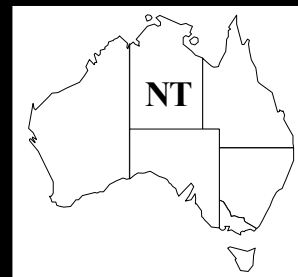




# THE NORTHERN TERRITORY DISEASE CONTROL BULLETIN



ISSN 1440-883X

Vol. 7, No. 4, December 2000

## Two cases of leptospirosis diagnosed in the Royal Darwin Hospital

*Marilyn Hassell, Medical Registrar, Royal Darwin Hospital*

Two confirmed cases of leptospirosis were diagnosed in the Royal Darwin Hospital in November 2000. Both cases contracted the disease in areas surrounding Darwin that had received significant rainfall, and may herald a growing number of cases during the coming wet season.

### Case 1

A 57 year old Chilean man resident in Darwin presented on 4 November having been unwell for two days. His symptoms were high fever with chills and sweats, headache, dry cough, mild generalised abdominal pain and nausea, conjunctival injection and aches in his knees and ankles. Seven days prior to presentation, he had been goose hunting at Harrison Dam, where he had walked barefoot through sharp grass, sustaining multiple cuts, and then through muddy water. He had no relevant past medical history.

On examination, he was sweaty and looked unwell with a temperature of 39.4°C, heart rate of 80 and regular and BP of 90/60. Conjunctival suffusion was present. There was no meningism. Cardiopulmonary examinations were unremarkable. Abdominal examination revealed mild right upper quadrant tenderness with no hepatosplenomegaly. There was no rash, joint tenderness or swelling or lymphadenopathy. There were multiple superficial scratches of the skin of both lower legs.

Investigations revealed a normal WCC of  $9.2 \times 10^9/L$  (4.0-11.0), mild neutrophilia of  $8.6 \times 10^9/L$  (2.0-7.5), lymphopaenia of  $0.2 \times 10^9/L$  (1.5-4.0), and thrombocytopaenia with a platelet count of  $123 \times 10^9/L$  (150-450). Renal function was normal

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with urea 5.9mmol/L(3.0-8.0) and creatinine 104umol/L(60-120), but urine microscopy revealed 10-40 WBC and 40-100 RBC with no bacteria grown. Liver enzymes were mildly abnormal with bilirubin 43umol/L(0-20), ALP 180U/L(39-117), ALT 49U/L(5-44) and GGT 268U/L(0-60). C-reactive protein was significantly raised at 281mg/L (<10).

The man was feeling much improved in the Emergency Department following hydration with IV normal saline. He was admitted to the medical ward with a provisional diagnosis of a viral infection, however was commenced on doxycycline 100 mg bd due to the possibility of leptospirosis. The next day, he remained unwell with ongoing high spiking fevers and right upper quadrant pain. An abdominal ultrasound was performed which showed no evidence of acute biliary changes, and no morphological hepatic abnormality. He was commenced on intravenous benzyl penicillin in addition to the doxycycline to treat severe leptospirosis infection. On 6 November, two days following admission, the man became afebrile, felt constitutionally much improved though he had some residual debilitating joint pains.

By 10 November, the CRP and platelet count had normalised. The liver enzyme abnormalities peaked on this date, with bilirubin 22, ALP 337, ALT 99, GGT 493 but were returning to normal by 20 November, with ALP 149 and GGT 117. By this stage, a ten day course of doxycycline had been completed.

Serology taken on 5 November for influenza, parainfluenza, Ross River Virus, Barmah Forrest Virus and Rickettsial infections were all negative, as was throat swab PCR for influenza and parainfluenza. Cultures for melioidosis were negative and melioidosis IHA titre was 1:20. Blood cultures were negative. Microagglutination titre for leptospirosis taken on 5 November was also negative, however convalescent serology taken on 20 November was positive for leptospirosis with a titre of >6400 for the serovar *australis*. At this time the patient was almost fully recovered.

## Case 2

A 20 year old Caucasian man, living and working on a farming property in Virginia (rural Darwin), presented on 16 November with a one week history of being unwell. His initial symptom was headache without photophobia, which persisted for two days followed by fever with sweats and chills, dry cough, nausea, dysuria and red eyes. There were no myalgias, arthralgias or rash. The patient stated his

place of work was inhabited by many animals, including feral pigs, cows and kangaroos, and that he frequently had close animal contact. In addition, he would wade through stagnant water, without wearing any protective clothing. The area had experienced significant amounts of rain in the weeks prior to presentation.

On examination, the man was febrile at 38.2°C with heart rate of 78 regular and BP of 115/50. Cardiopulmonary examinations were unremarkable. Abdominal examination revealed a palpable liver edge 2 cm below the costal margin with mild right upper quadrant abdominal tenderness. There was no rash or meningism. There were multiple breaks in the skin around the feet and toenails.

Investigations showed normal renal function, but mildly deranged liver enzymes with bilirubin 16, ALP 272, ALT 76, GGT 241. Urinalysis was also abnormal with RBCs and WBCs detected, but no nitrites and no bacterial growth on culture. Hb was normal at 135, WBC 7.1 with neutrophils 4.7 and lymphocytes 1.4, and a mild thrombocytopenia of 110. Chest X-ray was normal.

The patient was treated with IV normal saline hydration, and oral doxycycline 100 mg bd. The next day he was afebrile and feeling well, and was discharged from hospital the day following admission with advice to return in two weeks for convalescent serology testing.

Hepatitis A and B and Rickettsial serology were negative, as were blood cultures, urine, throat swab and rectal swab cultures for melioidosis, and melioidosis IHAT titre was 1:20. Urinary PCR for *Chlamydia trachomatis* and *Neisseria gonorrhoea* were also negative.

The diagnosis of leptospirosis was made with the initial microagglutination titre taken on 17 November being strongly positive for leptospirosis at 3200, again for the serovar *australis*.

## Discussion

These cases highlight the presence of leptospirosis, a ubiquitous zoonosis caused by the spirochaete *Leptospira interrogans*, in the Northern Territory. There is a need to be mindful of the illness in the patient with a febrile illness with exposure to animals or wet environmental conditions. Both of these cases were infected with the serovar *australis*, which is characteristically associated with severe disease, being the type most often implicated in cases of pulmonary haemorrhage, and together with the serovar *zanoni*, accounting for the majority of hospitalisations for leptospirosis in Queensland.<sup>1,2</sup>

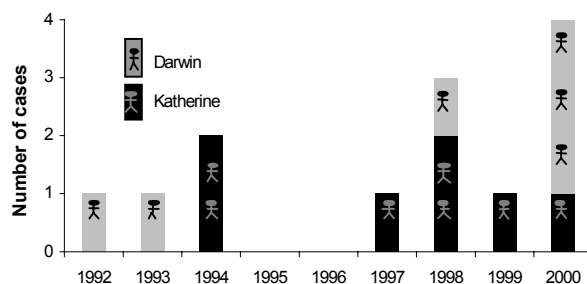
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## Editorial comment

From 1992 to December 2000 (see Figure) there have been 13 cases of leptospirosis notified in the Northern Territory (NT). Of note, all have been males aged 21 to 60 years (median age 34 years), 11 were non-Aboriginal, one was Aboriginal and the ethnicity of a 1993 case was unknown. Most cases (7) were from the Katherine area with the remaining 6 from the Darwin area. Occupation or 'risk activity' for acquisition of disease has not been systematically collected, however anecdotally, several Katherine cases were involved in activities around the Katherine Gorge – as tourists or guides.

**Figure NT leptospirosis notifications 1992 to December 2000**



This year, as of 8 December, 4 leptospirosis cases have been notified in the NT. In addition to the 2 cases reported above, a 45 year old Aboriginal male, tug-boat worker and duck shooter, contracted leptospirosis in late September 2000 and required 6 days hospitalisation in the Darwin Private Hospital. He presented afebrile, tachypnoeic, normotensive, with a tender epigastrium and a 5 day history of diarrhoea and vomiting. He was hyponatraemic (Na 129mmol/L) and hypokalaemic (K 2.8mmol/L) and had high serum urea and creatinine (13.7mmol/L and 226µmol/L, respectively). His gammaGT was high (165U/L) and total protein low (59g/l). He was mildly anaemic (Hb 113g/l); thrombocytopenic ( $92 \times 10^9/L$ ); mildly neutrophilic ( $8.4 \times 10^9/L$ ); and his ESR was elevated (125mm/hr). He was started on IV fluids and metronidazole. Apart from occasional rigors early in his admission, his gastrointestinal

and electrolyte disturbances steadily settled over a few days and he was discharged home with no further treatment. A positive leptospirosis IgM EIA (3.6) and an elevated Microscopic Agglutination Test (MAT) for leptospirosis were later reported satisfying the new standards proposed by the Public Health Laboratory Network for a notifiable leptospirosis case. The patient believes he contracted the disease while shooting at either Harrison Dam or Fogg Dam. He received no specific treatment for leptospirosis but recovered. Interestingly, the hunting partner of Case 1 above who was also duck shooting at Harrison Dam reported a similar leptospirosis-like illness, however further details are not yet available. Earlier in the year, (August 2000) a 43 year old male abattoir worker from Katherine was notified with leptospirosis making him the 4th reported case to date.

Of the 3 recent notified cases, 2 (and a possible 3<sup>rd</sup> suspect case) were duck hunters at the Harrison Dam - Fogg Dam area. This area is home to the 'dusky rat' (*Rattus colletti*), a native rat which collectively constitutes a higher biomass of herbivores than is found in the Serengeti in Africa. Leptospirosis is an occupational hazard to those working on the land or with animals and a recreational hazard to bathers, campers and sportsmen in infected areas. Contact of the skin (especially abraded skin) with water, wet soil or vegetation that is contaminated with urine of infected animals (notably rats, pigs, cattle and dogs) constitutes the main mode of transmission of leptospirosis. The need for those dealing with animals and this environment to wear protective footwear and clothing is highlighted and practitioners need to be aware of the disease. Parks and Wildlife and Environmental Health are aware of the cases and health alerts will be distributed to hunters, tourists and workers in the area.

Acknowledgements to Dr Mark Douglas, Dr Dale Fisher and Dr Sid Selva-Nayagam for their input to this report and also to Dr Bill Freeland and Bill Binns from Parks and Wildlife for the 'dusky rat' information.

## Melioidosis

### The Top End prospective study continues into another wet season and An update on treatment guidelines

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Despite the unusually dry start to this wet season, there were five cases of melioidosis, none fatal, in October-November 2000. The Top End prospective melioidosis study has now documented 294 culture-confirmed cases in the 11 years since October 1989, with 53 deaths (18%). During the 1999/2000 wet season there were 40 cases of confirmed melioidosis, with four deaths in the Top End. There were also two cases, one fatal, from central Australia.

#### Information about melioidosis

1. The bacterium, *Burkholderia pseudomallei*, is an environmental organism found in soils and water across the Top End. Most infection is thought to be acquired through percutaneous inoculation, although inhalation and ingestion are also possible.
2. Until new therapies recently became available it was the commonest cause of fatal community-acquired bacteremic pneumonia at RDH (and possibly also Katherine and Gove Hospitals).
3. Pneumonia is the commonest presentation of melioidosis. As well as severe septicaemic pneumonia with mortality often over 50%, many patients present with milder forms of pneumonia, which respond well to appropriate antibiotics. Other presentations of melioidosis include skin abscesses or ulcers, abscesses in the internal organs such as the prostate, spleen, kidney and liver, fulminant septicaemia with multi-organ abscesses and unusual neurological illnesses such as brainstem encephalitis and acute flaccid paraplegia.
4. Persons without symptoms or a known history of disease can also be found to be positive on serological testing, indicating asymptomatic infection. A small proportion of these people can "re-activate" from latent infection many years later in life, analogous to tuberculosis. However re-activation represents probably less than 5% of Top End cases, with the vast majority of presentations following infection during the current wet season.
5. The incubation period has been ascertained from the Top End study to be 1-21 days, with a mean incubation period of 9 days.
6. Epidemiological data shows that after periods of very intense rainfall, incubation period can be shorter and severity and mortality of melioidosis can be increased.
7. Diabetes is the most important risk factor for melioidosis, with around 40% of cases being diabetic. In addition, excessive alcohol consumption, chronic renal disease, chronic lung disease and excessive kava drinking are risk factors for melioidosis. While the majority of patients with melioidosis have one or more of these risk factors, melioidosis can also occur in children and healthy adults. However severe disease and death are extremely rare in people without identified risk factors.
8. 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.
9. Early diagnosis and appropriate antibiotic therapy decrease mortality.
10. Follow-up of cases and adherence to eradication therapy (usually at least 3 months of antibiotics after discharge) are critical to prevent relapse, which can be fatal.
11. Each monsoon, cases of melioidosis occur in travellers returning from tropical Australia to southern states or overseas countries.
12. Public education remains very important so that wherever possible people avoid contact with wet season soils or muddy water. Wearing footwear and the use of gloves whilst gardening or working outdoors are very important measures to avoid possible exposure. These preventive measures are especially important to emphasise for all diabetics.

## The NT Melioidosis Treatment Protocol

The Top End empirical protocol for adult community-acquired pneumonia is devised to cover melioidosis in patients with risk factors, as well as other important pathogens (see page 6 this *Bulletin*).

Once melioidosis is confirmed the usual treatment recommended is:

**Initial intensive therapy for at least 14 days with:**  
intravenous high dose ceftazidime or meropenem  
plus  
high dose cotrimoxazole.

This is followed by:

**Eradication therapy for at least 3 months of:**  
oral monotherapy with high dose cotrimoxazole.

Durations of intensive and eradication therapy may need to be prolonged in deep-seated infections, bone, joint and CNS infections.

In patients in ICU with melioidosis septic shock, a G-CSF protocol has been associated with decreased mortality.

## Recent References from the NT study

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Clin Infect Dis 2000;31:981-986.

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## Antibiotic protocol for adult community acquired pneumonia in the Top End

Bart Currie,<sup>1,2,3</sup> Dale Fisher,<sup>2</sup> Nick Anstey<sup>2,3</sup> and Gary Lum<sup>2</sup>

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The prime aim of treating community-acquired pneumonia is to prevent death.

Table 1 shows the four commonest organisms isolated in 255 cases of adult community-acquired bacteremic pneumonia treated at Royal Darwin Hospital (RDH). As elsewhere in the world, *Streptococcus pneumoniae* is the commonest organism overall. However, *Burkholderia pseudomallei*, the organism causing melioidosis, accounted for over one third of the deaths from severe community-acquired pneumonia at RDH. *Acinetobacter baumannii* is the second most important gram-negative organism, causing almost as many deaths as *S. pneumoniae*. *A. baumannii* has been increasingly recognised as an important cause of severe community-acquired pneumonia in tropical regions. This is in contrast to the situation in temperate city hospitals where *A. baumannii* is being increasingly recognised as a nosocomial pathogen causing secondary infection in patients in intensive care units. Community-acquired *A. baumannii* pneumonia in the tropics occurs usually in the wet season and usually following heavy alcohol consumption. Presentations are with fulminant lobar/total unilateral lung pneumonia and mortality rates are very high. There is a general impression that there are fewer cases of "atypical" pneumonia in patients admitted to RDH in comparison to southern hospitals, although formal assessment of this has not been done.

## Treatment Protocols

People with risk factors such as diabetes, alcohol excess, chronic lung disease, chronic renal failure and steroid therapy are at particular risk for melioidosis and *A. baumannii* pneumonia. However, melioidosis will sometimes occur in an immunocompetent person and therefore needs to be covered by antibiotics for any presentation with severe pneumonia in the Top End (see melioidosis article page 4 this *Bulletin*).

Table 2 outlines the initial therapy of adult community-acquired pneumonia recommended in the Top End. Irrespective of risk factors, penicillin is recommended for mild pneumonia and *S. pneumoniae* remains the commonest organism. If risk factors are present and the pneumonia is moderate or severe, then it is important to cover both *A. baumannii* and *B. pseudomallei*. Therefore gentamicin (to cover *A. baumannii*) is used with ceftriaxone or ceftazidime. In the wet season in a diabetic with severe pneumonia, melioidosis becomes very likely and therefore ceftazidime will often be commenced at RDH instead of ceftriaxone as initial therapy. For critically ill patients being admitted to the intensive care unit at RDH meropenem and gentamicin are usually the initial therapy. Apart from these above situations, ceftriaxone is considered to be adequate initial

empirical therapy to cover melioidosis if used in a dose of 2 grams per day, although it has been shown to be inferior to the specific melioidosis regimens (see below). The minimum inhibitory concentrations (MIC's) of ceftriaxone are around 2 - 4 times those of ceftazidime and meropenem, so if *Burkholderia pseudomallei* is isolated then ceftazidime or meropenem should be commenced as per the melioidosis guidelines (see article page 5 this *Bulletin*). Ceftriaxone however, has a better gram positive coverage than ceftazidime. When used with gentamicin, ceftriaxone will generally hold *Staphylococcus aureus* infection initially, although this is not the definitive therapy for *S. aureus*. Once *S. aureus* is isolated then the appropriate treatment for *S. aureus* pneumonia becomes usually flucloxacillin. Methicillin resistant *S. aureus* (MRSA) requires vancomycin therapy. Ceftriaxone will also provide excellent coverage for *S. pneumoniae*. However, once *S. pneumoniae* is

isolated, penicillin becomes the drug of choice. While average MIC's of penicillin for *S. pneumoniae* have been increasing, the level of resistance is usually intermediate and therefore high dose penicillin is quite adequate for respiratory (but not CNS) infections with these organisms. If *A. baumannii* is isolated from the blood then gentamicin is continued and the ceftriaxone is ceased and replaced by piperacillin or ticarcillin/clavulanic acid or piperacillin/tazobactam or meropenem.

In addition to two sets of blood cultures, an urgent Gram's stain of initial sputum may occasionally be helpful in directing therapy. However the results of sputum culture are often unreliable as they may just indicate throat and upper respiratory tract flora, especially if *Haemophilus influenzae* or *S. pneumoniae* is cultured.

**Table 1 Adult community-acquired bacteremic pneumonia: RDH 1986 - 1998**

255 cases 84 deaths (33%)	Admissions		Deaths		Mortality by organism
	Number of cases	Percentage of total admissions	Number of total deaths	Percentage of total deaths	Percentage
<i>Streptococcus pneumoniae</i>	100	39%	17	20%	17%
<i>Burkholderia pseudomallei</i>	60	24%	30	36%	50%
<i>Staphylococcus aureus</i>	29	11%	11	13%	38%
<i>Acinetobacter baumannii</i>	26	10%	14	17%	54%

**Table 2 Initial therapy of adult community-acquired pneumonia at RDH**

	Mild Pneumonia	Moderate Pneumonia	Severe Pneumonia
No risk factors present*	Penicillin	Penicillin	Ceftriaxone
Risk factors*	Penicillin	Ceftriaxone plus Gentamicin	+Ceftriaxone or Ceftazidime or Meropenem plus Gentamicin

For "atypical" pneumonia add or substitute erythromycin or ciprofloxacin

\*Risk factors include - alcohol, diabetes, chronic lung disease, chronic renal failure, steroid therapy and kava excess.  
+See text for choice.

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## Box-Jellyfish - An update from the Northern Territory and the NT *Chironex fleckeri* treatment protocol

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

The official “stinger” season for the Northern Territory (NT) is from 1 October until 1 June. This is longer than elsewhere in tropical Australia. However, the major box-jellyfish, *Chironex fleckeri* is also responsible for a few stings during the “safer” season (June until end of September) in the NT. Around 40 jellyfish stings present to Top End hospitals or health clinics each stinger season. The majority of these are confirmed by “sticky tape” test (see page 8) to be *C. fleckeri*. There are also 3-10 cases of classical Irukandji syndrome in the Top End each year. Irukandji syndrome is commoner in Far North Queensland, while *C. fleckeri* stings are more common in the NT. There are also a small number of stings in Darwin harbour each season from a four-tentacled (carybdeid) box-jellyfish which is bigger than the carybdeid usually associated with the Irukandji syndrome, namely *Carukia barnesi*. The “Darwin carybdeid” causes local sting marks and pain, which are less severe than with *C. fleckeri*. Whether the “Darwin carybdeid” can cause classical Irukandji syndrome and is the jellyfish responsible for the Irukandji syndrome in the NT remains unclear. Recent work in North Queensland suggests there are other species of carybdeids apart from *C. barnesi* that may cause Irukandji syndrome.

The last recorded stinger death in Australia was in January 2000 when a 6-year-old boy from Yarrabah in Far North Queensland died soon after presumed *C. fleckeri* envenoming. The last recorded death in the NT was from February 1996 in a 3 year old girl from a remote NT Aboriginal community with confirmed *C. fleckeri* envenoming. The last 10 stinger deaths in the NT have all been children, showing the greater risk of a smaller body mass exposed to the millions of stinging cells (nematocysts) on jellyfish tentacles injecting their venom threads into the dermis.

The rapidity of severe envenoming from *C. fleckeri* (and possibly also from related jellyfish elsewhere in the world) is unique in clinical toxinology. Death may be within a few minutes, and if it occurs is usually within 20 minutes of the sting.

Although the lethal toxins from *C. fleckeri* and their exact mechanisms of action remain poorly characterised, the prospective NT study of over 200 *C. fleckeri* stings has clarified some important clinical features, together with some recent

publications elsewhere. The ongoing support of Royal Darwin Hospital’s Accident and Emergency staff, and staff in coastal communities, and District Medical Officers in providing skin sticky tape samples for identification of jellyfish species by nematocyst microscopy, together with stinger report forms, has been invaluable in improving our understanding and clinical management.

A summary of the current status is:

1. Arrhythmias are seen with severe *C. fleckeri* stings, supporting a primary cardiotoxic role in potentially fatal stings. A baseline ECG is useful for all but minor stings.
2. Despite the dramatic nature of severe envenoming, by far the majority of *C. fleckeri* stings are mild to moderate, with the initial severe pain well controlled with ice-packs and, for moderate stings, a single injection of narcotic analgesia if ice-packs are insufficient.
3. The efficacy of *C. fleckeri* antivenom remains to be proven, with conflicting results from laboratory studies. There has yet to be a definitive report of *C. fleckeri* antivenom saving a life. There have now been three documented deaths despite *C. fleckeri* antivenom. While a number of severe envenomings have been given antivenom and survived, there are similar case reports where antivenom was not given. The suggestion from some animal studies that the antivenom is less effective than predicted initially has led to the NT and other recommendations that **in life-threatening envenoming as much antivenom as available (eg up to 6 ampoules) be given if there is no initial response** (see protocol on page 8). Because death is usually rapid if it occurs, the scenario for definitively showing benefit of antivenom will be a major sting with cardiorespiratory arrest near a health centre or hospital where immediate resuscitation and rapid use of intravenous (IV) antivenom is possible. The protocol on page 8 is written for this scenario.
4. Recent experimental work from Queensland supports the long-standing NT protocol of **not using pressure bandages and immobilisation (PI)** in jellyfish stings, because of potential harm and theoretical reasons why it is unlikely to work anyway (Pereira et al).

5. Delayed reactions are common with *C. fleckeri* stings and consist of an itchy "papular urticaria" appearing at the original sting sites around 7-14 days after the original sting. This is considered to be a delayed hypersensitivity reaction, possibly to jellyfish nematocyst products retained in the dermis. The reaction responds to topical steroid cream and oral antihistamines if required.

**The main message remains:**

**DO NOT ENTER THE SEA AND MOST IMPORTANTLY DO NOT LET CHILDREN ENTER THE SEA DURING THE STINGER SEASON - OCTOBER TO MAY**

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**Sticky tape method for identifying nematocysts:**

Ordinary transparent sticky tape, 4-8 cm long, is applied to the sting site, firmly stroked several times, then removed and stuck onto a glass slide. The slide is sent for microscopy, which is currently being done at the Menzies School of Health Research.

**Summary of treatment of *Chironex fleckeri* stings**

***On-the-beach treatment:***

1. Assess the conscious state and treat airway, breathing and circulation if necessary.
2. Liberally pour vinegar over the stung area for a minimum of 30 seconds to inactivate remaining stinging cells on skin and on any adherent tentacles.
3. If unconscious or if evidence of life-threatening cardiac or respiratory decompensation, and if antivenom is available, then 3 ampoules of antivenom (each containing 20,000 units) can be given IM by a trained health professional. However IV antivenom (see below) is preferable wherever possible.
4. In severe envenomation use oxygen if available. Entonox (50% nitrous oxide 50% oxygen) can be administered for severe pain.

***Hospital treatment:***

1. Continue treating airway, breathing and circulation if necessary and add oxygen. Apply vinegar as above.
2. If unconscious or if life-threatening cardiac or respiratory decompensation, or if a significant arrhythmia is present, administer a minimum of 1 ampoule of antivenom IV (each ampoule 20,000 units, diluted 1:10 with an isotonic crystalloid solution such as Hartmann's solution or isotonic saline, given over 5-10 minutes). In a life-threatening situation up to 3 ampoules may be given IV consecutively if the response remains inadequate.
3. Cardiopulmonary resuscitation should be continued and not abandoned in the patient with ongoing cardiac arrest until after further therapy with even more antivenom (at least 6 ampoules total dose if available) and consideration of cardioactive drugs.
4. In stings which are not life-threatening (no cardiac or respiratory decompensation) use ice-packs for initial pain relief, together with IV or IM administered analgesia if necessary (1 mg/kg of pethidine up to 50 mg adult dose initially). For pain not relieved by ice-packs and narcotic analgesia, administer 1 ampoule of antivenom IV as above.
5. Further IV narcotic analgesia may be administered when necessary in conscious patients.
6. The sting should subsequently be treated as for a burn (eg if blistering occurs), to avoid secondary infection. Steroid cream is contra-indicated in the acute sting, but can be used for delayed hypersensitivity reactions which occur at 7-14 days.

## Northern Territory HIV/AIDS Report

Jan Savage and Karen Dempsey, AIDS/STD Program, CDC, Darwin

### Introduction

Testing to diagnose HIV first became available in Australia in 1985. Infection at that time was considered to be invariably fatal. Since then there have been many effective public health preventative interventions introduced and enormous advances made in the clinical management of HIV leading to a decrease in rates of infection and an improvement in the quality and length of life of people living with HIV.

### Aim

This report will provide a description of the notifications of HIV and AIDS cases in the Northern Territory (NT).

### Methods

HIV and AIDS have been notifiable diseases under the Northern Territory *Notifiable Diseases Act* since 1985. Delinked data is maintained in a secure database in the AIDS/STD Program, Centre for Disease Control in Darwin. Access is limited to the program head and the clinical nurse consultant.

HIV is notified by the diagnosing laboratory (at Royal Darwin Hospital). A notification form provided by the National Centre for HIV Epidemiology and Clinical Research (NCHECR) is sent to the diagnosing medical practitioner to collect further information. Variables including name code (first 2 letters of first and surnames), date of diagnosis, date of birth, gender, exposure category, indigenous status, and CD4 cell (T cell) count at diagnosis are recorded. This is the data considered in this report.

AIDS is a clinician notified condition. De-identified information related to HIV diagnosis and clinical condition is requested by the AIDS/STD Program and also forwarded to the NCHECR. Data on cases who have been resident in Australia for at least 3 months prior to diagnosis are included in the national HIV and related diseases surveillance program at the NCHECR. Consultation with the National Centre decreases duplicate notifications of cases reported elsewhere in Australia. Although cases diagnosed in the NT in individuals resident in Australia for less than 3 months are included on the NT database, they will not be included in this discussion.

HIV testing (including pre-test information and post-test counselling) is available on request

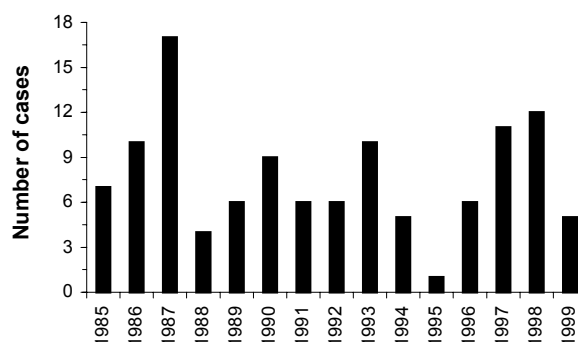
through NT sexual health clinics (Clinic 34), clinics of NT Family Planning Welfare Inc and general practitioners. Testing is also recommended for antenates and anyone diagnosed with a sexually transmitted infection (STI). It is the policy of the NT Department of Correctional Services that all prisoners be tested for HIV and other bloodborne viruses.

### Results

#### HIV

There have been 115 cases of HIV notified in the NT in the 15 years from 1985 to 1999. The average number of notifications per year is 8 (range 1 to 17, see Figure 1). There are 4 additional cases in individuals not considered here who had not been resident in Australia for 3 months.

**Figure 1 Annual HIV notifications, Northern Territory 1985–1999**

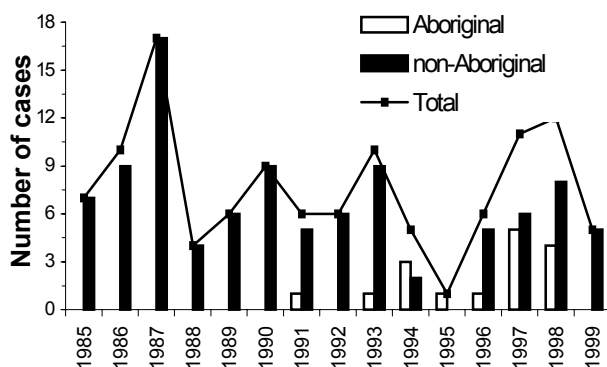


The vast majority (106) of notifications of HIV occurs in men (92%), with only 9 cases diagnosed in women (8%). Age at diagnosis ranges from 20 to 61 years, (median 35-39 years).

The first case of HIV in an indigenous person was diagnosed in 1991. Overall, 13% of NT notifications have been in indigenous people, however, when looking at the more recent period 1991-1999, 24% were indigenous (see Figure 2).

The most common exposure category for HIV infection (65%) remains male homosexual contact, followed by heterosexual exposure (23%) (see Table 1). Thirteen of the 24 cases acquired infection through heterosexual exposure with a person from a high prevalence country. Ninety one cases of HIV (79%) have been diagnosed in Darwin, with 12% and 3% diagnosed in Alice Springs and Katherine respectively. No cases have been documented in East Arnhem or Barkly (Tennant Creek) districts (see Table 2).

**Figure 2 Annual HIV notifications, by total Northern Territory and Indigenous status 1985–1999**



**Table 1 Exposure category, Northern Territory 1985–1999 and Australia 1991–1999**

Exposure category	Northern Territory		Australia <sup>1</sup>
	Cases	Percentage	Percentage
Male homosexual contact	68	64.8	78.4
Male homosexual contact & injecting drug use	6	5.7	3.8
Injecting drug use	5	4.8	4.5
Heterosexual	24	22.9	9.7
Haemophilia	1	1.0	1.8
Receipt of blood products	0	0.0	1.4
Mother with/at risk for HIV infection	1	1.0	0.4
Other/undetermined <sup>2</sup>	10	8.7	19.1
<b>Total</b>	<b>115</b>		

<sup>1</sup> Source: 2000 HIV/AIDS, hepatitis C & sexually transmitted infections in Australia: Annual surveillance report

<sup>2</sup> The 'Other/undetermined' category was excluded from the calculation of the percentage of cases attributed to each HIV exposure category

**Table 2 Cases of HIV notified by Northern Territory Health District, 1985–1999**

District	Northern Territory	
	Cases	Percentage
Darwin	91	79%
Katherine	4	3%
East Arnhem	0	0%
Barkly	0	0%
Alice Springs	14	12%
Unknown <sup>1</sup>	6	5%
<b>Total</b>	<b>115</b>	

<sup>1</sup> The six cases reported as address 'unknown' were notified during the 1980s

## AIDS

There have been 37 cases of AIDS notified in residents of greater than 3 months in the NT since 1985. The number of cases is small, ranging from 0–5 each year. There has been no apparent decline in the last 5 years. All cases have occurred in men; the median age at diagnosis is 30–34 years. Infections caused by *Mycobacterium avium* complex (MAC) and *Pneumocystis pneumoniae* pneumonia (PCP) have each accounted for 22% of AIDS defining diagnoses. AIDS and HIV diagnoses have been made simultaneously in 9 (24%) cases; in an additional 4 cases the AIDS diagnosis has been made within 2 months of the HIV diagnosis. Four of these late presentations (30%) have been notified since 1998. The range of time from HIV to AIDS diagnosis is 0–168 months with the median being 31 months. There have been no HIV related deaths in the NT since 1996.

## Discussion

The number of notifications of HIV in the NT remains stable. Determination of trend is difficult because of the small numbers. The Annual Surveillance report (2000) from the NCHECR<sup>1</sup> provides national data for comparison with the NT figures. Australia-wide, the number of new diagnoses has been steadily declining since testing became available in 1985. The rate of decrease has slowed in recent years. Last year there were 699 new diagnoses notified nationally. The cumulative rate of HIV notifications (59.5 cases per 100,000 population) is lower than the national figure (104.4 cases per 100,000 population),<sup>2</sup> but comparable with rates outside Sydney and Melbourne – the centres of the epidemic.

Nationally, 94% of notifications occur in men, which is similar to the NT (92%). The median age at diagnosis is also similar (35 years compared with 35–39 years in the NT). Rates of notification of HIV in Indigenous people per capita nationally are similar to rates per capita in non-Indigenous people. Nationally, infection through homosexual exposure remains the predominant mode of transmission although the proportion of heterosexually acquired cases is growing. This is also seen in the NT.

Patterns of exposure to HIV in the NT differ somewhat from the rest of the country. Although homosexual exposure is the major transmission factor, (64.8% of NT cases and 78.4% of national cases), 23% of NT cases are acquired heterosexually while only 9% are nationally. This may be explained by higher heterosexual

transmission in the NT Indigenous population, and infections acquired by heterosexual men from the NT travelling to high prevalence countries. Nationally now, nearly half of all *newly acquired* (as opposed to *newly diagnosed* which are reported here from the NT) infections due to heterosexual contact are in people from high prevalence countries or those with sex partners from high prevalence countries. The impact on HIV transmission associated with increasing travel to, and contact with high prevalence countries in the region, is considered an area for public health vigilance. The proportions of cases acquired through injecting drug use (IDU) are similar for the NT and Australia (4.8% and 4.5% respectively). In contrast with other countries these figures are very low and reflect the introduction and maintenance of needle and syringe programs early in the epidemic.

The number of AIDS cases reported nationally has dropped dramatically since the introduction of combination antiretroviral therapy in 1995, but this decrease has not been seen in the NT. The number of NT cases of late diagnosis who have AIDS at the time of HIV diagnosis or develop it soon after (13/37 – 35%) is a matter of concern when effective therapy for HIV infection is readily available. The national figure (1991-1999) for late diagnosis is 21%, although the proportion has been increasing over the last five years.<sup>1</sup> In Australia PCP is still the most common AIDS defining condition (28%). In the NT, PCP is the cause of 22% of AIDS cases. MAC infections contribute to only 5.5% of national AIDS cases, but 22% of NT cases. While the numbers are small this difference may also reflect

late presentations or increased exposure to MAC.

Despite the marked improvements in the health status of HIV positive people since antiretroviral therapy became available, prevention of new HIV infections must remain our focus. The improvement in national notification figures is not an excuse for complacency in this area. Many programs are in place in the NT to promote HIV prevention including:

- the improvement of access to early diagnosis and treatment of STIs in remote areas - a Territory-wide priority,
- education of health professionals and community members regarding prevention and treatment of STIs and HIV,
- urban activities such as needle and syringe programs,
- beat work,
- clinical and education services targeting particularly young people and men.

These programs are carried out across government and non government sectors and in health, education and other sections of the community.

## References

1. HIV/AIDS, Hepatitis C and Sexually Transmissible Infections in Australia Annual Surveillance Report 2000. National Centre in HIV Epidemiology and Clinical Research, University of Sydney, NSW. 2000.
2. Australian HIV Surveillance Report. National Centre in HIV Epidemiology and Clinical Research, University of Sydney, NSW. Vol 15 (3), July 1999.

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## Cervical screening in the Northern Territory

*Cynthia Croft and Chris Moon, Women's Cancer Prevention Program*

Regular Pap smear screening and appropriate follow up of detected abnormalities could prevent 90% of deaths from cervical cancer.

The time it takes for cervical abnormalities to progress to invasive cancer is unclear. However, on average it takes about ten years from the time of the first sign of abnormality to the development of invasive cancer. Therefore, regular two yearly screening provides opportunity for detection of cervical abnormalities before the development of cancer.

The age standardised annual incidence of cervical cancer in Australian women is 11.7 per 100,000,

with a peak of 24.4 per 100,000 in 65-74 year old women. Rates in the Northern Territory (NT) are higher, with non-Aboriginal women having a rate of 18.3 per 100,000 and Aboriginal women a rate of 36.2 per 100,000. NT incidence rates vary with age reaching a high of 49.7 per 100,000 among over 75 year old non-Aboriginal women, and 125.9 per 100,000 among 65-74 year old Aboriginal women.

Annual NT cervical cancer death rates are similarly dependent on age and Aboriginality. The death rate among NT Aboriginal women, at 25.5 per 100,000, is four times higher than that for NT non-Aboriginal women, at 6.7 per 100,000, and six times higher than the Australian rate of 4.0 per 100,000. The

death rates for both NT Aboriginal and NT non-Aboriginal women peak in the 75 and over age group, at 84.9 per 100,000 for Aboriginal women and 49.7 per 100,000 for non-Aboriginal women.

The National Cervical Screening program commenced in 1991 with the aim of reducing deaths from cervical cancer. The NT joined the National program in 1993 with joint funding from the Commonwealth and NT governments. The major components of the program are:

- increasing awareness among women
- encouraging women to attend for regular two yearly screening
- improving access to screening services
- education of health professionals
- operation of the Pap Smear Register that provides reminders to women who are overdue for their next Pap smear or follow up treatment
- provision of appropriate and accessible follow up of screen detected abnormalities

The Pap Smear Register enables the program to monitor participation rates, one of the earliest indicators of the effectiveness of the program. Screening rates are calculated each quarter over two year periods and have increased substantially since the beginning of the program. NT participation rates in 1993 were estimated to be 30%-40% with markedly lower rates in remote communities. Currently the overall NT screening rate is 64%, which compares favourably to interstate rates. Regional variations are shown in the Figure below, with the main points being:

- screening rates for Darwin urban (67.4%) Katherine (63.6%) Alice Springs urban (61.7%)

and Alice Springs remote (66.7%) are comparable to screening rates in the rest of Australia,

- screening rates in Darwin remote (56.9%) and East Arnhem (56.8%) are lower than interstate levels but are increasing steadily,
- the screening rate for Barkly, at 35.5%, continues to be substantially lower than interstate and other NT areas.

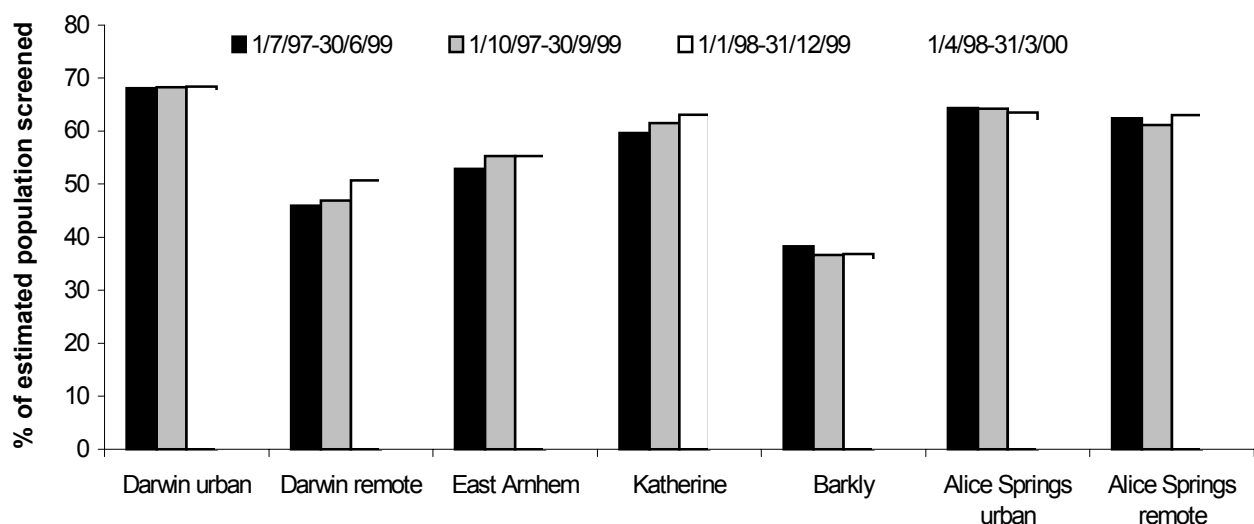
The increased rates in most remote communities are particularly pleasing and are due to a range of factors including the implementation of the Remote Areas Well Women's Screening Program that includes:

- the increased knowledge of Aboriginal women about western medicine women's health issues,
- the increasing role of Aboriginal Health Workers and grandmothers in communities,
- the work and dedication of Women's Health Educators in each of the districts,
- the increasing number of staff in clinics with skills in screening and the commitment of these staff to improving women's health,
- improvements in facilities and equipment in remote health centres,
- activities funded through the Cervical Screening program small grants program.

The consistently lower screening rates in the Barkly remain puzzling despite the implementation of a range of strategies.

If you wish to comment on this article or to make other comments about the Cervical Screening Program please contact Karen Finch by email or phone 8922 5501.

**Figure NT Pap Smear Screening Rates for Women with a Cervix, 20-69 years**



## The invisible and unknown battle

*Bryan Macdonald, Aboriginal Resource and Development Services, Nhulunbuy*

The Centre for Disease Control (CDC) in Nhulunbuy recently invited the Aboriginal Resource and Development Services (ARDS) to accompany them to an Arnhem Land community. ARDS provided health education based around microscopes while CDC offered a check-up/screening service to the men of that community. This was the first time these two organisations had worked together in this way.

Why do health education around microscopes you might ask? Read on.....

When asked what are bacteria, viruses, fungi etc many Yolngu (Arnhem Land Aboriginal people) say that they have heard these words but few have much understanding of what they really are, or accept that they cause many of the sicknesses that people suffer from. We hear comments like:

- “These words we hear doctors and nurses using are just that, words, with no meaning.”
- Others have said, “we have heard these words and know they have something to do with health from the Balanda (non-Aboriginal people) point of view, but what are bacteria and viruses. Are they spirit? Do they have bodies, if so, what do they eat and how do they reproduce?”
- “That is all very interesting but do you realise that most Yolngu people here do not believe all that germ story stuff?”

There is still a very limited understanding of the microbiological world out there in Arnhem Land today. In contrast, generally speaking, Yolngu people have a much better understanding of the visible animals and plants in their environment than Balanda.

How is it that the above situation is so? Here are two of the main reasons.

1. It is a fact of human dynamics that new information coming from another cultural group which is usually delivered in a foreign language (English) and often in a culturally inappropriate manner will always be difficult, if not impossible to receive and accept.
2. Yolngu have only very recently had exposure to microscopes and therefore the microscopic world.

ARDS sees the use of local languages and microscopes as important tools in overcoming these difficulties.

This has major implications in the areas of community and personal hygiene, people presenting at clinics when sick, being willing to comply with treatment and therefore, positive health outcomes for Yolngu people. If Yolngu people are not convinced that there are germs then why wash hands after going to the toilet? Why take the full course of antibiotics? Why put the used disposable nappies in the bins? Why endure the discomfort of a check-up/screening?

Having a good understanding of Yolngu people’s worldview is very important to the success of any education. This requires knowing what is accepted knowledge and then introducing new information that builds on the existing knowledge and working from the known to the unknown. I hasten to add that during these sessions I often find myself being the student and Yolngu being the teacher/s especially in the areas of matha (language) and rom (ways of life, law and world view).

Typical education sessions go something like this.

After some general introductions like working out relationships, malk (skin names) and bapurru (clans) we look at some Balanda history, in particular the great plagues of Europe. How millions of Balanda people died and how this was attributed to all sorts of weird, even laughable (now) causes like having too much blood in one’s body, washing too much, sorcery, evil spirits etc. Well at least it seems weird and laughable to us today. Back then it was no laughing matter. Through the development of lenses and then microscopes it was discovered that ‘little animals’ were the cause of much of the disease and death being experienced.

Significant dates such as the ones below are discussed. It should be noted that this was a slow and painful process for Balanda. It took them over 300 years to accept the germ theory and often the people putting forward these new theories and thereby challenging the accepted views of the day were rejected and laughed out of town.

- 1348 Plagues claim approximately 100 million in Europe.
- 1546 Girolamo Fracatoro proposes the germ theory.
- 1600’s Glass lenses are used in eyeglasses, telescopes and microscopes.
- 1674 Leeuwenhoek observes bacteria for the first time.

- 1880 Bacteria identified and positively connected to disease causation.  
 1898 M. Beijerinck proposes a virus theory.  
 1918 Influenza claims 20 million worldwide.  
 1928 Fleming discovers mould (penicillin) that kills bacteria.  
 1940 Viruses seen for the first time through an electron microscope.

We talk about how Yolngu people have detailed knowledge about how to live in their environment where there are many poisonous and dangerous plants and animals. Much of this information has been past down to them via the Ngathingu rom (old ways and laws). The fact that some of these old laws are not followed now-a-days and that Yolngu now live in larger, permanent communities mean that germs are much more of a problem than they were. All Yolngu readily agree that there is much more sickness and premature death in communities these days compared to even just twenty years ago.

Just as Yolngu people have learned to live with and avoid the harm that can be caused by the visible dangerous animals and plants in their environment, so they can learn to live with and avoid the harm caused by some of these invisible animals. Enter the microscope.

Firstly, a dissection microscope (low power, easy to use) is pulled apart and the group is shown that microscopes are just special glass lenses and mirrors. There are no tricks or 'magic' involved. Yolngu are familiar with glasses so this is our starting point to build on. We then view small insects through the dissection microscope, which is fitted with a camera and connected to a TV so the whole group can see together. This is done to allow people to become familiar with what microscopes are doing e.g. varying the magnification and focusing on different items. We call this microscope literacy. To start with the higher magnification compound microscope would be meaningless, as it does not work from the known to the unknown.

The group is encouraged to use the microscope themselves and start to become familiar with it. Some groups really lock in and collect many insects, leaves and dirt etc to investigate.

When the people are ready, we move on and explain that the compound microscope is much more powerful than the dissection microscope. Once again the compound microscope is fitted with a camera and connected to TV for easy group viewing. Discussion moves onto germs and how there are five 'clans' of germs. Balanda call these

'clans' Bacteria, Viruses, Fungi, Protozoa, and Parasites. Some examples from each 'clan' are discussed. We now focus on bacteria and where they are likely to live. We then obtain samples from the local environment, including sputum, stagnant water, pus from a sore, soil around dripping taps, from rubbish and view them through the microscope. The importance of obtaining live samples from the local environment can not be overstated. We have found that prepared slides do not work for microscope literacy education sessions.

When a 'good' slide has been obtained with lots of microscopic life, Yolngu people are usually surprised, shocked, and sometimes even frightened. While viewing bacteria we tap into the knowledge of the local people and draw some comparisons to the larger, visible animals. For example snakes. Each type of snake has their own distinct food, habits and habitats. Some are harmless, even helpful, whilst others can be very dangerous. It is the same with bacteria. We then discuss life cycles and breeding of bacteria (mitosis) and how under good conditions bacteria can reproduce very quickly.

From here we change to a dark field condenser, still on the compound microscope, and look at some live blood. In particular the white blood cells which make up part of the immune system. These are referred to as the 'body warriors' or 'protectors' in the local Yolngu Matha (Aboriginal language or tongue). Discussion centres on how these 'body warriors' attack any germs or invaders. Also if we don't look after our bodies, by eating a poor diet, abusing substances and drugs, not sleeping properly etc. then our body warriors will not be able to do their job of fighting off these invaders. No one in their right mind would send their soldiers to war hungry, tired, stoned or hung over.

The Yolngu of Arnhem Land consider the skeleton to be very special, even holy or sacred. When they learn that these body warriors (white blood cells) are made in the bones of our bodies this dovetails very easily into their own worldview and knowledge and adds onto it some new information. (This new information still has to be checked and validated before it will be accepted but now there are fewer barriers).

At this point we say that everyone has experienced this battle between the invading germs and our body warriors before today. Most in reply state firmly that this was the first time they have seen or heard anything on this subject to which we ask, "well,

what is boyara (pus)?" During the dialogue that follows we point out that the pain, redness or temperature experienced when germs invade our body (infection) is evidence of the battle or war that is going on in our body. The pus contains the dead invaders (germs) and the dead body warriors that have done their job and now all are being expelled from our body.

This microscope education session was usually conducted after the men had been through their check-up screening. It is pointed out that the samples of blood and urine that they have just given are sent to Darwin where skilled people with the aid of modern equipment look for any invading germs and also check how their body warriors are, amongst other things.

At this point it is a small step to introduce the AIDS/HIV story saying that the HIV virus attacks and kills some of the white blood cells (usually pointing to some on the TV screen). This then makes it easier for other invaders (germs) to come into our bodies and multiply quickly causing much sickness and possibly death.

Other STI's can be discussed at this time always reminding people that it is these very small invisible animals that cause these diseases. Here we introduce (only after microscope literacy has been completed) a large laminated poster with images of different pathogens and point out the many different shapes and sizes these 'little fellows' come in.

An ABC Foreign Correspondent documentary on AIDS in Africa recorded in November 1999 has also proved very useful in reinforcing the fact that HIV/AIDS is very serious and that we can not afford to be complacent. Images of the endless procession of funerals and of orphaned children

fending for themselves are powerful.

Here is a sample of comments from Yolngu people after they have seen live bacteria and blood cells through a microscope:

- "Ngunhi nganakmirr dhawu (that is good, meaty, meaningful information with no bones in it)."
- It is hard to understand how something that is so small that I cannot even see it, can hurt me, but there are so many of them.
- "Ah, now I understand! This is why we should not spit everywhere or leave used disposable nappies or other rubbish lying around. This is one reason why there is a lot of sickness around this community."
- After seeing all those things in my own spit I now believe they are real. Before I did not believe you. When you come back to this community next time and visit my house you will see some changes in the way we are living.
- For many years when I have been sick I have been given some medicine and sent home but dhudi dhawu bayangu (no intellectually satisfying information). Ngunhi gung'gayunhamirr dhawu (that is helpful information).

In any battle or contest, it is a huge advantage to know your enemy, to know what you are up against. As Yolngu people gain new understanding into microbiology they will develop their own strategies to enable them to live safely with these invisible enemies. They have survived successfully for thousands of years in this land of extreme contrasts.

It was both productive and a pleasure to work cooperatively with the CDC team. Thanks.

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## Influenza outbreak

In October 2000, a remote Top End Aboriginal community (population approximately 1370) experienced a dramatic increase in the incidence of an influenza-like illness. Over a three week period, there were almost 300 cases (~21% of the community) with the incidence peaking in the second week of the month. The age range of sufferers was from 5 months to 84 years. Of the 36 cases under 15 years, seven were under two years. Six of these seven were admitted to hospital, as were three adult cases. One of the adult cases died, an elderly woman with a presumptive diagnosis of influenza and a number of chronic conditions. Laboratory specimens from 12 cases during this

period showed influenza A Moscow/10/99 (H3N2)-like subtype confirming the initial suspicions. Approximately 50% of the cases had received influenza vaccine, but, in a retrospective analysis of the notes, it was not possible to determine symptom severity beyond those who were hospitalised in this group. Nevertheless, this high rate of disease causes concern. Additionally, given the proportion of children under two years who were hospitalised, vaccination in this young age group deserves consideration.

Liesa Clague (Master of Applied Epidemiology student) conducted the outbreak investigation.

## Points of correction/clarification/further information regarding the measles articles\* in the previous (September 2000) issue of the Bulletin

- Correction on page 4. A measles epidemic in Central Australia during 1994 with 259 cases reported and no deaths was inadvertently left out of Table 3. *This point has been corrected in the electronic copy on the Internet site.*
- Correction on page 7. The measles, mumps, rubella (MMR) vaccine used for the East Timorese evacuees in September 1999 was Priorix, Smith Kline Beecham; 0.5ml and was given by intramuscular injection - not subcutaneously. *This point has been corrected in the electronic copy on the Internet site.*
- Measles antibodies will develop in about 95% of children vaccinated at 12 months of age with the current MMR. There are various reasons why children who receive only one dose of MMR fail to respond – but most who fail to respond with the first dose will successfully respond to a second dose. Therefore, with two doses of measles vaccine (with the first dose given after the first birthday), over 99% of people will be immune to measles infection. So, a two-dose policy is in place for the second dose to immunise most of the people (~5%) who failed to become immune from the first dose. Additionally, the recommended age for the second dose was changed from 10 to 16 years to 4 years. Bringing the second dose forward reduces the absolute number of children who remain susceptible to measles despite having had a dose after their first birthday, and decreases the proportion of the population who are not immune. Measles is a very infectious disease and so a very high level of population immunity (>95%) is required in order to prevent disease transmission.. Vaccine-induced immunity to measles appears to be long-term and probably life long.<sup>1</sup>
- A proportion of young adults in Australia remain susceptible to measles infection because 1) they are too old to have received two doses of a measles containing vaccine (the two-dose MMR vaccination program was introduced in 1994), 2) they may never have received even one dose and 3) they may never have had measles infection because the circulation of measles virus fell after the vaccine was first introduced. As a group people aged over 40 years have a very high level of immunity to measles because they are more likely to have had the disease measles prior to the introduction of the measles vaccine into Australia in 1968. The two-dose MMR policy and the Measles Control Campaign of 1998 have resulted in high levels of immunity in Australians under 18 years of age. There is a need to promote two dose MMR coverage in this 18 to 40 year old age group.
- Twelve months has been chosen as the age for routine measles vaccination (given as MMR) in Australia where the rate of measles is low. The presence of maternal antibodies in infants under 12 months of age means that infants under 6 months are protected but those over 6 months will respond variably to the measles vaccine (the younger the infant, the less likely the vaccine will induce immunity; the older the infant the more susceptible to measles if unimmunised). So, during an *outbreak* of measles, infants aged 6-12 months are generally regarded to be susceptible to measles infection and are at risk of developing complications from the disease,<sup>2</sup> and consideration is given to vaccinating this age group. However, any infant who is vaccinated before 11 months of age i.e. during an outbreak should receive another dose of MMR vaccine on or after their 1<sup>st</sup> birthday. The new (2000) *Guidelines for the control of measles outbreaks in Australia* recommends lowering the age to 9 months during an outbreak rather than to 6 months as is currently recommended in the NT and this will be considered in the upcoming review of the NT measles policy.
- Five measles positive RT-PCR specimens from vaccinated East Timorese children have now been genotyped. These all have been confirmed as wild measles virus, not vaccine strain. Results are still pending on the other seven vaccinated East Timorese children notified as having measles. These interim results lend support to the conclusion in the report and to the paediatricians clinical diagnoses, that all notified cases were wild measles and not vaccine related illnesses.

### References

1. Atkinson W, Wolfe C, Humiston S, Nelson R (eds). *Epidemiology and Prevention of Vaccine-Preventable Diseases*. Centers for Disease Control and Prevention, Atlanta. Jan 2000: 128.
2. National Health and Medical Research Council. *The Australian Immunisation Handbook 7<sup>th</sup> Edition*. 2000:161-2. Australian Government Publishing Service.

### \*Articles:

- **Measles in the Northern Territory 1991-1999: implications for policy development (pages 1-6).**
- **An outbreak of measles amongst East Timorese evacuees in Darwin, 1999 (pages 7-13).**

## New vaccine policy in the NT: Availability of BCG

Bacille Calmette-Guerin (BCG) vaccine continues to be recommended for:

- Aboriginal neonates
- neonates and children <5 years of age who will live for >3 months in an Aboriginal community or in a country with high prevalence of tuberculosis (TB)
- neonates born to mothers who have been treated for leprosy.

In the NT, the majority of doses of BCG are administered to neonates in hospital. Administration of the vaccine is by intradermal injection, a technique which requires training, experience and familiarity with the protocol. In the past year, there have been two occasions when BCG was inadvertently injected into the forearm instead of PPD (purified protein derivative) for Mantoux testing. In one case this unintended administration lead to extensive reaction in the forearm and prolonged medical treatment.

To avoid this problem, BCG has been withdrawn

from health centres throughout the NT. It will no longer be available as a stock item. A recent review of the use of BCG in the community setting revealed that very little BCG was given at health centres and most did not stock it. The vaccine however will continue to be available for rural health centres by prescription through regional pharmacy for those few individual infants or children not administered BCG in hospital. An effort will be made to give BCG required outside the hospital setting through the TB/Chest Clinic in the NT. For patients over 6 months of age who require a BCG, a Mantoux test must be performed prior to the administration of the vaccine. Only patients with a negative Mantoux test should receive a BCG.

To read about BCG vaccination, refer to *The Australian Immunisation Handbook* (7<sup>th</sup> edition, 2000). Those with access to the Internet can refer to: [http://immunise.health.gov.au/handbook\\_7.pdf](http://immunise.health.gov.au/handbook_7.pdf)

For further information on the BCG vaccine, contact your nearest TB/Chest Clinic.

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## Ven Troppo - The inaugural Sexual Health College Conference - June 2000

The annual meeting of the Australasian College of Sexual Health Physicians was held in Darwin in June this year. This very successful conference attracted nearly three hundred delegates from across Australia and overseas.

The program focused on the status of sexually transmitted infections (STI) and blood borne viruses (BBV) in Northern Australia and regional areas and combined responses of the primary and public health sectors. Dr Heiner Grosskurth from the London School of Hygiene and Tropical Medicine opened the conference with the Gollow Lecture. He presented an excellent paper on HIV prevention through the treatment of STI's in Mwanza, Tanzania. A/Professor Verapol Chandeying discussed responses to STI's and BBV's across all levels of the health care system in Thailand and presented an analysis of strengths and weaknesses of these interventions. Dr Sue Crockett described the challenges of working in the area of STI and BBV prevention in Papua New Guinea and crafting an effective response in the context of competing health and development priorities. Dr Steven Skov and A/Prof David Plummer gave perspectives of sexual health service delivery in northern and southern Australia.

Diagnostic technology, epidemiology and public health, infection and adverse pregnancy outcomes, the clinical management of HIV and a range of other conditions were discussed at other sessions. There was considerable interest in the workshop and session on sexual assault which looked at clinical management and strategies to improve the training of sexual health professionals. Concurrent sessions presented aspects of men's and women's sexual health. The men's session included a presentation by the "sistagirls," an indigenous transgender group. The satellite meeting on "Sex, Sums and Cyberspace" linked information technology with disease modelling and education. The themes of the proffered papers covered clinical practice, primary health care, epidemiological trends, regional issues and potentially high risk groups.

The College also conducted a update directed towards trainees which addressed the basic sciences.

Other overseas speakers included Professor Stephen Morse from CDC, Atlanta, Professor David Eschenbach from the University of Washington, Dr Juliet Broadmore and Dr Carol Shand from NZ. All



**NT NOTIFICATIONS OF DISEASES BY DISTRICTS  
1 JULY TO 30 SEPTEMBER 2000 AND 1999**

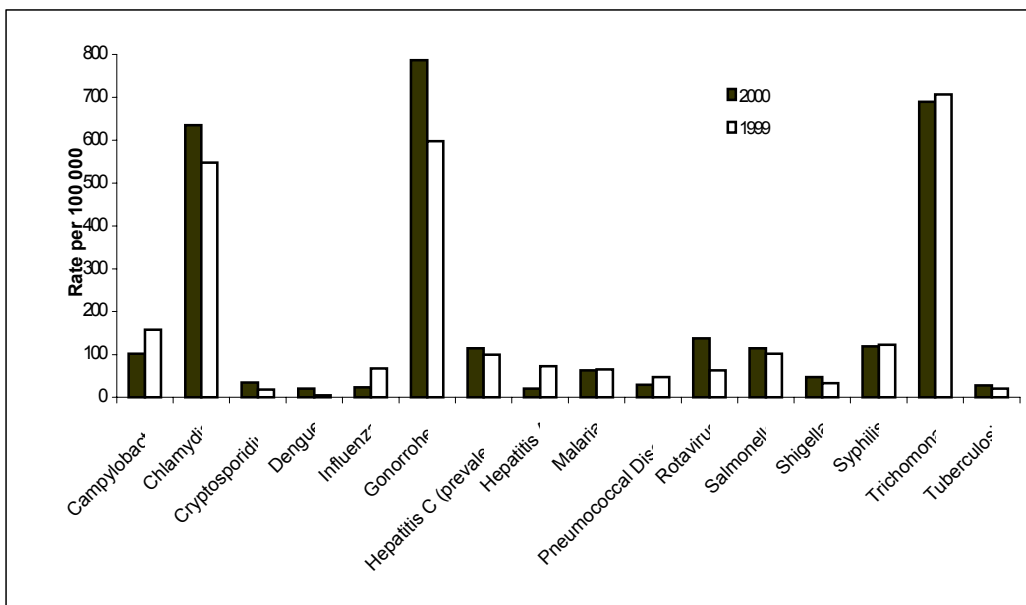
DISEASES	ALICE SPRINGS		BARKLY		DARWIN		EAST ARNHEM		KATHERINE		TOTAL	
	2000	1999	2000	1999	2000	1999	2000	1999	2000	1999	2000	1999
Acute Rheumatic Fever	0	0	0	0	0	2	1	0	0	5	1	7
Adverse Vaccine React.	0	0	0	0	4	0	0	1	1	1	5	2
Arbovirus infections												
Aust Encephalitis	0	0	0	0	1	0	0	0	0	0	1	0
Barmah Forest Virus	0	0	0	1	1	0	0	2	0	0	1	3
Dengue	0	0	0	0	9	1	0	0	1	1	10	2
Ross River Virus	0	0	1	0	6	15	0	1	1	1	8	17
Atypical Mycobacteria	0	0	0	0	1	0	0	0	0	0	1	0
Campylobacter	16	16	3	0	23	54	1	3	7	4	50	77
Chlamydia	166	91	2	9	92	113	23	22	28	33	311	268
Chlamydia Conjunct.	0	1	0	0	0	0	0	0	0	1	0	2
Cryptosporidiosis	5	0	1	0	4	3	3	4	4	2	17	9
Donovanosis	0	2	0	0	0	0	0	0	0	2	0	4
Glomerulonephritis	0	0	0	0	4	0	0	0	2	0	6	0
Gonococcal Disease	252	132	13	14	56	86	26	18	38	43	385	293
Gonococcal Conjunct.	2	1	0	0	0	0	0	0	0	0	2	1
Haemolytic Uraemic Syn	0	0	0	0	0	1	0	0	0	0	0	1
Haemophilus Inf type b	0	0	0	0	0	1	1	0	0	0	1	1
Hepatitis A	1	5	0	2	8	26	1	0	0	2	10	35
Hepatitis B	1	0	0	2	0	1	0	1	0	0	1	4
Hepatitis C (prevalence)	21	4	1	0	32	43	0	1	2	1	56	49
HIV infections	0	0	0	0	2	0	0	0	0	0	2	0
HTLV-1	8	2	0	0	0	1	0	0	1	0	9	3
Influenza	3	23	0	1	7	5	0	1	1	3	11	33
Legionnaires Disease	0	0	0	0	0	1	0	0	0	0	0	1
Leprosy	0	0	0	0	1	0	0	0	0	0	1	0
Leptospirosis	0	0	0	0	0	0	0	0	1	0	1	0
Malaria	1	1	0	0	28	30	0	1	2	0	31	32
Measles	0	0	0	0	0	3	0	0	0	0	0	3
Melioidosis	0	0	0	0	2	0	0	0	0	0	2	0
Meningococcal Infection	0	1	0	1	0	0	1	0	0	0	1	2
Mumps	0	0	0	0	0	2	0	1	0	0	0	3
Ornithosis	0	0	0	0	0	0	0	0	1	0	1	0
Pertussis	1	0	0	0	0	0	0	0	0	0	1	0
Pneumococcal Disease	9	12	0	0	3	8	0	0	2	3	14	23
Rotavirus	26	8	3	0	20	17	13	0	5	6	67	31
Salmonella	11	9	0	0	32	31	4	1	9	9	56	50
Shigella	11	5	1	0	7	9	3	1	1	1	23	16
Syphilis	13	16	0	9	26	17	10	10	9	8	58	60
Trichomonas	81	41	1	7	96	107	87	117	73	74	338	346
Tuberculosis	1	1	0	0	10	8	1	0	1	1	13	10
Typhus	0	0	0	0	1	0	0	0	0	0	1	0
Yersiniosis	0	0	0	0	2	0	0	0	0	0	2	0
<b>Total</b>	<b>629</b>	<b>371</b>	<b>26</b>	<b>46</b>	<b>478</b>	<b>585</b>	<b>175</b>	<b>185</b>	<b>190</b>	<b>201</b>	<b>1498</b>	<b>1388</b>

**NOTIFIED CASES OF VACCINE PREVENTABLE DISEASES IN THE NT  
BY REPORT DATE 1 JULY TO 30 SEPTEMBER 2000 AND 1999**

DISEASES	TOTAL		No. cases among children aged 0-5 years	
	2000	1999	2000	1999
Congenital rubella syndrome	0	0	0	0
Diphtheria	0	0	0	0
<i>Haemophilus influenzae</i> type b	1	1	0	0
Hepatitis B	1	4	0	0
Measles	0	3	0	2
Mumps	0	3	0	1
Pertussis	1	0	0	0
Poliomyelitis, paralytic	0	0	0	0
Rubella	0	0	0	0
Tetanus	0	0	0	0

- Mumps is largely under-reported.

**NT WIDE NOTIFIABLE DISEASES  
1 JULY TO 30 SEPTEMBER 2000 AND 1999**



Rates <10/100 000 not listed  
NT est. resid. pop – 195 905 supplied by Epidemiology & Statistical Branch, THS

## Points to note regarding notifications on page 19

- AIDS, Amoebiasis, Australian Encephalitis (Kunjin, Kokobera), Botulism, Brucellosis, Chancroid, Cholera, Congenital Rubella Syndrome, Congenital Syphilis, Diphtheria, Gastroenteritis, Hepatitis C (incidence), Hepatitis D, Hepatitis E, Hydatid Disease, Listeriosis, Lymphogranuloma venereum, Poliomyelitis, Rubella, Typhoid and Viral Haemorrhagic Fever are all notifiable but had "0" notifications in this period.
- The increase in gonorrhoea, chlamydia and trichomonas in the Alice Springs district was due to increased sub-regional screening and increased opportunistic testing and better case management (including contact tracing and the 3 months test of reinfection).
- As in previous quarters, the increase in dengue cases have all been acquired overseas, principally in East Timor.
- The decrease in hepatitis A cases this year reflect that there was no large clustering of cases as occurred during the same period last year which was associated with Darwin Child Care Centres.

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### Late breaking news!

Three further cases of leptospirosis have been confirmed in women turtle hunting in the Oenpelli area.

## Disease Control staff updates

### ALICE SPRINGS

**Helen Tindall** has recently been appointed to the temporary position of Public Health Nurse, CDC Alice Springs. Helen has spent the last three years in the NT with her most recent position as Clinical Nurse Consultant on the Children's Ward at Katherine Hospital. She is currently undertaking a Masters in Public Health through James Cook University in Townsville.

In November, **Lynette Purton** commenced as the Central Australian Rheumatic Heart Disease Program Coordinator. Lynette moved across to the Territory from Far North Queensland where she had been working for six months as a Public Health Nurse in the Torres Strait. Two years prior to this she was the Immunisation Coordinator at the Tropical Public Health Unit in Cairns.

In November, **Jenny Hains** commenced a 12 month temporary position as Continuous Quality Improvement Manager at Alice Springs Hospital. **Annie Tangy** is relieving in Jenny's positions in the Sexual Health Unit during December and January.

### TENNANT CREEK

**Jenny Midgley** is relieving in the second Public Health Nurse position in Tennant Creek until 18 December. Interviews for this position have

recently been conducted and permanent appointment is anticipated soon.

### KATHERINE

**Matthew Hansen**, Men's Health Educator/STD/AIDS has just started in CDC Katherine filling a much needed position. Matthew and his family are still considered relatively new to the Territory having only just arrived in March '99 from South East Queensland. Appreciating the lifestyle, they have decided to stay on a while longer. Although a new graduate, he is particularly interested in Men's Health and plans to do post graduate studies in this area. Initially, Matthew will be involved in the process of putting together a functioning Men's Health Team to visit communities and provide an optimal service. He is looking forward to the challenge of setting up and implementing a comprehensive service in Katherine. If there are any queries do not hesitate to come and find him on the first floor of O'Keefe House located opposite the Katherine District Hospital or phone 8973 9044.

### DARWIN

As of 18 December, **Sue Reid** will commence 12 months maternity leave. The position of Public Health Research Nurse has recently been advertised with interviews expected to be held soon.

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