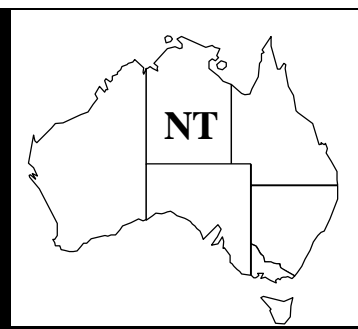




# THE NORTHERN TERRITORY DISEASE CONTROL BULLETIN



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## Usefulness of a self-reported history of chickenpox in adult women in the Top End

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### Introduction

Early determination of immune status is essential for the prevention and/or amelioration of disease following exposure to chickenpox. This is of particular significance for pregnant women because of the additional risks to the foetus or newborn.<sup>1</sup> To determine the usefulness of a self-reported history of chickenpox in adult women in the Top End, we compared it with serological evidence of immunity.

### Methods

As part of a seroprevalence survey of women over 14 years of age attending antenatal clinics at the Royal Darwin Hospital (unpublished data), a self-reported history of chickenpox was obtained. Data were collected with an interviewer-administered questionnaire. Women were asked, "Have you ever had chickenpox?" Responses were categorised as "Yes - definitely" if they could recall illness, "Yes - probably" if they responded "I think so" or "my mother told me I had", "No" if they believed they had never had chickenpox and "Unknown" if they didn't know.

Sera were tested for VZV IgG using a commercial enzyme immunoassay (Enzygost, Behring Diag-

nostics, Germany) at the Centre for Infectious Diseases and Microbiology, Institute of Clinical Pathology and Medical Research (ICPMR), Westmead NSW.

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The Joint Institutional Ethics Committee of the Royal Darwin Hospital and the Menzies School of Health Research approved the study.

## Results

Of 315 women approached, 298 agreed to participate. The median age was 26 years (range 15-45). Ninety two percent of women were seropositive; 40% reported a definite, and 11% a probable, history of chickenpox. A definite history of chickenpox was reported by 41% of seropositive and 29% of seronegative women giving positive and negative predictive values (PPV and NPV) of 96% and 7% respectively (Table 1).

Only 19% of indigenous women (n=98) gave a definite history of chickenpox, the sensitivity, specificity and PPV of which were 21%, 5% and 95% respectively.

**Table 1 Correlation between self-reported history of chickenpox and VZV antibody status**

History	Sero-positive	Sero-negative	Equi-vocal	History total (% all total)
Yes - definitely	113	5	0	118 (40%)
PPV (%)	(96)	-	-	-
Sensitivity [%]	[41]	-	-	-
Yes - probably	31	3	0	34 (11%)
PPV (%)	(91)	-	-	-
Sensitivity [%]	[11]	-	-	-
No	59	5	5	69 (23%)
NPV (%)	-	(7)	-	-
Specificity [%]	-	[29]	-	-
Unknown	72	4	1	77 (26%)
<b>Total %</b>	<b>275 (92)</b>	<b>17 (6)</b>	<b>6 (2)</b>	<b>298 (100%)</b>

Explanatory notes:

- Sensitivity – for all women who are antibody positive, the proportion who report a positive history (true +ve).
- Specificity – for all women who are antibody negative, the proportion who report a negative history (true –ve).
- Positive Predictive Value (PPV) – of all women who report a positive history, the proportion who are antibody positive.
- Negative Predictive Value (NPV) – of all women who report a negative history, the proportion who are antibody negative.

## Discussion

Although the majority (92%) of women were seropositive, recall of past illness in this study population was poor. Only 51% gave a definite or probable history of chickenpox. Other studies have reported sensitivities between 51% and 92%.<sup>2-4</sup> Differences may reflect variations in age at time of illness, the severity of disease, frequency of subclinical infection and/or differences in the socio-economic and demographic characteristics of the study populations.

Recall in indigenous women was considerably lower than in non-indigenous women. While language or cultural differences may contribute to this result, it could also reflect milder or more commonly subclinical disease in this population. Knowledge of chickenpox morbidity in the Aboriginal population is limited, and anecdotal reports indicate that it is uncommon in remote communities or maybe unrecognised, particularly in areas with a high incidence of scabies and impetigo, which can confuse the diagnosis.<sup>1</sup>

A PPV of 96% indicates that a self-reported history of chickenpox is a reliable predictor of immunity in both indigenous and non-indigenous women but a negative history is unreliable. It is recommended elsewhere that those with a positive history of chickenpox should be considered to be immune and not tested serological.<sup>5</sup> Consideration must be given however to the proportion of susceptible women who would be misclassified if histories alone are used for screening.

Nearly 30% of seronegative women gave a definite and 18% a probable history of chickenpox. Although the actual numbers are small, this means that nearly 50% of seronegative women overall and 80% of indigenous women, would not have been detected if history alone were used to determine immunity. In addition presumptive vaccination on history alone has not been shown to be cost effective.<sup>6</sup>

If a VZV vaccine is introduced to northern Australia, opportunistic screening of women of child-bearing age may be a useful approach to identify women needing immunisation prior to pregnancy. The Centres for Disease Control & Prevention, USA recently recommended a policy that requires evidence of varicella immunity for children entering high school (varicella vaccine is included in universal childhood immunisation schedules).<sup>7</sup> This approach has some merit,

particularly as there is the potential to validate self-reports with parental reports and reduce the need for serological testing. Young NT women not attending high school or child bearing at younger ages would require an alternative approach.

### Acknowledgements

The authors wish to thank: Dr Mahomed Patel at the National Centre for Epidemiology & Population Health; Dr Vicki Krause and Ms Sue Reid CDC, Darwin; Dr Gary Lum, Mrs Robyn Berghoff and staff at RDH; Assoc Professor Margaret Burgess at the National Centre for Immunisation Research and Vaccine Preventable Diseases; Ms Rosslyne Escott at the Centre for Infectious Diseases and Microbiology, Westmead; and the NT Interpreter and Translator Service, for their invaluable contribution to this project. This study was partially funded by a grant from SmithKline Beecham Pharmaceuticals.

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## Varicella vaccine workshop, Melbourne, 8 December 1999

*Christine Selvey, CDC, Darwin*

In December, I attended a national workshop aimed to inform about and discuss issues related to the use of varicella vaccine in Australia.

Some of the main points of discussion included:

- The crude annual death rate due to varicella in Australia is 0.03/100,000 which equates to an average of 5 deaths a year.
- The first varicella vaccine was developed in Japan over 20 years ago. The live attenuated vaccine is recommended in the United States as a single dose vaccine given at 12 months. Adults require two doses for adequate protection.
- While the vaccine can rarely cause mild zoster disease, the available evidence suggests that the vaccine protects against zoster. About 15% of the population will develop zoster infection following natural disease during their lifetime, some of whom who will have persistent severe pain.
- About 10% of vaccinees will develop breakthrough chickenpox but this is always mild disease, eg 5-10 lesions. A higher dose of vaccine increases efficacy.
- The development of the measles, mumps, rubella and varicella combination vaccine is ongoing.

Initial studies with this combination have shown reduced immunogenicity to the varicella component compared to the straight varicella vaccine.

- Concern was expressed at the possibility that low coverage rates of varicella vaccination (eg 50%) may increase the average age of onset of chickenpox and result in more cases in adults (as has occurred with measles). This may occur if the vaccine is readily available privately but is not on the routine schedule and is not funded.
- Varicella vaccine is not a cost effective vaccine if only health care costs are considered. However, if indirect costs are included, such as loss of production due to time taken off work to care for ill children, then the vaccine becomes highly cost effective.
- When available in Australia the vaccine will be recommended for health care and childcare workers who are not immune.

Anyone interested in receiving a copy of the proceeding should contact me on 8922 8825.

## Hospital separations in the Northern Territory for varicella-zoster virus related illnesses, 1993-1997

Kerry-Ann O'Grady, formerly CDC Darwin and NCEPH, ANU, Canberra

### Introduction

A varicella-zoster virus (VZV) vaccine is available overseas, and universal immunisation in childhood is recommended in the United States.<sup>1</sup> Any decision to introduce the vaccine to Australia must be based on an assessment of potential benefits and harms. While there has been some assessment of VZV significance in populations in southern Australia,<sup>2</sup> the impact on the NT population is not known. It is not a notifiable condition and information on morbidity and mortality is limited to a few data collections. These are hospital separation data, deaths registers, and in 1995 the inclusion of VZV congenital and neonatal complications in the Australian Paediatric Surveillance System. Hospital separation data were analysed to assess the importance of VZV as a cause of severe morbidity and mortality in the NT population.

### Methods

Hospital discharge diagnoses with ICD9 codes 052.0 to 053.9 inclusive were extracted from the Territory

Health Services client information database, Caresys, for the period 1993-1997. Data were entered into an EpiInfo V6.04 database for analysis. Crude population rates and crude death rates were calculated using the mid-period estimated NT and Australian resident populations. The Top End refers to the Darwin, Katherine and East Arnhem health regions and the Centre refers to the Alice Springs and Barkly health regions.

### Results

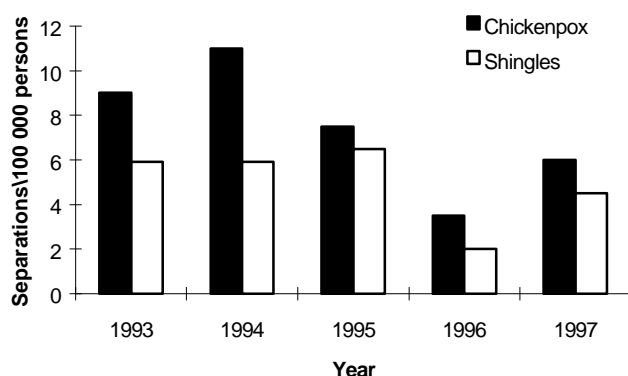
Over the five year period, there were 191 inpatients in NT hospitals with primary (PD) or secondary diagnoses (SD) of VZV related illnesses. Of these, 111 were for chickenpox (varicella) (58% PD) and 80 were for shingles (herpes zoster) (53% PD). The crude admission rate for the NT population for chickenpox (PD) was 7.3/100,000/year and 4.8/100,000/year for shingles (PD). Chickenpox or shingles without mention of complication were the most common ICD9 codes recorded (Table 1). There was a decline in admission rates for both diseases from 1993 to 1997 (Figure 1).

**Table 1** Frequency of VZV ICD9 codes in NT hospitals as primary or secondary diagnoses, 1993 - 1997

ICD9	Description (as per ICD9 Book)	Primary	Secondary	Total
0520	Post varicella encephalitis	1	0	1
0521	Varicella (haemorrhagic) pneumonitis	5	0	5
0527	CP with other unspecified complications	8	4	12
0529	Varicella without mention of complication	50	43	93
0530	Herpes zoster with meningitis	1	0	1
05312	Postherpetic trigeminal neuralgia	0	1	1
05319	HZ with other CNS complication	2	6	8
05320	HZ dermatitis of eyelid	4	0	4
05321	HZ keratoconjunctivitis	4	2	6
05329	HZ with other ophthalmic complication	10	4	14
05371	Otitis externa due to HZ	1	0	1
05379	HZ with other unspecified complication	2	2	4
0539	HZ without mention of complication	18	23	41
<b>Total</b>		<b>106</b>	<b>85</b>	<b>191</b>

Chickenpox (CP) = Varicella      Shingles = Herpes zoster (HZ)

**Figure 1 Annual NT hospital separations (per 100 000 persons) with a primary diagnosis of chickenpox or shingles, 1993-1997**



Rates in the indigenous population were two times higher for chickenpox and three times higher for shingles than the non-indigenous population. Admission rates for shingles in the 25-55 year olds were up to 13 times higher in the indigenous population (Table 2).

**Table 2 Mean annual age-specific hospital separation rates (per 100 000 persons), in the NT 1993-1997 by indigenous status**

	Chickenpox (per 100 000)		Shingles (per 100 000)	
	A	O	A	O
0-4	54	27	3	0
5-9	6	10	6	10
10-14	7	2	4	0
15-19	4	8	0	0
20-24	0	2	0	0
25-29	9	8	9	2
30-34	0	3	16	0
35-39	7	0	14	0
40-44	9	9	18	0
45-49	0	0	52	4
50-54	0	0	47	0
55-59	0	0	0	5
60-64	0	0	0	0
65-69	81	0	81	22
70-74	0	0	0	93
75+	0	0	0	69
Overall	12	6	10	3

A - Aboriginal : O - Other

Indigenous inpatients were younger than non-indigenous inpatients for both chickenpox (median 2 vs 7 years;  $p = 0.006$ ) and shingles (median 42 vs 64 years;  $p < 0.001$ ). Inpatients with a primary diagnosis of chickenpox were predominantly male (male:female ratio 2:1), as were those with a secondary diagnosis of shingles (male:female ratio 2.4:1).

The median length of stay for inpatients with a primary diagnosis of chickenpox was three days (Range 1-10) and 5 days (Range 1-175) for shingles. There was a total of eight deaths; two with a primary diagnosis of chickenpox (CDR 0.23/100,000/year) and one with a primary diagnosis of shingles (CDR 0.11/100,000/year). The details are listed in Table 3.

## Discussion

This analysis indicates that VZV does not cause a large number of hospital admissions or deaths in the NT. For the period 1993-1997, there was a yearly average of 22 inpatients in NT hospitals with chickenpox and 16 with shingles. Three people died, and a further five died who had illnesses complicated by VZV. The average age of these eight was 59 years (median 65 years). Inpatients with chickenpox were predominantly children and those with shingles were predominantly middle age to elderly adults. There was a decline in admissions over the study period but analysis of five years of data is inadequate for determining whether the decline was new, or simply related to the timing of epidemics.

Relative to data from elsewhere in Australia,<sup>2</sup> crude rates for the NT were higher for chickenpox and lower for shingles. In South Australia and New South Wales between 1988 and 1993, the average annual hospital separations were 72 (5/100,000/year) and 241 (4.1/100,000/year) respectively for primary chickenpox. For shingles they were, 208 (14.5/100,000/year) and 640 (10.9/100,000/year).<sup>2</sup> These differences, particularly with shingles, may reflect the younger age distribution of the NT population compared to the Australian population. There are however, biases inherent in comparing hospitalisation rates calculated on a population basis as they do not account for confounders such as variations in the incidence of disease in the community, access to services, differing age distributions, and variations in medical practice or the health of the population.

**Table 3 Deaths in NT hospitals with VZV related ICD9 codes, 1993 - 1997**

Year	Age	Sex	Indigenous	ICD9 Diagnosis	Primary Diagnosis
1993	65	M	Other	05319	No (shingles related)
1994	51	M	ATSI	0539	No (shingles related)
<b>1994</b>	<b>81</b>	<b>F</b>	<b>Other</b>	<b>0539</b>	<b>Yes (shingles related)</b>
<b>1995</b>	<b>65</b>	<b>M</b>	<b>ATSI</b>	<b>0521</b>	<b>Yes (chickenpox related)</b>
1995	65	M	ATSI	0527	No (chickenpox related)
<b>1996</b>	<b>5</b>	<b>M</b>	<b>ATSI</b>	<b>0529</b>	<b>Yes (chickenpox related)</b>
1996	67	F	ATSI	0539	No (shingles related)
1996	72	M	Other	0539	No (shingles related)

While in absolute terms the number of deaths was small, the crude death rate for chickenpox in the NT was approximately eight times higher than that reported for the Australian population (0.03/100,000/year) and 1.5 times higher for shingles (Australia: 0.07/100,000/year).<sup>2</sup> This possibly reflects the poorer health status of indigenous persons in the NT who constituted the majority of inpatients with VZV who died. The data do not however provide an indication of the case-fatality rate, as the incidence of the disease in the NT is not known. Elsewhere it has been estimated to approximately two per 100,000 cases in otherwise healthy children,<sup>1</sup> however increases with age to approximately 25 per 100,000 among persons over 30 years.<sup>3</sup>

The higher admission rates for indigenous persons are consistent with their excess hospitalisations for many other health problems in the NT. However, the numbers remain small indicating it is not a significant cause of severe morbidity or mortality. This is surprising given chickenpox can be complicated by Group A  $\beta$ -haemolytic streptococci (GAS) bacteraemia,<sup>4,5</sup> and a high prevalence of GAS in skin sores and throat infections has been reported in some Aboriginal communities.<sup>6,7</sup> Complicated chickenpox may have been expected more frequently. Standard treatment protocols for skin and throat infections in Aboriginal communities may contribute to preventing such complications.

Indigenous inpatients hospitalised with VZV were younger for both chickenpox and shingles, possibly reflecting the different age distributions of the two populations. Alternatively, with regards to chickenpox, it could indicate a younger age of infection in the indigenous population due to social or cultural factors

such as household densities and caring patterns. For shingles, the younger age may reflect a greater burden of risk factors such as stress, poor health, and chronic diseases.

There are obvious limitations with hospital separation data. Coding errors or inconsistencies may result in either an over or under estimate of actual admissions. This is reflected in two of the deaths that were coded as either chickenpox or shingles without mention of complication. Similarly, they do not reflect the incidence of disease in the community, nor the factors that influence whether a person with either disease is admitted for care. That the majority of admissions were coded as the primary disease without complication indicates factors other than the severity of disease are important.

Hospital separation data suggest VZV is not a significant contributor to mortality or excess hospitalisations in the NT. It is likely therefore that if a vaccine is introduced, the decision will be made on factors other than the cost of hospitalisation or mortality rates. Such factors may include morbidity and job absenteeism for parents and patients. This analysis may be of use to that decision-making process, however community based surveillance will be necessary to more accurately assess potential benefits and harms, and for monitoring the impact of a vaccine if it is introduced.

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## Varicella-zoster virus in pregnant women and babies

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### Introduction

Chickenpox or varicella caused by varicella-zoster virus (VZV) is endemic in the population at large and becomes epidemic among susceptible individuals during seasonal periods eg late winter/early spring. It is thought to be more common in temperate climates.

During outbreaks of chickenpox, nonimmune pregnant women will inevitably be exposed to the virus and this is therefore a common obstetric problem. Chickenpox is generally a benign infection for both the mother and her unborn baby. For a small number however, it has the potential to cause great harm. The mother may develop overwhelming and potentially fatal systemic illness while the foetus carries the risk of congenital varicella syndrome (CVS), stillbirth, prematurity, or perinatal chickenpox. The best possible outcome is immunity to chickenpox with a small risk of postnatal "shingles". The combination of postnatal exposure and no transplacental immunity carries the risk of neonatal chickenpox which may be severe.

CVS is a devastating condition with severe neurological, ophthalmological and skeletal manifestations. The risk is greatest following infection in the first trimester and careful counselling of the pregnant woman is required. Some women will elect to terminate the pregnancy following confirmation of a first trimester chickenpox infection.

Varicella Zoster Immune Globulin (VZIG) has radically improved the outcome for perinatal chickenpox and most susceptible exposed babies who receive VZIG will have mild disease. Prior to VZIG the fatality rate from neonatal chickenpox was up to 30%. The outcome and management of fetal or neonatal chickenpox exposure is clearly dependent on maternal immune status and this must be determined as soon as possible. Delay has consequences: VZIG is less effective, the baby may deliver too early for the beneficial effects of maternal antibody transfer, or the pregnancy may proceed to a point where the option of termination is not appropriate.

It is important that those counselling families in this situation are well aware of the risks and the appropriate management strategies in each case. For this reason the following fact sheet has been compiled. It covers all scenarios likely to be encountered both out of and within the hospital setting.

## FACT SHEET - Varicella-zoster virus in pregnant women and babies

### General information

- This is the virus that causes chickenpox which can reactivate later in life in a small percentage of people in the form of shingles.
- Incubation period: 10-21 days; up to 35 days after Varicella-Zoster Immune Globulin (VZIG).
- Contagious from 48 hours before rash until rash crusted over.
- Viraemia occurs 48 hours prior to rash; virus dormant in lymphoid tissue of nose and throat prior to that.
- Spread is by direct contact (vesicular fluid/respiratory secretions).
- If close household contact 90% develop chickenpox.
- Indoor contact greater than 1 hour risk of chickenpox is 20%.
- Chickenpox can occur after contact with patients with either chickenpox or shingles.
- VZIG reduces severity but does not prevent chickenpox.
- VZIG is most effective if given immediately after exposure, it remains partly effective up to 4 days and is of no use once symptoms appear.
- Varicella-zoster vaccine provides protective efficacy of greater than 90% within 3 days of exposure, but is a live attenuated virus and therefore should be avoided in pregnancy.

### Chickenpox in pregnancy

- Pregnant women who have had chicken pox in the past will not become reinfected.
- 5-10% pregnant women are non-immune.
- Attack rate of chickenpox in pregnancy is 5-7/10 000 pregnancies.
- Primary maternal chickenpox in pregnancy can be severe (thought to be because of depressed cell mediated immunity) but is usually benign. Pneumonitis can be particularly severe (up to 30% mortality).
- Possibly small increased risk of miscarriage.

### Risk to baby

- **Asymptomatic infection.** Self limited intrauterine infection occurs in 25% (is an *in utero* infection

without adverse effect; baby IgM positive but clinically unaffected).

- **Congenital varicella syndrome (CVS).** The 1st trimester is the period of greatest danger for CVS. Early small studies reported rates of up to 10%,<sup>1</sup> however these have not been substantiated by subsequent larger studies, all of whom had rates of 2% or lower.<sup>2,3</sup>
- The risk of CVS after the second or third trimester appears to be much less.
- The risk of CVS after shingles is also much less.
- **Perinatal chickenpox.** Maternal rash in the last 5 days to the first 2 days postpartum is the period of greatest danger for the baby.
- Fetal infection occurs with the initial viraemia, prior to the maternal rash.
- 1-2% of children exposed to chickenpox in utero may develop shingles later especially if exposed in the second and third trimesters.

### Management of the potentially exposed pregnant woman

1. Determine if a significant exposure has occurred. Significant exposure of a susceptible woman includes:
  - Household contact (family members).
  - Prolonged exposure (>1 hour).
  - Hospital contact - same room - prolonged face to face contact with an infectious person.
2. Determine immune status:
  - Immune status is determined by an IgG assay for VZV.
  - Results may take up to 36 hours to become available. This should be discussed with the laboratory on a case by case basis. Rapid results may be possible, particularly if there is serum already in the laboratory.
  - Women who know they have had chickenpox in their life or who have had a positive VZV assay in the past are immune and no further testing is required.
3. Administer VZIG in the following situations:
  - i. Where significant chickenpox exposure has occurred in a seronegative woman where the

time from exposure to VZIG administration would be less than 96 hours.

- ii. Where significant chickenpox exposure has occurred in a woman whose immune status is unknown and unlikely to be known within 96 hours of exposure.
- iii. Where significant chickenpox exposure has occurred in a woman with compromised immunity (regardless of her VZV status).

The dosage of VZIG for women greater than 50 kg is 600 IU (= 3 vials). The blood bank usually holds 10 vials in stock and are able to obtain further supplies within 24 hours. RDH usually hold a further six.

### Management of pregnant women with chickenpox

- Discuss maternal and fetal risks.
- Only give VZIG to immunocompromised women after consultation with an infectious disease physician.
- Monitor for progress to the severe systemic form of the illness.
- Consider acyclovir.
  - Uncertain if acyclovir reduces the incidence of CVS/fetal infection.
  - Acyclovir is considered safe in pregnancy (it has not been associated with any congenital anomaly).
  - Oral acyclovir is preferred - IV for severe cases (eg pneumonia).
  - Vigilance for preterm labour.

### PAEDIATRIC OUTCOMES

#### Congenital Varicella Syndrome (CVS)

##### Manifestations

- Chorioretinitis ± cataracts / microphthalmia / Horner's syndrome.
- Cicatricial skin (scars) in dermatomal distribution
- Hypoplasia of limbs/paralysis and atrophy of limbs/absence of digits.
- Growth deficiency and microcephaly.
- CNS calcification.
- Intellectual disability/seizures/cortical atrophy.
- Other (GI and genitourinary tract abnormalities).

### Pathophysiology

- Initial *in utero* infection results in systemic/CNS effects.
- *In utero* reactivation results in dermatomal lesions.
- 50% are premature.

### Diagnosis

- Clinical.
- Microbiological.
  - Evidence of maternal infection (VZ specific IgM, 4 fold increase in IgG).
  - Baby IgM or persistent IgG.
- Sensitivity of specific IgM as a marker of fetal infection is low; IgG better; (only 25% infants with proven infection are IgM positive).
- There may be a few residual skin lesions at birth
- Occasionally shingles develops in infancy.
- No need to isolate babies with CVS (not infectious).

### Perinatal chickenpox

- Infants whose mothers developed chickenpox 3 weeks before delivery may develop chickenpox.
- *The severity of baby's illness depends upon the timing of the mother's illness in relation to the delivery.*
- The infant is protected by the transplacental maternal antibody, only if onset of the mother's rash occurred more than 5 days before delivery.
- Incubation period (mother to baby disease) is 9–15 days, though may be less.
- If the infant has perinatal chickenpox, the rash in the baby will occur within 10 days of birth.
- The attack rate of the infant developing chickenpox in first 10 days is 25–50%.
- If *mother's rash* appears between 5 days prior to and 2 days after delivery the baby gets a large inoculum of virus but *no transplacental antibody* (IgG).
- These babies are born well. Symptoms usually include fever and haemorrhagic rash. Visceral involvement and pneumonia may also occur. The baby may become severely ill at 5–10 days. Mortality can be up to 30%.

### Management

- Delay labour if mother's rash occurs around her due date; the longer the infant remains *in utero* the more likely transplacental antibody transfer.
- Give VZIG to babies of mothers who have chickenpox rash from 5 days prior to and 2 days after delivery.
- VZIG ameliorates severity of disease but does not prevent or reduce incidence of infection (possibly because such intense exposure).
- Babies who are given VZIG and who develop chickenpox virtually all have mild disease. Hospitalisation and acyclovir are unnecessary unless baby develops high fever, cough, respiratory distress syndrome or extensive cutaneous lesions.
- Severe chickenpox (high fever, cough, respiratory distress syndrome or extensive cutaneous lesions) only very occasionally develop after VZIG.
- If signs of severe disease, hospitalise and give acyclovir 10–15 mg/kg tds (lower dose in renal failure).
- Babies who develop skin lesions at or shortly after birth do not require VZIG. The antibody has already crossed; the illness will be mild.
- *It is not necessary to isolate baby from mother if she has the chickenpox rash. By the time she has rash the baby has already been exposed to infection and the additional exposure risk is small. Secondly it is disadvantageous from a psychological and emotional perspective.*
- The baby should get VZIG irrespective of mother having had VZIG for exposure.

### Diagnosis

- Scrape cells from bottom of lesions  
Direct fluorescent antibody  
PCR
- Vesicle fluid  
Virus culture
- Serology  
VZ IgM  
Persistent IgG

### Postnatal chickenpox

- This occurs in babies who are exposed to chickenpox after birth.

- Non-immune babies may be at slight increased risk of severe disease.
- Maternal antibody does not always completely protect against infection.
- Unlikely to be severe disease in babies after 2 weeks of age, irrespective of mother's status.
- Generally does not require acyclovir.
- If the mother has contact with chickenpox before delivery but doesn't develop the rash until several days after delivery, the baby would not have received either transplacental virus or antibody. The baby is therefore at risk from postnatal contact and should receive VZIG.
- If a sibling of a new born baby gets chickenpox check mother's immune status (history or serology if necessary); if mother is immune do nothing; if mother is non-immune give VZIG to baby.

### INFECTION CONTROL ISSUES OF CHICKENPOX IN HOSPITAL

*If non-immune patient or staff member develops chickenpox:*

1. Inform the staff at Infection Control.
2. Isolate the index case for 5 days after onset of rash and for duration of vesicular lesions.
3. Neonates
  - Transmission remains unusual but infants should be divided into "exposed" and "nonexposed" groups.
  - Discharge all infants as early as possible.
  - Give VZIG to those exposed infants with seronegative mothers.
  - Give VZIG to exposed preterm babies less than 28 weeks gestation or <1000 g regardless of maternal history.
  - In neonatal intensive care units (NICUs) VZIG is administered to all neonates regardless of maternal history and gestation as many will be long term patients and even mildly infected infants will be infectious to others.
  - All infants should receive acyclovir even if they have VZIG if breakthrough infection develops.
  - Non exposed infants not in NICU do not need any specific treatment.
  - Neonates of mothers with rash need isolating for 21 days (or 28 days if they have received VZIG).
4. Other patients and staff members

- All exposed patients and staff members who do not recall a history of chickenpox should have VZ antibodies checked.
  - Exposed seronegative inpatients should be isolated from day 10 to day 21 days after exposure (ie index case rash) or day 28 days if had VZIG.
  - Exposed seronegative staff should be sent to an area where there are no immunocompromised patients from day 10 to day 28 after exposure.
5. Shingles
- Patients with shingles require secretion precautions unless all lesions crusted
  - Immunocompromised patients or patients with disseminated shingles should be isolated for duration of illness.
6. Isolation for chickenpox = "Airborne Precautions" (green card)

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## ***Cryptosporidium parvum*: An associated outbreak of diarrhoeal disease in Nhulunbuy, East Arnhem**

*Hartley Dentith and Ivor Alexander CDC, Nhulunbuy*

*Cryptosporidium parvum* is a spore-forming parasite similar to other enteric coccidia commonly found in wild and domestic animals. All infections are transmitted by contamination of the environment with resistant oocysts excreted by infected hosts. The parasite infects a wide range of vertebrate hosts and is associated with clinical disease primarily involving watery diarrhoea in mammals, diarrhoea and respiratory signs in birds and gastritis in reptiles and possibly fish.<sup>1</sup>

In humans, cryptosporidiosis is characterised by watery profuse diarrhoea, abdominal cramping, fatigue, myalgia and fever. In children, vomiting and low grade fever are common. Symptoms typically begin 6-7 days after exposure, (range 1-14 days). At the present time

there are no antimicrobials available for the treatment of cryptosporidiosis.<sup>2</sup> The parasite has been implicated in outbreaks associated with contaminated water. Because *Cryptosporidium* is highly chlorine resistant, inadequately filtered drinking water and swimming pools may be vehicles for infection.<sup>2</sup>

Notifications for cryptosporidiosis increased in East Arnhem District in October 1999. In June and July there were isolated cases in rural communities, two each at Milingimbi and Angurugu. During early October isolated single cases occurred at Gapuwiyak, Galiwinku, Yirrkala, Ramingining and Nhulunbuy. The first indication of an impending outbreak occurred during the week of 25 October when two otherwise healthy adult males were hospitalised for

rehydration. On examination their stools contained *Cryptosporidium* oocysts. One of these patients had been in contact with two adult males who had recently visited South East Asia, and who, on return to Australia, suffered severe gastrointestinal signs and symptoms. These two men were not able to be interviewed.

During the week of the 25 October, 18 people had contacted local health services complaining of gastroenteritis symptoms and eight stool specimens proved positive for *Cryptosporidium*. Up until 24 November when the outbreak abated a further 41 cases reported symptoms and were interviewed.

A total of 58 cases were investigated by interview of which 22 provided stool samples; 14 were positive for *Cryptosporidium* oocysts.

Of 34 adults, 26 or 72% were male. Of the children ten were under five years of age of whom two were hospitalised. In addition to the people interviewed CDC staff became aware that approximately 50 students at Nhulunbuy Primary School were absent with gastrointestinal symptoms during a two week period in early November.

Further anecdotal reports indicated a substantial number of people were symptomatic and absent from work. We estimate approximately 150 people were affected by gastrointestinal symptoms of varying degrees during this outbreak.

### Public health measures

Attempts to identify a source of the outbreak resulted in no scientifically based conclusions. Several lines of investigation were undertaken.

1. Foodborne - Environmental Health Officers (EHO's) visited all food outlets. Staff who provided stool specimens all tested negative. No consistent connections were made with the local food outlets, nor was there a pattern related to age, location or workplace. One suspicion concerned a fundraising event held involving about 500 people. This was catered for using finger food which was plated and distributed to each person. There is no evidence to implicate a food handler or the fund raising event in the outbreak.
2. Waterborne - The town water supply was investigated. Routine water sampling of the community supply was also conducted. The supply is from deep bores of about 80 metres. All 17 of the bores are secured and protected from surface contamination. The aerator has been inspected by the EHO and no evidence of contamination has been identified or sighted. The storage/header tanks on Mt Saunders are fenced and manholes, covered and locked.
3. Swimming pool - There was no scientific or epidemiological evidence to suggest the town swimming pool was implicated in the outbreak. However, the town board took the initiative and replenished the pool water in view of the fact that a large swimming carnival was being conducted in the near future. Bacterial testing of the pool water is routinely performed on a monthly basis. Hygiene notices were prominently displayed at the entrance to the pool.
4. Child Care facilities - All child care facilities were visited by EHO's and CDC staff and directors and staff were thoroughly briefed on routes of transmission for *Cryptosporidium* and the importance of the hygiene was emphasised. Copies of an information sheet (see next page) were distributed to all child care staff. Child care staff have been diligent by immediately excluding any child who has been sick.
5. Schools - The three schools in town were visited by EHO's and CDC staff and all available teaching and administration staff were briefed on the outbreak and given copies of the information sheet. The importance of handwashing was emphasised and the message to staff was to reinforce this message to students. The message was given again at the school assemblies and for the following five weeks the message was included in the school's weekly newsletter. A briefing session was conducted with the school nurse who was supplied with an initial batch of "poo pots".
6. A hygiene press release was prepared and approved and distributed to all children at the schools, the local community radio station (8EAR), and the local newspaper (Arafura Times).

The possibility that the *Cryptosporidium parvum* identified in this outbreak of diarrhoeal disease was introduced from South East Asia by returning holiday makers cannot be discounted. It was apparent that

human to human transmission was occurring in the community especially in the younger age groups.

### Thoughts

- The possible introduction of the Food Safe auditing system may help reduce the possibility of food borne outbreaks.
- Travel agents could assist by providing advice to overseas travellers, that if they are unwell on return home to seek medical advice.
- CDC staff and EHO's could provide hygiene education sessions/handouts to schools especially prior to sporting carnivals or school excursions.
- Regular contact by at least one CDC staff member liaising with the school nurse could be maintained with all schools in the area to promote the "healthy schools" concept, and include child care centres in the itinerary.
- Notices could be placed at fresh water swimming holes/creeks warning of possible contamination from animals.

### INFORMATION SHEET

- \* *No one who has diarrhoea and/or vomiting should be swimming in the public swimming pool or sharing home pools.*
- \* *Thorough hand washing with soap and water after going to the toilet, before preparing food and after changing nappies.*

- \* Do not allow children to sit on tables where food is prepared or eaten.
- \* Do not change nappies on tables or counters used for preparing or serving food.
- \* Do not share unwashed eating or drinking utensils.
- \* Children and young infants with either diarrhoea and or vomiting MUST be excluded from child care centres or school and also have prompt medical consultation should they become affected.

The above precautions should help in the prevention of spread of disease and your cooperation and assistance in this matter is greatly appreciated.

*Community members who have suffered from symptoms of gastroenteritis are requested to contact the Centre for Disease Control.*

### Acknowledgment

CDC staff thank Phil Donohoe (EHO, Nhulunbuy) for his critical input.

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### Another reason not to eat your oysters raw

*Source: Emerging infectious diseases: Press Release 30 August 1999  
Centers for Disease Control and Prevention, USA*

According to an article in an issue of Emerging Infectious Diseases, *Cryptosporidium* can be added to the list of reasons not to eat raw oysters.

Oysters feed by filtering water through their gills. When water is contaminated by run-off from pastures or sewage, oysters can keep the parasite in their gills and spread illness. Researchers tested oysters from seven sites used for commercial harvesting in the Chesapeake Bay area. Oysters from all the sites contained *Cryptosporidium* species both from cows and humans. This finding shows that

the water at these sites contained human and animal faeces during a period when the oysters were filtering. The risk of contamination is probably higher after a heavy rain, but some risk is present year-round.

Infection with *Cryptosporidium* can cause prolonged diarrhoea; it is especially dangerous for persons with weak immune systems. The good news is that heating to temperatures above 72°C kills the parasite, so the authors urge that oysters be cooked before they're eaten.

The full article can be accessed at <http://www.cdc.gov/ncidod/eid/vol5no5/fayer.htm>.

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## Incidence of interval breast cancers following round one mammography screening (December 1994-1997)

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### Abstract

**Objective:** To report on the incidence of interval cancer in first round mammography screening by NT BreastScreen.

**Design:** A retrospective analysis of NT BreastScreen data crossmatched with the NT Cancer Registry to identify interval cancers occurring in the 12 and 24 months following a negative mammography screen.

**Participants:** Women 40 years of age and over, who attended for their first round of mammography screening between December 1994 and December 1997.

**Results:** The incidence of interval cancer was 6.8 per 10,000 women-years for the 12 month interval (95% CI 1.4 - 19.9), and 10.5 per 10,000 women-years in the 24 month interval (95% CI 3.4 - 24).

**Conclusion:** The 12 month interval cancer rate of 6.8 per 10,000 women-years is slightly higher than the national accreditation minimum standard of less than 6 cancers per 10,000 women screened. However, as the study population was small, this rate may be quite unstable, and care needs to be taken when comparing this rate with larger programs.

### Introduction

The aim of mammography screening is to reduce mortality from breast cancer. NT BreastScreen commenced population-based breast screening for women over 40 years of age in December 1994. Up to December 1997, 6870 women have been screened, and 31 cases of breast cancer detected. This represents a detection rate of 4.5 per 1,000 women screened. NT BreastScreen conduct routine mammography screenings every 2 years, except for women at higher risk who may be screened annually.

The objective for reporting interval cancer rates is to measure the effectiveness of population-based breast screening on breast cancer mortality, and to allow comparisons between programs nationally and internationally. The interval cancer rate is one of the

performance indicators used by NT BreastScreen and the national BreastScreen program for evaluation and accreditation of their program.

**Definition:** An interval cancer is defined as an invasive breast cancer that is diagnosed in the period following a negative mammography screening, and before the next scheduled breast screening. This includes women who have returned for an early review or re-screen and may be subsequently diagnosed with breast cancer.

Information from the NT Cancer Registry was used to identify interval cancer cases. The NT Cancer Registry records all new cancer cases that are notifiable in the NT. Information is provided by NT based pathology laboratories, hospitals, and the NT Registrar of Births, Deaths and Marriages, with additional information provided by GPs. Interstate-based cancer registries also notify the NT Cancer Registry of any NT residents who are diagnosed with cancer interstate. The NT Cancer Registry at the time of the study was complete to the 31 December 1997.

### Objective

To determine the incidence of interval cancers in the first round of mammography screening by NT BreastScreen between December 1994 and December 1997.

### Study Population

The study population was all the women who attended for a round one mammography screening between December 1994 and December 1997.

### Methodology

NT BreastScreen records were cross-matched with NT Cancer Registry records. Matching of names was done electronically using SAS and SPSS by names and date of birth. Exact and possible matches were reviewed manually and confirmed by date of birth and address. Information of women appearing on both datasets were merged using SPSS.

Two datasets of the sample populations were created, excluding women who had cancer detected by screening, or had a previous personal history of breast cancer.

1. All women who had a round one screening before 31 December 1996 (12 month interval).
2. All women who had a round one screening before 31 December 1995 (24 month interval).

#### *Determining the number of interval cancers*

For a case to be identified as an interval cancer, it had to have the following characteristics:

- The case was recorded on the NT Cancer Register with breast cancer, or become known to NT BreastScreen as an interval cancer.
- The cancer diagnosis date recorded on the NT Cancer Register was within 12 months or 24 months from the round one screening date.
- The round one mammography screening had a negative result. This was ascertained by a woman having *not* been discharged from NT BreastScreen with a 'mammography detected cancer' within the study period.

#### *Calculating the denominator*

The denominator includes the woman-years of eligible women who had a round one screening and were at risk of an interval cancer in the relevant period (12 or 24 months). The following categories contributed the following to the denominator:

##### Category 1:

Women who had been found to have an interval cancer during the study period were at risk for the amount of time from the round one appointment date, to the date of diagnosis recorded on the NT Cancer Register.

##### Category 2:

Women who had been discharged from NT BreastScreen because they had moved interstate, died, or withdrew voluntarily from the service (and therefore it was unknown if they still resided in the NT), *and did not have cancer diagnosed in the relevant period* were at risk from their round one screening date to their discharge date.

##### Category 3:

All other women who were still active on the NT BreastScreen records contributed the full time (12 or 24 months) of the relevant interval, as they were at risk of having an interval cancer until their next screening was due.

#### *Calculating the interval cancer rate*

The interval cancer rate is the:

$$\frac{\text{Number of interval breast cancers} \times 10,000}{\text{Number of women-years at risk}}$$

The interval cancer rate is expressed per 10,000 women-years.

#### **Results**

The NT BreastScreen dataset had 6780 records from 1 December 1994 to 31 December 1997. The NT Cancer Register had 146 women diagnosed with breast cancer between December 1994 and December 1997. There were 42 women who were on both datasets. The 12 month interval dataset had 4418 records, and the 24 month interval had 2396 records.

**Table 1 Summary of person-time for each category of women at risk of an interval cancer following round one screening**

Category	12 months		24 months	
	Number of women	Person-time (yrs) at risk	Number of women	Person-time (yrs) at risk
Cat 1 = Interval cancer cases	3	2.7	5	5.8
Cat 2 = discharged within study interval	16	2.5	22	40.7
Cat 3 = women contributing full person-time at risk	4399	4399	2369	4738
<b>TOTAL</b>	<b>4418</b>	<b>4404</b>	<b>2396</b>	<b>4784.5</b>

$$\begin{aligned} \text{12 month interval} & & & 3 \times 10,000 \\ \text{cancer rate} & = & \frac{\text{-----}}{4404 \text{ women- yrs}} \\ & = & 6.8 \text{ per } 10,000 \text{ women- yrs (95\% CI } 1.4 - 19.9) \end{aligned}$$

$$\begin{aligned} \text{24 month interval} & & & 5 \times 10,000 \\ \text{cancer rate} & = & \frac{\text{-----}}{4784.5 \text{ women- yrs}} \\ & = & 10.5 \text{ per } 10,000 \text{ women- yrs (95\% CI } 3.4 - 24.4) \end{aligned}$$

### **12 Month Interval**

In the 12 months following the round one screening, 3 interval cancers were identified. The women at risk contributed 4404 women-years at risk to the denominator, resulting in an interval cancer rate of 6.8 per 10,000 women-years (95% CI 1.4 - 19.9) (Table 1).

### **24 Month Interval**

In the 24 months following the round one screening, 5 interval cancers were identified. The women at risk contributed 4784.5 women-years to the denominator, resulting in an interval cancer rate of 10.5 per 10,000 women-years (95% CI 3.4 - 24.4) (Table 1).

### **Discussion**

The interval cancer rates were calculated using NT BreastScreen data from December 1994 to December 1997 to enable matching with the NT Cancer Register. Due to the small numbers of women in the program, interval rates were not calculated for individual years, or age groups. The NT BreastScreen interval cancer rates were found to be 6.8 and 10.5 per 10,000 women-years for the 12 and 24 month intervals respectively (95% CI 1.4 - 19.9; 3.4 - 24.4). The 12 month interval rate is slightly higher than that reported by BreastScreen Victoria for women screened in 1994. BreastScreen Victoria reported an average one year rate of 5.85 per 10,000 asymptomatic women, and average two year rate of 11.91 per 10,000 asymptomatic women. Internationally, interval cancer rates have been

reported in the UK North Western Region between 3.7 and 7.4 per 10,000 women for round one screenings done between 1988 - 1992. In the Netherlands, the Nijmegen breast screen program reported the first round interval cancer rate of 8.5 per 10,000 women-years screened.

The NT 12 month interval cancer rate is higher than the national accreditation minimum standard of less than 6 cancers per 10,000 women screened in the 12 months following screening. As incidence rates have not been stratified by age groups care must be taken when comparing overall and average incidence rates in other states, due to the different composition of age groups between the NT and other States.

Factors that influence the interval cancer incidence rates are the accuracy of the numerator (interval cancer cases), and the denominator (person-time of those at risk). The identification of interval cancers is dependent on: the accuracy and completeness of information collected by the breast screening service; the completeness of the cancer registries; the timely registration of breast cancers to the cancer registries; the migration of women between states; and the methodology and types of software used for matching names.

It would be useful to include a field for the date that a client moved interstate to be entered, so the person-time of those at risk prior to leaving the NT could be calculated more accurately.

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## Pelvic inflammatory disease (PID): cessation of the PID treatment trial

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### Background

Pelvic inflammatory disease (PID) is an important cause of morbidity in women in the Top End of the Northern Territory (NT). In the three-year period 1991-1994 a total of 169 patients were admitted to Royal Darwin Hospital (RDH) with a diagnosis of sexually transmitted disease (STD)-related PID.<sup>1</sup> With the acknowledged problems of adherence to the standard PID treatment protocols, a study was commenced in May 1998, to investigate the value of an alternative azithromycin-based regimen.

Single dose azithromycin had been introduced in the NT in 1994 for the treatment of uncomplicated genital chlamydial infection. The advantages for compliance/adherence of this over the standard therapy of 10 days of doxycycline were considerable. The role of weekly azithromycin for the treatment of PID had not been determined in any studies, but there was pressure to change to azithromycin-based regimens in some local centres.

The study was an open-label, randomised clinical trial to compare a standard, antibiotic regimen, with an azithromycin-based regimen for the treatment of STD-related PID, for women admitted to hospital. Both arms received ceftriaxone 1.0g intravenously (iv) once daily for 3 days, plus metronidazole 500mg iv twice daily for 3 days. The standard arm then received doxycycline 100mg and metronidazole 400mg orally twice daily for 14 days, while the azithromycin arm received azithromycin 1.0g orally twice daily on day 1 and day 8, with no oral metronidazole. A total of 116 women would have been required to complete the study, but, it became apparent soon after commencement of the trial that the number of women eligible for enrolment was dramatically less than initially predicted.

### Results

1. To determine the reason for a decline in numbers, we reviewed all admissions to RDH which had been

ICD-9/10 coded as "PID" between 1 July 1997 and 30 June 1998, one year prior to the study. After correcting for miscoding, 19 cases of PID were identified, 12 with "chronic PID" (recurring problems with symptoms consistent with PID). An STD was identified in only 6/19 (Table 1).

**Table 1 Organisms identified in RDH admissions coded as "PID" 1/7/97 – 30/6/98**

Organism	Number
<i>Chlamydia trachomatis</i> alone	2
<i>Neisseria gonorrhoeae</i> alone	0
<i>N.gonorrhoeae</i> + <i>C.trachomatis</i>	2
<i>N.gonorrhoeae</i> + <i>Trichomonas vaginalis</i>	1
<i>T.vaginalis</i> alone	1
<b>Total</b>	<b>6</b>

2. Fourteen people were enrolled in the study between May 1998 to May 1999. In addition, a further 8 patients with a diagnosis of PID were identified at RDH in that time period, but were not enrolled. STD related organisms were isolated in 18/22. The microbiology is summarised in Table 2.

**Table 2 Organisms identified in patients eligible for enrolment in PID study**

Organism	Number (%)
<i>Chlamydia trachomatis</i> alone	0
<i>Neisseria gonorrhoeae</i> alone	13 (59%)
<i>N.gonorrhoeae</i> + <i>C.trachomatis</i>	1 (4.5%)
<i>T.vaginalis</i> alone	4 (18.2%)
No organism	4 (18.2%)
<b>Total</b>	<b>22 (100%)</b>

### Cases enrolled in the PID, azithromycin treatment trial (May 1998 to May 1999)

Case	PID diagnosis	Organism
1	Chronic* (post D&C)	<i>T.vaginalis</i> (PCR)
2	PID discounted, UTI	<i>Escherichia coli</i>
3	Acute	<i>N.gonorrhoeae</i> (PCR)
4	Chronic*	No organism isolated
5	Chronic	No organism isolated
6	Chronic*	No organism isolated
7	Acute, peritonitis	No organism isolated (PCR not done)
8	Chronic	<i>T.vaginalis</i> (PCR)
9	Chronic	<i>N.gonorrhoeae</i> (PCR)
10	Acute	<i>N.gonorrhoeae</i> (PCR + culture); <i>T.vaginalis</i> (culture)
12 <sup>+</sup>	Acute*	<i>N.gonorrhoeae</i> (PCR + culture)
13	Acute	<i>N.gonorrhoeae</i> (PCR)
14	Chronic*	No organism isolated
15	Acute*	<i>N.gonorrhoeae</i> (PCR + culture)

<sup>+</sup> Number 11 was a false start, enrolled but failed to meet the entrance criteria when reviewed

\* Rebound, to either clinic or RDH with persistent problems.

Despite significant efforts, no patients enrolled completed all follow-up visits (visits were scheduled to community health centre (CHC) of choice, on day 8, day 15 and at 3 months). Of the 14 on the trial, 6 rebounded to a CHC before the 3 month period with a similar complaint, of these 2 were on the standard treatment arm, and 4 on the azithromycin arm. A total of 4 were lost to follow-up, 2 from each regimen. Two were non-compliant, one from each regimen. Following discussions with the chief investigators the study was terminated on 15 May 1999. No conclusions about the role of azithromycin in PID can be drawn from these results.

### Discussion

We were able to identify 41 cases (~20/yr) of PID requiring hospitalisation in a period between June 1997 and May 1999, considerably less than the 56 per year rate reported earlier this decade.<sup>1</sup> The sources of data differed between the studies; the earlier RDH study used a database from the obstetrics and gynaecology department, which is no longer maintained. This was used by the researchers, as the RDH medical records were considered inaccurate and/or incomplete. The recent study used ICD-9/10 coding from medical records, which are more accurate since the introduction of hospital accreditation and quality-assurance within

the last 5 years. Despite the data having been obtained from different databases, the reduced rates may still reflect a true reduction in the rate of severe PID requiring hospitalisation. There are a number of possible explanations for this:

1. *Prevalence of chlamydia in the community has fallen.* This is supported by the notification data (Table 3) which shows a decrease in the number of new diagnosis of *C.trachomatis* in the Top End over the period 1997-1998.
2. *Patients with moderately severe PID are now being managed in the community.* There is anecdotal support for this contention (personal communication Margaret O'Brien).
3. *Suppression of symptomatic PID through partial treatment.* The current standard treatment for uncomplicated cervicitis is a stat dose of azithromycin 1g plus amoxicillin 3g, and probenecid 1g, +/- tinidazole. While 1g of azithromycin stat is 95% effective for uncomplicated cervicitis, its efficacy in PID is unknown. Unfortunately, we were unable to demonstrate the efficacy of azithromycin in higher multiple doses due to the cessation of this study. Partial treatment has serious implications as sub-acute infection with resultant tubal damage may occur.

**Table 3 Summary over 3 x 1 year periods of studies in the Darwin region**

Year	PID Cases	PID <i>C.trachomatis</i>	<i>C.trachomatis</i> Notifications	<i>C.trachomatis</i> Attack Rate /100 000	PID <i>N.gonorrhoeae</i>	<i>N.gonorrhoeae</i> Notifications	<i>N.gonorrhoeae</i> Attack Rate /100 000
1994	60	37% (3 yr ave)	360	431.4	23 % (3 yr ave)	190	227.7
1997-98	18	22%	275	303.1	16.7 %	302	332.9
1998-99	22	4.5% (1 patient had both)	289	309.7	27.3 %	378	405.1

$X^2 = 64.12$ ,  $P < 0.00001$  comparing PID cases with *C.trachomatis* to those with *N.gonorrhoeae*.

Attack Rates of *C. trachomatis* (for trend)  $X^2 = 18.18$   $p = 0.00002$  slope -ve

Attack Rates of *N. gonorrhoeae* (for trend)  $X^2 = 47.87$   $p < 0.00001$  slope +ve

### Lessons from this study

- PID diagnosis is notoriously difficult. The “gold standard” - laparoscopy (which itself may lack sensitivity) is not routinely available or acceptable. Clinical diagnosis suffers from lack of sensitivity and specificity.
- Follow-up of patients with PID in the NT is extremely difficult even in the context of a clinical trial with a dedicated research nurse. In the routine care setting the problems are accentuated.
- From the decrease in hospital admissions, there may be a decreased incidence of women with severe PID, but this proposition should be viewed with caution. There is, however, a definite change in the proportion of women with *N. gonorrhoeae* compared with *C. trachomatis* in the Darwin region (Table 3). This could in part be explained by the introduction of PCR, which has resulted in an increase in the sensitivity of testing for *N. gonorrhoeae* (previously reliant on microscopy and culture)

compared with *C. trachomatis* (already using antigen-based systems prior to PCR). Alternatively, the community-based azithromycin treatment of chlamydia may have truly reduced the prevalence of the latter pathogen.

- Before commencement, critical review of recruitment numbers using routine diagnostics is needed to establish feasibility of the study.

We were unable to establish the efficacy of an azithromycin based regimen. However, in light of the recognised difficulties with compliance we are currently reviewing the PID treatment protocol and considering the option of the addition of azithromycin to the standard antibiotic regimen.

### Reference

1. Mein J, Bowden FJ. A profile of inpatients STD-related pelvic inflammatory disease in the Top End of the Northern Territory of Australia [see comments]. *Med J Aust* 1997; 166(9):464-467.

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## Hepatitis A Vaccination Policy

### Background

The epidemiology of hepatitis A virus (HAV) is highly variable and constantly changing. Clinical HAV infection is largely determined by two factors: age and the prevalence of antibodies to hepatitis A virus (anti-HAV) which indicate the level of

population immunity.<sup>1</sup>

Worldwide, geographic areas can be characterised by HAV patterns of high, intermediate and low endemicity.<sup>1</sup> In most developed countries (ie Australia) endemicity is low and tends to occur among specific risk groups (ie child care workers) (Table 1).

**Table 1 Epidemiology of hepatitis A - worldwide**

Stage of development	Prevalence/ endemicity	Usual age of infection	Risk groups for clinical disease	Outbreaks
Developing	High	< 5 years		Rare
Transitional	Intermediate	5 – 15 years	Many	Common: community wide, food/water borne,
Developed	Low	> 20 years	Few eg: Travellers, Drug users, Child care workers	Occasional: daycare, food/water borne,

In the Northern Territory (NT) two levels of HAV endemicity are seen.

- In the general urban community prevalence is low with outbreaks limited to high risk occupational groups and other groups.
- In remote Aboriginal communities the prevalence is very high, similar to developing countries. A small study has shown a 90% prevalence of anti-HAV in children under 5 years of age in the Top End.<sup>2</sup>

### **Territory Health Services (THS) policy for hepatitis A vaccination**

In August 1994, a NT Hepatitis A Vaccination Program was introduced for:

1. Occupational risk groups:
  - a) High risk health care professionals
  - b) Other high risk occupations
2. Medical risk groups; and
3. Low risk THS staff.

All participants are required to enrol in the program as follows:

- THS hospital staff - Through staff vaccination clinic.
- All others - Through district CDC.

#### **1. Occupational risk groups**

- a) High risk health care professionals (vaccine paid by individual work unit for THS staff; all others as per decision of employer):
  - Paediatric staff - medical officers, registered and enrolled nurses;
  - Rural health staff - registered nurses, medical officers and Aboriginal health workers;
  - Rural environmental health officers;

- Menzies School of Health Research staff who make frequent visits to remote communities;
- Resident medical officers; and
- Medical students.

b) Other high risk occupations (vaccine paid by individual employee or employer):

- Staff in child day care centres;
- Teachers of the developmentally disabled;
- Teachers in remote Aboriginal and Torres Strait Islander communities;
- Staff of residential facilities; and
- Sewerage workers.

The vaccine is administered by THS or Aboriginal Medical Services through community health centres or staff vaccination clinics in THS hospitals.

#### **2. Medical risk groups**

Some individuals are not at increased risk of HAV infection but have medical conditions which put them at increased risk of complications if they have an HAV infection. The vaccine is provided free of charge through THS health centres or Aboriginal Medical Services for individuals with:

- Chronic hepatitis B;
- Chronic hepatitis C;
- Chronic liver disease;
- Haemophilia; and
- Haemodialysis or transplant recipients.

#### **3. Low risk THS staff (those not already listed in 1.a)**

Hepatitis A vaccination is available to all THS staff through THS health centres. The cost of the vaccine is the responsibility of the individual employee.

## Other risk groups

The following groups are also at increased risk of HAV infection and should consider vaccination:

- Persons travelling to areas of high or intermediate endemicity;
- Persons who share drug injecting equipment or bongs; and
- Persons engaging in frequent anal sex.

THS does not provide HAV vaccination services for individuals in these other risk groups. They should be referred to a medical practitioner or travellers to Health Services Australia on 8981 7492.

## Vaccination schedule

Three HAV vaccines are currently available in Australia: Havrix™ (SmithKline Beecham); VAQTA® (Merck Sharp & Dohme distributed by CSL); and a combined hepatitis A & B vaccine Twinrix™ (SmithKline Beecham). Twinrix is not available from THS hospital pharmacies (see Table 2).

## Adverse reactions and precautions

The vaccine is well tolerated with side effects being generally mild and self limited. The most common reported reaction is soreness, redness and swelling at the injection site. Frequency of symptoms decreases with successive doses.

The vaccine should not be administered to anyone with a hypersensitivity to any of the vaccine components.

## Pre and post vaccination testing

### Pre-vaccination testing

Pre vaccination testing for total hepatitis A antibody (anti-HAV) is recommended for all program participants. The consultation and pre-vaccination blood test are free through THS hospital laboratories for some occupational and all medical risk groups. Low risk THS staff are expected to pay for the pre-vaccination blood test.

### Post vaccination testing

Post vaccination testing is not required or recommended. The **Havrix™** and **VAQTA®** brands of HAV vaccine are highly immunogenic with detectable antibody documented as early as two weeks after one dose of vaccine. Seroconversion and protection results in 95% of adults and children 1 month after the primary dose, and booster dose(s) improve the 95% protection rate and extend the duration of protection.<sup>3</sup>

## Post-immunisation immunity

The duration of immunity following vaccination is not certain but mathematical models based upon the observed decline in antibodies suggest that protection persists for at least 20 years.

**Table 2 Standard HAV vaccines for Havrix™, VAQTA® and Twinrix™**

Brand		Age group (years)	HAV antigen dose (volume per dose and route)	Vaccination Schedule (months)	Injection site
Havrix™	junior	2-15	720 ELISA units (0.5 ml IM)	Two doses: 0, 6 to 12	Deltoid
	adult	≥ 16	1440 ELISA units IM (1.0 ml IM)	Two doses: 0, 6 to 12	Deltoid
VAQTA®	paediatric	2-17	25µ (0.5 ml IM)	Two doses: 0, 6 to 18	Deltoid
	adult	≥ 18	50µ (1.0 ml IM)	Two doses: 0, 6	Deltoid
Twinrix™	junior	1-15	HA 360 ELISA units + HB 10 mcg (0.5 ml IM)	Three doses: 0, 1, 6	Deltoid
	adult	≥ 16	HA 720 ELISA units + HB 20 mcg (1.0 ml IM)	Three doses: 0, 1, 6	Deltoid

## References

1. NHMRC Australian Immunisation Handbook 7<sup>th</sup> ed (draft) 1999. Australian Government Publishing Service, Canberra
2. Bowden FJ, Currie BJ, Miller NC, Locarnini SA, et al. Should Aboriginals in the "Top End" of the Northern Territory be vaccinated against hepatitis A? *MJA* 1994; 161:372.
3. Centers for Disease Control. Prevention of hepatitis A through active or passive immunization: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR* 1996; 45:1-30.

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## Diseases newly added to the NT Notifiable Diseases List

The NT Notifiable Diseases List has recently been modified (see *NT Disease Control Bulletin Vol 6 No 1 March 1999*) to further reflect:

1. Diseases of national importance, and
2. Diseases of local significance.

The clinical features, case definitions and public health action of diseases newly added to the list are outlined below. The diseases with ☎ next to them should be notified by telephone or by fax as soon as the diagnosis is made.

### 1. Diseases of national importance

#### ☎ Japanese Encephalitis

##### Clinical features

Fever, headache, meningeal symptoms, change in conscious state, coma, death. Infection may be mild or asymptomatic. Top End is a potential receptive area due to presence of the mosquito vector.

##### Case definition

- A clinically compatible illness

AND

- Demonstration of laboratory criteria for Japanese B encephalitis infection

**Laboratory and clinical notification** - urgent.

**Public health action:** If no history of travel outside Australia to JE endemic area institute formal investigation. Close liaison with Entomology Branch (if possibility of local acquisition).

#### ☎ Botulism (food borne)

##### Clinical features

Double vision, dry mouth, dysphagia, descending flaccid paralysis or bulbar palsy.

##### Case definition

- Clinically compatible illness with a history of exposure to a probable food source in the absence of a contaminated wound

AND

one of the following:

- Detection of *Clostridium botulinum* toxin in sera, faeces or food;
- Isolation of *Clostridium botulinum* from faeces;
- Epidemiological link to a confirmed case.

**Clinical and laboratory notification.**

**Public health action:** Urgent investigation of all cases in liaison with Environmental Health to exclude a common source outbreak.

#### Cryptosporidiosis

##### Clinical features

A parasitic infection causing diarrhoea, nausea, vomiting and abdominal colic. It can cause prolonged, life-threatening illness in the immune suppressed, especially in association with HIV infection. Faecal-oral transmission.

##### Case definition

- A clinically compatible illness

AND

- Detection of *Cryptosporidium parvum* oocysts in faeces

OR

- Detection of *C. parvum* life-cycle stages in intestinal biopsy specimens.

**Laboratory notification.**

**Public health action:** Exclusion from child care until diarrhoea has stopped. Exclude food handlers from food preparation until excretion of oocysts has stopped (usually within 30 days). Investigate contacts

and source of infection. Notify Environmental Health if a common source is suspected or an outbreak is identified. Notify Power and Water Authority if a waterborne outbreak is suspected.

### **☎ Toxigenic *E.coli* Related Illnesses (shiga-like/verocytotoxin producing - SLTEC/VTEC)**

- **Bloody diarrhoea**
- **Haemolytic Uraemic Syndrome (HUS) & Thrombotic Thrombocytopenic Purpura (TTP)**
  - **post diarrhoeal**
  - **SLTEC/VTEC related**
  - **sporadic**

HUS and TTP may occur as sporadic events and with no association with diarrhoea. However, epidemics of bloody diarrhoea associated with SLTEC/VTEC may result in large clusters of HUS and (less commonly TTP) with high morbidity and mortality, especially in children. Therefore all cases of HUS/TTP should be notified as a matter of urgency. For surveillance purposes the diseases are classified as follows: HUS/TTP, post diarrhoeal; HUS/TTP SLTEC/VTEC-related; HUS/TTP, sporadic.

#### **Clinical features**

HUS: microangiopathic haemolytic anaemia, renal injury, and low platelet count. TTP: as for HUS but can include central nervous system (CNS) involvement and fever. Diagnosis of each is dependant on the opinion of a specialist physician, nephrologist or paediatrician.

#### **Case definitions**

##### *i) Bloody diarrhoea, SLTEC/VTEC related*

- Bloody diarrhoea in an individual
- AND
- isolation of shiga-like toxin/verocytotoxin producing *E.coli* from a stool specimen
- OR
- identification of shiga-like toxin (verocytotoxin) in an *E.coli* isolate
- OR
- identification of the gene associated with the production of shiga-like toxin/verocytotoxin by nucleic acid amplification (e.g. PCR)

##### *ii) HUS, post diarrhoeal:*

All of the following:

- Microangiopathic anaemia (acute onset) (i.e., schistocytes, burr cells, or helmet cells) on peripheral blood smear

- Renal injury (acute onset) evidenced by either haematuria, proteinuria, or elevated creatinine level
- History of acute bloody diarrhoea in the preceding 3 weeks

Note: A low platelet count can usually, but not always, be detected early in the illness, but it may then become normal or even high. If a platelet count obtained within 7 days after onset of the acute gastrointestinal illness is not less than 150,000/mm<sup>3</sup>, other diagnoses should be considered.

##### *iii) TTP, post diarrhoeal*

- As for HUS
- PLUS
- fever
- AND
- CNS involvement

##### *iv) HUS/TTP, SLTEC/VTEC related*

- As for HUS/TTP, post diarrhoeal above
- AND
- isolation of shiga-like toxin/verocytotoxin producing *E.coli* from a stool specimen
- OR
- identification of shiga-like toxin (verocytotoxin) in an *E.coli* isolate
- OR
- identification of the gene associated with the production of shiga-like toxin/verocytotoxin by nucleic acid amplification (e.g. PCR)

##### *v) HUS/TTP, sporadic*

- As for HUS/TTP above but no history of preceding bloody diarrhoeal illness or evidence of SLTEC/VTEC infection.

**Laboratory and clinical notification:** urgent notification required.

**Public health action:** Identify source of SLTEC/VTEC *E.coli* infection and institute appropriate control measures. Notify Environmental Health..

### **Influenza**

Monitoring influenza activity in the NT is based on i) sentinel surveillance by selected GPs using a clinical case definition and ii) laboratory notifications based on virological and serological diagnosis.

#### **Clinical features**

Fever, cough, myalgia, malaise, sore throat

## Case definitions

### i) Sentinel surveillance (i.e. possible cases)

- Six or more of the following :
  - Sudden onset of symptoms (within 12 hours)
  - Cough
  - Fever
  - Rigors (shakes) or chills
  - Prostration and weakness
  - Myalgia or widespread aches and pains
  - No significant respiratory physical signs other than redness of the nasal mucosa and throat
  - Influenza in close contacts

### ii) Laboratory surveillance (i.e. confirmed cases)

- Isolation of influenza virus in cell culture  
OR
- A four-fold rise in antibody titre to influenza in sera taken 10 days apart during the acute and convalescent phase  
OR
- A single high titre\* in the presence of clinical features consistent with the sentinel surveillance definition in i) above.
- The level of the single high titre will vary according to the type of serology test performed and the particular laboratory used.

### **Clinical and laboratory notification.**

**Public health action:** Promote vaccination in high risk groups. Consider use of antiviral agents in susceptible, high risk contacts. Consider boosting immunity by re-vaccination in institutions/nursing homes.

## Lyssavirus: Australian Bat Lyssavirus

### Clinical features

Encephalomyelitis caused by a virus endemic in Australian bats which is antigenically related to the rabies virus.

### Case definition

- Clinically compatible neurological illness  
AND
- identification of ABL in a clinical specimen by specific PCR and/or culture

### **Laboratory notification.**

**Public health action:** Joint investigation of source, including a detailed travel history. Notify Australian Quarantine and Inspection Service, Environmental Health, DPI, Communicable Diseases Network Australia New Zealand urgently if local transmission is

suspected.

## Vibrio food poisoning

### Clinical features

Incubation period usually 10-20 hours. Abdominal colic and watery diarrhoea; nausea and vomiting may be present. Illness usually self limiting within 1-2 days. Usually associated with consumption of raw or partially cooked shellfish.

### Case definition

- A clinically compatible illness

AND

- culture of *Vibrio parahaemolyticus* from stool specimen.

### **Laboratory notification.**

**Public health action:** Not communicable from person to person, therefore exclusion unnecessary. Investigate clusters to identify potential source. Liaise with Environmental Health as appropriate.

## 2. Diseases of local importance

### **Chlamydia infections: Conjunctival (added to genital chlamydia infections)**

### Clinical features

- i) Genital: urethritis, cervicitis, pelvic inflammatory disease, proctitis. May be asymptomatic.
- ii) Conjunctival: follicular conjunctivitis but may be asymptomatic; late sequelae including corneal scarring and blindness.

### Case definition

- Detection of *Chlamydia trachomatis* by either culture, antigen or DNA techniques (including PCR/LCR) in conjunctival specimens.

### **Laboratory notification.**

**Public health action:** Appropriate treatment and education, contact tracing and other STD screening for genital disease. Promotion of general hygiene measures in trachomatous areas. Refer to *Guidelines for treatment of trachoma in the NT* (1998). \*Conjunctival chlamydia (trachoma) on clinical grounds only ie without laboratory confirmation is not notifiable in the NT. Information about prevalence is derived from occasional community surveys based on WHO diagnostic criteria.

## Melioidosis

### Clinical features

A bacterial infection causing a spectrum of disease ranging from sub-clinical infection, superficial and deep abscesses, to pneumonia, septicaemia and encephalitis. Immunodeficiency states (e.g. diabetes, alcoholism, malignancies, steroids, chronic lung, liver and renal disease) predispose to infection and increase the risk of mortality.

### Case definition

- A clinically compatible illness

AND

- culture of *Burkholderia pseudomallei* from appropriate specimens

### Clinical and laboratory notification.

**Public health action:** To be determined on a case by case basis. Environmental studies (e.g. sampling of water supply) to determine source of *B. pseudomallei*

during clusters of disease or when cases are reported from new geographical locations.

## Trichomoniasis

### Clinical features

Vaginal itch, discharge or odour in women; urethritis (rarely prostatitis) in men; most infections are asymptomatic. Associated with premature rupture of membranes and premature labour.

### Case definition

- *Trichomonas vaginalis* detected in appropriate specimens by one or more of the following:
  - microscopy of a wet preparation or Pap smear
  - culture
  - nucleic acid amplification test (e.g. PCR)

### Laboratory notification.

**Public health action:** Appropriate treatment, education, contact tracing, and screening for other STDs.

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## Culturally and linguistically diverse women's project

*Mahasti Farshidi, Women's Cancer Prevention Program*

One of the projects in the NT Women's Cancer Prevention Program is allocated to the health needs of women from diverse cultural backgrounds. The aim of this project is to increase knowledge and awareness about women's health issues focussing on Pap smear and breast screening and to maximise participation rate of the women from diverse cultural backgrounds in these screenings.

As a culturally and linguistically diverse (CALD) women's project officer, I network with individuals and groups within diverse cultural background communities and other organisations to promote health. Liaising with other health organisation is also an essential part of this project.

The strategies that we use to promote and improve the health status of the ethnic communities are:

- Provide education and information to CALD women through a variety of strategies developed in conjunction with individual CALD communities. These include conducting health information session for communities, training bilingual women

as health educators to pass information to their communities in their own languages.

- Organise women's health days in Darwin and other regions in the NT for women from diverse cultural backgrounds.
- Organising special well women's clinics for CALD women in Darwin and other regions throughout NT when possible.
- Consulting with ethnic communities about the health needs of women from diverse cultural backgrounds.

With the possible extension of this position across CDC, the above roles and strategies would be expanded in an effective way such as training of bilingual health educators in other health areas and working closely with CDC to work out other strategies to assist women from diverse cultural backgrounds in improving their health status.

For further information please call Mahasti Farshidi on 89 225513.

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## Immunisation update

### Influenza

Good News! The two Commonwealth funded influenza programs will continue in the year 2000. As in 1999, influenza vaccine will be funded by the Commonwealth for:

1. **Non-Aboriginal** people 65 years of age and older on the day of vaccination; and
2. **Aboriginal** people who are:
  - 50 years and older on the day of vaccination; or
  - 15 years and older who are in high risk groups as per the NHMRC recommendations.

Each year the vaccine is changed to protect against the strains most likely to circulate in the community. All high risk people therefore need annual influenza vaccination.

The influenza vaccine for the year 2000 is expected to be available by the middle of January. Please place your order with your THS regional pharmacy NOW and start vaccinating ALL high risk people as soon as your order arrives. Remember, influenza can occur at any time of the year in the tropical north of Australia.

As recommended by The Australian Influenza Vaccine Committee the influenza vaccine composition for the year 2000 season will be:

**A (H3N2):** a A/Sydney/5/97 (H3N2) - like strain,

**A (H1N1):** a A/New Caledonia/20/99 (H1N1) - like strain,

**B:** a B/Beijing/184/93 - like strain,

### Pneumococcal Disease

Good News! The Commonwealth funded pneumococcal program for at risk Aboriginal people will also continue in the year 2000. As in 1999, the pneumococcal vaccine will be funded by the Commonwealth for:

**Aboriginal** people who are:

- 50 years and older on the day of vaccination; or
- 15 years and older who are in high risk groups as per the NHMRC recommendations.

In the NT, Aboriginal people 15 to 49 years old have a rate of invasive disease of 104/100,000 or 18 times the non-Aboriginal rate of 7/100,000 for that age group and are at high risk of disease.

**In addition**, CDC will fund pneumococcal vaccine for:

- Children 2 years to 14 years who are in high risk groups.

The current recommendation for pneumococcal vaccine is every five years. Many Aboriginal people received pneumococcal vaccine in 1995 and therefore revaccination will be required in 2000.

All health care providers will receive information early in January about the influenza and pneumococcal programs. Meanwhile, if you have any questions please contact CDC on 8922 8044.

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## Addendum

The staff at Disease Control, Nhulunbuy would like to acknowledge the assistance and administrative support provided by Karen Blyth during the men's

health screening that took place in May and June 1999 (reports published in Vol. 6, No. 3, September 1999 edition).

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**NT MALARIA NOTIFICATIONS****July to September 1999***Merv Fairley, CDC, Darwin*

Thirty seven notifications of malaria were received for the third quarter of 1999. The following table provides details about where the infection was thought to be acquired, the infecting agent and whether prophylaxis was used.

ORIGIN OF INFECTION	REASON EXPOSED	AGENT	PROPHYLAXIS	COMMENTS
PNG	Visit	<i>P vivax</i>	No	Diagnosed RDH.
India	Resident	<i>P vivax</i>	No	Diagnosed RDH
India	Resident	<i>P falciparum</i>	No	Diagnosed RDH
India	Resident	<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
PNG	Visit	<i>P falciparum</i>	No	Diagnosed RDH
PNG	Holiday	<i>P vivax</i>	Yes	Diagnosed ASH
India/Indonesia	Holiday	<i>P falciparum</i>	No	Diagnosed RDH
Africa	Holiday	<i>P vivax</i>	Partial	Diagnosed QML
PNG	Work	<i>P vivax</i>	Yes	Diagnosed RDH
Indonesia	Holiday	<i>P falciparum</i>	No	Diagnosed WDP
Indonesia	Resident	<i>P falciparum</i>	No	Diagnosed RDH
Pakistan/East Timor	Work	<i>P falciparum</i>	Yes	Diagnosed DPH
PNG	Holiday	<i>P falciparum</i>	Yes	Diagnosed GDH
West Africa	Work	<i>P falciparum</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P falciparum</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P falciparum</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P falciparum</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P falciparum</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P falciparum</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P malariae</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P malariae</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P vivax</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P vivax</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P vivax</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P vivax</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P vivax</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P vivax</i>	No	Diagnosed RDH
East Timor	Evacuee	<i>P vivax</i>	No	Diagnosed RDH
PNG	Visit	<i>P vivax</i>	No	Diagnosed RDH
PNG	Visit	<i>P vivax</i>	No	Diagnosed RDH
East Timor	Work	<i>P vivax</i>	No	Diagnosed RDH
East Timor	Work	<i>P falciparum</i>	No	Diagnosed RDH
Indonesia	Fisherman	<i>P falciparum</i>	No	Diagnosed RDH
Indonesia	Fisherman	<i>P falciparum</i>	No	Diagnosed RDH
Indonesia	Fisherman	<i>P vivax</i>	No	Diagnosed RDH
Indonesia	Fisherman	<i>P vivax</i>	No	Diagnosed RDH

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**NT NOTIFICATIONS OF DISEASES BY DISTRICTS  
1 JULY TO 30 SEPTEMBER 1999 AND 1998**

DISEASES	ALICE SPRINGS		BARKLY		DARWIN		EAST ARNHEM		KATHERINE		TOTAL	
	'99	'98	'99	'98	'99	'98	'99	'98	'99	'98	'99	'98
Acute Rheumatic Fever	0	2	0	0	2	3	0	3	6	4	8	12
Adverse Vaccine React.	0	1	0	0	0	3	1	0	1	0	2	4
Arbovirus infections												
Barmah Forest Virus	0	0	1	0	0	2	2	1	0	0	3	3
Dengue	0	0	0	0	1	4	0	0	1	0	2	4
Ross River Virus	0	0	0	1	15	7	1	0	1	2	17	10
Campylobacter	16	17	0	0	54	21	3	5	4	4	77	47
Chlamydia	91	76	9	8	113	96	22	8	33	23	268	211
Chlamydia Conjunct.	1	-	0	-	0	0	0	-	1	-	2	-
Cryptosporidiosis	0	8	0	0	3	5	4	0	2	0	9	13
Donovanosis	2	2	0	0	0	1	0	0	2	0	4	3
Gastroenteritis	0	0	0	0	0	0	0	29	0	0	0	29
Glomerulonephritis	0	5	0	0	0	0	0	2	0	0	0	7
Gonococcal Disease	132	102	14	9	86	117	18	30	43	61	293	319
Gonococcal Conjunct.	1	0	0	0	0	1	0	0	0	0	1	1
Haemolytic Uraemic Syn	0	-	0	-	1	-	0	-	0	-	1	-
Haemophilus Inf type b	0	0	0	0	1	0	0	0	0	0	1	0
Hepatitis A	5	0	2	0	26	4	0	3	2	2	35	9
Hepatitis B	0	0	2	0	1	0	1	0	0	6	4	6
Hepatitis C (prevalence)	5	5	0	1	49	64	1	0	1	5	56	75
HIV infections	0	0	0	0	0	1	0	0	0	0	0	1
HTLV-1	2	8	0	1	1	0	0	0	0	2	3	11
Influenza	23	-	1	-	5	-	1	-	3	-	33	-
Legionnaires Disease	0	0	0	0	1	2	0	0	0	0	1	2
Leptospirosis	0	0	0	0	0	0	0	0	0	1	0	1
Malaria	1	0	0	0	30	3	1	0	0	0	32	3
Measles	0	0	0	0	3	0	0	0	0	0	3	0
Melioidosis	0	0	0	0	0	0	0	0	0	1	0	1
Meningococcal Infection	1	5	1	0	0	0	0	0	0	0	2	5
Mumps	0	0	0	0	2	1	1	0	0	1	3	2
Pertussis	0	0	0	0	0	9	0	0	0	0	0	9
Pneumococcal Disease	12	13	0	0	8	5	0	0	3	0	23	18
Rotavirus	8	6	0	7	17	51	0	8	6	5	31	77
Salmonella	9	14	0	1	31	47	1	8	9	9	50	79
Shigella	5	10	0	1	9	7	1	6	1	1	16	25
Syphilis	16	42	9	19	17	14	10	15	8	4	60	94
Trichomonas	41	-	7	-	107	-	117	-	74	-	346	-
Tuberculosis	1	0	0	0	9	7	0	0	1	0	7	7
<b>Total</b>	<b>372</b>	<b>316</b>	<b>46</b>	<b>48</b>	<b>592</b>	<b>475</b>	<b>185</b>	<b>118</b>	<b>202</b>	<b>131</b>	<b>1397</b>	<b>1088</b>

**Points to note regarding notifications**

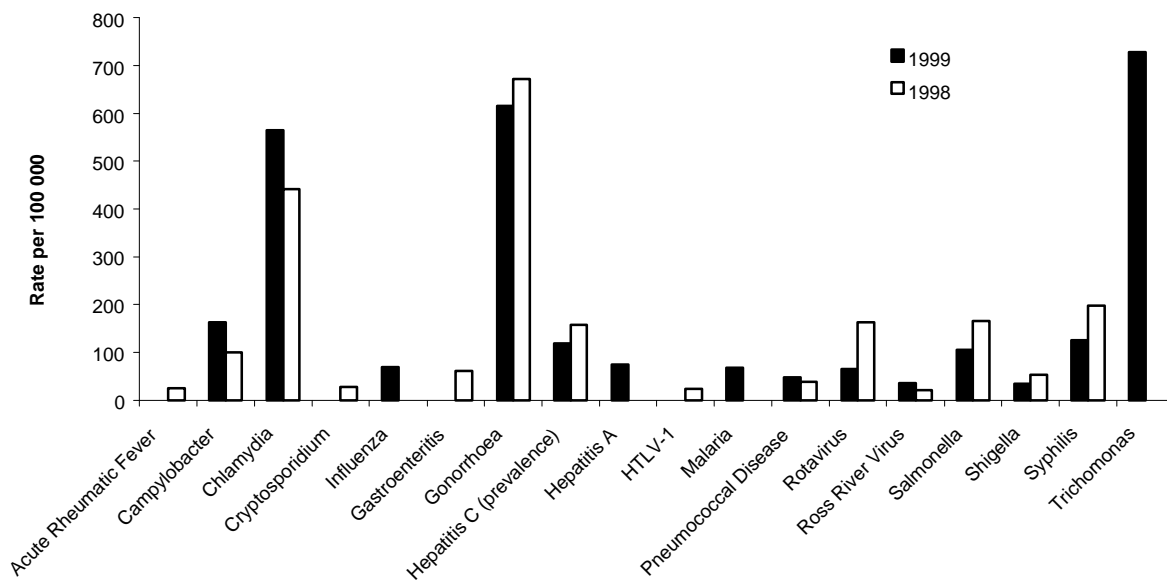
- Amoebiasis, Australian Encephalitis (MVE, Kunjin, Kokobera), Botulism, Brucellosis, Chancroid, Cholera, Congenital Rubella Syndrome, Congenital Syphilis, Diphtheria, Hepatitis C (incidence), Hepatitis D & E, Hydatid Disease, Leprosy, Listeriosis, Lymphogranuloma venereum, Poliomyelitis, Rubella, Typhoid, Typhus, Viral Haemorrhagic Fever and Yersiniosis are all notifiable but had "0" notifications in this period.
- The increase in East Arnhem and Alice Springs chlamydia cases this quarter reflects screening activity.
- Trichomonas was not notifiable in 1998.
- In Darwin, 26 cases of hepatitis A were notified to CDC during this quarter; a six-fold increase compared to the same period for 1998. The majority of the cases involved parents or care-givers of young children in childcare. Most of the cases were linked to one Darwin child care centre.
- The increase in malaria cases was secondary to East Timorese evacuees and workers from East Timor.
- The rise in campylobacter notifications for Darwin this quarter could not be attributed to a common source.

**NOTIFIED CASES OF VACCINE PREVENTABLE DISEASES IN THE NT  
BY REPORT DATE 1 JULY TO 30 SEPTEMBER 1999 AND 1998**

DISEASES	TOTAL		No. cases among children aged 0-5 years	
	'99	'98	'99	'98
Congenital rubella syndrome	0	0	0	0
Diphtheria	0	0	0	0
<i>Haemophilus influenzae</i> type b	1	0	0	0
Hepatitis B	4	6	0	0
Measles	3	0	2	0
Mumps	3	2	1	0
Pertussis	0	9	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 JULY TO 30 SEPTEMBER 1999 AND 1998**



Rates < 10 000 not listed

NT est. resid. pop – 189 987 as supplied by ABS

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## STAFF UPDATES

### DARWIN

After returning from Oxford, UK and acting Head of Surveillance for 3 months, **Frank Bowden** finally departed CDC at the end of October to take up the position of Director of the Gilmore Sexual Health Centre at Canberra Hospital. **Fay Johnston** is currently acting in the surveillance position on a part-time basis until it is filled. The position will be advertised mid January.

**Nathan Zweck** recently commenced a 12 months position as Medical Officer in the TB/Leprosy Unit. In addition to TB/leprosy work, his role will extend to assisting in setting up the National TB Program in East Timor. Previous programmatic experience includes working as Zonal Leprosy Supervisor in the Eastern Zone of Uganda from 1993-1996 (which also involved clinical work in the TB/leprosy referral hospital). He recently completed the MPH and DipTM&H at James Cook University while working at Townsville Aboriginal and Islanders Health Service from 1997-1999.

**Kathleen Hocking** is currently acting as the CDC Business Manager (formerly occupied by Helen Thistlethwaite). Not a stranger to the unit, Kathleen previously worked in administration in 1986 before moving to the Department of the Chief Minister's Office in 1991. Several other project positions in THS and a stint of maternity leave took her to Budget Management Coordinator in Family & Children's Services in 1998 before returning to CDC in September this year.

### ALICE SPRINGS

Staff in Alice Springs recently welcomed **Alex Brown** into the Disease Control Medical Officer position. Born and bred on the south coast of New South Wales, Alex never really contemplated becoming a doctor. He entered medical school at Newcastle University on a whim, and thrived until the chance of becoming a rock star beckoned. He returned to medicine when he realised that he was an awful musician and graduated in 1995 before proceeding to Gosford District Hospital on the NSW Central Coast, for intern and RMO1 years. He spent a further twelve months at Gosford as an ICU and anaesthetics registrar before receiving the inaugural Victorian Friends of Hebrew University Indigenous Doctor Scholarship to study for an MPH in Jerusalem, Israel. In September 1999, he completed the degree, with a thesis on Comparative analysis of Indigenous/Non-Indigenous cardiovascular mortality in Australia and New Zealand. His interests include enteric disease, HUS, CVD, NIDDM and Primary Health Care.

In November, **Jenny Hains** moved from the Public Health Nurse position into the Syphilis Database Officer position in the Sexual Health Unit, while **Margaret Stebbing** (formerly TB Control) took up the Public Health Nurse position. Interviews for the TB position will take place early in January.

### GOVE

**Simon Marrable** recently left the position of Men's Health Nurse in Nhulunbuy to commence medicine in Adelaide.



Happy New Year