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Elevated blood lead levels in the Northern Territory—an update for 2017
Pasqualina Coffey, Centre for Disease Control, Darwin

Abstract

A Steering Committee of key stakeholders was established in 2015 to investigate a cluster of children from some remote communities with elevated blood lead levels. The Steering Committee has taken on a broad range of actions to address the public health issue of lead exposure in the Northern Territory. This article provides an update on the key areas of health promotion, clinical guidelines development, regulation of lead shot, making elevated blood lead levels a notifiable disease, analysis of past blood lead results and the testing of magpie geese for lead.

Key words: Blood lead levels; shotgun ammunition; magpie geese; health promotion; public health legislation

Background

The Centre for Disease Control (CDC), Environmental Health Branch, Royal Darwin Hospital Paediatric Department, Top End Health Service (TEHS) - Outreach, Primary Health Care Management, Child and Youth Health Strategy, the Chief Health Officer, Northern Territory (NT) Police, the Chief Veterinary Officer, Legal Services and the Department of Environment and Natural Resources have worked together on a Steering Committee for Evaluating and Responding to Elevated Lead Levels since 2015. The Steering Committee was established after a cluster of children from several remote communities were found to have elevated blood lead levels (BLLs). Input has also been gathered from presentations or attendance by the Department of Lands, Planning and the Environment and the Department of Housing and Community Development.

Lead is a naturally occurring metal and is ubiquitous in the environment i.e. the soil, air and water, although generally in minute amounts. Human
exposure to lead has increased as its unique attributes became prized elements of many widespread products such as petrol, paint, piping and ammunition. Lead was known to have deleterious effects on human health as far back as in ancient Egypt, but only recently has it been recognised that even low levels of lead exposure are associated with harmful effects. The National Health and Medical Research Council currently recommends that if a person has a BLL > 5 micrograms per decilitre (mcg/dl), the source of exposure should be investigated and reduced, particularly if the person is a child or a pregnant woman.

The Steering Committee on elevated BLLs has taken on a broad range of actions to address the public health issue of lead. This article provides an update on 6 key areas:

- Health promotion
- Clinical guidelines development
- Regulation of lead shot
- Making elevated BLLs a notifiable disease
- A retrospective study of blood lead results in the Northern Territory (NT) and
- Testing magpie geese for lead.

**Health Promotion**

One of the TEHS Primary Health Care Outreach teams has driven the development and delivery of a suite of resources aimed at raising awareness of the dangers of elevated BLLs, ensuring key groups have access to information and resources to assist with preventing exposure to lead (unpublished report). These include: targeted presentations for children, communities and health services; posters; factsheets; a clinical protocol; a school lesson plan; internal communication; and wider media coverage.

Following on from visits undertaken in 2016, the Outreach team will be heading back out to communities to deliver face to face sessions throughout March 2017. To evaluate effectiveness of these health promotion activities and resources, the team will monitor attendance figures and number of services and groups they have engaged in order to assess reach. They will also seek feedback from target audiences to improve existing resources as the program is rolled out across the Top End.

**Figure 1. Case management guidelines for suspected elevated lead levels in the NT**

<table>
<thead>
<tr>
<th>Case Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>5 – 9.9µg/dL</td>
</tr>
<tr>
<td>• Clinical assessment</td>
</tr>
<tr>
<td>• Hb (or FBC) - treat iron deficiency as it increases lead absorption.</td>
</tr>
<tr>
<td>• Clinic to undertake lead questionnaire with parent/guardian</td>
</tr>
<tr>
<td>• Educate household on lead exposure reduction methods</td>
</tr>
<tr>
<td>• Recall for repeat lead level in 6 months</td>
</tr>
<tr>
<td>10-19.9 µg/dL</td>
</tr>
<tr>
<td>• As above</td>
</tr>
<tr>
<td>• Clinic to undertake lead questionnaire with parent/guardian and to send completed questionnaire to Environmental Health</td>
</tr>
<tr>
<td>• Clinical assessment by District Medical Officer</td>
</tr>
<tr>
<td>• Refer to paediatrician</td>
</tr>
<tr>
<td>• Put on recall for repeat lead level in 3 months</td>
</tr>
<tr>
<td>• If on repeat testing (at least 3 months after initial) blood lead level is in this range or rising, escalate as for levels 20 – 44.9µg/dL</td>
</tr>
<tr>
<td>20 – 44.9µg/dL</td>
</tr>
<tr>
<td>• As above</td>
</tr>
<tr>
<td>• Abdominal x-ray</td>
</tr>
<tr>
<td>• Follow-up with repeat test in 1 month</td>
</tr>
<tr>
<td>• Notify Environmental Health as per environmental assessment and response</td>
</tr>
<tr>
<td>&gt;=45µg/dL</td>
</tr>
<tr>
<td>• As above and urgent consult re children with paediatrician, and admission to hospital.</td>
</tr>
<tr>
<td>Consult with physician re adults.</td>
</tr>
</tbody>
</table>

**Clinical guidelines**

The Steering Committee has composed and disseminated “Guidelines for the Response to Elevated Lead Levels for Remote Clinics and Primary Health Care Providers.” The Guidelines set out indications for testing BLLs, a case definition (BLL >5mcg/dL), case management actions (stratified by lead level see Figure 1) and key advice for health practitioners to give affected individuals and their families. It also contains the Environmental Assessment and Response Protocol for elevated BLLs (Figure 2). These Guidelines assist environmental health officers, clinicians and public health practitioners in carrying out the appropriate course of actions to identify possible sources of lead exposure in a person’s diet or environment and subsequently to reduce or eliminate the source.
### Figure 2. Environmental assessment and response protocol for notifications of elevated blood lead levels

<table>
<thead>
<tr>
<th>Level</th>
<th>Blood lead level</th>
<th>Age category</th>
<th>NT Health Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>≥ 5&lt;9.9 µg/dL</td>
<td>Children</td>
<td>Clinic staff use factsheet ‘Lead exposure in children’ and lead prevention resources&lt;br&gt;Clinic staff undertakes environmental audit questionnaire with parents if repeat testing identifies lead blood levels in this range</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adults</td>
<td>Clinic staff uses appropriate general ‘Lead’ factsheet. English or translated versions&lt;br&gt;Clinic staff uses environmental audit questionnaire and interviews adults if repeat testing identifies lead blood levels in this range</td>
</tr>
<tr>
<td>2</td>
<td>≥ 10&lt;19.9 µg/dL</td>
<td>Children</td>
<td>Clinic staff uses factsheet ‘Lead exposure in children’ and lead prevention resources&lt;br&gt;Clinic provides factsheet/information pamphlets on risk identification and management to requesting doctor or case’s parents/guardians&lt;br&gt;Environmental Health staff undertake desk top study of lead sources in the specific community environment and checks with relevant NTG agencies and outstation resource centres on lead history of built environment and drinking water supplies&lt;br&gt;Clinic undertakes environmental audit questionnaire for interviewing parents/guardians and sends the Centre for Disease Control and Environmental Health a copy of completed audit&lt;br&gt;If repeat blood testing by the Clinic identifies lead blood levels above 15 µg/dL and other young children or pregnant woman/women are identified as at risk of exposure, contact Environmental Health to arrange for home risk assessment to be undertaken with consent of case’s parents. Environmental Health staff assists Clinic in personal counselling of parents on the results of the home risk assessment by phone or by visit</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adults</td>
<td>If occupational exposure, Clinic advises the patient to consult NT WorkSafe for further advice if appropriate&lt;br&gt;If non-occupational exposure, same actions as per children in level 2</td>
</tr>
<tr>
<td>3</td>
<td>Above 20 µg/dL</td>
<td>Children</td>
<td>As for level 2, plus&lt;br&gt;Environmental Health staff undertake home risk assessment with consent of case’s parents/guardians using X-ray fluorescence (XRF) apparatus (where available) for soils, inside of buildings etc as well as collecting water samples&lt;br&gt;EHO Guidance note used for investigation purposes&lt;br&gt;EHO provides information on results of sampling/analysis and recommendations on remediation options to the Clinic for liaison with case, as well as CDC and relevant authorities</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adults</td>
<td>As for Level 2, plus&lt;br&gt;Consult NT WorkSafe for further advice on occupational exposure, if appropriate.</td>
</tr>
</tbody>
</table>
Regulation of lead shot

In the investigation undertaken in 2015–16 as a response to a number of children in some NT remote communities having elevated BLLs, it was considered highly likely that the source of the lead was lead shot. The key routes of exposure children are threefold: firstly, from touching and playing with the shells or the lead pellets that have been taken out of shells; secondly, from direct consumption of bush tucker meat killed with lead shot, which may still contain lead pellets and fragments; and lastly, indirectly from consumption of magpie geese that have long-term accumulation of lead stores from ingesting spent lead shots when foraging in wetlands.

The use of lead shot by waterfowl hunters is illegal in all states in Australia other than the NT. In the NT the use of lead shot is prohibited for hunters requiring a permit to hunt waterfowl under the Territory Parks and Wildlife Conservation Act. In practice, this mostly affects non-Aboriginal hunters and lead shot continues to be widely used by Aboriginal people across the NT. A literature review undertaken for the Steering Committee determined that elevated BLLs were linked to use of lead shot in traditional hunting by many Indigenous and non-Indigenous populations globally (unpublished report available from author).

The Steering Committee is currently reviewing recommended legal options and will shortly commence stakeholder consultation to discuss this important public health issue with affected groups and key agencies, including Aboriginal Land Councils, NT Police Fire and Emergency Services and sporting and recreational shooters.

Elevated blood lead levels as a notifiable disease

Cases of elevated BLLs are legally required to be notified in most but not all jurisdictions in Australia. The management of each case depends on the blood level, the potential for harm, likely sources of lead and scale of lead exposure. Different responsibilities and actions are delegated to health practitioners, public health agencies and environmental health officers. Currently in the NT, there is no systematic means for surveillance of, and initiating a public health response to, elevated BLLs. There are clear advantages in making a disease of public health importance notifiable, in enabling prevention, control and management of that disease. Establishing an elevated BLL as a notifiable condition will not change the medical interventions offered to individual cases, but should augment and regulate case management and environmental assessment, as well as provide epidemiological data on incidence and trends.

In the NT, the Notifiable Diseases Act 1999 is the legal mechanism under which certain diseases must be reported by clinicians or laboratories, but in essence relates only to infectious diseases. Alternatively, the Public and Environmental Health Act sets out functions for the Chief Health Officer to “to develop and implement strategies to promote and protect public health” and to request the reporting of health information for “monitoring, protecting, maintaining or promoting public health”. Hence, there are options (guidelines or regulations) under this Act which would serve to mandate the reporting of an elevated BLL in the NT, which the Steering Committee under the auspices of the Chief Health Officer is exploring.

Retrospective analysis of elevated blood lead levels

A retrospective analysis of BLL pathology results in the NT between 01/11/2010 and 02/11/2016 was undertaken by CDC (unpublished data, personal communication Kate Hardie and Rebecca Jarman). The data provided were de-identified and no information regarding the indication for testing was available. The pattern and number of tests performed in 2014-2016 was affected by active case finding activities in both children and adults. Other than during that period, it was presumed that adult testing largely reflected occupational lead testing. Key findings included:

- Overall 17% of children and 25% of adults tested had a lead level ≥ 5mcg/dL
- The proportion of children with an elevated BLL increased with age, from 9% under 5 years to 27% aged 10–18 years
• Only 1 child under 5 years had a level above > 10mcg/dL and
• Among adults tested, the proportion ≥5mcg/dL was 20–30% for all age groups.

The results suggest that elevated BLLs in children are likely to be an issue beyond the communities previously identified. Furthermore, the rise in proportion of children with an elevated BLL with age supports either ongoing exposure throughout childhood, or an increase in behaviours associated with exposure as the child matures.

Testing lead levels in magpie geese

A study is being conducted by CDC in conjunction with the Wildlife Use and Pest Animals Unit, Department of Environment and Natural Resources, that will investigate the possible role of lead-burdened magpie geese (Anseranas semipalmata) in human lead exposure. As previously mentioned, geese can carry a lead load prior to human consumption through 2 potential pathways: long-term accumulation through the ingestion of spent lead shot; and short-term loading from shotgun pellets and pellet fragments lodged in muscle tissue, consequent to being shot.

A previous study in the NT revealed an extremely high density of lead shot in soil sediment at a popular Top End hunting reserve. The same study was also able to demonstrate that magpie geese caught at this reserve had high levels of lead exposure as indicated by high lead concentrations found in the liver (30%) and ingested shot in the gizzards (21%) of flying birds.

This new study will compare lead levels in geese shot with lead and steel shot. It will allow an estimation of background lead levels in the geese as well as the additional lead from the shot pellets and thus the amount of lead humans might be exposed to via consumption. Sampled birds will be collected using the same method employed by Indigenous hunters, and undergo X-ray imaging (Figure 3) prior to dissection and testing.

Summary

Lead exposure remains an important and concerning public health issue. In the NT, the multidisciplinary Steering Committee for Evaluating and Responding to Elevated Lead Levels is undertaking a wide range of activities to tackle this problem. Through the combined efforts of the multiple agencies involved, key stakeholder groups and community leaders, the NT population can look to benefit from a reduction in lead exposure.

References


Figure 3. Radiographic image of a magpie goose shot with lead pellets
Measles alert

On 27 March 2017 the media release below was posted from the Northern Territory Centre for Disease Control.

In addition, measles alerts were sent out to Darwin General Practitioners, paediatricians, physicians, the Royal Darwin Hospital Emergency Department and Community Care Clinics.

As of 31 March 2017 more than 400 contacts had been identified and contacted. Contacts have been provided with information about measles (see page 7) and being a measles contact (see page 9) and interventions, such as measles immunisations, as required.

It is a timely reminder to ensure that you are immunised against measles.

Department of Health

Are you measles immune? Travellers to be alert for measles

27 March 2017

Travellers are being warned to ensure they are immune to measles following a large number of returned travellers to Australia getting measles, including one case in Darwin.

Across Australia, 23 people have been notified with measles so far this year.

Of these cases, 19 have been associated with travel to Asia including travel to Bali.

In recent days a person returning to Darwin from Bali has been diagnosed with measles and extensive contact tracing has been undertaken to identify those who have been in contact with the case. Nearly 300 contacts have been identified and will now be interviewed to assess their measles immune status and to provide management, for those not immune, as well to provide important information about the disease.

“Travellers and holidaymakers should check to make sure they are immune to measles, especially before travelling overseas,” Centre for Disease Control Director, Dr Vicki Krause said.

“If you were born after 1966, make sure you have had two doses of a measles-containing vaccine, most often given as the measles, mumps rubella (MMR) vaccine. Most people born in or before 1965 (those 48 years or older) would have been exposed to circulating measles and are likely to be immune.”

“Parents should also ensure their children are vaccinated on time. Children receive a measles containing vaccine at 12 months and 18 months.”

People returning from overseas, in particular from Bali, are advised to be alert for symptoms.

“Measles is a highly contagious viral illness that is easily spread among people through coughing and sneezing. It can also cause serious complications that may require hospitalisation.”

Symptoms of measles include fever, cough, runny nose and sore eyes, which usually occur 7 to 10 days after exposure, followed by a red blotchy rash two to four days later that starts on the face and then moves down the body.

“Infected people can potentially pass measles on to others several days before the rash develops and for four days after it starts,” Dr Krause said.

“It is important to recognise the early symptoms and signs of measles to prevent further spread of this illness.”

People who might have measles should minimise contact with other people, not attend school or work and seek medical advice as soon as possible.

“When attending a GP clinic, people should phone ahead to advise that they may have measles so the clinic can make arrangements to minimise contact with other people.”

For further information visit nt.gov.au/wellbeing/health-conditions-treatments/viral/measles

Media Contact: Dimitra Grehl 0427 596 954

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What is measles?
Measles is a highly infectious viral illness, which can cause serious disease. Measles is now uncommon in Australia because of high levels of immunisation.

Annually in Australia since 2000 there have been years with 10 cases only and some years with up to 200, many in returned travelers. In the Northern Territory since 2000 there have been 0 to 5 cases per year.

How is measles spread?
Measles is spread by breathing in airborne droplets from the coughs and sneezes of people infected with the disease. Measles is one of the most highly infectious communicable diseases. In Australia most measles infection originates from returned overseas travelers or from foreign visitors who can then spread the infection to non-immune individuals.

What are the symptoms?
The symptoms of measles are fever, cough, runny nose and sore eyes, which usually occur about 7 to 10 days after exposure to a case followed by a red, blotchy rash 2 to 4 days later. The rash starts on the face and spreads down the body. One third of people with measles develop complications particularly young children and adults. These include ear infection, diarrhoea and pneumonia, which may require hospitalisation. Rarely, measles may result in encephalitis (infection of the brain).

What is the infectious period?
A person with measles is infectious from 24 hours before the onset of the first symptoms until 4 days after the appearance of the rash. They are most infectious before the rash appears so often do not know they have measles.

Who is at risk?
People who are not immune either by vaccination or previous infection are at risk of measles infection.

How can measles be prevented?
The best protection against measles infection is vaccination and people should receive 2 measles-containing vaccines. In Australia the vaccine is available as a combination vaccine containing measles-mumps-rubella (MMR) or measles-mumps-rubella-varicella (MMRV).

All children are currently recommended to get vaccinated for measles at 12 and 18 months of age as part of the National Immunisation Program. Children that did not receive the 2nd vaccine at age 18 months should receive the 2nd dose at 4 years.

People who were born before 1966 were most likely exposed to measles and are considered immune.

All people who were born after 1966 should have evidence of either receiving 2 measles-containing vaccines or evidence of having had the disease (by a blood test).

It is important for all overseas travelers to ensure that they are immune to measles.

No measles-containing vaccine should be given during pregnancy or to women contemplating pregnancy. Pregnancy should be avoided for 28 days after vaccination.

Disease in non-immune people exposed to measles can be prevented by administration of a measles-containing vaccine if given within 3 days of exposure, or by administration of immunoglobulin within 7 days of exposure. See the ‘Measles Contact’ fact sheet.
How is it diagnosed?
Measles can be difficult to diagnose early in the illness because there are many other viruses that cause similar symptoms (cough, conjunctivitis and runny nose) with fever and a rash. Sometimes the presence of white spots inside the mouth, called Koplik spots, the timing of the fever and the rash and the characteristics of the rash can help a doctor to make the diagnosis.

Whenever measles is suspected, swabs from the nose or throat, a urine sample or a blood test can be collected to confirm the diagnosis in the laboratory. Confirming the diagnosis is important so that other people who may be at risk of measles can be identified.

What is the treatment?
There is no specific treatment for measles. People with measles should have plenty of fluids and rest and treat symptoms as they occur. While the person remains infectious it is important that they stay at home to reduce the risk of spreading the disease to other people.

Where can I get vaccinated?
The free vaccine is available from your community health centre, Aboriginal Medical Service and most general practitioners.

How is measles controlled?
People who have measles should stay at home until they are no longer infectious which is usually 4 days after the onset of the rash. Doctors, hospitals, laboratories, schools and childcare centres must notify cases of measles to the local Centre for Disease Control. This is so that people at risk of infection can be identified and control measures can be implemented to prevent further spread of the virus.

For more information contact the Centre for Disease Control in your region
Alice Springs 8951 7540
Darwin 8922 8044
Katherine 8973 9049
Nhulunbuy 8987 0357
Tennant Creek 8962 4259

Measles contact information

The following information is specifically intended for people who may have been in contact with a case of measles while infectious.

What is measles?
Measles is a highly infectious viral illness. People generally develop symptoms of the infection after 7-10 days but may take up to 18 days after having been exposed to an infectious person. These symptoms begin with:
- Fever
- Cough
- Runny nose
- Sore eyes.

The characteristic measles rash usually begins 2-4 days after the first symptoms, generally starting on the face and then spreading down the body. Sometimes the rash peels. The rash will last for 4-7 days.

Measles is often thought of as a minor childhood illness but it can cause serious illness, particularly in young adults.

Up to a third of people infected with measles will experience a complication. Complications are more common in young children and in adults. Complications include ear infections, diarrhoea and pneumonia, and may require hospitalisation. About one in every 1000 people with measles develops encephalitis (infection of the brain).

How long does a person remain infectious?
A person with measles is infectious from 24 hours before the onset of the first symptoms until 4 days after the appearance of the rash. They are most infectious before the rash appears so often do not know they have measles.

Am I susceptible to measles?
People who are susceptible to measles are:
- Infants aged between 6 and 12 months of age. If the mother is immune natural immunity from maternal antibodies is protective for children under the age of 6 months;
- All those born after 1966 who have not been immunised with 2 doses of a measles-containing vaccine or do not have a history of having had measles.
- People who have had only 1 measles-containing vaccine. Routine childhood measles vaccination did not include 2 doses until 1986 so those born between 1966 and 1986 have often had just 1 dose of vaccine.
- People who are immunocompromised (i.e. have decreased immunity) are also at risk – at any age, even if immunised. This includes people with diseases such as Hodgkin’s lymphoma or cancer, HIV and people undergoing cancer treatment or on high-dose steroids.
- People born before 1966 most likely had measles and are therefore most likely to be immune. If you have no clear history of measles you should consider yourself susceptible.

What can I do to avoid measles?
If you have been in contact with someone with measles and you are susceptible to measles your risk of becoming infected may be reduced by seeing your doctor immediately for vaccination or immunoglobulin.
If it is less than 3 days since you came into contact with measles, immunisation with a measles-containing vaccine can prevent infection. Immunoglobulin contains antibodies against the measles virus and is especially recommended for infants and people with underlying illnesses who have a greater risk of developing complications if they catch measles. Subsequent immunisation with any measles, mumps, rubella or chickenpox containing vaccine should be deferred until at least five months after immunoglobulin as the immunoglobulin antibodies can prevent the vaccine from working. Discuss with your usual immunisation provider if you have received immunoglobulin and require vaccination against any of these diseases.

Sometimes measles contacts might need to be excluded from the workplace, school or childcare to prevent further spread of the infection. If you think you may be susceptible to measles you need to discuss your options with your local doctor as soon as possible.

**What do I do if I think I have measles?**

If you suspect that you might have measles, make an appointment with your local doctor. Let them know you think you might have measles, and ask for a home visit if possible. If not, try to get the last appointment of the day to avoid coming into contact with other patients in the waiting room.

While a person is infectious with measles it is important that they remain at home to reduce the possibility of spread to other people.

For more information contact your nearest Centre for Disease Control.

<table>
<thead>
<tr>
<th>Location</th>
<th>Phone Number</th>
</tr>
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<tbody>
<tr>
<td>Darwin</td>
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<td>Katherine</td>
<td>89739049</td>
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<tr>
<td>Nhulunbuy</td>
<td>89870357</td>
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<td>Tennant Creek</td>
<td>89624259</td>
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<tr>
<td>Alice Springs</td>
<td>89517540</td>
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</tbody>
</table>

Herpes zoster vaccine and individuals who are immunocompromised

Ros Webby, Centre for Disease Control, Darwin

Background

Zostavax® herpes zoster vaccine contains live attenuated varicella-zoster virus, containing 14 times more virus than childhood varicella vaccines. Administration to people who are immunocompromised is associated with risk of disseminated disease from the vaccine virus.

Zostavax® must not be given to the following people, including but not limited to:

- **Haematological or generalised malignancies (including those not on treatment)** e.g. lymphoma, acute or chronic leukaemia, Hodgkin’s disease
- **Solid organ or bone marrow transplant recipients** (with exceptions as advised by specialists)
- **HIV/AIDS** (with exceptions as advised by specialists). Persons must have asymptomatic HIV (not AIDS) with CD4+ counts >200 per μL. Serologic confirmation of previous VZV infection must be obtained prior to vaccination
- **Other congenital/acquired immunodeficiencies**

- **Current or recent high-dose systemic immunosuppressive therapy** e.g. chemotherapy and radiation therapy (including 6 months post chemotherapy and radiation therapy), oral corticosteroids, disease modifying anti-rheumatic drugs. Please see the table below with advice about timing and dose of immunosuppressive therapy and vaccination.

If someone is on a combination of medications or if there is any doubt whether Zostavax® is safe for your patient, defer vaccination and seek specialist advice.

Vaccination in persons anticipating being significantly immunocompromised in the future.

Persons anticipating being significantly immunocompromised in the future i.e prior to transplantation or chemotherapy, may be vaccinated at least 1 month prior to the onset of immunosuppression (after seeking specialist advice). Serological confirmation of previous VZV infection is recommended prior to vaccination.

### Table. Guide to safe doses of immunosuppressive therapy concurrent with Zostavax® administration

<table>
<thead>
<tr>
<th>Mechanism of Action</th>
<th>Examples</th>
<th>Safe Dose*</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-TNF</td>
<td>Etanercept, Infliximab, Adalimumab</td>
<td>None</td>
<td>Immunise 1 month prior to treatment initiation OR 12 months post-treatment cessation</td>
</tr>
<tr>
<td>IL-1 inhibition</td>
<td>Anakinra</td>
<td>None</td>
<td></td>
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<tr>
<td>Co-stimulation blockade</td>
<td>Abatacept</td>
<td>None</td>
<td></td>
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<tr>
<td>B-cell Depletion/Inhibition</td>
<td>Rituximab</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>Immuno-modulators (Antimetabolites)</td>
<td>Azathioprine, 6-Mercaptopurine, Methotrexate</td>
<td>≤ 3.0 mg/kg/day</td>
<td>If on higher dose, immunise 1 month prior to treatment initiation OR 3 months post-cessation refer to Immunisation handbook and NCIRS factsheet</td>
</tr>
<tr>
<td></td>
<td>Prednisone</td>
<td>≤ 1.5 mg/kg/day</td>
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<td></td>
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<td>≤ 0.4 mg/kg/week</td>
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<tr>
<td></td>
<td></td>
<td>&lt; 20 mg.kg for &lt;14 days</td>
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<tr>
<td>Corticosteroids</td>
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<tr>
<td>T-cell activation inhibition</td>
<td>Tacrolimus, Cyclosporine</td>
<td>None</td>
<td>Immunise 1 month prior to treatment initiation OR 3 months post-cessation</td>
</tr>
<tr>
<td>Others</td>
<td>Cyclophosphamide, Mycophenolate, Sulfasalazine</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

*See Australian Immunisation Handbook, Chapters 3.3.3 and 4.24
Interval between cessation of therapy and vaccination

The interval between ceasing biological or non-biological immune modulating therapy and administration of herpes zoster vaccine is variable (see Table) depending on the therapy given. At least 6 months after the end of chemotherapy and radiation treatment is recommended.

What if zoster vaccine has been inadvertently given to an immunocompromised patient?

Seek immediate specialist advice to determine if the patient is severely immunocompromised. The patient will likely need close monitoring for adverse effects related to vaccine virus-associated disease and may require antiviral therapy.

Further Information

National Centre for Immunisation Research & Surveillance fact sheets:


Meningococcal serogroup W and Y disease on the rise

Ros Webby, Centre for Disease Control, Darwin

Meningococcal disease is a rare but serious disease. It can cause blood poisoning (septicaemia) or inflammation of the lining of the brain and spinal cord (meningitis).

Complications from meningococcal infection include limb deformity/loss, skin scarring, deafness, neurological deficits and death.

There are several serogroups of meningococcal disease that cause invasive disease in Australia (A, B, C, W, X, Y). There are safe and effective vaccines for A, B, C, W, Y.

Which serogroups most commonly cause meningococcal disease in Australia?

Overall the number of cases of invasive meningococcal disease (IMD) is low in Australia with the predominant meningococcal serogroup from 2002 to 2015 being serogroup B. However in 2016, serogroup W disease became the most predominant meningococcal serogroup. IMD caused by meningococcal serogroup Y disease has also increased in 2016 and 2017. Meningococcal serogroup C disease has decreased since 2005 with the introduction of the meningococcal C vaccine at 12 months of age on the National Immunisation Program.

What are the symptoms of meningococcal disease?

The symptoms of meningococcal disease can include fever, headache, neck stiffness, muscle or joint pains, drowsiness, confusion, nausea and vomiting, and a rash (usually red-purple spots or bruises that appear late in the illness).

Common symptoms of meningococcal disease in babies include fever, rapid breathing, rash, vomiting, irritability, drowsiness, leg pain and altered skin colour.

Atypical IMD presentations such as septic arthritis, pneumonia, and epiglottitis occur more commonly with meningococcal W disease.

If IMD is suspected, early treatment with appropriate antibiotics is important with urgent referral to hospital for definite diagnosis and treatment. Please notify the Centre for Disease Control (CDC) for all suspected cases. For all confirmed cases, CDC will undertake an appropriate public health response and identify contacts that require clearance antibiotics and where appropriate vaccination.
How do you get meningococcal disease?

Meningococcal bacteria are only found in humans. The bacteria, *Neisseria meningitidis*, are carried in the throat by up to 10 per cent of the population without developing disease. These people are ‘carriers’ of the bacteria and can pass it on to others. The bacteria are spread by close contact with respiratory secretions such as kissing, sneezing and coughing. Adolescents have the highest rate of carriage of the bacteria, with carriage peaking at 19 years. This age group therefore plays an important role in transmission of the bacteria to others. As the carriage rate varies with age, around 3% of carriage is seen in children younger than 4 years and up to 24–37% in those 15–24 year olds, with a decrease in carriage to less than 10% in older age groups.

What is the epidemiology of meningococcal serogroup W and Y disease?

In 2016 the number of cases of meningococcal W cases increased across Australia with a doubling or tripling of cases in the last 2 years. Cases of serotype Y meningococcal disease have also increased nationally since 2014. There were 14 deaths due to meningococcal W in Australia in 2015 and 2016 which accounted for 60% of all meningococcal deaths. The NT has had 1 case of meningococcal W infection and 1 case of meningococcal Y infection in 2016 - early 2017.

The current strains of meningococcal W disease appear to spread more easily and cause more severe disease than other serogroups currently circulating.

The highest rates of meningococcal disease occur in young infants and in adolescents (15–19 years) but meningococcal W cases have occurred in middle age and older adults including those 65 years and over.

What vaccine will prevent meningococcal W and Y disease?

The meningococcal A,C,W,Y or ‘quadrivalent’ vaccine protects against 4 group types of meningococcal bacteria: A, C, W and Y. The meningococcal A, C, W, Y vaccine is not on the National Immunisation Program and is available on private prescription. This vaccine is usually recommended for those with high risk medical conditions such as asplenia or non-functioning spleen, complement disorders, HIV infection and haematological malignancy or for travellers to areas with high incidence of meningococcal A,C,W,Y infection such as in Africa or those going to the Hajj.

Conjugate A,C,W,Y vaccines are preferred to polysaccharide A,C,W,Y vaccines as they are more immunogenic in children and have a greater antibody response and immunological memory.

There are 3 brands of conjugate meningococcal A,C,W,Y vaccine—Menveo®, Menactra® and Nimenrix®.

Menveo® brand is the only brand that can be used in children from 2–12 months of age. Children aged 2–23 months may require more than 1 dose of vaccine to complete the primary series (see Table). People aged 2 years and over require a single dose of vaccine but may require a booster in 3–5 years if there is an ongoing risk of infection.

How long does it take for immunity to develop after having the vaccine?

It takes approximately 1 month for immunity to develop. The meningococcal A,C,W,Y vaccine is 80–85% effective in adolescents/adults and more than 90% effective in infants.

Are the vaccines safe?

Yes, the vaccines are very safe. Most vaccines can cause mild reactions which are usually short lasting and do not need medical treatment. Adverse reactions are rare. Common side effects are injection site pain, redness and swelling, myalgia, drowsiness, headache, decreased appetite, nausea and fever.

How do you give the vaccine?

The dose of all meningococcal conjugate vaccines is 0.5 mL, to be given by IM injection. Menveo® brand and Nimenrix® must be reconstituted.
### Contraindications

Anaphylaxis to any of the vaccine constituents or anaphylaxis following a previous dose of any meningococcal vaccine.

### Is there an NT vaccination program?

At this stage, targeted vaccination programs with meningococcal A,C,W,Y vaccine are being undertaken around cases of meningococcal A,C,W,Y disease. The existing childhood meningococcal vaccination program against serogroup C will continue at 12 months of age. An adolescent vaccination program in the NT is currently being considered. An A,C,W,Y vaccine replacing the ‘serogroup C-only’ coverage at 12 months of age is also being considered.

### Further information

For further information about surveillance and epidemiology of meningococcal W disease see:


For further information about meningococcal vaccination see:


### References


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### Table. Recommended use of conjugate meningococcal A,C,W,Y vaccine by age group

<table>
<thead>
<tr>
<th>Age at commencement of vaccine course</th>
<th>Recommended brand</th>
<th>Primary immunisation</th>
<th>Recommended interval between primary doses</th>
<th>Age to give booster dose if required</th>
</tr>
</thead>
<tbody>
<tr>
<td>2–6 months</td>
<td>Menveo®</td>
<td>3 doses</td>
<td>8 weeks</td>
<td>12–18 months</td>
</tr>
<tr>
<td>7–11 months</td>
<td>Menveo®</td>
<td>2 doses</td>
<td>12 weeks</td>
<td>3 years</td>
</tr>
<tr>
<td>12–23 months</td>
<td>Either Menveo®</td>
<td>2 doses</td>
<td>12 weeks</td>
<td>3 years</td>
</tr>
<tr>
<td></td>
<td>Or Nimenrix®</td>
<td>1 dose</td>
<td>Not applicable</td>
<td>3 years</td>
</tr>
<tr>
<td>2–6 years</td>
<td>Menactra®, Menveo®, or Nimenrix®</td>
<td>1 dose</td>
<td>Not applicable</td>
<td>3 years</td>
</tr>
<tr>
<td>≥ 7 years</td>
<td>Menactra®, Menveo®, or Nimenrix®</td>
<td>1 dose</td>
<td>Not applicable</td>
<td>5 years</td>
</tr>
</tbody>
</table>

Source: Australian Immunisation Handbook 3
As of 28 February 2017, a total of 452 cases of infectious syphilis had been reported in the Northern Territory (NT) since an outbreak was declared in mid-2014 among Aboriginal and Torres Strait Islander people living largely in remote and rural areas. Until January 2017, outbreak zones included Alice Springs, Barkly, Katherine and East Arnhem. However there have been multiple infectious syphilis cases consistent with outbreak cases reported in urban Darwin and the wider Darwin Region recently, making the entire NT, now, since January 2017, an outbreak region (see Figure 1).

Most outbreak cases have been reported in young people, predominately aged between 15 and 29 years. There have been 225 females diagnosed with infectious syphilis of whom at least 16% were pregnant. There have been 3 cases of congenital syphilis reported in the NT that occurred in 2014 as part of the outbreak.

Prevention, as well as control, requires dynamic approaches to achieve early diagnosis and treatment of syphilis. Treatment of sexual contacts of those with newly acquired syphilis is crucial in controlling the current outbreak and it is the most challenging task. Early detection of pregnancy in this population and frequent testing during pregnancy needs to occur to successfully identity and treat syphilis to protect the mother-to-be and the unborn child. This approach is essential to prevent further cases of congenital syphilis.

Syphilis also increases the risk of acquisition and transmission of HIV. It is critical to ensure that all of those with sexually transmitted infections, especially and including syphilis, are tested for HIV.
Testing for TB in a changing refugee demographic
Rowena Boyd, Centre for Disease Control, Darwin

Abstract

Over the past 4 years, the demographic of newly arrived refugees in the Northern Territory (NT) has changed from predominately African to Middle Eastern background. This study aimed to identify if there has been an associated change in Mantoux positivity. Proportions of Mantoux positive results where compared by year of arrival and TB incidence in the country of birth.

Between 2013 and 2016, the proportion of newly arrived refugees with a positive Mantoux result decreased significantly from 44% (78/177) to 24% (32/134) of people tested. While Mantoux positivity has decreased substantially, the overall prevalence remains high with 1 in 4 refugees affected. With risk of TB reactivation highest in the first years following immigration, timely testing of newly arrived refugees should continue.

Of note, the proportion of people testing positive was similar regardless of country of birth and TB incidence in the country of origin. Countries of birth therefore do not currently serve as a predictor of Mantoux positivity in the NT refugee population. Other factors such as time spent in refugee camps should be assessed to identify TB exposure and risk.

Key words: Latent tuberculosis infection; Mantoux test; Refugee health

Introduction

Exposure to active TB disease “triggers an immune response that contains the infection before it progresses to active disease, but live bacilli may persist for many years in a dormant state known as latent TB infection (LTBI). This immune response can be detected by the Mantoux test.”1 Roughly 1 in 10 people with LTBI will progress to active TB disease in their lifetime.2

Since 2013 the number of refugees arriving in the Northern Territory (NT) has fluctuated and the country of origin has changed to countries with lower TB incidence. This study aims to identify the effect that this changing demographic has on the proportion of refugees with positive Mantoux results.3 A secondary aim is to determine if TB incidence in the country of birth predicted the likelihood of Mantoux positivity.

Methods

Following arrival in the NT refugees receive Mantoux testing to assess LTBI as part of the refugee health assessment. Mantoux results, arrival dates and country of birth are received from refugee health services and entered on a centralised database at the NT Centre for Disease Control (CDC). People with a positive Mantoux undergo radiological and clinical assessment.

A Mantoux was considered positive if it measured 10mm and above, regardless of age. For children under 5 years of age, a Mantoux of 5mm or greater was considered positive.1

The chi squared statistic was used to compare proportions, with logistical regression analysis performed in Stata 13.1.

Results

The database recorded 417 refugees allocated to the NT between 2013 and 2016. Of these, 379 people received Mantoux testing. The remaining 38 moved interstate before testing or never arrived in the NT and were therefore excluded from the analysis. Over the 4 years, 126/379 (33%) had a positive Mantoux and 3 were subsequently diagnosed with active TB disease.

Figure 1 shows the majority of arrivals occurred in 2013 and 2016, with a marked decrease in arrivals in 2014 and 2015. Since 2013, the region of origin of the refugees has changed from predominately Central Africa to the Middle East. The Democratic Republic of the Congo, Kenya, Somalia and Sudan were the most commonly represented countries of birth among the African cohort. For the Middle East, representation has moved from a predominately Afghani cohort in 2013 to Iraqi and Syrian in 2016.
From 2013 to 2016, a declining trend in the proportion of people with a positive Mantoux is noted (Figure 1). Logistical regression analysis shows this declining trend in Mantoux positivity was statistically significant over time (Table 1).

Table 2 shows the association between Mantoux positivity and TB incidence in the country of birth as reported by the World Health Organisation (WHO) TB country profiles. While the odds of returning a positive Mantoux result was 1.5 to 2 times higher in refugees born in countries where TB cases were reported as 25 per 100 000 population or more, compared to countries where TB case incidence was less than 25 per 100 000, this finding was not statistically significant.

**Discussion**

With a change in demographic, the percentage of refugees with evidence of previous exposure to TB disease has halved. Prevalence of infection remains high however with 1 in 4 arriving refugees testing Mantoux positive. Thus TB testing should continue to be recommended as a part of refugee health assessment, providing an opportunity to detect cases of active TB disease and to identify LTBI and offer LTBI treatment to prevent progression to active TB in the future. Timely testing for LTBI is particularly indicated as the risk of progression to active TB is high within the first 2 years following immigration.

In 2016, there was an increase in the number of people who did not receive Mantoux testing (37 people) compared to previous years (0 to 1 person). This appears to be due to a change in reporting practice in 2016 that was better able to capture the number of refugees assigned to the NT but who never arrived in the NT or moved interstate within a short time frame.

*Note: for the purposes of our study the broader definition of the Middle East has been used which includes Afghanistan

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**Table 1. Mantoux positivity by year of arrival in Australia**

<table>
<thead>
<tr>
<th>Year of arrival</th>
<th>Total refugees</th>
<th>Mantoux Positive</th>
<th>Mantoux Negative</th>
<th>Not tested*</th>
<th>% positive †</th>
<th>Odds ratio (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>2013</td>
<td>178</td>
<td>78</td>
<td>99</td>
<td>1</td>
<td>78/177 (44%)</td>
<td>Reference</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>2014</td>
<td>30</td>
<td>9</td>
<td>21</td>
<td>0</td>
<td>9/30 (30%)</td>
<td>0.5 (0.2 – 1.3)</td>
<td></td>
</tr>
<tr>
<td>2015</td>
<td>38</td>
<td>7</td>
<td>31</td>
<td>0</td>
<td>7/38 (18%)</td>
<td>0.3 (0.1 – 0.7)</td>
<td></td>
</tr>
<tr>
<td>2016</td>
<td>171</td>
<td>32</td>
<td>102</td>
<td>37</td>
<td>32/134 (24%)</td>
<td>0.4 (0.2 – 0.7)</td>
<td></td>
</tr>
</tbody>
</table>

*Not included in analysis; † Calculated by dividing Mantoux positive by the total number of people tested
A smaller proportion of refugees tested Mantoux positive in 2016 compared to 2013. Concurrently there was a change in refugee demographic from Central African countries and Afghanistan which have higher TB incidence (>100 cases per 100 000 persons) to the Middle Eastern countries of Syria and Iraq which have lower incidence (<50 cases per 100 000 persons). Mantoux positivity was not significantly correlated to TB incidence in the country of birth, a finding that may be limited by lack of data on an individual’s time spent in other countries. Additionally, regional turmoil in areas refugees are fleeing may contribute to less functional health services and disease surveillance leading to greater prevalence of disease in countries such as Syria, previously associated with low TB incidence. The country of origin, therefore, does not appear to be a predictor of Mantoux positivity in refugees arriving in Australia, further supporting the need for continued TB testing of all newly arrived refugees.

**Acknowledgements**

I would like to acknowledge the hard work and dedication of staff at Melaleuca Refugee Centre, who tirelessly help newly arrived refugees in the NT settle-in and navigate their new environment. Acknowledgement also goes to the providers of refugee health assessments and management in the NT including Vanderlin Drive Surgery (prior to July 2016), Arafura Medical Clinics (from July 2016), Top End Medical Centres (from July 2016) and the NT refugee health program coordinator and co-workers at TB Unit CDC Darwin.

**References**


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**Table 2. Mantoux positivity by TB incidence in the country of birth for 2013 to 2016**

<table>
<thead>
<tr>
<th>TB incidence in country of birth per 100 000 population</th>
<th>Total refugees n=417</th>
<th>Mantoux positive</th>
<th>Mantoux negative</th>
<th>Not tested</th>
<th>% positive*</th>
<th>Odds ratio (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 to 24a</td>
<td>45</td>
<td>10</td>
<td>33</td>
<td>2</td>
<td>10/43 (23%)</td>
<td>Reference</td>
<td>0.53</td>
</tr>
<tr>
<td>25 to 99b</td>
<td>77</td>
<td>15</td>
<td>33</td>
<td>29</td>
<td>15/48 (31%)</td>
<td>1.5 (0.6–3.8)</td>
<td></td>
</tr>
<tr>
<td>100 to 199c</td>
<td>78</td>
<td>28</td>
<td>46</td>
<td>4</td>
<td>28/74 (38%)</td>
<td>2.0 (0.9–4.7)</td>
<td></td>
</tr>
<tr>
<td>200 to 299d</td>
<td>40</td>
<td>14</td>
<td>23</td>
<td>3</td>
<td>14/37 (38%)</td>
<td>2.0 (0.8–5.3)</td>
<td></td>
</tr>
<tr>
<td>≥ 300e</td>
<td>177</td>
<td>59</td>
<td>118</td>
<td>0</td>
<td>59/177 (33%)</td>
<td>1.7 (0.8–3.6)</td>
<td></td>
</tr>
</tbody>
</table>

*Egypt, Iran, Jordan, Lebanon, Syria; b Sri Lanka, Sudan, Iraq; c Burundi, Afghanistan, Bhutan, Ethiopia, Malawi, Nepal, Thailand; d Bangladesh, Kenya, Pakistan, Somalia; e Democratic Republic of the Congo, Namibia, Botswana, Zambia, Burma

* Calculated by dividing Mantoux positive by the total number of people tested
The last 5%: Elimination of trachoma from the Northern Territory

Gabrielle Watt, Centre for Disease Control, Darwin

Abstract

The elimination of trachoma from a number of low to middle income nations, including Morocco, in recent years is heartening. However Australia remains the only high-income country to still have endemic trachoma. Much has been achieved towards eliminating trachoma from Australia by 2020 largely due to screening, treatment and promoting facial cleanliness. However, there are still fundamental environmental issues that need to be addressed if trachoma is to be eliminated.

Key words: trachoma; elimination; hygiene; housing

Introduction

On 15 November 2016 Morocco was recognised by the World Health Organisation (WHO) as having eliminated trachoma as a public health problem. Oman, China, Gambia, Ghana, Iran and Myanmar are currently awaiting validation by the WHO for elimination of trachoma. However, trachoma continues to remain endemic in some areas of Australia and the push for elimination of trachoma by 2020 will, indeed, require serious pushing.

Defining elimination

In 2016 the WHO released a document titled ‘Validation of Elimination of Trachoma as a Public Health Problem.’ This document provides detailed guidance to countries that have had endemic trachoma regarding the elimination criteria. Interpreted in our context, elimination of trachoma in Australian requires the following:

- <5% prevalence of trachomatous inflammation-follicular (TF) in 1–9 year old children in each region (Northern Territory (NT) regions: Central Australia, Barkly, Katherine, Greater Darwin and East Arnhem)
- Trachomatous trichiasis ‘unknown to the health system’ of <1 case per 1000 population
- Active campaigns for promoting facial cleanliness
- Environmental health improvements, including improving access to sanitation in trachoma-endemic areas.

Successes to date

The NT-wide trachoma rate has plummeted in recent years from 14% in 2009 to 4% in 2012. This significant decline has been largely due to wide-spread distribution of single dose azithromycin to prevent trachoma spread in endemic communities. Productive engagement with clinical activities that control trachoma (screening and treatment) have been seen in individuals, communities, health care services and across government and non-government sectors. The intensive clinical work undertaken by this collaboration has led to great success in a short period of time. However since 2013 NT-wide trachoma rates have plateaued with screening continuing to identify some individual communities with extremely high prevalence rates. In 2016* community prevalence rates of active trachoma ranged from 0–38% with rates in Central Australia and the Barkly regions higher than those seen in the Greater Darwin and East Arnhem regions. The reasons for this may include reduced access to washing facilities and the dry, dusty climate, both which directly influence the ability to keep children’s faces clean.

This plateau in NT-wide rates, along with the high prevalence rates seen in some communities, suggests that clinical treatment alone is not sufficient to eliminate trachoma across all regions.

Tackling the hard yards

Morocco reports that they achieved elimination of trachoma as a public health problem through the provision of both clinical services and improved hygiene environments. In addition to providing mass single dose azithromycin within communities, health education was provided and significant improvements were made to rural water supply and sanitation. The WHO recognises the critical importance clean faces and environments that promote good face and

*All 2016 data are provisional
hand washing play in preventing the transmission of trachoma and in achieving elimination.

In Australia in recent years the focus has been primarily on the clinical management of trachoma, however, increased focus must be given to environments that support good face and hand washing to gain further improvements. This includes safe, functioning bathrooms in homes, schools and the community. When children live in houses that do not enable routine hand and face washing and easy access to bathe and to shower, due to overcrowding or through non-functioning bathrooms, they are at much higher risk of a number of hygiene-related illnesses including skin diseases (e.g. scabies and impetigo), rheumatic heart disease and invasive Group A streptococcal disease.4

The necessity to work together to improve environmental conditions for children in remote communities has been recognised by health care providers for a very long time. In early February 2017 the NT Trachoma Program organised a ‘Hygiene meeting’ in Alice Springs to discuss the prevention of hygiene-related illnesses. The response to this meeting was overwhelmingly supportive with representatives from the NT Departments of Housing, Education, and Health, regional councils, the Aboriginal Medical Services Alliance of the Northern Territory, numerous health programs and several other organisations attending. There was a strong commitment at the meeting to working together to promote improved hygiene conditions in remote communities. This enthusiasm provides a strong platform from which to launch concerted efforts to improve the environment and to increase understanding of the importance of good hygiene to prevent many diseases in remote communities.

Providing comprehensive clinical care for trachoma is not easy but the real challenge lies in working with families, the housing authorities and councils to ensure that homes have functioning washing facilities with clear pathways for maintenance. Additionally understanding of the need to support routine face washing in all remote communities is ongoing. The work may be challenging but the benefits are enormous. Knowing that Morocco, Oman, China, Gambia, Ghana, Iran and Myanmar have successfully eliminated trachoma is motivation to know it can be done.

References
Diagnosis of acute rheumatic fever (ARF) continues to be challenging because of 1) its rarity outside certain endemic regions or certain risk groups such as Northern Territory (NT) remote Indigenous communities, 2) the lack of sensitivity and specificity of clinical features, and 3) the absence of a diagnostic blood test. In 2014 Rheumatic Heart Disease Australia (RHDA), a Menzies-based, Commonwealth-funded National Coordinating Unit for supporting ARF and rheumatic heart disease (RHD) in Australia, sought to minimise ARF diagnostic complexities by developing a technology-based ARF diagnosis calculator.

The original project brief for the diagnosis calculator was to provide a simple and intuitive tool to embed the complex ARF diagnosis algorithm into a series of simple questions that could assist clinicians to diagnose ARF. To date there have been over 5500 iOS and Android downloads of the app (see Figure 1).

Since the initial launch of the diagnostic calculator there has been increasing ARF/RHD activity both in Australia and internationally. RHDA continues to coordinate education activities across Australia which have provided a platform for urban and remote clinicians to learn about ARF/RHD and give user feedback on the educational resources, including use of the diagnosis calculator in high and low risk settings. In 2015 the American Heart Association (AHA) revised the Jones diagnostic criteria for ARF to include a high/low risk tiered approach to ARF diagnosis. These changes were proposed by an international working group including researchers and medical professionals from Australia and were endorsed by the World Heart Federation (WHF). The changes were based on those already made for Australian ARF/RHD guidelines. In 2016 the Australian Medical Association (AMA) highlighted data showing that ARF/RHD, while disproportionately impacting people living in remote Australia, also has a significant impact in urban Queensland and NSW. The AMA put the focus on ARF/RHD for 2017 and outlined a plan to prevent new cases of RHD by the year 2031.

A changing understanding of national ARF/RHD epidemiology and the recent changes to the AHA’s diagnostic criteria which aligned a tiered approach to ARF diagnosis with the Australian guidelines prompted the diagnosis calculator working group to update the app. After piloting changes and incorporating end-user clinician feedback, the group expanded the functionality of the ARF diagnosis calculator to support clinicians globally to diagnose ARF in both high risk and low risk patient populations.

The first step was to develop and test an algorithm that calculates patient risk. This was achieved by providing clinicians with a series of 5 weighted questions (Figure 2). The questions are designed to simplify but maintain the integrity of the definition of high/low risk endorsed by the Australian and AHA guidelines (Figure 3).

Upon answering the 5 questions the patient is assigned a score which determines risk group and therefore which algorithm the diagnosis calculator uses.
If the patient scores 2 points or greater they are defined as high risk and directed to the high risk algorithm (Figure 4). If the patient scores 1 point they are assigned as intermediate risk and the clinician is prompted to make a decision based upon the individual case history and using the definition endorsed by the Australian and AHA guidelines (Figure 3). If the patient scores 0 they are defined as low risk (Figure 4).

While updating the app to incorporate a risk calculator, it was noted that the newly endorsed AHA low risk Jones criteria differ slightly from the current 2012 Australian guidelines. The working group considered the changes and opted to update the diagnosis calculator low risk algorithm to be entirely consistent with the 2015 AHA and WHF endorsed changes. The changes are minor and only affect the minor manifestations in the low risk diagnosis criteria (Figure 5). For global consistency it is anticipated that these endorsed changes will be adopted in the proposed 2017–2018 update of the Australian ARF/RHD guidelines. In conclusion, the new upgrades to the diagnosis calculator now effectively embed 3 separate algorithms into 1 tool and the benefits to assist diagnosis are numerous. The main benefit is that a complex diagnosis algorithm can be embedded into a simple series of questions, which minimises misinterpretation and error. This tool now provides an ARF diagnosis calculator that has the potential to assist clinicians to identify ARF in Australian high and low risk patients, and to assist clinicians globally to follow the AHA and WHF endorsed ARF diagnosis criteria.
The App is FREE and available by searching ‘rhdaustralia’ on google play or App store.

Acknowledgements

Bart Currie, Anna Ralph, Craig Boutlis, Kate Hardie, Joshua Francis, Claire Boardman and Vortilla Digital

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A summary of enteric disease in the Northern Territory in 2016

Anthony Draper1,2
1. OzFoodNet Epidemiologist, CDC, Darwin. 
2. National Centre for Epidemiology and Population Health, Australian National University.

Abstract

In 2016 there was a 24% increase in notifications of foodborne disease in the Northern Territory compared to 2015. There was a record number of salmonellosis notifications accounting for 51% of all foodborne disease notifications. Most of the increase in salmonellosis was due to an increase in S. Saintpaul associated with contaminated bean sprouts. Salmonellosis, campylobacteriosis, shigellosis and yersiniosis notifications were all higher than the 5 year mean. There were 10 foodborne and 14 non-foodborne disease outbreaks investigated in 2016.

Key words: OzFoodNet; salmonellosis; typhoid; shigellosis; outbreak; campylobacteriosis; cryptosporidiosis; foodborne disease; Northern Territory

Introduction

There has been an OzFoodNet epidemiologist in the Northern Territory (NT) since 2003. The position is based at the Centre for Disease Control (Darwin) within the Department of Health and is funded by the Australian Government. The purpose of the position is to enhance enteric and foodborne disease surveillance in the NT and to investigate outbreaks of foodborne and non-foodborne enteric disease.

Methods

Data were extracted from the NT Notifiable Diseases System (NTNDS) and analysed from the data warehouse using Business Objects and Intercooled Stata. Population figures were obtained from the NT Department of Health’s Health Gains Planning population data.1

Results and Discussion

In 2016 there were 1400 notifications of foodborne or potentially foodborne disease* reported in the NT which was 43% more than the 5 year mean (980) and 24% more than the previous year (1126). Salmonellosis notifications accounted for 51% of all foodborne disease notifications in the NT, followed by campylobacteriosis (35%) and shigellosis (13%).

There were 347 non-foodborne enteric disease† notifications reported in the NT in 2016 which was slightly (6%) more than the 5 year mean

*This includes total number of notifications for amoebiasis, botulism, brucellosis, campylobacteriosis, cholera, salmonellosis, shigellosis, STEC/VTEC, typhoid, yersiniosis, ciguatera, Vibrio food poisoning, and listeriosis. It does not include rotavirus, cryptosporidiosis, hepatitis A and hepatitis E.
† This includes notifications of rotavirus, cryptosporidiosis, hepatitis A and hepatitis E.
Cryptosporidiosis notifications (293) accounted for 84% of these non-foodborne disease notifications. There were 10 foodborne or suspected foodborne outbreaks and 14 non-foodborne outbreaks investigated in 2016.

**Salmonellosis**

The NT recorded the highest number of salmonellosis notifications ever (709) in 2016 which was 37% higher than the 5 year mean (519) and 24% more than the previous year (570). The majority of this increase was due to multiple outbreaks caused by *Salmonella* Saintpaul which was associated with contaminated bean sprouts. This occurred in April and May 2016 (Figure 1). These outbreaks were associated with Asian foods, particularly laksa where bean sprouts were used as a garnish or added late in the cooking process and with food premises that sold salads containing bean sprouts. These bean sprouts were traced back to a single interstate supplier. In 2016 there were 147 notifications of *S*. Saintpaul compared to a 5 year mean of 71.

Almost half of all salmonellosis notifications (290, 41%) came from the 0–4 year age group where the rate of disease was 152 cases per 100,000. There was no statistically significant difference in the rates of disease between Indigenous and non-Indigenous Territorians.

In 2016, 91% (646/709) of salmonellosis notifications were identified to the serovar level. Following *S*. Saintpaul (n=147) the next most commonly notified serovars were *S*. Virchow (n=73), *S*. Typhimurium (n=52) and *S*. Ball (n=27).

*S*. Ball, *S*. Virchow, *S*. Lansing, *S*. Reading and *S*. Saintpaul are usually the most commonly notified serovars in the NT (Figure 2) and are considered ‘environmental’ serovars. These environmental serovars are typically carried in the faeces of domestic and native animals that contaminate the environment. Young children are more at risk of becoming ill with environmental *Salmonella* by putting contaminated hands, objects or toys in their mouths, particularly during the hotter and more humid months.

**Campylobacteriosis**

In 2016 there were 465 notifications of campylobacteriosis in the NT which was 51% greater than the 5 year mean of 308 and 19% more than the previous year (390). The overall rate of campylobacteriosis was 186 cases per 100,000 which is 1.24 times the national rate (150 cases per 100,000). The median age of campylobacteriosis cases was 20 years (range 0–79 years). There has been an increase in the recorded rate of campylobacteriosis nationwide over the past few years (Figure 3) due largely to

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**Figure 1.** Salmonellosis notifications by month, Northern Territory, 2012–2016.
the introduction of more sensitive, culture independent testing.

We conducted a study over the 2015/2016 wet season whereby we sent an SMS to all people notified with campylobacteriosis in Darwin to ascertain restaurants, takeaways and overseas destinations that were visited prior to becoming ill. In total we sent 97 messages and the response rate was 49% (48/97). Of those who responded, 42% (20/48) reported travelling overseas prior to becoming ill. We did not detect any associated outbreaks.

The highest number of cases and rate of disease was seen in the 0–4 year age group with 125 notifications and a rate of 647 cases per 100 000. This age group represents 37% of all campylobacteriosis notifications in the NT. The rate of disease in this age group was significantly higher in the Indigenous population (1262 cases per 100 000) than the non-Indigenous population (240 cases per 100 000) with a rate ratio (RR) of 5.2 (95% CI 3.4-8.3, p = <0.01). In the remaining population (>4 years old), the non-Indigenous rate of campylobacteriosis (158 cases per 100 000) was significantly higher than the Indigenous rate (55 cases per 100 000) with a RR of 5.3 (95% CI 3.4-8.3, p = <0.01). In the total population, the rate of campylobacteriosis was 1.35 (95% CI 1.1-1.6, p<0.01) times higher for males (212 per 100 000) compared to females (157 per 100 000).

**Shigellosis**

There were 196 notifications of shigellosis notified in the NT which was 45% higher than the 5 year mean (135) and 35% higher than the number of notifications received in 2015 (145). The overall rate of shigellosis was 78 cases per 100 000 population which is over 13 times the national rate of 5.9 cases per 100 000 (Figure 4). Of the shigellosis notifications that were identified to the species level (87/196, 44%), *Shigella sonnei* biotype a (57) was the predominant biotype.

The highest number of cases (85) and rate of disease was seen in the 0–4 year age group, with 440 cases per 100 000 population. Of the 85 cases recorded in this age group, 77 (91%) were Indigenous. Shigellosis is spread through the oral-faecal route which is exacerbated by poor living conditions and poor hygiene. Shigellosis is historically more commonly reported in the Indigenous population and in those returning

![Figure 2. Notifications of selected Salmonella serovars, Northern Territory, 2012–2016.](image_url)
from travel to developing countries. The rate of shigellosis in the Indigenous population was 195 cases per 100 000 population (145) compared to 27 cases per 100 000 population in the non-Indigenous population (48). This represents a RR of 7.1 (95% CI 5.1–10.1, \( p<0.01 \)). Of the 48 non-Indigenous shigellosis notifications, 20 were known to have acquired their infection overseas, with 13/20 acquiring their infections in Indonesia.

As in previous years, there were higher rates of disease seen among older Indigenous people, particularly females (Figure 5). This may be a reflection of cultural practices where aunts and grandmothers are often involved in the care of young children. Overall though, the rate of disease did not vary between the sexes, with 80 cases per 100 000 in females (95) vs. 76 cases per 100 000 in males (101), RR 1.1 (95% CI 0.8–1.4, \( p=0.72 \)).

**Cryptosporidiosis**

There were 293 notifications of cryptosporidiosis which was almost 2.5 times more than 2015 (121) and 75% more than the 5 year mean of 167. Cryptosporidiosis was predominantly reported in children, with 221 of the 293 (75%) cases notified being in the 0–9 year age group.

The rate of disease was significantly higher in the Indigenous population with 165 cases per 100 000 (123) compared to 84 cases per 100 000 (148) in the non-Indigenous population (RR 2.0, 95% CI 1.5–2.5, \( p<0.01 \)). The rate of disease in the 0–4 year age group was also significantly higher in Indigenous children (1359 cases per 100 000, 98) than non-Indigenous children (736 cases per 100 000, 89), (RR 1.8, 95% CI 1.4–2.5, \( p<0.01 \)).

Cryptosporidiosis notifications typically peak in summer months and there was an increase in cases in Central Australia in early 2016 which extended until May. There was also a cluster of 3 cryptosporidiosis cases in family members who used a communal pool at an apartment complex.

**Typhoid**

There was 1 case of typhoid notified in 2016, acquired in Bangladesh.

The NT has averaged 2 cases of typhoid per year since 2007, all in people returning from typhoid endemic countries. Since 2012 all notified cases were either returning to their birth countries or visiting family members in typhoid endemic countries. This type of traveller is less likely to seek pre-travel health advice and seek vaccination.\(^6,7,8\)
In 2016, paratyphoid fever became a notifiable disease in its own right. Previously, infections caused by *Salmonella Paratyphi*, *Salmonella Paratyphi A* and *Salmonella Paratyphi B* were notified as salmonellosis.

There were 3 cases of paratyphoid notified in 2016. One was acquired in India (*S. Paratyphi A*) and there were 2 locally acquired cases of *S. Paratyphi B*. Paratyphoid is not a vaccine preventable disease.

There were no cases of hepatitis A notified in the NT in 2016. Since 2006, nearly all cases of hepatitis A reported in the NT have been overseas acquired; many of the cases are people living in Australia who have returned to their country of birth for a holiday or to visit family or, in irregular maritime arrivals arriving via boat from Indonesia. Since the introduction of the funded vaccine for all Indigenous children under 5 years of age in the NT (along with South Australia (SA) and Western Australia) in

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**Hepatitis A**

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November 2005, there has been a decline in the number of hepatitis A notifications in both the Indigenous and non-Indigenous population. There have been only 4 cases of locally acquired hepatitis A reported in the NT since 2006, the last one being in 2010.

**Hepatitis E**

There was one case of hepatitis E notified in the NT in 2016 in a returned traveller from India. This was the first case of hepatitis E notified in the NT since 2008.

**Listeriosis**

Listeriosis is not often reported in the NT and in 2016 no cases were notified.

**Amoebiasis**

There were 3 cases of amoebiasis notified in 2016:

- A 38 year old man had Entamoeba histolytica detected in faeces by polymerase chain reaction (PCR) and microscopy. No extraintestinal spread was noted and his last overseas travel was in 2013 to Nepal.
- A 73 year old non-Indigenous male with hepatic abscesses who regularly travelled to Bali, Indonesia was diagnosed by serological methods.
- A 54 year old non-Indigenous male was faeces PCR positive for E. histolytica with no extraintestinal spread. He regularly travelled to Dili in Timor-leste.

**Ciguatera**

There was 1 case of ciguatera fish poisoning notified in 2016. A 24 year old Indigenous female in Arnhem Land reported abdominal pain, vomiting, bradycardia, body aches and tingling after eating kingfish caught in a local estuary. Ciguatera is diagnosed on a spectrum of clinical symptoms rather than laboratory tests. No fish remained for testing.

Ciguatera was made a notifiable disease in the NT in 2010 and only 2 cases have been notified since (1 in 2013, 1 in 2016).

**Vibrio food poisoning**

There were no cases of Vibrio food poisoning notified in the NT in 2016.

**Shigatoxin producing Eschericia coli (STEC)/Haemolytic uraemic syndrome (HUS)**

There were no cases of HUS reported in the NT in 2016. There were 2 cases of STEC reported with both testing positive by PCR only. STEC/HUS is not often reported in the NT and is felt likely to be underreported. STEC is not routinely tested for by most of the laboratories that service the NT but as culture independent testing continually evolves, it is thought that STEC may be added to the list of pathogens tested for by the faeces multiplex PCR and may become more commonly notified in the NT.

**Yersiniosis**

There were 20 cases of yersiniosis reported in the NT in 2016 compared to the 5 year mean of 9 cases (Figure 6). Notifications of yersiniosis have increased since September 2013, when 1 of the private laboratories began performing a new multiplex enteric PCR and another private laboratory followed suit in 2015. Of the 20 cases reported in 2016, 15 were culture negative but PCR positive with 2 being cultured after initially being PCR positive. It is expected that more cases of yersiniosis will be reported in the coming years as a result of the introduction of this more sensitive testing method.

**Outbreak investigations**

In 2016 there were 10 foodborne or suspected foodborne outbreaks investigated (Table 1) and 14 non-foodborne enteric disease outbreaks investigated (Table 2). Salmonella Saintpaul was the aetiological agent in at least 5 foodborne outbreaks; all associated with contaminated bean sprouts from an interstate supplier. Most non-foodborne outbreaks with known aetiology were due to norovirus (5 outbreaks) with another 7 non-foodborne outbreaks suspected to be viral in the absence of confirmatory testing.
Description of key outbreaks

Salmonella Saintpaul associated with contaminated bean sprouts

In April and May 2016, there were 6 point source outbreaks in the Darwin area (DAR0416, DAR0516, DAR0616, DAR0716, DAR0816 and RDH0116—see Table 1) that were attributed to bean sprouts contaminated with S. Saintpaul. Of these, 3 outbreaks were point source outbreaks where the exposure was to Asian food purchased from market stalls and restaurants where bean sprouts were added as either a garnish or at the end of cooking to foods such as laksa and pad thai. The other 3 outbreaks were the result of the contaminated bean sprouts being served in salads at an institution, a commercial caterer and at a salad bar. These same contaminated bean sprouts caused a surge in S. Saintpaul notifications in South Australia (SA) at the same time. S. Saintpaul isolates from clinical cases in both jurisdictions, food retrieved from cases’ refrigerators in Darwin, bean sprout samples from an Adelaide supermarket and environmental swabs from the producer were all indistinguishable by whole genome sequencing.

In total there were over 370 cases of S. Saintpaul across SA, the Australian Capital Territory, New South Wales and the NT attributed to this multijurisdictional outbreak. The bean sprout producer withdrew the product and ceased production to undergo decontamination and maintenance in order to ensure that bean sprouts were safe for consumption upon reopening.

Staphylococcus aureus food poisoning associated with contaminated noodles

In October 2016 we were alerted to 2 outbreaks of acute gastroenteritis after people had consumed laksa; there were a number of cases who had eaten laksa from the same market stall and a separate cluster of cases that ate laksa prepared by a colleague and eaten at their workplace. Both exposures resulted in numbers of people presenting to the Royal Darwin Hospital Emergency Department with rapid onset of vomiting and diarrhoea within 1 hour of eating. Both outbreaks triggered an alert from the Emergency Department Syndromic Surveillance System which we use to detect possible outbreaks of foodborne illness. There were 2 ill people tested positive for staphylococcal enterotoxin and we identified 24 cases of staphylococcal food poisoning in the Darwin region associated with eating laksa between August and October 2016. The median incubation period was 3 hours (range 0.7–20 hours) and median illness duration was 6 hours (range 1–24 hours). Staphylococcal enterotoxin was also detected in 3 samples of cooked laksa. Uncooked noodles from Asian restaurants in Darwin were sampled but staphylococcal enterotoxin was not detected. However, unacceptable levels of Bacillus cereus and Staphylococcus aureus were detected in uncooked noodles originating from one supplier who was the supplier of the noodles implicated in the outbreak. In addition to positive laboratory tests, environmental health observations resulted in this supplier and a market stall trader being issued prohibition.

Figure 6. Yersiniosis notifications in the Northern Territory, 2007–2016.
### Table 1. Summary of foodborne and suspected foodborne outbreaks investigated in the Northern Territory in 2016

<table>
<thead>
<tr>
<th>Outbreak number</th>
<th>Onset month</th>
<th>Aetiology</th>
<th>No. Exposed</th>
<th>Cases</th>
<th>Transmission / Vehicle</th>
<th>Setting Exposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>DAR0316</td>
<td>March</td>
<td>Unknown</td>
<td>111</td>
<td>30</td>
<td>Sandwiches</td>
<td>Restaurant&lt;sup&gt;10&lt;/sup&gt;</td>
</tr>
<tr>
<td>KA0116</td>
<td>April</td>
<td>S. Litchfield (2)</td>
<td>40</td>
<td>2</td>
<td>Unknown</td>
<td>Institution</td>
</tr>
<tr>
<td>DAR0416</td>
<td>April</td>
<td>S. Saintpaul (15)</td>
<td>3000</td>
<td>38</td>
<td>Bean sprouts</td>
<td>Institution</td>
</tr>
<tr>
<td>RDH0116</td>
<td>April</td>
<td>S. Saintpaul (1)</td>
<td>Unknown</td>
<td>2</td>
<td>Bean sprouts</td>
<td>Fair/festival/mobile service</td>
</tr>
<tr>
<td>DAR0516</td>
<td>April</td>
<td>S. Saintpaul (4)</td>
<td>3000</td>
<td>20</td>
<td>Unknown</td>
<td>Community</td>
</tr>
<tr>
<td>DAR0616</td>
<td>April</td>
<td>S. Saintpaul (7)</td>
<td>43</td>
<td>17</td>
<td>Bean Sprouts</td>
<td>Commercial caterer</td>
</tr>
<tr>
<td>DAR0716</td>
<td>April</td>
<td>S. Saintpaul (3)</td>
<td>Unknown</td>
<td>3</td>
<td>Bean Sprouts</td>
<td>Restaurant</td>
</tr>
<tr>
<td>DAR0816</td>
<td>April</td>
<td>Salmonella Spp. (PCR only) (2)</td>
<td>Unknown</td>
<td>2</td>
<td>Unknown</td>
<td>Restaurant</td>
</tr>
<tr>
<td>DAR1616</td>
<td>August</td>
<td>Unknown</td>
<td>6</td>
<td>5</td>
<td>Unknown</td>
<td>Restaurant</td>
</tr>
<tr>
<td>DAR1716</td>
<td>October</td>
<td>Staphylococcus aureus (2)</td>
<td>Unknown</td>
<td>24</td>
<td>Noodles</td>
<td>Community (predominantly market stalls)</td>
</tr>
</tbody>
</table>

### Table 2. Summary of non-foodborne outbreaks investigated in the Northern Territory in 2016

<table>
<thead>
<tr>
<th>Ref No</th>
<th>Onset Month</th>
<th>Aetiology</th>
<th>No. Exposed</th>
<th>Cases</th>
<th>Transmission / Vehicle</th>
<th>Setting Exposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>DAR0116</td>
<td>February</td>
<td>Unknown</td>
<td>Unknown</td>
<td>6</td>
<td>Person-to-person</td>
<td>Child care</td>
</tr>
<tr>
<td>EA0116</td>
<td>February</td>
<td>Unknown</td>
<td>Unknown</td>
<td>8</td>
<td>Person-to-person</td>
<td>Community</td>
</tr>
<tr>
<td>DAR0216</td>
<td>February</td>
<td>Cryptosporidium parvum (3)</td>
<td>Unknown</td>
<td>3</td>
<td>Environmental</td>
<td>Community</td>
</tr>
<tr>
<td>DAR0916</td>
<td>June</td>
<td>Unknown</td>
<td>90</td>
<td>14</td>
<td>Person-to-person</td>
<td>Child Care</td>
</tr>
<tr>
<td>DAR1016</td>
<td>June</td>
<td>Unknown</td>
<td>20</td>
<td>9</td>
<td>Person-to-person</td>
<td>Private residence</td>
</tr>
<tr>
<td>RDH0216</td>
<td>June</td>
<td>Norovirus (2) Giardia spp &amp; Hymenolepsis nana (3)</td>
<td>7</td>
<td>4</td>
<td>Person-to-person</td>
<td>Private residence</td>
</tr>
<tr>
<td>BA0116</td>
<td>March</td>
<td>Unknown</td>
<td>Unknown</td>
<td>3</td>
<td>Environmental</td>
<td>Community</td>
</tr>
<tr>
<td>DAR1116</td>
<td>July</td>
<td>Unknown</td>
<td>19</td>
<td>2</td>
<td>Person-to-person</td>
<td>Private residence</td>
</tr>
<tr>
<td>DAR1216</td>
<td>July</td>
<td>Norovirus (1)</td>
<td>63</td>
<td>11</td>
<td>Person-to-person</td>
<td>Child care</td>
</tr>
<tr>
<td>DAR1316</td>
<td>July</td>
<td>Norovirus (1)</td>
<td>4</td>
<td>4</td>
<td>Unknown</td>
<td>Private residence</td>
</tr>
<tr>
<td>RDH0316</td>
<td>July</td>
<td>Norovirus (2)</td>
<td>Unknown</td>
<td>12</td>
<td>Person-to-person</td>
<td>Community</td>
</tr>
<tr>
<td>DAR1416</td>
<td>July</td>
<td>Unknown</td>
<td>47</td>
<td>7</td>
<td>Person-to-person</td>
<td>Institution</td>
</tr>
<tr>
<td>DAR1516</td>
<td>August</td>
<td>Norovirus (2)</td>
<td>2000</td>
<td>36</td>
<td>Person-to-person</td>
<td>Military</td>
</tr>
<tr>
<td>DAR1916</td>
<td>November</td>
<td>Unknown</td>
<td>3000</td>
<td>48</td>
<td>Person-to-person</td>
<td>Military</td>
</tr>
</tbody>
</table>
notices due to a number of breaches of the Food Act.

Contamination of the noodles likely occurred during production (*Staphylococcus aureus* was detected in unopened noodles in a restaurant fridge, as well as in samples taken from a private residence). The bacteria introduced to the noodles during the production were able to grow and produce toxin when it was served by the laksa vendors (i.e. it was exacerbated by handling conditions). Laksa noodles are often portioned out at room temperature prior to soup/broth being poured over them. The length of time the noodles were at room temperature facilitated bacterial growth and toxin production.

**Conclusion**

Continued surveillance of enteric disease and responses to outbreaks of illness are key functions of the OzFoodNet network of epidemiologists. The network works to ensure that Territorians consume food that is safe. This can be achieved by maintaining strong relationships between CDC, Environmental Health, laboratories, reference laboratories, food safety agencies, and food producers.

**Acknowledgments**

Members of the OzFoodNet network around Australia.

Dr Peter Markey, Mary Verus, Dr Vicki Krause, Mrs Chunya Rae and the staff at the NT CDC public health units.

Dr Stephanie Davis and academic staff at the National Centre for Epidemiology and Population Health, Australian National University.

NT Government Environmental Health. Staff from the NT Government Pathology Service (NTGPS). Western Diagnostic Pathology, Sullivan and Nicolaides Pathology, Australian Clinical labs Pathology, PathWest, IMVS and MDU.

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10. Georges N, Markey P, Draper A, Morton C. Was it the chicken, the egg or something else? A gastroenteritis outbreak most likely due to norovirus at a Top End school principals’ workshop. *NT Dis Control Bull*. 2016;23 (3);10-18.
The Darwin aerial salt marsh mosquito surveillance and control season 2016/17 and future implications

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Abstract

During the 2016/17 Darwin aerial salt marsh mosquito control season, high numbers of Aedes vigilax occurred in October and November despite successful mosquito control in the Shoal Bay Swamp located west of the Shoal Bay Receiving Station. Preliminary investigations have indicated that the high numbers were due to extensive breeding in the uncontrolled swamps associated with Kings Creek to the east of the Receiving Station, with mosquito dispersal occurring from these areas into the northern Darwin suburbs, Palmerston and other areas. To reduce the impact of Ae. vigilax and potential disease risks on residents, a comprehensive investigation of Ae. vigilax breeding in the Kings Creek swamps would be required with the aim to establish a comprehensive aerial mosquito control program.

Key words: Salt marsh mosquitoes, Aedes vigilax, aerial control, Ross River virus

Introduction

The northern salt marsh mosquito, Aedes vigilax is not only a major human pest, with an extreme flight range of over 50km, but a known vector for Ross River virus (RRV) and Barmah Forest virus, with the highest risk period for RRV transmission in the Northern Territory (NT) between December and March. In 1986, Medical Entomology (ME) of the NT Department of Health (DoH) established an aerial salt marsh mosquito surveillance and control program to reduce the impact of this pest and disease vector on the residents of the northern Darwin suburbs. Aerial larval control is carried out in the western part of the Shoal Bay swamp system, including the Leanyer, Holmes Jungle, Micket Creek and the Shoal Bay Receiving Station swamps, where Ae. vigilax breeds in very high numbers usually between September and January, following high tides and rainfall.

Aerial operations usually commence in the late dry season for tides ≥ 7.5m, with extensive control required in November and December following heavy rain and high tides. Total breeding areas controlled vary between years due to differences in tide height and rainfall, with an average of 1456 hectares controlled each year.
season since 2000/01 (Figure 1). During the late wet and early dry season, *Ae. vigilax* breeding sites are usually unavailable for oviposition following monsoonal rain or because tides are not big enough to trigger extensive breeding.\textsuperscript{14}

Aerial or ground post control surveys are carried out to confirm the success of the control operations. In addition, *Ae. vigilax* numbers are monitored through the ME adult mosquito monitoring program, with weekly CO\textsubscript{2} baited encephalitis virus surveillance (EVS) traps set overnight along the perimeter of the swamp system, with the Karama trap usually the most productive site (Figure 2).

**The 2016/17 aerial salt marsh mosquito control season**

During the 2016/17 *Ae. vigilax* season, a total of 5 successful aerial control operations were carried out between August and December, with 4 of these operations continuing over 2 to 3 consecutive days, with a total of 1797.75ha of breeding area controlled.

The season started with aerial control following a 7.51m tide on 21 August. However, *Ae. vigilax* numbers did not noticeably increase until after a 7.62m tide on 19 September associated with 60.2mm rain, even though a total of 297.5ha of breeding sites were controlled (Figure 3). A further 77mm of rain recorded in the western part of the Shoal Bay swamp system on 4 October, followed by a 7.92m tide on 18 October saw *Ae. vigilax* numbers explode to unprecedented numbers, despite control of 195.5ha and 488.75ha in early and mid-October (Figure 3). In early November, *Ae. vigilax* numbers decreased before another increase in late November following the highest tide of the year (8.02m) (Figure 3). Numbers increased regardless of the 459ha controlled on 17 and 18 November. Salt marsh mosquito numbers then decreased to very low levels in mid-December and remained low, with only 1 more control (272ha) required following a 7.96m tide on 15 December (Figure 3).

**Mosquito breeding and dispersal from outside the aerial control area**

On some occasions, adult mosquito traps set along the perimeter of the Leanyer and Holmes Jungle swamps collect high *Ae. vigilax* numbers in the late dry and early wet season. It was previously suggested that *Ae. vigilax* numbers generally increase following unseasonal rainfall, due to an increase in breeding area.\textsuperscript{16} This was the case in the 2016/17 season, with high numbers increasing following unseasonal rainfall. However, in 2016/17, control was effective in reducing these numbers.

**Figure 2. Weekly adult mosquito CO\textsubscript{2} baited EVS traps on the outer perimeter of the Leanyer and Holmes Jungle swamps**
Ae. vigilax numbers collected in the traps between September and early December, following high tides and extensive rainfall in late September and early October (Figure 3). However, post control larval surveys indicated successful salt marsh mosquito control during this period, leaving the exceptionally high Ae. vigilax peaks in October and November unexplained.

Following a high tide or rain, it usually takes 7 to 8 days for Ae. vigilax to fully develop and take their first blood meal. This is the time it takes before increased numbers of this species are collected in the traps from close by breeding sites. In September and October 2016, relatively high numbers were collected in the Karama trap on 27 September (7 days after the tide and rain event on 19 September), 11 October (7 days after 77mm rain) and 25 October, (7 days after the high tide on 18 October) (Figure 3). However, mosquito numbers trapped 14 days after the rain and tide events were considerably higher, indicating dispersal from uncontrolled areas (Figure 3).

While aerial control is currently carried out in swamps located between the northern Darwin suburbs and west of the Shoal Bay Receiving Station, extensive breeding is likely to occur in mangroves and saltmarsh areas associated with Kings Creek (Kings Creek, Noogoo Swamp, Milners Swamp) to the east of the Shoal Bay Receiving Station. To investigate possible Ae. vigilax dispersal from these uncontrolled swamps into the northern Darwin suburbs, the Karama trap was set on 25 October (7 days after the 7.92m tide) and again on 28 October (10 days after the 7.92m tide). While the trap set on 25 October collected 3204 Ae. vigilax, the trap set 10 days after the tide yielded 8021, the highest number ever recorded in the Karama trap, strongly suggesting mosquito dispersal from further afield.

To assess the Kings Creek swamps for salt marsh mosquito breeding and as a possible source for mosquito dispersal into the Darwin northern suburbs, ME carried out Ae. vigilax larval surveys on 3 occasions on the edges of the Kings Creek mangroves, Noogoo Swamp and Milners Swamp on 13 November 2014, 28 November 2015 and 20 October 2016. Surveys were carried out using a 250ml ladle dipper. During the 3 surveys average larval densities were found to be much higher compared to numbers in the western Shoal Bay swamps during the same tide events. While an average of 2, 3 and 24 larvae per ladle dip were collected in the Shoal Bay swamps during the 3 surveys respectively, an average of 24, 13 and 13 larvae per dip were collected in the Kings Creek swamps, with up to 100 larvae per dip (8900 larvae/m²) in some brackish grass areas bordering the upper Kings Creek mangroves.
Additional *Ae. vigilax* trapping carried out on 3 occasions following high tides between October and December 2015, with a number of traps set close to the Kings Creek swamps in addition to the routine Darwin traps, also showed very high *Ae. vigilax* numbers collected in the traps closest to the uncontrolled swamps compared to the traps adjacent to the Leanyer and Holmes Jungle swamps (Figure 4).

**Discussion**

Aerial salt marsh larval mosquito surveillance and control is carried out in the western part of the Shoal Bay swamp system following high tides and heavy rainfall. In 2016, extensive control was carried out between August and December. Although post control surveys confirmed that control operations were successful, mosquito numbers occurred in extremely high numbers in October and November. Preliminary *Ae. vigilax* larval surveys in the uncontrolled swamps associated with Kings Creek to the east of the Shoal Bay Receiving Station, in addition to adult mosquito trapping, showed high larval densities and very high adult mosquito numbers associated with these swamps. In addition, the investigation indicated dispersal of adult mosquitoes from the Kings Creek area occurring towards the northern Darwin suburbs.

This phenomena would explain the high productivity of the Karma trap, as mosquitoes are able to travel from their breeding sites through sheltered vegetation east of the Shoal Bay Receiving Station towards the upper Holmes Jungle mangroves, and further through sheltered vegetation around the Shoal Bay Waste Transfer Station towards the trap. The Karama trap itself is located amongst a relatively dense stand of eucalypts, providing wind protection and thus ideal harbourage. 

Despite the successful DoH aerial control program, the influx of high salt marsh mosquito numbers from the Kings Creek area has pest mosquito and disease risk implications for the northern Darwin suburbs. Furthermore, other areas within close proximity to the Kings Creek swamps, including Palmerston and some rural areas are also known to be severely impacted following extensive larval hatches.

**Figure 4. Average number of *Ae. vigilax* collected in CO2 baited EVS traps set on 3 occasions between October and December 2015**
To reduce the pest and potential disease risk associated with *Ae. vigilax* dispersal from the Kings Creek swamps a comprehensive investigation of the swamp system and further afield would need to be conducted. The aim would be to establish an aerial larval control program similar to the existing program in the western Shoal Bay swamps. To achieve this *Ae. vigilax* larval breeding sites within different vegetation types, as well as larval productivity of such sites following tide and rain events, would need to be determined.

**Acknowledgements**

We would like to thank all Medical Entomology staff involved in the aerial salt marsh mosquito program, Jayrow Helicopters for excellent aerial control and the Micket Creek Rifle Range and Australian Defence Force for providing access to the area.

**References**

Birth outcomes for Australian mother-infant pairs who received an influenza vaccine during pregnancy, 2012 – 2014: The FluMum study


Vaccine, 2017., http://dx.doi.org/10.1016/j.vaccine.2017.01.075

Introduction: In Australia, influenza vaccination is recommended for all women who will be pregnant during the influenza season. Vaccine safety and effectiveness are key concerns and influencers of uptake for both vaccine providers and families. We assessed the safety of receiving an influenza vaccination during any trimester of pregnancy with respect to preterm births and infant birthweight.

Methods: We conducted a nested retrospective cohort study of ‘FluMum’ participants (2012-2014). Our primary exposure of interest was influenza vaccination during pregnancy. The primary outcomes of interest were infant birthweight and weeks’ gestation at birth for live singleton infants. Analyses included comparisons of these birth outcomes by vaccination status and trimester of pregnancy an influenza vaccine was given. We calculated means, proportions, and relative risks and performed multivariable logistic regression for potential confounding factors.

Results: In the 7126 mother-infant pairs enrolled in this study, mean maternal age at infant birth was 31.7 years. Influenza vaccine uptake in pregnancy was 34%. Most mothers with a known date of vaccination received a vaccine in the second trimester (51%). Those mothers with a co-morbidity or risk factor were 13% more likely to have influenza vaccine during pregnancy compared to other mothers (RR 1.13, 95% CI 1.04 – 1.24, p = 0.007). Mean weeks’ gestation at birth was 38.7 for the vaccinated and 38.8 for the unvaccinated group (p = 0.051). Infants in the vaccinated group weighed 15 g less in birthweight compared to the unvaccinated infants (95% CI 12.8 to 42.2, p = 0.29).

Conclusion: Results arising from this large Australian cohort study are reassuring with respect to 2 critical safety outcomes; preterm births and low infant birthweights. Studies examining a broader range of birth outcomes following influenza vaccination during pregnancy are required, particularly now that maternal vaccination in pregnancy has expanded to include pertussis as well as influenza.

Whole genome sequencing to investigate a putative outbreak of the virulent community-associated methicillin-resistant Staphylococcus aureus ST93 clone in a remote Indigenous community


Microbial Genomics, 2016 2, doi: 10.1099/mgen.0.000098

We report 2 cases of severe pneumonia due to clone ST93 methicillin-resistant Staphylococcus aureus (MRSA) presenting from a remote Australian Indigenous community within a 2 week period, and the utilization of whole genome sequences to determine whether these were part of an outbreak. S. aureus was isolated from 12 of 92 nasal swabs collected from 25 community households (including the two index households); 1 isolate was ST93. Three of 5 skin lesion S. aureus isolates obtained at the community were ST93. Whole genome sequencing of the ST93 isolates from this study and a further 20 ST93 isolates from the same region suggested that recent transmission and progression to disease had not taken place. The proximity in time and space of the 2 severe pneumonia cases is probably a reflection of the high burden of disease due to ST93 MRSA in this population where skin infections and household crowding are common.
Holding back the tiger: Successful control program protects Australia from *Aedes albopictus* expansion


**Background:** The Asian tiger mosquito, *Aedes albopictus*, is an important vector of dengue, chikungunya and Zika viruses and is a highly invasive and aggressive biter. Established populations of this species were first recognised in Australia in 2005 when they were discovered on islands in the Torres Strait, between mainland Australia and Papua New Guinea. A control program was implemented with the original goal of eliminating *Ae. albopictus* from the Torres Strait. We describe the evolution of management strategies that provide a template for *Ae. Albopictus* control that can be adopted elsewhere.

**Methodology / Principal findings:** The control strategy implemented between 2005 and 2008 targeted larval habitats using source reduction, insect-growth regulator and pyrethroid insecticide to control larvae and adults in the containers. However, the infrequency of insecticide re-application, the continual accumulation and replacement of containers, and imminent re-introduction of mosquitoes through people's movement from elsewhere compromised the program. Consequently, in 2009 the objective of the program changed from elimination to quarantine, with the goal of preventing *Ae. albopictus* from infesting Thursday and Horn islands. We describe the evolution of management strategies that provide a template for *Ae. Albopictus* control that can be adopted elsewhere.

**Conclusions / Significance:** The program has successfully reduced *Ae. albopictus* populations on Thursday Island and Horn Island to levels where it is undetectable in up to 90% of surveys, and has largely removed the risk of mainland establishment via that route. The vector management strategies adopted in the later years of the program have been demonstrably successful and provide a practical management framework for dengue, chikungunya or Zika virus outbreaks vectored by *Ae. albopictus*. As of June 2016, *Ae. albopictus* had not established on the Australian mainland and this program has likely contributed significantly to this outcome.

Echocardiographic screening for Rheumatic Heart Disease in Indigenous Australian children: A cost–utility analysis


*J Am Heart Assoc.* 2017;6:e004515. DOI: 10.1161/JAHA.116.004515.

**Background:** Rheumatic heart disease (RHD) remains a leading cause of cardiovascular morbidity and mortality in children and young adults in disadvantaged populations. The emergence of echocardiographic screening provides the opportunity for early disease detection and intervention. Using our own multistate model of RHD progression derived from Australian RHD register data, we performed a cost-utility analysis of echocardiographic screening in Indigenous Australian children, with the dual aims of informing policy decisions in Australia and providing a model that could be adapted in other countries.

**Methods and Results:** We simulated the outcomes of 2 screening strategies, assuming...
that RHD could be detected 1, 2, or 3 years earlier by screening. Outcomes included reductions in heart failure, surgery, mortality, disability-adjusted life-years, and corresponding costs. Only a strategy of screening all Indigenous 5 to 12 year-olds in half of their communities in alternate years was found to be cost-effective (incremental cost-effectiveness ratio less than AU$50 000 per disability-adjusted life-year averted), assuming that RHD can be detected at least 2 years earlier by screening; however, this result was sensitive to a number of assumptions. Additional modeling of improved adherence to secondary prophylaxis alone resulted in dramatic reductions in heart failure, surgery, and death; these outcomes improved even further when combined with screening.

Conclusions: Echocardiographic screening for RHD is cost-effective in our context, assuming that RHD can be detected ≥ 2 years earlier by screening. Our model can be adapted to any other setting but will require local data or acceptable assumptions for model parameters.

Rheumatic Heart Disease severity, progression and outcomes: A multi-state model

Cannon J, Roberts K, Milne C, Carapetis J

J Am Heart Assoc. 2017;6:e003498. DOI:10.1161/JAHA.116.003498.

Background: Rheumatic heart disease (RHD) remains a disease of international importance, yet little has been published about disease progression in a contemporary patient cohort. Multi-state models provide a well-established method of estimating rates of transition between disease states, and can be used to evaluate the cost-effectiveness of potential interventions. We aimed to create a multi-state model for RHD progression using serial clinical data from a cohort of Australian patients.

Methods and Results: The Northern Territory RHD register was used to identify all Indigenous residents diagnosed with RHD between the ages of 5 and 24 years in the time period 1999-2012. Disease severity over time, surgeries, and deaths were evaluated for 591 patients. Of 96 (16.2%) patients with severe RHD at diagnosis, 50% had proceeded to valve surgery by 2 years, and 10% were dead within 6 years. Of those diagnosed with moderate RHD, there was a similar chance of disease regression or progression over time. Patients with mild RHD at diagnosis were the most stable, with 64% remaining mild after 10 years; however, 11.4% progressed to severe RHD and half of these required surgery.

Conclusions: The prognosis of young Indigenous Australians diagnosed with severe RHD is bleak; interventions must focus on earlier detection and treatment if the observed natural history is to be improved. This multi-state model can be used to predict the effect of different interventions on disease progression and the associated costs.

Letter

Crusted scabies in northern and central Australia — now is the time for eradication

Quilty S,1,2 Kaye TS2 and Currie BJ.3,4


TO THE EDITOR: There are very high rates of crusted scabies in remote Indigenous Australian communities, which is an important driver in skin infections and consequent illnesses, such as invasive streptococcal and staphylococcal sepsis, post-streptococcal glomerulonephritis and acute rheumatic fever.1,2 Patients with crusted scabies serve as sentinels that drive ongoing community infection. In order to understand the associated factors and movement patterns of patients with crusted scabies, we audited all the cases of patients presenting to Katherine Hospital.

Over a 5-year period, there were 42 admissions for treatment of crusted scabies, representing 30 patients of whom 4 presented on multiple (up to 6) occasions with re-infection—14 patients with a diagnosis confirmed by skin scraping and 16 with a presumptive diagnosis. All 30 patients were Indigenous people with comorbidities, including diabetes (n=13), hazardous alcohol use (n=13) and chronic renal impairment (n=5).
Moreover, 4 patients were infected with human T-lymphotropic virus type 1 and 7 patients were antinuclear antibody positive. The 5-year cumulative regional rate of crusted scabies was none for non-Indigenous Australians and 3 per 1000 for Indigenous Australians. By 6 months after the conclusion of the 5-year period, 7 patients were deceased.

Six patients were recorded as being homeless at the time of admission; however, this figure is likely to be an underestimation as homelessness is self-reported by patients, many of whom consider a tent in a fringe-dwelling town camp to be home. While 27 patients reported their home to be a remote community, only 14 of them were living in their home community at the time immediately before admission, thus 13 had either previously relocated to Katherine or were at least temporarily homeless (Box). Rates of homelessness in the region are 31 times higher than the national average, and extrapolating to Indigenous status, we estimate that 1 in 4 Indigenous people in the Katherine region is homeless.

Crusted scabies has recently been listed as a notifiable disease in the Northern Territory, raising opportunities for eradication. Regional scabies control programs have been successful in reducing the disease burden, and they now need to be extended and tailored to cater for extreme burdens of homelessness and inter-regional and interstate population movements. Regional and state health services need to work closely together to overcome these challenges.

Box. Distribution of patients presenting with crusted scabies

The circle size is proportional to the number of cases from a community. Red indicates the remote town community of origin, and blue shows the town where the patients was living immediately before admission. The map overlays existing Indigenous tribal groups (in watermark) and state boundaries, with town communities of origin in bold.
Homelessness is a profound problem and needs to be comprehensively managed with long term strategic policies immune from cyclic political interference.

1. Katherine Hospital, Katherine, NT.
2. Flinders University, Darwin, NT.
3. Menzies School of Health Research, Darwin, NT.
4. Royal Darwin Hospital, Darwin, NT.
simon.quilty@nt.gov.au

Competing interests: No relevant disclosures.
doi: 10.5694/mja16.00809


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**NT malaria notifications October—December 2016**

*Liz Stephenson, CDC, Darwin*

There were 3 confirmed cases of malaria notified in the 4th quarter of 2015. Below provides where the malaria was thought to be acquired, the malaria type, whether chemoprophylaxis was used and where the patient lived in the Northern Territory (NT).

<table>
<thead>
<tr>
<th>No. cases</th>
<th>Origin of Infection</th>
<th>Reason Exposed</th>
<th>Agent</th>
<th>Chemoprophylaxis</th>
<th>NT Region</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Republic of Cameroon</td>
<td>Expatriate visiting relatives</td>
<td><em>P. falciparum</em></td>
<td>No</td>
<td>Katherine</td>
</tr>
<tr>
<td>2</td>
<td>Rabaul, PNG</td>
<td>Recreation</td>
<td><em>P. falciparum</em></td>
<td>No</td>
<td>Darwin</td>
</tr>
</tbody>
</table>

************

**Measles**

*Before you travel*

Get protected not infected

The BEST protection against measles is VACCINATION

It is important to be immune to measles if you are travelling overseas.

Contact your local doctor or health centre to discuss vaccination.

www.nt.gov.au/health
### Immunisation coverage for children aged 12-<15 months at 31 December 2016

<table>
<thead>
<tr>
<th>Location</th>
<th>Number</th>
<th>%DTP</th>
<th>%Polio</th>
<th>%HIB</th>
<th>%Hep</th>
<th>%Pneumo</th>
<th>Fully vaccinated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Darwin</td>
<td>298</td>
<td>95.0%</td>
<td>95.0%</td>
<td>94.6%</td>
<td>95.0%</td>
<td>94.6%</td>
<td>94.3%</td>
</tr>
<tr>
<td>Winnellie PO Bag</td>
<td>51</td>
<td>90.2%</td>
<td>90.2%</td>
<td>90.2%</td>
<td>90.2%</td>
<td>90.2%</td>
<td></td>
</tr>
<tr>
<td>Palmerston/Rural</td>
<td>288</td>
<td>94.4%</td>
<td>94.4%</td>
<td>94.1%</td>
<td>94.8%</td>
<td>94.1%</td>
<td>93.8%</td>
</tr>
<tr>
<td>Katherine</td>
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<td>92.0%</td>
<td>92.0%</td>
<td>92.0%</td>
<td>89.3%</td>
<td>89.3%</td>
</tr>
<tr>
<td>Barkly</td>
<td>17</td>
<td>100.0%</td>
<td></td>
<td>100.0%</td>
<td></td>
<td>100.0%</td>
<td>100.0%</td>
</tr>
<tr>
<td>Alice Springs</td>
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<td>85.2%</td>
<td>85.2%</td>
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</tr>
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<td></td>
</tr>
<tr>
<td>East Arnhem</td>
<td>24</td>
<td>100.0%</td>
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<td>100.0%</td>
<td></td>
<td>100.0%</td>
<td>95.8%</td>
</tr>
<tr>
<td>NT</td>
<td>902</td>
<td>93.1%</td>
<td>93.0%</td>
<td>92.5%</td>
<td>92.9%</td>
<td>92.5%</td>
<td>91.7%</td>
</tr>
<tr>
<td>Non-Indigenous (NT)</td>
<td>634</td>
<td>94.2%</td>
<td>94.0%</td>
<td>93.4%</td>
<td>94.0%</td>
<td>93.5%</td>
<td>92.7%</td>
</tr>
<tr>
<td>Indigenous (NT)</td>
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<td>91.4%</td>
<td>91.4%</td>
<td>91.0%</td>
<td>90.7%</td>
<td>89.9%</td>
<td></td>
</tr>
<tr>
<td>NT</td>
<td>902</td>
<td>93.1%</td>
<td>93.0%</td>
<td>92.5%</td>
<td>92.9%</td>
<td>92.5%</td>
<td>91.7%</td>
</tr>
<tr>
<td>Australia</td>
<td>78551</td>
<td>94.6%</td>
<td>94.5%</td>
<td>94.3%</td>
<td>94.5%</td>
<td>94.2%</td>
<td>93.7%</td>
</tr>
</tbody>
</table>

### Immunisation coverage for children aged 24-<27 months at 31 December 2016

<table>
<thead>
<tr>
<th>Location</th>
<th>Number</th>
<th>%DTP</th>
<th>%Polio</th>
<th>%HIB</th>
<th>%Hep</th>
<th>%MMR</th>
<th>%MenC</th>
<th>%Varicella</th>
<th>Fully vaccinated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Darwin</td>
<td>280</td>
<td>94.6%</td>
<td>94.6%</td>
<td>94.3%</td>
<td>94.6%</td>
<td>89.3%</td>
<td>93.2%</td>
<td>89.6%</td>
<td>88.2%</td>
</tr>
<tr>
<td>Winnellie PO Bag</td>
<td>58</td>
<td>94.8%</td>
<td>94.8%</td>
<td>94.8%</td>
<td>96.6%</td>
<td>93.1%</td>
<td>94.8%</td>
<td>91.4%</td>
<td></td>
</tr>
<tr>
<td>Palmerston/Rural</td>
<td>242</td>
<td>95.0%</td>
<td>95.0%</td>
<td>94.2%</td>
<td>94.6%</td>
<td>93.8%</td>
<td>94.6%</td>
<td>93.4%</td>
<td>90.9%</td>
</tr>
<tr>
<td>Katherine</td>
<td>81</td>
<td>100.0%</td>
<td></td>
<td>100.0%</td>
<td></td>
<td>96.3%</td>
<td>98.8%</td>
<td>96.3%</td>
<td>95.1%</td>
</tr>
<tr>
<td>Barkly</td>
<td>17</td>
<td>100.0%</td>
<td></td>
<td>100.0%</td>
<td></td>
<td>88.2%</td>
<td>100.0%</td>
<td>82.4%</td>
<td>82.4%</td>
</tr>
<tr>
<td>Alice Springs</td>
<td>121</td>
<td>97.5%</td>
<td>97.5%</td>
<td>96.7%</td>
<td>97.5%</td>
<td>89.3%</td>
<td>94.2%</td>
<td>90.1%</td>
<td>88.4%</td>
</tr>
<tr>
<td>Alice Springs PO Bag</td>
<td>49</td>
<td>95.9%</td>
<td>95.9%</td>
<td>95.9%</td>
<td>95.9%</td>
<td>83.7%</td>
<td>95.9%</td>
<td>85.7%</td>
<td>79.6%</td>
</tr>
<tr>
<td>East Arnhem</td>
<td>35</td>
<td>100.0%</td>
<td></td>
<td>100.0%</td>
<td></td>
<td>100.0%</td>
<td>100.0%</td>
<td>100.0%</td>
<td>91.4%</td>
</tr>
<tr>
<td>NT</td>
<td>883</td>
<td>96.0%</td>
<td>96.0%</td>
<td>95.5%</td>
<td>96.0%</td>
<td>91.5%</td>
<td>94.9%</td>
<td>91.2%</td>
<td>89.2%</td>
</tr>
<tr>
<td>Non-Indigenous (NT)</td>
<td>568</td>
<td>95.1%</td>
<td>95.1%</td>
<td>94.4%</td>
<td>94.9%</td>
<td>92.4%</td>
<td>94.0%</td>
<td>92.1%</td>
<td>89.6%</td>
</tr>
<tr>
<td>Indigenous (NT)</td>
<td>315</td>
<td>97.8%</td>
<td>97.8%</td>
<td>97.5%</td>
<td>98.1%</td>
<td>89.8%</td>
<td>96.5%</td>
<td>89.5%</td>
<td>88.6%</td>
</tr>
<tr>
<td>Australia</td>
<td>79558</td>
<td>96.4%</td>
<td>96.30%</td>
<td>95.40%</td>
<td></td>
<td>96.10%</td>
<td>93.20%</td>
<td>95.20%</td>
<td>93.10%</td>
</tr>
</tbody>
</table>

### Immunisation coverage for children aged 60-<63 months at 31 December 2016

<table>
<thead>
<tr>
<th>Location</th>
<th>Number</th>
<th>%DTP</th>
<th>%Polio</th>
<th>%MMR</th>
<th>Fully</th>
</tr>
</thead>
<tbody>
<tr>
<td>Darwin</td>
<td>281</td>
<td>92.5%</td>
<td>92.5%</td>
<td>92.5%</td>
<td>91.5%</td>
</tr>
<tr>
<td>Winnellie PO Bag</td>
<td>62</td>
<td>95.2%</td>
<td>95.2%</td>
<td>95.2%</td>
<td>93.5%</td>
</tr>
<tr>
<td>Palmerston/Rural</td>
<td>254</td>
<td>93.3%</td>
<td>93.7%</td>
<td>94.1%</td>
<td>91.7%</td>
</tr>
<tr>
<td>Katherine</td>
<td>93</td>
<td>95.7%</td>
<td>95.7%</td>
<td>95.7%</td>
<td>94.6%</td>
</tr>
<tr>
<td>Barkly</td>
<td>12</td>
<td>91.7%</td>
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<td>91.7%</td>
<td>91.7%</td>
</tr>
<tr>
<td>Alice Springs</td>
<td>116</td>
<td>93.1%</td>
<td>93.1%</td>
<td>94.0%</td>
<td>92.2%</td>
</tr>
<tr>
<td>Alice Springs PO Bag</td>
<td>48</td>
<td>89.6%</td>
<td>89.6%</td>
<td>93.8%</td>
<td>89.6%</td>
</tr>
<tr>
<td>East Arnhem</td>
<td>43</td>
<td>100.0%</td>
<td></td>
<td>100.0%</td>
<td></td>
</tr>
<tr>
<td>NT</td>
<td>909</td>
<td>93.5%</td>
<td>93.6%</td>
<td>94.1%</td>
<td>92.4%</td>
</tr>
<tr>
<td>Non-Indigenous (NT)</td>
<td>571</td>
<td>92.5%</td>
<td>92.6%</td>
<td>93.0%</td>
<td>91.1%</td>
</tr>
<tr>
<td>Indigenous (NT)</td>
<td>338</td>
<td>95.3%</td>
<td>95.3%</td>
<td>95.9%</td>
<td>94.7%</td>
</tr>
<tr>
<td>Australia</td>
<td>79874</td>
<td>94.2%</td>
<td>94.3%</td>
<td>94.5%</td>
<td>93.5%</td>
</tr>
</tbody>
</table>
Immunisation coverage at 31 December 2016

Holly Carmichael, CDC, Darwin

Background information to interpret coverage

Children were assigned to regions based on the postcode taken from their Medicare address listed in the Australian Immunisation Register (AIR). Children with a PO Box address listed are counted towards their PO Box postcode. Winnellie PO Bag is postcode 0822, which includes most Darwin Rural District communities, some East Arnhem District communities and some residents of 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 31 December 2016 were born between 1 July 2015 and 30 September 2015 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 or 3 doses of PRP-OMP Hib or 3 doses of another Hib vaccine, 3 doses of hepatitis B vaccine and 3 doses of pneumococcal vaccine. All vaccinations must have been administered by 12 months of age.

The cohort of children assessed at 24 to <27 months of age on 31 December 2016 were born between 1 July 2014 and 30 September 2014 inclusive. To be considered fully vaccinated, these children must have received meningococcal C vaccination (given at the 12 month schedule point), and a second dose of measles, mumps, rubella (MMR) and the first dose of the varicella vaccination (given in combination as MMRV at the 18 months schedule point). All vaccinations must have been administered by 24 months of age.

The cohort of children assessed at 60 to <63 months of age on 31 December 2016 were born between 1 July 2011 and 30 September 2011 inclusive. To be considered fully vaccinated, these children must have received 4 or 5 valid doses of vaccines containing diphtheria, tetanus, pertussis antigens, 4 doses of poliomyelitis vaccine and 2 valid doses of MMR vaccine. All vaccinations must have been administered by 60 months (5 years) of age.

Interpretation and comment

Immunisation coverage rates for NT children by regions as estimated by the AIR are shown on page 42.

Compared to national data, children in the NT were less likely to be fully immunised in the 12 to <15 months cohort (NT 91.7%, National 93.7%), the 24 to <27 months cohort (NT 89.2%, National 91.5%) and the 60 to <63 months cohort (NT 92.4%, National 93.5%).

Indigenous children were less likely to be fully immunised than non-Indigenous children in the 12 to <15 month cohort (Indigenous 89.9%, Non-Indigenous 92.7%) and in the 24 to <27 month cohort (Indigenous 88.6%, Non-Indigenous 89.6%), but were more likely to be fully immunised in the 60 to <63 month cohort (Indigenous 94.7%, Non-Indigenous 91.1%).

Centre for Disease Control (CDC) is currently reviewing the reasons for lower immunisation coverage in NT children compared to the national average. CDC is working with the Australian Immunisation Register to review data quality and processing, and will send out reminders to overdue children.

Further information about the Australian Childhood Immunisation Register coverage may be found at: http://ncirs.edu.au/immunisation/coverage/index.php
### NT NOTIFICATIONS OF DISEASES BY ONSET DATE & DISTRICTS

1 January—31 December 2015 and 2016

<table>
<thead>
<tr>
<th>Disease</th>
<th>Alice Springs</th>
<th>Barkly</th>
<th>Darwin</th>
<th>East Arnhem</th>
<th>Katherine</th>
<th>NT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute post strep glomerulonephritis</strong></td>
<td>15</td>
<td>14</td>
<td>7</td>
<td>3</td>
<td>15</td>
<td>12</td>
</tr>
<tr>
<td><strong>Adverse vaccine reaction</strong></td>
<td>15</td>
<td>7</td>
<td>3</td>
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<td>0</td>
<td>0</td>
<td>0</td>
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<td><strong>Campylobacteriosis</strong></td>
<td>61</td>
<td>95</td>
<td>6</td>
<td>10</td>
<td>271</td>
<td>296</td>
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<td><strong>Chickenpox</strong></td>
<td>283</td>
<td>12</td>
<td>3</td>
<td>73</td>
<td>77</td>
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<td>784</td>
<td>841</td>
<td>50</td>
<td>75</td>
<td>1398</td>
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<td><strong>Gonococcal infection</strong></td>
<td>942</td>
<td>868</td>
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<td><strong>Gonococcal neonatal ophthalmia</strong></td>
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<td><strong>Group A strep invasive</strong></td>
<td>17</td>
<td>22</td>
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<td>9</td>
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<td><strong>Hepatitis B - new</strong></td>
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<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>1</td>
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<td><strong>Hepatitis B - unspecified</strong></td>
<td>43</td>
<td>6</td>
<td>1</td>
<td>0</td>
<td>106</td>
<td>89</td>
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<td><strong>Hepatitis C - new</strong></td>
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<td>0</td>
<td>0</td>
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<tr>
<td><strong>Hepatitis C - unspecified</strong></td>
<td>35</td>
<td>38</td>
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<td>0</td>
<td>0</td>
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<td>0</td>
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<tr>
<td><strong>H Influenza non-b</strong></td>
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<td>5</td>
<td>0</td>
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<td><strong>HTLV1 asymptomatic/ unspecified</strong></td>
<td>23</td>
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<td><strong>Influenza</strong></td>
<td>133</td>
<td>132</td>
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<td>6</td>
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<td><strong>Legionellosis</strong></td>
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<td><strong>Malaria</strong></td>
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<td><strong>Melioidosis</strong></td>
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<td><strong>Mumps</strong></td>
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<td>34</td>
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<td><strong>Non TB Mycobacteria</strong></td>
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<td>201</td>
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<td><strong>Q Fever</strong></td>
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<td><strong>Rheumatic Fever</strong></td>
<td>39</td>
<td>54</td>
<td>1</td>
<td>3</td>
<td>33</td>
<td>51</td>
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<td><strong>Ross River Virus</strong></td>
<td>16</td>
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<td>170</td>
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<td><strong>Rotavirus</strong></td>
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<td>36</td>
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<td><strong>Salmonellosis</strong></td>
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<td>68</td>
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<td><strong>Shigellosis</strong></td>
<td>34</td>
<td>58</td>
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<td>72</td>
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<td><strong>STEC/VTEC</strong></td>
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<tr>
<td><strong>Syphilis &lt;2 years duration</strong></td>
<td>79</td>
<td>53</td>
<td>4</td>
<td>1</td>
<td>31</td>
<td>72</td>
</tr>
<tr>
<td><strong>Syphilis &gt;2 years duration or unknown</strong></td>
<td>25</td>
<td>12</td>
<td>1</td>
<td>1</td>
<td>27</td>
<td>33</td>
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<td><strong>Trichomoniasis</strong></td>
<td>1047</td>
<td>959</td>
<td>135</td>
<td>156</td>
<td>1206</td>
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<td><strong>Tuberculosis</strong></td>
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<td><strong>Typhoid</strong></td>
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<td><strong>Varicella - unspecified</strong></td>
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<td><strong>Vibrio food poisoning</strong></td>
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<td><strong>Vibrio invasive</strong></td>
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<td>0</td>
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<tr>
<td><strong>Yersiniosis</strong></td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>10</td>
<td>15</td>
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<tr>
<td><strong>Zoster</strong></td>
<td>37</td>
<td>63</td>
<td>1</td>
<td>5</td>
<td>289</td>
<td>286</td>
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<tr>
<td><strong>Sum</strong></td>
<td>3,639</td>
<td>3,546</td>
<td>328</td>
<td>483</td>
<td>5,678</td>
<td>6,249</td>
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The Northern Territory Disease Control Bulletin Vol 24, No. 1 March 2017
Ratio of the number of notifications in 2016 to the 5 year mean (2011–15):
selected diseases

**DECREASE**
- Meningococcal infection
- Influenza
- Pertussis
- H Influenza non-b
- Rheumatic Fever
- Group A strep invasive
- Zoster
- Salmonellosis
- Dengue
- Shigellosis
- Campylobacteriosis
- Cryptoportidiosis
- Mumps 31.4

**INCREASE**
- Beyond 2SD of mean of previous 5 years

Ratio of the number of notifications in 2016 to the 5 year mean (2011–15):

**DECREASE**
- Syphilis congenital
- Hepatitis B - chronic
- HTLV1 asymptomatic/unspec
- Hepatitis B - new
- Hepatitis B - unspec
- Syphilis > 2 years duration or unknown
- Gonococcal infection
- Chlamydia
- Hepatitis C - new
- Hepatitis C - unspec
- Trichomoniasis
- HIV

**INCREASE**
- Beyond 2SD of mean of 5 previous years
- Syphilis < 2 years duration
  - Gonococcal conj

Ratio of 2016 cases to mean 2011-2015
Comments on notifications

Enteric diseases

The 4 major enteric pathogens, *shigella*, *salmonella*, *campylobacter* and *cryptosporidia* together with *yersinia* were all reported in numbers significantly greater than the 5 year mean in 2016. Cases of salmonellosis were 1.54 times more than expected (excess of 249 cases), shigellosis 1.79 times (88 cases), campylobacteriosis 1.85 times (213 cases), cryptosporidiosis 2.32 times (158 cases) and yersinosis 3.4 times (13 cases). It is likely that much of this increase is due to the increased sensitivity of the tests being performed with laboratories now universally using nucleic acid testing to detect pathogens.

However the increase in cases of cryptosporidiosis in 2016 was higher than expected even allowing for the testing bias, particularly in the months January to June. This increase was investigated and there was no common source identified.

In addition to more sensitive testing methods, the increase in salmonellosis was also attributed to multiple outbreaks caused by *Salmonella Saintpaul* associated with contaminated bean sprouts. There were 147 notifications of this Salmonella serovar in 2016 compared to a 5 year mean of 71 notifications.

Acute rheumatic fever and invasive group A streptococcal disease

There were 154 cases of acute rheumatic fever notified in 2016, 1.4 times the 5 year mean of 111. Likewise there were 82 cases of invasive group A streptococcal infection notified which was 1.4 times the expected (59). Some of this increase may be due to better case identification but it is also likely that there were higher numbers of the streptococci bacteria circulating in 2016. There was 1 cluster of invasive group A streptococcal infections detected in a hostel but no recognised outbreaks of acute rheumatic fever.

Mumps

2016 saw a mumps outbreak in the NT with 138 cases being notified compared to a total of 22 in the previous 5 years. The outbreak commenced in Western Australia in 2014 and crossed over into the Northern Territory in 2015, originally into the Katherine region and then to Alice Springs and the Top End. Community-based immunisation interventions aimed at improving population immunity have been successful in some communities. The outbreak is showing signs of decreasing.

Legionellosis

There were no cases of legionellosis notified in 2016. This is compared to a 5 year mean of 6 and is the first time there have been no notifications in a calendar year.

Syphilis under 2 years duration

The ongoing outbreak of syphilis effected the 2016 notifications of syphilis of less than 2 years duration. The outbreak was first identified in mid-2014 and has continued into 2016. This outbreak explains the ratio increase when compared to the previous 5 year mean when the disease had been on the decline in the Territory.

The population at risk continues to be Indigenous people mainly in the age group 15-29 years. A variety of public health control measures have been implemented and continue to be carried out. These include:

- NT-wide public health alerts plus delivery of education to a range of health professionals in primary and secondary care, in urban, rural, and remote settings
- Issuing guidelines
- Undertaking community screening in ‘hot spot’ areas
- Carefully targeted press and media alerts to the general public.

Gonococcal conjunctivitis

The number of notifications received for this condition increased from 5 in 2015 to 12 in 2016.

This condition should not be confused with gonococcal neonatal ophthalmia (ophthalmia neonatorum), which is also a notifiable disease and affects newborn infants.
Gonococcal conjunctivitis mainly affects adults, but can present occasionally in children. Transmission occurs through direct, and indirect, contact from infected genital areas to the eyes. Left untreated it can lead to corneal ulceration, scarring, and loss of vision.

In addition to prompt treatment, an STI screen should be done, and prevention messages provided regarding hygiene and safe sexual practice.

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Disease Control staff updates January—March 2017

**Top End**

**Charles Douglas** has returned to the Centre for Disease Control (CDC) replacing **Steven Skov**, who was farewelled in December 2016, as the Community Physician. Charles previously worked at CDC as a TB Medical Officer and Trachoma Medical Advisor before heading to West Africa for 2 years working on Ebola management and post-Ebola recovery programs.

**Philippa Binns** has also returned to CDC to job-share with **Ros Webby** as the Head of Immunisation. Philippa previously worked at CDC when completing a Masters of Applied Epidemiology. She subsequently worked in NSW public health programs before returning to the NT to work at the National Critical Care and Trauma Response Centre and as a remote medical practitioner in Milikapiti.

**Laura Francis** is working as a research nurse with the Paediatric Department at Royal Darwin Hospital and also working in the CDC on a surveillance research project with the national PAEDS (Paediatric Active Enhanced Disease Surveillance) network. The program focuses on hospitalised children with 8 childhood illnesses that are vaccine preventable or associated, to describe their epidemiology and evaluate vaccine effectiveness. PAEDS is already operational within paediatric hospitals in all other states (except Tasmania) with projects nationally coordinated through the National Centre for Immunisation Research and Surveillance.

Welcome to the new Medical Officers who commenced at CDC Darwin in January 2017. **Helena Chan**, **Pyae Pyae Phyo Maung** and **Winnie Chen** are working at CDC on a 6 monthly rotation as General Practice Registrars. **Sarah Lord**, **Thwe Thwe Win**, **Nurul Mohammed Bakri** and **Jacqueline Murdoch** completed their 6 month rotation in December 2016. **Swe Yen Tay** has replaced **Sarah Lynar** as the Infectious Diseases Registrar on a 6 month placement at CDC. **Nick Georges** has replaced **Pasqualina Coffey** in the Public Health Registrar role. Congratulations to Pasqualina who has qualified as a Public Health Specialist and is currently working as a TB Public Health Medical Officer at CDC. **Kate Hardie** has resigned from her position as the TB Public Health Medical Officer after 2 years in the role. She is enjoying some adventurous overseas travel.

**David Decolongon** has re-joined the Sexual Health and Blood Borne Virus (SHBBV) Team in the Senior Policy role taking the position vacated by Katherine Moriarty in November 2016. David has recently come from the Northern Territory AIDS and Hepatitis Council after his 12 months in the SHBBV Senior Policy role previously. The Remote Sexual Health Manager **Linda Garton** has taken extended leave.

**Adam Bourke** has commenced with Medical Entomology as a Technical Officer. **Jane Carter** has returned from 12 months leave and is acting on the Technical 4 level, taking over the responsibility for the aerial control program and the laboratory supervision.

Rheumatic Heart Disease (RHD) Clinical Nurse Specialist (CNS) **Cath Blacker** has taken maternity leave. Congratulations to Cath and **Peter Nihill** on the birth of their son Seamus. **Emma Childs** has joined the RHD team and will be covering the East Arnhem area while being based in Darwin. Emma is a CNS Nurse who has worked in various remote Top End communities, provided retrieval services for Careflight International and has been involved in humanitarian post-disaster work in Nepal and the Philippines. **Narelle Raiss** has also joined...
the RHD team as a CNS to cover Darwin urban, rural and remote area. Narelle has worked in Top End remote health in the Tiwi Islands and has been involved in humanitarian work with Médecines Sans Frontières in Pakistan prior to joining the RHD team at CDC.

Central Australia

The Central Australian Trachoma Program has welcomed and farewelled several staff at the start of 2017. Emma Kraft joined the Program in mid-January as the Trachoma Health Promotion Officer. Emma has experience across both Government and non-Government sectors and has worked in various health promotion programs. Emma Hunt has left her Trachoma CNS Nurse role to take up a role at Hearing Health—her enthusiasm and hard work will be greatly missed. Renee Ragonesi will join the trachoma program as a public health nurse commencing on 10 March. Renee will be joining us from Queensland where she has been studying a Master of Public Health and Tropical Medicine.

Farewell to Helen Tindall who has worked at CDC since 1999 as a CNS in both the Immunisation and Tuberculosis Programs. Helen has made an outstanding contribution to Immunisation, Tuberculosis and Surveillance during her time in CDC. Helen has moved to Cambodia to work for Médecins Sans Frontières. Farewell to Jacqui Arnold who has moved to New Zealand after working as a CNS at CDC both in the Trachoma and Tuberculosis Programs since April 2014.

Welcome to Jo Rhodes who has moved into the CNS Tuberculosis role from Palliative Care in the Central Australian Health Service.

Kate Wales and Leanne O’Connor have returned from maternity leave and are now job-sharing the CNS Hepatitis position in Clinic 34 Alice Springs. The clinic team also welcomed back Chelsea Lodge who resumed her Indigenous Liaison Officer position after maternity leave. Nina Missen has also returned from Maternity leave and is job-sharing the Rheumatic Heart Disease Register Coordinator position with Brianna Sanderson.

Rebecca Curr, CNS Immunisation, has started 12 months leave without pay to work for the Central Australia Aboriginal Congress as a midwife. Rebecca Payne returned to Clinic 34 Darwin after backfilling as the CNS for the Syphilis Register in Alice Springs. Helen Goodwin is acting as Remote Sexual Health Program Manager while Mark Russell is on long service leave. Bernard Longbottom is in the Remote Sexual Health CNS role backfilling for Helen Goodwin.

Noela Davies has joined the CDC Barkly team as a CNS. Noela has extensive experience in public health programs and primary health care having previously worked for CDC and Primary Health Care in the NT and in Queensland.

Congratulations to Noela Davies who received the Order of Australia Medal (OAM) on Australia Day this year. Noela received the OAM for her service to nursing and to international humanitarian healthcare programs.