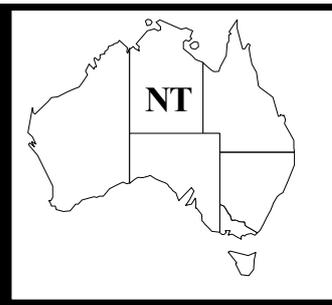




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Measles in the Northern Territory 1991-1999: implications for policy development

Karen Edmond, CDC, Darwin

Abstract

Objectives: To describe measles notification rates in the Northern Territory (NT) and Australia from 1991 to 1999 and to describe NT measles policy changes in the same period.

Design: This study was designed as a retrospective population based incidence study of national and NT measles notifications from 1991 to 1999.

Main outcome measures: Clinical or laboratory confirmed cases of measles that fulfil standard case definitions for national notification.

Results: The NT measles notification rates were higher than the Australian rates in all years except for 1993 and 1998. The NT average annual incidence for 1991-1999 (44.0/100,000) was almost 5 times the national rate (9.5/100,000). Age specific patterns were similar. In 1998 the MMR vaccination policy was changed to recommend that all NT infants (Aboriginal and non-Aboriginal) receive MMR at 12 months. There have been no cases of measles in Aboriginal children less than 24 months of age since that policy change (including no cases in infants aged 9 to 12 months), no measles deaths and no hospitalisations due to measles.

Conclusions: Recommendations will be made about improving accessibility of data from the

national system and enhancing the NT measles surveillance system. The NT guidelines will be reviewed in line with these recommendations and will be distributed in December 2000.

Contents

Measles in the Northern Territory 1991 - 1999: implications for policy development	1
Correction	6
An outbreak of measles amongst East Timorese evacuees in Darwin, 1999	7
NT quarterly immunisation coverage statistics for two birth cohorts as of 30 June 2000	13
Working together to beat measles - watch this space!	16
Don't give MMR or PedvaxHIB booster doses too early!	17
Attention all health care workers!	18
Fetal Alcohol Syndrome in Australia	18
Pelvic inflammatory disease (PID) in the Top End: a condition that needs closer attention	22
Towards a Sexual Health Strategy for remote communities in the Northern Territory	25
Points to note regarding notifications	26
NT notifications of diseases by districts 1 April to 30 June 2000 and 1999	27
Notified cases of vaccine preventable diseases in the NT by report date 1 April to 30 June 2000 and 1999	28
NT wide notifiable diseases 1 April to 30 June 2000 and 1999	28
NT malaria notifications - April to June 2000	29
CDC staff updates	29
NT Disease Control policies, protocols and guidelines	30

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Introduction

Measles has been a notifiable disease in the Northern Territory (NT) since 1990. Measles policies were developed and published in 1994 in the NT and much of the NT policy was incorporated in the national measles policy published in 1996. The national measles policy was revised very recently (July 2000). The NT policy is now up for review. This study has thus been conducted to determine if NT measles notification trends are similar to Australian trends. Additional information about NT trends in measles hospitalisation, mortality, immunisation coverage and policy change have also been obtained.

Methods

Case definitions

Confirmed cases of measles were defined according to recommendations by the Communicable Diseases Standing Committee of the National Health and Medical Research Council (NHMRC) for the purposes of national notification (Table 1).^{1,2} *Preventable cases of measles* were defined as those in children 13 months of age who lack documented evidence of vaccination against measles on or after the first birthday; had no medical contraindication to receiving the vaccine; and had no documented episode of serologically confirmed measles previously.^{1,2}

Table 1 Case definitions used for national surveillance in NT and Australia^{1,2}

The NHMRC definition of measles is an illness characterised by all the following features:

- a generalised maculopapular rash lasting 3 or more days;
- a fever exceeding 38.3°C; and
- cough or coryza or conjunctivitis or Koplik spots

A confirmed case was defined as a person with signs and symptoms consistent with measles and any one of the following:

- measles virus detected in an appropriate specimen, or
- the presence of measles-specific IgM, or
- a four-fold or greater change in measles antibody titre in sera obtained at least two weeks apart, or
- history of contact with a laboratory-confirmed case.

A measles hospitalisation was defined as a separation coded according to the International

Classification of Diseases – Clinical Modification Version 9 (ICD9-CM) code 055.³ A *measles death* was defined as a case coded according to the ICD9-CM code 055.³ *Date of notification* was defined as the date of notification to the relevant health authority in each state or Territory.⁴ *Immunisation coverage* for MMR was defined as the percentage of children who had received their first dose of MMR by two years of age.⁵ *NT cases of measles* were defined as persons with measles notified from within the NT.¹ *Australian cases of measles* were all cases of measles notified in Australia including NT cases.¹ An *imported* case of measles was defined as person who has confirmed measles and whose rash onset was within 18 days of arrival in Australia.¹ An *indigenous* case of measles was defined as person who has confirmed measles and whose rash onset was more than 18 days after arrival in Australia.¹ An *Aboriginal* case of measles was defined as any case of confirmed measles with Aboriginal ethnicity recorded in the NT CDC notifiable diseases data base.²

Population denominators

Denominators were calculated from 1996 census data for NT and Australia.⁶

Data collection

Australian notifications of measles were obtained from Communicable Diseases Network - Australia New Zealand (CDNANZ) - National Notifiable Diseases Surveillance System (NNDSS).⁴ *NT notifications* were obtained from the Darwin Centre for Disease Control (CDC) notifiable diseases database. Data were obtained for *measles separations* for ICD9 code 055 from the Business Information Management Unit, Territory Health Services, for all public hospitals in the NT. Data were obtained from the Registrar General of Births Deaths and Marriages for any death coded as a *measles death* or any death described by the NT coroner as a measles death. *NT measles policy changes and outbreak reports* were obtained by reviewing the published literature, the *NT Disease Control Bulletin*, the NT CDC measles filing system, and the Masters of Applied Epidemiology Bound Volume of Dr Douglas Lush.⁷ *NT immunisation coverage* was obtained from the Australian Childhood Immunisation Register (ACIR) and the NT immunisation register for March 1998 to September 1999.⁶

Data analysis

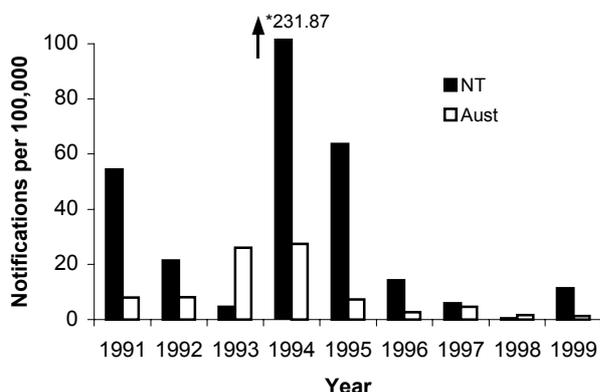
Annual Australian and NT measles notification rates were calculated per 100,000 population for 1991 to 1999. Average annual age specific rates

were also calculated for 1991 to 1999 for NT and Australia. NT measles notifications in the following subgroups were also collated: Aboriginality, district of notification, vaccination status, imported cases, preventable cases, confirmation (clinical, laboratory), age in months for children less than 2 years of age, hospitalisations, and deaths. NT immunisation coverage was also collated for March 1998 to September 1999 for the proportion of children who had received their first dose of MMR by two years of age.

Results

There were 709 cases of measles notified in the NT from 1991 to 1999 and 15,279 cases notified in Australia. The trends in measles notifications for Australia and the NT were similar (Figure 1). The NT measles notification rates were higher than the Australian rates in all years except for 1993 and 1998. The NT average annual incidence for 1991-1999 (44.0/100,000) was almost 5 times the national rate (9.5/100,000). The average annual measles incidence for 1991 to 1999 in the Aboriginal NT population (58/100,000) was twice that of the non-Aboriginal population (26/100,000).

Figure 1 Measles notifications per 100,000 population in Australia and NT 1991 - 1999



The highest age specific incidence was at 0-4 years for NT and Australia (Figure 2). The age specific rates decrease markedly from 20 years of age for both NT and Australia. Reporting of notifications by NT district is shown in Figure 3. Aboriginal status was almost complete, but there was little recording of information regarding vaccination status (Table 2). It was also not possible to determine which cases were confirmed by laboratory or clinical data. Acquisition site was listed for 582 of 709 cases. There was insufficient information to determine which NT cases were preventable.

Figure 2 Age specific notification rates of measles per 100,000 population in Australia and NT 1991-1999

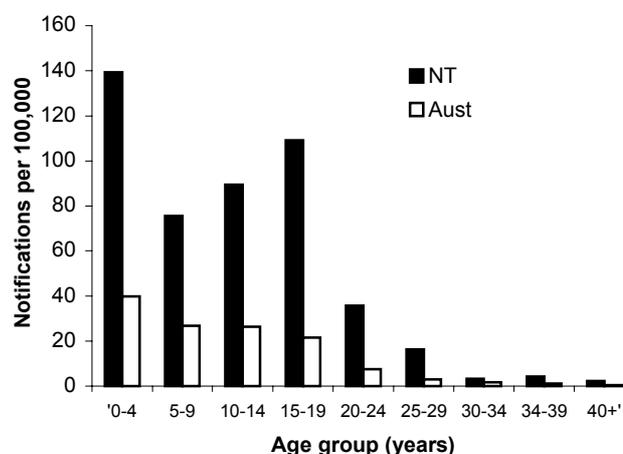


Figure 3 Measles notifications by NT district 1991-1999

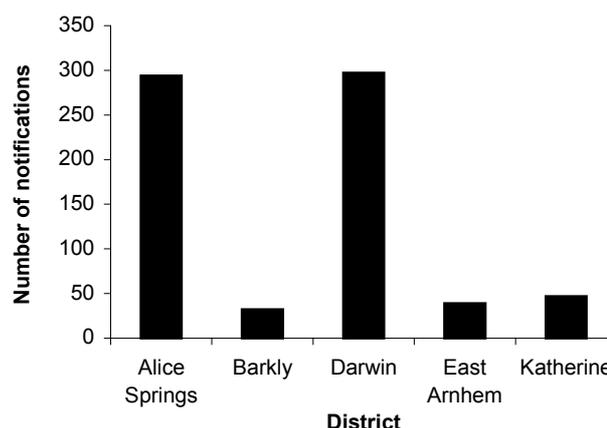


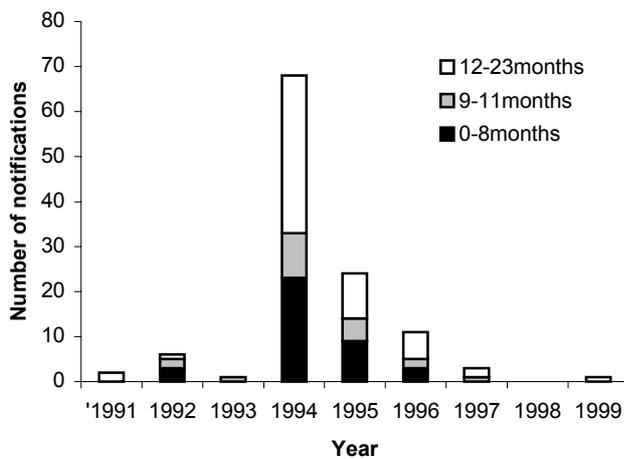
Table 2 Details of NT notifications 1991-1999

	Yes	No	Unknown*
Aboriginal	289	386	34
Vaccinated with MMR	89	0	637
Imported case	24	558	127

*Information not recorded in the NT CDC measles database

Of the 141 children under 2 years of age notified with measles, the date of birth was recorded in only 116 cases. Of these 116 children, 59 were aged less than 12 months and 21 were aged 9 to 11.9 months (Figure 4). Only one case under 2 years of age was reported as imported (an East Timorese child notified in 1999). With the exception of this East Timorese child, there have been no cases of measles in children less than 24 months of age since 1997.

Figure 4 Measles notifications in NT children aged less than 24 months with a recorded date of birth 1991-1999



There were no measles deaths in the ten year period in the NT. The trend in hospitalisation generally followed the trend in measles notifications (Figure 5) and 80% of hospitalisations were in those under five years of age. All the hospitalised cases in 1999 were linked to the East Timor outbreak and were non-Aboriginal.

Figure 5 NT measles hospitalisations, 1991-1999

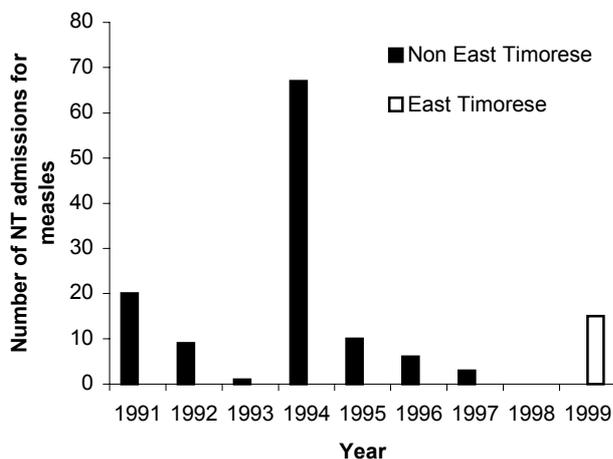


Table 3 displays NT measles policy changes and recorded outbreaks from 1968-1999. Vaccination coverage for MMR has gradually increased since the first data report on 31 March 1998. NT trends in measles coverage rates have generally followed those of Australia. The latest coverage figures reported by the ACIR and the NT immunisation register (December 1999) indicate that 88.9% of Australian children and 92.6% of NT children are vaccinated with MMR by two years of age.⁵

Table 3 NT measles policy changes and reported outbreaks 1968-1999 (NT outbreaks shown in *italics*)

1968	Monovalent Measles vaccination introduced for use at 12 months of age.
1979	<i>Measles epidemic in Central Australia - 237 cases, 5 deaths.</i> ⁸
1981	<i>Measles epidemic in Central Australia - 125 cases, 5 deaths.</i> ⁹
1983	Bivalent Measles Mumps vaccination introduced for use at 12 months of age.
1984	Age of Measles Mumps vaccination for Aboriginal children in the NT decreased to 9 months of age. Non-Aboriginal children vaccination remains at 12 months.
1989	Trivalent Measles Mumps Rubella vaccination introduced for use at 12 months of age.
1990	Measles becomes a notifiable disease in the NT.
1991	<i>Measles epidemic in Darwin - 76 cases, no deaths.</i> ¹⁰
1992	<i>Measles epidemic in Tennant Creek - 19 cases, no deaths.</i> ¹¹
1992	<i>Measles epidemic in Central Australia - 11 cases, no deaths.</i> ¹²
1992	<i>Measles epidemic prevented in Darwin by vaccination.</i> ⁷
1994	<i>Measles epidemic in Central Australia - 259 cases, no deaths.</i> ¹³
1994	Two dose Measles Mumps Rubella schedule introduced with second dose at 10 years.
1995	<i>Measles epidemic in Arnhem land - 60 cases, no deaths.</i> ⁷
1998	National enhanced measles control campaign (change of timing of second dose of MMR vaccine from 10-16 years to 4-5 years, change of timing of first dose of MMR vaccine for Aboriginal infants from 9 to 12 months, all primary school children [5-12 years] receive a second dose of MMR vaccine, all 2-5 year olds followed to ensure they receive their first dose of MMR vaccine, recommendation that all secondary school students receive a second dose of MMR, campaign coverage estimated to be 72.4% in the NT).
1999	Change of timing of second dose of MMR vaccine from 4-5 years to 4 years.
1999	<i>Measles epidemic in Darwin in East Timor evacuees - 17 cases in East Timorese evacuees, no deaths.</i> ¹⁴

Discussion

The descriptive epidemiology of measles in the NT appears to be similar to the rest of Australia. However the usefulness of this review is limited by deficiencies in the national and NT notifiable disease surveillance systems. National measles data was restricted to Aboriginality, age group and State/Territory of notification. Information about epidemiological linkage and imported or preventable (eg unimmunised) cases was not available. The NT database has very comprehensive data entry fields but much of the information is missing. Notification of Aboriginal status, district of notification and age was excellent but vaccination status, type of confirmation and date of birth were reported poorly.

Measles has been listed as a notifiable disease in Australia since 1986 and in the NT since March 1990. Case ascertainment is considered to be better in the NT than in most of the other States and Territories due to the smaller population and closely-knit networks. There are some difficulties surrounding laboratory testing in the NT eg. the average time for measles IgM results to be available can be long (when the laboratory is informed, turn around time is usually within 48 hours, otherwise it may be 4-5 days). Most NT specimens are sent interstate but one private laboratory performs their own testing in the NT. Difficulties were encountered in distinguishing vaccine and wild measles virus infection during the 1999 East Timorese measles outbreak when a large vaccination program was implemented two days after the first diagnosed case (see following article).¹⁴

Most years, measles rates were higher in the NT than nationally. Increased ascertainment and high rates in the NT Aboriginal population in 1994 were likely contributing factors. MMR coverage rates have risen from 1991 to 1999 and widespread protective immunity in the NT now appears likely. Importantly only two cases of measles extended beyond the East Timorese population during the 1999 outbreak surrounding the evacuees.¹⁴

Both NT and Australia had a high incidence of measles in young adults. This may indicate waning immunity, lack of exposure to the wild virus and unvaccinated individuals. This pattern is similar to other Western countries and unlike developing countries.¹⁵⁻¹⁸

From 1984 to 1998 NT policy recommendations

were to vaccinate all Aboriginal infants with MMR at 9 months of age because of concerns of waning maternal immunity and high morbidity and mortality.^{9,19} From 1991 to 1997, 21 cases of measles were notified in Aboriginal infants aged 9 to 11.9 months. This may indicate vaccine failure, but notification records were not adequate to determine if all of these infants had received MMR vaccination.

Vaccinating Australian Aboriginal children at nine months of age induced protective antibody levels in 93% of vaccinated children in 1989.¹⁹ Seroconversion rates however, are reported to be higher in children vaccinated at 12 months.^{18,20,21} In 1995, during a measles outbreak, Lush et al reported vaccination failure in 7.8% of NT Aboriginal infants vaccinated between 8 and 11 months and failure in only 3.2% of infants vaccinated after 11 months of age.²² Interestingly, during this outbreak clinical measles was notified in 38 Aboriginal infants less than 9 months of age but laboratory verification was not obtained. All infants were assessed by experienced paediatricians to have clinical measles. If these were true measles cases, low levels of acquired maternal antibody is the most likely explanation for disease in this young age group.¹⁸

In 1998 the MMR vaccination policy was changed to recommend that all NT infants (Aboriginal and non-Aboriginal) receive MMR at 12 months. Since then there have been no cases of measles in Aboriginal children less than 24 months of age, no measles deaths, and no hospitalisations due to measles.

The available data indicate that the descriptive epidemiology of measles in the NT is similar to the rest of Australia and our current immunisation policy is appropriate. There is no evidence to indicate that the national guidelines should not be adapted for use in the NT. Improvements in the NT measles notification system are recommended. Reporting of case details, especially information about vaccination status and epidemiological linkage must improve. Our NT notifiable diseases surveillance system should report the preventable cases regularly and enhance surveillance for imported cases and epidemiological linkage. Additionally it is hoped that access to national data by States and Territories will improve. This is likely to occur with the updating of the NNDSS. In particular States and Territories should be able to compare their epidemiological linkage and preventable cases with national rates.

The NT measles policy is under review. Issues to be considered include: the adaptation of national guidelines; an improved measles reporting form for disease control officers; guidelines for reporting of preventable and imported cases; guidelines for outbreak prevention and control in refugee populations; and guidelines on investigation and management of suspected vaccine associated measles. The target for finalising and distributing the NT policy is December 2000.

Acknowledgments

I would like to thank the Communicable Diseases Network - Australia New Zealand - National Notifiable Diseases Surveillance System, for provision of the national data. I would also like to thank Veronica Barrett and Christine Selvey for assisting with the CDC notification system, Nan Miller for providing information on immunisation policy and Douglas Lush for the information in his Masters of Applied Epidemiology bound volume.

References

1. National Health & Medical Research Council. Measles: Guidelines for the control of outbreaks in Australia, Series on Infectious Disease Control. Canberra: Australian Government Publishing Service, 1996, catalogue #9606629.
2. NT Measles Protocol. Guidelines for Disease Control. A manual for Communicable Disease Officers. NT Department of Health and Community Services, Disease Control Program, Darwin 1994.
3. International Classification of Diseases – Clinical Modification Version 9 (ICD9-CM).
4. Communicable Diseases Network - Australia New Zealand - National Notifiable Diseases Surveillance System, April 2000.
5. Australian Childhood Immunisation Register, Canberra December 1999.
6. Australian Bureau of Statistics. Population by age and sex, Australian states and territories. Canberra 1996, catalogue #3201.0.
7. Lush D, Masters of Applied Epidemiology, Bound Volume, December 1994.
8. Kettle E. Health Services of the Northern Territory-1 history 1842-1970. 1st edit. Vol1 and 2. Northern Australian Research Unit 1991.
9. Hanna JN, Kass RB. Immunisation status of Aboriginal children in Central Australia. *MJA* 1985;143:s56-7.
10. Merianos A, Miller NC, Patel MS. Control of a community outbreak of measles which started in a poorly immunised high school population. *Aust J Public Health* 1993;17:231-236.
11. Brennan R, Gokel G, Maloney M. Tennant Creek measles outbreak February to March 1992. *NT Comm Dis Bull* 1992;1(4):1.
12. Brennan R. Measles outbreak in Alice Springs. October to December 1992. Discussion document. Disease Control Centre files, Alice Springs 1992.
13. Lush D, Maloney M, Merianos A. Measles outbreak in Alice Springs region, Northern Territory, June to November 1994. *Comm Dis Intell* 1994;18(23):567-8.
14. Edmond KM, Ritchie B, Measles outbreak in East Timor refugees in Darwin, 1999. in progress
15. Markowitz LE, Preblud SR, Orenstein WA, Rovira EZ, Adams NC, Hawkins CE, Hinman AR. Patterns of measles transmission in measles outbreaks in the United States 1985-1986. *NEJM* 1989;320(2):75-81.
16. MMWR. Epidemiology of measles United States, 1998. Sept 1999;4834).
17. Lambert S, Lynch P, Morgan M, Gerovich D. Measles Outbreak – Young Adults at High Risk. *Vic Infect Dis Bull* 1999;2(2).
18. Mullholland K. Measles and Pertussis in developing countries with good vaccine coverage. *Lancet* 1995;345:305-6.
19. Hanna JN, Macintyre AB, Worswick DA, Burrell CJ. Seroconversion after administration of measles vaccine to central Australian Aboriginal children at nine months of age. *MJA* 1989;150:188-92.
20. Cutts FT, Markowitz LE. Successes and failures in measles control. *J Infect Dis* 1994;170(S1);S32-41.
21. Position statement by the expanded programme on immunisation of the world health organisation. Measles immunisation before the age of 9 months. *Lancet* 1988;1356-7.
22. Lush D, Patel M. Measles vaccine effectiveness in Aboriginal children vaccinated at or after eight months of age. *ANZJPH* 1998;22(6)729-30.

Correction

It may have come to the attention of some readers that on page 7 of the article East Timorese evacuees in Darwin - 1999, published in the June edition of the *Bulletin*, the last sentence in the paragraph under Results - *demographics* was cut off mid way

through (in a small number of the final printed copies). The sentence should have read: There were 38 people aged 60 years and above and this group included frail elderly.

An outbreak of measles amongst East Timorese evacuees in Darwin, 1999

Karen Edmond, CDC, Darwin

Abstract

Objectives: To describe the epidemiological features of the measles outbreak that occurred on the arrival of the East Timorese evacuees in Darwin in 1999, and to identify measles surveillance and control measures needed for prevention of further measles outbreaks in evacuees in the Northern Territory (NT).

Design: This study was designed as a prospective descriptive cross sectional study from 9 September 1999 to 19 November 1999.

Subjects and setting: 1863 East Timorese refugees evacuated to Darwin from 10 – 14 September 1999. MMR vaccinations were given to the first 202 evacuees on arrival. The first case of suspected measles was notified on 19 September 1999. MMR vaccination was administered to all evacuees aged 6 months to 30 years on 21 September 1999 and 4 October 1999.

Main outcome measures: Cases of suspected, probable and confirmed measles according to standard case definitions.

Results: 17 cases of measles were confirmed from 9 September 1999 to 19 November 1999. Cases 1-3 were unimmunised East Timorese children, cases 4-15 were East Timorese children who had been immunised on 21 September 1999 and cases 16-17 were unimmunised Darwin resident adults. 13 children with confirmed measles had clinical complications. All children made a full recovery.

Conclusions: The measles outbreak was rapidly controlled with 95% MMR immunisation coverage (in evacuees aged 6 months to 30 years). However, there are lessons to be learnt from this outbreak; particularly that early measles immunisation in such a situation is a priority. Recommendations for measles prevention and control in NT evacuees have been developed and will be incorporated into the NT measles control policy.

Introduction

In August 1999 the pro-independence referendum vote by the people of East Timor resulted in an unstable political situation in East Timor. From the 10 - 14 September 1999, 1863 East Timorese people were evacuated to Darwin, Northern Territory (NT). This paper documents the measles outbreak that occurred following the arrival of the East Timorese

evacuees in Darwin. It describes the epidemiological features of the outbreak and the investigations and measures that were instituted to control it. It also identifies measles surveillance and control measures for prevention of further measles outbreaks in evacuees in the NT.

Methods

Design

This was a prospective descriptive cross sectional study from 9 September 1999 to 19 November 1999.

Subjects and setting

East Timor was estimated to have a population of 850,000 with 100,000 children under five years of age in 1999.¹ Of the 1863 East Timor refugees evacuated to Darwin, 790 were aged less than 15 years, 293 were aged under 5 years and 53 were less than 1 year of age.² There were two groups of evacuees; Group 1 consisted of 347 United Nations Mission to East Timor (UNAMET) workers and their families, and Group 2 consisted of 1516 UNAMET workers and Dili residents.²

A camp was set up to house the 1863 evacuees in the northern suburbs of Darwin (Kalymnios camp). The camp was considered to have good conditions with no overcrowding (one family or four persons to each tent). The camp had its own primary health care clinic staffed by Territory Health Services (THS) medical and nursing staff and volunteers. Notifiable diseases detected at the camp were reported to the Centre for Disease Control (CDC) in Darwin.

Group 1 evacuees were intended to be in Darwin for the duration of their safe haven, and those aged 9 months to 30 years were given immunisations including Measles Mumps Rubella vaccine (Priorix, Smith Kline Beecham) 0.5ml, intramuscularly on 10 September 1999. Group 2 evacuees were intended for rapid transport to Southern havens where full immunisation would be given and therefore were not initially immunised. However, it was not until 18 September 1999 that the first East Timorese people were ready to fly out of Darwin and many stayed for much longer. On 19 September 1999 the first case of suspected measles was notified to CDC.

Case definitions

The following case definitions were applied prospectively:

- A *suspected case* of measles was defined as any rash illness with fever.³
- A *probable case* of measles was defined as: a generalised maculopapular rash lasting 3 or more days, fever exceeding 38.3°C and cough or coryza or conjunctivitis or Koplik spots.^{3,4}
- A *laboratory confirmed case* of measles was defined as a person with signs and symptoms consistent with measles and any one of the following: measles virus detected in an appropriate specimen (this included detection by culture and by reverse transcriptase polymerase chain reaction (RT-PCR)), presence of measles specific IgM, or a four fold or greater change in measles antibody titre in sera obtained at least two weeks apart.⁴
- A *clinically confirmed case* of measles was defined as a person with signs and symptoms consistent with measles who had negative laboratory tests or no laboratory test performed but a history of contact with a laboratory confirmed case.⁴
- A *vaccine-induced case* of measles was defined as a probable case of measles that had RT-PCR genotyping consistent with the measles vaccine strain. It was planned that these cases would be excluded from further analysis.^{5,6}
- A *measles complication* was defined as a laboratory or clinically confirmed case of measles in a hospitalised patient plus one of the following diagnosed by a NT paediatrician: otitis media, pneumonia, laryngotracheobronchitis, febrile convulsion, acute encephalomyelitis, enteritis, stomatitis, keratitis, candidiasis, or pyoderma.
- A *measles death* was defined as a laboratory or clinically confirmed case of measles confirmed by the NT coroner as a measles death.
- An *underweight child* was defined as a child with a weight for age standard deviation score less than -2.0, Marasmus was defined as a weight for height standard deviation score (z score) less than -2.0, and Kwashiorkor was defined as a child with a weight for age z score less than -2.0 and oedema.⁷

Case ascertainment and surveillance

On 19 September 1999 the first suspected case of

measles in a child resident in Kalymnios camp was notified to CDC. Two other cases quickly followed on 20 and 21 September 1999. On 19 September the Kalymnios camp primary health care staff were requested to commence active surveillance for more cases and to notify all new cases of suspected measles by phone or facsimile. The current NT measles outbreak protocol⁴ (which describes policies for the diagnosis and management of cases and contacts as well as community control measures) was distributed to the primary health care staff at Kalymnios camp to assist in surveillance and control. A specific rash and fever protocol was also developed and sent out to the camp.⁸ All medical practitioners in Darwin, accident and emergency departments and community health centre staff in the Top End of the NT were informed of the outbreak. They were also asked to commence active surveillance for more cases and to notify all new cases by phone or facsimile to CDC. Laboratory specimens were obtained from all suspected cases of measles.

Control measures

A media release was prepared informing the general community of the outbreak. Any person with suspected measles was advised to contact a doctor, their community care centre or Darwin CDC to notify the case and to obtain information about how to prevent the disease spreading to siblings and other children. Parents were advised to check their children's immunisation status and to organise a MMR immunisation if there was any doubt that their children may not be protected. Parents of children aged 4-15 years who had not received a second MMR vaccine were advised to have a second MMR vaccine given to their children. MMR vaccination had been a priority for all health staff and others working with the East Timorese in the first days of the evacuation. This was re-emphasised after the first measles cases were notified. Workers were advised to recheck their vaccination status and to organise a MMR immunisation if there was any doubt that they may not be immune. MMR vaccine was provided free of charge to all health care workers and the general public at CDC and community care centres.

All probable cases of measles were hospitalised immediately so that specimens could be obtained and severity of illness established. Restriction of movement of the 2,000 people inside Kalymnios camp was considered during the incubation period but proved impractical. Fliers were distributed to all those entering the camp informing them of the

measles outbreak and the need for individuals to be immune or to get immunised.

On 21 September 1999 MMR vaccine was offered to all evacuees between the ages of 9 months and 30 years who were not pregnant. On 4 October 1999 infants 6 to 9 months of age were also immunised. High dose vitamin A was given to all hospitalised cases of measles. The standard dose of vitamin A according to age was given to all East Timorese children aged 6 months to 14 years residing in Kalymnios camp.⁹

Laboratory tests

The following laboratory tests were performed for each probable case according to standard procedures.

- Measles specific IgM and IgG were assayed at PathCentre, the Western Australian Centre for Pathology and Medical Research, using a commercial enzyme immunoassay according to standard methods.¹⁰
- Heparinised blood (EDTA), urine and nasopharyngeal aspirate (NPA) were cultured for measles virus at PathCentre, using standard methods.^{11,12}
- Heparinised blood (EDTA), urine and NPA measles reverse transcriptase polymerase chain reaction (RT-PCR) testing was performed at PathCentre, using standard methods.¹³
- Positive RT-PCR specimens were sent from PathCentre, to the Victorian Infectious Diseases Reference Laboratory (VIDRL) for genotypic classification according to standard methods.^{5,6}

Data recorded

Information was collated prospectively using a standard reporting form. Documentation of vaccination and hospitalisation details were also provided on a standard form to each family of each child.

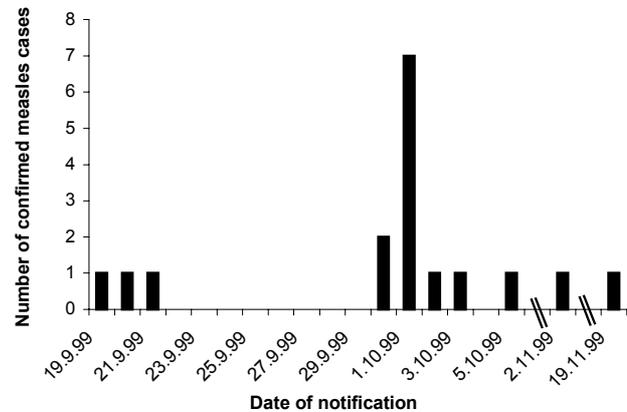
Data analysis

Data was prospectively recorded in Excel Version 7.0, Windows 95, Microsoft IBM 1995. Only descriptive analysis was performed because of small study numbers.

Results

Twenty three cases of suspected measles were reported from 9 September 1999 to 19 November 1999 with 6 unable to be confirmed by clinical or laboratory criteria. Of the remaining 17 cases, 14 were laboratory confirmed and 3 were clinically confirmed. Figure 1 shows the epidemic curve of the confirmed cases of measles.

Figure 1 Epidemic curve for 17 confirmed measles cases 19 September to 19 November 1999



Demographic characteristics of the 17 confirmed measles cases are shown in Table 1. The first 15 cases were all East Timorese children (age range 18-104 months). Cases 1-3 were unvaccinated, cases 4-15 all received MMR vaccination on 21 September 1999. Cases 16 and 17 were two Darwin resident adults who had been working with the East Timorese evacuees from 9 September 1999 to 9 October 1999. They were both uncertain of their vaccination history.

Table 1 Demographic characteristics of confirmed cases by ethnicity

	East Timorese	Non East Timorese
Total cases (n)	15	2
Chronological order of cases	1-15	16 & 17
Mean age (years)	3.75	20.8
Gender (male: female)	9:6	0:2
Vaccinated with MMR on 21/9/99	12	0

Laboratory results are displayed in Tables 2 and 3. Genotype has been reported on only three RT-PCR specimens to date and all were confirmed as wild measles virus. Of note, these three genotyped specimens were obtained from the unvaccinated index cases (cases 1-3) and no vaccinated cases have yet been genotyped. However, the epidemiological linkage, severity and spectrum of measles symptoms in the 12 vaccinated cases convincingly fit the standard definitions and strongly suggest that the cases are not vaccine associated measles. Thus, the 12 vaccinated cases have been considered as true cases of confirmed measles.

Table 2 Laboratory results for laboratory and clinically confirmed measles cases

	IgM* positive RT-PCR† positive	IgM positive, RT-PCR negative	IgM negative RT-PCR positive	IgM negative RT-PCR negative	IgM positive RT-PCR not performed	Total
Laboratory confirmed measles	10	0	2	0	2	14
Clinically confirmed measles	0	0	0	3	0	3
Total	10	0	2	3	2	17

*Measles specific immunoglobulin-M, †Measles specific reverse transcriptase polymerase chain reaction

Table 3 Laboratory results for unvaccinated and vaccinated confirmed measles cases

	Case no	IgM* positive RT-PCR† positive	IgM positive, RT-PCR negative	IgM negative RT-PCR positive	IgM negative RT-PCR negative	IgM positive RT-PCR not performed	Total
Unvaccinated East Timorese children	1-3	3	0	0	0	0	3
Vaccinated East Timorese children	4-15	7	0	2	3	0	12
Unvaccinated non East Timorese adults	16,17	0	0	0	0	2	2
Total		10	0	2	3	2	17

*Measles specific immunoglobulin-M, †Measles specific reverse transcriptase polymerase chain reaction

A total of 807 MMR immunisations were given (202 on 10 September 1999, 410 on 21 September 1999 and 195 on 4 October 1999). MMR vaccine coverage was 95% for East Timorese evacuees aged 6 months to 30 years resident at Kalymnios camp from 10 September to 9 October 1999.

All East Timorese children with confirmed measles were hospitalised. The mean length of stay was 4.71 days (range 2-13 days). Seven children were malnourished as defined by a weight for age z score less than -2.0 (Table 4).

Table 4 Nutritional status of East Timorese children with confirmed measles at admission

	Nutritional status
Mean WFA z score (range)*	-1.68 (-3.65 - +1.19)
Mean SWFA (range)†	87 (63 - 116)
Number of children underweight (%) ‡	7 (46%)
Number of children with marasmus§	0
Number of children with kwashiorkor	0

* Weight for age standard deviation score †Standard weight for age ‡Weight for age standard deviation score < -2.0, § Weight for height standard deviation score < -2.0

Thirteen children (87%) with confirmed measles had clinical complications (Table 5). There were no long term sequelae and all children were discharged after full recovery. There were no cases of laryngotracheobronchitis, febrile convulsions, encephalomyelitis, stomatitis, candidiasis, or pyoderma. There were no deaths and no children had clinical signs of vitamin A deficiency.

Table 5 Measles complications in hospitalised East Timorese children with confirmed measles by nutritional status

	Complications in malnourished children* (n)	Complications in children who were not malnourished† (n)	Total
Otitis media	3	2	5
Pneumonia	1	4	5
Enteritis	0	2	2
Keratitis	4	3	7
≥ 2 complications	1	6	7

*Weight for age standard deviation score < -2.0

†Weight for age standard deviation score ≥ -2.0

Discussion

Measles is a highly infectious disease and can spread rapidly in susceptible populations. Measles control programs in emergency settings have two major components: measles prevention through routine immunisation and measles outbreak control. In Darwin, Group 1 evacuees were immunised on arrival but Group 2 evacuees were not immunised as rapid transport to southern havens where full immunisation would be given was planned. Group 2 evacuees however, did not commence transportation south for 8 days, with the majority not transferred for 17 days. Some evacuees stayed several months since they were hospitalised, diagnosed with tuberculosis, or were unfit to fly for other reasons. The first measles case occurred 9 days after the first evacuees arrived and 5 days after those last to arrive on 14 September. The first 3 cases were probably unavoidable but all of the following 12 subsequent cases in evacuees were preventable. The World Health Organization (WHO) protocol may be too liberal in allowing up to 15 days for MMR immunisation after arrival and should consider 3-5 days.⁹ Evacuees should have been immunised on arrival.

In the event of an outbreak the main control strategy is to immunise the population at risk with live measles vaccine as quickly as possible to prevent further transmission.^{9,14,15} Rapid immunisation and excellent coverage was achieved in Darwin 48 hours after the first measles case was diagnosed. Current guidelines for measles control in refugee populations include vaccinating children aged 6 months to 5 years as a high priority with secondary priority given to persons 5 years to 30 years.^{9,15} Infants aged 6-9 months do not respond well to the vaccine however, because of interference by maternal antibodies.¹⁴ Current recommendations state that those children receiving MMR before the age of 9 months should be flagged for reimmunisation as soon as possible after nine months of age.¹⁴ Infants aged under 6 months are protected by high levels of maternal antibodies and do not require vaccination even in an outbreak situation.¹⁴

The vaccination status of the East Timorese population was unknown prior to the outbreak. MMR coverage was likely to be poor because there had been few functioning East Timorese primary health care facilities for the previous year and probably longer.¹ In developing countries with poor vaccination coverage, measles is primarily a disease of children aged less than 5 years.^{9,14,15} In

populations that have received one dose of vaccine, disease primarily occurs in young adults with waning immunity.¹⁶ Current Australian rates are also highest in the 16-30 year age group.¹⁷ The epidemiology of the Darwin outbreak also followed these patterns; only young children were affected in the East Timorese population and the two cases of measles that extended beyond the East Timorese population were young adults. This also suggests that there is quite good immunity against measles in the Darwin population.

Cases 1-3 were clearly cases of wild measles illness. However cases 4 to 15 (all recently vaccinated with MMR vaccine) posed diagnostic problems. The date of notification of cases 4 to 15 (13 to 19 days post contact with confirmed cases and 10 to 16 days post vaccination) correlated with the incubation period for the wild disease (7 to 18 days) and the usual time of onset of vaccine induced measles (5 to 15 days post MMR vaccination). So, did these 12 children have wild measles illness or vaccine induced measles? Approximately 5% of nonimmune vaccinees may develop transient malaise, rash and fever within 5-12 days post-immunisation but with little disability.^{5,19,20} Coryza, mild cough and Koplik spots have been quoted as 'occasionally' occurring post MMR vaccination but no exact proportions are quoted in the published literature.²⁰ In the Darwin outbreak 612 MMR vaccinations had been given by the 30 September 1999. Thus, up to 30 mild cases of vaccine induced measles could have been expected.

It was hoped that laboratory testing would clarify the diagnosis. However, measles virus is rarely shed for more than a few days after the onset of the rash^{11,12} and false negative and positive measles specific IgM results occur.^{10,18} Vaccine induced measles IgM is also detectable 8 days to 6 weeks post immunisation.^{10,18} False positive and negative RT-PCR results can occur.^{6,13} Measles RNA has been detected in well vaccine recipients 14-16 days post MMR vaccination.⁶

Genotyping and characterisation of measles isolates is the most helpful test to distinguish between vaccine virus and wild strains.^{6,13} However, only the unvaccinated index cases (cases 1-3) have been genotypically confirmed as wild measles virus. The vaccinated cases are still awaiting genotyping.

Clinical symptoms and epidemiological linkage were more helpful in finalising the diagnosis. All 12 cases were considered by experienced paediatricians to be true wild measles infection. All 12 cases were also convincingly epidemiologically linked to the 3 index cases. The NHMRC guidelines state that

'probable measles cases who received measles containing vaccinations 8 days to 8 weeks before testing are considered to be confirmed measles if they are also linked epidemiologically to another confirmed case'.³ Thus, the final diagnosis of all 12 vaccinated cases was considered to be confirmed measles. The true vaccination induced measles cases, which are often mild events, possibly went unnoticed.

None of the East Timorese children with measles died. The most common complications were mild keratitis, otitis media and pneumonia. There were no neurological complications and no clinical vitamin A deficiency, although there were at least two other East Timorese children subsequently brought to Australia and diagnosed with severe xerophthalmia in the ensuing months. Measles has been reported to be the leading cause of death in children under 5 years of age during the initial phase of emergency relief.^{9,21-24} The good outcomes in the East Timorese children with measles is likely to be due to their nutritional status and access to high quality medical care in Darwin. Additionally Vitamin A was administered both in the camp and in hospital. Days of hospitalisation may have been increased for comfort in some cases as children were going back to tents in hot-humid weather.

The success of the measles control program and other communicable disease control measures during September and October 1999 was due to the hard work of many people including CDC staff, THS staff, non government organisations, volunteers from the Darwin community and some interstate workers. It was also due to the rapid and co-ordinated mobilisation of public health and private health care providers including pathology services. NT protocols and guidelines were produced within hours and there was a rapid response to the measles outbreak with high MMR coverage. However there are lessons to be learnt from this 1999 outbreak of measles in the East Timorese evacuees. At the time the scenario was evolving, authorities were not 100% clear on the timing and final destinations of the evacuees. Speed of initial screening and housing was seen as the priority. It is clear now that all East Timorese evacuees 9 months - 30 years should have been immunised with MMR on arrival in Darwin. Ideally a coordinated Australia-wide plan would have been useful. CDC Darwin is currently developing recommendations for measles prevention and control in evacuee crisis settings. These will be published in the December 2000 issue of *The NT Disease Control Bulletin*.

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References

1. United Nations Inter-Agency and Non-Governmental Organisation Preliminary Assessment of needs for humanitarian assistance for east Timorese. Oct 1999.
2. Evans C, Noonan S, Krause V. East Timorese evacuees in Darwin, 1999. *NT Dis Con Bull* 2000; 7 (2): 5-11.
3. National Health & Medical Research Council. Measles: Guidelines for the control of outbreaks in Australia, Series on Infectious Disease Control. Canberra: Australian Government Publishing Service, 1996, catalogue #9606629.
4. NT Measles Protocol. Guidelines for Disease Control. A manual for Communicable Disease Officers. NT Department of Health and Community Services, Disease Control Program, Darwin 1994.
5. Jenkin GA, Chibo D, Kelly H, Lynch PA, Catton MG. What is the cause of a rash after a measles-mumps-rubella vaccination? *Med J Aust* 1999; 171: 194-5.
6. Rota S, Health JL, Rota PA, Molecular epidemiology of measles virus, identification of pathways of transmission and implications for measles elimination. *J Infect Dis* 1996; 173:32-37.
7. Waterlow JA. Definitions of malnutrition Oxford University Press 2nd ed, Oxford 1996.
8. CDC Darwin. Protocol for east Timorese evacuees with rash and fever. September 1999.
9. Toole M, Stektee RW, Waldman RJ, Neiburd P, Measles Prevention and control in Emergency settings. *Bull Wld Hth Org* 1989; 381-8.
10. Hefland RF, Health JL, Anderson LJ Diagnosis of measles with an IgM captures EIA the optimal timing of specimen collection after rash onset. *J Infect Dis* 1997; 175: 195-99.

11. Ruckle G, Rogers K. Studies with measles virus: II: isolation of virus and immunological studies in persons who have had the natural disease. *J Immunology* 1957; 78:341-55.
12. Gresser I, Katz SL. Isolation of measles virus from urine. *New Engl J Med* 1960; 275: 516-23.
13. Chibo D, Birch C, Rota P, Catton M. Genetic characterisation of measles viruses isolated in Victoria, Australia 1973-1998. *Immunisation beyond 2000*. 6th National Public Health Association Immunisation conferences 1998 Nov 4,5 Melbourne Canberra Public Health Association Australia. 1998.
14. Position statement by the expanded programme on immunisation of the world health organisation. Measles immunisation before the age of 9 months. *Lancet* 1988; 1356-7.
15. Mullholland K. Measles and Pertussis in developing countries with good vaccine coverage. *Lancet* 1995; 345: 305-6.
16. Lambert S, Lynch P, Morgan M, Gerovich D. Measles Outbreak – Young Adults at High Risk. *Vic Infect Dis Bull* 1999; 2(2).
17. Hefland RF, Kebede S, Gary HE. Timing of development of measles specific immunoglobulin M and G after primary measles vaccination. *Clinical and Diagnostic Laboratory Immunology* 1999; 6: 178-80.
18. National Health and Medical Research Council. The Australian immunisation handbook. 6th ed Canberra: AGPS, 1997.
19. Chin J, editor. Control of Communicable Disease Manual. An official report of the American Public Health Association. 17th ed. Washington DC, 2000.
20. Shears P. Epidemiological assessment of the health and nutrition of Ethiopian refugees in emergency camps in Sudan. *BMJ* 1985; 295: 314-8.
21. Cutts FT, Markowitz LE. Successes and failures in measles control. *J Infect Dis* 1994; 170 (S1): S32-41.
22. Ramakrishan K. Measles: a clinical study of 600 cases. *Ind J Pediatr* 1978; 15: 1036-1037.
23. Ayra LS, Taana I, Tahiri C, Saida, I A, Singh M. Spectrum of complications in Afghanistan: a study of 784 cases. *J Trop Med Hyg* 1987; 90: 117-22.
24. Ayra LS, Taana I, Tahiri C, Saida, I A, Singh M. Spectrum of complications in Afghanistan: a study of 784 cases. *J Trop Med Hyg* 1987; 90: 117-22.

NT quarterly immunisation coverage statistics for two birth cohorts as of 30 June 2000

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Introduction

The Australian Childhood Immunisation Register (ACIR) publishes immunisation coverage rates for two birth cohorts of children each quarter. These cohorts are aged 12 to <15 months and 24 to <27 months on the date of assessment. NT coverage estimates of the same cohorts of infants are calculated from the NT Childhood Immunisation Database (CID) on the same day that the ACIR coverage estimates are determined, allowing comparisons between the two estimates.

Methods

Coverage rates for each NT region were calculated using data on the NT CID on 30/06/00 for children aged 12 to <15 months on 31/03/00 (ie born between 01/01/99 and 31/03/99 inclusive). To be considered as fully immunised an infant must have received 3 doses of DTP (diphtheria-tetanus-pertussis vaccine), 3 doses OPV (oral polio vaccine) and either two doses of PedvaxHIB[®] or three doses of a different Hib (*Haemophilus influenzae* type b) preparation by 12 months of age.

Similarly, coverage rates for each region were calculated using data on the NT CID on 30/06/00 for children aged 24 to <27 months on 31/03/00 (ie born between 01/01/98 and 31/03/98 inclusive). To be considered as fully immunised an infant must have received 4 doses of DTP, 3 doses OPV, 3

doses of PedvaxHIB[®] or 4 doses of a different Hib preparation and 1 MMR (measles, mumps, rubella vaccine) by 24 months of age.

Only valid Australia-wide vaccinations as defined in the rules used by the ACIR are included in the assessment of each child's immunisation status. Hepatitis B (HepB) vaccination statistics for both cohorts were also calculated for local NT information but were not included in the estimates of the number of children who were fully immunised as reported in Tables 1 to 4.

The denominator used for the coverage estimates was the number of children in the respective birth cohorts on the CID on the date of assessment. All children who are born in public hospitals in the NT are entered onto the CID via reports from the Hospital Information System. Other children are entered onto the CID when immunisations given in the NT are reported to the Centre for Disease Control (CDC). Children who are deceased or who move away from the NT are archived from the CID when these details are reported to CDC.

Results

Table 1 shows the proportion of the younger cohort, children aged 12 to <15 months, in each region that are fully immunised by vaccine group. Corresponding rates from the ACIR are shown in Table 2.

Table 3 shows the proportion of the older cohort, children aged 24 to <27 months, in each region that are fully immunised by vaccine group.

Corresponding population denominators and coverage rates for children aged 24 to <27 months from the ACIR are shown in Table 4.

Table 1 Population denominators and coverage rates by vaccine group for NT children aged 12 to <15 months on 31/03/00 vaccinated by 12 months of age as at 30/06/00

	No.	DTPa3 ¹	OPV3 ²	Hib ³	HepB3 ⁴	Fully Immunised ⁵ (Not including HepB)
Darwin Urban	458	92%	92%	95%	85%	91.5%
Darwin Rural	84	92%	89%	98%	89%	89.3%
Katherine	119	86%	85%	89%	80%	82.4%
East Arnhem	70	97%	96%	97%	96%	95.7%
Alice Springs	237	87%	87%	97%	83%	85.7%
Total	968	90.2%	89.9%	95.2%	85.0%	89.0%

¹Diphtheria-tetanus-pertussis vaccine dose 3, ²Oral polio vaccine dose 3, ³*PedvaxHIB*[®] vaccine dose 2 or dose 3 of another Hib vaccine, ⁴Hepatitis B vaccine dose 3, ⁵Diphtheria-tetanus-pertussis vaccine dose 3, oral polio vaccine dose 3 and Hib vaccine dose 2 or 3.

Table 2 Population denominators and coverage statistics from the NT CID and the ACIR for NT children and the ACIR for all Australian children aged 12 to <15 months on 31/03/00 assessed on 30/06/00

	No.	DTPa3	OPV3	Hib2/3	Fully Immunised
NT - CID	968	90.2%	89.9%	95.2%	89.0%
NT – ACIR ¹	941	84.2%	84.6%	88.7%	82.7%
Australia – ACIR ¹	63,130	89.8%	89.8%	89.3%	88.4%

¹Childhood Immunisation Coverage, *Communicable Diseases Intelligence* 2000;24(7):225

Table 3 Population denominators and coverage rates by vaccine group for NT children aged 24 to <27 months on 31/03/00 vaccinated by 24 months of age as at 30/06/00

	No.	DTPa4 ¹	OPV3 ²	Hib ³	MMR1 ⁴	HepB3 ⁵	Fully Immunised ⁶ (Not including HepB)
Darwin Urban	477	72%	93%	82%	87%	85%	67.5%
Darwin Rural	72	90%	97%	92%	96%	97%	83.3%
Katherine	108	73%	89%	86%	90%	83%	67.6%
East Arnhem	76	86%	100%	93%	100%	96%	82.9%
Alice Springs	237	84%	97%	86%	94%	94%	78.9%
Total	970	77.4%	94.2%	85.3%	90.5%	88.8%	72.7%

¹Diphtheria-tetanus-pertussis vaccine dose 4, ²Oral polio vaccine dose 3, ³*PedvaxHIB*[®] vaccine dose 3 or dose 4 of another Hib vaccine, ⁴Measles-mumps-rubella dose 1, ⁵Hepatitis B vaccine dose 3, ⁶Diphtheria-tetanus-pertussis vaccine dose 4, oral polio vaccine dose 3, Hib vaccine dose 3 or 4 and MMR dose 1

Table 4 Population denominators and coverage statistics from the NT CID and the ACIR for NT children and the ACIR for all Australian children aged 24 to <27 months on 31/03/00 assessed on 30/06/00

	No.	DTPa4	OPV3	Hib3/4	MMR1	Fully Immunised
NT – CID	970	77.4%	94.2%	85.3%	90.5%	72.7%
NT – ACIR¹	956	79.4%	90.0%	86.3%	89.8%	74.6
Australia – ACIR¹	62,732	87.5%	91.9%	87.2%	91.0%	81.7

¹Childhood Immunisation Coverage, *Communicable Diseases Intelligence* 2000;24(7):225

Discussion

The algorithm used to calculate the coverage rates from NT CID data is the same as that used by ACIR and therefore the rates are directly comparable. This algorithm allows the last vaccinations on the child's schedule to be administered up to 6 months after their due date (according to the Australian Standard Vaccination Schedule) and still be included as valid vaccinations. It also allows another 3-6 months for the data to be recorded on the relevant database. These are generous allowances.

Coverage rates estimated by the NT CID for infants aged 12 to <15 months are highest in East Arnhem at 95.7%. Similarly, East Arnhem shows a high coverage rate of 82.9% for the infants aged 24 to <27 months, second only to Darwin Rural with 83.3%. These high immunisation coverage rates reflect diligence on the part of health staff and communities in vaccine administration, as well as an emphasis on reporting immunisation data.

Small numbers of children in the birth cohorts in regional areas of the NT reflect small numbers in each community so that poor coverage performance at a community level may make a large difference to the coverage estimates at a regional level.

For infants aged 12 to <15 months, the ACIR coverage rate for full immunisation is 7.3% lower than the CID rate. This difference is probably due to the problems matching NT immunisation data with ACIR records generated by Medicare enrolments, which are discussed in detail in a recent report.¹ A sustainable process to ensure a single Medicare enrolment for all NT children, together with accurate reporting of the Medicare number with all immunisations, is necessary before ACIR rates for NT children will approach those of the CID.

The NT CID immunisation coverage rate for 12 to <15 month old infants at 89.0% is on par with the national rate of 88.4%. Despite chronic staff shortages, the difficulties associated with working in an isolated community, and parents who do not receive ACIR immunisation reminder letters (as do parents in other States and Territories), NT immunisation service providers are able to achieve high coverage rates.

The ACIR immunisation coverage rate for NT children aged 24 to <27 months for the second quarter of 2000 is higher than the NT CID coverage rate. This is probably explained by over-enumerated and therefore inaccurate CID denominator data. CDC is usually not notified when children move away from the NT, so that these children remain on the CID. When their next immunisations become due, they will appear as under-immunised on the CID, as immunisations given elsewhere in Australia are not generally notified to the CID. This contrasts with the situation for the ACIR, where all immunisations given in Australia should be recorded on a child's record (via the Medicare number) and where the State/Territory of residence is updated when the child's Medicare address is changed. As the gap between scheduled immunisations increases (ie. when children become older), it is more likely that the child may have moved from the NT between immunisations and appears under-immunised on the CID. So, the CID denominator is less accurate for the 24 to <27 month old cohort than the cohort aged 12 to <15 months. This is of particular relevance to the larger urban areas of the NT, where child migration details are not known to the community care centre staff. In many remote community health centres, staff are more aware of where and when children have moved and report this information to the local CDC and hence the CID. On the other hand, children who have recently moved into the NT will not appear on the CID if they are not immunised and therefore

under-enumerate the real denominator, potentially resulting in an over-estimation of coverage. However, as the ACIR reports that 81.7% of all Australian children aged 24 to <27 years are fully immunised, it is most likely that the CID estimates for the 24 to <27 month cohort are an underestimate of the true coverage rate.

As data matching problems still exist for the 24 to <27 month cohort, the NT ACIR estimate of 74.6% is a minimum estimate. This figure is 7.1% below the ACIR national rate. The biggest component to the low ACIR estimate for full immunisation coverage is due to the coverage of DTP4: there is less than 2% difference between the NT ACIR and ACIR Australian rates for OPV3, Hib3/4 and MMR1 but a 6.1% difference for DTP4. While it is well recognised nationally and internationally that immunisation coverage rates fall slightly in older children, the marked lower rate in the older NT children is an area requiring NT parents' and immunisers' attention. Indeed, there is a need to make administration *and* reporting of dose 4 of

DTPa a priority for immunisation service delivery in those regions where coverage of this vaccination is lowest, namely Darwin Urban and Katherine regions. Reporting interstate and overseas migration to the CID will help to increase rates estimated from the CID, and overseas migration reported to the ACIR by CDC assists in improving coverage on the ACIR. Because of the large number of children living in Darwin and surrounds, the low coverage for Darwin Urban contributes significantly to the low rate for the NT. Strategies being considered by CDC to improve the uptake of the 18 month immunisation include identification of under-immunised two year olds to target their parents for individual follow-up.

Reference

1. Report on immunisation coverage data for children aged 0-7 years in the Northern Territory. Centre for Disease Control, Territory Health Services, in collaboration with General Practice Divisions Northern Territory. *Copies available on request to CDC Darwin.*

Working together to beat measles – watch this space!

The Federal Minister for Health and Aged Care, Dr Wooldridge, has announced the Commonwealth will fund the purchase of measles, mumps and rubella (MMR) vaccine for administration to all Australians aged 18-30 years. This is part of a long-term national strategy to eliminate measles from Australia and concurs with the WHO plan to eradicate measles from the world (as was done with smallpox and will soon be achieved for polio).

The NT CDC has for several years been targeting this older age group, recommending all adults born after 1960 (ie now aged up to 40 years) should receive MMR vaccination. This recommendation remains.

Adults born before 1960 are likely to have had wild measles virus infection before measles vaccination was introduced in Australia in 1968 and consequently will be immune. There are high levels of immunity to measles in adolescents and children aged under 17-18 years most of whom should have received two doses of MMR vaccine (with the Measles Control Campaign conducted in 1998 and the two dose MMR policy in place since 1994). This leaves the 18-40 year age group who may not have received effective measles vaccination but who may also not have had wild measles infection when circulation of the virus decreased after measles vaccination was introduced. Sero-surveys

have confirmed that this age group is at risk of measles infection.

Anecdotal evidence from the 1999 Victorian measles outbreak (where 84% of the cases are now aged 18-32 years) suggests young adults may incorrectly believe they are immune to measles infection. A verbal report of past vaccination or of measles disease from young adults in Australia should be viewed with caution and MMR vaccination recommended.

At this stage, it is anticipated that vaccination of young adults will be done by community care centres, community health centres and general practitioners together with hospital emergency departments, university health services and sexual health centres. Planning and collaboration with these service providers is just beginning. However, it is recognised nationally that achieving a high vaccination uptake by 18-40 year old people presents a strong challenge. In acknowledgment of this, there is a possibility of extending the program to include more innovative methods of vaccine delivery such as visiting workplaces and sporting clubs and supporting vaccination in remote communities. Watch this space! For further information please contact Christine Selvey or Nan Miller on 8922 8044.

Don't give MMR or PedvaxHIB booster doses too early!

Measles Mumps Rubella vaccine (MMR)

For optimal protection against measles infection, in an area with a low rate of measles infection, MMR vaccine should not be administered before 12 months of age. If it is given before 12 months of age, maternal antibodies may interfere with the infant's response to the vaccination so that the infant is not adequately protected against measles infection.

Before August 1998, the NT Childhood Vaccination Schedule included MMR vaccination for Aboriginal children at 9 months of age. Therefore there are many children in the NT who received their first dose of MMR vaccination before their first birthday. Some children continued to be vaccinated at 9 months of age after August 1998. In order to avoid having to catch up those children vaccinated before 12 months of age in the past, but to ensure that children do not continue to be vaccinated too early, the following rules apply (see Table).

Children born before 1 May 2000

A child who received MMR vaccination before he

or she turned 9 months of age, must receive another dose of MMR vaccine at 12 months of age and another at 4 years (ie dose 1, dose 2 and dose 3). Children who received their first dose of MMR vaccine at or after 9 months of age require only one booster at 4 years (doses 1 and 2).

Children born on or after 1 May 2000

A child who received MMR vaccination before he/she turned 11 months of age must receive another dose of MMR vaccine at 12 months of age and another at 4 years (ie dose 1, dose 2 and dose 3).

Haemophilus influenzae type b vaccine (Hib)

The schedule for Hib vaccination has not changed (PedvaxHIB due at 2, 4 and 12 months). A child who received a third dose of PedvaxHIB before he/she turned 11 months of age requires another dose after the child has turned 12 months of age. The Australian Childhood Immunisation Register will count this as the valid dose 3 (see Table).

Table Immunisation schedules when dose 1 MMR or dose 3 PedvaxHIB are given too early

MMR schedule				Hib schedule	
Child born before 1 May 2000		Child born after 1 May 2000		PedvaxHIB	
1 st MMR given before 9 months of age	1 st MMR given at or after 9 months of age	1 st MMR given before 11 months of age		All infants	
12 months	√	12 months	√	2 months	√
4 years	√	4 years	√	4 months	√
				12 months	√
Catch up doses should be given so that a total of 3 MMR doses are administered.		Catch up doses should be given so that a total of 3 MMR doses are administered.		If 3rd dose PedvaxHIB is given before child turned 11 months of age, another dose is required at 12 months of age.	

Attention all health care workers

Are you at risk of hepatitis B infection?

A recent article in *Communicable Disease and Public Health* (Volume 3, No.3, September 2000) by Alvahrani, Vallely and Klapper entitled "Needlestick Injuries and Hepatitis B Vaccination in Health Care Workers" highlighted the large number of staff members in hospitals in greater Manchester in the UK who were not protected against hepatitis B virus (HBV) infection. In a study spanning the years April 1992 to April 1999, 10% (191) of 1967 staff members with needlestick injuries who submitted specimens from 2646 needlestick injuries, had never been vaccinated against HBV. With an additional 27% of those who reported being vaccinated having a hepatitis B surface antibody (HBsAb) level of less than 10 IU/ml, over a third of all staff observed were either unvaccinated or technically HBsAb negative.

All health care workers in contact with patients' blood or body fluids should have three injections of 20 mcg of hepatitis B vaccine over a six month period.

- First injection 0
- Second injection 1 month after the first
- Third injection 6 months after the first*

*A minimum of three months should elapse between the second and third dose.

Post vaccination testing for HBsAb is recommended 1-2 months after the third dose.

Overall the risk of blood borne virus transmission can be reduced by following general infection control guidelines (ie safe work practices) and by successful vaccination of all health care workers at risk.

Don't delay! Make an appointment with your staff vaccination clinic and get fully vaccinated against HBV - then 1-2 months later have your HBsAb level checked.

Fetal Alcohol Syndrome in Australia

Ingrid Bucens, Paediatrician, Royal Darwin Hospital

Fetal Alcohol Syndrome (FAS) is a condition that affects some infants who have been exposed to alcohol *in-utero*. It is characterised by the triad of 1. pre +/- postnatal growth deficiency, 2. a characteristic facies, and 3. evidence of damage to the central nervous system. There may also be associated congenital anomalies. The term, FAS, was coined by Smith and Jones in 1973¹ though infants fitting this description had earlier been reported. There have been references to the ill effects of alcohol on the fetus as far back as the Roman times, when couples were forbidden alcohol on their wedding night "for fear of a defective child". The precise amount of alcohol and the timing of its intake that is teratogenic to the fetus is unclear, as is the mechanism of its teratogenicity. Most children with FAS however are born to mothers who are daily heavy alcohol users or relatively frequent heavy intermittent users ("binge drinkers").² Lesser alcohol intakes have been associated with partial expression of FAS^{3,4} (sometimes termed fetal alcohol effect, or alcohol related birth defects). The box on page 21 lists the

clinical features of FAS in more detail.

The most important facts about FAS are:

- a) the effects of FAS on the individual are permanent, and***
- b) FAS is an entirely preventable condition***

The majority of literature on FAS has emerged from the United States of America (USA) and Canada where Indigenous populations have been found to have particularly high rates of FAS. Reported rates of fully manifest FAS range from 0.3 - 4.6 per 1000 live births though some communities with known high rates of alcoholism have had much higher rates.⁵ These figures have attracted not only a large amount of publicity but also large amounts of funding which has led to the development of prevention campaigns and FAS diagnostic and rehabilitation clinics for those already affected. FAS has since been labelled as "the leading cause of intellectual handicap in the world."⁶

Over the last 2 years the New Zealand (NZ)

Paediatric Surveillance Unit (PSU) has been addressing the prevalence of FAS in NZ. Figures are not yet available as notified cases are still under review. Of the 21 cases notified to date, Maori children are represented more than other ethnic groups (communication via NZ PSU).

There has been little published in Australia about FAS in either Indigenous or non-Indigenous populations. The literature consists of a handful of case reports, noting children of varying ethnic groups.^{2,7,8} Data from the Western Australian Birth Defects register found a prevalence of 0.13 per 1000 live births between 1980-97. When the data was extended to include the rural paediatric service the figure was 0.18 per 1000 live births and the frequency in Indigenous children was 100 times that in non-Indigenous children. In South Australia the prevalence of FAS between 1986-1987 was 0.04 per 1000 live births (communication via Australian PSU). ***The national prevalence of FAS is unknown.***

Many Australian paediatricians suspect the prevalence of FAS is much higher than the estimates above. Given the level of alcohol consumption in Australia, FAS rates may well approach those reported from the USA. A recent medical record search at Royal Darwin Hospital (RDH) including the years 1990 to 2000 uncovered 37 children with the diagnosis of FAS.

There are inherent difficulties in making the diagnosis of FAS that are likely to explain the under-reporting of FAS. There is no biological marker for FAS; the diagnosis is clinical, and without documentation of alcohol use by the mother the diagnosis of FAS cannot be made. Several factors contribute to the under-ascertainment of FAS including:

1. Self-reporting of alcohol use is notoriously unreliable.
2. Many health professionals feel uncomfortable discussing substance use with patients because they fear the patient's response (guilt, anger, embarrassment) and/or they are untrained or unskilled to do so.
3. Most Australian antenatal clinics do not have established alcohol screening programs.
4. Professionals may fail to consider the diagnosis of FAS through lack of awareness of the condition.
5. Of those who are aware of the condition, some may lack confidence to make the diagnosis of FAS. In particular, professionals may be uncertain about the facial characteristics as

there are no "gold standard" objective criteria for confirming the presence of the features. Even more recently introduced FAS screening tools have not been validated across populations and ethnic groups. The facial phenotype also varies with age and thus its recognition depends on age at presentation.

6. There may even be a reluctance to make the diagnosis of FAS because of the social implications for the mother/family/child.
7. The inherent delay in presentation of all features necessary for the diagnosis. The facial features of FAS may not be easily recognisable in the newborn period (though some dispute this) and the neurodevelopmental delay will not be obvious until later in infancy. The possibility of alcohol induced teratogenicity may not be considered.

Failure to recognise or diagnose FAS has consequences for the individual, for other family members and for society as a whole. For the individual it means a lifetime without "a reason for their disability" and a lack of appropriately directed therapy. Siblings may be born affected if the mother continues to drink because the problem was not addressed following the effects on the first child. For society it means large costs associated with low birth weight, surgery for malformations, institutionalisation and lost productivity. ***Failure to diagnose FAS also perpetuates ignorance about the condition.***

This year there has been an increasing national focus on potentially damaging substance use in pregnancy, and specifically on FAS. There was an entire day devoted to the topic at the National Perinatal Society Conference and there was a recent feature article on FAS in "The Australian". In April the Australian Brewers' Foundation announced \$50,000 grants for alcohol related research, in particular that focussing on Indigenous issues. Shortly afterwards a national prevalence study was proposed by a group of paediatricians based in NSW. The study aims to establish the current prevalence of FAS in Australia by linking FAS diagnoses to the existing Australian Paediatric Surveillance Unit (APSU). The APSU is an active surveillance scheme that enables estimates of national incidence (or birth prevalence) of selected diseases in Australian children. Each month every Australian paediatrician (currently 971) is contacted either by e-mail or a reply-paid report card listing conditions currently being studied by APSU. They are asked to report any child newly diagnosed with any of the listed conditions seen by them in the past month. All notified cases are then investigated

further by the Unit. The Unit, which has been in place since 1992, reports a 98% response rate. By adding FAS to the notification card the APSU hope not only to estimate the incidence and prevalence of FAS in Australia but also to describe the cases by severity, by geography, socio-economic class and ethnicity. By doing so they hope to attract funding to areas with the highest rates of FAS. The group has co-investigators based in WA, SA and NT (the author). All information reported to the study is anonymous.

Since February this year a FAS Interest Group has been meeting regularly in Darwin. The group includes representatives from RDH, Territory Health Services, Menzies School of Health Research (Aboriginal Health Unit), Alcohol and Other Drugs and Danila Dilba Medical Services. The group formed with the aim of trying to assess the problem of alcohol in pregnancy/children with FAS in the Top End, in both Indigenous and non-Indigenous populations. Initially a local prevalence study was planned but this was abandoned as the group felt a study without an associated intervention was unacceptable. A community based intervention study (of antenatal education about alcohol in pregnancy) was designed but review of Top End birth rates then showed the study was not feasible. Even assuming FAS rates from the USA we could expect only 0.5 cases of FAS/year in a rural community; it would not be possible to show a difference following an intervention within "a reasonable" period of time. The group then changed its direction; ***the NT FAS Interest Group is now undertaking to improve education and understanding about the dangers of alcohol in pregnancy, and specifically about FAS, throughout the Top End.*** Currently we are in the process of contacting health and education providers (both urban and rural) in order to establish the extent of current understanding about the dangers of alcohol in pregnancy and FAS. Individuals, establishments and communities are being asked if they would like further education or information about FAS and individually tailored assistance is planned. We hope to channel all information and teaching through pre-existing health and education services.

To date our group has received a positive response from most who have been contacted about FAS. Examples of requests for further input about FAS include writing a lesson for rural Aboriginal school classes on "Alcohol and the Baby" and supplying reference materials for Northern Territory University drug and alcohol courses. We are

negotiating the introduction of alcohol screening via interview at RDH antenatal clinic and we hope to establish in-hospital education of antenatal and postnatal mothers about the dangers of alcohol in pregnancy. Other Top End health services have also expressed preliminary interest in accessing education and information about FAS.

Ultimately we hope that a widespread increase in awareness about the harmful effects of alcohol on the unborn child will reduce the numbers of those who drink alcohol in pregnancy. By inference this will reduce the number of children born with FAS.

Our group has also contacted USA, Canadian and NZ FAS experts to determine the availability of FAS teaching resources, for both Indigenous and non-Indigenous populations. Resources will be screened for local suitability by the reference group and where appropriate will be incorporated into education projects.

In the interim, a group in the Top End independently designed a laminated colour illustrated flip chart about substance use in pregnancy. The idea for the chart emanated from the women of Kalkaringi community and was designed with the help of "Living With Alcohol". The chart has been well received and, though not officially released yet, it will be a great teaching aid in FAS education.

For those children already affected by FAS the outlook is bleak. Follow-up studies have repeatedly shown persistence of neurocognitive disability (ie. intellectual handicap) into adulthood.^{9,10} There are no controlled trials demonstrating improvement with interventions although small pilot studies and anecdotal reports suggest some improvement is possible with tailored special educational assistance.¹¹ In the Top End, resources for intellectually disabled children and children with learning difficulties and behavioural problems are perceived to be inadequate, particularly for those in rural and remote areas. Currently there are insufficient resources to specifically provide services for children with FAS. Resources for special needs children in general will need to increase before this becomes a possibility. If the national prevalence study documents high rates of FAS in the NT then possibly funding will follow to address the problem.

For further information about the FAS Interest Group contact the Paediatric Departmental secretaries (extension 28373) or Dr Ingrid Bucens (through the RDH switchboard).

Clinical features of FAS

Craniofacial

- ◆ Microcephaly (80%)
- ◆ short palpebral fissures
- ◆ thin upper lip
- ◆ hypoplastic long/smooth philtrum
- ◆ retrognathia
- ◆ maxillary hypoplasia
- ◆ short upturned nose
- ◆ ptosis
- ◆ epicanthic folds
- ◆ abnormal tooth enamel
- ◆ prominent ears
- ◆ rounded forehead

Many of these features result from facial bone hypoplasia. Ophthalmic structural and functional abnormalities are common (visual impairment is present in > 50% patients).

Damage to Central Nervous System

- Functional:
- ◆ intellectual handicap (70-80% have IQ 50-70)

- ◆ hyperactivity and inattention (behavioural problems)
- ◆ poor coordination/hypotonia
- ◆ developmental delay
- ◆ speech problems
- ◆ fine motor dysfunction
- ◆ epilepsy (10%)
- ◆ auditory and visual deficits
- Structural:
- ◆ Microcephaly
- ◆ *numerous other structural abnormalities have been described*

Congenital Malformations

- ◆ Skeletal (eg joint abnormalities, tapering phalanges, hypoplastic nails etc)
- ◆ Cardiac (VSD, ASD etc)
- ◆ other (includes renal, genital, hirsutism etc)

Neonatal abnormalities

- ◆ abnormal habituation/arousal/sleep/attention
- ◆ withdrawal phenomena
- ◆ poor feeding/irritability

References

1. Jones KL and Smith DW. *Lancet* 1973; 2: 999.
2. Clarren SK, Smith DW. The Fetal Alcohol Syndrome. *N Engl J Med* 1978; 298 (19): 1063-7.
3. Larroque B, Kaminski M et al. Moderate Prenatal Alcohol Exposure and Psychomotor Development at Preschool Age. *Am J Pub Health* 1995; 85 (12): 1654-9.
4. Streissguth AP, Martin JC et al. The Seattle Longitudinal Prospective Study on Alcohol and Pregnancy. *Neurobehavioural Toxicology and Teratology* 1981; 3: 323-3.
5. Sampson PD, Streissguth AP et al. Incidence of Fetal Alcohol Syndrome and Prevalence of Alcohol Related Birth. *Neurodevelopmental Disorder. Teratology* 1997; 56: 317-26.
6. Walpole IR, Hockey A. Fetal Alcohol Syndrome: Implications to Family and Society in Australia. *Aust Paediatr J* 1980; 16: 101-5.
7. Collins E, Turner G. Six Children Affected by Maternal Alcoholism. *Med J Aust* 1978; 606-7.
8. Lipson AH, Walsh DA, Webster WS. Fetal Alcohol Syndrome. A Great Paediatric Imitator. *Med J Aust* 1983; 1: 266-9.
9. Streissguth AP, Clarren SK, Jones KL. Natural History of the Fetal Alcohol Syndrome: a 10 year follow-up of 11 Patients. *Lancet* 1985; ii, 85-91.
10. Spohr HL, Willms J, Steinhausen HC. Prenatal Alcohol Exposure and Longterm Developmental Consequences: a 10 year follow-up of 60 Children with Fetal Alcohol Syndrome. *Lancet* 1993; 341: 907-10.
11. Morse BA and Weiner L. Rehabilitation Approaches for Fetal Alcohol Syndrome. Ch 13 in *Alcohol, Pregnancy and the Developing Child*. Ed Spohr HL, Steinhausen HC. 1st ed. 1996. Cambridge University Press.

Editorial

The above article highlights that Fetal Alcohol Syndrome (FAS) is a preventable condition and that recognition of the syndrome has been difficult and therefore prevention strategies problematic. A group from the University of Washington in the USA developed the FAS Diagnostic and Prevention

Network (FAS DPN) with the aim of identifying the populations of women at highest risk of producing FAS children, ie. mainly women who had already given birth to an alcohol affected child.¹

A study from this Network (1993-1997) revealed

some interesting findings. Of 87% of caregivers (1192/1374) responding to a questionnaire the referral source most commonly identified was social service agencies (28%) which included identifying persons through photographic screening of high risk populations. The next most common source was medical care providers (22%), followed by mental health providers (15%), FAS support organisations (12%), self referrals (10%), school personnel (5%) and others (4%). That only 22% were being referred by medical care providers raises the importance of a multi-disciplinary approach to identify the population at risk and to facilitate the proper diagnosis. This is further emphasised by the variety of reasons prompting referral to the FAS DPN in the study with the top 5 reasons being:

- conduct disorder or extreme anger (46%),
- learning disabilities or cognitive delays (32%),
- short attention span (29%),
- poor judgement or inability to function independently (19%), and
- poor self control, disorganised or unpredictable behaviour (19%).

The syndrome presents itself in ways that may not be recognised in the traditional medical setting.

Reference

1. Clarren SK, Astley SJ. Identification of children with Fetal Alcohol Syndrome and opportunity for referral of their mothers for primary prevention Washington, 1993-19.

Pelvic Inflammatory Disease (PID) in the Top End: a condition that needs closer attention

*Steven Skov, Matthew Parnaby, Karen Dempsey, David McDowall, Adeline Drogemuller, Simon Morgan
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Over the last eight months a major review of STD/HIV programs in a large Top End region has been conducted. This was done in partnership between the region's health services and the Territory Health Services AIDS/STD program. The review was part of a process to develop a regional STD/HIV strategy. The first stage of this strategy has uncovered a number of areas where the service can be improved. Specific plans have been made and are currently being implemented to do so. Progress will be evaluated next year and the overall strategy revised by the region's health services with the assistance of the AIDS/STD program. This paper aims to raise awareness of pelvic inflammatory disease (PID) as a significant public health problem in this region and make recommendations to improve its prevention and management.

PID in the Top End

In the Top End of the Northern Territory rates of gonorrhoea and chlamydia are close to the highest in the nation.^{1,2} Both of these infections can progress to cause PID³ with its sequelae of chronic pelvic pain, infertility and ectopic pregnancy. Until now there has been little knowledge of how much PID and infertility there is in the Top End. However, two recent studies suggested that 26% and 30% of women in two Top End communities were infertile and that 26% of women in one of the communities had probably had at least one episode of PID.^{4,5} A recent random case note audit of 10%

of women in all communities of a large Top End region suggested that 30% of women had probably had an episode of PID and that 20% of women over the age of 24 did not appear to have had a child and may be infertile. In comparison, during the height of the gonorrhoea epidemics in Europe and the US in the 1960's and 1970's, only 15% of women in Sweden and 10% of women in the US reported ever being treated for PID.^{6,7}

Gonorrhoea, chlamydia, PID and sequelae

PID develops when infection from the lower genital tract ascends to affect the uterus and fallopian tubes. Organisms other than gonorrhoea and chlamydia can cause PID. Gonorrhoea or chlamydia may be present in the fallopian tubes but not be detectable at the cervix. Between 20-40% of women with untreated chlamydial or gonococcal infections will develop symptomatic PID.^{8,9} PID can present in a variety of ways.³ Women can have a severe illness with fever, severe abdominal pain and vaginal discharge. They can also present with no fever and much milder symptoms and signs: abdominal pain, dysuria, dyspareunia, menstrual irregularities and no vaginal discharge. Or they can have no symptoms at all, so-called "silent PID", particularly when it is due to chlamydial infection.

The risk of a woman becoming infertile or having an ectopic pregnancy increases with each episode of PID. By the third episode the risk of infertility is

increased by 54%.¹⁰ Animal studies suggest that PID can develop within 5 days of infection with chlamydia.¹¹ If a woman is not treated within three days of the onset of pain, her risk of infertility increases three times.¹² One study found that among women whose treatment was delayed six days, one third became infertile.¹³

Reductions in rates of gonorrhoea and chlamydia have been strongly associated with a fall in hospital admissions for PID and ectopic pregnancy.^{14,15} Chlamydia screening programs have been shown to reduce the incidence of PID.^{16,17}

How are these conditions being managed in the Top End?

Treatment of diagnosed PID

There are a number of recommended treatments for PID in various references and treatments manuals. However, they all include two weeks of treatment for chlamydia usually with doxycycline. Single dose treatment is only appropriate for *uncomplicated lower genital tract infections*.

During the Top End region's review it was found that, in the majority of cases where the specific diagnosis of PID was made, management was inappropriate. The most common error was in not giving two weeks of treatment for chlamydia. Close follow-up and clinical re-assessment of women with PID is generally also recommended but this was infrequently done. The Top End is not alone in this situation: a recent review in England and Wales found that general practitioners diagnosed and managed PID in a very variable and generally sub-standard fashion.¹⁸

Single dose treatment of gonorrhoea and chlamydia

Many infections with these organisms are diagnosed with a screening test (eg well-woman's test or opportunistic test) and the woman needs to be followed up for treatment. The Top End region's review found that there was almost never any assessment of these women for possible PID. They were simply given the single dose treatment. This even occurred in women who had presented initially with lower abdominal pain. Guidelines in the NT recommend single dose treatment *only* for *uncomplicated lower genital tract infections*: PID requires two weeks of treatment.

Women presenting with lower abdominal pain +/- dysuria

This is a very common presentation to clinics and is very often managed as a urinary tract infection. Sometimes a tampon or urine PCR test is done. In the Top End region review it was found that when a PCR test was done 20% of women with dysuria alone and 24% of women with abdominal pain and dysuria had infection with either gonorrhoea, chlamydia or trichomonas. All of these infections can present in this way whether as PID or as lower genital tract infections.

Where to from here for controlling PID?

There are four key points to bear in mind.

- Gonorrhoea and chlamydia are very common in this region, as is PID.
- Active detection and treatment programs for gonorrhoea and chlamydia can reduce progression to PID.
- Infections must be detected and treated as soon as possible, because in women with symptoms of PID, even minor delays in treatment can lead to a greatly increased risk of infertility.
- PID requires two weeks of treatment.

Be more aware of the possibility of STDs and PID

When seeing women with lower abdominal pain refer to the protocol on page 214 of the third edition of the Nganampa/Alukura Women's Business Manual. Consider the possibility of STDs and offer a PCR test. If there is reasonable suspicion of PID then the woman should have a speculum or bimanual examination. This should include endocervical swabs for both PCR *and* culture. If you think that PID is not very likely, or a full examination is not possible, then a PCR test is still worth doing. A tampon or self-administered swab is the best, but a urine test will still do if the others are not possible. Think about whether PID is possible in any woman with lower abdominal pain.

Think about PID when treating for gonorrhoea and chlamydia

When following up a woman who has had gonorrhoea or chlamydia diagnosed previously *DO NOT just give out the single dose treatment*.

Ask the woman if she has had:

- any lower abdominal pain
- any deep dyspareunia (pain deep inside with sex)

- any vaginal discharge
- any period problems recently (eg bleeding between periods, heavier than usual)

If she answers "no" to all these questions then give single dose treatment.

If she answers "yes" to any of them, then consider doing a full assessment with a speculum and bimanual examination. Take endocervical swabs for MC&S and PCR.

If there is any adnexal tenderness, or pain when moving the cervix or discharge from inside the cervix then treat as for PID. If none of these are present then give the single dose treatment.

Management of PID

The Women's Business Manual is now available. There was some concern in the Top End about the antibiotics it recommends for the treatment of PID. This has been reviewed and consultations undertaken with staff throughout the Top End. As a result the Top End has determined that PID should be treated as follows.

- **Amoxicillin** 3g with **probenecid** 1g and **azithromycin** 1g by mouth once.
- If the woman is allergic to penicillin or pregnant, talk with a doctor.
- Continue treatment the next day with:
 - **Doxycycline** 100 mg by mouth twice a day for 1 week, AND **metronidazole** 400mg by mouth twice a day for 1 week
- SEE THE WOMAN IN ONE WEEK to give:
 - **Azithromycin** 1g by mouth once more AND the same doses of **doxycycline** and **metronidazole** for one more week.
- If, on the first day, you are not sure of being able to see the woman again, give her two weeks worth of doxycycline and metronidazole.
- give **paracetamol** or **paracetamol-codeine** 2 tablets by mouth every 4 hours as needed for pain relief.

What is most important is that:

- A woman has *two weeks* of treatment (even if the swab results are negative).
- She be clinically reviewed. Ideally, she should be seen at three days to see that she is improving (if not improving, send to hospital). The *absolutely minimal follow-up* would be to see her at one week to make sure she is taking her medication and that she is improving. She

should also be seen at two weeks to have another bimanual examination.

- Her partner(s) be treated for both gonorrhoea and chlamydia.

Community Education

The AIDS/STD Unit has developed a new story book for community education on this issue. This book was developed over the course of four workshops for female Aboriginal Health Workers from the region. The story book will be used by AIDS/STD staff and will be available for community staff to use also.

As part of the ongoing process of developing the regional STD/HIV strategy, further ideas will be explored and developed between the region's health services and the AIDS/STD Unit.

References

1. HIV/AIDS and related diseases in Australia. Annual Surveillance Report 1998. National Centre in HIV Epidemiology and Clinical Research.
2. NT notifiable diseases database.
3. Westrom L, Eschenback D. Pelvic inflammatory disease. In: K.K. Holmes et al (eds) Sexually Transmitted Diseases (3rd ed), 1999 McGraw Hill, New York.
4. Kildea S. A retrospective epidemiological study comparing fertile and infertile women in a remote indigenous community in Australia. Unpublished masters thesis Southern Cross University.
5. Anonymous. The prevalence, aetiology and management of infertility in an Aboriginal community. Unpublished report, Territory Health Services, Darwin.
6. Westrom L. Decrease in incidence of women treated for acute salpingitis in Sweden. *Genitourin Med* 1988; 64: 59-63.
7. Eschenbach D, Harnisch J, Holmes KK. Pathogenesis of acute pelvic inflammatory disease: role of contraception and other risk factors. *Am J Obstet Gynecol* 1977; 128: 838-50.
8. Platt R, Rice PA, McCormack WM. Risk of acquiring gonorrhoea and prevalence of abnormal adnexal findings amongst women recently exposed to gonorrhoea. *JAMA* 1983; 250: 3205-9.
9. Stamm WE, Guinan ME, Johnson C, Starcher T, Holmes KK, McCormack WM. Effect of treatment regimes for *Neisseria gonorrhoea* on simultaneous infections with *Chlamydia trachomatis*. *N Engl J Med* 1984; 310: 545-9.
10. Westrom L. Sexually transmitted diseases and infertility. *Sex Transm Dis* 1994; March-April Supplement: 32-37.
11. Swenson C, Schacter J. Infertility as a consequence of chlamydial infection of the upper genital tract in

- female mice. *Sex Transm Dis* 1984; 11: 64-67.
12. Hillis S, Joesoef R, Marchbanks P, Wasserheit JN, Cates W Jr, Westrom L. Delayed care of pelvic inflammatory disease as a risk factor for impaired fertility. *Am J Obstet Gynecol* 1993; 168: 1503-9.
 13. Viberg L. Acute inflammatory conditions of the uterine adnexa. *Acta Obstet Gynecol Scand* 1964; 4 (suppl 4): 1-28.
 14. Kamwendo F, Forslin L, Bodin L, Danielsson D. Decreasing incidences of gonorrhoea- and chlamydia associated acute pelvic inflammatory disease. *Sex Transm Dis* 1996; 23: 384-90.
 15. Kamwendo F, Forslin L, Bodin L, Danielsson D. Programmes to reduce pelvic inflammatory disease - the Swedish experience. *Lancet* 1998; 351 (suppl III) S25-8.
 16. Scholes D, Stergachis A, Heidrich FE, Andrilla H, King K, Holmes KK et al. Prevention of pelvic inflammatory disease by screening for cervical chlamydial infection. *N Eng J Med* 1996; 334: 1362-1366.
 17. Hillis S, Nakashima A, Amsterdam L, Pfister J, Vaughn M, Addiss D et al. The impact of a comprehensive chlamydia prevention program in Wisconsin. *Fam Plan Perspect* 1995; 27: 108-111.
 18. Simms I, Vickers M, Stephenson J. National assessment of PID diagnosis, treatment and management in general practice: England and Wales. Thirteenth meeting of the International Society for Sexually Transmitted Diseases Research. Denver, USA, 1999. Abstract No 305.

Towards a Sexual Health Strategy for remote communities in the Northern Territory

Steven Skov, AIDS/STD Unit, Darwin

In late 1998 it became apparent that very few primary health care services had a specific STD/HIV strategy, there was very little collaboration between health services, and there were no regional or NT policies to provide a strategic framework.

Therefore, in March 1999, a project officer began work half-time with the Darwin AIDS/STD Remote Team to coordinate the development of an NT wide sexual health strategy for remote communities. All primary health care agencies, THS, community controlled, and grant-in-aid, were approached *to seek a partnership in developing such a strategy*. The National Indigenous Australians' Sexual Health Strategy would form a framework for the NT strategy. Rather than attempt to write a strategy from regional offices in Darwin or Alice Springs, it was decided to try to build the strategy from the ground up.

In central Australia, the Tri-State STD/HIV project and the THS Sexual Health Unit in Alice Springs had been doing collaborative work with all health services in central Australia for some years. After discussions with central Australia, it was agreed that the Darwin AIDS/STD remote team would focus on the Darwin, Katherine and East Arnhem districts and the Tri-State and SHU would remain responsible for central Australia.

In the Top End, a practical decision was made to focus on one region at a time and to begin where there was some enthusiasm for the idea. At about

the same time, the Darwin Remote district was beginning to develop their business plan. At meetings of community based staff and regional management, it was agreed to include STD/HIV as one of the six priority areas for the district and to work with the AIDS/STD team to develop a strategy for the district. The Darwin Remote strategy was developed via the following process:

- conduct a thorough review of all aspects of STD/HIV programs at community level,
- consult with staff about possibilities for STD/HIV programs,
- develop a draft strategy on the basis of the initial review and consultations, and
- refine the strategy via a dialogue between Darwin Remote and the AIDS/STD program.

There are five major facets to the strategy which is now in an implementation phase:

- Improve treatment rates of STDs by establishing a system within the AIDS/STD unit to assist clinics with treatment and follow-up.
- Re-establish coding of all HIV tests.
- Increase awareness and improve management of PID.
- Enhance contact tracing by establishing simple clinic based systems with support from the AIDS/STD unit.
- Develop community and regional plans to prepare for an HIV positive diagnosis.

A key concept in this process is that the ultimate responsibility for the strategy remains with Darwin Remote while the role of the AIDS/STD unit is to assist and support. It is a partnership between the two.

This very clinically orientated program should be seen as phase one of a longer term strategy. An appropriate time for a first evaluation will probably be early in 2001. Once these aspects of health service delivery are working well, the region can broaden its focus. Engaging community members and groups in the process will be an essential step. The ultimate aim will be to develop a comprehensive program consisting of good clinical care, community education and health promotion and early detection and treatment strategies.

A similar review has been done for the Tiwi Health Board and the process of devising their STD/HIV program will begin shortly. In the Katherine region both the Katherine West Health Board and THS remote have expressed interest in the same process

and it will be also be offered in the East Arnhem region.

The Tri-State STD/HIV project and the Sexual Health Unit in Alice Springs are proceeding in a very similar fashion working with both remote community health services and with Congress in town. This recognises the links between remote community and town based health services and the importance of bringing them together as part of a coherent, regional strategy.

The development of regional strategies "from the ground up" is the first step of building an overall NT strategy. The process used should ensure that these regional strategies are appropriate, feasible, have grass roots support and are focused on day to day, easily measurable and practical outcomes. The Top End and central Australia will then be able to draw together the common and key principles of the regional strategies to define a policy and strategy framework for the whole of the NT.

Points to note regarding notifications on page 27

- AIDS, Amoebiasis, Kunjin, Kokobera, Botulism, Brucellosis, Chancroid, Cholera, Congenital Rubella Syndrome, Congenital Syphilis, Diphtheria, Gastroenteritis, Gonococcal Conjunctivitis, Haemolytic Uraemic Syndrome, Haemophilus Influenza type b, Hepatitis C (incidence), Hepatitis D & E, Hydatid Disease, Leprosy, Leptospirosis, Listeriosis, Lymphogranuloma venereum, Mumps, Pertussis, Poliomyelitis, Rubella, Typhoid, Typhus, Vibrio Food Poisoning and Viral Haemorrhagic Fever are all notifiable but had "0" notifications in this period.
- There were 33 cases of dengue in this quarter compared to none the year before. All cases were acquired overseas with 30 from East Timor and 3 from Indonesia.
- Similarly for malaria cases, there were 21 and 8 in the second quarter this year and last year respectively with 12 of this year's cases acquired in East Timor compared to none for the same time last year.
- A cluster of Murray Valley Encephalitis (MVE) cases followed unusually heavy rainfall and high mosquito (*Culex*) numbers this year, particularly around Alice Springs, an area not usually reporting any MVE activity. A similar explanation may account for the increase in Ross River Virus notifications.
- It is likely that the higher number of trichomoniasis notifications this year compared to last year may reflect increased screening. Trichomoniasis only became a notifiable disease last year.
- Syphilis notifications are being monitored to see if this year's decrease in number will be sustained over time.
- Although there was a large increase in the number of cases of cryptosporidiosis, no common source was found.
- There were a number of outbreaks of acute post-streptococcal glomerulonephritis in remote Top-End aboriginal communities. Such outbreaks occur every few years. Interventions to control scabies and skin sores were implemented in these communities. Review articles of these outbreaks will be featured in the next issue of the *Bulletin*.
- The 19 TB notifications this quarter include 2 non-Aboriginal, non overseas-born cases, 5 overseas-born cases (2 of whom are unauthorised persons presently in custody), and 12 cases in Aboriginal people (reflecting an increase in disease specifically in one Top End community and also in one central Australian community).

**NT NOTIFICATIONS OF DISEASES BY DISTRICTS
1 APRIL TO 30 JUNE 2000 AND 1999**

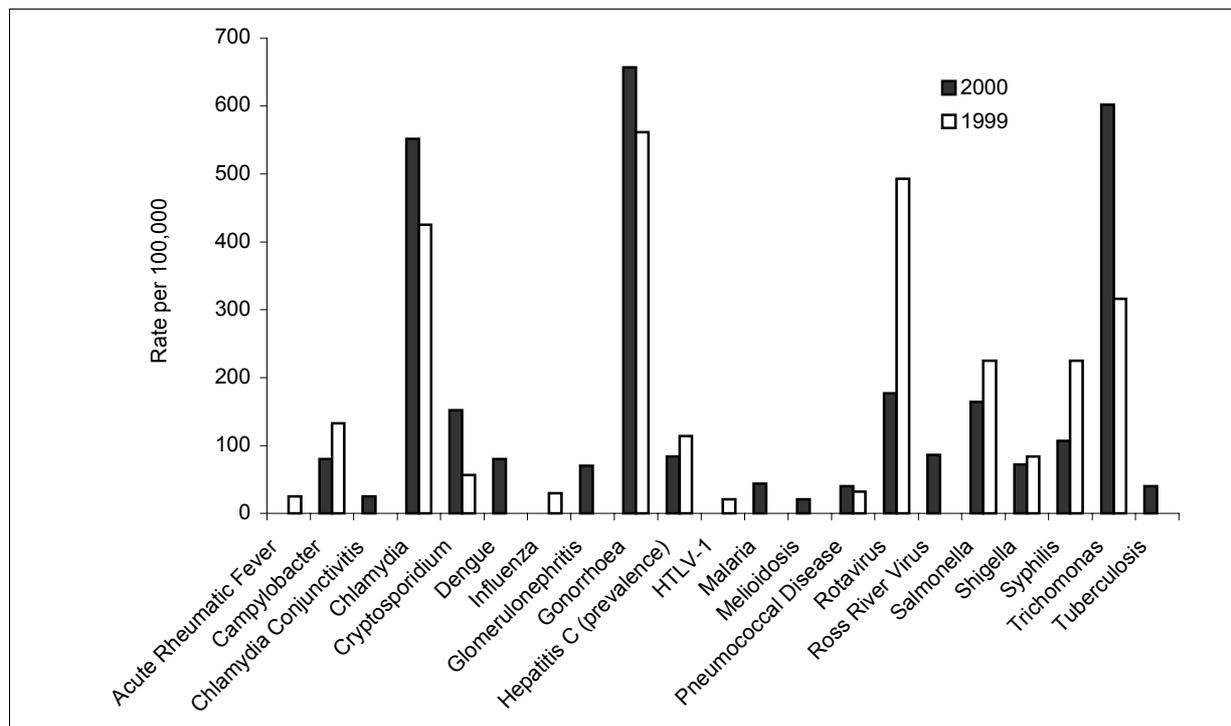
DISEASES	ALICE SPRINGS		BARKLY		DARWIN		EAST ARNHEM		KATHERINE		TOTAL	
	2000	1999	2000	1999	2000	1999	2000	1999	2000	1999	2000	1999
Acute Rheumatic Fever	3	2	1	2	2	1	1	3	2	4	9	12
Adverse Vaccine React.	0	0	1	1	4	1	0	0	1	2	6	4
Arbovirus infections												
Barmah Forest Virus	1	0	0	0	4	2	0	1	0	0	5	3
Dengue	0	0	1	0	37	0	0	0	0	0	38	0
Kunjin Virus	1	0	0	0	1	1	0	0	0	0	2	1
Murray Valley Enceph.	4	0	0	0	2	0	0	0	0	0	6	0
Ross River Virus	16	0	4	0	14	7	1	0	6	2	41	9
Atypical Mycobacteria	0	0	0	0	1	0	0	1	0	1	1	2
Campylobacter	13	20	1	0	18	39	2	2	4	2	38	63
Chlamydia	133	79	1	5	77	92	17	7	34	19	262	202
Chlamydia Conjunct.	1	3	0	0	11	1	0	0	0	1	12	5
Cryptosporidiosis	19	8	3	0	34	11	0	2	16	6	72	27
Donovanosis	0	0	0	0	1	0	0	0	0	0	1	0
Glomerulonephritis	0	0	0	1	5	0	24	0	4	0	33	1
Gonococcal Disease	201	90	8	16	44	99	14	29	45	33	312	267
Hepatitis A	1	0	0	1	7	2	0	0	0	0	8	3
Hepatitis B	1	0	0	1	0	2	0	0	2	0	3	3
Hepatitis C (prevalence)	3	1	0	0	36	50	0	1	1	2	40	54
HIV infections	0	0	0	0	2	3	0	0	0	0	2	3
HTLV-1	3	7	0	1	0	2	0	0	1	0	4	10
Influenza	2	1	0	0	1	9	2	2	0	2	5	14
Legionnaires Disease	0	0	0	0	0	0	0	0	0	1	0	1
Malaria	1	0	0	0	20	7	0	0	0	1	21	8
Measles	0	0	0	0	0	4	0	0	0	0	0	4
Melioidosis	0	0	0	0	9	3	0	1	1	1	10	5
Meningococcal Infection	0	1	0	0	2	0	1	1	0	1	3	3
Pneumococcal Disease	11	7	0	0	7	5	1	0	0	3	19	15
Rotavirus	72	63	2	37	4	67	1	26	5	41	84	234
Salmonella	23	14	3	4	35	60	3	7	14	22	78	107
Shigella	12	23	0	2	14	12	7	1	1	2	34	40
Syphilis	25	34	0	17	22	20	1	21	3	15	51	107
Trichomonas	77	10	6	5	79	69	54	50	70	16	286	150
Tuberculosis	3	1	1	0	14	4	0	0	1	1	19	6
Vibrio Food Poisoning	0	0	0	0	0	0	0	0	0	1	0	1
Total	626	364	32	93	507	573	129	155	211	179	1505	1364

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

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

• Mumps is largely under-reported.

**NT WIDE NOTIFIABLE DISEASES
1 APRIL TO 30 JUNE 2000 AND 1999**



Rates <10/100,000 not listed
NT estimated residential population - 189,987 supplied by Epidemiology & Statistical Branch, THS

NT MALARIA NOTIFICATIONS - APRIL TO JUNE 2000

Merv Fairley, CDC, Darwin

Eighteen notifications of malaria were received for the second quarter of 2000. 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	COMMENTS
PNG	Work	<i>P. vivax</i>	No	Diagnosed RDH
PNG	Study	<i>P. vivax</i>	Yes	Diagnosed RDH
Indonesia	Work	<i>P. vivax</i>	No	Diagnosed RDH
Indonesia	Holiday	<i>P. falciparum</i>	No	Diagnosed RDH
Indonesia	Holiday	<i>P. falciparum</i>	No	Diagnosed RDH
Indonesia	Resident	<i>P. falciparum</i>	No	Diagnosed RDH
Indonesia	Resident	<i>P. falciparum</i>	No	Diagnosed RDH
Indonesia	Holiday	<i>P. vivax</i>	No	Diagnosed RDH
East Timor	Work	<i>P. falciparum</i>	Yes	Diagnosed RDH
East Timor	Work	<i>P. falciparum</i> & <i>P. vivax</i>	Unknown	Diagnosed RDH
East Timor	Work	<i>P. vivax</i>	Yes	Diagnosed RDH
East Timor	Work	<i>P. vivax</i>	Yes	Diagnosed Westerns
East Timor	Work	<i>P. vivax</i>	Yes	Diagnosed Westerns
East Timor	Work	<i>P. vivax</i>	Yes	Diagnosed Westerns
East Timor	Work	<i>P. vivax</i>	No	Diagnosed RDH
East Timor	Work	<i>P. vivax</i>	Yes	Diagnosed RDH
East Timor	Work	<i>P. vivax</i>	No	Diagnosed RDH
S America	Work	<i>P. vivax</i>	No	Diagnosed RDH

CDC staff updates

East Arnhem

Vivien Braybrook has been appointed to the STD/HIV position, CDC Gove, replacing **Karen Blyth** who is on extended leave until 1 August 2001. Vivien is well known to most East Arnhem District staff having worked at Marn Garr Health Centre and more recently with the Chronic Diseases Recall team. Her experience and knowledge will be of great value to the team.

Ross Jackson commenced as the Men's Health Educator in June. Ten of his 24 years nursing experience has involved working in Indigenous health, taking him to places such as Alice Springs, Katherine, Maningrida, Gapuwiyak, Normanton and Yarrabah. He most recently worked in men's sexual health in the Torres Strait.

Alice Springs

Margaret Stebbing recently took up a 12 month

position in China with MSF Holland to work on the Nujiang TB Assistance Project. The CDC Public Health Nurse position has recently been advertised and is expected to be filled soon.

Tennant Creek

Robin Freeman recently resigned from the Public Health Nurse position. **Celina Bond** (formerly remote area relief nurse for the Barkly region as well as theatre nurse in Tennant Creek Hospital) has been appointed into the AIDS/STD position.

Darwin

Steve Morton finished up in his position as Chronic Disease Project Officer on 30 September. In line with recommendations from an evaluation carried out in July of this year, the position has been re-designed and should soon be advertised in its new form. Many thanks to Steve for all his hard work over the last three years.

Disease Control Policies, Protocols and Guidelines

The table below lists policies, protocols and guidelines relevant to the Disease Control Program. Copies of individual documents can be downloaded from the Disease Control Bulletin Board (now referred to as discussion databases), the THS Intranet Site (under Public Health/Disease Control/Guidelines & Protocols) or by contacting your regional CDC.

Policies, protocols & guidelines
Acute post streptococcal glomerulonephritis Guidelines for the control of acute post-streptococcal glomerulonephritis (August 1997)
Anaphylaxis Management of anaphylaxis in the urban setting (March 2000) Management of anaphylaxis in the rural setting (March 2000)
Communicable Disease Surveillance in the NT Guidelines for the reporting of notifiable conditions (February 2000) Notifiable conditions to be reported by all clinicians in the NT (March 2000) - WALL CHART Notifiable conditions to be reported by all laboratories in the NT (March 2000) - WALL CHART
Congenital syphilis Guidelines for the investigation and treatment of infants at risk of congenital syphilis in the Top End of the NT (November 1998)
Diphtheria Guidelines for the control of diphtheria in the NT (January 1998)
Exclusion periods 'Time Out' – Recommended minimum periods of exclusion from school, pre-school and child care facilities for children or staff with, or exposed to, infectious diseases (March 1999) – WALL CHART
Gonococcal conjunctivitis Guidelines for the control of gonococcal conjunctivitis (August 1997)
Hepatitis A NT Hepatitis A vaccination policy and public health management guidelines (February 2000)
Hepatitis B NT Hepatitis B vaccination policy and public health management guidelines (June 2000)
Invasive haemophilus influenzae type b infections Refer to relevant section in the current edition of <i>The Australian Immunisation Handbook</i> (NHMRC)
Leprosy Guidelines for leprosy control in the NT (1996)
Lyssavirus - FLOW CHART Australian bat lyssavirus post-exposure prophylaxis (PEP) - (April 2000)
Malaria Guidelines for health professionals in the NT - 3rd edition (January 1997) Revision and review of NT protocol (memorandum dated 7 October 1999)
Measles NHMRC – Guidelines for the control of outbreaks in Australia (August 1996)
Meningococcal disease Guidelines for meningococcal meningitis/septicaemia chemoprophylaxis (December 1997) NHMRC - Guidelines for the control of meningococcal disease in Australia (October 1996)
Outbreak management A framework for investigating outbreaks in the Northern Territory (May 2000)
Pertussis Communicable Diseases Network Australia New Zealand: Technical Report Series No 1: Guidelines for the control of pertussis in Australia (November 1997) http://www.health.gov.au/pubhlth/strateg/communic/tech/pertus.htm
Scabies Guidelines for community control of scabies and skin sores (September 1997)
Trachoma Guidelines for treatment of trachoma in the NT (1998)
Tuberculosis Guidelines for the control of tuberculosis in the NT (September 1997) The Central Australian Tuberculosis Control Program – TB Control Strategy (June 1997)
Vaccination Schedules Northern Territory Standard Childhood Vaccination Schedule, 1/5/2000 (March 2000) Northern Territory Adult and Special Groups Vaccination Schedule (March 2000)

