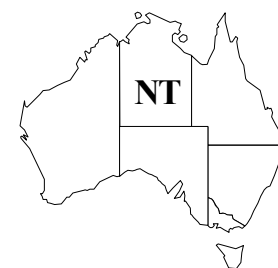




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Outbreak of non-sexually transmitted gonococcal conjunctivitis in Central Australia and the Kimberley region, 13 February to 27 June 1997

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Introduction

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 (NT), Western Australia (WA) and South Australia (SA). The outbreak involved Aboriginal communities predominantly in Central Australia (which included the western Alice Springs region, NT, the Ngaanyatjarra central desert area and the Pitjantjatjara Lands of SA) and the Kimberley region in WA. 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.

Gonococcal conjunctivitis causes considerable morbidity in Aboriginal communities in Central Australia during outbreaks.^{1,2,3,4,5} The disease is characterised by acute painful conjunctivitis with rapid non-sexual transmission between individuals caused by person to person contact.

Bushflies may act as a mechanical vector of *Neisseria gonorrhoeae*. There have been five previously documented gonococcal conjunctivitis outbreaks in Central Australia. These are summarised in Table 1.

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Table 1 Time and size of reported gonococcal conjunctivitis outbreaks in Central Australia

Year of outbreak	Cases*	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

* Includes both clinical and laboratory confirmed cases

Similar environmental factors leading up to and during previous outbreaks were observed with this outbreak. These factors included above average summer rainfall preceding the outbreak, summer changing to winter temperatures, an increase in the percentage of *Haemophilus sp.* 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.

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 when *N. gonorrhoeae* was isolated on culture, detected by PCR or Gram negative diplococci were seen in 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 clinical unconfirmed cases, 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. Information regarding the number of clinical cases was not obtained from the Kimberley.

Laboratory Investigation

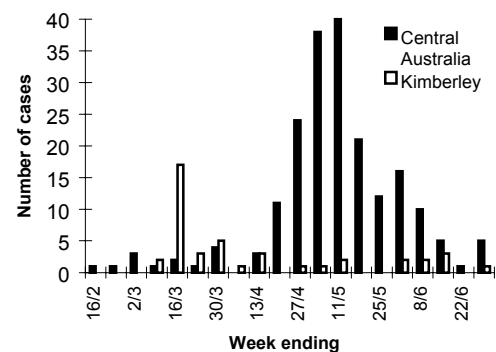
N. gonorrhoeae strains from Central Australia were sent to the Prince of Wales Hospital, Sydney, for serotyping, auxotyping and MIC testing and included 89 eye, 34 genital and 2 joint isolates. Records at the Pathology Laboratory, ASH, were examined to determine the percentage of *Haemophilus sp.* identified from eye swabs between June 1996 and June 1997 and also 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 e.g. joint fluid.

Results

Number of cases

There were 242 confirmed cases (including 5 reinfection cases) and 205 unconfirmed cases of gonococcal conjunctivitis for a total of 447 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 appears to have been two epidemics during the outbreak (Figure 1). The smaller epidemic peaked in the Kimberley during March before a larger epidemic, involving many more communities in Central Australia, peaked in May.

Figure 1 242 laboratory confirmed cases of gonococcal conjunctivitis by week and geographic region, Feb-June '97



Age distribution of cases

Over three quarters of the confirmed cases were children under 10 years old with the greatest number recorded in the 5 to 9 group (Table 2). The youngest child was four months old.

Table 2 Age distribution of confirmed cases

Age group (yrs)	Cases	% of cases
0 to 4	73	30.2
5 to 9	114	47.1
10 to 14	35	14.5
15 and over	12	4.9
Unknown	8	3.3
Total	242	100.0

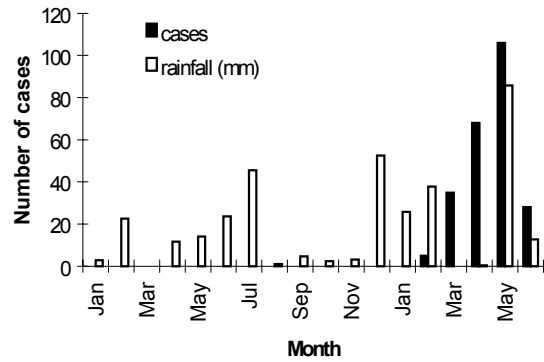
Gender

There were 113 males and 129 females with confirmed infection.

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 (Figure 2). The first cases of the outbreak 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.

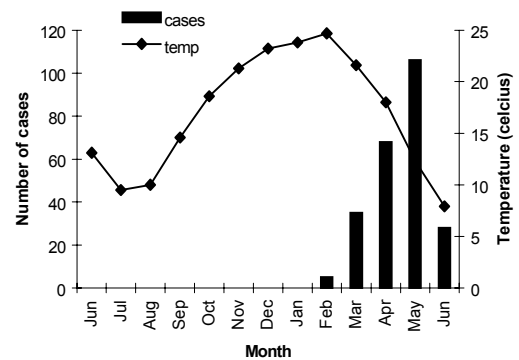
Figure 2 Number of cases compared to the monthly rainfall at Giles, January 1996 to June 1997



Temperature

The mean daily minimum temperature at Giles in February was 24.7°C (Figure 3) at the start of the outbreak and was below 10°C in June at the end of the epidemic. The mean daily maximum temperature in February was 36°C at the start of the epidemic. Typical summer temperatures preceded the outbreak.

Figure 3 Number of cases compared to the mean daily minimum temperatures at Giles for each month, June 1996 to June 1997



Sexually transmitted gonorrhoea in Central Australia

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

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

Using *Haemophilus sp.* as an indicator of the amount of general bacterial/viral eye disease in communities the total number of *Haemophilus sp.*

identified each month from eye swabs was expressed as a proportion of the monthly total number of eye swabs received. The highest proportion (47%) of *Haemophilus sp.* during the thirteen months occurred in March 1997. The mean monthly proportion was 33%. There was a gradual increase in the proportion of eye disease in which *Haemophilus sp.* was isolated at the start of the outbreak.

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

Fly density

Thirteen Central Australian communities involved in the epidemic were contacted and asked when they thought flies were at their worst 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 flies were at their worst in March and April. Four communities had their worst flies in May. Fly density was dramatically reduced in June.

Auxotyping, serotyping and MIC testing from Central Australia

Fifty three eye isolates involved in the outbreak, that have been fully typed, belonged to auxotype/serovar class Wt/IB3. Fourteen STD background isolates obtained during the outbreak were typed and included six Wt/IB3 strains as well as seven other types. Two eye isolates not involved in the outbreak, consisted of two different strains which were not Wt/IB3. Two joint isolates consisted of a Wt/IA4 strain and a IB3 serovar (the Wt/IA4 strain was not involved in the outbreak but included as a background strain). 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. All 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 direction 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 both at the Central Australia Disease Control Coordinating Committee (CADCCC) meeting/teleconference on 9/5/97 and again at the CARPA Conference on 10-11/5/97.

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 amount of clinical disease that was treated. Delays in transport resulted in the death of *N. gonorrhoeae* in swabs and confirmation of infection could only be made from smear and PCR. All the Kimberley cases were tested by PCR.

The sharp peak in the number of Kimberley cases in Figure 1 is artefactual. When Public Health Unit staff arrived at the community many patients had been experiencing symptoms of conjunctivitis for several days or more. Cases had been observed during late January and early February but no swabs had been taken as gonococcal conjunctivitis had not been suspected.

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 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 crop of bushflies, however, further work is still in progress to ascertain whether the number of organisms carried is sufficient to cause trachoma (unpublished work by Dr Ian Dadour, entomologist).

The environmental factors present in this outbreak were similar to previous outbreaks, including: 1) heavy summer rainfall at least one month before the first cases appeared, 2) a mean daily minimum temperature above 20°C for the first two months of the outbreak, 3) eye infections caused by *Haemophilus sp.* increased to the highest monthly percentage for the year and 4) an extremely high fly population occurred in February, March and April. Sexually transmitted *N. gonorrhoeae* cases did not increase before the outbreak.

Factors which contributed to the outbreak include: a) *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. b) The breeding conditions, including humidity and temperature, for the bushfly were ideal⁹ just after the rainfall in December and then deteriorated markedly in June. Bushfly pupae cannot survive at all when the temperature fluctuates between 6°C and 18°C. c) Small adult flies were present (indicating a reduced larval stage) during the outbreak and there was extreme pressure for available protein necessary for the breeding cycle and moisture/food for survival. Flies were theoretically attracted to moist, pussy eyes and became 'sticky' flies.

In addition to these factors there is a large reservoir of sexually transmitted *N. gonorrhoeae* in remote Aboriginal communities in Central Australia.⁷

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 investigator⁶ found three asymptomatic oropharyngeal carriers of *N. gonorrhoeae* in the 1991 gonococcal conjunctivitis outbreak. Two of these carriers were children under ten 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 pussy eye to hand to mouth and throat. There may be conjunctival autoinoculation to a child's throat and transmission via droplet to another child's eye. 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.⁸ Single dose treatment with less effective antibiotic regimens will achieve an approximate 70% cure rate. The most highly effective single dose treatments are likely to cure at least 80% of pharyngeal infections, although the same treatments will cure > 95% of uncomplicated anogenital gonorrhoea infections. The failure to eliminate any pharyngeal carriage which may be present has three consequences: a) the probability of a continuing reservoir of infection to further infect the community; b) the possibility of recurrent infection in the individual; and c) 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.

There is a great deal of mobility of Aboriginal people between communities. This contributes to the spread of disease. Examination of dates of

onset of disease figures and talking to staff at community clinics has 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.

There is the possibility of mini-outbreaks 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. So far there has been only one strain identified from the Central Australian eye isolates obtained during this outbreak. There were no organisms for typing from the Kimberley. Two eye background isolates (not contributing to the outbreak) were tested and found to be different strains. As has happened in previous outbreaks there were numerous sexually transmitted *N.gonorrhoeae* background strains and eight different strains were detected in the sample tested preceding and during this outbreak. It appears that different strains are predominant during different times of the year and any strain is capable of causing a gonococcal outbreak.

Control measures

We have enough information to help predict when an outbreak is likely to occur (see table 3) and communities should monitor their populations in these circumstances to detect index cases. Central Australian outbreaks have been occurring approximately every five years since 1981. 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 sensitivity monitoring.

Once the disease is established contact tracing can become extremely difficult due to limited local resources. The disease is self limiting when it becomes colder and the majority of

clinical cases have been treated, but sporadic cases can still surface (note epidemic curve of the Kimberley outbreak) and if conditions become favourable 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 e.g.~~ 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* has to be evaluated further.

Table 3 Factors which may predict outbreaks of gonococcal conjunctivitis

-
1. Heavy above average summer rainfall.
 2. Mean daily elevated minimum temperature of 20°C following summer rain in early autumn.
 3. Increase in *Haemophilus sp.* eye infections.
 4. Large fly populations.
-

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

The above article is a welcome review of a recent outbreak of gonococcal conjunctivitis and the discussion raises many points regarding the disease, its transmission and treatment to consider. Most importantly, however, it highlights to health care providers the fact that childhood conjunctivitis may be caused by *Neisseria gonorrhoeae* which is highly contagious and requires systemic antibiotics. This is in contrast to the more common causes of conjunctivitis - allergic, viral and the non gonococcal bacterial, ie *Haemophilus influenzae*, *Streptococcus pneumoniae*, *S. pyogenes* and *Staphylococcus* species which are usually treated by topical antiseptics or in more severe case, topical combination antibiotic drops or eye ointment¹. So, *N. gonorrhoeae* does need to be considered in conjunctivitis especially in areas where there is endemic high prevalence of venereal gonorrhoeal disease and environmental factors which may promote spread to the eyes. Swabs and cultures must be taken to establish the diagnosis. The NT Centre for Disease Control (CDC) endorsed protocol for individual management and public health response is provided below. It was being prepared as the outbreak evolved and it was formulated after consultation with various centres involved in past outbreaks (eg in WA, Alice Springs and Katherine) and after

examining a number of protocols. It generally still reflects past informal NT CDC control measures.

The paper supports that one notified case of non-sexually transmitted gonococcal conjunctivitis should be treated as a potential outbreak and responded to as per the protocol including treating household contacts or childcare or school contacts. Emphasising thorough hand and face washing and making sure that there are provisions for this in affected households or schools is important. Knowing the penicillin MICs for the Central Australian isolates is very reassuring. Procaine penicillin or amoxycillin with probenecid are still very adequate to treat the recurrent gonococcal conjunctivitis being transmitted. The knowledge that in general penicillinase producing *N.gonorrhoeae* has not emerged in Central Australia and the NT is also reassuring. The isolates fall within the fully or less sensitive range (see Table) and will respond to standard treatment.² It is prudent, however, to culture and susceptibility test at least sentinel samples during an outbreak to monitor MICs. The question of what place, if any, pharyngeal carriage has in acting as a reservoir for transmission, does need to be considered but the ability to control outbreaks with penicillin or amoxycillin with probenecid rather than ceftriaxone would make this appear less important. A preparedness to investigate

this in the event of another outbreak should be considered.

Table Penicillin MIC and response to treatment²

• Fully sensitive to penicillin, MIC \leq 0.03 mg/L	Usually respond to standard treatment
• Less sensitive to penicillin, MIC 0.06- 0.5 mg/L	Usually respond to standard treatment
• Relatively resistant to penicillin, MIC \geq 1 mg/L	Usually fail to respond to penicillin
• Penicillinase producing <i>N. gonorrhoeae</i> (PPNG)	Usually fail to respond to penicillin

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Guidelines for the Control of Gonococcal Conjunctivitis**Background**

Gonococcal conjunctivitis is a highly contagious eye infection caused by *Neisseria gonorrhoeae*. It is a notifiable disease. Cases are rarely seen in isolation in the Northern Territory (NT) and follow up and treatment of contacts is essential.

Case Definition***Clinical features***

A clinical illness characterised by intense inflammation of the conjunctivae, copious purulent discharge with or without periorbital oedema.

Case definition

Neisseria gonorrhoeae detected on culture of a conjunctival specimen.

OR

Positive diagnosis using a molecular technique such as PCR (or LCR).

OR

Clinically compatible illness *and either*

- gram negative intracellular diplococci visible on microscopy of a conjunctival specimen

OR

- epidemiologically linked to a laboratory confirmed case. This includes any other proven case in a remote Aboriginal community.

Clinical Picture

Gonococcal conjunctivitis is usually a localised infection of the conjunctivae, however corneal ulceration, perforation and blindness can occur if treatment is not given promptly. More disseminated infection including gonococcal arthritis has been reported in the NT.

Neonatal infection (ophthalmia neonatorum) is a potentially more serious clinical picture than gonococcal infection seen in older children and adults, and the mode of transmission is different, which means that management is different.

Mode of Transmission

In children and adults transmission is either direct from person to person from contaminated fingers, or indirect transmission from contaminated fomites, or from flies. Heavy rains and an increase in fly numbers may precipitate an outbreak.

Neonatal infection occurs during passage through the birth canal.

Incubation

Usually 2 to 7 days, but sometimes can be longer.

Period of Communicability

May extend for months in untreated people. Infectivity ceases within hours of appropriate antibiotic therapy. Patients should be isolated/excluded from school for 24 hours after treatment.

Diagnosis

Swabs should be taken from all patients with a discharging eye for microscopy, culture and sensitivity.

- Wet the swab with sterile normal saline and swab the eye.
- Roll the swab on a slide and let the slide air dry.
- Place the swab in Stuart's transport medium.
- Do not refrigerate.
- If there is a delay in receiving the specimen in the laboratory, culture may fail to grow *Neisseria gonorrhoeae*, however gram negative intracellular diplococci will be visible on microscopy, and is diagnostic.

Management of Cases

- Collect a swab as above.
- Irrigate the eyes with saline solution to remove the discharge.
- If possible check visual acuity, and check cornea for ulceration. If abnormal refer to local doctor for management.
- Treat with single dose antibiotics (see below).
- Encourage frequent washing of face and hands with warm water and soap.
- Warn families to represent if there is persisting eye infection, fevers or other symptoms such as arthritis.

- Exclude from school or child care for 24 hours after treatment.
- Report to the Centre for Disease Control (CDC).
- Treat contacts (see below).
- If a neonatal infection, do a full STD screen on the mother and then treat.

Treatment

Standard

Neonates: < 1 month (ophthalmia neonatorum)

⇒ **Admit to hospital urgently** for intravenous antibiotics.

Children and adults:

⇒ Procaine penicillin intramuscularly (IM) as a single dose

50,000 units = 50 mg/kg (to a maximum of 1,500,000 units = 1.5g)

OR

⇒ Amoxicillin plus probenecid as a single dose

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

Alternative

For cases who are allergic to penicillin, or where the standard treatment has failed, or if infection is known to be due to penicillinase producing *Neisseria gonorrhoeae* (PPNG) alternative treatment should be used. Failed treatment is defined as no substantial decrease in discharge, conjunctival injection or peri-orbital oedema within 24 hours of treatment.

Additionally, if pharyngeal swabs have been taken and are positive, alternative treatment should be given. In an outbreak situation,

pharyngeal swabs are not routinely recommended unless symptomatic.

The above cases should be treated with **ceftriaxone**:

- ⇒ Weight ≤ 25 kg: 125 mg dissolved in 1% lignocaine hydrochloride, as a single intramuscular dose.
- ⇒ Weight > 25 kg: 250 mg dissolved in 1% lignocaine hydrochloride, as a single intramuscular dose.

Note: Infants below 6 weeks of age should not be given ceftriaxone. Refer to hospital.

Management of Contacts

- Treat all household contacts of a case with a single dose of the standard treatment of procaine penicillin or amoxicillin with probenecid (or alternative treatment as above, if allergic to penicillin).
- For those with symptoms/signs follow the steps taken for management of cases.
- If the case attends a child care or school, then those in the same classroom need treatment.
- Encourage good personal hygiene and regular washing of face and hands with water and soap.

Meningococcal disease - 2 NT cases in August 1997 - The view from CDC

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Case 1

On 1 August 1997, the Friday afternoon of the start of a three day weekend (NT Picnic Day Monday 4 August) a case of suspected meningococcal disease was notified to CDC Katherine by an experienced, long serving, local GP.

The patient

A 4 year old female vomited the previous day once at pre-school and was described by her mother as being "a bit flat". The child complained of a sore elbow, shoulder and left leg but no headache. She had a fever overnight and vomited 3-4 times. The morning of admission, 1 August, she complained of feeling "dizzy" and her head "hurting". During the course of the morning she developed a purple rash on her legs and became comatose during the short drive to the hospital. Over the next 3 hours while being resuscitated by Emergency Department staff the rash spread to cover her trunk, arms and face but became more pink and less typically purpuric. There was no neck stiffness and negative Kernig's signs. She received a stat dose of penicillin and IV ceftriaxone. She was admitted to hospital under her local GP and placed in respiratory isolation. Her past history revealed that she

was fully immunised including Hib. The clinical picture was very suggestive of meningococcal disease. One reportedly inadequate blood culture was obtained prior to antibiotics. A skin lesion aspirate was cultured post antibiotic. No lumbar puncture was attempted as within hours the patient was showing signs of improvement. Blood and skin lesion cultures showed no growth and urine antigen tests were negative. Of interest, no nasopharyngeal swab was done. The patient made a full recovery within a week. No serum was available to send for further meningococcal serology investigation.

The public health response

The NT CDC definition of a contact for invasive meningococcal or *Haemophilus influenzae* disease is:

1. Any person who has spent 4 hours or more a day for 5 consecutive days or more than 24 hours with the index case in the week prior to the onset of illness.
2. Any person who has significant contact with the nasopharyngeal secretions of the index case during the previous week.

The current NH&MRC Guidelines case definition of meningococcal meningitis requires laboratory confirmation. In a small town with a public health minded GP, the local Disease Control Unit was promptly notified even before the possibility of laboratory confirmation. The case was convincing as a highly probable meningococcal meningitis. The child had close contacts with children in a pre-school setting, neighbourhood playmates and a long weekend was approaching with children likely to disperse and a decision was needed. Initially a list of contacts was drawn up. At 24 hours with no growth from blood cultures or skin and after consultation with the CDC Darwin and RDH Infectious Diseases Specialist it was decided to inform the contacts about meningococcal disease, give out an information pamphlet and dispense rifampicin prophylaxis to 22 children and 7 adults and ceftriaxone to a pregnant contact. A newspaper article on meningococcal disease appeared in the local paper the following week. At six weeks post this probable meningococcal case there have been no secondary cases.

Case 2

On 12 August 1997 at 1 pm the CDC Darwin was notified of a suspected case of meningococcal disease by the public health conscious Emergency Department at Royal Darwin Hospital.

The patient

A 14 year old male boarder student at a local Darwin high school became unwell on the evening of 11 August with fever, vomiting, diarrhoea and a headache. He had first complained of headache at 3 pm that afternoon. He was taken to a private hospital at 8 pm with a provisional diagnosis of viral illness and dehydration. IV fluids and paracetamol were given and he was sent home. He again presented at 9 am the following morning, 12 August, with persistent fever 38°C, vomiting, photophobia, drowsiness, headache, generalised aches and pains and diarrhoea. A widespread purpuric rash had developed. There was no neck stiffness and a Kernig's sign was negative. A provisional diagnosis of meningococcal meningitis was made and IM ceftriaxone given. He was transferred to the Royal Darwin Hospital, admitted and placed in respiratory isolation and commenced on IV

ceftriaxone and penicillin. The patient had no significant medical history and no recent illnesses other than a "blocked nose". He had not been out of Darwin since the school holidays 5 weeks previously and had only left the school on weekends to stay with a friend's family. One member of that family had been ill with fever and mild abdominal cramping the preceding week which was diagnosed as a viral illness by the family GP. Blood cultures and a small amount of CSF were obtained after antibiotic treatment and vesicular fluid from a skin lesion associated with the rash was cultured. The patient did not have a nasopharyngeal swab. CSF and blood cultures were negative, however fluid from the skin lesion grew *Neisseria meningitidis* at 24 hours. The isolate was sent to the National Neisseria Network and serogroup identification is still pending. The isolate was fully sensitive to penicillin. The patient made a full recovery within a week.

The public health response

By 8 pm on the evening of 12 August before the isolation of *N.meningitidis*, 111 people had been identified who had met the above NT CDC definition of a contact for prophylactic rifampicin. These included dormitory residents, house parents, a football team who had shared water bottles, the family the patient boarded with on the weekend and his classmates. Additionally 4 people insisted they be considered as contacts despite minimal contact and assurance that their level of risk was minimal. 20 contacts considered most at risk according to the degree of contact were medicated on the evening of admission. These consisted of the patient's room mates, the family he stayed with on the weekend and houseparents and others who had come in contact with vomitus etc on the evening of 11 August. A decision was made to wait until laboratory confirmation was obtained before rifampicin was given to the remaining contacts. Confirmation was received at 10 am 13 August. By 4 pm that day, all remaining contacts had received the first dose of rifampicin. A letter from CDC, an information pamphlet on meningococcal disease and rifampicin were provided to all contacts and their families. There were numerous calls to CDC and the school nurse from concerned parents, friends and school staff. Information

sessions were held with staff and students at the school. A notice was prepared for the school's newsletter at the principal's request and a press release prepared for the local media. Staff involved in the public health response included the school nurse, 3 pharmacists and 2 CDC staff. It had taken at least 70 personnel hours in contact tracing, education and medication distribution. The cost of rifampicin was approximately \$3000.

Discussion

These two cases have raised a few issues in regards to the NH&MRC October 1996 endorsed Guidelines for the Control of Meningococcal Disease in Australia¹, which are generally very thorough and useful. The issues are also timely as the NT Guidelines for Meningococcal Meningitis/Septicaemia Prophylaxis are currently being revised.

The first issue is the definition of disease. Both the NH&MRC case definition and the NT CDC current definition of meningococcal disease require laboratory confirmation, ie isolation of *N.meningitidis* from a normally sterile site or detection of meningococcal antigen in joints, blood or CSF or detection of gram negative intracellular diplococci in blood or CSF. There is no provision for probable cases. The USA CDC case classification² allows for a probable case where there is a positive antigen test in CSF or clinical purpura fulminans in the absence of positive blood cultures. The UK Meningococcal Working Group³ define meningococcal disease as confirmed, probable or possible. Confirmed disease includes microbiological confirmation and probable cases include a clinical diagnosis of meningococcal meningitis/septicaemia without microbiological confirmation where the Disease Control or Public Health Specialist in consultation with the clinician managing the case consider that meningococcal disease is the likeliest diagnosis. Confirmed and probable cases require intervention. The first case presented meets these definitions of probable meningococcal meningitis deserving intervention. The current NH&MRC Guidelines do not address a probable category and therefore no public health intervention is considered. As earlier and appropriate patient treatment occurs laboratory confirmation will be obtained less frequently.

When meningococcal infection is suspected, particularly where actual or insipient shock is evident, immediate empiric therapy in the absence of formal diagnosis is indicated. Treatment should commence immediately before transfer to hospital and not be withheld until *N.meningitidis* or another organism has been identified. This is particularly important with patients with a haemorrhagic rash.¹

There is a need to at least address what to do when there is no microbiologic confirmation.

The second issue follows from the first. Even when a probable case category exists emphasis should still be placed on obtaining laboratory evidence. A full range of specimens (beyond CSF) for testing should be taken on every case suspected of meningococcal disease. The traditional lumbar puncture to obtain CSF is now frequently deferred until therapy and supportive measures have been established and investigations such as a CT have ruled out increased intracranial pressure. Alternative diagnostic specimens therefore are important. Collection of blood for culture should be attempted prior to antibiotics (but not delay antibiotics) and a throat/nasalpharyngeal swab should always be obtainable. Unfortunately on page 1 of the NH&MRC Guidelines, the fifth point reads "There is no indication for use of throat/nasalpharyngeal swab in routine management". This actually should go on to read "in routine contacts" and is misleading. On page 10, under "Patient Management", subsection, "Diagnostic Studies" the correct advice is given: "All patients with suspected meningococcal infection should have blood and a throat/nasalpharyngeal swab collected as soon as possible for culture". The public health physician's role on notification of a case is to recommend to the managing clinician that a throat swab be taken if antibiotics were given to the patient before referral to hospital or if the organism has not been cultured from a normally sterile site. Additionally, a collection of fluid from skin lesions for meningococcal culture or presence of gram positive diplococci should be encouraged. Routine microbiological surveillance for meningococcal infections

should include the serogroup and antibiotics sensitivity of all isolates of *N.meningitidis* and isolates should be referred to the National Neisseria Network (NNN) to confirm identification, antibiotic sensitivities and provide further characterisation. As the public health response to an outbreak is also influenced by these findings some timeliness in reporting is expected. This becomes more of an issue when there is more than one case. Participation in projects to explore the utility of other means of enhancing laboratory diagnosis eg newer or novel tests should be encouraged.

A third issue is the definition of a contact. The NT definition has historically been the same for meningococcal and *Haemophilus influenzae* disease and differs somewhat from the 8 point NH&MRC definition on page 33. Possibly they complement each other.

A final issue which has come up in revising the NT Guidelines both for meningococcal and for gonococcal conjunctivitis contacts is the dosing for ceftriaxone, should this alternative to rifampicin be required. The dosage of 5 mg/kg as per the Antibiotic Guidelines⁴ is impractical to measure in low doses and the NH&MRC Guidelines are somewhat confusing and based on age rather than weight.

The simplest rational method would appear to be to give 250 mg dissolved in 1% lignocaine hydrochloride, as a single intramuscular (IM) dose for those over 25 kg and 125 mg dissolved in 1% lignocaine hydrochloride, as a single IM dose in those 25 kg or under, recognising that it is not to be used in infants below 6 weeks of age.

The revised NT Guidelines for Meningococcal Meningitis/Septicaemia Prophylaxis will be published in the next *Bulletin*.

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El Nino - ? drier wet season, but still expect melioidosis and other "tropical" infections

Bart Currie, Royal Darwin Hospital and Menzies School of Health Research

Melioidosis (disease from infection with *Burkholderia pseudomallei*) predominantly occurs during the wet season. However the amount of rainfall does not definitively predict the number of melioidosis cases. With the record wet in 1996/97, between November and April, there were 24 Top End cases of melioidosis with 5 deaths plus an additional case of non fatal relapsed melioidosis associated with non-adherence to eradication therapy. From May to August 1997 there have been 2 new cases and 2 relapsed cases, all non fatal.

Over the last eight years the average number of cases and deaths per year was 23 and 5, with 181 cases and 40 deaths overall. Even with the predicted drier wet season ahead, melioidosis is likely to still be the commonest cause of fatal community-acquired septicaemic pneumonia at Royal Darwin, Gove and Katherine Hospitals.

It will once again be important for staff new to our Top End hospitals and to urban and rural health centres in the Top End to be made aware of melioidosis and other infectious diseases uncommonly seen in southern Australia.

Other details on melioidosis

1. Apart from pneumonia, other presentations of melioidosis include skin abscesses or ulcers, abscesses in internal organs such as prostate, spleen and liver, fulminant septicaemia with multi-organ abscesses and unusual neurological illnesses (such as brainstem encephalitis). Individuals without symptoms or a known history of disease have also been found to be serologically positive.
2. While melioidosis can occur in children and healthy adults, almost 40% of cases are diabetic and 40% are excessive alcohol consumers. Excessive kava drinking also appears to be associated with melioidosis.
3. The likelihood of diagnosis is increased by using selective culture media (modified Ashdown's broth), frequent sampling (sputum, throat, rectal and ulcer swabs) and collection of blood cultures. Clinicians should liaise with laboratory staff to ensure selective media are available, including for remote communities.
4. Mortality is decreased by early diagnosis and appropriate antibiotic therapy.
5. Follow up of cases and adherence to eradication therapy (usually at least three months of antibiotics after discharge) is critical to prevent relapse, which can be fatal.
6. The Top End empirical treatment protocol for adult community-acquired pneumonia is devised to cover both melioidosis in patients with risk factors, as well as other important pathogens (Table over).
7. Once melioidosis is confirmed the treatment recommended is:

Initial INTENSIVE therapy for usually 14 days or more of:

- intravenous high dose ceftazidime
plus either
- high dose cotrimoxazole
or
- high dose doxycycline.

This is followed by ERADICATION therapy for at least three months of:

- oral monotherapy with either high dose cotrimoxazole or doxycycline.

Examples of other infections of particular local interest include:

- i. Scrub typhus from Litchfield Park - the only known NT focus.
- ii. Murray Valley Encephalitis - no case since 1993, but a nasty disease with mortality over 25% in those with encephalitis - will periodically "break-out" of its endemic northern Western Australia - Top End NT mosquito-animal cycles to cause human cases sporadically or in clusters or a larger epidemic. Most infections are actually subclinical.
- iii. Barmah Forest Virus - although increasing as a cause of fever, rash and polyarthritis in southern Australia, the first true epidemic was described from Nhulunbuy in the 1991/92 monsoon.
- iv. Cryptococcal meningitis - should be considered in anyone with persistent severe headache, especially in remote Aboriginal communities. The "tropical" variant, *Cryptococcus neoformans var gattii*, occurs in overtly immunocompetent people and may also present as a lung mass(es) on CXR. The connection with eucalypts remains to be sorted out in the NT.
- v. Crusted (Norwegian) scabies - often the source of community or hospital outbreaks of scabies due to the thousands, even millions, of mites in the hyperkeratotic skin plaques. Requires intensive therapy and infection control measures.
- vi. Community-acquired ("tropical") *Acinetobacter baumannii* pneumonia - unusual (1-2 cases/year Top End), usually wet season and fatal and almost always in alcoholics. Presents as fulminant lobar/total lung pneumonia.

Because of melioidosis and *Acinetobacter* pneumonia, while recognising that *Streptococcus pneumoniae* is the commonest overall cause of pneumonia (and second to melioidosis as a documented cause of death in the Top End), the following is the Top End protocol for initial therapy of adult community-acquired pneumonia. With the wet season approaching please make new staff aware of this.

Table Initial therapy of adult community-acquired pneumonia in the Top End¹

	Mild pneumonia	Moderate pneumonia	Severe pneumonia
No risk factors ² present	Penicillin	Penicillin	Ceftriaxone
Risk factors ² present	Penicillin	Ceftriaxone plus gentamicin	Ceftriaxone or ceftazidime plus gentamicin

1. For 'atypical pneumonia' consider adding erythromycin.

2. Risk factors include: alcohol, diabetes, chronic lung disease, chronic renal failure and steroid therapy.

Guidelines for Community Control of Scabies and Skin Sores

Background

Scabies and skin sores are one of the commonest problems seen in health centres.

Scabies sores often become infected. Severe scabies contributes to malnutrition in children, because their body needs extra energy to heal the skin sores.

The high rates of streptococcal skin sores cause Acute Post Streptococcal Glomerulonephritis (APSGN), which may be severe with swelling of the face, high blood pressure and dark (blood stained) urine. It can also be very mild with haematuria (blood) found just on urine test. Research in the Northern Territory (NT) suggests that frequent mild episodes of APSGN in children are contributing to high rates of kidney disease in adults. High rates of skin infection allow the streptococcus to remain circulating in communities, leading to the extremely high rates of rheumatic fever and rheumatic heart disease seen in the NT.

Treating individuals, or even whole families for scabies will not be successful in reducing community rates. Up to 50% of children are infected in some communities and people get reinfected very quickly. This causes continual frustration for community members and health staff.

Community Scabies Program

A successful community program to control scabies has occurred in one Top End community over the past two years.¹ This was based on a successful program devised to control scabies among Kuna Indians in Panama.² A simple form of those programs

would help to reduce and control scabies in other communities.

There are three requirements:

1. Community support and education.
2. Single community scabies treatment of all residents at the same time.
3. Maintenance program involving:
 - simple screen of all children less than 15 years of age, three times per year to check skin for scabies and skin sores
 - ongoing community education and evaluation of the program

1. Community support and education

Treating all community members at the same time regardless of whether an individual has scabies is the only effective method to significantly reduce the high prevalence rate. Most communities will need extra staff to assist with community screening and treatment. Although this involves a large commitment of time and effort by local health staff and the community, it will show results within a short time and significantly reduce the clinic work following the scabies program.

Health staff education

Health staff should arrange education sessions for themselves:

- to ensure everyone understands the issues
- to ensure staff are confident in diagnosing scabies

- to obtain appropriate educational materials for community education
- to organise the community screening and treatment program

Community support

Discuss issues with key leaders and decision makers to enable them to recognise:

- the serious short and long term outcomes of continuing high rates of scabies
- the high rate of success with full community involvement

Ask council members, community elders, teachers, Health Boards, Arts Centres, CDEP workers, Woman's Centre members, outstation resource centres and other appropriate people to take the message to their families.

Community education

Education is essential to promote full participation. AHW's and other local trusted health staff are the best people to provide this education. Arrange school and community education sessions and include information on personal health practices, such as regular hand and face washing, daily bathing, toothbrushing and washing of clothes and linen.

Resources

The issue of resources is currently being looked at by CDC, Rural Services, Health Promotion and Environmental Health. The plan is to develop educational materials such as posters, videos, booklets and pamphlets to assist community staff in providing this program. Funding will also need to be established to cover costs such as the scabies treatment, publicity materials and transport of extra staff.

2. Initial screen and community treatment

Arrange community screening and treatment. The time taken will depend upon the size of the community and number of outstations. Allocate at least one week.

There are two main reasons for screening:

1. To establish the baseline prevalence rate for your community which provides a comparison at follow-up screening.

2. To determine which individuals have infected sores and need antibiotics.

Who to screen

- Children are the main group that must be seen, as they have the highest rates of infected scabies.
- It is desirable but not essential to screen adults, but if adults are *not screened* it is important to ensure that *all adults* as well as children are *treated*.

How to screen

- Organise resources prior to screening (eg staff, scabicide).
- Organise all equipment prior to screening day.
- Organise appropriate screening area (eg school, clinic, Woman's Centre).

Screening

Scabies:

- Look for small papules and scratch marks around web spaces between fingers and toes and around wrists. Children often have scabies papules on elbows, armpits and trunk. Babies can have widespread scabies including the head, and pustules on their palms and feet. Adults mostly have lesions in web spaces. Scabies is almost always itchy. Document if scabies present with a simple Yes/No and record presence of moderate to severe scabies, as these people need repeat treatment.

Skin sores:

- Document presence of sores (Yes/No) only for infected sores. Do not include non-infected cuts, scratches or insect bites. Sores will be moist or have pus, or have a yellow/brown crust. Treat all people with sores with a single intramuscular (IM) dose of benzathine penicillin (or erythromycin for 10 days if allergic to penicillin).
- Permethrin can be applied at the time antibiotic treatment is given. There is no need to wait for healing, as permethrin has very low skin irritation.

Age groups:

- Young children: check all of skin, including scalp.
- School children: check hands, arms, legs, feet and waist. Only check rest of skin if scabies or sores noted, or if itching is present on other parts of the body.
- Adults: check hands, arms and feet, unless scabies or sores found.
- Document and offer treatment for other skin problems (eg ringworm).

Treatment

- Give out scabies treatment to the *whole community* at the same time.
- Health staff should visit households to demonstrate the correct way to use the cream by treating some young children.
- Use 5% permethrin cream for everyone older than 2 months. Permethrin is recommended because it is simple to use and has low rates of skin irritation. (For babies under 2 months, see below). Everyone should treat themselves late in the afternoon or evening by applying permethrin cream from head to toe, ensuring the whole body is covered, avoiding eyes and mouth. *Previously we only treated young children head to toe, but in endemic areas many older children and adults have scabies on their head and neck.* The cream should be left on overnight (8-12 hours) and washed off in the morning.
- Repeat scabies treatment in two weeks for all people with moderate to severe scabies. This includes infants with pustules on hands and feet, and other people with multiple scabies lesions.
- Babies younger than 2 months of age can be treated with sulphur 5% cream daily for 2 to 3 days or crotamiton 10% cream (Eurax) daily for 3 to 5 days, depending on how quickly they respond. Wash off and reapply the cream once each day. Do not use permethrin as it may be absorbed and cause problems.

People with crusted scabies (Norwegian scabies) are very infectious as they have

thousands of scabies mites on their skin. These people usually need initial hospital treatment and their houses need special cleaning (see Crusted Scabies protocol).

Reducing the high rate of scabies depends upon treating all possible human hosts at the same time. Cleaning of sheets, blankets and clothes and treatment of mangy dogs is not essential for success. However, community members may wish to encourage these activities as part of personal health practices and to improve dog health.

3. Maintenance Program

It is important to continue screening children's skin regularly over the long term, until endemic scabies has disappeared from Aboriginal communities. If health staff don't continue four monthly skin checks, the prevalence of scabies will slowly increase until it returns to pre-treatment levels.

Screening

- screen all children one month after community treatment
- then screen all children three times per year (this can be combined with regular de-worming in the Top End)
- treat any skin sores with penicillin (or erythromycin if allergic) and
- treat any child with scabies AND all family members living with them

The clinic and school could decide on three screening dates at the start of each school year. Ideally, this screening could be done on the same three dates within a region eg 1st week in February, June and October.

Individual clinics should decide how they will combine maintenance skin checks with the school screening program.

Community education

In order to maintain community interest:

- evaluate the success of your program and feedback the information on a regular basis to community decision makers such as Councils, Woman's Centres, community elders and teachers

- encourage community residents to present early for treatment of scabies or skin infections
- encourage community residents to refer visitors to health centre for early treatment continue positive promotion of the benefits of personal health practices; and encourage community councils to lobby for improved "health hardware" ie appropriate housing, showers, toilets, washing machines

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Editorial

Following discussions at a recent Menzies School of Health Research streptococcal symposium attended by international experts, the recommended treatment of scabies for individuals has been changed. These new recommendations will appear in the next edition of the Antibiotic Guidelines, but all staff should adopt these changes now. The changes are:

1. Infected scabies: treat with permethrin and antibiotics **at the same time.**
Permethrin causes very little irritation on broken skin, so the 2 day delay is not required.

2. Treat all adults and children with scabies **from head to toe.**

In tropical and endemic areas, scabies lesions are frequently found on the scalp and behind ears in adults as well as young children.

3. Retreat people with moderate to severe scabies **in two weeks.**

Treating in one week is too early, as some eggs may not have hatched and will not be killed by the permethrin.

Contact CDC, Darwin on 8922 8044 for a copy of the guidelines including Appendices 1-3.

Non-Communicable Diseases Update: No.3

Message: Cholesterol reduction: base your primary prevention strategy on overall cardiovascular risk, and emphasise non-drug options first

Tarun Weeramanthri, Community Physician, CDC, Darwin

In the March edition of the *Communicable Diseases Bulletin*, the importance of aggressive drug treatment of hyperlipidaemia in those known to have cardiovascular disease was stressed. This article looks at the evidence for more aggressive primary prevention strategies (both drug and non-drug) in those asymptomatic individuals at risk of, but without currently diagnosed, cardiovascular disease.

The WOSCOP Study

The results from the West of Scotland Coronary Prevention Study (WOSCOP) were reported in late 1995.¹ The study was restricted

to middle aged men. 6595 men with no history of myocardial infarction were randomly assigned to pravastatin or placebo and followed up for an average of 4.9 years. There was an approximate 30% reduction in definite coronary events and death from coronary heart disease in the pravastatin group. The reduction in coronary events was independent of baseline levels of LDL cholesterol. Overall mortality was 22% lower in the pravastatin group, but this was just short of statistical significance (p=0.051). Strangely, this was not a pure primary prevention study, since men with angina (5% of the study population) were not excluded. The results of this trial are

supportive of a more aggressive approach to primary prevention, but it should be remembered that the study population had a high average baseline total cholesterol of 7.0 mmol/L and LDL of 5.0 mmol/L. In addition, no women were included. So the study results cannot be directly extrapolated to the whole population.

A subsequent analysis of six trials of primary prevention, including WOSCOP, has estimated that one would have to treat 53 patients without known atherosclerosis with lipid-lowering drugs for five years to prevent one additional non-fatal infarction or cardiac death, compared to only 16 patients with known atherosclerosis.²

In the March edition of the *Bulletin*, it was noted that the cost effectiveness of giving lipid-lowering drugs to those with known coronary disease had been estimated by the Scandinavian Simvastatin Survival Study group to be \$A 12,400 per year of life saved, comparable to bypass grafting for the usual indications. In those without coronary disease, the cost per year of life saved is estimated to be at least four times that amount.³

The Cholesterol Screening Debate

The WOSCOP results, however, have not settled the issue of who should be screened for high cholesterol levels.⁴ For example in the United States, the American College of Physicians recommends only limited screening, primarily for middle-aged men, or those with two coronary risk factors, whereas the National Cholesterol Education Program recommends that all adults over the age of 20 years know their cholesterol level. The National Heart Foundation (NHF) of Australia has adopted a similarly aggressive position.⁵

Proponents of limited screening point out that clinical trials have not been done in young adults and that the number of women included in trials has been small. They are worried about the costs of cholesterol-lowering medications and the possibility of over-prescription driven by a rising consumer demand. For example, the NHF of Australia's 'Guide to Plasma Lipids for Doctors' recommends that drug therapy be considered in asymptomatic people with a total cholesterol over 6.5 mmol/L but only if they have other risk factors and dietary therapy has not worked after 6-8 weeks. How many people would that apply to in Australia? Well, the

1989 NHF Risk Factor Prevalence Study found that, in adults aged 20-69 years, 16% of men and 14% of women had levels of 6.5 mmol/L or more.⁶ Treating large numbers of people with drugs would be expensive and the opportunity costs would be large. Any benefits that could theoretically accrue based on the trial data would also be lessened in real life, if the high discontinuation rates (60% after 12 months) seen in one Australian study were to prevail.⁷

Proponents of widespread screening point out that knowledge of cholesterol levels does not automatically lead to drug therapy and that measuring one's cholesterol may reinforce behavioural change by focusing attention on diet, smoking and other cardiac risk factors, although they concede that the magnitude of any such changes are likely to be modest.⁸ In any case, if drug treatment is needed, it can yield as much benefit as the treatment of mild and moderate hypertension, and the numbers in the population needing treatment are similar to those with hypertension.

Proponents of widespread screening also stress that the distinction between primary and secondary prevention is not especially relevant as those most likely to benefit from primary prevention are most likely to have advanced but asymptomatic atherosclerosis at the time treatment is started. There is a risk in waiting for symptoms to develop as in approximately 25% of patients with atherosclerosis, sudden death is the first manifestation.

Assessing overall cardiovascular risk

All in all, the approach to primary prevention of cardiovascular disease through lipid-lowering therapy in Australia is changing and becoming more aggressive. As previously mentioned, the recently updated NHF guidelines recommend that all adults, excluding pregnant females, should have their total cholesterol, HDL cholesterol and triglycerides measured, and proposes lower treatment targets than the PBS guidelines.

The proposal for universal testing irrespective of predicted risk is contentious, but the principle of assessment of overall risk prior to drug prescription is now agreed upon and has been incorporated into the PBS guidelines first published in 1994. Paradoxically, in deciding whether to treat elevated cholesterol levels in

the asymptomatic individual, the level of cholesterol is not the only or even the most important factor. Instead of treating the isolated cholesterol level, one should base the decision to treat on an assessment of the overall cardiovascular risk, taking into account age, gender, family history and other risk factors such as diabetes, hypertension and smoking.

There are an increasing number of risk charts being produced to help practitioners calculate the overall cardiovascular risk for an individual. For example the Sheffield risk table identifies those with a risk of coronary death over 1.5% per year based on age, gender, cholesterol concentration and the presence of hypertension, smoking, diabetes and left ventricular hypertrophy on ECG.⁹ However, there is considerable uncertainty as to how to integrate such risk tables into routine clinical practice and how to incorporate other relevant information such as client preferences, cost of treatment, years of life gained, and probability of adverse events.¹⁰

In the future, more effective non-drug therapies are needed that could be applied in population interventions to reduce hypercholesterolaemia. Effective population interventions to increase exercise and activity levels, and to change national dietary intakes of salt and saturated fats must be developed. There are also emerging options such as using plant sterols, that are known to inhibit cholesterol absorption, dissolved in margarine.¹¹

We await further trials of primary prevention of coronary heart disease in groups other than middle-aged men. Meanwhile there is a need to focus on risk factor modification, and development of expertise in tailoring dietary and activity advice to an individual's personal circumstances and in offering options such as smoking cessation. The preferences and values of the patient must be taken into account whenever lipid-lowering therapy is considered. Any decision to start and monitor a medication, that may need to be continued over a lifetime, needs to be made jointly.

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Acute respiratory illness in 2 Darwin schools

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Towards the end of May 1997, Darwin Centre for Disease Control (CDC) received reports of an illness characterised by a sore throat, runny nose, fever, lethargy, cough and occasionally vomiting and diarrhoea, affecting large numbers of staff and students in Darwin schools. An investigation was undertaken of staff with the objectives of identifying the illness, establishing if it was an influenza outbreak, documenting the impact on the two schools and determining whether any public health action could be taken.

A case was defined as a member of staff who had experienced a sore throat plus 2 or more of the following symptoms any time since 1 May 1997: fever, runny nose, cough, lethargy, generalised aches and pains, vomiting, diarrhoea, nausea, chills or rigors and weakness. People meeting the case definition had blood taken for influenza A and B, adenovirus, mycoplasma, respiratory syncytial virus (RSV) and parainfluenza. If acutely unwell, a throat swab was also collected for influenza cultures.

School A (n=26) had 84% and School B (n=47) had 91% of staff participate in the investigation. The remaining staff were on leave at the time. School A had 14 staff (63%) and School B had 26 staff (60%) who met the case definition. In addition to the sore throat, a cough, runny nose and weakness were reported most frequently. Fever was reported by 45% of all staff. The median length of illness was 3 days (range: 2-42 days).

School A had 43% and School B had 50% of staff off work for a median of 2 days (range: 1-12 days). There was a total of 50 days of lost productivity in staff alone. Student absenteeism in one school ranged from 15-75% throughout May. Fifty three percent of all staff had family members who had illnesses similar to their own in the same time period.

Blood was collected from 11 staff members in School A and 18 in School B. No throat swabs were obtained as no staff were acutely ill at the time of the investigation. No staff had viral titres considered diagnostic for acute infection, however levels suggestive of past or early infection were found in 80% of ill staff, with

RSV and parainfluenza 3 being the most common.

Unfortunately a specific organism was not identified, but we are reasonably certain this illness was not Influenza A or B. The impact on the schools, however, was significant. There had been considerable disruption to school routine and class progress. In addition, the attack rates in staff's families suggest the illness may have been widespread in the community.

While results were inconclusive, benefits have arisen from this investigation. Firstly, it was an avenue for promoting CDC's role in outbreak investigations, the potential public health importance of acute respiratory illness (ARI) outbreaks and the reasons for early notification. School staff are in a position to alert public health authorities if an unusual health event occurs so that, when appropriate, immunisations and environmental investigations can be implemented and, where possible, the public can be informed and reassured.

Secondly, public education regarding the prevention and control of communicable diseases was delivered. Inservices were given to provide feedback and answer questions. There was a particular interest in the modes of transmission of viral illnesses and in how they could be controlled in the school setting. The sessions provided a forum for discussing the transmission of acute respiratory illness (ARI), pushing the general immunisation message, emphasising the benefits of healthy diets, exercise, not smoking, handwashing, covering one's mouth when coughing or sneezing, staying home when unwell, and for encouraging staff to alert CDC early whenever they are concerned about illness in their environment.

Active campaigns would be beneficial in these environments at the start of each year. While ARIs will not be eradicated, raising awareness of the public health implications of disease may facilitate early public health initiatives (eg immunisation campaigns) and encourage institutions to consider strategies for reducing the impact of these diseases in their environments. These strategies could include the promotion of handwashing and encouraging staff to stay at home when ill.

Hepatitis C Community Awareness Campaign

Naomi Oliver, AIDS/STD Unit, CDC, Darwin

It is thought that hepatitis C is greatly underdiagnosed and that the current hepatitis C notifications are only the tip of the iceberg. Dr. Alex Wodak (Director, Alcohol and Drug Services, St. Vincent's Hospital, Sydney) estimates that between 50,000 to 200,000 people Australia wide are infected with the virus. Unlike hepatitis B, only 20% of people, once infected, will eliminate this virus from their body. Currently there is no vaccine or cure and treatment is only effective and available for a small percentage of people. Since testing began in 1990 to 26 August 1997, there have been 1,352 notification in the NT - 56 in the last two months.

The Hepatitis C Network NT report that many people who are hepatitis C positive do feel unsupported by the community and medical organisations. Fear of discrimination by employers, insurance companies and others who are ignorant of the facts, is often a complaint. There are still a lot of unanswered questions. The results of a small survey show that whilst most people have heard about hepatitis C, there is little understanding within the community of the virus, its transmission modes and its consequences.

In January of this year staff of the AIDS/STD Unit, Alcohol and Other Drugs and other key organisations decided to conduct a community awareness campaign for the purposes of increasing knowledge of hepatitis C and providing the community with an avenue through which they could access further information. Staff working on this project were mindful of the possible stigma which can be associated with hepatitis C and carefully planned materials and media information.

The focus of the campaign was the implementation of a hepatitis help line using a 1 800 number that is available Territory wide. During the first month, when the majority of the advertising occurred, the line was staffed from 9am - 5.30pm. Calls were monitored and approximately 50 calls came in within the first

9 days. As was expected, calls have tailed off, and the line is now staffed for two hours per day on an ongoing basis. Members of the public can ring between 9am - 11am or alternatively leave a message for their call to be returned.

Levels of transmission are the highest amongst people who have injected drugs. Accurate figures are difficult to ascertain, but it is estimated that after two years of injecting drugs, approximately 66% of people will be Hepatitis C positive, and 100% after 8 years of injecting drugs. Dr. Wodak estimates that there is one new hepatitis C infection per hour in Australia amongst people who inject drugs (8,000-10,000 estimated new infections per year in injecting drug users).

Other high risk groups include young people in general, and prisoners, both possibly reflecting use and experimentation with injecting drugs. In the southern states a high proportion of prisoners are convicted for drug related crimes. The percentage in the NT is smaller, but the overall rate of hepatitis C in NT prisons is still about 9 times higher than in the general community. For these reasons part of the campaign focuses on the three identified groups. Specifically targeted messages have been placed in venues which these risk groups are likely to frequent, and face to face education is occurring via different agencies and Territory Health Services (THS).

The campaign was launched on Wednesday 6 August by The Minister for Health, Family and Children's Services, The Hon Denis Burke. The event attracted considerable interest from the media and was attended by approximately 40 people from a number of non government organisations as well as THS personnel.

Future work will focus on education and prevention amongst the target groups specified above.

The help line number is 1 800 353 755

Chris Nagy, CDC, Darwin

The Federal Government's *Immunise Australia* campaign was launched on 28 July 1997. The campaign aim is to increase the level of age appropriate immunisation within the community by raising awareness to the benefits of immunisation and to provide Australians with the information they need to make informed immunisation decisions. In addition, three *National Immunisation Days* have been designated to offer the public immunisation at locations and times which may be more accessible. These dates are three Saturdays, 2 August, 4 October and 6 December 1997.

Territory Health Services has participated so far in the campaign by holding immunisation clinics at Casuarina Community Care Centre, Katherine Health Centre and the Palmerston Shopping Centre on the 2 August. The clinics were supported by national and local advertising and shopping centre displays in the week leading up to the immunisation day.

Attendance and public response to the clinics was pleasing, with 60 adults and children being immunised and many more requesting information. Immunisation rates in the Northern Territory are lowest in the urban and urban satellite areas, hence this is where the campaign is being focussed. Providing

accessible Saturday clinics should work to improve coverage in the target groups e.g. the children of working parents or parents with transport difficulties and for adults. Regular weekday baby clinics are provided at the Casuarina Community Care Centre in the northern suburbs, however, Saturday clinics are not usually available. Provision of a Saturday immunisation clinic at Katherine Health Centre was very successful and well attended. At Palmerston, a vacant shop within the shopping centre was transformed into a clinic and it was by far the busiest clinic on the day. Users described its convenient location as its main advantage. A full report is available from CDC on 8922 8487.

Increasing public awareness to immunisation through advertising and taking immunisation to the public in their preferred locations and time should ultimately fulfil the goals of this campaign. A full evaluation of the Program is being undertaken by the Commonwealth.

Immunisation clinics are planned for 4 October at Gove Infant and Maternal Health Centre, Palmerston Shopping Centre, Tennant Creek and Alice Springs shopping areas. The final National Immunisation Day locations are yet to be finalised.



**NT NOTIFICATIONS OF DISEASES BY DISTRICTS
1 APRIL TO 30 JUNE 1997 AND 1996**

DISEASES	ALICE SPRINGS		BARKLY		DARWIN		EAST ARNHEM		KATHERINE		TOTAL	
	'97	'96	'97	'96	'97	'96	'97	'96	'97	'96	'97	'96
Acute Rheumatic Fever	2	1	0	0	1	8	0	1	0	2	3	12
Adverse Vaccine React.	1	2	0	0	2	3	1	1	2	0	6	5
Arbovirus infections												
Barmah Forest Virus	4	1	5	0	7	4	0	2	4	0	20	7
Kunjin Virus	0	0	1	0	0	0	0	0	0	0	1	0
Dengue	0	0	0	0	0	1	0	0	0	0	0	1
Ross River Virus	35	0	10	4	40	17	1	1	6	2	92	24
Campylobacter	40	3	1	2	15	51	2	0	4	9	62	65
Chlamydia	62	48	11	5	67	53	21	22	8	35	169	163
Cong.Syphilis	0	0	0	0	0	0	0	0	0	1	0	1
Donovanosis	2	3	0	1	0	0	0	1	0	0	2	5
Glomerulonephritis	0	0	0	0	2	0	0	0	0	0	2	0
Gonococcal Disease	125	72	13	7	141	29	27	23	34	40	340	171
Gonococcal Conjunct.	93	0	2	0	0	0	0	1	0	0	95	1
Haemophilus Inf type b	1	0	0	0	0	0	0	0	0	0	1	0
Hepatitis A	17	3	3	1	8	10	0	2	13	6	41	22
Hepatitis B	1	2	4	0	2	0	0	0	4	1	11	3
Hepatitis C (prevalence)	8	9	0	1	65	62	0	0	2	2	75	74
Hepatitis D	1	0	0	0	0	0	0	0	0	0	1	0
HIV infections	0	0	0	0	2	2	0	0	0	0	2	2
HTLV-1	6	9	0	1	0	0	0	0	0	1	6	11
Leprosy	0	0	0	0	0	2	0	2	0	0	0	4
Malaria	1	1	0	0	11	4	2	0	2	0	7	5
Measles	0	0	0	0	0	0	1	0	0	0	1	0
Meningococcal Infect.	1	0	0	0	2	1	2	1	0	0	5	2
Mumps	0	0	0	0	1	1	0	0	1	0	2	1
Pertussis	0	2	0	9	3	0	0	0	0	0	3	11
Pneumococcal Disease	9	3	0	2	9	5	1	0	0	2	19	12
Rotavirus	3	14	0	5	0	12	0	0	3	6	6	37
Rubella	1	0	0	0	1	1	0	0	0	0	2	1
Salmonella	22	20	6	11	39	50	3	8	17	18	87	107
Shigella	22	9	6	2	3	7	0	4	3	5	34	27
Syphilis	32	17	2	0	8	10	7	11	15	11	64	49
Tuberculosis	1	2	0	0	10	4	0	0	2	2	13	8
Typhoid	1	0	0	0	0	0	0	0	0	0	1	0
Yersiniosis	0	0	0	0	1	0	0	0	0	0	1	0
Total	491	221	64	51	440	337	68	79	120	143	1183	831

Points to note regarding notifications:

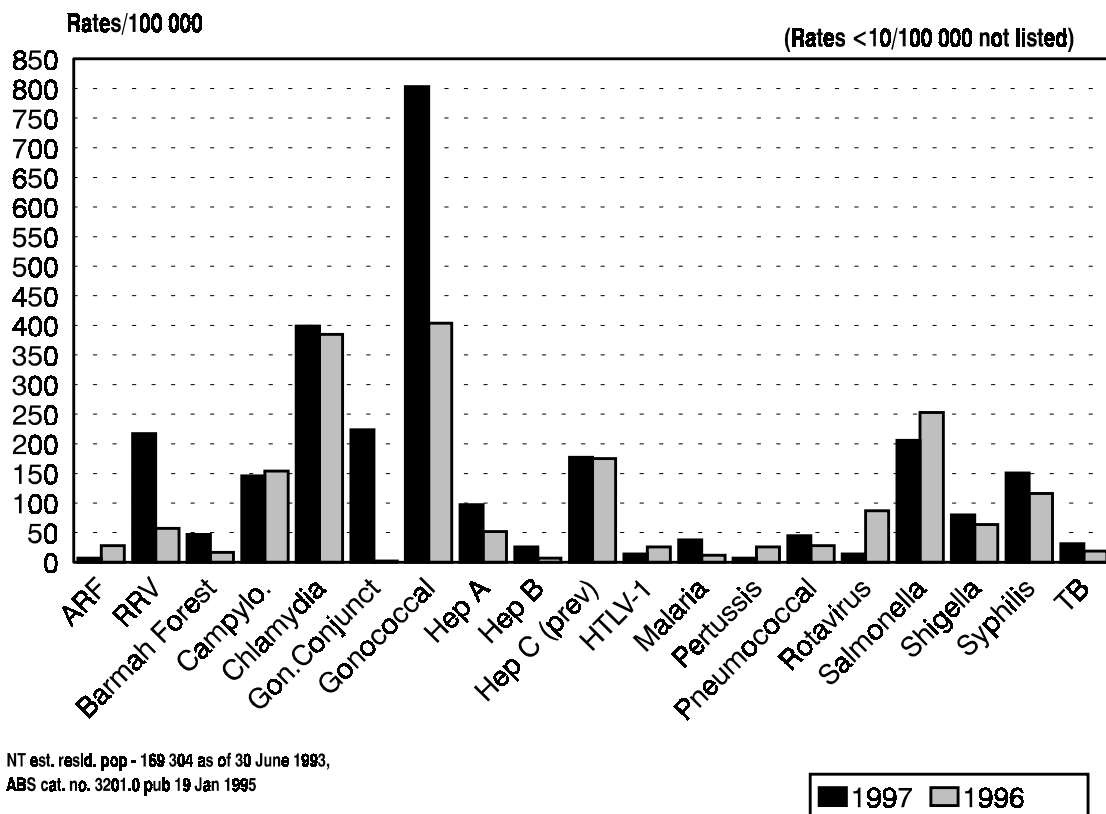
- Australian Encephalitis (MVE,) Amoebiasis, Botulism, Brucellosis, Chancroid, Cholera, Congenital Rubella Syndrome, Diphtheria, Hepatitis C (incidence), Hepatitis E, Hydatid Disease, Legionnaires Disease, Leptospirosis, Listeriosis, Lymphogranuloma venereum, Poliomyelitis, Typhus and Viral Haemorrhagic Fever are all notifiable but had "0" notifications in this period.
- Rotavirus increased in early July 1997 and therefore is not reflected in this quarter.
- Arboviruses - The unusual amount and late rain in the Alice Springs and Barkly areas contributed to the highest RRV and Barmah Forest notifications recorded since 1991. Though not reflected in our notifications due to travel - 1 case of MVE and 3 cases of Kunjin were acquired in these areas during this period. Late rains in the Top End also showed higher numbers in April to June than in previous years.
- Gonococcal disease in Darwin increased due to inclusion of positive results of tampon testing after 1/7/96 and possibly wider PCR screening. For Alice Springs the increase is due to more screening including improved techniques for antenatal specimen collection, and opportunistic STD testing.
- Gonococcal conjunctivitis increase shows the outbreak as reported in the first article.
- Hepatitis A - A major outbreak in a child care centre in Alice Springs (12 cases) accounts for the increase in notified cases for this quarter. The increase in Katherine is less clear as all were sporadic unrelated cases except for one family which resulted in 4 cases.

Notified cases of Vaccine Preventable Diseases in NT by Report Date 1 April to 30 June 1997 and 1996

DISEASES	TOTAL		No. cases among children aged 0-5 years	
	'97	'96	'97	'96
Congenital rubella syndrome	0	0	0	0
Diphtheria	0	0	0	0
<i>Haemophilus influenzae</i> type b	1	0	1	0
Hepatitis B	11	3	0	0
Measles	1	0	1	0
Mumps	2	1	0	0
Pertussis	3	11	1	2
Poliomyelitis, paralytic	0	0	0	0
Rubella	2	1	1	0
Tetanus	0	0	0	0

- Mumps is largely under-reported.
- Hepatitis B increase was across all regions

NT wide Notifiable Diseases 1 April to 30 June 1997 and 1996



MALARIA NOTIFICATIONS, NORTHERN TERRITORY

April to June 1997

Compiled by Peter Knibbs and Mervyn Fairley, CDC, Darwin

Seven notifications of malaria were received for the second quarter of 1997. The following table provides details about where the infection was thought to be acquired, the infecting agent and whether chemoprophylaxis was used.

ORIGIN OF INFECTION	REASON EXPOSED	AGENT	CHEMO-PROPHY-LAXIS	COMMENTS
PACIFIC				
PNG	Business visit	<i>P.vivax</i>	No	Travels regularly between NT and PNG
PNG	Holiday	<i>P.vivax</i>	Yes	
PNG	PNG resident	<i>P.falciparum</i>	Yes	Vague history of chemoprophylaxis. In Darwin for Arafura Games.
ASIA/SE ASIA				
Indonesia	Illegal immigrant	<i>P.falciparum</i>	No	Travelled by boat from Malaysia to Kupang.
Indonesia	Indonesian resident	<i>P.vivax</i>	No	Indonesian fisherman detained in Australian waters.
Indonesia	Holiday	<i>P.vivax</i>	No	On Roti for four days. Son diagnosed with malaria in Adelaide.
Indonesia	Diving holiday	<i>P.falciparum</i>	Yes	Symptoms developed 5 weeks after return.

STAFF UPDATES

Sandy Thompson is the new medical officer in the Population Health Unit in Alice Springs, having most recently been working at Macfarlane Burnet Centre for Medical research in their Epidemiology and Social Research Unit. She has a wide range of interests in public health, particularly in immunisation and infectious diseases epidemiology.

Michael Howard recently joined the Sexual Health Unit in Alice Springs as the Donovanosis Project Officer. The project aims to establish a donovanosis register, increase notification rates and ensure individuals receive effective treatment and follow-up. He has previously worked in Katherine and Utopia as a Remote Area Nurse.

Angela Merianos has returned to her position as head of Immunisation and Surveillance, CDC Darwin, after spending 6 months working on a hepatitis E project in Nepal.

Fay Johnston, who was acting in Angela's position has gone on 2 months leave and **Tania Wallace**, TB/leprosy medical officer, CDC, Darwin has recently commenced 3 months maternity leave. **Sarah Huffam** (formerly the infectious diseases and microbiology registrar, RDH) is filling in for Tania for the three months.