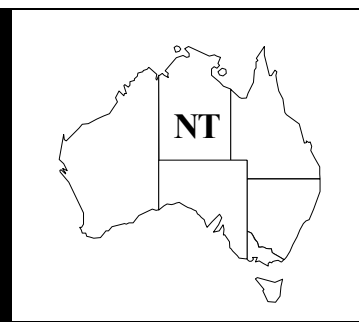




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An outbreak of salmonellosis linked to a marine turtle

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Introduction

On 18 September 1998 the Centre for Disease Control (CDC), Darwin was notified of an outbreak of gastroenteritis predominantly affecting adults in a Top End coastal community. There had been no previous presentations to the community clinic in the month of September with vomiting or diarrhoea. On 14 September, a green turtle (*Chelodonia mydas*) was cooked and distributed throughout the community. Water collected from a water hole near the community (known as the aerator) was used as drinking water at the cook site and to cook the meat. In addition, there were reports that kava, a plant derived tranquilliser,¹ had been consumed the night before using water from the same source. An investigation was conducted to determine the aetiology and source and to instigate prevention and control measures.

Methods

Community X is a coastal Aboriginal community in the Top End of the Northern Territory with a fluid population of between 700 - 1500 people. Approval to interview cases was obtained from community representatives.

A case was defined as any person who resided in, or had visited, Community X with onset of diarrhoea

and/or vomiting in the week beginning 12 September 1998. Data were collected through an interviewer administered questionnaire at the person's home with the assistance of an Aboriginal Health Worker (AHW).

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Water, food and stool samples were obtained for microscopy, culture and sensitivity testing. Water was analysed by the Department of Primary Industries in Darwin. Food samples and isolates of stools were referred to the Institute of Medical and Veterinary Sciences in Adelaide. Faecal specimens were also sent to Path Centre, Perth for viral studies including electron microscopy. Inspections of the waterhole, town water supplies and community food outlets were conducted by Public Health Officers (PHOs), Environmental Health Officers (EHOs) and the community's Essential Services Officer.

Results

Thirty six cases were detected, with onset of illness from 14 September to 21 September 1998. Twenty-nine cases (81%) were interviewed. Of the remainder, three had left the community and four refused interview. Of the 36 cases, 33 (92%) were Aboriginal, 21 (58%) were female and 28 (n=78%) were 10 years of age or older. The median age was 20 years (Range: 1-59).

The first case had an onset of illness on the morning of 14 September and the last on the evening of 21 September. The frequency of reported symptoms is presented in Table 1. The median duration of illness was two days (Range: 1-7 days). Six (17%) cases were hospitalised and a further two cases required intravenous rehydration at the clinic.

Table 1 Frequency of self-reported symptoms in respondents (n=29)

Symptom	Number	%
Watery diarrhoea	25	86
Abdominal pain	24	83
Nausea	22	76
Vomiting	20	71
Fever	21	72
Headache	12	43
Bloody diarrhoea	0	0

Eighteen (62%) respondents reported turtle consumption within a median of 24 hours (Range: 8-96 hours) prior to onset of illness. Seven (21%) reported exposure to the aerator water and of these, six had also consumed turtle. The remaining case reported exposure to the water 36 hours prior to onset of illness. Only four cases (14%) reported kava consumption, each at different times during the week. General food history recall for all other food items was limited.

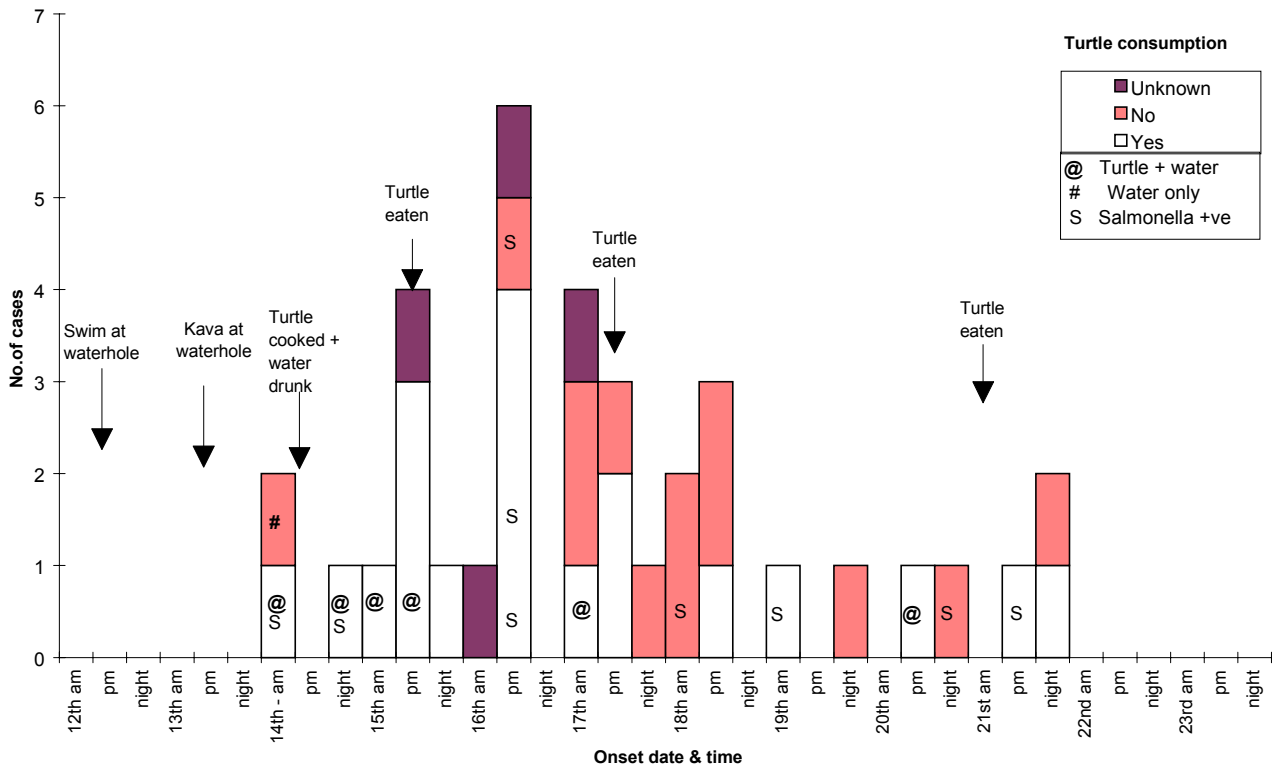
A total of nine stool specimens were collected. Eight (89%) were positive for *Salmonella chester*. *S. chester* was also isolated from a sample of partially cooked turtle meat. Table 2 presents cases in the context of exposure history and stool culture results. Figure 1 presents the epidemic curve relative to the sequence of events, stool results and exposure history.

Table 2 Exposure history and stool culture results

Exposure	Responder	Non-responder	Stool culture
Turtle only	12		4 <i>S. chester</i>
Turtle + water	6	1	2 <i>S. chester</i>
Water only	1	1	N/A
Household only*	5	5	1 <i>S. chester</i>
Other contact only ⁺	3		1 <i>S. chester</i>
None	2		1 <i>Enterovirus</i>
Total	29	7	9

* All lived in same house as someone who had exposure to turtle or water

+ Non-household contact of another case

Figure 1 Epidemic curve of a salmonellosis outbreak in a Top End NT community

Environmental results

The run-off creek from the waterhole had been obstructed by a make-shift dam causing the water to stagnate. The grass around the perimeter had been recently mowed and dirty nappies were found in the water and around the site. Water samples had a faecal coliform count of 710 per 100ml. None of this sample was retained for culture. One week after the dam was broken, the faecal coliform count was 50 per 100ml and *Aeromonas sp.* was cultured. Town water supplies were reported as not contaminated. EHOs reported no detected deficient food handling, storage or store hygiene practices in the two community food stores.

Discussion

The laboratory evidence from this investigation supported consumption of the green turtle as the likely vehicle of transmission for this outbreak of salmonellosis; with a high rate of secondary transmission. The temporal relationship and biologic plausibility between the consumption of turtle and onset of the outbreak, together with the isolation of *S. chester* from both the meat and stools from cases, provides evidence to support our conclusion. Exposure to pet turtles is a recognised risk factor for salmonellosis.² National and international outbreaks

have occurred,^{3,4} and legislation enacted in Canada and the US on the control of pet turtle breeding farms and sales resulted in a marked decrease in the frequency of salmonellosis in children.^{5,6} While there are reports, however, of a toxic illness associated with consumption of turtle,⁷ none could be found reporting salmonellosis from consumption of meat, despite sea turtles being a popular food source throughout tropical areas, including Australian Aboriginal coastal communities.⁷

In Australia, *Salmonella sp.* have been isolated from wild green turtles in Western Australia and southern Queensland.⁸ Prolonged carriage of *Salmonella sp.* of up to 11 months in other turtle species has been demonstrated, together with high rates of infected eggs and hatchlings.⁶ Latent carrier states can be affected by periods of stress precipitating a bacteraemia.⁹ This particular turtle had been tied up to rocks for nearly two days prior to slaughter.

It is possible the original source of infection was water either consumed at, or collected from the aerator site. While the water was used to cook part of the meat, it was reported as boiling at the time and it is unlikely the meat was contaminated at that stage of the process. Cross-contamination is possible as the water may have been used to wash the meat or

cool it down after cooking, however, information that precise was not obtained at the time of interview.

There were two cases who became ill on the Monday morning prior to the turtle being cooked. Both had exposure to the water prior to onset of illness. The first, a child, had no contact with anyone else ill in the community until after his diarrhoea had begun and his only common exposure was the waterhole. However, his only reported symptom was diarrhoea and stools were not cultured. The high rate of hospitalisations in this outbreak suggested a particularly virulent strain and more severe disease in a young child may have been expected. Infection with a different pathogen is plausible given the high faecal coliform count in the aerator water.

The second case, an adult, from whom *S. chester* was isolated, was drinking kava mixed with the water the night before onset of illness and ate turtle after she became unwell. However, she had also been drinking alcohol on the Sunday night which may have precipitated gastrointestinal symptoms the following morning; later exacerbated by turtle consumption. Unfortunately, her stool specimen was collected after turtle consumption, and determining which source was responsible was not possible.

That the water is not as likely to be the source as the turtle is supported by reports that the waterhole is a popular swimming spot for the residents of a nearby town and more cases from outside the community may have been expected. Similarly, reports indicated a large group of people were present at the waterhole on the Sunday drinking kava and there were no cases detected who had this event as their sole exposure.

While 38% of cases had no exposure to either the turtle or the aerator water, only two reported no known personal contact with another case. As the majority were household or workplace contacts, and occurred 24 - 48 hours after consumption of turtle by index cases in those environments, it suggests person-to-person transmission through contamination of other food sources occurred, possibly during preparation of meals. For those with no contact, *Enterovirus* was detected in one person's stool, suggesting a different illness; and for the other, history is uncertain as she gave a negative

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response to all questions on the questionnaire other than symptoms.

Public health action resulting from this outbreak included: notifying the community and nearby township through media channels, schools and council offices; encouraging personal and domestic hygiene and use of Oral Rehydration Solution on a house-to-house and community wide basis; and distributing educational material through businesses, schools and community organisations. Warning signs were posted regarding the suitability of the water from the waterhole for swimming and drinking. Feedback was given to the AHW and the community council illustrating the relationship between inadequately cooked animal food and diarrhoeal disease for dissemination through the community.

This investigation also provided the opportunity to evaluate the usefulness of a standard foodborne disease outbreak questionnaire in these settings and raised issues that will need to be addressed in the future. Importantly, it highlighted the significant contribution of AHWs to the success of investigations and associated public health responses; demonstrating support for the continuing development of specific training in this field.

Acknowledgements

We would like to thank the local public health staff, the community's council and health centre, and the AHWs for their invaluable contribution to this investigation.

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Report on ciguatera poisoning, Groote Eylandt, October 1998

Wendy Williams, District Medical Officer, Groote Eylandt and Hartley Dentith, CDC, Gove

On the evening of the 5 October 1998 a Groote Eylandt family were given a gift of reef fish, a barracuda, measuring approximately 75cm x 23cm and weighing about 4 kg. The fish was caught at North East Island on the morning of 5 October and was brought into Groote Eylandt through Bartalumba Bay.

The fish had been gutted but was otherwise intact. The fish was cut into three sections, one to be cooked and eaten straight away and the other two pieces were put in the refrigerator. The first section was not left out in the sun and was cooked soon after it was received by placing fillets of fish onto the embers of an open fire. No wrapping of bark or foil was used.

Seven family members ate the fish without any other food being consumed.

Clinical presentations and management

In the early hours of 6 October, six of the family members presented to the Health Centre with symptoms varying in severity which began 4-5 hours after eating the fish. The 38 year old husband presented 6 - 7 hours after eating the fish with vomiting, diarrhoea, abdominal pain, a burning sensation in his mouth and dizziness. He had consumed the largest serving of fish. The 38 year old wife presented with dizziness weakness and diarrhoea, but no burning sensation in the mouth. A 16 year old male family member complained of vomiting and a sensation of tingling in his tongue and mouth. Three children, aged 9, 10 and 11, all experienced vomiting and a burning sensation in the mouth with two also experiencing diarrhoea and abdominal pain.

The seventh family member who ate the fish remained at home with moderate nausea and vomiting.

On examination, none of the family suffered from temperature reversal. The two adults and teenager were hypotensive. All had bradycardia with the pulses ranging from 50 to 60 BPM.

The 38 year old male had a pulse of 51 BPM and BP of 100/70 with previous routine pulse recordings of 80 BPM and BP of 120/85. He was treated with intravenous fluids. At 8 - 9 hours after eating the fish his pulse dropped to 36 BPM and cardiac monitoring revealed single focus ectopics. After treatment with 0.6 mg intravenous atropine his pulse increased to 70-80 BPM and BP was 120/90. He developed burning chest pain requiring pethidine for relief. He was evacuated to hospital where he continued to have sinus bradycardia with ectopic beats, hypotension, a tingling sensation around his mouth, nausea, vomiting, abdominal and thoracic spine pain and diarrhoea. All of these signs and symptoms lasted for two days. He was treated with intravenous fluids, 375 mls of mannitol and PRN atropine. He had no temperature reversal on testing. On discharge three days post admission he was still mildly bradycardic with various pains and nausea.

The wife presented 13 - 14 hours after eating the fish with a pulse of 52 BPM and BP of 86/50. She was treated with intravenous fluids. At 14 - 15 hours after ingestion her pulse dropped to 40 BPM with a BP of 80/53. She was treated with intravenous atropine and evacuated to hospital where sinus bradycardia without ectopics and hypotension continued requiring ongoing intravenous fluids and PRN atropine for the next 24 hours. She complained of tiredness for one day post ingestion, but reported no other symptoms. She had

no temperature reversal on testing. Two days post ingestion she felt well, but sinus bradycardia continued until discharge on the third day.

The 16 year old's pulse was 50 BPM with a BP of 95/60 on presentation. He was given atropine and intravenous fluids before evacuation to hospital. Sinus bradycardia continued until discharge on day three and he reported two days of nausea.

The children were also given intravenous fluids and evacuated to hospital. Two of the children complained of weakness, lethargy and 'pins and needles' in the legs which lasted for one day. One of these children also reported a painful left knee about 30 hours post ingestion. The third child complained of nausea, headache and lethargy which lasted for one day. All were asymptomatic on discharge at day three.

The six hospitalised family members were reviewed one week post ingestion. All were experiencing residual symptoms of pain and/or itching. The husband was experiencing left arm, chest and thoracic back pain and an itchy sensation all over his body. The wife and two of the children had all over body itching and the third child had left foot pain. The teenager complained of headache and sore eyes. On examination there was no temperature reversal. The husband had a pulse of 50 BPM and ECG showed sinus rhythm. The rest of the family had normal heart rates and ECG's.

Two weeks post ingestion all of the family were asymptomatic with the exception of the husband who complained of intermittent body pains. His pulse was 62 BPM and ECG showed sinus rhythm.

Public Health Action

The remaining fish was retrieved by the doctor and sent to Royal Darwin Hospital Pathology for storage until the appropriate laboratory destination was established.

The husband and 16 year old were interviewed in the afternoon of 6 October to obtain information regarding the fish in this apparent ciguatera poisoning event.

After the interviews the following steps were taken to prevent any further ingestion of possible ciguatera toxin containing fish:

- Environmental Health Officers were contacted so they could notify local food outlets about the risks of selling locally caught fish
- the fisherman who caught the fish was interviewed to check distribution of fish he had caught at North East Island
- all local Community Health Centres were contacted and faxed copies of the 'Ciguatera Pamphlet'
- all Local Community Councils were faxed copies of the 'Ciguatera Pamphlet'
- all Health Centres on Groote Eylandt were sent pamphlets for distribution
- police assisted by contacting another family who were given fish to advise them not to eat it
- police were contacted to inform via radio a fishing party still reported to be fishing at North East Island regarding the risks of eating reef fish from the area.

The main problem encountered during the initial interviews was in establishing time frames. All of the people involved had English as a second language.

On 7 October fish samples of the barracuda were sent to a laboratory in Queensland Department of Agriculture in the Biotechnology Department. (No result by end of December 1998).

The police contacted the involved family to retrieve any other fish from the 5 October catch for analysis as a precautionary measure. One further fish was found which had been cleaned and cut into pieces with the species unknown at this stage.

Ciguatera poisoning occurs throughout the tropical and subtropical waters of the Pacific, Indian and Atlantic Oceans and surrounding Seas. Outbreaks and sporadic cases occur in residents and visitors of these areas and also in unsuspecting persons far from tropical waters who have eaten fish transported from these areas. This was highlighted in a recent newspaper report of a group from Melbourne with alleged ciguatera poisoning.¹

Ciguatera toxins are produced by dinoflagellates which are usually attached to algae growing in reef areas. The toxin is eaten by plant-eating fish who are in turn eaten by larger predatory fish who may be eaten by even larger fish. The toxin accumulates and concentrates in the fish, especially in the head, viscera (guts) and roe (eggs). The occurrence of the toxin is unpredictable but can be very localised eg, in one specific side or area of a reef. The toxin is usually associated with large (> 2 kg) predatory reef fish such as barracuda, coral trout, coral cod, trevally, grouper, mackerel and red emperor. In the NT, fish implicated in ciguatera poisoning have been caught around Bremer Island, Bonner Rocks, Miles Island, the Cape Arnhem area, north of Borrooloola, at Nhulunbuy (Gove)² and, for the first time, as reported above, from North East Island near Groote Eylandt. Deep sea fish eg tuna and dolphin are not known to carry ciguatoxins.

The toxin does not harm the fish and cannot be removed by freezing, cooking or cleaning the fish. The toxin is colourless, odourless and tasteless. The fish does not look spoilt.

There is no approved human assay for measuring ciguatera toxin and the diagnosis is based on having the characteristic signs and symptoms after eating large predatory reef fish. The symptoms usually come on 1 to 30 hours after eating the fish with the mean time being about 8 hours. The toxin can be tested for in samples of the fish - though often not in a timely fashion. Symptoms experienced are a combination of:

1. Acute gastrointestinal
 - nausea and vomiting
 - diarrhoea
 - abdominal pain;
2. Neurological
 - tingling and numbness around the lips, hands and feet
 - muscle weakness
 - headaches
 - severe pruritus

- hot and cold reversal; and occasionally
3. Cardiovascular
 - bradycardia
 - hypotension
 - respiratory depression.

Additional general complaints include tiredness and joint pains.

Neurological symptoms may be made worse by alcohol consumption, exercise, sexual intercourse and changes in dietary behaviour eg restricted diet or high-protein diet.³

The duration of the acute illness ranges from 1 to 8 days however neurological symptoms can last for months.

There is no known antidote or antitoxin and therefore treatment is supportive and to relieve pain and discomfort. Administering IV mannitol has been reported by some to be effective as an early treatment^{4,5} but results from controlled trials are not available.³

General precautions need to be followed when living or travelling in areas known to have ciguatera toxin carrying fish and include:

1. Fish species which are locally implicated should be avoided.
2. Large warm water reef fish should be treated with suspicion (or avoided altogether eg barracuda) with no more than 250 grams of flesh being eaten at a first sitting.
3. Under no circumstances should the head, viscera or roe of reef fish be eaten.
4. People who develop symptoms of ciguatera poisoning should seek medical advice immediately.
5. Fisherman should avoid known implicated areas and vendors should not sell fish from those areas.

There are public health actions to be taken when one case or an outbreak of ciguatera poisoning occurs. By notifying public health authorities as in the above outbreak further cases are avoided. Additionally, by gathering information on the number of incidents and the locations of ciguatera poisoning, the public, the departments of fisheries and medical services become more informed. Therefore the argument can be made, that like other foodborne illnesses with a public health response, ciguatera poisoning deserves consideration to

become a notifiable disease not only in the NT but nationwide.

Ciguatera reporting forms can be obtained through CDC, Darwin on 8922 8044.

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What's happening with sexually transmitted diseases in the NT?

Steven Skov, Medical Officer, AIDS/STD Unit, Darwin and CRC project

What is the situation in the NT?

A review of STD notifications in the Northern Territory (NT) over the last few years suggests a modest decline in the amount of syphilis.¹ However, notified cases of gonorrhoea and chlamydia have increased recently, probably due to the introduction of urine and tampon PCR tests which have facilitated detection. Table 1 displays the rates per 100,000 population for syphilis, gonorrhoea and chlamydia for the NT compared to Australia as a whole in 1997.² While the rates in the NT are higher for both Aboriginal and non-Aboriginal people, it is clearly a more serious problem for Aboriginal people. Studies done in central Australia suggest that in many remote communities the prevalence of infection with either gonorrhoea or chlamydia is extremely high.³⁻⁶ The excess in these prevalence rates is mainly for gonorrhoea as the rates for chlamydia are quite similar to those which have been reported recently in some non-indigenous populations.^{7,8}

Table 1 Rates of STDs per 100,000 population in the NT and Australia²

	Syphilis	Gonorrhoea	Chlamydia
Australia	6.7	23.4	50.4
NT Non-Aboriginal	17	204	204
NT Aboriginal	496	1790	787

Clearly there is a large problem, but what does this mean in real life? Or, why do we worry about gonorrhoea or chlamydia anyway? Two very real reasons are 1) They roughly double the risk of HIV transmission^{9,10} and 2) they cause PID, infertility and ectopic pregnancies. In the NT there is very

little information on the extent of PID and infertility, but recent studies in two remote Top End communities found that 26% and 30% of the women were infertile.^{11,12} From what we know of the nature of infertility and studies done elsewhere, we can estimate that *at least* half of these women are probably infertile *because of an STD*.^{13,14} Why do we worry about syphilis? Again, because of increased risk of HIV transmission and also because of the miscarriage, stillbirth and congenital syphilis it causes. Information is not available on the number of babies lost in this way or on the number of babies who have a lumbar puncture and/or are treated empirically at birth to make sure that they do not have or get syphilis.

What more do we need to understand about STDs?

In order to develop rational programs to address STDs we need to understand more about the diseases themselves, the dynamics of their transmission at both individual and population levels and how people with STDs interact with clinical services. We need to know what type of programs community members would actively support and clinic staff could successfully run on a sustainable basis.

The majority of people with an STD probably will not present to a clinic. A very large proportion of STDs are asymptomatic. As many as 10% of all men and more than 50% of women with gonorrhoea will have no symptoms. About 70% of women and 30% of men with chlamydia will not know they are infected.¹⁵ Even if symptoms are present it does not necessarily mean the person will go to the clinic. Symptoms may be mild or the person may not

understand what is causing the symptoms. People may be unwilling to go to the clinic because they are ashamed, afraid of the tests, concerned about confidentiality or the clinic may not have separate men's and women's areas and practitioners. If a person does present to the clinic both they and their partner(s) need to be properly treated. In some African countries it has been estimated that 5% or less of all people with an STD get to this point.¹⁶ Figure 1 shows schematically STD prevalence in the community, the steps needed to ensure effective treatment and therefore the percentage of STDs effectively treated. This model is based on studies from Uganda, Zaire, and Tanzania.

Figure 1 Piot-Fransen model of STD management in rural women¹⁶

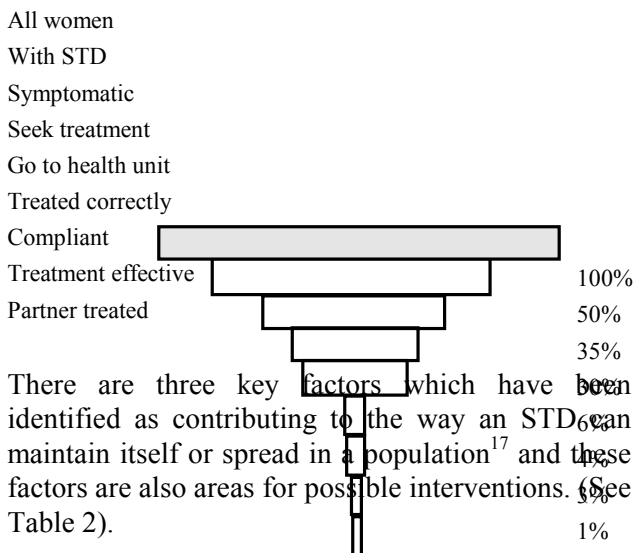


Table 2 Factors contributing to STD transmission

Factor	Possible intervention
Rate of sexual partner change	Education to reduce number of sexual partners
Risk of infection per act of unprotected intercourse	Health education and promotion about condom use to reduce risk of infection
Duration of infection	Early detection strategies Effective treatment Short delays between diagnosis and treatment Partner notification and treatment Education to encourage people to self-present

The probability of transmission of STDs varies with the organism and circumstances. For example, it is thought that the risk of transmission of gonorrhoea from an infected man to a woman can be as high as between the Primary Health Care (PHC) sector and the public health sector.^{19,20}

80% while from a woman to a man is less than 50%. The dynamics of syphilis transmission is different to that of gonorrhoea and chlamydia. Syphilis usually has two relatively brief periods (4-6 weeks) of infectiousness which resolve spontaneously: ie when the chancre is present and during secondary syphilis. The risk of transmission from a chancre is about 30% but the infectiousness of secondary syphilis may be quite low if there are no condylomata or mucous patches present. However, a woman with untreated gonorrhoea or chlamydia may be *continuously* infectious for perhaps as long as eighteen months while a male with gonorrhoea may be infectious every day for up to six months.¹⁷ Therefore, the types of strategies we use, especially for early detection and treatment, may need to be different for the different STDs.

It is now widely believed that an important factor in transmission of STDs within a population is the presence of a so-called "core-group" of high risk individuals.¹⁸ Groups of this type probably exist within most societies and probably consist of a relatively small number of people who have a large number of sexual partners and very high rates of STDs. The sexual interaction between the core-group and the rest of the population acts to maintain a higher level of infection in the whole. It is thought that by focusing on the core-group with education and testing, treatment and follow-up strategies, it may be possible to confer a benefit on the whole community.

Where does this lead us?

In the NT a great many health services are relying on symptomatic persons to present to the clinic as their main STD "strategy". This we know will probably result in most people with infection remaining untreated and possibly continuing to infect others. Though effort has been put into educational programs and opportunistic screening over the years there are very few health services in the NT with comprehensive STD/HIV programs in place. These health services need specific aims, and strategies which are informed by the data and an understanding of transmission dynamics to achieve desired outcomes. Unfortunately, the approach to STD control has often been ad-hoc with little coordination between different health services or

STD/HIV control from Operations North and Central needs to assist and support the PHC services to provide good programs. STD programs need to be

integrated into PHC clinic routines as advocated by the WHO.¹⁵ Nganampa Health Council have been doing so for some years and have reduced their prevalence of active syphilis by more than 90% since 1985.⁵ They have also reduced their prevalence of gonorrhoea by 46% and of chlamydia by 20% in the past two years.²¹ Ngaanyatjarra Health Service and Pintupi Homelands Health Service have embarked on an effort to put in place a comprehensive set of strategies. The challenge remains for other health services to improve their STD/HIV programs. As with any communicable disease a regional, coordinated approach involving all health services is essential. The good efforts of one health service will be compromised if adjacent health services are doing little.

There are many ways in which STD/HIV programs can be improved and innovations introduced. There are many examples of successful initiatives from around the world and within Australia. Strategies for the early detection and treatment of bacterial STDs are being increasingly advocated around the world not only as a means to address the morbidity they cause but also to reduce HIV transmission.²²⁻²⁴ Community health promotion and education strategies are very important and must be reinforced. However, they will be insufficient on their own to deal with the problem. The National Indigenous Australians' Sexual Health Strategy emphasises that activities for early detection and treatment of STDs need to be conducted as well.²⁵ What remains to be found is the willingness to do so.

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Editorial

The review of sexually transmitted diseases (STDs) in the Northern Territory and the above article are very timely as the AIDS/STD Program is embarking on the development of a Remote STD/BBV (blood borne virus) Strategy. This is in response to the continuing high rates of chlamydia, gonorrhoea and syphilis and concerns about the potential for HIV and Hepatitis C entering and spreading throughout remote communities.

The aim of the Strategy will be to prevent the serious complications which result from these infections by decreasing their prevalence and incidence. Here we have a number of bacterial infections which are frighteningly common and often unnoticed. However, they are readily prevented through safe sexual practices, and where these fail, can be simply diagnosed and easily treated. A program leader's dream! To date, however, despite these features, little impact has been made on the burden of STDs.

Skov outlines some of the barriers to effective STD control: lack of knowledge, asymptomatic disease, sense of shame, lack of suitable services, extended infectivity and the existence of a "core group" of transmitters. The fundamental block to disease

prevention is the lack of recognition and ownership of the problem and its magnitude, at all levels, from the individual through to community and political leaders. Major aims of the Strategy will be to provide information about STDs and barriers to their control to people, to seek opinions about possible interventions and to assess community and individual commitment to STD control. More regular reports on STD/BBV rates and general results of preventive activities (screening, community educational initiatives, etc) are planned.

This article describes the importance of community health services integrating STD programs into their core (primary health care) business as well as coordinating their activities with that of neighbouring communities. This will require central and regional support, good communication, planning, flexibility and cooperation from all involved. Programs must be effective and sustainable as the benefits of intervention will only be seen in the medium to long term. Those programs which *are* seeing health gains for their communities are distinguished, in part, by their recognition of their problems and commitment to their solutions as a priority.

Letters to the Editor are most welcome to provide a brief report, a comment on eg should ciguatera be notifiable? or to convey additional information and/or experience on topics printed.

Revised guidelines for the investigation and treatment of congenital syphilis in the Top End of the Northern Territory (NT)

Background

Congenital syphilis is a rare but preventable cause of death and disability in children of mothers infected with syphilis. Infection is passed from the usually asymptomatic mother to her unborn infant. The infant requires prompt treatment and follow-up to minimise long term complications.

It is also important to treat the mother and her contacts to prevent further pregnancies from being put at risk. In the NT there are between one and seven cases notified to CDC each year, but the true number is probably higher.

Clinical Features

Although infants may be asymptomatic, congenital syphilis can lead to abortion, usually in the second trimester, stillbirth, or signs of infection in the infant including those described below.

Clinical Description

Congenital syphilis is a condition caused by infection *in utero* with *Treponema pallidum*. A wide spectrum of disease exists and only severe cases are clinically apparent at birth. Congenital syphilis is divided into two categories, early and late. For the purposes of these guidelines we have emphasised the manifestations of early congenital syphilis.

Early Congenital Syphilis - less than two years of age.

Clinical signs include:

low birth weight, jaundice, pseudoparalysis, hepatosplenomegaly, skin rash (including red palms and soles), mucocutaneous lesions, nephrotic syndrome and pancreatitis.

For a more comprehensive description of congenital syphilis see elsewhere.^{1,2}

Common investigation findings:

- abnormal LFTs
- abnormal FBE (high or low white cell counts, low platelets)
- syphilitic changes on long bone X-rays

Laboratory criteria for diagnosis

Demonstration of syphilis IgM from *venous* blood (not cord blood as spillage from maternal circulation may occur). If cord blood only is available, discuss

positive result with experienced clinician for interpretation.

Late Congenital Syphilis - greater than two years of age.

An older child may have stigmata such as interstitial keratitis, nerve deafness, anterior bowing of shins, mulberry molars, Hutchinson's peg teeth, saddle nose, rhagades, or Clutton's joints.

Case Definition

A definition adapted to NT conditions has been used here.³ Case classifications in textbooks may be slightly different from our guideline classifications. There are two reasons for this.

- 1) Congenital syphilis is a treatable disease. It is better to treat a few infants who do not have syphilis than it is to miss treating infants who are infected.
- 2) It is better to base treatment decisions largely on maternal status as the mother is the source of infection. Tests results on the infant may take time and some results are not particularly reliable.

Confirmed: a positive darkground examination in fetal/placental tissue (rarely performed)

Presumptive:

Maternal reasons - RPR positive and any one of the following:

- Untreated or inadequately treated or non-penicillin treated syphilis
- Treatment not completed before 30 days prior to delivery
- Maternal RPR titre \geq 1:8 or not declining as expected
- Mother likely to be lost to follow-up
- Mother treated with non-penicillin regimen

Infant reasons - any two of the following:

- Any evidence of congenital syphilis on physical examination or on long bone X-ray
- A positive venous syphilis IgM (if only cord blood available, consult with experienced clinician)
- An elevated CSF cell count or protein (without other obvious cause)
- A positive CSF VDRL

- Infant RPR \geq 4 times mother's titre

Management of suspected congenital syphilis

The following are summarised in flow charts in the guidelines (not shown here).

1. Categorise neonatal risk status

A. No risk

Maternal perinatal RPR negative.

B. Low Risk

Any one of the following:

- Maternal syphilis in pregnancy with adequate treatment completed prior to 30 days before delivery with no possibility of reinfection after treatment
- Maternal perinatal RPR \leq 1:4 and past history of adequately treated syphilis
- No maternal antenatal care (NB evaluate carefully to ensure infant is not high risk)

C. High risk

Maternal RPR positive and any one or more of the following:

- Treatment of the mother absent or inadequate
- Maternal treatment not completed 30 days before delivery
- Maternal reinfection post-treatment likely (eg partner not treated)
- Maternal RPR \geq 1:8 at delivery
- Mother/infant highly likely to be lost to follow-up
- Mother treated with non-penicillin regimen
- Clinical, radiological or CSF exam of infant strongly suggestive of syphilis
- Infant RPR \geq 4 times titre of mother
- Positive darkground microscopy of fetal/placental tissue

2. Investigate and treat appropriately

A. No risk

- No investigations/treatment

4. Arrange adequate follow-up

Follow-up should be ensured *prior* to discharge by:

B. Low risk

- Examine for clinical signs
- Check RPR/TPPA. If RPR \geq 4 times mother's, go to high risk
- Enter name in Congenital Syphilis Register
- **Administer benzathine penicillin 50,000 units/kg IM single dose.**
- Follow-up at six months of age with venous RPR

C. High Risk

- Examine for clinical signs
- Check RPR/TPPA, FBE, LFT
- Long bone X-rays
- Lumbar puncture only if facilities available
- Enter name in Congenital Syphilis Register
- If all above are negative, treat as for low risk (as above)
- If any of above are positive, **administer benzyl penicillin 50,000 units/kg/dose IV 12 hourly for 10 days**
- Follow-up at six months of age with venous RPR. LP only if previous LP suggested neurosyphilis

3. Notify Syphilis Register

All low and high risk babies should be notified to the Register. This is done by photocopying and filling in the notification sheet at the back of the guidelines (shown on page 17 of this *Bulletin*) and sending it via internal mail to "Community Paediatrician, CDC Darwin".

The information is required so that:

- The relevant district medical officer (DMO)/local medical officer/Clinic can be informed in writing of the time of each review, and to estimate numbers of children requiring treatment each year.
- To ensure adequate treatment of the mother has taken place.
- To monitor low risk babies to see whether follow-up of this group is necessary.
- Notifying the DMO/doctor caring for the mother and infant in writing and specifying the six month follow-up on the discharge summary.

- Where possible the child should be reviewed by a visiting paediatrician.

If a repeat LP is required, a return to hospital should be arranged.

Pitfalls in CSF examination

The upper limits of normal CSF findings for protein and leucocytes in the neonate are variable.

- The levels of protein in a normal neonate can be as high as 1.5mg/ml, and
- the normal white cell count can be as high as 30 leucocytes/ml.

In addition not all centres in the Top End will have facilities to perform lumbar punctures.

Therefore for practical purposes all infants classified as high risk are treated as if they have congenital syphilis.

- Where facilities are available, CSF examination should be performed in high risk children, as a positive CSF VDRL test as well as a high protein level and large number of white cells may confirm the diagnosis.
- Equivocal levels of protein and/or white cells should not change the need for treatment.
- Equivocal cases should be discussed with the consultant paediatrician caring for the child and/or a physician from the AIDS/STD Unit, Darwin and/or an Infectious Diseases physician.

Health care professionals

Responsibilities to mother

Every mother should receive at least one test for syphilis during pregnancy.

Health care professionals providing ante-natal care for the mother will ensure that all mothers who are at high risk of syphilis be identified and additional appropriate tests be performed, eg mothers who are classified as "greater risk" for syphilis should receive a further RPR at the time of confinement.

Alice Springs. An effort has been made to make the guidelines more applicable to rural settings, incorporating some suggestions particularly from Katherine and Tennant Creek Disease Control staff eg lumbar puncture is no longer an integral part of

For a number of reasons Aboriginal mothers, particularly those from rural areas, should be considered amongst those who are at "greater risk". Each mother should have the need for the test explained, for both mother (and also the infant if testing is required), and verbal consent obtained prior to the tests being performed. The mother with a positive RPR should be treated with penicillin if there is no history of adequate treatment. If there has been no ante-natal care, take an RPR as soon as possible and consider whether empiric treatment of the mother and infant is needed.

Responsibilities to infant

Health care professionals caring for the infant will ensure that all infants be classified as high risk, low risk or no risk for congenital syphilis.

Health care professionals caring for the infant will undertake appropriate investigations and other necessary steps for the infant depending on their classification. The mothers of infants who are to receive venous testing for syphilis should be informed of the need for the test and verbal consent obtained prior to the test being performed.

Paediatric staff will notify all children of mothers considered at risk (high or low) to the Syphilis Register for follow-up of congenital syphilis.

Tests on cord blood are unreliable and should not be performed in most instances

There is a risk of mixing or spillage of syphilis antibodies from the maternal circulation, which may result in false positive results for the infant. This is why venous sampling of the infant is important. If there is a question about staff ability to take venous blood, cord blood is better than no blood at all. Take cord blood and store for use only if venous blood cannot be taken.

Discuss all positive results on cord blood with an experienced clinician.

These guidelines have had input from hospital, community and Disease Control staff including those from Katherine, Tennant Creek, Gove, and management as it is not feasible to perform in some of the smaller centres in the NT. These guidelines are currently for use in the Top End of the NT. As Alice Springs has its own syphilis database and a different system for diagnosis, recall, and follow-up,

their guidelines differ slightly from what is printed here. Alice Springs presently follows the CARPA Standard Treatment Manual for the management of congenital syphilis.

If there are any comments or suggestions regarding the revised guidelines please send them to Dr Jacki Mein or Dr Ingrid Bucens (currently running this project) by email via THS or phone 8922 8788.

Acknowledgements

Thankyou to all people who assisted with comments in the revision of these guidelines. These include Dr Jan Bullen, Katherine CDC, Sharon Doyle and Fiona Maslin, Tennant Creek CDC, Liz Stephenson, Gove CDC, Dr Janet Knox and Penny Kenchington, Sexual Health Unit, Alice Springs, Dr Karen Edmond, Community Paediatrician Darwin CDC, Dr Jan Savage, Co-ordinator, Darwin AIDS/STD

Unit, Dr Gary Lum, Microbiology Unit RDH, Dr Angela Merianos, Head, Immunisation and Surveillance, Darwin CDC, and Dr Bart Currie, Infectious Diseases Consultant, RDH, Territory Health Services.

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Editorial comment - Ingrid Bucens, Paediatrician, Royal Darwin Hospital

Congenital syphilis (CS) is a devastating but entirely preventable outcome of a sexually transmitted disease. As recently as this year, outbreaks of CS are being reported in epidemiological literature.¹ The October 1998 *MMWR* (CDC Atlanta) reports an epidemic of CS in Baltimore. From 1993 to 1996 the rate of CS increased from 62 to 282 per 100,000 live births. It was determined that 90% of cases could have been prevented by adequate prenatal care i.e. by timely syphilis screening and treatment. The numbers may even be underestimates, as CS is known to cause stillbirths and these figures are not reported. Fifty eight percent of mothers of cases had no antenatal care or initiated antenatal care late in the third trimester.

This year members of staff at Royal Darwin Hospital (RDH) and the Darwin Centre for Disease Control (CDC) revised preexisting RDH CS guidelines. The preexisting guidelines, written in 1994 (Ruben et al), were based on the CARPA manual.⁴ Knowledge of their existence amongst staff was limited and they were underutilised.

These revised CS guidelines are intended for use in the Top End. A CS register in Darwin has also been established with the aim of being able to describe the prevalence of CS in our population. This register will generate reminder phone calls at appropriate times to the relevant

regions/communities regarding follow-up tests and examination of children notified to the register. It is located within the Community Paediatric Unit, Block 4, RDH. The new guidelines require notification of all Top End cases to the CS register.

The guidelines still determine an infant's risk category for CS (no risk/low risk/high risk) based on maternal screening and treatment status. There are now a few differences between the new guidelines and the CARPA manual⁴ and/or the preexisting RDH guidelines which include:

- 1) The CARPA manual⁴ states that maternal syphilis treatment is adequate if, along with other requirements, it is completed before 29 weeks gestation. These new guidelines push forward that date of completed treatment to *30 days* before delivery. All recent guidelines including CDC, Atlanta^{2,3,5,6,7} use this latter classification. Thus, not only is it consistent with international recommendations, but it also limits the numbers of infants who would be questionably classified as high risk.
- 2) The CARPA⁴ manual uses the maternal RPR titre of 1:64 to classify infants at risk of CS. We have chosen a titre of 1:8 or greater. This choice of titre is clearly more conservative. There is controversy surrounding what single titre is best

used as a "cut off". In Queensland a titre of 1:8 is notifiable.⁸ The *MMWR* 1990 use a titre of 1:4.² We consider it prudent to use 1:8 as a cut off, being aware that we are, in this case, being over rather than under inclusive.

- 3) The CARPA⁴ manual states that all high risk infants should undergo lumbar puncture (LP) to determine/exclude the diagnosis of neurosyphilis. We support this recommendation but acknowledge its impracticality in certain circumstances. A neonatal LP requires a person with appropriate training and skill to be present. Clearly this will not always be the case, particularly in smaller regional hospitals. It is inappropriate and possibly dangerous to insist upon this procedure if skilled personnel are not present. Even in larger centres a prompt CSF specimen often does not provide a diagnosis or alter treatment initially. Neurosyphilis can be difficult to diagnose. Firstly, the cell count and protein concentration in normal newborns is often high therefore 'clouding' the picture. Secondly, results are not timely as CSF "syphilis tests" (VDRL etc) are transported to Darwin and then interstate with results often taking over a week. Thirdly, many CSF specimens are bloodstained and this too interferes with diagnosis (alters cells, protein and "syphilis tests"). Thus, a LP need not be performed if a skilled person is unavailable. In these cases, or in the case of CSF being nondiagnostic, a full course of penicillin should be given, with the assumption that the infant has neurosyphilis. Treatment should not await the CSF result which may take a week or more to return.

It is well recognised in the NT population that there are high rates of sexually transmitted diseases and late presentations for antenatal care. It is essential to screen all antenates for syphilis and to ensure adequate response to treatment when it is initiated in pregnancy. All infants must be evaluated for their risk of CS as CS is a preventable and easily treatable condition.

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Late Update - As of 31 December 1998 there have been 17 cases of melioidosis from the Top End with 2 deaths. Almost half have been from the Tiwi Islands following the heavy rains of Cyclone Thelma (see page 18).

CONGENITAL SYPHILIS REGISTER NOTIFICATION SHEET

Please send via internal mail to:
"Community Paediatrician, CDC, Block 4, Darwin"

Referring Centre: RDH KDH TCH GDH

Notifying Doctor: Date: ___ / ___ / ___

(If available, affix hospital stickers below, child on left, mother on right)

Child's name:

Child's DOB: ___ / ___ / ___

HRN:

Mother's name:

Mother's DOB: ___ / ___ / ___

HRN:

Address/Community:

Date of first day of child's treatment: ___ / ___ / ___

Six month follow-up mentioned in discharge letter: Yes No

Treatment given: high risk,
 benzyl penicillin 50,000 units/kg/dose IV 12/24 for 10/7

low risk,
 ie benzathine penicillin 50,000 units/kg IM stat

other, please specify

 Drug:

IM IV oral

 Length of course days

Mother's RPR (if known) 1: _____ Date of test: ___ / ___ / ___

Serology sent on child: Yes No

If yes, results: RPR TPPA Don't know

Lumbar puncture on child: Yes No

If yes, results: RCC WCC Protein

 VDRL

Keep melioidosis in mind in the monsoon

*Bart Currie, Dale Fisher, Nick Anstey, Gary Lum, Adam Jenney, Dianne Stephens and Susan Jacups,
 Royal Darwin Hospital and Menzies School of Health Research*

With the heavy rains in the coastal Top End there have been 9 cases of melioidosis admitted to Royal Darwin Hospital (RDH) in the first half of December, 1998. Six of these 9 have been blood culture positive and 1 diabetic woman has died with fulminant melioidosis pneumonia. During the

1997/98 wet season there were a record 45 cases of confirmed melioidosis, with 4 deaths. The prospective melioidosis study at RDH has now documented over 240 cases of melioidosis in the Top End in the last 9 years, with 215 confirmed by culture of *Burkholderia pseudomallei*.

Important facts about melioidosis

1. The bacterium, *Burkholderia pseudomallei*, is an environmental organism found in soils and muddy water in the Top End. Most infection is thought to be acquired through percutaneous inoculation.
2. It is 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 a mortality of 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 septicemia with multi-organ abscesses and unusual neurological illnesses such as brainstem encephalitis and acute paraplegia. Persons without symptoms or a known history of disease can also be found to be positive on serological testing.
4. 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 occasionally in children and healthy adults.
5. 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.
6. Mortality is decreased by early diagnosis and appropriate antibiotic therapy.
7. Follow-up of cases and adherence to eradication therapy (usually at least 3 months of antibiotics after discharge) is critical to prevent relapse, which can be fatal.
8. The Top End empirical protocol for adult community-acquired pneumonia is devised to cover both melioidosis in patients with risk factors, as well as other important pathogens. (see page 19 this *Bulletin*).
9. Once melioidosis is confirmed the 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.

Recent studies in Thailand have confirmed that imipenem (at RDH we use meropenem) is at least as good as ceftazidime for the initial intensive therapy of melioidosis. Studies in Thailand have also shown that doxycycline monotherapy for the eradication of melioidosis has a higher relapse rate and therefore doxycycline monotherapy is generally not used at RDH anymore as first line therapy or eradication therapy of melioidosis. However, some patients will require doxycycline or other antibiotics, as an alternative to cotrimoxazole for various reasons.
10. Each monsoon cases of melioidosis occur in travellers returning from tropical Australia to southern states or overseas countries.
11. 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 are especially important to emphasise for all diabetics.

Treatment of adult community acquired pneumonia in the Top End

Bart Currie, Dale Fisher, Nick Anstey and Gary Lum,
Royal Darwin Hospital and Menzies School of Health Research

The prime aim of treating community-acquired pneumonia is to prevent death. We have been looking at the more severe pneumonias admitted to Royal Darwin Hospital (RDH), represented by patients with positive blood cultures (bacteraemic). The findings have been used to define treatment guidelines based on severity of pneumonia and presence or absence of risk factors. Of interest is the very few numbers of cases at RDH diagnosed as "atypical pneumonia". While there may well be more cases of "atypical pneumonia" presenting in the community, it appears likely that the relative importance of these is clearly less than in southern Australia.

Table 1 shows the 4 commonest organisms isolated in 255 cases of adult community-acquired bacteremic pneumonia. As elsewhere in the world, *Streptococcus pneumoniae* is the commonest organism overall. However, *Burkholderia pseudomallei*, the organism causing melioidosis, accounts 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.

Treatment Protocols

People with risk factors such as diabetes, alcohol, chronic lung disease, chronic renal failure and steroid therapy are at particular risk for melioidosis and *A. baumannii* pneumonia. Recently excessive kava consumption has been associated with melioidosis as well. However, melioidosis will occasionally occur in an apparently immunocompetent person and therefore needs to be

covered by antibiotics for any presentation with severe pneumonia in the Top End.

Table 2 defines the initial therapy of adult community-acquired pneumonia in the Top End. Irrespective of risk factors, mild pneumonia is treated with penicillin 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 we have recently changed therapy to meropenem and gentamicin. 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. 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 "Keep melioidosis in mind in the monsoon," previous page). Ceftriaxone however, has a better gram positive coverage than ceftazidime. When used with gentamicin, ceftriaxone will generally hold *Staphylococcus aureus* infection initially. Once *S. aureus* is isolated then the appropriate treatment becomes usually high dose 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 ticarcillin/clavulanic acid (Timentin) or piperacillin or meropenem is added.

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 Cefazidime 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.
+See text for choice

Brief reports - recent outbreaks in late 1998

Jacki Mein^{1,2}

¹CDC, Darwin, ²MAE program, NCEPH, ANU, Canberra

Hepatitis A

There have been five confirmed cases and one probable case of hepatitis A in one class area of a Darwin primary school in November and December 1998. Five students and one teacher were affected and all are now recovering. No further cases at the school have been identified to date and the timing of cases suggests it was due to a common source of infection. Possible transmission links are still being investigated by CDC. Families and school staff have been educated regarding transmission and prevention of hepatitis A and immunoglobulin has been offered to appropriate household contacts.

Salmonella kinondoni

S. kinondoni is a rare *Salmonella* serotype seldom notified in Australia. Over the past seven years the annual number notified in the Northern Territory (NT) has varied from zero to five cases per year. Occasional isolates are notified from Queensland and New South Wales but the majority in Australia appear to come from the NT. These isolates are most often recovered from non-human specimens such as nuts, buffalo and kangaroo meat.

Ten human cases from two laboratories were notified to CDC in Darwin in late November 1998.

Although the cases all occurred within ten days of one another they were widely scattered all over the Top End. Seven of the ten cases were able to complete extensive food surveys but no definitive link to particular foods was established. The three week time delay prior to notification of Salmonella serotype results often made recall by the cases (of food eaten at the time they would have been infected) difficult and possibly unreliable. No further cases have been identified to date.

Acute Rheumatic Fever

Even in the Top End of the NT where rheumatic fever is endemic, it is unusual to get clusters of cases occurring. However over the period August to November 1998 in one Arnhem land community

there were three cases of acute rheumatic fever (ARF) in children between eight and ten years, all presenting with chorea. Over the last twenty years there have been only eight other non-clustered cases identified in the same community. All three were children in this cluster and fulfilled the modified Duckett-Jones criteria for diagnosis of ARF and had been resident in the community for at least six weeks prior to becoming ill. Although unrest within the community prevented full scale screening of children at risk for Group A streptococcal throat carriage, limited screening was performed and no further cases have come to the attention of health centre staff.

Adult immunisation - new initiatives for 1999

National Influenza Vaccine Program

Federal Government health initiatives will provide annual influenza vaccination free of charge to all Australians 65 years and over from 1999. Funding has been made available for 82% of the target population. In the Northern Territory, vaccination for the 1999 'flu season' will commence as soon as the vaccine becomes available (approximately early to mid February). Vaccine distribution will be through Territory Health Services (THS) hospital pharmacies in each of the five districts using the pathology courier system. General practitioners and other vaccination providers are advised to start placing orders for influenza vaccine as soon as possible after the New Year to enable the pharmacies to gauge the number of vaccines likely to be required.

National Indigenous Pneumococcal and Influenza Immunisation Program

Federal funding has also been allocated for a National Indigenous Pneumococcal and Influenza Immunisation Program. This program, developed in association with the National Aboriginal Community Controlled Health Organisation (NACCHO) and funded by the Commonwealth Office for Aboriginal and Torres Strait Islander Health Services (OATSIHS) aims to increase immunisation coverage within the indigenous population in order to reduce the high rates of acute respiratory illness and death. Five yearly pneumococcal and yearly influenza vaccine will be

available free of charge through community controlled Aboriginal Medical Services, State/Territory Health Authorities and general practitioners for the indigenous target group. Funding will be provided initially for all indigenous people over 50 years and indigenous people in the 15-50 year old age group who meet the NHMRC recommendations for immunisation based on risk factor assessment. The vaccines will be distributed as for the over 65 influenza program, through THS hospital pharmacies.

The Commonwealth will require the following aggregated non-identified information for accountability purposes only:

- number of vaccines used;
- demographic information on age, sex and Aboriginality of each client given the vaccines (to ensure vaccines are reaching the target population); and
- estimation of vaccine wastage levels.

THS have been promoting increased uptake of influenza and pneumococcal vaccine as part of the annual NT Adult Immunisation Campaigns since 1995. We are therefore ahead of most States in that a large proportion of Aboriginal people with risk factors for pneumococcal disease has already been vaccinated against pneumococcal disease.

Further meetings are planned with key stakeholders early in the New Year to discuss implementation options. Detailed information outlining both programs will be sent to all doctors and relevant health professionals as soon as they are finalised.

Flu shots for health staff - reminder for 1999

Immunisation guidelines recommend annual influenza vaccination for staff working in Accident and Emergency (A&E), Coronary Care Unit (CCU), Intensive Care Unit (ICU), special care nursery and general medical wards. In A&E the recommendation is made because of high risk of exposure of staff to influenza virus and the potential for many concurrent staff absences due to illness. ICU, CCU, special care nursery and most staff on general medical wards work directly with patients who would be at risk of serious complications if influenza is acquired. The vaccine is therefore recommended for these groups to reduce the risk of patients acquiring influenza from the staff as well as to reduce staff illness during outbreaks of influenza which are invariably associated with increased hospital admissions.

Since outbreaks of influenza have been documented in the Top End as early as February it is important that staff and high risk individuals (see below) are

vaccinated as soon as the vaccine is released. It is anticipated that the 1999 influenza vaccine will be available early February.

A staff immunisation clinic is held every Friday in the RDH Outpatients Department from 2-3 pm. Appointments for influenza (and other appropriate vaccinations) MUST be made in advance by ringing 28699. In addition to the service provided through the staff immunisation clinic, the acting Clinical Nurse Consultant (A/CNC), RDH Outpatients Department will visit work areas identified as 'high risk' and offer 'on the spot flu shots' in February 1999. Staff must provide the A/CNC with their Hospital Record Number before the flu vaccine will be given.

Health staff working in remote communities, or other situations in which there is limited relief available for ill staff, should also ensure they are vaccinated against influenza.

Annual influenza vaccine is recommended for the following groups:

- All Australians 65 years and over and Aboriginal people over 50 years of age.
- Those with chronic heart, lung, kidney, liver or metabolic disorders such as diabetes.
- Adults and children receiving immunosuppressive therapy.
- Children with cyanotic congenital heart disease.
- Residents of nursing homes and other chronic care facilities.
- Staff of nursing homes, chronic care facilities and carers of immunocompromised patients.
- Health staff in Accident and Emergency, Coronary Care Unit, Intensive Care Unit, Neonatal Intensive Care Unit and general medical wards.
- Health staff in remote communities, or other situations in which there is limited relief available for ill staff.

NT MALARIA NOTIFICATIONS

July to September 1998

Compiled by Merv Fairley, CDC, Darwin

Six notifications of malaria were received for the third quarter of 1998. 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
PNG	Visit	<i>P. vivax</i>	Yes	Diagnosed RDH
PNG	PNG National	<i>P. falciparum</i>	Yes	Diagnosed RDH.
W Africa	W. African National	<i>P. falciparum</i>	Yes	Diagnosed RDH.
PNG	Holiday	<i>P. vivax</i>	No	Diagnosed ASH. Relapse.
PNG	Holiday	<i>P. vivax</i>	No	Diagnosed ASH. Relapse.
Indonesia	Fisherman	<i>P. falciparum</i>	No	Diagnosed RDH.

**NT NOTIFICATIONS OF DISEASES BY DISTRICTS
1 JULY TO 30 SEPTEMBER 1998 AND 1997**

DISEASES	ALICE SPRINGS		BARKLY		DARWIN		EAST ARNHEM		KATHERINE		TOTAL	
	'98	'97	'98	'97	'98	'97	'98	'97	'98	'97	'98	'97
Acute Rheumatic Fever	2	3	0	0	3	0	3	0	4	1	12	4
Adverse Vaccine React.	1	2	0	0	3	0	0	0	0	0	4	2
Arbovirus infections												
Barmah Forest Virus	0	0	0	1	2	4	1	0	0	1	3	6
Dengue	0	0	0	0	4	0	0	0	0	0	4	0
Kunjin Virus	0	0	0	1	0	0	0	0	0	0	0	1
Ross River Virus	0	3	1	0	7	8	0	0	2	3	10	14
Campylobacter	17	12	0	0	21	32	5	1	4	1	47	46
Chlamydia	77	80	9	8	98	60	8	5	24	17	216	170
Cryptosporidium	8	-	0	-	5	-	0	-	0	0	13	-
Donovanosis	2	5	0	0	1	1	0	0	0	2	3	8
Gastroenteritis	0	0	0	0	0	3	29	0	0	0	29	3
Glomerulonephritis	5	2	0	0	0	4	2	0	0	0	7	6
Gonococcal Disease	104	137	9	13	125	32	29	9	63	18	330	209
Gonococcal Conjunct.	0	4	0	4	1	0	0	0	0	0	1	8
Haemophilus Inf type b	0	1	0	0	0	0	0	0	0	1	0	2
Hepatitis A	0	3	0	1	4	12	3	0	2	2	9	18
Hepatitis B	0	1	0	1	0	2	0	0	6	2	6	6
Hepatitis C (incidence)	0	0	0	0	0	1	0	0	0	0	0	1
Hepatitis C (prevalence)	5	12	2	1	65	83	1	0	5	1	78	97
HIV infections	0	0	0	0	2	1	0	0	0	0	2	1
HTLV-1	8	7	1	0	0	1	0	0	2	0	11	8
Legionnaires Disease	0	0	0	0	2	0	0	0	0	0	2	0
Leptospirosis	0	0	0	0	0	0	0	0	1	0	1	0
Malaria	0	4	0	0	3	2	0	0	0	0	3	6
Measles	0	0	0	0	0	1	0	0	0	0	0	1
Melioidosis	0	-	0	-	0	-	0	-	1	-	1	-
Meningococcal Infect.	6	0	0	0	0	2	0	1	0	1	6	4
Mumps	0	0	0	0	1	4	0	0	1	0	2	4
Pertussis	0	2	0	0	9	2	0	0	0	1	9	5
Pneumococcal Disease	13	22	0	0	5	7	0	0	0	1	18	30
Rotavirus	6	29	7	8	51	142	8	30	5	12	77	221
Rubella	0	1	0	0	0	1	0	0	0	0	0	2
Salmonella	14	13	1	3	48	31	8	25	9	10	80	82
Shigella	10	6	1	7	7	13	6	9	1	6	25	41
Syphilis	42	47	19	15	14	6	15	3	4	7	94	78
Tuberculosis	0	1	0	0	7	5	0	1	0	1	7	8
Yersiniosis	0	0	0	0	0	2	0	0	0	0	0	2
Total	320	397	50	63	488	462	118	84	134	88	1110	1094

Points to note regarding notifications:

- Australian Encephalitis (MVE, Kunjin), Amoebiasis, Botulism, Brucellosis, Chancroid, Cholera, Congenital Rubella Syndrome, Congenital Syphilis, Diphtheria, Gastroenteritis in an institution or food handler, Hepatitis D and E, Hydatid Disease, Leprosy, Leptospirosis, Listeriosis, Lymphogranuloma venereum, Poliomyelitis, Typhoid, Typhus, and Viral Haemorrhagic Fever are all notifiable but had "0" notifications in this period.
- It is unclear why there is a marked increase in gonococcal notifications from 1997 to 1998.
- The high numbers seen in the above quarter of 1997 as compared to that of 1998 are similar to those seen in the same quarter in 1995 and show the yearly variations observed in the NT with the cause being uncertain.
- This is the third consecutive quarter to show a decrease in invasive pneumococcal disease.

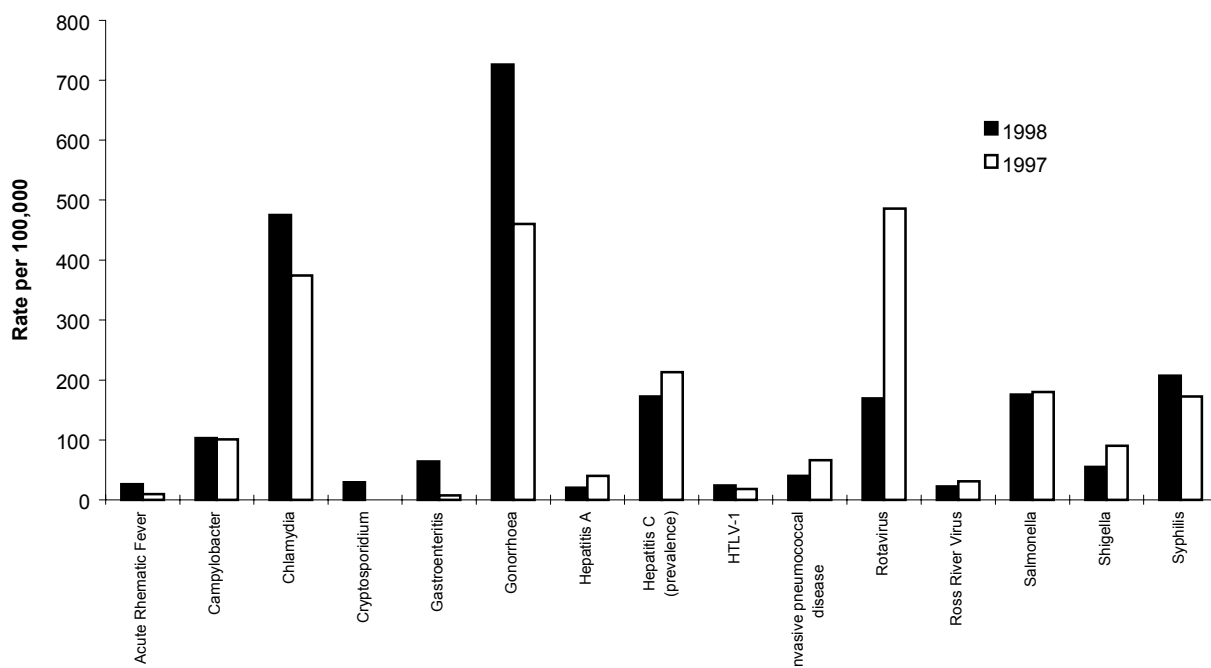
NOTIFIED CASES OF VACCINE PREVENTABLE DISEASES IN THE NT

BY REPORT DATE 1 JULY TO 30 SEPTEMBER 1998 AND 1997

DISEASES	TOTAL		No. cases among children aged 0-5 years	
	'98	'97	'98	'97
Congenital rubella syndrome	0	0	0	0
Diphtheria	0	0	0	0
<i>Haemophilus influenzae</i> type b	0	2	0	2
Hepatitis B	6	6	0	1
Measles	0	1	0	0
Mumps	2	4	0	3
Pertussis	9	5	1	3
Poliomyelitis, paralytic	0	0	0	0
Rubella	0	2	0	1
Tetanus	0	0	0	0

- Mumps is largely under-reported.

**NT WIDE NOTIFIABLE DISEASES
1 JULY TO 30 SEPTEMBER 1998 AND 1997**



Rates <10/100 000 not listed
NT 1996 mid year est resid. pop - 181 923 as supplied by ABS