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NORTHERN TERRITORY

THE NORTHERN TERRITORY
DISEASE CONTROL
BULLETIN

Vol. 10, No. 1, March 2003 ISSN 1440-883X

Central Australian Rheumatic Heart Disease Control Program

A report to the Commonwealth November 2002

Prepared by Alex Brown, Lynette Purton, Gabrielle Schaeffer, Gavin Wheaton and Andrew White for the Central Australian RHD Steering Committee

Summary

Acute Rheumatic Fever (ARF) and Rheumatic Heart Disease (RHD) are important causes of morbidity and mortality in the Australian Aboriginal population in Central and Northern Australia with rates among the highest recorded.

The Central Australian RHD program operates in line with World Health Organisation recommendations (1994) for regions with high incidence. The program covers a population of 50,000 and collects epidemiological data, assists with client follow-up, provides client, family and health staff education and has a focus on improving secondary prophylaxis. The report outlines achievements to date.

A centralised database has been established with appropriate security and ethical approval. There are currently 445 active clients. Data related to diagnosis, hospitalisations, compliance with prophylactic benzathine penicillin, clinical progress, surgery and mortality has been collected and analysed. A number of strategies have been developed to update database information. Retrospective hospital chart audits have been undertaken for all clients.

Active surveillance has **improved the diagnosis and timeliness of notifications** of ARF and recurrences. The percentage of known cases that are notified increased from 23% in 1995 to 96% in 2002.

Recurrences represented between 23% and 58% of ARF notifications during the years 1995-2002. These are preventable by secondary prophylaxis. Incident

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cases of ARF have been between 19 and 33 in these years with an apparent increasing trend. When broken down by age the increase appears to have been highest in the 15-24 year group. This may represent an increase in incidence and/or improvement in ascertainment. The prevalence of RHD in the Central Australian Aboriginal population is 12.5 per 1000.

Adherence to secondary prophylaxis has been monitored by setting up 4 communities as sentinel sites. In these communities overall compliance was 55% in 2000 rising to 68% in 2002. Some barriers to compliance have been identified.

Education of clients and primary health care staff has occurred in a variety of settings. The aim is to improve understanding and awareness of ARF / RHD to improve management.

Consultation with stakeholders including Aboriginal Community Controlled Health Organisations has occurred and agreements to share information and resources have been made. Liaison between primary, secondary and tertiary health services has occurred in order to **facilitate best practice management of ARF/RHD.**

Future planned developments include:

- Strategies to follow-up high risk clients (there are 159 individuals with moderate or severe valvular disease). These clients will be given priority and intensive work. Some have previously been lost to follow up.
- Further educational resources targeted to various stages of the disease process, which are culturally appropriate, will be developed.
- Work to improve management of clients with RHD during pregnancy.
- Development of multidisciplinary outreach services to visit remote communities.

Introduction

"Acute Rheumatic Fever (ARF) and Rheumatic Heart Disease (RHD) continue to be important causes of morbidity and mortality in the Aboriginal population of central and northern Australia, as well as in Maori and Pacific Islanders in New Zealand, despite access to high-quality health services. In contrast, rheumatic fever is now extremely rare in non-Aboriginal children throughout Australia"[1]

The establishment of the Central Australian Rheumatic Heart Disease Program has been in line with World Health Organisation recommendations (1994) for regions with a high incidence of RHD.

The recommendations include:

- The establishment of a register based control program
- A focus on health education
- An emphasis on secondary prophylaxis
- A collection of epidemiological data

The Central Australian program covers a huge area of the Northern Territory, which encompasses the Alice Springs and Barkly regions, the Anangu-Pitjantjatjara lands in South Australia, the Ngaanyatjarra lands in Western Australia, and extends to the Queensland border. It services almost 52,000 people, 46% of whom are Aboriginal, and covers an area of almost 1,000,000sq kilometres.

An initial program was temporarily funded for 12 months, from November 2000 until September 2001. In that time the Coordinator established the register and set up lines of communication with service providers.

The program became fully operational in June 2002, with the commencement of two part time coordinators.

Objectives

A) The establishment and maintenance of a centralised data-base.

Achievements:

The database was established on Microsoft Access.

The register currently has 445 clients who have been diagnosed with Acute Rheumatic Fever (ARF) or Rheumatic Heart Disease (RHD). A further 66 clients that have been previously identified and added to the register have been removed following their death.

The database contains the following information:

- Identification and demographic data
- Hospital separation data

- Diagnosis
- Compliance with prophylactic benzathine penicillin
- Mortality
- Updated Medical Review, Condition Report and Clinical Progress
- Surgery for RHD
- Recommended future care plans

In addition, the database has recall facilities in order to send reminders and recall lists to health care providers. The database is contained on a secure network with strictly limited access, is confidential and is subject to the Department of Health & Community Services information privacy act.

Approval for the program, database and dissemination of findings was obtained from the Central Australian Human Research Ethics Committee prior to the commencement of the initial program in 1999.

Client Identification:

The identification of clients for the data-base is sourced from numerous places including hospital discharge coding, cardiology clinic lists and letters, specialists review letters, notification records, primary and secondary health care staff, paediatric liaison team communication and echocardiogram records.

Information Updates:

To enable regular update of information on clients on the database, various steps were taken to ensure information flow to the coordinator.

Steps included:

- Copying of review letters from specialists in primary, secondary and tertiary health care.
- Utilising hospital information systems.
- Echocardiography reports.
- Provision of monthly hospital death notifications allowing those names to be removed from the 'current client' list thereby preventing 'deceased persons' names appearing on subsequent recall lists.

Retrospective Chart Review:

Retrospective chart audits have been undertaken on every client identified on the database. This has been done to collate individual data on their diagnosis, recurrences, follow up, and to develop urgent recall lists for high-risk clients.

Community Visits:

Remote community visits are used as an opportunity to retrospectively audit case notes and clinical histories, assess penicillin compliance and audit clinical care.

B) To improve the diagnosis and timeliness of notifications of ARF.

ARF was added to the list of notifiable conditions in 1995. Therefore NT wide Notifiable Diseases data is only available after this time. Diagnoses of ARF and recurrences are made according to the Revised Jones Criteria (1992). Recurrence can only be diagnosed 3 months or more after the last episode of rheumatic fever.

As Figure 1 and Table 1 demonstrate, there has been a small, but evident increase in the total number of incident cases of ARF from 21 in 1995 to 27 in 2002, with a peak of 33 in 1999. Across the eight-year period, recurrences have accounted for between 23.3% (2001) and 57.9% (1997) of all cases of ARF. The average annual percentage of cases accounted for by recurrences was 40.1% (63/157) prior to the commencement of the program, falling to 26.3% (15/57) for the years 2001/2002. Although it is too early to know if these trends are sustainable, and as a direct result of the program itself, these are important positive signs.

Figure 1. ARF cases, Central Australia, 1995-2002.

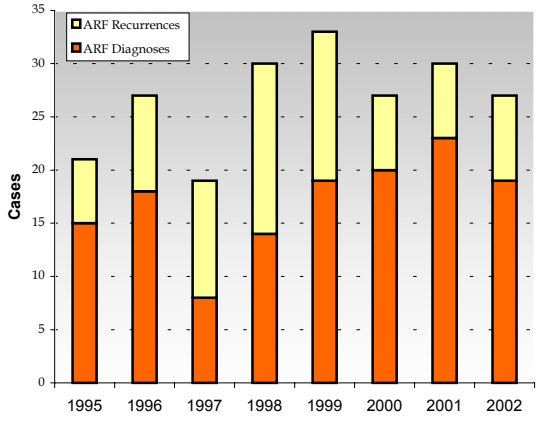


Table 1: Central Australian ARF diagnoses, recurrences and notification system accuracy, 1995-2002.

Year	New ARF Diagnoses (N)	ARF Recurrences (N)	Total Cases (N)	Recurrence as % of ARF Diagnoses	NT Notifications (N)*	Notification Coverage (%)*
1995	15	6	21	28.6	5	23.8
1996	18	9	27	33.3	11	40.7
1997	8	11	19	57.9	9	47.4
1998	14	16	30	53.3	6	20.0
1999	19	14	33	42.4	3	9.1
2000	20	7	27	25.9	10	37.0
2001	23	7	30	23.3	25	83.3
2002†	19	8	27	29.6	26	96.3

*Excludes cases diagnosed in the A-P Lands of South Australia
† Up to the end of October

Furthermore, since the inception of the program, notification system coverage has improved significantly, from an average of 28% (44/157) of all documented cases of ARF being included on the notification system prior to 2001, to 89.5% for 2001/2002 (51/57) ($\chi^2=63.96$, $p<0.001$).

Retrospective case note auditing and improved active surveillance activities have identified a large deficit in ARF notifications, especially in reporting recurrences. Problems identified in the notification of episodes of ARF and recurrences of ARF include:

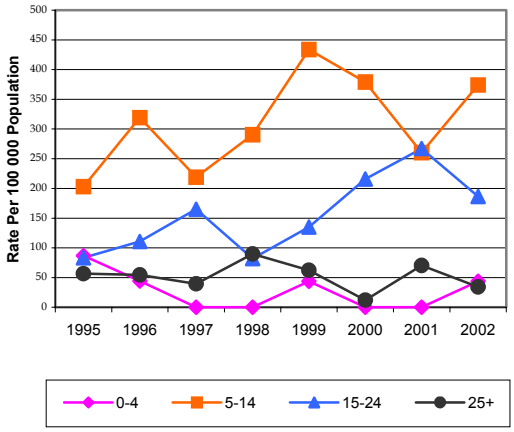
- Some staff are unaware that ARF and recurrences are notifiable.
- Recurrences of ARF may not be diagnosed because clients may not present, symptoms may not be recognised and appropriate investigations may not be performed.

To improve the system of notification of ARF the following strategies are being developed:

- Education of all health staff in ARF guidelines and the Jones Criteria.
- Distribution of the Central Australian Rural Practitioners Association (CARPA) guidelines for diagnosis and treatment to all health staff.
- Client education stressing the need to present with symptoms of a recurrence.

Figure 2 highlights the incidence of ARF among Aboriginal persons, by age group, for the Central Australian region of the Northern Territory for the years 1995-2002. Diagnoses among clients from SA and WA have been excluded from this analysis because of difficulties ascertaining appropriate denominators over the specified time period.

Figure 2: Incidence rates of ARF among indigenous clients in Central Australia, 1995-2002.



The incidence of ARF among Aboriginal clients of Central Australia has been relatively stable for the 0-4 and 25+ age groups between 1995 and 2002. Furthermore, they account for only 2.3% (5/214) and 15.4% (33/214) respectively of all cases between those years.

The highest incidence rates were found in the 5-14 year age group for every year except 2001. These rates have risen from 203.1 per 100,000 population in 1995 to 374.2 per 100,000 in 2002. High rates were also noted for the 15-24 year age group, rising from 83.1 per 100,000 in 1995, to 186.6 per 100,000 in 2002.

There is an apparent rise in incidence rates among Aboriginal persons aged 5-24 years between 1995 and 2002. It is possible that there was under ascertainment of cases in the earlier part of the study period. Subsequent improvements in case finding through active surveillance since the commencement of the program could thus overestimate the relative increase in incidence. Most notably, increased hospital surveillance, especially of clients admitted with a provisional diagnosis of arthritis who have subsequently been demonstrated to have ARF, has served to increase total case numbers.

Despite some concern with potential bias, the trend of increasing incidence among the 5-14 and 15-24 year age groups is of concern.

Table 2: Prevalence of rheumatic heart disease in the NT, 2001, by age group (in Central Australia) and ethnicity and region.

Age Group	Aboriginal		Non-Aboriginal	
	Number of Cases	Prevalence per 1000	Number of Cases	Prevalence per 1000
Central Australia				
5-14 yrs	37	7.64	0	0
15-24 yrs	80	18.6	2	0.51
25-44 yrs	101	15.8	4	0.36
45+ yrs	49	14.9	10	1.4
Total	**267**	**12.5**	**16**	**0.57**
Top End (1995)[2]				
Total	**271**	**9.6**	**14**	**0.14**

Table 2 shows the prevalence of RHD by age group and ethnicity in Central Australia. Top End figures are given for comparison. There are currently 283 people with documented RHD in the region, 94% being Aboriginal. Overall the prevalence of RHD was 12.5 per 1000 for the Aboriginal population, and 0.57 per 1000 for the non-Aboriginal population. The highest prevalence was seen in the Aboriginal 15-24 year age group. Almost 2% of this group have rheumatic heart disease. This highlights the large burden of significant heart disease occurring across the region, particularly at young ages.

The prevalence of RHD in Central Australia is higher than that demonstrated among both Aboriginal and non-Aboriginal populations in the Top End of the Northern Territory, based on comparison with figures from 1995.

C) To monitor and increase adherence to secondary prophylaxis

One of the main priorities in the prevention of ARF recurrences is to ensure adequate levels of compliance with secondary prophylaxis. The recommended prophylaxis is a monthly injection of two mls IMI benzathine penicillin. Carapetis (1998) stated that, in Top End communities, the percentage of required injections actually given was as low as 28% without intensive follow up and recall.

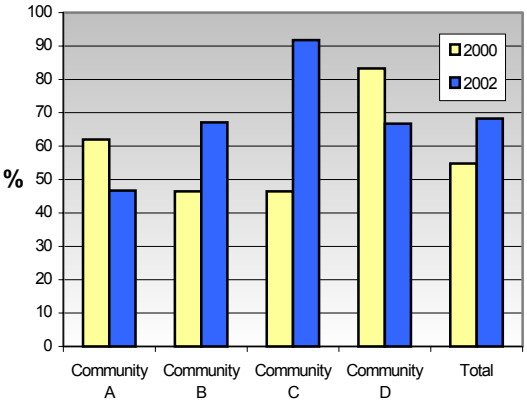
One of the objectives of the program is to monitor the level of compliance. It is hoped that education of clients and families will increase the level of compliance.

Achievements

Several communities were established as sentinel sites to assess compliance over time. Data is currently available for 4 communities, with the aim to extend data collection to all communities in the region.

Figure 3 shows compliance data from the 4 sentinel communities. The overall compliance with benzathine penicillin was 54.8% in 2000, rising to 68.3% in 2002 (χ^2 *18.61, p < 0.001*). These compliance data are a long way from

Figure 3: Benzathine penicillin compliance in 4 Central Australian communities, 2000-2002



ideal. A number of barriers to improving compliance have been identified. They included:

- Clinics only keep recall lists for the current year
- Poor response rate of clinics to requests for compliance information
- High staff turnover
- Mobility of the population
- Lack of communication between clinics
- Difficulty in providing services to multiple remote out-stations
- Attitudes of health staff to active follow up

Overall, the success or failure of any approach is dependent on the health staff. A consistent finding is that when a dedicated person is given the task of active follow up, the level of compliance improves.

D) To improve the support and education of clients with ARF/RHD and their families.

and

E) To improve the education of health staff and the community in order to increase the understanding and awareness of ARF/ RHD.

There are numerous forms of education that are provided by the coordinators.
Education is aimed at:

- newly diagnosed clients
- clients with ARF and normal heart valves
- clients with significant heart valve damage
- pre and post surgical clients
- all health staff

Education is provided on a one-to-one basis with clients, their families and carers, as well as in small informal groups and in formal education sessions.

Achievements:

During the 5 months since recommencement of the program, 50 clients have been educated on a one-to-one basis in hospital, during general and specialist outpatient clinics, and in communities. Resources such as 'The Rheumatic Fever Story' video, 'The Heart Story' flip chart, ARF booklets and anatomical models are used to help clients achieve an understanding of the disease process. Education is provided on the recognition of disease signs and symptoms, medication regimes and surgery.

Education sessions and presentations have also been provided to:

- Aboriginal Health Workers
- Medical students
- Families/carers
- Community members
- Remote Area Nurses
- Hospital nurses
- Hospital doctors
- District Medical Officers
- Public Health Staff

Information that is provided is specific for the 'target' group. For example, a presentation to the Maternity unit will include a general talk on ARF and RHD, but also more specific information on problems in pregnancy.

Information packages have been sent to all communities. These packages include an educational video, information about the ARF/ RHD program, and guidelines for the diagnosis, treatment and follow up of clients with ARF or established RHD.

An information help line is provided, which assists with the day to day management of cases, aiding information flow between primary and secondary health care settings, assisting with the arrangement of further follow-up and investigations and re-emphasising appropriate management of clients.

F) To establish lines of communication and consultation with relevant stakeholders, including Aboriginal community controlled health organisations.

Achievements

- Networks and lines of communication have been established with relevant hospital departments and personnel, independent health services, remote Aboriginal communities (both government and independent) and tertiary referral centres.
- The RHD Program Steering Committee has been established and meets on a regular basis to discuss progress and issues arising

from the program. The committee meets at least 6-8 weekly. Membership includes the Director of Paediatrics, the Director of Medicine, a Public Health Medical Officer, the Outreach Physician, Outreach Paediatrician, the Cardiac Educator and the Coordinators. The Coordinators are also involved in the Top End Steering Committee.

- Awareness of the program in remote clinics has been increased by regular tele-conferencing, the circulation of a laminated poster, and attendance at remote area nurse orientation.
- A RHD Help Line has been established and publicised.
- Established links with the Commonwealth funded Medical Specialist Outreach Assistance Program (MSOAP).

G) To improve clinical care and follow up of people with established RHD

and

H) To facilitate best practice management of ARF/RHD.

The program has a goal of coordinated care across all levels of service provision through the establishment of the coordinator as the central point of contact for clinical care, education and prevention of RHD.

Achievements

- Updated client lists are sent to all communities.
- The database is continually updated with current information on the client's condition and details of recent reviews/investigations/ treatment plans. This information is then disseminated to relevant care providers.
- Standardised best-practice guidelines for clinical care have been disseminated.
- The program has sought to identify all clients on the register with moderate or severe heart disease as defined by echocardiography to develop a high-risk approach to management and follow-up.
- Development of a specialised paediatric RHD clinic, providing an opportunity for intensive education.
- Identification and care planning for clients who have been recommended for or have had valve surgery.
- Coordination of opportunistic care and investigation for clients whilst they are in hospital.
- Coordination of care between the primary health care sector and secondary/tertiary referral centres when clients require urgent care or specialist treatment.

I) Future Needs and Developments

1) High Risk Strategy

A total of 159 clients have been identified with moderate to severe valvular heart disease and 46 post surgical clients identified who require intensive education and follow up. The adoption and formulation of a high-risk strategy will ensure these clients are given priority, and intensive education developed and implemented.

Some of these clients have been lost to follow up for a number of identified reasons, including:

- Lack of continuity of care for clients passing from an age group where they are cared for by the paediatric team to adult physician care. The identified lack of formalised hand-over is being addressed through the steering committee.
- High patient mobility.
- Lack of awareness among staff and clients of the progression and prevention of disease.
- Non attendance at outpatient clinic appointments results in removal from hospital recall lists.
- High turnover of staff.

2) The development of educational resources

There is a need to design, develop, implement and evaluate educational resources for individuals and groups at increased risk of ARF and RHD. The program is focused on patient and family education. Resources are required for a number of stages of the disease process, from initial diagnosis of ARF, to severe heart disease and subsequent surgery.

Furthermore, the material must cover a wide spectrum of ages.

There is a clear need to build on the resources available (Rheumatic Fever Story), to assist client educators to interact with patients and their families. The inclusion of audio-visual education modalities, which can be translated into local Aboriginal languages, is an important future step in this program.

3) Antenatal Protocols

As pregnancy is a high-risk time for clients with RHD, the monitoring and management of heart disease throughout gestation needs to be meticulous. Staff need to be well informed of the need for good antenatal care and the potential for complications, particularly at the time of delivery. Education and the development of specific protocols are future priorities.

4) Echocardiogram guidelines

It is acknowledged that echocardiography should be more readily accessible for clients with significant heart disease, and for clients who are ceasing treatment. However, there are currently no specific recommendations regarding the frequency of echocardiography, according to type and severity of valve lesions. Guidelines will be developed, standardised and disseminated.

5) Provision of Multidisciplinary Outreach Services

The program will develop and implement a multidisciplinary outreach service that will visit remote communities. It is intended that these services be provided to both government and independently run health services, utilising the coordinator, paediatrician, physician/medical officer and public health staff for the provision of echocardiograms, clinical review and education to clients in the community. It is anticipated that community governance structures will be educated about the program, the illnesses associated and the preventative activities that could be developed in collaboration with the program.

Conclusion

RHD in Aboriginal Australians is a major public health problem. It is a cause of very significant morbidity and mortality in young people. Almost 2% of 15-24 year old Aboriginal people in Central Australia have RHD and this would be completely unacceptable in any other society. Historically there has been a failure to appreciate the extent of this problem and to provide adequate funding for public health programs.

The Central Australian RHD Control Program has taken some important steps. The initial program goals were clearly defined and have been achieved. The centralised database is now well established and robust systems for ongoing data collection are in place. A strong focus on clinical care and education has been maintained. The potential limitations of the data are recognised, but the indications are that there has already been a significant improvement in rates of notification and adherence to secondary prophylaxis.

The program is now entering a consolidation phase. Maintenance of the database will require less time and efforts will be focused on other priorities including the management of high risk clients and the development of outreach services. Although it is anticipated that there will be significant positive outcomes in the next 12-18 months, much work is still to be done, and there is a strong consensus that the program will need to be sustainable beyond the currently funded period to see major gains.

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1. Currie BJ, Brewster DR. Rheumatic Fever in Aboriginal children. *J Paediatr Child Health* 2002 38(3):223-225.
2. Carapetis JR, Wolff DR, Currie BJ. Acute rheumatic fever and rheumatic heart disease in the Top End of Australia's Northern Territory. *MJA* 1996; 164: 146-149.

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Top End Rheumatic Heart Disease Program

A Report to the Commonwealth: February – November 2002 (abbreviated)

Prepared by Angela Kelly, Rheumatic Heart Disease Program Coordinator,
Top End, Northern Territory
December, 2002.

Background

In the Top End of the Northern Territory (NT) the incidence of acute rheumatic fever (ARF) in children in 2001 was 142 per 100,000, higher than anywhere else in the world. The systematic prevention of ARF recurrences in order to reduce the burden of rheumatic heart disease (RHD) is our major public health strategy.

Introduction

This report outlines the achievements of the Top End RHD program for the period between February and November 2002. Funding provided by the Commonwealth Department of Health and Aging has enabled the part-time employment of a coordinator (0.7 FTE) for one year commencing February 2002.

The Top End is divided into Darwin urban, Darwin rural, East Arnhem and Katherine regions consisting of a population of 154,000 people. Of these, 38,000 are Aboriginal people, of whom 27,000 live outside the Darwin urban region.

Commencing in 1997, the program has, in line with World Health Organisation (WHO) recommendations:

- developed and maintained a centralised, computerised database containing all known cases of ARF and RHD
- provided staff and patients with education about these ailments; developed best practice guidelines
- collated epidemiological data about these conditions.

A steering committee meets regularly to formulate priorities for the program.

As the coordinator's position formally ceases in April 2003, the priorities for this period have been to ensure the continuation of the various parts of the program, i.e. maintenance of the database, staff and patient education.

Existing database/register

The database/register provides information to determine the incidence and prevalence of ARF/RHD, to assist with the coordination of ongoing patient care and to increase and maintain awareness of rheumatic heart ailments.

Lists of clients on the database have been sent to the client's home community health centre and to the attending paediatrician and physician in June and October this year, with another run scheduled for December. These lists assist the health centre in planning and provision of client care. Feedback from the community health centre provides for updating of the register and assists in informing community centres of client whereabouts if the client has relocated.

The database has been modified so that a designated administrative person can maintain it without having to interpret medical information. The database now consists of only the information that is fed back to the community and that required by the Notifiable Diseases Act.

Two of the functions of the database i.e. maintaining a priority patient list and maintaining a list of those who have had heart valve surgery, have successfully been transferred to NT Cardiac (cardiologists) who now organise and provide these services.

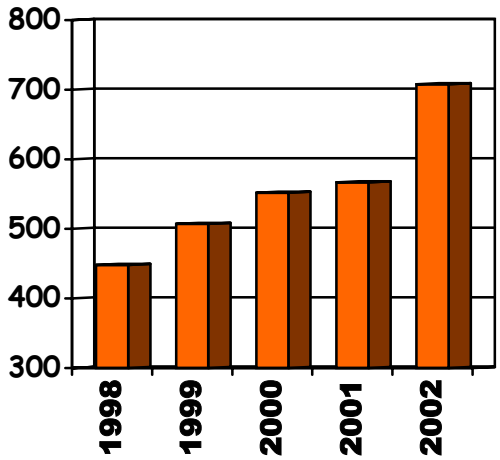
There are currently 900 people on the Top End RHD database, of whom 708 have RHD, 170 have had ARF, and 22 have had suspected ARF (Figure 1).

There have been 88 cases of ARF, notified to the register, between Jan 2000 and Nov 14, 2002 in the Top End. Nineteen out of 40 cases of ARF this year have been recurrences. The number of identified established cases of RHD has increased by 136 in 2002 (Figure 1).

Table 1. ARF cases notified in the Top End 2000-2002

	ARF	<15 yrs	Hospitalised	Recurrences
2002 (Nov 14)	40	28 (70%)	33 (82%)	19 **(48%)**
2001	28	19 (68%)	19 (67%)	8 (28%)
2000	20	14 (70%)	14 (70%)	6 (30%)
Totals	88	61 (69%)		

Figure 1. RHD in the Top End 1989-2002



Death due to RHD

Between 1993 and November 2002, 138 people on the RHD register died. RHD was the cause of death in 70 of these people (table 2). It is unknown if RHD was a contributing cause in 27 deaths. Five people have died from RHD in 2002.

Table 2. Age at death from RHD 1993-2002

Age in years	<15	<30	31-40	41-50	>50	All ages
Number of deaths	4	23	18	13	12	70

Best practice management

Best practice guidelines state that secondary prophylaxis for the prevention of recurrences of ARF is 4 weekly intramuscular benzathene penicillin. Recurrences accounted for 48% of ARF episodes this year. The high recurrence rate indicates that the secondary prophylaxis regimens are failing. People are not receiving the 4 weekly benzathene penicillin injection or, if they are on oral penicillin, they are not taking every dose. A memo was sent to all health care centres and all medical officers on behalf of the RHD steering committee outlining best practice guidelines.

In education sessions and at every opportunity, best practice guidelines are promoted with regard to having a functioning recall system in every community health centre with, preferably, a designated staff member in charge of ARF/ RHD management with the aim of increasing the secondary prophylaxis uptake.

A power-point presentation has been developed over the year, which covers definitions, diagnosis, management of ARF/RHD, the role of the RHD program and discussion/tips about improving the uptake of secondary prophylaxis. This presentation has been developed in collaboration with district medical officers (DMO), registered nurses and Aboriginal health workers, utilising feedback from many education sessions. This power point presentation is available from Disease Control on request.

As the coordinator position finishes in April 2003, it was decided that the education function of the position be transferred to other educators.

- Two staff in the Department of Health and Community Services, Workforce Development will incorporate ARF/RHD education into their sessions for Darwin urban and remote health staff.
- The Preventable Chronic Disease Strategy public health nurses have been approached and will discuss further in the new year
- A DMO is reviewing the educational material for use in education sessions in remote health care centres.
- A workshop was held with the AHW educators recently where the powerpoint presentation was modified for their use in

educating colleagues in remote areas. These educators will utilise this educational resource to assist with the assessing of AHW competencies.

Move to consent process

Early in 2002, concern was expressed by 2 independent health services about the data being collected by the RHD register and its usage. The impact of the NT Privacy Information Bill and establishment of the proposed NT Independent Health Zones have implications for the completeness of the RHD register. A decision was made by the steering committee to obtain individual consent from those on the RHD register and new people as diagnosed, to be included on the RHD register.

A meeting was held with the NT Privacy Information Project Officer, Ms Robyn Cooke. A draft consent form and accompanying fact sheet outlining the function of the register has been circulated and approved by members of various stakeholder groups. It is planned that the consent process will be introduced early in 2003.

Conclusion

As of November 14, 2002, 40 cases of ARF had been notified to the RHD register in the Centre for Disease Control. More than 48% of these cases were recurrences. This is obviously a worrying trend as recurrences of ARF are almost entirely preventable and recurrences of ARF may lead to further potentially debilitating heart disease.

Challenges for the program:

- Improving secondary prophylaxis compliance rates.
- Continuing education to staff, patients and families in the Top End, including to the remote areas, without a program coordinator position.

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Newspaper surveillance. Chikungunya spreads to West Timor and Central Sulawesi in Indonesia

Summary report from Jakarta Post online, Wed 26 Feb 2003
Byline: Nana Rukmana and Yemris Fointuna

Chikungunya, caused by an alphavirus and transmitted by the mosquito *Aedes albopictus,* has been spreading across Indonesia during the past rainy season, particularly in regions with high rainfall.

The head of intensive care at the Kupang General Hospital, Frank Touw, said that chikungunya had spread to urban and rural areas in Kupang regency. He said "Though the disease is not fatal, those affected will be physically weak for a fairly long time and it takes 3 to 5 days to recover". Based on the Jakarta Post's monitoring of 5 hospitals and a number of health centres in Kupang, 50 to 100 chikungunga patients were admitted daily in the last weeks of February.

The disease chikungunga, which first emerged in Bandang, West Java in December 2002, has spread to East Nusa Tenggara (West Timor) and Central Sulawesi. Hundreds of people in Kupang, the capital of East Nusa Tenggara, have been treated at hospitals and public health centres for the disease. The chikungunga disease symptoms are similar to dengue fever which is also caused by a flavivirus and transmitted primarily by the mosquito *Aedes aegypti.*

Chikungunga is considered to be a relatively recent introduction into Southeast Asia, and an increasing public health problem.

Comment

In light of the occurrence in West Timor, there is a good possibility that chikungunga disease could occur in East Timor. Travellers to Timor and other areas in the region should consider mosquito protection while travelling and advising doctors of travel history when presenting with suggestive symptoms after returning.

This story documents another good reason to keep the NT free of exotic *Aedes* vectors such as *Aedes aegypti* and *Aedes albopictus*.

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Meningococcal Group C Vaccine

CDC Fact sheet

Meningococcal Disease

Meningococcal disease is caused by bacteria called meningococci. In the Northern Territory, about two thirds of cases of meningococcal disease are due to group B meningococci, and the rest are mainly due to group C. While there are fewer cases of C than B disease, group C meningococci generally cause more severe disease than group B. About 1 in 10 people with C disease will die.

Meningococci are commonly found at the back of the throat or nose of healthy people. Although most people who 'carry' this germ in their throat or nose remain quite well, they are able to spread it to others, a few of who may subsequently become very ill. It is spread in the fine droplets that are passed to other people through coughing, sneezing and kissing.

Cigarette smoking, both active and passive, increases the risk of a person developing meningococcal disease.

What is meningococcal disease?

Meningococcal disease is a rare (around 10-15 cases of per year in the NT) but severe infection, which occurs when the meningococci 'invade' the body from the throat or nose. The infection often develops very quickly and can cause long term complications or death.

It occurs in two main forms: **septicaemia,** when there is blood poisoning and widespread infection throughout the body; and **meningitis,** when there is infection in the outer lining around the brain and spinal cord.

Symptoms of meningococcal disease include fever, lethargy, severe headache, vomiting, joint or muscle pains, stiff neck, dislike of bright lights and drowsiness. There may be a rash, which starts anywhere on the body as tiny red or purple spots but soon spreads to look like bruises.

The rash must be taken seriously as the person requires urgent medical attention.

The symptoms of meningococcal meningitis in young babies may differ from those above and include: refusing feeds, vomiting, a high pitched moaning cry, irritability and a dislike of being handled, a blank staring expression, lethargy or drowsiness and a pale blotchy complexion.

Babies and children under 5 years of age, and young adults aged 15-24 years are at the highest risk of getting meningococcal disease.

Meningococcal group C vaccines

There is no vaccine against meningococcal group B disease. Parents must continue to be on the lookout for signs of meningococcal disease, even if their child has been vaccinated against meningococcal infection.

The new conjugate meningococcal group C vaccines give protection against group C disease that lasts for many years. They also work well in babies and young children. One dose provides protection for adults and children aged 12 months or older. Infants younger than 12 months need 2 or 3 doses depending on their age.

The older polysaccharide meningococcal vaccines provide protection against 4 groups of meningococci (A, C, W135 and Y). However, these vaccines do not work in children under 2 years of age and only give protection for 3-5 years. Anyone who has received polysaccharide meningococcal vaccine in the past and who requires protection against C disease should receive a conjugate meningococcal C vaccine (at least 6 months after the polysaccharide vaccine).

Who is eligible for free vaccine?

Children turning 12 months of age from January 1 2003 will receive meningococcal C conjugate vaccine with their routine immunisations due at 12 months.

Children and adolescents aged 1-19 years in 2003 will be eligible for free vaccine from 2003 to 2006 through a staged national meningococcal program.

In the Northern Territory, vaccine will be available as shown below.
School-aged children will mainly be vaccinated via school based programs.

2003	**Children turning 1-5 years Senior high school students (15-19 years)**
2004, 2005	**Children aged 6-14 years**
late 2005, 2006	**Those aged 1-19 years in 2003 who are not yet vaccinated**

Does the vaccine work?

Meningococcal C conjugate vaccines are about 92% effective in protecting toddlers against invasive meningococcal C disease and about 97% in teenagers.

Is the vaccine safe?

Meningococcal C conjugate vaccines are not live vaccines and cannot cause infective meningitis.

The most common side effects of the vaccine are redness, soreness and swelling at the site of the injection. Other possible side effects are:

- low grade fever;
- irritability;
- change in appetite;
- headache (adolescents and adults);
- malaise and muscle pain.

These side effects are usually mild and get better by themselves. If they do occur, they can be reduced by paracetamol (Panadol) and extra fluids to drink.

Serious side effects, such as allergic reactions are rare and occur in less than 1 in 10,000 doses.

Further information

Contact your immunisation provider or your nearest Centre for Disease Control on:

Darwin	8922 8044
Katherine	8973 9049
Nhulunbuy	8987 0359
Tennant Creek	8962 4259
Alice Springs	8951 7549

Information can also be obtained from http://immunise.health.gov.au/

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Severe Acute Respiratory Syndrome (SARS) in mid March 2003

SARS is characterised by atypical pneumonia-like symptoms of fever, cough, shortness of breath or difficulty breathing. Symptoms may be severe and require oxygen and mechanical ventilation. There is a fatality of around 4%.

SARS has been largely limited to a number of Asian countries, Southern China, Hong Kong, Vietnam and Singapore.

Updates and information on clinical diagnosis and management can be obtained from Disease Control on 89228044 or:

Australian Department of Health and Aging
http://www health.gov.au/sars.htm

Table of cases from the World Health Organisation (WHO)
http://www.who.int/csr/sarscountry/en/

WHO discharge and followup guidelines
http://www.who.int./csr/sars/discharge/en/

WHO case management
http://www.who.int.csr/sars/management/en/

Centre for Disease Control (Atlanta)
http://www.cdc.gov/ncidod/sars

Information for General Practitioners and other Health Professionals at:
http://www.health.gov.au/pubhlth/cdi/cdna/press/saraha2.htm

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Guidelines for the Control of Nontuberculous Mycobacteria in the Northern Territory

Centre for Disease Control, Darwin, October 2002

Introduction

The genus *Mycobacterium* includes the species responsible for causing tuberculosis (TB) (*M. tuberculosis, M. bovis, M. africanum*: also called *Mycobacterium tuberculosis* complex) and leprosy (*M. leprae*). There are several other species of mycobacterium that are collectively called nontuberculous mycobacteria (NTM). NTM have been recognised since late in the nineteenth century but only since the 1950s have they been well recognised as a cause of human disease. With advances in molecular biology and the ability to map the entire genome of a bacterium new NTM are identified on a regular basis these days. More reliable identification techniques have helped elucidate the pathological potential of some infrequently isolated NTM. The pathological potential of several species however is still unclear. NTM disease is notifiable in the Northern Territory (NT) but not in other states of Australia.

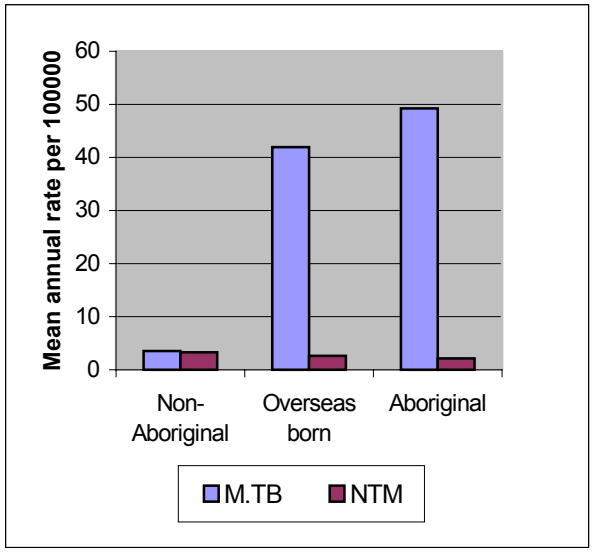
Epidemiology

Incidence

NTM disease presents most commonly as pulmonary disease. Other manifestations include: disseminated disease; skin, soft tissue and skeletal disease; and lymphadenitis. An extensive review of NTM occurring in the NT from 1989 to 1997, by O'Brien et al, was published in *Clinical Infectious Diseases* in 2000[1]. There is now data available to 30/06/02. There were 74 cases of NTM disease reported in the NT between January 1989 and June 2002. This gives an average incidence rate per year of 3.2 per 100 000.

Rates of NTM disease are more than double for males compared with females. The majority (86%) of notifications were among people born in Australia. The mean duration of residence in Australia for people born overseas was 25 years. This suggests NTM infection is acquired locally and progresses to disease with limited latency, unlike TB. Also, unlike TB, the incidence of NTM disease is higher in non-Aboriginal people compared with Aboriginal people. See Figure 1.

Figure 1. Ethnic background of persons in the NT with disease due to *M. tuberculosis* and NTM 01/01/89 to 31/12/2001



Source of NTM

NTM occur naturally in the environment and are therefore often referred to as environmental mycobacteria. Most NTM organisms have been isolated from water and soil. NTM disease occurs in animals but transmission from animals to humans is felt not to be important in human infection. Human to human transmission is rare. This means public health measures such as isolation and contact tracing are not necessary with NTM disease. In the NT there is no significant difference in the likelihood of developing disease due to NTM in rural versus urban areas. Of the 74 cases notified in the NT in the last 13 years, 41 people came from the Darwin Urban Area as opposed to 33 people from other parts of the NT (RR = 1.1 (95% CI 0.7-1.7)).

Risk factors

In O'Brien et al's review of NTM disease in the NT risk factors identified included: smoking;

Table 1. Classification of NTM recovered from humans[2,3,4]

Clinical disease	Common etiologic species	Disease ever notified in the NT	Unusual etiologic species
Pulmonary disease	*M. avium* complex (MAC)*	Yes	*M. asiaticum*
	M. kansasii	No	*M. branderi*
	M. abscessus	No	*M. celatum*
	M. xenopi	No	*M. fortuitum*
	M. malmoense	No	*M. haemophilum*
			M. scrofulaceum
			M. shimoidei
			M. simiae
			M. smegmatis
			M. szulgai
Lymphadenitis	MAC	Yes	*M. abscessus*
	M. scrofulaceum	No	*M. chelonae*
	M. malmoense	No	*M. fortuitum*
			M. haemophilum
			M. interjectum
			M. kansasii
			M. szulgai
Skin and soft tissue disease #	*M. marinum*	Yes	MAC
	M. fortuitum	Yes	*M. branderi*
	M. chelonae	No	*M. haemophilum*
	M. abscessus	Yes	*M. kansasii*
	M. ulcerans	No	*M. mucogenicum*
			M. scrofulaceum
			M. simiae
			M. smegmatis
			M. szulgai
			M. terrae complex$^{\phi}$
Disseminated disease	MAC	Yes	*M. abscessus*
	M. kansasii	No	*M. conspicuum*
	M. chelonae	No	*M. fortuitum*
	M. haemophilum	No	*M. genavense*
			M. gordonae
			M. malmoense
			M. marinum
			M. scrofulaceum
			M. simiae
			M. szulgai
			M. xenopi

**M. avium* complex (MAC) consists of two distinct species *M. avium* and *M. intracellulare*.

#*M. marinum, M. fortuitum*, MAC, *M. scrofulaceum*, *M. haemophilum* and *M. szulgai* are also causative organisms for osteomyelitis.

$^{\phi}$*M. terrae* complex includes *M. terrae*, *M. nonchromogenicum*, and *M. triviale*

chronic lung disease; and immunosuppression due to HIV infection, alcoholism and immunosupressive therapy.[1]

Table 2. NTM whose pathogenicity is unclear[2,3]

M. gastri	*M. aichiense*
M. flavescens	*M. aurum*
M. neoaurum	*M. chubuense*
M. thermoresistible	*M. gadium*
M. mageritense	*M. celatum*
M. phlei	*M. lentiflavum*
M. vaccae	*M. triplex*

Table 3. NTM growth rate classification[2,3]

Slow growing NTM	Rapidly growing NTM
MAC	*M. abscessus*
M. asiaticum	*M. aichiense*
M. branderi	*M. aurum*
M. celatum	*M. chelonae*
M. conspicuum	*M. chubuense*
M. flavescens	*M. fortuitum*
M. gastri	*M. gadium*
M. genavense	*M. mageritense*
M. gordonae	*M. mucogenicum*
M. haemophilum	*M. neoaurum*
M. interjectum	*M. phei*
M. kansasii	*M. smegmatis*
M. lentiflavum	*M. thermoresistible*
M. malmoense	*M. vaccae*
M. marinum	
M. scrofulaceum	
M. shimoidei	
M. simiae	
M. szulgai	
M. terrae	
M. triplex	
M. ulcerans	
M. xenopi	

Pathogenic species

An extensive list of NTM recovered from humans is included in Table 1. Pathogenicity is unclear for some NTM (Table 2). This is usually because there are so few cases reported and some of the reports lack sufficient documentation of the organism's identification, or its disease association, to confirm the validity of the case. Though it is unclear, those listed in Table 2 are probably non-pathogenic. NTM fall into 2 categories, slow growing and rapidly growing. Mycobacteria that form colonies clearly visible to the naked eye within 7 days on subculture are termed rapid growers, while those requiring longer periods are called slow growers. Those identified in Tables 1 and 2 are categorised as either slow or rapidly growing in Table 3.

The clinical distribution of NTM disease in the NT is shown in Table 4. NTM isolated in association with disease in the NT are included in Table 5. Interestingly *M. kansasii*, which is commonly identified as a cause of NTM disease in the USA, Europe and other parts of Australia, has not been associated with disease in the NT in the last 13 years. Other common causative organisms such as *M. xenopi, M. malmoense, M. chelonae, and M. ulcerans* have not been associated with disease in the NT in this time period either.

Table 4. Clinical distribution of NTM disease in the NT 01/01/89 to 30/06/02.

SITE	HIV associated case	Non-HIV associated case	HIV status not tested	Total
Pulmonary	4	37	4	**45 (61%)**
Disseminated	9	1	-	**10 (13%)**
Skin/soft tissue	3	7	4	**14 (19%)**
Nodal	-	5	-	**5 (7%)**
Total	**16**	**50**	**8**	**74**
Percentage	**22%**	**68%**	**11%**	**100%**

Table 5. NTM isolated from patients with NTM disease in the NT 01/01/89 to 30/06/02.

ORGANISM	Pulmonary	Disseminated	Skin/soft tissue	Nodal	Total	Percentage
MAC	42	9	2	2	**55**	**74%**
M. fortuitum	-	1	6	-	**7**	**9%**
M. haemophilum	-	-	2	-	**2**	**3%**
M. scrofulaceum	2	-	-	-	**2**	**3%**
M. terrae	-	-	2	-	**2**	**3%**
M. abscessus	-	-	1	1	**2**	**3%**
M. gordonae	-	-	-	2	**2**	**3%**
M. marinum	-	-	1	-	**1**	**1%**
M. simiae	1	-	-	-	**1**	**1%**
TOTAL	**45**	**10**	**14**	**5**	**74**	**100%**

Table 6. Outbreaks of iatrogenic NTM infection reported in medical literature.

Mycobacterium	Disease	Cause
M. abscessus	Surgical wound infections	Rinsing surgical equipment in contaminated tap water and then inadequate sterilisation of equipment[5]
M. abscessus	Injection site infection	Not identified but all cases from one physician's office[6]
M. abscessus	Injection site infection	Contaminated unlicensed injectable alternative medicine[7]
M. chelonae	Injection site infection	Contaminated external surface of penicillin vial lids[8]
M. chelonae	Post liposuction cutaneous abscesses	Rinsing surgical equipment in contaminated tap water and then inadequate sterilisation of equipment[9]
M. chelonae	Post podiatry procedure foot infection	Contaminated jet injector[10]
M. chelonae	Injection site infection	Contaminated normal saline solution[11]
M. chelonae	Post rhinoplasty nasal cellulitis	Rinsing surgical equipment in contaminated tap water and then inadequate sterilisation of equipment[12]
M. chelonae	Bacteraemia, soft tissue infection and disseminated disease post haemodialysis	Inadequate sterilisation of haemodialysis machines[13]
M. fortuitum	Post electromyography infection at site of electrode insertion	Needles sterilised then rinsed in tap water[14]

Outbreaks of NTM disease

Outbreaks of NTM disease have been reported in the literature. Most reports are related to skin and soft tissue disease secondary to iatrogenic infection from medical intervention. A search of the Medline database back to 1990 identified many such outbreaks (see Table 6).

In 1992-1995 a large localised outbreak of 29 cases of *M. ulcerans* infection occurred in a 4km square area of Phillip Island, Victoria, Australia. Cases were mostly with the elderly and had distal limb lesions. A golf course irrigation system was suspected as the source of infection and with limitation of the use of this there was a reduction in the number of new cases.[15] In a northern Californian nail salon an outbreak of *M. fortuitum* furunculosis occurred as a result of contaminated foot baths. The inlet suction screens of these footbaths had large amounts of hair and skin debris behind them, as they were never cleaned.[16] In Colorado restriction fragment length polymorphism analysis was

used to identify an indoor hot tub as the source MAC pulmonary disease in a family of 5, with varying degrees of respiratory illness.[17] No outbreaks of NTM disease have been reported in the NT.

Pseudo-outbreaks have been reported where NTM contamination has caused respiratory tract colonisation, rather than disease, and contamination of laboratory specimens. Ice machines and water fountains on hospital wards have been implicated as sources of NTM respiratory tract colonisation. Inadequate sterilisation of endoscopy, bronchoscopy, and colonoscopy equipment, and contamination of distilled water, and culture medium in the laboratory have been implicated in NTM contamination of diagnostic specimens.[18-29]

Clinical Manifestations

Pulmonary disease

Patients with NTM lung disease are generally older adults. Except for patients with cystic fibrosis, children rarely develop this form of NTM disease. Although many NTM patients have a history of underlying chronic lung disease not all do. Signs and symptoms of NTM pulmonary disease are variable and non-specific. They include:

- Chronic cough
- Sputum production
- Fatigue

Less commonly:

- Malaise
- Dyspnoea
- Fever
- Haemoptysis
- Weight loss (usually with advanced NTM disease)

Evaluation is often complicated by the symptoms caused by co-existing lung diseases. These conditions include chronic obstructive airway disease often associated with smoking, bronchiectasis, previous mycobacterial disease, cystic fibrosis and pneumoconiosis (asbestosis and silicosis).

Though there is some uncertainty, it does seem that in an HIV negative person, NTM can colonise the respiratory tract in the absence of tissue invasion. To compensate for this uncertainty multiple isolations of the same NTM and evidence of progressive disease (chest x-ray and symptoms) are required for diagnosis. The interpretation of NTM in the sputum of an HIV positive person is difficult as these patients are frequently felt to be infected with NTM without evidence of pulmonary disease. Such infection may be transient but it may also reflect disseminated NTM disease or subclinical NTM pulmonary disease. In addition some NTM species that are generally considered non-pathogenic have been associated with pulmonary disease in the HIV infected host.[2] This is an area of ongoing evaluation. Since the advent of triple drug therapy and in some cases preventative regimens, NTM disease in those with AIDS has markedly decreased.

Disseminated disease

Dissemination of NTM occurs in adults and children. From a global perspective and before HIV infection became prevalent, disseminated disease was rare and confined to immunocompromised individuals such as those with leukaemia, lymphoma, or on immunosuppressive therapy.[2, 30-34]

Patients without AIDS

In general, in patients without AIDS, disseminated disease caused by *Mycobacterium avium* complex (MAC) presents as fever of unknown origin whereas *M. kansasii, M. chelonae, M. abscessus*, and *M. haemophilum* generally present as multiple subcutaneous nodules or abscesses that drain spontaneously.[2]

Patients with AIDS

Disseminated disease due to NTM in patients with HIV infection usually occurs only in those with very advanced immunosuppression. The diagnosis is exceedingly rare in persons with >100 CD4 cells, but should be suspected in patients with <50 CD4 cells. Most patients have prolonged fevers, which may be as high as 39.5-40.0^{o}C and also frequently have night sweats. Weight loss is common and some people complain of abdominal pain and diarrhoea. Physical findings may only be those of advanced HIV disease, although abdominal pain, retroperitoneal adenopathy and hepatosplenomegaly may be present.[34] Again

disease has decreased with the advent of triple drug therapy and some preventative regimens.

Skin, soft tissue, and skeletal disease

NTM skin, soft tissue, and skeletal disease may occur in any age group. In the NT *M. fortuitum* is the most common causative organism (see Table 5). NTM may cause localised abscess formation and drainage at the site of puncture wounds. Infection due to long term intravenous or peritoneal catheters, post injection abscess or surgical wound infection can also occur due to NTM. Chronic granulomatous infection caused by NTM may develop in tendon sheaths, bursae, joints and bones after direct inoculation of the organisms through accidental trauma, surgical incision, or injections. Occasionally axial bones and extremities have been infected without apparent trauma and are due presumably to haematogenous spread.[2]

Lymphadenitis

Infection of the submandibular, submaxillary, cervical, or periauricular lymph nodes in children between 1 and 5 years old is the most common presentation of NTM lymphadenitis. It is the most common disease manifestation of NTM in children in the absence of HIV infection and rarely affects adults. The 5 cases of NTM nodal disease in the NT in the last 13 years, have been aged 1, 2, 3, 9 and 14 years. Two of these 5 (40%) were Aboriginal children whereas for the same time period 21 of 25 (84%) aged less than 16 years, notified with TB were Aboriginal children. The involved lymph nodes are usually unilateral and non-tender. The nodes may enlarge rapidly and even rupture, with formation of sinus tracts that result in prolonged local drainage.[2]

Diagnosis

Diagnostic criteria for lung disease in HIV-seropostive and HIV-seronegative hosts.

The following criteria (Figure 2) are from the Official Statement of the American Thoracic Society on the Diagnosis and Treatment of Diseases Caused by Nontuberculous Mycobacteria, 1997.[2] These criteria best fit for disease cased by MAC, *M. abscessus* and *M. kansasii*. Too little is known of other NTM to be certain how applicable these criteria will be.

Figure 2. Diagnostic criteria for NTM lung disease.

Applies to ***symptomatic*** patients with:

- infiltrative, nodular or cavitary disease **or**
- a high resolution CT that shows multifocal bronchiectasis and/or multiple small nodules

A) If three sputum/bronchial washing results are available from the previous 12 months[#]:

i) three positive cultures with negative AFB smear results **or**
ii) two positive cultures and one positive AFB smear

A) If only one bronchial wash is available:

i) positive cultures with a 2+, 3+, or 4+ AFB smear or 2+, 3+, or 4+ growth on solid media.

A) If sputum/bronchial washing evaluations are nondiagnostic or another disease cannot be excluded:

i) transbronchial or lung biopsy yielding a NTM **or**
ii) biopsy showing mycobacterial histopathologic features (granulomatous inflammation and/or AFB) and one or more sputums or bronchial washings are positive for an NTM even in low numbers

[#] Sputum samples must be collected at least seven days apart.

The traditional presentation of MAC lung disease has been as apical fibrocavitary lung disease, sometimes with huge cavities, in males in their late 40s and early 50s with a history of heavy cigarette smoking and, frequently, alcohol abuse. This form of disease is usually progressive within one to two years if left untreated. More recently it has become apparent that MAC lung disease also presents as bilateral nodular and interstitial/nodular disease or as isolated right middle lobe or lingular disease, predominantly in elderly non-smoking females.

Earlier it was thought that MAC colonised bronchiectatic lung. However, high resolution computerised tomography studies show some patients have MAC related parenchymal disease in addition to their bronchiectasis. The appropriate distinction is not between colonisation and invasive disease but between those patients with the disease of nodular bronchiectasis who require immediate therapy directed at MAC and those in whom such a decision can be delayed. If the decision is made to observe such a patient they need long term follow up as the MAC disease will likely progress at some stage and require treatment. Treating earlier rather than later is recommended.[2]

Differentiating NTM from *M. tuberculosis* complex is not possible radiographically or on acid fast bacilli (AFB) smear. Because of the public health risk of *M. tuberculosis* complex, disease suggestive of mycobacterial disease should be managed as TB until proven to be otherwise. The traditional method of identification is by culture and then biochemical testing. Some of these tests require inoculation of subcultures and subsequent delay in testing until adequate growth has occurred.

Genetic techniques are useful if a more urgent identification is required, for example to avoid the large public health undertaking associated with TB, or to definitively diagnose TB in someone who may need follow up. Detection and identification of *M. tuberculosis* complex directly from a clinical specimen can be done using the Polymerase Chain Reaction (PCR) test. Determining the presence of NTM or differentiation beyond *M. tuberculosis* complex using the PCR test is not possible, however, this can be done on special request using band techniques. The PCR test can be completed in one working day.

Once isolates arise on solid culture or from broth culture DNA probes can be used to identify the mycobacterium. Probes are available for *M. tuberculosis* complex, MAC, *M. kansasii*, *M. gordonae* and others are being developed. Probes to further differentiate *M. tuberculosis* complex are not yet available. The DNA probe test, on the cultured specimen, takes about two hours.

Where NTM is deemed more likely than TB or a diagnosis of TB has very wide public health implications the laboratory used for diagnosis should be called directly to have a PCR test requested.

Diagnosis of disseminated NTM disease

Patients without AIDS

The isolation of organisms from sterile closed sites such as bone marrow, blood, liver or from a skin biopsy (in the setting of multiple lesions) is diagnostic.

Patients with AIDS

The diagnosis of disseminated disease is most commonly confirmed by isolation of MAC in blood so it is important to request this investigation and liaise with the laboratory staff so they are aware of the organism being sought. Blood cultures will need to be collected in blood culture bottles containing mycobacterium culture medium. This media differs to the culture medium routinely used for non-mycobacterial bacteraemia. The bacteraemia is ongoing and a single blood culture in a symptomatic patient has a sensitivity of 90%. In a prospective study of HIV positive patients with < 50 CD4 cells approximately 67% of patients with MAC in sputum or stool had disseminated disease within one year however only one third of patients with disseminated disease had a prior positive stool.[2]

Diagnosis of skin, soft tissue, and skeletal disease.

Diagnosis is made by microscopy and culture of the specific NTM pathogen from drainage material or tissue biopsy if the site is sterile. If the site is not sterile microscopy and culture of

an NTM in the absence of other more culpable pathogens may indicate NTM infection.[2] In a non-sterile site histological findings of granulomatous changes can support the diagnosis.

Diagnosis of NTM Lymphadenitis

When investigating lymphadenitis in the NT it is important to remember TB lymphadenitis is a much more common disease in an Aboriginal child. A definitive diagnosis of NTM lymphadenitis is made by recovery of the causative organism from lymph node cultures. Fine needle aspirate (FNA) or complete excision of the lymph node is recommended for diagnosis rather than biopsy or incision and drainage as these procedures may be followed by fistulae formation with chronic drainage. However, even with an FNA or excised node and compatible histopathology, only about 50% and 50-80% respectively will yield positive cultures.[2] See footnote* History should involve questioning about exposure to patients with TB. A CXR should be taken looking for signs of *M. tuberculosis* disease or past infection. Dual skin testing with Tuberculin purified protein derivative (PPD) human and Avian Tuberculin PPD may contribute to a working diagnosis and empirical treatment if cultures are negative.

Treatment

Because of the extensive list of NTM causing disease only treatment for the more common forms are discussed here. Treatment and outcomes for other types and for HIV positive patients will need to be discussed with a specialist. O'Brien et al reported treatment and outcomes of the 1989 to 1997 case series in the NT.[1]

MAC pulmonary disease in HIV negative adults

Though there are no definitive trials to guide duration of treatment, generally treatment should continue until the patient is culture negative for 10 to 12 months. Sputum should be tested monthly. Clinical improvement is expected

Figure 3. Treatment regimen for NTM pulmonary disease in HIV negative adults[§]

- Clarithromycin*: 500mg twice a day or
- Azithromycin*: 250mg/day *or* 500mg three times/week

AND

- Rifabutin: 300mg/day or
- Rifampicin: 600mg/day

AND

- Ethambutol: 25mg/kg/day for 3 months *then* 15mg/kg/day

+/-

- Intermittent Streptomycin for extensive disease for the first two to three months of treatment (see Table 5 for dosing schedule).

[§] This regimen and its doses have not been validated in paediatric patients

*Patients with small body mass and/or >70 years clarithromycin at 250mg twice per day or azithromycin 250mg three times per week may be better tolerated

[*] Two FNA passes should be performed, one fixed on a slide for cytology and one for culture. FNA specimens for culture should be placed in a sterile container with a few drops of sterile saline on top. Lymph node biopsies should be placed in an empty sterile container. Unfortunately often FNA and lymph node biopsy specimens are not cultured because they are fixed onto a slide or placed in formalin immediately after collection. A reminder to the specimen collector, to place a specimen in saline, prior to the procedure may avoid this problem.

within 3 to 6 months. Sputum should clear within 12 months. Failure to improve or to clear sputum should warrant checks on compliance and sensitivity. Post treatment the patient should be followed and have sputum samples tested to check for relapse. One sputum sample should be collected at 4, 5, and 6 months post treatment and then 3 sputums a week apart at 18 months. If one of the sputum samples collected at 4, 5 or 6 months is negative then it should be followed by another 2 sputum samples at weekly intervals. The patient should be reviewed clinically at 6 and 18 months. Patients with disease localised to one lung and who can tolerate resectional surgery may be candidates for surgery if there has been poor response to drug therapy.[2]

Table 5. Suggested doses of streptomycin relative to age and weight in patients with normal serum creatinine

Weight and age	Initial therapy †	Maintenance therapy ‡
≥50 kg and ≤50 yrs	1g 5x/wk	1g 3x/wk
<50 kg and ≤50 yr	500mg 5x/wk	750mg 2x/wk
>50 kg and 50-70 yr	500mg 5x/wk	750mg 2x/wk
>70yr	750mg 2x/wk	750mg 2x/wk

† For the first 6 to 12 weeks of therapy as tolerated
‡ For subsequent therapy as tolerated

Disseminated MAC disease

Drug combinations will be as those for HIV negative patients with pulmonary disease. MAC prophylaxis should be strongly considered in high-risk patients and therapy should be offered to all patients with established disease.[2]

Skin/soft tissue/bone disease

The most common cause of skin and soft tissue disease in the NT has been *M. fortuitum*. In general with rapidly growing mycobacteria some minor infections will resolve spontaneously or after surgical debridement. For the more common types causing infection, *M. fortuitum, M. abscessus and M. chelonae*, no controlled trials of pharmacological treatment have been performed. However patients with cutaneous disease, due to rapidly growing NTM, treated on the basis of in vitro susceptibilities have shown good results.[2]

Because of variable drug susceptibility among rapidly growing NTM species susceptibility testing of all clinically significant isolates is essential for good management. A primary panel of drugs for testing could include amikacin, cefotoxin, ciprofloxacin, clarithromycin, doxycycline, imipenem and a sulphonamide.[2]

Several studies of post injection abscess in which no therapy was given revealed disease

Table 6. Treatment plan for NTM pulmonary disease (disseminated disease - should follow the same plan except CXR and sputum may not be necessary if there is no pulmonary involvement).

Pre-treatment	Treatment – monthly	Treatment – 3 monthly	Post treatment
HIV Glucose LFTs FBC U&Es Vision* Weight	Symptoms – NTM disease and side effects Compliance Weight Vision* Sputum§ LFTsϕ FBCϕ U&Esϕ	First 3 months-stop streptomycin CXR three-monthly	Sputum at 4, 5, 6 and 18 months. If any are negative then collect a further 2 samples at weekly intervals.

*Visual acuity and colour discrimination
§ If the sputum is AFB smear negative then a week and two weeks later a further sputum sample should be collected.
ϕMonthly for the first three months then reassess the need for monthly collection.

that persisted for eight to twelve months before spontaneously resolving. For serious disease a minimum of 4 months of treatment is recommended and for bone disease 6 months of treatment is recommended. Surgery is generally indicated with extensive disease, abscess formation or where drug therapy is difficult.[2]

Lymphadenitis due to MAC

Excisional surgery without chemotherapy is recommended for children with NTM cervical lymphadenitis. For children with recurrent disease a second surgical procedure is usually performed. When surgery is contraindicated the use of a clarithromycin multi drug regimen such as those used for pulmonary disease is recommended, however ethambutol should not be used in children who cannot reliably report loss of visual acuity[2].

New treatments on the horizon

Oxazolidinones are a new class of antibiotic active against gram positive bacteria. Linezolid, one of the first oxazolidinones to be registered for use, has been shown to be effective in vitro against rapidly growing mycobacteria[35]. Limited research has shown it to be successfully in treating MAC disseminated skin disease[36]. Further research is necessary before its use can be widely recommended in NTM disease.

Figure 4. Treatment regimen for serious skin, soft tissue or bone disease caused by *M. fortuitum* and *M. abscessus*

- Amikacin 10-15mg/kg IV in two divided doses (average 400mg BD) in patients with normal renal function*.
- Cefoxitin 12g/day IV is recommended for initial therapy (min 2 weeks) until clinical improvement is evident**.**

*10mg/kg should be used in patients over the age of 50; once daily dosing is unproven clinically but appears reasonable.

Table 7 Drug side effects and interactions[37]

SIDE EFFECTS		COMMON DRUG INTERACTIONS	
CLARITHROMYCIN			
▪ GI upset ▪ Hepatitis ▪ Headache ▪ Dizziness ▪ Rash ▪ Steven-Johnson Syndrome ▪ Psychiatric and CNS effects ▪ Pseudomembranous colitis ▪ Altered taste	**Precautions** ▪ Duodenal ulcer ▪ Renal impairment ▪ Elderly ▪ Pregnancy ▪ Lactation ▪ Children ▪ Immunocompromised	**Contraindicated with:** ▪ Astemizole ▪ Terfenadine ▪ Cisapride ▪ Pimozide Increases the levels of: ▪ Theophylline ▪ Anticonvulsants ▪ Warfarin ▪ Ergot alkaloids ▪ Benzodiazepines ▪ Disopyramide ▪ Digoxin ▪ Tacrolimus ▪ Cyclosporin ▪ Rifabutin	▪ Cilostazol ▪ Methylprednisolone ▪ Quinidine ▪ Sildenafil ▪ Vinblastine Decreases levels of: ▪ Zidovudine Clarithromycin levels increased by: ▪ Fluconazole ▪ Fluoxetine ▪ Retinovir Rhabdomyolysis reported when taken with: ▪ Simvastatin ▪ Lovastatin

SIDE EFFECTS		COMMON DRUG INTERACTIONS	
AZITHROMYCIN			
▪ GI upset ▪ Superinfection ▪ Pseudo-membranous colitis ▪ Vaginitis ▪ Angiooedema ▪ Cholestatic jaundice ▪ Hepatitis ▪ Hearing impairment	**Precautions** ▪ Renal impairment ▪ Elderly ▪ Pregnancy ▪ Lactation ▪ Severe hepatic impairment	Increases the levels of: ▪ Ergot derivatives ▪ Cyclosporin ▪ Digoxin ▪ Zidovudine ▪ Terfenidine ▪ Astemizole ▪ Warfarin	Decreases levels of: ▪ Zidovudine Levels decreased by: ▪ Antacids
RIFAMPICIN			
▪ Pink/orange -urine -sweat -tears (stains contact lenses) ▪ Nausea ▪ Vomiting ▪ Pseudo-membranous colitis ▪ Hepatitis ▪ Rash ▪ Drug induced fever ▪ CNS disturbance With prolonged unscheduled breaks: ▪ Shock ▪ Acute renal failure ▪ Thrombocytopenia purpura ▪ Haemolytic anaemia ▪ Shortness of breath ▪ Flu like syndrome (myalgia, arthralgia, fever, malaise, mild haemolysis)	**Precautions:** ▪ Hepatic impairment ▪ Malnourishment ▪ Sodium metabisulphite allergy ▪ Diabetes ▪ Pregnancy ▪ Lactation ▪ Premature and newborn infants ▪ Porphyria	Antacids may reduce absorption. Rifampicin causes decreased activity of: ▪ Oral anticoagulants ▪ Anticonvulsants ▪ Antiarrhythmics ▪ Antifungals ▪ Barbiturates ▪ Benzodiazepines ▪ β blockers ▪ Calcium channel blockers ▪ Chloramphenicol ▪ Clarithromycin ▪ Corticosteroids ▪ Cyclosporin ▪ Cardiac glycosides (digoxin) ▪ Clofibrate ▪ Hormonal contraceptives ▪ Dapsone ▪ Enalapril	▪ Doxycycline ▪ Fluoroquinolones ▪ Oral hypoglycaemics ▪ Levothyroxine ▪ Narcotic analgesics ▪ Methadone ▪ Quinine ▪ Tacrolimus ▪ Theophyline ▪ Tricyclic antidepressants ▪ Zidovudine When taken with Rifampicin: ▪ PAS decreases Rifampicin levels. ▪ Atovaquone levels increase and Rifampicin levels decrease. ▪ Halothane may cause hepatotoxicity
RIFABUTIN			
▪ Pink/orange ▪ -urine ▪ -sweat ▪ -tears (stains contact lenses) ▪ Nausea ▪ Vomiting ▪ Hepatitis ▪ Rash ▪ Drug induced fever ▪ Myalgia ▪ Arthralgia ▪ Uveitis ▪ Cytopenias	**Precautions:** ▪ Hepatic impairment ▪ Severe renal impairment ▪ Pregnancy ▪ Lactation ▪ Children	Rifabutin causes ↓ activity of: ▪ Oral anticoagulants ▪ Anticonvulsants ▪ Antiarrhythmics ▪ Antifungals ▪ Barbiturates ▪ ß blockers ▪ Calcium channel blockers ▪ Chloramphenicol ▪ Clarithromycin ▪ Corticosteroids ▪ Cyclosporin ▪ Cardiac glycosides (except digoxin) ▪ Atovaquone ▪ Bactrim ▪ Cisapride	▪ Lignocaine ▪ Terfenadine ▪ Erythromycin ▪ Lovastatin ▪ Hormonal contraceptives ▪ Dapsone ▪ Benzodiazepines ▪ Oral hypoglycaemics ▪ Narcotic analgesics ▪ Methadone ▪ Quinidine ▪ Tacrolimus ▪ Antiretrovirals Many of the above drugs along with fluroquinolones. can increase rifabutin levels

SIDE EFFECTS		COMMON DRUG INTERACTIONS
ETHAMBUTOL		
▪ Optic neuropathy - Loss of visual acuity - Loss of red green discrimination ▪ Nausea ▪ Vomiting ▪ Hepatitis ▪ Rash ▪ Arthralgia ▪ Peripheral neuropathy	**Precautions:** ▪ Renal impairment ▪ Visual defects	None noted
AMIKACIN AND STREPTOMYCIN		
▪ Auditory and vestibular impairment ▪ Renal impairment ▪	**Precautions:** ▪ Renal impairment **Contraindications:** ▪ Pregnancy ▪ Lactation	The neurotoxic and nephrotoxic potential of this drug is increased by other Aminmoglycosides, some diuretics, some anaesthetic and neuromuscular blocking drugs
CEFOXITIN		
▪ Superinfection ▪ Pseudomembraneous colitis ▪ Phlebitis ▪ Local pain ▪ Hypotension ▪ Blood abnormalities ▪ Hypotension ▪ GI upset ▪ Hepatitis	**Precautions** ▪ Renal or hepatic impairment ▪ Meningitis ▪ Brain abscess ▪ Pregnancy ▪ Lactation ▪ Neonates ▪ Premature infants	None noted

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Medically important insects in the Northern Territory and how disasters may affect them

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Introduction

Insects are the largest class in the animal kingdom. They belong to the Phylum Arthropoda, which also includes spiders, crustaceans, ticks, centipedes and millipedes. All members of the phylum are characterized by having a segmented body and segmented legs. Insects and some other arthropods are the vectors of some of the most destructive diseases in the world, some of which are listed in Table 1. The vectors of disease that are of relevance to Australia are mosquitoes, flies, ticks and mites. Of these, mosquitoes are considered to be the most important.

Background

Mosquito borne diseases of Australian significance

Malaria

Malaria is a debilitating disease caused by protozoa of the genus *Plasmodium*. There are 120 different species of *Plasmodium*, of which *P. vivax, P. falciparum, P. malariae* and *P. ovale* infect humans. Depending on the species of *Plasmodium,* symptoms of malaria include fever, chills, sweats and headache. In *P. falciparum* it progresses to blood coagulation, shock, renal and liver failure, coma and death. *Plasmodium* undergoes its sexual cycle in mosquitoes and asexual cycle in humans. Malaria in nature is only transmitted to humans through the bite of *Anopheles spp.* mosquitoes.

Endemic malaria last occurred in Australia in the Northern Territory (NT) in 1962. Australia has been certified free of endemic malaria since 1981. However, there is still potential for malaria transmission from people infected in malarious countries. Local transmission is only possible where known vectors occur, generally north of 19°S. In northern Australia, any of the 3 sibling species of *Anopheles farauti* are considered to be the primary potential malaria vector.

Dengue fever

Dengue fever (DF), and dengue haemorrhagic fever (DHF) results from infection by any of the

Table 1. Arthropod vectors of human diseases (after Sutherst, 1993).

Vector	Disease	Pathogen
Mosquitoes *(Anopheles spp.*)	Malaria	*Plasmodium spp.*
Mosquitoes *(Aedes spp.*)	Dengue fever Yellow fever	Dengue virus Yellow fever virus
Mosquitoes *(Aedes, Ochlerotatus* and *Culex spp.*)	Encephalitides Ross River Virus Disease	Various viruses Ross River virus
Sand-flies *(Phlebotomus)*	Leishmaniasis	*Leishmania spp.*
Triatome bugs	Chagas' disease (American trypanosomiasis)	*Trypanosoma cruzi*
Tsetse flies (*Glossina*)	African Trypanosomiasis	*Trypanosoma spp.*
Black flies (*Simulium*)	Onchocerciasis	*Onchocerca*
Mosquitoes (*Culex, Anopheles, Mansonia* and *Ochlerotatus spp.)*	Lymphatic filariasis	*Wuchereria spp., Brugia spp.*
Ticks	Tick typhus Lyme disease	*Rickettsia spp.* *Borrelia burgdorferi*
Mites	Scrub typhus	*Orientia tsutsugamsushi*
House flies & Blow flies	Food poisoning	*Shigella, Salmonella, Escherichia coli*

4 serotypes of dengue virus. Patients suffering from classic dengue fever experience a sudden onset of fever, headache, retro-orbital pain, arthralgia, myalgia and minor haemorrhaging. Manifestations of DHF include severe haemorrhage from vascular permeability leading to shock through blood loss and encephalopathy with hepatitis. Dengue was common in northern Australia earlier in the twentieth century with epidemics occurring in Queensland, the NT and New South Wales. All available evidence suggests that dengue is not endemic in Australia, although outbreaks do occur in Queensland from virus introduced by viraemic travelers.

Dengue virus transmission occurs through the bite of infected *Aedes* mosquitoes, of which *Aedes aegypti* is the only vector in Australia. *Aedes aegypti* is not present in the NT but it and the other dengue vector, the Asian tiger mosquito *Aedes albopictus*, have been occasionally imported and eradicated. There is a continuous risk of these dengue vectors being introduced to the NT.

Murray Valley encephalitis

Murray Valley Encephalitis (MVE) is caused by a flavivirus, first isolated from a fatal human case from the Murray valley of northern Victoria in 1951. Most human infections are subclinical, with a clinical to sub-clinical ratio varying from 1:500 to 1:5000. The presentation of MVE cases is variable but major symptoms include sudden onset with fever, nausea, headache, vomiting and non-specific dizziness, followed by symptoms of meningitis and brain dysfunction. Approximately one third of cases are fatal, with one third of survivors suffering neurological sequelae with physical and/or intellectual handicap. Since 1974, as many as 50 clinical cases of MVE have been recorded from northern Australia, with the majority from the north of Western Australia and the NT.

Humans are infected by mosquitoes, which acquire the virus from a vertebrate reservoir. Major vertebrate hosts of MVE are waterbirds (particularly night herons), although a range of avian (including ducks and parrots) and mammalian (such as kangaroos and rabbits) vertebrates have been implicated on serological grounds. Extensive field studies have confirmed that *Culex annulirostris* is the major vector of MVE in Australia, accounting for more than 90% of isolates.

Japanese encephalitis

Japanese encephalitis (JE) is a mosquito-borne arboviral disease of major public health concern in Asia, responsible for 35,000 cases and 10,000 deaths annually. In 1995 an outbreak of JE resulted in three cases at Badu Island in the Torres Strait, indicating the first incursion of this disease into Australia. Infection with JE virus results in similar clinical symptoms as MVE, although the clinical to subclinical ratio ranges from 1:25 to 1:400. Fortunately, a JE vaccine exists, which gives up to 5 years protection.

During the Badu Island outbreak an extensive mosquito trapping program resulted in the isolation of JE virus from *Culex annulirostris,* implicating this species as the most likely Australian vector. Potential vectors also include *Culex palpalis* and the recently established *Culex gelidus*. Wading birds and pigs are the principle vertebrate amplifying hosts, while humans are dead-end hosts. Active surveillance is currently being undertaken to determine when JE is introduced or becomes established on the Australian mainland. Risk areas in the NT are the ones closest to Papua New Guinea or East Timor.

Ross River virus disease (formerly epidemic polyarthritis)

Ross River (RR) virus, is the aetiological agent for Ross River virus disease, the most common arboviral disease in Australia. RR virus has been reported from all Australian states including Tasmania, with several hundred to several thousand cases reported annually. In northern and central Queensland and the Top End of the NT, particularly in coastal regions, the virus is active throughout the year. In the Top End of the NT the principle transmission season is from December to March inclusive. Elsewhere in Australia, virus activity tends to be epidemic, following summer rain and/or tidal inundation of saltmarsh in coastal regions of Australia. The major clinical features include various combinations of arthralgia and arthritis, usually involving joints of the extremities, myalgia, lethargy, a maculopapular rash, headache and (less often) fever. RR virus infection is often subclinical, particularly in adolescents, with as many as 80% of cases being asymptomatic.

RR virus is sustained in a mosquito-mammal cycle, with kangaroos, wallabies and flying foxes being the main vertebrate hosts. There are indications that humans may act as primary hosts in epidemic situations and serve to distribute the virus geographically. So far, at least 30 species of mosquitoes have been shown to carry RR virus in nature. The most important species associated with RR virus in the NT are: *Verrallina funerea, Ochlerotatus notoscriptus, Ochlerotatus vigilax, Culex annulirostris* and *Ochlerotatus normanensis.* In the Top End of the NT *Ochlerotatus vigilax and Culex annulirostris* are the major vectors, while in inland areas *Ochlerotatus normanensis* and *Culex annulirostris* are the major vectors.

Barmah Forest virus disease

Barmah Forest (BF) virus was originally isolated from *Culex annulirostris* collected in the Barmah Forest on the Murray River in northern Victoria. BF virus and RR virus infection exhibit similar symptomatology. However, BF virus causes significantly more rash than RR virus, while arthralgia and arthritis are less common than with RR virus. Again, like RR virus, BF virus has a high ratio of subclinical to clinical infection.
Sporadic cases of BF infection have been reported Australia wide, with most of the cases in the NT occur in the Top End from December to March. The first major Australian outbreak of BF was recorded in the NT at Nhulunbuy in February 1992.

Very little is currently known about the ecology and epidemiology of BF virus. The vertebrate hosts of BF virus are yet to be elucidated, although marsupials, cattle, horses and waterbirds have been implicated as a source of infection. The major mosquito vectors of BF virus are considered to be *Ochlerotatus vigilax* and *Culex annulirostris.*

Biology of important vector and pest mosquito species

Apart from their ability to transmit disease, mosquitoes can be a considerable nuisance in some areas. Of the species that are important vectors of disease in the NT, *Verrallina funerea, Ochlerotatus vigilax, Ochlerotatus normanensis* and *Culex annulirostris* can proliferate after high tides, heavy rainfall or flooding associated with cyclones and floods. Others such as *Aedes aegypti* could become introduced to the NT or prolific following cyclones and widespread rain filling exposed receptacles such as drains and tyres.

***Aedes aegypti* (Not present in the NT)**

Aedes aegypti is a peridomestic mosquito that breeds in artificial water containers such as tyres, ice cream containers and pot plant bases, and occasionally in natural receptacles such as leaf axils of *Bromeliads* and palm fronds. *Aedes aegypti* is predominantly a day biter, being most active in the early morning (0600 hr-0800 hr) and late afternoon (1500 hr-1800 hr). *Aedes aegypti* has a preference for human blood and is found frequently biting and resting inside buildings.

Verrallina funerea

Verrallina funerea is a recently discovered RR virus vector, which can be major pest in areas close to *Melaleuca* swamps or mangroves. This species breeds in slightly brackish to fresh pools, usually well shaded, in swampy areas of *Melaleuca* and sedges adjoining tidal areas. *Verrallina funerea is* a common daytime biter, with activity also on dusk. It rarely bites outside of well-shaded areas during the day.

Ochlerotatus notoscriptus

Ochlerotatus notoscriptus is arguably the major domestic pest species in Australia. It breeds in artificial containers, such as tyres, pot plant drip trays, roof gutters and boats. Its habitat is similar to *Aedes aegypti. Ochlerotatus notoscriptus* readily bites indoors or outdoors throughout the day, with peak activity at dusk.

***Ochlerotatus vigilax* (Saltmarsh mosquito)**

Ochlerotatus vigilax (formerly called *Aedes vigilax*) is the main coastal vector of RR virus in northern and north-western Australia. Most commonly found breeding in temporary, brackish water left in saltmarsh or mangrove pools or shallow depressions on tidal flats by higher than normal tides or rainfall. *Ochlerotatus vigilax* usually bites outdoors at sunrise and sunset but will bite throughout the day in shaded

areas within 5-10km of coastal breeding sites. It is more common in the Top End from August to January, usually reaching a peak in the December to January period.

Ochlerotatus normanensis

Ochlerotatus normanensis is usually associated with drier, sparsely populated areas (i.e. outback), where it can become an abundant pest nuisance at times. This northern species breeds in a variety of freshwater breeding sites, ranging from temporary ground pools and animal footprints to large temporary pools. *Ochlerotatus normanensis* will readily bite during the afternoon and night.

***Anopheles farauti* (Australian malaria mosquito)**

Anopheles farauti breeds in semi-permanent brackish or freshwater sites, such as swamps, lagoons or ponds, usually with emergent vegetation. *Anopheles farauti* is predominantly a night biter (1900 hr-0030 hr) with a peak for the first hour after sundown. It bites both inside and outside houses.

***Culex annulirostris* (Common banded mosquito0**

Culex annulirostris is the major vector of arboviral disease in Australia, particularly in inland areas, where proliferation of this species occurs after summer rain. Typical *Culex annulirostris* breeding habitats include freshwater swamps, lagoons and transient grassy pools, as well as saltmarsh pools, irrigation channels, raw sewage effluent and artificial containers. *Culex annulirostris* is mainly a nocturnal biting species, with peak activity during the first hour after sundown (1700 hr-2000 hr) and a small peak before dawn (0530hr-0630 hr).

Culex gelidus

Culex gelidus is a new introduction to Australia and is currently in Queensland and the NT. It is found in Indonesia, Papua New Guinea and East Timor and other areas of Asia where it is regarded as a vector of JE. In the NT it is widely distributed from Darwin to Alice Springs, breeding in freshwater ground pools, marshes and containers, with productive sites in wastewater ponds with high organic levels. It is found mainly breeding during the wet season. If *Culex gelidus* breeds in close association with pigs, it could play a major role in JE transmission in Australia. It is reported to bite humans readily, particularly in the absence of other animals.

Culex palpalis

Culex palpalis is a potential vector of MVE and other arboviruses. It breeds in situations similar to *Culex annulirostris,* although generally it is found more often in clear freshwater swamps. It is widespread in the NT and is often found in high numbers in sub coastal swamps in the latter wet season and early dry season. It bites humans readily after sundown and can reach very high population levels.

Other insect species

Houseflies and BlowFlies.

Domestic flies can occur in pest numbers throughout the year in the Top End of the NT, with a tendency for higher populations in the wet season, when suitable breeding materials are kept moist and soil can be more suitable for maggot entry and survival. Populations of flies can be annoying but more importantly they play a role in disease transmission.

Domestic flies carry disease-causing organisms in a number of ways. This includes:

- On their mouthparts.
- Through their vomit.
- On their body and legs
- On the sticky pads on their feet.
- Through their intestinal tract to faeces.

Diseases transmitted mechanically by domestic flies include typhoid, cholera, bacillary dysentery, infantile diarrhea, amoebic dysentery, giardiasis, pinworm, roundworm, whipworm hookworm and tapeworms. This includes transmission of organisms such as *Salmonella typhimurium* and *Shigella spp.*, which cause food poisoning. Recently enterohaemorrhagic *Escherichia coli* have been shown to proliferate in houseflies and to be excreted by the flies.

The house fly *Musca domestica* and the green/ blue blow fly *Chrysomyia megacephala* are the

most common problem domestic flies in the Top End of the NT.

The housefly breeds in a wide range of garbage and is the most likely species to cause a nuisance and lead to food spoilage inside a house. It commonly breeds in wheelie bins in poorly packaged garbage. It also breeds in prolific numbers in horse dung and moist chicken manure.

Over a few days, a female housefly can lay 4 to 6 batches of eggs (each batch about 20 eggs). The eggs hatch in about 12 hours and the maggot stage takes about 5 days. The full-grown maggot migrates to a drier area or enters the soil to develop into a pupa. Pupae are often found in wheelie bins that have missed a collection. The pupa stage lasts about 4 days before the adult fly emerges. The development from egg to adult takes about 9 to 10 days in summer conditions. The adult flies feed on a great variety of materials such as faeces, meat, sugar, milk, or any other foodstuff. The fly vomits and deposits faeces on food and in this way can spread disease.

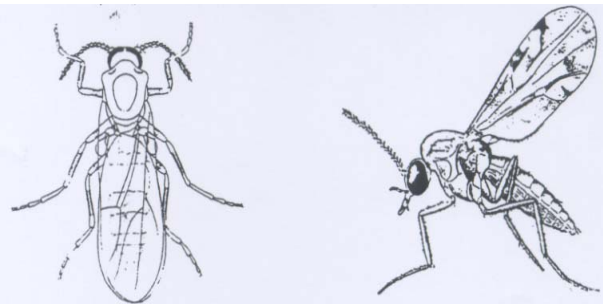
The blowfly tends to be less common inside houses but is more obvious because of its larger size and very active flight habits. It breeds mainly in meat products, in other garbage with a high protein content, and also on dead animals. Common breeding places including wheelie bins with unpackaged garbage, waste pet food, discarded bones, and dead rats.

Blowflies can lay from 100 to 400 eggs in a single batch and the eggs hatch in about 8 hours. After hatching, maggots can take as little as 3 days to mature and reach the wandering stage. Over 5000 mature maggots can be produced from 300 grams of food garbage in a single wheelie bin under Darwin conditions. Wandering maggots crawl out of garbage storage areas at night and then enter moist soil, where they pupate and can emerge as adults 5 to 7 days later. The adult flies are attracted by smell and feed on a variety of materials such as faeces, blood, sugar, milk, or any other foodstuff.

Biting Midges (Culicoides *spp.*)

Biting midges (often erroneously called sandflies) are small (less than 2mm), relatively robust looking flies, that are annoying pests of man and domestic animals (Figure 1). They usually breed in wet soil in the zone between aquatic and terrestrial habitats, such estuarine mud in mangroves (*Culicoides ornatus*), freshwater pond margins (*Culicoides marksi*) and wet soils with a high content of animal wastes (*Culicoides brevitarsis*).

Figure 1. Lateral and dorsal view of a female *Culicoides spp.*



The eggs are laid in batches (30-450 eggs depending on the species) and hatch within a few days. The larva, which emerges from the egg, feeds on a wide range of organisms, which they encounter in their movement through the substrate. There are four larval instars, which last anywhere from 2-3 weeks to 2-3 months (depending on the species). The larva then pupates and after several days, the adult emerges.

The adult *Culicoides* only disperse a short distance from their breeding sites (usually less than 1.0 km but up to 3.0 km for *Culicoides ornatus*). *Culicoides* generally feeds at dusk (1800 hr-2000 hr) and dawn (0600 hr-0800 hr), although biting can be experienced throughout the day near breeding sites. The bites from *Culicoides* cause itching and, in sensitive individuals, welts and lesions that may persist for several days. These are sometimes complicated by secondary infections resulting from scratching.

Disasters of relevance to medically important insects

It should be noted that disease outbreaks following a disaster are rare, as many of the diseases, such as malaria and dengue do not occur in Australia unless imported by travelers. Other diseases such as MVE and JE only have a

small and often isolated foci of activity. It is possible that transmission of RR virus and BF virus could increase, although appropriate self protection and control measures should limit this threat. Houseflies and blowflies can cause diseases which can be life threatening for old people and young children. In Australia biting midges are not vectors of any human disease, although they can cause considerable irritation through their bites.

There are several disaster scenarios that can directly result in an increase in the potential for vector and pest insects to become a menace to the human population. In situations that involve widespread flooding, *Aedes* or *Ochlerotatus spp.* eggs usually hatch first, as they have drought resistant eggs in ground depressions or receptacles such as tyres. *Anopheles spp.* and *Culex spp.* lay their eggs on the waters surface, where they undergo development for a further 1-3 days before hatching into larvae. House flies and blowflies can breed in spoiled food, dead animals or wet animal faeces.

The disaster situations that are applicable to mosquitoes and other flies are:

i) Cyclones with destructive winds and flooding.

Flooding from cyclones can come in two forms: a) the associated torrential rainfall and b) possible storm surge created by a significant rise in tides. In wet tropic areas, wet season rainfall has usually flooded most mosquito breeding sites, so that additional flooding will not increase the extent of mosquito breeding. However, in periods of dry weather or in drier areas (such as Katherine to Alice Springs), flooding associated with cyclones could produce significant numbers of *Ochlerotatus normanensis* and *Culex annulirostris.* Extensive local flooding could have also the beneficial effect of flushing out breeding sites. However as waters recede, mosquitoes will colonize the pools as they dry up. Mosquitoes hatched by flooding associated with cyclones would emerge within 1-2 weeks depending on the temperatures. House flies and blowflies can begin breeding in 1-2 days after a disaster and need control within 4-5 days before they reach the wandering maggot stage and become difficult to treat with insecticides. As biting midges have a long larval stage, significant increases in *Culicoides* populations would not be expected for up to 2-3 weeks after the cyclone, although excessive flooding will not produce higher numbers of *Culicoides.*

Apart from the flooding of breeding sites for mosquitoes, strong winds associated with cyclones could cause widespread damage to housing and other structures. Insect screens, walls and roofs could be damaged allowing increased access of mosquitoes, biting midges and flies into otherwise protected houses. Structural damage of water supply and sewage drainage will provide a wide range of habitats for container breeding species such as *Aedes aegypti, Ochlerotatus notoscriptus* and *Culex quinquefasciatus* (a nocturnal biter that breeds in polluted water), near human habitation.

Severe cyclones could require the relocation of residents to temporary housing in tents, schools or community halls that without adequate screening, would expose a large number of people to mosquito and biting midge bites and flies.

ii) Flooding of coastal rivers, creeks and floodplains

Torrential rain, causing flooding of coastal rivers, creeks and floodplains (e.g. Daly, Mary, South and East Alligator Rivers and around the Roper River) may result in a large hatch of mosquitoes in 1-2 weeks after the waters abate. Typically, flooding of coastal rivers and swamps creates localized flooding, so damage to residential areas will not be as widespread as that encountered after a cyclone. The mosquitoes hatched during these floods would be mainly *Ochlerotatus vigilax* and *Culex annulirostris.*

iii) Flooding of inland rivers

The flooding of inland rivers and low lying areas (e.g. Roper, Katherine and the Todd River) would result in large hatchings of mosquitoes.

There are 3 separate types of flooding of inland rivers:

a) Heavy rains in the upper catchment causing flooding downstream, resulting in the production of large numbers of mosquitoes. The flushing of the streams may result in reduction of mosquito

breeding sites in the upper catchment and extreme mosquito breeding where flooding occurs (e.g. Katherine River).

b) Local heavy rains causing flooding of low country with only minor stream rises. This would stimulate mosquito breeding in temporary swamps and storm drains (e.g. around Tennant Creek).

c) General heavy rain causing high stream flow plus local flooding. Under these conditions, large numbers of mosquitoes are produced over wide areas (e.g. Barkly, VRD and Central Australia Regions).

Mosquitoes would not be expected to become a problem with inland flooding until the water level has dropped, and isolated pools without wave motion form (Figure 2). Mosquito species that could potentially reach plague proportions after inland flooding include *Ochlerotatus normanensis* and *Culex annulirostris.*

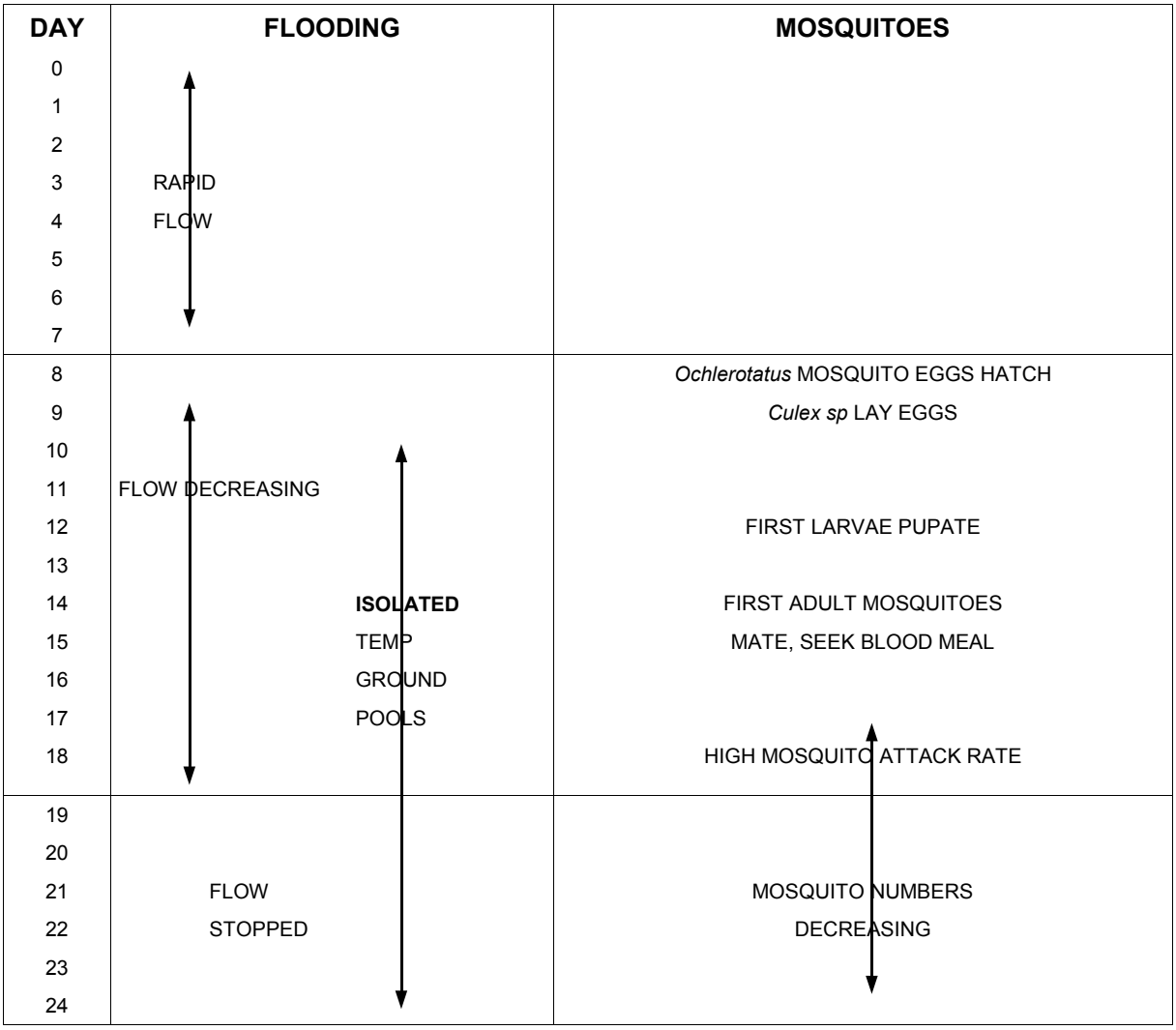
Control of mosquitoes and flies associated with disaster

Each disaster will produce a unique mosquito problem, so no strict set of guidelines for control will apply. Instead, flexibility and competent planning must be done before, during and following a disaster situation.

In preparation for the disaster

Local councils should periodically undertake adult and larval mosquito surveys to locate potential mosquito breeding sites, and identify particular species that may become a pest in the advent of flooding of breeding sites. Appropriate larval and adult control strategies can then target these specific areas. Local Government Authorities should liaise with Environmental Health and Medical Entomology personnel to assist in mosquito surveys. Any reports of

Figure 2. Chronology of mosquito and blackfly production following flooding.



previous mosquito outbreaks can provide valuable information on potential breeding sites, adult behavior and control methods employed.

Local councils should also ensure they have sufficient equipment and supplies to cope with any outbreak of mosquitoes or flies. Councils should have access to necessary pesticides and pesticide application equipment, repellents, maps of affected areas, etc. It is important to have a resource list available to allow rapid identification of deficiencies in equipment and other resources. If the situation overwhelms the resources of the Local Authority, the Medical Entomology and Environmental Health Branches of Department of Health and Community Services should be contacted to provide consultation and support on appropriate mosquito prevention strategies. Additional support and assistance can be provided by other Local Government Authorities, who may possess the necessary equipment, personnel and expertise to deal with problems such as drainage or rubbish collection. Larger councils may have the necessary mosquito control equipment, such as vehicle-mounted foggers or all terrain vehicles, which are ideal for surveys in outbreak situations. It is therefore necessary for smaller local authorities to liaise with larger councils in their region, to organize any support that may be required during an outbreak following a disaster.

During the disaster

During the disaster and in the first day or 2 after, mosquitoes are not a priority, so resources should be concentrated on other issues. Again, refer to Figure 4 for the chronology for the production of mosquito populations. However, preparation for larviciding and adulticiding can be done. In addition, contact should be made with relevant authorities for support and assistance.

After the disaster

Mosquito control

- **Larval control**

  Mosquitoes will not become a problem until a week after the floodwaters recede. Known mosquito breeding sites, as well as any new sites created by flooding or torrential rains, should be surveyed and treated with appropriate larvicides.

  Larvicides that will provide adequate control include:

  - **Methoprene (trade name Altosid®)**

    Methoprene is what is known as an insect growth regulator. It is a synthetic juvenile hormone that prevents the mosquito larva from maturing into an adult mosquito. Altosid® formulations include liquid, sand, granules, pellets and extended residual briquettes, each with different applications and control periods. When used as directed Altosid® products do not harm mammals, waterfowl and other non-target organisms. It is approved by the World Health Organization to control container-breeding mosquitoes in drinking water receptacles. It should be noted that due to the mode of action of methoprene, larvae would not actually die. Instead pupae should be sampled and emergence inhibition monitored.

  - ***Bacillus thuringiensis* var. *israelensis* (trade names Vectobac®, Skeetal® and Teknar®)**

    Bacillus thuringiensis var. *israelensis* (or B.T.I.) is a bacteria (actually crystalline spores and protein byproducts) that kill mosquito larvae. B.T.I. is ingested by the mosquito larva where it destroys the midgut epithelium, causing paralysis and death within 24 hours. The most common formulation of B.T.I. is a liquid suspension that can be applied through vehicle mounted boom sprayers, hand-operated pump sprayers and Ultra Low Volume (ULV) misters.

  - **Temephos (trade name Abate®)**

    Abate is an organophosphate compound that acts as a nerve poison. Temephos contains esters of phosphoric acid, which inactivate acetylcholine esterase, an enzyme that is essential for nerve transmission. There are three formulations of Abate®, a 10 SG sand granule, 50 SG sand granule and 100 E emulsifiable concentrate. Unfortunately, widespread use of Abate® has led to resistance in some mosquito species in other areas. Coupled with this has been the toxicity of Abate" to non-target organisms such as crustaceans. Because

of these concerns, it is recommended that either B.T.I. or methoprene be used for most larval control, and temephos only be applied where late fourth instar larvae occur in high concentrations and there are no appreciable non-target animals.

- **Adult Control**

  Adult mosquitoes will not emerge until at least one week after the flood waters recede. Biting midges will emerge at least three weeks after the flooding abates. Again, this will depend on the species present. Adult control of mosquitoes and biting midges is carried out by either a space spray, which kills flying insects or residual spraying which kills resting adults.

  There are currently three types of chemicals that are available for adult mosquito and biting midge control.

  - **Natural pyrethrins (trade names Drift® and Chemfog®**)

    This chemical is extracted from the flowers of the Chrysanthemum plant. These chemicals enter the mosquito through the cuticle. Once inside the body they act as nerve poisons, by preventing the transmission of nerve impulses. The advantages of natural pyrethrins is that they have rapid insecticidal properties, they breakdown rapidly and have very low dermal and oral toxicity to mammals. The disadvantages are that they are relatively expensive, have no residual effect and may be toxic to other aquatic life.

  - **Synthetic pyrethrins.(trade names Reslin®, Cislin® and Coopex®)**

    These have the same insecticidal action of natural pyrethrins. Depending on the particular synthetic pyrethroid, control may be achieved for periods of up to 3-4 months. Reslin (bioresmethrin) is the insecticide of choice for rapid adult control near residential areas. Cislin (deltamethrin) and Coopex (permethrin) have longer residential effects and should not be sprayed as a large-scale aerosol treatment in residential areas. Synthetic pyrethrins also have low mammalian toxicity, although they can kill fish, so should be used carefully near water.

  - **Organophosphate compounds (Malathion, trade name Maldison®)**

    These chemicals act as nerve poisons, which interrupt nerve impulse transmission in insects. Maldison has a rapid insecticidal effect, with low toxicity to mammals. Unfortunately, it has a strong odour, which may draw complaints from residents. Maldison® also has a detrimental effect on non-target insects.

Pesticide Application Equipment

Equipment for vector control is mainly of three types:

a) sprayers used to apply liquid larvicides or residual deposits on surfaces;

b) sprayers designed to produce aerosols, mists and fogs, principally as adult space spray applicators (Ultra Low Volume (ULV) misters and thermal foggers are examples of space spray applicators); and

c) granular spreaders that are used to apply granular and pellet forms of larvicide.

Each of these types of pesticide application equipment can be hand held, motor vehicle mounted, boat mounted or aircraft mounted. Adulticides and larvicides may have to be applied using a wide variety of application equipment. Again, each disaster situation may require different vehicles or a combination of vehicles to access difficult areas. For instance in the Katherine floods in 1998 Territory Health Services sprayed large areas of mosquito breeding with B.T.I. by helicopter and rapidly reduced the number of adult mosquitoes for up to 3 weeks after spraying. For aerial application of pesticides, the Disaster District Coordinator should liaise with the NT Departmnet of Health and Community Services to assist in the organization of procedures for the use of helicopters or aeroplanes.

When using larvicides and adulticides, the dosage rate on the label should be strictly observed. Always ensure that the correct safety equipment and correct handling procedures are used. Furthermore, when using adulticides, there are several other factors that should be observed.

Always inform the public (especially apiarists and butterfly farmers) before carrying out adulticiding activities. Adulticiding should not be carried out in winds that exceed 15km per hour. Adulticides should never be applied or disposed of where they may enter watercourses or other marine habitats. Finally, never apply adulticide to areas that have beehives or other insect colonies (i.e. butterfly farms). Adulticides should not be applied to residential areas unless there is a disease outbreak or a demonstrated high risk of an outbreak, and rapid reduction of adult mosquitoes is required.

- **Fly control**

A previous survey of wheelie bins in Darwin indicated that over 50% of bins and up to 70 % of bins could be infested with maggots. If wheelie bins or other garbage receptacles are collected once per week, maggots can readily develop to the wandering stage and crawl out of the receptacle before the next collection. During disasters the non-collection of garbage or the exposure of spoiled food or dead animals can produce prolific amounts of maggots.

The prevention of domestic fly breeding relies on the correct treatment, storage and disposal of household garbage by the householder or sanitation officers. The shorter the period of exposure of the garbage to flies, the less production of flies. Adult flies can readily burrow into loosely packaged garbage.

Double bagging of garbage with plastic bags can reduce blow fly production in wheelie bins by up to 600 % compared with non bagged garbage. Although double bagging can reduce blow fly production, it is important to also prevent exposure of garbage before bagging. In practice with the use of kitchen tidies and the ability of flies to quickly enter wheelie bins it is difficult to prevent exposure of garbage to flies. During disasters meticulous care is required to prevent exposure of flies to garbage in all stages of production until the garbage is disposed in landfill. Clearly double bagging is useful however control of adult flies or maggots in wheelie bins can also be very effectively achieved with off the shelf products such as impregnated insecticide strips or blocks. The installation of impregnated pest strips in wheelie bins can kill adult flies in 30 minutes and maggots in a few hours. For effective use of pest strips, the bins should be in a sunny position and the lid left closed. If all wheelie bins or garbage holding receptacles were installed with pest strips there would be a dramatic reduction in domestic fly numbers.

- **Adult fly control**

Many methods may be used to reduce adult flies in and around the home, including:

Screening air curtains
fly swats fly baits
fly traps electrocution devices
knock down insecticides containing resmethrin or similar

Screening can be very effective in separating food and people from flies and is vital in most Top End residential areas and food preparation and consumption sites. Fly traps and fly baits, while useful for survey purposes outside the house, usually only harvest a proportion of the large population of flies. They do not clear a residential area of flies and offer little control unless the breeding sites are also removed. The other devices are very useful inside buildings by destroying those flies that enter, but offer little in the way of controlling the outside population of flies. Electronic sound repellers do not work against insects and are completely useless as fly control devices.

Electrocution devices with attracting ultra violet light are excellent devices for killing adult flies in food preparation premises. They are best positioned close to fly entry points. They should be out of sight of flies outside the premises to prevent attracting flies inside. They must be well away from food preparation or consumption surfaces as dead fly debris can contaminate food.

With disasters, large-scale aerial immediate knockdown sprays may need to be used. For

example after Cyclone Tracy in Darwin, aerial ULV sprays of maldison was used to reduce the fly problem.

Personal protection

Personal protection can be employed by residents to reduce pest problems with mosquitoes or flies. Residents should be encouraged to use repellents when being bitten. Any repellent containing DEET (Diethyl-m-toluamide) or picaridin are suitable for use (common repellent trade names include Aerogard®, Rid® Repel® and Bushman®). Note that Aerogard® is formatted for flies while Aerogard Tropical Strength® is formatted for mosquitoes. It should be noted that repellents containing DEET should not be applied to children less than 12 months. Residents can also be encouraged to use mosquito coils (any commercially available brand will suffice) to repel biting insects in sheltered areas. In housing or emergency accommodation, people can be protected from biting insects by using bed nets (these are available from army surplus or camping equipment stores). It should be noted that some mesh sizes would not be small enough to exclude biting midges. Finally, residents should be encouraged to wear light, long-sleeved, loose-fitting clothing, and avoid times and locations where biting insects are a problem. Local Government Authorities should ensure that they have an adequate supply of the above materials, especially repellent, or access to suppliers immediately following a major disaster. This can facilitate the rapid dispersal of materials to emergency accommodation camps and unscreened/damaged houses.

Dissemination of information

In the advent of a large outbreak of mosquitoes, flies or biting midges, a media campaign should be implemented to update the public on the nature of the situation, provide information on the council or government control program and to recommend the best methods to avoid being bitten. Each Local Government Authority and State Government body has their own media release policies, which should be strictly adhered too. Apart from using the media, information can be disseminated to the public through appropriate health promotion procedures, such as brochures and posters, as well as liaison with community groups (such as Neighborhood Watch).

Conclusion

Each disaster situation will provide a unique insect problem, so thorough knowledge and planning are the best weapons against any possible outbreak of mosquitoes or flies. Each State or Local Government Authority should be aware of potential problems following a disaster, their available resources and which government organizations to collaborate with to obtain the necessary cooperation or resources. Control programs should be thoroughly planned, so that swift implementation can limit the impact of mosquitoes, flies and biting midges on the community.

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For Information regarding pesticides please contact Medical Entomology Branch 08 89228901 and refer to 'Disasters and Medically Important Insects in the Northern Territory. November 2001.'

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Red imported fire ant still threatens the NT

Jim Swan, Operations Manager, NT Quarantine, DBIRD

The Red Imported Fire Ant (RIFA) is an important pest, which was detected in South-East Queensland during early 2001. The ant is a serious public nuisance and pest of agriculture in North America. It has a painful sting and can attack both humans and animals. Where they are present, fire ants will affect everyone. They have the potential to destroy Australia's outdoor lifestyle, environment and agricultural production if they spread beyond the current infested areas.

An eradication program funded by the Commonwealth and all Australian States has been operating in SE Queensland for the past 18 months.

Although RIFA is not known to occur in the NT, they are very similar to the commonly occurring 'Ginger Ant'. RIFA are between 2 to 6 mm long, golden to reddish brown in colour, with nests as dome shaped mounds that may be up to 40cm high.

The ants can attack aggressively when their mounds are disturbed. Unlike Ginger Ants, RIFA can sting repeatedly. The ants grasp skin with their jaws and use their rear stingers to inject venom up to eight times in a circular pattern. The venom contains a high concentration of toxin, which causes intense burning and irritation, especially to those allergic. In humans, the stings can induce anaphylactic shock and have caused death in severe cases.

NT Quarantine needs your help to keep this pest out and to ensure that it does not become established in the Northern Territory. Reports of severe ant stinging may indicate that RIFA is present in the NT and follow-up inspection and specimen collection would ascertain if this has occurred. Quarantine authorities would appreciate your notification when suspect ant sting cases causing any medical condition are presented to clinics or hospitals.

Previously, health workers were advised of this pest through an article published in the Northern Territory Disease Control Bulletin, Volume 8 No. 1, March 2001. All cases where ants are suspected to be involved should be reported to the Quarantine Office for further investigation

Contact Bruce Dilley at Quarantine on:
Ph: 89992392 during business hours
OR mobile 0417 821 086 after hours.

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Northern Territory Notifiable Diseases 2002 – A summary

Peter Markey, Head, Surveillance, CDC

Top 10 Notifiable Diseases

In 2002, the top 10 notifiable diseases included the 4 major sexually transmitted diseases (gonorrhoea, trichomonas, chlamydia and syphilis) and the four major enteric infections (salmonella, campylobacter, rotavirus and cryptosporidia). The other 2 diseases were Hepatitis C and chlamydial eye infection (trachoma). The ranking of the top 6 remained unchanged from 2001, while the only new disease to the top 10 was trachoma which replaced Ross River Virus.

Disease	Rank 1999	2000	2001	2002
Gonorrhoea	1	2	1	1
Chlamydial Genital Infection	3	3	2	2
Trichomoniasis	2	1	3	3
Rotaviral infection	4	6	4	4
Syphilis	6	5	5	5
Salmonellosis	5	4	6	6
Cryptosporidiosis	10	7	8	7
Campylobacteriosis	7	8	7	8
Hepatitis C	8	9	10	9
Chlamydial eye infection	22	22	15	10

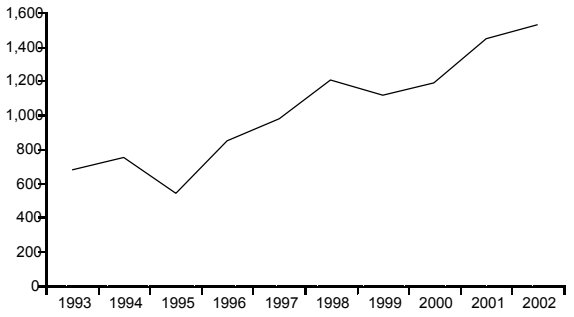
Notable trends 1993-2002

- Gonorrhoea and genital chlamydial infection (Fig 1 and 2) have continued to rise over the recent years. The relative contributions of real increases or better case ascertainment to the continuing rise is difficult to determine. It should be noted that the population rates for chlamydia and gonorrhoea continue to be highest in the 15-19 year age group at 2995/100,000 and 2902/100,000 respectively.
- Cases of acute rheumatic fever have risen over the last 3 years (Fig 3). This may be due to improved surveillance through the NT Rheumatic Heart Disease Programs. Nevertheless, these cases represent an unacceptably high incidence of the disease in the NT Aboriginal community.
- Rotavirus infections have continued to rise in a saw-tooth pattern with a peak every 2 years (Fig 4). Case ascertainment has improved with more sensitive testing but nevertheless some of this rise may be real.
- Notifications of chlamydia eye infections have increased over the last 5 years from one in 1998 to 146 in 2002 (Fig 5). This has been almost certainly due to an increase in the number of PCR tests being carried out in rural centres. Nevertheless it indicates that trachoma is still an issue for eye health in the NT.
- Notifications of salmonellosis fluctuate from year to year but have, perhaps, shown a downward trend over the last decade (Fig 6). There were 461 in 1994 and 330 in 2002. Rates are still many times higher than the national average. Enhanced surveillance through OzFoodNet in 2003 may result in an increase in notifications.
- Trichomonas infections have been decreasing over the last 2 years from a peak in 1,224 in 2000 to 684 in 2002 (Fig 7). PCR screening for trichomonas is no longer routinely done. This would account for much of the decrease.
- The number of Ross River Virus cases in 2002 was 63 a 70% reduction from the previous year (225) and the lowest recorded since 1990 (Fig 8). This may have been due to a particularly dry wet season in 2001-02 and a late start to the 2002-03 wet.
- There were fewer cases of melioidosis in 2002, the least since it became a notifiable disease in 1998 (Fig 9). This is likely to reflect the drier wet season of 2001-02, and lack of flooding and cyclonic conditions which had occurred in previous years as well as possible increased individual self protection.
- Pertussis cases were down in 2002, following an epidemic in 2001. Figure 10 illustrates this together with the epidemic of 1994-95.

Other important diseases

Meningococcal disease – In 2002, there were 9 cases of meningococcal disease. Seven of these were group B, one was group C and the other was not able to be grouped. This compared with 13 in 2001 (10 group B, one C and 2 unknown) and 9 in 2000 (6 group B, 2 unknown and one W).

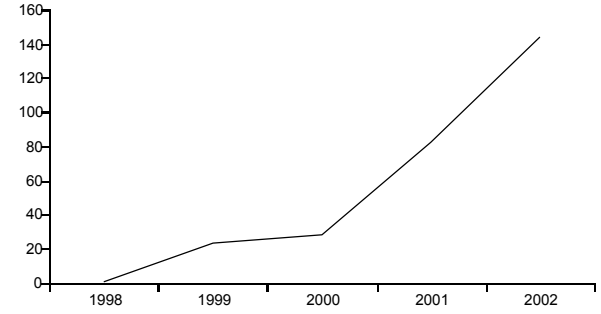
Chlamydial Genital Infection



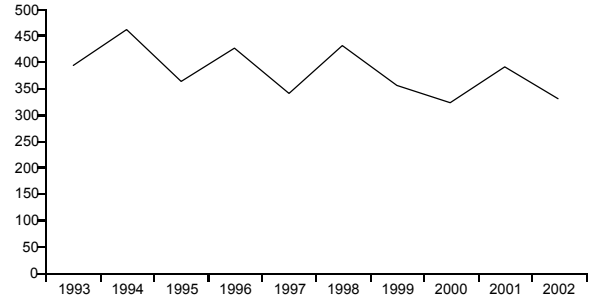
Gonorrhoea

Acute Rheumatic Fever

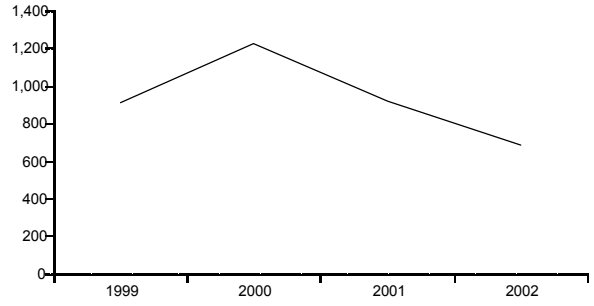
Rotaviral infection



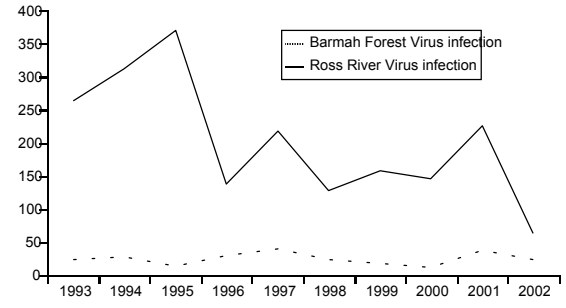
Chlamydial conjunctivitis



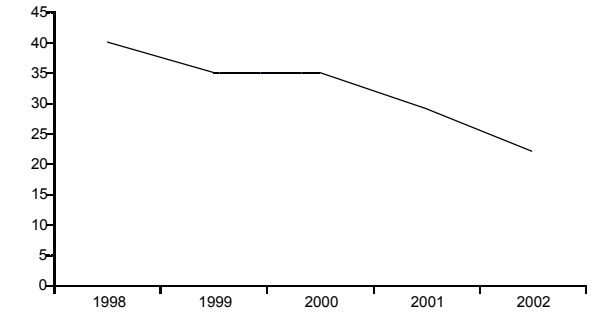
Salmonellosis



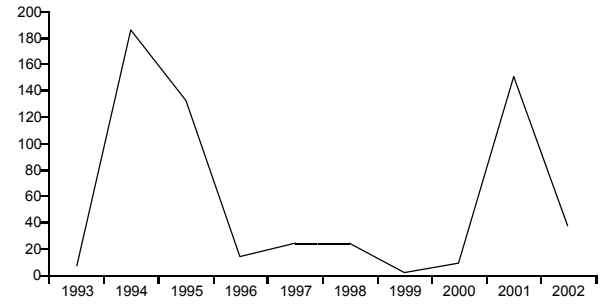
Trichomoniasis



Ross River Virus and Barmah Forest Virus Infections



Melioidosis



Pertussis

Measles – There were no measles cases in 2002 and the last NT cases were in 1999.
Rubella – There was one case in 2002, the first since in the NT since 1998.
Malaria – There were only 24 cases of malaria a fall from 61 in 2001. There were no locally acquired cases. The last locally acquired case was in 1962.
Dengue – There were 32 cases of Dengue compared with 43 in 2001. All were imported.
Q fever – There was one case of Q fever which was the first case notified since electronic records began in 1991.
Murray Valley Encephalitis - There were no cases in the NT in 2002, despite sentinel chicken sero-conversion in March. Likewise there was no Kunjin/Kokobera.
Invasive Pneumococcal disease – There were 65 cases, the lowest recorded since it became notifiable in 1995. This may reflect the trend towards reduction in the under 5 year olds in response to conjugate pneumococcal vaccination of at risk groups started in June 2001.
Yersinia – There were 7 cases notified, the highest since 1992. There was no connection found between the cases.

Data reporting

As mentioned in previous editions of The Bulletin, we are now reporting diseases by their "onset date". That is, the figures in this report and accompanying graphs and tables are the number of cases who had their onset of disease in the year reported (2001 or 2002). Because of this the numbers for 2001 reported in previous editions of The Bulletin will be different from those presented here.

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NT malaria notifications

Merv Fairley, CDC, Darwin

October- December 2002

Five notifications of malaria were received for the 4th quarter of 2002. The following table provides details about where the infection was thought to be acquired, the infecting agent and whether chemoprophylaxis was used.

Origin of infection	Reason exposed	Agent	Chemoprophylaxis
Indonesia	resident	P.vivax	no
East Timor	working	P vivax	no
West Timor	holiday	P vivax	yes
Thailand	resident	P.vivax	no
Thailand	holiday	P.vivax	no

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Points to note regarding notifications page 42

Anthrax, Kokobera, Botulism, Brucellosis, Chancroid, Cholera, Congenital Rubella Syndrome, Hepatitis C (incidence), Hepatitis D & E, Hydatid Disease, Listeriosis, Lymphogranuloma venereum, Plague, Poliomyelitis, Rabies, Tetanus, Vibrio Food Poisoning, Viral Haemorrhagic Fever and Yellow Fever are all notifiable but had "0" notifications in this period.

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NT NOTIFICATIONS OF DISEASES BY ONSET DATE & DISTRICTS 2002 AND 2001

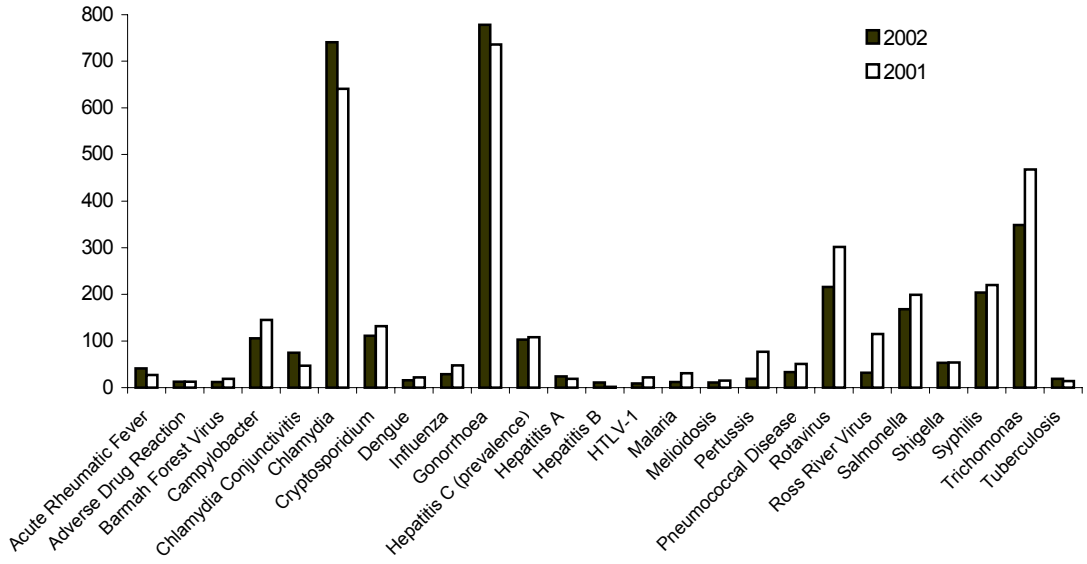
DISEASES	ALICE SPRINGS		BARKLY		DARWIN		EAST ARNHEM		KATHERINE		TOTAL	
	2002	2001	2002	2001	2002	2001	2002	2001	2002	2001	2002	2001
Acute Rheumatic Fever	30	25	0	2	21	12	12	8	18	5	81	52
Adverse Vaccine React.	2	2	1	4	18	16	4	3	0	1	25	26
Amoebiases	0	2	0	0	2	0	0	0	1	1	3	3
Arbovirus infections												
Murray Valley Enceph	0	1	0	0	0	1	0	0	0	0	0	2
Barmah Forest Virus	1	1	1	5	14	20	5	4	2	7	23	37
Dengue	0	0	0	0	30	41	0	1	2	1	32	43
Kunjin	0	2	0	0	0	0	0	0	0	0	0	2
Ross River Virus	1	7	2	68	40	109	4	6	16	35	63	225
Atypical Mycobacteria	0	1	0	0	1	4	0	0	0	0	1	5
Campylobacter	70	93	4	8	112	145	7	17	15	21	208	284
Chlamydia	547	588	24	18	596	444	140	98	144	107	1451	1255
Chlamydia Conjunctivitis	3	1	0	0	74	72	45	0	24	9	146	82
Congenital Syphilis	13	6	0	0	0	5	0	3	0	3	13	17
Cryptosporidiosis	44	60	11	4	110	98	20	29	32	67	217	258
Diphtheria	0	0	0	0	0	1	0	0	0	0	0	1
Donovanosis	1	9	1	0	2	2	0	0	5	2	9	13
Gastroenteritis	0	0	0	0	1	0	0	0	0	0	1	0
Glomerulonephritis	1	4	0	0	1	10	1	2	3	3	6	19
Gonococcal Disease	707	914	40	39	452	257	133	86	193	145	1525	1441
Gonococcal Conjunctivitis	5	0	0	0	0	0	1	0	0	0	6	0
Gon. Opthalmic. Neonatal	0	1	0	0	0	0	0	0	0	0	0	1
Haemolytic Uraemic Syn	0	0	0	0	1	0	0	0	0	0	1	0
Haemophilus Inf type b	1	0	0	0	2	2	0	0	0	1	3	3
Haemophilus Inf not typeb	1	1	0	0	2	1	0	0	1	1	4	3
Hepatitis A	8	7	5	1	14	20	1	8	19	2	47	38
Hepatitis B	13	0	2	1	5	0	1	1	1	1	22	3
Hepatitis C (prevalence)	25	40	3	2	157	148	3	6	14	16	202	212
HIV infections	3	0	0	0	6	6	0	1	0	0	9	7
HTLV-1	12	33	0	0	4	8	0	0	2	2	18	43
Influenza	20	46	0	0	29	39	4	5	3	4	56	94
Legionnaires Disease	0	0	0	0	1	3	0	0	0	0	1	3
Leprosy	0	0	0	0	0	0	0	0	1	0	1	0
Leptospirosis	0	0	0	0	3	3	0	0	0	1	3	4
Malaria	1	3	0	0	20	54	0	3	3	1	24	61
Melioidosis	1	0	1	1	18	22	1	3	1	3	22	29
Meningococcal Infection	1	4	0	0	4	8	3	1	1	0	9	13
Mumps	0	0	0	0	1	1	0	0	0	0	1	1
Ornithosis	0	0	0	0	2	1	0	0	0	0	2	1
Pertussis	3	54	0	4	32	77	2	8	0	7	37	150
Pneumococcal Disease	31	63	2	4	20	25	1	1	11	6	65	99
Q Fever	0	0	0	0	1	0	0	0	0	0	1	0
Rotavirus	130	189	19	15	157	245	49	44	69	99	424	592
Rubella	0	0	0	0	1	0	0	0	0	0	1	0
Salmonella	66	84	6	12	181	209	10	21	67	63	330	389
Shigella	46	38	7	8	36	37	5	7	9	16	103	106
Syphilis	189	232	4	30	120	67	16	36	70	65	399	430
Trichomonas	228	236	14	23	240	285	86	158	116	214	684	916
Tuberculosis	3	2	0	0	23	15	0	1	12	9	38	27
Typhoid	0	0	0	0	0	2	0	0	0	0	0	2
Typhus	0	0	0	0	0	1	0	0	0	0	0	1
Yersiniosis	0	0	0	0	7	1	0	0	0	0	7	1
Total	**2207**	**2749**	**147**	**249**	**2561**	**2517**	**554**	**561**	**855**	**918**	**6324**	**6994**

NOTIFIED CASES OF VACCINE PREVENTABLE DISEASES IN THE NT 2002 AND 2001

DISEASES	TOTAL		No. cases among children aged 0-5 years	
	2002	2001	2002	2001
Congenital rubella syndrome	0	0	0	0
Diphtheria	0	1	0	0
Haemophilus influenzae type b	3	3	2	3
Hepatitis B	22	3	0	0
Measles	0	0	0	0
Mumps*	1	1	0	0
Pertussis	37	150	11	40
Poliomyelitis, paralytic	0	0	0	0
Rubella	1	0	0	0
Tetanus	0	0	0	0

* Mumps is largely under-reported.

NT WIDE NOTIFIABLE DISEASES 2002 AND 2001



Rates, < 10/100,000 not listed
NT established residential population—195,905 supplied by Epidemiology & Statistics Branch DHCS.

Revised CDC guidelines

The following Disease Control Guidelines have been developed or revised in 2002:

- Control of Nontuberculous Mycobacteria in the Northern Territory
- Control of Tuberculosis in the Northern Territory
- Community Control of Scabies, Skin Sores and Crusted Scabies
- Control of Gonococcal Conjunctivitis in the Northern Territory
- Control of Acute Post Streptococcal Glomerulonephritis in the Northern Territory
- Control of Leprosy in the Northern Territory

Updated Immunisation schedules with the addition of Meningococcal C vaccine and accompanying fact sheets are also available.

Treatment guidelines, fact-sheets and immunisation updates can be accessed on the DHCS internet site at *http://www.nt.gov.au/health/cdc/cdc.shtml*. A hard copy is available by contacting the Project/Research Officer at Disease Control on 0889228089 or lesley.scott@nt.gov.au

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Access your Northern Territory Disease Control Bulletin on the internet

If you would prefer to access your Bulletin from our internet site at *www.nt.gov.au/health/cdc/bulletin/index.shtml* rather than receive a mailed copy please contact Lesley Scott on 0889228089 or lesley.scott@nt.gov.au and I will add you to our E–mailing list.

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Disease Control staff updates

Darwin

Welcome back to **Karen Dempsey** who has recently completed her MAE in Mt Isa. Her experience as a remote health nurse and public health background will ensure she is well equipped in her position as enteric disease epidemiologist.

The child health team has gained **Alison Cupitt**, as Community Paediatric Registrar. Alison has been working at RDH and recently passed her RACP fellowship exams. From November 2002 to February 2003 **Brad Palmer** has worked in a coordinator position to develop the service model and job descriptions for the Top End staff as part of the new Child Health Initiative for remote communities. **Helen Lourigan** has moved back to the maternal and child health bank at RDH after her stint as community child health nurse replacing Brad. **Angie Salter** has realised a long held ambition and moved on to become a police auxiliary.

Meredith Neilson comes to the Chronic Disease and Injury Prevention Project Officer position from a physiotherapy background having worked in the public and private system. She has worked in NSW, UK, and for the last 2 years in Darwin and Nhulunbuy.

Matthew Parnaby has returned to the rural AIDS/STI coordination position from working on an HIV program in Kenya and with an under 5 year old nutrition program in the Philippines. **Janine Weston** has replaced **Craig Atkinson** as administration assistant in Clinic 34.

Alice Springs

Welcome to **Dr Jackie Glennon,** the new Medical Officer for CDC in Alice Springs. Jackie trained in Ireland, and has a MSc in Community Health for developing countries from London School of Hygiene, she has worked in developing countries for most of her career most recently in Solomon Islands. Jackie came to Australia in 1999 and worked as DMO remote health and has been working as Medical officer in Congress for the past 6 months before taking up this post.

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CUMULATIVE INDEX
Vol. 1 Nos.(1-10) to Vol. 9 No. 4 (Nov 1991 - December 2002)