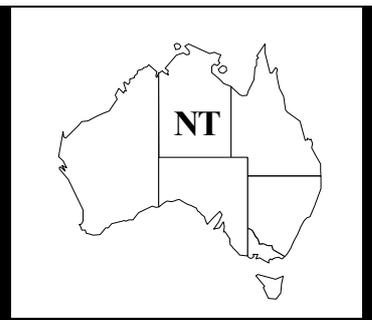




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Changes to the Northern Territory Childhood Vaccination Schedule - MMR

Nan Miller, CDC, Darwin

Australia is in the first stage of a strategy to eliminate measles nationwide. The strategy has included special programs such as Immunisation Days, where the public had access to vaccination clinics in shopping malls and a school program where measles-mumps-rubella (MMR) vaccine has been offered and administered with the aim to provide 5 to 11 year olds with a second MMR (MMR2). This later program acknowledges the National Health and Medical Research Council's (NHMRC) recommendation to shorten the interval between the two doses of MMR by bringing the second dose forward to 4-5 years of age (pre-school) instead of 10-12 years of age (currently offered in year six in the NT) and allows the entire cohort of children between these ages to be protected within a few months.

In this climate of change to the MMR schedule the NT differential policy of giving MMR1 to Aboriginal children at 9 months rather than 12 months of age has been reviewed and revised. There are therefore now two changes to the NT Childhood Vaccination Schedule and the rationale for these changes are as follows:

CHANGE 1: Shortening the interval between the two doses of MMR

The policy for measles vaccination has changed over the past decade from "just one shot", to "two

doses of measles containing vaccine" in 1990 and "two doses of MMR" in 1994. It is well recognised that measles is a highly infectious disease which spreads rapidly within a susceptible population. Greater than 95% immunity within a population is required to stop transmission. It is also recognised that no vaccine is 100% effective. The MMR vaccine is up to 95% effective after one dose if stored and administered correctly. It is at least 99%

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effective after 2 doses. Even if 100% (3,600) of the NT annual birth cohort receive one dose of MMR, 5% (180) will remain susceptible. At present there are approximately 10 years between the first and the second dose of MMR. Thus a pool of 1800 susceptible children can build up, which is enough to sustain an epidemic and put children too young for vaccination at risk of measles infection. Recognising that 100% coverage for MMR1 is not attained, the number of susceptibles would be considerably more than 1800. By moving the second dose forward to age 4-5 years, there are only 3 years available for susceptibles to build up prior to the second dose, lessening the risk of an epidemic. Striving towards 100% coverage, however, is still important.

CHANGE 2: First dose of MMR at 12 months of age for all infants

In 1984, it was recommended that all Aboriginal children be vaccinated against measles at 9 months of age.¹ This was and still is the WHO recommendation for developing countries. This policy was based on the parallels in morbidity and mortality profile of measles in Aboriginal children to those in developing countries. The policy also followed four deaths from acute measles illness in Aboriginal children less than one year of age reported in Central Australia in 1979 and a further epidemic in 1980-1981 which resulted in additional morbidity and mortality in Aboriginal children less than two years of age.² The excess mortality experienced and the assumption that the frequent cycles of outbreaks could continue contributed to the policy implementation.

The epidemiology of measles in the NT however, has changed since the 1980s. The NT has not experienced frequent cycles of epidemics. The average age of infection with measles in the Aboriginal population, which should guide recommendations for the age of MMR1, has risen.

In a 1994 outbreak in Aboriginal communities the average age of infection was older than 12 months³ and showed a similar age profile to that seen in developed countries. There were no measles related deaths during the epidemic.

In addition to the change in measles morbidity and mortality in NT Aboriginal children less than 12 months of age a comprehensive measles outbreak control policy exists which includes lowering the age to 6 months during outbreaks.

Therefore, the NHMRC recommendation that MMR1 be given at 12 months of age is now recommended policy for **all** NT children.

Conclusions

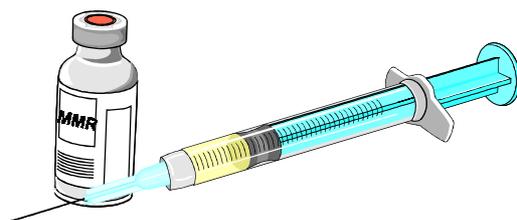
The following changes were incorporated into the NT Childhood Vaccination Schedule on 1 August 1998:

- **MMR1 - for all infants at 12 months of age; and**
- **MMR2 - at 4-5 years of age (pre-school).**

A copy of the new schedule is attached. Please discard all other schedules. A coloured wall chart of the new schedule will be available soon.

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Age	Vaccine	Route
Birth	Hepatitis B Vaccine (HBV)	IM
	* Hepatitis B Immunoglobulin	IM
	* BCG	Intradermal
1 month	HBV	IM
2 months	Diphtheria, tetanus, pertussis (DTPa) [#]	IM
	Oral polio vaccine (OPV)	Oral
	Haemophilus influenzae type b (Hib) [†]	IM
4 months	DTPa [#]	IM
	OPV	Oral
	Hib [†]	IM
6 months	DTPa [#]	IM
	OPV	Oral
	HBV	IM
12 months	MMR	IM
	Hib [†]	IM
18 months	DTPa [#]	IM
4 - 5 years	DTPa [#]	IM
	OPV	Oral
	MMR	IM
10 years (rural areas)	Tuberculin skin test	Intradermal
1st year High School (urban areas)	Tuberculin skin test	Intradermal
15 years, and then at 10 year intervals	Adult diphtheria, tetanus (ADT)	IM
15 years	OPV	Oral

[†]PRP-OMP (PedvaxHIB)

*Specified risk groups only

[#]diphtheria-tetanus-acellular pertussis

Notes on the schedule in the Northern Territory which varies from "The Australian Immunisation Handbook", 6th Edition NHMRC, 1997. This replaces the previous insert from the Centre for Disease Control (CDC), dated August 1997.

1. **BCG:** We recommend BCG at birth for Aboriginal neonates, neonates who will live in Aboriginal communities and neonates born to mothers who have been treated for leprosy.

For more information contact the CDC in your district, Community Health/Care Centre or your doctor.

☎ 8922 8044 Darwin
8951 6920 Alice Springs
8962 4259 Barkly
8973 8795 Katherine
8987 0359 East Arnhem

CDC, Darwin, August 1998



A review of BCG complications since the introduction of a different BCG vaccine

Lyn Barclay, CDC, Darwin

Background

Bacillus Calmette-Guerin, BCG vaccine, consists of a live attenuated strain of *Mycobacterium bovis* administered intradermally. The recommended dosage as well as the recommended groups to receive BCG have changed over the years. In 1991 BCG vaccination was no longer offered to the general population by way of the school BCG program but was recommended for those infants and young children at increased risk of tuberculosis (TB). Presently in the NT BCG vaccination is recommended for the following:

1. Aboriginal neonates;
2. Children under 5 years living in or travelling to countries or communities of high risk for TB and staying for greater than 3 months; and,
3. Neonates born to families of leprosy patients.¹

BCG was first used in 1921. Because BCG was never cloned, there is strain variability among BCG preparations produced by different manufacturers.² There are 4 main strains of BCG in the world today with varying reactivity.³

Data from 14 case-control studies in 12 countries show that the protective efficacy for BCG against TB has a range of 0% to 83%. The efficacy against TB meningitis and miliary TB in children under 5 years is 58% to 100%.⁴

The Commonwealth Serum Laboratories (CSL) stopped producing BCG in Australia in early 1996 and since July 1996 BCG has been supplied by Connaught of Canada. This BCG is a different strain to the CSL product.

In August 1997, the South Australian Health Commission expressed concerns regarding an apparent increase in adverse reactions following BCG that was linked to the Connaught strain. They asked if the NT had any data to support this observation.

The aim of this study was to determine if the introduction of the Connaught vaccine led to an increase in adverse reactions reported to the Centre for Disease Control (CDC) in Operations North (which includes Darwin, Katherine and East Arnhem districts)

Methods

Prior to August 1997, the NT had kept a database of BCG complications dating back to 1989. This consisted of a simple questionnaire with limited information. This information was updated by expanding the previous questionnaire and doing a clinic/hospital file search. All districts in the NT were then notified to advise that all adverse reactions be reported on the new questionnaire.

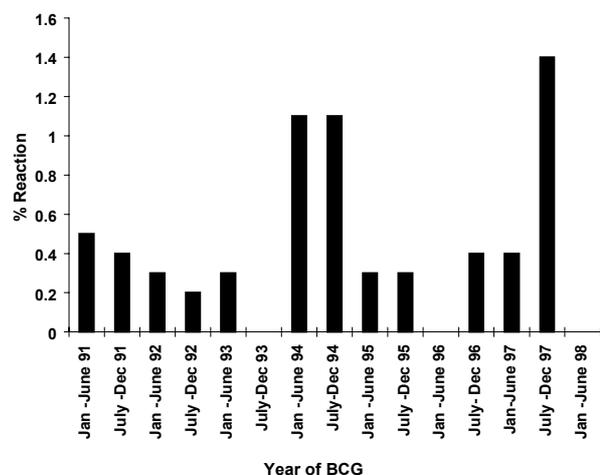
Data from Alice Springs and Barkly districts were omitted from this study as there were no data recorded in those districts prior to July 1996 to allow for comparison. Reactions recorded in the Darwin, Katherine and East Arnhem districts prior to 1991 were also omitted as the number of BCG vaccinations given in that time frame was unknown.

The number of BCG vaccinations given over successive 6 month periods was determined from the childhood immunisation databases starting in 1991 for the Darwin Urban and Rural, Katherine Urban and Rural, and East Arnhem districts. The total number of BCG vaccinations given has been used as the denominator for the calculation of adverse event rates, expressed as percentages.

Results

A total 8954 doses of BCG were administered to infants and young children from January 1991 to June 1998 with 38 associated adverse reactions; 27 occurred up to June 1996 and 11 from July 1996 to the end of the observation period. Figure 1 presents the rate (%) of adverse reactions by 6 monthly intervals over the total observation period.

Figure 1 Adverse reactions by 6 monthly intervals



The average rate of adverse reactions following BCG vaccination over the entire observation period was 0.5%. Prior to July 1996, 6819 doses of BCG were administered with 27 adverse reactions (0.4%). From July 1996, 2135 doses of BCG were given

with 11 adverse reactions (0.5%). There was no statistically significant difference in adverse event rates before and after the introduction of the Connaught vaccine.

Figure 2 Adverse reactions by region

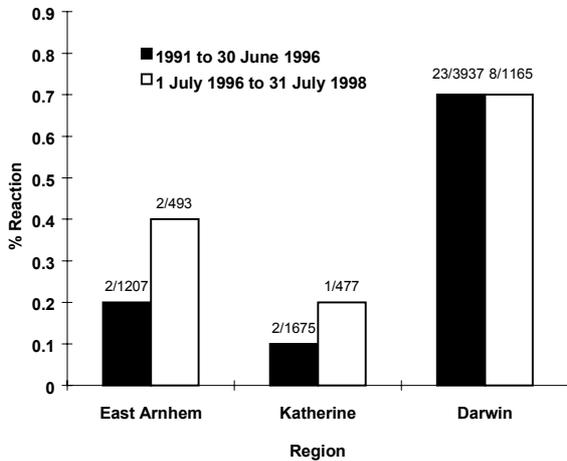


Figure 2 shows the differences (which did not reach statistical significance) in the adverse event rates before and after introduction of the Connaught BCG vaccine by district of administration.

Of the 38 adverse reactions that were investigated, 16% occurred in infants who did not belong to the risk groups for whom BCG is recommended under the NT protocol (see Figure 3).

Figure 3 Reason BCG given to the 38 cases resulting in an adverse reaction

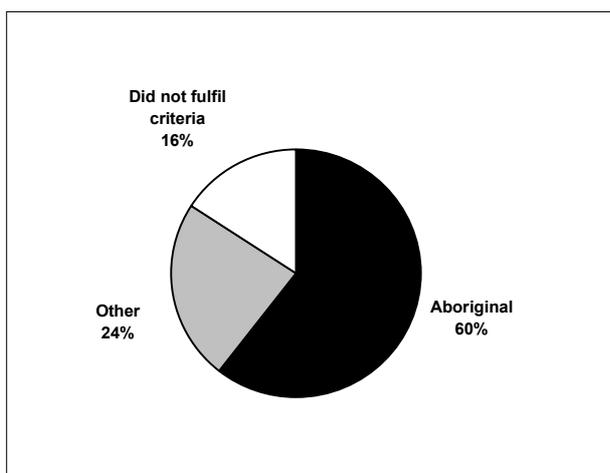


Table Classification of adverse reactions by BCG strain of the 38 cases

	CSL BCG	Connaught BCG
Enlarged or suppurative lymph node	11 (35.5%)	4 (26.7%)
Local abscess or local suppuration	11 (35.5%)	7 (46.7%)
Secondary bacterial infection	7 (22.6%)	2 (13.3%)
Incorrect dosage or documented technique error*	2 (6.4%)	2 (13.3%)
Total	31#	15#

* Documentation of technique was limited.

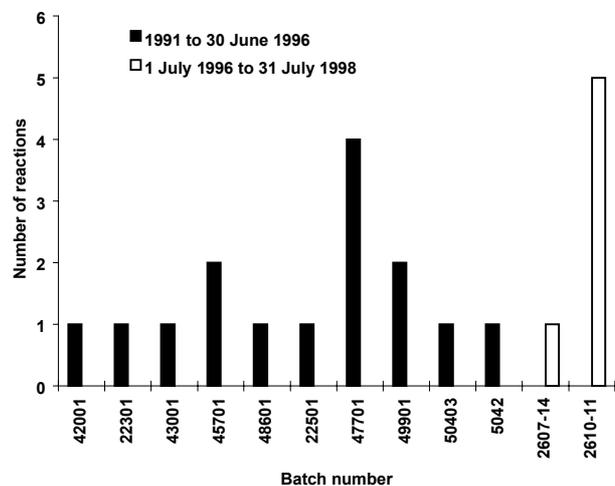
Total number of adverse reactions (4 cases had one or more adverse reactions).

Fourteen among the 38 cases who developed adverse reactions to BCG were admitted to hospital, 5 of whom were admitted for reasons unrelated to the BCG complications. Eleven of the 14 had axillary or supraclavicular nodes on the side where the BCG had been given.

Of the 15 cases with nodal involvement in our study, 5 were excised and 2 were drained. Of the 2 that were drained, 1 was given 6 months of isoniazid after drainage and the other was later excised. Fifty percent were left alone and resolved on their own.

There was no significant difference found in the nature or severity of reactions between the CSL and Connaught BCG preparations.

Figure 4 Batch numbers of BCG given



Batch numbers were recorded for only 21 of 38 complications reported making the data difficult to interpret. From July 1996, 5 adverse events were reported after administration of Batch 2610-11 and 1 after Batch 2607-14.

Discussion

This study failed to support the South Australian findings that Connaught BCG is associated with a higher rate of adverse vaccine reactions than the CSL BCG preparation. However, these data should be interpreted with caution. Complications were reported by people with varying experience and knowledge, eg ranging from rural community health centres, district hospital staff to TB Control Unit staff at Chest Clinics. With no standard definitions of the nature of reactions and their severity, documentation of these events is subjective to varying degrees.

As with all passively collected data, the TB Control Unit is reliant on people first of all presenting to health care providers with complications, and secondly having the incident recorded and reported by the health care provider to the TB Control Unit. The number actually reported therefore may only be a fraction of the whole. When memos were sent to all regions in August 1997 to report adverse BCG events, it may have prompted more reporting than in previous years thereby inflating the number of BCG reactions reported more recently.

Data on children from southeast Queensland reviewed by Queensland's Specialised Health Services showed the incidence of adverse events in 1994 using CSL vaccine was 0.7% (14 of 1945 vaccinated) as compared to 0.9% (19 of 2100 vaccinated in 1996-97) using Connaught.⁵ There did not appear to be a significant difference in the numbers of reactions.

In South Australia the adverse reaction rate reported from 1989 for the CSL vaccine was 0.7% and 3.1% for the Connaught BCG preparation. Errors in administration were considered unlikely as all personnel are trained and accredited.³

In our study only 55% of batch numbers were actually recorded. This makes any interpretation of adverse vaccine reactions associated with specific batch numbers difficult. The batch number now being used in the NT is 2612-12. Only one reaction has been reported from this batch to date.

It is well documented that when BCG vaccines are given properly, complications are rare.⁶ In our study there was limited documentation on technique and

so it is unknown how many reactions were due to technique errors. In 1994, when the Darwin region had 11 reported reactions, we found on investigation that BCG were being given by nursing staff regardless of experience. BCG can be given by all registered nurses but as it is not a universally administered vaccine and it is given intradermally rather than eg intramuscularly (IM) experience and additional training is seen as beneficial. An education program was set up by the Clinical Nurse Consultant of the maternity ward at RDH and only staff who were experienced in giving BCG were to give the vaccination. In 1997, when the percentage of reactions were relatively high (average 1.0%), again, there was no documented information to determine the underlying cause of the reactions.

Although Central Australian data were excluded from this analysis, the following anecdotal information is of interest. In the period 1997/98 there were 14 adverse BCG reactions reported from Alice Springs. Of these cases, 7 developed enlarged axillary nodes, 1 child developed an enlarged inguinal node following BCG given IM, 4 cases developed local and suppurative reactions (1 following documented incorrect dosage) and 2 others were given BCG subcutaneously rather than intradermally with no complications reported.

Adverse reactions by district show a higher percentage in Darwin than in Katherine and East Arnhem. It is difficult to explain this difference in reactions as all districts experience an overall high turnover of staff and BCG vaccinations are mainly given by nurses working in the midwifery wards in each district's hospital(s). Still, the experience and duration of service varies, with one non Darwin hospital known to have a long serving midwife. Alternatively, under reporting of reactions from Katherine or East Arnhem may account for some of the difference.

The results showed that 16% of the infants who developed adverse reactions did not fulfil the recommended criteria for BCG administration used in the NT Childhood Vaccination Schedule. Reasons for giving the BCG included staff not being aware of protocol changes over the years and pressure from a parent to give the vaccine.

Local reactions, including abscesses and lymphadenopathy, are reported in the literature to occur in 1% to 5% of people vaccinated with BCG.⁷ The percentage of complications was much lower than this in the Darwin, Katherine and East Arnhem

districts, both prior to (0.4%) and after (0.5%) Connaught commenced supplying BCG.

In Japan, Mori et al found that 0.73% of 34,516 BCG vaccinations resulted in lymphadenopathy.⁶ The study showed that in 222 cases with nodes greater than 10 mm in diameter, 90% showed shrinkage or disappearance within 7 months. Surgery accelerated healing and achieved a good cosmetic result. Whether or not we should excise nodes is a question always asked by treating medical personal and the answer remains controversial.

Hegster et al advocate surgical removal of lymph glands greater than 15 mm which are fluctuating.⁸ In their article, the authors report that spontaneous regression occurred in 62% to 67% of cases using conservative treatment or no treatment but again surgery made the area heal faster and gave a reported better cosmetic result. This corresponds with our study which showed 50% healed without surgical intervention.

Conclusions

There is a need to monitor adverse reactions following BCG administration and these reactions should be reportable to the Adverse Drug Reactions Advisory Committee for national assessment. Staff need to be vigilant in reporting to allow a true picture to be presented. With the introduction of an accredited course in vaccinology (*About Giving Vaccines*), available to all vaccine providers in the NT, faulty technique and incorrect dosage will hopefully be rare events and this will reduce the number of adverse reactions.

The sample size of this NT study was small but found little difference in the percentage of adverse BCG reactions before and after the introduction of the Connaught BCG preparation. The NT reported an adverse event rate well below the internationally accepted level. It may be that the initial Connaught batch imported to Australia was more reactogenic than the CSL preparation but reactions have been reduced with subsequent batches. It is important to record batch numbers and errors in administration technique for continued evaluation.

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Editorial

The above article brings to our attention several issues about BCG vaccination in the NT - and Australia.

Firstly, the NT records BCG vaccinations on its immunisation databases and transmits this information to the Australian Childhood Immunisation Register (ACIR). The ACIR records the BCG vaccination and makes an information payment but does not recognise the administering of BCG as a vaccination encounter for the purpose of funding service incentive payments to private

providers or for sending recall reminder letters and national coverage rates are not generated. There is, therefore, no nationwide monitoring of BCG coverage or assessment of its use in the recommended 'at risk' groups. These 'at risk' groups are those recommended by the NHMRC TB Working Party and include 1) Aboriginal and Torres Strait Islander neonates in regions of high TB incidence, 2) neonates born to parents with leprosy, and 3) children under the age of 5 years living or travelling in countries of high TB prevalence for more than 3 months. Secondly, there is no national

monitoring of BCG side effects by any surveillance system to answer such questions as whether a change in vaccine product may lead to increased side effects or whether administering techniques may need updating or, importantly, whether BCG side effects are becoming more of a concern for the present 'at risk' group than TB. Thirdly, though universally recognised definitions of BCG side effects may be problematic, a method to attain comparable data should be achievable if information of possible BCG adverse events is widely distributed to vaccine providers/recipients and parents and a specific BCG adverse events form is available. Finally, it is well recognised that many State/Territory Health Departments, institutions and health care employers do not follow the NHMRC TB Working Party recommendations for BCG vaccination. Even in the NT where these recommendations are actively supported and promoted, 16% of side effects are accounted for by infants/children outside of these 'at risk' groups. No doubt nationwide, many side effects could be prevented by more appropriate selection of those to be vaccinated.

Efforts are currently being focused on these issues. Presently The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) is coordinating a prospective study of BCG vaccine adverse events at the request of the National Centre for Disease Control and as endorsed by the Communicable Diseases Network Australia and New Zealand. An NCIRS BCG Vaccination and Adverse Events form will be used to collect and record information with adverse events identified according to the World Health Organization's (1997) definitions. This should provide useful information to guide BCG practices and hopefully prompt the continuous collection of BCG coverage and adverse reaction information.

More work is needed to achieve "practice consensus" on who is offered and administered BCG. Present evidence supports continuing BCG vaccination according to the NHMRC Working Party recommendations above.¹ The Paediatric Special Interest Group of the Australasian Society of Infectious Diseases (ASID) and the Australasian Paediatric Respiratory Group have recommended that 2 further groups be included for BCG vaccination based on consensus opinion.² These 2 groups are 1) neonates who will be living in a household which includes migrants or visitors recently arrived from countries of high TB

prevalence as well as neonates who have returned to visit the homes of relatives in countries of high incidence and 2) children and adolescents aged less than 16 years who continue to be exposed to a patient with active TB where the child or adolescent cannot be given preventive isoniazid therapy, or the person with active disease has organisms resistant to both rifampicin and isoniazid. Concerning these suggested additional recommendations, there are now many studies which show that children born in Australia with one or both parents born overseas have no higher risk of infection than those with Australian born parents^{3,4,5,6,7} and neonates who travel to countries of high TB incidence for more than 3 months are already recommended for BCG. The group that involves children being exposed to active TB without the possibility of successful preventive therapy would hopefully be a very limited number. Another area where there is diverse opinion and practice is in the administering of BCG among health care workers. Presently the Australasian Society of Infectious Diseases (ASID) council is seeking written views from its members for the formulation of a BCG and Health Care Worker position paper.

The role of BCG in reducing transmission and achieving TB control worldwide is limited. BCG continues to be recognised, however, as an important component of immunisation programs in neonates and children 'at risk' of TB to prevent disseminated disease. Side effects from BCG, though uncommon and largely self limiting, do occur. Documentation of BCG coverage and side effects, as well as comprehensive national TB surveillance data, is needed to guide best practice and correct selection of those to be vaccinated.

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Territory Day Celebrations 1998 in the Top End of the NT - an outbreak of firework-related injuries

Jacki Mein^{1,2}

¹CDC, Darwin, ²MAE program, NCEPH, ANU, Canberra

Introduction

Background

At present the Northern Territory (NT) and the Australian Capital Territory (ACT) are the only two jurisdictions in Australia that allow members of the public to buy shopgoods fireworks without a permit during a set period of time each year. In the NT from 1993-1997, the major cause of admission to public hospitals in the 5-14 year age group was Injury/Poisoning (indigenous rates in the 0-24 year age group being 86.7/1,000 population years, and non-indigenous rates 55.5 for the same age group).¹ The NT division of Kidsafe conducted an Injury Surveillance Program from July 1993 to July 1994 looking at accident and emergency presentations among admitted patients and outpatients.² There was no marked seasonal variation over the 12 month period and 2% (37/2315) of all accidental injuries were burns. Of the 37 burns, one was reported as being firework related (3%).

In the NT for the three days leading up to Territory Day on 1 July each year the public can buy certain approved fireworks without a permit, and set them off between the hours of 6 to 11pm on 1 July only. A somewhat liberal interpretation of these regulations ensues. It was therefore somewhat surprising against this background that the official number of firework related injuries for 1998 was three, according to the NT Workhealth Authority. CDC Darwin therefore reviewed other possible sources of data on fireworks related injuries for the week after Territory Day.

Objectives

1. To assess the number, nature, and severity of injuries sustained by people in the urban Darwin area presenting to health services from 24 June 1998 to 8 July 1998 when fireworks are available, and to compare these data with injuries reported in the preceding two weeks.
2. To make some working recommendations regarding the safety of fireworks based on the findings in the NT setting.

Methods

All case histories of people presenting to the Accident and Emergency department of the Royal Darwin Hospital (RDH) with injuries arising from fireworks or fires over the two week period Wednesday 24 June to Tuesday 7 July 1998 were assessed by prospective collection of data by the department's triage nurses. The same data were compared with a two week period averaged from the eight week period immediately preceding Wednesday 24 June, when the number of fireworks in Territory households was expected to be similar to the rest of the year.

Data on fireworks related injuries were also requested from the St John Ambulance Service NT, general practices in Darwin and Palmerston, urban Australian Defence Force (ADF) health services, urban Aboriginal Medical Services and Darwin Urban community care centres immediately after Territory Day and then again one week after Territory Day. These services were also asked about whether they had seen anyone with firework

related injuries in the two week control period before the fireworks became available.

Definitions used for injury severity

Severe: admitted to hospital for dressings, grafts or other procedures

Moderate: burn or eye injury requiring greater than one review by a health practitioner. This included infected burns.

Mild: requiring only one visit to a health practitioner.

Results

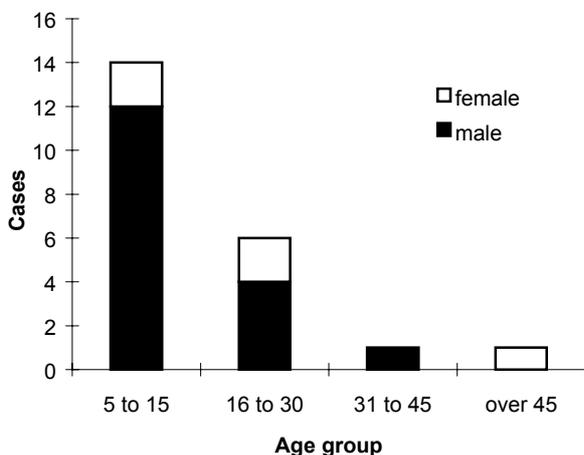
All of the 34 GP practices operating during the study period, all Aboriginal Medical Services, community care centres, ADF aid posts and hospitals in the Darwin urban area participated in the study. The only service that was contacted that could not provide details of firework related injuries for administrative reasons was the St John Ambulance Service, resulting in an overall response rate of 98%.

There were more firework related injuries (22) seen around the Darwin area than the NT Workhealth Authority official tally of three for 1998. There were no fatalities. Two were identified as being of Aboriginal or Torres Strait Islander descent.

Injuries by age group and sex

77% of all firework related injuries were sustained by males (17 cases) and 64% were sustained by the 5-14 year old age group (14 cases). There were no injuries reported in children below the age of five years.

Figure 1 Firework related injuries 1998 by age and sex



Nature and severity of injury

Figures 2 and 3 present data on the nature and severity of injury.

Figure 2 Severity of firework related injury 1998

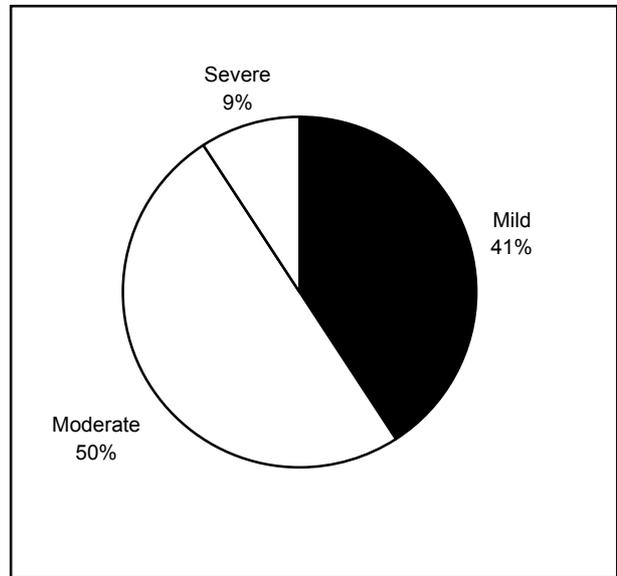
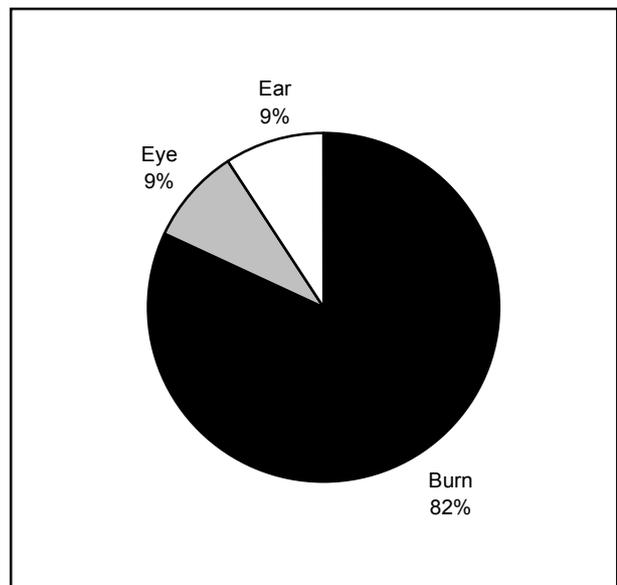
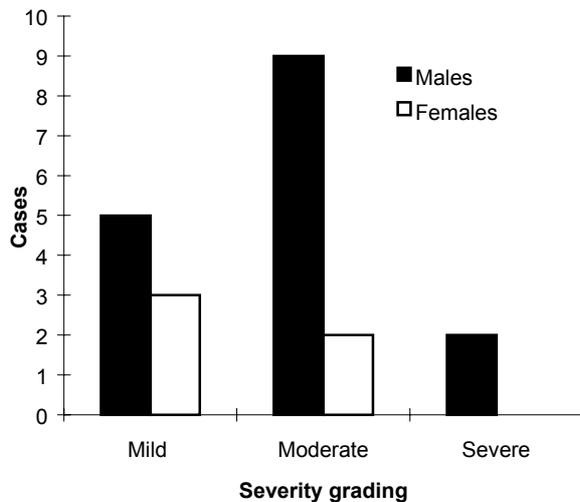


Figure 3 Types of firework related injury 1998



82% of injuries were burns and 59% were moderate or severe. Males with moderate or severe injuries comprised half of the total number of all firework related injuries (Figure 4).

Figure 4 Firework related injury severity 1998 by sex

Cause of injury

77% of injuries were preventable using simple safety rules.

Time of presentation

The majority of people presented and were injured on the night of 1 July, with a small number of presentations up to a week later. Six of the latter cases were injured in the five days after Territory Day.

Discussion

Over half of the firework related injuries that occurred or were treated in the Darwin area were males between the ages of 5-15 years. A similar age and sex distribution has been found elsewhere in Australia.³ This should be the target group for safety education campaigns. A review of the causes of injury indicates that over 75% of injuries that occurred were preventable if standard safety guidelines were followed. This reinforces the need for safety campaigns regarding fireworks in the NT.

Almost 80% of all injuries seen were burns, and 67% were classified as moderate to severe. Aside from small warnings on packets of fireworks there are at present no clear guidelines in the NT for the safer use of fireworks, aimed either at children or adults.

Also of note is the delay to presentation of many injuries related to fireworks. This probably relates to the delay before complications of burns such as infection develop, and to injuries sustained outside

the legal use period. It is clear from these data that an accurate assessment of the numbers of injuries sustained as a result of fireworks cannot be made until a week or more has elapsed after Territory Day. There were no injuries in the week before Territory Day, suggesting that most people do not let their fireworks off before the event.

There were seven firework related injuries which presented to hospital during the fortnight in which fireworks were available, compared to none in the control period. One of 15 control period burns (7%) was sustained by a child aged 5-14 years, compared with 35% (6/17) in the firework period ($p > 0.05$, NS). Five of the 6 firework period burns in this age group were firework related injuries. Burns made up 1% (1/79) of all-cause control period injuries in this age group. This finding is similar to the 2% overall reported by NT Kidsafe during its 1993-4 period surveillance period. In contrast, burns made up 26% (17/64) of all fireworks period injuries. Nine percent (6/64) of all-cause injuries in the 5-14 year age group were firework related in the fortnight around Territory Day.

Conclusions

In Darwin the rate of burns is higher in the 5-14 year age group around Territory Day than it is at other times of the year. At present there is little awareness of firework risks to health or adherence to the legal period for using fireworks.

Recommendations

1. Any assessment of the injury impact of Territory Day should be based on surveillance data collected for up to a week or more after 1 July.
2. The five hour legal period for the setting off of fireworks should be reinforced to assist in minimising the number of injuries.
3. Restricting the number of outlets able to sell fireworks would make adherence to sale regulations easier to police.
4. As the majority of injuries happen are easily preventable, simple, sensible guidelines for the use of shopgoods fireworks may decrease the rates of firework related injury and prevent the more severe ones. Guidelines are currently being developed in a joint effort by the Darwin Centre for Disease Control, the Workhealth Authority and Kidsafe NT for next year's Territory Day celebrations.
5. A campaign to distribute the new safety guidelines via fireworks packs and the media

immediately before Territory Day celebrations in 1999 is being developed. CDC plans to review the number, severity and type of injury prospectively to determine whether the campaign has had any impact on firework related injuries.

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1. Kennedy K, d'Espaignet ET, Measey ML. From infancy to young adulthood: Health status in the Northern Territory. Territory Health Services, Darwin 1998.
2. A report on the Northern Territory Injury Surveillance Program 1994, Kidsafe, Profile Publishing 1994.
3. Abdulwadud O, Ozanne-Smith J. Injuries and Fires associated with fireworks in Victoria: an epidemiological overview. Abstract 23, Second National Conference on Injury Prevention and Control 23-24 February 1998, Melbourne, Australia.

Box-Jellyfish in the Northern Territory

Bart Currie, Menzies School of Health Research and Royal Darwin Hospital

The official "stinger" season for the Northern Territory (NT) is from 1 October until 1 June. This is longer than elsewhere in tropical Australia. However, the major box-jellyfish, *Chironex fleckeri* is also responsible for around 5 sting cases each "safer" season (June until end of September) in the NT.

The last recorded death from *C. fleckeri* in Australia was in early 1996 in a 3 year old girl from a remote NT Aboriginal community. The last 10 stinger deaths in the NT have all been children, showing the greater risk of a smaller body mass exposed to the millions of stinging cells (nematocysts) on jellyfish tentacles injecting their venom threads into the dermis.

The rapidity of severe envenoming from *C. fleckeri* (and possibly also from related jellyfish elsewhere in the world) is unique in clinical toxinology. Death may be within a few minutes and if it occurs it is usually within 20 minutes of the sting.

Although the lethal toxins from *C. fleckeri* and their exact mechanisms of action remain poorly characterised, the prospective NT study of over 200 *C. fleckeri* stings has clarified some important clinical features, together with some recent publications elsewhere. The ongoing support of Royal Darwin Hospital Accident & Emergency staff and staff in coastal communities and district medical officers in providing skin sticky tape samples for identification of jellyfish species by nematocyst microscopy, together with stinger report forms, has been invaluable in improving our understanding and clinical management.

A summary of the current status is:

1. Arrhythmias are seen with severe *C. fleckeri* stings, supporting a primary cardiotoxic role in potentially fatal stings. A baseline ECG is useful for all but minor stings.
2. Despite the dramatic nature of severe envenoming, by far the majority of *C. fleckeri* stings are mild to moderate, with the initial severe pain well controlled with ice-packs and, for moderate stings, a single injection of narcotic analgesia if ice-packs are insufficient.
3. The efficacy of *C. fleckeri* antivenom remains to be proven, with conflicting results from laboratory studies.
 - i. There has yet to be a definitive report of *C. fleckeri* antivenom saving a life. There have now been three documented deaths despite *C. fleckeri* antivenom. While a number of severe envenomings have been given antivenom and survived, there are similar case reports where antivenom was not given. The suggestion from some animal studies that the antivenom is less effective than predicted initially has led to the NT and other recommendations that in life-threatening envenoming as much antivenom as available (eg up to 6 ampoules) be given if there is no initial response (see protocol below). Because death is usually rapid if it occurs, the scenario for definitively showing benefit of antivenom will be a major sting with cardiorespiratory arrest near a health centre or hospital where immediate resuscitation and rapid use of intravenous (IV) antivenom is possible. The protocol below is written for this scenario.
 - ii. As the natural history of the majority of stings is for pain to settle and as the majority of stings leave no scarring (especially if managed as with burns to prevent secondary infection of any blisters), the enthusiasm of earlier case reports

attributing pain response and lack of scarring to antivenom requires some scepticism. The prospective NT study has resulted in a protocol restricting use of antivenom to the rare life-threatening sting and occasional moderate or severe cases where there is persisting pain at the stings despite ice and narcotics. In such cases pain relief from antivenom has been sometimes but not always excellent. Antivenom given after several hours may prove unhelpful. Because controlled or comparative (eg antivenom versus narcotic) studies are unlikely, careful documentation of each use of antivenom remains important in improving our assessment of antivenom efficacy. The issue is also clouded by a reported shortage of *C. fleckeri* antivenom and apparent secrecy regarding the manufacturing process and whether the original innovative but crude method is still used for producing the jellyfish venom which is injected into sheep.

4. A recent publication from Tibballs et al of experiments in mechanically ventilated piglets has shown preincubation of tentacle extract with antivenom was effective in neutralising IV tentacle extract toxicity. The same study however showed verapamil actually increased mortality, in contrast to earlier animal studies suggesting a protective effect for verapamil. The only documented use of verapamil in human *C. fleckeri* envenoming was in the 1996 NT fatality, where it was given after the child probably had irreversible hypoxic damage from cardiopulmonary arrest. While verapamil has not yet been removed from the NT protocol (below), it remains as a last resort before abandoning resuscitation.
5. Recent pharmacokinetic studies of antivenoms show slow absorption of antivenom from intramuscular (IM) injections, with peak levels in blood taking up to 48 hours, with less than half the antivenom reaching the blood stream. The rapid envenoming with *C. fleckeri* and the uncertainties regarding efficacy of antivenom make IV antivenom clearly the route of administration of choice for life-threatening envenoming. Reports of "successful" use of IM antivenom therefore also raise scepticism and re-appraisal of the case reports suggests the improvement (which occurs in the vast majority of similar stings where antivenom is not given), may have been coincidental with the IM antivenom. The NT protocol still includes the possible use of IM antivenom for an "on-the-

beach" scenario of life-threatening envenoming, but now states the clear preference for IV antivenom.

6. The NT protocol continues to differ from the Queensland Surf Life Saving Association protocol in not including the use of pressure bandages and immobilisation (PI). PI was developed in Australia to delay lymphatic absorption of venom in snake bites and case reports also support its use in funnel-web but not red-back spider bites. Unlike snake venom, *C. fleckeri* venom is extremely rapidly absorbed, most likely directly into the vascular system and not via lymphatics. In life-threatening stings where time is so critical, the area of stings is extensive and almost invariably involves more than one limb and usually also the trunk. Bandaging and immobilisation in such circumstances is cumbersome and likely to distract the from the priorities of resuscitation and access to antivenom. Because the pressure may discharge unfired tentacle nematocysts, bandaging has only been recommended for after adequate application of vinegar to inactivate undischarged nematocysts. By the time this has all occurred the reality is that deposited venom is likely to have been already absorbed, even if adequate pressure to prevent venous absorption could be applied to all the stung area. The combination of all these factors make it unlikely that PI will be of benefit for the truly life-threatening extensive jellyfish envenoming. While the use of PI in "stinger drills" for the majority of stings will at least not be harmful to the patient, attempts at PI in serious stings may delay or interfere with potentially life-saving resuscitation and antivenom use.

Given the uncertainties regarding antivenom and other therapies for *C. fleckeri* envenoming, the message that prevention is simple becomes even more important.

DO NOT ENTER THE SEA AND MOST IMPORTANTLY DO NOT LET CHILDREN ENTER THE SEA DURING THE STINGER SEASON

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Riviere G, Choumet V, Audebert F, et al. Effect of antivenom on venom pharmacokinetics in experimentally envenomed rabbits: towards an optimization of antivenom therapy. *J Pharmacology and Experimental Therapeutics* 1997; 281: 1-8.

Tibballs J, Williams D, Sutherland SK. The effects of antivenom and verapamil on the haemodynamic actions of *Chironex fleckeri* (box jellyfish) venom. *Anaesth Intens Care* 1998; 26: 40-45.

Williamson JA, Fenner PJ, Burnett JW, Rifkin JF, editors. *Venomous & Poisonous Marine Animals. A Medical & Biological Handbook*. UNSW Press 1996.

Sticky tape method for identifying nematocysts:

Ordinary transparent sticky tape, 4-8 cm long, is applied to the sting site, firmly stroked several times, then removed and placed onto a glass slide, with the ends secured to the slide with additional tape. The slide is sent for microscopy, which is currently being done at Menzies School of Health Research.

Summary of treatment of *Chironex fleckeri* stings

On-the-beach treatment

1. Assess the conscious state and treat airway, breathing and circulation if necessary.
2. Liberally pour vinegar over the stung area for a minimum of 30 seconds to inactivate remaining stinging cells on skin and on any adherent tentacles.
3. If unconscious or if evidence of life-threatening cardiac or respiratory decompensation, and if antivenom is available, then 3 ampoules of antivenom (each containing 20,000 units) can be given IM by a trained health professional. However IV antivenom (see below) is preferable wherever possible.
4. In severe envenomation use oxygen if available. Entonox (50% nitrous oxide 50% oxygen) can be administered for severe pain.

Hospital treatment

1. Continue treating airway, breathing and circulation if necessary and add oxygen. Apply vinegar as above.
2. If unconscious or if life-threatening cardiac or respiratory decompensation, or if a significant arrhythmia is present, administer a minimum of 1 ampoule of antivenom IV (each ampoule 20,000 units, diluted 1:10 with an isotonic crystalloid solution such as Hartmann's solution or isotonic saline, given over 5-10 minutes). In a life-threatening situation up to 3 ampoules may be given IV consecutively if the response remains inadequate.
3. Cardiopulmonary resuscitation should be continued and not abandoned in the patient with ongoing cardiac arrest until after further therapy with even more antivenom (at least 6 ampoules total dose if available) and consideration of cardioactive drugs.
4. If supplies of antivenom are exhausted and there is persisting cardiac arrest or life-threatening arrhythmia, then before abandoning resuscitation consider giving verapamil IV (0.1 mg/kg up to 5 mg adult dose) while continuing cardiopulmonary resuscitation. Inotropes such as adrenaline should also be considered.
5. In stings which are not life-threatening (no cardiac or respiratory decompensation) use ice-packs for initial pain relief, together with IV or IM administered analgesia if necessary (1 mg/kg of pethidine up to 50 mg adult dose initially). For pain not relieved by ice-packs and narcotic analgesia, administer 1 ampoule of antivenom IV as above.
6. Further IV narcotic analgesia may be administered when necessary in conscious patients.

Endemic scabies in dogs and people are different

Shelly Walton¹, Annie Bonson¹, Bart Currie^{1,2} and David Kemp¹
¹Menzies School of Health Research, ²Royal Darwin Hospital

Molecular techniques, based on a *Sarcoptes scabiei* specific DNA fingerprinting system, were employed to ascertain whether dog scabies were infesting people in scabies endemic communities.¹ Parasites were collected from dog and human hosts in six Aboriginal communities in northern Australia. Additionally, with the establishment of some international collaborations, parasites were obtained from regional locations worldwide. Despite the difficulties encountered in the collection of mites the results of these studies clearly identified a number of important findings. All evidence compiled to date suggests mites consist of two host-associated populations and that interbreeding or cross-infection is extremely rare (Figure). Although no diagnostic microsatellite genotype (or combinations of genotypes) enabled the unambiguous discrimination of dog-derived or human-derived mites, a significant proportion of dog-derived mites exhibited a mutation (up to 98%) at the Sarms 20 locus. This feature was not observed in the human-derived mites and further investigations into this region of the *S. scabiei* genome may provide a fixed genetic marker. Although there are occasional reports of dog scabies causing an itch in humans, this first DNA typing study of scabies mites shows it is very unlikely that dog scabies is contributing significantly to scabies in people living in scabies endemic regions. Control

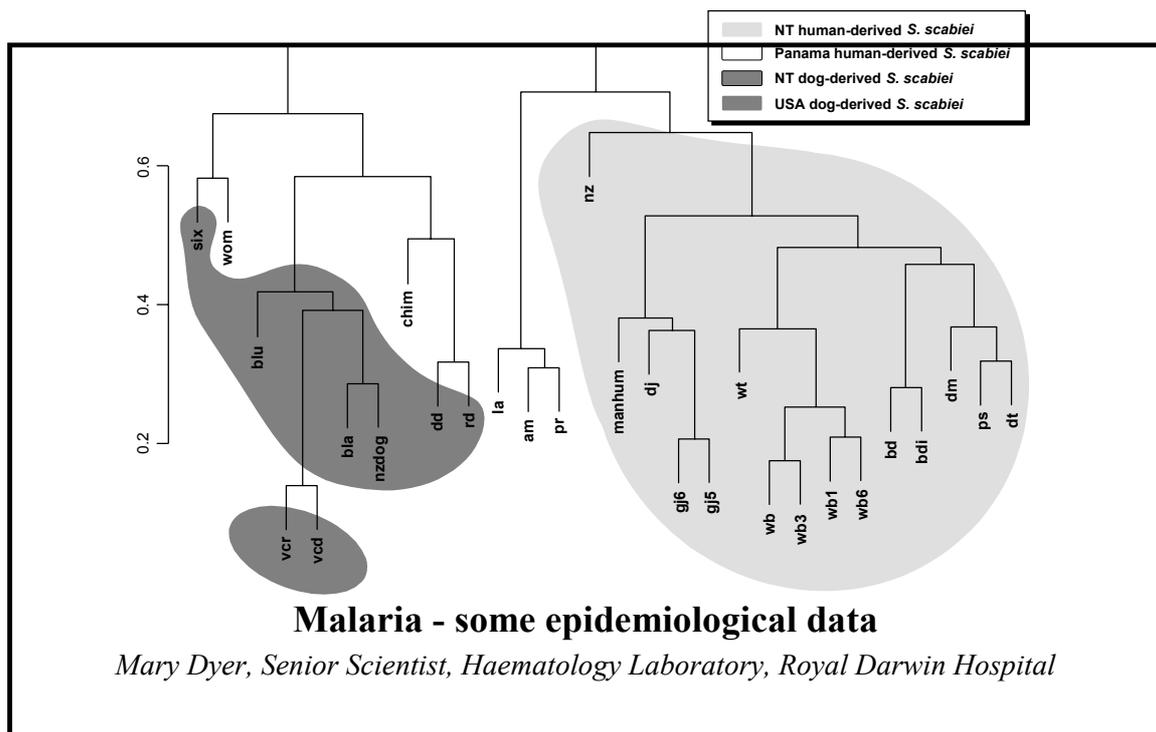
programs directed against human-derived *S. scabiei* in Aboriginal communities do not require resources directed towards zoonotic infection.

Parasites were also collected from a number of patients with sequential episodes of crusted scabies. Management of these patients has proved troublesome due to the difficulties in eradicating mites from heavily crusted areas of skin. Genetic studies on mites collected from relapsed patients suggested some similarities between sequential mite populations. This suggests that while most of the scabies relapses after ivermectin therapy are reinfestations from inadequately treated contacts, some severe cases may not have full clearance of mites even after 3 fortnightly doses of ivermectin.²

See *NT Communicable Diseases Bulletin* Vol. 4, No 3 for Guidelines for Community Control of Scabies and Skin Sores or contact Sue Reid at CDC on 8922 8089 for a hard copy.

References

- Walton SF, Currie BJ, Kemp DJ. A DNA fingerprinting system for the ectoparasite *Sarcoptes scabiei*. *Mol Biochem Parasitol* 1997; 85: 187-196.
- Huffam SE, Currie BJ. Ivermectin for *Sarcoptes scabiei* hyperinfestation. *Inter J Infec Dis* 1998; 2: 152-154.



Malaria is no longer endemic in mainland Australia however the mosquito vector flourishes in the NT and there is an ever present danger that malaria could be re-introduced. The haematology laboratory at Royal Darwin Hospital (RDH) examines between 400 and 600 thick and thin blood films each year; approximately 10% of patients screened are positive for malaria.

Chart 1 shows the number of positive cases for each of the last three years. Of the four Plasmodium species which cause malaria, *P. vivax* and *P. falciparum* are most commonly identified in this laboratory.

Chart 1 Malaria 1995 - 1997

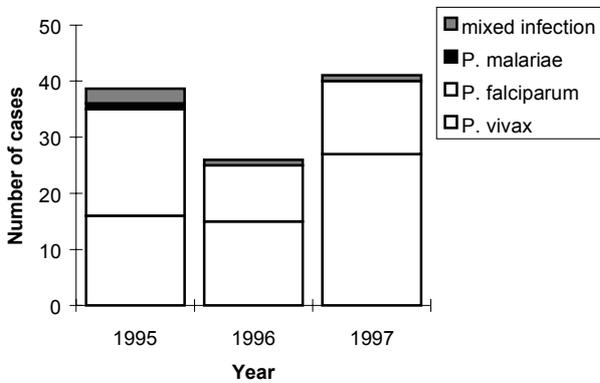
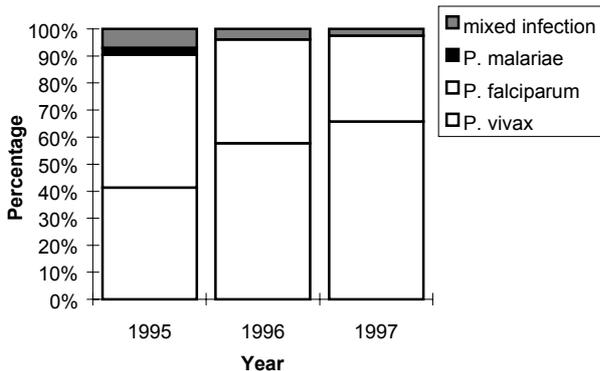


Chart 2 shows the proportions of the different types of infection.

Chart 2 Malaria 1995 - 1997 Relative Proportions



Charts 3-5 show the number of cases of each type of malaria and the country or region where the infection was contracted.

Chart 3 Malaria - Where From 1995

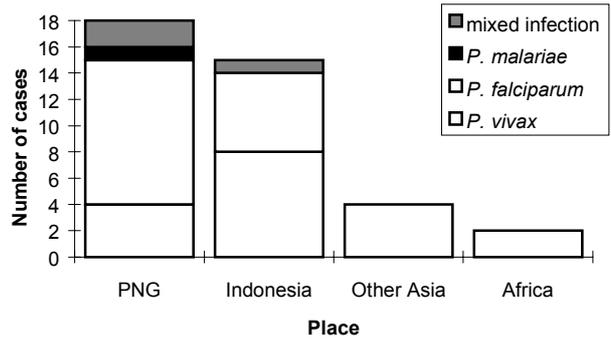


Chart 4 Malaria - Where From 1996

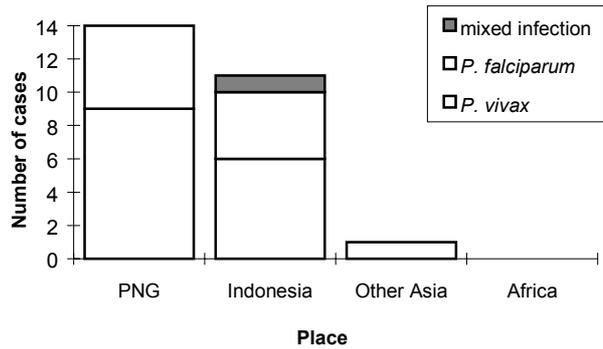
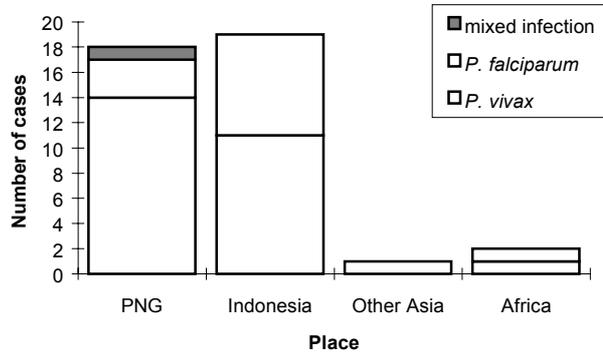


Chart 5 Malaria - Where From 1997



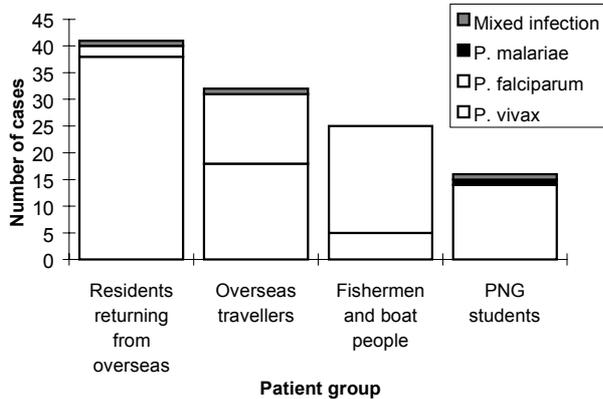
Not surprisingly, most of our cases of malaria are imported from Indonesia and PNG. If we look at the patients presenting with malaria, we see four distinct groups:

1. Australian residents returning from vacation or work overseas who present at RDH or other NT hospitals.
2. Travellers from overseas.
3. Indonesian fishermen and illegal immigrants (boat people) from Asia who arrive via Indonesia.

4. PNG students attending the NTU and NT high schools, screened routinely on their return from holidays.

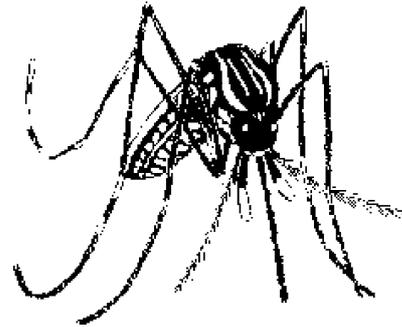
Chart 6 shows the distribution of Plasmodium species within these four patient groups.

Chart 6 Malaria - Patient Group



Broadly speaking, Indonesian fishermen, illegal immigrants and returning PNG students more commonly present with *P. falciparum*; travellers present with *vivax* or *falciparum* malaria in about equal numbers; and returning Australians more commonly present with *P. vivax*.

Thanks to Lyn Barclay, Peter Knibbs and Merv Fairley at CDC for providing data for this review.



**NT MALARIA NOTIFICATIONS
April to June 1998**

Compiled by Merv Fairley, CDC, Darwin

10 notifications of malaria were received for the first quarter of 1998. The following table provides details about where the infection was thought to be acquired, the infecting agent and whether chemoprophylaxis was used.

ORIGIN OF INFECTION	REASON EXPOSED	AGENT	CHEMO-PROPHY-LAXIS	COMMENTS
PNG	Work	<i>P.vivax</i>	Yes	Diagnosed RAAF Base, Darwin.
PNG	Holiday	<i>P.vivax</i>	Yes	Diagnosed RDH.
PNG	Work	<i>P.vivax</i>	Yes	Diagnosed RAAF Base, Darwin.
PNG	Holiday	<i>P.vivax</i>	Yes	Diagnosed RDH.
Africa/PNG	Holiday/Work	<i>P.malariae</i>	Yes	Diagnosed Tennant Creek Hospital.
Hong Kong	Student	<i>P.vivax</i>	No	Diagnosed RDH.
SE Asia	Holiday	<i>P.vivax</i>	Yes	Diagnosed ASH.
India/SE Asia	Holiday	<i>P.falciparum</i>	No	Diagnosed RDH.
Indonesia	Holiday	<i>P.vivax</i>	Yes	Diagnosed RDH.
Indonesia	Student	<i>P.vivax</i>	No	Diagnosed RDH.

**NT NOTIFICATIONS OF DISEASES BY DISTRICTS
1 APRIL TO 30 JUNE 1998 AND 1997**

DISEASES	ALICE SPRINGS		BARKLY		DARWIN		EAST ARNHEM		KATHERINE		TOTAL	
	'98	'97	'98	'97	'98	'97	'98	'97	'98	'97	'98	'97
Acute Rheumatic Fever	3	2	0	0	0	1	0	0	1	0	4	3
Adverse Vaccine React.	0	1	1	0	3	2	2	1	0	2	6	6
Arbovirus infections												
Barmah Forest Virus	0	4	0	4	4	7	1	0	3	4	8	19
Kokobera Virus	0	0	0	0	1		0	0	0	0	1	0
Kunjin Virus	0	0	0	1	0	0	0	0	0	0	0	1
Dengue	0	0	0	0	2	0	0	0	0	0	2	0
Ross River Virus	1	35	0	10	18	38	1	1	2	6	22	90
Campylobacter	19	40	0	1	15	15	2	2	4	4	40	62
Chlamydia	50	62	9	11	58	62	22	21	26	8	165	164
Congenital Syphilis	1	0	0	0	0	0	0	0	0	0	1	0
Cryptosporidiosis	13	-	0	-	22	-	0	-	3	-	38	-
Donovanosis	4	2	0	0	1	0	0	0	1	0	6	2
Glomerulonephritis	0	0	0	0	0	2	2	0	0	0	2	2
Gonococcal Disease	79	123	6	13	87	129	32	27	65	34	269	326
Gonococcal Conjunct.	0	95	0	2	1	0	0	0	1	0	2	97
Haemophilus Inf type b	0	1	0	0	0	0	0	0	0	0	0	1
Hepatitis A	0	17	0	3	5	8	3	0	4	13	12	41
Hepatitis B	0	1	2	1	0	2	1	0	0	2	3	6
Hepatitis C (prevalence)	6	9	0	0	46	64	1	0	3	2	56	75
Hepatitis D	0	1	0	0	0	0	0	0	0	0	0	1
HIV infections	0	0	0	0	4	2	0	0	0	0	4	2
HTLV-1	3	6	0	0	0	0	0	0	0	0	3	6
Malaria	1	1	1	0	8	11	0	2	0	2	10	16
Measles	0	0	0	0	0	0	0	1	0	0	0	1
Melioidosis	0	-	0	-	7	-	1	-	5	-	13	-
Meningococcal Infect.	3	1	0	0	0	2	0	2	0	0	3	5
Mumps	1	0	0	0	0	1	0	0	0	1	1	2
Pertussis	1	0	0	0	2	3	0	0	1	0	4	3
Pneumococcal Disease	11	8	0	0	1	7	0	2	0	1	12	18
Rotavirus	24	3	0	0	6	0	0	0	1	3	31	6
Rubella	0	1	0	0	1	1	0	0	0	0	1	2
Salmonella	19	22	2	6	60	36	6	3	14	17	101	84
Shigella	6	22	4	6	6	3	4	0	1	3	21	34
Syphilis	25	32	13	2	12	8	3	7	5	15	58	64
Tuberculosis	1	1	0	0	2	10	0	0	1	2	4	13
Typhus	0	1	0	0	0	0	0	0	0	0	0	1
Yersiniosis	0	0	0	0	1	1	0	0	0	0	1	1
Total	271	491	38	60	373	415	81	69	141	119	904	1154

Points to note regarding notifications:

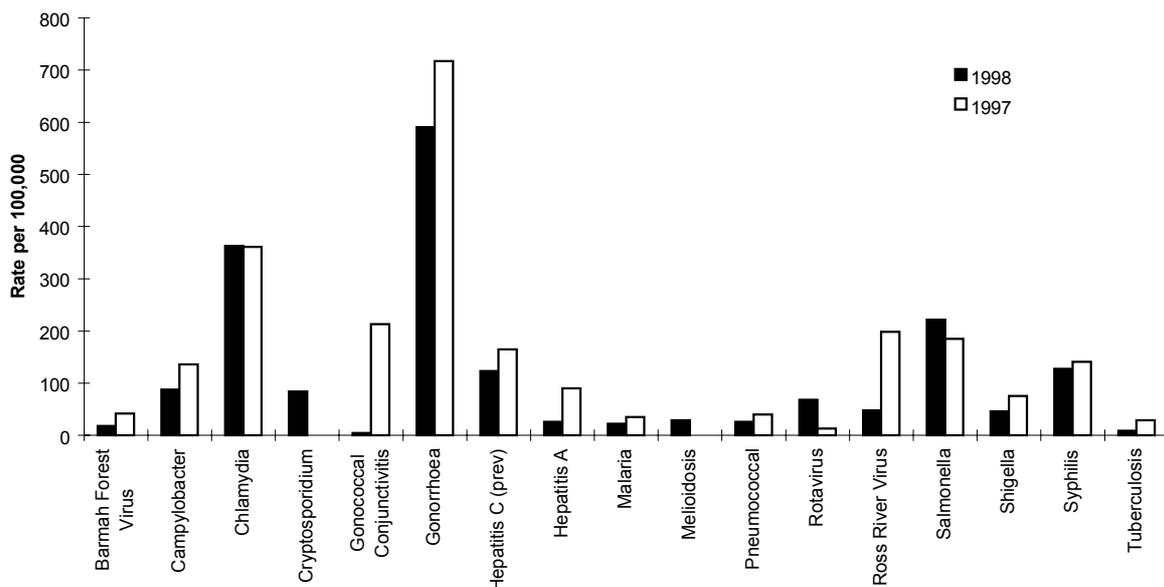
- Australian Encephalitis (MVE), Amoebiasis, Botulism, Brucellosis, Chancroid, Cholera, Congenital Rubella Syndrome, Diphtheria, Gastroenteritis in an institution or food handler, Hepatitis C (incidence) Hepatitis E, Hydatid Disease, Legionnaires Disease, Leprosy, Leptospirosis, Listeriosis, Lymphogranuloma venereum, Poliomyelitis, Typhoid, and Viral Haemorrhagic Fever are all notifiable but had "0" notifications in this period.
- Cryptosporidiosis will be notified routinely.
- The decrease in chlamydia and gonococcal disease in Alice Springs, Barkly and Darwin from 1997 to 1998 reflects the decrease in screening activity in 1998. The increases in Katherine may reflect increased staffing levels which allowed increased screening.
- The gonococcal conjunctivitis numbers reflect the large outbreak which occurred in Alice Springs in 1997 and contribute to the overall discrepancy of total notified diseases in Alice Springs for 1997 and 1998.
- Ross River Virus: The fall in numbers in the Top End this quarter compared to 1997 is due to a variety of factors. In Darwin; this includes increased effectiveness of control in the Leanyer swamp, drainage engineering works at the Casuarina Coastal Reserve, a shorter Wet Season and the flushing away of mosquito larvae by heavy rainfall during the Wet. Extensive control activities were implemented in Katherine after the flood.

**NOTIFIED CASES OF VACCINE PREVENTABLE DISEASES IN THE NT
BY REPORT DATE 1 APRIL TO 30 JUNE 1998 AND 1997**

DISEASES	TOTAL		No. cases among children aged 0-5 years	
	'98	'97	'98	'97
Congenital rubella syndrome	0	0	0	0
Diphtheria	0	0	0	0
<i>Haemophilus influenzae</i> type b	0	1	0	1
Hepatitis B (acute)	3	6	0	0
Measles	0	1	0	1
Mumps	1	2	0	0
Pertussis	4	3	1	1
Poliomyelitis, paralytic	0	0	0	0
Rubella	1	2	1	1
Tetanus	0	0	0	0

- Mumps is largely under-reported.

**NT WIDE NOTIFIABLE DISEASES
1 APRIL TO 30 JUNE 1998 AND 1997**



Rates < 10/100,000 not listed
NT 1996 mid year est. resid. pop - 181,923 as supplied by ABS

STAFF UPDATES

TENNANT CREEK

Sharon Doyle has recently been appointed to the position of Public Health Nurse in Disease Control, Tennant Creek. Prior to this (since January 1996) she was working at Tennant Creek Hospital as a Registered Nurse and midwife.

Fiona Maslin recently returned from maternity leave to resume her position as AIDS/STD Public Health Nurse in Disease Control, Tennant Creek.

DARWIN

Mark Di Francesco has recently joined forces with Lyn, Lesley, Merv and Michael in the Darwin TB/leprosy Unit. Mark's varied background

includes working in the Disease Control Unit in Katherine (1990/1991), remote area nursing and the last two years lecturing at Batchelor College.

EAST ARNHEM

Karen Blyth trained as a registered nurse in Scotland before moving to Australia 14 years ago. She completed midwifery training in Victoria and has spent the last 5 years living and working in the NT, mainly in remote communities. Having completed the Nursing Degree through NT University she is now currently filling a temporary position in Disease Control, working in women's health and STD's in the East Arnhem district.
