Abstract

In 2017, a mature gentleman developed pulmonary tuberculosis (TB) and it appeared that his canine companion also fell ill with the same illness. This review article was born out of the contemplations that subsequently followed. Namely: Can animals contract TB from humans? If so, can they infect other humans? More broadly, is TB in animals an important issue in the global campaign to eradicate TB? What measures can be undertaken to control TB in animals?

After reviewing the literature, the main findings were that: Many animal species are as vulnerable in contracting TB as humans are. In terms of animal prevalence and zoonotic transmission, Mycobacterium bovis is a more significant pathogen than M. tuberculosis. The interspecies transmission of bovine TB beyond the human-animal interface is a theoretical obstacle to global eradication of TB. A One Health collaborative approach combining medical and veterinary disciplines is critical for future success in TB eradication programs.

Key words: Tuberculosis; Mycobacterium tuberculosis; M. bovis; zoonosis; transmission.

Case study

An older male with several chronic medical conditions was living on his own and minding an aged dog. Over a few months, he developed a chronic productive cough, night sweats, considerable weight loss, and had functional deterioration and deconditioning. After several weeks his canine companion also became unwell with a respiratory infection consisting of cough, fever and secondary heart failure.

The local GP referred the gentleman to the local hospital Emergency Department (ED) where a CT scan of the chest was taken and revealed diffuse nodules and cavitary lung disease. Sputum AFB cultures grew Mycobacterium tuberculosis (MTB).
and the patient was placed on appropriate TB treatment. CDC staff members were concerned the dog may have contracted TB from its owner and, taking this into consideration, they liaised with local veterinary services. Thankfully the dog’s condition settled and it did not require diagnostic specimens to be taken.

In the process of treating the patient and managing the dog, the treatment team pondered that across a city, region, country there are many domesticated animals in close contact with their owners. These pets are potentially vulnerable to contracting TB from owners with active disease. Of some concern was the thought that animals that got TB disease from their owners may pass it on to other humans. There are important public health implications if this is the case.

This review article describes TB in animals and the implications for human health.

Background

TB continues to be an important disease worldwide in both humans and animals causing morbidity, mortality, economic loss as well as animal conservation concerns due to outbreaks among endangered species in captive environments.1-3

TB is caused by MTB complex which consists of the following species: M. tuberculosis, M. canetti, M. africanum, M. bovis, M. pinnipedi, M. caprae and M. microti. These species can cause disease in a range of animal hosts and in humans. For instance, M. pinnipedi has been reported in fur seals, and in captive environments the bacterium has been transmitted from seals to their human handlers.4

The tubercle bacillus infects around one-quarter of the world’s population and is estimated to kill 1.5 to 2 million people every year.1,5 In 2016 there were 1.3 million deaths from TB in HIV negative patients and 370,000 deaths from TB in HIV positive patients.5 A total of 95% of cases occur in developing nations.1

In industrialised countries, there has been significant progress towards elimination of disease caused by MTB complex. In many countries with insufficient resources to support TB programs, only limited progress has been experienced.1

TB in humans

The majority of human TB is due to MTB, but the zoonotic pathogen M. bovis continues to be reported in developed nations and in immigrants from regions of the world where TB in cattle is endemic.1,3 Globally in 2016 there were an estimated 147,000 new cases of zoonotic (bovine) TB and 12,500 deaths due to M. bovis.5 In the NT since 1990, there were 8 cases of bovine TB out of a total of 1087 TB cases (NT Notifiable Disease System); a low rate following successful local and national bovine TB eradication programs. In 1994 there were 1057 notifications of TB in Australia (with 97 cases being due to relapse or reactivation) at an annual incidence rate of 5.9 cases per 100,000 population.5 Of the 708 notifications where the causative organism was reported, 704 were due to MTB and only 4 cases were due to M. bovis.7 National notifications of M. bovis dropped from a peak of 10 cases in 1996 to be between 0 and 4 cases annually from 1999 to 2011.8-21 Between 1994 and 2014 the national annual TB incidence rate remained steady moving from 5.9 to 5.7 cases per 100,000 population.8,22 The incidence rate in the NT fell from 18.1 to 11.4 cases per 100,000 population over the same period.6,22

In developing nations the real incidence of zoonotic (bovine) TB is grossly underestimated due to the scarcity of resources and laboratory facilities to isolate M. bovis and to differentiate it from MTB.1,23 In addition, bovine TB is often associated with extrapulmonary disease which may be misdiagnosed. To complicate matters, M. bovis is largely foodborne and therefore the epidemiology and transmission dynamics differ substantially from airborne disease.23 In resource-restricted settings it is likely that there are higher rates of bovine TB as eradication programs in cattle are not usually undertaken and pasteurisation of dairy products may not be routinely practised.

In agrarian environments in developing nations, bovine TB follows from close contact between humans and cattle particularly as some groups keep their animals indoors and have a custom of drinking raw milk and consuming raw meat and
meat products.\textsuperscript{3} Human-to-human airborne transmission of \textit{M. bovis} can also occur.\textsuperscript{1}

**TB in animals**

TB occurs in both domestic and wild animals. Disease due to \textit{M. bovis} is far more common than that due to \textit{MTB}. The primary reservoir for \textit{M. bovis} is domestic cattle, but it has been reported in most mammalian species and is endemic in animal populations throughout the world.\textsuperscript{3,24} \textit{MTB} infection mainly results from spillover of cases from humans to animals.\textsuperscript{1-3}

There has been increased interest in recent years regarding TB in wild and captive animals following outbreaks of disease in zoos, primate centres, animal colonies and game parks.\textsuperscript{1,25} The susceptibility of an animal host to \textit{MTB} complex varies according to the animal species, the host’s level of interaction with humans and other animals, and host-specific immune function. Also relevant is the route of exposure, dose of organisms and virulence of the strains.\textsuperscript{1,3}

Mechanisms of transmission of \textit{M. bovis} between animals include inhalation of respiratory secretions carried as aerosols or droplets; ingestion of feed and water contaminated with urine, faecal material or exudates from infected animals; consumption of infected carcasses by carnivores and scavengers; through fomites such as thermometers, cages or food containers; via congenital transmission; and through the suckling of mothers with tuberculous mastitis.\textsuperscript{1} Genital lesions have been reported and can be an important means of spread.\textsuperscript{1} Pulmonary exudates of cows are usually swallowed and organisms therefore pass with the faeces to contaminate the ground and feed.\textsuperscript{1}

**Domestic animals**

In regard to our study patient and his dog, the literature reveals that there has been genotype confirmed transmission of the same TB strain from owners to their pet dogs,\textsuperscript{26} but there is limited evidence to suggest that domestic animals infected with human TB can infect other humans. An exception is a 1971 study quoted by Erwin et al.,\textsuperscript{26} which reported several cases of transmission of \textit{MTB} back to humans from dogs that acquired the infection from humans. There is much stronger evidence of genotype matched transmission between humans and elephants, and humans and monkeys.\textsuperscript{1}

Cattle, rabbits and cats are susceptible to \textit{M. bovis} but are quite resistant to \textit{MTB}.\textsuperscript{1,3} Ingestion of contaminated materials including milk and offal from infected cattle or wildlife has been identified as the most common source of infection for cats. Wild hoofed stock are generally susceptible to \textit{M. bovis} but there is little evidence for \textit{MTB} infection.\textsuperscript{1} Swine, like dogs, are susceptible to both. Horses can be infected with \textit{M. bovis}, \textit{M. avium} and \textit{MTB}, but on experimental inoculation are relatively resistant. TB in sheep is rare and is mostly due to \textit{M. bovis} and \textit{M. avium} infection. Goats are quite resistant to \textit{MTB} but are susceptible to \textit{M. bovis} and \textit{M. caprae}.\textsuperscript{1} \textit{M. caprae} has also been isolated in a dromedary camel.

**Wild animals**

TB has been infrequently reported in wild animals when they have been exposed to domestic animals or to humans that have the disease.\textsuperscript{1} Despite this, TB is one of the most common infectious diseases and causes of death of captive wildlife in zoological collections worldwide, especially in primates and in countries where there is a high burden of TB.\textsuperscript{25} Contributing factors have been poor hygiene in enclosures as well as lack of adequate veterinary care, overcrowding, unsuitable environments and close contact with visitors.\textsuperscript{25} TB infected humans have been the source of \textit{MTB} infection in Asian elephants and primates as well as other captive wild animals such as a black rhinoceros, oryx and wild goats. The majority of these are considered spillover cases, where infection is present but does not appear to be sustainable in the wild population.\textsuperscript{1} The high genetic diversity among strains of \textit{MTB} in captive animals in a South African Zoological Garden led to the conclusion that the visiting public was the most likely source of the \textit{M. tuberculosis} infection.\textsuperscript{25} Foci of infection may persist in some wild animal populations for long periods of time.\textsuperscript{1}

Clinical signs are only rarely apparent in wild animals. Those with pulmonary disease may exhibit a cough, dyspnoea and enlarged lymph nodes, which may rupture and drain to the
surface in advanced cases. Other presentations include organomegaly, emaciation and a roughened hair coat. Many diseased animals are only found to have TB at necropsy.

The magnitude of the problem

In 2016, the World Health Organization (WHO) estimated that there were 147,000 new cases of zoonotic TB in people and 12,500 deaths due to the disease. However, estimates of the global burden of zoonotic TB are imprecise. Experts believe that the burden of M. bovis is much higher, particularly in developing nations, where the bacterium has a disproportionate impact on communities that are least equipped to treat it. The number of people with zoonotic TB largely exceeds the number of people affected by other diseases that have received greater attention, funding, and resources.

In the United States, M. bovis accounts for 1-4% of human TB cases annually. In areas with large foreign-born populations the prevalence is steadily increasing. In San Diego, California, M. bovis accounted for 45% of TB cases in children and 6% of adult TB cases. The risk for disease increases in areas where bovine TB is endemic and where people live in conditions that favour direct contact with infected animals (i.e. farmers, veterinarians, and slaughterhouse workers) or with animal products (unpasteurised milk and untreated animal products). Areas where bovine TB is endemic sometimes overlap with areas where HIV prevalence is high (namely in some African countries) adding further to the impact of the disease.

The burden of bovine TB also extends into the socioeconomic sphere through economic losses due to livestock deaths, restrictions for trading animals both at the local and international level and losses in productivity due to chronic disease in livestock and workers. This economic loss has an important effect on livelihoods, particularly in poor marginalised communities. Further impacts relate to the need for funding for surveillance activities and regular testing of cattle; the removal and culling of infected animals and other in-contact animals in the same herd; and movement control of infected herds. In view of the subsistence nature and reliance on animals as a source of livelihood in low-income countries, it is expected that the economic effect to the individual farmer will be important.

Bovine TB eradication efforts

The United Nations Sustainable Development Goals (SDGs) include a target for ending the global TB epidemic by 2030 (SDG 3, Target 3.3). The WHO, World Organization for Animal Health (Organization International for Epizootics, OIE), Food and Agricultural Organization of the United Nations (FAO) and International Union Against Tuberculosis and Lung Disease (The Union) all advocate that a strategy to end global TB must include measures to address zoonotic TB. The FAO has prioritised bovine TB as an important infectious disease that should be controlled at the animal-human interface through national and regional efforts. The prevention and control of zoonotic TB needs a cross-sectoral and multidisciplinary approach, linking animal, human and environmental health. This ‘One Health’ approach is increasingly being endorsed by many prominent organisations.

For humans, M. bovis is naturally resistant to pyrazinamide, one of the four medications used as standard first-line antituberculosis treatment. This is problematic as most patients worldwide begin TB treatment without identification of the causative mycobacterium species and risk having inadequate treatment if they have bovine TB. Globally in 2014, only 12% of 2.7 million new bacteriologically confirmed TB cases were tested for drug resistance. Due to the innate resistance of M. bovis to pyrazinamide, the standard 6 months of therapy for MTB is not ideal and a longer course of 9 months of antituberculous therapy is recommended. This leads to the additional challenges of decreased patient adherence and increased costs associated with prolonged therapy.

M. bovis is a multi-host pathogen that thrives in complex systems at the wildlife-livestock interface. This makes eradication unlikely unless the role of wildlife hosts in transmission cycles is understood and all relevant reservoirs are targeted at the same time. Measures such as tuberculin skin testing (TST) and drug treatment,
which are important in control and elimination of TB in humans, are unreliable and impractical in many animal species.\textsuperscript{1,3} Necropsies are the gold standard for investigating TB but are not conducted frequently. Bacillus Calmette-Guérin (BCG) vaccines are not utilised in animals as they fail to protect against infection, they do not prevent progression of disease and they interfere with interpretation of TST results.\textsuperscript{3} Vaccination with BCG has been attempted in cattle with limited success.\textsuperscript{1}

Eradication of \textit{M. bovis} in cattle and pasteurisation of dairy products remain the cornerstones of prevention of human disease. Control measures based on surveillance in abattoirs, test-and-slaughter policy on farms and disease notification have been intensified in the last decades in several South American countries.\textsuperscript{3} Elimination of TB from cattle stock may not be enough however, as there are now several species of wildlife that are recognised to be reservoirs of \textit{M. bovis} that can sustain the infection and cause spillback to cattle.\textsuperscript{1} These include ungulates such as buffalo and bison, and cervids such as deer and elk. European badgers are a reservoir in the United Kingdom as are brushtail possums in New Zealand.\textsuperscript{1}

Disease control in wildlife is difficult, and outbreaks of TB in cattle due to exposure from wildlife are costly. Approaches to address this include reducing wildlife reservoir populations and using biosecurity measures to separate infected wildlife from cattle, feed, water and shelter.\textsuperscript{1} Wildlife free buffer areas reduce contact opportunities between wildlife and livestock. Elimination of TB from wild animals has also been hampered by the lack of suitable regulations to limit the sale and transportation of infected or exposed wild animals.\textsuperscript{1}

Transmission modelling research raises hope that if the TB load is controlled in important animal reservoirs such as cattle, then significant progress toward TB eradication can follow. When TB in cattle in the same geographic areas was eliminated or reduced, comparable declines in the prevalence in wildlife were also seen.\textsuperscript{1}

In New Zealand there is hope that bovine TB will be eliminated from wildlife species. Since its first detection in 1967, bovine TB has spread to wildlife populations in over 35% of its land area.\textsuperscript{2} Through the combined control measures in livestock and wildlife, only 0.2% of herds were infected.\textsuperscript{2} These measures included improvements in the diagnosis and eradication of TB in cattle and deer herds, and stringent movement control and animal identification of livestock. An eradication campaign against wildlife reservoirs is being pursued as none of the key wildlife hosts are native animals (namely possums, deer, pigs and ferrets). This can be achieved through improved methodology for large-scale toxin deployment backed by the development of new surveillance methods to detect wildlife at low population densities.\textsuperscript{2}

There is international consensus that in order to address bovine TB there is a need for collaboration between clinicians, researchers, and public health practitioners in the medical, veterinary, social science and economic fields, under the umbrella of One Health.\textsuperscript{1,24,27} Combining expertise and efforts from different fields and institutions will broaden the scope of options to address the challenges at the animal-human interface and reduce unnecessary duplication and efforts in disease control programs.\textsuperscript{3,23} Targeted research is needed to assess the spillback risks from less relevant wildlife hosts to domestic animals and then to humans.\textsuperscript{2}

An alliance of WHO and UN bodies (OIE, FAO, The Union) commissioned the ‘Roadmap for Zoonotic Tuberculosis’ in 2017 with 10 priority areas for tackling zoonotic TB (see the Box).\textsuperscript{24} These are divided into 3 areas: improving the scientific evidence; reducing transmission at the animal-human interface; and strengthening intersectoral and collaborative approaches.

**Conclusion**

From this review the following conclusions can be drawn: humans can transmit TB to animals; animals can transmit disease to humans; the transmission of bovine TB between different animal species is an important issue in the global campaign to eradicate TB; a number of measures
can be undertaken to control or eliminate zoonotic TB; to be successful, action must be undertaken as an intersectoral and collaborative effort under the umbrella of One Health.

Acknowledgements

Our thanks go to the dedicated TB staff for closely monitoring and supporting the patient and for not overlooking the pooch either.

References


Figure. Which of the following animals are said to have been infected with TB from humans? Which of these animals have had bovine TB?

The animals featured are a goat, gorilla, possum, elephant, rabbit, hippopotamus, pig, bison, monkey, badger, deer/elk, cow, cat, dog, sheep, camel and koala.

Answers to Figure questions
The following animal species have been known to have been infected with human TB: gorilla, elephant, pig, bison, monkey, deer/elk, cat and dog. It should be noted that cats are quite resistant to human TB.

All of the featured animals bar the koala and hippopotamus are documented to have had bovine TB.

The image was collated and formatted from silhouettes of individual animals as free clipart images from the following internet sources: www.Clipartqueen.com, www.openclipart.org and www.GetDrawings.com.

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An outbreak of gastroenteritis caused by contaminated drinking water at a school camp in the Top End, May 2018

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Abstract

An outbreak of gastroenteritis occurred following attendance at an overnight school camp on 24 and 25 May 2018, in a remote area of the Top End of the Northern Territory (NT), Australia.

Environmental Health (EH) Officers from the EH Branch undertook an investigation and collected water samples. A retrospective cohort study was conducted via telephone interviews, using a structured questionnaire that recorded symptoms and exposures to foods and activities during the camp.

Of the 31 people who attended the camp (22 students and 9 adults), 18 (58%) became ill. We interviewed 29/31 (95%) of attendees and 16 were cases (2 cases declined to participate however they did provide information on their symptoms). The most commonly reported symptoms were diarrhoea (100%, 18/18), abdominal pain (17/18, 94%), lethargy (16/18, 89%) and nausea (11/18, 61%). Two people sought medical attention but none required hospitalisation.

Illness was significantly associated with drinking water from the kitchen tap at the camp (aOR 18.0, 95% CI 1.3-248, p=0.03). Escherichia coli was detected in water samples above safe drinking water levels, indicating that it was unsuitable for drinking. The camp was not supplied with mains reticulated drinking water and a combination of factors in the private water supply are likely to have led to the unsuitability of the water for drinking, but the causative organism was not identified. This outbreak highlights the challenges of maintaining a safe private drinking water supply and the risks it may present.

Key words: outbreak; gastroenteritis; waterborne disease; cohort study; public health; camp; Northern Territory.

Background

Enteric illness is caused by the presence of organisms and toxins in food or water and can result in symptoms such as diarrhoea, nausea, vomiting, fever, abdominal pain and even death. Foodborne illnesses can be caused by bacteria, parasites, viruses, or toxins produced by bacteria or algae.1 It is estimated that 80% of foodborne illness in Australia is due to unknown causes.2

On 29 May 2018, the Northern Territory (NT) Environmental Health Branch (EHB) was alerted to a possible outbreak of gastroenteritis among school students who had recently attended an overnight school camp on 24 and 25 May 2018. This was reported to the EHB by the parent of one child and confirmed by the school who were notified of several other reports of illness. The school camp was at a remote outback location and was attended by 31 people who slept in tents. Food and drink was prepared and served from a camp kitchen by the school group. An outbreak investigation was initiated to identify the cause of illness and implement appropriate public health measures to prevent illness in groups attending the campsite in future. This paper describes the outbreak investigation.

Methods

Environmental health investigation

Environmental Health Officers (EHOs) from the EHB undertook an initial investigation at the campsite on 31 May 2018 and a follow-up visit on 5 June 2018. The EHOs inspected food preparation and storage areas, collected information about food preparation, storage and procurement procedures and inspected public health facilities including water supply and ablation facilities at the camp.

Water samples were collected from a storage tank, ablutions, the camp kitchen and a nearby creek and tested at the NT Department of Primary Industry and Fisheries (DPIF) Water Microbiology Laboratory in Darwin, NT for the presence of coliforms, Escherichia coli (E. coli)
Environmental health investigation

The campsite as visited on 31 May 2018 had basic facilities to enable groups to run self-sufficient camping trips. No fixed accommodation or catering was provided to guests. A camp kitchen, ablation facilities and drinking water supply were provided for camp users. The facilities appeared to be generally clean and in good repair.

Food and a minimal supply of personal drinking water (up to 2L) was provided by the school or brought by each of the students for the duration of the camp. The menu items were considered low-risk and included breakfast cereal, biscuits, fruit, baked potatoes, baked beans, cheese and sausages in bread. Meals were prepared by the group in the camp kitchen. No food remained for testing.

The wastewater from the ablation blocks was collected in a septic tank on site. It appeared to be in good condition and no odour or pooling was observed. Wastewater from the kitchen was disposed of to the ground and not to the septic tank.

The camp facilities were supplied with a private water supply (not mains reticulated) that was sourced from surface water pumped directly from a nearby creek. The water was pumped into holding tanks and reticulated around the site. The lid to one of the storage tanks was not secured indicating the tank was open to environmental exposure. The water supplied to the 2 main sinks in the camp kitchen was passed through an ultraviolet (UV) treatment system, however all other water access points around the camp site were supplied with untreated water. The water supply tank for the caretakers’ residence was supplied with a separate rainwater supply. There was only one sign in the female ablutions which may have indicated the unsuitability of the water for drinking. It stated that the water was ‘unfiltered’ and to use the kitchen tap for ‘filtered water’. The sign however did not indicate whether the water was safe for drinking. Other water taps were located around the site including one outside the camp kitchen and one outside the ablation blocks but without signage as to whether the water was suitable for drinking. It is believed that 1 of these taps was used to fill containers for a children’s water play activity; a water fight.

Results of water testing on samples collected on 31 May 2018 are shown in Table 1. The results did not meet ADWG for drinking water which states that E. coli should not be detected in any 100mL sample of water. There are no specific targets for total coliform or heterotrophic colony counts in...
the ADWG but counts show the presence of bacteria in the water supply. The presence of *E. coli* in the water sampled from the kitchen tap indicates that the UV treatment system in place was ineffective. There is no standard for total coliform or heterotrophic colony count in the ADWG but these were tested as part of the laboratory’s standard testing panel.

**Epidemiological and laboratory investigation**

Of the 31 people who attended the camp (22 students and 9 adults), 18 became ill (attack rate 58%). We interviewed 29 of 31 (response rate 95%) of those who attended the camp (2 cases provided details of symptoms but declined to participate further). The most commonly reported symptoms were diarrhoea (100% 18/18), abdominal pain (17/18, 94%), lethargy (16/18, 89%) and nausea (11/18, 61%). Two people sought medical attention but none required hospitalisation. Table 2 describes demographics of the cases and their symptoms.

The epidemic curve was typical of a point source outbreak (Figure 1). There was no significant difference in age and sex between cases and other attendees.

We measured exposure against 48 variables and univariate analysis showed that, while no exposures reached statistical significance, drinking water from the kitchen tap had the highest relative risk and lowest p value (RR 3.9, 95% CI 0.6-24.0, p=0.0638).

Multivariate analysis revealed that, when adjusted by age and gender, drinking water from the kitchen tap was significantly associated with illness (Table 3).

**Table 1. Results of water sampled on 31 May 2018**

<table>
<thead>
<tr>
<th></th>
<th><em>E. coli</em> (MPN* per 100mL)</th>
<th>Total coliforms (MPN* per 100mL)</th>
<th>Heterotrophic colony count (CFU†/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rainwater tank</td>
<td>&lt;1</td>
<td>299</td>
<td>620</td>
</tr>
<tr>
<td>Male toilet tap (slug)</td>
<td>6</td>
<td>23</td>
<td>280</td>
</tr>
<tr>
<td>Male toilet tap (flush)</td>
<td>47</td>
<td>&gt;2420</td>
<td>2000</td>
</tr>
<tr>
<td>Kitchen tap (slug)</td>
<td>50</td>
<td>816</td>
<td>3500</td>
</tr>
<tr>
<td>Kitchen tap (flush)</td>
<td>99</td>
<td>1553</td>
<td>13000</td>
</tr>
<tr>
<td>Ablution hose tap (slug)</td>
<td>30</td>
<td>345</td>
<td>2200</td>
</tr>
<tr>
<td>Ablution hose (flush)</td>
<td>18</td>
<td>461</td>
<td>380</td>
</tr>
<tr>
<td>Concrete storage tank</td>
<td>140</td>
<td>&gt;2420</td>
<td>3800</td>
</tr>
<tr>
<td>Creek</td>
<td>172</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* MPN = most probable number, † CFU = colony forming units

**Table 2. Characteristics and symptoms of cases who attended a school camp on 24-25 May 2018 (n=18)**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographic</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Student</td>
<td>15</td>
<td>83</td>
</tr>
<tr>
<td>Adult</td>
<td>3</td>
<td>17</td>
</tr>
<tr>
<td>Female</td>
<td>10</td>
<td>56</td>
</tr>
<tr>
<td>Male</td>
<td>8</td>
<td>44</td>
</tr>
<tr>
<td><strong>Symptoms</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>18</td>
<td>100</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>17</td>
<td>94</td>
</tr>
<tr>
<td>Lethargy</td>
<td>16</td>
<td>89</td>
</tr>
<tr>
<td>Nausea</td>
<td>11</td>
<td>61</td>
</tr>
<tr>
<td>Vomiting</td>
<td>5</td>
<td>28</td>
</tr>
<tr>
<td>Headache</td>
<td>3</td>
<td>18</td>
</tr>
<tr>
<td>Fever</td>
<td>2</td>
<td>11</td>
</tr>
<tr>
<td>Bloody diarrhoea</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**Table 3. Results of multivariable analysis for all interviewed attendees who attended a school camp in the Top End from 24-25 May 2018 (n=29)**

<table>
<thead>
<tr>
<th>Exposure</th>
<th>aOR*</th>
<th>95%CI†</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drinking water from kitchen</td>
<td>18.0</td>
<td>1.3-248</td>
<td>0.031</td>
</tr>
<tr>
<td>Being male</td>
<td>0.4</td>
<td>0.1-2.3</td>
<td>0.277</td>
</tr>
<tr>
<td>Being a child</td>
<td>3.6</td>
<td>0.5-28</td>
<td>0.220</td>
</tr>
</tbody>
</table>

* aOR = Adjusted odds ratio, † CI = Confidence interval
Two cases submitted stool samples; both were negative on culture and 1 tested positive for astrovirus by PCR.

**Public health response to findings**

Following notification of the water sampling results, the camp owner hand-dosed the water tanks with chlorine. Sampling after this chlorine treatment indicated that the disinfection treatment was effective and the water results met the ADWG (Table 4).

The UV treatment system was tested by a service engineer who confirmed that the unit was operating, but changed the UV bulbs in case they were defective.

The camp owner implemented the practice of hand-dosing the storage tanks with chlorine at an appropriate concentration, prior to all future groups attending the site. Signage warning campsite users of the untreated water supply were also put up at all water access points except the camp kitchen.

**Discussion**

Outbreaks of waterborne illness at places that do not use a treated mains water supply are rare but require investigation to prevent future occurrences. Our investigation concluded that the consumption of contaminated drinking water caused the outbreak of gastroenteritis. The conclusion is supported by the detection of ‘indicator’ E. coli above ADWG acceptable levels and a statistically significant association between drinking the water from the kitchen and illness.

The UV treatment of the water supply appeared to be working, however did not result in a safe drinking water supply. This may be due to gross contamination levels in the surface water supply or the ineffectiveness of the UV system to treat the water due to high levels of organic material and lack of maintenance of the unit. Ongoing service and maintenance should be implemented to ensure the UV treatment system is effective and an ongoing testing regimen should be put in place.

We were unable to determine the organism that caused the outbreak; the presence of E. coli in drinking water samples is merely an indicator of contamination of the water supply with bacteria and E. coli should not be considered to be the cause of the outbreak. E coli are not usually pathogenic and illness caused by pathogenic E

**Table 4. Results of water sampled on 5 June 2018 (post chlorination)**

<table>
<thead>
<tr>
<th></th>
<th>E. coli (MPN* per 100mL)</th>
<th>Total coliforms (MPN* per 100mL)</th>
<th>Heterotrophic colony count (CFU†/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kitchen tap (flush)</td>
<td>&lt;1</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Concrete storage tank</td>
<td>&lt;1</td>
<td>&lt;1</td>
<td>26</td>
</tr>
</tbody>
</table>

* MPN = most probable number, † CFU = colony forming units
coli would be more severe than that of this outbreak.

One stool sample tested positive for astrovirus but this was not likely to be the cause of the outbreak and is considered an incidental finding. Astrovirus has a typical mean incubation period of 4.5 days and is characterised by mild diarrhoea associated with vomiting, fever, anorexia and abdominal pain. It is typically spread from person to person. The person who tested positive had an onset of symptoms on 27 May; 2 days after the conclusion of the camp. Furthermore, the shape of the epidemic curve supports a point source exposure rather than person-to-person transmission.

Attendees at this school camp had been instructed to bring at least 2 litres of water with them from home to consume during the camp. It is feasible that once this supply was exhausted, people filled their drink bottles up from the kitchen at the campsite. This would likely explain the close clustering of onset times and suggests a common exposure at the end of 24 May or at the start of 25 May. Only 2 cases submitted samples as they were still symptomatic at the commencement of the cohort study. Most cases experienced only mild diarrhoea of short duration.

We minimised measurement bias by using a standard questionnaire based on the menu, itinerary and activities undertaken at the camp. Selection bias was minimised by obtaining the list of attendees at the camp enabling a large proportion of attendees to be contacted 29/31 (95%).

Conclusion

We conclude that an outbreak of gastroenteritis affecting attendees at a 2 day school camp was caused by drinking contaminated water. The presence of E. coli in samples of the water supply from the camp confirmed that it was contaminated. The causative organism of the outbreak however is unknown.

Camp groups attending locations away from mains water supplies should consider carrying sufficient water from a reputable source for attendees to consume throughout their trip or ensure that a safe drinking water supply can be obtained while on site. Camp ground operators need to ensure that untreated private water supplies are safe to drink by establishing an appropriate water management plan which includes sufficient treatment of the water supply and appropriate verification, testing and monitoring procedures to demonstrate its safety. Clear signage is required to indicate what taps are safe for drinking.

Acknowledgements

The authors acknowledge Dominique Giese for her assistance in the investigation and the camp ground operator and school for their cooperation in the investigation.

References

OzFoodNet is a network of epidemiologists in the health departments of every Australian state and territory who focus on foodborne illness surveillance. The network is funded and coordinated by the Commonwealth Department of Health. OzFoodNet is a member of the Communicable Disease Network of Australia (CDNA).

OzFoodNet's mission is to apply concentrated effort at a national level to investigate and understand foodborne disease, to describe more effectively its epidemiology and to identify ways to minimise foodborne illness in Australia. Information sharing is crucial for the success of OzFoodNet and members communicate frequently about issues such as unexpected clusters of foodborne disease, current outbreak investigations and changes in laboratory testing and surveillance.

The Northern Territory (NT) OzFoodNet Epidemiologist hosted the 55th OzFoodNet face-to-face meeting in Alice Springs on 19-20 June; the first time the meeting has been held outside a major metropolitan area. Foodborne disease experts from around Australia discussed outbreaks currently under investigation, surveillance issues and current and future projects.

Staff from the NT Centre for Disease Control and the Environmental Health branch presented on the epidemiology of enteric disease in Central Australia as well as some of the unique aspects and challenges of delivering public health services in remote Australia. A large portion of the first day of the meeting was spent discussing the current increase in shigellosis which has been occurring in the NT and South Australia for the past 18 months. Other interstate presenters spoke about innovative surveillance methods and the increasing use of whole genome sequencing as a tool in outbreak investigations.

Photo. Delegates at the 55th OzFoodNet face-to-face meeting, Alice Springs, 19-20 June 2018
Epidemiology and aetiology of severe community acquired pneumonia at Royal Darwin Hospital Intensive Care Unit
Ian Marr,1,2 Resy van Beek,3 Rob Baird2 Anna P. Ralph1,4
1. Infectious Diseases Department, Royal Darwin Hospital (RDH); 2. Territory Pathology, RDH; 3. Intensive Care Department, RDH; 4. Menzies School of Health Research, Darwin.

Abstract

The aetiology of severe community acquired pneumonia (CAP) differs between tropical Australia and temperate parts of the country. We undertook an audit to describe the implementation of a new testing algorithm to determine the additional diagnostic yield, if any, of a newly-introduced rapid diagnostic test in severe CAP in an Intensive Care Unit (ICU) in a tertiary facility in Australia’s Northern Territory.

All adult patients with severe CAP admitted during a 12-month period were included. A diagnostic algorithm was implemented whereby patients in whom the microbiological diagnosis remained uncertain at 48 hours of intubation were eligible to have an endotracheal tube aspirate tested using a multiplex real-time polymerase chain reaction (RT-PCR) (BioFire Diagnostics, Salt Lake City, UT). This detects Adenovirus, Coronaviruses, Human metapneumovirus, Rhinovirus, Enterovirus, Influenza A and B, Parainfluenza 1-4, Respiratory syncytial virus (RSV), Bordetella pertussis, Chlamydophila pneumoniae and Mycoplasma pneumoniae.

There were 86 eligible CAP diagnoses during the study period. We found that a microbiological aetiology was detected using standard diagnostics in 50/86 (58%). Most common pathogens were: Streptococcus pneumoniae (10), Burkholderia pseudomallei (9), Influenza (9), Staphylococcus aureus (8), Acinetobacter baumannii (3), Pseudomonas aeruginosa (3), Mycobacterium tuberculosis (2) and Nocardia sp (1).

Eight patients were eligible for testing using the RT-PCR according to the algorithm; 5 tests were performed showing Rhinovirus/Enterovirus (1), Parainfluenza-3 (1) and Respiratory Syncytial Virus (1) or no pathogens (2). Therefore, the additional diagnostic yield using the algorithm in this patient subset was low.

Multiplex testing for respiratory infections may have an important place, but few patients were eligible using a strict algorithm, and clinician uptake was low. Implementation of a new test requires careful consideration about strategies for targeted use and clinician education. Current local guidelines on empirical CAP antimicrobial therapy are appropriate for the range of pathogens which predominate in this tropical environment.

Keywords: Community acquired pneumonia; reverse transcriptase polymerase chain reaction; Northern Territory.

Background

Within Australia, the tropical north has a unique spectrum of seasonal severe pneumonia.1 The dominant aetiologies include Burkholderia pseudomallei and Acinetobacter baumannii; in addition to Streptococcus pneumoniae and Staphylococcus aureus.1-5 This contrasts with data from southern Australia. One large study found the dominant aetiological agents were Streptococcus pneumoniae (14%), Mycoplasma pneumoniae (9%) and respiratory viruses (15%; including influenza, picornavirus, respiratory syncytial virus, parainfluenza virus, adenovirus).6 Another found the top microbiological causes to be Streptococcus pneumoniae, Mycoplasma pneumoniae and Haemophilus influenzae.7

Pneumonia studies consistently demonstrate that identification of a causative pathogen is the exception rather than the rule, at 35-45% in prospective studies,1,3 due to challenges in obtaining and interpreting microbiological information from the respiratory tract. Meanwhile community acquired pneumonia (CAP) continues to be a leading cause of morbidity and mortality,8 the investigation of which consumes significant resources.9,11

Contributing to the low diagnostic yield are difficulties in accessing uncontaminated specimens, bacteria with fastidious growth requirements, and a reliance on suboptimal
serological assays. Many of these assays require extended time before seroconversion, have prolonged turnaround times and limited positive predictive value.12

The growing availability of nucleic acid amplification test (NAAT) based multiplex platforms provides optimism for improved accuracy in the diagnosis of respiratory tract infections.

The aims of this prospective descriptive audit were to define the microbiological aetiology of severe CAP in the setting of a tropical Intensive Care Unit (ICU), and to determine the additional diagnostic yield, if any, of a newly-available FilmArray real-time polymerase chain reaction (RT-PCR) multiplex platform.

Methods

All patients admitted to the ICU at the Northern Territory (NT) Top End tertiary referral centre (Royal Darwin Hospital, RDH) between 31 August 2015 and 1 September 2016 with severe CAP requiring endotracheal intubation and ventilation were identified. Inclusion criteria comprised 2 of the following symptoms: cough, sputum production, fever > 37.8°C, pleuritic chest pain, dyspnoea, altered mental status or white cell count greater than 12 x 10⁹/L, plus a radiologically confirmed new infiltrate on imaging taken within 24 hours of admission.

An algorithm was developed for application of the newly-available FilmArray respiratory RT-PCR multiplex screening (BioFire Diagnostics, Salt Lake City, UT) by agreement between the ICU, Infectious Diseases and Microbiology departments. The algorithm indicated that a lower respiratory tract sample could be submitted for multiplex RT-PCR in patients with severe CAP who were still ventilated at 48 hours and in whom no microbiological diagnosis was apparent, or if there was an alternative strong clinical indication to do the test. This multiplex assay includes primers for Adenovirus, 4 separate Coronaviruses, Human metapneumovirus, Rhinovirus, Enterovirus, Influenza A (H1, H1-2009 and H3), Influenza B, Parainfluenza 1-4, RSV, B. pertussis, C. pneumoniae and M. pneumoniae multiplex RT-PCR.

Other microbiological investigations were undertaken at clinicians’ discretion. These included blood culture, in which growth of a feasible pneumonic pathogen (in addition to the clinical and radiological criteria) was considered significant; sputum culture, where growth was considered significant if >25 leukocytes and <10 epithelial cells were present on microscopy; urinary antigen testing for Legionella pneumophila serogroup 1 and Streptococcus pneumoniae; polymerase chain reaction (PCR) for selected respiratory viruses on upper respiratory tract swab specimens or lower respiratory tract samples for Influenza A/B and RSV 1/B; and serology for respiratory pathogens, where a 4-fold rise in titre was considered significant for C. pneumoniae enzyme immunoassay (EIA) IgA and IgG, L. pneumophila and L. longbeachae immunofluorescent assay (IFA) and M. pneumoniae (EIA) IgM and IgG. B. pseudomallei serology using indirect hemagglutination assay (IHA) was interpreted as previously described.11

Patient data were collected prospectively from the ICU Australian and New Zealand Intensive Care Society (ANZICS) database and medical files, and included: clinical, demographic, radiological and microbiological results; final diagnosis, APACHE score, duration of intubation, and final outcome. Descriptive data analyses were used. Summary statistics are presented as medians and interquartile ranges.

The study was submitted to the Human Research Ethics Committee of the NT Department of Health and Menzies School of Health Research as a Quality Assurance Audit and was approved for inclusion in the Quality Assurance Audit Register (QAAR 2015-2475). No funding was required for this study.

Results

There were 86 diagnoses of CAP among adults in the ICU during the study period. Patient characteristics are shown in Table 1. The median age was 56 years (IQR 17.7). Females accounted for 51% (44/86) of cases, the median length of ICU stay was 6.5 days and 33/86 required intubation for over 24 hours. Mortality occurred in 7 cases (8%).
Conventional microbiology

Culture of blood and/or sputum samples provided a microbiological aetiology for CAP in 58% (50/86) of individuals. Of these, *S. pneumoniae* (n=10), *B. pseudomallei* (n=9), *S. aureus* (n=8), and *A. baumannii* (n=3) were the top 4 bacteriological agents identified (Table 2). Influenza was the most prevalent viral agent identified (n=9). Co-infections included combinations of *S. pneumoniae* with *Influenza B* or *Moraxella*, *S. aureus* with *Pseudomonas*, and *A. baumannii* with *Stenotrophomonas spp.*

Table 1. Patient characteristics for ICU CAP at Royal Darwin Hospital (2015-2016)

<table>
<thead>
<tr>
<th>Patient Characteristics</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>86</td>
</tr>
<tr>
<td>Age in years: median (IQR)*</td>
<td>56 (17.7%)</td>
</tr>
<tr>
<td>Female: n (%)</td>
<td>44 (51%)</td>
</tr>
<tr>
<td>COPD†</td>
<td>12 (7%)</td>
</tr>
<tr>
<td>ESRF‡/ CRF§</td>
<td>8 (8.6%)</td>
</tr>
<tr>
<td>Immunosuppression</td>
<td>7 (8%)</td>
</tr>
<tr>
<td>Non-invasive ventilation</td>
<td>27 (31.4%)</td>
</tr>
<tr>
<td>Intubated</td>
<td>34 (40%)</td>
</tr>
<tr>
<td>Inotropic support</td>
<td>50</td>
</tr>
<tr>
<td>ICU length of stay in days: median</td>
<td>5.2</td>
</tr>
<tr>
<td>ICU unit length of stay in hours: median</td>
<td>131</td>
</tr>
</tbody>
</table>

*IQR (Interquartile range), †COPD (Chronic obstructive airways disease), ‡ESRF (End stage renal failure), §CRF (Chronic renal failure)

Multiplex respiratory PCR

Eight individuals fulfilled the criteria for multiplex respiratory PCR testing (i.e. were without a microbiological diagnosis and still intubated at 48 hours). Five underwent testing of endotracheal aspirate samples with the respiratory BioFire multiplex PCR. Pathogens were identified in 3 of the 5 individuals tested, with 1 result each positive for Rhinovirus/ Enterovirus, Parainfluenza-3 and Respiratory syncytial virus. Two multiplex samples were negative. Of note, Rhinovirus was not considered to be a cause of severe CAP. The test was also done in an additional 2 ICU patients with severe CAP not fulfilling criteria for testing, following clinician-initiated requests.

Table 2. Twelve-month aetiology and severity indicators of CAP at Royal Darwin Hospital 2015-2016

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Cases</th>
<th>ICU LOS* (median days)</th>
<th>APACHE† III (median)</th>
<th>Intotropes needed no.</th>
<th>Age (median years)</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Streptococcus pneumoniae</em></td>
<td>10</td>
<td>7.2</td>
<td>72.4</td>
<td>8</td>
<td>46</td>
</tr>
<tr>
<td><em>Burkholderia pseudomallei</em></td>
<td>9</td>
<td>9.2</td>
<td>78.5</td>
<td>7</td>
<td>49</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-MSSA</td>
<td>5</td>
<td>7.8</td>
<td>57.6</td>
<td>1</td>
<td>46</td>
</tr>
<tr>
<td>-nmMRSA‡</td>
<td>3</td>
<td>5.9</td>
<td>70.4</td>
<td>1</td>
<td>48</td>
</tr>
<tr>
<td>-mMRSA§</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Influenza</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- A</td>
<td>7</td>
<td>7.1</td>
<td>70</td>
<td>3</td>
<td>47</td>
</tr>
<tr>
<td>- B</td>
<td>2</td>
<td>5.2</td>
<td>78</td>
<td>0</td>
<td>58</td>
</tr>
<tr>
<td><em>Pseudomonas aeruginosa</em></td>
<td>3</td>
<td>5.9</td>
<td>67</td>
<td>2</td>
<td>57</td>
</tr>
<tr>
<td>RSVI</td>
<td>1</td>
<td>4.7</td>
<td>11</td>
<td>0</td>
<td>61</td>
</tr>
<tr>
<td><em>Acinetobacter baumannii</em></td>
<td>3</td>
<td>6</td>
<td>101.3</td>
<td>3</td>
<td>55</td>
</tr>
<tr>
<td><em>Mycobacterium tuberculosis</em></td>
<td>2</td>
<td>11.6</td>
<td>27</td>
<td>0</td>
<td>43</td>
</tr>
<tr>
<td>Nocardia</td>
<td>1</td>
<td>4.8</td>
<td>29</td>
<td>1</td>
<td>71</td>
</tr>
<tr>
<td><em>Haemophilus influenzae</em></td>
<td>1</td>
<td>1.9</td>
<td>35</td>
<td>0</td>
<td>56</td>
</tr>
<tr>
<td><em>Group A streptococcus</em></td>
<td>1</td>
<td>2.8</td>
<td>90</td>
<td>1</td>
<td>64</td>
</tr>
<tr>
<td>Parainfluenza-3</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td>48</td>
</tr>
<tr>
<td>Leptospirosis</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td>52</td>
</tr>
<tr>
<td>No microbiological cause</td>
<td>36</td>
<td>5.9</td>
<td>70</td>
<td>19</td>
<td>58</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>86</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*LOS (Length of stay), †APACHE (Acute Physiology and Chronic Health Evaluation III), ‡nmMRSA (non-multi-resistant Methicillin resistant *Staphylococcus aureus*), §mMRSA (multi-resistant Methicillin resistant *Staphylococcus aureus*), IRSV (respiratory syncytial virus).
Results comprised 1 Rhinovirus/Enterovirus positive case with concurrent Streptococcus pneumoniae, and 1 Rhinovirus/Enterovirus in a case that was confirmed to be Mycobacterium tuberculosis (MTB) positive on subsequent culture.

**Serology and urinary antigen testing**

Of the 36 cases without a microbiological diagnosis, 25 had Mycoplasma spp. IgM and IgG serology tested at presentation; none showed an elevated titre acutely and no convalescent serology was performed at our institution. Legionella spp. serology was tested in 23 individuals. One case had convalescent serology at 14 days, showing no rise in titre. Chlamydophila pneumoniae serology was tested at presentation in 22 individuals; 7 had low-level IgG and 8 low level IgA, not fulfilling diagnostic criteria. No Chlamydophila convalescent serology or follow up of equivocal results was performed at our pathology provider. Streptococcus pneumoniae urinary antigen was performed in 28 individuals within 48 hours and Legionella pneumophila urinary antigen in 24 with no positive results for either being recorded.

Of individuals without a microbiological diagnosis (i.e. not including those with B. pseudomallei detected on culture), 22 had B. pseudomallei (IHA) serology performed, and 4 had 10-14 day convalescent serology, with none showing a subsequent rise of titre. One individual had a B. pseudomallei (IHA) titre of 1:2560 which was actively investigated but no B. pseudomallei was cultured and in the clinical context, the serology was interpreted as representing past exposure. Targeted B. pseudomallei treatment was not prescribed and the patient improved with empirical CAP therapy, therefore active B. pseudomallei accounting for the current presentation was considered unlikely.

**Discussion**

In this study, we enrolled the most unwell subset of patients with CAP at a single tropical Australian institution to determine the additional diagnostic yield of a rapid diagnostic test. Key findings were that a microbiological aetiology was detected more commonly than anticipated overall, that the new diagnostic test was underutilised despite an algorithm for its use, and that the results from the new diagnostic were unlikely to change clinician choices on antimicrobials.

We detected a microbiological aetiology in 57% of patients. While this is substantially higher than previous reports in the general hospitalised CAP population,\(^3\) we included only patients with the most severe disease who are more likely to have high-organism burden disease, therefore more likely to be detected. Secondly, leading tropical pathogens Burkholderia pseudomallei and Acinetobacter baumannii are readily culturable (when using the correct selective media).

The spectrum of bacterial causes of CAP in this severely unwell, ventilated group closely mirrors findings from a previous report from our setting of bacteraemic pneumonias;\(^1\) both studies found Streptococcus pneumoniae as the leading cause followed closely by Burkholderia pseudomallei, then Staphylococcus aureus and Acinetobacter baumannii. The finding of nocardia in 1 case and tuberculosis in 2 is not unexpected in this setting.\(^{14,15}\)

The median APACHE III score of 65 approximated those from previous ICU studies for pneumonia.\(^9\) The mortality rate from this study of severe CAP in a tropical ICU in Darwin was 7/86 (8%). This approximates mortality rates reported in studies of CAP in ICU in other Australian settings.\(^{16,17}\)

Institution of RT-PCR multiplex testing for diagnostically uncertain cases gave few satisfactory diagnoses in this study. Restricting use to those intubated patients without a microbiological cause at 48 hours limited absolute numbers tested. In this most severely unwell patient cohort, treatment changes are unlikely to be implemented on the basis of results; detection of 1 of the 8 viral genera included in the RT-PCR would be unlikely to result in antibiotic de-escalation due to clinical concerns that it may be a co-pathogen rather than the primary cause. Similarly detection of B. pertussis, C. pneumoniae or M. pneumoniae might also not change treatment since patients already receive a macrolide antibiotic as part of the empirical regimen in ICU - although a strong
argument could be made to cease beta-lactam therapy if C. pneumoniae or M. pneumoniae were identified. It has long been thought that agents of 'atypical' pneumonia (in particular C. pneumoniae and M. pneumoniae) are uncommon in our setting. This study supports that conclusion, but low uptake of the RT-PCR and convalescent serology meant that an accurate estimate of the proportion of CAP attributable to these pathogens is still lacking. In the tropical regions of Australia there are also few data on rates of L. longbeachae, a recognised cause of severe pneumonia elsewhere in Australian and New Zealand.18,19

The expansion of multiplex testing for respiratory illnesses may well have a valuable role in the diagnosis of upper and lower respiratory tract infections. We chose to use the test in the most unwell patients in the first instance to determine whether this would change our understanding of severe pneumonia aetiology in this setting. However, the utility of this test may be better in settings such as paediatric infection and general practice management of respiratory infections. In those settings, there is scope for better antimicrobial stewardship by avoiding antibiotics when a virus is identified, or tailoring antibiotics for a detected bacterium.

Limitations include that since the study sought to document clinical practice rather than enrol patients for additional testing, the algorithm for RT-PCR testing and other testing such as convalescent serology, was not enforced. We collected results from the hospital database only, hence testing done at other providers was not captured. We did not collect data on impact of the RT-PCR on clinical decision making, and also did not follow patient outcomes after discharge form the ICU, since the focus of the study was on CAP aetiology rather than outcome.

Conclusion

In this 12-month prospective audit, the microbiological causes identified in cases of severe CAP were very consistent with previous studies from this setting. The high rate of organism detection in this most unwell patient subgroup meant that few remained eligible for testing according to the stringent algorithm, and clinician uptake was low. Implementation of a new test requires careful consideration about strategies for targeted use and clinician education. Current local guidelines on empirical CAP antimicrobial therapy are appropriate for the range of pathogens which predominate in this tropical environment.

Conflicts of interest and sources of funding

The authors have no conflicts. This study did not receive funding.

Acknowledgements

We thank the ICU nursing staff at RDH for facilitating data collection for the study. APR is supported by a National Health and Medical Research Council fellowship (1113638).

Availability of data and materials

Data are available on request from the lead author via the editor.

References


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Dengue mosquito detection at HMAS Coonawarra, Darwin
Nina Kurucz and William Pettit, Medical Entomology, Centre for Disease Control, Darwin

Abstract

On 17 April 2018 dengue mosquito larvae were detected in a routine sentinel tyre trap set by the Commonwealth Department of Agriculture and Water Resources at the Royal Australian Naval Base HMAS Coonawarra in Darwin. In response to the detection all receptacles at the Naval Base facility and adjacent Larrakeyah Defence Precinct military residential areas were treated with residual insecticide and adult mosquito control (fogging) and residual barrier spraying was carried out. Enhanced exotic mosquito surveillance was established to monitor for exotic mosquitoes. There have been no further detections of Aedes aegypti at HMAS Coonawarra.

Key words: Exotic mosquitoes; mosquito surveillance; NT ports.

Background

The dengue mosquito, Aedes aegypti is a vector for dengue, chikungunya, Zika and yellow fever.1-6 While this mosquito is not present in the Northern Territory (NT), it occurs in northern Queensland and neighbouring South East Asian countries, posing a serious threat to the NT. Since 2000/01, A.e. aegypti has been detected on 114 occasions on illegal fishing vessels and at NT First Points of Entry, including sea and airports.7,8 Three separate incursions with established populations have also occurred in the NT since 2004, with elimination of the dengue mosquito achieved through dedicated programs.9-12

To allow early detection and control of exotic mosquitoes at First Points of Entry in the NT, the Medical Entomology (ME) unit of the NT Department of Health (DoH) has a close working relationship with the Commonwealth Department of Agriculture and Water Resources (DAWR) as well as air and sea port stakeholders, with routine surveillance traps set by DAWR and identification of all adult mosquitoes carried out by ME. In the event of a positive trap, an immediate response occurs following the recently established Australian Government Department of Health’s ‘Response Guide for Exotic Mosquito Detections at Australian First Points of Entry.’13

In April 2018, A.e. aegypti has for the first time been detected in a DAWR routine surveillance trap at the HMAS Coonawarra Naval Base in Darwin, NT. This article outlines the response to the detection.

Detection and identification

On 17 April 2018 a routine sentinel tyre trap located at the HMAS Coonawarra Naval Base in Darwin was serviced by DAWR, with larvae collected from the tyre (Figure 1). As a precautionary measure, while samples were being processed, 2 sticky traps were deployed in the vicinity of the sentinel tyre trap by DAWR on the same day. To enable accurate identification, the larvae were reared to bigger life stages and on 24 April 2018 were identified by DAWR as larvae of the dengue mosquito, A.e. aegypti. They were delivered to the ME laboratory for confirmation on the same day and were confirmed as A.e. aegypti, with the sample containing 6 x 4th instar and 1 x 3rd instar larvae, 1 x 4th instar larval skin and 1 x pupae.

Response to exotic mosquito detection

Adult mosquito control

Following the positive identification of A.e. aegypti an immediate response occurred in the afternoon of the same day, in line with the ‘Response Guide for Exotic Mosquito Detections at Australian First Points of Entry.’ Under the guidelines, the land owner has the responsibility for exotic mosquito control activities as advised by the State or Territory Health Departments. As the detection occurred on Defence land, a Defence Contractor, BroadSpectrum carried out adult mosquito control (fogging), with Aqua K’Othrine® (active constituent: deltamethrin) applied with a ULV thermal fogger, as advised by ME (Figure 2).

The contractor also commenced barrier spraying to buildings and vegetation in the area using Biflex Ultra® (active constituent: bifenthrin)
applied with a backpack mister (Figure 2). Barrier spraying was continued and finalised on 1 May 2018, including the vegetation at the base of the escarpment leading to the adjacent Larrakeyah Defence Precinct military base residential area (Figure 2).

**Larval survey and receptacle treatment**

On 24 April, ME in liaison with DAWR commenced a larval survey and receptacle treatment at the Coonawarra wharf below the escarpment, which was continued on 26 April including outer areas of Coonawarra and the residential areas of the adjacent Larrakeyah Defence Precinct military base on top of the escarpment (Figure 2). During the survey only 2 receptacles with water, but no mosquito breeding, were found close to the wharf, while a number of receptacles breeding mosquitoes were located in the residential area. All receptacles
were treated with Temprid® (active constituent: beta-cyfluthrin and imidacloprid) and methoprene pellets. No exotic mosquitoes were detected.

**Enhanced surveillance**

Following the guidelines, the routine sentinel tyre was removed on the day of detection and enhanced surveillance commenced on 24 April 2018, with 2 Biogents® (BG) sentinel traps and the previously established sticky traps set by DAWR and an additional 2 BG traps set by BroadSpectrum (Figure 1). All 6 traps were serviced daily by DAWR for 10 days, then every second day for 1 week and then weekly for another 2 weeks. No exotic mosquitoes were collected in the enhanced surveillance traps.

**Discussion**

This was the first detection of *Ae. aegypti* at a military establishment in the NT. The good sanitation around the Coonawarra wharf area needs to be acknowledged, rendering the area extremely hostile to mosquito breeding, with only 2 receptacles holding water detected during the larval survey. Good sanitation at First Points of Entry is extremely important to prevent the establishment of exotic mosquitoes in an area. This detection once again showed the importance of established surveillance programs at First Points of Entry enabling detection and response to exotic mosquito incursions in a timely manner. The response was carried out following the Response Guide for Exotic Mosquito Detections at Australian First Points of Entry and every stakeholder involved in this response should be commended for their cooperation and timely response actions, which ensured that the NT remains free of the dengue vector.

**Acknowledgements**

We would like to acknowledge and thank all ME, DAWR, Australian Defence Force and BroadSpectrum staff involved in the exotic mosquito surveillance and control response at Coonawarra.
Update on meningococcal disease in the Northern Territory (NT)

In the 5 years, 2012 to 2016, there were 12 cases of invasive meningococcal disease (IMD) notified in residents of the NT with 11 cases caused by serotype B (MenB), 1 by serotype C (MenC) and none by serotype W (MenW) or Y (MenY).

In 2017 alone there were 32 cases of IMD notified in NT residents with serotypes as follows; 3 MenB, 26 MenW, 3 MenY and no MenC cases. The increased number of MenW was due solely to an outbreak of MenW in Alice Springs, Barkly and Katherine regions with 94% of cases under 15 years of age. This outbreak prompted an intense public health response to cases, their contacts and communities and lead to a change in the NT Childhood Immunisation Schedule in December 2017, when MenC vaccine at 12 months of age was replaced with Men ACWY vaccine for all NT children. A stepwise roll-out of MenACWY vaccine programs started in October 2017 for 1 to 19 years olds in the outbreak regions (Alice Springs, Barkly and Katherine) and in January 2018 extended to 1 to 19 year olds in Rural Darwin and East Arnhem regions and to Darwin boarding schools, residential care and correctional facilities.

In the first 6 months of 2018, January to June, there have been 4 cases of IMD including 1 case of MenB, 2 cases of MenW and 1 case of ungroupable meningococcus. MenW cases have markedly reduced but there is no room for complacency and while the coverage in the 1 to 19 year olds in targeted areas is good, at 75% for Aboriginal children and 62% for non-Aboriginal children overall, some age groups and regions have lower coverage than others. Particularly 15 to 19 year olds, an age group at increased risk of disease and who are felt to have high nasopharyngeal carriage rates of the organism, have the lowest coverage (65% in Aboriginal 15-19 years old compared to 85% in Aboriginal 1-4 year olds and 44% in non-Aboriginal 15-19 year olds compared to 88% in non-Aboriginal 1-4 year olds). The 15 to 19 age group needs to be opportunistically encouraged to receive their MenACWY vaccine.

Now is the time to make sure people are protected.

Below is the schedule for targeted free MenACWY vaccine on the NT immunisation schedule. The vaccine can only prevent disease if requested from/offered by a vaccine provider and given.

Current NT funded meningococcal ACWY vaccines on the NT immunisation schedule:

- All 12 months olds in the NT as per the NT immunisation schedule
- All 1 to 19 years olds in Alice Springs, Barkly and Katherine, Rural Darwin and East Arnhem regions including Darwin boarding schools, residential care and correctional facilities.

Other meningococcal vaccines recommended but not funded on the NT immunisation schedule:

Meningococcal ACWY and B vaccines are recommended for anyone aged 2 months and over who wants to protect themselves from meningococcal ACWY and meningococcal B disease.

The higher risk groups for disease include:

- children aged 2 months to 2 years of age
- Aboriginal people aged 2 months to 19 years
- adolescents aged 15-19 years and
- young adults aged 20-24 years who live in close quarters (such as new military recruits and students living in residential accommodation) or who are current smokers.

Children under 12 months will require additional doses of vaccine and those not eligible for vaccine on the NT immunisation schedule will need to obtain a prescriptions for both meningococcal ACWY and meningococcal B vaccine from their general practitioner.

People with medical conditions associated with an increased risk of IMD such as complement disorders, asplenia and other immunocompromising conditions are recommended to receive both meningococcal ACWY and meningococcal B vaccines and require additional doses of these vaccines.
For more information


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Contacts of person with meningococcal disease - should I be concerned?

If you have any of these symptoms, go to your health clinic or doctor immediately

- Fever
- Headache or neck stiffness
- Drowsiness or confusion
- Joint pain
- Rash of purple spots
- Dislike of bright lights
- Vomiting

YOUNG BABIES may have a high pitched cry, skin rash, dislike being carried, hard to wake from sleep or refuse to eat or drink.

What is meningococcal disease?
Meningococcal disease is an uncommon but severe infection that occurs when the meningococcal bacteria (germ) ‘invades’ the body from the throat or nose.

Someone I know has been in contact with a person with meningococcal disease - should I be concerned?

Meningococcal is a very serious disease, but meningococcal is also very difficult to catch.

People do not catch the meningococcal germ through casual contact or by just breathing air or by touching a surface where someone with meningococcal has been.

The germ is spread by respiratory droplets from close person to person contact. The Centre for Disease Control will identify ‘high risk’ contacts of confirmed cases. These people are followed up and given antibiotic medication and vaccination as per the Australian Guidelines.

A ‘high risk’ contact is a person

- That has lived in the same house or dormitory as the case in the 7 days prior to onset of illness
- That engaged in intimate kissing or was a sexual partner in the 7 days prior to onset of illness
- Any child or staff at child care, kindergarten or preschool where the case spent 2 full days (6-8 hours per day) or a total of 20 cumulative hours in the 7 days prior to onset of illness
- That sat directly next to a case in a vehicle (bus, plane, train) for greater than 8 hours
- Healthcare worker who has had unprotected close exposure to large particle respiratory droplets of a case during airway management (Intubation, suction)

In circumstances where a person with meningococcal disease has attended primary or high school for 2 full days (6-8 hours per day) or a cumulative 20 hours in the 7 days before onset of illness, the Centre for Disease Control will work with the Education Department and may decide to offer vaccination to the immediate class including the teacher.

For further information about meningococcal disease

Further information contact the Centre for Disease Control
Darwin 8922 8044 Alice Springs 8951 7540 Katherine 8973 9049 Tennant Creek 8962 4259 Nhulunbuy 8987 0357
Meningococcal disease is an uncommon but severe infection that occurs when the meningococcal bacteria (germ) ‘invades’ the body from the throat or nose.

The meningococcal germ is spread by close or long term person to person contact. People do not catch the meningococcal germ through casual contact or by just breathing air where someone with meningococcal has been.

Symptoms of meningococcal disease include:

- Fever
- Headache
- Drowsiness or confusion
- Neck stiffness or joint pains
- Rash of purple spots
- Dislike of bright lights
- Vomiting

Young babies may have a high pitched cry, skin rash, dislike being carried, hard to wake from sleep or refuse to eat or drink.

**If you have any of these symptoms, go to your health clinic or doctor immediately**

Well people in the same household of someone with meningococcal disease will be treated with antibiotics (medicine) to kill the meningococcal germ. This will reduce further spread of the disease to others.

Health centre staff are offering meningococcal vaccination to people in your community to help prevent further disease.

For more information contact the Centre for Disease Control

- Alice Springs 8951 7540
- Darwin 8922 8044
- Katherine 8973 9049
- Nhulunbuy 8987 0357
- Tennant Creek 8962 4259

September 2017  Centre for Disease Control – Meningococcal disease
What is meningococcal disease?
Meningococcal disease is a rare but very serious bacterial infection caused by *Neisseria meningitidis* which is also known as the *meningococcus*. About 1 in every 10 people carry this germ in the nose or throat. Although most carriers remain well, they are able to spread it to others, who, if infected may become very unwell.

There are 6 different groups of the meningococcal bacteria that cause nearly all disease globally (A, B, C, W, X and Y). The most common in Australia and the Northern Territory has been group B but since 2014 groups W and Y have been increasing. Group C disease is now rarely seen because children are vaccinated against group C at the age of 12 months. In the Northern Territory over the last 5 years there have been 2-4 cases per year of meningococcal disease.

Meningococcal disease occurs in 2 main forms:
- meningococcal septicaemia or ‘blood poisoning’
- meningococcal meningitis.

Sometimes both septicaemia and meningitis can occur at the same time.

Meningococcal disease can develop very quickly and cause death in around 5-10% of those affected. However, if diagnosed early and treated with antibiotics promptly most people will make a full recovery.

Meningococcal septicaemia
Meningococcal septicaemia develops when the germ gets into the bloodstream and causes ‘blood poisoning’.

Symptoms of meningococcal septicaemia may include:
- fever
- rash, this may start anywhere on the body as tiny red or purple spots which can spread and enlarge to look like fresh bruises. The rash does not fade when pressure is applied to it
- joint or muscle pains.

The rash must be taken seriously as the person requires urgent medical attention.

Meningococcal meningitis
Meningococcal meningitis occurs when the germ infects the outer lining around the brain and spinal cord.

Symptoms of meningococcal meningitis include:
- fever
- stiff neck
- headache
- dislike of bright lights
- vomiting and/or diarrhoea
- rash of tiny red or purple spots or larger bruises
- joint or muscle pains
- drowsiness, confusion or even coma.

The symptoms of meningococcal meningitis in young babies may be more subtle.

They can include:
- disinterest in feeding
- vomiting and/or diarrhoea
- a high pitchedmoaning cry
- irritability and a dislike of being handled
- a blank staring expression
- turning away from light
- extreme tiredness or floppiness
- rash or a pale blotchy complexion
- convulsions or twitching.

How easy is it to catch meningococcal disease?
Although the germ is spread in droplets from the nose or throat it is fortunately not easy to catch the disease.
The bacteria do not survive for long outside the body. Close and prolonged contact with a carrier is usually required for the germ to spread to other people. The bacteria cannot be picked up from surfaces, water supplies or animals and are not easily spread by sharing drink bottles, food or cigarettes.

Meningococcal disease can affect all ages, but babies and young children under 5 years of age and young adults (15-24 years of age) are most at risk. People of any age regularly exposed to tobacco smoking are also at increased risk.

How can meningococcal disease be prevented?
Meningococcal disease can be prevented by vaccination. The vaccines available provide protection against:

- meningococcal C
- meningococcal ACWY
- meningococcal B.

Meningococcal C vaccine is given routinely to 12 month-olds in a combination vaccine which also provides protection against another bacteria *Haemophilus influenzae B*.

Meningococcal ACWY vaccine is recommended for:

- travelers to countries such as Africa and Asia and pilgrims to the Hajj
- people with high risk medical conditions.

As disease caused by group W and Y is on the increase ACWY vaccine is being considered for additional age groups known to be most at risk of transmitting the meningococcal bacteria.

Meningococcal B vaccine is available for use in individuals over 2 months of age. This is recommended for:

- children aged 2 months to 2 years
- adolescents aged 15-19 years
- people with high risk medical conditions.

ACWY and group B meningococcal vaccines can be purchased privately with a prescription from your doctor. Please see The Australian Immunisation Handbook online version for dose information


What happens when a case occurs?
There is a small but real risk for very close contacts of the person with meningococcal disease to also develop disease. Sometimes cases of meningococcal disease can also occur in clusters of people when bacteria spread from a carrier to more than 1 person.

Treatment of a carrier of meningococcal bacteria with antibiotics has been shown to stop further spread. However, because there is no quick and accurate test to identify carriers, all of the ‘household contacts’ of a case are considered as potential carriers and recommended to have antibiotic treatment. The purpose of the antibiotic is to eliminate the germ from the nose or throat of the carrier in an effort to prevent further spread to others.

Vaccination may also be offered to contacts.

Contacts must be told to be alert for the symptoms of the disease even if they have taken the antibiotic. Contacts of an infected person should share the information about meningococcal disease with their close contacts to raise awareness about signs and symptoms of meningococcal disease. Early presentation of possible cases to medical care is important. The treating doctor should be made aware if the person presenting is a possible meningococcal contact.

For more information contact the Centre for Disease Control in your region

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<th>Contact Number</th>
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<td>8987 0357</td>
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<tr>
<td>Tennant Creek</td>
<td>8962 4259</td>
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</table>

Abstracts from peer reviewed published articles related to the Northern Territory

**Rheumatic heart disease in Timor-Leste school students: an echocardiography-based prevalence study**


*MJA, 208(7),16 April 2017, 303-307*

**Objectives:** To determine the prevalence of rheumatic heart disease (RHD) in school-aged children and young people in Timor-Leste.

**Design:** Prospective cross-sectional survey. Echocardiography was performed by Australian cardiologists to determine the presence of RHD. Demographic data were also collected. Patients in whom RHD was detected were entered into a register to allow monitoring of adherence to secondary prophylaxis; the first dose of benzathine penicillin G (BPG) was administered on the day of screening.

**Setting:** Schools in urban (Dili) and rural (Ermera) Timor-Leste.

**Participants:** School students aged 5-20 years.

**Outcome measures:** Definite and borderline RHD, as defined by World Heart Federation echocardiographic criteria.

**Results:** 1365 participants were screened; their median age was 11 years (IQR, 9-14 years), and 53% were girls. The estimated prevalence of definite RHD was 18.3 cases per 1000 population (95% CI, 12.3-27.0 per 1000), and of definite or borderline RHD 35.2 per 1000 (95% CI, 26.5-46.4 per 1000). Definite (adjusted odds ratio [aOR], 3.5; 95% CI, 1.3-9.4) and definite or borderline RHD (aOR, 2.7; 95% CI, 1.4-5.2) were more prevalent among girls than boys. Eleven children (0.8%) had congenital heart disease. Of the 25 children in whom definite RHD was identified, 21 (84%) received education and a first dose of BPG on the day of screening; all 25 have since received education about primary care for RHD and have commenced penicillin prophylaxis.

**Conclusions:** The rates of RHD in Timor-Leste are among the highest in the world, and prevalence is higher among girls than boys. Community engagement is essential for ensuring follow-up and the effective delivery of secondary prophylaxis.

**Rheumatic heart disease prophylaxis in older patients: A register-based audit of adherence to Guidelines**

*Holland J, Hardie K, de Dassel J, Ralph A*


**Background:** Prevention of rheumatic heart disease (RHD) remains challenging in high-burden settings globally. After acute rheumatic fever (ARF), secondary antibiotic prophylaxis is required to prevent RHD. International guidelines on recommended durations of secondary prophylaxis differ, with scope for clinician discretion. Because ARF risk decreases with age, ongoing prophylaxis is generally considered unnecessary beyond approximately the third decade. Concordance with guidelines on timely cessation of prophylaxis is unknown.

**Methods:** We undertook a register-based audit to determine the appropriateness of antibiotic prophylaxis among clients aged ≥35 years in Australia's Northern Territory. Data on demographics, ARF episode(s), RHD severity, prophylaxis type, and relevant clinical notes were extracted. The determination of guideline concordance was based on whether (1) national guidelines were followed; (2) a reason for departure from guidelines was documented; (3) lifelong continuation was considered appropriate in all cases of severe RHD.

**Results:** We identified 343 clients aged ≥35 years prescribed secondary prophylaxis. Guideline concordance was 39% according to national guidelines, 68% when documented reasons for departures from guidelines were included and 82% if patients with severe RHD were deemed to need lifelong prophylaxis. Shorter times since last echocardiogram or cardiologist review were associated with greater
The likelihood of guideline concordance ($P < 0.001$). The median time since last ARF was 5.9 years in the guideline-concordant group and 24.0 years in the nonconcordant group ($P < .001$). Thirty-two people had an ARF episode after age 40 years.

**Conclusions:** In this setting, appropriate discontinuation of RHD prophylaxis could be improved through timely specialist review to reduce unnecessary burden on clients and health systems.

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**Scabies and impetigo in Timor-Leste: A school screening study in two districts**

**Korte L, Bowen A, Draper A, Davis K, Steel A, Teodora I, Mascarenhas I, Dingle B, Francis J**

*PLOS Neglected Tropical Diseases* 12(5): e0006400. https://doi.org/10.1371/journal.pntd.0006400

**Introduction:** Scabies and impetigo are common and important skin conditions which are often neglected in developing countries. Limited data have been published on the prevalence of scabies and impetigo in Timor-Leste. Sequelae including cellulitis, bacteraemia, nephritis, acute rheumatic fever and rheumatic heart disease contribute significantly to the burden of disease.

**Methods:** School students were recruited from schools in Dili (urban) and Ermera (rural) in Timor-Leste for an epidemiological study in October 2016. A standard questionnaire was used to record demographics, anthropometry and skin examination results. Impetigo and scabies were diagnosed based on clinical examination of exposed surfaces, and clinical photographs were reviewed for correlation by an infectious diseases paediatrician. Prevalence of scabies and impetigo were calculated and binary risk factor associations were described using relative risks and 95% confidence intervals. Adjusted odds ratios were calculated using logistic regression multivariate analysis. Continuous variables were analysed for associations using the Mann-Whitney Rank Sum test.

**Results:** The study enrolled 1396 students; median age 11 years (interquartile range (IQR) 9-15). The prevalence of scabies was 22.4% (95% CI 20.2–24.7%) and active impetigo 9.7% (95% CI 8.3–11.4%); 68.2% of students had evidence of either active or healed impetigo. Students in Ermera were more likely than those in Dili to have scabies (prevalence 32.0% vs 5.2%, aOR 8.1 (95% CI 5.2–12.4, $p<0.01$). There was no difference in the prevalence of active impetigo between urban and rural sites. More than a third of participants were moderately or severely underweight. Stunting was markedly more common in the rural district of Ermera.

**Conclusion:** Scabies and impetigo are common in Timor-Leste, with very high prevalence of scabies in the rural district of Ermera. Improvements in prevention and treatment are needed, with prioritised activities in the rural areas where prevalence is highest.

**Priorities for preventing a concentrated HIV epidemic among Aboriginal and Torres Strait Islander Australians**

**Ward J, Hawke K, Guy R**


A summary of this article is provided here by Dr Tasnim Hasan, Infectious Diseases Registrar, CDC

The rate of new human immunodeficiency virus (HIV) infection diagnosis in Aboriginal and Torres Strait Islander (ATSI) people has increased by 33% from 2012 to 2016, compared to a decrease of 22% in rate for the non-Indigenous population, in the same time period. This poses a considerable public health concern, and the risk of an epidemic in the ATSI population. This can threaten Australia’s goal of eliminating transmission of HIV by 2020.

The reason for this increase may be attributable to poorer health outcomes and education, as well as poorer uptake of HIV interventions aimed at prevention of HIV, such as pre-exposure prophylaxis (PrEP). Diagnosis of HIV in the Indigenous population also remains sub-optimal. The epidemiological risk factors also differ in the Indigenous populations, with greater rates of HIV in heterosexual contacts. Due to this, Australia is at risk of failing to achieve the UNAIDS global 90-90-90 target, by 2020, where 90% of people with HIV know their status, 90% are on treatment and 90% have an undetectable viral load.
This necessitates effective intervention strategies to improve diagnosis of HIV and prevent spread of infection in the Indigenous population. This includes an overall reduction in the gaps in health and social outcomes in the Indigenous population and also implementing HIV prevention strategies which are targeted at Indigenous populations and those at risk.

**An outbreak of Salmonella Muenchen after consuming sea turtle, Northern Territory, Australia, 2017.**

Draper A, James C, Pascall J, Shield K, Langrell J, Hogg A

CDI, 41(4) 2017, E290-294

An outbreak of *Salmonella* Muenchen gastroenteritis occurred in a remote coastal Aboriginal community in the Northern Territory of Australia. There were 22 people sick (attack rate 55%); 7 had laboratory confirmed *S.* Muenchen infection; 2 required medical evacuation and admission to the intensive care unit. We conducted a descriptive case series to investigate the outbreak. All cases ate meat from a single green turtle (*Chelonia mydas*). The animal’s pre-death stress, improper butchering, insufficient cooking and the unsatisfactory storage of meat all likely contributed to the outbreak. Turtle meat requires safe preparation which includes thorough cooking and appropriate storage to avoid *Salmonella* infection.

**Firework-related injury in the Top End: a 16-year review**

Read D, Bradbury R, Yeboah E

*ANZ J Surg* 87 (2017) 1030–1034

**Background:** On July 1st on ‘Territory Day’, the public in the Northern Territory (NT) are permitted to purchase and operate consumer fireworks without a licence. Serious permanent injuries from fireworks are well described, leading to their banning in many other jurisdictions. This study describes those seriously injured by fireworks in the Top End of the NT, with the aim of identifying opportunities for prevention and harm minimization.

**Methods:** This is a retrospective audit of all admitted patients with an injury from fireworks at the Royal Darwin Hospital between 2000 and 2015. The variables collected included demographic data and the circumstances around injury (operator versus bystander, alcohol involvement and day of device operation). The consequences such as injuries, operating theatre visits, length of stay and outpatient visits are described.

**Results:** Fifty-five patients (including 17 children) suffered 67 injuries over the study period, resulting in 68 operating theatre visits, 322 hospital days and 380 outpatient appointments. Burns, hand and eye injuries predominate. Females (P= 0.000) and children (P= 0.029) were more likely to be injured as bystanders. Injuries on a day other than Territory Day were more likely to have alcohol involvement (P= 0.01), and occur in the operator (P= 0.017).

**Conclusion:** Consumer firework usage results in a small number of life altering injuries annually. Previous prevention campaigns focusing on device user safety should be expanded to include the safety of bystanders and children and reduce firework usage outside of the Territory Day.

**Murray Valley Encephalitis Virus: An Ongoing Cause of Encephalitis in Australia’s North**


*Trop Med Infect Dis 2018, 3, 49; doi:10.3390/tropicalmed3020049*

Murray Valley encephalitis virus (MVEV) is a mosquito-borne virus endemic to Australia and New Guinea. Encephalitis due to MVEV is potentially devastating, and no therapeutic interventions of proven value exist. Prevention relies largely on personal protective measures against mosquito bites. We present a case of MVEV encephalitis with a favourable outcome following intensive care management and prolonged rehabilitation, and the epidemiological features of a further 21 cases notified to the health department of Australia’s Northern Territory. As cases occur in travellers, and epidemics occur sporadically in south-eastern Australia, clinicians across Australia and further abroad should be familiar with the disease and its diagnosis and management.
## NT NOTIFICATIONS OF DISEASES BY ONSET DATE & DISTRICTS

### January — March 2017 and 2018

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<td>3</td>
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<td>8</td>
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<td>16</td>
<td>2 8</td>
<td>8 7</td>
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<td>Ross River Virus</td>
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<td>8</td>
<td>3 2</td>
<td>28</td>
<td>54</td>
<td>1 8</td>
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<td>Rotavirus</td>
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<td>7</td>
<td>0 1</td>
<td>13</td>
<td>8</td>
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<td>1 6</td>
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</tr>
<tr>
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<td>16</td>
<td>7 2</td>
<td>22</td>
<td>21</td>
<td>8 9</td>
<td>34 6</td>
</tr>
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<td>1</td>
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<td>0</td>
</tr>
<tr>
<td>Syphils &lt; 2 years duration</td>
<td>18</td>
<td>7</td>
<td>1 1</td>
<td>23</td>
<td>38</td>
<td>15 5</td>
<td>13</td>
</tr>
<tr>
<td>Syphilis &gt; 2 years duration or unknown</td>
<td>6</td>
<td>6</td>
<td>2 0</td>
<td>17</td>
<td>10</td>
<td>3 0</td>
<td>2</td>
</tr>
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<tr>
<td>Trichomoniasis</td>
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<td>278</td>
<td>65</td>
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</tr>
<tr>
<td>Tuberculosis</td>
<td>2</td>
<td>1</td>
<td>0 1</td>
<td>1</td>
<td>1</td>
<td>5 0</td>
<td>0</td>
</tr>
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<td>Typhoid</td>
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<td>0 0</td>
<td>0</td>
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<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Varicella - unspecified</td>
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<td>0 0</td>
<td>1</td>
<td>0</td>
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<tr>
<td>Vibrio invasive</td>
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<td>0 0</td>
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</tr>
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<tr>
<td>Zoster</td>
<td>19</td>
<td>13</td>
<td>2 2</td>
<td>88</td>
<td>66</td>
<td>6 7</td>
<td>15</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>1,071</td>
<td>872</td>
<td>154</td>
<td>150</td>
<td>1,320</td>
<td>1,701</td>
<td>368</td>
</tr>
</tbody>
</table>

*The Northern Territory Disease Control Bulletin Vol 25, No. 2, June 2018*
Ratio of the number of notifications in the 1st quarter 2018 to the 5 year mean (2013-17): selected diseases

**DECREASE**
- Barnah Forest
- Acute Post Strept GN
- Dengue
- Ross River Virus
- Tuberculosis
- Chickenpox
- Influenza
- Pneumococcal disease
- Rotavirus
- Cryptosporidiosis
- Campylobacteriosis
- Pertussis
- Malaria
- Group A strep invasive
- Salmonellosis
- Rheumatic Fever
- Melioidosis
- Adv Vacc Reaction
- Zoster
- Meningococcal disease
- Shigellosis

**INCREASE**
- Mumps

Ratio of 1st quarter 2018 cases to the mean 1st quarter 2013-17

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Ratio of the number of notifications in the 1st quarter 2018 to the 5 year mean (2013-2017): sexually transmitted diseases

**DECREASE**
- Hepatitis B - unspecified
- Hepatitis C - unspecified
- HTLV1

**INCREASE**
- Chlamydia
- Gonococcal infection
- Trichomoniasis
- Syphilis > 2 years or unknown duration
- HIV
- Syphilis < 2 years duration

Ratio of 1st quarter 2018 cases to the mean 1st quarter 2013-17
Comments on notifications

Shigellosis

The shigellosis outbreak in remote areas is continuing with 140 cases of shigellosis being notified in the 1st quarter compared with the 5 year mean of 49 cases. Transmission is likely to be person-to-person and the outbreak response has involved ensuring cases are treated, giving hygiene information and encouraging health hardware maintenance.

Herpes zoster

There were 130 cases of zoster notified in the 1st quarter which is 1.7 times the expected of 77 based on the 5 year mean. Zoster notifications continue to rise and whether this is real or just a result of the increase in testing and reporting is hard to ascertain but will be the subject of further investigation in the future.

Dengue

The number of dengue cases significantly decreased in the 1st quarter with only 7 cases notified compared with the 5 year 1st quarter mean of 23. This is good news and may be due to better control measures being implemented in dengue endemic countries or better awareness of the risk by travellers. There is promise worldwide in new biological control methods and vaccines.

NT malaria notifications January—March 2018

Elizabeth Stephenson, CDC Darwin

There were 5 cases of malaria notified in the 1st quarter of 2018. The following Table provides details about where the infection was thought to be acquired, the reason exposed, the infecting agent, whether chemoprophylaxis was used and where the patient lived.

<table>
<thead>
<tr>
<th>No. of 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>Nigeria</td>
<td>Recreation</td>
<td><em>P. falciparum</em></td>
<td>No</td>
<td>Darwin</td>
</tr>
<tr>
<td>1</td>
<td>Uganda</td>
<td>Recreation</td>
<td><em>P. falciparum</em></td>
<td>No</td>
<td>Darwin</td>
</tr>
<tr>
<td>1</td>
<td>Democratic republic of Congo (DRC)</td>
<td>Refugee</td>
<td><em>P. falciparum</em></td>
<td>No</td>
<td>Darwin</td>
</tr>
<tr>
<td>1</td>
<td>South Africa</td>
<td>Recreation</td>
<td><em>P. falciparum</em></td>
<td>No</td>
<td>Darwin</td>
</tr>
<tr>
<td>1*</td>
<td>Papua New Guinea</td>
<td>Refugee</td>
<td><em>P. falciparum</em></td>
<td>No</td>
<td>Darwin</td>
</tr>
</tbody>
</table>

* Probable case. Antigen detection – weakly positive, microscopy negative. Treated as a clinical case in Royal Darwin Hospital.
## Immunisation coverage for children aged 12-<15 months at 31 March 2018

<table>
<thead>
<tr>
<th>SA3 Name</th>
<th>Number of individuals in SA3</th>
<th>%DTP</th>
<th>%Polio</th>
<th>%HIB</th>
<th>%HEP B</th>
<th>%Pneumo</th>
<th>% Fully vaccinated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Darwin City</td>
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<td>93.03</td>
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<td>93.03</td>
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<tr>
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<td>91.94</td>
<td>91.94</td>
<td>91.94</td>
<td>91.94</td>
<td>91.94</td>
</tr>
<tr>
<td>Palmerston</td>
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<td>93.92</td>
<td>93.92</td>
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<td>94.48</td>
<td>93.92</td>
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<td>97.47</td>
<td>97.47</td>
<td>96.2</td>
<td>96.2</td>
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<td>92.59</td>
<td>92.59</td>
<td>96.3</td>
<td>96.3</td>
<td>92.59</td>
</tr>
<tr>
<td>Daly - Tiwi - West Arnhem</td>
<td>25</td>
<td>92</td>
<td>92</td>
<td>92</td>
<td>92</td>
<td>96</td>
<td>92</td>
</tr>
<tr>
<td>East Arnhem</td>
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<td>95.65</td>
<td>95.65</td>
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<td>95.65</td>
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<td>97.98</td>
<td>93.94</td>
<td>93.94</td>
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<tr>
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<td>94.7</td>
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<td>93.90</td>
<td>95.50</td>
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## Immunisation coverage for children aged 24-<27 months at 31 March 2018

<table>
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<tr>
<th>SA3 Name</th>
<th>Number of individuals in SA3</th>
<th>%DTP</th>
<th>%Polio</th>
<th>%HIB</th>
<th>%HEP B</th>
<th>%MMR</th>
<th>%MenC</th>
<th>%Varicella</th>
<th>% Fully vaccinated</th>
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<tbody>
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<td>90.4</td>
<td>91.92</td>
<td>90.91</td>
<td>86.87</td>
</tr>
<tr>
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<td>92</td>
<td>96</td>
<td>96</td>
<td>90</td>
<td>94</td>
<td>90</td>
<td>86</td>
<td></td>
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<td>92.7</td>
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<td>96.81</td>
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<td>96.81</td>
<td>92.55</td>
<td>95.74</td>
<td>90.43</td>
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<tr>
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<td>97.22</td>
<td>97.22</td>
<td>97.22</td>
<td>97.22</td>
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<td>97.22</td>
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</tr>
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<td>92</td>
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<td>88</td>
<td>76</td>
<td>76</td>
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<td>93.81</td>
<td>91.92</td>
<td>90.03</td>
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<td>90.71</td>
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<td>95.50</td>
<td>96.30</td>
<td>93.30</td>
<td>95.40</td>
<td>91.70</td>
<td>89.80</td>
</tr>
</tbody>
</table>

* Not mapped: Individual could not be mapped to a specific location. For example a PO Box cannot be mapped to a geographical area
## Immunisation coverage for children aged 60–<63 months at 31 March 2018

<table>
<thead>
<tr>
<th>SA3 Name</th>
<th>Number of individuals in SA3</th>
<th>%DTP</th>
<th>%Polio</th>
<th>% Fully vaccinated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Darwin City</td>
<td>97</td>
<td>87.63</td>
<td>87.63</td>
<td>87.63</td>
</tr>
<tr>
<td>Darwin Suburbs</td>
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<td>93.3</td>
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</tr>
<tr>
<td>Litchfield</td>
<td>60</td>
<td>93.33</td>
<td>93.33</td>
<td>93.33</td>
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<tr>
<td>Palmerston</td>
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<td>90.85</td>
<td>90.24</td>
</tr>
<tr>
<td>Alice Springs</td>
<td>86</td>
<td>93.02</td>
<td>93.02</td>
<td>93.02</td>
</tr>
<tr>
<td>Barkly</td>
<td>25</td>
<td>96</td>
<td>96</td>
<td>96</td>
</tr>
<tr>
<td>Daly - Tiwi - West Arnhem</td>
<td>27</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>East Arnhem</td>
<td>35</td>
<td>91.43</td>
<td>91.43</td>
<td>91.43</td>
</tr>
<tr>
<td>Katherine</td>
<td>69</td>
<td>98.55</td>
<td>97.1</td>
<td>97.1</td>
</tr>
<tr>
<td>Not mapped*</td>
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<td>97.3</td>
<td>97.3</td>
<td>97.3</td>
</tr>
<tr>
<td>Non-Aboriginal (NT)</td>
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<td>91.43</td>
<td>91.25</td>
</tr>
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<td>94.60</td>
<td>94.60</td>
<td>94.50</td>
</tr>
</tbody>
</table>

* Not mapped: Individual could not be mapped to a specific location. For example a PO Box cannot be mapped to a geographical area

## Immunisation coverage at 31 March 2018

_Holly Carmichael, Centre for Disease Control, Darwin_

**Background information to interpret coverage**

Immunisation coverage will be reported by Australian Bureau of Statistics (ABS) Statistical Area Level 3 (SA3). SA3s are ABS standardised geographical areas to which children have been assigned based on their Medicare address as recorded on the Australian Immunisation Register (AIR). The region ‘Not mapped’ captures the children whose residency could not be mapped to a specific location within the Northern Territory (NT), this includes PO Box addresses. Maps of these geographic area boundaries can be found at [http://www.ausstats.abs.gov.au/ausstats/subscriber.nsf/0/B0AC271BC8160338CA257801000E0692/$File/1270055001_asgs_2011_nt_maps.pdf](http://www.ausstats.abs.gov.au/ausstats/subscriber.nsf/0/B0AC271BC8160338CA257801000E0692/$File/1270055001_asgs_2011_nt_maps.pdf)

The cohort of children assessed at 12 to <15 months of age on 31 December 2017 were born between 1 October 2016 and 31 December 2016 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 2017 were born between 1 October 2015 and 31 December 2015 inclusive. To be considered fully vaccinated, these children must have received meningococcal C vaccination (given at the 12 month schedule point), 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 2017 were...
born between 1 October 2012 and 31 December 2012 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 SA3 and Aboriginal status, as estimated by the AIR, are shown on page 35. Coverage for all Australian children is also provided.

Children in the NT were less likely to be fully immunised in all cohorts in comparison to Australia wide coverage rates: 12 to <15 months (NT 93.4%, National 93.8%), 24 to <27 months (NT 87.8%, National 89.8%) and 60 to <63 months (NT 92.9%, National 94.5%).

Aboriginal children were less likely to be fully immunised than non-Aboriginal children cohorts 12 to <15 months (Aboriginal 91.64%, non-Aboriginal 94.95%) and 24 to <27 months (Aboriginal 82.9%, non-Aboriginal 90.03%), however, more likely to be fully immunised in the 60 to <63 months cohort (Aboriginal 96.09%, non-Aboriginal, 91.25%).

Coverage by SA3 in the Table shows variation between high and low coverage areas. Darwin Suburbs had the lowest Aboriginal coverage in the 24 to <27 months cohort. The highest Aboriginal coverage area was in the Daly-Tiwi-West Arnhem region for the 24 to <27 months cohort and the Darwin City, Darwin Suburbs, Barkly and Daly-Tiwi-West Arnhem Region for the 60 to <63 month cohort. The lowest coverage area for non-Aboriginal children was in Darwin City for the 60 to <63 months cohort. The area that had the highest coverage for non-Aboriginal children was Katherine in the 60 to <63 months cohort.

Centre for Disease Control (CDC) are currently reviewing the reasons for the lower coverage in both Aboriginal and non-Aboriginal children. CDC is working with the AIR to review data quality and processing of vaccine recording, and reviewing other strategies to improve childhood immunisation coverage. Further information about the AIR coverage may be found at: http://ncirs.edu.au/immunisation/coverage/index.php

<table>
<thead>
<tr>
<th>Age group</th>
<th>Aboriginal</th>
<th>Non-Aboriginal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lowest SA3</td>
<td>Highest SA3</td>
</tr>
<tr>
<td>12&lt;15 months</td>
<td>Palmerston</td>
<td>Alice Springs</td>
</tr>
<tr>
<td></td>
<td>81.58%</td>
<td>97.5%</td>
</tr>
<tr>
<td>24&lt;27 months</td>
<td>Darwin Suburbs</td>
<td>Daly-Tiwi-West Arnhem</td>
</tr>
<tr>
<td></td>
<td>72.41%</td>
<td>100%</td>
</tr>
<tr>
<td>60&lt;63 months</td>
<td>Alice Springs, Darwin Suburbs, Barkly, Daly-Tiwi-West Arnhem</td>
<td>90%</td>
</tr>
</tbody>
</table>

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Disease Control staff updates April-June 2018

**Top End**

Lesley Scott retired from the Department after 33 years of dedicated service. Lesley, who has worked extensively across the Top End, started her career with the Northern Territory Government (NTG) in Mataranka as a Remote Area Nurse in 1985. From 1986 to 1992, she worked for the Centre for Disease Control (CDC) in Katherine as a Sexual Health nurse, a TB and Surveillance nurse and finally as the CDC Coordinator for Katherine. From 1992 to 1997, Lesley transferred to Timber Creek Community Health Centre as a Remote Area Nurse with a focus on maternal and child health but really dealt with a wide range of health issues and in an era with no air evacuations after last light. In these early years she went on many leprosy review trips to communities for follow-up and support of leprosy patients and to review their contacts. She has provided expertise in this area throughout her career.

Lesley has worked at CDC Darwin from 1998 to 2018, first in the TB clinic, then as a Project Research Officer for the Director updating policies and guidelines and as an educator and surveillance nurse. The role evolved over the years to be the CDC senior nurse position and nurse coordinator for public health responses.

In 2014 Lesley received the Excellence in Nursing/Midwifery Education, Research and Innovation Award at the Northern Territory (NT) Nursing and Midwifery Excellence Awards. Lesley has made a significant contribution to public health in the NT and will be greatly missed.

Dae Sharrock has retired after 28 years of service to the NTG and 25 years working at Royal Darwin Hospital Pathology as a microbiologist with extensive experience with mycobacterial organisms. She spent many years providing laboratory service to the leprosy services of the NT working with Dr John Hargrave.

Natasha Murray has commenced in the Community Paediatric Allied Health professional role. Natasha has worked as a dietician in various places across the NT and more recently in project management at the Heart Foundation. Toni Stokes commenced in the Clinical Nurse Manager Top End Remote Sexual Health role in May. Toni had been working as a Remote Area Nurse at Beswick and prior to that worked in Cox’s Bazaar in Bangladesh as a clinical midwife. Katelin Gallagher, Administration Officer at Medical Entomology, has resigned and is now working in administration with the Department of Education.

Helen Cleary started work at Gove CDC in the Healthy Skin Nurse position. Helen has previously worked in Gove District Hospital (GDH) in the wards and specialist clinics. Kate Ranford commenced in April at Gove CDC as the Remote Sexual Health Clinical Nurse Specialist (CNS). Kate had previously worked at GDH where she had worked in the operating theatres and also at the General Practice surgery in Gove. Emma Childs has transitioned from the Healthy Skin Nurse position to the Acting Gove CDC Manager role.

Tresa Joseph joined the Katherine CDC team as the TB CNS on a short-term contract. Tresa has transferred from the Katherine Hospital to fill the TB position vacated by Judy Creighton. Tarrant Tolotta has completed his contract with CDC in the Healthy Skin Nurse position and has returned to his position at Katherine Hospital.

**Central Australia**

Renae Williams started in CDC in April as a Trachoma CNS. Renee had previously worked in the Surgical Unit of Alice Springs Hospital. Nicola (Nikki) Eaton has also joined the trachoma team as a Project Officer. Nikki had previously worked as an Enrolment and Attendance Officer (Barkly) for the Department of Education. Elizabeth Delaney is backfilling the CNS Immunisation role for 6 months vacating her Trachoma CNS position.

Anirrudha (Ani) Goswami, Trachoma Administration and Data Officer, has resigned and moved to Melbourne where she is working as a Project Officer at the Western and Central Melbourne Integrated Cancer Service.