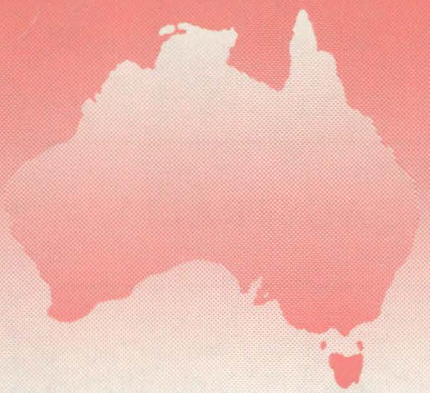

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CONTENTS

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DEPARTMENT OF HEALTH, HOUSING AND COMMUNITY SERVICES

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

OUTBREAK OF NON-SEXUALLY TRANSMITTED GONOCOCCAL CONJUNCTIVITIS IN CENTRAL AUSTRALIA, 31 JANUARY TO 6 JUNE 1991

(A Merianos[1], G Mulvey[2], S Jayathissa[1], J Stewart[1], P Linehan[3] and R Matters[1])

Summary

From 31 January to 6 June 1991 251 cases of gonococcal conjunctivitis were reported to the Communicable Disease Control Centre in Alice Springs. All but one case occurred in Aboriginal people. Most cases have been reported from an area extending in a circle of 400km radius centred on Alice Springs. One hundred and twenty-two cases were from the Alice Springs and Barkly Tablelands region of the Northern Territory, 118 were diagnosed among the communities of the Pitjantjatjara Lands in northern South Australia and 9 cases were from Western Australian communities near the NT border. Two further cases were reported from Oodnadatta and Coober Pedy in SA.

This is the third outbreak of non-sexually transmitted gonococcal conjunctivitis in Central Australia since 1981. The outbreaks have occurred at approximately 5 year intervals.

Subjects and Methods

The Central Australian Aboriginal population is divided into several language groups living in an area of approximately 800,000 square kilometres, and is highly mobile between related communities. The Pitjantjatjara people in South Australia have traditional ties and sociological homogeneity with their relatives in the Northern Territory.

Case definition

We have accepted a clinical case definition of acute gonococcal conjunctivitis in communities with at least one microscopy- and/or culture-proven case.

Investigations

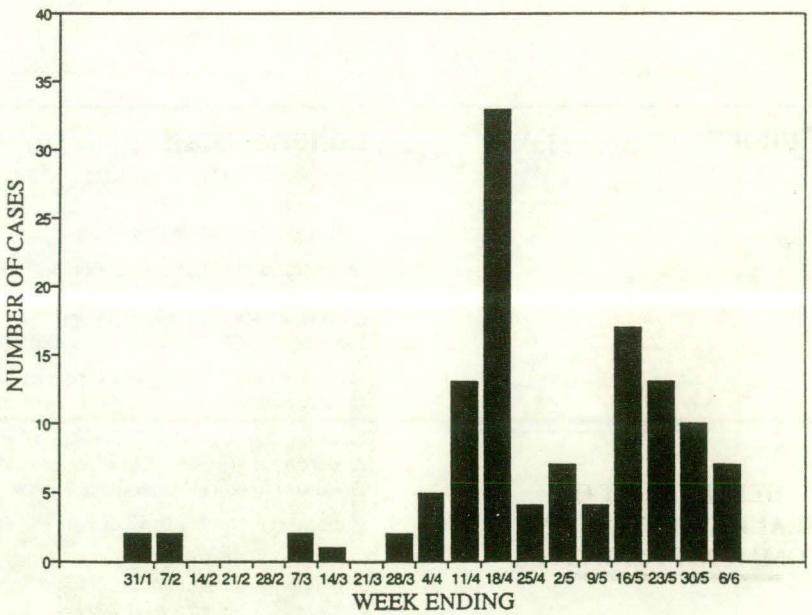
Conjunctival swabs for gram stain and culture were collected from 220 cases. Swabs for culture were placed in either Stuart's or charcoal media for transportation to the regional laboratories in Alice Springs, but delays of up to 5 days occurred between collection and processing. Samples of conjunctival and genital gonococcal isolates have been forwarded to the Gonococcal Reference Laboratory in Sydney for serotyping, auxotyping and determination of penicillin MICs.

Screening of household contacts of known cases was undertaken in order to determine the role, if any, of subclinical and mild clinical disease in transmission.

Results

Cases occurred sporadically throughout the region from January 1991 until the first week of April when 27 patients were diagnosed. The epidemic curve peaked in the week ending 18 April 1991 in both the NT (Figure 1) and the Pitjantjatjara Lands (Figure 2). (Active case finding had been undertaken during that week.) Three communities reported sudden increases in patient numbers within 2 weeks of festivals attended by symptomatic members of related communities. Case numbers have consistently fallen in SA, but a second smaller peak occurred in the week ending 16 May 1991 in the NT, reflecting disease transmission to previously unaffected communities. The crude attack rates were 10.4/1000 and 60.9/1000 for the Central Australian Aboriginal population and the Pitjantjatjara Lands communities respectively. (The NT denominators are based on 1986 census data (n=11,698) and the SA denominators are based on Nganampa Health Council population files (n=1,939).)

Figure 1. Epidemic curve of non-sexually transmitted gonococcal conjunctivitis in NT



1. NT Department of Health and Community Services;
2. Nganampa Health Services, Pitjantjatjara Lands, SA;
3. Western Diagnostic Pathology, Alice Springs

Table. Age-specific attack rates of gonococcal conjunctivitis, Central Australia, 31 January-6 June 1991 (n=240*)

Age goup (yrs)	Cases	% of cases	Attack Rate /1000	Relative Risk (RR)**	95% CI of RR
0-4	105	43.7	55.8	31.5	17.4<RR<51.1
5-9	60	25.0	35.7	19.1	10.9<RR<33.5
10-14	31	12.9	15.8	8.5	4.6<RR<15.7
15 & over	15	6.3	1.9	1.0	
Unknown	29	12.1			
Total	**240**	**100.0**			

* Only NT and Pitjantjatjara Lands cases included

** Relative risk calculated using age 15 and over as referent

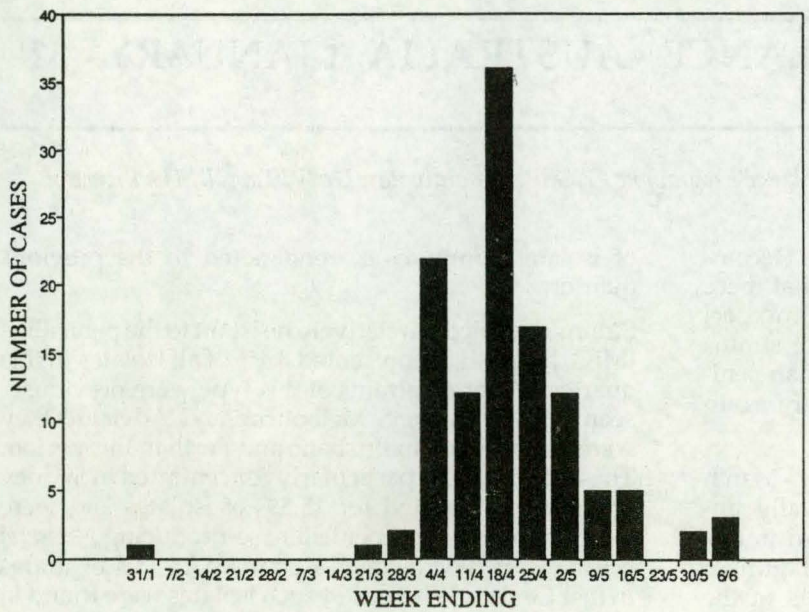
The Table shows the age-specific attack rates for the NT and the Pitjantjatjara Lands combined. The highest attack rates occurred in children aged 0-4 years in both areas, and there was a statistically significant age-related trend in attack rates (linear trend test $p<0.001$). This finding is consistent with the age distribution of cases during the 1987 outbreak[1]. Although ages were not available for 12.1% of cases, clinic staff have stated that most were children less than 10 years of age. There was no gender-related difference in attack rates.

Most children presented with acute, usually bilateral, purulent conjunctivitis and periorbital cellulitis. The important sequelae which have been associated with gonococcal ophthalmia neonatorum and genitally acquired gonococcal ophthalmitis in adults (corneal scarring, ulceration and prolapse, and pan-ophthalmitis) did not occur. The only complications reported were disseminated gonococcal infection with arthritis in a 3 year old child, and transient large joint arthralgia in 4 other children.

Both microscopy and culture were performed on conjunctival specimens collected from 220 patients. The air-dried smears were positive for intracellular gram-negative diplococci in 208 cases (94.5%), and *Neisseria gonorrhoeae* was cultured in 90 cases (40.9%). In only two cases where the microscopy findings were negative did the swab yield a positive culture. All isolates have been sensitive to penicillin by disc diffusion testing.

Multiple cases within affected households was a feature of this outbreak. In a review of 10 affected families (93 people), there were 7 secondary and one co-primary cases (9.6%). All but one secondary case occurred among children aged 0-9 years. In contrast, screening of asymptomatic community contacts in 3 communities (171 people) only yielded one additional case of gonococcal conjunctivitis (0.6%).

Figure 2. Epidemic curve of non-sexually transmitted gonococcal conjunctivitis in the Pitjantjatjara Lands, SA



Control measures

Control measures included early detection through active case finding and prompt treatment. Single dose treatment with either procaine penicillin or oral amoxycillin with probenecid was recommended as standard treatment, as it proved effective in the 1987 outbreak. Community education was aimed at encouraging early presentation of patients and discussion of the role of hygiene in transmission. Cross-border networking facilitated the adoption of a standard treatment regimen and improved awareness of the outbreak.

Comment

Epidemic gonococcal conjunctivitis[1,2] appears to differ from both gonococcal ophthalmia neonatorum and adult gonococcal eye infections associated with

anogenital gonorrhoea, in severity, the development of sight-threatening sequelae and in response to treatment[3,4]. The reasons for this are unclear, but it is hoped that typing of genital and conjunctival isolates from this outbreak will help to clarify this question.

Effective single dose treatment has proven acceptable to the Aboriginal communities in Central Australia, and reduces the logistical problems of follow-up for multidose treatment in a highly mobile population. At present, early case detection and effective standardised treatment are the easiest interventions for control. We are collecting data on treatment regimens used, and the incidence of recrudescence and re-infection.

This outbreak also highlights the importance of the air-dried smear in the diagnosis of gonococcal infections in remote communities where transportation of specimens to the regional laboratories is often delayed, and culture is unsuccessful.

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CDI EDITORIAL COMMENT

Gonococcal conjunctivitis is not notified separately from other gonococcal diseases, so little data are available on its overall incidence in Australia. (The exception is gonococcal conjunctivitis acquired by babies specifically from their mother's infected birth canal (gonococcal ophthalmia neonatorum). This disease is notifiable in New South Wales, Queensland, South Australia, Western Australia and the Northern Territory. No cases were reported for 1990 and no more than 5 cases have been reported each year since 1986.)

Further details on the Western Australian gonococcal conjunctivitis epidemic were published recently in the *Western Australian Notifiable Diseases Bulletin*. That report is reproduced, with acknowledgment, as follows:

Aboriginal communities in the Ngaanyatjarra Homelands (far north eastern Goldfields region, near the 3-state WA/SA/NT border) have been experiencing an outbreak of acute purulent conjunctivitis since April. The outbreak began in the Northern Territory in March and then spread to the adjacent areas of Western Australia and South Australia.

The causative organism is a penicillin-sensitive strain of *Neisseria gonorrhoeae.*

June notifications for gonorrhoea include 19 cases of gonococcal eye infection, mainly in young children in small communities near the border. The attack rate in children under 5 living in one of these communities is 36.4%. This brings to 35 the cumulative number of cases diagnosed in the district since April.

The reservoir of infection is believed to be an untreated pool of genital gonorrhoea among sexually active adults. The mechanism of transmission is uncertain, but flies and poor personal hygiene are thought to play important roles. Laboratory tests are under way to compare the prevalent genital, pharyngeal and conjunctival strains of *N.gonorrhoeae.*

GONOCOCCAL SURVEILLANCE - AUSTRALIA, 1 JANUARY - 31 MARCH 1991

(Contributed by the Australian Gonococcal Surveillance Programme - AGSP. Co-ordinator, Dr JW Tapsall, The Prince of Wales Hospital, Sydney, NSW 2031)

In the last report of the AGSP for the October - December quarter 1990, (*CDI* **15**:143) it was noted that there had been a decline in the total number of gonococci isolated and an increase in the proportion of strains resistant to penicillins by mechanisms other than penicillinase production. Both of these trends are again evident in the January-March quarter of 1991.

The number of strains examined in the January - March quarter in the years 1988 - 1990 was virtually unchanged. However, the 407 isolates examined in this period in 1991 represent only 75% of the total number of strains seen in the corresponding periods in the previous three years and this reduction in the number of isolates continues a trend noted in the previous quarter.

Strains classified as relatively resistant to the penicillins (MIC 1.0 mg/L) represented 8.4% of all isolates in this quarter. Whereas strains of this type were previously seen only in Sydney, Melbourne and Adelaide they were also isolated in Brisbane and Perth in this period. These strains were particularly concentrated in Sydney were they accounted for 15.5% of isolates and were more numerous that penicillinase-producing *Neisseria gonorrhoeae* (PPNG) which accounted for 11% of strains in that Centre. Whereas 24 such isolates were found in Sydney only small numbers were present in the other

Table. Penicillin sensitivity of isolates of *Neisseria gonorrhoeae* 1 January - 31 March 1991*

CENTRE	PERCENTAGE OF ISOLATES					
	SENSITIVE		LESS SENSITIVE		PPNG	
Brisbane	13	(17.1)	49	(52)	14	(17.1)
Sydney	15	(3.8)	48	(58)	11	(16.6)
Melbourne	8	(6.9)	48	(59.7)	28	(16.4)

* Sensitive, MIC = 0.004 - 0.016 mg/L

Less Sensitive, MIC = 0.06 - 0.25 mg/L

PPNG = Penicillinase-producing *N. gonorrhoeae*

Figures in parentheses repesent data for the same period in 1990

centres. Additional information on levels of intrinsic resistance in those three centres with large numbers of isolates is contained in the Table.

The number of infections with PPNG in Australia in this quarter was 64 or 15.7% of all gonococcal infections. However, the distribution of cases was not uniform, with 28% of isolates in Melbourne being PPNG and lower percentages in other centres. The 11% PPNG rate in Sydney is the lowest seen in that centre for a considerable time, but overall, resistance to the penicillins remains high given the large number of chromosomally resistant strains referred to earlier. Data on the geographic site of acquisition was available for only 44 of the 64 PPNG. Strains acquired through contact in Australia were isolated in Adelaide, Brisbane, Sydney, Melbourne and Hobart although in some of these cases the immediate contact had acquired the infection outside Australia. Imported infections were acquired mainly in Thailand and the Philippines, other sources of acquisition being Bali, Malaysia and Vietnam. For the third period in succession imported cases of PPNG outnumbered locally acquired cases, this trend being observed in all centres except Sydney where endemic transmission of PPNG still occurs at a significant level.

TUBERCULOSIS BRIEFS 1 - NOTIFICATION RATES

Introduction

This is the first of a series of reports on tuberculosis (TB) in Australia, based on data collected recently from all States and Territories.

National data on the epidemiological aspects of tuberculosis have not been published since 1985.

The rate of notification of tuberculosis in Australia fell, from 46.8 per 100,000 in 1948 to 7.0 per 100,000 in 1985[1], the year that national data on tuberculosis were last published. Those data consisted of several separate categories, including new cases of tuberculosis, atypical tuberculosis and relapses (reactivations). Notification rates reported in the past consisted of "all forms" of tuberculosis including new cases plus atypical cases, but excluded relapses. This report on the notification rates, post 1985, differentiates between the different categories of notifiable cases.

Definitions used

In this analysis, the definitions used are:

1. Tuberculosis (New Cases)

- a case which has been confirmed by the identification of *Mycobacterium tuberculosis* culture or by microscopy
- infectious agents are usually from the Mycobacterium Tuberculosis Complex - *M. tuberculosis, M. africanum* and *M. bovis* (not including BCG - bovis).
- a case of TB which has been diagnosed to be active clinically and which has been accepted, as such, by the State or Territory Director of TB.

2. Relapse (Reactivation)

A case of active tuberculosis diagnosed again (bacteriologically, radiologically or clinically) following previous full treatment (as deemed appropriate by the Director of TB) and considered to be inactive or quiescent.

3. 'Atypical' Mycobacterial Infection

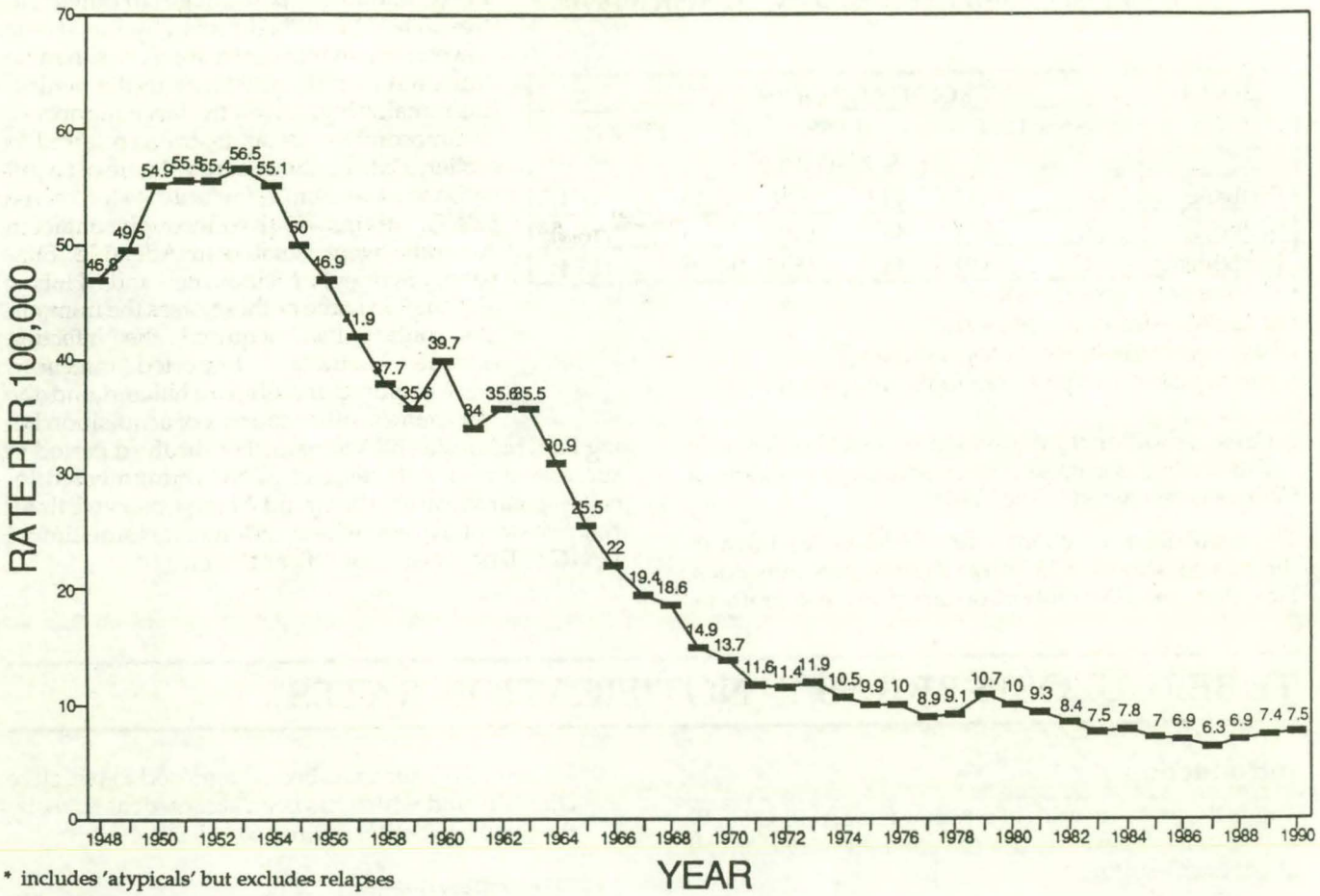
An active case of 'atypical' mycobacterial disease is one when there are clinical features consistent with one or more of the following:

- presence of compatible disease process which is clinically, radiologically and/or pathologically not due to other causes
- consistent repeated recovery of the same organism from the same site in moderate to abundant amounts
- recovery of 'atypical' mycobacterium from sites which are normally sterile.

4. Incidence Rate

The number of new cases of tuberculosis diagnosed or reported during a defined period of time (usually one year), divided by the number of persons in the stated population.

Figure 1. Tuberculosis rates in Australia 1948-90: notifications for all forms*



* includes 'atypicals' but excludes relapses
1989 and 1990 data exclude NSW

5. Population

The number of persons living in an area at mid year. This information is supplied by the Australian Bureau of Statistics.

6. Tuberculosis Deaths

Deaths from tuberculosis (all forms including relapses) reported by a clinician during the year, including deaths due to, and incidental to the disease.

Results

The rate of notification of all forms of TB excluding relapses has fallen from a peak of 56.5 per 100,000 in 1953 and has been below 10 per 100,000 since 1980 (Figure 1). Since 1987 there has been a slight upward trend from 6.3 to 7.5 per 100,000. For 1989 and 1990 NSW data are unavailable and denominators have been adjusted accordingly. The rate of notified new cases of TB has been fairly constant at between 5.71 and 5.34 per 100,000 over the last five years but there has been an upward trend in 'atypical' disease over the same period (Figure 2). The infected population (comprising new cases and relapses) has remained fairly constant between 5.59 and 5.89 per 100,000 since 1986, and the crude death rate has increased from 0.32 to 0.53 per 100,000 over that period (Figure 3). Numbers of new cases, 'atypical' cases new and relapsing cases and deaths since 1986 are shown in the Table.

Discussion

The incidence of notified new tuberculosis cases in Australia has not increased in recent years. This is in contrast with the United States, which has had an in-

Table. New cases, atypicals, new cases + relapses and deaths, 1986-1990*

	1986	1987	1988	1989	1990
NEW CASES	899	870	922	591	633
'ATYPICALS'	212	163	218	228	209
NEW+RELAP	944	909	951	626	663
DEATHS	51	68	60	35	35

* 1989 and 1990 data excludes NSW

Figure 2. Tuberculosis rates in Australia 1980-1990: new cases and 'atypicals'

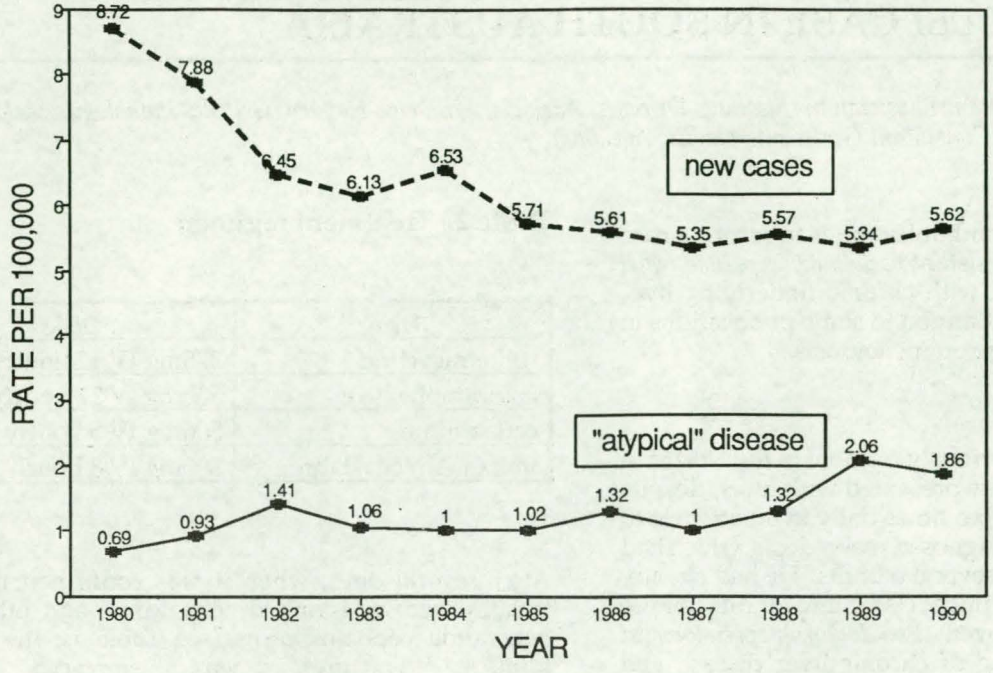
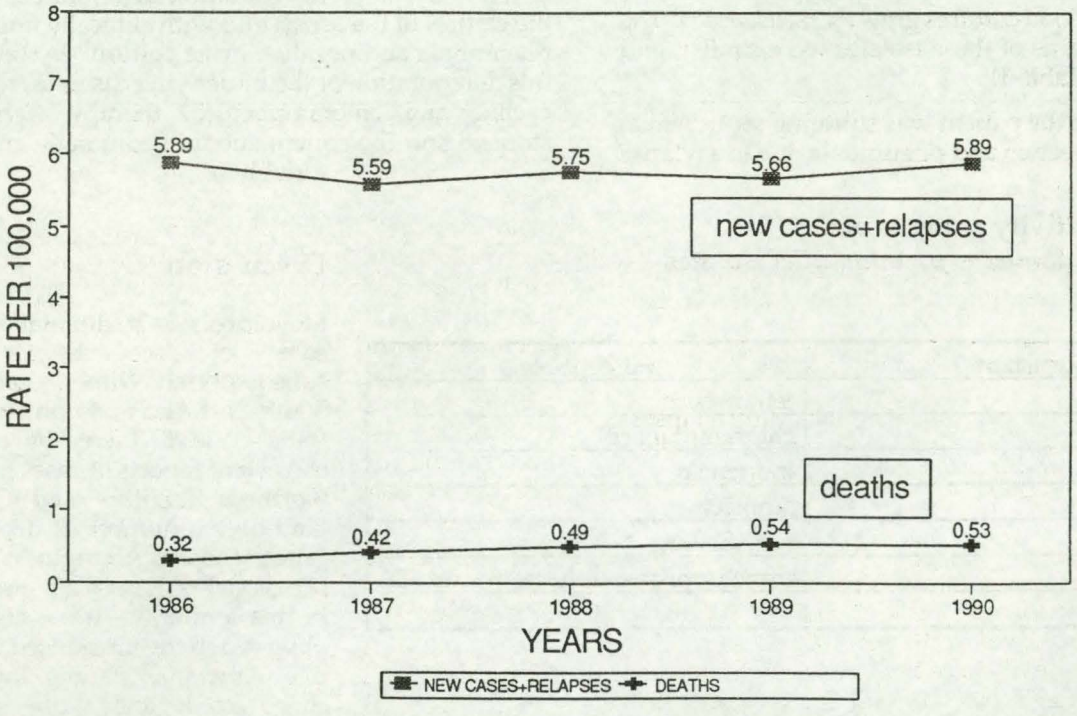


Figure 3. Tuberculosis rates in Australia 1986-90: new cases & relapses, and deaths



creasing rate since 1986, attributed to HIV disease[2]. In Australia, closer analysis of the data may be necessary to detect any relationship between the HIV epidemic and TB notifications. The rate of reported atypical disease has increased since 1985.

Future reports on tuberculosis in *CDI* will include analysis of 'atypical' disease, patterns of disease, age group and country of birth of reported cases.

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MULTIPLE ANTIBIOTIC RESISTANT *PSEUDOMONAS PSEUDOMALLEI* CASE IN SOUTH AUSTRALIA

(Dr Ross Philpot, Consultant Physician in Infectious Diseases, Adelaide; Mr Denis Hayden, Gribbles Pathology, Adelaide; and Dr Geoffrey Gibson, Consultant Gastroenterologist, Adelaide)

Introduction

The purpose of this communication is to record a case of multiple antibiotic resistant *Pseudomonas pseudomallei* (MARPP) in a patient with chronic underlying liver disease, and to draw attention to some publications in the Australian literature on melioidosis.

Case Report

A 64 year old man, previously resident in the Northern Territory for many years presented with fever, despite taking 'Augmentin' three times daily in an attempt to suppress previously diagnosed melioidosis, which had been symptomatic for several months. He had already required intensive treatment elsewhere for one relapse involving culture-proven *Pseudomonas pseudomallei* peritonitis, in a setting of chronic liver disease and chronic renal failure.

He was found to have Xray evidence of pneumonia, and urine and blood cultures grew *Ps. pseudomallei*. The sensitivity patterns of these isolates were similar, and are detailed in Table 1.

It was clear that the patient was suffering septicaemia, urinary tract infection and pneumonia due to a relapse of *Pseudomonas pseudomallei* infection, despite the attempted suppressive therapy with 'Augmentin'. It was therefore decided to employ triple intravenous antimicrobial therapy as a life-saving measure. He was started on a regimen of ceftazidime, rolitetracycline and chloramphenicol, as detailed in Table 2.

Table 1. Sensitivity patterns of the *Pseudomonas pseudomallei* isolates

Resistant	Susceptible
ticarcillin	tetracycline
piperacillin	chloramphenicol
gentamicin	imipenem
cefoxitin	amikacin
ceftazidime	piperacillin
ampicillin	trimethoprim-sulphamethoxazole
Augmentin	

Within two days, his temperature began to resolve, and he improved symptomatically. Within several days, his temperature had returned to below 37°C, and remained so for over a week.

Table 2. Treatment regimen

Drug	Dose
rolitetracycline	275mg IV 12 hourly
chloramphenicol	500mg IV 12 hourly
ceftazidime	500mg IV 8 hourly
imipenem/cilastatin	500mg IV 8 hourly

After several days, when it was confirmed that the isolates were resistant to ceftazidime and other late generation cephalosporins (see Table 1), the ceftazidime ('Fortum') was replaced with imipenem/cilastatin ('Primaxin').

Several days later, his condition deteriorated, despite the control of the sepsis and with clinically improving pneumonia and negative urine culture. In the face of this deterioration of the underlying diseases rolitetracycline and chloramphenicol therapy were both stopped and the patient succumbed quietly and comfortably.

Discussion

Melioidosis is traditionally a disease of the tropics and subequatorial climes, including South-East Asia and northern Australia[1-6] and there have been consistent reports of cases from the Northern Territory and Queensland over a number of decades in caucasians and Aborigines[7-11] with occasional reports from elsewhere in this country[12]. It has also been observed in the introduced and native Australian fauna, including sheep, cattle and swine, a tree-climbing kangaroo, and a galah[13-16]. An alarmingly high proportion of cases in Darwin last year proved fatal[1] (Bart Currie and Val Asche, personal communications), as was the outcome in our report. This is despite the introduction in recent years of newer generation cephalosporins added to the more traditional regimens of trimethoprim-sulphamethoxazole, and tetracycline therapy, supplemented in some cases with chloramphenicol. Use of the newer generation cephalosporins has been based on encouraging results of in vitro susceptibility

testing of a wide range of antimicrobial agents against this organism 17-23. Currently the Royal Darwin Hospital is using ceftazidime as primary therapy (Asche, personal communication). Yet the emergence of resistance to the later-generation cephalosporins *in vitro* and during treatment has been documented[23] and bodes ill for the clinical management of future patients afflicted with this pathogen.

Conclusions

We present this case to warn that:

1. resistance to newer generation cephalosporins may develop during the course of treatment of infections involving *Pseudomonas pseudomallei*, resulting in the appearance of multiply antibiotic resistant *Pseudomonas pseudomallei* (MARPP);
2. this will require alteration to chemotherapy, which is a particular concern in patients who are either (a) intolerant of one of the otherwise relevant antibiotics, as in our patient, or (b) when the patient is generally very ill, as also was the situation;
3. despite apparently adequate antibiotic combination chemotherapy and control of sepsis, a fatal outcome can still occur because of (a) the severity of the underlying disease or diseases, plus (b) complications of diverse types that may arise during the course of a patient's management.

We therefore recommend that:

1. patients should be carefully monitored for the emergence of antibiotic resistant strains during treatment of melioidosis, as recommended by Dance *et al* [23];
2. patients should be given the benefit of aggressive combination chemotherapy to control their sepsis and to discourage the emergence of resistant strains;
3. control must be kept on the patient's underlying disease(s), and a close watch maintained for development of any complications.

Acknowledgments

It is a pleasure to acknowledge the assistance of staff of Gribbles Pathology Microbiology Department in the work done on the isolates, the experience of Drs Bart Currie and Val Asche in providing information regarding antimicrobial treatment; Dr D Worthley and the staff of the private hospital in which the patient was cared for during this final episode of his long illness. Isolates have been sent to a reference laboratory for detailed sensitivity testing, and it hoped that results will be available for publication as an addendum to this report.

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CDI EDITORIAL COMMENT

The melioidosis case reported above is one of an increased number of cases of this disease diagnosed in northern Australia in the last year. In the Northern Territory, since the beginning of the 1990-91 heavy 'wet season' in November 1990, there have been 32 cases of melioidosis diagnosed at Royal Darwin Hospital. Most, but not all, have had risk factors such as diabetes or alcoholism. Sixteen were septicaemic on admission, and 12 patients (including the one in the above report) have died. A similar increase in diagnosed cases has occurred in north Queensland (Ashdown, *CDI*, this issue).

After initial intensive therapy with appropriate IV antibiotics, the continuation of maintenance therapy for two or more months needs emphasis to prevent relapse. Of the 4 relapses (3 patients) in the Darwin patients, 3 have been in patients not compliant with maintenance therapy. The fourth relapse is the case above, and this failure of amoxycillin/clavulanic acid maintenance therapy has been seen previously at Royal Darwin Hospital (RDH). Cotrimoxazole or doxycycline are now used for the maintenance therapy at RDH, and no failures have occurred in compliant patients to date. Resistance such as in the case described is occasionally seen, usually in patients with previous melioidosis not fully treated. Surveillance for resistance is therefore important, although for the majority of cases standard melioidosis protocols are adequate.

EPIDEMIOLOGICAL ASPECTS OF MELIOIDOSIS IN AUSTRALIA

(Les Ashdown, PhD, FASM, FATCM, Consultant Microbiologist, Department of Pathology, Townsville General Hospital)

By 4 July of this year, 28 cases of active infection with *Pseudomonas pseudomallei*, all from northern Queensland, had been notified to the Queensland Department of Health compared to 8, 10, 1 and 2 cases in the first six months of 1990, 1989, 1988 and 1987 respectively[1]. The increase in incidence of reported cases of melioidosis after 1988 was most probably due to an amendment to the Health Act following Queensland government legislation in 1988 which required pathology laboratories to notify the Department of their finding of any notifiable disease in the State of Queensland.

The marked increase in incidence of disease in the first six months of this year is most probably due to the much heavier rainfall which occurred during the last wet season in northern Australia, compared to the wet seasons of the previous four years or so, and resulted in a more widespread distribution and larger numbers of the bacterium in the soil, as well as prolonged contaminated surface water. Consequently, the chances of exposure to soil or surface water contaminated with *Ps. pseudomallei*, and to a heavier inoculating dose of the bacterium, and subsequent infection, would have been considerably greater.

The bacterium is a natural resident of soil in certain tropical regions, presumably with an ecological niche similar to *P. cepacia*. With respect to human and animal infection, the bacterium is, in most cases, an opportunist, and, following exposure to the bacterium, infection results because of impaired host defences, the virulence of the strain, a heavy inoculating dose or a combination of these factors[2]. The preponderance of clinical cases of melioidosis during the tropical wet seasons is well documented in northern Australia and in South-East Asia[2,3]. During the dry-season, the bacterium recedes with the surface water as it drains below the surface of the soil; in sandy soils, the organism dissipates well and may be deep in the subsoil, but in soils with high clay content, the bacterium in attached to the clay with the water by hydrostatic attraction, and may reside close to the surface of the soil. When the wet-season commences, the bacterium is brought to the surface of the soil by the water where exposure may subsequently occur[4]. The organism is found most commonly in soil and surface water that receives filtered light rather direct sunlight, and the numbers of the bacterium in the soil or surface water is directly related to the rainfall, temperature and humidity[2, 4]. Although the tropics (north of Rockhampton, Longreach, Alice Springs, Newman) is the main endemic area of Australia, sporadic autochthonous infections in animals have occurred in the Burnett River Valley[5], the Brisbane Valley[6] and in southwest Western Australia[7] and in a 47 year-old man in southwest Western Australia[8].

Melioidosis is a saprozoonose and, as such, presents few possibilities for control and, unlike the control of spread of glanders, a nonendemic, zoonotic disease with similar clinical features but with quite different epidemiological characteristics, major internal and international preventative and control measures have not been adopted. An imported ram may have been responsible for the disease outbreak in Aruba[9], and imported animals may have introduced the organism into Iran[10], while imported non-human primates are known carriers of the infection. Endemic regions were established in France probably following the importa-

tion from Iran of infected horses which subsequently contaminated stables[11], and their experiences exemplify the ease with which the organism spread from one zone to another and demonstrate that the bacterium can exist quite readily in conducive environments in temperate climates.

The extent to which decaying carcases and contaminated faeces, urine, sputum and exodate contribute to the maintenance of the organism in the soil is unknown. In one report, recycling of the organism from infected foot lesions onto the soil provided a continual source of contamination[4]. Also, there is significant movement of individuals and animals to and from endemic areas in Australia. Darwin, Cairns, Townsville and Rockhampton are well visited tourist destinations as well as being cities with large transient populations, and many persons return to temperate Australia, sometimes after a considerable sojourn in these centres. Some 5-10% of these individuals will have been exposed to *Ps. pseudomallei*[12], and some may present later in temperate climates with recrudescent clinical disease or may inadvertently disseminate the bacterium to the environment. The importation of the disease to previously nonendemic areas illustrates that the organism can exist beyond its traditional habitat and attention should be directed to other areas that are climatically conducive to the existence of the bacterium.

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OVERSEAS BRIEFS

The cholera pandemic is continuing its spread in the Americas and Africa. The following information regarding recent cases and recently infected areas has been supplied by the World Health Organization in the last two weeks.

CHOLERA IN AFRICA UPDATE

Parts of **Rwanda** have been declared cholera-infected. There were 24 cases reported from Cyangugu and Gisenyi Prefectures for the period 1 January to 31 March 1991.

The Mayo-Danai Department of the Province de L'Extrême-Nord in **Cameroon** has recently been declared chorea-infected. No new reports of cases have been received, however.

In **Nigeria**, the Federal Ministry of Health reports that cholera has now spread to 14 out of the 21 states of the country. A total of 7,674 cases have been reported, with 990 deaths (case-fatality rate of 13%). Control measures have been taken.

The Diffa, Dosso and Zinder Departments of **Niger** were declared cholera-infected on 11 July 1991. There were 1,202 cases and 87 deaths reported for the country for the period 1-7 July.

Cholera has spread to the Borkou-Ennedi-Tibesti, Logone oriental and Tandjile Prefectures of **Chad** recently. There were 994 cases and 109 deaths reported for the period 8-15 July.

Other reports from Africa are:

Angola - 493 cases and 15 deaths from 11 June to 7 July

Ghana - 6,713 cases and 181 deaths from 1 January to 5 July 1991

Liberia - 68 cases and 26 deaths up to 30 June

Mozambique - 520 cases and 5 deaths from 28 April to 25 May

Zambia - 450 cases and 141 deaths from 27 April to 10 July.

CHOLERA IN THE AMERICAS UPDATE

Guatemala is the latest country in the Americas to begin reporting cases of cholera. The Ministry of Health reported 1 confirmed case on 24 July. It occurred in La Gloria, San Marcos Department.

The Santander Department of **Colombia** has recently been declared cholera-infected. There were 331 cases with 2 deaths in the period 20-26 July.

Other reports from the Americas are:

Brazil - 6 cases reported for the period 6 June to 15 July

Ecuador - 7,446 cases and 117 deaths for the period 16 June to 13 July

Mexico - 38 cases reported from 29 June to 22 July

Peru - 11,855 cases and 210 deaths from 23 June to 22 July.

CHOLERA IN ASIA UPDATE

Recent reports of cholera from Asia are:

Iraq - 61 cases for the period 4 June to 30 June

India - 48 cases for the period 1-30 April

Singapore - a single case was reported during the period 30 June to 6 July

Japan - 51 cases (49 imported from elsewhere) for the period 9 January to 12 July.

INFLUENA IN PAPUA NEW GUINEA

An epidemic of influenza-like illness started in early May in an isolated community in the Highlands Region, according to a report dated 6 July 1991. Forty-five per cent of the population, in all age groups, has been affected so far, and at least 1 death has been attributed to the outbreak. Influenza A (H3N2) has been implicated on serolgical evidence in patients for whom paired sera were available.

INFLUENZA IN NEW ZEALAND

July reports of data collected by the New Zealand national network of sentinel general practitioners suggests that influenza activity is increasing in New Zealand, particularly in the Auckland region and the whole of the South Island. The predominant strain identified from throat swabs was influneza B, but an influenza A (H1N1) was also identified from Gisborne in the North Island.

Influenza B remains the most common influenza type in other countries now experiencing influenza: Argentina, Brazil, Chile, Hong Kong, Thailand and Australia (*CDI*, this issue). Influenza A appears far less frequently but recently a case of influenza A (H1N1) was confirmed in Brazil and a case of influenza A (H3N2) was confirmed in Ecuador.

CDI NOTICES TO READERS

NEW PUBLIC HEALTH PUBLICATION

Readers of the *CDI* will be interested to learn of a new public health publication. The *Western Australian Notifiable Diseases Bulletin* will be published monthly by the Epidemiology and Research Branch, Health Department of Western Australia. Copies are available by contacting:

Epidemiology and Research Branch
1st Floor C Block
189 Royal Street
East Perth WA 6004

ph (09) 222 4241
fax (09) 222 4236

SUSPENSION OF INDIVIDUAL PATIENT USAGE (IPU) AUTHORISATIONS FOR JAPANESE ENCEPHALITIS VACCINE

Several episodes of major adverse effects following the use of Japanese Encephalitis (JE) vaccine have recently been reported. These effects were delayed hypersensitivity reactions following the second dose which resulted in varying degrees of urticaria, angioedema and circulatory collapse with one patient requiring resuscitation and support in intensive care.

In view of the fact that hypersensitivity as a complication of JE vaccine has been recognised in the USA and Denmark, and that the risk of contracting the disease during travel to endemic or epidemic areas is estimated to be very low, the Communicable Diseases Section of the Department of Health, Housing and Community Services has now suspended approval for JE vaccine release pending evaluation of a marketing application by the Australian Drug Evaluation Committee (ADEC).

The NH&MRC recommends that as an alternative to the JE vaccine, the following measures will decrease the risk of infection[1]:

- minimise travel in areas where JE is epidemic;
- be aware that risks are greater in rural areas than in cities;
- take precautions against mosquito bites, for example, by:

- minimising outdoor exposure from dusk to dawn and on overcast days;
- sleeping in screened quarters and/or under mosquito netting;
- wearing clothing leaving a minimum of bare skin; and
- using insect repellents on exposed skin surfaces (repellents shold not contain more than 20% DEET active ingredient).

REFERENCE

1. NH&MRC. *Immunisation Procedures*, 4th Edition. AGPS Canberra. 1991.

COMMUNICABLE DISEASES SURVEILLANCE

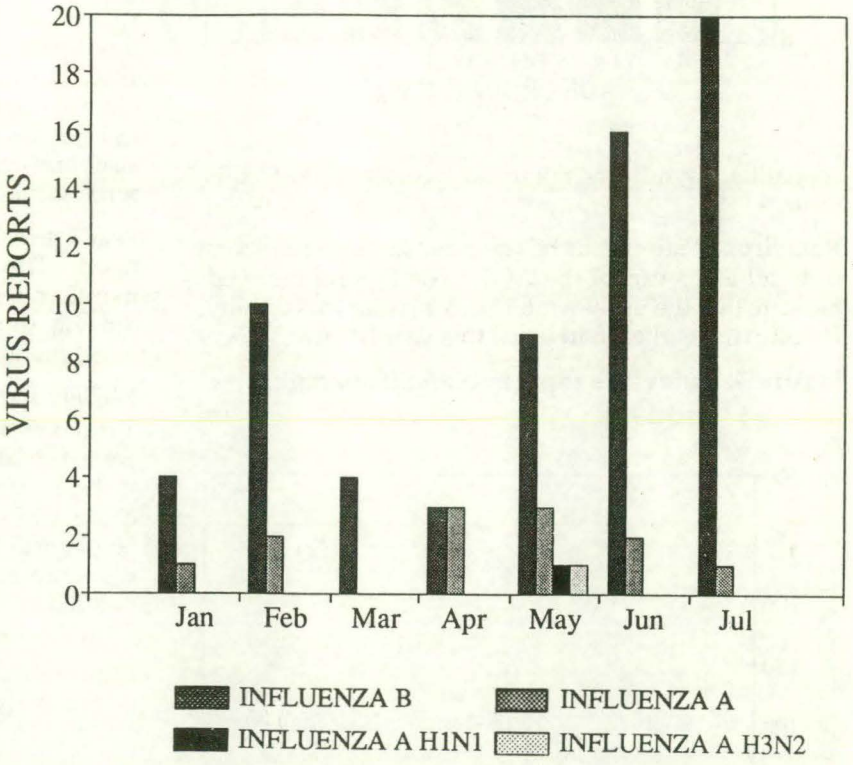
CDI LABORATORY REPORTING SCHEMES

A total of 1 305 reports were processed for the latest reporting period (17 July - 30 July 1991).

Following the recent introduction of a new code, ***Chlamydia pneumoniae*** (strain TWAR) has been reported for the first time. The patient was a 66 year old man who had lower respiratory tract disease; the diagnosis was made by the demonstration of specific IgM by immunofluorescence.

C. pneumoniae has distinct morphological and serological differences from *C. psittaci* and *C. trachomatis*, causes clinical disease mainly in young adults[1], and may be causally associated with asthma[2]. Seropositivity is rare in children, but increases with age to a plateau of about 50% by age 20-30. In Australia, an antibody survey conducted in South Australia and the Northern Territory showed that many persons had higher IgG titres against *C. pneumoniae* than against *C. trachomatis*, but no isolations of *C. pneumoniae* were made[3].

Figure 1. Influenza virus reports 1991, by month of specimen collection



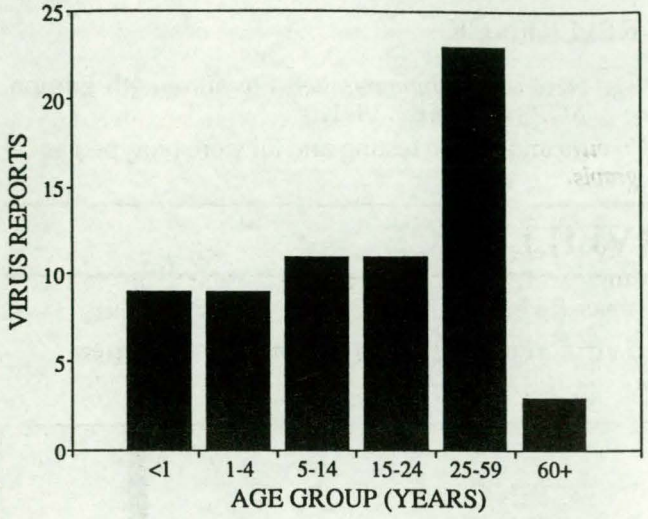
The number of **influenza B** reports has been increasing recently but there is still very little influenza A activity of any subtype (Figure 1). There were 21 reports of influenza B in this period, the highest number since the epidemic in mid-1989, when there was a total of 435 reports of this virus. This year most of the 66 cases reported so far have occurred in adults (Figure 2). Syndromes reported have been respiratory (47 cases), muscle and joint disease (3 cases), fever of unknown origin (8 cases), fever/malaise (6 cases), hepatic disease (1 case) and CNS symptoms (1 case). Most cases (42) have been diagnosed serologically. Eight were diagnosed by direct identification methods (6 by immunofluorescence and 2 by immunoenzymatic techniques) and for 23 cases, virus isolations were made.

It appears that Australia is following 1-2 months behind New Zealand with this increase in influenza B reports, as is typically the case (*CDI*;15:228, and see *Overseas Briefs*, this issue of *CDI*). ASPREN reports of influenza have also increased since mid-June, with a peak of 65 reports in week 23:15/7-21/7 (page 278 *CDI*, this issue). In this reporting period, influenza B reports have come from laboratories in Melbourne, Sydney, Adelaide and Canberra.

One further case of **echovirus type 17** infection has been reported, bringing the total for the year to 15. The patient was a six month old girl who was suffering general malaise/mild fever. The virus was identified in CSF samples.

A total of 290 reports of **respiratory syncytial virus** were received. This is a lower number than is usual for the seasonal peak which normally occurs around July each year (*CDI* 90/10). One report was of a 3 month old girl who had lower respiratory tract disease, and en-

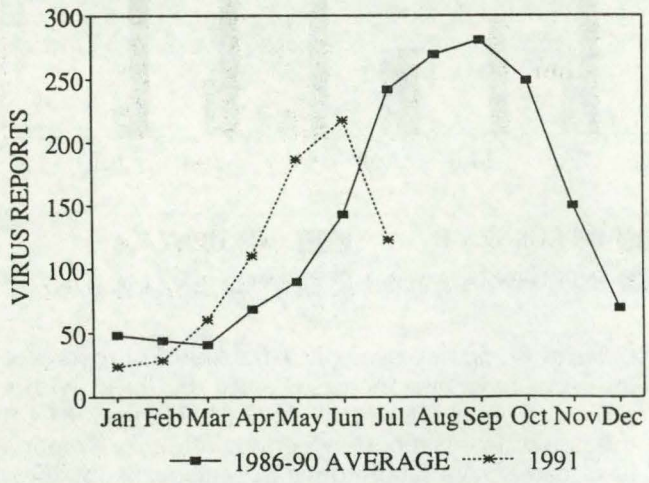
Figure 2. Influenza B virus reports 1991, by age group



cephalitis, a syndrome not usually associated with this virus.

Rotavirus continues to be reported in large numbers from all areas except the ACT. The reports received indicate that the usual winter peak in rotavirus activity is occurring earlier than usual this year (Figure 3).

Figure 3. Rotavirus reports 1986-90 average and 1991



Four cases of *Legionella pneumophila* have been reported. Three cases were notified by the Institute of Clinical Pathology and Medical Research, Westmead: 2 male patients, age group 25-44 years (1 renal transplant recipient) and 1 female, age group 65-74 years. The fourth case was notified by Royal Canberra Hospital (no details supplied).

Rubella was reported in 6 patients. Three of these were women of child-bearing age (34 yrs, 13 yrs, 13 yrs), for whom no clinical information was supplied. All diagnoses were by detection of specific IgM.

No exposure details were provided for the 4 cases of **Q fever** reported. One case was described as 'not vaccinated'.

Syphilis was reported in a woman (age group 25-44 yrs) who was 14 weeks pregnant.

There were 2 reports of invasive ***Haemophilus influenza*** type b. One was reported from blood cultures collected from a 3 month old boy with facial cellulitis (Toowoomba) and the other was from a 2 year old girl with meningitis (Nambour).

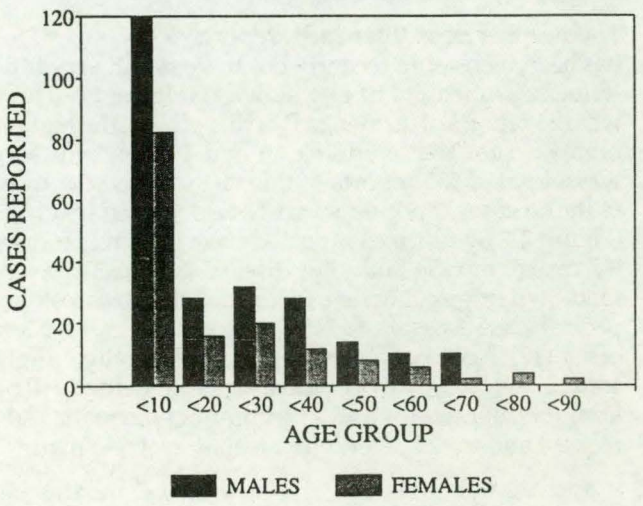
Serological tests detected past infection with ***Yersinia enterocolitica*** in an adult woman and an adult man with joint disease (Rockhampton).

A total of 93 cases of *Yersinia* have been reported through the *CDI* laboratory reporting schemes since 1986, but these are the first cases of associated joint disease that have been reported apart form a single case in 1988: an adult male who had suffered general malaise and joint disease and whose diagnosis was serological.

Reactive polyarthritis in adults is increasingly recognised. The onset of joint symptoms may be days to months after onset of acute diarrhoea. Studies in Scandinavia show 10-30% of adults with *Y. enterocolitica* infection develop polyarthritis[4].

Yersinia infections are notifiable in all States (except Tasmania) and in the Northern Territory. In 1990 there were 413 laboratory-confirmed cases notified and 216 in 1989. Unfortunately, these notifications do not include site of organism isolation or clinical history. Serological tests are therefore not discriminated from isolations of *Yersinia* in faeces or blood. Of the cases notified in 1990, 60% were in males, 38% in females and 2% unknown. Most cases were in children (Figure 4).

Figure 4. Yersinia infection notified, 1990, by age group and sex



Dr Suzanne Proudman, Royal Adelaide Hospital, (08) 223 0230, is interested in hearing of any further cases of *Yersinia* reative polyarthritis.

A new pilot surveillance programme is underway in three Sydney Laboratories collecting reports of isolates from sterile sites. The scheme, entitled **LabDOSS**: Laboratory Database of Organisms from Sterile Sites, is intended to become a national scheme in the future. The aim of LabDOSS is to improve surveillance of significant isolates, to provide epidemiological data on organisms that are not otherwise notifiable, and to provide a reporting nidus for research. The system is aimed for ease of use by the laboratory (using computerised data where available) and to be timely in nature. It collects demographical data, minimal clinical data, and microbiological results. The pilot program in the South West Area Pathology Service collected the following information on the meningococcal meningitis outbreak in Sydney.

Six cases of **meningococcal meningitis** have been reported recently from the Sydney area. All the cases were meningitis; none have been fatal. *Neisseria meningitidis* type C has been isolated in all cases except for one in which an untypable *N. meningitidis* was isolated from blood culture (Table).

Table. Sydney meningococcal cases: patient and isolate details

Patient Age/Sex	Positive Samples	Group	Date of Organism Isolation
2yrs F	CSF	C	17 July
14yrs F	CSF, blood	C	18 July
5mths M	CSF, Blood	C	28 July
2 yrs M	Blood	untypable	31 July
6yrs F	CSF, Blood	C	1 August
9 yrs M	Blood	C	3 August

Corynebacterium diphtheriae endocarditis: over the last 12 months, the Clinical Microbiology Unit, Institute of Clinical pathology and Medical Research, Westmead Hospital, has received five isolates of *C. diphtheriae* from blood cultures taken from patients admitted to hospitals in the Sydney region. All patients had endocarditis and two patients had proven and one patient possible septic arthritis. All five isolates were nontoxigenic by *in vitro* and *in vivo* testing and all were biotyped as *var gravis*.

The number of **hepatitis C** reports has increased rapidly in recent months, with 180 diagnoses reported for June, and 112 reported for July so far. This large increase is because Fairfield Hospital (Melbourne) and the Institute of Medical and Veterinary Science (Adelaide) have only recently begun reporting their hepatitis C tests. It does not necessarily represent an increased incidence of the disease in the community.

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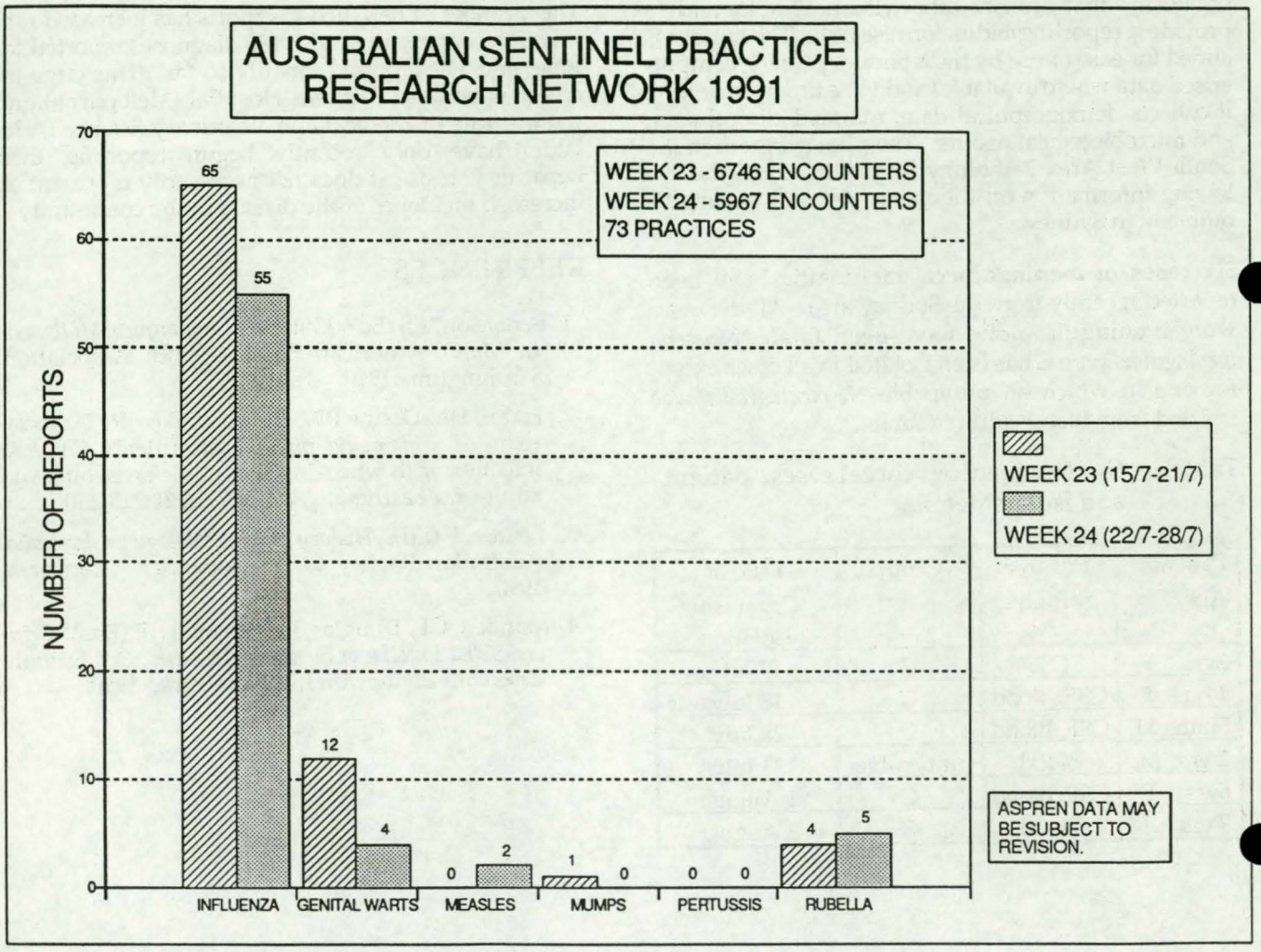
AUSTRALIAN SENTINEL PRACTICE RESEARCH NETWORK 1991
WEEK 23 - 6746 ENCOUNTERS
WEEK 24 - 5967 ENCOUNTERS
73 PRACTICES
NUMBER OF REPORTS
70
60
50
40
30
20
10
0
65
55
12
4
0
2
1
0
0
0
4
5
INFLUENZA
GENITAL WARTS
MEASLES
MUMPS
PERTUSSIS
RUBELLA
WEEK 23 (15/7-21/7)
WEEK 24 (22/7-28/7)
ASPREN DATA MAY BE SUBJECT TO REVISION.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES
BASED ON DATE OF REPORTING

PERIOD 17/07/91 TO 30/07/91

CODE 019 - FAIRFIELD HOSPITAL, MELBOURNE (VIC)
CODE 066 - PRINCESS MARGARET HOSPITAL, PERTH (WA)
CODE 110 - INSTITUTE OF MEDICAL & VETERINARY SCIENCE, ADELAIDE (SA)
CODE 112 - INSTITUTE OF CLINICAL PATHOLOGY & MEDICAL RESEARCH, WESTMEAD (NSW)
CODE 113 - PRINCE HENRY/PRINCE OF WALES HOSPITALS, SYDNEY (NSW)
CODE 114 - ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, CAMPERDOWN (NSW)
CODE 115 - STATE HEALTH LABORATORY, BRISBANE (QLD)
CODE 116 - WODEN VALLEY HOSPITAL, GARRAN (ACT)

		019	066	110	112	113	114	115	116	TOTAL
0100	ADENOVIRUS NOT TYPED	0	5	10	7	2	2	33	0	59
0101	ADENOVIRUS TYPE 1	2	0	1	2	0	0	0	0	5
0102	ADENOVIRUS TYPE 2	1	0	0	1	0	0	0	0	2
0103	ADENOVIRUS TYPE 3	1	0	0	3	0	0	0	0	4
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	1	0	0	0	0	0	0	0	1
0106	ADENOVIRUS TYPE 6	0	0	2	0	0	0	0	0	2
0108	ADENOVIRUS TYPE 8	0	0	0	0	1	0	0	0	1
0114	ADENOVIRUS TYPE 14	0	0	0	1	0	0	0	0	1
0126	ADENOVIRUS TYPE 26	1	0	0	0	0	0	0	0	1
0129	ADENOVIRUS TYPE 29	0	0	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	1	0	1	0	0	2	0	0	4
0203	INFLUENZA B VIRUS	2	0	9	2	0	4	0	4	21
0301	PARAINFLUENZA VIRUS TYPE 1	0	2	0	0	0	0	0	0	2
0302	PARAINFLUENZA VIRUS TYPE 2	0	1	1	0	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	7	0	6	4	1	3	3	0	24
0400	RESPIRATORY SYNCYTIAL VIRUS	38	10	25	53	20	69	69	6	290
0500	RHINOVIRUS (ALL TYPES)	3	0	0	4	0	13	4	0	24
0600	MYCOPLASMA PNEUMONIAE	1	1	2	1	3	1	0	0	9
0700	ORNITHOSIS-PSITTACOSIS	5	0	0	0	2	0	0	0	7
0809	COXSACKIEVIRUS A9	3	0	0	0	0	0	0	0	3
0816	COXSACKIEVIRUS A16	0	0	0	1	0	1	0	0	2
0824	COXSACKIEVIRUS A24 = ECHOVIRUS	1	0	0	0	0	0	0	0	1
0902	COXSACKIEVIRUS B2	0	0	0	2	0	1	0	0	3
0904	COXSACKIEVIRUS B4	1	0	0	3	0	1	0	1	6
0905	COXSACKIEVIRUS B5	1	0	0	0	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	0	0	1	0	0	0	0	1
1014	ECHOVIRUS TYPE 14	0	0	0	1	0	0	0	0	1
1017	ECHOVIRUS TYPE 17	0	0	0	0	0	1	0	0	1
1022	ECHOVIRUS TYPE 22	0	0	0	3	0	0	0	0	3
1101	POLIOVIRUS TYPE 1	0	0	0	1	0	0	0	0	1
1102	POLIOVIRUS TYPE 2	0	0	0	2	0	0	0	0	2
1103	POLIOVIRUS TYPE 3	0	0	0	2	0	0	0	0	2
1200	MUMPS VIRUS	0	0	0	1	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	1	0	0	0	0	0	0	0	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	1	3	0	25	0	0	0	3	32
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	3	0	11	8	5	0	0	0	27
1303	VARICELLA-ZOSTER VIRUS	5	0	3	2	0	0	1	0	11
1306	HERPES SIMPLEX TYPE 1	55	1	15	4	0	0	48	0	123
1307	HERPES SIMPLEX TYPE 2	67	0	17	25	0	0	28	0	137
1401	COXIELLA BURNETII	0	0	2	2	0	0	0	0	4
1502	PICORNIA VIRUS - NOT TYPED	0	0	0	1	11	0	19	0	31
1515	CONTAGIOUS PUSTULAR DERMATITIS	1	0	0	0	0	0	0	0	1
1521	MEASLES VIRUS	1	0	1	0	1	0	0	0	3
1522	RUBELLA VIRUS	1	0	1	3	1	0	0	0	6
1532	HEPATITIS B ANTIGEN	15	0	0	25	5	1	33	0	79
1535	HEPATITIS A ANTIBODY	2	0	3	3	1	0	3	0	12
1536	HEPATITIS C VIRUS	92	0	23	0	0	0	0	0	115
1537	HEPATITIS, DELTA	0	0	0	0	0	0	5	0	5
1541	CHLAMYDIA TRACHOMATIS - UNSPEC	1	0	31	12	6	0	0	4	54
1544	CHLAMYDIA PNEUMONIAE	0	0	0	1	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	21	20	0	6	2	5	20	0	74
1563	CORONAVIRUS	0	0	0	1	0	0	0	0	1
1564	ROTAVIRUS	7	10	8	38	17	6	0	0	86
1566	NORWALK AGENT	1	0	0	2	0	0	0	0	3
1571	ENTEROVIRUS TYPE 71 (BCR)	1	0	0	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	1	0	0	0	0	5	0	0	6
9992	ROSS RIVER VIRUS	0	0	0	2	0	0	0	0	2
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	1	0	0	1
TOTAL		346	53	172	256	78	116	266	18	1305

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS FROM CONTRIBUTING LABORATORIES BY STATE OF CONTRIBUTING LABORATORY

PERIOD 17/07/91 TO 30/07/91

NSW: ICPMR; PHH/POW; RACH; ST GEORGE HOSP, KOGARAH; ROYAL NEWCASTLE HOSP; TAMWRTH LAB.
VIC: FAIRFIELD; RCH; MDU, UNI MELB.
QLD: STATE LAB, BRIS; TOOWOOMBA PATH LAB; ROYAL BRIS HOSP; DR TB LYNCH, PATHOLOGIST, ROCKHAMPTON.
WA: STATE LAB, PERTH; PMH.
SA: IMVS.
TAS: ROYAL HOBART HOSP; DIAGNOSTIC SERVICES, LAUNCESTON; LAUNCESTON GEN HOSP; DIAGNOSTIC SERVICES, HOBART; HOBART PATH; MERSEY GEN HOSP, LATROBE.
ACT: WVH.

		NSW	VIC	QLD	WA	SA	ACT	TOTAL
0100	ADENOVIRUS NOT TYPED	11	0	33	5	10	0	59
0101	ADENOVIRUS TYPE 1	2	2	0	0	1	0	5
0102	ADENOVIRUS TYPE 2	1	1	0	0	0	0	2
0103	ADENOVIRUS TYPE 3	3	1	0	0	0	0	4
0104	ADENOVIRUS TYPE 4	0	1	0	0	0	0	1
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	1
0106	ADENOVIRUS TYPE 6	0	0	0	0	2	0	2
0108	ADENOVIRUS TYPE 8	1	0	0	0	0	0	1
0114	ADENOVIRUS TYPE 14	1	0	0	0	0	0	1
0126	ADENOVIRUS TYPE 26	0	1	0	0	0	0	1
0129	ADENOVIRUS TYPE 29	1	0	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	2	1	0	0	1	0	4
0203	INFLUENZA B VIRUS	6	2	0	0	9	4	21
0301	PARAINFLUENZA VIRUS TYPE 1	0	0	0	2	0	0	2
0302	PARAINFLUENZA VIRUS TYPE 2	0	0	0	1	1	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	8	7	3	0	6	0	24
0400	RESPIRATORY SYNCYTIAL VIRUS	142	38	69	10	25	6	290
0500	RHINOVIRUS (ALL TYPES)	17	3	4	0	0	0	24
0600	MYCOPLASMA PNEUMONIAE	5	1	0	1	2	0	9
0700	ORNITHOSIS-PSITTACOSIS	2	5	0	0	0	0	7
0809	COXSACKIEVIRUS A9	0	3	0	0	0	0	3
0816	COXSACKIEVIRUS A16	2	0	0	0	0	0	2
0824	COXSACKIEVIRUS A24 = ECHOVIRUS	0	1	0	0	0	0	1
0902	COXSACKIEVIRUS B2	3	0	0	0	0	0	3
0904	COXSACKIEVIRUS B4	4	1	0	0	0	1	6
0905	COXSACKIEVIRUS B5	0	1	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	1	0	0	0	0	0	1
1014	ECHOVIRUS TYPE 14	1	0	0	0	0	0	1
1017	ECHOVIRUS TYPE 17	1	0	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	3	0	0	0	0	0	3
1101	POLIOVIRUS TYPE 1	1	0	0	0	0	0	1
1102	POLIOVIRUS TYPE 2	2	0	0	0	0	0	2
1103	POLIOVIRUS TYPE 3	2	0	0	0	0	0	2
1200	MUMPS VIRUS	1	0	0	0	0	0	1
1300	HERPES VIRUS GROUP - NOT TYPED	0	1	0	0	0	0	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	25	1	0	3	0	3	32
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	13	3	0	0	11	0	27
1303	VARICELLA-ZOSTER VIRUS	2	5	1	0	3	0	11
1306	HERPES SIMPLEX TYPE 1	4	55	48	1	15	0	123
1307	HERPES SIMPLEX TYPE 2	25	67	28	0	17	0	137
1401	COXIELLA BURNETII	2	0	0	0	2	0	4
1502	PICORNIA VIRUS - NOT TYPED	12	0	19	0	0	0	31
1515	CONTAGIOUS PUSTULAR DERMATITIS	0	1	0	0	0	0	1
1521	MEASLES VIRUS	1	1	0	0	1	0	3
1522	RUBELLA VIRUS	4	1	0	0	1	0	6
1532	HEPATITIS B ANTIGEN	31	15	33	0	0	0	79
1535	HEPATITIS A ANTIBODY	4	2	3	0	3	0	12
1536	HEPATITIS C VIRUS	0	92	0	0	23	0	115
1537	HEPATITIS, DELTA	0	0	5	0	0	0	5
1541	CHLAMYDIA TRACHOMATIS - UNSPEC	18	1	0	0	31	4	54
1544	CHLAMYDIA PNEUMONIAE	1	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	13	21	20	20	0	0	74
1563	CORONAVIRUS	1	0	0	0	0	0	1
1564	ROTAVIRUS	61	7	0	10	8	0	86
1566	NORWALK AGENT	2	1	0	0	0	0	3
1571	ENTEROVIRUS TYPE 71 (BCR)	0	1	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	5	1	0	0	0	0	6
9992	ROSS RIVER VIRUS	2	0	0	0	0	0	2
9994	SMALL VIRUS (LIKE) PARTICLE	1	0	0	0	0	0	1
TOTAL		450	346	266	53	172	18	1305

NOTE: DIRECT COMPARISION BETWEEN STATES IS NOT POSSIBLE SINCE:
- SOME STATES HAVE MORE THAN ONE CONTRIBUTING LABORATORY; AND
- INTERSTATE REFERRRALS OCCUR REGULARLY.

AUSTRALIA - COMMUNICABLE DISEASES INTELLIGENCE

VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 1

PERIOD 17/07/91 TO 30/07/91

1. CODE 00, 99 ..... - NO ILL OR DATA
2. CODE 01, 02, 11, 12 - RESPIRATORY
3. CODE E3 ..... - ENCEPHALITIS
4. CODE M3 ..... - MENINGITIS
5. CODE 04 ..... - PARALYSIS
6. CODE 05, 13 ..... - CNS OTHER UNSPEC
7. CODE 07, 49 - GASTRO INTESTINAL
8. CODE 17, 47 - HEPATIC
9. CODE 19 ... - CVS
10. CODE 89 ... - URINARY TRACCT
11. CODE 06 ... - SKIN MUCOUS

		1	2	3	4	6	7	8	9	10	11	TOTAL
0100	ADENOVIRUS NOT TYPED	0	30	0	0	0	22	0	0	0	2	54
0101	ADENOVIRUS TYPE 1	0	4	0	0	0	1	0	0	0	0	5
0102	ADENOVIRUS TYPE 2	0	1	0	0	0	1	0	0	0	0	2
0103	ADENOVIRUS TYPE 3	0	2	0	0	0	1	0	0	0	0	3
0105	ADENOVIRUS TYPE 5	0	1	0	0	0	0	0	0	0	0	1
0106	ADENOVIRUS TYPE 6	0	2	0	0	0	0	0	0	0	0	2
0114	ADENOVIRUS TYPE 14	0	0	0	0	0	1	0	0	0	0	1
0126	ADENOVIRUS TYPE 26	0	0	0	0	0	1	0	0	0	0	1
0199	ADENOVIRUS TYPING PENDING	0	2	0	0	0	0	0	0	0	0	2
0203	INFLUENZA B VIRUS	1	14	0	0	0	0	0	0	0	0	15
0301	PARAINFLUENZA VIRUS TYPE 1	0	2	0	0	0	0	0	0	0	0	2
0302	PARAINFLUENZA VIRUS TYPE 2	0	2	0	0	0	0	0	0	0	0	2
0303	PARAINFLUENZA VIRUS TYPE 3	0	24	0	0	0	0	0	0	0	0	24
0400	RESPIRATORY SYNCYTIAL VIRUS	11	275	0	0	0	2	0	0	0	0	288
0500	RHINOVIRUS (ALL TYPES)	2	19	0	0	0	0	0	0	0	0	21
0600	MYCOPLASMA PNEUMONIAE	2	3	0	0	0	0	0	0	0	0	5
0700	ORNITHOSIS-PSITTACOSIS	0	4	0	0	0	0	0	0	0	0	4
0809	COXSACKIEVIRUS A9	1	0	0	0	0	0	0	0	0	0	1
0816	COXSACKIEVIRUS A16	1	0	0	0	0	0	0	0	0	1	2
0902	COXSACKIEVIRUS B2	1	1	0	1	0	0	0	0	0	0	3
0904	COXSACKIEVIRUS B4	1	3	0	1	1	0	0	0	0	0	6
0905	COXSACKIEVIRUS B5	0	0	0	0	0	1	0	0	0	0	1
1006	ECHOVIRUS TYPE 6	0	0	0	1	0	0	0	0	0	0	1
1014	ECHOVIRUS TYPE 14	0	0		0	0	1	0	0	0	0	1
1022	ECHOVIRUS TYPE 22	0	0	0	0	0	3	0	0	0	0	3
1102	POLIOVIRUS TYPE 2	1	0	0	0	0	0	0	0	0	0	1
1103	POLIOVIRUS TYPE 3	0	1	0	0	0	0	1	0	0	0	2
1300	HERPES VIRUS GROUP - NOT TYPED	0	0	0	0	0	0	0	0	0	1	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	6	1	1	0	0	0	0	0	0	7	15
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	5	2	0	0	0	0	0	1	0	1	9
1303	VARICELLA-ZOSTER VIRUS	0	0	0	0	0	0	0	0	0	8	8
1306	HERPES SIMPLEX TYPE 1	0	9	0	0	0	0	0	0	0	80	89
1307	HERPES SIMPLEX TYPE 2	4	0	0	0	0	0	0	0	0	37	41
1401	COXIELLA BURNETII	2	0	0	0	0	0	0	0	0	0	2
1502	PICORNIA VIRUS - NOT TYPED	0	8	1	0	2	20	0	0	0	0	31
1515	CONTAGIOUS PUSTULAR DERMATITIS	0	0	0	0	0	0	0	0	0	1	1
1521	MEASLES VIRUS	0	0	0	0	0	0	0	0	0	2	2
1522	RUBELLA VIRUS	3	0	0	0	0	0	0	0	0	1	4
1532	HEPATITIS B ANTIGEN	34	0	0	0	0	0	44	0	0	0	78
1535	HEPATITIS A ANTIBODY	3	0	0	0	0	0	9	0	0	0	12
1536	HEPATITIS C VIRUS	113	0	0	1	0	0	0	0	0	0	114
1537	HEPATITIS, DELTA	0	0	0	0	0	0	5	0	0	0	5
1541	CHLAMYDIA TRACHOMATIS - UNSPEC	8	0	0	0	0	0	0	0	0	0	8
1544	CHLAMYDIA PNEUMONIAE	0	1	0	0	0	0	0	0	0	0	1
1556	CMV - CYTOMEGALOVIRUS	4	32	0	0	0	3	3	0	5	0	47
1563	CORONAVIRUS	0	0	0	0	0	1	0	0	0	0	1
1564	ROTAVIRUS	0	0	0	0	0	85	0	0	0	0	85
1566	NORWALK AGENT	0	0	0	0	0	3	0	0	0	0	3
1571	ENTEROVIRUS TYPE 71 (BCR)	0	0	0	1	0	0	0	0	0	0	1
1599	ENTEROVIRUS TYPING PENDING	0	3	0	0	0	0	0	0	0	1	4
9994	SMALL VIRUS (LIKE) PARTICLE	0	0	0	0	0	1	0	0	0	0	1
TOTAL		203	446	2	5	3	147	62	1	5	142	1016

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VIRAL IDENTIFICATIONS BY CLINICAL INFORMATION TABLE 2

PERIOD 17/07/91 TO 30/07/91

12. CODE 10 - EYE
13. CODE 59 - GENITAL
14. CODE 39 - ENDOCRINE/SALIVARY GL.
15. CODE 38 - RETICULO-ENDOTHELIAL
16. CODE 29 - MUSCLE/JOINT
17. CODE 69 - CONGENTIAL
18. CODE P8 - PUO
19. CODE G8 - FEVER/MALAISE
20. CODE 09 - OTHER
21. CODE A1 - SIDS

		12	13	14	15	17	18	19	20	21	TOTAL
0100	ADENOVIRUS NOT TYPED	3	0	0	0	0	1	0	1	0	5
0103	ADENOVIRUS TYPE 3	1	0	0	0	0	0	0	0	0	1
0104	ADENOVIRUS TYPE 4	1	0	0	0	0	0	0	0	0	1
0108	ADENOVIRUS TYPE 8	1	0	0	0	0	0	0	0	0	1
0129	ADENOVIRUS TYPE 29	0	0	0	0	0	0	0	1	0	1
0199	ADENOVIRUS TYPING PENDING	0	0	0	0	0	0	0	1	1	2
0203	INFLUENZA B VIRUS	0	0	0	0	0	0	4	2	0	6
0400	RESPIRATORY SYNCYTIAL VIRUS	0	0	0	0	0	0	1	1	0	2
0500	RHINOVIRUS (ALL TYPES)	0	0	0	0	0	0	1	2	0	3
0600	MYCOPLASMA PNEUMONIAE	0	0	0	1	0	1	0	2	0	4
0700	ORNITHOSIS-PSITTACOSIS	0	0	0	0	0	0	3	0	0	3
0809	COXSACKIEVIRUS A9	0	0	0	0	0	0	1	0	0	1
0824	COXSACKIEVIRUS A24 = ECHOVIRUS	1	0	0	0	0	0	0	0	0	1
1017	ECHOVIRUS TYPE 17	0	0	0	0	0	0	1	0	0	1
1101	POLIOVIRUS TYPE 1	0	0	0	0	0	0	0	0	1	1
1102	POLIOVIRUS TYPE 2	0	0	0	0	0	0	0	0	1	1
1200	MUMPS VIRUS	0	0	0	0	0	0	0	1	0	1
1301	HERPES SIMPLEX VIRUS - NOT TYP	0	16	0	0	0	0	0	1	0	17
1302	EPSTEIN-BARR VIRUS (EB VIRUS)	0	0	9	2	0	0	3	2	0	16
1303	VARICELLA-ZOSTER VIRUS	1	0	0	0	0	0	0	2	0	3
1306	HERPES SIMPLEX TYPE 1	2	28	0	0	0	0	1	3	0	34
1307	HERPES SIMPLEX TYPE 2	0	95	0	0	0	0	0	0	0	95
1401	COXIELLA BURNETII	0	0	0	0	0	1	0	1	0	2
1521	MEASLES VIRUS	0	0	0	0	0	0	0	1	0	1
1522	RUBELLA VIRUS	0	0	0	0	0	0	0	2	0	2
1532	HEPATITIS B ANTIGEN	0	0	0	0	0	0	0	1	0	1
1536	HEPATITIS C VIRUS	0	0	0	0	0	0	1	0	0	1
1541	CHLAMYDIA TRACHOMATIS - UNSPEC	1	43	0	0	0	0	0	1	0	45
1556	CMV - CYTOMEGALOVIRUS	0	3	0	0	2	1	1	19	0	26
1564	ROTAVIRUS	0	0	0	0	0	0	0	1	0	1
1599	ENTEROVIRUS TYPING PENDING	0	0	0	0	0	0	1	1	0	2
9992	ROSS RIVER VIRUS	0	0	0	0	0	0	1	1	0	2
TOTAL		11	185	9	3	2	4	19	47	3	283