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Biting Midges or 'Sandflies' in the Northern Territory

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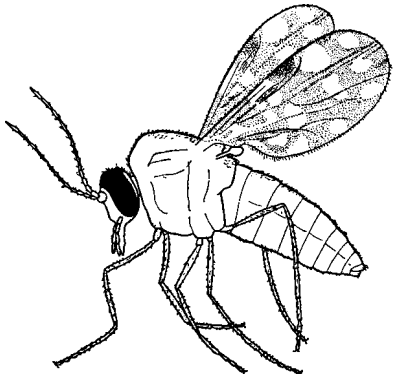
Introduction

Biting midges are small blood sucking flies in the family *Ceratopogonidae* (figure 1). They are commonly referred to as "sandflies" in northern Australia. The term "sand fly" is a misused term for a number of families of small biting flies. This includes the true sandflies, Family *Psychodidae,* (figure 2) which are not pests of humans in Australia, as well as black flies, Family *Simulidae,* (figure 3) which are serious pests in the inland areas of Qld and NSW following flooding, and the biting midges, Family *Ceratopogonidae* (figure 4).[1]

Figure 1. *Culicoides* – a female "Biting Midge"



Entomology for Students of Medicine.
Blackwell Scientific Ltd. 1962

Biting midges are the major midge pest problem in Northern Australia.[2] A number of members of this family bite people in the Northern Territory. They include two species in the genus *Lasiohelea,* which are found biting in small numbers in shaded areas in or near dense forests during the day. A species of *Styloconops* is found in small numbers biting and swarming around the head on open sandy beaches

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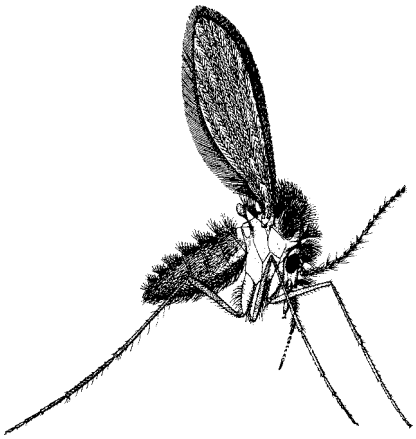
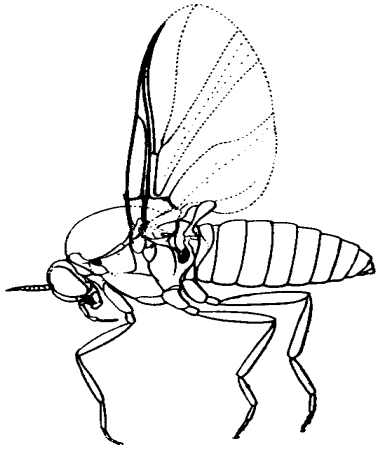
Figure 2. *Phlebotomus* – a female "Sand Fly"



Insects of Medical Importance
British Museum 1956

Figure 3. *Simulium* – a female "Black Fly"



Insects of Medical Importance.
British Museum 1956

during the day. The members of the *Culicoides* genus are more common, with many species and a wide range of breeding sites and biting habits.

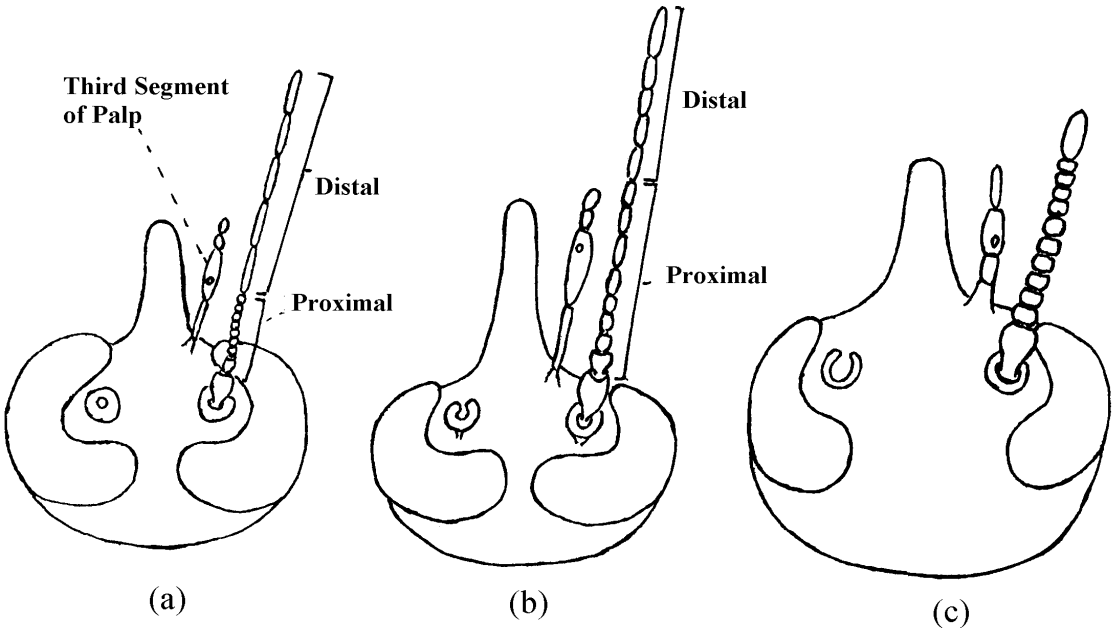
Thirty-three species of *Culicoides* have been recorded from the Darwin area.[3] The *Culicoides* species include some species that don't bite vertebrates, some which preferentially bite cattle and other domestic animals, and the few species that are serious pests of people. The breeding sites include fresh water margins and cattle dung. Most of the serious human pest species breed in tidal and estuarine sites.

Culicoides midges are small, robust flies, approximately 1 mm in length with two wings usually showing a pattern of clear patches on a grey background. They have a short, forward directing proboscis or mouthparts for piercing skin and sucking blood.

Two species, *Culicoides flumineus* and *Culicoides* species near *subimmaculatus* can be severe human pests in mangrove areas across the Top End of the NT, but are rarely found outside mangrove forests.[4]

One species, *Culicoides ornatus,* sometimes referred to as the "mangrove midge", is found in association with mangroves across northern Australia, and is usually responsible for severe biting midge pest problems near the coast. This

Figure 4. Heads of *Ceratopogonidae* (Biting Midges) (a) *Lasiohelea* (b) *Culicoides* (c) *Styloconops*



Atlas of Common Queensland Mosquitoes. Queensland Institute of Medical Research, 1982

species is a major pest because it occurs in very high numbers and has a habit of invading nearby residential or recreational areas.

Culicoides ornatus is becoming an increasing problem across northern Australia due to urban development encroaching nearer to their major breeding places.[5,6,7] They can impose serious restrictions on outdoor activities within flight range of their mangrove breeding sites due to the extremely annoying and painful bites, and to the discomforting after effects of the bites.

Bites of biting midges

It is only the female midges that bite. Biting midges do not transmit disease to humans in Australia. Their main human medical importance is as a biting pest.

Midges must take a blood meal for their eggs to mature. They do not, as is sometimes believed, urinate on people to cause discomfort. In the process of biting and sucking blood, they inject a salivary secretion that produces a skin reaction of varying intensity, depending on an individual's reaction. Bites usually produce a classic allergic response, with the first bite producing no noticeable effect, and the subsequent bites producing the reactions. If the exposure to midges is reasonably continuous, a process of desensitization may follow. People continuously exposed are usually tolerant to the bites, and generally have no reaction or show a mild reaction with a small red spot.[8]

The average reaction for newly exposed people is a red spot that develops a small dome shaped blister with a hole at the top. In people who are more sensitive to bites, the reaction may result in a red swelling over an area of a few centimetres. The bite area can be extremely itchy, and scratching is very difficult to avoid. Reactions may last 3 - 4 days with slowly decreasing irritation. Sometimes scratching breaks the skin and allows secondary bacterial infections that lead to unsightly sores and residual scarring.

Treatment of bites

Mild reactions from bites require little treatment other than the application of soothing lotions. Proprietary products such as Eurax, Stingose, Medicreme, Katers Lotion, Democaine and Paraderm cream can give relief from bites or prevent secondary infection. Useful non-proprietary products include tea tree oil, eucalyptus oil, aloe vera gel, or methylated spirits. Painful reactions to bites can be appreciably reduced by the intermittent application of ice packs to the bite site.

More severe reactions may need medical advice and systemic treatment using antihistamine products such as Phenergan, Telfast or Vallergan. Check with your doctor or pharmacist for available products and safety information.

Breeding sites of *Culicoides ornatus*

Culicoides ornatus is by far the most common biting midge pest around the coast of the Northern Territory.[3]

This midge breeds in the highest numbers in the dry season in the mangrove mud in the creek banks of upper tidal tributaries around the mean high water neap tide mark. This corresponds to an area reached by tides from 4.8 to 6.0 m in Darwin Harbor.[4,9] The prime breeding sites are in a narrow zone in the upper section of the creek bank associated with the occurrence of pneumatophores of the mangrove species *Avicennia marina* on narrow creek banks. The prime dry season breeding site has an upper limit where the *Avicennia* reduces in height and predominance, and a lower limit where the creek opens out from the overhanging *Avicennia* canopy.[4] Broad mangrove areas with many tidal tributaries will have a considerable area of breeding sites.

Other breeding sites of low to medium productivity occur at the front edge of the mangrove forest in the *Sonneratia* or woodland mangrove zone facing open water. These breeding sites are usually associated with mud substrates and not with sandy substrates. Narrow beach fringing mangrove areas are usually not appreciable sources of *Culicoides ornatus,* particularly in areas with sandy substrates.[4]

Another site exploited only in the wet season is in the *Ceriops* transition zone at the back of the creek bank forest. This is just below MHWS (Mean High Water Spring or average high tide mark) or 6.6m ACD (Admiralty Chart Datum) in Darwin harbor. This is where the mixed *Ceriops* starts in a transition from the taller creek bank mangroves to the smaller mangroves in drier,

less frequently flooded areas only reached by tides from 6.5 to 6.8m.

The larvae are small active worm-like creatures that are confined to the surface mud. The larvae take in excess of 6 weeks to mature, when they change into a relatively inactive, air-breathing pupa. The pupa stage lasts only two to three days and the adults emerge around the time of neap tides.[9]

The flight activity of *Culicoides ornatus*

The numbers of adults emerging from pupa cases is related to the lunar cycle, with sudden rises in numbers inside their mangrove breeding sites of the order of 16 times the number occurring on the previous day. The peak in emergence occurs in the two days around the neap tide, although emergence of adults can continue for up to 4 days after the neap tide.[4]

The adults mate soon after emergence. The males are short lived while the females stay in the mangroves to develop and lay their first batch of eggs. The females then start to disperse from the mangroves in an active flight inland in search of blood meals. The dispersal starts about 2 days before the spring tide, and reaches a peak around the day of the spring tide. They show a marked abundance around spring tides with full moons, but are also numerous around spring tides of the new moon.[3]

The adults seek shelter in winds above 8 km/ hour, so that there is little tendency for them to be borne long distances by strong winds. Light breezes from their breeding areas will however aid their dispersal flight. They are active fliers and despite their small size, are relatively hardy insects.

Mass movements of adults can occur to 0.5 to 1.5 km from the mangrove margin of their major breeding sites, although they will move greater distances up creeks and rivers with dense tree cover which form avenues of humidity for dispersal. The dispersal is a purposeful one, with the midges actively flying away from the mangroves. Often higher numbers can be found up to 1.0 km from the mangroves compared to numbers in the mangroves or at the mangrove margin. Elevated hills or escarpments within 1.5 km of prolific biting midge breeding sites often exhibit higher biting midge numbers compared with lower adjacent areas. Minor pest numbers can be detected up to 3 km from the nearest mangrove margin.

Most *C. ornatus* bite in the morning and evening. There is a peak in biting activity in the one hour either side of sunset, with a smaller peak in the one-hour after sunrise of about half the sunset peak. However there is a low level of activity throughout the night.

Seasonal abundance of *Culicoides ornatus*

The annual peak of *Culicoides ornatus* adults in the NT is in the August to October period in the late dry season, with lowest numbers in January and February during the wet season. Populations start to build up from the end of the wet season to the late dry season with a slight decrease in the coldest months of June and July. Populations start to decline rapidly after the first heavy rains occur. However pest numbers can still be present during the seasonal lows in the mid dry season and the mid wet season.

There are three different breeding sites in the mangroves, with varying seasonal productivity from the different breeding sites. Mangroves with small tidal tributaries that contain the prime creek bank breeding sites are dry season breeding sites. The greatest productivity from these creeks occurs in the August to October period. They are not significant sources of midges in the wet season.[4] The back of small mangrove creeks in the *Ceriops* transition zones has moderate productivity in the wet season.[4,9] Areas with extensive *Sonneratia* zones will have moderate productivity at least in the dry season[4] and probably all year around.

Highest numbers of *Culicoides ornatus* occur for the four days around the full moon, with high numbers to a lesser extent, four days around the new moon.

Protection from bites of *Culicoides ornatus*

Avoidance

Culicoides ornatus bite primarily in the early morning or evening around sunrise and sunset. Attacks can occur in the daytime in shaded areas adjacent to the mangroves near major mangrove

breeding areas or in dense creek vegetation that is continuous with the mangrove breeding places. They will continue to bite throughout a still, humid day and warm humid night, particularly in sheltered areas outside the mangroves but close to their breeding areas. Often there is only a little biting activity in the mangroves during the day during and just after the spring tide, as all midges have usually dispersed landward.

Landward areas that are close to and within one kilometre of broad areas of mangroves with many tidal creek tributaries, especially near densely vegetated creeks that run into the mangroves, should be avoided. This particularly applies to the two days either side of the spring tides in the August to November period. Spring tides on full moons have roughly twice as many biting midges as spring tides on new moons.[3]

Minimum pest problems occur in the June-July period during the mid dry season or in January and February in the middle of the wet season. During any month the least pest problems occur in the two to three days either side of the neap tide, particularly neap tides following a new moon. A calendar marked with the 4 days around full moons and new moons, with highlights of seasonal peaks of abundance, can serve as a good midge avoidance reminder.

Biting midges are active under calm conditions and are generally inhibited by wind. Wind protected areas adjacent to and within 1.5km of large expanses of mangroves should be avoided around the spring tide period. People in open areas exposed to winds will experience less pest problems compared to other areas.

Elevated houses and high rise buildings have less pest problems than ground dwellings. Although midges probably fly over dense tree canopies and can fly in appreciable numbers at least 3 metres above the landscape surface, they are generally more numerous lower to the ground surface.[11]

The worst pest problems around Darwin include areas include landward areas adjacent to the mangroves and tidal areas of Sadgroves and Reichardt Creeks, Hudson Creek, Elizabeth River, and Buffalo Creek. The north shore of Frances Bay near Sadgroves Creek in the Charles Darwin National Park is a particularly troublesome area. This is due to the dendrite pattern of numerous narrow mangrove creeks and an extensive Sonneratia zone nearby. Urban areas of Stuart Park, the Narrows, and near Winnellie, which are closest to the Sadgroves creek mangroves, can experience seasonal moderate to minor pest problems. There are some minor pest problems near the lower reaches of Ludmilla creek and Alawa near Rapid Creek. Darwin city itself is relatively free from midges due to the relative lack of mangroves, the exposed cliffs, and the fact that the prevailing SE and NW winds do not blow from mangrove areas.

Clothing and netting

Full-length trousers, socks and shoes, and long sleeved shirts will usually provide considerable protection from midge attack. Pale clothing is generally less attractive than dark clothing. Any exposed part of the body will still be subject to midge bites, with most bites occurring on the legs. Protective clothing should be supplemented with the application of repellants on exposed skin.

Clothing impregnated with permethrin or bifenthrin insecticide offers considerable protection for people continually exposed to biting midges. Impregnation involves soaking the clothing in a prescribed volume and concentration of certain formulations of the insecticide. Protective clothing such as overalls and mosquito nets impregnated with permethrin or bifenthrin will remain effective through one or two washes at the most, and will need reapplication. The insecticides in these treatments can kill the insects after they land on them, but they can also have the effect of interfering with the normal biting behaviour. Impregnated clothes with the additional use of insect repellents can provide extremely good protection.

Normal insect nets and screens are usually not adequate to restrict entry to midges unless the mesh is very fine. Tents screens in particular should have mesh diameter approximately half that of normal mosquito netting. Clothing, screens, netting or tents can be impregnated with permethrin or sprayed with permethrin, bifenthrin or repellents containing Deet to increase their efficiency.

Houses should have outward opening doors and insect screens to prevent entry when opening doors during midge activity.

Repellents

Most repellents have limitations because of their short duration of effectiveness (about 2-4 hours) and their irritability to mucous membranes around the eyes and mouth. Care is needed with young children to avoid the spread of repellent to their eyes or mouth. Repellents are also removed by perspiration.

Repellents that contain Deet (diethyl toluamide) or Picaridin as the active constituent offer considerable protection. Mixtures of natural oils or oils with natural ingredients such as herbs or antiseptics are not as effective as repellents containing Deet or Picaridin. In general effective repellents require above 10% Deet and 9% Picaridin. Repellents in lotions are more effective than alcohol based spray-ons, while gels are the most effective formulations. Repellents can also be applied to mosquito netting or insect screens, although a sample application on a small piece of netting is wise as some repellents affect synthetics. Repellents containing relatively high amounts of Deet can melt some plastics, although those containing Picaridin don't have the same effect.

Other methods of repelling biting midges include the use of coils, repellent oil lamps, and electric vapor pads impregnated with insecticide. These work satisfactorily in closed situations such as rooms, or sheltered patio and veranda situations out of the wind, where a cloud of vapour or smoke can build up. However they cannot provide satisfactory protection in windy and exposed situations.

Smoke from a fire with green leaves will give some protection in emergency situations. Burning aromatic and oil producing foliage of plants such as *Hyptis* (horehound), *Calytrix* (turkey bush), *Melaleuca* species (paperbark) and *Eucalyptus* species (gum trees) can give appreciable protection. Rubbing the skin with the leaves of some of these plants can also provide some protection, but this is not as good as recommended repellents.

The so-called "electronic mosquito repellers" that emits a frequency that is supposed to repel biting midges by imitating the noise of males do not work and offer no protection against biting insect attack.

There is an urban myth that taking Vitamin B1 or thiamin can act as a repellent. There is no scientific evidence that Vitamin B1 acts as a repellent, or helps to reduce the reaction to insect bites by developing some immunity to the bites.[13] Other topical applications such as a Dettol™ and baby oil mixture do offer some physical barrier to biting midges, but are not as effective as Deet or Picaridin based repellents. The best protection from biting insects remains the avoidance of the problem areas at times of abundance and the use of protective clothing in combination with efficient repellents.

Use of lights

Biting midges can be attracted to lights. Houses in biting midge problem areas should have dull outside lighting, with little internal light visible from outside. Lightproof curtains that can be drawn at night offer a good alternative. Outside lights should be away from insect screens, as the midges attracted to the light can then penetrate the screens. Outside lights should be yellow (or red, which is even better) to reduce their attractiveness to biting insects. Attractive lights such as large incandescent bulbs or white or ultra violet fluorescent tubes positioned a distance away from a house or building can deflect biting midges to some extent. However rows of streetlights positioned between mangroves and residential areas are not effective barriers to midge dispersal inland.[11]

The reduction of vegetation

The reduction of vegetation around houses or recreation areas can reduce problems by removing shelter for the midges. A buffer of clear open space between the mangroves and residential areas can reduce biting midge numbers in a residential area, as long as the buffer is wide and subject to winds. However clear open buffers by themselves offer little protection unless they are at least 1 km wide. Mowing a wide margin around houses to eliminate dense grass can help reduce the available areas where midges can harbor.

The use of attractant traps

There are a number of insect attracting traps on the market. They generally use light or carbon dioxide as an attractant and either trap the insect in a container, electrocute, or drown the insects. Some are more useful than others but can not be relied to give considerable protection from bites for unprotected people in close proximity to the traps. In most cases they attract biting insects to the general vicinity and these are then diverted to

people, who are more attractive targets. Some traps can help to reduce the overall population, as long as there are enough traps, the biting insect population is relatively small, and the area is isolated from re-invasion from other areas. However most trapping techniques can not cope with the huge populations of midges at one time, and those not trapped still result in a pest problem.

Evaluation of biting midge problems

The Medical Entomology Branch of the Department of Health and Community Services has conducted numerous investigations into biting midge problems in the Top End of the NT.[2,3,5] Potential problems have been investigated by trapping midges overnight using special carbon dioxide (CO2) baited traps. The number of midges collected can be counted or estimated by weight or volume and identified to species under a microscope in the laboratory.

The number of bites by biting midges that constitute a pest problem will largely depend on an individual. It has been suggested that over 60 bites per hour for most experienced biting midge workers are the thresholds of acceptability. For people unaccustomed to biting midge bites, even 1 to 5 bites per hour may be considered unbearable.

There is an approximate relationship between the number of midges collected in a CO2 trap and the number of bites that can be expected at the peak biting period. For an unprotected person, the number of bites in an hour at the peak biting time is approximately one quarter of the number collected in a CO2 trap over one night at the same position. Thus CO2 collections of over 240 per carbon dioxide trap per night are likely to represent a pest problem (equal to over 60 bites per hour) to unprotected people with prior experience of biting midges. Collections of over 1000 per trap per night represent over 250 bites in an hour and would constitute a major pest problem. Trap collections of over 5,000 per trap would constitute a severe pest problem.[3]
The numbers of *C. ornatus* collected by CO2 traps in different locations can indicate the magnitude of the human pest problem in each location. Trapping on a constant day in relation to the tide cycle over every month in a year can give an indication of the seasonal population fluctuations. Trapping at different distances from the mangroves and in different vegetation types can give an indication of the dispersal of midges into various areas.

Control of *Culicoides ornatus*

Insecticide fogging for adult midges

Insecticide fogging is the application of aerosol size particles directed against active flying insects. Insecticide fogging operations in residential areas by vehicle or hand held equipment are usually not very effective measures to eliminate pest problems, due to the rapid re-infestation of midges from nearby breeding and harborage areas. Sometimes re-infestation occurs very soon after the fog has cleared, although up to 12 hours protection can be achieved in some localized situations.

For effective midge control, the entire midge breeding and harboring area near residential development needs to be fogged each day over the 3-4 day period of peak emergence. This has to be timed to coincide with the time just after the midges have emerged and before they begin to disperse out of their breeding areas. This area would also have to be relatively isolated from other such areas to prevent re-invasion. Fogging also has to be carried out during the peak activity period in the evening and early morning.

For vehicle ground based operations, the fog has to be able to drift into the target area on favorable winds of the right velocity and in the right direction. This often reduces the opportunity for effective fogging. Fogs do not usually penetrate more than 50m into dense forested areas such as mangroves, monsoon forests and other thick vegetation.

One of the major problems is determining the level of control required. A reduction of *C. ornatus* numbers by 99% may be required to reduce a large pest problem to an appreciable level. This may be impossible to achieve for various operational purposes, and if there were still any remaining pest problem, the control would not be cost effective.

In the Darwin situation, the mangrove breeding and harboring areas are generally inaccessible, too wide, or too extensive for ground based application methods to effectively reduce midge

numbers, although some temporary relief would be possible in some areas.

Aerial application of insecticides aimed at adult midges in breeding and harborage areas has given the best results in overseas investigations, but in some instances there has been immediate re-infestation. It is a difficult practice, as the breeding grounds have to be closely delineated and fogging must be based on an accurate forecast of adult emergence times. The fogging has to be with sufficient regularity to kill all the emerging dispersing females over the night and fog drift to nearby residential areas has to be avoided. Fogging is not carried out regularly for midge control in Australia and requires more local research. Fogging involves large continuing costs, which is often beyond the resources of many local authorities. Insecticide resistance and the killing of other insects pose additional potential problems.

Barrier spraying

The application of insecticides to create an artificial barrier or an insect killing zone around houses where biting insect harbor before biting offers some promise as a new control method. The application of residual insecticides to exterior walls, screens, patio plants, nearby hedging plants or lawns and other close vegetation may kill midges attracted to houses or people.[14] Insecticides that can be used include permethrin, deltamethrin and bifenthrin. Bifenthrin has the advantage over other similar insecticides, as it appears to have less of a repellent or agitation effect on insects, is less irritant to people, is ultra violet resistant, and binds very well to surfaces to give it a good residual effect.[14] As with all synthetic pyrethroids, it must only be applied as per the label and kept out of fish habitats.

Insecticide control of larval habitats

Breeding site treatment by applying insecticides to kill larvae before emergence of adults is a possible control method but there have been very few examples of successful larval treatment in mangrove areas. Larval habitat treatment involves considerable costs and organization, which is impractical in extensive breeding areas such as those surrounding Darwin. Insecticides would need to have good residual qualities and be able to penetrate dense mangrove tree cover and mud in a tidal situation. Most insecticides with these qualities would generally kill non-target insects. The problem of accurately delineating all the significant breeding sites and the seasonal fluctuation of breeding sites pose additional problems.

Elimination of breeding habitats

Reclamation of mangroves has been successful in eliminating biting midge breeding sites in various localized situations. This usually requires large amounts of fill material which is neither cheap or readily available. For *Culicoides ornatus*, the reclamation needs to extend from near the average high tide level to below the outer mangrove forest. This may involve significant engineering considerations posed by deep mud and erosion of the filled area.

Reclamation would not be practicable in most of the Darwin area because of the extensive areas involved. The destruction of large areas of mangroves would be environmentally undesirable and unacceptable to public opinion. This potential solution would only be practicable in localized areas if the breeding site was small, in close proximity to residential development, was regarded as an area of reduced environmental importance, and the filling could create a stable shore environment.

There should be conclusive evidence that the site to be reclaimed is a significant source of biting midges and that the midges are significant pests to nearby residential development. Mangroves can be an indicator of biting midge breeding sites, but the presence of mangroves does not confirm any site as the breeding place. Other specific factors such as substrate types are involved in productive breeding sites.

Buffer zones

There is some evidence that creating a buffer zone between urban residential development and mangrove areas can reduce the dispersal of biting midges into residential areas. Clearing of vegetation and mowing to allow wind disruption, or extensive streetlights or roads with active traffic in the buffer zone may enhance the buffer to some extent.[3] However extensive testing of a modified buffer with lights and different vegetation types in Darwin have shown that unmodified buffers and lights by themselves are not effective barriers to *C. ornatus* dispersal from mangroves to urban areas.[11] The effectiveness of buffers is generally related to the width of the buffer and the presence of blood

sources or other attractions such as light in the buffer zone. However semi-urban residential or industrial development between mangroves and urban areas can reduce midge dispersal inland. In general, unmodified buffers need to be in the order of 1.5km, and modified buffers in the order of one kilometre to offer significant reduction in numbers.

Planning guidelines to prevent biting insect problems

The Medical Entomology Branch is involved in the planning process to reduce the effects of biting insects. Guidelines have been prepared for preventing biting insect problems in new urban and semi rural residential developments, industrial, and other developments.

In 1974 the planning for the new satellite town of Palmerston near Darwin included a buffer of at least 1-km from the mangrove boundary to urban residential development.[12] Palmerston is one of the few urban areas in Australia that has been specifically designed to minimize biting insect problems.

Good urban planning is required to;

- reduce the risk of biting insect pests
- recognize and avoid areas of biting insect breeding or harborage
- avoid costly and environmentally undesirable rectification methods
- avoid costly and ongoing biting insect control programs

The Medical Entomology Branch gives advice on what may constitute a potentially significant biting insect breeding site. In some instances detailed entomological investigations are necessary to gather sufficient information before the detailed planning stage. The avoidance of biting insect problems can be achieved in the initial planning process by consideration of development location, easements, buffer zones, and sub division design.

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Initial survey of underground mosquito breeding sites in Darwin, NT

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Introduction

Several studies on the dengue vector *Aedes aegypti* in northern Queensland have revealed a considerable percentage of the larval population to be living and surviving the unfavourable dry or winter season in underground breeding sites.[1,2] Service pits and manholes have been found to contain mosquito larvae.[1,3] The presence of natural predators of mosquito larvae such as *Mesocyclops* (Crustacea, Copepoda) species is known to considerably reduce mosquito breeding in service pits and manholes.[2, 3]

Darwin and the NT are free from the two main dengue vectors *Aedes aegypti* and *Aedes albopictus*.[4, 5] Darwin is considered a locality of high risk for the introduction of these exotic *Aedes* species.[5, 6] The Medical Entomology Branch (MEB) and the Australian Quarantine and Inspection Services (AQIS) carry out a comprehensive exotic mosquito surveillance program with the main goal to enable early detection of any exotic mosquito importation and institute elimination procedures.[7]

Since surveys began in 1974 all importations of exotic *Aedes* mosquitoes in Darwin have been successfully eliminated.[4, 8, 9] However, there is always the possibility of late detection, which could lead to the establishment of dengue vectors. The current paper is a preliminary investigation of the abundance of, and potential for, mosquito breeding in underground sites such as Telstra pits and manholes in Darwin. The purpose of the survey was to confirm the presence of water in pits and manholes, and sample these for mosquito larvae and aquatic predators such as *Mesocyclops*.

Methods

A Telstra officer and a MEB officer jointly carried out the initial survey on 27th March 2003. The Telstra officer opened the pits and manholes according to Telstra safety procedures and requirements. A manhole allows the entry of personnel to inspect and work inside the facility. The smaller pits are designed for inspection and maintenance purposes and do not allow physical entry by personnel. The manholes and pits sampled were selected for their likelihood of holding water near the end of the wet season.

The presence or absence of water was recorded. Water depth was measured using a ruler. Sampling for mosquito larvae and other aquatic invertebrates, including plankton, was carried out using a 100 ìm mesh plankton net of 20 cm length with an opening of 20 cm x 10 cm on an extendable handle.[10] The net was swept along the sides and corners of the manhole/pit on the surface and then in deeper water for 50-60 seconds. The net was emptied into a photographic tray and the sample transferred to sealable containers. The samples were taken to the MEB laboratory and placed into 70% ethanol. Mosquito larvae were identified using a stereo microscope. Plankton was sorted and *Mesocyclops* specimens were identified to genus level under a compound microscope.

Results

During the initial survey, ten service manholes and six pits were sampled (Table 1). Of these, more than half of the manholes were found to hold water, but only 1 pit held water (Table 1). The service pit holding water did not contain mosquito larvae or *Mesocyclops*. Of the 6 manholes holding water, 3 were breeding mosquitoes and 1 had *Mesocyclops* (Table 1). The copepods remain to be identified to species level to determine if they are potential mosquito larval predators. The mosquito larvae were all *Ochlerotatus (Mac) tremulus*.

Discussion

This survey of service manholes and pits at the end of the wet season in Darwin revealed the presence of water and mosquito larvae. The only mosquito species recorded was *Ochlerotatus tremulus*, an endemic species that is known to use both natural and artificial containers as breeding sites.[11] This species was also commonly found in the surveys of underground breeding sites in Queensland.[2, 3, 12] These studies often found a close association between the presence of *Ochlerotatus tremulus* and *Aedes aegypti*.[2, 12] The presence of *Ochlerotatus*

Table 1. Summary of survey of Telstra service pits and manholes in Darwin, 27 March 2003

Type	Number sampled	Number holding water	Number positive for mosquito larvae	Number positive for *Mesocyclops*
Manhole	10	6 (60%)	3 (30%)	1 (10%)
Pit	6	1 (16.7%)	0 (0%)	0 (0%)
Total	**16**	**7 (43.8%)**	**3 (18.8%)**	**1 (6.3%)**

tremulus in manholes in Darwin indicates their potential to serve as potential breeding sites for *Aedes aegypti*.

A larger survey is planned for the wet season 2003/04 to establish the amount of endemic mosquito breeding in manholes and pits, and the duration of breeding throughout and after the wet season. Part of the study will aim at establishing the relationship between a manhole or pit holding water with the recent rainfall, the soil type and topography. It is also planned to survey other underground breeding sites, such as PowerWater manholes and roadside side entry pits (SEP's) of underground stormwater drains. The results of these surveys will form the basis of a dengue management plan for Darwin and the Northern Territory.

Conclusions

Mosquito breeding does occur in underground manholes, indicating the possible use of underground breeding sites by exotic *Aedes* mosquitoes. These sites will require specific precautionary treatment in the quarantine area for all future risk importation of exotic *Aedes* mosquitoes. A larger survey in cooperation with Telstra will investigate mosquito breeding in the underground sites on a temporal and spatial scale.

Acknowledgments

Special thanks to the following people for their support: Ananda Waniganayake, John Stopp, and Andrew Donnolly from the Telstra Corporation.

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Firework related injuries during Territory Day Celebrations 2003

Meredith Neilson, Chronic Disease and Injury Prevention Project Officer, CDC; Didier Palmer, Director Emergency Department Royal Darwin Hospital; Jackie Glennon, Public Health Medical Officer, Alice Springs; Jan Bullen, Coordinator CDC Katherine; Hartley Dentith, Manager CDC Nhulunbuy

Introduction

Active surveillance of firework injuries has been conducted annually by the Centre for Disease Control (CDC) in the Darwin region over the last 5 years. To gain a Territory-wide perspective of injuries, the information was collected over the entire Northern Territory (NT) in 2003.

Methods

Between the 28th of June and the 8th of July 2003 nursing staff in all NT hospitals collected data from people presenting to the Emergency Departments with injuries sustained from fireworks. Data was also collected over the same period at General Practices, the Australian Defence Force Health Services, Aboriginal Medical Services, Community Health Clinics, and Community Care Centres in the NT.

Survey forms were sent to the above health services and completed forms were faxed back at the end of the study period. A consent form allowed officers from Worksafe to conduct follow-up interviews to determine whether the injuries were due to faulty or illegal fireworks or unsafe use.

Results

In 2003 there were 32 people reported with injuries due to fireworks: 21 in Darwin/ Palmerston, 6 in Katherine, 3 in Alice Springs and 2 in Jabiru. The Top End of the NT reported 91% of the injuries. All injuries were sustained at private displays, predominantly by bystanders.

Of all persons injured by fireworks, 60 % occurred in males and 45% were in the 16-30 year age group (Figure 1).

Injuries to the hand (23%), lower extremities (23%) and eye and facial regions (31%) were the most common body regions affected (Figure 2). Of all persons injured, 62% were classified as moderate or severe (Figure 3). One injury required immediate admission and an eye injury required extensive review and day surgery at a later date.

Mechanisms of injury

Injuries in 2003 were caused in the following ways:

- 2 incidents where several sparklers were held together and burned rapidly resulting in severe hand burns;

Figure 1. Firework related injuries 2003 by age and sex

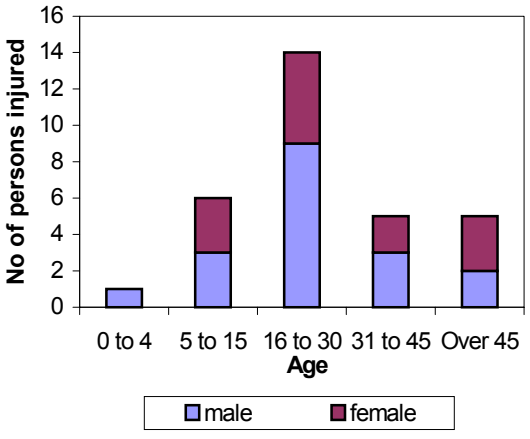


Figure 2. Body region affected by firework related injury

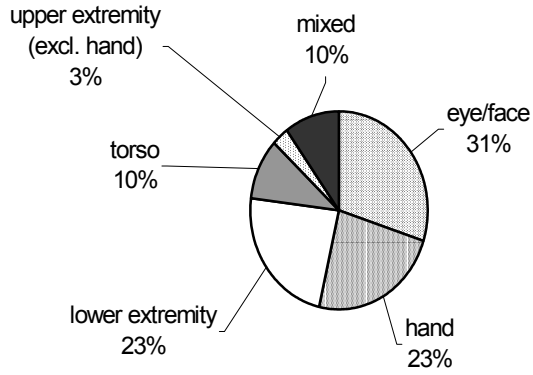
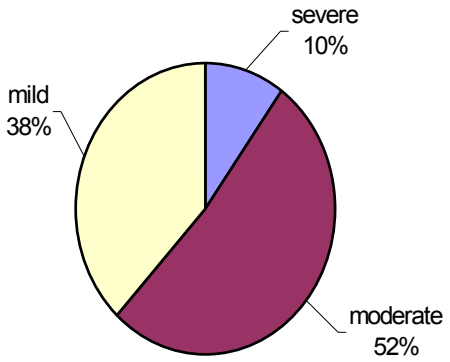


Figure 3. Severity of Firework related injury



Severe: admitted to hospital for IV antibiotics, analgesia, dressings, grafts
Moderate: requiring 2 or more reviews by a health practitioner
Mild: requiring only one visit to a health practitioner

- 3 incidents where fireworks exploded near faces creating eye injuries and facial burns (1 person continues to have treatment and requires day surgery for his eye);
- A 4 year old child was injured when his trousers were set alight by wayward fireworks;
- A wayward firework exploded under a plastic chair resulting in burns to the buttocks and thighs, and shrapnel injuries requiring a 3 day hospital admission.
- A firework hit a male in the genital and upper thigh area causing partial thickness burns; and
- Extinguishing a cigarette in an ash tray containing gunpowder residue from fireworks created a forceful explosion causing full thickness hand burns.

In the NT there have been 16 presentations to Emergency Departments, 28 appearances at the Burns Clinic, 1 immediate hospital admission, 2 surgical procedures, 12 Ophthalmology consultations, and 47 GP clinic visits from 28 June to 30 August 2003.

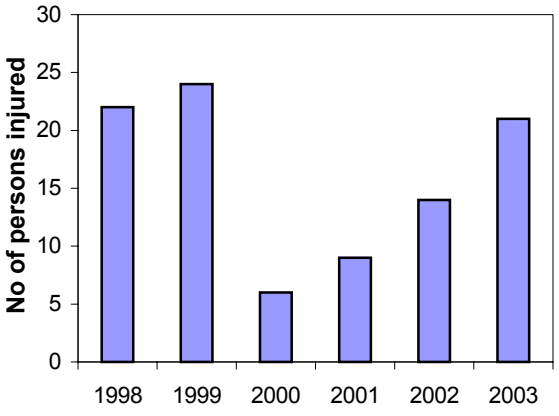
Trends

Comparison with previous years data from Darwin urban region allowed trends to be examined. Following an initial decline in the number of persons injured in 2000 there has been an increase between 2000 and 2003 (figure 4).

There were 7 more persons injured by fireworks reported this year compared to 2002 (Figure 4). Of these, 40% were sustained by 16-30 year olds (Figure 5) and 52% sustained by males. There were fewer severe injuries (2) reported this year compared to 2002 (5) with 57% of all firework injuries classified as moderate.

The estimated cost of health care to treat

Figure 4. Total number of persons injured by fireworks in Darwin 1998-2003

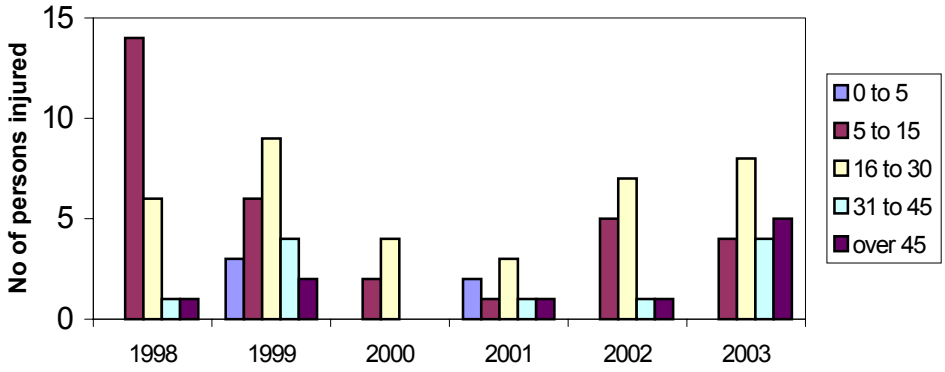


firework related injuries in the Darwin/ Palmerston region decreased this year compared to last year from $33,421 to $15,839. This may have been due to fewer severe injuries this year. These costs were calculated by the NT Department of Health and Community Services (DHCS) with assistance from the Hospital Services Program.

Other Community Reports

The Fire Department in the Darwin/Palmerston region reported 27 call outs this year compared to 66 in 2002. The Police also reported fewer call outs and the RSPCA reported fewer complaints compared to previous years.

Figure 5. Number of persons injured by fireworks by age group in Darwin 1998-2003



These reports stated that people were generally "well behaved". However there were several dangerous incidents on Territory Day. Many people took private fireworks to Mindil Beach in Darwin, the site of a public fireworks display. The combination of a high tide reducing the area for release of fireworks, and the vast number of people present (estimated at 14,000), created a hazardous environment.

Discussion

The number of persons injured by fireworks has steadily increased in the Darwin/Palmerston area in the last three years. In 2000 there was a decline in cases which coincided with the implementation of the Worksafe coordinated Fireworks Working Group. The Group consisted of representatives from Worksafe, Fire and Emergency Services, Department of Employment Education and Training, NT Police, and the DHCS. The aim of the group was to promote safer firework celebrations on Territory Day and provide ongoing implementation of safety campaigns.

In 2003 the strategies employed by the group included reducing the duration of sale of fireworks from 3 days to 2 days, conducting Burns Awareness Week in the week prior to Territory Day, and running a Safe Use of Fireworks campaign in schools. School age children were targeted for safety messages this year with 16,500 fliers placed in school newsletters across the NT. The 2003 safety campaigns obtained a higher profile than previous years. Interviews and paid newspaper advertisements promoted safe use of fireworks, exposed illegal wholesalers, and discussed the School Campaign. Despite these measures the injury rate has increased.

The data obtained from the 2003 survey across the NT will assist in allowing comparisons in future years and help direct safety campaigns. Of concern in 2003 was the relatively high number injured in Katherine compared with other areas.

Future safety campaigns need to be directed towards young adult males as a large proportion of injuries in 2003 were sustained in this population group. The public also needs to be educated on the safe use of sparklers as annually there are several injuries due to mishandling. Worksafe are currently investigating each person injured to determine whether the cause was incorrect use or faulty product.

As a result of the injury surveillance from 2003 the DHCS has come up with the following recommendations:

- Continue the DHCS Firework-related Injury Survey across the NT
- Ban private fireworks displays at Mindil Beach and other public displays
- Coordinate a media campaign to encourage the public not to take fireworks to public displays
- DHCS to continue to work with the Fireworks Safety Group to target young adult males in future safety campaigns
- Educate community on safe use of sparklers

Acknowledgments

Special thanks to Jackie Mein who first implemented the community surveillance of firework injuries in 1998.

TB control in the Darwin Correctional Centre and the crew of boats carrying asylum seekers

Nathan Zweck, Merv Fairley, Paul Kelly, Vicki Krause – TB unit, CDC Darwin

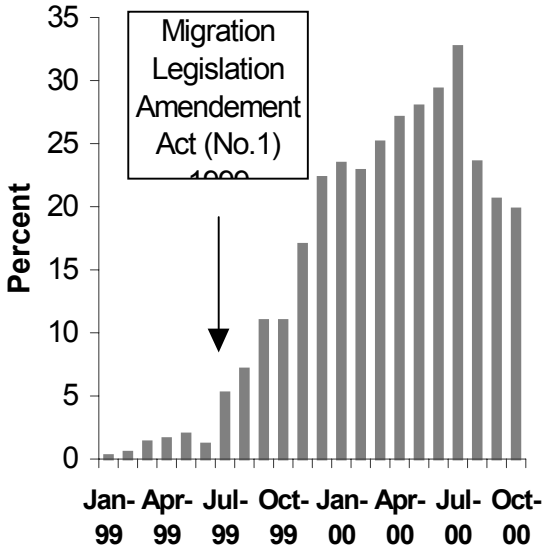
Introduction

Until 1999, screening of inmates for tuberculosis (TB) in the Darwin Correctional Centre (DCC) consisted of a Mantoux test on reception, which if positive (≥10mm induration) was followed by a chest x-ray days or weeks later. Each inmate also underwent a reception clinical examination by the prison's medical officer which included the symptoms and signs of TB.

However, a recent change in Commonwealth legislation necessitated an intensified approach. The *Migration Legislation Amendment Act (No. 1) 1999* was introduced in July 1999 in the Northern Territory (NT) and provided for harsher penalties for the Indonesian crew of boats carrying asylum seekers to Australia (up to 10 years in prison and a maximum fine of $110,000).[1] Prior to this, the maximum penalty was 2 years imprisonment[2], but crewmembers were usually deported soon after their cases were heard in the Darwin Magistrate's Court. Since September 1999 they have been tried and sentenced (usually for several years) in the Supreme Court of the NT. The previously negligible fraction of the prison population who were Indonesian nationals subsequently burgeoned to 33% (119/364) by July 2000 (S. LaBrooy, personal communication, November 2000, Figure 1). This influx was additional to small background numbers of Indonesian fishermen who have been incarcerated in DCC for days or weeks for offences under the *Fisheries Management Act 1991*.

The estimated annual TB incidence in Indonesia in 2001 was 271 per 100,000 population[3], around 10-fold that in the NT. Therefore, crew from Indonesia have a higher probability of entering DCC with active pulmonary TB (PTB) than Australian-born prisoners. The standard process for TB screening had the potential for unacceptable delays of days or weeks before disease detection. Therefore, since August 1999 Indonesian nationals have been screened with a chest x-ray as soon as possible after reception *regardless of Mantoux results*, and since March 2000, with the cooperation of the Department of Immigration, Multicultural and Indigenous Affairs (DIMIA), Australian Federal Police, Northern Territory Correctional Services, and the Royal Darwin Hospital, a chest x-ray has been performed at the Royal Darwin Hospital *prior* to reception at the DCC.

Figure 1. Indonesian nationals as a proportion of the Darwin prison population, January 1999 to October 2000



This report describes the prevalence of latent TB infection (LTBI) and active TB in a cohort of Indonesian inmates and judges whether intensified control activities for this group are justified.

Methods

Data collection and analysis

Data were collected in an Access database about DCC inmates (or those in custody and destined for DCC) who were clinically assessed at least once by staff of the NT Department of Health and Community Services TB Unit in the years 1999-2002. Data described by quarter or by year, refers to the date first assessed by the TB unit. Statistical analysis was performed using SPSS software (SPSS for MS Windows, release 6.1).

Table 1. Summary of screening procedures for TB in different groups of prisoners and detainees

Crew carrying asylum seekers	♦ CXR and report by TB Unit or Emergency Department medical officer prior to reception at DCC*
Unauthorised fishermen	♦ Symptom screen while detained on Darwin Harbour; ♦ assessment by medical officer (general practitioner) if symptom screening positive; ♦ CXR and TB Unit assessment prior to court appearance if medical
All prisoners	♦ Mantoux test, and assessment by prison medical officer at reception in DCC; ♦ CXR and review by TB Unit medical officer if Mantoux test positive (>=10mm) or symptoms suggestive of TB

*since March 2000; † since February 2001

Diagnosis of LTBI and TB

Screening of prisoners and those destined for prison varied according to a classification summarised in Table 1. At reception all prisoners underwent Mantoux testing with 10 international units of purified protein derivative intradermally (left forearm, flexor aspect) by staff of the private prison clinic contractor, unless they had a history of a positive Mantoux test or clinical TB. Inmates with ≥10mm induration had a chest x-ray and a clinical assessment by visiting staff of the TB Unit. If active TB was excluded, LTBI was diagnosed. Treatment of LTBI was offered if the sentence length allowed completion before discharge and there were no medical contraindications.

Screening crew of boats carrying asylum seekers

Since August 1999, all Indonesian crew were screened with a chest x-ray as soon as possible after reception regardless of Mantoux results. Since March 2000 those crew charged by the Australian Federal Police were screened for signs of active pulmonary TB with a chest x-ray at the Royal Darwin Hospital *prior* to reception at the DCC. If necessary, they were admitted to a respiratory isolation ward-room, investigated, and treatment was initiated there. When considered non-infectious (3 consecutive sputum specimens negative for acid-fast bacilli on microscopy) they were transferred to DCC where treatment was continued.

Screening of fishermen

Those charged with unauthorised fishing in Australian waters were detained in their vessels on Darwin Harbour awaiting arraignment to the Darwin Magistrate's Court, and a small proportion were ultimately incarcerated in DCC, usually for several weeks. Since February 2001, harbour detainees who were likely to be arraigned (captains and repeat offenders, around 100 per year) underwent screening for TB with a symptom questionnaire administered by staff of Barefoot Marine Pty Ltd with an Indonesian interpreter. If TB was suspected, they were reviewed by a designated Darwin general practitioner, and if required, chest x-ray screening was performed.

Definition of active TB

The diagnosis of active TB was according to the case definitions for TB in the NT[4]:

1. Isolation of *Mycobacterium tuberculosis* complex (*M tuberculosis, M bovis, M africans*) from a clinical specimen, or
2. Demonstration of AFB in a clinical specimen or in a histopathological specimen when a culture is not available in a patient with compatible symptoms or clinical or radiological signs, or
3. Compatible signs and symptoms and evidence of resolution following anti-TB treatment.

Definition of the completion of LTBI or TB treatment

Treatment for LTBI constituted 182 doses of isoniazid (6 months, daily administration) within 8 months or 60 doses of rifampicin and pyrazinamide or "2RZ" (2 months, daily administration) within 3 months. Treatment success was defined as ingestion of >=80% of

Table 2. Characteristics of 743 inmates clinically assessed in Darwin by the TB Unit, 1999-2002, by ethnicity

Factor	All n=743	Ethnicity			
		Aboriginal n=292	NAAB* N=85	Indonesian N=326	Other OSB† n=40
Age					
Median (Range)	29 (10-75)	31 (10-55)	36 (11-65)	25 (21-75)	39 (15-65)
Gender					
Male %	97.8	96.9	92.9	100	97.5

*NAAB = Non-Aboriginal Australian-born; †OSB = Overseas-born

the intended doses within the allowed time-frame. Treatment for TB constituted 182 doses of combined treatment (6 months, daily administration) within 8 months. Treatment success was defined as ingestion of >=80% of the intended doses (i.e. 146 doses) with 8 months, provided that 28 or more consecutive doses were not missed. If 28 or more consecutive doses were missed, a full course of treatment was recommenced.

Results

There was a total of 743 inmates clinically assessed on at least one occasion by the TB unit on, or prior to, arrival at the Darwin Correctional Centre in the 4-year period 1999 to 2002. The demographic details of the group are displayed by ethnic classification in Table 2. Of 743 inmates, 326 (44%) were Indonesian citizens and all Indonesians were male. Of the Indonesians, 284 (87%) were charged with transporting asylum seekers and 42 (13%) were charged with unauthorised fishing in Australian waters. The median age of the Indonesians was significantly lower than that of the other 3 ethnic groups combined (25 v 32 years; Wilcoxon Rank Sum test, $p < 0.0001$). Almost one-third (104/326) of the Indonesians were aged 12 to 19 years. None of the Indonesians were HIV-seropositive.

Latent TB

Of the total of 743 inmates assessed, 656 (88%) were diagnosed with LTBI, 17 (2%) had active TB, and 70 (9%) were not infected with TB (Table 3). The majority of those not infected (56/70) were Indonesians who were assessed by the TB unit whether their Mantoux result was positive or negative, in contrast to other ethnic classifications who were seen only if they recorded a positive Mantoux test or were symptomatic.

Of 326 Indonesians assessed, 323 (99%) underwent Mantoux testing and 268 (83%) of those tested had induration measuring ≥10mm (Figure 2). The 3 Indonesians not Mantoux tested included 2 fishermen who were deported prior to incarceration but after clinical and radiological assessments. After excluding 13 with active TB, 255 (95%) of the 268 with positive Mantoux tests were diagnosed with LTBI, 173 (68%) of whom accepted and commenced treatment. The completion rate was 96%.

Figure 2. Mantoux results and outcome of treatment of LTBI for Indonesian inmates

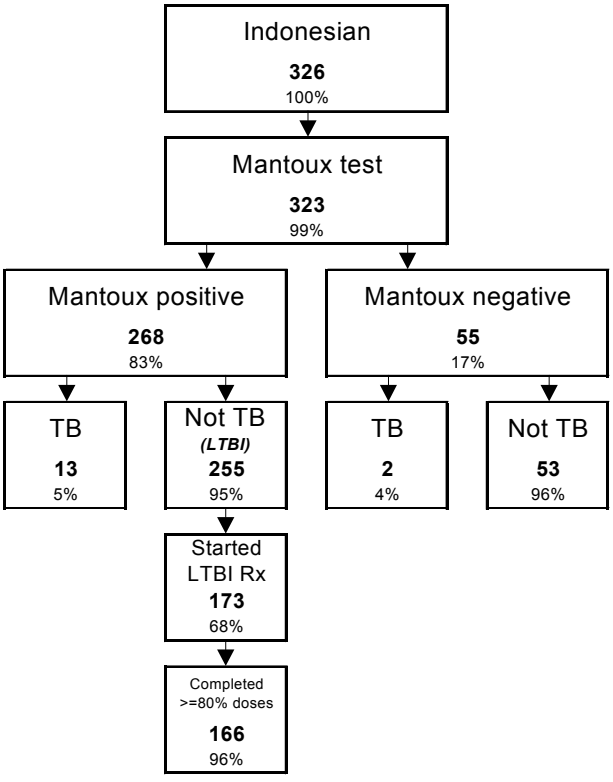


Table 3. Diagnoses, and outcomes of treatments, for all inmates assessed in Darwin by the TB unit, 1999-2002, by ethnicity

	All		Aboriginal		NAAB		Indonesian‡		Other OSB	
	n	%	n	%	n	%	n	%	N	%
Diagnosis										
LTBI	**656**	*88.3*	**285**	*97.6*	**80**	*94.1*	**255**	*78.2*	**36**	*90*
TB	**17**	*2.3*	**1**	*0.3*	**0**	*0*	**15**	*4.6*	**1**	*2.5*
Not infected	**70**	*9.4*	**6**	*2.1*	**5**	*5.9*	**56**	*17.2*	**3**	*7.5*
Total seen '99-2002	**743**	*100*	**292**	*100*	**85**	*100*	**326**	*100*	**40**	*100*
LTBI treatment										
INH completed pre-99	**17**	*2.6*	**11**	*3.9*	**5**	*6.3*	**0**	*0*	**1**	*2.8*
INH started 1999-2002	**222**	*33.8*	**35**	*12.3*	**9**	*11.3*	**173**	*67.8*	**5**	*13.9*
2RZ started 1999-2002	**8**	*1.2*	**5**	*1.8*	**3**	*3.8*	**0**	*0*	**0**	*0*
LTBI Rx not started	**409**	*62.3*	**234**	*82.1*	**63**	*78.8*	**82**	*32.2*	**30**	*83.3*
Total	**656**	*100*	**285**	*100*	**80**	*100*	**255**	*100*	**36**	*100*
Outcome of INH for LTBI 1999-2002										
Completed 100% doses	**191**	*86*	**23**	*65.7*	**3**	*33.3*	**161**	*93.1*	**4**	*80*
80-99% of doses	**8**	*3.6*	**3**	*8.6*	**0**	*0*	**5**	*2.9*	**0**	*0*
< 80% of doses	**12**	*5.4*	**4**	*11.4*	**2**	*22.2*	**5**	*2.9*	**1**	*20*
Stopped ADR	**4**	*1.8*	**1**	*2.9*	**1**	*11.1*	**2**	*1.2*	**0**	*0*
Still taking	**7**	*3.2*	**4**	*11.4*	**3**	*33.3*	**0**	*0*	**0**	*0*
Total	**222**	*100*	**35**	*100*	**9**	*100*	**173**	*100*	**5**	*100*
Outcome of 2RZ for LTBI 1999-2002										
Completed 100% doses	**6**	*75*	**3**	*60*	**3**	*100*	**0**	*0*	**0**	*0*
< 80% of doses	**1**	*12.5*	**1**	*20*	**0**	*0*	**0**	*0*	**0**	*0*
Stopped ADR	**1**	*12.5*	**1**	*20*	**0**	*0*	**0**	*0*	**0**	*0*
Total	**8**	*100*	**5**	*100*	**3**	*100*	**0**	*0*	**0**	*0*
Outcome of TB treatment 1999-2002										
Completed 100% doses	**14**	*82.4*	**1**	*100*	**0**	*0*	**13**	*86.7*	**0**	*0*
< 80% doses (deported)	**1**	*5.9*	**0**	*0*	**0**	*0*	**1**	*6.7*	**0**	*0*
Deported pre-diagnosis	**2**	*11.8*	**0**	*0*	**0**	*0*	**1**	*6.7*	**1**	*100*
Total	**17**	*100*	**1**	*100*	**0**	***0***	**15**	*100*	**1**	*100*

‡ All Indonesian inmates were assessed whether Mantoux positive or negative; other ethnic groups were assessed if Mantoux positive at reception or previously

NAAB, non-Aboriginal Australian-born; OSB, overseas-born; LTBI, latent TB infection; TB, active tuberculosis; INH, isoniazid; 2RZ, rifampicin and pyrazinamide for 2 months; ADR, adverse drug reaction

Excluding those who had completed LTBI treatment before 1999, 36% (230/639) of all inmates with LTBI accepted treatment with either 6 months of isoniazid (97%) or 2RZ (3%). The acceptance of treatment for LTBI by eligible Indonesians was high (68%) compared with eligible members of other groups, ie. 15% for Aboriginal, 16% for NAAB, and 14% for non-Indonesian overseas-born (Figure 3). The proportion of all inmates who did not complete LTBI treatment due to discharge from prison or an adverse drug reaction was 7% for isoniazid (16/222) and 25% for 2RZ (2/8). Only 4% (7/173) of the Indonesian cohort failed to complete LTBI treatment compared with 14% of Aboriginal, 33% of NAAB, and 20% of other OSB inmates (Figure 2).

Active TB

Pulmonary TB was diagnosed in 17 persons including 15 Indonesian nationals, 1 Aboriginal, and 1 Vietnamese national (Table 4). Diagnosis was by pre-reception or reception screening for 16 cases, while 1 case arose later during the

period of incarceration after pre-reception assessment had excluded active TB (Box 1, Case B, p 23). Respiratory specimens were culture-positive for *Mycobacterium tuberculosis* (MTB) in 7 cases (41%) and 1 of these was sputum smear-positive for acid-fast bacilli. The remaining 10 cases (59%) were smear-negative and culture-negative for MTB and the diagnosis was made by excluding other aetiologies and taking into account clinical and radiological findings and their improvement with anti-TB treatment.

Of 326 Indonesians, 15 (4.6%) were diagnosed with active PTB during the study period including 14 cases diagnosed at the time of arrival in Australia (prevalent cases). The point prevalence of TB in this cohort on arrival was 14/326 or 4.3% (95%CI: 2.1-6.5%) and for culture-positive cases 6/326 or 1.8% (95%CI: 0.4-3.3%). Of the 312 Indonesians who did not have prevalent TB on arrival, 1 (0.32%) later developed incident disease.

All 7 culture-positive cases of TB (6 Indonesians, 1 Vietnamese) had isolates sensitive to the 4 standard anti-TB drugs used in the NT - rifampicin, isoniazid, pyrazinamide, and ethambutol.

Table 4. Details of 17 persons diagnosed with TB disease in custody or in prison in Darwin, 1999-2002.

Case no.	Quarter-Year first seen	Age	Ethnicity	Symptoms (a)	Offence (b)	Mantoux test(mm)	Sputum mycobacteriology results				CXR (e)	Case def.initio n (f)
							Smear (c)	Culture	ID (d)	Sensitivity Profile		
1	3-1999	60	Indonesian	NS	Crew	30	-	+	MTB	RZHE (g)	F, C	1
2	3-1999	43	Indonesian	-	Crew	32	-	+	MAC		C	3
3	3-1999	53	Indonesian	C, F	Crew	0	-	-	-		F, C	3
4	3-1999	30	Indonesian	C	Crew	12	- (h)	+	MTB	RZHE	F, C	1
5	4-1999	30	Indonesian	W, NS	Crew	45	-	-	-		C	3
6	4-1999	49	Indonesian	-	Crew	22	-	+	MTB	RHZE	F, C	1
7	4-1999	30	Aboriginal	C, H	Other	16	-	-	-		F, C	3
8	4-1999	50	Indonesian	C, F	Crew	14	-	-	-		F, C	3
9	1-2000	36	Indonesian	H, W, P	Crew	25	-	-	-		C	3
10	2-2000	19	Indonesian	W	Crew	28	-	+	MAC		C	3
11	2-2000	58	Indonesian	C, H	Crew	10	-	-	-		F, C	3
12	4-2000	60	Indonesian	C, S, W, F, NS	Crew	15	-	-	-		F, C	3
13	4-2000	48	Indonesian	C, H	Crew	20	-	-	-		F, C	3
14	4-2000	35	Indonesian	C, W	Fisherman	0	-	+	MTB	RHZE	C	1
15	3-2001	42	Indonesian	S	Crew	30	-	+	MTB	RHZE	C	1
16	4-2002	41	Indonesian	NS	Fisherman	27	- (i)	+	MTB	RHZE	C	1
17	4-2002	43	Other OSB	W, H	Visa expiry	17	+	+	MTB	RHZE	F	1

Key: (a). Symptoms – C, Cough; S, Sputum production; W, Weight loss; H, Haemoptysis; F, Fever; NS, Night Sweats; P, Chest pain (b). Offence – Crew, crewmember of a boat carrying asylum seekers; Fisherman, unauthorised fishing in Australian waters (c). -, negative; +, positive (d). ID, Species identification; MTB, Mycobacterium tuberculosis; MAC, Mycobacterium avium complex (e). CXR, chest x-ray findings; F, fibrosis; C, consolidation (f). See methods section for 3 case definitions (g). R, rifampicin; Z, pyrazinamide; H, isoniazid; E, ethambutol (h). Bronchoalveolar lavage specimen (I). Induced sputum specimen

Table 5. Univariate analysis for selected variables and the prevalence of TB among Indonesian inmates at reception

	Sample N	TB	Not TB	Prevalence per 100,000	Odds Ratio	95% Confidence Interval	p-value
Mantoux result							
Median (mm)		21	18				0.29
>= 10mm	268	12	256	4478	1.24	0.38-4.0	1.0
< 10mm	55	2	53	3636			
>= 30mm	30	4	26	13333	4.35	1.28-14.8	0.03
< 30mm	293	10	283	3413			
Total	323	14	309				
Age							
Median (yrs)		45.5	24				< 0.0001
>= 40 yrs	54	10	44	18519	15.2	4.56-50.5	< 0.0001
< 40 yrs	272	4	268	1471			
Total	326	14	312				
Year assessed							
1999	77	7	70	9091	3.46	1.17-10.2	0.03
2000-02	249	7	242	2811			
Total	326	14	312				
Offence							
Crew	283	12	271	4240	0.89	0.10-7.57	0.7
Fishing	42	2	40	4762			
Total	325	14	311				

Predictors of active TB among the Indonesian cohort at reception

Table 5 shows the univariate analysis for possible predictors of all TB cases diagnosed in Indonesians at the time of prison reception. The median age at the time of first assessment of the 14 cases with active TB (45.5 years) was significantly higher than for the 312 who did not have active TB (24 years). The odds ratio for TB for those 40 years and over was 15.2 (95%CI: 4.6-50.5) compared with those under 40 years of age. A positive Mantoux result (>=10mm) is not significantly associated with TB. However, Mantoux results of 30mm and above are associated with a significantly higher prevalence of TB (13%) than results under 30mm. The prevalence of TB was not significantly different for Indonesians charged with transporting asylum seekers compared with those charged with unauthorised fishing. The proportion of Indonesians who had TB fell from 9.1% (7/77) in 1999 to 5.5% (6/109) in 2000 to 0.8% (1/131) in 2001. The proportion of arrivals aged 40 or more reduced from around 20% in 1999 and 2000 to 11.5% in 2001 (Figure 3).

Logistic regression using CXR category (normal/abnormal) in addition to the variables shown in Table 4 shows that age is the only independent significant predictor of TB ($p < 0.0001$), but the best model includes age (continuous), Mantoux category (30mm cutoff), and year of assessment (1999-2000 v. 2001-2002).

Utility of screening modalities

If we accept as a gold standard for the diagnosis of TB the combination of clinical, radiological, and bacteriological assessments, the sensitivity and specificity of a CXR alone (100% and 76%

Figure 3. Percentage of Indonesians in each age category by year of first assessment

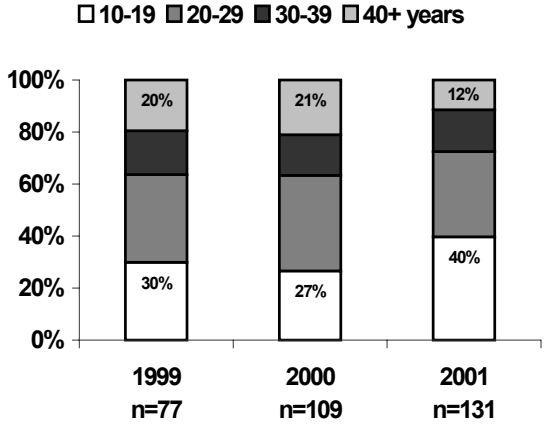


Table 6. Comparison of the Mantoux test (10mm cutoff) and chest x-rays for detection of active TB in 326 Indonesian prisoners in Darwin, 1999-2002.

	TB	Not TB	Total
Mantoux positive	13	255	268
Mantoux negative	2	53	55
Total	15	308	323*
Mantoux test: sensitivity = 87%, specificity = 17%, positive predictive value = 5%, negative predictive value = 96%			
Abnormal CXR	15	74	89
Normal CXR	0	236	236
Total	15	310	325†
CXR: sensitivity = 100%, specificity = 76%, positive predictive value = 17%, negative predictive value = 100%			

*3 did not have a Mantoux test; †1 did not have a CXR (not boat crew nor fisherman, inmate for only 2 days)

respectively) to detect active TB among the Indonesian cohort was superior to the Mantoux test with a 10mm cutoff (76% and 17% respectively, Table 6). The proportion of Indonesians with a positive Mantoux test who had TB (5%) was similar to the proportion with a negative Mantoux test who had TB (4%, Figure 2).

Discussion

This report describes a high prevalence of active TB at or prior to reception in the Indonesian cohort. Although the non-Indonesian, overseas-born cohort during 1999-2002 was small (40), the only smear-positive case among 743 prisoners was diagnosed in a citizen of Vietnam giving a comparable point prevalence (2.5%, 95%CI: -2.3-7.3%) to the Indonesian cohort. Countries in our region with estimated rates of TB over 100 per 100,000 population whose citizens have been incarcerated in Darwin include Vietnam (179), China (113), Philippines (297), Thailand (135) and Cambodia (585).[3] Chest x-ray screening regardless of Mantoux results is also appropriate for these citizens, but coordinating this to occur *prior* to incarceration will be more difficult than for Indonesian crew or fishermen whose arrival is less sporadic and more predictable.

Between 1989 and 1998 there were only 3 cases of TB notified in the NT among prisoners - 2 Indonesian fishermen in 1994 in Darwin, and 1 Aboriginal in Alice Springs Prison in 1996 – equating to a mean of 0.3 cases per year for the 10 year period (unpublished data). The current study period 1999-2002 has seen a total of 17 cases notified in 4 years, a mean of 4.25 cases per year representing an increase of 13 times compared with the earlier period.

The absence of smear-positive PTB among the Indonesian cases suggests that screening allowed disease detection at an early stage when mycobacteriology was either culture-positive alone, or culture-negative. This benefits the individual since earlier treatment improves their prognosis. Failing to detect these early cases at reception could allow silent deterioration to smear-positive disease increasing the risk of transmission within the prison. More advanced cases of smear-positive PTB may not have appeared in this Indonesian cohort since more severe illness (with malaise, weight loss, and debility) could preclude a case from embarking on the arduous journey by sea to Australia.

If only the culture-positive cases of PTB among Indonesians are considered, the point prevalence on arrival of 1.8% was high enough to demand that intensive screening for active TB should continue for this group. Although cases with smear-negative, culture-positive PTB have been shown to be only 22% as likely to transmit infection as those with smear-positive disease, transmission has still been shown to occur, and screening should therefore occur *prior* to prison reception.[5] A relatively high proportion (57%) of the Indonesian PTB cases were culture-negative compared to 28% of 377 cases of PTB diagnosed in the NT between 1989 and 2001 (unpublished data). High proportions of culture-negative cases have been documented in other settings where active screening rather than passive presentation with symptoms has been the method of case finding[6,7] (Table 7). This study documents a similar prevalence of TB of approximately 4000/100,000, and of culture-positive TB of approximately 2000/100,000, to 2 recent studies where refugees from South-East Asia were screened for TB.[6,8]

Table 7. Prevalence of TB and culture-negative proportion diagnosed by screening in selected cohorts

Authors	Sample	Prevalent TB cases	TB Prevalence/ 100,000			% Culture negative
			Overall	Culture positive	Culture negative	
MacIntyre & Plant, 1999	1101 refugees in camps in Laos, Cambodia, Vietnam	41	3724	2452	1272	34
Kelly et al, 2002	1863 East Timorese evacuees in Darwin	76	4079	2040	2040	50
van Burg et al, 2003	46424 European, Middle-Eastern, African asylum seekers in the Netherlands	103	222	86	136	61
This study	326 Indonesians in custody in Darwin	14	4294	1840	2454	57

There is no infirmary or respiratory isolation facility in the DCC. Thus, workup of inmates suspected of having PTB must be performed in the Royal Darwin Hospital, with each prisoner under 24 hour guard by 2 prison officers at an approximate cost to the DCC of $2000 per day. Furthermore, adequacy of staffing levels in the DCC is compromised by this exercise. There is therefore some urgency for TB to be rapidly ruled in or out if 3 sputum smears are negative and discharge to the DCC is imminent. Culture negativity will only be ascertained 8 weeks later so it is preferable to commence TB treatment and make a definitive decision about continuing treatment based on clinical, bacteriological and radiological parameters after 8 weeks.

The utility of using chest x-rays to screen all Indonesians regardless of Mantoux results or symptoms suggestive of TB is supported by the data concerning TB cases. Of the 15 Indonesian cases, 2 (12%) had a Mantoux result of 0mm, and a further 2 (12%) were asymptomatic (Table 4). Therefore, the presence of symptoms, or a positive Mantoux test alone should not be relied upon to correctly indicate a chest x-ray for disease detection. What is required in the crowded environment of a prison where the stakes are high if a missed diagnosis occurs is a test which is 100% sensitive, and the CXR fulfilled this for the Indonesian cohort (Table 6). The absence of HIV infection among the Indonesians is notable, since in other settings, 7 to 14% of HIV-seropositive persons with active TB have had a normal CXR.[9,10]

There was also a very high rate of LTBI in this Indonesian cohort. Unpublished NT data for the period 1989 to 2000 has previously shown that Indonesian inmates have higher rates of Mantoux positivity than the other 3 ethnic classifications (Indonesian 80%, Other OSB 49%, Aboriginal 39%, NAAB 29%). During 1999-2002, 83% of the Indonesian cohort were Mantoux positive, and 78% had LTBI.

The alteration in Commonwealth legislation relating to penalties for transportation of asylum seekers to Australia introduced the new prospect of incident cases among Indonesian inmates from reactivation of LTBI during sentences of several years duration. This risk has been reduced by the prompt treatment of LTBI in the majority of the cohort. A lifetime risk of active TB of up to 20% in persons born in high-prevalence countries who have a positive tuberculin skin test[11] translates to the prevention by this program of approximately 20 to 30 cases of active TB. Since the initial years after migration is the period of highest risk for immigrants to develop TB in a new country,[8,12] many of these "prevented cases" among Indonesians are likely to have arisen during incarceration in Australia.

The major reason for the higher uptake of LTBI treatment among Indonesians compared with other groups is the comparatively longer sentences being served by the 284 Indonesians convicted of transporting asylum seekers. The adults have been sentenced for at least 1 year, which allows a 6-month course of treatment to be completed. By contrast, 66% of the total prisoner population were sentenced for less than 6 months during 1998-1999.[13]

Heightened suspicion for active TB is required when screening older Indonesians in custody.

Age of 40 years or over is now included as a risk factor in a proposed clinical screening tool for use by DIMIA compliance officers to determine which deportees in their custody require chest x-ray screening prior to incarceration. It is also included along with symptoms suggestive of TB as an indication for referral of detained fishermen to a designated general practitioner for clinical TB screening, and chest x-ray if indicated.

One could debate the utility of prosecuting and incarcerating crew for several years as a deterrent to the arrival of asylum seekers. Long term solutions to forced mass population movements lie with addressing the root causes of civil conflict in countries of origin, increasing aid to countries of first transit and the United Nations High Commission for Refugees, and improving access to migration for persons from war-torn regions. However, while people from countries with high rates of TB continue to be imprisoned in the NT, heightened surveillance, and activities which prevent the progression, reactivation, and transmission of TB in the DCC must be maintained.

Acknowledgements

We are grateful to the following persons and agencies whose cooperation made the intensified screening possible: Chris Wake & Corrections

Box 1. Selected histories of cases with active TB

Case A. Prevalent case in a crewmember
A 60 year old Indonesian charged with people-smuggling in 1999 complained only of night-sweats and the Mantoux result was 30mm. His CXR showed bilateral upper lobe linear shadows and volume loss consistent with old TB, but with superimposed patchy consolidation consistent with reactivation. He was isolated in the Royal Darwin Hospital until 3 sputum specimens were shown to be smear-negative for acid-fast bacilli, empirical treatment for TB was commenced, and he was discharged to the prison. Later, the sputa cultured *Mycobacterium tuberculosis*, and following completion of a full course of anti-TB treatment his weight increased from 45 to 54kg. After serving 12 months of his sentence, he was returned to Indonesia.

Case B. Incident case in a crewmember
A case of PTB in a 36 year old Indonesian arose 6 months after reception. He had previously been detained in Australia for unauthorised fishing in 1997. His chest x-ray in 1997 showed bilateral apical fibrosis, but clinical and bacteriological assessments did not diagnose active PTB, and he was returned to Indonesia. When charged with people-smuggling in 2000, the chest x-ray changes were unchanged in comparison with 1997, sputum mycobacteriology was negative, and with a Mantoux reading of 25mm, he was treated for LTBI. After 5 months of LTBI treatment he developed chest pain, haemoptysis and weight loss, and a chest x-ray showed new nodular left apical infiltrates suggestive of TB reactivation. Despite negative sputum mycobacteriology, he commenced anti-TB treatment, clinically and radiologically improved, and completed a full course.

Case C. Prevalent case in a fisherman
A 35 year old Indonesian charged with unauthorised fishing in Australian waters in 2000 and destined for the DCC was instead isolated and investigated in the Royal Darwin Hospital since his pre-reception chest x-ray showed patchy consolidation in the right upper lobe. He had symptoms of an occasional dry cough and weight loss in the previous month and his Mantoux reading was 0mm. After 5 days under guard in the Royal Darwin Hospital, his sentence was served, 2 sputum specimens were smear negative for AFB, and he was returned to Indonesia. However, both sputa cultured *Mycobacterium tuberculosis* 2 months later. No follow up to recommend treatment was possible.

Case D. Prevalent case in a DIMIA deportee
A 43 year old citizen of Vietnam whose visitor's visa had expired was briefly incarcerated in DCC in 2002 prior to deportation under the Migration Act. Being neither in the custody of the Australian Federal Police, nor the Australian Fisheries Management Authority, he was not screened for TB prior to reception according to the established algorithms for crew transporting asylum seekers and unauthorised fishermen. His Mantoux result at reception was 17mm so a chest x-ray was performed. This was suggestive of old healed PTB with bilateral upper lobe fibrosis. There was a history of 6kg weight loss in the previous year and occasional haemoptysis. Three sputa were requested for mycobacteriology and the first 2 were smear negative. He was deported on the day the 3rd sputum was obtained, and this was subsequently reported as smear positive for acid-fast bacilli and cultured *Mycobacterium tuberculosis*. Negotiations are currently underway with DIMIA staff regarding clinical screening of deportees by compliance officers, and if indicated, chest x-ray screening at the Royal Darwin Hospital prior to incarceration in Darwin prisons or police holding cells.

Medical Services staff; Angela Brannelly and St Vincent's Correctional Health Service staff; Tony Tucker, Director Department of Immigration, Multicultural and Indigenous Affairs, Darwin; Roy McKay, Australian Fisheries Management Authority, Darwin; Jenny Scullion, Barefoot Marine Pty Ltd; Stewart LaBrooy, Northern Territory Correctional Services; Rod Williams, Steve Russell, and all prison officers who facilitated clinics, Darwin Correctional Centre; Ramzi Jabbour & Geoff Eyles, Australian Federal Police; Didier Palmer, Emergency Dept, Royal Darwin Hospital; Roger Weckert & Mark Palmer, Radiology Dept, Royal Darwin Hospital; and Dom Modoh and the many interpreters who assisted us.

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TB in Two Katherine Region Communities

Joy Pascall and Margaret Cooper, CDC Katherine

(Excerpts from a presentation at the September 2001 CDC Conference in Darwin)

Introduction

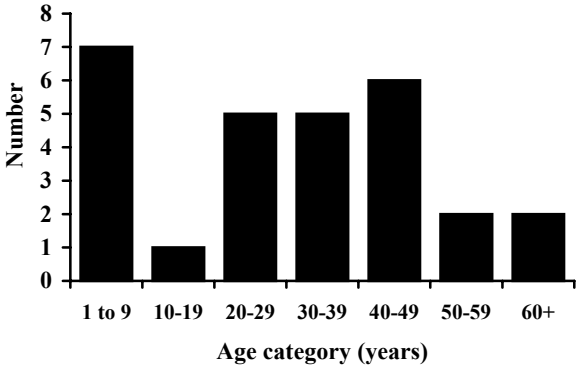
Communities A and B, which are located in the Katherine region, have historically had high rates of TB. Community A has a health clinic which provides primary health services to both communities and was staffed at the time by a Registered Nurse and an Aboriginal Health Worker (during the tourist season 2 Registered Nurses). Community B is in close proximity and the people inter-relate with community A to a high degree.

This article is a summary of a community based project to treat cases of latent tuberculosis infection (LTBI) in these communities.

TB epidemiology 1987-1999

TB has been a major health problem within these 2 communities. The first recorded case was in 1966, and from 1987-1999 28 cases of TB were notified (Figure 1). There was 1 TB-related death in 1989. Assuming population stability, this equates to a mean annual incidence of 560 per 100,000 for the 13-year period, compared with mean NT and national rates of approximately 25 and 5 per 100,000 respectively. Of the 28 cases, the site of disease was pulmonary in 20 (71%), nodal in 7 (25%) and peritoneal in 1 (4%). A majority of the cases were among men (61%), and those aged 20 to 49 (57%) (Figure 2). Of note is the high proportion of cases which were children under the age of 10 years (25%) including 3 of the last 6 cases in the period. This reflects recent transmission of TB in these communities as well as diagnosis of early disease due to active screening.

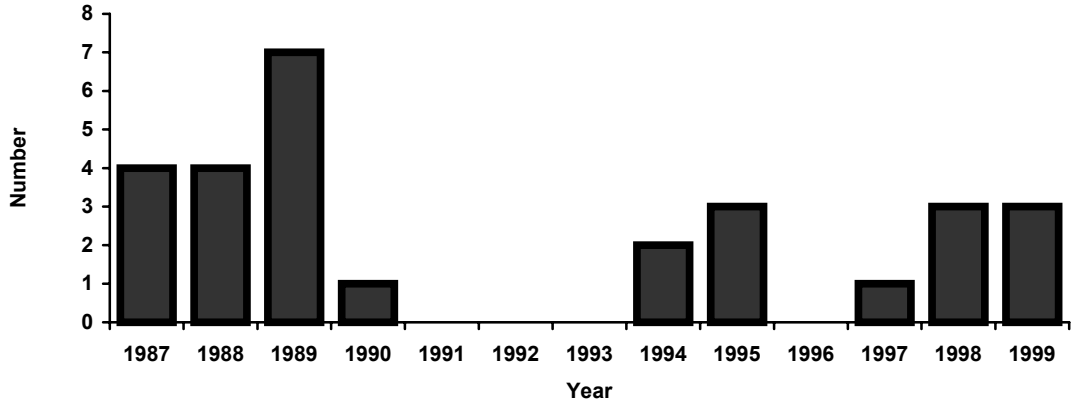
Figure 2. Age distribution of 28 cases of TB, 1987-1999.



The mean number of contacts followed up for the 28 cases was 31 (range 2-81). Additionally, 2 community-wide screens have been performed (since 1992) of both communities, in 1995 and in 1999.

Statistical modelling predicts that persons with a positive Mantoux test who are household contacts of an active case, or who have recently

Figure 1. Notifications of TB in Communities A and B, 1987-1999.



converted from a negative to a positive test have a lifetime risk of active TB of up to 20%.[1] The completion of a course of treatment for LTBI can reduce this risk by approximately 90%.

Community screening in 1999

In December 1999, 3 cases of pulmonary TB were notified, 2 smear positive and 1 culture positive. The finding of 2 unrelated smear positive cases prompted a TB screen of both communities in accordance with NT protocols. Of 385 people screened, 60 had a previously recorded positive Mantoux and required a chest x-ray, and 325 required a Mantoux test. No additional cases of TB were found. However, 35 persons with latent TB were identified as priorities for preventive treatment, 3 because of recent acquisition of infection (Mantoux conversion), and 32 who had pre-existing latent TB but were recent contacts of infectious cases.

Following discussions between Katherine CDC staff and a community elder, the communities became supportive of the need for those with both TB disease, and latent TB infection, to be adequately treated and monitored. This required a person to distribute directly-observed treatment three times each week who was not involved in the day to day running of the clinic. In March 2000 a local enrolled nurse, well known to the communities, was employed on a part-time basis for this purpose and the outcomes have been excellent. Of 35 persons with latent TB, 33 (94%) completed a 6-month course of preventive treatment substantially reducing their risk of active TB in the future. The 3 cases also received curative courses of treatment. In June 2001 funding for this position ceased including the leasing costs of a car to follow patients up.

Extra resources provided for TB control in Communities A and B allowed positive outcomes to occur, and the incidence of active TB in these communities will be reduced in years to come. On the other hand, failure to comprehensively treat LTBI identified among contacts leads to ongoing smouldering endemicity in communities.

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The Australian Immunisation Handbook 8th edition 2003

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The *Australian Immunisation Handbook* 8th Edition 2003 was endorsed by the National Health and Medical Research Council (NHMRC) on 18 September 2003.

The Australian Standard Vaccination Schedule (ASVS), the National Immunisation Program (NIP) and the Northern Territory Childhood Vaccination Schedule

- The Australian Standard Vaccination Schedule (ASVS) is the new recommended schedule in the 8th edition of the *Australian Immunisation Handbook*. It is different to the National Immunisation Program (NIP) and to the NT Childhood Vaccination Schedule. The ASVS lists "best practice" recommendations for vaccinations made by the Australian Technical Advisory Group on Immunisation. Not all the vaccines on the ASVS are funded. The NIP and the NT Childhood Vaccination Schedule list all standard vaccinations funded by the Australian Government and provided free to the Australian population.

What's new for the NT Childhood Vaccination Schedule and the NIP?

- The 18 month dose of diphtheria-tetanus-acellular pertussis (DTPa) has been removed from the NT schedule and the NIP. This is the recommendation made in the 8th Edition Handbook and is effective immediately. Please note that children who have already received DTPa dose 4 at 18 months of age still need another dose of DTPa at 4 years of age. This is because immunity to pertussis wanes with time, and a dose at 18 months of age will not provide protection in late childhood and early adolescence. However, the primary course of DTPa vaccination at 2, 4 and 6 months of age will provide protection against pertussis until 4 years of age, when the next booster is due.
- The 15 year old adult diphtheria and tetanus vaccine (ADT) will be replaced by the adult/ adolescent formulation diphtheria-tetanus-acellular pertussis (dTpa) vaccine (Boostrix™) due at 15-17 years. It is hoped that this additional pertussis immunisation will help prevent the spread of whooping cough. This will be effective from 1 January 2004.
- The list of medical risk factors that predispose children to invasive pneumococcal disease has been expanded. Children with these risk factors are eligible to receive conjugate pneumococcal vaccine funded by the Australian Government under the NIP. These conditions include diabetes, Down's syndrome, premature infants born at less than 28 weeks gestation and cystic fibrosis. The complete new list of medical risk factors is in the 8th Edition Handbook and on the Immunise Australia website at http://immunise.health.gov.au/pneumococcal/index.htm. NT information on medical risk factors for pneumococcal disease will be updated and available in the near future.
- Vaccination requirements for the assessment of immunisation status to determine eligibility for the Maternity Immunisation Allowance and Childcare Benefit will use the NIP schedule. This means that the 4th dose of DTPa at 18 months will not be required for a child to be considered fully immunised. Similarly, the calculation for NT immunisation coverage rates, and for General Practice Immunisation Incentives (GPII) Scheme coverage will not include the 18 month DTPa.
- All vaccinations administered to children up to 7 years of age, including those that are not on the NT Childhood Vaccination Schedule or the NIP should be reported to the NT Childhood Immunisation Database in the usual way. If an immunisation provider reports directly to the Australian Childhood Immunisation Register (ACIR), data on all childhood vaccinations should be submitted to ACIR with a copy forwarded to the NT database. This will ensure that the NT immunisation database and the ACIR maintain a complete immunisation history for each individual child.

What's new on the ASVS?

- The revised ASVS records "best practice" vaccination recommendations. Not all vaccines on the ASVS are funded and parents/ caregivers can purchase these vaccines if they

wish their child to receive them. The ASVS includes several new childhood vaccine recommendations as follows.

- Conjugate pneumococcal vaccine is recommended for all infants at 2, 4 and 6 months with no booster required. The 8th Edition Handbook states that catch-up for low risk children aged 3 –23 months can be considered. The previous recommendations for high risk groups are unchanged, so in the NT the recommendations (and funding) for Indigenous children (booster of 23-valent polysaccharide vaccine at 18 months) and for non-Indigenous children in Central Australia are unchanged.
- Inactivated poliomyelitis vaccine (IPV) is recommended on the ASVS at 2, 4 6 months and 4 years. However, notes to the ASVS state that "IPV is preferred to OPV, subject to the availability of IPV-combination vaccines, but both IPV and OPV are acceptable for use in the ASVS". The 8th Edition also states that "Although both OPV and IPV are appropriate alternatives, providers should inform parents/caregivers that IPV-combination vaccines are preferred because of the proven but extremely rare risk of VAPP following OPV". IPV-combination vaccines are **not** yet available in Australia. If parents/carers elect not to have any polio containing vaccine they should be advised that their Maternity Immunisation Allowance and child care benefit may be affected.
- Varicella vaccine is recommended at 18 months of age, with catch-up at 10-13 years for those without a clinical history of chicken pox or previous varicella vaccination.
- Diphtheria-tetanus-acellular pertussis (dTpa) vaccine for adolescents (as for the NIP above).
- The ASVS also no longer recommends the 18 month dose of DTPa vaccine. This is effective immediately

What's new in the handbook

- Booster doses of 23-valent polysaccharide pneumococcal vaccine are no longer required every 5 years. Instead, 1 to 3 re-vaccinations are recommended, depending on the age of first vaccination and on the risk of disease. NT information adult pneumococcal re-vaccination will be updated and circulated as soon as possible.
- 23-valent polysaccharide pneumococcal vaccine is now recommended for all adults who smoke tobacco.
- The Handbook contains new information on vaccines for Aboriginal and Torres Strait Islander people, international travel, certain occupational groups and other special risk groups.
- Information on vaccine preventable diseases has been updated, as has information on adverse events following immunisation.
- Levels of evidence for new recommendation have been included in the fully reference electronic version of the 8th Edition Handbook. The print version of the 8th Edition will not be fully referenced nor include levels of evidence.

Obtaining the new Handbook

- A PDF version of the 8th Edition Handbook is now available on the Immunise Australia website: http://immunise.health.gov.au
- Printed versions of the 8th Edition Handbook will be distributed by December 2003 to all immunisation providers listed on the Australian Childhood Immunisation Register (ACIR) and Medicare databases.
- Each printed version of the 8th Edition Handbook will be distributed with an interactive CD-ROM version. This interactive version will be available on the Immunise Australia website from October 2003 onwards.
- If you have not received a copy of the new 8th Edition Handbook by the end of the year and you would like one, visit Immunise Australia at http://immunise.health.gov.au, email handbook@health.gov.au or contact the Immunisation Infoline on 1800 671 811.

Further information

- Contact your local Centre for Disease Control.

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Will SARS re-emerge? The epidemic part 2.

Peter Markey, CDC Darwin

Introduction

Between mid-March and mid-July this year, the world was gripped by the global epidemic of Severe Acute Respiratory Syndrome (SARS). Emerging from south-eastern China in late February, SARS spread to North America and Europe within days and triggered an unprecedented global public health response. In Australia, development and implementation of national policies and guidelines was done by the Communicable Diseases Network Australia (CDNA). The Network is constituted under the National Public Health Partnership and consists of representatives from the communicable diseases units of each jurisdiction and other peak bodies. The Northern Territory's Department of Health and Community Services' response has been documented in a previous edition of this bulletin.[1] This article summarises the final stages of the SARS epidemic, describes the process of scaling down the national guidelines and outlines the current strategies in place to help maintain a degree of vigilance in case SARS re-emerges.

The epidemic ends

On the 5th July 2003 the last country was removed from the list of 'SARS affected areas' and WHO declared that transmission had ceased.[2] In the following weeks CDNA developed new guidelines allowing for relaxation of health service and airport screening but still maintaining some baseline vigilance. In the NT, signs at hospitals were removed on 25th of July and nurses remained at airports until 15th August.

The latest WHO table of SARS cases (to the end of July) revealed that there were 8099 cases reported from 32 countries with 774 deaths.* There were 1707 cases in health care workers or 21% of the total and the overall case fatality rate was 9.6%.[3]

In Australia, there were, in the end, only 6 probable cases of SARS reported to WHO. The last of these was an interesting case notified and confirmed only after the epidemic was over. A 26 year old female German tourist, who had stayed at the Metropole Hotel in Hong Kong at the same time as the original index case, flew to Melbourne in early March. She travelled by road up the east coast of NSW to Queensland and became ill during the trip, visiting a GP but not requiring hospitalisation. She had recovered and returned home to Germany before the world-wide alert for SARS was announced by WHO on 14th March. It was only after the serological test for SARS became available some months later that she tested positive. Neither the GP nor her travelling companion showed evidence of exposure to the virus.[4]

The control of the epidemic demonstrated that traditional disease control measures such as isolation, quarantine, community awareness and the co-ordination of government action were still the most effective tools to reduce transmission. It was also notable that countries with limited resources such as Vietnam were more successful at containing the epidemic than other more developed countries.[5] Speculation still remains whether other natural factors assisted in controlling the spread of the virus, such as the changing season or a natural loss of virulence or communicability.

The origin of SARS and the chance of its re-emergence

The most likely explanation of the origin of the SARS coronavirus was that it arose through a mutation in an animal coronavirus. The markets of south-eastern China, where large numbers of multiple species of live animals are sold for food, have often been considered as environments favourable to trans-species migration of viruses. Researchers have recently focussed their attention on several exotic species of mammal sold at the live animal market in Shenzhen. Investigations on animals in the markets revealed coronaviruses similar, but not identical, to the SARS coronavirus, and antibodies to these viruses in market traders.[6]

* The number of total SARS cases reported to WHO has been decreasing presumably due to the results of serological blood tests establishing other diagnoses.

There have been 2 episodes in recent weeks which raised the anxiety of public health officials. Firstly, there was an outbreak of a respiratory illness in a nursing home in Vancouver with high attack rates but low mortality and which was associated with positive SARS coronavirus serology. This turned out to be caused by another coronavirus which cross-reacted with the SARS coronavirus. Secondly, there was a laboratory worker in Singapore who didn't quite fulfil the case definition of SARS but nevertheless had a positive PCR test. It was concluded that this was indeed a case, acquired from cross contamination in the laboratory.

Two possibilities exist for SARS to re-emerge. Even though transmission has ceased and a carrier state does not seem to exist, there may be parts of the world where low rates of unrecognised disease is still occurring. On the other hand, re-emergence of the virus may occur from an animal reservoir. If the decline of SARS transmission has been due to seasonal weather changes we could see a re-emergence of the disease this November. We can only speculate on this until the season arrives.

National preparedness

Following the relaxation of hospital screening protocols and incoming passenger screening it was apparent that there needed to be some continued vigilance for SARS in case of sudden re-emergence of the disease. However, it was also understood that the intensity (and associated cost) of the surveillance should be commensurate with this substantially reduced risk. CDNA drew up and distributed new guidelines in mid-July and these were updated on 15th September taking into account the latest WHO guidelines.[7]

The new recommendations make it clear that it is unlikely, although not impossible, that Australia will be the first place where SARS re-emerges; the most likely scenario being that we will have some warning of the re-emergence. The guideline recommends maintaining surveillance for 'alert' clusters of cases, with a SARS 'alert' being 2 or more health care workers fulfilling the new case definition or hospital-acquired illness in three or more persons.[7] Until SARS re-emerges the emphasis is now on hospital acquired disease and less focussed on travellers. Disease control units are implementing passive surveillance for requests for SARS-coronavirus testing, with laboratories being asked to contact disease control units, either directly, or indirectly through the requesting doctor, on receipt of a request.

The new (public health) case definition does not include a travel condition. It has 4 requirements;

- fever (>=38^{o}C)
- lower respiratory tract symptoms
- X-ray changes
- no alternative diagnosis

The guideline also emphasised that clinicians should remain vigilant and be suspicious about SARS in all cases of atypical pneumonia and seriously consider isolating cases of severe illness without apparent cause.

Outcomes for policy

The SARS epidemic generated some unexpected benefits that have already shaped future policy directions in the public health and disease control environment. Firstly, the outbreak response emphasised the importance of a close relationship between hospital infection control teams and disease control units and illustrated both the similarity and the differences in the work that they do. This has laid the foundation for further projects and policy development in the future.

Secondly, the threat posed by SARS to the borders strengthened the relationship between the Australian Quarantine and Inspection Service (AQIS) and jurisdictional disease control units. While these bodies have, in the past, worked together as required and have had standing arrangements for the management of quarantinable diseases, the fact that they were bureaucratically and physically distant, meant that they rarely met. SARS broke the ice and allowed for a firmer working relationship to be developed for the future.

Finally, SARS arrived during a period in history where countries around the globe are preparing emergency public health responses as part of disaster management, in particular for disasters caused by infectious agents, both natural (the influenza pandemic) and man-made (bioterrorism). The epidemic allowed

jurisdictional disease control units, infection control teams and other agencies to gain unrivalled experience and practice in a real but less hurried setting.

Fortunately, SARS wasn't highly contagious and transmission was mostly limited to close contacts of cases with advanced disease. If it re-emerges it is likely that, with a world now well prepared and sensitive to the condition, the outbreak will be quickly controlled. The real challenge for public health bodies, national governments and world agencies is to be ready for bigger and nastier threats. There seems to be no question that bioterrorism, if it happens, or the influenza pandemic, when it happens, will be SARS 'writ large'.

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The response to SARS: Building infection control capacity in the Pacific

Meredith Hansen-Knarhoi, CNC TB/Leprosy, CDC Darwin

Introduction

"Is there SARS in Fiji?" This was the typical response from colleagues in Darwin when it became known I had been chosen to work in the South Pacific as an infection control nurse as part of the WHO global response to the SARS epidemic. It was a big step from tuberculosis in the Top End, but, on the grounds that the basic principles of isolation and hospital hygiene apply universally, I considered my background in public health and TB work to be more than adequate for the task ahead.

Infection control as a discipline is in its infancy in many countries in the Pacific, due mainly to the competing pressures in building sustainable health systems. Some hospitals have infection control practitioners, but with limited resources and poor access to education their efficacy is limited. Many of Australia's neighbours do not have the luxury of separate isolation rooms, negative pressure facilities (for most, this means a decent draught from the afternoon sea breeze) and functioning ventilators with equipment and staff who know how to use them. Many Pacific Island countries deal with outbreaks of vaccine preventable illness on a day to day basis. Nosocomial infection of dengue occurs due to unscreened wards, lack of mosquito nets and insufficient hospital environmental health controls. Infections like TB, typhoid and suspected measles cases are commonly managed in the general ward environment with no separate isolation facilities.

Many hospital wards do not have sufficient hand washing facilities for staff and soap is a luxury. Laboratories are not able to perform more than basic blood tests, even doing blood cultures is dependent on the hospital having culture medium. Many more complicated immunological analyses are sent to other countries such as New Zealand, Australia and

Japan for testing. Results become available weeks after the event, instead of 24 hours as in Australia! This makes it difficult when determining the cause of a rash illness or early identification of the beginning of an epidemic.

The Pacific reaction to SARS

The Pacific Island countries and territories are comprised of a huge variety of cultures, languages and traditions. Despite the fact that 96% of SARS cases were occurring in the Western Pacific region, countries reacted in differing ways to the crisis on their doorstep. Many instituted health screening at airports very quickly (as early as April 2003), with passengers completing questionnaires prior to arrival. Some chose to ban flights completely from affected countries, some imposed home quarantine to all visitors who had travelled to SARS affected countries in the last 14 days or insisted that returning residents had 14 days quarantine in Australia or New Zealand prior to returning home. The political, economic and diplomatic ramifications of the decisions made by these countries had wide reaching implications. WHO worked closely with the Pacific Public Health Surveillance Network (PPHSN) via Epinet to keep countries up to date with the constantly changing epidemiological scenario. This enabled countries with limited resources to make reasoned decisions when reacting to the crisis. For many countries the risk of importing SARS was relatively low, but the threat to the health system, where there were no local resources available and no functioning infection control committees to help hospitals react appropriately to a suspect SARS case, made it imperative that these fragile systems were protected.

The situation for Fiji was made even more precarious by the imminent arrival, in June, of competitors for the South Pacific Games, a 4 yearly event held in the Pacific, with 2003 being Fiji's turn. Purpose built sports facilities had been constructed and a large economic upturn anticipated. There were 5000 visitors expected from the 22 participating countries with many athletes travelling from their home country via training facilities in South East Asia. The overriding concern was the importation not just of SARS but also diseases like dengue, measles and malaria. WHO worked closely with the organizers to ensure that a surveillance system was in place, with close collaboration and training of hospital and volunteer medical staff in infection control. The Colonial War Memorial Hospital in Suva (the largest hospital in Fiji) set aside a ward from March through to the end of the Games in July specifically to manage possible SARS cases.

There had been several trial runs with SARS suspects in Fiji following the return of the rugby sevens teams from Hong Kong at the height of the SARS epidemic in April. Japan International Cooperation Agency (JICA) in consultation with WHO arranged for donations of N95 masks, gloves, and other infection control materials ensuring that Pacific Island countries had the basic equipment to contain SARS. WHO assisted the various governments in the development of locally appropriate infection control guidelines, ensuring that staff were familiar with the management of a SARS case. In some instances facsimile machines were purchased by individual countries to allow immediate and accurate dissemination of information from the WHO website.

My role in building capacity

The terms of reference for my mission were to assess current infection control measures, assess current local stock levels of personal protective equipment (including the contingency planning for further purchases), assist in rapid development of guidelines (given a resource-poor environment) and enable rapid dissemination of new developments in the SARS epidemic.

When I arrived in mid May 2003, 'unknown' local transmission was occurring in Hong Kong and Canada and cases were still increasing in Taiwan, while Vietnam and Singapore were having success in bringing the epidemic under control. WHO was concerned about the potential spread of SARS to the Pacific, and sought to strengthen the preparedness of the Pacific nations by building their capacity in surveillance and infection control.

During the course of the mission, concentration on the development of local capacity was paramount in SARS preparedness and response. Fundamental to my role was active participation in the Fijian National SARS taskforce (with key

stakeholders including customs, quarantine, tourism and the Ministry of Health), initiation of weekly meetings and coordination with the other Fijian health divisions. Training and meetings with hospital boards were also held in other parts of Fiji; namely Lautoka hospital in the west (the hospital receiving patients from Nadi international airport) and Labasa in the north (the third largest hospital). Assessment and training was also held in Savu Savu, a favorite overseas tourist destination. Staff of the Colonial War Memorial Hospital in Suva had set aside an empty ward specifically to manage potential SARS cases. Weekly infection control meetings were attended as well as infection control training (a total of 8 trainings), culminating in a mock exercise where a 'suspect' passenger was identified at the airport, transferred, admitted and isolated in hospital. Laboratory and radiology staff were included as well as housekeeping staff, as these sectors had often missed out on any infection control training. Suva Private Hospital was also included as it was a key health facility for overseas visitors. The use of scenarios and mock exercises enabled hospital staff to assess their own performance and correct the flaws discovered in their contingency plans. Liaison with the local health promotion unit allowed this activity to be filmed for further training and media purposes for the Ministry of Health.

Another core activity was also compiling bi-weekly faxed updates for Fijian authorities, utilizing data from the WHO website, because many airport and hospital staff did not have access to the internet. This update was also emailed to other island neighbours, particularly those with no local WHO office.

Whilst most of my time was spent in Fiji, I also undertook an assessment and training visit to Tuvalu, Tonga and Samoa. In each country meetings were held with local WHO staff, Ministry of Health officials and national taskforce stakeholders to reinforce the recommendations already communicated by WHO and PPHSN on SARS. The visit also allowed information distribution to other key stakeholders (such as infection control staff), training of hospital staff and inspection of the identified hospital isolation facility.
Recommendations had to be practical and appropriate to the available resources. For example, in Tonga the hospital had only one ventilator and the major preoccupation of medical staff was whether or not they would ventilate a SARS patient. Staff were encouraged to 'walk' through scenarios, to try and anticipate the needs of a SARS case. Creative local solutions were found for both the supply of alcohol handrub – pharmacy concocted its own from a mixture of methylated spirits and chlorhexidine – and for its dispensing – with dispensers recycled from aloe vera moisturizer bottles.

Lessons learnt from the SARS epidemic

1. Travel history is important.

Where have you been? A fundamental lesson learnt the hard way was that ALL 'cough/fever/ pyrexia of unknown origin' admissions to a hospital environment should routinely be screened for recent travel history **as part of the triage history**. This allows for the earliest recognition and isolation of possible SARS cases, minimising further contact with staff and other patients or visitors.

2. Infection control works!

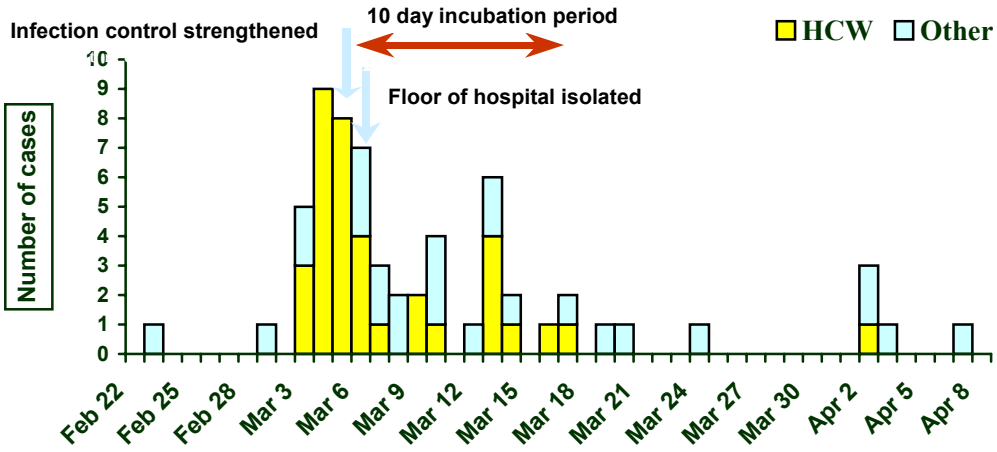
The evidence from the experiences of hospitals in Vietnam, Singapore, Canada and China suggests that SARS can be contained in a hospital environment, where adequate procedures are put in place.[1] The graph illustrates the impact on SARS cases of basic infection control practices such as isolation and decontamination in a Vietnamese hospital where resources were limited. Complacency amongst hospital staff and decreased vigilance on the part of infection control staff ensured, in some cases, that SARS persisted in the hospital environment.[2] Preliminary research has begun on the most important aspects of infection control, shedding light on the key factors in controlling for secondary spread.[3]

A primary part of my role in infection control with WHO was to raise the profile of infection control as a core activity of hospitals.

3. Human resource management is critical

Globally, 21% of SARS cases occurred in health care workers.[4] This means that quality human resource management is vital to the prevention

Figure 1. No of cases by date of onset of symptoms, Viet Nam



Source: Professor Aileen Plant, Team Leader WPRO, Vietnam

of local transmission in the hospital environment. It is essential that hospital staff are fully trained in the use of infection control equipment, that the equipment is available, the appropriate policies and contingency plans are in place and hospital staff caring for SARS patients are supported by hospital management, particularly in staff rostering. In this scenario the potential SARS case can be managed safely and effectively.

Infection control staff in hospitals should ensure that national guidelines on SARS preparedness are followed, with the policies readily adaptable in the event of new developments and findings on the nature of SARS. Practice drills and exercises are fundamental in ensuring staff are familiar with all aspects of the infection control equipment and procedures. Many of these recommendations are available via the Communicable Disease Network Australia, WHO and PPHSN websites.

Summary

There were no cases of SARS or probable SARS in the Pacific. As a result of training and the elevated profile of infection control staff, the future impact of diseases like SARS in countries such as Fiji will be minimised. This is wholly dependent on a number of factors including the sustained political commitment of countries to fighting SARS, ongoing investment in equipment and vigilance on the part of infection control and admitting staff in all health facilities.

Much is still unknown about SARS. The most important lesson learnt was that ALL health facilities have to be trained and prepared for the accidental or deliberate importation of an unidentified infectious agent. It is only with global collaboration and open international communication that diseases like SARS can be contained, a salutary lesson for the inevitable influenza pandemic.

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An audit of malaria management in the Top End

Geoffrey Menzies and Peter Markey, CDC Darwin

Introduction

There has been no local transmission of malaria in the Northern Territory (NT) since 1962, despite the presence of Anopheles mosquitoes and the steady influx of malaria cases into the Top End particularly from tropical South East Asia and Papua New Guinea. Credit for this goes to a well developed surveillance system, strict treatment and isolation measures, the expertise of infectious disease physicians and public health personnel and the diligence of entomologists in keeping mosquito numbers to a minimum in populated areas.

Malaria is a notifiable disease, so that cases are notified to the Centre for Disease Control (CDC) by both clinician, when the diagnosis is suspected, and laboratory, following the result of a positive test. Cases are managed according to locally developed guidelines and are routinely followed up by CDC staff; where appropriate, entomological investigation is done by the Medical Entomology Branch (MEB).

The third edition of the Northern Territory CDC Malaria Guideline[1] was developed in 1997 by the Malaria Working Group (MWG), which included infectious diseases clinicians and staff from CDC, MEB and the Royal Darwin Hospital (RDH) laboratory. It was consistent with the nationally developed therapeutic guidelines.[2] In 1999, a supplementary note made some revisions to the guideline.[3]

As a prelude to the scheduled revision of the guideline this year and as a commitment to continuous quality improvement in the management of malaria, it was thought that a clinical audit of a series of cases be made to assess the adherence to the 1997 guidelines. Previous work in Queensland had noted that as few as 20% to 50% of malaria patients were being treated in accordance with the relevant guideline.[4,5] This paper describes a clinical audit of malaria management at CDC Darwin, conducted by a final-year medical student (GM) under the auspices of the MWG, in March 2003.

Methods

The audit included confirmed malaria cases in Darwin during 2001and 2002, as listed in the notifiable diseases database in CDC. The NT Hospital Morbidity Database (HMD) and data in MEB were also checked to see if there were any cases that were not notified. Cases with no RDH casenote record of malaria were excluded. To conform to the CDC database format, concurrent infections with of multiple *Plasmodium* species were considered to be multiple 'cases'.

The audit considered the question 'Have malaria patients been treated in accordance with the 1997 (amended 1999) NT CDC Malaria Guideline?' Methodology included a review of literature and a brief search for auditing tools used elsewhere which may have been applicable. A meeting of the MWG, consisting of three infectious disease physicians, an epidemiologist, an entomologist and two public health physicians, was arranged and the key performance criteria by which the records were to be assessed were decided by consensus of the group. Data were retrieved from the CDC database, RDH casenotes and the MEB database and analysis performed in Microsoft Excel. Incomplete documentation of management decisions in hospital notes was interpreted as 'not done' where it was thought that the action should have been documented in the record (Questions 3, 5).

There was an emphasis on patient privacy, through de-identifying of data and indexing by Hospital Reference Number (HRN). Data storage was protected through use of password-accessed files. There was also a strong emphasis on documentation of the audit process to allow repeatability in future audits. The findings were later presented to the MWG.

Results

There were 87 cases of malaria notified in 2001-02 in the NT. In 2 cases records could not be found; therefore they were excluded, so the audit examined 85 malaria cases, 61 in 2001 and 24 in

2002, in 70 individual patients. There were 64 cases of *P. vivax* and 21 cases of *P. Falciparum* with 5 cases of concurrent infections. Relapse of *P. vivax* occurred in 5 patients, a total of 10 times (1 with 5 separate admissions). There were no cases of *P. ovale* or *P. malariae*.

The audit considered 14 key questions which are listed in Table 1 along with results. Most procedures were correct in at least 70 percent of cases, with a more detailed analysis providing explanations for some apparent deficiencies. Access to medical records and the integrity of the record affected the completeness of the audit. Cases transferred to a private hospital, treated within the military system, or imprisoned or deported as illegal immigrants had incomplete data (Question 1). Likewise it was often difficult to glean answers from the medical record and, for certain questions, even the notifiable disease data did not allow verification. (Question 2). Infrequently, there were inconsistencies between the database and hospital record. (Questions 12, 13). Only 18 patients had records of a 14-day follow-up appointment. There were no cases found through MEB data or the HMD which had not been notified to CDC.

Discussion

The scope of the audit was confined to CDC and clinical responsibilities and excluded prophylaxis, antimalarial drug choice and other external activities. The emphasis was on procedural compliance, which contributed to patient treatment. There was no formal audit of actual patient outcomes, such as symptom control, cure, recrudescence, outbreaks or mortality. There were, however, no documented fatalities.

Differences between the 1997 NT Malaria Guideline, with or without the 1999 amendment and updates to Therapeutic Guidelines provided varying management recommendations and this was reflected in decreased adherence to the audit questions (Questions 2, 5, 9). For example, a stat dose of primaquine for *P. falciparum* infection, introduced in 1999, was not well adopted. Whether this was due to clinical decision-making or not being aware of the guidelines could not be ascertained as decisions such as these were not consistently documented in casenotes (Questions 10, 11).

The audit gave rise to several recommendations.

- Inconsistencies, which developed between the Malaria Guideline and the associated documents, could be avoided by referring readers to the external document (e.g. Therapeutic Guidelines[6]) and not duplicating their contents within the guidelines, as they may become outdated.
- The possibility for recommendations to be tailored by clinical judgement in special circumstances, should be indicated in the guidelines and discussion with CDC recommended to help clarify and inform these decisions.
- The importance of adequate documentation of patient casenotes with clinical considerations and decisions was emphasised.
- Ongoing dialog between RDH, CDC and MEB as well as with GPs, military and other agencies should continue to ensure adequate coordination and information exchange.
- The continuing quality improvement cycle will involve further audits or the establishment of continual performance monitoring system, with trigger levels for reviews. Data routinely gathered by CDC should facilitate future audits.
- Future audits might examine patient follow-up and the measurement of clinical outcomes, in addition to process compliance.

Overall, the audit showed that the management of malaria in Darwin is consistent with guidelines, with procedural compliance in the majority of cases. Understanding the performance limitations may further improve the care of malaria patients in the Top End.

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Table 1. Analysis of Malaria Guideline procedures as performance against 14 key questions

No	Question	Justification	Data Source	Denom	No	%
1	If the species was *P. falciparum*, was the patient admitted?	Possibility of complications	RDH	16	12	**75**
			CDC	21	15	**71**
2	If the species was *P. falciparum*, was the patient treated with a gametocidal single dose primaquine prior to discharge?	Eradication of gametocytes		16	5*	**31***
3	For all patients, was glucose-6-phosphate dehydrogenase (G6PD) enzyme deficiency checked?	Primaquine is contraindicated in patients with G6PD deficiency.		58	43	**74**
4	If species was *P. vivax*, was there 'no evidence of' *P. falciparum* before discharge?	Check for co-infection – treatments are different.	Not collected			
5	If species was *P. vivax* and management was as an outpatient, was the patient able and advised to remain in screened or air-conditioned accommodation from dusk to dawn?	Patients with *P. vivax* may convalesce at home, if they are isolated from *Anopheles* mosquitoes.		15	7*	**47***
6	If species was *P. vivax*, was the patient notified to CDC for a public health risk assessment prior to decision to treat as an outpatient?	The patient is notified to CDC for assessment of public health risk.		14	10	**71**
7	If species was *P. vivax*, did the ID consultant physician review the patient before discharge?	Consultant review is advised.		15	14	**93**
8	If species was *P. vivax*, and patient resides in malaria receptive area, was 14/7 radical treatment with primaquine recommended?	Prevention of recurrence.	RDH	42	36	**86**
			CDC	64	57	**89**
9	If species was *P. falciparum*, was the patient treated with 3/7 of doxycycline, tetracycline or Fansidar or, as an alternative, mefloquine before discharge?	Check for correct treatment.	Not collected			
10	If species was *P. falciparum*, was the patient's blood slide negative before discharge?	Treatment effectiveness.		16	8*	**50***
11	If species was *P. falciparum*, was the patient well and afebrile for 24/24 before discharge?	Proof of treatment effectiveness		16	11	**69**
12	Was Medical Entomology Branch (MEB) advised of the case?	CDC coordinates with MEB to contain public health risks.	RDH to CDC	56	54	**96**
			CDC to MEB	85	74	**87**
13	What was the timescale in advising Medical Entomology Branch (MEB) of the case?	Prompt MEB notification important for response.	RDH to CDC	54	40	**74**
			CDC to MEB	74	52	**71**
14	Was each element of required information passed to MEB correctly, including gametocytes and travel information, etc?	MEB rely on correct information and travel history.		76	62	**82**

* Likely to be an underestimate due to difficulties with ascertainment

Notes: RDH – based on data obtained from review of RDH patient casenotes
CDC – based on data obtained from CDC malaria database
MEB – based on data obtained from MEB database

Fact sheet

Hand Foot and Mouth Disease

What is Hand, Foot and Mouth Disease?

Hand, Foot and Mouth Disease is an infection caused by the human coxsackievirus, or enterovirus family, in which blisters appear on the hands and feet and inside the mouth. It affects all age groups, but commonly children aged less than 10, and is found all over the world.

How is it spread?

Hand, Foot and Mouth Disease is spread by airborne droplets of respiratory secretions (saliva, sputum), when people sneeze or cough, by direct contact with the fluid in blisters and by the faeces of infected people. It only affects humans, and is not related to the similar sounding "foot and mouth" disease in cattle.

Symptoms of Hand, Foot and Mouth Disease

Within 3 to 5 days of exposure to the virus, the person may have the symptoms of fatigue, loss of energy and appetite, a sore throat and mouth and a mild fever. In a baby, this may present as a decreased interest in feeding.

Several days after these initial symptoms a non-itchy rash develops on the palms, hands, soles of the feet and on the inside of the mouth. Mouth lesions can be widespread and occur on the inside surfaces of the cheeks and gums and sides of the tongue.

How serious is Hand, Foot and Mouth Disease?

For most people with the infection, the rash disappears and they feel better within about a week. Very rarely, the illness can be more serious, so if the person complains of severe headache, or their fever persists and they are not getting better, it is advisable to contact your doctor immediately. Fatal cases have been reported.

Treatment

There is no specific treatment and antibiotics are not helpful.

For most people, symptomatic relief with fluids, paracetamol and rest is all that is required.

How can Hand, Foot and Mouth Disease be controlled?

There is no vaccine for Hand, Foot and Mouth Disease. On recovery from the illness, a person will develop immunity against the one specific type of virus that caused the infection. However, as there are several viruses which can cause Hand, Foot and Mouth Disease, further episodes of the disease are possible from a different virus.

Promoting good hygiene is the best way to stop the spread of the virus. Therefore, practise frequent hand washing, especially when changing nappies and other stool-soiled material. Wash toys contaminated with saliva, and cover the mouth when sneezing and coughing.

People with Hand, Foot and Mouth Disease are infectious during the acute stage of the illness and perhaps longer, since these viruses persist in stools for several weeks after recovery.

The blisters should be allowed to dry naturally, and should not be pierced as the fluid inside them is infectious.

Children with Hand, Foot and Mouth Disease should be excluded from school, preschool and childcare until all blisters have dried.

Further fact sheets and treatment protocols are available at:
http://www.nt.gov.au/health/cdc/fact_sheets/fact.shtml

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Gastroenteritis outbreak due to *Staphylococcus aureus*

Karen Dempsey (CDC), Kelly Monaghan (Environmental health)

Background

At 10am on the 1st of August 2003 a rural health nurse alerted the Darwin Centre for Disease Control (CDC) to an outbreak of apparent food poisoning. She reported that 5 (3 children and 2 adults) of 11 passengers travelling on a bus to a destination in Arnhem Land had just presented to the community health clinic for treatment of severe abdominal cramps, prostration, vomiting and diarrhoea. Foodborne intoxication (food poisoning caused by bacterial toxins) was suspected because of the abrupt onset of explosive gastro-enteric symptoms in a group of people within 2 hours of consuming breakfast at a roadhouse.

Initial investigations

The patients were asked to recall what they had eaten at the roadhouse, what they had eaten on the bus prior to arrival and what they had eaten the previous night. Attempts to gain an accurate food history were challenging in view of the level of prostration in the patients and the clinical imperative to initiate fluid replacement therapy as soon as possible. Consequently the following history transpired via the non-ill passengers. The bus picked up the 11 passengers between 4 and 5am that morning to travel back to Arnhem Land. None had eaten breakfast prior to leaving so the bus stopped at the roadhouse for breakfast around 7-30am. Of the eleven passengers, 7 had bacon and egg sandwiches while the remaining passengers ate pies and chips. One and a half hours later 1 of the 7, a 3 year old child, became violently ill with severe abdominal cramps, explosive vomiting and diarrhoea. Within 30 minutes 4 more of the 7 became ill with similar symptoms triggering the bus driver to seek medical assistance at the nearest health facility. All passengers, including those who became ill, had 'empty stomachs' before consuming food at the roadhouse, a fact that was reiterated repeatedly. Food eaten on the previous evening was not implicated as the sick children and adults had each eaten different meals. Since none of the passengers who ate pies and chips were ill, the bacon and egg sandwiches appeared to be the prime suspect.

The 5 patients were given intravenous fluids at the community health clinic. Despite this intervention 4 of the 5 (2 children and 2 adults) were still unwell by late afternoon prompting clinic staff to organise their evacuation by Aerial Medical Services to the Royal Darwin Hospital. Specimens of vomitus and stool were collected and sent with the patients to a Darwin pathology laboratory.

Public health interventions

The implication that a roadhouse was possibly involved in a food poisoning incident meant that swift instigation of a public health investigation was essential. The manager of the Darwin Environmental Health Branch immediately allocated an environmental health officer (EHO) to facilitate interventions in conjunction with CDC personnel.

The initial investigation involved contacting health clinics and bus companies and the manager/owner of the roadhouse. The 3 clinics in adjacent communities were contacted and asked to inform CDC of any presentations of foodborne gastroenteritis. All commercial tourist bus companies who regularly stop for breakfast at the roadhouse were asked to get in touch with their drivers for details of ill passengers. The manager/owner of the roadhouse was asked to cease selling bacon and egg sandwiches and hold any left over sandwiches for testing. He was informed that personnel from environmental health and CDC would be visiting later that day to conduct an investigation involving inspection of the roadhouse and an examination of the food handler(s).

Results of initial investigations

There were no presentations of foodborne gastroenteritis to any clinic that day and there were no incidents of illness reported among passengers of the commercial bus companies

A Hazard Analysis of Critical Control Points (HACCP) auditing approach was used in investigating the process of producing a bacon and egg sandwich. Hazard analysis involves the

identification of ingredients and products that might have a pronounced effect on food safety.[1,2] The audit followed the process of an egg and bacon sandwich from food transport, reception and cold storage through to preparation, cooking and handling of the final product before sale. The audit also included an inspection of the roadhouse in terms of cleanliness and hygiene as well as structural aspects that may involve a risk to food safety and could contribute to, or be the cause of, the outbreak.

All hazards identified within this process were being controlled; food transport and storage temperatures were below 5°C for cold food storage and above 60°C for hot-bain maire foods.

The main preparation area was found to be generally clean and uncluttered however there were several areas that were identified for improvement. There was evidence to suggest that the hand wash basin was not being utilised even though it was well located, equipped with soap and handtowels, and the staff were aware that they should be washing their hands frequently. The investigation also revealed that a staff member working in the food roadhouse had diarrhoea. The staff member was not directly involved in preparing the bacon and egg sandwich, however as a precautionary measure and in line with the Food Safety Standards he was requested to perform other duties.[3] These duties included those not associated with food handling, preparation and storage and were required until the diarrhoea had ceased.

Information and advice on food handling practices and temperature control and monitoring was provided to the proprietors and staff at the time of the investigation. The proprietor was also provided with copies of the Australia New Zealand Food Authority, Food Safety Standards.[3]

Foods that are frequently involved in staphylococcal food poisoning are those that require considerable handling during preparation and that are kept heated in temperature less than 60 degrees for prolonged periods.[4] Furthermore, in classical staphylococcal foodborne outbreaks purulent skin lesions may be found on the hands and/or face of food handlers.[5]

The bacon and eggs went through a 'cook' process before being assembled and were not handled with bare hands at any time during the preparation process. In addition none of the food handlers had lesions on their hands or faces. A printout of the morning's takings was examined to determine the time between preparation and consumption. This information revealed a rapid turnover of sandwiches and that the average length of stay in the bain-marie was 30 minutes.

In summary the investigation did not identify any reason to implicate the roadhouse as the source of infection and it looked as if that another source might be involved. In view of this we decided to reinterview the patients evacuated to RDH.

Follow-up investigation

Later that evening the 4 patients who had been evacuated to RDH had recovered and were able to provide more history regarding their food intake. They recalled that a small take-away meal of fried rice and beef and black bean sauce was taken onto the bus that morning. It was at least 24 hours old and had not been refrigerated since purchase the previous day. The 4 patients plus 1 other passenger who subsequently became ill shared the meal prior to stopping at the roadhouse for breakfast. The other passengers did not eat on the bus nor were they aware that food was eaten, hence the repeated denial of food consumption other than that eaten at the roadhouse. Since there was no left over rice and the source was not established in order to test food handlers the diagnosis of food intoxication hinged on the detection of *Bacillus cereus* (*B. cereus*) or *Staphylococcus aureus* (*S. aureus*) in the faecal and/or vomitus specimens.

Results

No organisms were isolated in the faeces and *B. cereus* was not isolated in the vomitus. A very heavy growth of *S. aureus* grew in the vomitus fitting the clinical picture of staphylococcal food intoxication. For further confirmation the vomitus specimens were sent for toxin testing but staphylococcus enterotoxin was not detected, possibly due to delay in specimens reaching the appropriate laboratory.

Discussion

From the outset this outbreak had all the hallmarks of food poisoning. The short incubation period, the sudden onset of violent gastro-enteric symptoms in a group of people, and the self limiting nature of the illness all pointed to the consumption of a food containing a toxin.

S. aureus and *B. cereus* both produce pre-formed toxin causing food poisoning syndromes which include nausea, vomiting, abdominal cramps and diarrhoea.[5,6] In addition fried rice is frequently implicated in *B. cereus* food poisoning outbreaks, particularly if left in warm conditions for prolonged periods.

Public health interventions, including an investigation of the roadhouse were based on the presumption that bacon and egg sandwiches were the source, and were initiated early to prevent further illness. The investigation failed to implicate the bacon and egg sandwiches as the source of infection and a more plausible source ensued implicating a meal of fried rice and beef and black bean sauce eaten shortly before the bacon and egg sandwiches. The likelihood that the meal was the source of *S.aureus* is borne out by 2 factors; the meal was not refrigerated from the time of purchase until consumption the following day, at least 24 hours later. Secondly infected lesions were observed on the hands of both affected children by the clinic staff. It is possible that the children handled the meal during the previous evening causing the *S. aureus* to be present in the meal. The combination of warm tropical conditions and the prolonged delay between purchase and consumption would have ensured the proliferation of S. *aureus* to the concentration required to produce a toxin dose.

The clinical diagnosis was supported by the isolation of *S.aureus* in the vomitus specimens but could not be confirmed since no leftover food was available for entertoxin testing. Vomitus is not routinely collected for testing in gastroenteritis outbreaks, however in the case of suspected food intoxication recommended specimens include faeces and vomitus from the patient(s) and leftover food. Additional specimens including nasal and wound swabs from food handlers may also be taken if *S. aureus* is suspected. Staphylococcal food intoxication is confirmed if *S.aureus* is isolated from the vomitus or faeces and the phage type matches that isolated from nasal orifice or wound lesion swabs; where more than 10^5 organisms per gram are isolated from the food; or if staphylococcal enterotoxin is demonstrated in the food. Bacillus food intoxication is confirmed if *B.cereus* is isolated from the vomitus or faeces or more than 10^5 organisms are isolated in the food.[5]

S.aureus is not a common cause of foodborne disease outbreaks in Australia (7% of foodborne disease outbreaks[8]). The incidence of *S.aureus* outbreaks in the Northern Territory (NT) is unknown but likely to be significant given the high rate of skin infections and the favourable climatic conditions.

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Quarterly notifiable disease surveillance

In this edition of the bulletin we are introducing a new way of illustrating notifiable disease data. In addition to the usual table, we have displayed a selection of notifiable diseases from the April-June quarter on a bar graph. Each bar represents the ratio of the number of cases of the disease in the last quarter to the mean number of cases in the same quarter for the preceding 4 years. This allows a comparison of the actual number of cases with the number you might expect for that quarter based on previous years' experience. For example the number of cases of malaria notified in the second quarter this year was 17, whereas the mean number for the second quarter for the 4 previous years was 13. Therefore the ratio of actual to expected was 17/13 = 1.31.

We have also employed a method to establish whether the difference between actual and expected is "significant". This is by seeing whether the number of cases observed in the last quarter falls outside a range of 2 standard deviations either side of the mean of the previous four years. This method is very similar to that used by the Morbidity and Mortality Weekly Review in its regular reporting. However, it is only one of many, at times complicated methods to examine the significance of a difference in case numbers.

We hope that trialing this method will allow us to gain a greater understanding of the trends we observe in notifiable disease occurrence. We would welcome feedback from readers of the *Bulletin* on this new system of reporting and analysis.

In examining the April-June data there are a number of diseases worthy of some comment. There were four diseases that fell outside the two standard deviation limit: trichomonas and tuberculosis showed reductions while chlamydial conjunctivitis, and hepatitis C showed increases. When observing such differences we must explore whether they represent true changes in the occurrence of disease or can be explained by other factors.

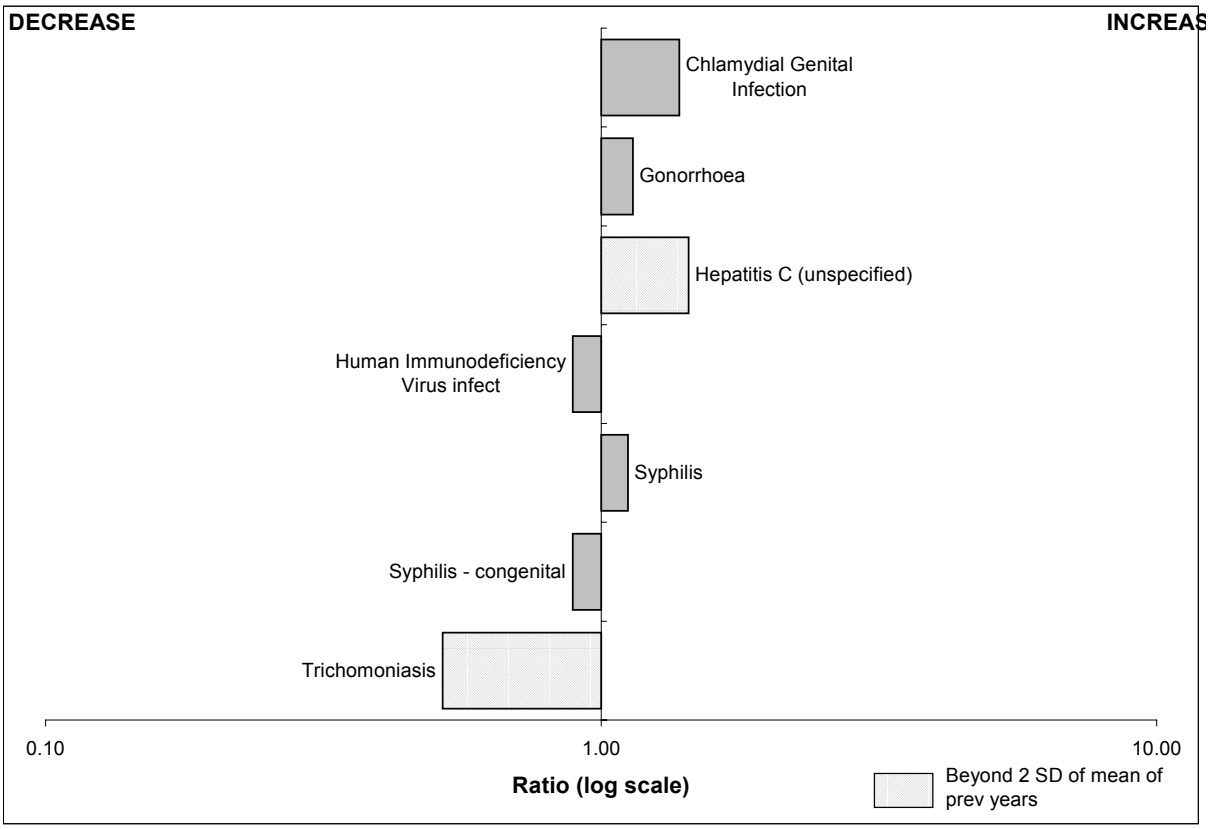
Chlamydial conjunctivitis: Notifications of this have increased each year over the past 3-4 years. The vast majority is in young children in remote communities. Reports from those communities, the eye health committee and pathology laboratories indicate that the number of PCR tests for chlamydia taken from eyes has dramatically increased during the same period. Many practitioners are now doing PCR tests on kids with possible trachoma or conjunctivitis. The overall impression is that the increase is due to extra testing and not an increase in trachoma.

Hepatitis C: It is likely that this increase is related to increased testing. Since the Opiate Pharmacotherapy Program (buprenorphine and methadone program) began in September 2002 in both Darwin and Alice Springs over 100 people presenting to that program have been tested for hepatitis C approximately 70% of whom were positive. During this quarter 11 of the 60 cases detected in the NT came from the Opiate Pharmacotherapy Programs. Without these 11 cases the number for this quarter would not have been in excess that expected from previous years.

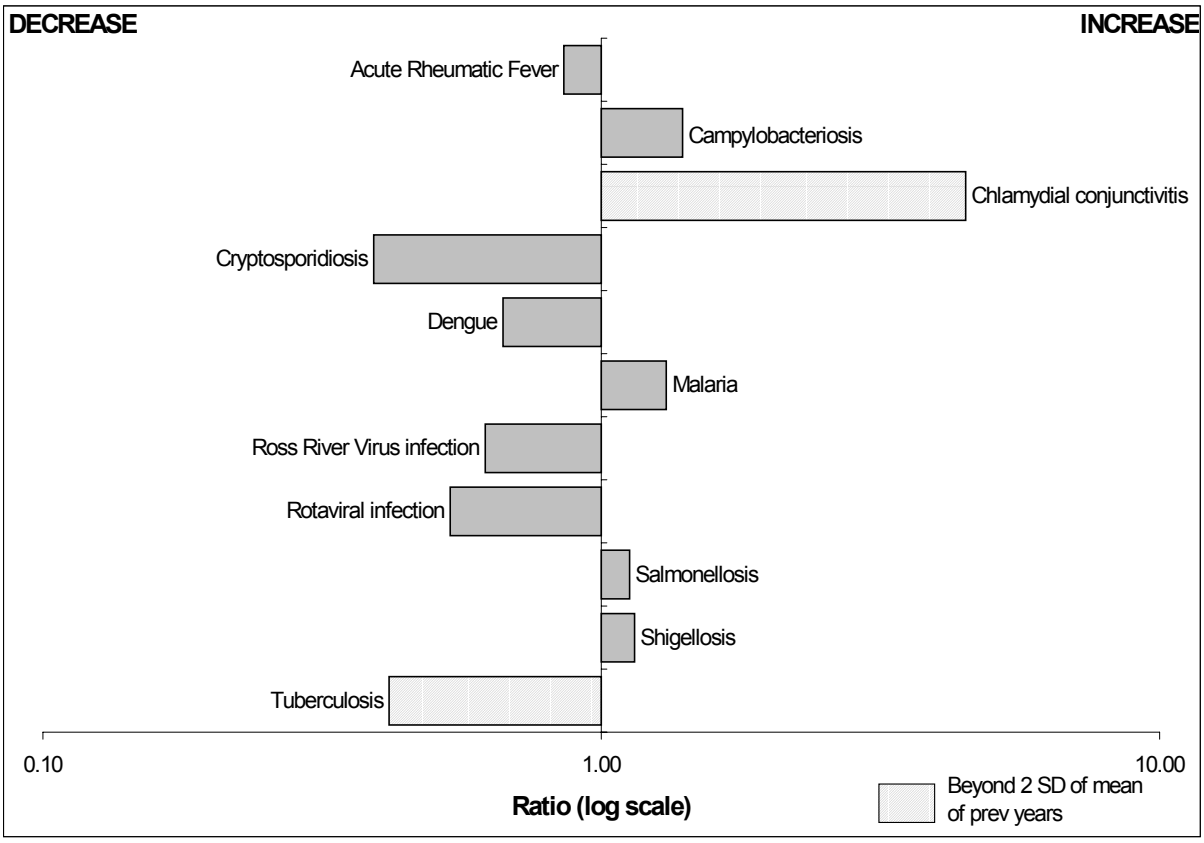
Trichomonas: Notifications of trichomonas for this quarter have declined in the Darwin, East Arnhem and Katherine regions particularly since 2001. They have remained stable in Alice and the Barkly. Detection of STIs, particularly trichomonas which is very often asymptomatic, is very dependent on the amount of testing done. We will need to explore whether the amount of testing done and proportion of tests that were positive in these regions has changed before drawing any conclusions about the change in notifications.

Tuberculosis: Not only does this quarter show a reduction, but the year to date total is down compared to previous years with only 19 notifications compared to 40 last year, 35 in 2001 and 60 in the year 2000. Nathan Zweck of the TB program believes we may be starting to reap the benefits of seriously applying latent TB treatment to infected persons. He feels there has been significant improvements in screening for latent infection and in people completing isoniazid treatment in the past few years.

Change in April June quarter 2003 compared to mean of corresponding quarter of previous four years: sexually transmitted infections and blood borne viruses



Change in April June quarter 2003 compared to mean of corresponding quarter of previous four years: selected diseases



Enteric diseases in the Northern Territory April – June 2003

Karen Dempsey, CDC Darwin

Gastroenteritis outbreaks

There were no confirmed foodborne gastroenteritis reported to Northern Territory (NT) Centre for Disease Control (CDC) during the April-June 2003 quarter.

A large national outbreak of hepatitis A was linked to the NT in June 2003. Hepatitis A was diagnosed in 29 interstate residents within 24 to 42 days of participating in a 4-day youth camp in Central Australia.

An outbreak of non-foodborne gastroenteritis occurred in a Darwin Childcare Centre. The attack rate was high with 12 out of 16 children under 2 years of age becoming ill with gastroenteritis within 24 to 48 hours of exposure to a child vomiting in the nursery. The symptoms were consistent with a viral infection; vomiting was the dominant symptom followed by diarrhoea. In addition the attack rate was high in older siblings and adults with 15 people (3 siblings and 12 parents) became symptomatic following exposure. Stool testing identified the causative organism as a calicivirus, which was subsequently typed as norovirus.

Campylobacteriosis

During the April-June 2003 quarter there were 87 *Campylobacter* notifications (45 males and 42 females). The median age was 1 year reflecting the high proportion of cases among the 0 to 4-year old age group.

The number of *Campylobacter* notifications reported this quarter exceeded the number reported during the same period in 2002. This is consistent with the trend observed during the first quarter of this year, although for females only. Female notifications were 3 times higher (14 in 2002 and 42 in 2003) and male notifications were similar for both periods (41 in 2002 and 45 in 2003). Indigenous notifications increased markedly (20 in 2002 and 51 in 2003) while non-Indigenous cases remained the same. Alice Springs region, including Alice Springs City and rural communities, reported the largest rise in C*ampylobacter* notifications increasing from 18 to 48. It is not known why this region has experienced such a large increase and the cause is currently undergoing investigation.

Cryptosporidiosis

There were 22 *Cryptosporidium* notifications reported during the April-June 2003 quarter. All were sporadic cases and none were associated with swimming pools or child-care centres.

The number of *Cryptosporidium* notifications for this quarter were fewer in number compared to the same period in 2002 (40 in 2002 and 22 in 2003).

Hepatitis A

Only 6 hepatitis A notifications reported during the April-June 2003 quarter, mostly in the Alice Springs region. This was far fewer than the number notified during the first quarter of this year (18 notifications). None of the 4 NT cases detected during June were linked to the previously described nation-wide outbreak.

The number of hepatitis A notifications for this quarter were slightly less than recorded for same period in 2002 (9 in 2002 and 6 in 2003).

Rotavirus

There were 125 rotavirus notifications (77 males and 48 females) significantly higher than the previous quarter (12 notifications). The majority of cases were Indigenous children aged less than 5 years of age living in the Alice Springs region.

The NT has experienced biennial epidemics of rotavirus gastroenteritis since 1995 and it was anticipated that the 2003 winter incidence would match this trend. This was not the case and the number of notifications recorded this quarter were considerably less than that recorded for the same period in 2002 (262 in 2002 and 125 in 2003). The decline was observed in all regions.

Since peak activity during past epidemic years has occurred during April, June, July and August it is possible that the peak is late this year and may occur during the ensuing month.

Salmonellosis

There were 95 *Salmonella* notifications reported during the April-June 2003 quarter. More males than females were reported (51 males and 44 females) and the median age was 2 years reflecting the high incidence of salmonellosis among children aged less than 5 years of age.

Salmonella Ball (14 notifications) was the dominant serovar followed by Salmonella Saintpaul (9 notifications). One notification of Salmonella Paratyphi B var Java was reported in a 21-month-old Indigenous child living in an Arnhem Land community. There were no notifications of Salmonella Enteritidis.

The number of *Salmonella* notifications was similar during this quarter to the same period in 2002. Male notifications were down slightly (57 in 2002 and 51 in 2003) while notifications among females increased by ten (34 in 2002 and 44 in 2003). There was no significant change in the number of notifications by Indigenous status over the two periods. For most regions the incidence was relatively unchanged with the exception of Alice Springs region where there was a slight reduction.

Shigellosis

During the April-June 2003 quarter there were 35 *Shigella* notifications (13 males and 22 females). The majority of cases were children aged less than 9 years of age and the median age was 7 years.

The number of *Shigella* notifications reported this quarter exceeded the number reported during the same period in 2002. Female notifications were three times higher (6 in 2002 and 22 in 2003) and male notifications were similar for both periods (16 in 2002 and 13 in 2003). The majority were Indigenous and there was little change in the ethnic distribution of cases over the two periods. Alice Springs region, including Alice Springs City and rural communities, reported the largest increase in *Shigella* notifications, increasing from 8 during the second quarter of 2002 to 24 during the second quarter of 2003.

Other enteric diseases

There were no notifications of yersinia, listeria or haemolytic uraemic syndrome during the April-June 2003 quarter.

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NT Malaria notifications April - June 2003

Merv Fairley, CDC, Darwin

There were 17 notifications of malaria received for the second 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
10	West Guinea	refugee	P.falciparum	no
1	East Timor	working	P.falciparum	no
1	East Timor	working	P vivax	yes
1	? South Africa	holiday	P.falciparum	yes
1	Uganda	refugee	P.falciparum	no
2	PNG	holiday	P.falciparum	no
1	PNG	holiday	P vivax	yes

NT NOTIFICATIONS OF DISEASES BY ONSET DATE & DISTRICTS 1 APRIL TO 30 JUNE 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	0	0	0	1	1	1
Acute Rheumatic Fever	3	8	1	0	3	7	0	2	2	1	9	18
Adverse Event after Immunisation	0	1	0	0	4	4	3	2	0	0	7	7
Barmah Forest Virus infection	1	0	0	0	2	5	1	0	0	0	4	5
Campylobacteriosis	48	18	4	2	29	29	3	0	3	6	87	55
Chlamydial conjunctivitis	25	0	18	0	45	26	0	9	1	0	89	35
Chlamydial Genital Infection	201	175	2	8	159	129	23	27	25	40	410	379
Cryptosporidiosis	18	7	0	0	3	25	1	5	0	3	22	40
Dengue	0	0	0	0	6	6	1	0	0	2	7	8
Donovanosis	0	1	0	0	0	1	0	0	0	1	0	3
Gonococcal conjunctivitis	0	1	0	0	2	0	0	0	1	0	3	1
Gonococcal ophthalmic neonatorum	0	0	1	0	0	0	0	0	0	0	1	0
Gonorrhoea	223	198	4	13	122	120	18	19	35	42	402	392
Haemophilus influenzae (not type b)	0	0	0	0	0	0	1	0	0	0	1	0
Haemophilus influenzae (type b)	1	1	0	0	0	0	0	0	0	0	1	1
Hepatitis A	4	2	0	0	1	3	0	0	1	4	6	9
Hepatitis B (incident)	1	0	0	2	3	1	0	0	1	1	5	4
Hepatitis C (unspecified)	12	2	1	1	42	37	1	1	4	2	60	43
Human Immunodeficiency Virus infect	0	0	0	0	2	3	0	0	0	0	2	3
Human T-Cell Lymphotrophic Virus	5	6	0	0	2	0	0	0	2	0	9	6
Influenza	0	1	0	0	1	0	0	0	0	0	1	1
Leptospirosis	0	0	0	0	1	1	0	0	0	0	1	1
Malaria	2	1	0	0	15	4	0	0	0	1	17	6
Melioidosis	0	0	0	1	1	2	0	0	0	0	1	3
Meningococcal infection	4	0	0	0	0	2	0	2	0	0	4	4
Mumps	0	0	0	0	0	1	0	0	0	0	0	1
Pertussis	0	0	0	0	0	6	0	0	0	0	0	6
Pneumococcal Disease (Invasive)	10	9	4	0	1	5	1	0	3	1	19	15
Ross River Virus infection	0	1	4	1	5	9	0	0	3	1	12	12
Rotaviral infection	58	97	2	17	25	83	11	9	30	56	126	262
Salmonellosis	18	24	6	1	54	48	5	3	12	15	95	91
Shigellosis	24	8	4	4	4	6	1	2	2	2	35	22
Syphilis	58	73	1	2	13	23	7	3	16	5	95	106
Syphilis - congenital	2	7	0	0	0	0	0	0	0	0	2	7
Trichomoniasis	62	56	2	5	32	76	16	27	10	33	122	197
Tuberculosis	0	1	0	0	4	5	0	0	1	5	5	11
Typhus (scrub)	0	0	0	0	1	0	0	0	0	0	1	0
Total	781	698	54	57	582	667	93	111	152	222	1,662	1,755

Points to note regarding notifications:

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

NOTIFIED CASES OF VACCINE PREVENTABLE DISEASES IN THE NT BY ONSET DATE 1 APRIL TO 30 JUNE 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	1	1	1	1
Hepatitis B	5	4	1	0
Measles	0	0	0	0
Mumps	0	0	0	0
Pertussis	0	6	0	2
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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Disease control staff updates

CDC

Darwin: Vicki Krause has taken sabbatical leave until January 2004 with **Peter Markey** acting Director of Disease Control during this time.

Alice Springs: Jackie Glennon has moved to Noosa, with **Alex Brown** covering her position part time until it is filled.

Surveillance

Darwin: Steven Skov is working part time as surveillance medical officer.

Immunisation

Alice Springs: Dyan Kelaart is now working as a remote area nurse at Yuendumu and **Helen Tindall** has returned to the Public Health Nurse position after her time overseas. A short term immunisation catch up program is being completed by **Beth Rowan**.

Katherine: Nancy Nyberg has moved to fill an audiometry nurse position with **Ruth Baillie** joining the team from Aerial Medical Services.

AIDS/STD

Darwin: Brian Hughes has joined The AIDS/ STD Program with the departure of **Sarah Huffam**. Brian has Previously worked at Sydney Sexual Health Centre and the Albion Street Centre in Sydney. He is a dual fellow of the Royal Australasian College of Physicians (Infectious Diseases) and the Australasian College of Sexual Health Physicians. The coordination position for the remote sexual health team has been filled by **Sandra Downing** on return from her overseas work with a polio eradication program in Ethiopia and an obstetrics program in Sri Lanka. **Di Clare** and **Darryl Thomas** are moving to the Aboriginal Medical Alliance Service Northern Territory (AMSANT) working with health boards and councils promoting HIV/AIDS awareness. **Andrew Seymour** is moving on from the administration position in October.

Katherine: Greg Henschke has accepted a position with Katherine West Health Board as programs manager to develop annual plans, assist with implementing programs and liason with other health services,

Non Communicable Diseases

Darwin: Tarun Weeramanthri will be leaving as Community Physician on returning from his leave to fill the Principal Medical Advisor position. **Angela Kelly** has returned as the Rheumatic Heart Disease project officer for 6 months developing a model of best practice 'tool kit' for the TopEnd of Australia and will be presenting workshops in the NT, Queensland and Western Australia. A Change of paediatric registrars brings **Rhys Parry** with **Alison Cupitt** returning to RDH. **Brad Palmer** will be taking leave to work overseas leaving the Community Child Health position to be filled.

Alice Springs: Lynette Purton is moving to a public health nurse position in Broome leaving the Rheumatic Heart Disease Program.

TB/Leprosy

Alice Springs: Jeannette Berthelsen's return to the Emergency Department has brought **Minnie Blythman** as Public Health Nurse TB/Leprosy. Minnie previously worked with the Centre for Remote Health. Recruitment to this position is currently underway.

Katherine: Maria Chandler is returning to a new position with clinic administration. **Kerry Bettison** joins the team as administration support and **Margaret Richards** will be leaving in the next month.

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Centre for Disease Control Conference

The CDC conference planned for the 28th-30th October 2003 has been cancelled. We hope to be able to convene another conference next year. Apologies to anyone that may have been inconvenienced.