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New treatments for hepatitis C virus: the future is now

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Abstract

Chronic hepatitis C infection affects over 220,000 Australians. Between 10-30% of those with chronic infection will develop decompensated cirrhosis or hepatocellular carcinoma after an average of 30 years, both of which are generally fatal without a liver transplant. Chronic hepatitis C is the most common causative factor in patients receiving liver transplants in Australia, United States of America (USA) and Europe each year. Due to significant side effects, current treatments are difficult to tolerate and lead to clearance of the virus in only 50-60% of those treated. Better treatments are needed and the first wave of new treatments will be available for use in Australia in 2012.

Key words: hepatitis C; treatments

Introduction

The hepatitis C virus (HCV) was first identified in the late 1980s and its genome was cloned and sequenced within the following few years. However, unlike HIV and hepatitis B virus (HBV) there was no easily available animal model and HCV could not be grown in cell culture. It was only in 2005 that a complete cell culture system for propagating HCV was first described and from this followed a detailed understanding of the life cycle of the virus. Hence direct acting antiviral agents for HCV have lagged behind those for HIV and HBV by approximately 20 years.

Current standard treatment for chronic hepatitis C virus infection

Current treatment of HCV infection is based on interferon, a cytokine which harnesses the host's

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immune response to the virus. Cure rates with interferon treatment have incrementally improved over the past 15 years with serial refinements in treatment regimens. Conventional interferon alpha (which was injected 3 times per week and gave an 8% cure rate) has been replaced with the longer half-life pegylated-interferon alpha (PEG-IFN), and combined with the non-specific antiviral drug ribavirin. Ribavirin has no significant direct anti-HCV activity *in-vitro* but when added to interferon in treatment regimens it greatly improves response rates. PEG-IFN plus ribavirin cause side effects in nearly 100% of those treated, the most common being 'flu-like symptoms (in 90% of patients), major depression (10-20%), anaemia, neutropenia and thrombocytopenia.

The current standard treatment for HCV depends on the genotype of the virus. Genotype 1 (the most common in Australia, accounting for around 65% of infections) is treated with 48 weeks of PEG-IFN (an injection once per week) plus ribavirin (tablets twice per day) with a resultant chance of cure of 45%. Genotypes 2 and 3 are treated with 24 weeks of PEG-IFN and ribavirin with an 85% chance of cure. Hence, the first wave of new treatments will be targeted at genotype 1 HCV which are currently the most difficult to treat.

First generation direct acting antiviral agents for HCV: boceprevir and telaprevir

The new treatments for HCV are collectively referred to as 'direct acting antiviral agents for hepatitis C' or DAAs. The first DAAs, boceprevir and telaprevir, have already hit the market in the USA and Europe, and are likely to be TGA-approved in Australia in early 2012 and PBS-funded in Australia possibly by late 2012. These drugs are protease inhibitors, properly known as NS3/4a serine protease inhibitors, a similar concept to the protease inhibitors used for HIV treatment such as lopinavir or ritonavir. They prevent the virus polyprotein precursor being cut up and processed into new virions. They only have useful activity against genotype 1 HCV infection.

Boceprevir or telaprevir are used in addition to current standard treatment. Monotherapy with either of these drugs, or indeed any of the DAAs leads to rapid emergence of viral drug resistance, and so they should only ever be used in combination with other effective therapies. The addition of either boceprevir or telaprevir to current standard treatment leads to improved cure rates in genotype 1 infection by 45% to 70-80% and can shorten the required duration of treatment from 48 weeks down to 24-28 weeks in those who show an early favourable virological response.

However, the addition of a protease inhibitor is at the expense of more side effects and an increased pill burden. The main treatment-limiting side effect of telaprevir is rash which occurred in 40-50% of patients in pre-registration trials and resulted in cessation of telaprevir in 5-15%. Boceprevir causes anaemia in over 40% of patients but rarely lead to treatment discontinuation in pre-registration trials, primarily because of the use of erythropoietin to support haemoglobin levels.

Boceprevir and telaprevir have also been trialled in patients with previous treatment failure to standard therapy and have shown very good cure rates, even in this difficult to treat group, with cure rates of up to 80% in prior relapsers, 60% in prior partial responders and 30% in prior null responders.

It is likely that boceprevir and telaprevir will be TGA approved for use in both treatment-naïve and treatment-experienced patients with genotype 1 HCV infection. Treatment will be for 24-44 weeks of boceprevir or 12 weeks of telaprevir in combination with 24-48 weeks of PEG-IFN and ribavirin.

Subsequent generations of direct acting antiviral agents for HCV

There are at least 4 other classes of DAAs in the pipeline, the most important of which are nucleoside polymerase inhibitors (NPIs) and non-nucleoside polymerase inhibitors (NNPIs). These inhibit the NS5b RNA-dependant RNA polymerase and are analogous to HIV reverse transcriptase inhibitors. NPIs will work in all genotypes of HCV, whereas protease inhibitors and NNPIs will be tailored to work in specific genotypes, usually genotype 1. There are over 5 NPIs and 5 NNPIs (eg. filibuvir) in phase 1 and 2 clinical trials.

NS5a inhibitors are another class of drug that inhibit the NS5a protein. The NS5a protein-specific function is uncertain, but is important in viral replication. There are over 4 such drugs currently in phase 1 and 2 trials.

Subsequent generations of NS3/4a protease inhibitors are also in development, with over 12 candidate drugs in phase 1 and 2 trials (eg. danoprevir, vaniprevir). These are generally more potent than telaprevir and boceprevir with a higher barrier to resistance, less frequent dosing and less adverse effects.

Other drug classes include those which interact with host proteins (cyclophilin and caspase inhibitors) and those with an unknown mechanism of action (nitazoxanide).

Interferon-free combination treatment for HCV

The availability of drugs in several different drug classes opens up the possibility of the holy grail of HCV treatment: interferon-free treatment with DAAs. Some people cannot tolerate interferon or cannot be safely given it due to contraindications such as severe mental illness, autoimmune conditions or renal failure.

This concept has already been tested in phase 1 studies. In the INFORM-1 study, 72 treatment-naïve genotype 1 patients were treated with danoprevir (a protease inhibitor) plus RG7128 (a polymerase inhibitor) and the majority achieved an undetectable viral load after 14 days of treatment. It is unclear if such a strategy will lead to eradication of the virus (as with interferon treatment) or if long-term suppressive therapy will be needed (as with antiviral treatment of HBV and HIV). Interferon-free combination therapies for HCV are at least 5 years away from the market.

Conclusions

We live in an exciting time in the treatment of HCV with a paradigm shift occurring in the way the virus is treated and a large number of new therapies in the pipeline. As well as improved outcomes for patients, these new therapies will also bring with them a new set of challenges: antiviral drug resistance and novel adverse effects. The majority of currently infected patients with HCV have not undergone treatment or have failed to be cured by current treatments and are awaiting future developments. For such patients, the future is now.

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Measles cases in Darwin June 2011

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Abstract

In June 2011 there were 2 linked cases of measles reported in Darwin. The index case (Case 1) was a 12 year old girl arriving in the Northern Territory (NT) on an overseas flight. Case 2 was a 35 year-old social contact of Case 1. A public health response was mounted in accordance with National Guidelines. There were 36 people followed up during contact tracing for Case 2. Of these 8 were immunised against measles and none received human immunoglobulin. No further cases of measles were notified in the NT in the following 2 week period.

Key words: *measles; Northern Territory*

Introduction

Australia is deemed to be free of endemic measles.[1] However, measles cases still occur from time to time with most linked to travel or exposure to returned travellers.[2-4] In addition, vaccine failure can contribute to measles transmission in highly vaccinated populations.[4]

On 9 June 2011 a 12 year old female Australian citizen (Case 1) living in Singapore became unwell with a fever, coryza and cough. Later that day she travelled to Darwin to visit relatives. The fever persisted and 3 days later she developed a rash over her face and trunk. While unwell, she stayed with relatives and had no contact with the public apart from a brief visit to a supermarket. She was seen by a general practitioner on 12 June but no diagnosis was made as a result of this consultation.

Case 2 was a 35 year old female family friend of Case 1 who was exposed to Case 1 on 12 June and became unwell on 22 June with fever cough, coryza and conjunctivitis. On 24 June she developed a morbilliform rash, appearing first on her face and later becoming generalised to her trunk and limbs. She attended the Royal Darwin Hospital (RDH) Emergency Department (ED) on 28 June and measles virus RNA was detected via a throat swab polymerase chain reaction (PCR) test carried out by the RDH laboratory within 6 hours of her presentation.
This article describes the public health response and management of this 'measles cluster' in Darwin during June 2011.

Methods

An immunisation and contact history was sought from Case 1. As she was infectious until 17 June but not notified until 28 June (when Case 2 was notified), the 6 day window of opportunity for public health action had passed. The National Incident Room in Canberra was alerted regarding Case 1's flight details to better inform possible modes of transmission for any further measles notifications recorded in other jurisdictions. No media release was issued concerning the flight because of her delayed diagnosis.

Following National Guidelines,[1] a detailed immunisation and contact history (from 21 June) was obtained from Case 2. Despite being unwell, she had continued to work as a sole operator of a small business and reported face to face contact with up to 50 persons each day, most of whom were 'backpackers'. A list of these clients together with their contact details was made available and attempts were made to contact them by mobile phone and text messages. Backpacker hostels in the central Darwin area were informed and posters and copies of the measles factsheets were placed in all backpacker hostels.[5]

An alert was distributed NT-wide to all health-care providers requesting that they contact the Northern Territory (NT) Centre for Disease Control (CDC) regarding possible cases of measles. A media release identifying the business was published with the request that those who attended while the case was infectious contact NT CDC in Darwin.

Results

Case 1 had received 2 measles-containing vaccines during her childhood in Australia which were verified through the Australian Childhood Immunisation Register. No further

Table. Contact tracing and outcomes for contacts with unknown immunity to measles

Contact tracing outcome	Royal Darwin Hospital	Household	Social	Workplace	Total
Number	0	2	8	26	36
Measles immune*	-	0	3†	13†	16
Measles immunity unknown	-	2	5	13	20
Interventions for contacts with unknown immunity to measles					
Received measles vaccine	-	0	4	4	8
Received immunoglobulin	-	0	0	0	0
Health advice only	-	2	1	4	7
Declined follow-up	-	0	0	1	1
Lost to follow-up	-	0	0	4	4

Notes: *Documented receipt of 2 measles-containing vaccines or measles IgG positive; †One social and one workplace contact were unwell with a rash or febrile illness but were found to be measles immune (IgG positive) and had a negative throat swab (PCR) for measles RNA.

cases of measles were identified among her contacts and no further cases in Australia were linked to her flight from Singapore.

Case 2 reported that she had lived overseas during her early childhood years and did not have her immunisation records. Born in 1975, she therefore may have received none or only 1 measles-containing childhood vaccine.

Contact tracing for Case 2 included (i) RDH ED, (ii) household contacts, (iii) social contacts and (iv) workplace contacts (Table).

At RDH ED Case 2 spent less than 5 minutes at triage with no others in the queue. She was immediately provided with a mask and within 10 minutes of her triage assessment placed in a negative pressure respiratory isolation room. No ED patient contacts were identified. ED staff exposed to Case 2 had their measles immunisation status verified and managed appropriately by RDH infection control personnel. Of the household contacts 2 were exposed more than 6 days prior to the diagnosis being made and received health advice only.[1] In addition, 8 social and 26 workplace contacts were followed up. No contacts of Case 2 received normal human immunoglobulin.

In response to the media alert, a further 5 persons were referred to CDC by health practitioners in the greater Darwin area with suspected measles. None of these had measles on laboratory investigations. No further cases of measles were notified in the NT in the 2 week period following the diagnosis of the Case 2.

Discussion

This article has described the management and outcomes of a 'measles cluster' in Darwin in June 2011. Several points warrant discussion. First, although Australia's population is generally well immunised against measles and does not have endemic measles virus transmission,[1] measles importation is still possible.[2, 4] Second, Australian adults born between 1960 and 1984 may not have immunity to measles due to (i) less exposure to wild measles and (ii) receipt of only 1 measles-containing vaccine as the 2-dose schedule for measles vaccine was only introduced in Australia from 1984 onward.[1] Recognition of this cohort and offering a 2nd measles vaccine, particularly prior to travel overseas to measles-endemic countries, may play an important role in minimising the importation of wild measles virus.[3] Third, vaccine failure, while uncommon, may also contribute to transmission of wild measles virus as illustrated by Case 1.[4, 6]

Further aspects of these measles cases are also worth noting. First, the model ED response, including infection control procedures, minimised the risk of measles transmission to

other patients and staff and reduced the scope of contact tracing. Second, the PCR test for measles virus enabled the public health response to be initiated early and effectively.[7] A further issue that arose during this public health response was the need to ensure that all health-care providers working in disease control or primary/emergency care have documented immunity to measles virus either through a history of wild measles infection or documented receipt of 2 measles-containing vaccines. Finally, the relatively high measles immunisation coverage in the NT, 89% in 0-5 yrs (90% nationally),[8] that has been achieved was a factor limiting further measles transmission.[1] However, as this 'measles cluster' demonstrates, there is no room for complacency because measles importation continues to occur related to large gatherings at sporting events such as the rugby world cup and the soccer world cup. [3]

In summary, this case study illustrates (i) an effective public health response, (ii) good coordination with primary care and acute care domains and (iii) appropriate communication to reach those at risk from exposure to measles virus.

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Editorial on measles. Everyone needs to be immune.

Vicki Krause, CDC Darwin

No further measles cases were reported in relation to these 2 cases in June 2011. However, 2 additional, unrelated cases of measles were diagnosed in Darwin, Northern Territory (NT) on 7 and 8 December with 1 having acquired the illness in Darwin from an unknown/ unrecognised source case.

A large public health response was mounted as a result of these 2 cases and to-date no further cases of measles have been diagnosed in the NT. These 2 additional cases underscore the need for vigilance and the need to consider measles when patients present with rash and a fever. Equally it points to the need to assure one's own immunity to measles in the health workplace and to check the status of patients' immunity opportunistically during clinical visits, especially if they are going travelling.

To date (as of 21 December 2011) there have been 5 cases of measles diagnosed in the NT (1 was resident in Western Australia) for 2011. For this same timeframe there have been 181 measles cases notified in Australia in 2011 with all states and territories, except Tasmania, reporting cases. This is in contrast to 69 cases of measles in 2010 and 105 in 2009 in Australia.[1]

Recently published numbers of cases of measles in Europe, where the disease is notifiable in all 53 member states, lists 26,074 cases for 2011 as of 26 October.[2] France leads the member states for this timeframe with 14,025 cases of measles notified. Whether travelling near or far - just getting on a plane with the travelling public puts one at risk of possible exposure to measles.

Assuring one's immunity to measles either from wild measles (which was common in those born before 1960) or by having 2 measles-containing vaccines a month apart (now as measles-mumps-rubella (MMR) vaccine) needs to be everyone's responsibility. Healthcare providers need to conscientiously assist the public with this undertaking.

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Measles in the NT. Information for GPs, ED staff … December 2011

The Centre for Disease Control (CDC) wishes to advise health care professionals to be alert for cases of measles. In early December 2 cases of measles were notified in the NT, one of whom was infected in Darwin. While infectious this person attended work for several days, went to the Palmerston cinema on the evening of Thursday 1 December and also spent time in Casuarina shopping centre.

Measles is highly contagious and can be spread by brief casual contact so the potential for other persons to have been infected is considered high. A media release was issued to inform the public and to warn them of the possibility of becoming ill with the need to seek medical attention. In addition, advice was given to patients to alert health care providers before arriving of the possibility that they may have measles.

Measles is characterised by a prodrome of 2-4 days of fever, coryza, cough and conjunctivitis followed by a rash, which usually starts on the face and appears while the patient is still febrile.

Those at greatest risk of measles are:

- children less than 12 months of age;
- children over 12 months of age who have not been fully vaccinated against measles (full vaccinated means having had one dose at 12 months and one at 4 years of age);
- adults born after 1960 who have never had measles or have not had 2 doses of vaccine against measles given at least 4 weeks apart.

We are asking general practitioners to please:

- Maintain a high index of suspicion for measles in those presenting with fever and rash.
- Notify the Centre for Disease Control immediately of any suspected cases (see numbers below).
- Make every effort to ensure that patients presenting with fever and rash do not sit in the general waiting area and are seen in a separate room (this room can not be used for susceptible patients/staff for 2 hours following the consultation with the suspected case).
- Do not send patients with suspected measles to pathology collection centres.
- **The testing procedure for patients with suspected measles is:**
  - ⇒ **1 urine sample (PCR measles)**
  - ⇒ **1 throat swab (PCR measles)**
  - ⇒ **1 nose swab up both nostrils (PCR measles)**

Samples should be forwarded to Royal Darwin Hospital following discussion with Centre for Disease Control on 8922 8044.

Finally, please ensure that all your staff are immune, ie they have either had measles or have had 2 doses of measles-containing vaccine at least 4 weeks apart.

Acute post-streptococcal glomerulonephritis and opportunistic trachoma screening in an Indigenous community in the Northern Territory, 2011

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Abstract

Acute post-streptococcal glomerulonephritis (APSGN) outbreaks are sporadically seen in the Northern Territory (NT), although their global prevalence is declining. We report the methodology and results of a large-scale interventional program initiated after 1 such outbreak, and provide feedback on achievements and limitations to guide further public health response field trips.

Children between the ages of 1 and 17 years were screened for signs of glomerulonephritis, skin sores, and scabies. Of the targeted population 56.8% were able to be screened, 40.5% of whom were found to have skin sores requiring treatment and 3.1% of whom had scabies. Screening for trachoma was also opportunistically undertaken on a subset of this population (those aged 4 to 17 years). Overall, 19 cases of trachoma were found 18 of 19 were aged <10 years signifying a prevalence of 5.4% of those screened. In this context we discuss the methodology behind the screening and public health response with the aim of improving access and participation in future screening programs.

Key words: Acute post-streptococcal glomerulonephritis; (APSGN); Group A streptococcus (GAS); trachoma; public health response

Introduction

Acute post-streptococcal glomerulonephritis (APSGN) is a non-suppurative complication of a skin or throat infection caused by a Group A beta-haemolytic streptococcus (GAS). The incidence of clinically detectable APSGN in children with impetigo is about 25%, with an average incubation period of 3 to 6 weeks.[1]

It is the most common cause of nephritis worldwide, with 97% of cases occurring in the undeveloped world.[2]

Only specific strains of the streptococcus bacterium are known to be nephritogenic.[3] These strains promote immune complex deposition which trigger complement activation and inflammation causing glomerular damage. Repeated cases of APSGN are a risk factor for renal disease later in life.[4]

Trachoma is caused by repeated episodes of ocular infection with the bacteria *Chlamydia trachomatis*. This causes chronic kerato-conjunctivitis and eventual scarring of the eyelid and cornea leading to blindness. Australia is the only developed nation where trachoma is found at endemic levels.[5]

Screening for active trachoma is conducted as part of the Healthy School-Aged Kids Program (HSAK) with support from the Centre for Disease Control (CDC). This program occurs annually in communities at risk of trachoma. Screening for active trachoma was combined with the public health response for APSGN in this instance due to the poor attendance rates during the recent HSAK screening.

It has been reported that Indigenous communities in the Northern Territory (NT) experience epidemics of APSGN approximately every 5-7 years in association with the circulation of a nephritogenic strain of GAS.[6] The last outbreak occurred in 2008. Previous responses, by the CDC and remote health staff, to APSGN outbreaks in remote Indigenous communities in 2004, 2005 and 2008 have achieved screening of approximately 38%, 20% and 50% of the target population respectively.[7,8,9]

This report describes the intervention methodology and discusses potential improvements for future programs.

Methods

There were 2 confirmed cases of APSGN identified from 1 community that, were not contacts of each other within a 1 week period

justifying the need for a community public health response to prevent further cases of APSGN.[3]

The following definition for a community outbreak is taken from the *Northern Territory Guidelines for Acute-Post Streptococcal Glomerulonephritis*.[3]

Community outbreak:

- 2 cases, either probable or confirmed, living in the same community; and
  onset within 1 week of each other
  at least 1 case has a low C3
  the cases are not contacts of each other

OR

- 1 confirmed case and 2 probable cases living in the same community; and
  onset within 1 month of each other
  none are contacts of each other.

The community trip was preceded by a meeting with CDC staff, local community health centre staff and the District Medical Officer. A strategy for implementing the public health response was developed.

Preparation by the clinic and the CDC involved the doctor at the clinic airing both television and radio broadcasts (translated by local Aboriginal Health Workers (AHW), which told the community what to expect during visits. This preparation aimed to improve community acceptance and involvement. The clinic also sent personalised letters to the elders of each local family re-iterating the need for screening and inviting them to participate. It was initially agreed that for the week of screening, the clinic was to be closed for all consults save emergencies to free all available staff.

Figure 1. The team at work

The 5-day interventional screening program used 4 CDC staff, 1 Maternal, Child and Youth Health staff member, 3 medical students as well as 2-4 local staff (Figures 1 and 2). Transport was provided by the local health service in the form of 2 clinic vehicles.

Figure 2. The trachoma 'screeners'

A list of required items was discussed as follows.

Supplied by local clinic stocks:

- Medications: Benzathine penicillin (Bicillin L-A) x600 prefilled syringes, scabies ointment x300 tubes, paracetamol liquid and tablets
- Injecting supplies: alcohol swabs, syringes, needles (drawing up and injecting)
- Prophylaxis equipment: adrenaline and oxygen

Brought by CDC staff:

- Trachoma screening kits: magnifiers, cotton wool balls
- Scales
- 1000 x soaps (5 bars for each household)
- Stickers, plastic sticky hand toys
- Health promotion t-shirts
- Hand washing posters (no germs on me) http://www.health.nt.gov.au/Environmental_Health/No_Germs_on_Me_Campaign/index.aspx#OtherNoGermsonMeCampaignMaterial

Population lists of children aged 12 months to less than 17 years of age, in order of surname, were downloaded from the Primary Care Information System (PCIS). Other columns of information included in the lists were Northern

Territory (NT) Hospital Registration Number (HRN), age and DOB. Additional columns for information to be collected were added to the lists including weight, scabies, sores, Bicillin L-A, date given, clean face and eyes (whether trachoma follicles were present).

The method agreed upon was to divide into 2 teams of equal expertise. Both teams consisted of an AHW for translation and identification, 1 person in charge of screening for skin lesions and data recording into the community population lists, at least 1 person able to draw up and administer intramuscular (IM) injections (2 was preferable) and 1 trachoma public health nurse.

Our visit time was aimed to be as soon as possible after the outbreak was identified, allowing time for both CDC to organise the necessary personnel to fly out but also for the clinic to inform the community of the screening program. There was an 11 day difference between the identification of the outbreak and the teams arriving and commencing screening.

All children aged 12 months to less than 17 years of age were screened for facial and peripheral oedema, skin sores and scabies, Those identified with skin sores were treated with an appropriate dose of benzathine penicillin (Bicillin L-A) according to the *Northern Territory Guidelines for Acute Post-Streptococcal Glomerulonephritis*[3] and a single dose of weight-appropriate paracetamol. Families with children affected by scabies were given tubes of permethrin 5% with verbal advice on use.

Children aged 4 to 16 years were also screened for clean or dirty faces and trachoma. Facial cleanliness is seen as 'the critical, final common pathway of all the environmental risk factors' of trachoma. Therefore all children are screened for facial cleanliness during trachoma screening. A clean face is described as absence of 'ocular or nasal discharge, flies, dirt or crusting'. Personal hygiene health promotion messages such as 'Did ya wash ya hands' and 'Clean Face-Strong Eyes' were delivered at each intervention.

Children identified as having trachoma were followed up for treatment by the CDC Trachoma Team over June / July 2011.

The message of daily face-and hand-washing was reiterated with all families treated.

Screening results for the 2 teams were documented on 1 of 2 identical population lists and collated each evening to produce a running list to be used the following day by both teams. Trachoma cases were identified and recorded separately for ease of follow-up.

Results

The community screen occurred 4-8 April 2011.

The community population lists identified a total number of 950 Indigenous children aged 12 months to less than 17 years from the Health Centre records.

The team visited every house in the community and screened a total of 540 children (56.8% of the population) over a period of 5 days.

The rate of skin sores requiring treatment was found to be 40.5% (219 cases). The majority of these were treated with Bicillin L-A. There were 9 children who were unable to be treated when screened due to known allergy to penicillin (1 case) or absconding from treatment (8 cases). These were to be followed up by local clinic staff. Many more children had healing or healed skin sores which were deemed to not require penicillin prophylaxis.

Scabies was found in 17 children within 3 separate households throughout the community (prevalence 3.1%). This is below the level suggested as endemic (>5%) according to NT Guidelines.[9] Treatment with Permethrin 5% cream was explained and initiated for all cases found.

Children were concurrently screened for active trachoma and included those from 4 to 16 years who would tolerate the procedure which required eversion of the eyelid. A total number of 540 children were screened for trachoma with 19 cases of trachoma identified. There was a prevalence of 5.4% of active trachoma in children under the age of 10. Prevalence of clean faces was 80%.

The process and organisation required for the treatment of the 19 children and all their household contacts was commenced and

antibiotic treatment was undertaken in early June.

Although it was not specifically screened for or recorded, it was noted by the screening team that ringworm seemed particularly prevalent.

A further case of APSGN was identified on 12 April following the community screen. The APSGN was diagnosed in a child that had missed being screened.

An additional 2 cases of APSGN were diagnosed at a nearby community between 10-12 April 2011, resulting in a community screen in that community.

There has only been 1 further case from this area since the community screening.

Discussion

The successful access to the community for screening lay in the initial groundwork undertaken by the local health service and because of their support during the program, particularly in the form of willing and able AHWs to accompany each team.

The local health service was pleased and satisfied with the result of 56.8% of targeted children being screened. However it was found that certain factors prevented the teams from achieving a higher degree of access.

The ability of the CDC team to have such a successful program was wholly dependent on both the community engagement beforehand and the invaluable expertise of the AHW in identifying children and families. Children can have a number of alias's and they may not know their dates of birth, making the contribution of the AHWs on the team utterly indispensable, particularly in their capacity as interpreters.

Due to large social gatherings, school holidays and irregular housing arrangements it was difficult to reach all children on our population list, despite visiting every house in the community and outlying areas. It was proposed that for the initial day of screening, a collective barbeque or swimming pool day provided by the visiting clinicians where screening could take place would aid initial penetration. This would decrease subsequent home visits done on the other days, minimizing the labor intensive and inefficient nature of repeated home visits. However this was not attempted during our program due to time frame and logistical issues. It remains a sensible and valid option for planning of any subsequent interventions.

The issues that arose with the screening methods were mostly related to privacy of the patients. Given that the age ranges to be screened included teenagers, it was very difficult to adequately screen them, particularly young girls, for skin sores without a private space in which to do so (very often unavailable). In these situations, the teams relied heavily on self-reporting after examining readily exposed areas.

A similar problem was found when giving injections ie many children over the age of around 5 years were not only frightened of having an injection, but also appeared embarrassed about having the injection in front of others. Unfortunately there were no private places in which to inject. For many children the only method of successfully giving the injection was to put the child over their Auntie's lap and have 2-3 others hold them still. This was especially traumatic for some children and for 1 child, the distress was significant enough for the treating team to refuse to give an injection at that time. She was referred to the clinic for follow up and education.

Clinicians had varying approaches to treating skin breaks, scabies and/or ringworm with some having a more preventative approach or lower threshold for penicillin treatment. A more consistent approach may be needed for future responses.

Further issues arose in the community during the screening program with regards to the safety of the CDC clinicians. A short period of inter-familial violence coincided with the screening program, resulting in a number of local residents having to be flown to Darwin for management of life-threatening injuries. There were many discussions regarding the safety of home visits during this time but local health workers were able to direct screening to uninvolved areas.

This violent episode also had an impact on the capacity of the AHWs to visit certain houses or families. Of the 3 available AHWs, 2 were members of families heavily involved in the

dispute and so screening had to be dictated by the families they could speak with and visit.

Of note, we found that the plastic Sticky Hand (developed by the Environmental Health Program) given out as a reward to children who received injections became a sought-after commodity on the toy-market and were a very good bargaining tool for children about to receive injections (particularly boys who were about to abscond). These 'high quality' rewards could be well-used in the future to attract more children to screening and treatment.

Conclusion

APSGN continues to be a sporadic yet ongoing issue for Indigenous communities in the NT requiring regular responsive screening programs.

The key to a successful screening program is based on the degree of access to the target population, made possible only by local health worker experience and co-operation.

Clinicians undertaking screening programs in the future can build on previous access techniques and lessons learned to maximise their success.

Acknowledgements

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Northern Territory Government

Immunisation Newsletter

Please circulate this newsletter to all immunisation providers in the Northern Territory.

Issue 1 - December 2011

Commonwealth changes to Maternity Immunisation Allowance and Family Tax Benefit

From 1 July 2012

- Families will need to have their children "fully immunised"* to receive the Family Tax Benefit Part A supplement. This will replace the Maternity Immunisation Allowance.
- The immunisation eligibility requirements for the Family Tax Benefit Part A supplement will continue to use the existing age-based check points for children at two and five years of age. In addition, a new immunisation check point will be introduced for one year olds.

*Fully immunised means up to date with age appropriate diphtheria, tetanus, pertussis, polio, hepatitis B, measles, mumps, rubella, Hib vaccines

From 1 July 2013

- Meningococcal C, pneumococcal and varicella vaccines will be included in the list of vaccines that are needed for a child to be considered 'fully immunised'.
- For further information please see Immunise Australia website www.immunise.health.gov.au

Reminder

Pneumococcal vaccine for children with underlying medical conditions:

Children with underlying medical conditions include:

- All premature infants with chronic lung disease
- Cystic fibrosis
- All infants born at less than 28 weeks
- Cardiac disease associated with cyanosis
- Insulin dependent diabetes
- Intracranial shunts and cochlear implants
- HIV infection
- Renal failure
- Down syndrome
- Congenital immune deficiency
- Congenital or acquired asplenia
- Haematological malignancies
- Immunosuppressive therapy or radiation therapy, when sufficient immune reconstitution for vaccine response

Children with underlying medical conditions under 6 years of age should receive Prevenar® 13 (13vPCV) at 2, 4, 6 months **with a fourth dose at 12 months and Pneumovax®23 (23vPPV) at 4-5 years of age.**

Immunisation Conference 2012 in Darwin

Public Health Association Australia (PHAA) 13th immunisation conference will be in Darwin in 19-21 June 2012. Call for abstracts are now open and closes on 7 February 2012. See the website:

www.phaa.net.au13thImmunisationConference.php

National Pertussis Campaign

The Commonwealth Government has released a national campaign to increase awareness amongst new parents about pertussis.
New parents will receive a letter from the Commonwealth advising them how to identify, protect and prevent their child from pertussis.
Please see the pertussis brochure at the Immunise Australia website:
http://www.www.immunise.health.gov.au

Flu season 2012

The influenza vaccine for the Australian 2012 influenza season contains the following three virus strains:

- A (H1N1): an A/California/7/2009 (H1N1) - like strain, 15 µg HA per dose
- A (H3N2): an A/Perth/16/2009 (H3N2) - like strain, 15 µg HA per dose
- B: a B/Brisbane/60/2008 - like strain, 15 µg HA per dose

Influenza 2012 vaccine will be available in February 2012. Although the 2012 influenza vaccine has the same 3 virus strains as 2011 it is important that vaccination occurs annually to provide adequate protection. Antibodies to influenza viruses decrease by 12 months in those previously vaccinated.

About Giving Vaccine (AGV) training for 2012

Information about AGV training and the list of 2012 workshops are now available on the NT Department of Health Immunisation website at:

www.health.nt.gov.au/Centre_for_Disease_Control/Immunisation/About_Giving_Vaccines_Course

CDC Contact Numbers

Alice Springs 8951 6907	Barkly 8962 4250
Katherine 8973 9049	East Arnhem 8987 0357
Darwin 8922 8044	

Haemophilus influenzae type b (Hib) carriage in Northern Territory children

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The full version of this paper is available at - Jacups SP, Morris PS, Leach AJ. *Haemophilus influenzae* type b carriage in Indigenous children and children attending childcare centers in the Northern Territory, Australia, spanning pre- and post-vaccine eras. Vaccine. 2011 Apr 5;29(16):3083-8.

Abstract

Background

The incidence of Hib disease in children decreased dramatically, after Haemophilus influenzae type b (Hib) conjugate vaccine was introduced to Australia in 1993. This paper reports on Hib carriage rates in Indigenous children and children attending childcare centres from the Northern Territory (NT) between 1992 and 2005.

Methods

Hib carriage was reviewed in nasopharyngeal or nasal swabs collected between 1992 and 2005; from over 2000 children (61% Indigenous) aged 0 to 6 years enrolled in 7 otitis media or carriage surveillance studies in the NT.

Results

More than 10 years after the introduction of the Hib conjugate vaccine, PRP-OMP (PedvaxHIB®), Hib carriage persists at low levels, but at a higher rate in Indigenous children (3.4%, 2003-2005) than children attending childcare centres (0.2%, 2004), in the NT.

Conclusions

This is the first Australian study to examine Hib carriage spanning the pre and post vaccination eras. Internationally there is growing concern that increasing carriage rates are the driving force behind Hib disease resurgence especially in those with higher disease burdens, such as remote Indigenous Australians. Ongoing carriage surveillance provides a sentinel warning system, which notifies public health professionals of potential invasive disease. This is valuable information, especially if Australian Indigenous children and children attending childcare centres follow current international trends with high rates of carriage preceding invasive disease - despite high vaccination rates. Changes to the vaccination schedule in the NT from PRP-OMP (PedvaxHIB®), to PRP-T (2, 4, 6, 12 months) from October 2009 may affect carriage and in time, invasive disease rates. Additional to sentinel warning systems, this work is important for national and international comparisons of the Hib carriage environment.

Keywords:Haemophilus influenzae type b (Hib); vaccine; carriage rates; Indigenous children

Introduction

Worldwide *Haemophilus influenzae* type b (Hib) is responsible for hundreds of thousands of deaths each year plus debilitating consequences to survivors.[1] The World Health Organization estimates that Hib causes 2-3 million cases of serious disease and 450 000 deaths in young children each year.[2,3] In the Northern Territory (NT) prior to the introduction of immunisation (1989-1993), rates of invasive Hib disease in all 0-4 year olds were 141/100 000 persons and in Indigenous children the rates were almost double at 278/100 000 persons.[4] Vaccination commenced in the NT in April 1993. An Australia-wide ,'catch-up' program commenced in July 1993 (April 1993 for Aboriginal children), to ensure all children under 5 years were vaccinated. Non-Indigenous children were vaccinated with HibTITER® while Indigenous children received PedvaxHIB®. From April 1993 until October 2009 however the vaccination schedule for *all* children in the NT was PedvaxHIB® at 2, 4 and 12 months; then, due to a national supply shortage, this was changed to PRP-T (2, 4, 6, 12 months) from October 2009.[5] In the NT, invasive Hib disease in children 0-5 years dropped from an average of 24 cases per year (1989 to June-1993) to 3.4 cases per year (July-1993 to 1996).[6] For

Indigenous children this represented a reduction in incidence from 278/100 000 to 37/100 000,[4] which is similar to the pre-Hib vaccine rate for Australian 0-4 year old children. The reduction in nasopharyngeal carriage, in those vaccinated and un-vaccinated (herd effect), is considered the key to success of Hib vaccination programs. The aim of this study was to collate data on Hib carriage among Indigenous and non-Indigenous children in the NT between 1992 and 2005 and to describe any risk of Hib disease, especially for those living in remote Indigenous communities.

Materials and methods

Patients

We reviewed all the nasopharyngeal or nasal swabs where Hib was isolated (indicating carriage) from children aged 0 to 6 years enrolled in ear-disease or bacterial surveillance studies with Menzies School of Health Research between 1992 and 2005. Indigenous status was formally requested for children residing in remote Indigenous communities in the NT or Pitjantjatjara Lands in South Australia. In urban childcare centre studies, Indigenous status was self reported. Informed consent was collected from participants and each study was approved by the Joint Human Research Ethics Committee of NT Department of Health and Families and Menzies School of Health Research.

Swabs and isolates

Nasopharyngeal swabs were cultured for respiratory bacterial pathogens using selective plates for *H. influenzae*[7] The Phadebact® Haemophilus test (Bactus, Sweden) was used to differentiate capsular types, as previously described.[8]

Statistical analysis

Carriage days were calculated by subtracting the first Hib positive date from the most recent date, plus 1 day. Where only 1 swab was positive, carriage was reported as having persisted for 1day only. Epidemiological tables were produced using STATA version 10.0 (Stata Corp, College Station, TX, USA).

Results

Carriage prevalence in Indigenous children was observed to decrease from 24% (median duration 2.9 months) (Table, study 1-a) during the pre-vaccination era to 3.4% between 2003 and 2005 ($p<0.0001$) (Figure), with carriage incidence echoing this (Table.) In contrast, the incidence of Hib carriage among non-Indigenous children in the post-vaccination era (2004) was approximately 5 times lower (Table, study 7). *Haemophilus influenzae* type b carriage results from cross sectional studies 6 and 7, with few Indigenous children, indicate a decrease in point prevalence, 0.7% (2001) to 0.2% (2004), (Table, study 6 & 7). This compares with the cross sectional study involving only Indigenous participants from 27 communities across the NT where point prevalence was 0.7% (Table, study 5).

Discussion

It has been over 12 years since the introduction of Hib vaccination, yet carriage among Indigenous children in the NT persists, despite high vaccination coverage in NT (98.5% fully immunised).[9] The disparity in Hib colonization rates between Indigenous and non-Indigenous children is associated with disproportionate invasive Hib disease rates and the higher number of reported deaths and vaccine failures in Indigenous children.[10] Invasive Hib disease is occurring in both vaccinated and non-vaccinated Australians; of the 532 cases between 1993 and 2000, 74 were recognised as true vaccine failures.[11] For Indigenous Australians, 41 cases of invasive Hib disease were reported and of these 20 had received no immunisations and 9 of 41 (22%) were defined as true vaccine failures- all 9 had received at least 2 doses of PRP-OMP, and 5 had received 3 doses.[11] In the light of vaccine failures, our findings of persisting Hib carriage highlight the need for ongoing vigilance.

Our data reveal that the age of colonisation in Indigenous children was particularly young; 3 studies from 1996-2005 had median Hib colonisation ages of 5.5, 10 and 16 months respectively, in a well vaccinated population,[9] suggesting an inadequate vaccine response or antibody levels low enough to permit colonisation. Vaccine failures during the late 1990s in the United Kingdom (UK) report a median age of invasive Hib disease of 23 months.[12] In the UK, the point prevalence of Hib carriage in fully vaccinated (3 doses of PRP-T) school-aged (6-16 years) children in 2005 was 4.2%,[13] with a very low prevalence (1.3%) similarly reported among fully vaccinated pre-school aged children during years 1995-6.[14]

Figure. Hib prevalence in Indigenous children and children attending childcare centres, NT, 1992-2005

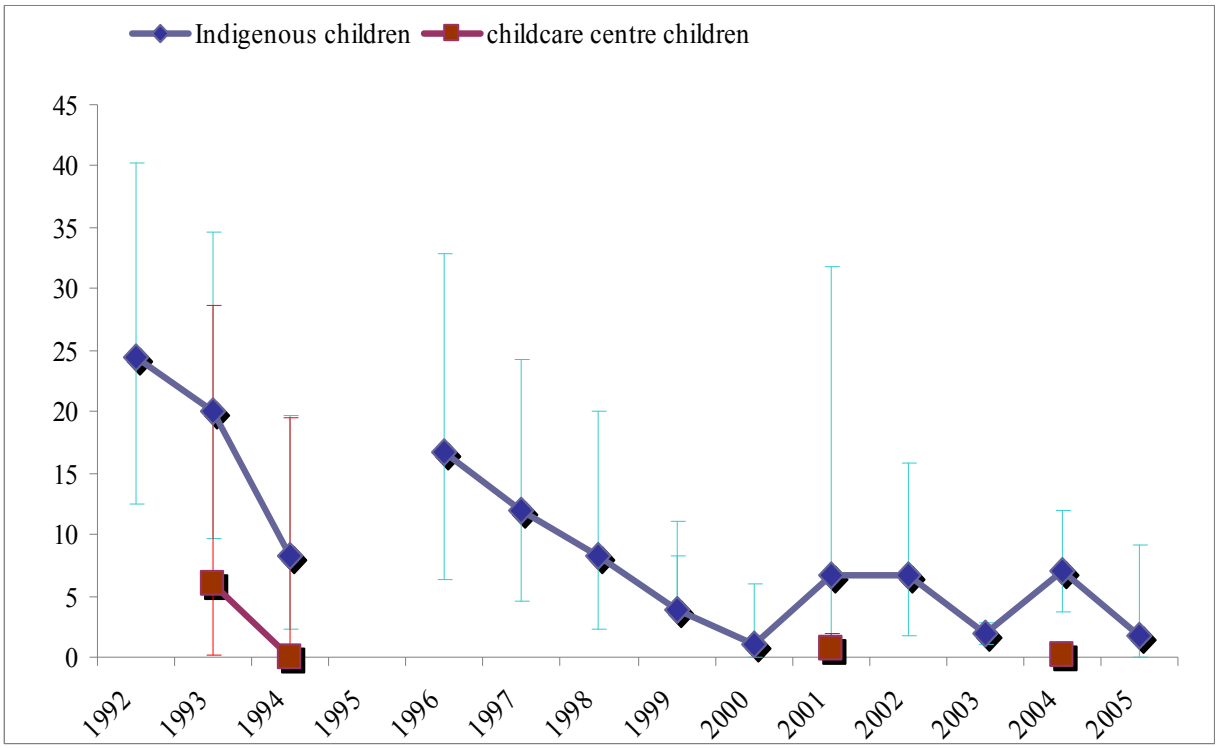

Table. Nasopharyngeal carriage with Hib, 1992-2005, NT

Study number	1- a	1-b	1-c	2	3	4	5	6	7	P
Number(%) of swabs HIB +ve	18 (9%)	3 (4%)	13 (7.1%)	27 (2.6%)	15 (1.6%)	15 (2.3%)	10 (2%)	3 (0.7%)	1 (0.2%)	
Number of children HIB carriers	10	1	4	19	10	11	10	3	1	
Hib prevalence	24%	6%	20%	15%	10.30%	3.40%	1.60%	0.70%	0.20%	<0.001*
Hib carriage annual incidence/100 000	16 260	3922	13 333	2533	2575	1146	1553	658	218	
Median duration of carriage (months) (range)	2.9 (0-7.2)	2.5	3.0 (1.4-7.0)	3.9 (0-21.9)	0.3 (0-1.9)	0.03 (0-0.5)	0.03	0.03	0.03	
Median duration of carriage (days) (range)	78 (1-219)	77	57 (42-212)	17 (1-666)	8 (1-58)	1 (1-16)	1	1	1	
Median age (months) of first colonisation (range)	3.5 (1.0-5.5)	6.9	4.2 (3.6-5.4)	5.5 (1-34)	10 (2.9-25.8)	15.9 (7.7-72.6)	18 (7-28.2)	29 (26.7-38.4)	Age data unavailable	
Median age (months) of last colonisation (range)	6.2 (1-10.1)	9.4	6.4 (5-11.6)	6.6 (1.3-34)	11 (3.1-25.8)	15.9 (7.9-72.6)	18 (7-28.2)	29 (26.7-38.4)	Age data unavailable	

*Chi square trend analysis for proportions (χ^2= 142.171, p < 0.0001)

Previous studies have demonstrated that upon introduction of Hib conjugate vaccine nasopharyngeal Hib colonisation no longer occurred.[2] While others, mostly in Indigenous populations report lower levels of Hib carriage.[2,15,16] For the Australian Indigenous population, duration of protection using the conjugate Hib vaccine is unknown.[17] Serologic responses to PRP-OMP have been compared in 2 populations in Australia, NT rural Indigenous children with non-Indigenous children from Sydney. Guthridge et al reports that the NT Indigenous cohort had consistently lower PRP-specific IgG levels than their non-Indigenous counterparts from Sydney (1.98 verses 6.04 µg/mL p=0.002). Only 29.2% of those in the Indigenous group had antibody levels >5 µg/mL 1 month post 3rd dose; markedly lower than the non-Indigenous group 59.2%.[10] The current threshold concentration for protection from invasive Hib disease is >1µg/mL[15] PRP-specific IgG levels; however, the findings of Fernandez et al indicate that a higher level may be required for protection against Hib carriage, and they suggest a level of ≥5µg/mL become the standard.[15] When these newly suggested figures are applied to the Guthridge et al results above, a concentration of 1.98 µg/mL from the NT Indigenous cohort children versus non-Indigenous children at 6.04 µg/mL implies inadequate protection for the Indigenous children.[10] Thus, lower responses to vaccination could explain the persistence of Hib colonisation in Indigenous populations.

It has been speculated that a reduction in carriage rates in adults and older children in a vaccinated population results in a decrease in natural boosting, causing serum antibody titres to wane in the absence of further doses of vaccine. In countries which schedule a Hib booster at 18 months of age, no increase in vaccine failures has been reported and antibody levels remain high at 10 years of age.[12] Several European countries now include a Hib booster dose in the second year of life[18] including the UK, which now schedules 4 doses (2, 3 and 4 month plus booster at 12 months).[13]

This study reports that Hib carriage persists in Indigenous children but is rarely detected in non-Indigenous children in the NT. We are unable to determine if carriage persists in this population as a result of poor timeliness (despite good overall vaccination rates)[19,20] or a poor immune response to Hib vaccine. Further immunogenicity studies may be warranted to determine if booster Hib vaccine doses will improve PRP-specific IgG levels in Australian Indigenous children. Since the schedule has recently changed to PRP-T (2, 4, 6, 12 months), vigilance in surveillance should be continued, to ensure carriage rates are not increasing under the radar.

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19 - 21 June 2012
Darwin Convention Centre

Immunisation: New Frontiers

Abstracts submission is now open and closes on 7 February 2012.

http://www.phaa.net.au/documents/Immunisation2012CallForAbstracts.pdf

The program has been designed to provide lively and productive discussions and contributions from all professionals engaged in immunisation from laboratory bench to clinic, from academia to the service provision coalface. We invite you to submit abstracts under the following session themes:

1. Influenza
2. Human papillomavirus
3. Rotavirus
4. Pertussis
5. Varicella and other viruses
6. Pneumococcal and other bacterial diseases
7. Vaccine Preventable Diseases in Aboriginal and Torres Strait Islander peoples'
8. Service delivery
9. Social science of immunisation (aka knowledge, attitudes, and practice)
10. Vaccine safety
11. Coverage and data linkage

15 food safety tips for the Christmas season (or any time of year)

Tracy Ward, Environmental Health, Darwin

With Christmas and New Year upon us, many people are planning parties to celebrate with family and friends. Christmas is a great time to get together but it can also increase the likelihood of food borne illness. Following a few simple rules will help to minimise the risk of getting sick.

1. Before preparing foods and between handling raw meat or raw chicken wash hands thoroughly with soap and warm water and dry thoroughly.
2. Avoid keeping food in the temperature danger zone between 5°C and 60°C where food poisoning bacteria grow best.
3. Keep hot foods steaming hot over 60°C and keep cold foods refrigerated at or below 5°C.
4. Ready to eat food should always be defrosted in the fridge or microwave, never on the bench top, unless the manufacturer recommends that you do so.
5. You can defrost the turkey in the fridge, or ask your butcher to defrost it in the cool room but make sure it is completely defrosted in the centre before cooking. Because stuffing slows down cooking and cooling, it is best cooked separately.
6. Before preparing food for Christmas make sure that there is enough room in the fridge to keep cold food at or less than 5°C.
7. If there is not enough room in the fridge, remember that soft drinks and alcohol, jams, pickles and other acidic condiments do not require refrigeration to remain safe. Drinks can be kept cold in an esky with ice.
8. Prepare foods as close as possible to eating time.
9. Use separate cutting boards and utensils for raw meats and poultry and ready to eat foods.
10. Cook foods properly. All rolled & stuffed roasts, poultry, sausages, mince dishes and liver need to be fully cooked. Steaks, chops and solid pieces of meat can be eaten rare.
11. If you cook large amounts of food in advance, divide it into smaller portions or shallow containers, cover and place in fridge or freezer. Make sure there is good air circulation around the containers.
12. Refrigerate leftovers immediately after the meal.
13. Always store perishable leftovers in the fridge and use them up within 2 to 3 days.
14. When reheating food ensure that it is steaming hot all the way through (at least 75°C).
15. Your Christmas ham will keep several weeks with proper handling by removing it from its plastic wrap, covering with clean cloth soaked in water and vinegar so it doesn't dry out, following any instructions on the packaging and storing it in the fridge below 5°C. Reduced salt hams are now becoming popular but will not last as long as conventional hams so follow instructions on the packaging.

More information is available from the Food Safety Information Council at http://www.foodsafety.asn.au/factsheets/

The Food Safety Information Council is a partnership of government agencies, industry and professional groups with the objective of educating consumers about safe food practices.

The Environmental Health Program wishes you a safe and Merry Christmas.

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Northern Territory Government

DEPARTMENT OF HEALTH

ENVIRONMENTAL HEALTH FACT SHEET NO. 119

FLY CONTROL

fact sheet

The house fly (*Musca domestica*) and the blue blow fly (*Chrysomyia megacephala*) are the most common problem domestic flies in the Top End. The warm climate means that house and blow flies can be a problem year round, although there is a tendency for fly numbers to increase during the wet season.

In developing countries domestic flies are recognised as carriers of a number of severe communicable diseases including amoebic dysentery, cholera and typhoid fever. In Australia flies can transmit germs that cause diseases such as gastroenteritis.

Flies pick up bacteria while they are feeding. They are attracted to warm, moist locations with a good supply of organic matter including rubbish dumps, bins, animal and human excreta, decaying food and rotting carcases. Bacteria stick to their hairy legs from which they can be transferred to food and other surfaces that the flies touch. Mature flies use saliva to liquefy their food which can also result in the transference of pathogens collected from feeding on organic matter.

Where do they breed?

Houseflies usually lay their eggs in rotting vegetation or food found in rubbish bins, piles of lawn clippings, compost heaps or other organic matter. Blowflies tend to lay their eggs in rotting carcases, meat products and garbage with a high protein content. A female housefly can lay 4 to 6 batches of about 20 eggs each over a period of a few days. In normal Darwin conditions, it takes about 9 -10 days for a house fly to develop from egg to adult. Blow fly maggots take only 4 days to develop from eggs to the wandering stage. House and blow fly maggots are commonly found in wheelie bins in Darwin.

How do I stop them from breeding?

- Use a kitchen tidy bin with a sealable lid and a liner for temporary storage. Keep the tidy bin inside a screened house or in a screened area to prevent fly entry. Install a pest strip in the tidy.
- Wash out all food containers such as milk cartons, meat trays, pet food tins and similar containers before placing the washed non-recyclable items in the kitchen tidy bin. Reduce all fluids in garbage as much as possible.
- Bury or collect and securely wrap or bag all uneaten pet food, including old bones, as well as any animal faeces and place in a wheelie bin.
- Wrap food scraps securely in newspaper or seal them in plastic bags prior to depositing in a kitchen tidy bin. Alternatively, keep wrapped meat and seafood scraps in the freezer until bin collection day. Double bagging of garbage has been found to reduce blow fly production by 600% compared with non-bagged garbage (Whelan, 2001).
- Tie all kitchen tidy bin liners with a knot before depositing the contents in the wheelie bins. This will prevent fly entry and more importantly prevent fly or maggot exit.

DEPARTMENT OF HEALTH

fact sheet

- Ensure the kitchen tidy bin and recycling bin/bag contents are placed in the appropriate wheelie bin daily and at least the day of wheelie bin collection.
- Ensure that the lid of your wheelie bin is closed at all times.
- Ensure wheelie bins are placed out in time for scheduled collections. A missed collection can lead to fly breeding.
- Install a pest strip such as Binkill® inside a wheelie bin and replace the strips every 2 months. These strips can kill adult flies in half an hour and maggots within a few hours. They are available at local supermarkets.
- Wash all bins out frequently and allow drying out.
- If maggot problems in the bins persist, then spray the bottom of a cleaned out bin and the under side of the lid and internal sides with a residual insecticide such as permethrin, deltamethrin, bifenthrin or lambda-cyhalothrin.
- Make sure your compost bin seals well to keep flies out.
- If you have fowls or caged birds keep their yards and cages clean at all times.
- Spread lawn clippings thinly on the garden. Don't leave them in heaps.
- Apply blood and bone and animal manures thinly on your garden and dig it in.

References

Whelan, P. 2001. Guidelines to prevent fly breeding in domestic situations in the Top End of the Northern Territory. *The Northern Territory Disease Control Bulletin Vol. 8, No 1*

FOR COPIES OF THE FACT SHEET SEE:
http://www.health.nt.gov.au/publications/environmental_health_publications/index.aspx#factsheets

FURTHER INFORMATION
CONTACT ENVIRONMENTAL HEALTH ON 1800 095 646 OR YOUR LOCAL OFFICE
Email: envirohealth@nt.gov.au

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Northern Territory Government

DEPARTMENT OF HEALTH

media release

Community can help eliminate dengue mosquitoes in Tennant Creek

Wednesday 30 November 2011

The Department of Health is seeking the Tennant Creek community's assistance in eliminating an infestation of the potentially dengue fever carrying mosquito *Aedes aegypti* recently detected in the town.

An initial control effort by the medical entomology team will include a property by property program on both public and private land to treat all water holding receptacles with a residual pyrethrum-like insecticide that kills wrigglers and prevents adult reinfestation. .

"The team will also conduct an extensive survey to establish the full extent of the infestation," NT senior medical entomologist Peter Whelan said today.

"It is possible that the elimination of this mosquito will require an intensive and ongoing program for up to two years.

"The Department is seeking the cooperation of residents to help stop the spread of mosquitoes by emptying and cleaning all water containers, and taking other preventive measures (*see below*)."

Mr Whelan stressed that there is no present dengue transmission in Tennant Creek or in any other part of the NT as there is no one identified in Tennant Creek with dengue fever who would introduce the virus.

"The current risk of dengue transmission in Tennant Creek is extremely remote. It would require someone with dengue fever coming here from, for instance overseas and being bitten by one of these mosquitoes. This infected mosquito would have to live to bite another person to transmit the dengue virus.

"We are putting every effort into eliminating the mosquito to avoid that possibility. Initial detection of the presence of mosquitoes that could carry the disease was in a special egg trap and a preliminary follow up survey on 24-25 November.

"We identified *Aedes aegypti* adults and larvae in a wide area that includes at least the northern and central half of the town."

The dengue mosquito breeds in all types of water-holding receptacles, including old tyres, pot plant bases, plastic sheeting, disused tanks or ponds, unsealed rain water tanks - anything that can hold rain or sprinkler water.

This is the first establishment of this mosquito in the NT since the importations that were eliminated from Tennant Creek in 2006 and from Groote Eylandt in 2008.

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"It is significant because this species is a recognised vector of the dengue virus and is the main vector involved in the almost annual transmission of dengue disease that occurs in Queensland," Mr Whelan explained.

"It is extremely important that this mosquito species does not become permanently established in Tennant Creek or other places in the NT. "

Residents are asked to assist in stopping the spread immediately by:

- emptying out water from all potential breeding sites (water receptacle, refuse etc) in backyards or inside.
- cleaning up your backyard breeding or inside house sites and spray household residual insecticide in any mosquito breeding receptacle.
- turning over all water holding receptacles and leaving them outside for treatment by the control teams.
- advising the control teams of all receptacles that have held water or wrigglers.
- not transporting any previously water filled receptacles from Tennant Creek to other NT centres.
- wiping out all emptied receptacles with wrigglers with an undiluted solution of household bleach to kill any eggs present.
- treating all vehicles inside the cabin or protected carrying sections with an aerosol knock down spray each morning before moving the vehicle in Tennant Creek or from Tennant Creek to another town or location.
- reporting any infestations of wrigglers in receptacles that can't be emptied or day biting mosquitoes to the toll free during business hours hotline (1-800-008-002).
- assisting the elimination teams by allowing access to your yard when they visit to check receptacles or leave mosquito traps in yards.
- leaving a contact number in your letter box or on the hot line and advising of suitable times for inspections over the initial control period from now until Christmas.

Media Contact: Bridget Wild 899 92818 or 0401 116 203

Abstracts from peer reviewed published articles related to the Northern Territory

Acute post-streptococcal glomerulonephritis in the Northern Territory of Australia: A review of 16 years data and comparison with the literature.

Catherine S. Marshall, Allen C. Cheng, Peter G. Markey, Rebecca J. Towers, Leisha J. Richardson, Peter K. Fagan, Lesley Scott, Vicki L. Krause and Bart J. Currie

***Am J Trop Med Hyg* 2011; 5(4):703–710.**

Data relating to acute post-streptococcal glomerulonephritis (APSGN) from the notifiable diseases surveillance system in the Northern Territory of Australia was extracted and analyzed. Isolates of Streptococcus pyogenes from confirmed cases were emm sequence typed. From 1991 to July 2008, there were 415 confirmed cases and 23 probable cases of APSGN notified. Four hundred fifteen (94.7%) of these were Indigenous Australians and 428 (97.7%) were people living in remote or very remote locations. The median age of cases was 7 years (range 0–54). The incidence of confirmed cases was 12.5/100,000 person-years, with an incidence in Indigenous Australian children younger than 15 years of age of 94.3 cases/100,000 person-years. The overall rate ratio of confirmed cases in Indigenous Australians to non-Indigenous Australians was 53.6 (95% confidence interval 32.6–94.8). Outbreaks of disease across multiple communities occurred in 1995 (N = 68), 2000 (N = 55), and 2005 (N = 87 [confirmed cases]). Various emm types of S. pyogenes were isolated from cases of APSGN including some types not previously recognized to be nephritogenic. The widespread outbreak in 2005 was caused by emm 55.0 S. pyogenes . Acute post-streptococcal glomerulonephritis continues to occur in remote Indigenous communities in Australia at rates comparable to or higher than those estimated in developing countries. Improvements in preventative and outbreak control strategies are needed.

Assessment and management of latent tuberculosis infection in a refugee population in the Northern Territory.

James M Trauer and Vicki L Krause

***Med J Aust* 2011; 194 (11):579-582.**

Objective: To assess the prevalence of latent tuberculosis infection (LTBI) in recently arrived refugees in the Northern Territory and to obtain comprehensive data for rates of treatment acceptance and completion for this condition.
Design, setting and participants: Prospective data collection and follow-up of all 471 newly arrived refugees seen at the Centre for Disease Control, NT refugee health clinic from February 2006 to January 2009.
Main outcome measures: Rates of LTBI determined by tuberculin skin testing; subsequent assessment and treatment compared with local protocols.
Results: 458 of 465 eligible refugees were adequately assessed for LTBI, of whom 146 (31.9%) were diagnosed with LTBI. Older age, male sex and World Health Organization Eastern Mediterranean region of birth were associated with increased prevalences of LTBI. Of the refugees diagnosed with LTBI, 10 failed to attend for follow-up and 15 were not offered treatment. Isoniazid therapy was accepted by 93 of 121 refugees (76.9%), and 41 of these (44.1%) completed treatment. The most common reasons for discontinuation of therapy were medication-related side effects (most often gastrointestinal) and loss to follow-up. Increasing age was associated with failure to complete treatment.
Conclusion: Outcomes of assessment and treatment for LTBI in newly arrived refugees in the NT are comparable to those for other target groups screened in developed countries. Loss to follow-up caused significant attrition in numbers, but complete data were obtained for a large proportion of eligible refugees. Most refugees who are offered treatment for LTBI accept, but less than half complete treatment.

Feasibility of latent tuberculosis infection diagnosis by interferon-gamma release assay remote from testing facilities

James M Trauer, Krispin M Hajkowicz, Kevin G Freeman and Vicki L Krause

***Commun Dis Intell* 2011;35(2):168–171.**

Although the tuberculin skin test (TST) has been the mainstay of the diagnosis of latent tuberculosis infection (LTBI) for many decades, interferon-gamma release assays (IGRAs) are gaining acceptance and are more specific for this diagnosis. The characteristics of one such IGRA, the QuantiFERON®-TB Gold Whole Blood

In-Tube, make it feasible for use in a remote setting. This study performed 62 IGRAs with this test on individuals testing positive by TST, in a clinical setting over 3,000 km from the testing laboratory. Of these, 42 patients (68%) recorded negative results, 19 (31%) were positive, with only 1 result (2%) indeterminate. Negative, and therefore discordant in this study, test results were more common in those known to have been previously vaccinated with bacille Calmette-Guérin. These results are consistent with other reports, indicating that this approach to testing is logistically feasible, and has the potential to complement LTBI screening to assist tuberculosis control programs in settings remote from the testing laboratory.

The epidemiology of community acquired bacteremic pneumonia, due to *Streptococcus pneumoniae*, in the Top End of the Northern Territory, Australia-Over 22 years.

Susan P Jacups and Allen C Cheng

Vaccine. 29(33): 5386-5392.

Background: Diseases caused by Streptococcus pneumoniae continue to cause substantial morbidity and mortality throughout the world. Furthermore, detrimental outcomes are more pronounced in some populations- such as those living in third world poverty, and Indigenous people who live in developed nations.
Methods: This study describes the epidemiology of blood culture positive S. pneumoniae community-acquired pneumonia (CAP) in the Top End of the Northern Territory. Demographics, Indigenous status, medical risk factors, serotype and outcomes were collected from adults presenting to Royal Darwin Hospital with blood culture positive S. pneumoniae CAP, from 1987-2008.
Results: Reported were 205 cases; median age 40 years. The average overall incidence rate ratio was 10.3 for Indigenous adults compared with non-Indigenous adults. There was no statistical difference between incidence rates pre and post-23-valent pneumococcal polysaccharide vaccine (23vPPV) introduction. Serotypes in presenting cases were predominantly (84.7%) 23vPPV types. The whole-population logistic regression model identified significant adjusted relative risks: 95% CI, for; age 45 and older 1.6: 1.1, 2.2, Indigenous 5.9: 3.7, 9.5, diabetes 2.3: 1.6, 3.3, excess alcohol 4.8: 2.8, 8.3, smoking 2.7: 1.9, 3.7 with Indigenous + excess alcohol 18.5: 17.3, 19.7 as predictive for bacteremic S. pneumoniae CAP presentation.
Conclusions: These results suggest that the national 23vPPV program appears to be under-utilized. An integrated Public Health strategy, vigorously targeting vaccination of Indigenous adolescents, before substances such as alcohol and smoking are habitual, could reduce the burden of pneumococcal disease in this population.

Case Report: West Nile virus (Kunjin subtype) disease in the Northern Territory of Australia—A case of encephalitis and review of all reported cases.

Timothy J. Gray, James N. Burrow, Peter G. Markey, Peter I. Whelan, Justin Jackson, David W. Smith and Bart J. Currie

***Am J Trop Med Hyg* 2011;85(5):952–956.**

West Nile virus Kunjin subtype (WNV/KUNV) is enzootic across the tropical north of Australia, with epizootic spread into other jurisdictions. The clinical spectrum of illness in humans is poorly described. We report a clinical case of WNV/KUNV encephalitis and performed a retrospective chart audit of all cases of WNV/KUNV notified in the Northern Territory from 1992 to 2010. Thirteen cases of WNV/KUNV disease were identified; case notes were available for 10 of these presentations. Six of these patients had confirmed infection and presented with neuroinvasive illness, whereas the other four suspect cases comprised three cases with arthralgia, myalgia, and/or rash and one case with fever alone. On the available evidence, WNV/KUNV is of lower virulence

compared with the New York 1999 strain. Difficulties in serological diagnosis, especially when paired acute and convalescent sera are not available, may adversely impact the accuracy of the epidemiological and clinical understanding of this virus.

Differential effects of pandemic (H1N1) 2009 on remote and Indigenous groups, Northern Territory, Australia, 2009.

James McCracken Trauer, Karen Louise Laurie, Joseph McDonnell, Anne Kelso, and Peter Gregory Markey

***Emerg Infect Dis* 2011;17(9).**

Pandemic (H1N1) 2009 influenza spread through the Northern Territory, Australia, during June–August 2009. We performed 2 cross-sectional serologic surveys on specimens from Northern Territory residents, with 445 specimens obtained prepandemic and 1,689 specimens postpandemic. Antibody titers were determined by hemagglutination inhibition against reference virus A/California/7/2009 on serum samples collected opportunistically from outpatients. All specimens had data for patients' gender, age, and address, with patients' indigenous status determined for 94.1%. Protective immunity (titer >40) was present in 7.6% (95% confidence interval [CI] 5.2%–10.1%) of prepandemic specimens and 19.5% (95% CI 17.6%–21.4%) of postpandemic specimens, giving a population-standardized attack rate of 14.9% (95% CI 11.0%–18.9%). Prepandemic proportion of immune persons was greater with increasing age but did not differ by other demographic characteristics. Postpandemic proportion of immune persons was greater in younger groups and around double in indigenous persons. Postpandemic proportion immune was geographically heterogeneous, particularly among remote-living and indigenous groups.

Habitat modification for mosquito control in the Ilparpa swamp, Northern Territory, Australia.

Susan Jacups, Nina Kurucz, Raelene Whitters and Peter Whelan

***J Vector Ecol* 2011;36(2):292-9.**

Habitat modification is an established method of effective long-term mosquito management particularly in salt-marsh environments. It is especially pertinent when mosquitoes are known vectors of life-threatening disease and their larval breeding habitat is in close proximity to residential areas. The Ilparpa Swamp is located less than 10 km from Alice Springs, Northern Territory. Wet season rainfall, often followed by effluent discharges to the swamp from the adjacent sewage treatment plant create ideal sites for the immature stages of the common banded mosquito *Culex annulirostris* (Skuse). A major vector of Murray Valley encephalitis (MVEV) and Kunjin (KUNV) viruses. Subsequent to increases in notifications of MVEV disease cases in 2000 and 2001, a drainage system was established in the Ilparpa Swamp in early 2002. This paper evaluates the drainage intervention effects. Results indicate a significant reduction in mosquito numbers following habitat modification which remain low. There have been no seroconversions in sentinel chickens to MVEV or KUNV and no human infections from these viruses in the Alice Springs urban region since the drains were completed. Habitat modification has successfully reduced mosquito numbers and minimized the risk for mosquito-borne disease to residents in Alice Springs urban and surrounding areas which has never before been documented in Australia.

Bronchiectasis is associated with Human T-Lymphotropic Virus 1 infection in an Indigenous Australian population.

Lloyd Einsiedel, Liselle Fernandes, Tim Spelman, Daniel Steinfort and Eduardo Gotuzzo

***Clin Infect Dis 2012 Jan:54(1):43-50. Epub 2011* Nov 17.**

Background: Recent studies suggest that infection with human T-lymphotropic virus 1 (HTLV-1) might be associated with bronchiectasis among Indigenous Australians. The present study compared the clinical characteristics and outcomes of bronchiectasis in this population, according to HTLV-1 serologic status.

Methods: We performed a retrospective cohort study of Indigenous adults with bronchiectasis and known HTLV-1 serologic status admitted to Alice Springs Hospital, central Australia, from January 2000 through December 2006.

Results: Among 89 Indigenous adults whose HTLV-1 serologic status was confirmed, 52

(58.4%) were HTLV-1 seropositive. Differences between HTLV-1–seropositive and HTLV-1–seronegative groups were apparent in childhood presentations and adult outcomes. Among adults, an increasing number of bronchiectatic lobes (univariable odds ratio [OR], 1.51; 95% confidence interval [CI]; 1.03–2.20; P 5 .033) and the presence of ground-glass opacities at chest high-resolution computed tomography (univariable OR, 8.54; 95% CI, 1.04–70.03; P 5 .046) predicted HTLV-1 infection. Cor pumonale (HTLV-1–positive group, 10/52; HTLV-1–negative group, 1/37; P 5 .023) was more frequent among HTLV-1–seropositive adults, who also experienced a higher disease-specific mortality (univariable OR, 5.78; 95% CI, 1.17–26.75; P 5 .028). Only HTLV-1–seropositive patients were admitted specifically for the treatment of infected skin lesions, and this finding predicted death (multivariableOR, 6.77; 95% CI, 1.46–31.34; P5.014). Overall mortality was high; 34.2% of the cohort died at a median age of 42.5 years.

Conclusions: HTLV-1 infection contributes to the risk of developing bronchiectasis and worsens outcomes among Indigenous Australians.

The impact of sexually transmissible infection programs in remote Aboriginal communities in Australia: a systematic review

Rebecca Guy, James S. Ward, Kirsty S. Smith, Jiunn-Yih Su, Raelin Huang, Annie Tangey, Steven Skov, Alice Rumbold, Bronwyn Silver, Basil Donovan and John M. Kaldor

***Sex Health*. Nov 2011. Online.**
http://dx.doi.org/10.1071/SH11074

Objective: To systematically review evaluations of the impact of sexually transmissible infection (STI) programs delivered by primary health care services in remote Aboriginal communities.

Methods: PubMed, Google Scholar, InfoNet, Cochrane Controlled Trials Register, Australian New Zealand Clinical Trial Registry, conference proceedings and bulletins were searched to April 2011 using variations of the terms 'Aboriginal', 'programs' and 'STI'. The primary outcome of interest in the review was the change in bacterial STI infection prevalence in the target age group assessed through cross-sectional screening studies over a 5-year period or more. The characteristics of the primary health care service, STI programs and other clinical service outcomes were also described.

Results: Twelve reports described four distinct STI programs in remote communities and their impact on STI prevalence. In the Anangu Pitjantjatjara Yankunytjatjara (APY) lands of northern South Australia, there was a reduction in the age-adjusted chlamydia and gonorrhoea prevalence by 58% and 67%, respectively (1996–2003). In the Tiwi Islands of Northern Territory (NT), chlamydia and gonorrhoea positivity decreased by 94% and 34%, respectively (2002–2005). In the Ngaanyatjarra Lands of Western Australia, crude chlamydia and gonorrhoea prevalence decreased by 36% and 48%, respectively (2001–2005), and in the central Australian region of NT, there was no sustained decline in crude prevalence (2001–2005).

Conclusion: In three of the four programs, there was some evidence that clinical best practice and well coordinated sexual health programs can reduce STI prevalence in remote Aboriginal communities.

The health of newly arrived refugees to the Top End of Australia: results of a clinical audit at the Darwin Refugee Health Service.

Vanessa Johnston, Le Smith and Heather Roydhouse

Aust J Prim Health. 9 December 2011. Online. http://dx.doi.org/10.1071/PY11065

Accurate data on the health of refugees in primary care is vital to inform clinical practice, monitor disease prevalence, influence policy and promote coordination. We undertook a retrospective clinical audit of newly arrived refugees attending the Darwin refugee primary health service in its first 12months of operation. Data were collected from the clinic files of refugee patients who attended for their initial health assessment from 1 July 2009 to 30 June 2010 and were analysed descriptively. Among 187 refugees who attended in 2009–2010, ~60% were from Asia and 42% were female. The most common diagnoses confirmed by testing were vitamin D deficiency (23%), hepatitis B carrier status (22%), tuberculosis infection (18%), schistosomiasis (17%) and anaemia (17%). The most common documented health conditions

recorded by the GPs were vitamin D deficiency or insufficiency (66%), followed by schistosomiasis (24%) and dental disease (23%). This clinical audit adds to a limited evidence base suggesting a high prevalence of infectious disease, nutrient deficiency and dental disease among refugees arriving to Australia. GPs involved in the care of refugees must be aware of the epidemiology of disease in this group, as some diseases are rare among the general Australian population. Our results also highlight the ongoing need for advocacy to address service constraints such as limited public dental access for this population.

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Zoonoses 2012

"Bringing Docs and Vets together"

…an ASID Workshop on Emerging Issues in Animal and Human Infections in Australia

With support from the AVA

The Australasian Society for Infectious Diseases, with support from the Australian Veterinary Association, is holding a 2 day meeting **Friday-Saturday July 27/28 2012**. The venue is the **Eastern Avenue Complex** at the **University of Sydney**.

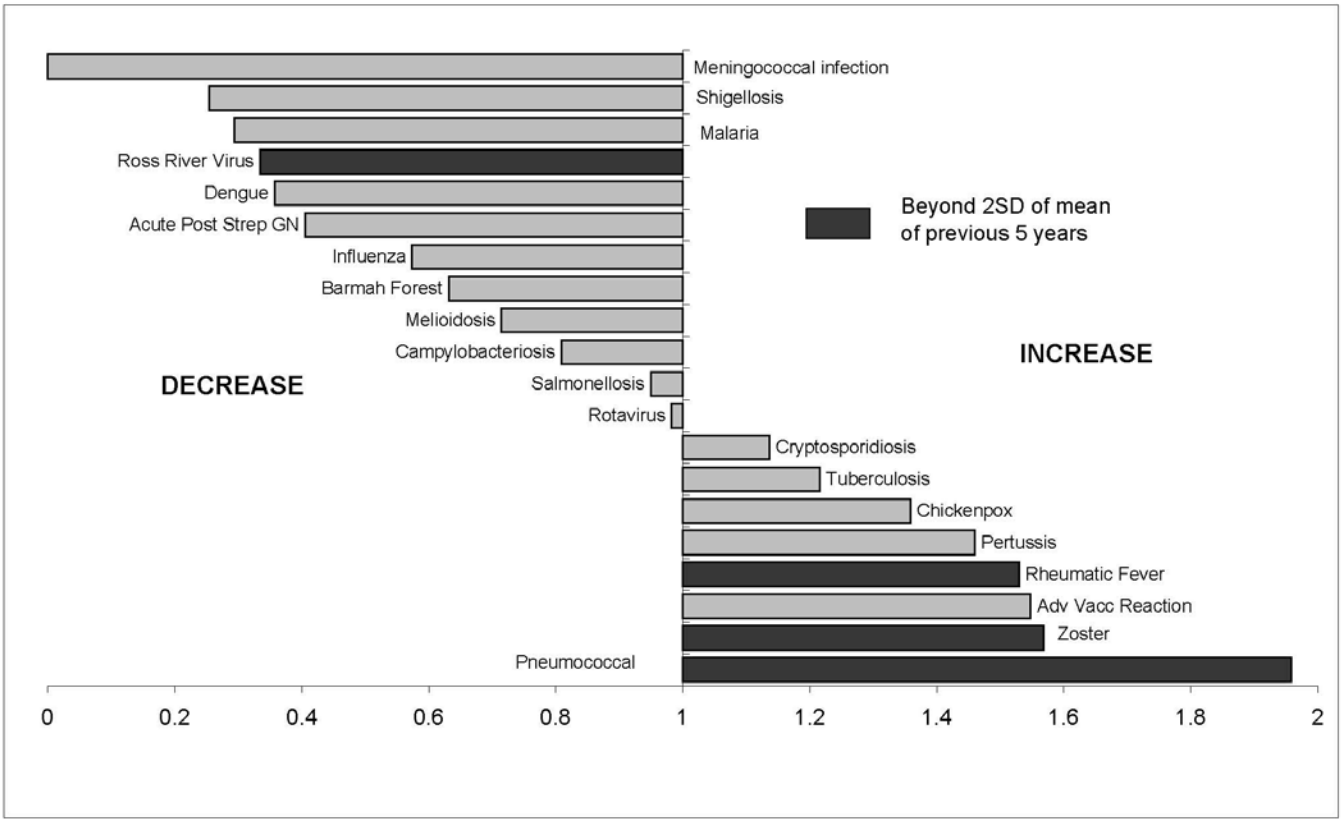
The meeting aims to bring together veterinarians, medical practitioners and laboratory scientists around the theme of zoonoses in Australia. Topics covered include: hazards of human and animal interaction including OH&S issues for veterinarians, wildlife officers and animal carers; emerging antimicrobial resistance; food-borne zoonoses; hazards from the environment following natural disasters such as floods; and updates on specific Australian zoonoses including Q fever, Hendra, Mycobacterial infections and whatever emerges over the next 6 months….

The final programme will be available in February/March 2012. Any queries can be directed to: asid@racp.edu.au

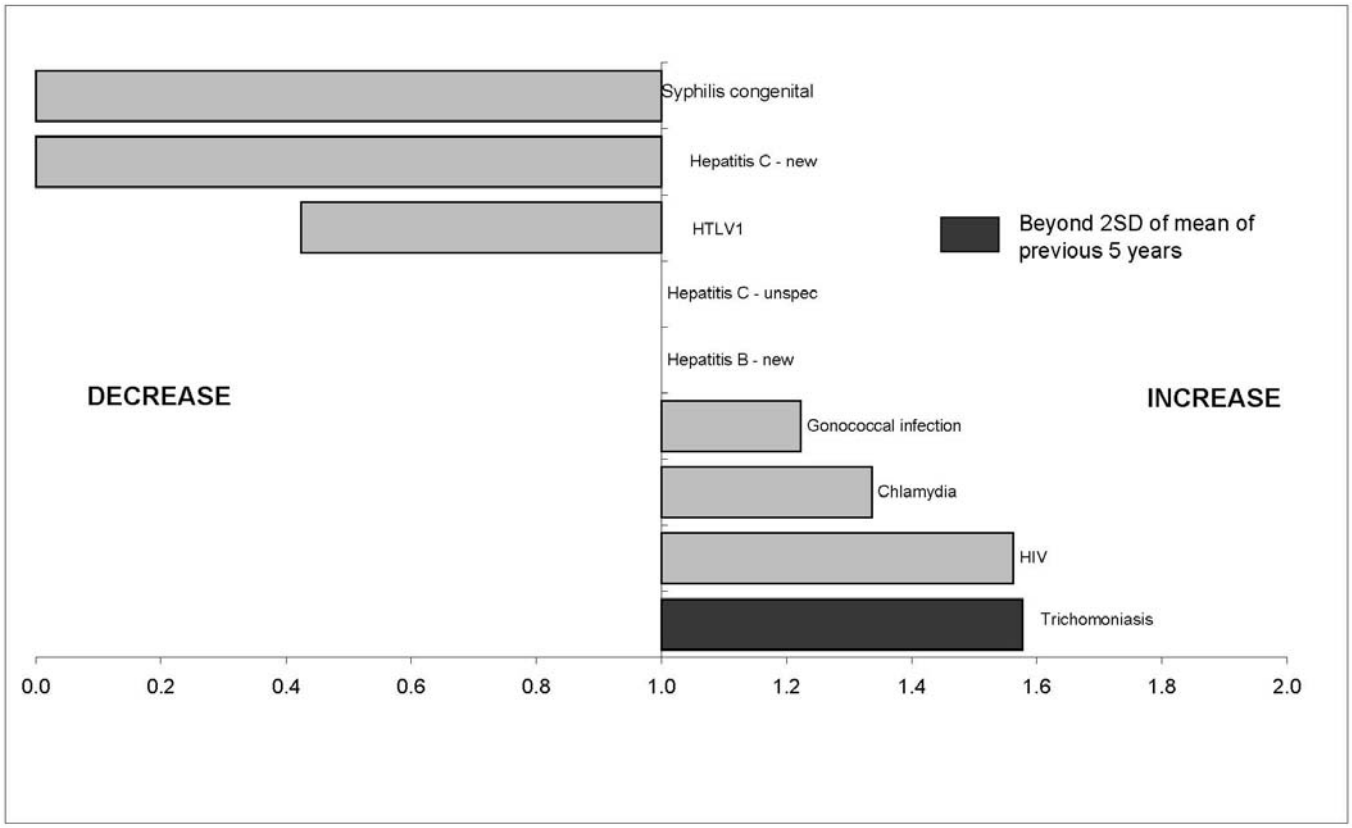
NT NOTIFICATIONS OF DISEASES BY ONSET DATE & DISTRICTS
1 July –30 September 2011 & 2010

	Alice Springs		Barkly		Darwin		East Arnhem		Katherine		N T	
	2011	2010	2011	2010	2011	2010	2011	2010	2011	2010	2011	2010
Acute post-streptococcal glomerulonephritis	1	1	0	0	1	1	0	1	1	2	3	5
Adverse vaccine reaction	4	2	0	0	7	5	1	2	1	0	13	9
Amoebiasis	0	0	0	0	1	0	0	0	0	1	1	1
Arbovirus infection - not otherwise specified	0	0	0	0	0	0	0	1	0	0	0	1
Barmah Forest virus infection	1	2	0	0	10	7	0	0	0	0	11	9
Campylobacteriosis	7	6	0	4	33	26	3	2	6	4	49	42
Chickenpox	13	7	9	0	31	18	7	2	12	0	72	27
Chlamydial genital infection	197	258	12	16	354	373	55	54	95	84	713	785
Chlamydial conjunctivitis	0	4	0	0	3	5	0	0	0	0	3	9
Cryptosporidiosis	4	2	0	0	2	3	2	1	2	3	10	9
Dengue virus infection	0	0	0	0	2	10	0	0	0	2	2	12
Food or water borne disease	0	0	0	1	0	0	0	0	0	0	0	1
Gonococcal conjunctivitis	0	0	0	0	0	0	0	0	0	1	0	1
Gonococcal infection	244	324	14	19	93	103	30	33	93	86	474	565
Gonococcal neonatal ophthalmia	1	0	0	0	0	0	0	0	0	0	1	0
Group A streptococcal infection (invasive)	4	0	4	0	13	0	1	0	0	0	22	0
Hepatitis A	0	0	0	0	3	4	0	0	0	0	3	4
Hepatitis B - chronic	12	20	0	3	6	5	12	12	1	8	31	48
Hepatitis B - new	1	0	0	0	1	1	0	0	0	0	2	1
Hepatitis B - unspecified	15	13	0	0	19	17	0	1	2	10	36	41
Hepatitis C - unspecified	7	12	0	1	42	30	0	0	0	5	49	48
***Haemophilus influenzae* b**	1	0	0	0	0	0	0	0	0	0	1	0
***Haemophilus influenzae* non-b**	1	1	0	0	0	0	0	0	0	0	1	1
HIV	0	0	0	0	5	2	0	0	0	0	5	2
HTLV1 asymptomatic/unspecified	10	26	0	0	0	0	0	0	0	2	10	28
Influenza	167	3	10	0	23	201	5	13	20	6	225	223
Legionellosis	0	1	0	0	1	1	0	0	0	0	1	2
Malaria	0	0	0	0	1	1	0	1	1	0	2	2
Measles	1	0	0	0	0	1	0	0	0	0	1	1
Melioidosis	0	0	1	0	0	10	1	1	1	0	3	11
Meningococcal infection	0	2	0	0	0	0	0	0	0	0	0	2
Non-tuberculosis mycobacterial disease	0	0	1	2	0	0	0	0	0	0	1	2
Pertussis	4	81	0	1	81	17	0	0	14	0	99	99
Pneumococcal disease (invasive)	26	10	3	0	11	6	1	0	6	2	47	18
Rheumatic fever	6	6	0	1	8	6	9	4	3	3	26	20
Ross River Virus infection	1	5	0	0	15	64	0	3	1	3	17	75
Rotavirus infection	54	22	4	1	15	10	3	4	13	9	89	46
Salmonellosis	17	12	2	1	55	92	6	7	7	19	87	131
Shigellosis	0	3	1	0	4	5	0	0	1	2	6	10
Strongyloidiasis (disseminated)	0	0	0	0	1	0	0	0	0	0	1	0
Syphilis < 2y	3	3	1	0	1	2	0	0	1	3	6	8
Syphilis > 2y or unknown	3	8	1	1	3	5	0	5	2	6	9	25
Trichomoniasis	261	205	29	43	206	196	133	102	160	177	789	723
Tuberculosis	1	0	0	1	6	5	1	0	1	2	9	8
Typhoid	0	0	0	0	1	0	0	0	0	0	1	0
Varicella - unspecified	0	0	0	0	2	1	0	0	0	0	2	1
Vibrio food poisoning	0	0	0	0	2	1	0	0	0	0	2	1
Zoster	4	1	1	1	31	23	3	3	9	3	48	31
Total	**1,071**	**1,040**	**93**	**96**	**1,093**	**1,257**	**273**	**252**	**453**	**443**	**2983**	**3088**

Ratio of the number of notifications (3rd Quarter 2011 cases to the mean Q3 2006-10): selected diseases



Ratio of the number of notifications (3rd Quarter 2011 cases to the mean Q3 2006-10): sexually transmitted diseases



Comments on notifications p 30

Trichomoniasis

There were 789 cases of trichomoniasis this quarter compared with the 5 year mean of 500 for the same quarter. Trichomoniasis has increased yearly since it became notifiable in 1999 and particularly since the introduction of the nucleic acid testing in 2006. This year rates have increased further perhaps due to the increased testing due to the STRIVE study.

Zoster (shingles)

This quarter there were 48 cases of zoster notified which was 57% more than the expected number of 30. This number represents only a small proportion of the real number of zoster cases. While the recent increase might reflect a true increase in zoster cases it may be due to an increased awareness by clinicians of the availability of the nucleic acid test for zoster and therefore better capture of cases. It has been hypothesised that the introduction of the varicella-zoster virus vaccine in 2006 might contribute to increased rates of zoster as time goes on; further study is being done to look into zoster rates in the vaccination era.

Invasive pneumococcal disease

The trend of having higher than normal numbers of invasive pneumococcal disease notifications continued in this quarter with 47 cases notified in the Northern Territory. This is more than twice the number reported for the same period last year. This is attributed to the continuing serotype 1 outbreak (49% of all cases) with notifications from residents in central Australia as well as the Katherine region. Over 40% of the serotype 1 cases are children aged <15 years but no cases have been reported in children who have received a conjugate pneumococcal vaccine containing serotype 1.

Rheumatic Fever

During this quarter there has been a marked increase in ARF cases in 3 communities in the Top End. Six new cases of 1st episode of ARF and 4 recurrences ARF were notified. Historically 1 of the communities has consistently high rates of Rheumatic Heart Disease (RHD), so the recent increase in ARF episodes is not unusual. This community now has a dedicated RHD Program Coordinator which benefits overall ARF and RHD patient care. A structured systems-based approach is being implemented to work with these 3 health services to improve their administration and management of the RHD Program.

Ross River virus infections

Ross River virus case numbers were well below average this quarter due to relatively low numbers of *Culex annulirostris* (fresh water common banded mosquito) and very good *Aedes vigilax* (salt marsh mosquito) aerial larval control in the brackish and saline swamps around Leanyer swamp.

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Immunisation coverage 30 September 2011

Compiled by Charles Strebor, CDC, Darwin

Immunisation coverage rates for NT children by regions based on Medicare address postcode as estimated by the Australian Childhood Immunisation Register are shown on page 34.

Background information to interpret coverage

Winnellie PO Bag is postcode 0822, which includes most Darwin Rural District communities, some East Arnhem District communities and some people who live in the Darwin "rural area" who collect mail from the Virginia store or Bees Creek. Alice Springs PO Bag is postcode 0872, which includes Alice Springs District, Nganampa and Ngaanyatjarra communities.

The cohort of children assessed at 12 to <15 months of age on 30 September 2011 were born between 1 April 2010 and 30 June 2010 inclusive. To be considered fully vaccinated, these children must have received 3 valid doses of vaccines containing diphtheria, tetanus, pertussis, and poliomyelitis antigens, either 2 doses of PRP-OMP Hib or 3 doses of another Hib vaccine, and 2 doses of hepatitis B vaccine (not including the birth dose) (latest doses due at 6 months of age). All vaccinations must have been administered by 12 months of age.

The cohort of children assessed at 24 to <27 months of age on 30 September 2011 were born between 1 April 2009 and 30 June 2009 inclusive. To be considered fully vaccinated, these children must have received 3 valid doses of vaccines containing diphtheria, tetanus, pertussis, and poliomyelitis antigens, either 3 doses of PRP-OMP Hib or 4 doses of another Hib vaccine, and 2 doses of hepatitis B vaccine (not including the birth dose) and 1 dose of measles, mumps, rubella vaccine (latest doses due at 12 months of age). All vaccinations must have been administered by 24 months of age.

The cohort of children assessed at 60 to <63 months of age on 30 September 2011 were born between 1 April 2006 and 30 June 2006 inclusive. To be considered fully vaccinated, these children must have received 4 valid doses of vaccines containing diphtheria, tetanus, pertussis antigens, 4 doses of poliomyelitis vaccine and 2 valid doses of measles, mumps, rubella vaccine (latest doses due at 4 years of age). All vaccinations must have been administered by 60 months (5 years) of age.

Interpretation

Immunisation coverage in NT children was above the national average for the 12 to <15 and 24 to <27 month cohorts although below the national average for the 60 to <63 cohort.

Immunisation coverage for Indigenous NT children was above the national Indigenous average across all 3 cohorts as well as being above the national average in the 24 to <27 months cohort.

Immunisation coverage for children aged 12-<15 months at 30 Sept 2011

Region	Number in District	% DTP	% Polio	% HIB	% Hep B	% Fully vaccinated
Darwin	262	94.7%	95.0%	94.7%	95.0%	94.7%
Winnellie PO Bag	94	92.6%	92.6%	92.6%	92.6%	92.6%
Palm/Rural Area	224	94.2%	94.2%	94.2%	94.2%	94.2%
Katherine	104	89.4%	89.4%	89.4%	89.4%	89.4%
Barkly	24	95.8%	95.8%	95.8%	95.8%	95.8%
Alice Springs	136	92.6%	92.6%	92.6%	91.9%	91.9%
Alice Springs PO Bag	55	92.7%	92.7%	92.7%	92.7%	92.7%
East Arnhem	58	94.8%	94.8%	94.8%	94.8%	94.8%
NT	957	93.4%	93.5%	93.4%	93.4%	93.3%
NT Indigenous	396	91.7%	91.7%	91.7%	91.7%	91.7%
NT Non-Indigenous	561	94.7%	94.8%	94.7%	94.7%	94.5%
Australia Indigenous	3,501	86.0%	85.9%	85.9%	85.9%	85.9%
Australia Non Indigenous	71,427	92.8%	92.8%	92.7%	92.5%	92.4%
Australia total	74,928	92.5%	92.5%	92.4%	92.2%	92.1%

Immunisation coverage for children aged 24-<27 months at 30 Sept 2011

Region	Number in District	% DTP	% Polio	% HIB	% Hep B	% MMR	% Fully vaccinated
Darwin	293	92.5%	92.5%	92.8%	91.5%	90.1%	87.7%
Winnellie PO Bag	114	99.1%	99.1%	99.1%	99.1%	99.1%	99.1%
Palm/Rural Area	237	97.0%	97.0%	98.3%	96.6%	95.8%	94.1%
Katherine	102	98.0%	98.0%	98.0%	98.0%	98.0%	97.1%
Barkly	19	94.7%	94.7%	94.7%	94.7%	100.0%	94.7%
Alice Springs	138	95.7%	95.7%	95.7%	95.7%	94.2%	92.8%
Alice Springs PO Bag	70	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
East Arnhem	50	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
NT	1023	96.2%	96.2%	96.6%	95.8%	95.1%	93.6%
NT Indigenous	448	98.0%	98.0%	98.2%	98.0%	97.5%	96.9%
NT Non-Indigenous	575	94.8%	94.8%	95.3%	94.1%	93.2%	91.1%
Australia Indigenous	3,516	94.9%	94.9%	95.7%	94.8%	94.7%	92.7%
Australia Non Indigenous	71,044	95.0%	95.0%	95.2%	94.6%	94.0%	92.8%
Australia total	74,560	95.0%	95.0%	95.3%	94.6%	94.1%	92.8%

Immunisation coverage for children aged 60-<63 months at 30 Sept 2011

Region	Number in District	% DTP	% Polio	% MMR	% Fully vaccinated
Darwin	252	82.1%	82.1%	82.1%	82.1%
Winnellie PO Bag	84	98.8%	98.8%	98.8%	98.8%
Palm/Rural Area	218	88.1%	88.1%	87.6%	87.2%
Katherine	92	95.7%	95.7%	95.7%	95.7%
Barkly	29	86.2%	86.2%	89.7%	86.2%
Alice Springs	105	87.6%	87.6%	86.7%	86.7%
Alice Springs PO Bag	62	96.8%	96.8%	96.8%	96.8%
East Arnhem	55	89.1%	89.1%	89.1%	89.1%
NT	897	88.7%	88.7%	88.6%	88.4%
NT Indigenous	394	90.6%	90.6%	90.6%	90.4%
NT Non-Indigenous	503	87.3%	87.3%	87.1%	86.9%
Australia Indigenous	3,184	86.0%	85.8%	86.3%	85.5%
Australia Non Indigenous	68,930	89.9%	89.9%	89.8%	89.4%
Australia total	72,114	89.8%	89.7%	89.6%	89.3%

24 to <27 month immunisation coverage, December 2010—June 2011 recalculated

Compiled by Charles Strebor, CDC, Darwin

As noted in the March, June and September 2011 issues of this *Bulletin*, immunisation coverage rates for children 24 to <27 months for *Haemophilus influenza* type B as calculated by the Australian Childhood Immunisation Register (ACIR) were initially 10% and then over 40% lower for the NT than would be expected.

On 1 October 2009, the NT Immunisation Schedule changed from having a 3 dose schedule of a PRP-OMP HIB containing vaccine to a 4 dose schedule of a PRP-T HIB containing vaccine which consequently affected the calculation for this age cohort. As a result of the lower-than-expected coverage estimates as supplied by the ACIR, the NT Centre for Disease Control asked that these coverage rates be re-calculated taking this information into account.

These recalculations have been undertaken and are provided below.

Immunisation coverage for children aged 24-<27 months at 31 December 2010

Region	Number in District	% DTP	% Polio	% HIB	% Hep B	% MMR	% Fully vaccinated
Darwin	274	93.8%	93.8%	93.1%	92.7%	92.7%	90.9%
Winnellie PO Bag	96	97.9%	97.9%	97.9%	97.9%	96.9%	96.9%
Palm/Rural Area	242	95.0%	95.0%	93.4%	94.6%	93.4%	91.7%
Katherine	100	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Barkly	11	81.8%	81.8%	81.8%	81.8%	90.9%	81.8%
Alice Springs	122	97.5%	97.5%	95.9%	97.5%	95.9%	95.1%
Alice Springs PO Bag	61	100.0%	100.0%	98.4%	100.0%	98.4%	98.4%
East Arnhem	48	100.0%	100.0%	100.0%	100.0%	97.9%	97.9%
NT	954	96.2%	96.2%	95.3%	95.8%	95.1%	93.9%
NT Indigenous	385	96.6%	96.6%	95.6%	96.6%	96.4%	95.1%
NT Non-Indigenous	569	96.0%	96.0%	95.1%	95.3%	94.2%	93.1%
Australia Indigenous	3,444	94.7%	94.7%	94.9%	94.7%	94.5%	92.4%
Australia Non Indigenous	73,395	94.9%	94.9%	95.1%	94.4%	94.0%	92.7%
Australia total	76,839	94.9%	94.9%	95.1%	94.4%	94.0%	92.7%

Immunisation coverage for children aged 24-<27 months at 31 March 2011

Region	Number in District	% DTP	% Polio	% HIB	% Hep B	% MMR	% Fully vaccinated
Darwin	274	94.9%	94.9%	93.4%	94.5%	94.9%	93.1%
Winnellie PO Bag	76	98.7%	98.7%	97.4%	98.7%	98.7%	97.4%
Palm/Rural Area	245	96.7%	96.3%	94.7%	96.7%	95.9%	93.9%
Katherine	78	97.4%	97.4%	94.9%	97.4%	97.4%	93.6%
Barkly	23	95.7%	95.7%	95.7%	95.7%	100.0%	95.7%
Alice Springs	118	96.6%	96.6%	94.1%	96.6%	94.9%	93.2%
Alice Springs PO Bag	69	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
East Arnhem	61	98.4%	98.4%	96.7%	96.7%	96.7%	91.8%
NT	944	96.7%	96.6%	95.0%	96.5%	96.3%	94.2%
NT Indigenous	378	95.0%	95.0%	95.0%	94.7%	96.0%	93.4%
NT Non-Indigenous	566	97.9%	97.7%	95.1%	97.7%	96.5%	94.7%
Australia Indigenous	3,419	94.2%	94.1%	94.8%	94.1%	94.2%	91.8%
Australia Non Indigenous	71,271	95.0%	95.0%	95.2%	94.5%	94.0%	92.7%
Australia total	74,690	95.0%	94.9%	95.2%	94.5%	94.0%	92.7%

Immunisation coverage for children aged 24-<27 months at 30 June 2011

Region	Number in District	% DTP	% Polio	% HIB	% Hep B	% MMR	% Fully vaccinated
Darwin	274	94.9%	94.9%	93.4%	94.5%	94.9%	93.1%
Winnellie PO Bag	9	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
Palm/Rural Area	312	97.1%	96.8%	95.2%	97.1%	96.5%	94.6%
Katherine	78	97.4%	97.4%	94.9%	97.4%	97.4%	93.6%
Barkly	23	95.7%	95.7%	95.7%	95.7%	100.0%	95.7%
Alice Springs	118	96.6%	96.6%	94.1%	96.6%	94.9%	93.2%
Alice Springs PO Bag	69	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
East Arnhem	61	98.4%	98.4%	96.7%	96.7%	96.7%	91.8%
NT	944	96.7%	96.6%	95.0%	96.5%	96.3%	94.2%
NT Indigenous	389	96.9%	96.9%	96.4%	96.9%	97.2%	95.6%
NT Non-Indigenous	555	96.6%	96.4%	94.1%	96.2%	95.7%	93.2%
Australia Indigenous	3,510	95.0%	95.0%	95.7%	94.9%	95.2%	93.1%
Australia Non Indigenous	70,157	94.9%	94.9%	95.1%	94.5%	94.0%	92.8%
Australia total	73,667	94.9%	94.9%	95.1%	94.5%	94.0%	92.8%

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NT Malaria notifications July—September2011

Frances Daily, CDC, Darwin

There were 3 notifications of malaria received this quarter. The following table provides details about where the infection was thought to be acquired, the infecting agent and whether chemoprophylaxis was used.

No. cases	Origin of infection	Reason for exposure	Agent	Chemoprophylaxis
1	Bali	Holiday	*P. falciparum*	No
1	Liberia	Resident	*P. falciparum*	No
1	West Papua	Holiday	*P. vivax*	No

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Disease Control staff updates

Darwin

Merv Fairley, RN in the TB/Leprosy Unit and team member in CDC Darwin for 14 years passed away in October 2011. His presence is sorely missed. He has left CDC staff members with many memories. His beautiful and self sustaining tropical garden reminds us daily of his spirit.

Mahesh Menon, Public Health Registrar joined CDC TB Unit in October for 4 months and generously volunteered for the recent 'mosquito duty' in Tennant Creek. **Nor Shaharuddin,** Public Health Registrar, returned to the TB unit in November after a period at RDH in infectious diseases and will return to Malaysia in the New Year. Her talents will be missed. **Frances Daily,** Public Health Registrar, has left CDC to return to Cambodia. Frances will continue to work on the TB guidelines update.

Helen Wassman, Project Officer in Community Paediatrics resigned to take up a position in NSW to be closer to family. **Debra Liddle**, AO5, commenced 3 month employment with Medical Entomology in November. **Meredith Neilson** has returned to CDC as the Injury Prevention Policy and Project co-ordinator. Meredith previously worked at CDC in 2003 as the Chronic Diseases Coordinator and is now working part-time Monday – Tuesday. **Mary Verus** has returned following her successful knee reconstruction. Thanks to **Gemma Farmer** for acting in the surveillance position. **Lindsay Stirrat**, Administration Officer TB Unit Darwin, has taken 12 months leave without pay. **Gemma Farmer** will act in Lindsay's position for the next 12 months. **Lisa Panton,** PHN, has resigned from the Rheumatic Heart Disease program to take up a surveillance officers position at the Randwick Surveillance Unit, NSW.

Brenda Santi, Remote Sexual Health Coordinator from SHBBV Unit has taken 12 month leave without pay. **Suzanne Connor** will be joining the team to act in the Remote Sexual Health coordinator while Brenda is on leave. **Susannah O'Brien**, Blood Borne Virus Policy Officer has taken on a position as A/Executive and Strategic Policy Officer Health Protection Division with Barbara Paterson. **Kishan Kariippanon** Youth Health Policy Officer has resigned from the SHBBV unit and will be continuing his studies. **Jocelyn Perry**, Co-ordinator Early Indigenous Childhood Development Project has gone on 12 months maternity leave. **Michael Borenstein** transferred from Alice Springs to Darwin to take up the position of Sexuality Education Project Officer while Jocelyn is on maternity leave. **Autumn Goodall,** CNC SHBBV unit, has returned from maternity leave.

Congratulations to ex-CDC staff member **Meredith Hansen-Knarhoi** on her award of the degree of BMBS with Honours from Flinders University. Dr Hansen-Knarhoi will be an intern next year at RDH.

Vicki Krause was a recipient of the Chief Minister's Public Sector Medal in 2011, a medal awarded for outstanding and meritorious service (slipped in by the production team).

Katherine

The **Hon Kon Vatskalis**, Minister for Health and **Jenny Cleary**, Executive Director Top End Hospital Network visited Katherine Hospital on 28 September 2011 to present service awards to 102 staff for a total of 551 years of service given to the community of Katherine. There were 4 categories of service awards - 5, 10, 20 and 30 years. CDC staff receiving awards were **Maria Chandler** (30 years), **Judy Barritt** (20 years), **Kaye McGough** (20 years), **Carmel Whalley** (20 years) and **Kerrie Bettison** (5 years). Congratulations all!

Louis Geri, Public Health Nurse commenced on 28 November in the TB and Leprosy RN position at Katherine CDC. **Kyle Osborne**, has joined the SHBBV unit in Katherine as Adolescent Sexual Health Promotion Officer

Disease Control staff updates continued

with the Early Indigenous Childhood Development Project. **Raenae Reeves** - has joined the SHBBV Unit as the Adolescent Sexual Health Promotion Officer with the Early Indigenous Childhood Development Project.

Alice Springs

Robyn Puls, Trachoma Data Manager and Administrative Support AO4 resigned from the Trachoma Unit. **Andrew Mellows,** Aboriginal Men's Health Worker, has joined SHBBV unit in Alice Springs. **Michael Borenstein** has transferred from Alice Springs to Darwin to take up the position of Co-ordinator Early Indigenous Childhood Development Project. **Heather Wilson** resigned from the Trachoma Team on 8 December and **Julie Pedersen** also finishes in the Trachoma Team in December. **Cate Coffey** starts long service leave from 19 December 2011 till April 2012. **Prathibha Chelemella** returned in November after 4 months extended leave. **Kaylene Prince** is on leave from 28 October – 2 January 2012. **Wendy Mactaggart** resigned in November from the Remote Sexual Health Team. Wendy is moving to Canberra to start a midwifery refresher course. **Helen Tindall** reverted back to the TB Public Health Nurse position in December, after backfilling the Immunisation Public Health Nurse for several months.

Gove

Marion Smith, CNC SHBBV East Arnhem has resigned after 2½ years in the position and will initially be doing the midwifery up-skilling in Katherine before moving west. **Kathy Shield** will be taking over the position from Marion.