8	0	0	0	0	8	7	31	16
Treponema pallidum	0	1	24	14	49	0	0	2	90	113	366	166
Entamoeba histolytica	0	0	0	0	0	0	0	1	1	1	9	1
Toxoplasma gondii	0	0	0	0	1	0	0	0	1	0	7	5
Echinococcus granulosus	0	0	0	0	1	0	0	0	1	0	14	0
Total	22	375	47	358	414	8	317	591	2,132	2,439	11,201	11,018

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. Australian Sentinel Practice Research Network reports, weeks 22 to 25, 2000

Week number	22		23		24		25	
Week ending on	4 Jun 2000		11 Jun 2000		18 Jun 2000		25 Jun 2000	
Doctors reporting	63		65		69		69	
Total encounters	7,682		7,769		7,408		8,264	
Condition	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters	Reports	Rate per 1,000 encounters
Influenza	66	8.6	48	6.2	45	6.1	44	5.3
Chickenpox	12	1.6	11	1.4	10	1.3	13	1.6
Gastroenteritis	59	7.7	51	6.6	53	7.2	70	8.5
Gastroenteritis with stool culture	4	0.5	20	2.6	7	0.9	13	1.6
ADT immunisations	24	3.1	34	4.4	41	5.5	35	4.2

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 Health and Medical Research Council (NHMRC). 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.*

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 every four weeks. 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. Between 7,000 and 8,000 consultations are reported each week, with special attention to 14 conditions chosen for sentinel surveillance in 2000. Communicable Diseases Intelligence *reports the consultation rates for five of these. For further information, including case definitions, see* Commun Dis Intell *2000;24:7-8.*

Additional Reports

Sentinel Chicken Surveillance Programme

Sentinel chicken flocks are used to monitor flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVE) and Kunjin which cause the potentially fatal disease Australian encephalitis in humans. Currently 28 flocks are maintained in the north of Western Australia, seven in the Northern Territory, nine in New South Wales and ten in Victoria. The flocks in Western Australia and the Northern Territory are tested year round but those in New South Wales and Victoria are tested only from November to March, during the main risk season.

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

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5. Department of Health and Community Services, Northern Territory

Sentinel chicken serology was carried out for 23 of the 28 flocks in Western Australia in May and June 2000. A new flock was established at the Bidyadanga Aboriginal community, approximately 150km south of Broome and was bled for the first time in June 2000. Widespread MVE was still detected in the Kimberley, Pilbara, Gascoyne and Midwest regions in May. however, the number of seroconversions declined in June, except for some areas of the Pilbara, particularly those sites near permanent water (Harding and Ophthalmia dams). The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 6. A number of the later seroconversions have not yet been confirmed.

High levels of MVE virus activity occurred in 2000 as a result of high wet season rainfall in the Kimberley region and high cyclonic rains and extensive flooding in the Pilbara, Gascoyne, Murchison and Midwest regions. MVE virus antibodies have been detected in chickens in the Murchison and Midwest regions for the first time this year. This is the furthest south the virus has ever been detected. A survey to determine MVE antibody levels in domestic chickens located in this region and areas further south and east is being carried out to determine the limit of MVE virus activity in Western Australia this year. A number of news media

Table 6. Flavivirus seroconversions in Western Australian sentinel chicken flocks in May and June 2000

Location	May 2000			June 2000		
	MVE	MVE/KUN	KUN	MVE	MVE/KUN	KUN
Kimberley						
Wyndham	1	1	1			
Kununurra	1					
Halls Creek	2					
Fitzroy Crossing	1					1
Derby*	3		1			
Curtain Air Base	4					
Lombadina				2		
Broome*	8#					
Pilbara						
South Hedland	1	1				1
Karratha				4#		
Harding Dam*				4	1	
Nullagine	1		1			
Tom Price				1		
Paraburdoo	2		1	4		
Ophthalmia Dam	6	1		2		
Newman	1					
Exmouth	4	2		1		1
Gascoyne						
Carnarvon	3					
Mid-West						
Dongara	4					

* 2 flocks of 12 chickens at these sites

\# These results have not yet been confirmed.

MVE Antibodies to Murray Valley encephalitis virus detected by ELISA.

KUN Antibodies to Kunjin virus detected by ELISA.

Table 7. Flavivirus seroconversions in Northern Territory sentinel chicken flocks in May and June 2000

Location	May 2000			June 2000		
	MVE	MVE/KUN	KUN	MVE	MVE/KUN	KUN
Alice Springs	1				1[#]	
Howard Springs				1[#]		
Leanyer	2					
Gove	1			3[#]		
Tennant Creek	6[#]					
Katherine	1					

[#] These results have not yet been confirmed.

MVE Antibodies to Murray Valley encephalitis virus detected by ELISA.

KUN Antibodies to Kunjin virus detected by ELISA.

warnings have been issued by the Health Department of Western Australia to alert residents living in the northern areas of Western Australia to the increased risk of disease. Additional warnings were also sent out by the Regional Public Health Units to Aboriginal communities in the regions. To date eleven cases of Australian encephalitis caused by MVE virus have been confirmed from Western Australia. In addition there have been several cases of non-encephalitic disease caused by Kunjin virus reported from Western Australia.

Serum samples from all seven of the Northern Territory sentinel chicken flocks were tested in our laboratory in May 2000 and from six flocks in June 2000. There were a number of seroconversions to flaviviruses in the flocks located at Alice Springs, Leanyer, Katherine, Tennant Creek and Gove in May and at Alice Springs, Howard Springs, and Gove in June. The number of chickens positive for flavivirus antibodies by ELISA at each site and the identity of the infecting virus(es) are shown in Table 7. A number of news media warnings have been issued by the Northern Territory Health Department and to date there have been four cases of Australian encephalitis confirmed from central Australia.

The MVE surveillance programs using sentinel chickens in New South Wales and Victoria finished in April 2000.

HIV and AIDS Surveillance

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

Tabulations of diagnoses of HIV infection and AIDS are based on data available three months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in HIV/AIDS and related diseases in Australia Annual Surveillance Report. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Telephone: (02) 9332 4648; Facsimile: (02) 9332 1837; http://www.med.unsw.edu.au/nchecr.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 to 29 February 2000, as reported to 31 May 2000, are included in this issue of Communicable Diseases Intelligence *(Tables 8 and 9).*

Table 8. New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 to 29 February 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	1	0	2	0	0	0	1	4	7	13	10
	Male	0	20	0	12	1	0	5	4	42	39	111	85
	Sex not reported	0	0	0	0	0	0	0	0	0	0	1	0
	Total[1]	0	21	0	14	1	0	5	5	46	46	126	95
AIDS diagnoses	Female	0	2	0	1	0	0	0	0	3	1	7	1
	Male	0	3	0	2	1	0	4	1	11	12	30	22
	Total[1]	0	5	0	3	1	0	4	1	14	13	37	23
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	0	3	0
	Male	1	3	0	3	1	0	2	0	10	6	13	27
	Total[1]	1	3	0	3	1	0	2	0	10	6	16	28

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

Table 9. Cumulative diagnoses of HIV infection, AIDS and deaths following AIDS since the introduction of HIV antibody testing to 29 February 2000, by sex and State or Territory

		State or Territory								
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
HIV diagnoses	Female	26	604	11	153	61	5	212	116	1,188
	Male	223	10,901	109	1,990	674	79	3,892	918	18,786
	Sex not reported	0	252	0	0	0	0	24	0	276
	Total[1]	249	11,775	120	2,150	735	84	4,142	1,038	20,293
AIDS diagnoses	Female	9	188	0	48	25	3	69	26	368
	Male	86	4,644	36	823	346	44	1,616	350	7,945
	Total[1]	95	4,844	36	873	371	47	1,692	378	8,336
AIDS deaths	Female	4	113	0	32	15	2	49	16	231
	Male	66	3,171	24	567	231	29	1,270	248	5,606
	Total[1]	70	3,292	24	601	246	31	1,325	265	5,854

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

Childhood Immunisation Coverage

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

The data show the percentage of children fully immunised at age 12 months for the cohort born between 1 January and 31 March 1999 and at 24 months of age for the cohort born between 1 January and 31 March 1998, according to the Australian Standard Vaccination Schedule.

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

Acceptance of a report does not imply a causal relationship between administration of the vaccine and the medical 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 six years.

Table 10. Percentage of children immunised at 1 year of age, preliminary results by disease and State for the birth cohort 1 January to 31 March 1999; assessment date 30 June 2000

	State or Territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,068	21,412	941	12,332	4,509	1,541	15,102	6,225	63,130
Diphtheria, Tetanus, Pertussis (%)	91.5	88.2	84.2	90.4	91.9	92.8	91.2	88.8	89.8
Poliomyelitis (%)	91.2	88.4	84.6	90.3	91.8	92.6	91.3	88.8	89.8
Haemophilus influenzae type b (%)	91.3	87.6	88.7	90.4	90.7	91.7	90.8	88.0	89.3
Fully immunised (%)	91.1	86.5	82.7	89.7	90.2	91.1	90.0	86.9	88.4
Change in fully immunised since last quarter (%)	-0.7	-0.1	-0.3	0.0	+1.1	+2.8	+0.6	+1.1	+0.3

Table 11. Proportion of children immunised at 2 years of age, preliminary results by disease and State for the birth cohort 1 January to 31 March 1998; assessment date 30 June 2000[1]

	State or Territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,102	21,148	956	12,392	4,579	1,517	14,842	6,196	62,732
Diphtheria, Tetanus, Pertussis (%)	90.0	85.6	79.4	90.1	89.4	87.2	88.3	86.4	87.5
Poliomyelitis (%)	93.6	90.1	90.0	92.7	94.0	93.4	93.2	91.0	91.9
Haemophilus influenzae type b (%)	89.0	84.8	86.3	90.1	88.8	86.4	88.3	85.6	87.2
Measles, Mumps, Rubella (%)	92.5	89.3	89.7	92.1	92.4	92.9	92.1	90.2	91.0
Fully immunised (%)[2]	87.0	78.0	74.6	86.2	84.2	82.7	83.4	79.5	81.7
Change in fully immunised since last quarter (%)	+4.4	+4.2	+1.6	+4.7	+6.3	+4.0	+5.7	+6.2	+5.0

1. The 12 months age data for this cohort was published in *Commun Dis Intell* 1999;23:232.
2. These data relating to 2 year old children should be considered as preliminary. The proportions shown as “fully immunised” appear low when compared with the proportions for individual vaccines. This is at least partly due to poor identification of children on immunisation encounter forms.

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

Bulletin Board

The Australasian Society for HIV Medicine
12th Annual Conference
12-14 October 2000
Sofitel, Melbourne, Victoria
Phone: 02 9368 2700
Fax: 02 9380 9528
Email: ashm@sesahs.nsw.gov.au

VTEC 2000
4th International Symposium and Workshop on 'Shiga Toxin (Verocytotoxin) - Producing *Escherichia coli* Infections
29 October-2 November 2000
Kyoto, Japan
Phone: +81 3 3423 4180
Fax: +81 3 3423 4108
Email: vtec@mx6.mesh.ne.jp

The NSW Infection Control Association
23rd Annual Conference
2-3 November 2000
Civic Hall, Newcastle, New South Wales
Phone: 02 4921 8777
Fax: 02 4921 8778
Email: simpson@mail.newcastle.edu.au
Early bird registration 29 September 2000

Public Health Association of Australia
32nd Annual Conference
26-29 November 2000
National Convention Centre, Canberra
Phone: 02 6285 2373
Fax: 02 6282 5438
Email: conference@phaa.net.au
Website: http://www.phaa.net.au

Royal North Shore Hospital
Outpatient Parenteral Therapy - beyond 2000
TBA 2001
Fairmont Resort
Leura, New South Wales
Phone: 02 9956 8333
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Email: confact@conferenceaction.com.au

Institute for Microbiology of Medical Faculty of Masaryk University & St Anna's Faculty Hospital
10th Tomasek Days
Annual conference of young microbiologists
6-8 June 2001
Brno, Czechia
Contact: Ondrej Zahradnicek
Phone: ++420 5 4318309
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Contributions to the Bulletin Board are invited from those organisations with forthcoming events relevant to communicable disease control.

Overseas briefs

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

Cholera in the Federated States of Micronesia

An outbreak of cholera was first reported on 17 April by the public health authorities of Pohnpei island. As of 26 June, 954 cases and 9 deaths had been reported to WHO, on the basis of inpatient and outpatient data from the Pohnpei hospital and community-based dispensaries. *Vibrio cholerae* serotype Ogawa has been isolated. Preventive and control measures are being implemented.

Foodborne intoxication due to Staphylococcus aureus in Japan

On 10 July, the Ministry of Health and Welfare reported a total of 13,809 cases of food poisoning in 8 western states. Of 180 cases requiring hospitalisation, 17 were still in hospital. The source of the outbreak was found to be 3 different kinds of milk which had been contaminated by *Staphylococcus aureus* in a production-line valve, at a major dairy-products processing plant in Osaka. Those affected suffered diarrhoea and vomiting due to *Staphylococcus aureus* enterotoxin after drinking low-fat milk. The local authorities have ordered the company to recall all products which might have been contaminated.

Acute haemorrhagic fever syndrome in Afghanistan - Update

To date, there have been 27 suspected cases, including 16 deaths, reported from an isolated village in Gulran district, Herat Province, Afghanistan. An international team, including experts from the WHO Collaborating Centre at the National Institute for Virology (NIV- South Africa), Epicentre and WHO have completed their investigation. Extensive virologic and serologic tests for a wide variety of pathogens have not yet identified the etiologic agent.

Anthrax in Ethiopia

WHO has received reports of clusters of cases of suspected anthrax in the Afar region of Ethiopia. This area is inhabited by pastoralists who depend on livestock and cases of anthrax are known to occur. Reports from organisations (e.g. United Nations Development Programme, Médecins Sans Frontières) working in the area indicate clusters of cases and increased numbers of cases of a clinical syndrome consistent with anthrax. No systematic epidemiological investigation has been carried out thus far. WHO, in collaboration with MSF/Epicentre and the Ministry of Health in Ethiopia, have planned to carry out an investigation in the region in order to confirm the diagnosis, assess the true extent and impact of the disease and plan and implement control measures.

Source: 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.

Influenza, summer outbreak - USA (Texas)

In what may be this season's first outbreak nationally, 2 influenza cases have been confirmed and at least 68 more are being investigated at a children's camp south of Waco, US health officials say. There is no evidence that the viral illness has spread outside the McLennan County camp and it is unclear how the outbreak started. State health officials, who are now working with CDC officials, haven't yet identified the viral strain.

The first flu-like symptoms in campers and staff were identified on about 11 July. The camp has about 425 campers, ages 9 to 16, and more than 150 staff. The sick children have been kept isolated in one area of the camp and none of the campers have left without getting preventive medication. Parents are being alerted to watch for flu-like symptoms.

The central Texas outbreak follows news from the CDC that this season's vaccine will be delivered late. CDC officials say that one of the three strains to be included in the vaccine is slow growing and there may be a month's delay in delivery. Shots may not be available until November.

Lassa fever in UNAMSIL peacekeeping force in Daru

There has been another probable case of Lassa fever in Freetown, unrelated to the four cases (and four deaths) reported earlier. A 21-year-old male member of the Indian contingent of UNAMSIL peacekeeping forces stationed in Daru, Kailahun District, presented with fever on about 10 July. He was treated presumptively for malaria. Subsequently malaria and typhoid were ruled out by laboratory tests. He was acutely ill, with temperatures up to 41 C. Lassa fever was suspected, and he was transported to the UNAMSIL referral hospital in Freetown, where he has been maintained in strict isolation, with careful precautions in handling of blood and body fluids. He developed pharyngitis, facial and pharyngeal oedema and erythema, and bleeding and ecchymoses at venipuncture sites. He was started on IV ribavirin. On Day 3 of treatment, his temperature had come down and he was much improved. He continues to have albuminuria, but there is no evidence of renal, hepatic or pulmonary failure.

Prothrombin times remain moderately elevated. He was very well managed by the UNAMSIL medical services in Daru and Freetown. All contacts are being followed with temperatures recorded twice daily. UNAMSIL's preventive medicine officer is travelling to Daru to assess conditions at the camp, including food security and rodent control.

This is the third introduction of Lassa fever into urban Freetown. (The first was the DFID worker who later died in London). This is the first case involving UN peacekeeping forces. There is no laboratory capacity to confirm the diagnosis, and no definitive investigation has been done.

Lassa fever – Netherlands ex Sierra Leone

Lassa fever has been diagnosed in a surgeon working in Sierra Leone (not in Freetown), while visiting his relatives in the Netherlands. While in Sierra Leone on 11 July, a bout of fever was diagnosed as malaria (on the basis of a blood smear). The patient was treated with artemisine. On 14 July he arrived in the Netherlands for a short leave. He still had a fever, but no cough; and a thick smear was negative. On 15 July, he was admitted to hospital. On 16 July he exhibited fever, diarrhoea, and vomiting, but no cough. By 20 July, clinical deterioration was apparent and intravenous ribavirin treatment was begun. On 22 July, the results of a PCR test from a blood sample taken on 19 July revealed 10^7 Lassa fever virus RNA molecules/ml. An antibody test, using the Lassa fever virus, Josiah strain (the strain prevalent in Sierra Leone), was negative. By 24 July, the condition of the patient was poor with multiple organ failure, and he was transferred to intensive care. The patient has been in isolation since 17 July.

Contacts, including hospital staff-at-risk and airline cabin attendants, have been asked to report body temperature twice daily for three weeks. The airline company is tracing passengers who were seated close to the patient on the flight from Sierra Leone on 13 July.

Global salm-surv on internet

Global Salm-Surv (GSS), WHO's *Salmonella* surveillance and laboratory support project, is now accessible on the Internet, at: http://www.who.int/salmsurv.

Initiated in January 2000, GSS is a collaborative project of WHO, the WHO Collaborating Centre for Foodborne Disease Surveillance (Atlanta, United States) and the Danish Veterinary Laboratory (Copenhagen, Denmark). GSS is a global network of over 150 individuals from 108 laboratories and 66 countries involved in the surveillance of *Salmonella* from humans, animals and food. The primary goal of this network is to strengthen the *Salmonella* surveillance capacities of national and regional laboratories.

Global Salm-Surv consists of the following:

1. An international, online accessible database that contains: contact information of national or regional salmonellosis laboratories; descriptions of laboratory responsibilities, laboratory methods and types of samples received; and, annual surveillance summary results of most frequently isolated *Salmonella* serotypes.

2. Data sharing and communication between laboratories and individuals via e-mail, web, electronic discussion group and/or fax.

3. Participation in internal and external quality assurance systems. This is a useful tool for the production of reliable laboratory results of consistently good quality. A recent WHO survey determined that less than 50% of laboratories performing antimicrobial resistance testing of *Salmonella* isolates participated in any formal system of quality assurance.

4. Training courses on surveillance of salmonellosis and antimicrobial resistance in *Salmonella*.

The aim is to provide training in the following:

(1) standardized laboratory methods for the isolation, identification and antimicrobial-susceptibility testing of foodborne *Salmonella*;
(2) interpretation of results; and
(3) utilization of foodborne disease surveillance and antimicrobial resistance.

5. Selected reference testing services for a limited number of *Salmonella* isolates per participant. Selected testing services available to GSS participants include serotyping and phage typing of *Salmonella*, and antimicrobial-susceptibility testing of *Salmonella* (and occasionally other foodborne bacteria).

Global Salm-Surv is part of WHO's endeavours to strengthen the capacities of its Member States in the surveillance and control of major foodborne diseases and to contribute to the global effort of surveillance and containment of antimicrobial resistance in foodborne pathogens. Eventually, it is intended to extend the network to other major foodborne pathogens.

Individuals and laboratories who are not currently members but are interested in participating in WHO's Global Salm-Surv should fill out the request form available on the website.

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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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