COMMUNICABLE DISEASES INTELLIGENCE

ISSN 0725-3141 VOLUME 19 NUMBER 6 20 March 1995

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COMMUNICABLE DISEASES NETWORK-AUSTRALIA
A National Network for Communicable Diseases Surveillance

REPORT OF A MULTIDRUG RESISTANT CLONE OF *STREPTOCOCCUS PNEUMONIAE* (MR*SPN*) IN ABORIGINAL INFANTS IN THE NORTHERN TERRITORY

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Summary

During pilot studies of antibiotic treatment of otitis media in Aboriginal infants in a remote community we detected multidrug resistant *Streptococcus pneumoniae* in the nasopharynx of infants studied and showed, using selective media, that such MR*Spn* can be present, without being the dominant strain, in infants with multiple strains of pneumococci. The carriage rate of MR*Spn* significantly increased over the period of observation, probably because antibiotic treatment reduced the prevalence of sensitive strains and provided pre-existing MR*Spn* with a selective advantage and thus facilitated their more rapid spread.

Introduction

Australia had had no published report of multidrug resistant *Streptococcus pneumoniae* nor *S. pneumoniae* with high level penicillin resistance[1] until the recent *CDI* report that described the latter finding[2]. *S. pneumoniae* is a common cause of otitis media in Aboriginal infants. Otitis media (OM) is endemic in the Northern Territory[3] and nasopharyngeal carriage of *S. pneumoniae* and/or *Haemophilus influenzae* and to a lesser extent *Moraxella catarrhalis* within weeks of birth is predictive of the onset of OM which persists throughout childhood[4].

We have recently conducted studies on the natural history and microbiology of OM and on antibiotic treatment of small numbers of Aboriginal infants with otitis media. Treatment regimens were based on the Australian antibiotic guidelines[5] for otitis media in children; we found that short (five day) courses of antibiotic were not followed by resolution of middle ear effusions or by loss of bacterial pathogens from the nasopharynx. Longer courses were evaluated in some children; eradication of *S. pneumoniae* from the nasopharynx was occasionally seen although ear disease was unresolved and *H. influenzae* and *M. catarrhalis* continued to colonise the nasopharynx. However, in other infants, *S. pneumoniae* was not eradicated, and in a proportion of such infants multidrug resistant *S. pneumoniae* (MR*Spn*) was recovered. Details of these studies will be reported elsewhere.

We detected a multidrug resistant *S. pneumoniae* during these studies and determined its characteristics and changing prevalence over time.

Subjects and methods

The study was approved by an Institutional Ethics Committee working to National Health and Medical Research Council guidelines.

Phase 1: Prospective study from January 1992 to June 1993

Infants from a remote Aboriginal community were enrolled from birth in a prospective study of the natural history and microbiology of otitis media[4]. During this time infants were also seen in the clinic by health service staff and offered treatment on clinical indications. Compliance with antibiotic regimens was not documented during this period although we believe it to have been poor, because it was necessary to provide assistance with compliance during our subsequent (Phase II) treatment studies.

Phase II: Antibiotic treatment studies from July 1993 to June 1994

From July 1993 mothers in the same community were asked to enrol their newborn infants and infants with OM in a study of antibiotic treatment. Seventeen children who were included in the Phase I of the study were also included in Phase II. Initially, infants with OM were given bicillin (IM) on days one and three, or if the mother did not consent to bicillin injection, infants were randomised to cefaclor (50 mg/kg/day in three doses) or amoxycillin (50 mg/kg/day in three doses) for five days. If OM had not resolved at the 14 day follow-up examination, infants received additional five day courses of the same oral antibiotics. A later trial of 28 days amoxycillin (50mg/kg/day in two doses) began in March 1994. An Aboriginal Health Worker (AHW) and a mother living in this community who were given specific training[6] visited each household daily to record antibiotic use and to assist the mothers with any problems. Additional antibiotics prescribed during this time for other clinical indications were recorded from clinic notes.

Nasopharyngeal swab collections and ear examinations

During Phase I nasopharyngeal swabs were taken and ear states were determined (according to an algorithm based on tympanometry and pneumatic otoscopy)[3] during monthly visits to the community. More frequent swab collections were made during Phase II.

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Microbiological methods

Standard culture methods were used as previously described[4].

Selective culture medium for MR*Spn* screening

A selective medium, SSChlor (Staph Strep agar (Oxoid) with 1.5μg chloramphenicol/mL) was developed for detection of dual carriage of antimicrobial sensitive *S. pneumoniae* (S*Spn*) and MR*Spn* strains. A 100μL inoculum was spread over a full plate and large and small colonies were counted following a 24 hour incubation at 37°C in 5% CO_2; when MR*Spn* was the only type detected or when the number of sensitive strains was several fold less on SSChlor plates this was defined as MR*Spn* dominance; MR*Spn* was described as a minority clone when detected in small numbers, usually less than 1:40 MR*Spn*:S*Spn*.

Antimicrobial susceptibility testing

Up to four colonies of *S. pneumoniae* were selected from each specimen culture for susceptibility testing by the CDS method[7]. Antimicrobials tested were oxacillin (1.0μg), penicillin (0.5μg), tetracycline (30μg), erythromycin (5μg), cotrimoxazole (25μg) and chloramphenicol (30μg) (BBL or Oxoid). The minimal inhibitory concentrations (MICs) for penicillin, cefotaxime and ceftriaxone were determined by E-test[8] for isolates showing multiple drug resistance. Additional colonies selected from the SSChlor plates were similarly tested.

Serotyping

All MR*Spn* isolates were serotyped (MG) by the capsular swelling (Quellung) method[9] with antisera made by the Statens Seruminstitut, Copenhagen, Denmark.

Results

Infants enrolled and swabs collected

During Phase I, 41 infants were enrolled and 204 swabs were taken (an average of five swabs per infant). The infants were less than one month of age at the beginning of the study; there were 23 females and 18 males. For Phase II treatment studies there were 31 enrolled infants, 28 of whom received treatment; three began treatment on the last day of this analysis. An average of 11.4 swabs was collected from each of the 28 treated infants; 321 swabs in total.

Nasopharyngeal colonisation

All infants became colonised by *S. pneumoniae* during the 2.5 year study period and absence of *S. pneumoniae* from the nasopharynx was rarely observed. Seventeen infants were colonised by MR*Spn*, three during Phase I and 14 during the treatment studies (Table 1). The frequency of isolation of MR*Spn* increased significantly ($\chi^2_1 = 14.02$, $p < 0.0002$) from 3/41 (7%) in Phase I to 14/31 (45%) in Phase II of the study. Although antibiotic prescription rates were similar in both Phases (data not shown), it was noted that during Phase II, compliance with antibiotic regimens was enhanced by daily visits by the AHW and trained local assistant. Thus the frequency of isolation of MR*Spn* increased during the period of greatest antibiotic consumption.

We noted that six of the 17 (36%) infants (all with OM) were colonised by MR*Spn* before or at the time treatment began; we therefore analysed the data according to history of antibiotic use (that is, none versus any), irrespective of the Phase of study. Table 2 shows that MR*Spn* was detected in infants not known to have had antibiotics, as well as those with history of prior or current treatment; treated infants were in fact less likely to be carriers of MR*Spn* ($p < 0.02$) than carriers of sensitive strains; this is paradoxical.

Table 1. Infants colonised by sensitive *S. pneumoniae* and MR*Spn* and average number of antibiotic courses prescribed per infant during natural history (Phase I) and treatment (Phase II) studies

Infants	*S. pneumoniae* (*Spn*) positive cases	MR*Spn* negative cases	MR*Spn* positive cases	Total
Phase I	41	38	3	41
Phase II	31	17	14	31
Total	72	55	17	72

$\chi^2_1 = 14.02$, $p < 0.0002$

Table 2. Infants colonised by MR*Spn* and average number of antibiotic courses prescribed, by history of antibiotic use, irrespective of Phase

Infants	No antibiotic use	Any antibiotic use	Total
MR*Spn* negative	3	35	38
MR*Spn* positive	6	11	17
Total	9	46	55

Fisher exact test: $p < 0.02$.

The selective medium, SSChlor, enabled isolation of MR*Spn* clones from specimens initially identified as having only sensitive pneumococcal isolates. Of the 42 MR*Spn* isolates collected in Phase II, eight (19%) were found in specimens that initially yielded sensitive strains (Table 3). All 17 infants were repeatedly swabbed and MR*Spn* was identified as the dominant clone in all 17 infants in at least one of the swabs; thus the selective medium did not increase the number of individuals identified as MR*Spn* carriers, although it did reveal the presence of MR*Spn* when it was not the dominant strain.

Table 3. Swabs cultured and isolation of MR*Spn* during Phases I and II

Swabs	MR*Spn* positive	MR*Spn* negative	Total
Phase I	7	197	204
Phase II	42[1]	279	321
Total	49	476	525

$\chi^2_1 = 13.73$, $p = 0.0002$.

1. 8/42 (19%) MR*Spn* positive swabs detected only with use of selective medium.

Characteristics of the MR*Spn* clone

The MR*Spn* isolates were relatively resistant to penicillin (MIC values in the range 0.5 to 1.0μg/mL in the E-test), resistant to oxacillin, erythromycin, tetracycline, co-trimoxazole and chloramphenicol and sensitive to rifampin and vancomycin (CDS method). MICs in the E-test for cefotaxime and for ceftriaxone were in the intermediate range (0.25 and 0.5μg/mL)[10]. There was no significant rise in the penicillin MIC for strains isolated in 1994 compared to those isolated in 1992 and 1993.

All isolates tested belong to serotype 6B.

Discussion

We have previously shown high carriage rates of *S. pneumoniae, H. influenzae* and *M. catarrhalis* in the nasopharynx of Aboriginal infants; the timing of colonisation is predictive of onset of OM within the first weeks of life[4]. These infants are persistently colonised by multiple species and types of bacteria during the first months and years of life and this irreversible colonisation we believe to be responsible for chronic otitis media throughout infancy and childhood. Such infants are exposed to multiple courses of antibiotics, although compliance is often poor.

In this report we show a serotype 6B MR*Spn* clone to be present in the community, the prevalence of which increased significantly between Phase I and Phase II of our studies. Infants living in the community but not receiving antibiotics were at risk of colonisation by MR*Spn*.

We also show that the MR*Spn* strain is detectable, using selective antibiotic plates, even when it is not the dominant strain in the nasopharynx; this is a particular example of our general observation that individual Aboriginal infants are colonised with multiple strains (serotypes) of *S. pneumoniae* at the same time.

How is the paradox of treated infants being less likely to be carriers of MR*Spn* than carriers of sensitive strains explained? The average age of the six infants who became colonised with MR*Spn* prior to antibiotic use was only 46.3 days, and five of these colonisations were detected during Phase II when MR*Spn* were more common. Thus it is likely that neonates in this remote Aboriginal community became colonised by a clone of MR*Spn* that had gained a selective advantage over sensitive strains in older (multiply colonised) infants who had received antibiotics; naturally also these infants had had less time to be exposed to antibiotics, thus explaining the paradoxical association in Table 2.

These data indicate that if one of the pneumococcal strains carried in the nasopharynx is antibiotic resistant (in our case MR*Spn*) it will derive a selective advantage when exposed to antibiotics and high compliance rates, as during Phase II, the studies of treatment for OM. This selective effect probably explains the increasing prevalence of MR*Spn* (in comparison with antibiotic sensitive strains) over the time course of our observations. Thus the antibiotic use has revealed the presence of MR*Spn* both by inhibiting the growth of sensitive strains, and probably as a consequence by allowing MR*Spn* to be propagated and transmitted preferentially.

A significant reduction in the carriage rate of *S. pneumoniae* was recently described from a Swedish study of the ecological effects of placebo versus penicillin V on nasopharyngeal flora[11]. The authors concluded that there had been no obvious ecological drawbacks. However, unlike the pneumococcal population in our study, the Swedish study found that all pneumococcal strains were sensitive.

This report is the first to describe a possible effect of antibiotic in promoting the growth and transmission of a pre-existing strain of MR*Spn*. It is of particular relevance to groups who live in overcrowded circumstances where carriage rates of pneumococci are high, such as children in remote Aboriginal communites, large day-care centres or overcrowded hospitals.

The good news from this study is that when compliance is good, as in Phase II, antibiotics are effective in reducing the frequency of (sensitive) pneumococcal strains which contribute to conditions such as otitis media; the bad news is that a resistant strain if present in the population can thereby gain a selective advantage. Thus treatment of pneumococcal infection requires careful consideration and monitoring to ensure that if resistant strains are detected, they can be treated appropriately. It is noteworthy that the multidrug resistant serotype 6B clone detected here shows only intermediate resistance to penicillin, ceftriaxone and cefotaxime.

Acknowledgments

We thank all the mothers and their infants who participated in this study as well as Kath Kemp, Maureen Tipuamantumirri, Colleen Kantilla and Judith Boswell for specimen collection, consent procedures and compliance monitoring. Absorbed factor serum which enabled the differentiation of serotype 6 was generously provided by Dr Jorgen Henrichsen (Statens Seruminstitut, Copenhagen, Denmark).

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ACUTE POSTSTREPTOCOCCAL GLOMERULONEPHRITIS AND ACUTE RHEUMATIC FEVER IN FAR NORTH QUEENSLAND, 1994

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Introduction

A large outbreak of acute poststreptococcal glomerulonephritis (APSGN) that occurred in Cape York communities in 1993[1], has re-emphasised the importance of Group A streptococcal (GAS) infections, and their sequelae, APSGN and acute rheumatic fever (ARF), to indigenous Australian children. In an attempt to understand the epidemiology of APSGN and ARF better, surveillance for the two conditions was commenced in Far North Queensland (FNQ) in 1994.

Methods

A case of APSGN was defined[1] as having haematuraia (>10 red blood cells per μL upon urine microscopy) and at least two of:

i. facial oedema
ii. hypertension (diastolic pressure ≥ 85mm Hg)
iii. hypocomplementaemia (C3 ≤ 0.6/L), and
iv. *either* an antistreptolysin O titre (ASOT) > 512 IU/mL *or* GAS cultured from a skin swab.

A case of ARF was defined according to updated (1992) Jones criteria[2].

Raised anti-DNAase B (ADB) titres were not considered as being indicative of recent GAS infections for either case definition. This was because several surveys[3,4] have demonstrated high prevalences of very high ADB titres in Aboriginal children in north Australia, presumably reflecting that GAS infections are ubiquitous in these children. However, an ASOT of > 512 IU/mL was included in both case definitions because it has been suggested that this level is indicative of a recent GAS infection in north Australian Aboriginal children[3].

From January to June 1994 laboratory based surveillance was undertaken, using hypocomplementaemia and raised ASOT as the reportable serological markers. This surveillance was undertaken primarily at the Cairns Base Hospital laboratory, but two private laboratories were also included. Follow-up of each case was undertaken with clinicians who requested the tests.

For the second half of the year, not only laboratory based surveillance, but also clinical case surveillance was undertaken. Eleven hospital superintendents and six specialist physicians in FNQ were asked, on a monthly basis, if any new APSGN or ARF cases had been seen in the preceding month. Follow-up questionnaires requesting more detailed information were utilised when a clinician indicated that a new case of either condition had been recently diagnosed.

Population data from the 1991 national Census (Australian Bureau of Statistics) were used to calculate incidence rates.

Results

APSGN

A total of 100 incident cases of APSGN was identified during the year (Figure). Eighty-five cases were identified through surveillance and 15 were identified through a screening procedure (detailed in reference 1) mounted in response to two community outbreaks of APSGN.

Fifty-eight cases were Aboriginal, and 38 Torres Strait Islanders; therefore 96% of the cases occurred in Aboriginal and Torres Strait Island persons. The crude incidence of APSGN for indigenous persons was 424 cases/100,000.

The median age was 5.0 years (range 15 months to 57 years); the incidence for indigenous children less than 5 years of age was 1344 (95% confidence interval (CI) 988-1787) cases/100,000 children. Both sexes were equally affected.

Hypocomplementaemia and facial oedema were the most common clinical features of the APSGN cases (Table 1). There was one death, of an eight year old Torres Strait Island child; there had been considerable delay before assistance was sought for this child. Seventy-two of the cases were hospitalised; severe hypertension was the most common of the major complications that developed (Table 2). An 18 month old child, the cousin of the child who died, developed pneumococcal pericarditis and a purulent effusion; he underwent pericardotomy in Brisbane. A 17 year old pregnant Aboriginal woman required dialysis to manage acute renal failure.

ARF

Twenty-one episodes of ARF were identified; six were recurrences in patients with a past history of ARF/rheumatic carditis. Of the 15 new (that is incident) cases, 14 occurred in indigenous persons (11 in girls and three in boys).

The median age of the incident indigenous cases was 12.8 years (range 4.5 to 39 years). The crude incidence of ARF for indigenous persons was 62 cases/100,000, and the incidence for indigenous school aged children five to 14 years of age was 161/100,000.

Figure. Acute poststreptococcal glomerulonephritis cases, Far North Queensland, 1994, by month and detection method

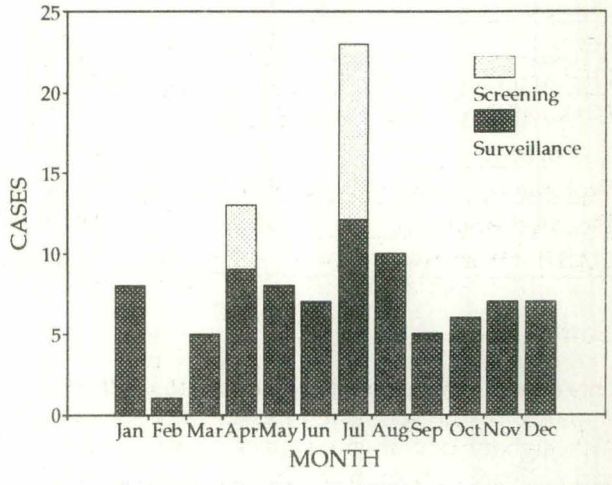

Table 1. Clinical features of the APSGN cases

Clinical features	Number examined	Number (%) with feature
Macroscopic haematuria	100	36 (36)
Facial oedema	100	68 (68)
Hypertension	100	64 (64)
Hypocomplementaemia	96	84 (88)
Elevated ASOT	96	37 (39)
GAS in skin swab	44	22 (50)

Table 2. Complications of the APSGN cases

Complication	Number (%)
Severe hypertension (diastolic > 100 mmHg)	40 (40)
Acute renal failure	18 (18)
Fluid overload/ pulmonary oedema	12 (12)
Bacteraemic infection	6 (6)

Eleven of the new cases and three of the recurrence patients (67% of all patients) were hospitalised; an 11 year old Aboriginal female developed severe congestive cardiac failure requiring repeated and prolonged admissions. The clinical features of the incident cases are shown in Table 3. Only six of the incident cases are known to have had an echocardiogram; two children had mild to moderate, and two had severe valvular disease reported.

Table 3. Clinical features of the incident cases of ARF

Clinical feature	Number examined	Number (%) with feature
Carditis	15	10 (67)
Polyarthritis	15	12 (80)
Chorea	15	1 (7)
Fever	15	13 (87)
Elevated ESR	15	15 (100)
Elevated C-reactive protein	5	5 (100)
Prolonged PR interval	12	5 (42)
Elevated ASOT	15	6 (40)
GAS in throat swab	4	1 (25)

Comments

This report details an initial attempt to undertake surveillance of the sequelae of GAS infections in FNQ. Although the documented attack rates of both APSGN and ARF are very high in Aboriginal and Torres Strait Island persons, we nevertheless believe that our data underestimate the true incidences. Clinical case surveillance was not used in the first six months, further cases of APSGN may have been identified if the screening procedure had been implemented on a more widespread basis, and asymptomatic cases of ARF ('silent carditis') are well recognised[5].

We believe that the high incidence of ASPGN in indigenous children indicates that the epidemic that began in 1993[1] continued into 1994. If so, there were nearly 200 documented cases in FNQ and the epidemic was of the same magnitude as an epidemic that occurred in Aboriginal communities in the Top End of the Northern Territory in 1980 in which 'several hundred' persons were affected[6]. Prospective surveillance will determine the trends in incidence; meanwhile hygiene education and effective case management of skin sepsis and scabies should be considered priority tasks for those providing primary health care for indigenous persons in FNQ.

The incidence of ARF in FNQ indigenous persons is of the same order as that reported in Aboriginal persons in central Australia[7], and in Maori and Pacific Island persons in Auckland, New Zealand[8]. Because we do not have denominator data for all past ARF cases, we do not know the recurrence rate, but we are concerned that nearly one third of all episodes of ARF were recurrences. This suggests that the standard recommendations for the prevention of recurrences of ARF[9] are not being adequately implemented in FNQ.

Rheumatic fever is recognised as being an important, and expensive, public health priority in New Zealand[8]; the response there has been to establish regional rheumatic fever registers, and to administer, via the district nursing service, monthly intramuscular benzathine penicillin to those on the registers. This secondary prophylaxis program has considerably reduced the recurrence rate of ARF[8]. To date no similar program has been established for indigenous persons in FNQ. Rather, a community based primary prevention ARF program has been attempted[10], but the difficulty experienced by that program[11] reflects just how difficult it is to reduce the prevalence of GAS carriage given the contemporary circumstances that prevail in indigenous communities in FNQ.

The 1994 data can perhaps form the basis of a register, and could be used to evaluate current secondary prevention practices. They emphasise the continuing importance of ARF to indigenous persons in north Australia, and the need to implement the standard recommendations for the prevention of rheumatic fever[9].

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AN OUTBREAK OF ACUTE POSTSTREPTOCOCCAL GLOMERULONEPHRITIS IN AN ABORIGINAL COMMUNITY

Jonathan Carapetis, Menzies School of Research, Darwin, Northern Territory; reproduced from the Northern Territory Communicable Diseases Bulletin 1994;2(3):1-4

Introductions and methods

Outbreaks of acute poststreptococcal glomerulonephritis (APSGN) occurred in one Aboriginal community in the Top End of the Northern Territory in 1980 and 1987[1,2]. APSGN outbreaks are recognised to occur in six to eight year cycles in some places[3], so it was predicted that a further outbreak would occur in this community in 1994. Informal conversations between health care professionals in early June 1994 alerted staff at the Menzies School of Health Research (MSHR) that a number of cases of APSGN had apparently been noted in the community in March and April. The District Medical Officer (DMO) for the community confirmed that there had been five cases admitted to Royal Darwin Hospital (RDH) and a further four cases incompletely investigated, but which were highly suspected of being APSGN. Following discussions with the Disease Control Centre at the Northern Territory Department of Health and Community Services, it was decided to conduct an on-site investigation, although it seemed likely that the outbreak was over.

The objectives of the outbreak investigation were:

- to ascertain individual cases of APSGN which had occurred in this community in 1994,
- to describe the illnesses suffered by these individuals and to ensure that appropriate medical follow-up had occurred,
- to describe the extent of the outbreak,
- to determine if there remained a risk of further cases stemming from this outbreak and to decide on appropriate interventions to reduce the risk of this happening.

The community involved has a population of 1046 residents according to the Australian Bureau of Statistics' 1991 Census. Case finding was undertaken using details of confirmed or suspected cases of APSGN provided by the community DMO and health centre staff. The case definitions used were:

Confirmed case

1. clinical picture consistent with the diagnosis (for example puffy face),
2. abnormal urinary sediment (10×10^6/L red blood cells with 40% dysmorphic cells),
3. evidence of a recent Group A streptococcal infection (elevated ASOT or anti-DNAase B), and
4. reduced complement C3 level.

Possible case

1. clinical picture consistent with the diagnosis,
2. abnormal urinary sediment *or* heavy haematuria with or without proteinuria on urinalysis,
3. absence of results for C3 level and streptococcal serology.

Results

Between 7 March and 27 April 1994 there were five confirmed cases of APSGN and four possible cases (Figure). There was one further possible case in mid-June, seven weeks after the previous last case. Most of the possible cases were unable to be confirmed due to incomplete investigation at the time, particularly the absence of formal urine microscopy and C3 levels.

Figure. Confirmed and possible APSGN cases, 7 March to 27 April 1994, by week

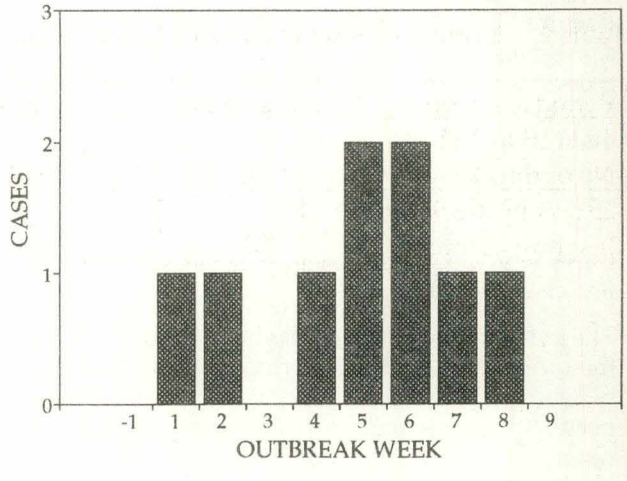

Table 1. Clinical features of the ten confirmed and possible APSGN cases

Clinical feature	Cases
Facial puffiness only symptom	7
Hypertension	4[1]
Elevated urea or creatinine	4[1]

1. All of these were hospitalised.

Of the ten confirmed and possible cases, nine were female. The age range was three to 21 years, with four cases being older than ten years. Six cases were admitted to Royal Darwin Hospital. Seven patients had facial puffiness as the only symptom (Table 1). One required antihypertensive treatment and none required dialysis.

During the outbreak investigation, hypertension was found in only one young adolescent. At subsequent follow-up of this individual, the blood pressure had normalised. In addition, this person and another adolescent case were found to have markedly elevated urinary albumin:creatinine ratios. This measure of proteinuria is thought to be a marker of underlying renal disease. These two were first cousins and had a strong family history of end stage renal disease. They were referred for ongoing review.

On follow-up, all other individuals were well and appeared to have fully recovered from their illness. Of the eight cases whose urine was re-checked, persistent glomerular haematuria was found in six.

Discussion

Because of the retrospective nature of the investigation, the true extent of this outbreak, in particular the number of mild clinical cases (which may not present to the clinic or be noted by health staff) and subclinical cases is not known. This lack of information on these cases may explain the outbreak curve which demonstrates an even spread of cases over the weeks of the outbreak instead of the more usual rapid increase in the early weeks followed by a prolonged tail of secondary cases.

Previous outbreaks of APSGN in this community were documented as more extensive. In 1987 there were 57 cases and in 1980 over 40 cases were found. The reports of these outbreaks do not detail the proportions of symptomatic and asymptomatic cases. During epidemics of APSGN, particularly among families, asymptomatic APSGN episodes outnumber symptomatic episodes by a factor three or four to one[4].

As one means of attempting to assess the true extent of this outbreak, the MSHR Renal Disease Survey team kindly allowed us to scrutinise their records of urine dipstick screening of children from the local school of the affected community. Although this survey has always found a number of children at any one time with blood in their urine, examination of the results revealed an apparent increased rate of haematuria in the paediatric population of this community between November-December 1993 and May-June 1994 (Table 2). This screening did not take place during the period of the detected outbreak, but the results may indicate a number of children in the active or recovery phase of APSGN.

As expected, it was concluded that the outbreak was over by the time of the investigation, and no program of widespread screening or administration of prophylactic benzathine penicillin was carried out. However, as it was possible that the responsible Group A streptococcus (GAS) was still circulating in the community, the health centre staff were encouraged to be extra vigilant about detecting and treating scabies and skin sores.

An interesting feature of this outbreak was the relatively large proportion of cases over the age of ten years. This has been noted in previous studies of APSGN outbreaks in Aboriginal communities[2], whereas the literature implicates the three to ten year old age group as being the most at risk[3,5,6]. Whilst the reasons for this age distribution in Aboriginal communities are not clear, it must be taken into account when planning screening and intervention programs for future outbreaks.

Table 2. Urine screening of children in the community

Period of screening	Total screened	Blood 2+	Blood 1+ or less	Total blood > trace
10.11.93 to 7.12.93 No outbreak	60	6 (10%)	7 (12%)	13 (22%)
25.5.94 to 16.6.94 Just post outbreak	257	53 (21%)	42 (16%)	95 (37%)

The clinical presentation of these cases was typical, in that the majority of clinical cases did not present with the full 'nephritic syndrome' (frank haematuria, reduced urine output, hypertension and oedema) but rather with facial puffiness and microscopic haematuria. The excellent outcome is also in keeping with what is known about the disease[4,7]. In general the prognosis of APSGN is good. In most patients, symptoms and signs will resolve within a week, although persistent urinary abnormalities may be noted for months or years. In a New Zealand study[5], proteinuria was found in 20% of patients two years after APSGN, although haematuria had virtually disappeared by 12 months. Epidemic (outbreak) APSGN has a better outcome than endemic (sporadic) disease, with a mortality of less than 1%[4].

The question of whether APSGN can lead to lasting or progressive renal impairment is unresolved. Most studies have found very little evidence that such progression occurs[4,8-11], although few studies have occurred in populations exposed to persistent GAS infection as is found in Aboriginal communities of Northern Australia. It is possible that the extremely high rates of end-stage renal disease seen in this population may be explained to some extent by recurrent clinical and subclinical episodes of APSGN in early life.

The textbook diagnosis of APSGN requires evidence of nephritis, evidence of current or preceding GAS infection and, in most cases, reduction of serum C3 levels. In Aboriginal communities, GAS infection is endemic and serological evidence of GAS infection (ASOT and anti-DNAase B) is almost invariably present. Moreover, due to reasons unknown, glomerular haematuria and/or proteinuria is also widespread in non-endemic circumstances. In many cases, therefore, the most important laboratory diagnostic criterion is a reduced C3 level.

It is apparent that the health care staff from the community and from RDH were not aware of the need to notify cases of APSGN to the Northern Territory Department of Health and Community Services. Had such notifications occurred at the time it may have been possible to assess this outbreak properly, and to intervene to prevent further cases. This has resulted in two further strategies. First, the Disease Control Centre will be alerting staff at all levels about the diseases which are notifiable, and the importance of notification. Second, a comprehensive protocol is presently being drawn up for investigation and intervention in future outbreaks of APSGN, so that health staff can be assured that notification of this disease will result in practical action to stem the outbreak.

There is the further issue of the reasons for a number of these cases being incompletely worked up and followed up. Investigation of possible cases should include urine microscopy and serum for ASOT, anti-DNAase B, and C3 levels. In questioning the community health staff, it was apparent that the main limitations to their ability to perform these duties were lack of time and staff. This illustrates that even in a community with an on-site DMO the workload is extreme and the provision of facilities may be inadequate. This staffing problem is a long-term one for most remote communities and requires continued emphasis. Service providers in communities need support from researchers, hospital staff and rural health/disease control staff in making their situation a priority.

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OVERSEAS BRIEFS

In the last two weeks, the following information has been supplied by the World Health Organization.

Yellow fever in Gabon

The outbreak of yellow fever in Gabon has caused 44 cases (28 fatal) in the town of Makokou and three secondary foci in neighbouring areas in the north-east of the country. Makokou in the Province of Ogooue-Ivindo has been declared infected. Yellow fever vaccination has been carried out in the Province and in adjacent departments and those with frontiers with neighbouring countries. Measures are also being undertaken to extend yellow fever vaccination to the whole country.

Polio eradication efforts in Europe-Asia

The Eastern Mediterranean and European Regional Offices of the World Health Organization are currently organising Operation MECACAR, an effort to eradicate poliomyelitis from the Middle East, the Caucasus and the Central Asia Republics. It is the largest multi-country immunisation campaign to date; during the period March to May 1995, mass campaigns will be conducted to immunise the nearly 70 million children in the area under the age of five years. All children will receive two doses of oral polio vaccine during two rounds of national immunisation campaigns about one month apart. Conducting a campaign in such a wide area will reduce the likelihood of wild poliovirus being introduced from a neighbouring country.

Influenza in the Northern Hemisphere

By the beginning of March, influenza activity may have reached a peak in Europe but was continuing to increase in North America. In Europe, sporadic influenza or local outbreaks associated with influenza A H_3N_2 and/or influenza B viruses have been reported this season in Belgium, Finland, France, Italy, Norway, Poland (also one influenza A H_1N_1 isolate), Slovakia, Spain, Switzerland, the United Kingdom (much lower rates for clinical indices than last winter, and influenza B predominating) and Yugoslavia. Many European countries have reported very low or no activity this season.

Nineteen States in the United States reported widespread or regional activity in the second week of February. Influenza A continued to be the most common type in all regions (72%; all H_3N_2 except for one H_1N_1), except the South-Atlantic where influenza B has accounted for 56% of the isolates. Pneumonia and influenza mortality has remained at, or near expected levels so far for the season. Canada has reported mainly sporadic activity, with influenza A H_3N_2 and influenza B strains isolated.

Increasing activity was reported in Israel in February and influenza A H_3N_2 and influenza B viruses were isolated. Influenza B and influenza A H_3N_2 were also isolated from sporadic cases in Beijing and other areas of China in February.

WHO northern hemisphere influenza vaccine recommendations

The World Health Organization has issued its recommendations for the influenza strains to be included in influenza vaccines for the 1995-1996 northern winter. They are an A/Johannesburg/33/94 (H_3N_2)-like strain, an A/Singapore/6/86 (H_1N_1)-like strain and a B/Beijing/184/93-like strain[1]. The influenza A H_3N_2 and influenza B strains differ from those in previous vaccines and reflect changes in the strains that are currently circulating. Recent isolates of influenza A H_1N_1 have been closely related to the current vaccine viruses (A/Singapore/6/86 and the closely related A/Texas/36/91).

Cholera update

Gaza has been removed from the list of cholera infected areas.

Cholera cases have been reported for December, January and February from Afghanistan, Angola, Cambodia, Cameroon, Cape Verde, Chile, Djibouti, El Salvador, Gaza, Ghana, Guinea Bissau, Guyana, India, Iraq, Italy, Kenya, Philippines, Sierra Leone, Tanzania (in Rwandan refugee camps) and Zaire (in Rwandan refugee camps).

Reference

1. Recommended composition of influenza virus vaccines for use in the 1995-1996 season. *Wkly Epidemiol Rec* 1995;70:53-56.

CDI NOTICE TO READERS

Microbes in the ACT: Australian Society for Microbiology 1995 Annual Scientific Meeting and Exhibition

The 1995 Annual Scientific Meeting of the Australian Society for Microbiology is to be held at the National Convention Centre, Canberra from 24 to 29 September 1995.

The scientific program features plenary sessions on contemporary microbiology topics presented by leading international speakers. Symposia covering a diverse range of interests and a number of workshops are also planned.

Topics to be covered at the meeting include Aboriginal health, foodborne disease surveillance, pneumococci and antimicrobial resistance, mosquitoes and malaria and the 1994 horse (and human) virus disease, amongst many others.

Contributed oral papers and posters on any area of micriobiology are invited. Participation is open to all persons interested in microbiology. Registration brochures and abstract forms are available from the conference Secretariat:

ASM '95 Secretariat
GPO Box 128
SYDNEY NSW 2001

Telephone: (02) 262 2277
Facsimile: (02) 262 2323
Email: KHALL@TOURHOSTS.COM.AU

COMMUNICABLE DISEASES SURVEILLANCE

Virology and Serology Reporting Scheme

There were 2,180 reports received in the *CDI* Virology and Serology Reporting Scheme this fortnight (Tables 6, 7 and 8).

- Twenty reports of **measles** were received this period, for 10 males and 10 females. Diagnosis was by IgM detection (19) where method of diagnosis was reported.
- **Rubella** was reported for 47 patients this fortnight including 15 females (11 in the 15 to 44 year age group) and 32 males. Forty-three diagnoses were by IgM detection and four by a fourfold rise in titre. The number of reports has declined after peaking in October.
- Twenty-seven reports of **hepatitis A** were received including 15 males and 8 females (4 sex unknown), age range 5 to 74 years.
- Positive **hepatitis B** serology was reported for 147 patients this fortnight, 80 males and 58 females (9 sex not reported). Sixty-six patients were in the 25 to 44 year age group, and 40 in the 15 to 24 year age group.
- Positive **hepatitis C** serology was reported for 383 patients this fortnight including 224 males and 156 females (3 sex not reported). Two hundred and ninety-six reports were for the 25 to 44 years age group. Included were 54 injecting drug users, one patient with HIV infection, one patient with renal failure and 4 pregnant women.
- **Hepatitis E** was reported for one patient from Tasmania.
- **Ross River virus** was reported for 84 patients this fortnight, 35 from Queensland, 37 from the Northern Territory, 9 from Western Australia and three from New South Wales. The diagnosis was confirmed (fourfold rise in titre) for one patient , the remainder being presumptive diagnoses (IgM detected). The number of reports has increased for January but is consistent with the seasonal peak of previous years.
- Nineteen reports of **Barmah Forest virus** were received this period, 16 from Queensland, two from New South Wales and one from the Northern Territory. Diagnsosis was by IgM detection for all patients.
- **Untyped flavivirus** was reported for a 20 year old female Victorian returned traveller diagnosed by IgM detection.
- Thirty-nine reports of **adenovirus** were received this fortnight diagnosed by virus isolation (16), antigen detection (13), single high titre (two) and total antibody (two). Method of diagnosis was not reported for 6 patients. Included were **adenovirus types 2** (one case), **3** (two cases), **9** (one case) and **47** (one case).
- **Herpes simplex virus type 1** was reported for 265 patients this fortnight, 262 isolations and 3 antigen detections.
- There were 84 reports of **cytomegalovirus** (CMV) this fortnight, 45 virus isolations, one antigen detection, one single high titre and 36 IgM detections. Included were 7 transplant recipients, one immunosuppressed patient, 3 patients with HIV infection, one pregnant female, one postnatal female and two preterm neonates.
- **Varicella-zoster virus** was reported for 68 patients this period. Method of diagnosis included virus

isolation (27), antigen detection (30), single high titre (one) and IgM detection (10).

- One hundred and thirty-three reports of **Epstein-Barr virus** were received this period.
- Thirteen reports of **parvovirus** were received, all diagnosed by IgM detection.
- **Coxsackievirus B3** was reported for one patient this fortnight.
- Ten reports of **rhinovirus** were received this period, all diagnosed by virus isolation. Five patients were under the age of 4 years.
- **Influenza A** was reported for 4 patients this fortnight, 3 males and one female, all aged less than 5 years. Diagnosis was by virus isolation (three) and single high titre (one).
- Two reports of **influenza B** were received this period, for one male and one female aged in the 25-64 years age group.
- Seventeen reports of **parainfluenza virus type 3** were received this fortnight, 14 for patients under the age of 5 years. Diagnosis was by virus isolation (11) and antigen detection (6).
- Eighteen reports of **respiratory syncytial virus (RSV)** were received this fortnight, 14 for patients under one year of age. Diagnosis was by virus isolation (4), antigen detection (11) and single high titre (two).
- **Rotavirus** was reported for 40 patients this period including 20 males and 18 females (two sex not stated). Twenty-five patients were under the age of 4 years.
- One hundred and forty-seven reports of ***Chlamydia trachomatis*** were received this fortnight, for 54 males and 93 females. One hundred and thirty-nine patients were in the 15 to 44 year age group and two were aged less than one month. Diagnosis was by culture (28), antigen detection (67), nucleic acid detection (51) and serology (one).
- **Q fever** was reported for 6 patients this period, all males aged between 22 and 70. Diagnosis was by fourfold rise in titre (3), single high titre (one) and IgM detection (2).
- One hundred and six reports of ***Bordetella*** were received this fortnight, 94 ***Bordetella pertussis*** and 12 ***Bordetella*** species. Forty-five patients were male and 60 female (one sex not reported). Two patients were aged less than one month. Forteen reports of *Bordetella pertussis* were for diagnoses by PCR detection of nucleic acid in nasopharyngeal specimens, the first such reports received by the *CDI* Virology and Serology Reporting Scheme.

Australian Sentinel Practice Research Network

Data for week 8 (ending 26 February) and week 9 (ending 5 March) are included in this issue of *CDI* (Table 1). There were 8471 consultations reported for week 8 and 8020 reported for week 9. The influenza reporting rate rose during this fortnight, with increased reports from New South Wales, Victoria and Queensland.

Sterile Sites Surveillance (LabDOSS)

Data for this fortnight have been provided by 11 laboratories. There were 179 reports of recent significant sepsis:

New South Wales: John Hunter Hospital 18; Liverpool Hospital 36; Prince of Wales Hospital 39; Royal Prince Alfred Hospital 19.
Queensland: Central Queensland Pathology Laboratory 2; Nambour General Hospital 5; Sullivan, Nicolaides and Partners 10.
ACT: Woden Valley Hospital 21.
Tasmania: Northern Tasmanian Pathology Service 4; Royal Hobart Hospital 15.
Western Australia: Princess Margaret Hospital for Children 10.

Table 1. Australian Sentinel Practice Research Network, weeks 8 and 9, 1995

Condition	Week 8, to 26 February 1995		Week 9, to 5 March 1995	
	Reports	Rate per 1000 encounters	Reports	Rate per 1000 encounters
Influenza	52	6.1	50	6.2
Rubella	1	0.1	2	0.2
Measles	0	0	0	0
Chickenpox	12	1.4	11	1.4
Pertussis	11	1.3	5	0.6
Gastroenteritis	115	13.6	96	12.0

Table 2. LabDOSS reports of blood isolates, by organism and clinical information

Organism	Clinical information						Risk factors				Total[1]
	Bone/Joint	Lower respiratory	Endocarditis	Gastrointestinal	Urinary tract	Skin	Surgery	Immunosuppressed	IV line	Neonatal	
Staphylococcus aureus	2				1	4	1	6	4	1	24[2]
Staphylococcus epidermidis							1			2	7
Staphylococcus coagulase negative	1			1			2	3	2		9
Escherichia coli				2	8	1	1	4			32
Klebsiella pneumoniae				1				2	1		9
Proteus mirabilis				1	1		3		2		6
Pseudomonas aeruginosa		1			1		1	2	1		6

1. Only organisms with 5 or more reports are included in this table.
2. MRSA 1.

Organisms reported 5 or more times from blood are detailed in Table 2. Other blood isolates not included in Table 2 were:

Gram positive: 1 *Bacillus* species, 1 *Corynebacterium jeikeium*, 2 *Corynebacterium* species, 3 *Enterococcus faecalis*, 2 *Enterococcus* species, 2 *Listeria monocytogenes* (65 year old male, and a 75 year old male, both from ACT), 1 *Staphylococcus warneri*, 1 *Streptococcus* Group A, 3 *Streptococcus* Group B, 1 Streptococcus Group C, 3 *Streptococcus* 'milleri', 2 *Streptococcus pneumoniae*, 3 *Streptococcus sanguis*, 1 *Streptococcus* 'viridans'.

Gram negative: 2 *Acinetobacter baumannii*, 1 *Aeromonas* species, 1 *Campylobacter jejuni*, 4 *Enterobacter cloacae*, 1 *Enterobacter* species, 2 *Haemophilus influenzae* (26 year old female from NSW, and a 4 year old female with epiglottitis from Tasmania), 3 *Klebsiella* species, 1 *Morganella morganii*, 4 *Neisseria meningitidis* (all from New South Wales: one, serogroup B, from a 34 year old septicaemic female with history of injecting drug use; one from a 30 year old female with chronic meningococcaemia; one, serogroup B, from a one year old male with a rash and fever; one with polyvalent ACYW135 agglutination from a 9 month old female with rash and fever), 1 *Pasteurella multocida*, 1 *Providencia rettgeri*, 1 *Pseudomonas paucimobilis*, 1 *Pseudomonas stutzeri*, 1 *Salmonella* Paratyphi A (reported in a 17 year old male from New South Wales with a history of overseas travel), 4 *Salmonella* species (18 year old female from ACT, one year old female and 7 year old male, both from New South Wales, all with gastrointestinal disease; one group B reported as a hospital acquired infection in a 25 year old male from New South Wales), 2 *Salmonella* Typhi (37 year year old male from ACT, 6 year old male from New South Wales; no risk factors reported for either case), 1 *Serratia marcescens*.

Anaerobes: 1 *Bacteroides fragilis*, 1 *Clostridium perfringens*, 1 *Clostridium septicum*, 3 *Clostridium* species.

Fungi: 3 *Candida albicans*, 1 *Candida glabrata*, 2 *Candida* species.

There were twelve blood isolates from patients aged less than one year and 80 from patients aged 55 years and over (Figure 1).

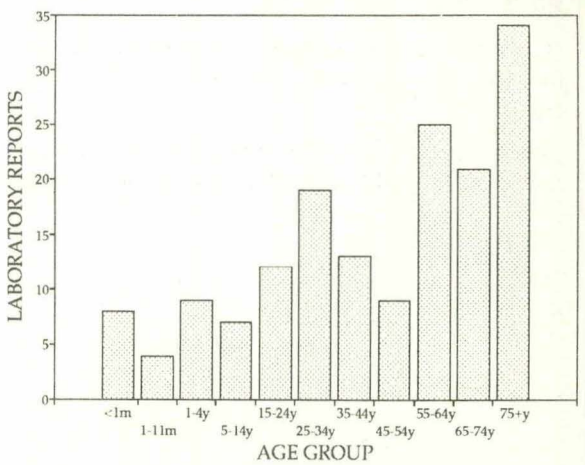
Hospital acquired blood isolates

Twenty-five isolates were reported as hospital acquired. The five most commonly reported organisms were: 7 *Staphylococcus aureus* (including 1 MRSA), 3 *Escherichia coli*, 2 *Enterobacter cloacae*, 2 *Klebsiella pneumoniae*, 2 *Pseudomonas aeruginosa*.

Meningitis and/or CSF isolate reports

There were 3 reports of meningitis and/or CSF isolates. *Pasteurella multocida* was isolated, from blood and CSF, in an 87 year old female from New South Wales. One isolate, from CSF, was *Staphylococcus aureus* reported in

Figure 1. LabDOSS reports of blood isolates, by age group



a 61 year old female with a risk factor of surgery, from the ACT. One isolate was *Staphylococcus epidermidis* in a 62 year old female with a risk factor of neurosurgery, from Tasmania.

Isolates from sites other than blood or CSF

Joint fluid: 1 *Enterobacter* species, 2 *Pseudomonas aeruginosa*, 2 *Staphylococcus aureus*, 1 *Streptococcus* Group A, 1 *Streptococcus* Group B.

Pleural fluid: 1 *Enterobacter cloacae*, 1 *Klebsiella oxytoca*, 1 *Pseudomonas aeruginosa*, 1 *Streptococcus* 'milleri'.

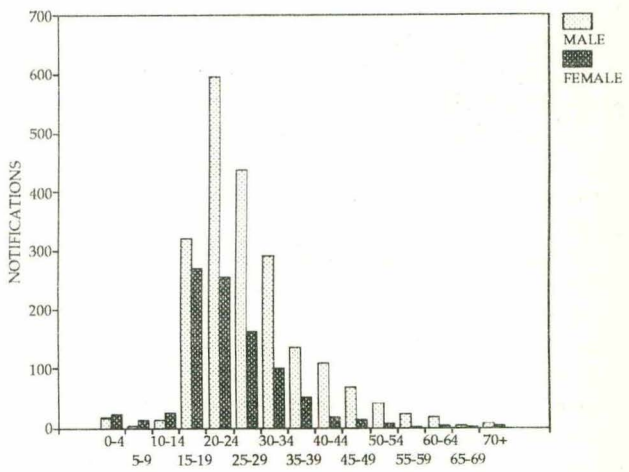
Other: 1 *Candida albicans*, 1 *Staphylococcus aureus*, 2 *Staphylococcus* coagulase negative.

National Notifiable Diseases Surveillance System, 19 February 1995 to 4 March 1995

There were 1963 reports received in the period (Tables 3, 4 and 5 and Figure 4).

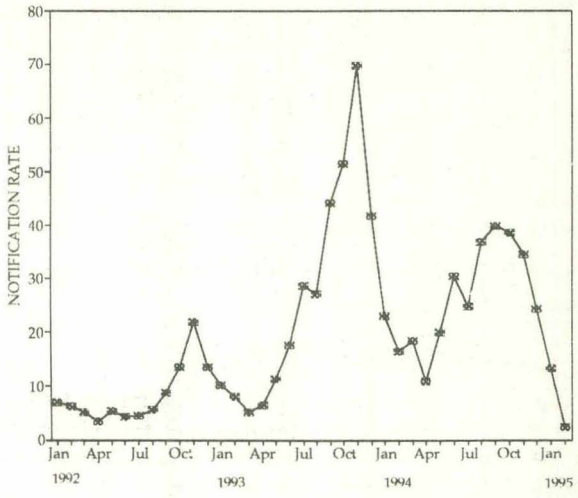
- There were 54 notifications of **Ross River virus infection**; 31 cases were male and 23 cases were female. Recorded ages were between the 10-14 and the 75-79 years age group. Forty-one per cent of the cases were from the Northern Territory. Onset dates were November (one), December (2), January (11), and February (40).
- A single case of **dengue** was reported for a female in the 25-29 years age group resident in the Statistical Division of Sydney.
- Two cases of **brucellosis** were reported; one case was male and one case was female. The cases were aged in the 45-54 years age group.
- There were 395 cases of **campylobacteriosis** reported; 210 cases were male, 183 were female and the sex of 2 cases was unrecorded. The cases were aged between the 0-4 and the 85-89 years age groups with 26% of cases aged less than 10 years.
- Eighty cases of **gonococcal infection** were reported; 53 cases were male, 25 cases were female, and the sex of 2 cases was unrecorded. The cases were aged between the 10-14 and the 70-74 years age groups with 73% of cases in the 15-29 years age group, for which most reports have been received since January 1994 (Figure 2).
- A single case of ***Haemophilus influenzae* type b infection** was reported for a male in the 0-4 years age group.
- There were 42 cases of **hepatitis A** reported; 21 cases were male and 21 cases were female. Recorded ages were between the 0-4 and the 45-49 years age groups.
- Fourteen incident cases of **hepatitis B** were reported; 7 cases were male and 7 were female. Recorded ages were between the 20-24 and the 70-74 years age groups.
- A single incident case of **hepatitis C** was reported for a female in the 30-34 years age group.
- A single case of **hydatid infection** was reported for a male in the 15-19 years age group resident in the Statistical Division of Melbourne.
- There were 3 cases of **legionellosis** reported. All cases were male and recorded ages were between the 30-34 and the 70-74 years age groups.
- A case of **leprosy** was reported for a female in the 0-4 years age group resident in rural Queensland.
- There were 5 cases of **leptospirosis** reported for males aged between the 25-29 years and the 70-74 years age groups.
- Two cases of **listeriosis** were reported; one case was male and one case was female. Recorded ages were in the 25-29 years age group and the 75-79 years age group.
- Five cases of **malaria** were reported; 3 cases were male and 2 cases were female. Recorded ages were between the 5-9 and the 35-39 years age groups. Onset dates were in January (one) and February (4).

Figure 2. Notifications of gonococcal infection, 1994 to 1995, by age group and sex



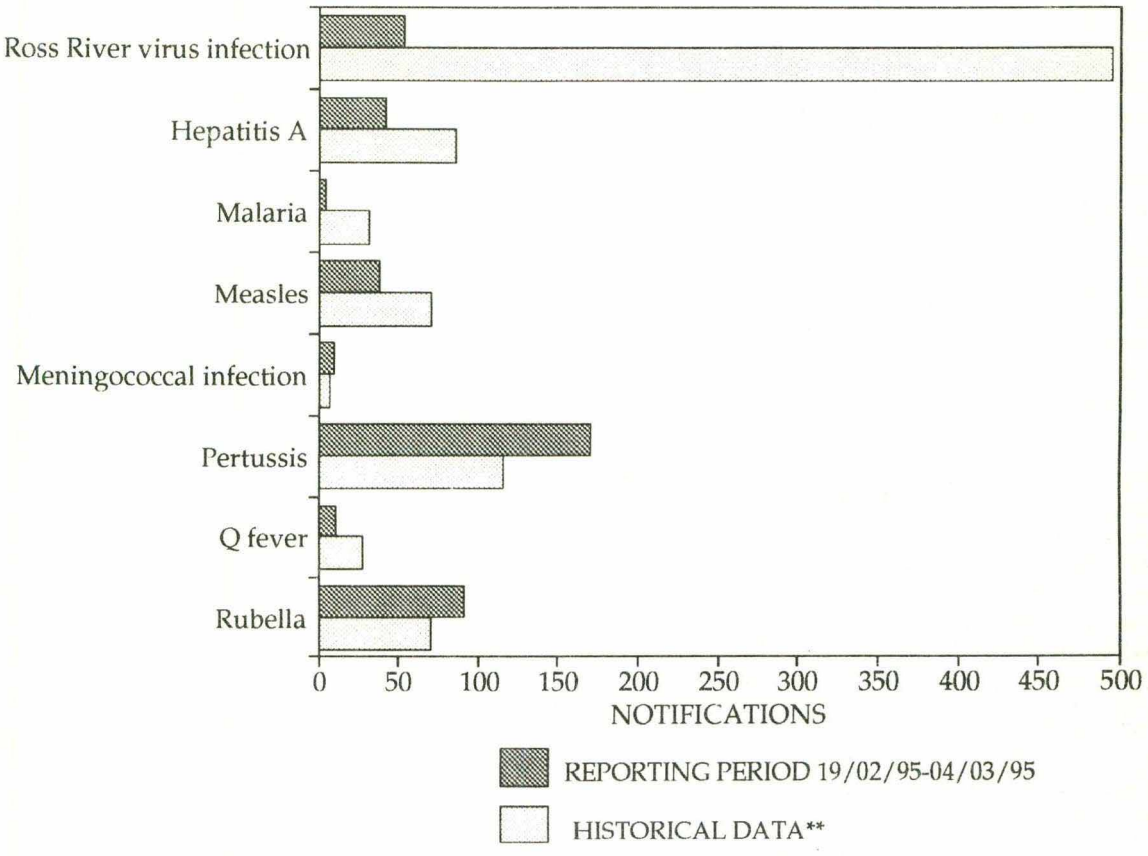
- The rate of reporting of **measles** continues to decline with 38 cases reported for the period (Figure 3). Twenty-two cases were male and 16 cases were female. Recorded ages were between the 0-4 and the 40-44 years age groups with 7 cases aged less than one year. There were 5 apparent clusters of between 2 and 3 cases each in the same postcode area. Apparent clusters were in the Northern Territory (one), New South Wales (2), the Australian Capital Territory (one), Victoria (one), and Queensland (one).
- There were 9 cases of **meningococcal infection** reported; 4 cases were male and 5 cases were female. Recorded ages were between the 0-4 and the 80-84 years age group with 4 cases in the 0-4 years age group. All recorded onset dates were in February. There were no apparent clusters.
- There were 170 cases of **pertussis** reported; 67 cases were male and 103 cases were female. Recorded ages were between the 0-4 and the 65-69 years age groups with 10 cases aged less than one year. There were 27 apparent clusters of between 2 and 4 cases each in the same postcode area. Clusters were in New South Wales (3), Victoria (one), Queensland (13), Western Australia (8), and Tasmania (2).
- Eleven notifications of **Q fever** were received; 10 cases were male and one case was female. Recorded ages were between the 0-4 and the 55-59 years age groups.
- There were 91 cases of **rubella** reported; 63 cases were male, 27 cases were female, and the sex of one case was unrecorded. The cases were aged between the 0-4 and the 65-69 years age groups with 11 cases reported for females in the 15-44 years age group.
- There were 347 cases of **salmonellosis** reported; 176 cases were male, 163 cases were female and the sex of 8 cases was unrecorded. The cases were aged between the 0-4 and the 85-89 years age group with 47% of cases in the 0-4 years age group.

Figure 3. Measles notifications per 100,000 population, January 1992 to February 1995,



- Forty-five notifications of **syphilis** were received; 27 cases were male, 15 cases were female, and the sex of 3 cases was unrecorded.
- There were 34 cases of **tuberculosis** reported; 19 cases were male, 14 cases were female, and the sex of one case was unrecorded. Cases were aged between the 5-9 and the 75-79 years age groups.
- There were 3 cases of **typhoid** reported; two cases were male and one case was female. Recorded ages were between the 15-19 and the 45-49 years age groups. Two cases were reported for residents of the Statistical Division of Sydney and one case was reported for a resident of the Australian Capital Territory.
- Sixteen cases of **yersiniosis** were reported; 8 cases were male and 8 cases were female. Recorded ages were between the 0-4 and the 70-74 years age groups.

Figure 4. Selected National Notifiable Diseases Surveillance System reports, and historical data[1]



1. The historical data are the averages of the number of notifications in 9 previous 2-week reporting periods: the corresponding periods of the last 3 years and the periods immediately preceding and following those.

Table 3. Notifications of diseases preventable by vaccines recommended by the NHMRC for routine childhood immunisation, received by State and Territory health authorities in the period 19 February to 4 March 1995

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[1] This period 1995	This period 1994	Year to date 1995	Year to date 1994
Diphtheria	0	0	0	0	0	0	0	0	0	4	1	5
Haemophilus influenzae b infection	0	1	0	0	0	0	0	0	1	5	12	39
Measles	2	12	2	12	0	1	9	0	38	110	376	746
Mumps	1	0	NN	NN	1	NN	0	2	4	1	11	5
Pertussis	1	20	20	64	2	9	19	35	170	218	938	1412
Poliomyelitis	0	0	0	0	0	0	0	0	0	0		
Rubella[2]	3	6	0	47	0	0	23	12	91	69	564	426
Tetanus	0	0	0	NN	0	0	0	0	0	0	1	1

1. Totals comprise data from all States and Territories. Cumulative figures are subject to retrospective revision, so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

2. Tas: CRS only.

NN Not Notifiable.

Table 4. Notifications of other diseases[1] received by State and Territory health authorities in the period 19 February to 4 March 1995

DISEASES	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	TOTALS FOR AUSTRALIA[2] This period 1995	This period 1994	Year to date 1995	Year to date 1994
Arbovirus infection												
Ross River virus infection	0	2	22	23	0	-	1	6	54	399	405	1386
Dengue	0	1	0	0	0	-	0	0	1	0	3	3
NEC[3]	0	3	1	13	0	0	0	0	17	29	97	112
Campylobacteriosis[4]	16	-	5	78	33	24	191	48	395	413	1834	1773
Chlamydial infection (NEC)[5]	4	NN	6	109	0	2	36	32	189	284	1055	1187
Donovanosis	0	NN	2	0	NN	NN	0	1	3	7	15	19
Gonococcal infection[6]	1	11	6	36	0	0	7	19	80	168	450	552
Hepatitis A	1	9	0	13	0	1	10	8	42	83	300	366
Hepatitis B incident	0	1	0	9	0	0	4	0	14	11	44	45
Hepatitis C incident	-	1	0	-	0	-	-	-	1	0	3	3
Hepatitis C unspecified	25			119		2	151	45	342	410	1449	1623
Hepatitis (NEC)	0	0	0	0	0	0	1	NN	1	4	10	15
Legionellosis	0	2	0	0	0	0	1	0	3	7	31	33
Leptospirosis	0	1	0	2	0	1	1	0	5	3	29	45
Listeriosis	0	1	0	0	0	0	1	0	2	1	15	7
Malaria	0	1	1	0	0	0	1	2	5	42	88	103
Meningococcal infection	0	2	0	3	0	0	2	2	9	8	58	55
Ornithosis	0	NN	0	1	0	0	0	1	2	2	29	22
Q fever	0	4	0	3	0	0	4	0	11	29	62	126
Salmonellosis (NEC)	6	70	11	132	15	11	70	32	347	354	1380	1328
Shigellosis[4]	0	-	2	5	0	0	3	9	19	34	154	140
Syphilis	1	11	5	15	0	0	12	1	45	114	298	419
Tuberculosis	0	6	0	6	6	0	15	1	34	48	141	206
Typhoid[7]	1	2	0	0	0	0	0	0	3	2	7	10
Yersiniosis (NEC)[4]	0	-	0	6	7	0	1	0	14	26	92	106

1. For HIV and AIDS, see Tables 2 and 3 *CDI* 1995;19:121. For rarely notified diseases, see Table 5.
2. 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.
3. Tas: includes Ross River virus and dengue.
4. NSW: only as 'foodborne disease' or 'gastroenteritis in an institution'.
5. WA: genital only.
6. NT, Qld, SA and Vic: includes gonococcal neonatal ophthalmia.
7. NSW, Vic: includes paratyphoid.

NN Not Notifiable.

NEC Not Elsewhere Classified.

\- Elsewhere Classified.

Table 5. Notifications of rare[1] diseases received by State and Territory health authorities in the period 19 February to 4 March 1995

DISEASES	Total this period	Reporting States or Territories	Year to date 1995
Botulism	0		0
Brucellosis	2	Qld 1, Vic 1	9
Chancroid	1	Vic	1
Cholera	0		0
Hydatid infection	1	Vic	3
Leprosy	1	Qld	2
Lymphogranuloma venereum	0		0
Plague	0		0
Rabies	0		0
Yellow fever	0		0
Other viral haemorrhagic fevers	0		0

1. Fewer than 50 cases of each of these diseases were notified each year during the period 1988 to 1993.

Table 6. Virology and serology laboratory reports by State or Territory[1] for the reporting period 23 February to 8 March 1995, historical data[2], and total reports for the year

	State or Territory [1]								Total this fortnight	Historical data [2]	Total reported this year
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA			
MEASLES, MUMPS, RUBELLA											
Measles virus		7	1	7			1	4	20	20.8	211
Mumps virus				2	1		2		5	1.3	20
Rubella virus		2		35	2	1		7	47	23.7	413
HEPATITIS VIRUSES											
Hepatitis A virus		4	1	11	2		4	5	27	21.7	130
Hepatitis B virus	2	22	5	62	1		13	42	147	95.7	543
Hepatitis C virus	20	38	15	112	50	38	2	108	383	154.3	1,533
Hepatitis E virus						1			1	.2	4
ARBOVIRUSES											
Ross River virus		3	37	35				9	84	128.0	322
Barmah Forest virus		2	1	16					19	12.8	65
Dengue not typed			1					1	2	1.5	5
Flavivirus (unspecified)							1		1	2.2	5
ADENOVIRUSES											
Adenovirus type 2					1				1	3.0	11
Adenovirus type 3							2		2	1.8	21
Adenovirus type 9							1		1	.0	1
Adenovirus type 46							1		1	.3	2
Adenovirus not typed/pending		9		4	12			9	34	42.7	239
HERPES VIRUSES											
Herpes simplex virus type 1		8	3	119	46	1	44	44	265	185.2	1,228
Herpes simplex virus type 2		9	1	146	35	6	35	56	288	183.5	1,145
Herpes simplex not typed/pending	7	11			1		2	10	31	26.8	128
Cytomegalovirus	1	21		22	3	2	25	10	84	57.2	353
Varicella-zoster virus	1	9		34			9	15	68	31.7	295
Epstein-Barr virus		16	3	75	23		6	10	133	66.2	566
Herpes virus group - not typed		1						1	2	1.0	6
OTHER DNA VIRUSES											
Parvovirus				1				12	13	2.8	49
PICORNA VIRUS FAMILY											
Coxsackievirus B3	1								1	.2	15
Echovirus type 3		1			1				2	.0	10
Echovirus type 6	1						4	1	6	.5	26
Echovirus type 9							1		1	3.2	1
Echovirus type 30		6						1	7	4.3	26
Poliovirus type 1 (uncharacterised)		1				1			2	1.5	6
Poliovirus type 3 (uncharacterised)		1							1	1.0	2
Rhinovirus (all types)		1		8				1	10	28.8	141
Enterovirus not typed/pending		4		11				7	22	33.5	199
ORTHO/PARAMYXOVIRUSES											
Influenza A virus		2			1			1	4	7.7	42
Influenza B virus					1		1		2	4.3	9
Parainfluenza virus type 1							1		1	3.3	3
Parainfluenza virus type 2					3				3	1.3	6
Parainfluenza virus type 3		2		7	1		4	3	17	9.7	156
Respiratory syncytial virus		5		5	1	1	1	5	18	14.5	100

Table 6. Virology and serology laboratory reports by State or Territory[1] for the reporting period 23 February to 8 March 1995, historical data[2], and total reports for the year, continued

	State or Territory [1]								Total this fortnight	Historical data [2]	Total reported this year
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA			
OTHER RNA VIRUSES											
HIV-1				2					2	2.2	18
HTLV-1								1	1	.3	2
Rotavirus	2	13			11		1	13	40	25.2	244
OTHER											
Chlamydia trachomatis not typed	3	9	9	76	14	4	8	24	147	127.2	615
Chlamydia psittaci							6	1	7	2.3	51
Chlamydia spp typing pending				1					1	.7	3
Chlamydia species					12				12	.3	18
Mycoplasma pneumoniae		1	1	14	4		1		21	59.3	91
Coxiella burnetii (Q fever)		2			1		2	1	6	14.3	76
Streptococcus group A		2	3	21					26	9.3	104
Salmonella Typhi		1							1	.0	1
Brucella species				1					1	.0	4
Bordetella pertussis	1	2	4			1	1	85	94	15.2	220
Bordetella species			1	11					12	8.7	48
Legionella species								2	2	.2	20
Cryptococcus species		1							1	.3	8
Treponema pallidum	1	20	2	5				7	35	16.0	172
Toxoplasma gondii		5		1		1			7	1.5	26
Echinococcus granulosus							1		1	.3	5
TOTAL	40	243	88	844	229	57	180	499	2,180	1,461.5	9,770

1. State or Territory of postcode, if reported, otherwise State or Territory of reporting laboratory.
2. The historical data are the averages of the numbers of reports in 6 previous 2 week reporting periods: the corresponding periods of the last 2 years and the periods immediately preceding and following those.

Table 7. Virology and serology laboratory reports by clinical information for the reporting period 23 February to 8 March 1995

	Encephalitis	Meningitis	Other CNS	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	Total
MEASLES, MUMPS, RUBELLA													
Measles virus		1						5				14	20
Mumps virus												5	5
Rubella virus					1			17				29	47
HEPATITIS VIRUSES													
Hepatitis A virus							10					17	27
Hepatitis B virus							40					107	147
Hepatitis C virus				1			34					348	383
Hepatitis E virus												1	1
ARBOVIRUSES													
Ross River virus						1	1	5		27		50	84
Barmah Forest virus			1					1		3		14	19
Dengue not typed										1		1	2
Flavivirus (unspecified)												1	1
ADENOVIRUSES													
Adenovirus type 2					1								1
Adenovirus type 3									2				2
Adenovirus type 9						1							1
Adenovirus type 46								1					1
Adenovirus not typed/pending					6	18		1	1			8	34
HERPES VIRUSES													
Herpes simplex virus type 1					7			168	3		83	4	265
Herpes simplex virus type 2					1			76			204	7	288
Herpes simplex not typed/pending	1							13	2		5	10	31
Cytomegalovirus				4	18	2	2	1	2	2	1	52	84
Varicella-zoster virus		1	1					53	2	1		10	68
Epstein-Barr virus		1			5					2		125	133
Herpes virus group - not typed								2					2
OTHER DNA VIRUSES													
Parvovirus								4		1		8	13
PICORNA VIRUS FAMILY													
Coxsackievirus B3												1	1
Echovirus type 3					1							1	2
Echovirus type 6		5						1					6
Echovirus type 9									1				1
Echovirus type 30		4			1							2	7
Poliovirus type 1 (uncharacterised)					1	1							2
Poliovirus type 3 (uncharacterised)												1	1
Rhinovirus (all types)					9							1	10
Enterovirus not typed/pending					8	5						9	22

Table 7. Virology and serology laboratory reports by clinical information for the reporting period 23 February to 8 March 1995, continued

	Encephalitis	Meningitis	Other CNSI	Congenital	Respiratory	Gastrointestinal	Hepatic	Skin	Eye	Muscle/joint	Genital	Other/unknown	Total
ORTHO/PARAMYXOVIRUSES													
Influenza A virus					2							2	4
Influenza B virus					1							1	2
Parainfluenza virus type 1					1								1
Parainfluenza virus type 2					3								3
Parainfluenza virus type 3					15			1				1	17
Respiratory syncytial virus					17							1	18
OTHER RNA VIRUSES													
HIV-1												2	2
HTLV-1												1	1
Rotavirus					1	39							40
OTHER													
Chlamydia trachomatis not typed									1		111	35	147
Chlamydia psittaci					4							3	7
Chlamydia spp typing pending												1	1
Chlamydia species					1						11		12
Mycoplasma pneumoniae					9					1		11	21
Coxiella burnetii (Q fever)							1					5	6
Streptococcus group A					1	1		1		4		19	26
Salmonella Typhi												1	1
Brucella species												1	1
Bordetella pertussis					54							40	94
Bordetella species					7							5	12
Legionella species					2								2
FUNGI													
Cryptococcus species												1	1
Treponema pallidum											5	30	35
Toxoplasma gondii										1		6	7
Echinococcus granulosus												1	1
TOTAL	1	12	2	5	178	68	88	350	14	45	421	996	2180

Table 8. Virology and serology laboratory reports by contributing laboratories for the reporting period 23 February to 8 March 1995

STATE OR TERRITORY	LABORATORY	REPORTS
Australian Capital Territory	Woden Valley Hospital, Canberra	44
New South Wales	Institute of Clinical Pathology & Medical Research, Westmead	18
	Prince Henry/Prince of Wales Hospitals, Sydney	46
	Royal Alexandra Hospital for Children, Camperdown	18
	Royal Prince Alfred Hospital, Camperdown	32
	South West Area Pathology Service, Liverpool	63
Queensland	Queensland Medical Laboratory, West End	861
	State Health Laboratory, Brisbane	116
South Australia	Institute of Medical and Veterinary Science, Adelaide	229
Tasmania	Northern Tasmanian Pathology Service, Launceston	4
	Royal Hobart Hospital, Hobart	49
Victoria	Microbiological Diagnostic Unit, University of Melbourne	8
	Monash Medical Centre, Melbourne	16
	Victorian Infectious Diseases Reference Laboratory, Fairfield Hospital	158
Western Australia	Princess Margaret Hospital, Perth	96
	State Health Laboratory Services, Perth	422
TOTAL		2180