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An outbreak of non-sexually transmitted gonococcal conjunctivitis in Central Australia and the Kimberley region

Rex Matters,¹ Ignatius Wong,² and Donna Mak³

Abstract

From 13 February to 27 June 1997, 447 cases of gonococcal conjunctivitis were identified by Communicable Disease and Public Health Centres and Community Clinics in the Northern Territory, Western Australia and South Australia. The outbreak involved Aboriginal communities predominantly in Central Australia and the Kimberley region in Western Australia. This was the first outbreak recorded in the Kimberley region. It is not yet known whether the Kimberley cases were part of the larger Central Australian outbreak or whether they represented a separate and unrelated outbreak. Environmental factors associated with this outbreak were similar to those seen in previous outbreaks. Control measures were based on early recognition and treatment of index cases and identifying and treating contacts. Until sexually transmitted *Neisseria gonorrhoeae* is controlled in communities gonococcal conjunctivitis is likely to appear again. The role of oropharyngeal carriage of *N. gonorrhoeae* needs to be evaluated further. *Comm Dis Intell* 1998;22:52-58

Introduction

Gonococcal conjunctivitis is an acute painful conjunctivitis characterised by rapid transmission between individuals through non-sexual person to person contact. It has caused considerable morbidity in Aboriginal communities in Central Australia during five previously documented outbreaks (Table 1).^{1,2,3,4,5}

Environmental factors associated with these outbreaks included: above average summer rainfall preceding the outbreak; summer temperatures at the onset changing to winter temperatures towards the end; an increase in the percentage of *Haemophilus* species isolates from eye swabs; and increased fly numbers at the start of the outbreak. None of the outbreaks were characterised by an increase in notifications of sexually transmitted gonorrhoea in the time period preceding the outbreak.

This article reports on an outbreak of gonococcal conjunctivitis in Aboriginal communities predominantly in Central Australia during the period 13 February to 27 June 1997, and examines some of the environmental factors associated with the outbreak. The Central Australian area involved included the western Alice Springs region in the Northern Territory (NT), the Ngaanyatjarra central desert area and the Pitjantjatjara Lands of South Australia (SA). Cases also occurred in the Kimberley region in Western Australia (WA).

Methods

Case definition

A clinical illness was defined as intense inflammation of the conjunctivae, copious purulent discharge with or without periorbital oedema. A clinical case was confirmed

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when *Neisseria gonorrhoeae* (*N. gonorrhoeae*) was isolated on culture, detected by polymerase chain reaction (PCR) or Gram negative diplococci were seen by microscopy. Unconfirmed clinical cases were included in this analysis if there was a laboratory confirmed case notified from the same community. Date of onset was defined as the date on which the eye swab confirming the diagnosis was taken.

Collection of data

Patient information including age, gender, date of onset of illness and address was obtained from Disease Control Centres in Alice Springs, NT and Adelaide, SA; Goldfields Public Health Services, Boulder, WA; Kimberley Public Health Unit, Derby, WA; Western Diagnostic Pathology, Alice Springs, NT and Pathology, Alice Springs Hospital (ASH), NT. Information on the number of unconfirmed clinical cases, the spread of disease and fly density was obtained from Community Health Clinics in Central Australia. Population demographic statistics were obtained from Nganampa Health Council, Ngaanyatjarra Health Service and Rural Health - Alice Springs, Territory Health Services (THS). Rainfall and temperature data were obtained from the Bureau of Meteorology, Darwin and Alice Springs Regional Offices NT and WA. The number of unconfirmed clinical cases was not obtained from the Kimberley and only laboratory confirmed cases from this area were included in the analysis.

Laboratory investigation

N. gonorrhoeae strains from Central Australia were sent to the Prince of Wales Hospital, Sydney, for serotyping, auxotyping and minimum inhibitory concentration (MIC) testing. Records at the Pathology Laboratory, ASH, were examined to determine the percentage of *Haemophilus* spp. identified from eye swabs between June 1996 and June 1997 and the number of cases of disseminated gonococcal infection (DGI) between 1 August 1995 and 18 July 1997. DGI was defined as the isolation of *N. gonorrhoeae* from a sterile site, for example joint fluid.

Results

Number of cases

A total of 447 cases of gonococcal conjunctivitis were reported, 242 confirmed cases (including 5 reinfections) and 205 unconfirmed cases. Of the confirmed cases, 120 were culture positive, 53 PCR positive and 69 microscopy positive. The NT had 121, WA 105 and SA 16 confirmed cases. There appear to have been two epidemics during the outbreak (Figure 1). The smaller epidemic peaked in the Kimberley during March before the larger epidemic, involving many more communities in Central Australia, which peaked in May. The sharp peak in the number of Kimberley cases, however, is artefactual. Cases had been observed during late January and early February, but no swabs had been taken as gonococcal conjunctivitis had not been suspected at that time.

Gender and age distribution and of cases

There were 113 males and 129 females with confirmed infection. Over three-quarters of the confirmed cases were children under 10 years old, with the greatest number recorded in the 5 to 9 years age group (Table 2). The youngest child was four months old.

Figure 1. Gonococcal conjunctivitis, laboratory confirmed cases by week and geographic region

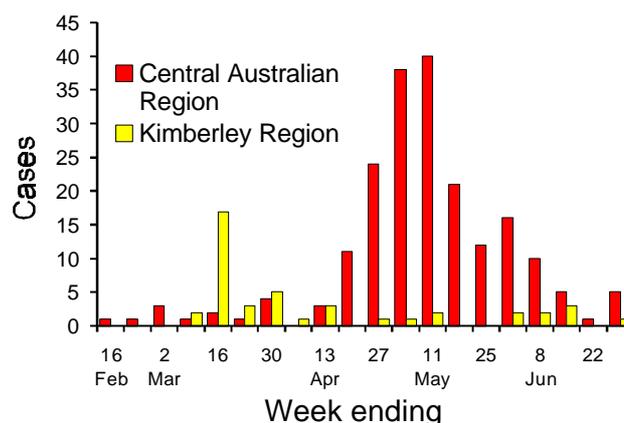


Table 1. Gonococcal conjunctivitis outbreaks in Central Australia, by date and number of cases

Year of outbreak	Cases ¹	Period of outbreak
1934	91	-
1981	35	May to June
1986-87	>140	November to May
1991	432	January to July
1992	>62	April to July

1. Includes both clinical and laboratory confirmed cases

Table 2. Age distribution of confirmed cases

Age group (years)	Number of cases	Per cent of cases
0 to 4	73	30
5 to 9	114	47
10 to 14	35	15
15 and over	12	5
Unknown	8	3
Total	242	100.0

Rainfall

The Giles Meteorological Station is in WA, approximately 150 kms north of the junction of the WA, NT and SA borders. This station was one of the closest to the communities which were involved in the start of the outbreak. The rainfall at Giles in December was above average and there was substantial rainfall in January and February preceding the outbreak (Figures 2a and 2b). The first cases appeared in the middle of February. Alice Springs received 80 mm of rain in January and 242 mm in February. The Kimberley region received 136 mm of rain in January and 274 mm in February. This is well above the average rainfall for these months.

Temperature

The mean daily minimum temperature at Giles in February was 24.7°C (Figures 2a and 2c) at the start of the outbreak and was below 10°C in June at the end of the epidemic. At the start of the epidemic the mean daily maximum temperature in February was 36°C, which is a typical summer temperature.

Sexually transmitted gonorrhoea in Central Australia

There was no increase in the notifications of sexually transmitted *N. gonorrhoeae* preceding the outbreak. The notification information from the NT Centre for Disease Control indicated that sexually transmitted disease was endemic at nearly the same level all year round.

Proportion of *Haemophilus* spp. isolates from eye swabs received at Alice Springs Hospital

The total number of *Haemophilus* spp. identified each month from eye swabs, expressed as a proportion of the monthly total number of eye swabs received, can be used as an indicator of the amount of general bacterial/viral eye disease in communities. During the period June 1996 to June 1997, the highest proportion of *Haemophilus* spp. (47%) occurred in March 1997. The mean monthly proportion was 33%. There was a gradual increase in the proportion of eye disease in which *Haemophilus* spp. was isolated at the start of the outbreak.

Fly density

Thirteen Central Australian communities involved in the epidemic were contacted and asked when they thought flies were at their most dense and when the flies decreased in number. Five communities where the first gonococcal conjunctivitis index cases appeared reported the flies were 'terrible' in February. Ten communities reported that the fly density was greatest in March and April. Fly density in four communities was greatest in May. Fly density reduced dramatically in June.

Laboratory investigation

Delays in transport frequently resulted in the death of *N. gonorrhoeae* in swabs and in many cases confirmation of infection could only be made from smears and PCR. All the Kimberley cases were tested by PCR.

Auxotyping, serotyping and MIC testing from Central Australia

The *N. gonorrhoeae* strains submitted for serotyping, auxotyping and MIC testing included 89 eye, 34 genital and 2 joint isolates. Of the 53 fully typed eye isolates, all were auxotype/serovar class Wt/IB3. Fourteen background isolates from standard STD surveillance obtained during the outbreak were typed as six Wt/IB3 strains, three Pro/IB3 strains, and one each of strains Pro/IB1, Pro/IB5, Wt/IA6, Pro/IA4 and Pro/IA6. Two eye isolates not involved in the outbreak, consisted of a Pro/IB3 strain and a Pro/IB1 strain. Two joint isolates consisted of a Wt/IA4 strain and a IB3 serovar (the Wt/IA4 strain was not involved in the outbreak but was included as a background strain). STD background isolates from April 1996 consisted of one Wt/IB3, thirteen Pro/IB3, four Wt/IA6 and three Wt/IA4 strains. The majority of the eye isolates tested so far have had a penicillin MIC of between 0.125 and 0.25 mg/L, with one isolate having a penicillin MIC of 0.5 mg/L. According to the criteria established by the Australian Gonococcal Surveillance

Figure 2a. Gonococcal conjunctivitis, number of cases at Giles, June 1996 to June 1997, by month

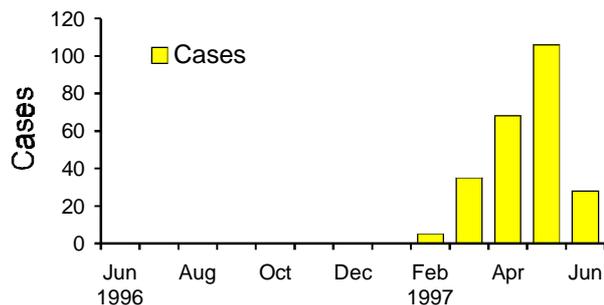


Figure 2b. Rainfall at Giles, June 1996 to June 1997, by month

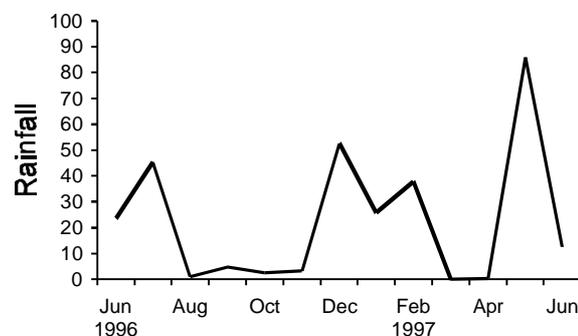
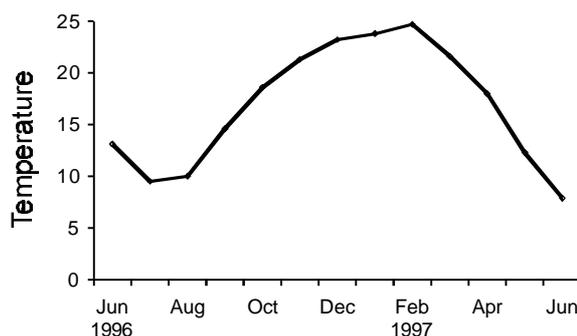


Figure 2c. Mean daily minimum temperatures at Giles, June 1996 to June 1997, by month



Programme, all the eye isolates are classified as sensitive to penicillin, but in the less sensitive range.

Public health response

An alert was sent by Alice Springs Disease Control to Central Australian communities (NT, WA, SA) in March after several cases had been reported. This alerted the communities to the presence of current cases, urged that swabs and cultures be taken and that procaine penicillin or amoxicillin with probenecid be given. A second alert was

sent out in April. In May, with over 40 cases reported, a further alert was sent out which included advice to treat household contacts, a direction not explicitly stated in the Central Australian Rural Practitioners Association (CARPA) standard treatment manual. An alert also went to all remote schools telling of the outbreak and the need for treatment. The outbreak, protocol, and need to treat all household contacts were discussed at both the Central Australia Disease Control Coordinating Committee (CADCCC) meeting/teleconference and the CARPA Conference, during May 1997.

Public Health Unit staff in the Kimberley region arrived at the affected community within 24 hours of the first notification (though not necessarily the index case) of gonococcal conjunctivitis. They then assisted local staff with screening and treatment.

Discussion

The number of cases reported is undoubtedly an underestimate of the true number of gonococcal eye infections, as not all patients presenting with conjunctivitis had an eye swab and smear taken for laboratory confirmation by either culture, smear or PCR. Direct input from Central Australian communities was invaluable in obtaining a more accurate estimate of the amount of clinical disease that was treated.

Fly counts were not performed in any community and the subjective assessment of fly numbers by staff may be inaccurate, but the survey covered many communities and a reasonable picture was obtained.

There is some indication that conjunctivitis caused by *Haemophilus* spp. is endemic throughout the year and *Haemophilus* conjunctivitis rises prior to and during gonococcal conjunctivitis outbreaks. This has been observed in every previous outbreak and is possibly due to the same environmental and other stimuli which promote gonococcal transmission.

The environmental factors present in this outbreak included:

1. heavy summer rainfall at least one month before the first cases appeared;
2. mean daily minimum temperature above 20°C for the first two months of the outbreak; and
3. an extremely high fly population in the first three months of the outbreak.

In addition, the percentage of eye infections caused by *Haemophilus* spp. increased to its highest monthly level for the year during the second month of the outbreak (March). These findings were similar to those seen in the previously reported outbreaks.

Factors which may have contributed to the occurrence and spread of the epidemic include: a background of high levels of sexually transmitted gonorrhoea; environmental conditions favouring increased survival of the organism; bushflies acting as a mechanical vector of *N. gonorrhoeae*; and, a high level of mobility of the population.

There is a large reservoir of sexually transmitted *N. gonorrhoeae* in remote Aboriginal communities in Central Australia,⁶ however, in common with previously reported outbreaks, the incidence of sexually transmitted *N. gonorrhoeae* did not increase before the outbreak.

N. gonorrhoeae can only survive in warm moist conditions and will die rapidly in a dry cold atmosphere. Survival of the organism on fomites would have been optimal at the start of the outbreak, rapidly decreasing as the temperature dropped below 10°C.

The breeding conditions, including humidity and temperature, for bushflies were ideal⁷ just after the rainfall in December and deteriorated markedly in June. A study conducted by the Commonwealth Scientific and Industrial Research Organisation (CSIRO) in Central Australia in 1980-1982, where fly counts were performed, recognised that the only major increase in bush fly abundance occurred after the first summer rainfall.⁸ Bushfly pupae cannot survive at all when the temperature fluctuates between 6°C and 18°C. During the outbreak small adult flies were present (indicating a reduced larval stage) and there was extreme pressure for the available moisture/food necessary for the breeding cycle and for survival. It is likely that flies were attracted to moist, purulent eyes and became 'sticky' flies.

There is still insufficient evidence to determine whether flies definitely contribute to the spread of gonococcal conjunctivitis. Recent studies in WA have shown that *Chlamydia trachomatis* can survive in the bushfly population, however, further work is still in progress to ascertain whether the number of organisms carried is sufficient to cause trachoma (personal communication, Dr. Ian Dadour, entomologist).

Aboriginal people have a high level of mobility between communities. This contributes to the spread of disease. Examination of 'dates of onset of disease' figures and discussions with staff at community clinics, have indicated that the outbreak seemed to have started at the WA/NT border and moved eastwards into the Alice Springs region as well as down into the top of SA.

Mini-outbreaks may occur in different communities. Auxotyping and serotyping of gonococcal isolates from the 1986-87 and 1991 outbreaks identified four different strains of gonococci in one outbreak and four in the other. Only one strain has been identified to date from the Central Australian eye isolates obtained during this outbreak. As no organisms were available for typing from the Kimberley, it is not possible to determine whether this was part of the Central Australian outbreak or was a separate outbreak. Two non-outbreak isolates were tested and found to be different strains. As has happened in previous outbreaks there were numerous sexually transmitted *N. gonorrhoeae* strains, and eight different strains were detected in the sample tested preceding and during this outbreak. It appears that different strains predominate during different times of the year and that any strain is capable of causing an outbreak.

Injectable penicillin or amoxycillin with probenecid worked well during this outbreak to eradicate gonococcal conjunctivitis. The highest penicillin MIC recorded against the isolates of *N. gonorrhoeae* causing this outbreak was 0.5 mg/L, which lies in the less sensitive range.

Another possible site of *N. gonorrhoeae*, though not necessarily involved in transmission to the eye, is oropharyngeal carriage of the organism. One investigation⁹ found three asymptomatic oropharyngeal carriers of *N. gonorrhoeae* in the 1991 gonococcal conjunctivitis outbreak. Two of these carriers were children under 10 years of age. During the same outbreak three individuals had confirmed gonococcal conjunctivitis as well as oropharyngeal carriage

of *N. gonorrhoeae*. It was thought there was autoinoculation of the oropharynx via the nasolacrimal duct. In one report gonococci were detected in the saliva of pharyngeal carriers.¹⁰ Autoinoculation may also occur from purulent eyes to hands to mouth and throat. There may be autoinoculation to the eyes from the throat via saliva. The prevalence of pharyngeal carriage in outbreaks of non-sexually transmitted gonococcal conjunctivitis in Central Australia is unknown.

Oropharyngeal carriage of *N. gonorrhoeae* is more difficult to eradicate than uncomplicated infections at other mucosal sites.¹¹ The cure rate for a single dose treatment with less effective antibiotic regimens is approximately 70%. The most highly effective single dose treatments are likely to cure at least 80% of pharyngeal infections, although the same treatments will cure greater than 95% of uncomplicated anogenital gonorrhoea infections. The failure to eliminate pharyngeal carriage may have three consequences:

1. the probability of a continuing reservoir of infection to further infect the community;
2. the possibility of recurrent infection in the individual; and
3. a possible source of disseminated gonococcal infection (DGI) in an individual.

DGI in a 3 year old boy was documented as a complication in the 1991 epidemic.⁴ Two cases of DGI were diagnosed at the ASH in May 1997. *N. gonorrhoeae* was isolated from the joint fluid of a 14 year old boy and a 26 year old woman. The isolate from the boy was serovar IB3. Unfortunately the isolate from the woman was not kept for testing. Both of these patients had no history of sexually transmitted *N. gonorrhoeae* at the time and both lived in communities which were affected by the outbreak.

Control measures

Central Australian outbreaks have been occurring approximately every five years since 1981. We have enough information to help predict when an outbreak is likely to occur and communities should monitor their populations in these circumstances to detect index cases. If a case is detected, the relevant Public Health and Disease Control Centres should be notified immediately. Interstate Disease Control Centres also need to be notified so all adjacent communities can be informed of a possible epidemic.

Following the collection of the specimen, including a direct smear, treatment should be given to cases and their close contacts prior to laboratory confirmation as some specimens, due to transportation availability, can take up to one week to reach the laboratory. Direct smears are essential for a rapid diagnosis. PCR is more sensitive than either culture or smear but culture is essential for antibiotic susceptibility monitoring.

Once the disease is established, contact tracing can become extremely difficult due to limited local resources. The epidemic is self limiting once the weather becomes colder and the majority of clinical cases have been treated, but sporadic cases can still occur, as can be seen in the Kimberley outbreak (Figure 1). With favourable conditions, a further epidemic could eventuate.

Treatment failures must be detected rapidly. Community members must be educated on the transmission of the disease from eye to eye between children. Poor hygienic practices have been observed in previous outbreaks, for

example, wiping an infected child's eyes and then using the same material to wipe another child's face.⁴

Flies may act as a mechanical vector to establish index cases from reservoirs of *N. gonorrhoeae* and then, along with person to person transmission, contribute to the spread of the disease. However, fly control is virtually impossible during the start of the outbreak because of the extremely high population of flies.

Early recognition and treatment of index cases and identifying and treating contacts is currently the only way of preventing an epidemic. Strategies are in place to detect and treat sexually transmitted *N. gonorrhoeae* in communities. Until this reservoir of disease is controlled gonococcal conjunctivitis is likely to appear again. The role of oropharyngeal carriage of *N. gonorrhoeae* needs to be evaluated further.

Acknowledgments

We would like to thank Nganampa Health Council, Ngaanyatjarra Health Service and community clinic staff; Western Diagnostic Pathology staff and microbiology staff - Pathology Dept, ASH; Virginia Sitzler - Disease Control Centre, Alice Springs and John Tapsall, Department of Microbiology, Prince of Wales Hospital, Sydney.

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Editorial comment

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The above is a comprehensive review of the most recent large outbreak of non-sexually transmitted gonococcal conjunctivitis. It raises several interesting points regarding the circumstances which 'set the scene' for such an outbreak, the possible methods of disease transmission and treatment considerations. Most importantly, however, it highlights to health care providers that childhood conjunctivitis may be caused by *N. gonorrhoeae* which is highly contagious and requires systemic antibiotics. This contrasts with the more common causes of conjunctivitis: allergy, viral infection and infection with the non gonococcal bacteria *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Streptococcus pyogenes* and *Staphylococcus* species; which are usually treated topically.¹

The possibility of *N. gonorrhoeae* needs to be considered in childhood conjunctivitis, especially in areas with endemic high prevalence of venereal gonorrhoea disease and environmental factors which may promote its spread to the eyes. Swabs and cultures should be obtained to establish the diagnosis. The use of air-dried smears to detect Gram negative diplococci is useful for presumptive diagnosis in remote communities where transporting specimens to regional laboratories may be delayed and cultures unsuccessful.²

This outbreak as well as past experience² suggests that one case of non-sexually transmitted gonococcal conjunctivitis should be treated as a potential outbreak. Effective control measures include a standardised approach to case and contact management. Single dose treatment with an appropriate penicillin is recommended^{2,3,4} as it reduces the problem of multidose treatment, especially in a mobile population. Standard and alternative treatment recommendations are shown below. Alternative treatment regimens are indicated for: patients allergic to penicillin; when standard treatment has failed; when infection is known to be due to penicillinase producing *N. gonorrhoeae* (PPNG); or, where a pharyngeal swab has been taken and is positive.

In this outbreak the greatest number of cases were in the 5 to 9 years age group, whereas the highest attack rate in the large 1991 epidemic was in the 0 to 4 years age group. The carers of cases under 10 years need to be advised to monitor the treated child for persisting eye infection, reinfection, fevers or other symptoms such as arthritis. Cases should be excluded from school or child care for 24 hours after treatment has been given. Where neonatal infection is found a full STD screen is indicated for the mother.

Gonorrhoea is a notifiable disease and, in view of the potential for the very rapid spread of this clinical form, gonococcal conjunctivitis should be immediately reported by phone or facsimile to the local Disease Control or Public Health Unit. Early reporting will assist in contact tracing and enable adjacent health services and communities to be informed.

Treatment for gonococcal conjunctivitis

Standard

Neonates < 1 month (Ophthalmia neonatorum)	Admit to hospital urgently for intravenous antibiotics
Older infants, children and adults	Procaine penicillin intramuscularly (IM) as a single dose 50,000 units/kg (50 mg/kg) to a maximum of 1,500,000 units or 1.5g

OR

Amoxycillin plus probenecid as a single dose

Weight	Amoxycillin	Probenecid
3kg to <6kg	500mg	nil
6kg to <10kg	1g	nil
10kg to 15kg	1.5g	250mg
15kg to <20kg	2g	500mg

Alternative

Infants <6 weeks of age	Should not be given ceftriaxone. Refer to hospital
Older infants, children and adults	Weight ≤25 kg Ceftriaxone - 125 mg dissolved in 1% lignocaine hydrochloride, as a single intramuscular dose
	Weight >25 kg Ceftriaxone - 250 mg dissolved in 1% lignocaine hydrochloride, as a single intramuscular dose

Household contacts of cases should be treated with a single dose of a standard treatment or alternative treatment if allergic to penicillin. If a case attends a childcare centre or school the childcare or class mates should be treated. For cases and contacts, and their families, emphasis should be given to thorough hand and face washing, the use of individual clean towels and to ensuring that these are available in affected households or schools.

It is reassuring to know that the penicillin MICs for the Central Australian isolates continue to fall in the fully sensitive to less sensitive range, 0.0125 to 0.5mg/L, and that PPNG has not emerged as a problem in Central Australia and in the NT⁵. Standard treatment, therefore, is still adequate to treat conjunctivitis caused by the current strains of *N. gonorrhoeae*. While recognising the diagnostic role for air-dried smears in remote settings, and for PCR testing as used in the Kimberly, it is prudent to culture and susceptibility test at least sentinel samples during an outbreak in order to monitor the antibiotic MICs.

The place, if any, of pharyngeal carriage as a reservoir for transmission does need to be further considered. However, the ability to control outbreaks with penicillins, and without ceftriaxone or spectinomycin, suggests that pharyngeal

carriage was not an important contributor to this outbreak. Pharyngeal swabs are not routinely recommended in outbreaks unless patients are symptomatic. A preparedness to investigate this issue in the event of another outbreak should be considered.

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Current issues in immunisation

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

This occasional report series in Communicable Diseases Intelligence provides commentary on topical immunisation issues.

Measles vaccine, inflammatory bowel disease and pervasive developmental disorder: is there cause for concern?

Janaki Amin, Peter B. McIntyre, Timothy C. Heath

On 28 February 1998, the *Lancet* published a report of a case series from the Royal Free Hospital, London suggesting a temporal association between measles-mumps-rubella (MMR) vaccine and an apparently new syndrome, consisting of an unusual type of inflammatory bowel disease (IBD) with pervasive developmental disorder (PDD).¹ This report was published with an editorial by Dr Robert Chen,² head of the Vaccine Safety and Development Activity National Immunization Program, US Centers for Disease Control and Prevention, which refuted its conclusion. Despite this, the subsequent intense media attention and public concern has challenged the integrity of the MMR immunisation program in the United Kingdom.

The hypothesis generated by the Royal Free Hospital group is that MMR is associated with IBD, and that IBD is associated with PDD. To examine this further, both microbiological and epidemiological evidence need to be considered.

The microbiological evidence

An association between wild and vaccine strains of the measles virus and IBD has been postulated since 1993.³ Previous studies by the Royal Free group have reported detection of measles vaccine viruses in biopsies from patients with IBD, but other investigators have not been able to reproduce their findings.^{4,5} Using nested polymerase chain reaction (PCR), a much more specific test than those used by the Royal Free Hospital group, Azafal et al.,⁶ could not detect measles virus in the gut mucosal biopsies of patients with Crohn's disease or ulcerative colitis. The recent Royal Free Hospital study provided no evidence of vaccine virus in the bowel, brain or any other tissue of the reported subjects.

The epidemiological evidence

As highlighted in the *Lancet* editorial,² the Royal Free Hospital report is essentially one of hypothesis generation. The study design is a case series and does not enable conclusions to be drawn about causation. In addition, both selection and recall biases are likely to have affected the findings.

In the Royal Free Hospital case series, any association between MMR and IBD is likely to be inflated by selection bias arising from the referral of subjects to a group known to