Australian Bat Lyssavirus in the Northern Territory 2000 – 2002: an overview of exposure and treatment.

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Introduction

"Do not touch or try to rescue bats" is the main message being promoted to all residents and visitors to the Northern Territory (NT) by the Department of Health and Community Services. A television advertisement, newspaper and radio articles, and fact sheets have been designed in an attempt to discourage people from handling bats, and reduce the number of people inadvertently exposed to the potentially fatal Australian Bat Lyssavirus (ABL). Whilst it is a simple message, an alarming number of people continue to attempt to rescue bats. Many people present to health centres, hospitals or general practitioners for post exposure treatment but an unknown number of people do not seek treatment and remain potentially at risk.

The Virus

ABL is one of the seven genotypes in the Lyssavirus family of Rhabdoviruses. It is most similar to rabies virus genetically, serotypically, and in its presentation of symptoms in humans. It was first described in May 1996[1] when it was discovered in a flying fox in NSW. Two strains of ABL have subsequently been isolated from several species of insectivorous bats and flying foxes

found throughout Australia.[2] Surveillance over the past 8 years has recorded an infection prevalence throughout Australia of 6.0-9.4% in flying foxes submitted for testing.[2] The rate of ABL is highest in sick bats; however the virus has been detected in bats that do not display any signs of being unwell.[2] ABL has not been detected in any other wildlife.

Contents

The Injury

Bat bites and scratches can be fatal if proper preventative post-exposure treatment is not undertaken. In 1996 and 1998 there were 2 recorded cases of human ABL infection in Australia. Bats had bitten both people, neither received post-exposure treatment and both died. If bitten or scratched, the wound should be thoroughly washed with copious amounts of soap and running water as soon as possible and an antiseptic applied if available. The person should then present to a health facility for assessment and referral to CDC regarding the need for post-exposure vaccination. Suturing is not recommended.

Post-exposure Treatment

The treatment for bat bites and scratches is both painful and expensive. Due to the similarity between ABL and rabies virus, rabies immunoglobulin (RIG) and rabies vaccine are effective for post-exposure treatment. Non-immune people sustaining a bat inflicted injury require RIG to be administered as soon as possible after the injury at a dose of 20 IU / kg. (eg an 80kg patient requires 10.6mls of RIG). The aim of treatment is to infiltrate the nerve supply around the wound by injecting as much of the RIG into the wound site as possible. The remainder should be injected intramuscularly into sites between the bite and the brain. Five doses of rabies vaccine over a period of 1 month are also required to afford protection. The first vaccine should be administered at the same time as the RIG and then on days 3, 7, 14 and 28. Tetanus vaccine should also be offered if the patient is not fully vaccinated. The price of rabies immunoglobulin has increased dramatically over the last year. Together with the cost of 5 doses of vaccine, post-exposure treatment for an 80 kg person is $1320. This cost is borne by the Centre for Disease Control (CDC) public health program.

Pre-exposure Prophylaxis

Pre-exposure Prophylaxis (PEP) is recommended for people who have contact with bats. This includes wild care workers, bat handlers, vets, power and water workers who regularly remove bats from power lines, as well as expatriates who plan to live in rural parts of rabies endemic areas for more than 1 month. PEP requires the administration of 3 vaccines over a 1 month period (Day 1, 7 and 28). People who have completed PEP still require 2 post-exposure treatment vaccines if they are exposed to ABL through a bite or scratch, but no immunoglobulin is required. Table 1 displays the number of persons presenting to CDC Darwin for pre–exposure prophylaxis in the past 3 years. Other regional CDCs routinely offer this service. The client or their employer funds all vaccines administered in this program. Rabies vaccine costs $65 per dose.

Table 1. Number of people attending CDC Darwin for ABL PEP 2000-2002

Occupation	Number
Wildlife Park worker	23
Private bat handler	12
Vet/Vet nurse	6
Other	5
TOTAL	**46**

Injury Incidence

Darwin CDC maintains a database of reported bat related injuries in the NT. The majority of injuries in 2000-2002 were sustained during bat rescues; people attempting to free bats from barbed wire fences being the most common reported reason. Accidental injuries occurred frequently in people who handle bats through either the course of their work or volunteer duties. Several injuries resulted from bats becoming disorientated and flying into people's homes and yards and subsequently injuring the occupants (Table 2).

Children are more likely to be injured than adults; their natural inquisitiveness and desire to rescue an injured animal featuring strongly in the incident reports for 2000-2002. A large proportion of those people who sustain bat inflicted injuries are people who work with bats either professionally or as wild care workers

(Table 3). Not all of the people who sustained this type of injury had participated in a pre–exposure prophylaxis program.

Table 2. Cause of bat inflicted injuries in NT 2000-2002

Exposure type	Number	Percentage (%)
Rescue	20	57
Accidental	12	34
Unknown	3	9
TOTAL	**35**	**100%**

Table 3. Persons sustaining bat inflicted injuries in NT 2000-2002

Occupation	Number	Percentage (%)
Student	9	26
Home duties	5	14
Wildlife Park worker	5	14
Private bat handler	3	8
Labourer	3	8
Tourist	2	6
Power and Water worker	2	6
Vet / Vet nurse	1	4
Other	5	14
TOTAL	**35**	**100%**

Summary

ABL is present in many species of bats and flying foxes throughout Australia and has been isolated in bats sampled from Darwin[3]. Whilst there have been no reported cases of human ABL in the NT, nor in Australia since 1998, the ongoing number of people who sustain bat inflicted injuries either accidentally or while attempting a rescue is cause to continue public education on ABL and the risks it imposes. People who routinely handle bats should be encouraged to participate in pre-exposure prophylaxis programs in an attempt to provide them with some protection from ABL and reduce the need for expensive post exposure treatment. We should all discourage people from touching or handling bats.

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Fact Sheet

Australian Bat Lyssavirus (ABL)

What is ABL?

Australian bat lyssavirus (ABL) has been found in several species of flying foxes and insectivorous bats. It was first identified in Australia in 1996. Previously 6 types of lyssavirus were recognised throughout the world, 5 of which are found in bats. The most well known lyssavirus is rabies, which is closely related to ABL. There have been 2 deaths from ABL in Australia.

How is it spread?

ABL is usually transmitted to humans via bites or scratches that provide direct access of the virus in bat saliva to breaks in the skin and exposed tissue and through mucous membranes (eyes, nose and mouth).

The virus cannot survive more than a few hours outside the bat. ABL is not spread by bat urine, faeces or blood. Fruit soiled with bat saliva, urine or faeces is not a risk but should be washed before eating.

There is no risk of ABL infection from eating flying foxes that have been thoroughly cooked.

Who is at risk?

Anyone who handles bats is potentially at risk through scratches, bites and direct saliva contact to the mucous membranes of your mouth, eyes or nose.

Handling bats

Do not touch or try to rescue bats. If you find a sick or injured bat, contact your nearest wildlife rescue service for assistance.

Anyone who regularly handles or cares for bats (members of bat care groups, wildlife officers, vets etc) should be vaccinated. Routine vaccination is not recommended for other people.

If involved in handling bats you should:

- ensure you are vaccinated before handling bats
- cover any unhealed cuts or wounds on your skin
- wear puncture proof gloves and long sleeved clothing of thick material and protective glasses
- avoid handling any new bat in your care for 24 hours and if it displays signs of illness take it to the vet

- pick up sick bats by wrapping them in thick cloth to reduce the chance of being bitten or scratched
- take soap and water when rescuing bats so you can thoroughly clean any bites or scratches as soon as possible

If you are scratched or bitten:

- Wash the wound thoroughly for a minimum of 5 minutes with soap under running water as soon as possible. Proper cleaning of the wound is the most effective way to reduce transmission of the virus. Apply an antiseptic solution after washing if possible (i.e. povidone-iodine).
- If you get bat saliva in your mouth, eyes or nose you should flush the area with water.
- Cover the wound and seek medical attention *immediately*. Vaccination is still protective against ABL if given promptly.
- Even if already vaccinated, medical attention should be sought as soon as possible for further (treatment) vaccine.

Symptoms of ABL in a sick bat

- muscular weakness such as partial wing or hind limb paralysis;
- difficulty or inability to fly;
- unusually docile or unusually aggressive;
- depressed and unresponsive.
- Any bats with these symptoms should be reported to your nearest wildlife rescue service.

NB Some infected bats may not exhibit any unusual behaviour.

Disposal of dead bats

If the bat had any of the above symptoms your nearest wildlife rescue service should be contacted for appropriate disposal of the body. Other bats may be disposed of by placing them in a bag and burying.

Although ABL is not thought to live long in a dead bat, precautions should be taken to avoid being scratched when disposing of the body.

For more information contact your nearest Centre for Disease Control:

	Darwin	89228044
Katherine 89739040	Nhulunbuy	89870359
Alice Springs 89516907	Tennant Ck	89624259

Leishmaniasis: an overview in the context of an emerging pathogen in Top End wildlife.

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Introduction

In 2000, investigation into an ulcerative disease in the tails of captive Red Kangaroos (*Macropus rufus*) resident in the Top End led to the identification of a probable *Leishmania* species as the cause.[1] The discovery led to some media interest[2,3] and the concern of the possibility of spread to humans. This article is a very brief overview of human leishmaniasis with the aim of informing health care professionals and the public about this disease and examining the risks to human health in the NT.

The organism and its life cycle

Leishmania is a genus of a protozoan which is characterised by two life stages; the intracellular amastigote stage which occurs in mammals and the extracellular promastigote stage which occurs in the phlebotomine sandfly vector. There are many species of *Leishmania* which cause disease in humans (14 main ones often grouped into sub-genera) but they all appear similar on light microscopy and need further enzyme testing or nucleic acid amplification for further species differentiation.[4]

The sandfly is infected by ingesting amastigotes with a blood meal. These are then liberated in the sandfly's stomach and develop into motile promastigotes, which migrate to the mouthparts and are injected into the mammalian host at the next meal. They are then taken up in the host's macrophages where they revert to amastigotes and multiply.

Although leishmaniasis is usually transmitted to humans from a mammalian reservoir via a sandfly (zoonotic spread), human-sandfly-human (anthroponotic) spread is recognised in some areas as the major mode of transmission (particularly the Indian sub-continent). Transmission through blood transfusion is well documented; other routes such as sexual spread, inoculation, congenital transmission and direct spread are possible but are either extremely rare or unreported.[4]

The vector

The only recognised vectors for leishmaniasis are the so-called "true" sandflies in the sub family *Phlebotominae* of the family *Psychodidae*[5]. These are often referred to as phlebotomine sandflies. The 2 important vector genera are *Phlebotomus* in the Old World and *Lutzomyia* in the New World. These genera are not present in Australia, although *Phlebotomus* may extend through Southeast Asia to Timor (personal communication, Alan Dyce).

Figure 1. *Phlebotomus − a female "Sand Fly"*
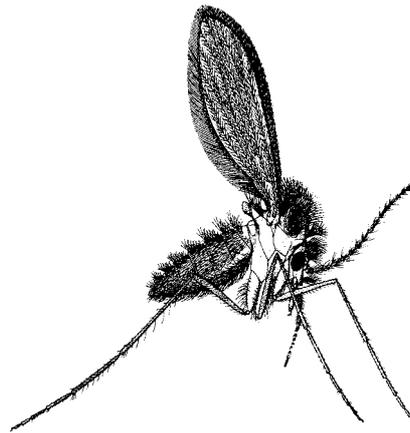
Insects of Medical Importance
British Museum 1956

Phlebotomine sandflies (Figure 1) can transmit viruses (sandfly fever and others), bacteria (*Bartonella bacilliformis* which causes Oroya Fever in South America) and protozoa (*Leishmania*).[4] They are small flies, 1.5-3.5 mm in length and look like very small, hairy mosquitoes. Unlike mosquitoes their wings are held in a V above the body rather than flat across the back.[4] They are about a quarter the size of mosquitoes and slightly larger than the so-called sandflies (biting midges) which are prevalent in the Top-End. Phlebotomine sandflies can inhabit a large range of habitats from sea level deserts to tropical mountain ranges, although

Table 1. Species, distribution, vector and mamalian hosts of visceral leishmaniasis

Species	Distribution	Vector	Mammalian hosts
L. donovani	India	*Phlebotomus*	Humans
	East Africa (Kenya)		
L. infantum	Mediterranean	*Phlebotmus*	Dogs
	North Africa		Foxes
	Middle East		Jackals
	Central Asia		Rodents
	China		
L. chagasi	Central America	*Lutzomyia*	Humans and dogs
	South America		

Note: This table is an approximate guide only.

each species has fairly specific ecological requirements. They breed in a variety of habitats involving darkness, humidity and organic matter on which the larvae feed. Breeding places include under leaves in rain forest floor litter, hollow trees, tree buttresses, rocky outcrops, and animal burrows. Some species have adapted to the peridomestic setting and breed in cracks in floors and walls, and stone fences[6].

The illness

There are two major forms of leishmaniasis; visceral and cutaneous. Even though there is a close association between the species of *Leishmania* and the clinical disease, the distinction is somewhat blurred with some species recognised as causing visceral leishmaniasis also causing the cutaneous form in exceptional circumstances or in the presence of severe immunosuppression.[4]

Visceral leishmaniasis (also known as kala-azar)

This is a chronic disease characterised by infection of the visceral organs such as the spleen and liver and giving rise to fever, hepatosplenomegaly, lymphadenopathy, pancytopenia and progressive emaciation and weakness.[4,7] The incubation period is generally some months, but can be as long as 10 years.[4] The disease has an effect on both humoral and cell mediated immunity such that secondary bacterial infection is the common cause of death.[8] The prognosis following infection depends on the host's cell mediated immunity, but once infection is established in the spleen, mortality without treatment is almost invariable.

Visceral leishmaniasis is widely distributed around the Mediterranean basin, tropical Africa, Central and Eastern Asia and parts of South

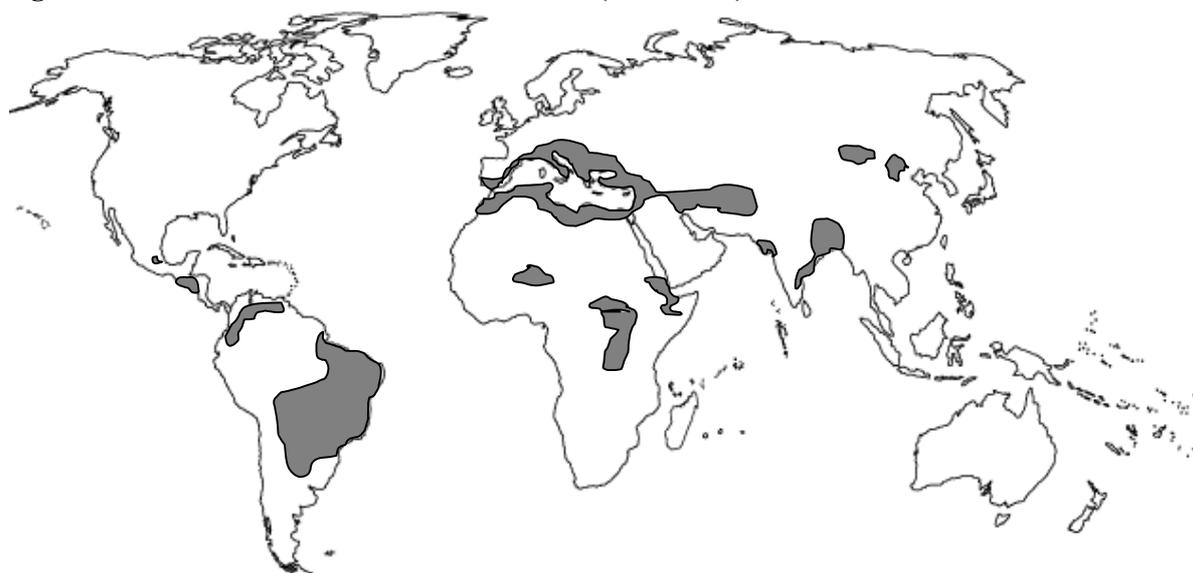**Figure 2. Distribution of visceral leishmaniasis (from Bell[8])**

Figure 3. Distribution of cutaneous leishmaniasis (from Bell[8])

America[8] (see Figure 2). The recognised causal species are *L. donovani, L. infantum* and *L. chagasi*. Distribution, vectors and mammalian hosts are given in summary in Table 1.

Cutaneous leishmaniasis

This is a chronic ulcerative and deforming disease of the skin and mucous membranes. There is a wide clinical spectrum of disease which is determined among other things by the species of *Leishmania* and the host's cell mediated immunity. Some forms are mild and self-healing, others can remain indolent and recrudesce while others can be diffuse and disseminate.[8] The typical ulcer may be single or multiple and may be on any part of the body, but usually the extremities or the face. The edge is raised and infiltrated but not undermined. Most are painless and lymph nodes can be involved.[8] Depending on the species they can be dry (*L. tropica*) or exudative (*L. major*), although the clinical picture does not always match the species. If cell mediated immunity is poor lesions can be diffuse.[8] One form of the disease (mucocutaneous leishmaniasis) occurs at the mucocutaneous junction, particularly the nose, and after many years can involve ulceration and destruction of the cartilaginous septum and surrounding tissues resulting in severe disfigurement.

The incubation period is at least a week and can be up to many months.[7]

Cutaneous leishmaniasis is generally divided into Old World and New World forms that are determined by the location and causative species. In the Old World the disease is distributed around the Mediterranean, in north, east and Sub-Saharan Africa, the Middle East, Central Asia and parts of India. In the New World it is seen in south Texas, Central America and in all countries in South America except Chile and Uruguay (see Figure 3).

The skin lesions have local common names such as oriental sore, Baghdad boil, pian bois, chiclero's ulcer, uta and espundia (mucocutaneous leishmaniasis). A summary is given in Table 2.

Diagnosis

Leishmania are easy to detect by light microscopy but the correct specimen is required. For visceral leishmaniasis the most sensitive test is microscopy of splenic biopsy, but bone marrow or lymph node can also be used.[9] The organism can also be cultured *in vitro* or in hamsters.[4] There are several serological tests available, perhaps the most innovative is an immuno-chromatographic dipstick test for a specific antigen of *L. infantum* used in India.[9] There is a test for cell-mediated immunity, the Leishmanin test, which involves injection of antigen intradermally and is analogous to the Mantoux test for tuberculosis. It has no role in the diagnosis but can be used at the population level to map disease patterns and outbreaks.

Table 2. Species, distribution, vector, reservoir and clinical picture for leishmania species causing cutaneous disease. (from Bell[8])

Species	Distribution	Vector	Mammalian reservoirs	Clinical picture
L. tropica	India Pakistan Afghanistan Mediterranean	Phlebotomus	Humans (Anthroponotic cutaneous leishmaniasis)	Oriental sore Urban CL, Recidivans CL
L. major	North Africa Middle East Central Asia	*Phlebotomus*	Various desert rodents such as gerbils and rats	Oriental sore Rural CL
L. aethiopica	Ethiopia Kenya	*Phlebotomus*	Hyraxes (small rodents)	Oriental sore Disseminated CL
L. braziliensis complex	Central and South America	*Lutzomyia*	Forest rodents Sloths	Mucocutaneous CL, Pan Bois South American CL
L. mexicana complex	Central America, Brazil, Venezuela	*Lutzomyia*	Forest rodents	Chiclero's ulcer South American CL
L. peruviana	Peruvian Andes, Argentina	*Lutzomyia*	?dog	Uta

Note: This table is an approximate guide only.

Traditionally, diagnosis of cutaneous leishmaniasis is done through either a "touch smear" (from a biopsy) or a slit-skin smear as in the diagnosis of leprosy. This is best taken from the edge of the lesion. Culture can also be used but serology is generally not reliable. None of these tests have a high sensitivity so a combination is often recommended in areas of high prevalence.[10] There is some promising progress made with diagnosis by polymerase chain reaction (PCR) technology.[11]

Amastigotes are usually seen within tissue macrophages (2-3μm across) and are also known as Leishman-Donovan bodies. They have characteristic large nuclei with an extra dark kinetoplast in the cytoplasm.

Epidemiology

It is estimated that globally there are about half a million new cases of visceral leishmaniasis and 1.5 million new cases of cutaneous leishmaniasis each year.[10,12] It affects all ages. The most eastern regions in distribution maps for leishmaniasis are; Bangladesh, eastern India and northern China for the visceral form and the central Asian republics for the cutaneous form. However, there were 46 cases reported in East Timor following the conflict there in 1999.[13] There have been no other cases reported in the literature in South East Asia or the Pacific, although potential vectors are present in some countries in these regions.[4] There have been reports of travellers returning to Australia with leishmaniasis, the most recent report being from Darwin in 1998.[14]

Leishmaniasis is an opportunistic infection acquired by people living with AIDS. The AIDS pandemic in regions of high incidence of leishmaniasis together with increased migration to the rural-urban fringe has led to an increase in many countries. Co-infection with HIV and *Leishmania* is emerging as an important new disease complex with clinical, epidemiological and economic implications.[15] The ongoing conflict in Southern Sudan, in combination with the increasing HIV prevalence in that region has led to an epidemic of visceral leishmaniasis in that country in recent years.[16] In Darwin, we have had an increase in the number of refugees arriving from Sudan and kala-azar should be considered in the differential diagnosis of unexplained fever with hepato-splenomegaly.

Treatment

The mainstay of treatment for both visceral and cutaneous leishmaniasis has been the pentavalent antimonial drugs such as sodium antimony gluconate and meglumine antimonate. These

need to be given parentally and are associated with numerous and serious side-effects and with the development of resistance. Newer drugs such as liposomal amphotericin B and oral miltefosine (a cytotoxic agent) have been used in visceral leishmaniasis with excellent results.[9,17,18]

Many cutaneous leishmaniasis lesions heal without treatment, however size, position or cosmesis may necessitate intervention. Physical removal such as curettage or cryotherapy has been used in the past together with the antimonials. Miltefosine has been shown to be effective and for some species the antifungal azoles (in particular fluconazole) have shown promising results.[9,17] Local treatments such as intra-lesional antimonials, paromomycin ointment and Imiqimod (a wart treatment) have also been shown to be effective.[10]

Control

The essential step in the control of leishmaniasis is to separate humans from sandflies or the animal reservoir. In the past, malaria control programs have had a large impact on the transmission of *Leishmania* due to the impact on sandfly populations.[4] Successful sandfly control has been achieved through household spraying (with pyrethroid sprays) and with the use of bed nets either treated with pyrethroids or untreated.[9] Control of the mammalian reservoir by environmental change,[4] or culling or treating local dogs[9] has been successful in some settings.

Leishmaniasis has always been considered a good candidate for vaccine development. Milestones for progress here have been; the recognition of the role of CD8 cells in the immune response,[9] the *Leishmania* genome project,[19] the funding boost from the Bill and Melinda Gates foundation, and WHO support for a program to resurrect "leishmanisation", whereby infants are inoculated with *L. major* to develop a sore and thereby prevent cutaneous disease later in life.[9]

Risks of transmission to humans in the NT

In the Northern Territory, the risk of spread from kangaroos to the human population is indeed very small. The known sandfly vectors for transmission to humans do not exist in Australia and there are no phlebotomine species that are pests of humans in Australia. Phebotomine attack on humans in the Australasian region is very rare overall and transmission of diseases of humans or domesticated animals in the region has been regarded as most unlikely.[20]

However there could be a local, but previously undetected, *Leishmania* in Australia being transmitted to local hosts that do not exhibit symptoms of disease. There are a number of phlebotomine sandflies species in Australia that could be local vectors. This includes the genera *Australophlebotomus, Sergentomyia and Idiophlebotomus*. This last genus is known from a single species which bites bats and has been found in caves.

There are 8 species of *Australophlebotomus*, of which one, *Australophlebotomus brevifiloides* has been recorded once as biting a human.[20] The 2 most abundant species dominate in the south of Australia and favour moderate or lower rainfall environments. *Australophlebotomus brevifiloides* normally bites small animals, birds and reptiles. It has been recorded in the NT, mostly from the Barkly and south of (and including) Katherine.

Sergentomyia has the most species (24), with *S. queenslandi* and *S. englishae* the 2 most abundant and widespread species. *S. queenslandi* is the most frequently collected phlebotomine species from Australia, with the biggest densities found in the northern half of Australia from low rainfall areas, often associated with rocky outcrops.[20] This species has been found in the Darwin region including Beatrice Hill, Howard Springs and Mudginberri. Adults have been collected from burrows of small reptiles and it is reported to feed readily on lizards.[20] However human attack by *S. queenslandi* is unknown in Australia.

It is interesting to postulate why this disease has appeared only in the Red Kangaroo and how it was transmitted to these animals, at least one of which was geographically isolated from the others. The Red Kangaroo is an exotic species in the Top-End and, given that it probably exists under some ecological stress, may lack immunity to local pathogens, leaving it susceptible to disease from a previously undetected local *Leishmania*. If this is the case, and presuming that the local parasite has been around since at least European settlement of the

NT, it appears not to cause illness in its natural local host, nor indeed spread to humans or domestic animals.

Studies are being planned to ascertain what arthropods are biting the kangaroos and whether there are local animal reservoirs of *Leishmania*. The evidence that reptiles are preferred hosts for some of the Australian phlebotomines and the collection of adult sandflies from reptile burrows indicates that reptiles, and particularly goannas and their burrows, are good candidates for the investigation of a local *Leishmania* in the Top End.

Summary

Leishmaniasis is an exotic protozoal disease which is transmitted between humans or between animals and humans by phlebotomine sandflies. The recent discovery of a possible *Leishmania* species in captive kangaroos in the NT has raised the possibility of an emerging pathogen which could impact on human health. Even though a theoretical risk exists, the lack of human biting phlebotomine sandflies in Australia, and the lack of any evidence of spread between other marsupials or mammals, suggests that the risk to human health in the NT is minimal. Nevertheless, clinicians should keep the theoretical risk in mind when assessing non-healing ulcers. It is also worthwhile being aware of the possibility in the context of refugee health, given that both internecine conflict and the HIV pandemic have led to an increase in leishmaniasis in other parts of the world.

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Chronic suppurative otitis media (CSOM)
Ear Toilet has not gone far enough!
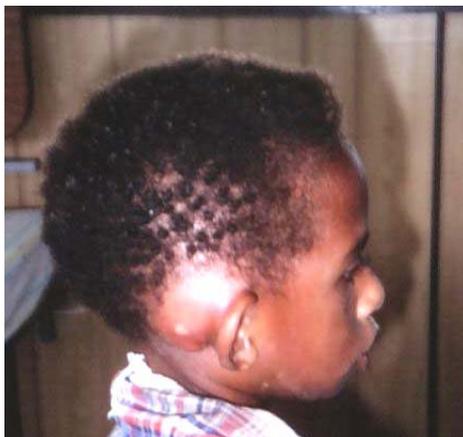*Keith Edwards, Community Paediatrician, CDC Darwin.*

Background

Suppurative Otitis Media is an inflammation of the middle ear cavity which leads to pus formation, bursting of the ear drum and pus discharging from the ear canal. In urban children, this condition is usually self limiting and healing of the perforation occurs after a few days. In many Aboriginal children living in remote communities in the tropical north of Australia, healing does not occur and the ear continues to discharge, intermittently or continuously, for many months or years causing deafness and language development delays as well as learning difficulties in school. Children who cannot hear well are often unwilling to attend school. Rates of CSOM vary from 5% of children under five years in some remote communities to more than 50% in others. These rates have not changed significantly over the past 10 years despite a standard management protocol of ear toilet, topical antibiotic / anti-inflammatory drops (Sofradex) and oral antibiotics as recommended in the CARPA Standard Treatment Manual.[1]

Serious Complications

In some children, the infection spreads locally to infect the bone and cause a mastoid abscess (Fig. 1), often leading to long term osteomyelitis of the bony structures around the middle ear making treatment difficult and requiring surgical intervention. Rarely, infection can spread inwardly from the middle ear leading to bacterial meningitis and serious neurological deficits or death. Uncommonly, the infection may involve branches of the facial nerve and lead to paralysis of that side of the face (Fig. 2). Other rare complications are labrynthitis and brain abscess.[2] Pneumonia may also result from aspiration of the bacteria-laden pus from the inner end of the eustachian tube whilst the child is sleeping.

Causation

Colonisation of the naso-pharynx with pathogenic bacteria early in life (infancy) is thought to be one of the important reasons why Aboriginal children are afflicted with this chronic infection. These bacteria reach the middle ear from the naso-pharynx through the eustacian tube. The inflammation is primarily sited in the middle ear cavity which is midway between the naso-pharynx and the external ear and that is where the pus is formed and is trapped until the drum is pushed outwards and bursts under the pressure. Pus is then able to drain out along the external auditory canal and is visible dripping from the external ear (Fig. 3).

Figure 1. Mastoid abscess

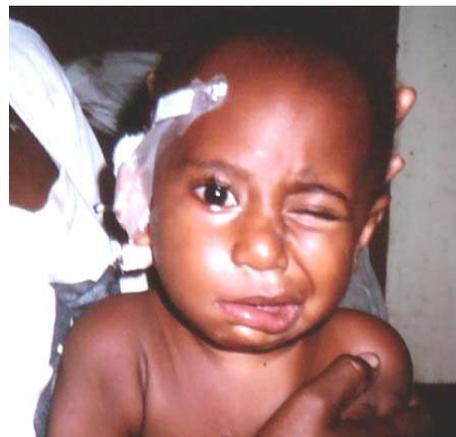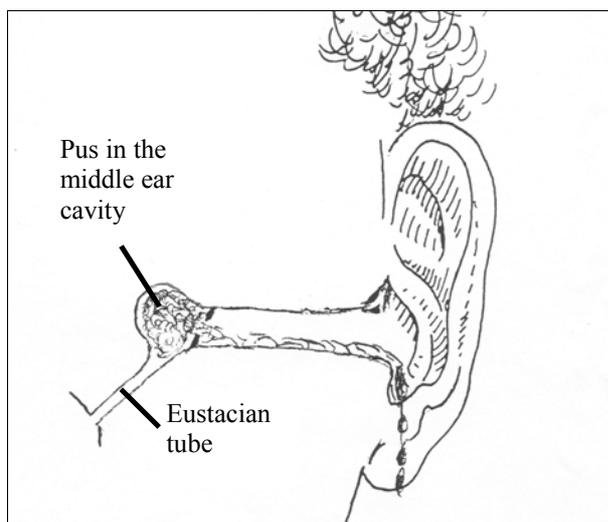
Figure 2. Facial nerve paralysis

Figure 3. Chronic middle ear infection

Pus in the middle ear cavity
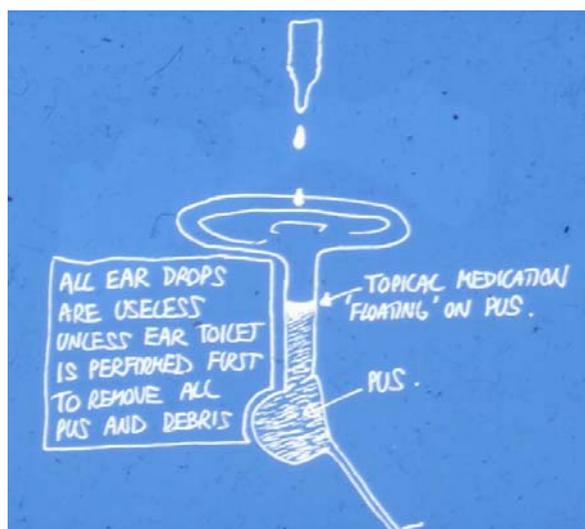
Eustacian tube

Benefits of effective Ear Toilet

If ear toilet is performed well and often enough, then many of the causes for treatment failure are addressed[3,4]:

- Mucopus is removed from the middle ear
- Resistant bacteria 'hiding' in the pus are removed
- Moisture is reduced by tissue absorption and results in drying of the middle ear, making it less suitable for bacterial survival.
- Ear drops can reach the inflamed surfaces of the middle ear.
- Hearing is improved as the sound muffling effect of the pus in the middle ear is removed.

Possible reasons for treatment failure

1. The pus in the middle ear is mucoid in nature (mucopus) and it is this characteristic which reduces the effectiveness of topical medication which cannot easily reach the inflamed surfaces or the bacteria within the pus (Fig. 4).

2. The mucopus in the middle ear still contains live bacteria which are "insulated" from systemic as well as topical antibiotics by the pus.

3. Over time, the repeated use of the same antibiotic preparations both topically and systemically selects out bacteria which are resistant and these bacteria multiply and continue to cause local inflammation.

4. The moisture and temperature in the middle ear cavity are conducive to bacterial growth and water-based ear drops, e.g., Sofradex, add to the level of moisture in the middle ear cavity.

5. Failure to remove the pus from the middle ear cavity with tissue spears (ineffective Ear Toilet). This failure occurs for two main reasons:

   i. the spear does not reach far enough into the ear canal because:

      - the tissue spear is made "too fat" and gets stuck in the outer canal

      - the carer is afraid to push the tissue spear far enough into the ear canal

   ii. ear toilet is performed infrequently so that pus re-accumulates

Figure 4. Ear drops prevented from reaching middle ear by pus

ALL EAR DROPS ARE USELESS UNLESS EAR TOILET IS PERFORMED FIRST TO REMOVE ALL PUS AND DEBRIS

TOPICAL MEDICATION 'FLOATING' ON PUS.

PUS.

PERFORMING EFFECTIVE EAR TOILET
(1) MAKING THE BEST TISSUE SPEAR

STEP 1.	Use a sheet of absorbent toilet paper. You can also use other tissue paper instead but you should separate the two layers and use one only to make a spear otherwise it will be "too fat" and not reach far enough into the canal.
STEP 2.	Hold the sheet at one of the corners with your best hand (right) whilst holding the rest of the tissue in your other hand (left) --- Start twisting the corner with the finger and thumb of your best hand (right).
STEP 3.	Keep twisting the tissue and you can also help to twist using the finger and thumb of your other hand (left) whilst continuing to hold onto the other end of the tissue with your 3rd, 4th and 5th fingers of your other hand (left).
STEP 4.	Keep twisting the tissue from the corner until it is tightly wound. It should be "thinner" at the "corner end" and "fatter" at the other end.
	This tissue has been twisted well.
STEP 5.	The tip is always too floppy and thin, so about 1cm should be broken off and discarded as shown. This provides a "fluffy" absorptive surface at the tip to maximise pus removal from the middle ear cavity.
STEP 6.	Similarly, the "back" of the tissue is "too fat" and should be broken off as shown as it makes insertion more difficult.
	This is a well twisted spear which is ready to be inserted into the child's ear.
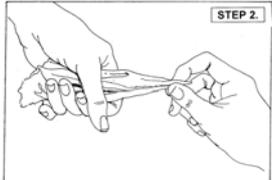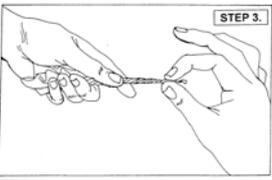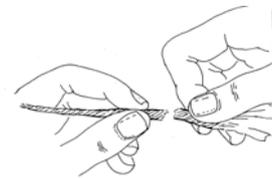
PERFORMING EFFECTIVE EAR TOILET
(2) INSERTING THE TISSUE SPEAR INTO THE EAR

	It is very important to hold a young child steady with one hand on the head, keeping it firm against the holder's chest whilst the other hand wraps around the arms, steadying them. This is so that they do not move whilst the tissue is inserted. At first, 2 people are needed, one to hold the child and the other to insert the tissue spear.
	Later, when the child is used to the cleaning, they will cooperate and one person can perform the ear toilet on their own. It is important to be very gentle so as not to cause pain or a sudden loud noise in the ear from the tissue movement.
	Some small children are happy to lie on their carer's lap whilst their ear is gently cleaned. Gently pull the outer ear backward and outward to straighten the ear canal. Push the tissue in with a slight twist in the direction that the spear was twisted so that it does not come untwisted. Stop pushing the tissue when the child blinks, coughs or cries. The tissue should be at least one inch into the ear canal, measured from the tragus (front of the ear opening).
	Gentle removal of the tissue after 3-5 minutes often results in a string of mucopus being removed on the end of the tissue.
	Initially, the ear should be cleaned 3 times or until the inserted tissue comes out dry. This cleaning should be repeated 3 to 4 times a day till improved, then twice daily for a week, then once a day for a month and can then be stopped if the ear remains dry.

PERFORMING EFFECTIVE EAR TOILET
(3) USING EAR DROPS

Ear drops* should be inserted after the ear has been effectively cleaned with toilet tissue spears. 2 drops are inserted with the child lying flat. Keep the child lying on his or her side for a minute. Pressing gently on the ear flap (tragus) can help the drops reach the middle ear through the perforation

*Current guidelines recommend Sofradex ear drops which are water based. Alcohol based ear drops also kill bacteria and are most effective at drying the ear when the alcohol evaporates. Consideration should be given to using alcohol based drops e.g. Aquaear in cases which do not respond to standard treatment.

PERFORMING EFFECTIVE EAR TOILET
(4) IMPORTANT MESSAGES TO GIVE
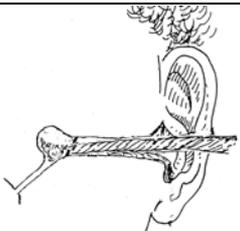
"The tissue needs to go inside the ear at least 1 inch and must touch the ear drum. If the tissue is too fat, it will not go in far enough".

"Do not be afraid, the tissue is soft and will not harm the child's ear or head inside."

"When the ear starts to get dry you will see that the pus is on the end of the tissue spear only, showing that the sore place is at least one inch inside".

"Ear cleaning is important to help your child hear the teacher when he or she goes to school."

"Used tissue spears have bad germs on them and should be disposed of carefully in a plastic bag then burned on the fire at the end of the day or thrown out in the garbage".

How to improve ear toilet technique in remote communities

Obtain a copy of the powerpoint presentation on CD of "How to perform effective ear toilet" from the author (e-mail keith.edwards@nt.gov.au). Use the presentation to teach yourself and your colleagues how to perform effective ear toilet. Also use the presentation to improve ear toilet technique for parents and family members. Community members can be taught in groups in the women's centre. Child care centre staff can be taught to perform ear toilet on children with discharging ears in their care. Teachers and children can be taught in schools. The more often discharging ears are cleaned, the less likely they will become chronic and those affected will have better hearing and better learning opportunities.

References

1. Central Australian Rural Practitioners Association Alice Springs. CARPA Standard Treatment Manual. 4th Edition. CARPA. 2003.

2. Kangsanarak J, Fooanant S, Ruckphaopunt K, Navacharoen N, Teotrakul S. Extracranial and intracranial complications of suppurative otitis media. Report of 102 cases. *J Laryngol Otol* 1993; 107(11): 999

3. Acuin J, Smith A, Mackenzie I. Interventions for chronic suppurative otitis media. Cochrane Database Syst Rev. 2000; (2): CD000473.

4. Edwards K. Chronic Suppurative Otitis Media Study, Port Moresby PNG 1986 presented to the Paediatric Society Annual Symposium (unpublished)

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Clinic 34 - on the move!
Peter Knibbs, Clinical Nurse Consultant, CDC Darwin

After 10 years of security in the folds of the Disease Control mothership, Clinic 34 is venturing into unchartered territory and moving to the city. From early 2004, it will be located in the rooms previously occupied by the Darwin Community Care Centre in Mitchell St (on the ground floor of Health House).

Clients attending Clinic 34 have long told us of how difficult the clinic is to find and people attending a sexual health clinic are often reluctant to ask for directions. We have been concerned that clients who have made appointments, but don't arrive, may have had difficulty finding us and were too embarrassed to ask for directions. In addition the hospital campus is difficult to access using public transport and given these issues it takes a lot of motivation for the asymptomatic person to attend for sexual health screening.

The new location will be much easier to find and reach by public transport. By having the clinic in Mitchell St, which is recognised as the social hub of the city, we will be accessible to the many young people who are identified as one of our target groups. It is also envisaged that the clinic will provide more flexible hours allowing people working in the city to attend after work or during their lunch breaks.

The move will only involve clinical staff and other AIDS/STD program staff remain in Building 4. Telephone numbers will be changing but the well known 8922 8007 will be redirected to our new number during the transitional period.

All in the AIDS/STD program look forward to a bigger, busier and better service for our clients. Come and check out the cafe latte, Clinic 34 team in the Mitchell Street precinct!

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Gastroenteritis outbreak due to *Salmonella*

Karen Dempsey, OzFoodNet Enteric Disease Epidemiologist, and
Leah Campbell, Environmental Health Officer, CDC Darwin.

Background

The Darwin Centre for Disease Control (CDC) was notified on Monday 10th November by a member of the public that she and several other people had become ill with apparent food poisoning 24 to 48 hours after eating a meal at a catered function.

Initial investigation

That afternoon the OzFoodNet epidemiologist informed the environmental health branch of the outbreak and initiated an investigation. Important preliminary information, including the names of the caterers, the menu and the names of the 21 people who had eaten at the function were collected. A local restaurant had provided the main meal including an entrée of spicy quail followed by a variety of Asian dishes. A cake shop, not related to the restaurant, provided the dessert.

During the ensuing 3 to 4 days the majority of guests were interviewed using standard gastroenteritis outbreak hypothesis-generating questionnaires. Information was obtained relating to food items eaten at the function together with onset and duration of any symptoms. Convalescent stool samples were collected from 3 cases and an Environmental Health Officer (EHO) conducted an inspection of the restaurant and the cake shop on Tuesday 12th November.

Initial results

In total 17 people (8 females and 9 males) out of 21 people who attended the function were interviewed. The median age was 57 years and the attack rate was 59% (10 out of 17 people). The median incubation period was 34 hours with a median duration of illness of 41 hours. The most common symptom was diarrhoea (90%) followed by moderate to severe abdominal pain (60%). Vomiting, fever and headache were less common complaints with only 40% of cases experiencing any of these symptoms. The

hypotheses-generating interviews were entered into a spreadsheet and analysis was performed using EpiInfo 2002. The risk ratios for the prawns, squid and wonton soup all exceeded 1 but none were statistically significant (see Table 1). The risk ratio for the quail was undefined because all 17 interviewees had eaten it.

The food handling practices and hygiene of the cake shop and staff were found to be satisfactory; however, the restaurant was found to be in a poor level of cleanliness and staff were noted to be using inappropriate hygiene and food safety practices. The following day, under Section 11 of the *Food Act*, a Notice was served on the proprietor of the business to ensure that the premises were in a clean and sanitary condition within twenty-four hours. Furthermore, the manager was advised that if he did not comply with the Notice, the restaurant would be required to cease trading until all requirements had been met. Re-inspection 24 hours later revealed good compliance with the Notice.

All 3 convalescent stool samples were negative for bacterial and viral pathogens including norovirus.

Follow-up investigation

During the re-inspection, further discussion with the manager revealed that preparation of the spicy quail meat involved excessive handling including thawing, stuffing, marinating, and refreezing. On the day of cooking quantities of the frozen processed quail (often large) were taken out of the freezer, defrosted in the microwave and deep-fried for 10 to 15 minutes. This, together with a clinical picture suggestive of a bacterial cause, prompted the sampling of raw quail meat, both processed and unprocessed for microbiological analysis at a local laboratory.

Follow-up results

Results of the food testing revealed that the raw unprocessed quail was negative for *Salmonella*,

Table 1. Attack rate of food items eaten at function, their risk ratio and confidence interval

Exposure	Attack rate	Risk ratio	95% Confidence Interval
Prawns	67%	1.67	0.53–5.24
Spicy quail	59%	Undefined	Undefined
Squid	62%	1.23	0.42–3.59
Wonton soup	60%	1.20	0.28–5.10
Spring roll	58%	0.97	0.41–2.30
Fried rice	57%	0.86	0.34–2.15
Dim sim	50%	0.75	0.33–1.72
Pork	50%	0.70	0.53–5.24
Chicken	50%	0.72	0.34–1.53
Steamed rice	54%	0.72	0.34–1.53
Beef	50%	0.50	0.30–0.84

Notes: 1 Attack rate is the proportion of people who ate each food item who became ill.
2 Risk ratio is the ratio of the risk of illness in people who ate each food item to the risk of illness in people who did not eat the food item.

yeast and moulds. In contrast, the raw processed quail was found to contain high levels of yeasts and moulds and coliforms plus unacceptable levels of *Salmonella.* The stuffing was also found to have a high level of yeasts and mould present.

Discussion

The outbreak was reported to CDC more than a week after onset of illness resulting in a substantial delay before case investigation was completed. Although the guests were no longer symptomatic it was possible that a pathogen could still be identified from stool specimens, particularly if the causative agent was *Salmonella*, as the median duration of excretion of *Salmonella* may be as long as 5 weeks after onset of acute salmonellosis. On this basis we decided to request samples from the 2 people whose duration of illness was the longest. Despite this neither were positive for any enteric pathogens.

The epidemiological analysis was unenlightening even though the risk ratios for certain foods exceeded one. For these foods the risk of becoming ill from consuming them was greater than the risk of becoming ill if you had not consumed them. However, as there were few cases among those who ate these foods, the results were not statistically significant. In addition a risk ratio for the consumption of quail could not be obtained because all guests ate quail. Consequently epidemiological analysis was unable to implicate any of the food items.

Samples of the raw quail, both processed and unprocessed were sent to a local laboratory compliant with Food Standards Australia. The laboratory microbiologist conducted 6-point testing including viable bacterial count, total coliforms, *Escherichia coli*, *Staphylococcus aureus*, *Salmonella* and *Clostridium perfringens,* and isolated *Salmonella* in the raw processed quail meat. Serotyping of the *Salmonella* was not carried out on site as this process involved further testing in a southern state laboratory and a comparator pathogen had not been isolated from stool.

The presence of *Salmonella* in a sample of raw processed quail strongly implicated the spicy quail as the probable source of contamination in this outbreak. The absence of *Salmonella* in the unprocessed sample indicated the quail was being contaminated during the marinating and stuffing process. Furthermore, the presence of yeasts and moulds indicated that the stuffing was being kept for prolonged periods after preparation enabling the amount of yeasts and moulds present to proliferate.

Conclusion

This outbreak highlights the need for vigilance in relation to food processing. Smaller batches of food condiments such as stuffing and marinade should be prepared on an "as needs" basis with any leftovers being discarded after use. A contributing factor in this outbreak was that large quantities of frozen processed quail were thawed in the microwave prior to cooking, which may have led to an inadequate thawing process and subsequent inadequate cooking. All poultry therefore should be defrosted in a cool room or refrigerator and cooked thoroughly before being served.

Reference

Jay LS, Dovas D, Dundas M, Frankish E, Lightfoot D. *Salmonella*. In: Hocking AD, ed. Australian Institute of Food Science

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Quarterly Notifiable Disease surveillance

In the last edition of the *Bulletin* we introduced a new system for illustrating and analysing notifiable disease data. We have done this as a means of better understanding the variations that occur from year to year. A table is presented comparing the numbers in each district of the quarter this year compared to last year. We also compare the number this year to the mean of the same quarter for the previous 4 years. If this year's number differs from that mean by more than 2 standard deviations, we consider it to be potentially significant.

Chlamydia conjunctivitis

It was noted in the last edition that notifications of chlamydial conjunctivitis were up compared to previous years and that this was related to a general increase in PCR testing for chlamydia in eye specimens rather than an increase in clinical trachoma. This quarter saw 38 notifications compared with a mean of 8 for the same period over the past 4 years. In the September quarter last year there were 26 notifications while there were 89 in the April-June quarter this year.

Influenza

This year there was an NT wide epidemic of influenza type A which occurred later in the year than is usual. With 151 notifications for the year and 118 for the September quarter, there were more notifications than for any other year on record. It is difficult to say whether this was due to more influenza or more testing. This issue is being further investigated.

HTLV I

During September quarter this year there were 15 notifications of HTLV I compared with only 1 during the same period last year. However, the mean number of HTLV I infections for this period over the last 4 years was 7.5 with 13 being observed in 2001. The increase this year seems therefore to be within the variation we see from year to year and probably has to do with physician interest in testing.

Melioidosis

There were 7 cases of melioidosis during the quarter; this was unusual for the time of year so each of the cases were investigated. Several were relapses or delayed presentations of earlier infection. Two cases occurred in the same remote community, but further analysis of the organisms revealed them to be different. We could find no link between any of the other cases. Therefore it would seem that the increased number this quarter was a chance occurrence.

Adverse event after Immunisation

There were 13 notifications of adverse events following immunisation (AEFI) in this quarter, compared with 2 for the same quarter in 2002 and 7 for the previous quarter. Twelve of the AEFI were from the Darwin district and 8 were adverse events after meningococcal C conjugate vaccination given in Darwin high schools in September 2003. The meningococcal C vaccine AEFI were 3 vasovagal reactions (known to be common in the adolescent age group), 2 injection site reactions, and 3 reactions with 2 each of the following symptoms: headache, fever and nausea.

Tuberculosis

There were 8 cases this quarter compared to 4 last year. However, the mean over the last 4 years was 21 cases: a figure which is raised due to the very high number notified in 1999 (65 cases). In the years 2000-2002, 10, 6 and 4 cases were notified respectively.

Salmonella

There was a sizeable increase in Salmonella notifications during the third quarter, 49% higher than previous September quarters (see comments page 22)

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Change in July to September quarter 2003 compared to mean of corresponding quarter of previous four years: sexually transmitted infections and blood borne viruses
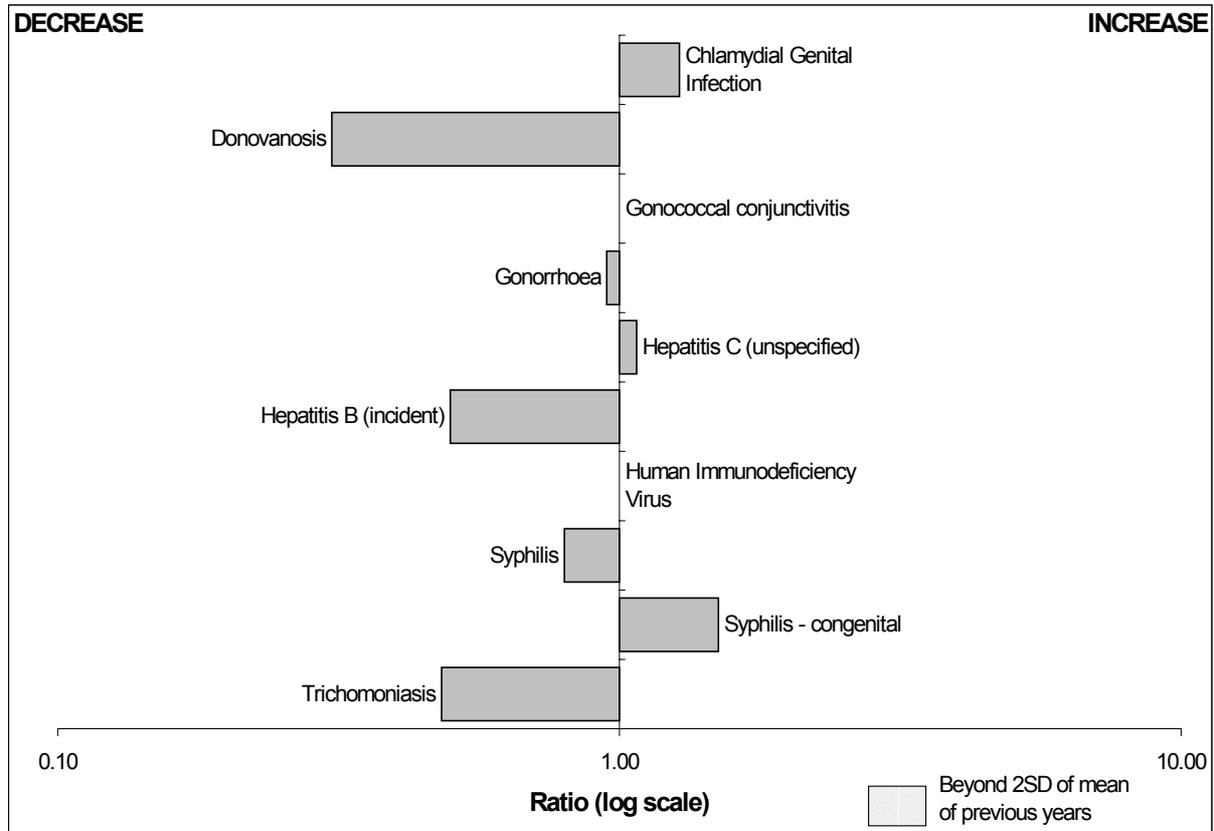
DECREASE   INCREASE

Disease	
Chlamydial Genital Infection	
Donovanosis	
Gonococcal conjunctivitis	
Gonorrhoea	
Hepatitis C (unspecified)	
Hepatitis B (incident)	
Human Immunodeficiency Virus	
Syphilis	
Syphilis - congenital	
Trichomoniasis	

0.10   1.00   10.00

Ratio (log scale)   Beyond 2SD of mean of previous years

Change in July to September quarter 2003 compared to mean of corresponding quarter of previous four years: selected diseases

DECREASE   INCREASE

Disease	
Adverse Event after Immunisation	
Campylobacteriosis	
Chlamydial conjunctivitis	
Cryptosporidiosis	
Influenza	
Melioidosis	
Rotaviral infection	
Salmonellosis	
Shigellosis	
Tuberculosis	

0.01   0.1   1   10

Ratio (log scale)   Beyond 2SD of mean of previous years
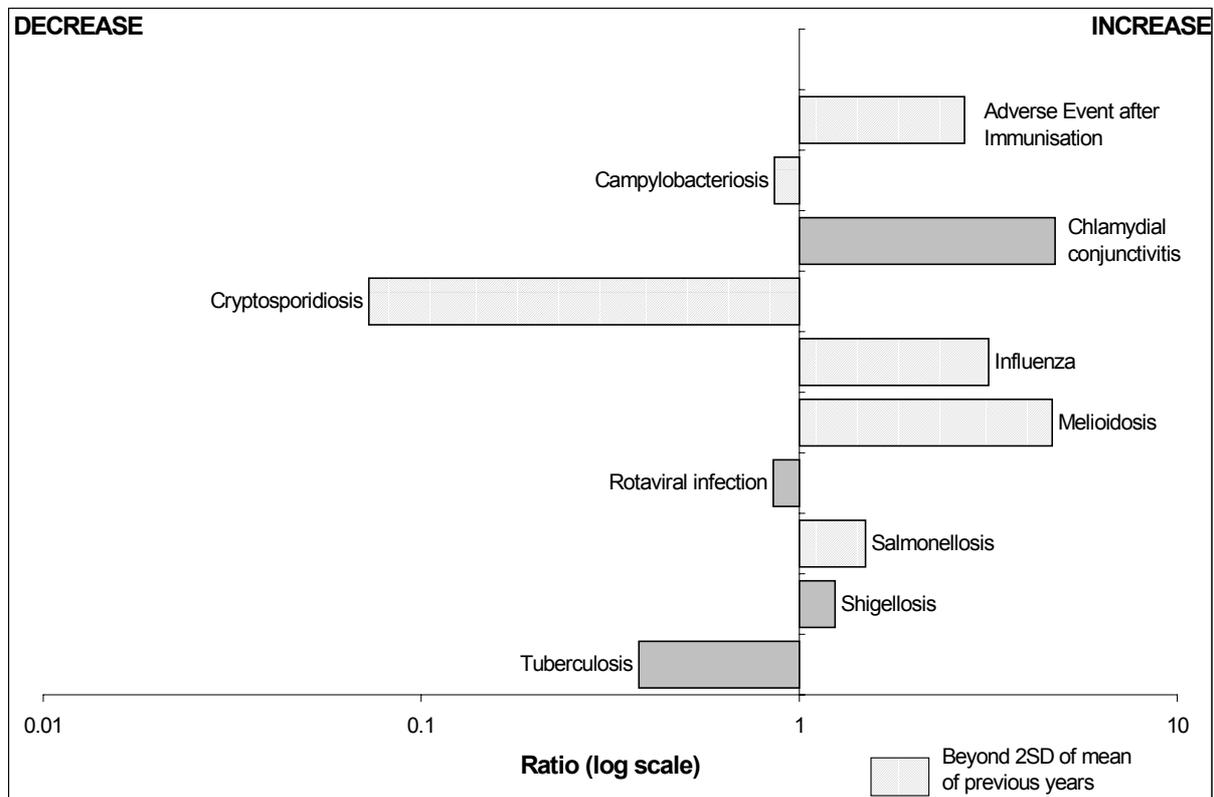
Enteric diseases in the Northern Territory
July - September 2003
Karen Dempsey, OzFoodNet Enteric Disease Epidemiologist,
CDC Darwin.

Gastroenteritis outbreaks

During the third quarter, 2 foodborne disease outbreaks occurred in the Darwin region and were reported to the Centre for Disease Control for follow-up. The first, which involved 5 people and was considered to be caused by *Staphylococcus aureus,* has been reported previously.[1] The second involved 18 people who became ill following consumption of pizza at a party. No food nor stools were made available for testing due to the delay with reporting. Norovirus was considered to be the likely causative organism because more than 50% of cases vomited and there was a long incubation period (mean 37 hours). There were no reports of foodborne disease outbreaks occurring elsewhere in the NT.

One non-foodborne disease outbreak occurred in an extended family of 5 (2 parents, 2 grandparents and a 2-year-old child). Norovirus was isolated in 2 stools and person-to-person transmission from child to adult carers was the probable cause of illness.

One cluster investigation was conducted following 4 cases of *Salmonella* Typhimurium phage-type 9 during a 1-week period, 4th to 9th July 2003. No common exposures were identified apart from the fact that all 4 cases resided in urban Darwin.

Campylobacteriosis

During the third quarter, 52 cases of campylobacteriosis were reported throughout the Northern Territory. In contrast to the first 2 quarters of 2003, the number of campylobacteriosis cases was lower than the mean number reported during the same quarter for the previous 4 years (61 cases). The number of male cases exceeded female cases this quarter (30 compared to 22).

The median age was 24 years reflecting a preponderance of cases in older age groups. In total, children less than 5 years of age only accounted for 33% of cases with 27% attributed to infants less than 2 years of age. Despite this, the age group with the highest rate of disease was 0 to 4 years (388 cases per 100,000 population).

Alice Springs region still experienced the highest rate of campylobacteriosis, although less than previous quarters. The Alice Springs rate was almost 6 times higher than the lowest rate reported by East Arnhem region (169 cases per 100,000 population compared to 29).

The rate of campylobacteriosis in Indigenous persons was almost twice that of non-Indigenous persons (119 cases per 100,000 population compared to 75). The rate difference was lower than previously reported reflecting a substantial decline in Indigenous cases this quarter. Children less than 5 years of age accounted for a large proportion of Indigenous cases (82%). In contrast, children accounted for few non-Indigenous cases with older children (older than 4 years of age) and adults accounting for the majority (93%).

Cryptosporidiosis

Only 1 case of cryptosporidiosis was reported during this quarter. It was a sporadic case and not associated with a swimming pool or child-care centre. This count was considerably lower than the mean number (14) reported during the same quarter for the previous 4 years (Fig. 1).

Figure 1. Cases of cryptosporidiosis, by month; NT, 2000-Sept 2003
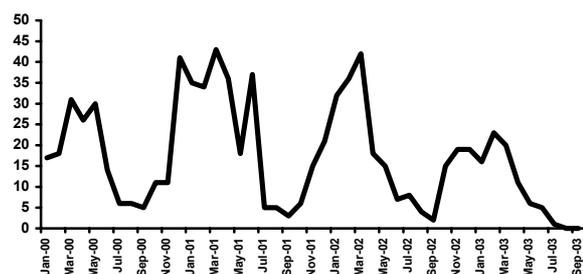
Hepatitis A

A total of 7 hepatitis A cases were reported, mostly in the Alice Springs region (4 cases). The number of hepatitis A cases for this quarter was less than half the mean number (18) reported during the same quarter for the previous 4 years.

Rotavirus

There were 89 cases of rotavirus (61 males and 28 females) reported, much lower than the previous quarter (125 cases). Forty were Indigenous while 42 were non-Indigenous. Children less than 5 years of age accounted for most cases and the majority (59) occurred in the Darwin region, representing the end of the annual May to July epidemic. The total for the 3 months May to July was less than expected this year with 180 cases being reported in those months compared to 329 in corresponding months in 2002 and 504 in 2001. This reduction goes against the recent biennial pattern of larger epidemics every odd numbered year (Fig. 2).

Figure 2.   Cases of rotavirus, by month; NT, 2000-Sept 2003
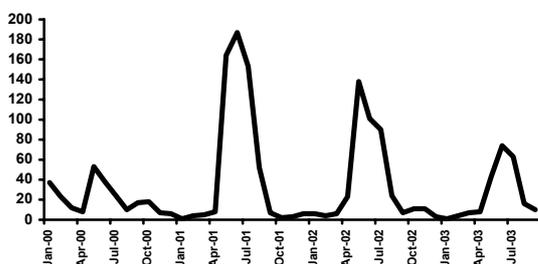
Salmonellosis

During the period July to September 2003 there were 82 cases of salmonellosis reported throughout the Northern Territory, 49% higher than the mean number (55) reported during the same quarter for the previous 4 years. Male cases were the same as females (41 each). The rate of infection was highest in children aged between 0 to 4 years of age (1,119 cases per 100,000 population) and the median age was 2 years. Children aged less than 2 years of age accounted for 44% of cases while children aged less than 5 years of age accounted for 60%. East Arnhem region experienced the highest rate, one

and a half times higher than Darwin region which reported the lowest rate (228 cases per 100,000 population compared to 145).

The rate in the Indigenous population was nearly 3 times higher than in the non-Indigenous population (266 cases per 100,000 population compared to 100).

The predominant serovar was *Salmonella* Ball (11 cases). *Salmonella* Ball is an environmental serovar that has established an ecological niche in the NT and is usually the predominant serovar. *Salmonella* Typhimurium was the next leading serovar with 9 cases, 4 of which were phage-type 9.

There was 1 case of *Salmonella* Enteriditis and 3 cases of *Salmonella* Paratyphi. *Salmonella* Enteriditis RNDC was reported in a 48-year-old non-Indigenous male who became ill during a 2 week visit to East Timor. *Salmonella* Paratyphi B Var Java was reported in 2 people who had no travel history nor contact with aquarium fish; a 27-year-old Indigenous woman from a remote East Arnhem community and a 10-month-old non-Indigenous female infant. The third case of *Salmonella* Paratyphi B Var Java was a 28-year-old non-Indigenous female who became ill during a 1-week visit to Bali.

Shigellosis

During the third quarter 23 cases of shigellosis were reported, 15 of which were in the Alice Springs region. The number reported this quarter was slightly higher than the mean number (19) reported during the same quarter for the previous 4 years.

The rate of infection was highest among children aged between 0 to 4 years of age (365 cases per 100,000 population). The median age was 2 years with children aged less than 2 years of age accounting for 35% of cases and children aged less than 5 years of age accounting for 70%.

The rate in the Indigenous population was almost 12 times higher than that in the non-Indigenous population (133 cases per 100,000 population compared to 11). There were few non-Indigenous cases (4) and the majority of Indigenous cases were reported in children younger than 5 years of age (74%).

Other enteric diseases

There were no cases of yersinios, listeriosis or haemolytic uraemic syndrome during the July to September 2003 quarter.

Reference

1. Dempsey K, Monaghan K. Gastroenteritis outbreak due to *Staphylococcus aureus*. NT Disease Control Bulletin 2003;10(3):39-41.

NOTIFIED CASES OF VACCINE PREVENTABLE DISEASES IN THE NT BY ONSET DATE 1 JULY TO 30 SEPTEMBER 2003 AND 2002

DISEASES	TOTAL		No. cases among children aged 0-5 years	
	2003	**2002**	**2003**	**2002**
Congenital rubella syndrome	0	0	0	0
Diphtheria	0	0	0	0
Haemophilus influenzae type b	0	0	0	0
Hepatitis B	1	2	0	0
Measles	1	0	1	0
Mumps*	0	0	0	0
Pertussis	0	2	0	1
Poliomyelitis, paralytic	0	0	0	0
Rubella	0	0	0	0
Tetanus	0	0	0	0

* Mumps is largely under-reported

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NT NOTIFICATIONS OF DISEASES BY ONSET DATE & DISTRICTS 1 JULY TO 30 SEPTEMBER 2003 AND 2002

Diseases	Alice Springs		Barkly		Darwin		East Arnhem		Katherine		NT	
	2003	2002	2003	2002	2003	2002	2003	2002	2003	2002	2003	2002
Acute post-Streptococcal GN	1	0	0	0	0	0	1	0	1	0	**3**	**0**
Acute Rheumatic Fever	3	10	0	0	2	4	0	3	2	5	**7**	**22**
Adverse Event after Immunisation	1	0	0	1	12	1	0	0	0	0	**13**	**2**
Amoebiasis	0	0	0	0	0	2	0	0	0	0	**0**	**2**
Barmah Forest Virus infection	0	1	0	0	0	2	0	0	1	1	**1**	**4**
Campylobacteriosis	17	21	3	1	29	26	1	5	2	7	**52**	**60**
Chlamydial conjunctivitis	9	1	3	0	23	21	0	2	3	2	**38**	**26**
Chlamydial Genital Infection	165	127	18	4	150	174	26	41	37	34	**396**	**380**
Cryptosporidiosis	0	6	0	1	1	4	0	1	0	2	**1**	**14**
Dengue	0	0	0	0	0	4	0	0	0	0	**0**	**4**
Donovanosis	1	0	0	0	0	0	0	0	0	1	**1**	**1**
Gonococcal conjunctivitis	0	1	0	0	0	0	0	1	0	0	**0**	**2**
Gonorrhoea	167	178	19	5	76	112	24	45	42	65	**328**	**405**
Haemophilus influenzae (not type b)	0	1	0	0	0	2	0	0	0	0	**0**	**3**
Hepatitis A	4	2	0	5	2	6	1	0	0	4	**7**	**17**
Hepatitis B (incident)	0	1	0	0	1	1	0	0	0	0	**1**	**2**
Hepatitis C (unspecified)	5	3	1	1	47	41	1	1	1	5	**55**	**51**
Human Immunodeficiency Virus infect	0	2	0	0	2	2	0	0	0	0	**2**	**4**
Human T-Cell Lymphotrophic Virus	15	1	0	0	0	0	0	0	0	0	**15**	**1**
Influenza	48	12	0	0	63	23	7	4	0	2	**118**	**41**
Legionellosis	0	0	0	0	0	1	0	0	0	0	**0**	**1**
Malaria	0	0	0	0	6	3	0	0	0	2	**6**	**5**
Measles	0	0	0	0	1	0	0	0	0	0	**1**	**0**
Melioidosis	1	0	0	0	6	3	0	0	0	0	**7**	**3**
Meningococcal infection	3	0	0	0	0	1	0	0	1	1	**4**	**2**
Ornithosis (Psittacosis)	0	0	0	0	1	2	0	0	1	0	**2**	**2**
Pertussis	0	0	0	0	0	1	0	1	0	0	**0**	**2**
Pneumococcal Disease (Invasive)	10	9	1	0	11	7	0	0	2	6	**24**	**22**
Ross River Virus infection	0	0	0	0	0	1	0	0	0	0	**0**	**1**
Rotaviral infection	12	11	12	2	59	64	4	36	2	8	**89**	**121**
Salmonellosis	19	4	3	1	44	31	8	3	8	9	**82**	**48**
Shigellosis	15	6	3	2	4	7	0	1	1	2	**23**	**18**
Syphilis	47	57	1	1	13	36	4	5	15	32	**80**	**131**
Syphilis - congenital	2	2	1	0	0	0	0	0	0	0	**3**	**2**
Trichomoniasis	59	52	4	2	40	48	13	26	10	26	**126**	**54**
Tuberculosis	3	0	0	0	5	4	0	0	0	0	**8**	**4**
	607	**508**	**69**	**26**	**598**	**634**	**90**	**175**	**129**	**214**	**1,493**	**1,557**

Points to note regarding notifications page 24:

Anthrax, Murray Valley Encephalitis, Kunjin, Kokobera, Atypical Mycobacteria, Botulism, Brucellosis, Chancroid, Cholera, Congenital Rubella Syndrome, Diphtheria, Gastroenteritis, Gonococcal Opthalmic Neonatal, Haemolytic Uraemic Syndrome, Haemophilus Inf type b, Hepatitis C (incidence), Hepatitis D & E, Hydatid Disease, Leprosy, Leptospirosis, Listeriosis, Lymphogranuloma venereum, Mumps, Plague, Poliomyelitis, Q Fever, Rabies, Rubella, Tetanus, Typhoid, Typhus, Vibrio Food Poisoning, Viral Haemorrhagic Fever, Yellow Fever, Yersiniosis and SARS are all notifiable but had "0" notifications in this period

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NT Malaria notifications July September 2003

Merv Fairley, Clinical Nurse Consultant, CDC Darwin.

Six notifications of malaria were received for the third quarter of 2003. The following table provides details about where the infection was thought to be acquired, the infecting agent and whether chemoprophylaxis was used.

Number of cases	Origin of infection	Reason exposed	Agent	Chemoprophylaxis
4	East Timor	working	P vivax	yes
1	Solomon Islands	holiday	P vivax	yes
1	Papua New Guinea	holiday	P falciparum	no

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Disease Control staff updates

AIDS/STD

Darwin: Catriona Arnold-Nott has commenced maternity leave and **Steven Skov** will be returning as Public Sexual Health Medical Officer in January after his work with the surveillance team. **Diedre Ballinger** continues to work remotely from Adelaide 2 days per week hoping to return by early April. **Danielle Bament** will be replaced at the end of January by **Maggie Richardson** who is moving from Health Development and has previous experience as acting manager with the Women's Health Strategy Unit. **Glen Hall** joins the team as male educator. Glen is an Aboriginal Health Worker who has previously worked at Danila Dilba. The new receptionist for Clinic 34 is **Tracey Hagger**.

Non Communicable Diseases -

Community Child Health Darwin: Welcome to **Catherine Moody** who has replaced **Brad Palmer** as the community child health nurse.

Immunisation

Darwin: Linda McDonell has moved for Family, Youth and Childrens Services to fill the immunisation data entry position.

TB/Leprosy

Alice Springs: Mini Blytheman has departed and **Christina Hosking** commenced as TB/Leprosy public health nurse. Christina completed a Post Grad Diploma in Public Health and Trop Medicine in 2000 and has recently worked at Mareeba, in forensic mental health nursing.

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