CDC
CENTRE FOR DISEASE CONTROL
NORTHERN TERRITORY

THE NORTHERN TERRITORY
DISEASE CONTROL
BULLETIN

Vol. 23, No. 1, March 2016 ISSN 1440-883X

Melioidosis awareness campaign ……..Don't get melioidosis….

Rowena Boyd,[1] Sarah McGuinness,[1] Anthony Draper,[1,2] Meredith Neilson[1] and Vicki Krause,[1]
[1]Centre for Disease Control, Darwin
[2]National Centre for Epidemiology and Population Health, Australian National University

Abstract

In 2015–16 the Centre for Disease Control in partnership with local health care providers, services and industries intensified efforts to increase melioidosis awareness and prevention messages. In particular we aimed to target high risk populations with 5 key messages which included advice to 1) wear shoes during the Wet Season, 2) wear gloves when working outside, 3) stay indoors during storms, 4) take it easy with alcohol and 5) wear a mask when using a high pressure hose. We updated, developed and distributed a new factsheet and poster resources aimed at people with low English literacy, ran announcements on local radio in local Indigenous languages, provided greater education for those sick with melioidosis and we became involved in a 'Shoe Art Project' which served the dual purpose of providing education and shoes to a vulnerable population. Sponsorship for this project was sourced from local businesses and organisations. We are now individually interviewing and educating melioidosis patients with the aim to reduce reinfection and increase community awareness of melioidosis. Raising and maintaining awareness about melioidosis in the population is a continuous process requiring innovation and the ability to reach those most at risk.

Key words: Melioidosis; awareness campaign; Northern Territory; Burkholderia pseudomallei.

Contents

Melioidosis awareness campaign. Don't get melioidosis... . 1
CDC fact sheet update January–March 2016 4
Melioidosis plain language fact sheet 5
Establishment of a paediatric injury surveillance system in the Northern Territory 7
What is new in immunisation March/April 2016..... 11
NT Pneumococcal Vaccination and Revaccination Guideline 14
Adult and Special Groups Vaccination Schedules..... 15
Childhood Vaccination Schedule 16
Enteric disease in the Northern Territory in 2015..... 17
Audit of the availability of condoms in remote communities of the Northern Territory 26
Northern Territory Congenital Syphilis Guidelines in 2015: a review of the recommendations against major clinical guidelines and the literature..... 30
End TB—World TB Day 24 March 36
Fish for dinner? Some fish is good for you, just not too much fish 37
NT malaria notifications October–December 2015 38
Campaign to increase awareness of Murray Valley encephalitis in remote communities across the Northern Territory, Australia 39
Abstracts from peer reviewed published articles related to the Northern Territory 40
NT notifications of disease by onset date and district..... 49
Graphs of selected diseases and STIs 50
Comments on notifications 51
Immunisation coverage..... 52
Disease control staff updates 54

Editor: Vicki Krause
Email: vicki.krause@nt.gov.au

Assistant Editors: Lesley Scott, Kristen Overton, Jiunn-Yih Su, Manoji Gunathilake, Matthew Thalanany, Peter Markey, Anthony Draper, Linda Garton, Heather Cook, Kate Hardie

Centre for Disease Control
PO Box 40596
Casuarina
Northern Territory 0811

Production Design: Meredith Neilson
Website:http://www.nt.gov.au/health/cdc/cdc.shtml

Introduction

Between 20 and 106 people are notified with melioidosis in the Northern Territory (NT) each year. Melioidosis is a disease caused by a bacterium that lives below the soil's surface in the tropical regions of northern Australia. The disease is more common during the Wet Season, when periods of rain disrupt the soil and can be found in soil, ground water and even in the air during periods of windy weather. The bacteria enter the body through cuts in skin, by inhalation or on rare occasions by ingestion. Clinical presentations and syndromes vary but most commonly include pneumonia, genitourinary infection, skin infection and abscesses in internal organs such as the liver, spleen and prostate.[1] While improved diagnosis and treatment in the NT has greatly reduced mortality from 30% in the 1990's to less than 10% currently, melioidosis is still recognised as a severe and preventable disease.[1]

Certain groups of people are at higher risk of melioidosis and its complications. Groups at increased risk for melioidosis include those with diabetes, hazardous alcohol users (including binge drinkers) and those with chronic lung disease and chronic renal disease.[1,2] In 2015, the NT Centre for Disease Control (CDC) identified a need to increase melioidosis awareness in the Top End with specific emphasis on targeting those most at risk. We embarked on a campaign to meet this objective.

Consultations prior to melioidosis awareness campaign

We formed a committee to assess what the key messages might be and to make a plan as to how to go about reaching those at risk with the appropriate messages and support. We commenced a series of consultations, starting with experts in the field of melioidosis including infectious disease physicians at Royal Darwin Hospital, CDC staff and the Menzies School of Health Research researchers. We then consulted stakeholders supporting populations at increased risk of melioidosis including health professionals caring for people with diabetes, renal disease and cancer. We also met with Larrakia Nation who support Indigenous homeless people and Danila Dilba who provide Indigenous health care services in the Darwin region.

Coming up with the messages

From these consultations we identified a need for public health messages to target people most at risk of disease and communicate health messages with appropriate language, minimal words and to utilise pictures. We looked at what was used in previous years and what needed to be improved. There was agreement that the previous melioidosis poster was too dark in colour with too many words and a new poster was required.

The initial aim was to have a wide-ranging campaign with a very recognisable slogan that could be used on posters and in other print material. A tune or jingle that could be readily identified with the message about melioidosis for radio, TV or other media was also thought to be desirable. With limited resources we started by asking the CDC staff to identify a catchy, action-provoking slogan similar to our measles "Get protected, not infected" slogan. Alas we got "Melioid; the bug that lives in the mud"; "Stay out of the bog when you've been on the grog"; "Melioidosis this way comes……."; "Dirt and rain? No shoes, no play"; "In the mud?..Boot the bug" and similar. Together with input from the media department we settled on a straight forward message that started as

"Are YOU at risk of melioidosis this Wet Season?"

to

"Will you get Melioidosis?"

to

"Protect yourself from melioidosis"
to finally

"Don't get melioidosis"

We prioritised 5 messages as follows;

1) wear shoes during the Wet Season, 2) wear gloves when working outside, 3) stay indoors during storms, 4) take it easy with alcohol and 5) wear a mask when using a high pressure hose.

Icons were produced to represent each of these 5 messages.

To augment CDC program funding additional sources were found by approaching local industries, organisations and retail centres.

Awareness campaign strategies implemented

As in previous years, CDC sent out media releases prior to and during the Wet Season which were taken up by print and radio media.

We updated our melioidosis poster simplifying language and graphically representing our main messages. Posters were again distributed to nurseries, hardware stores, workplaces with outdoor workers, airports, health centres, and public spaces where people with risk factors receive services.

In addition to revising our current melioidosis factsheet we produced a second factsheet in plain language aimed at people for whom English is not their first language. The poster and factsheets can be viewed at:

http://www.health.nt.gov.au/Centre_for_Disease_Control/Publications/CDC_Factsheets/index.aspx

Following translation of the melioidosis awareness and prevention messages into 5 Top End Indigenous languages we partnered with Yolgnu Radio, Larrakia Radio, Aboriginal Radio Vast, and TEABBA Radio who financially assisted the broadcast of these messages on local Top End radio between December 2015 and March 2016.

In looking for ways to reach the homeless population we contacted the Larrakia Nation who engages the homeless community in a weekly community arts workshop known as 'Arts in the Grass'. Partnering with Larrakia Nation and with generous sponsorship from Inpex, Big W, Target, and the Rotary Club of Darwin Sunrise we are running a 'Shoe Art Project.' Artists are encouraged to paint shoes at the weekly workshop and learn about melioidosis and ways to protect themselves from getting the disease. CDC staff members attend the workshops to deliver these messages and talk with the artists. The shoes are photographed (see Photos 1 and 2) when finished and then the artist keeps and wears their newly painted shoes. This collaborative project will finish at the end of the Wet Season and will continue next Wet Season.

Also starting this Wet Season, CDC implemented a new practice of having a CDC team member formally interviewing and providing education to all people newly diagnosed with melioidosis. These interviews help identify risk factors (e.g. undiagnosed diabetes), screen for problem drinking and if found provide education on strategies to reduce alcohol intake and also promote life style activities and strategies that will reduce reinfection with melioidosis in future.

Photos 1 and 2: Painted shoes from the 'Shoe Art Project'

Future aspirations

During the consultation process, a recurring theme identified was a need to provide a wider reaching campaign and to not just rely on people reading information regarding melioidosis and

its prevention. Ideally a television advertisement, similar to the well-known 'don't play in pipes and drains' advertisement was viewed as a high priority that possibly would provide information to a wider audience via a visual and audio media. A TV advertisement will require funds to develop and to air it each Wet Season.

Conclusion

Following recognition of the need for greater melioidosis awareness and prevention strategies, CDC and partners have improved and expanded strategies with this focus. Sponsorship from local organisations has been greatly appreciated in helping to implement this campaign and to contribute to improved health in our tropical climate. Raising and maintaining awareness about melioidosis in the population is a continuous process requiring innovation and the ability to reach those most at risk.

Acknowledgements

Without the contribution of many people, local industry and retail centres, this campaign would not have gone ahead. Thanks go to Joel Carter and the Media and Corporate Communications Unit, Department of Health; Menzies School of Health Research; the Infectious Diseases, Diabetes, Podiatry, Oncology and Renal Departments at Royal Darwin Hospital; and Peter Markey, Centre for Disease Control. Thanks also for the contributions of Larrakia Nation, Nightcliff Renal Unit and Danila Dilba. Our special appreciation goes to Inpex, Big W, Target, the Rotary Club of Darwin Sunrise, Yolgnu Radio, Larrakia Radio, Aboriginal Radio Vast, and TEABBA Radio for their financial support of this campaign.

References

1. Currie BJ, Ward L, Cheng AC. The epidemiology and clinical spectrum of melioidosis: 540 cases from the 20 year Darwin prospective study. *PLoS Negl Trop Dis* 2010; 4(11): e900.
2. Currie BJ, Jacups SP, Cheng AC, Fisher D, Anstey N, Huffam S, Krause V. Melioidosis epidemiology and risk factors from a prospective whole population study in northern Australia. *Trop Med Int Health* 2004; 9(11): 1167-74.

****

CDC fact sheet updates January-March 2016

The Centre for Disease Control (CDC) fact sheets and guidelines are updated on a regular basis and can be found on the CDC website at:

http://health.nt.gov.au/Centre_for_Disease_Control/Publications/CDC_Factsheets/index.aspx .

Updated fact sheets

- Group A streptococcal infection – invasive
- Hand, foot and mouth disease
- Melioidosis plain language
- Mosquito-borne diseases
- Pneumococcal disease

****

Save the date

Northern Territory Centre for Disease Control 2016 Conference

6-8 September, Darwin

Centre for Disease Control

December 2015

Melioidosis (plain language)

What is melioidosis?

Melioidosis is a disease caused by germs that live in the soil. After rain the germs come to the surface and are found in mud and water on the ground. When it's rainy and windy, the germ can get blown around in the air.

How would I get melioidosis?

The germs enter your body through cuts or scratches in your skin. You can also breathe in the germs if you go outside in heavy rain or wind.

Am I at risk of melioidosis?

You are more at risk if you have **diabetes**, **kidney disease**, **lung disease** or **cancer**, if you drink lots of **alcohol**, or take **medicines that weaken your immune system**. Healthy people can also get the disease if they work outside with mud and water and don't protect their hands and feet.

How do I know if I have melioidosis?

The most common symptoms include fever, cough with mucus and difficulty breathing. Other symptoms include skin sores that don't heal or, for men, pain or difficulty when trying to pass water. If you are sick with any of these symptoms, you should go to a health clinic and see a doctor.

What is the treatment?

Melioidosis is a serious disease that can kill. Treatment with antibiotics needs to be started in hospital and then continued when you go home for a total of at least 3 months.

Can melioidosis be prevented?

By avoiding contact with the germs you can reduce your risk. When it is rainy and windy, stay inside. If you go outside, wear shoes. If you are working with mud or soil protect your hands and wear gloves. If you are using a high pressure hose outside, wear a mask. Take it easy with alcohol - even one big drinking session can increase your risk.

CENTRE FOR DISEASE CONTROL

For more information contact the CDC in your region

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

or

www.nt.gov.au/health/cdc

Establishment of a paediatric injury surveillance system in the Northern Territory

Kate Tessmann and Keith Edwards, CDC, Darwin

Abstract

Injuries are the leading cause of death for Australian children and the cause of many hospitalisations and Emergency Department (ED) presentations. National data show that death due to injury is almost 3 times higher in the Aboriginal and Torres Strait Islander populations. Injury surveillance in the Northern Territory (NT) has been limited almost entirely to reporting of deaths and hospitalisations. While this information is valuable, extending the scope of injury surveillance to include data on less serious injuries presenting to the Royal Darwin Hospital (RDH) ED and their causes will provide valuable knowledge of the epidemiology of childhood injuries. Such knowledge is essential for the effective planning, implementing and evaluation of primary prevention. This project commenced data collection at the RDH ED 1 January 2016 and aims to enhance the knowledge of paediatric injury in the NT.

Key words: injury; surveillance; paediatrics; Northern Territory; prevention.

Background

Injuries are the leading cause of death among children aged 0-14 years in Australia.[1] Deaths that result from injuries represent only a small fraction of those injured, with many more people suffering injuries that lead to hospitalisation, Emergency Department (ED) or general practitioner treatment or treatment that does not involve formal medical care.[2] Injuries sustained during childhood can have profound and lifelong effects on health and development. Children who are permanently disabled from injury may experience lifelong pain, or suffer loss of motor or cognitive functioning. Injuries affect quality of life, impact future ability to work and impose a financial burden on many segments of society.[3]

National data show that Aboriginal and Torres Strait Islander (ATSI) children, compared with their non-Indigenous counterparts, are over-represented in relation to injury, with higher rates of hospitalisation and higher rates of mortality. In 2003–2007, deaths due to injury contributed 13% of all deaths in non-Indigenous children aged 0-17 years. The death rate for Indigenous children was almost 3 times the rate of non-Indigenous children with 37% of all deaths due to injury.[4]

In 1989, delegates to the first World Conference on Injury Prevention and Control resolved to cooperate on finding ways to prevent injury but recognised that there was a serious shortage of information on which to base plans for action.[5] Since this time, government reports have emphasised that injury is preventable, that primary prevention is the best means of injury control[6] and recommendations have been made to improve data collection from trauma treatment centres, especially hospital EDs, for the purpose of guiding prevention.[7]

In recent years, a number of Australian States and Territories have established injury data collection systems. These include comprehensive systems such as the one created by the Victorian Injury Surveillance Unit which has been used to collect, analyse and interpret Victorian data on injury deaths, hospital admissions and ED presentations for more than 20 years. Another comprehensive data collection system is used by the Queensland Injury Surveillance Unit which currently collects injury data from 17 hospitals across 4 sample regions in Queensland. Western Australia's Department of Health reports on Paediatric injury data collected from the Princess Margaret Hospital in Perth[8] and New South Wales have developed and used several surveillance systems including an ED based system.[9]

Surveillance is defined by the World Health Organization (WHO) as the ongoing systematic collection, analysis, and interpretation of health data essential to the planning, implementation, and evaluation of health practice, closely integrated with the timely dissemination of these data.[5] The final link of the surveillance chain is in the application of these data to prevention and control. Surveillance is the best way of monitoring trends, detecting emerging problems, identifying interventions and assessing the results of interventions.

While injury data from EDs is often routinely collected and reported on, including at Royal Darwin Hospital (RDH), the data is commonly

limited to information about the patient's demographics and the injury itself, for example: 12 year old male presents with a dislocated shoulder. The information that is not being collected but is essential for the understanding of injury epidemiology is causation data. Causation data refer to details of the external cause of the injury and is principally about the nature of the injury event (e.g. a fall or a collision in a motor vehicle) but can also include information such as the place where the child was injured, and what activity they were doing when injured. The collection and analysis of injury causation data enable the monitoring of trends and the detection of emerging problems by identifying the role that the person, a product or environmental factors have in the cause of the injury. Essentially, it is the causation data that provide the knowledge necessary to understand childhood injury and guide the development and evaluation of prevention strategies and measures.

RDH is the largest hospital in the Northern Territory (NT) servicing approximately 150,000 people and has significant geographic and demographic differences to hospitals in other Australian jurisdictions. The NT's population of approximately 212,000 has the highest percentage of ATSI people of all Australian States and Territories, making up almost a third of its population,[10] with current statistics indicating that 43.6% of NT ED presentations are patients identifying as ATSI.[11] Appreciating the uniqueness of the NT and understanding through national data on hospitalisation and mortality rates that Indigenous children are over represented and more likely to experience injury, increases the value in conducting quality injury surveillance in the NT.

The rationale for this project was to try to improve childhood injury surveillance in RDH ED by including cause of injury data. From 1 January 2016, a trial of improved paediatric injury surveillance began at the RDH ED.

Methods

The inclusion criteria for collecting data are all children aged 0–15 years, on their first presentation to the RDH ED for a particular injury. Patients treated at another health centre or hospital for their current injury and then presenting to the RDH ED will still qualify as first presentation and will be included. Data collected include demographic details of the patient, injury information and the previously absent injury causation details.

When a child presents to the RDH ED with an injury, the injury is routinely assessed by a doctor and an interview is conducted with the child and/or their guardian. During this interview, the doctor asks questions about how the injury occurred; when, where and what caused the injury and what the child was doing at the time they were injured. Information collected is recorded in the patient's case notes. For each presentation, the patient's presenting problem e.g. pain, affected limb, is electronically recorded by the triage nurse on arrival and the diagnosis e.g. elbow dislocation, is electronically recorded by the treating doctor on discharge. The diagnosis is selected from an 'illness' or 'injury' classification list, a modified version of the WHO's International Statistical Classification of Diseases and Related Health Problems codes (ICD codes).

For this project, a report is generated weekly which lists all patients aged 0–15 years presenting to the RDH ED in the week prior. The report includes demographic details of each patient, and details of their presentation including; date and time of presentation, the presenting problem, the diagnosis and whether the diagnosis was selected from the illness or injury list.

In order to determine if a presentation meets the inclusion criteria, all presentations assigned an injury code are further investigated. Presentations assigned an illness code but with a presenting problem that indicates a potential injury, e.g. pain to lower leg, are further investigated. Presentations assigned an illness code and a presenting problem not indicating injury, e.g. appendicitis, are not investigated further and are not included in the database.

Further investigation includes reading the child's case notes to confirm that the reason for their presentation is due to injury and not illness, and to ensure that this is the first presentation to RDH ED for this particular injury. Once deemed to meet the inclusion criteria, any data recorded in the case notes that correlate to the injury surveillance data set items (see Table 1) are added to the injury surveillance database.

Case notes may include the electronic triage comments recorded when a patient first presents to the triage nurse in ED, the electronic discharge summary or clinical letter provided to the patient

on discharge, or review of the handwritten notes relating to the ED presentation, recorded within the patients medical file.

Data set

The data set items align with national and international recommendations regarding the core information required for quality surveillance data, and include the WHO's core minimum data set for injury surveillance, plus data set items from additional modules.[5] This includes data items regarding the individual demographics, the circumstances of the injury event and the injury outcome.

In addition, many data set items correlate with the Australian National Data Standards for Injury Surveillance (NDS-IS). The NDS-IS was created by the Australian Institute of Health and Welfare's (AIHW) National Injury Surveillance Unit at Flinders University, and specifies a set of data items, definitions and classifications to enable the nationwide collection of uniform data about injury and the events leading to injury.[12]

The International Classification of External Causes of Injury (ICECI) Version 1.2 is a system of classifications to enable systematic description of how injuries occur.[13] ICECI components utilised in this project will assist in the coding of injury events and the classification of injury. ICECI modules include intent, mechanism, object, place, activity, personal countermeasures, environmental countermeasures and sport.

The completed list of data set items collected for the NT paediatric injury surveillance project is documented (alphabetically) in Table 1.

Ethics approval

Ethics Approval (ref: HREC-2015-2494) has been granted by the Human Research Ethics

Table 1.NT paediatric injury surveillance data set items

Data set item	Description of data set item
Activity when injured	Activity of occurrence at the time of the incident
Bodily location of injury	The bodily location of the injury chiefly responsible for occasioning the attendance of the injured individual to ED
Date (and day) of attendance	The date the individual attended RDH ED
Date (and day) of injury	Date of injury
Date of birth and age	Date of birth and age of the injured individual
Environmental countermeasures	Measures in the competitive or recreational environment that are designed to protect against injury, if appropriate
Establishment identifier	Identifier for the establishment in which the injured individual presents and where data is collected from
ICD 10 Code	Classification of injury sustained
Indigenous origin	Indigenous origin of the injured individual
Injury factor	Object/substance producing injury
Injury summary	Free text description of the incident
Intent	Role of human intent in the incident
Mechanism	Cause of the injury
Mode of separation/ disposition	Status at separation of person (discharge/transfer/death) and place to which person is released (where applicable)
Nature of Injury	Nature of injury sustained
Patient identifier	Patient's Hospital Reference Number (HRN) which is used to uniquely identify each case and avoid double counting
Personal countermeasures	Equipment used or worn by the participant to protect against injury, if appropriate
Place	Place of occurrence of the incident
Residence	Injured persons usual residence – suburb & locality
Severity	Triage category given based on the speed of which the patient requires medical attention, which can indicate the severity of injury
Sex	Sex of the injured individual
Sport	The type of sport or exercise activity in which the injured individual was engaged at the time of the injury
Time of attendance	The time the individual attended RDH ED
Time of injury	Time of injury
Triage comment	Description given by triaging nurse regarding injured individuals ED presentation

Committee of the NT Department of Health and Menzies School of Health Research.

Data analysis and reporting

Data analysis will provide information on injury epidemiology e.g. distribution of injuries according to sex, age, Indigenous status, mechanism of injury. Further analyses may include how one data item relates to another e.g. the percentage of drowning's/near drowning's (mechanism of injury) that occurred with the reported use of a pool fence (safety countermeasure).

Data analysis will be presented in a non-identifiable, aggregated format, with updates published in the *NT Disease Control Bulletin* and a final report published as an ePublication on the NT Department of Health online library catalogue at the end of the data collection period.

Summary

From the 1 January 2016, 12 months of data will be collected from the RDH ED for all patients aged 0-15 years on their first presentation to RDH ED for an injury. Successful injury prevention rests on a foundation of quality surveillance data,[14] and the meaningful and reliable classification of injury event information is an essential prerequisite.[15] All data collected correlates with national and international recommendations and includes the critical but previously absent injury causation data.

The aims of the project are to add to the knowledge of childhood injury in the NT through the regular reporting of analysed data. It is hoped that this knowledge will then be used to underpin injury prevention policies, stimulate research and to develop and evaluate prevention strategies and measures, including community awareness initiatives and education (e.g. KidsafeNT programs), legislative and regulatory changes and safety-related environmental, equipment and product design improvements.

Acknowledgements

We would like to thank Didier Palmer, Director of RDH ED; David Read, Director of Trauma and Burns, NCCTRC; and KidsafeNT for their ongoing support in the establishment of a paediatric injury surveillance system for the NT.

References

1. Australian Institute of Health and Welfare 2012. A picture of Australia's children 2012. Cat. no. PHE 167. Canberra. AIHW.
2. Injuries and violence: the facts. Geneva, World Health Organization, 2010.
3. Mercy JA, Sleet DA, Doll L 2006. Applying a developmental and ecological framework to injury and violence prevention. In: Liller K (ed.*) Injury prevention for children and adolescents: research, practice and advocacy*. Washington, DC: American Public Health Association, 1–14.
4. Australian Institute of Health and Welfare 2011. Aboriginal and Torres Strait Islander child safety. Cat. no. IHW 50. Canberra: AIHW.
5. Holder Y, Peden M, Krug E, Lund J, Gururja G, Kobusingye O (Eds). *Injury Surveillance guidelines*. Geneva, World Health organization, 2001.
6. AIHW and Commonwealth Department of Health and Family Services 1997. First report on the national health priority areas, full report. Cat. no. PHE 1. Canberra: AIHW.
7. Commonwealth Department of Health and Family Services and Australian Institute of Health and Welfare 1998. National Health Priority Areas Report : Injury Prevention and Control 1997. AIHW Cat. No. PHE 3. Canberra : DHFS and AIHW.
8. Mohamad-Isa I, Richards J, Skarin D. WA Childhood Injury Surveillance Bulletin: Annual Report 2014-2015. Perth WA: Kidsafe WA (AUS); 2015 Oct.
9. Mitchell R, Bambach M. Examination of narratives from emergency department presentations to identify road trauma, crash and injury risk factors for different age groups. *Health Information Management Journal* 2015; l 44 (1).
10. Estimates of Aboriginal and Torres Strait Islander Australians, June 2011, Australian Bureau of Statistics.
11. AIHW 2012. Australian hospital statistics 2011-12: emergency department care. Health services series. Cat. no. HSE 126. Canberra: AIHW.
12. National Injury Surveillance Data Dictionary (NISDD) and Guidelines for Use. AIHW National Injury Surveillance Unit, Research Centre for Injury Studies, Flinders University.
13. ICECI Coordination and Maintenance Group (2004). International Classification of External Cause of Injury (ICECI) version 1.2. Consumer Safety Institute, Amsterdam and AIHW National Injury Surveillance Unit, Adelaide.
14. Mitchell R, McClure R, Williamson A and McKenzie K. Implementing the national priorities for injury surveillance. *MJA* 2008; 188: 405-408.
15. Scott D, Harrison J, Purdie D, et al. The properties of the International Classification of the External Causes of Injury when used as an instrument for injury prevention research. *Inj Prev* 2006; 12: 253-257.

****

What is new in immunisation—March/April 2016

Ros Webby, Centre for Disease Control, Darwin

The new 2016 immunisation schedules are now available.

Changes in 2016 are:

- The introduction of the 18 month booster dose of diphtheria, tetanus and pertussis vaccine (DTPa) as part of the National Immunisation Program in March 2016.
- The introduction of quadrivalent influenza vaccines for eligible groups. Vaccine will be available to order from April 2016.
- BCG vaccine is currently not available.
- The Adult and Special Groups vaccination schedule has also been updated, information is now identified through eligibility groups rather than disease. Vaccination details remain unchanged for adults, only the brand of influenza vaccine has changed.

Diphtheria, tetanus and pertussis (DTPa) vaccine at 18 months

All children at 18 months require an additional dose of diphtheria, tetanus, pertussis vaccine from March 2016.

Why is there an additional dose of DTPa vaccine at 18 months?

To reduce pertussis infection in young children, the DTPa vaccine has been added to the National Immunisation Program for 18 month olds from March 2016. The reason for this change is that although the 3 dose primary series of DTPa vaccine given in infancy is 84% protective against severe pertussis disease,[1] the immunity wanes with time and therefore a booster is required at 18 months.[2]

Which brands of DTPa vaccine?

The initial brand of DTPa vaccine will be Infanrix®. Infanrix® contains only the diphtheria, tetanus, pertussis components and is different from Infanrix®IPV as it does not contain inactivated poliomyelitis vaccine. An alternative brand Tripacel® will be used later in the year. DTPa vaccine can be given at the same time as other vaccines at 18 months but should be given 6 months after the last DTPa containing vaccine (Infanrix®Hexa).

The common side effects associated with DTPa vaccine are:

- Localised pain, redness and swelling at injection site
- Low-grade temperature (fever)
- Irritability, crying, drowsiness or tiredness
- An injection-site nodule that resolves without treatment

Less common side effects include extensive limb swelling which is a recognised adverse event that occurs rarely following booster doses of DTPa vaccine. Such reactions commence within 48 hours of vaccination, last for 1–7 days and resolve completely without any long term problems.

Please report all adverse events following immunisation to the Centre for Disease Control on 89228044 and complete the adverse event form available on the NT Department of Health immunisation website at:

http://health.nt.gov.au/Centre_for_Disease_Control/Immunisation/Recording_and_Reporting_Forms/index.aspx.

Catch up

There is no catch up program. Because the 18 month booster dose was not routinely recommended between 2003 and 2015, there will be children who have only had 4 doses of DTPa containing vaccine by 4 years of age. As long as the 4th dose of DTPa-containing vaccine was administered after 3.5 years of age, they do not require any further doses until 13 years of age. There is no need to give additional DTPa vaccine to these children. Children aged ≥18 months to ≤3.5 years can have an extra dose of DTPa if they request it but there should be a minimum interval of 6 months between the 3rd and 4th dose and 4th and 5th dose. The 5th dose should be given at 4 years of age.

Resources

The Australian Government has produced a fact sheet and poster about the program please see:

http://immunise.health.gov.au/internet/immunise/publishing.nsf/Content/IT0204-cnt

and

http://immunise.health.gov.au/internet/immunise/publishing.nsf/Content/IT0203cnt

Pregnant women and diphtheria, tetanus, pertussis (DTPa) vaccine

The introduction of the 18 month dose aims to decrease pertussis infection in young children and reduce spread to infant siblings. Pertussis is a serious disease in young babies under 6 months of age.[3] Pertussis vaccine is now given from the 28th week of pregnancy to protect the mothers and their infants. The vaccine protects infants by passive transfer of antibodies from mother to infant and reduces the possibility of maternal exposure.[4] The best time to give the vaccine in pregnancy is between 28–32 weeks to allow placental transfer of antibody from mother to child.

Influenza vaccine 2016 available in early April

Influenza is a serious disease

In 2015, there were 84,201 laboratory confirmed notifications of influenza across Australia with 7% of cases admitted to the intensive care unit (ICU) and 86 influenza associated deaths.[5] Notifications of influenza associated deaths are likely to underestimate the true number of influenza associated deaths occurring in the community.

The median age of deaths notified was 85 years (range 4 to 102 years). Two deaths occurred in children with 53 hospitalisations associated with severe complications of influenza reported in children.[5]

Influenza vaccine

Quadrivalent influenza vaccine will be available on the National Immunisation Program for the first time in 2016 from early April.

This influenza vaccine protects against 4 different strains of influenza. In 2016 the strains in the vaccine are:

- A/California/7/2009 (H1N1) - like virus
- A/Hong Kong/4801/2014 (H3N2) - like virus
- B/Brisbane/60/2008 - like virus
- B/Phuket/3073/2013 - like virus

The groups eligible for free influenza vaccine people in 2016 are the same as last year and include:

- Pregnant women in all trimesters
- Indigenous children aged 6 months to 5 years
- Indigenous adults aged 15 years and over
- People over 6 months of age with chronic medical conditions
- Non Indigenous people aged 65 years and over

There are 2 brands of influenza vaccine available on the National Immunisation Program in 2016. They are:

- Fluarix®Tetra for all people 3 years and over including pregnant women, give 0.5ml intramuscularly
- FluQuadri®Junior for children 6 months to less than 3 years, give 0.25ml intramuscularly

A common vaccine error last year was giving children over 3 years the incorrect dose of influenza vaccine. Please check the age of the child at the time of administration and select the correct vaccine.

Two doses of influenza vaccine are only required in children under 9 years of age who have never had an influenza vaccine before. If a child had a trivalent or quadrivalent vaccine at any time in a previous year they only require 1 influenza vaccine this year.

For people who are not eligible for influenza vaccine on the National Immunisation Program

Influenza vaccine is still recommended for anyone over the age of 6 months who wants to protect themselves from influenza. Remember Fluvax® brand should not be given to children under 10 years of age.

Pregnant women and influenza

Influenza is a serious disease for pregnant women and their babies

Influenza infection in pregnancy can lead to higher hospitalisations, deaths and increased premature deliveries and stillbirths especially in the second and third trimesters.[6-7] During non-pandemic periods, pregnant women are 5 times more likely to be hospitalised while pregnant compared to a period when they are not pregnant.[8] Influenza vaccine given to their mothers can also protect babies in the first 6 months of life with a study showing that influenza vaccine given to mothers in pregnancy was 91.5% effective in preventing hospitalisation of infants for influenza in the first 6 months of life.[9]

Influenza coverage in 2015 for Indigenous children

Due to high rates of influenza infection in Indigenous children, in 2015 influenza vaccine was provided free on the National Immunisation program for Indigenous children aged 6 months to less than 5 years. Coverage was very good in the NT (see Table).

Table: Coverage in Indigenous children aged 6 months to less than 5 years, 2015

	6 months<3 years	3 years to <5 years
Dose 1	2760 (72%)	2022 (66%)
Dose 2	1924 (50%)	1376 (45%)

Coverage for pregnant women in 2013 was estimated at 30%, please ensure we continue to promote influenza vaccine for all pregnant women.

Thank you to all the immunisation providers who worked hard in 2015 to implement the new Indigenous influenza vaccine program for children. Hopefully we can have even greater coverage for Indigenous children, adults, pregnant women and people with chronic medical conditions in 2016.

References

1. Zhang L, Prietsch SO, Axelsson I, Halperin SA. Acellular vaccines for preventing whooping cough in children. *Cochrane Database of Systematic Reviews* 2012; March 14(3).
2. Quinn HE, Snelling TL, Macartney KK, McIntyre PB. Duration of protection after first dose of acellular pertussis vaccine in infants. *Pediatrics* 2014;133:e513-9.
3. Munoz FM. Pertussis in infants, children, and adolescents: diagnosis, treatment, and prevention. *Seminars in Pediatric Infectious Diseases* 2006;17:14-9.
4. Amirthalingam G, Andrews N, Campbell H, Ribeiro S, Kara E, Donegan K, et al. Effectiveness of maternal pertussis vaccination in England: an observational study. *Lancet* 2014384: 1521-1528.
5. The Department of Health Australian influenza surveillance report No 9, 2015 12 to 25 September 2015 available at http:// www.health.gov.au/internet/main/publishing.nsf/ Content/48F7894CC1A055F9CA257EDF00133D 17/$File/Australian-Influenza-Surveillance-Report.pdf.
6. Centers for Disease Control and Prevention (CDC).Maternal and infant outcomes among severely ill pregnant and postpartum women with 2009 pandemic influenza A (H1N1)--United States, April 2009-August 2010. *MMWR Morb Mortal Wkly Rep*. 2011 Sep 9;60(35):1193-6.
7. ANZIC Influenza Investigators and Australasian Maternity Outcomes Surveillance System. Critical illness due to 2009 A/H1N1 influenza in pregnant and postpartum women: population based cohort study. *BMJ* 2010Mar 18;340:c1279.
8. Dodds L, McNeil SA, Fell DB, Allen VM, Coombs A, Scott J, MacDonald N. Impact of influenza exposure on rates of hospital admissions and physician visits because of respiratory illness among pregnant women *CMAJ*. 2007 Feb 13;176 (4):463-8.
9. Benowitz I Esposito DB Gracey KD Shapiro ED Vazquez M. Influenza vaccine given to pregnant women reduces hospitalisation due to influenza in their infants. *CID* 2010; 51 (12): 1355-1361.

****

NT Pneumococcal **Vaccination** and Revaccination Guideline

March 2016

www.nt.gov.au/health

For Indigenous people aged 15 years and older and non Indigenous people aged 18 years and older

#Prevenar 13® Category A only at time of diagnosis

Pneumovax 23®	All persons with Category A or B condition (including tobacco smokers)	No Category A or B condition
Dose 1	At time of diagnosis of Category A or B condition	Indigenous people ≥15 years of age Non Indigenous people ≥65 years of age
Dose 2	Due at least 5 years after dose 1	Indigenous people ≥50 years of age or 5 years after 1st dose, whichever is later Non Indigenous people – Not required
Dose 3	Indigenous people ≥ 50 years of age or 5 years after the 2nd dose whichever is later Non Indigenous people ≥ 65 years of age or 5 years after the 2nd dose whichever is later *Dose 3 not required if Category B and 1st dose given ≥50 years of age (Indigenous) or ≥65 years of age (non Indigenous) people	Not required

Does your client have a condition in Category A or B?

Category A - highest risk group Australian Immunisation Handbook (AIH) 10th Edition p326

Asplenia (including splenectomy, sickle cell disease or other haemoglobinopathies), immunocompromising conditions including undergoing immunosuppressive therapy, HIV infection, chronic kidney disease: eGFR < 30ml/min (stages 4 or 5) or receiving dialysis, CSF leak, organ transplant, cochlear implant, intracranial shunt, haematological and other malignancies, haemaopoietic stem cell transplants (Refer to AIH 10th Edition p156)

Category B - increased risk group (AIH 10th Edition p327)

Diabetes, chronic lung disease including severe asthma, chronic liver disease, chronic cardiac disease, alcoholism, Down syndrome, tobacco smoker

Vaccine Notes

All people with a Category A condition should receive a single dose of Prevenar 13® if no previous Prevenar 13® doses have been given. Give at least 12 months after any previous Pneumovax 23® dose. Wait 2 months between Prevenar 13® and any subsequent Pneumovax 23® dose.

Pneumovax 23® (23vPPV)– maximum 3 adult doses – can be given at/over 15 years of age. Doses given under 15 years of age do not count as adult doses.

Refer to Australian Immunisation Handbook 10th edition or contact Centre for Disease Control for people aged <15 years of age (Indigenous) or <18 years of age (non Indigenous).

Refer to AIH 10th Edition p329-330 for people who have received additional doses of Pneumovax 23® in childhood (apart from previously scheduled 18 month dose for Indigenous children)

Information:

NT Immunisation Register:

Top End 8922 8315

Central Australia 8951 6928

NORTHERN TERRITORY GOVERNMENT

Effective from March 2016

Adult and Special Groups **Vaccination** Schedule

Eligible groups	Diseases covered	Vaccine	Administration	Additional information
All people All people born after 1966 who have not recieved 2 MMR vaccines or who are not immune	Measles, mumps, rubella (MMR)	MMRII ® Priorix®	0.5 ml SC 0.5 ml IMI	Give two MMR vaccines at least 28 days apart
All people at 65 years of age	Diphtheria, tetanus, pertussis	Boostrix®, Adacel®	0.5 ml IMI	**Self funded -** If not given in the last 10 years
Indigenous people Indigenous people 15 years of age and over	Pneumococcal	Pneumovax23®	0.5ml IMI	See NT pneumococcal vaccination and revaccination guidelines
	Influenza	Fluarix Tetra®	0.5ml IMI	Annually
Indigenous people 20 years to 50 years *if not previously vaccinated or who do not have immunity through natural infection.*	Hepatitis B	Engerix B® adult or H-B Vax II®	1ml IMI	0, 1, 6 month
Non-Indigenous Non-Indigenous people over 65 years of age and over	Pneumococcal	Pneumovax23®	0.5ml IMI	See NT pneumococcal vaccination and revaccination guidelines
	Influenza	Fluarix Tetra®	0.5ml IMI	Annually
Pregnancy related Pregnant women (every pregnancy)	Influenza Diphtheria, tetanus, pertussis	Fluarix Tetra® Boostrix®	0.5ml IMI 0.5ml IMI	Any stage of pregnancy From the 28th week of pregnancy
Fathers and carers in the same household of infants under 7 months	Diphtheria, tetanus, pertussis	Boostrix®	0.5ml IMI	From the 28th week of the mother's pregnancy, if no dTpa vaccine in the last 10 years
People with chronic medical conditions All people 6 months of age and over with chronic medical conditions *Other vaccines may be required, see Australian Immunisation Handbook (AIH) for specific conditions.*	Pneumococcal	Pneumovax 23® (Prevenar13® - high risk)	0.5ml IMI	See NT pneumococcal vaccination and revaccination guidelines
	Influenza		0.5ml IMI	Annually
Household and sexual contacts of people with Hepatitis B *Hepatitis B vaccine is also recommended for other groups see AIH for details.*	Hepatitis B	Engerix B® adult or H-B Vax II®	1ml IMI	0, 1, 6 month

More Information:

- NT Immunisation Register - Top End 8922 8315 - Central Australia 8951 6928
- Australian Immunisation Handbook (AIH)

NORTHERN TERRITORY GOVERNMENT

Childhood **Vaccination** Schedule

Effective from **March 2016**

	Hepatitis B	Rotavirus	Diphtheria Tetanus Pertussis Hepatitis B Poliomyelitis *Haemophilus influenzae* type b	Conjugate Pneumococcal (13vPCV)	*Haemophilus influenzae* type b Meningococcal C	Measles Mumps Rubella	Hepatitis A	Diphtheria Tetanus Pertussis	Measles Mumps Rubella Varicella	Diphtheria Tetanus Pertussis Poliomyelitis	Human Papillomavirus	Varicella	Polysaccharide Pneumococcal	Adult Diphtheria Tetanus Pertussis	Influenza REPEAT YEARLY	
	Engerix®-B 0.5ml IMI	Rotarix® 1.5ml ORAL	INFANRIX®hexa 0.5ml IMI	Prevenar 13® 0.5ml IMI	Menitorix® 0.5ml IMI	M-M-R®II 0.5ml SC or Priorix® 0.5ml IMI	VAQTA® 0.5ml IMI	Tripacel® 0.5ml IMI or Infanrix® 0.5ml IMI	Priorix-Tetra® 0.5ml SC or ProQuad® 0.5ml SC	Infanrix®IPV 0.5ml IMI	Gardasil® 0.5ml IMI	Varivax® 0.5ml SC or Varilrix® 0.5ml SC	Pneumovax23® 0.5ml IMI	Boostrix® 0.5ml IMI	Flu Quadri® Junior 0.25ml IMI <3 years	Fluarix Tetra ® 0.5ml IMI ≥3 years
Birth ●	✓															
6 weeks		● ✓	✓	✓												
4 months		● ✓	✓	✓												
6 months			✓	✓												
12 months					✓	✓	◘									
18 months				■			◘	✓	✓ ‡							
4 years										✓						
5 years																
12 years											✓✓✓					
13 years												✓		✓		
15 years													■			■

✸ 6 Months — <3 yrs — ≥3 yrs — 5 yrs ✸

Vaccine notes:

✓	= All children.	✸	= Indigenous children only aged 6 months to less than 5 years. All children with a chronic medical condition 6 months of age and over. Give 2 doses, 28 days apart to children under 9 years of age who are receiving influenza vaccine for the first time in their life.
●	= ORAL VACCINE: First dose must be given by 14 weeks and 6 days of age; Second dose must be given by 24 weeks and 6 days of age.		
■	= Indigenous people only.	‡	= NEVER to be given as the 1st dose of MMR containing vaccine.
◘	= All Indigenous children. Non-Indigenous children living in remote communities.	●	= BCG vaccine - for eligible groups and vaccine eligibility please refer to the CDC Immunisation Website: www.health.nt.gov.au/Centre_for_Disease_Control/Immunisation
✓✓✓	= Requires 3 doses given at 0, 2 and 6 months.		

Information:

NT Immunisation Register:

Top End **8922 8315**

Central Australia **8951 6928**

NORTHERN TERRITORY GOVERNMENT

Enteric disease in the Northern Territory in 2015

***Anthony Draper*[1,2]**
[1]OzFoodNet Epidemiologist, Centre for Disease Control, Darwin
[2]Master of Philosophy (Applied Epidemiology) Scholar, National Centre of Epidemiology and Population Health, Australian National University

Abstract

In 2015, there was an increase of 34% in notifications of foodborne disease in the Northern Territory compared to 2014. Salmonellosis notifications accounted for 51% of all foodborne disease notifications in 2015. Salmonellosis, campylobacteriosis and shigellosis notifications were all higher than the previous 5 year mean. There were a number of foodborne and non-foodborne disease outbreak investigations conducted in 2015.

Key words: OzFoodNet; Northern Territory; outbreak; foodborne disease.

Introduction

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

Methods

Data was extracted from the NT Notifiable Diseases System (NTNDS) and also analysed from the data warehouse using Business Objects. Population figures were obtained from the NT Department of Health's Health Gains Planning population data.[1]

Results and Discussion

In 2015 there were 1128 notifications of foodborne or potentially foodborne disease* reported in the Northern Territory. This is 34% more than the 5 year mean (5YM) (840) and 25% more than the previous year (902). Salmonellosis notifications accounted for 51% of the foodborne disease notifications in the NT, followed by campylobacteriosis notifications (35%) and shigellosis notifications (13%).

There were 318 non-foodborne enteric disease notifications reported in the NT in 2015. This is on par with the 5YM (318) but 66% more than the previous year (192). Rotavirus notifications made up 60% of these non-foodborne disease notifications, followed by cryptosporidiosis (38%). There were 6 foodborne or suspected foodborne outbreaks and 16 non-foodborne outbreaks investigated in 2015.

Salmonellosis

In 2015 there were 572 notifications of salmonellosis in the NT which is 22% more than the 5YM (468 cases) and 18% more than the previous year (484). The median age of salmonellosis cases was 3 years (range 0–80).

The highest rate of disease was seen in the 0–4 year age group with a rate of 1441 cases per 100 000. This age group represents 48% of all salmonellosis notifications in the NT (277 cases).

The rate of salmonellosis in the Indigenous population was 238 cases per 100 000 (150 cases) compared to 218 cases per 100 000 (379 cases) in the non-Indigenous population. This is the second year that there has been no statistically significant difference in the rates of salmonellosis between Indigenous and non-Indigenous Territorians (rate ratio (RR) 1.3, 95% confidence interval (CI) 0.9–1.6, p=0.06) and represents a closing of 'the gap.'

In the 0–4 year age group there was also no significant difference in the rate of disease in the Indigenous population (1219 cases per 100 000,

* This includes total number of notifications for amoebiasis, botulism, brucellosis, campylobacteriosis, cholera, salmonellosis, shigellosis, STEC/VTEC, typhoid, yersiniosis, ciguatera, *Vibrio* food poisoning, and listeriosis. It does not include rotavirus, cryptosporidiosis, hepatitis A and hepatitis E.

88 cases) compared to the non-Indigenous population (1541 cases per 100 000, 185 cases) with the RR being 1.3 (95% CI 0.9–1.6, p=0.07) (Figure 1).

Reports of salmonellosis normally increase during the wetter, warmer months of the year. In 2015, salmonellosis cases again peaked during the wet season and declined during the dry.

In 2015, 93% (531/572) of salmonellosis notifications were identified to the serovar level compared to 91% of salmonellosis notifications in 2014. One of the major pathology service providers servicing the NT introduced a faecal multiplex polymerase chain reaction (PCR) assay in September 2013 and this has resulted in an increase in salmonellosis notifications detected by PCR but culture negative for *Salmonella* Spp. In 2015, the serovar with the highest number of notifications was *Salmonella* Virchow (n=88), followed by *S.* Saintpaul (n=76), *S.* Typhimurium (n=65) and *S.* Ball (n=25). In the past, *S.* Ball, *S.* Virchow, *S.* Lansing, *S.* Reading and *S.* Saintpaul were the most commonly reported serovars in the NT, with these serovars thought to have established an ecological niche in the NT.[2,3,4]

There were 2 foodborne outbreaks of salmonellosis in 2015; 1 was caused by *S.* Typhimurium phage type (PT) 9 and associated with duck prosciutto served at a restaurant and another outbreak was due to *S.* Typhimurium PT168a and was associated with a cultural festival.

Notifications of the typical NT 'environmental' *Salmonella* serovars generally increased in 2015. Reported case numbers for *S.* Saintpaul were 41% higher than 2014 (76 vs 54) and 46% higher than the 5YM (37). Case numbers for *S.* Virchow were 44% higher than 2014 (88 vs 61) and 55% higher than the 5YM (57). *S.* Lansing, and *S.* Ball were notified at about the same level as the 5YM and as 2014 (Figure 2).

There was a variety of different phage types of *S.* Typhimurium reported in 2015. Most *S.* Typhimurium cases reported in the NT in 2015 were *S.* Typhimurium PT9 (31%, 20/65 cases). There was an outbreak of 8 cases of this phage type associated with consuming duck prosciutto mentioned above. There were 12 notifications of *S.* Typhimurium PT135 and 10 notifications of *S.* Typhimurium PT108 which are phage types that have been implicated in numerous egg associated outbreaks in Australia in the last few years.

Figure 1. Salmonellosis rates by age group and Indigenous status, Northern Territory, 2015.

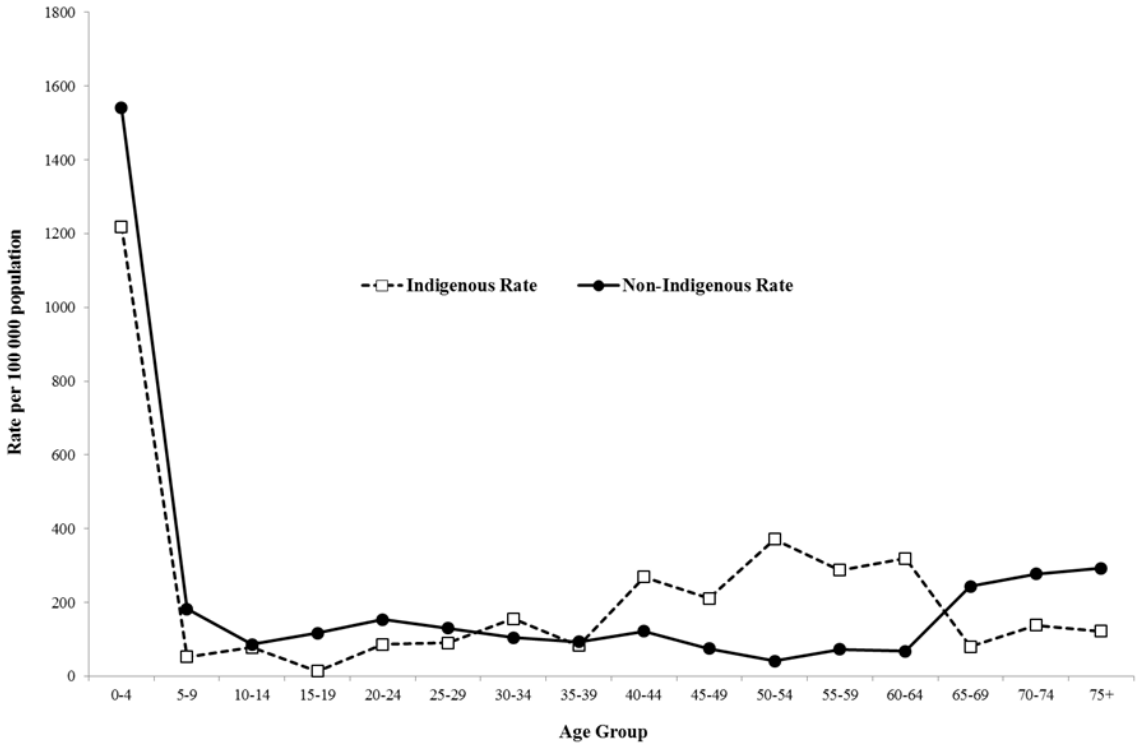
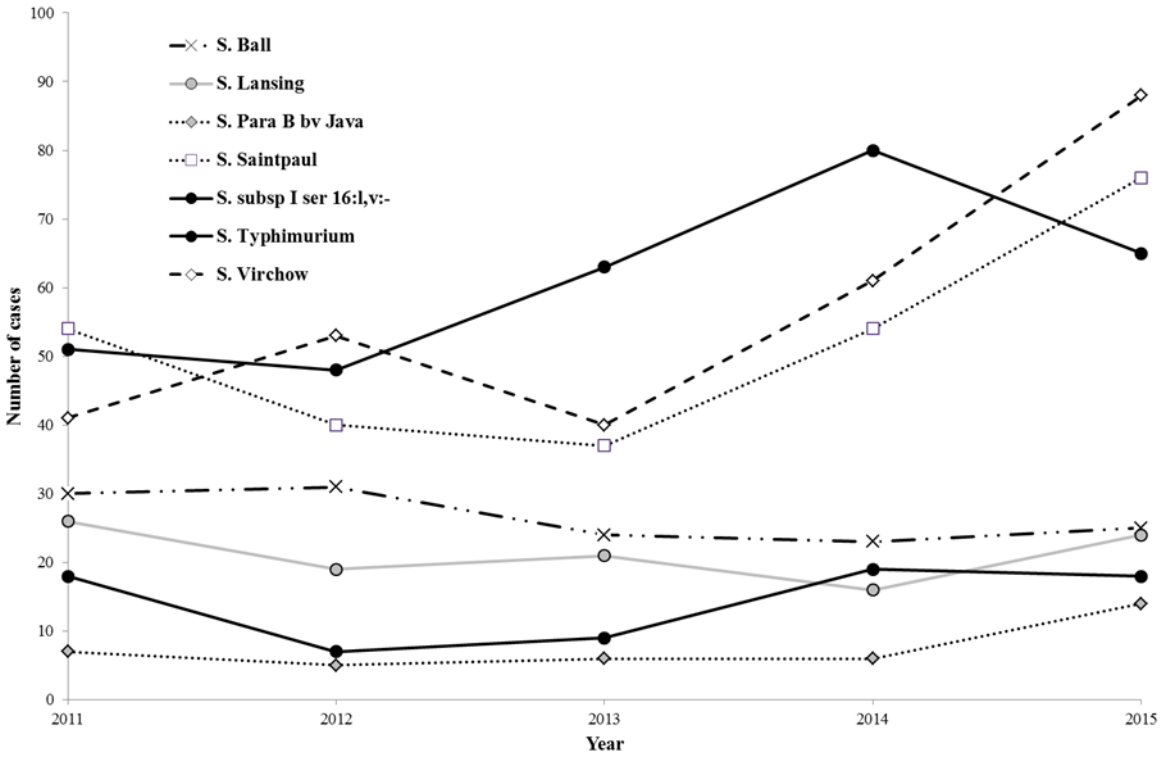


Figure 2. Notifications of selected *Salmonella* serovars, Northern Territory, 2015.



Typhoid

There were no cases of typhoid notified in 2015.

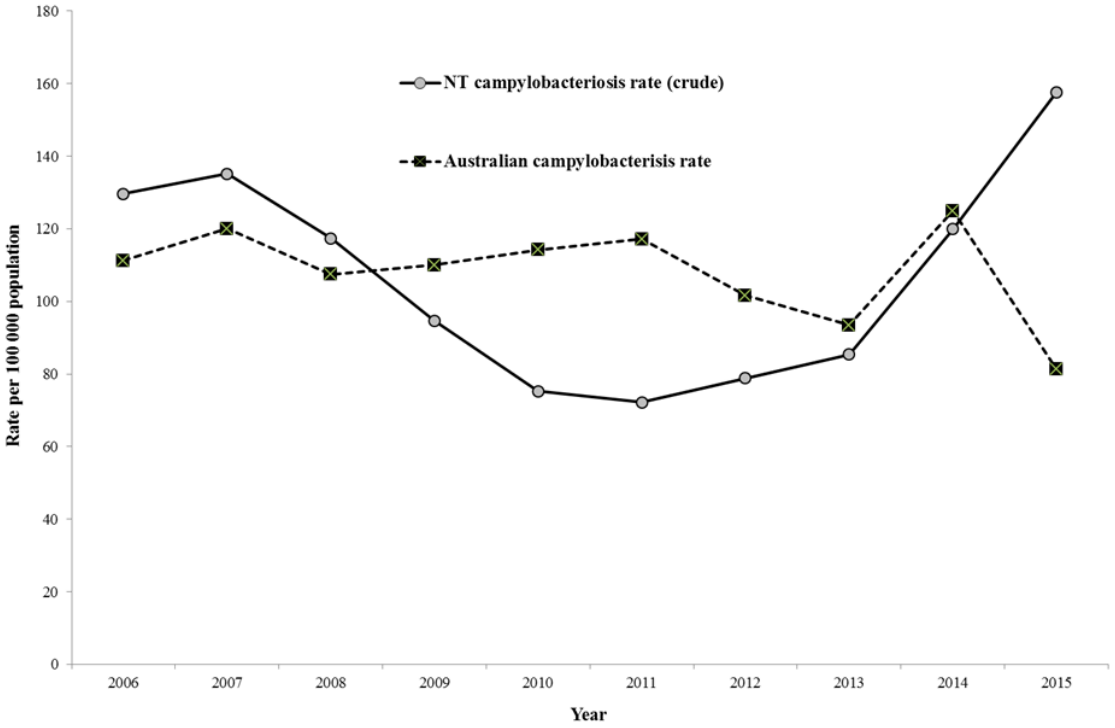
The NT has averaged 2 cases of typhoid per year since 2009, all in people returning from typhoid endemic countries. The last 6 reported cases since 2012 were either returning to their birth countries or visiting family members in typhoid endemic countries. This type of traveller is less likely to seek pre-travel health advice and vaccination.[5]

Campylobacteriosis

In 2015 there were 390 notifications of campylobacteriosis in the NT. This was 90% higher than the expected number of cases (5YM=205) and 33% more than the previous year (294). The overall rate of campylobacteriosis was 158 cases per 100 000. The median age of campylobacteriosis cases was 20 years (range 0–79 years). There has been an increase in the rate of campylobacteriosis in the NT over the last few years, and the rate in 2015 was the highest since 2007 when the rate was 135 cases per 100 000 population. Since 2010, the implementation of the Primary Production and Processing (PPP) Standard for Poultry Meat saw rates fall significantly lower than those in the 5 years preceding its implementation. Nearly all of the chicken meat in the NT is supplied from interstate; there is essentially no local NT chicken meat industry (Frost, pers comm.). Much of the product that enters the NT is frozen, and the rest is chilled (Frost, pers comm).

The introduction of the multiplex PCR by a private pathology provider probably accounts for most of increase in campylobacteriosis seen in 2014 and 2015. Of the 270 campylobacteriosis notifications received in the Darwin CDC office in 2015, 141 (52%) were PCR positive but culture negative. The sharp increase in the rate of campylobacteriosis in the NT mirrors the increase seen across Australia in 2014 (Figure 3) however the rate of campylobacteriosis in the NT also increased in 2015 and was higher than that seen in the rest of Australia. A project was begun in November 2015 whereby campylobacteriosis cases in Darwin were sent an SMS in order to ascertain restaurants, takeaways and overseas destinations that were visited prior to becoming ill. The results of this project will be finalised and published in 2016.

Figure 3. Rates of campylobacteriosis cases in the NT and Australia, 2006-2015



The highest number of cases and rate of disease was seen in the 0-4 year age group with 145 notifications and a rate of 754 cases per 100 000. This age group represents 37% of all campylobacteriosis notifications in the NT. The rate of disease in this age group was significantly higher in the Indigenous population (1510 cases per 100 000) than the non-Indigenous population (250 cases per 100 000) with a RR of 6.0 (95% CI 4.0-9.0, p=0.01). However, in the remaining population (>4 years old), the non-Indigenous rate of campylobacteriosis (110 cases per 100 000) was significantly higher than the Indigenous rate (23 cases per 100 000) with a RR of 4.9 (95% CI 2.9–8.2, p=<0.01).

Shigellosis

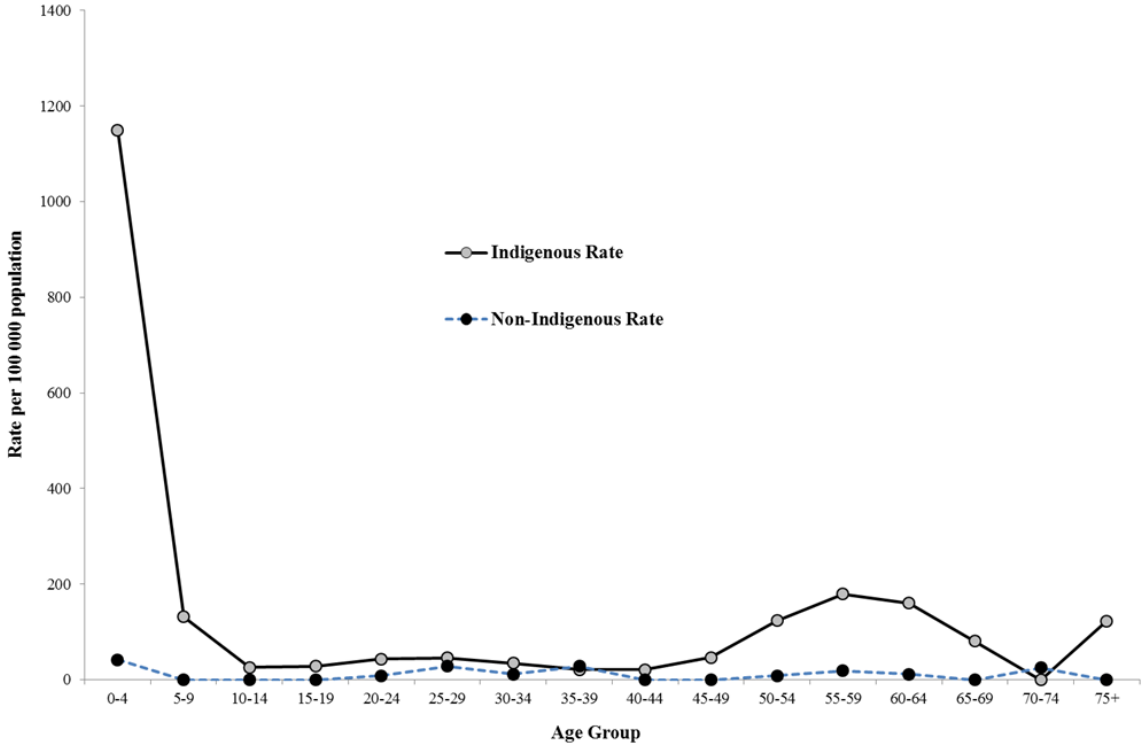
In 2015 there were 146 notifications of shigellosis reported in the NT. The number of cases was 48% higher than the previous 5YM (99 cases) and 42% higher than the number of notifications received in 2014 (103). The overall rate of shigellosis was 59 cases per 100 000 population. The median age of cases was 3 years (range 0–75 years). The introduction of the multiplex PCR resulted in a higher proportion of notifications where *Shigella* Spp. was detected by PCR but was not cultured and hence not able to be biotyped. Of the shigellosis notifications that were biotyped (63 cases), *S. sonnei* biotype a (45 cases) was the predominant biotype.

The highest number of cases (88/146, 60% of all notifications) and rate of disease was seen in the 0-4 year age group, with 458 cases per 100 000 population. Of the 88 cases recorded in this age group, 83 (94%) were Indigenous. This demographic accounted for over half (57%) of all shigellosis cases in the NT and had rate of disease of 1150 cases per 100 000. There were also moderately high rates of disease seen among older Indigenous people, particularly females (Figure 4). This may (in part) be a reflection of cultural practices where aunts and grandmothers are often involved in the care of young children in Indigenous populations.

The rate of disease did not vary between the sexes, with 57 cases per 100 000 in females (67 cases) vs. 60 cases per 100 000 in males (79 cases) (Figure 4).

Shigellosis is more commonly reported in the Indigenous population. The rate of shigellosis in the Indigenous population was 59 cases per 100 000 population (123 cases) compared to 13 cases per 100 000 population in the non-Indigenous population (22 cases), with a RR of

Figure 4. Shigellosis rates, by age and Indigenous status, NT, 2015.



13.3 (95% CI 8.4–20.1, p=<0.01). This represents a considerable 'gap' between Indigenous and non-Indigenous health outcomes that can only be closed by addressing the social determinants of health. There were 22 cases of shigellosis in the non-Indigenous population which is higher than previous years; the majority of this is due to increased detection by PCR of shigellosis in returned travellers; 8 cases were known to have acquired their infection overseas with the majority (5/8) acquiring their infection in Indonesia (Bali and Lombok). In 2015 there were 2 outbreaks of shigellosis in remote Indigenous communities following cyclones.

Cryptosporidiosis

In 2015 there were 122 notifications of cryptosporidiosis. This is on par with the 5YM (99 cases) and 37% more than the previous year (89 cases). The median age of cases was 1 year (range 0–37 years).

Cryptosporidiosis is predominantly a disease reported in children, with 99 of the 122 (81%) cases notified being in the 0–4 year age group. In this age group the rate of disease was similar in males and females with 519 cases per 100 000 in males (51 cases) vs. 511 cases per 100 000 in females (48 cases). In the overall population the rate of disease between males (48 cases per 100 000, 63 cases) and females (51 cases per 100 000, 59 cases) was also similar.

The rate of disease was significantly higher in the Indigenous population with 102 cases per 100 000 (75 cases) compared to 25 cases per 100 000 (44 cases) in the non-Indigenous population (RR 4.0, 95% CI 2.8–5.9, p=<0.01). The rate of disease in the 0-4 year age group was significantly higher in Indigenous children (1025 cases per 100 000, 74 cases) than non-Indigenous children (208 cases per 100 000, 25 cases), (RR 4.9, 95% CI 3.1–7.7, p<0.01).

In 2015, the peak number of cases occurred in December; typically numbers usually peak during and immediately following the wet season which is October through to May. There were no large outbreaks reported in 2015 (Figure 5).

Hepatitis A

There were 5 cases of hepatitis A notified in the NT in 2015.

The first was a non-Indigenous male teenager who spent his entire exposure period in Bangladesh. He was born in Bangladesh and returned to his country of birth on holiday.

Figure 5. Cryptosporidiosis notifications by month in the NT, 2011-2015

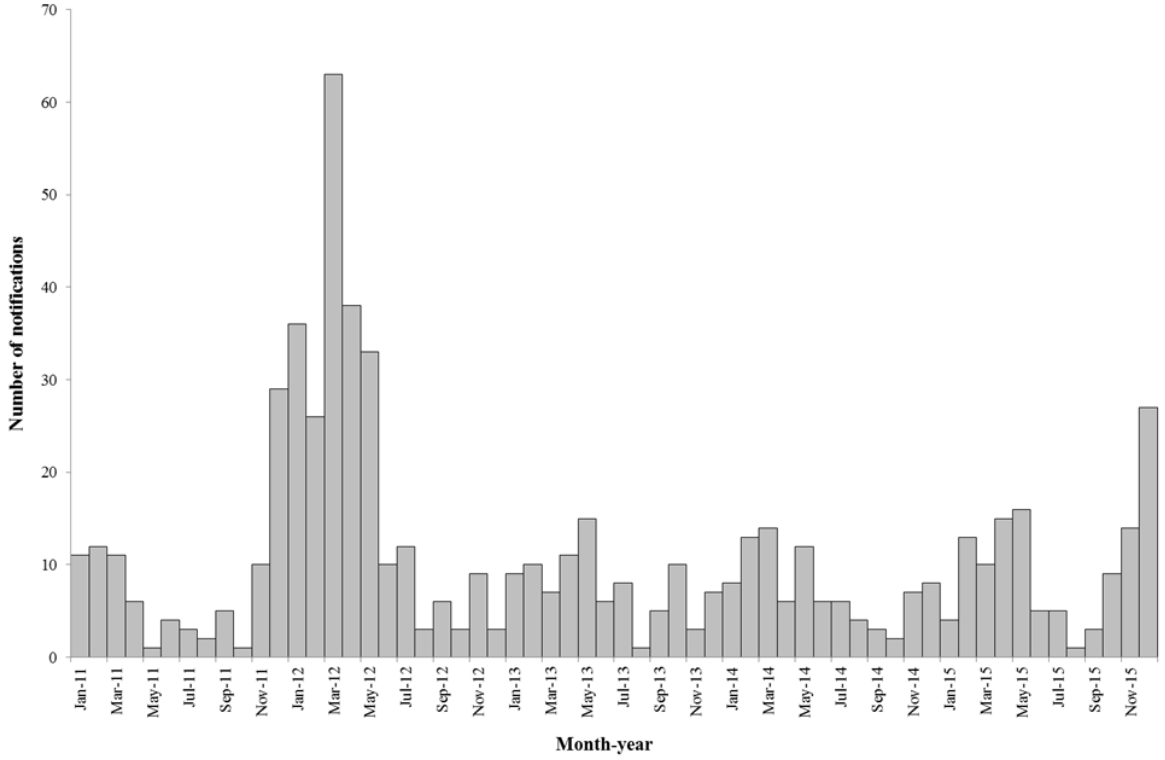
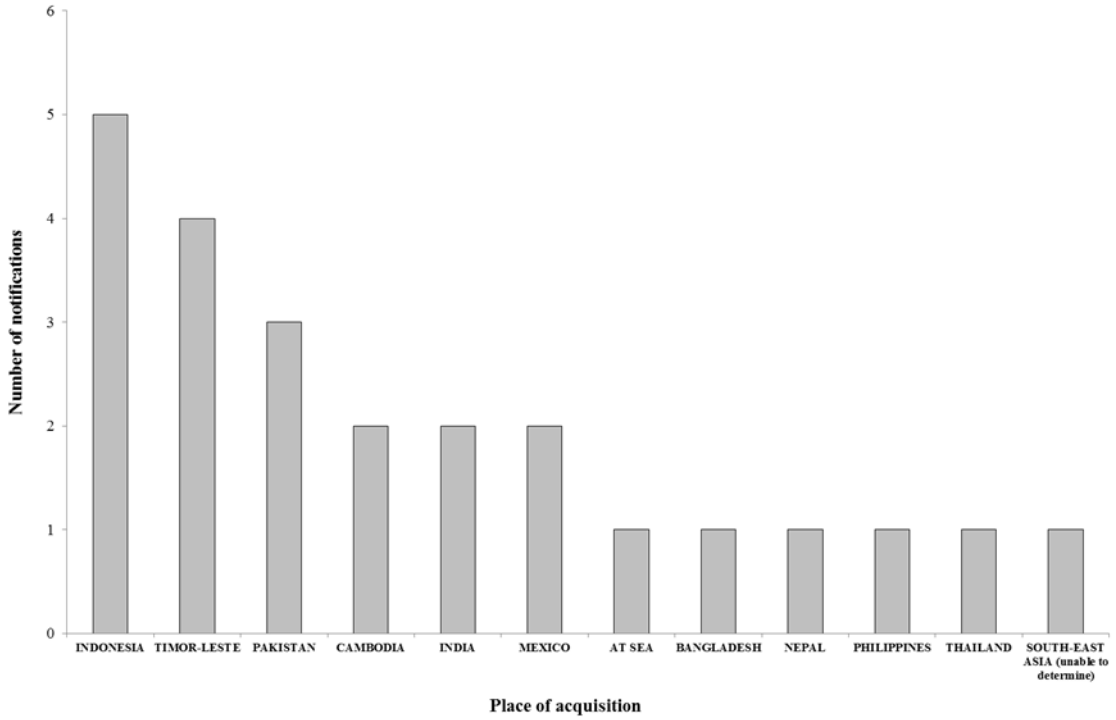


Figure 6. Imported hepatitis A notifications in the NT by country of acquisition, 2006-2015 (n=24)



The second case was an Indian born male returning to Australia after holidaying in his country of birth. His entire exposure period was spent in India.

The third case was a child under 5 years who was diagnosed with hepatitis A upon returning from his parent's homeland of Pakistan.

The fourth case was a middle-aged Japanese national who was evacuated from Timor-Leste to Darwin and subsequently diagnosed with hepatitis A. He was living in congregate style accommodation in Dili for his entire exposure period. It was advised that his co-workers and contacts in Timor-Leste be provided with post-exposure prophylaxis (PEP).

The fifth case was a middle aged non-Indigenous woman who acquired her infection in Bali, Indonesia.

CDC's public health response for all typhoid notifications included providing screening of co-travellers of cases as well as household contacts and contacts who worked in sensitive occupations such as the food industry.

Since 2006, 24/40 (60%) cases of hepatitis A reported in the NT have been overseas acquired; many of the cases are people living in Australia who have returned to their country of birth for a holiday or to visit family or, irregular maritime arrivals arriving via boat from Indonesia (Figure 6).

Since the introduction of the funded vaccine for all Indigenous children under 5 in the NT (along with South Australia and Western Australia) in November 2005, there has been a decline in the number of hepatitis A notifications in the both the Indigenous and non-Indigenous population. There have been only 4 cases of locally acquired hepatitis A reported in the NT since 2006, the last one being in 2010. The vaccination program in NT Indigenous children has resulted in a herd effect in the non-Indigenous population of the NT.

STEC/HUS

There were no cases of Shiga toxin (verotoxin) producing *Eschericia coli* (STEC/VTEC) or haemolytic uraemic syndrome (HUS) reported in the NT in 2015.

STEC/HUS is not often reported in the NT and is in fact likely underreported.[5] STEC/VTEC is not routinely tested for by most of the laboratories that service the NT. The sporadic cases that have been reported historically have generally been in the region where IMVS/ Medvet laboratory provided a pathology service (Central Australia) as all blood-stained stool samples were screened for STEC/VTEC as part of their standard procedure.

Yersiniosis

There were 14 cases of yersiniosis reported in the NT in 2015 compared to 10 cases in 2014. Of the 14 cases reported in 2015, 10 were culture negative but PCR positive with *Yersinia* being included in the enteric multiplex testing introduced in 2013. It is expected that more cases of yersiniosis will be reported in the coming years as a result of this more sensitive method.

Of the 14 yersiniosis cases, 2 likely acquired their infections overseas in the United Arab Emirates and Fiji. There was only 1 yersinisosis case in 2015 in the Indigenous population. In 2014 there were no yersiniosis cases in the Indigenous population.

Listeriosis

Listeriosis is not often reported in the NT. In 2015 there were only 2 cases notified.

A 19 year old Indigenous female with sepsis tested positive for *Listeria monocytogenese* 1 day prior to giving birth by planned caesarean section at term. The neonate was asymptomatic and subsequently tested negative for *L. monocytogenese*. The mother had poorly controlled type 2 diabetes.

A 62 year old non-Indigenous man was notified with listeriosis. He had type 2 diabetes, was immunocompromised, undergoing steroid therapy and had chronic renal disease.

In both cases, no high risk foods were identified as being consumed.

Amoebiasis

There were 2 cases of amoebiasis notified in 2015.

A 50 year old non-Indigenous male likely acquired his infection in the Philippines. A PCR test detected the presence of *Entamoeba histolytica* in his faeces. There was no extraintestinal spread.

A 39 year old Timorese man was medivacced to Australia for treatment. Upon arrival in Australia he was diagnosed with amoebiasis by serological methods. He had extensive hepatic abscesses.

Ciguatera

There were no cases of ciguatera fish poisoning reported in 2015 in the NT.

Ciguatera was made a notifiable disease in the NT in 2010 and there has only been 1 case (in 2013) notified to date.

Vibrio food poisoning

There was 1 case of *Vibrio* food poisoning reported in the NT in 2015.

A 65 year old Indigenous woman was notified after *Vibrio parahaemolyticus* was isolated from her stool culture. She had multiple comorbidities and had eaten oysters during her incubation period that were likely not commercially purchased (i.e. wild).

Outbreak and cluster investigations

In 2015 there were 6 foodborne, suspected foodborne or waterborne outbreaks investigated. The settings exposed for the outbreaks were varied and included various types of food businesses (Table 1). *S.* Typhimurium was identified as the likely cause of 2 outbreaks. *Campylobacter* was also identified as the aetiological agent of 1 outbreak. The number of suspected foodborne outbreaks investigated in 2015 was less than the number investigated in 2014 (13).

There were 16 non-foodborne outbreaks investigated in the NT during 2015. These outbreaks mainly occurred in childcare centres (4) and aged care facilities (2). Norovirus was implicated in 4 of the outbreaks, *Shigella* Spp. in 2 outbreaks and rotavirus in 3 outbreaks. The rest were of undetermined aetiology. A summary of these outbreaks is included in Table 2.

An outbreak of note

An outbreak of *S.* Typhimurium PT9 at a restaurant resulted in 23 cases of illness and 8 laboratory confirmed cases of *Salmonella* infection in diners who attended the restaurant. There were 4 hospitalisations as a result of the outbreak. A cohort study was conducted involving 76/80 (95%) patrons and 3 staff. An association was detected between consuming duck prosciutto and illness (p=0.0008) with an undefined RR as duck prosciutto was universally eaten by all 23 cases. The attack rate for the duck prosciutto was 38%. An environmental health inspection of the restaurant identified that the duck proscuitto was cured on the premises and that quality control of the process was inadequate. Duck prosciutto was immediately removed from the menu. Samples of duck meat and duck prosciutto were collected and tested negative for *Salmonella* spp. Samples of duck meat tested negative for coliforms however samples of duck prosciutto prepared from the duck meat contained high levels of coliform bacteria which indicated contamination of the prosciutto during the curing process.

Acknowledgments

Members of the OzFoodNet network around Australia.

Table 1. Summary of foodborne and suspected foodborne outbreaks investigated in the NT in 2012

Outbreak register / number	Onset month	Aetiology (no lab confirmed cases)	No. Exposed	Cases	Transmission / Vehicle	Setting Exposed
DAR0515	Jun	*S.* Typhimurium PT9 (8)	83	23	Duck prosciutto	Restaurant
DAR0815	Aug	*S.* Typhimurium PT168a (2)	Unknown	4	Unknown	Outdoor food festival
DAR0915	Aug	Unknown	Unknown	3	Raw egg aioli	Café/Restaurant
DAR1015	Sep	*Campylobacter* Spp. (3)	Unknown	19	Unknown	Overseas/ Indonesia
DAR1215	Dec	Unknown	40	8	Unknown	Private residence
KA0315	Dec	Unknown	7		Fruit smoothie	Outdoor festival/ market

Table 2. Summary of non-foodborne outbreaks investigated in the NT in 2015

Ref No	Onset Month	Aetiology (no. lab. confirmed cases)	No. Exposed	Cases	Transmission / Vehicle	Setting Exposed
EA0115	Jan	*Shigella* Spp.(20)	Unknown	20	Unknown	Community
DAR0115	Feb	Norovirus (3)	137	33	Person-to-person	Aged care
DAR0215	Mar	Unknown	Unknown	8	Person-to-person	Child care
DAR0315	Mar	Rotavirus (27)	1600	27	Unknown	Community
KA0115	Mar	Unknown	22	8	Unknown	Child care
EA0215	Mar	*Shigella* Spp.(1)	Unknown	4	Unknown	Community
DAR0415	Mar	Unknown	159	13	Person-to-person	Child care
DAR0615	Jul	Norovirus (2)	65	20	Person-to-person	Aged Care
KA0215	Jul	Norovirus (2)	Unknown	14	Person-to-person	Military base
DAR0715	Jul	Norovirus (2)	5000	42	Person-to-person	Military base
DAR1115	Oct	Rotavirus (1)	Unknown	9	Person-to-person	Child care
AS0315	Oct	Unknown	Unknown	24	Person-to-person	Hospital
DAR1315	Nov	Unknown	37	25	Person-to-person	Child care
AS0115	Apr	Unknown	25	16	Person-to-person	School camp
EA0315	May	Rotavirus (15)	800	15	Person-to-person	Community
AS0215	Jun	Unknown	20	16	Person-to-person	Camp

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

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

Northern Territory Government Environmental Health.

Staff from the Northern Territory Government Pathology Service (NTGPS). Western Diagnostic Pathology, Sullivan and Nicolaides Pathology, Healthscope (Australian Clinical labs) Pathology, PathWest, SA Pathology and Microbiological Diagnostic Unit at Melbourne University (MDU).

References

1. NT Department of Health, Staff Intranet. Health Gains Planning Publications. http:// internal.health.nt.gov.au/healthstatistics/library/ Pages/default.aspx (accessed 19/02/2016).
2. Northern Territory OzFoodNet Annual Report (2009).
3. Williams S, Patel M, Markey P, Muller R, Benedict S, Ross I, Heuzenroeder M, Davos D, Cameron S, Krause V. Salmonella in the tropical household environment - Everyday, everywhere. *J Infect*. 2015 Dec;71(6):642-8.
4. Williams S, Markey P, Harlock M, Binns P, Gaggin J, Patel M. Individual and household-level risk factors for sporadic salmonellosis in children. *J Infect*. 2016;72:36-44.
5. Draper A, Markey P. Notifications of haemolytic uraemic syndrome and Shiga-toxin producing E. coli infections to the Northern Territory Centre for Disease Control – an audit. *The NT Dis Control Bull*. 2014;21(1).

****

Audit of the availability of condoms in remote communities of the Northern Territory

Kelly Hosking,[1] Jan Holt[1] and Linda Garton[1,2]
[1]Centre for Disease Control, Darwin
[2]Master of Philosophy (Applied Epidemiology) Scholar, National Centre of Epidemiology and Population Health, Australian National University, Canberra

Abstract

Historically the Northern Territory (NT) has the highest rates of sexually transmissible infections (STIs) in Australia. People in remote communities in the NT suffer this burden disproportionally. Condom access is an important strategy for the reduction of STIs. The Centre for Disease Control, Remote Sexual Health Program advocates and collaborates with remote primary health centres to provide condoms, with a focus on 24 hour availability, and facilitates community consultation around condom dispensers, their locations and installation. This audit compares 2014 to 2015 condom access data for government and non-government health centres throughout the NT.

Keywords: Condoms; dispensers; access; STIs; remote communities; Indigenous health.

Background

The Northern Territory (NT) has the highest rates of bacterial sexually transmissible infections (STIs) in Australia. All cases of syphilis, gonorrhoea, chlamydia and trichomonas are notifiable diseases. In 2014, there were 5,275 STI notifications in the NT, with over 75% of individuals diagnosed, from remote communities.[1,2] Increasing condom access in the NT is a key strategy of the Sexual Health and Blood Borne Virus Unit (SHBBV Unit) to prevent transmission and reduce high rates of STIs.

The Remote Sexual Health Program (RSHP) uses a continuous quality improvement (CQI) approach to support the implementation of a best practice program delivery for prevention and control of STIs and Blood Borne Viruses (BBV). The program aims to support primary health care services by providing education, technical assistance and clinical leadership. The RSHP team also facilitate condom distribution throughout the NT. This involves community consultation around appropriate condom access sites and assisting with condom dispenser installation. The team works with clinic staff to identify gaps in 24 hour/7 day per week (24/7) access, and improving accessibility in established condom locations, such as clinic toilets, by developing systems for restocking routinely.[3]

In 2014, the RSHP team conducted a baseline audit on condom access within clinics, 24/7 access within communities, and the total number of distribution points within the clinic and community. The aim of the baseline audit was to identify gaps, so that targeted support could be provided to increase condom availability in the NT, with the ultimate aim of contributing towards a decreased STI/BBV prevalence (and transmission). A subsequent audit was undertaken in 2015 to compare data initially collected in 2014 and to assess if improvements had been made to condom accessibility and identify if any gaps and challenges remained.

Methodology

The SHBBV Unit RSHP teams from the Top End, Alice Springs, Katherine, Tennant Creek, and Nhulunbuy, together with the Tiwi Islands Sexual Health Coordinator and Public Health staff from Katherine West Health Board and Sunrise Health Service, conducted the condom distribution audit, by region, which included all Primary Health Care Centres in the NT. A standardised excel template was used by all staff conducting the audit, to ensure data was collected systematically. The team either visited or telephoned community clinics. Information collected included:

- Condom access within the clinic
- Availability of dispensers
- Type of dispensers
- Number of condoms dispensed
- 24 hour condom access
- Responsibility for refilling the dispensers
- Ceremony kit availability, where appropriate
- Generalised comments relevant to the audit.

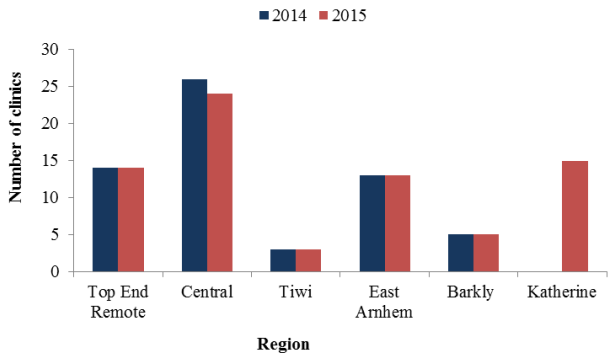
All data were reviewed and collated by the SHBBV Unit Top End team to ascertain the number of condom distribution points and any gaps or issues with access. In 2015 data was collected from a total of 76 out of 81 (94%) remote primary health centres, which included both government and community controlled health centres. One East Arnhem community and 1 Top End West community were excluded from analysis as they are intermittently staffed by outreach clinicians. Due to staffing limitations data from Katherine region health centres were only available for 2015 and were therefore excluded from the data analysis. The number of clinics audited in 2014 was 61 out of 81. This audit therefore compares 61 primary health clinics, which represents 75% of clinics in the NT. The audit has focused only on remote communities where the highest burden of infection lies. The urban communities, clinics and sites have not been included in this process.

Results

Condom availability within clinics

In 2014, all clinics had condoms available, with freely accessible condoms available in 60 out of 61 clinics audited (98%). Ease of access was defined as having condoms available in dispensers, bowls or paper bags in clinic rooms and/or toilets. In comparison, 2015 data showed a slight downward trend to freely accessible condoms in clinics to 58 out of 61 (95%) (see Figure 1).

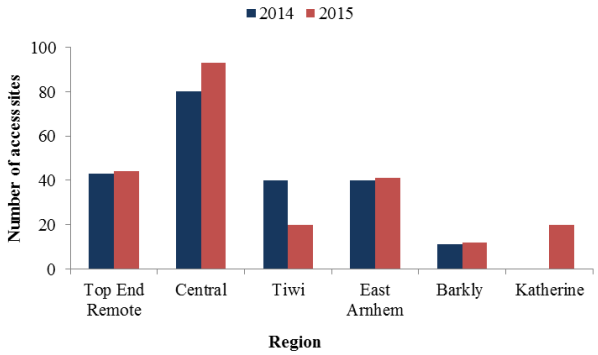
Figure 1. Number of clinics where condoms are freely available — by year and region



Condom access points within clinic and community

In terms of access points to condoms within the clinic and community there was a slight increase in the number of condom access points from 2014 to 2015 across most regions. However, in Tiwi there was a 50% reduction in condom access points from 40 in 2014 to 20 access points in 2015 (see Figure 2).

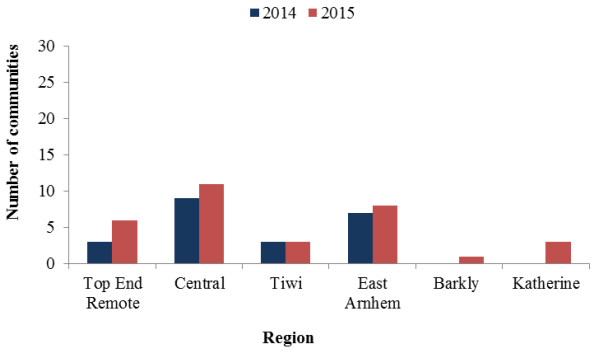
Figure 2. Total number of condom access sites — by year and region



Condom 24/7 access within community

In 2014, there were 22 out of 61 (36%) communities with 24/7 access with an increase to 29 out of 61 (48%) in 2015. Therefore, there has been a 12% increase in the number of communities with 24/7 condom access within a 12 month period (see Figure 3).

Figure 3. Number of communities with 24/7 condom access — comparison by year and region



Responsibility for restocking condom dispensers ranged from routine to ad hoc with many clinics not knowing who was responsible for this job.

Ceremonial kits were only available in a few clinics. Other clinics where ceremony occurs are aware of the protocols around this and assemble them as required. Clinic staff report that men in the community will approach male Aboriginal Health Practitioners (AHPs) and male nurses for ceremonial equipment as required.

Discussion

As part of the CQI process and aligning with the objectives of the STRIVE project,[4] RSHP staff have developed a tailored Sexual Health Action Plan with individual clinics. Improving condom access and availability was identified as a key goal for the majority of clinics. Each visit RSHP staff assesses how remote staff are progressing with this goal. RSHP staff use the clinic Action Plan as a tool to keep condom access on the agenda and reiterate 24 hour access as an important component for STI control. Despite this many clinics have made little progress with community consultation and facilitating permission to install dispensers within the community. Several shires were supplied with condom dispensers and informed that the cost of installation would be covered by RSHP but few installations had occurred. However some community representatives have agreed in principle to installation of dispensers in the near future.

A key factor for community reluctance to condom dispensers has been attributed to reported issues of vandalism and in 1 community reports that a metal dispenser was used as a weapon. Several clinic staff reported community dissatisfaction at the "condom rubbish" lying around. As a result many community representatives have refused to have dispensers re-installed. Clinicians stated that there is also a lack of community understanding around the need for condoms and have requested more education in schools and the community about sexual health and the consequences of STIs on fertility and neonatal complications.

Another challenge identified by clinic staff is ensuring dispensers are routinely stocked. Many clinics have a high staff turn-over and there is often no system in place to allocate restocking responsibility. In some of the larger communities where there are many dispensers, new staff are often not aware of the location of all the dispensers. Discussions with Primary Health Care managers, AHPs and Aboriginal Corporations around mapping of where clients can access condoms 24/7 are being undertaken. This will enable clinicians to inform their clients of condom availability.

The audit process has also been a useful tool for education and keeping condom accessibility on the agenda of clinics. It has inspired many managers to commit to discuss 24/7 access with the community and revisit the clinics condom supplies. Dispensers, condoms, lubricant, condom wallets and stickers have been sent out to many clinics as a result of requests for resources during the audit.

The rationale for a decrease in access points in the Tiwi's could be attributed to an initial overestimate of access points needed and the difficulty in sustaining routine restocking processes. Some have therefore been removed in 2015 to make it a more streamlined and easy process.

The objective of this audit was to assess condom access rather than usage. It is understood that simply having access to condoms does not necessarily translate to use. However 24/7 access is an important component for STI prevention and control and it is essential the RSHP help to facilitate access in remote communities due to the unacceptable burden of STIs. Once improved 24/7 access to condoms can be demonstrated it would be important to assess whether increased condom access leads to a sustained decrease in STI prevalence.

The RSHP continues to advocate for the importance of 24 hour access to condoms. As installing dispensers in communities has not always been possible or prompt the RSHP have been facilitating other ways that people can access condoms such as using night patrol, local youth organisations, men's and women's days, ceremonies and women's centres. The RSHP have also been trialling other approaches including glow-in-the-dark condom stickers which have been developed and distributed. Feedback from clinics on acceptability of stickers by community has been positive. A new condom wallet, which fits 2 condoms and a water-based lubricant, are currently available and being distributed through RSHP staff to clinics. Although not formally evaluated, anecdotally the condom wallets have had a positive response from clinic staff, AHPs and clients in the Top End remote and East Arnhem regions.

Conclusion

The SHBBV Unit remains committed to working towards decreasing the prevalence of STIs in the NT particularly in remote communities. The RSHP will continue to work with primary health staff and key stakeholders in NT remote communities to improve condom availability and increase the number of 24/7 condom access points. An annual condom audit will be undertaken to further assess access and availability in remote settings and monitor condom access trends.

Acknowledgments

Mark Russell, Helen Goodwin, Sarah Wyatt, Anne Davis, Natalie Newman, Kathy Shield, Annette Cotterill, Judy Burke, Shane Wing and Julie Scudder.

References

1. Northern Territory Department of Health. Sexual Health and Blood Borne Viruses Unit Surveillance Update. 2014; Vol. 15, No. 1.
2. Kirby Institute. HIV, viral hepatitis and sexually transmissible infections in Australia. Annual Surveillance Reports. 2014. The Kirby Institute for infection and immunity in society. The University of NSW. 2014.
3. Holt, J, Garton, L, Ryder, N. Auditing the Availability of condoms in Remote and Urban Settings in the Northern Territory. *NT Dis Control Bull.* 2014:21(4), 11-12.
4. Ward J, McGregor S, Guy RJ, et al. STI in remote communities: improved and enhanced primary health care (STRIVE) study protocol: a cluster randomised controlled trial comparing 'usual practice'STI care to enhanced care in remote primary health care services in Australia. *BMC Infect Dis* 2013;13:425.
5. Australian Government, Department of Health. Third National Sexually Transmissible Strategy 2014-2017.

****

Calling on all NT GPs and Primary Healthcare Nurses

Free Hepatitis B Vaccine for Indigenous Adults in the NT between 20 to 50 years of age.

A simple 2-step process:

Look up the immunisation status of Indigenous patients aged 20 to 50 years on your systems.
Offer the vaccine to those who are non-immune or never been vaccinated.

If you have any queries about the vaccine, please phone 08 8922 8315 or 08 8922 8044

To place an order, use this link to download the order form:
http://www.health.nt.gov.au/Centre_for_Disease_Control/Immunisation/Recording_and_Reporting_Forms/index.aspx#Vaccineorderform

To get the Hepatitis B Vaccination and Public Health Guidelines, view at:
http://www.health.nt.gov.au/Centre_for_Disease_Control/Publications/CDC_Protocols/index.aspx

Comment on the following article

Matthew Thalanany and Manoji Gunathilake, SHBBVU, CDC, Darwin

This focus on syphilis and congenital syphilis is important and timely in the context of the current syphilis outbreak in the NT.

The article that follows is a literature review undertaken by Dr Erana Gray and Dr Nathan Ryder, past clinicians in the Sexual Health Unit at the time the Congenital Syphilis Guidelines 2005 were to be reviewed and updated.

It is being presented as an example of how CDC Guidelines are updated, and illustrates the fact that guideline development is a process underpinned by the best evidence base available at the point in time. The literature review upheld the Guidelines, and added further points, principally focussing on management of syphilis in pregnancy and neonatal assessment.

As a result of this literature review, the NT Congenital Syphilis Guidelines have been updated to reflect the recommendations made in the review.

The updated 2015 Guidelines can be found at:

http://www.health.nt.gov.au/library/scripts/objectifyMedia.aspx?file=pdf/10/91.pdf&siteID=1&str_title=Congenital

This focus on syphilis and congenital syphilis is important and timely in the context of the current syphilis outbreak in the NT.

We urge all clinicians involved in the care of mothers and neonates to refer to these 2015 Guidelines.

****

Northern Territory Congenital Syphilis Guidelines 2015: a review of recommendations against major clinical guidelines and the literature

Erana Gray[1] and Nathan Ryder[2]
[1]Division of Medicine, Waikato Hospital, Hamilton, NZ
[2]Clinical Director, Newcastle Sexual Health Services
[1,2]CDC Sexual Health and Blood Borne Virus Unit Darwin

As part of the review and updating process of the Northern Territory Congenital Guidelines 2005, a systematic comparison was made between the recommendations in the NT with those contained in major local, national and international guidelines. Based on this comparison a series of clinical questions were identified and a targeted review of published literature was conducted to identify evidence regarding these questions. It must be noted that as there have been no high quality comparative trials conducted among pregnant women current recommendations are largely based on expert opinion from decades past, reinforced by case series data.[1]

1. First line treatment of syphilis in pregnancy

NT CDC Guideline recommendation (unchanged)

Syphilis in pregnancy should be treated with standard doses of benzathine penicillin:

- **Early syphilis (<2 years) 1.8 grams intramuscularly single dose**
- **Late/unknown duration 1.8 grams intramuscularly for 3 weekly doses**

Benzathine penicillin is listed as the only first line agent in the NT Guidelines, consistent with Australian Antibiotic guidelines,[2] US Centres for Disease Control[3] and World Health Organization (WHO)[4,5] guidelines. The British[6] and European[7] guidelines also list procaine penicillin as an option. Historically these guidelines advocated procaine penicillin rather than benzathine penicillin due to concerns raised at least 2 decades ago regarding the central nervous system (CNS) penetration of benzathine penicillin however the evidence currently supports the effectiveness of long acting benzathine penicillin in preventing vertical transmission and tertiary syphilis.[1] Additionally

local expert opinion suggests compliance with a 10 to 17 day course of daily intramuscular procaine penicillin is impractical in the NT context and it should not be a recommended alternative regimen.

A second dose of benazthine penicillin for women with early syphilis in the third trimester of pregnancy is recommended in the British guideline and listed as an option in the US CDC guideline, whereas the Australian, European and WHO guidelines recommend a single dose only. One case control[8] and 1 retrospective cohort study[9] found evidence of the failure of single dose benzathine penicillin to universally prevent congenital syphilis and an uncontrolled pharmacokinetic study[10] showed some women at term fail to reach the commonly accepted effective penicillin level. However 2 systematic reviews[1,11] concluded there was insufficient evidence to recommend a second dose of benzathine penicillin in pregnancy. Due to of lack of convincing evidence for better outcomes with a second dose of benzathine the NT Guidelines recommend treating pregnant women with a single dose consistent with non-pregnant people.

2. Role of non-penicillin regimens

NT CDC Guideline recommendation (unchanged)

All women with syphilis in pregnancy should receive treatment with penicillin; women who are allergic to penicillin undergo desensitisation.

The Australian, US and WHO guidelines recommend all women receive penicillin whereas British and European guidelines provide the alternatives of ceftriaxone, azithromycin and erythromycin.

Erythromycin does not reliably cross the placenta[12,13] and is associated with a greater risk of treatment failure.[14,15] Azithromycin was shown to be effective for treatment of early syphilis in 4 randomised studies and a subsequent meta-analysis,[16] however there have been many reports of clinical failures and high levels of both erythromycin and azithromycin resistance reported, including in a small number of samples obtained from remote Aboriginal communities.[17,18]

Use of ceftriaxone regimens have been described in very small case series reports in early and late syphilis, however there is very little evidence for use in pregnancy[19] and none in preventing congenital syphilis. Ceftriaxone treatment requires daily intramuscular injections and is contraindicated in severe penicillin allergy, the most common reason for avoiding penicillin. Due to these factors ceftriaxone is not listed as an alternative regimen in the NT Guideline.

3. Testing for HIV and effect on maternal and congenital syphilis

NT CDC Guideline recommendation (new)

All pregnant women should be tested for HIV.

HIV has no impact on management of syphilis.

The NT and Australian guidelines make no recommendation about HIV testing whereas all other guidelines recommend universal HIV testing of all people with syphilis. While there is no known association between HIV infection and syphilis among pregnant women in the NT in most populations women diagnosed with syphilis are at increased risk of HIV. Australian antenatal guidelines recommend universal HIV testing for all pregnant women.

The 2005 NT Guidelines make no mention of any impact of HIV infection on treatment of syphilis. Most guidelines specifically recommend testing all women but providing standard treatment and follow-up for syphilis. While the British guideline note a possible increased risk of vertical transmission in HIV positive women this is based on 2 papers reporting on a total of 3 cases.[20,21] As HIV infection is uncommon among women with syphilis in pregnancy in the NT no specific guidance is provided on this topic.

4. Maternal ultrasound monitoring

NT CDC Guideline recommendation (new)

Women who are treated for syphilis after 20 weeks of pregnancy should have foetal and placental ultrasound examination at least once.

Foetal morphology ultrasound scanning is recommended by US CDC, British and European guidelines. Indications are variable, either diagnosis after 20 weeks of pregnancy, or all women diagnosed with syphilis in pregnancy. There are very limited data to suggest a greater risk of treatment failure where foetal abnormalities are detectable,[22] additionally detection of foetal abnormalities prior to delivery allows closer follow-up by obstetrics team and counselling of pregnancy options including termination.

5. Management of maternal Jarisch-Herxheimer reaction

NT CDC Guideline recommendation (new)

Women treated for early syphilis should be advised to seek advice should any contractions or decrease in foetal movements occur.

Treatment of early syphilis can result in a Jarisch-Herxheimer reaction, an immune mediated response to treatment that may cause foetal distress, preterm labour or still birth. As there is no evidence for prevention or management for this reaction, and early detection has not been shown to improve outcomes, routine hospitalisation is not recommended excepting in the British guideline. Routine admission or monitoring of all pregnant women treated for syphilis is impractical in the NT and in the absence of any evidence of benefit cannot be recommended; however women should be advised to seek advice should any contractions or decrease in foetal movements occur.

6. Neonatal treatment criteria and regimen

NT CDC Guideline recommendation (unchanged)

Benzathine penicillin 37.5mg/kg intramuscularly as a single dose for all neonates at risk of congenital syphilis regardless of clinical assessment.

Benzyl penicillin 50mg/kg intravenously 12 hourly for 10 days for all neonates with proven or higher risk of congenital syphilis.

The Australian and European guidelines recommend treatment of neonates with clinical evidence of congenital syphilis following a thorough investigation of all infants born to seropositive mothers. The British guidelines also recommend investigation but additionally recommend presumptive treatment for high risk cases where the mother was not proven to have been treated greater than 4 weeks prior to delivery. The NT, WHO and US CDC guidelines all recommend a risk based assessment similar to the British guidelines but with the addition of a single prophylactic dose of benzathine penicillin to neonates at low risk infection. Low risk of infection is defined as adequate treatment given in the current pregnancy.

The NT Guidelines remain unchanged in the treatment recommendation for all potentially exposed neonates. While neonates whose mothers received adequate treatment during pregnancy are at very low risk of infection[23] treatment failures have been reported.[8,9] Reinfection is more likely in higher prevalence regions such as the NT, and thorough investigation and timely treatment of all neonates with signs of congenital syphilis is difficult in the NT context. Presumptive treatment with a single birth dose of benzathine penicillin is both justified and has evidence of effectiveness.[24,25] Similarly as many cases of congenital syphilis are asymptomatic at birth, and cerebrospinal fluid (CSF) examination on the neonate is both difficult to perform and interpret, all neonates at high risk of congenital syphilis should be treated with a regimen recommended for neurological involvement. While some guidelines allow daily intramuscular procaine penicillin, the NT Guidelines recommend admission for intravenous treatment on the basis of better CSF penetration and assured completion of treatment.

Australian and NT guidelines recommend 12 hourly dosing of IV penicillin whereas UK, US CDC and WHO guidelines recommend increased frequency after 7 days of age. European guidelines recommend 6 hourly dosing throughout the course. While studies have shown increases in clearance rates with post-natal age, other factors such as gestational age and weight also impact upon clearance.[26] Additionally as the effective dose is unknown it

is very difficult to determine whether these changes are clinically significant. In the absence of convincing contrary evidence the NT Guidelines neonatal dosing schedule remains consistent with the Australian antibiotic guidelines and local expert opinion.

7. Definition of prior adequate maternal treatment

NT CDC Guideline recommendation (new)

Adequate treatment of prior maternal syphilis requires completion of a penicillin regimen appropriate to stage of infection and an adequate serological response for the clinical situation.

The 2005 NT Guidelines explicitly defined adequate prior maternal treatment of a prior infection. The mother must have completed a course of penicillin appropriate to the stage of infection, have a documented serological treatment response and maintained an RPR titre that is both stable and below 1:8. These recommendations are closely based on the US CDC recommendations.

Up to 50% of those treated for syphilis will not respond serologically with the expected 2 titre fall with 1 year.[27,28] Retreatment only leads to a small improvement in this rate.[29] While meta-analysis found 1 study showing an increased risk of congenital syphilis where maternal RPR was greater than 1:8, this study included both treated and untreated women. There is no data to indicate that people with a stable RPR following appropriate treatment, and have no evidence of reinfection, are at risk of progression or transmission regardless of whether the RPR is high or low.

The revised 2015 NT Guideline retains the recommendation to retreat women without evidence of a serological response.

8. Role of neonatal CSF and long bone x-ray analysis

NT CDC Guideline recommendation (unchanged)

CSF and long bone x-ray evaluation should only be performed for specific clinical indications on advice from a paediatrician

Examination of CSF and long bone x-rays are recommended in all guidelines. US CDC recommends these tests for high risk neonates not given IV treatment. The UK guidelines recommend where there is laboratory evidence of congenital infection. The European and WHO guidelines provide no guidance on indications for these tests.

The primary purpose of CSF examination is to determine whether neurosyphilis is present and hence IV therapy is mandated. The interpretation of CSF findings are notoriously difficult and rarely changes management, with WCC and protein levels being non-specific, VDRL poorly sensitive and with decreasing availability, PCR is specific but with significant delays in obtaining results inmost regional and remote settings.[30]

Long bone x-rays may provide clinical evidence of congenital syphilis which is not otherwise apparent but are unlikely to alter the management of a case.

As the NT Guidelines continue to recommend IV therapy for all high risk or infected neonates both CSF and long bone x-rays should only be performed by a paediatrician in evaluating individual cases where the information may be clinically useful, particularly where the diagnosis is unclear. Unlike previous guidelines this decision is solely based on clinical utility rather than availability of expertise.

9. Role of neonatal IgM testing

NT CDC Guideline recommendation (unchanged)

Neonatal IgM testing should be considered for neonates at high risk of congenital infection where the diagnosis is in doubt.

The 2005 NT Guidelines previously made no mention of IgM assays. The British guidelines recommend IgM EIA and the European guidelines IgM by EIA, 19S FTA-abs or immunoblot. The US guidelines specifically recommend against all commercially available IgM assays. WHO guidelines do not make reference to IgM assays.

Neonatal IgM has been consistently shown to lack sensitivity and hence a negative IgM cannot

be used to rule out congenital infection. While older tests lack specificity data suggests some tests (EIA, 19S FTA-abs and immunoblot) are sufficiently specific to aid in the diagnosis.[31] However as doubts on the actual specificity remain it is not recommended IgM assays are used as a screening test. In the NT testing would require referral to an interstate laboratory and hence is unlikely to alter immediate clinical management and should not be used to guide immediate presumptive treatment.

10. Role of neonatal nucleic acid amplification (NAA) testing (new)

NT CDC Guideline recommendation

All high risk neonates and symptomatic neonates should have syphilis NAA tests on relevant clinical samples such as CSF, skin lesions, nasal secretions, etc.

Nucleic acid amplification (NAA) tests, such as PCR, have evolved significantly since the 2005 NT Guideline. The NT Guideline reserved NAA tests for use in specific cases on the advice of a paediatrician. The British and European guidelines consider positive NAA tests to be diagnostic of congenital syphilis but do no not give specific indications for testing. The US CDC and WHO do not refer to NAA tests at all in this context.

NAA tests can be performed on samples from clinical lesions, CSF, placental tissue and amniotic fluid. While sensitivity is highly variable specificity is considered to be very high.[32,33,34,35] However NAA tests are simple and minimally invasive tests with a low false positive rate that may aid in detecting definitive cases of congenital syphilis. Testing should be performed on appropriate clinical samples from all symptomatic neonates.

11. Follow-up schedule for neonates

NT Guideline recommendation (minor changes)

The NT and US CDC guidelines outline the follow-up schedule for neonates whereas all guidelines do not. The UK guidelines state that all infants should be jointly managed with a paediatrician, a situation impractical and unwarranted to be applied to all low risk neonates in the NT.

An internal audit in the NT found very few exposed infants received the recommended clinical and serological follow-up. The revised guideline retains essentially the same recommendations as the 2005 version. Minor changes made are to recommend high risk neonates receive paediatric review at 3 and 6 months, guidance on the management of infants with positive EIA but negative RPR (repeat at 15-18 months) and defining congenital syphilis as EIA positive at 15-18 months rather than 12, in-line with other guidelines.

References

1. Walker GJA. Antibiotics for syphilis diagnosed during pregnancy. *Cochrane database of systematic reviews* 2001, Issue 3. Art. No.: CD001143. DOI: 10.1002/14651858.CD001143.
2. Therapeutic Guidelines, Antibiotic version 15, 2014, http://www.tg.org.au/index.php?sectionid=41.
3. Centres for Disease Control and Prevention, Sexually Transmitted Diseases Treatment Guidelines. MMWR 2010;59. http://www.cdc.gov/std/treatment/2010/STD-Treatment-2010-RR5912.pdf#page=38.
4. World Health Organization. Guidelines for the Management of Sexually Transmitted Infections, 2003, http://www.who.int/hiv/pub/sti/en/STIGuidelines2003.pdf.
5. World Health Organization. Management of Sexually Transmitted Infections –regional guidelines. 2011.
6. Kingston M, French P, Goh B, Goold P, Higgins S, Sukthankar A et al. UK National Guidelines on the Management of Syphilis. DOI: 10.1258/ijsa.2008.008279. *Int J STD AIDS* 2008; 19: 729 –740.
7. French P, Gomberg M, Janier M, Schmidt B, van Voorst Vader P, Young H et al. European Guidelines on the Management of Syphilis. *Int J STD AIDS* 2009; May 20(5):300-9. doi: 10.1258/ijsa.2008.008510.
8. Sheffield S, Sanchez P, Morris G, Mabery M, Zeray F, McIntyre D et al. Congenital syphilis after maternal treatment for syphilis during pregnancy. *Am J Obstet Gynecol* 2002; 186(3): 569-573.
9. Donders G, Desmyter J, Hooft P, Dewet G. Apparent Failure of One Injection of Benzathine Penicillin G for Syphilis During Pregnancy in Human Immunodeficiency Virus-Seronegative African Women. *Sex Transm Dis* 1997; 24(2): 94-101.

10. Nathan L, Bawdon R, Sidawi E, Stettler W, McIntire D, Wendel G. Penicillin levels following the administration of benzathine penicillin G in pregnancy. *Obstet Gynecol* 1993; 82(3): 338-342.
11. Wendel G, Sheffield J, Hollier L, Hill J, Ramsey G, Sanchez P et al. Treatment of syphilis in pregnancy and prevention of congenital syphilis. *Clin Infect Dis* 2002; 35 Supp 2: S200-S209.
12. Kiefer L, Rubin A, McCoy J, Foltz E. The placental transfer of erythomycin. *Am J Obstet Gynecol* 1955; Jan;69(1):174-7.
13. Philipson A, Sabath L, Charles D. Transplacental passage of erythromycin and clindamycin. *N Engl J Med* 1973; 288: 1219–1221.
14. Fenton L, Light I. Congenital syphilis after maternal treatment with erythromycin. *Obstet Gynecol* 1976;47:492–4.
15. South M, Short D, Knox J. Failure of erythromycin estolate therapy in utero syphilis. *JAMA* 1964;190:70–1.
16. Bai Z, Yang K, Liu Y, Tian J, Ma B, MI D et al. Azithromycin vs. benzathine penicillin G for early syphilis: a meta-analysis of randomized clinical trials. *Int J STD AIDS* 2008; 19(4): 217-221.
17. Read P, Jeoffreys N, Tagg K, Guy R, Gilbert G, Donovan B. Azithromycin-resistant syphilis-causing strains in Sydney, Australia: prevalence and risk factors. *J Clin Microbiol* 2014;52(8): 2776-2781.
18. Whiley, D. Personal communication 2012.
19. Zhou P, Gu Z, Xu J, Wang X, Liao K. A study evaluating ceftriaxone as a treatment agent for primary and secondary syphilis in pregnancy. *Sex Transm Dis* 2005; 32(8): 495-498.
20. McFarlin B, Bottoms S, Dock B, Isada N. Epidemic syphilis: maternal factors associated with congenital infection. *Am J Obstet Gynecol* 1994; 170:535–40.
21. Rawstron S, Bromberg K. Failure of recommended maternal therapy to prevent congenital syphilis. *Sex Transm Dis* 1991;18:102–6.
22. Hollier L, Harstad T, Sanchez P, Twickler D, Wendel G. Fetal syphilis: clinical and laboratory characteristics. *Obstet Gynecol* 2001;97:947–53.
23. Watson-Jones D, Gumodoka B, Weiss H, Changalucha J, Todd J, Mugeye K et al. Syphillis in pregnancy in Tanzania. 11. The effectiveness of antenatal syphilis screening and single does benzathine penicillin treatment for the prevention of adverse pregnancy outcomes. *J Infect Dis* 2002;186(7):948-57.
24. Paryani S, Vaughn A, Crosby M, Lawrence S. Treatment of asymptomatic congenital syphilis: benzathine versus procaine penicillin G therapy. *J Pediatr* 1994;125(3): 471-475.
25. Radcliffe M., Meyer M, Roditi D, Malan A. Single-dose benzathine penicillin in infants at risk of congenital syphilis: results of a randomised study. *S Afr Med J* 1997;87; 62-64.
26. Pacifici G, Labatia J, Mulla H, Choonara I. Clinical pharmacokinetics of penicillins in the neonate: a review of the literature. *Eur J Clin Pharmacol* 2009; 65(2): 191-198.
27. Fiumara N. Serologic responses to treatment of 128 patients with late latent syphilis. *Sex Transm Dis* 1979;6(4): 243-246.
28. Knaute D, Graf N, Lautenschlager S, Weber R, Bosshard P. Serological response to treatment of syphilis according to disease stage and HIV status. *Clin Infect Dis* 2012;55(12): 1615-1622.
29. Seña A, Wolff M, Behets F, Van Damme K, Martin D, Leone P et al. Response to therapy following retreatment of serofast early syphilis patients with benzathine penicillin. *Clin Infect Dis* 2013;56(3): 420-422.
30. Talati A, Padmaja K. Neonates at risk for congenital syphilis: radiographic and cerebrospinal fluid evaluations. *South Med J* 2011;104(12): 827-30.
31. Herremans T, Kortbeek L, Notermans D. A review of diagnostic tests for congenital syphilis in newborns. *Eur J Clin Microbiol Infect Dis* 2010;29(5): 495-501.
32. Grimprel E, Sanchez P, Wendel G, Berstain J, McKracken G, Radolf J et al. Use of polymerase chain reaction and rabbit infectivity testing to detect Treponema pallidum in amniotic fluid, fetal and neonatal sera, and cerebrospinal fluid. *J Clin Microbiol* 1991;29(8): 1711-1718.
33. Sánchez P, Wendel G, Grimpel E, Goldberg M, Hall M, Mireles O et al. Evaluation of molecular methodologies and rabbit infectivity testing for the diagnosis of congenital syphilis and neonatal central nervous system invasion by Treponema pallidum. *J Infect Dis* 1993;167(1): 148-157.
34. Gayet-Ageron A, Lautenschlager S, Ninet B, Perneger T, Combescure C. Sensitivity, specificity and likelihood ratios of PCR in the diagnosis of syphilis: a systematic review and meta-analysis. *Sex Transm Infect* (2012): 89(3) 251-256.
35. Grange P. Evaluation of a PCR test for the detection of Treponema pallidum in swabs and blood. *J Clin Microbiol* 2012: JCM-00702.

****

END TB — World TB Day March 24

Kristen Overton and Vicki Krause, CDC, Darwin

World TB Day, on March 24th provides the opportunity to raise public awareness about tuberculosis (TB). It marks the day in 1882 when Dr Robert Koch announced to the scientific community that he had discovered the cause of tuberculosis, the bacterium, *Mycobacterium tuberculosis*. Koch's discovery opened the way to diagnosing and curing TB.

TB however remains a significant global health problem and worldwide each year there are approximately 9.5 million new TB cases and 1.5 million deaths from this treatable and preventable disease. In Australia 1200 to 1300 new cases of active TB are detected each year, including 25 to 35 cases in Territorians. It is important to recognise that TB can develop many years after one has been exposed, usually when the body's defenses are weakened. It is estimated that a third of the world's population is infected with *M. tuberculosis* with a potential to progress to active TB during their lifetime.

In May 2014, at the World Health Assembly governments agreed on an ambitious new 20-year (2016-2035) strategy. The 'End TB Strategy' envisions a world free of TB with zero deaths, disease and suffering. It sets targets and outlines actions for governments and partners to carry out to end the epidemic and eliminate TB.

The NT Department of Health's Centre for Disease Control (CDC) is committed to TB control efforts that will work to end TB. These include screening for those at risk of TB, contact tracing, preventive treatment for potential TB cases, observed treatment for active TB cases and supporting high standard laboratory work on disease identification and drug testing.

This year's World TB Day theme 'Unite to end TB' highlights that TB is both a curable and preventable disease. The first step is to detect active TB disease and cure it but also to identify latent TB infection (LTBI) which has the potential to go on to active TB disease and treat it effectively as a preventive measure. At a local level, the NT CDC with the help of Casuarina and Palmerston Community Care Centres, are encouraging all people who have been born or lived in areas where TB is common such as in countries in Asia, the Pacific Islands and in Africa or those with a known TB exposure, to have the Mantoux skin test* to check if they have LTBI. If LTBI is found and treated the risk of progressing from latent infection to active TB at some later date is markedly reduced.

While progress has been made in TB control it still remains a vast worldwide problem and ongoing commitment is needed in Australia and elsewhere to identify those at greatest risk of developing the disease through screening and education so they can receive preventative treatment. More information on TB can be found at
http://www.health.nt.gov.au/Centre_for_Disease_Control/Publications/CDC_Factsheets/index.aspx

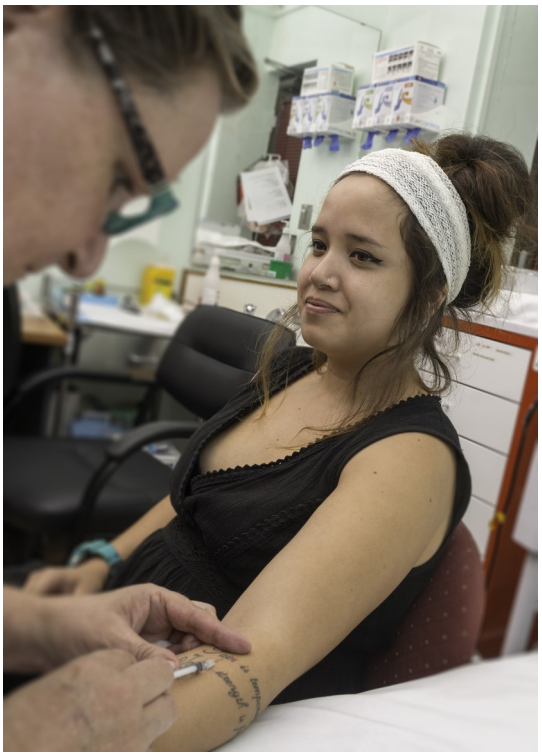


Photo: Jazmine Srakaew-Stephenson receiving a Mantoux test

*A Mantoux skin test is a simple and safe test — a small amount of tuberculin purified protein derivative is injected just under the top layer of skin on a person's arm using a small sterile needle and syringe. The skin is examined 2 to 3 days later and if a reaction (lump) is present it is measured and recorded.

****

Fish for dinner? Some fish is good for you, just not too much fish

Steven Skov, Community Physician, CDC, Darwin

Fish is a popular food in the Northern Territory (NT). Aboriginal people in the Top End have eaten fish and other aquatic foods for millennia as a key source of protein and calories. Fishing is extremely popular in all cultural groups in the NT and many people eat fish regularly.

Fish is generally perceived as being good food: good for cardiovascular health and for neurological development ('brain food'). However, fish often contains whatever contaminants are in the water or the sediments in the area in which they live. The most common and well known of these around the world is methylmercury. Methylmercury is toxic to humans and most notably can cause neurological disease. The developing foetus is the most at risk of developing neurological harms.

For this reason, fish consumption needs to be balanced. There is a line to be drawn between eating some fish to gain the benefits of fish without eating too much to cause possible problems from the methylmercury.[1]

Methylmercury is ubiquitous in fish. This subject is very well studied and the world scientific literature describes the presence of methylmercury in fish from every corner of the globe. The great majority of the methylmercury present in the environment is as a result of the normal breakdown of minerals in rocks and soils, volcanic eruptions and smoke from natural fires over the eons. However, since the industrial age, human sources, particularly from smoke from fossil fuel combustion and fires of human origin have become important sources as are industrial sources such as mining.[2]

Methylmercury can be found in virtually all species of fish, although some species are known to have higher levels. In general, methyl mercury levels are higher in larger, older predatory fish as well as other species that are long-lived. As a predatory fish eats other smaller fish, mercury levels gradually build up over time. In Australia, sharks, swordfish, marlin, the larger species of tuna and orange roughy are some of the species known to have higher levels. Other fish such as atlantic salmon, snapper, whiting and yellowfin tuna (usually used in tinned tuna) tend to have lower levels.

In Australia, Food Standards Australia New Zealand (FSANZ) has issued guidelines for safe fish consumption based on the presence of methylmercury that apply Australia wide.[3] In brief, the recommendation is for 300-450 grams, or 2-3 serves of 150 grams each, per week, with half that amount for children up to 6 years of age. This applies for most species of fish with less frequent consumption of fish known to have higher concentrations of methylmercury particularly for pregnant women or women likely to become pregnant. The FSANZ guidelines are also in line with the National Heart Foundation recommendations[4] to consume 2-3 serves of fish per week.

As for fish elsewhere, fish in the NT also contain methylmercury. A study from the 1980s showed that many sharks in Top End waters had high levels of methylmercury.[5] In 2012, fish, including barramundi, were tested for a range of metals in the Edith and Daly Rivers following a train derailment and release of water from a tailings dam. Mercury was not a metal considered to be a contaminant from these events, but testing revealed it was present at levels such that the FSANZ guidelines are appropriate advice for the NT.[6] The same was found recently in the review conducted into metals contamination in the McArthur River region.[7] The Department of Primary Industries and Fisheries (DPIF) has been testing barramundi across the NT for methylmercury in recent times also.[8] Analysis of the preliminary data from DPIF again shows that while methylmercury is present in NT barramundi, the FSANZ guidelines do provide appropriate advice for consumption.

What does this mean for Territorians? If a person was regularly eating substantially more fish than recommended by the FSANZ guidelines over a prolonged period, then there might be an increased risk of illness. This would most likely take the form of neurological symptoms such as tingling or abnormal sensations in the skin, poor memory or difficulty

with thinking. In children, this might manifest also in behavioural or school difficulties. There might also be some deterioration in kidney function as measured by laboratory tests. While rare, such cases can occur in people who have been eating significant amounts of fish on a daily or near daily basis over a prolonged period of at least many months.

In summary, there is methylmercury in NT fish and the FSANZ guidelines for safe fish consumption are relevant to the NT. The guidelines have been constructed with large safety margins built in so a person will not be at risk if they regularly eat a bit more than the guidelines or on occasion eat a lot more. People can and should still eat fish: it is a good source of protein and important nutrients. Eating some fish is good, just not too much fish.

Acknowledgement

Thanks to Ms Janis Baines of Food Standards Australia New Zealand for her helpful comments on an early draft of this paper.

References

1. FAO/WHO (2011). Report of the Joint FAO/ WHO Expert Consultation on the Risks and Benefits of Fish Consumption. Rome, Food and Agriculture Organization of the United Nations; Geneva, World Health Organization, Available at http://www.fao.org/docrep/014/ba0136e/ ba0136e00.pdf.
2. ATSDR (1999) Toxicological profile for mercury. US Department of Health and Human Services, Public Health Service Agency for Toxic Substances and Disease Registry, Atlanta, Georgia. Available at: http: //www.atsdr.cdc.gov/ toxprofiles/tp.asp?id+115&tid=24.
3. Food Standards Australia and New Zealand. Mercury in fish; Advice on fish consumption, 2004. Available at http:// www.foodstandards.gov.au/consumer/chemicals/ mercury/Pages/default.aspx.
4. National Heart Foundation, 2015, Healthy Hearts Position Statement. Fish and Seafood. Available at: https://heartfoundation.org.au/images/uploads/ main/Programs/PRO-169_Fish_and_seafood_position_statement.pdf.
5. Lyle JM. Mercury in shark from Northern Territory waters. Vol 2, Fishery Report no 12, Dept of Primary Production, Northern Territory, April 1984. Available at http://www.nt.gov.au/d/ Content/File/p/Fish_Rep/FR12_2.pdf.
6. Northern Territory Department of Health 2013. Summary and analysis of risk assessments relating to the testing of trace elements in fish from the Edith and Daly Rivers. Available at http://www.nt.gov.au/d/Minerals_Energy/Content/ File/Media/Overview-document.pdf.
7. Hydrobiology. 2016. Bioaccumulation of heavy metals in McArthur River and tributaries. Investigation and assessment of potential impacts. Study conducted for the NT Department of Mines and Energy. Available at http://www.nt.gov.au/d/ Minerals_Energy/Content/File/news/MRM-biaccumulation-tributaries-Jan2016.pdf.
8. NT Department of Primary Industries and Fisheries. 2016 Analysis of bioaccumulation of mercury in Barramundi. Unpublished preliminary report.

****

NT malaria notifications October—December 2015

Liz Stephenson, CDC, Darwin

There were 3 confirmed cases of malaria notified in the 4th quarter of 2015. The following table provides details about where the infection was thought to be acquired, the infecting agent, whether chemoprophylaxis was used and where the patient lived.

No. cases	Origin of Infection	Reason Exposed	Agent	Chemoprophylaxis	NT Region
1	Republic of Cameroon	Expatriate visiting relatives	*P. falciparum*	No	Katherine
2	Rabaul PNG	Recreation	*P. falciparum*	No	Darwin

****

Campaign to increase awareness of Murray Valley encephalitis in remote communities across the Northern Territory, Australia

Nina Kurucz, Medical Entomology, CDC, Darwin

Background

Murray Valley encephalitis (MVE) is a potentially fatal disease, transmitted by the common banded mosquito, *Culex anulirostris.*[1,2,3] Symptoms of MVE include headache, fever, nausea, vomiting and muscle aches, which can progress to drowsiness, confusion and seizure and in severe cases to delirium and coma.[4] In the Northern Territory (NT), the high risk period for MVE is February to July, with a total of 36 MVE cases reported since recording began in 1974. A high proportion (46.7%) of these cases, with the age of the patient determined, are reported in young Indigenous children under the age of 10.

In the NT, MVE virus activity is monitored through the Department of Health's (DoH) flavi virus surveillance program. If virus activity is detected, a media warning is issued to warn the public of potential MVE risks. These warnings are issued in the form of newspaper articles, radio and TV interviews. However, it is unknown how effective warnings, delivered in the English language are to prevent potential MVE cases in remote Indigenous communities.

MVE radio campaign 2016

In an effort to reduce the incidence of MVE in remote communities across the NT, DoH launched a radio campaign, with MVE risk messages delivered in 8 Indigenous languages. Messages in Yolgnu Matha, Murrinth Patha, Anindilyakwa, Modern Tiwi and Kriol were initially produced by the DoH Environmental Health unit to warn people in remote Top End communities about mosquito borne disease following Cyclone Lam in 2015. To cover Central Australian communities, DoH further produced messages in Arrente, Pitjantjatjara and Warlpiri. All messages were produced in liaison with the Aboriginal Interpreter Service, and were delivered by the Australian Indigenous Radio, Yolngu Radio and CAAMA Radio between 15 February and 12 March 2016.

To further increase coverage, the English version of the message was advertised on Facebook. The MVE Facebook advertising was marketed to the target audience using Facebook's Custom Audience Targeting. The Custom Audience used were people living in the NT, at the age of 18 to over 65 years, with an interest in Indigenous related themes. Using this method, a potential 21,000 people were reached. The Facebook advertising can be found on the NT Government YouTube channel using the following links;

https://www.youtube.com/watch?v=p47B_a8FHpk (Yolgnu Matha version)

https://www.youtube.com/watch?v=62G1BsKLpUk (English version).

References

1. Whelan PI and Weir R. The isolation of alpha and flavi viruses from mosquitoes in the Northern Territory 1982 -1992. *Arbovirus Research Australia*. 1993; 6:270-78.
2. Russell RC. Vectors vs. humans in Australia- who is on top down under? An update on vector-borne disease and research of vectors in Australia. *J Vector Ecol.* 1998; 23:1-46.
3. Russell RC and Dwyer D. Arboviruses associated with human disease in Australia. *Microbes Infect.* 2000; 2:1693-1704.
4. Centre for Disease Control Murray Valley encephalitis Fact Sheet 2013.

****

Abstracts from peer reviewed published articles related to the Northern Territory

Invasive *Staphylococcus aureus* infections in children in tropical northern Australia

Engelman D, Hofer A, Davis J, Carapetis J, Baird R, Giffard P, Holt D, Tong S

J Pediatric Infect Dis Soc 2014 Dec; Vol. 3 (4), pp. 304-11.

Background: Despite a high burden of staphylococcal skin disease in children and high incidence of *Staphylococcus aureus* bacteremia in adult Indigenous populations in northern Australia, there are few studies describing incidence or clinical information of invasive *S aureus* (ISA) infections in children.

Methods: We conducted a retrospective review for all cases of *S aureus* bacteremia and sterile site infections, for children under 15 years, in northern Australia over a 4-year period (2007-2010). Cases were categorized as neonatal (<28 days) and pediatric (≥28 days).

Results: Forty-four cases (9 neonatal, 35 pediatric) were identified. The annual incidence of ISA was 27.9 cases per 100 000 population. Among pediatric cases, the annual incidence was significantly higher in the Indigenous (46.6) compared with the non-Indigenous (4.4) population (IRR: 10.6 [95% confidence interval, 3.8-41.4]). Pediatric infections were predominantly community-associated (86%). Clinical infection sites included osteoarticular (66%), pleuropulmonary (29%), and endocarditis (9%), and multifocal disease was common (20%). Eighty-three percent of pediatric cases presented with sepsis; 34% resulted in intensive care admission. Neonatal cases were all born prematurely; 89% were late-onset infections. Overall, 27% of infections were due to methicillin-resistant *S aureus* (MRSA). Compared with methicillin-sensitive *S aureus* (MSSA), there was no difference in severity or presentation in pediatric MRSA cases, but a higher proportion of MRSA cases were readmitted.

Conclusions: The annual incidence of ISA infection in this study is among the highest described, largely due to a disproportionate burden in Indigenous children. Infections are frequently severe and infection with MRSA is common. Children presenting with suspected ISA in this region should be treated empirically for MRSA.

Staphylococcus aureus infections: Epidemiology, pathophysiology, clinical manifestations, and management

Tong S, Davis, J, Eichenberger E, Holland T, Fowler V

Clin. Microbiol. Rev. July 2015 28 (3), pp. 603-661.

Summary: *Staphylococcus aureus* is a major human pathogen that causes a wide range of clinical infections. It is a leading cause of bacteremia and infective endocarditis as well as osteoarticular, skin and soft tissue, pleuropulmonary, and device-related infections. This review comprehensively covers the epidemiology, pathophysiology, clinical manifestations, and management of each of these clinical entities. The past 2 decades have witnessed 2 clear shifts in the epidemiology of *S. aureus* infections: first, a growing number of health care-associated infections, particularly seen in infective endocarditis and prosthetic device infections, and second, an epidemic of community-associated skin and soft tissue infections driven by strains with certain virulence factors and resistance to β-lactam antibiotics. In reviewing the literature to support management strategies for these clinical manifestations, we also highlight the paucity of high-quality evidence for many key clinical questions.

Community and clinic-based screening for curable sexually transmissible infections in a high prevalence setting in Australia: a retrospective longitudinal analysis of clinical service data from 2006 to 2009

Silver B, Kaldor JM, Rumbold A, Ward J, Smith K, Dyda A, Ryder N, Yip TW, Su JY, Guy RJ

Sex Health. 2015 Dec 18. doi: 10.1071/ SH15077

Background: In response to the high prevalence of sexually transmissible infections (STIs) in many central Australian Aboriginal communities, a community-wide screening program was implemented to supplement routine primary health care (PHC) clinic testing. The uptake and outcomes of these 2 approaches were compared.

Methods: Chlamydia trachomatis (CT) and Neisseria gonorrhoeae (NG) community and clinic screening data for Aboriginal people aged 15-34 years, 2006-2009, were used. Regression analyses assessed predictors of the first test occurring in the community screen, positivity and repeat testing.

Results: A total of 2792 individuals had 9402 tests (median: 4 per person) over 4 years. Approximately half of the individuals (54%) were tested in the community and clinic approaches combined, 29% (n = 806) in the community screen only and 18% (n = 490) in the clinic only. Having the first test in a community screen was associated with being male and being aged 15-19 years. There was no difference between community and clinic approaches in CT or NG positivity at first test. More than half (55%) of individuals had a repeat test within 2-15 months and of these, 52% accessed different approaches at each test. The only independent predictor of repeat testing was being 15-19 years.

Conclusions: STI screening is an important PHC activity and the findings highlight the need for further support for clinics to reach young people. The community screen approach was shown to be a useful complementary approach; however, cost and sustainability need to be considered.

Chlamydia trachomatis genotypes in a cross-sectional study of urogenital samples from remote Northern and Central Australia

Giffard P, Brenner N, Tabrizi S, Garland S, Holt D, Andersson P, Lilliebridge R, Tong S, Karimi M, Boylan P, Ryder N, Johns T, Singh G

BMJ Open 2016;6:e009624 doi:10.1136/ bmjopen-2015-009624

Objectives: The objective was to determine the frequency of trachoma genotypes of *Chlamydia trachomatis*-positive urogenital tract (UGT) specimens from remote areas of the Australian Northern Territory (NT).

Setting: The setting was analysis of remnants of *C. trachomatis* positive primarily UGT specimens obtained in the course of clinical practice. The specimens were obtained from two pathology service providers.

Participants: From 3356 *C. trachomatis* specimens collected during May 2012–April 2013, 439 were selected for genotyping, with a focus on specimens from postpubescent patients, in remote Aboriginal communities where ocular trachoma is potentially present.

Primary and secondary outcome measures: The primary outcome measure was the proportion of successfully genotyped UGT specimens that were trachoma genotypes. The secondary outcome measures were the distribution of genotypes, and the frequencies of different classes of specimens able to be genotyped.

Results: Zero of 217 successfully genotyped UGT specimens yielded trachoma genotypes (95% CI for frequency=0–0.017). For UGT specimens, the genotypes were E (41%), F (22%), D (21%) and K (7%), with J, H and G and mixed genotypes each at 1–4%. Four of the five genotyped eye swabs yielded trachoma genotype Ba, and the other genotype J. Two hundred twenty-two specimens (50.6%) were successfully genotyped. Urine specimens were less likely to be typable than vaginal swabs ($p<0.0001$).

Conclusions: Unlike in some other studies, in the remote NT, trachoma genotypes of

C. trachomatis were not found circulating in UGT specimens from 2012 to 2013. Therefore, *C. trachomatis* genotypes in UGT specimens from young children can be informative as to whether the organism has been acquired through sexual contact. We suggest inclusion of *C. trachomatis* genotyping in guidelines examining the source of sexually transmitted infections in young children in areas where trachoma genotypes may continue to circulate, and continued surveillance of UGT *C. trachomatis* genotypes.

Notification and management of congenital syphilis in the Northern Territory 2009 to 2014

McLeod C, Su JY, Francis JR, Ishwar A, Ryder N

Commun Dis Intell Q Rep. 2015 Sep 30;39 (3):E323-8

Objective: To determine whether cases of congenital syphilis in the Northern Territory between 2009 and 2014 were correctly notified based on probable or confirmed case criteria stipulated by the Communicable Diseases Network Australia (CDNA).

Methods: Pregnant women with positive syphilis serology defined as reactive treponemal test and rapid plasma reagin titre ≥1:8 were identified from the Northern Territory Syphilis Register Information System. Risk classification was performed based on local guidelines, and CDNA criteria for probable/confirmed cases of congenital syphilis were applied to determine whether cases were appropriately notified.

Results: Thirty-four cases of positive maternal syphilis serology in pregnancy were identified from 31 women; all were Indigenous. Twenty-one cases fulfilled criteria for probable congenital syphilis; 1 case was formally notified to the Centre for Disease Control. Twenty cases (95%) fulfilling CDNA criteria for probable congenital syphilis were not notified over the study period.

Conclusions: Application of standard case definitions significantly increases the rate of congenital syphilis cases in the Northern Territory. Improved education regarding CDNA criteria for notification of congenital syphilis is necessary for clinicians and public health staff. Emerging evidence has supported the recent simplification of CDNA criteria for notification of congenital syphilis, effective 1 July 2015.

Editorial

This article demonstrates the difficulty in defining and monitoring cases of congenital syphilis.

The surveillance case definition for congenital syphilis at the time of the audit was complex, and relied heavily on the changes to RPR titres (non-treponemal serology test) following treatment in antenates to exclude 'probable' congenital syphilis in the newborn.

Following the audit, each of the qualifying cases was reviewed and detailed follow up revealed that only 2 newborns were qualified as 'probable congenital syphilis', while the others were considered either very low risk or not cases. Further follow up of the 2 probable cases revealed negative serology suggesting that there was no congenital infection. Partly as a result of this study, the case definition was reviewed and, consistent with other congenital syphilis case definitions internationally, the requirement for serological proof of maternal treatment response was excluded.

Large-scale comparative genomics identifies unprecedented melioidosis cases in northern Australia caused by an Asian *Burkholderia pseudomallei* strain

Price E, Sarovich D, Smith E, MacHunter B, Harrington G, Theobald V, Hall C, Hornstra H, McRobb E, Podin Y, Mayo M, Sahl J, Wagner D, Keim P, Kaestli M, Currie B

Appl Environ Microbiol, 2015, DOI: 10.1128/ AEM.03013-15

Melioidosis is a disease of humans and animals that is caused by the saprophytic bacterium *Burkholderia pseudomallei*. Once thought to be confined to limited locations, the known presence of *B. pseudomallei* is expanding as more regions of endemicity are uncovered.

There is no vaccine for melioidosis, and mortality remains as high as 40% in some endemic regions, even with antibiotic administration. Despite high levels of recombination, phylogenetic reconstruction of *B. pseudomallei* populations using whole-genome sequencing (WGS) has revealed surprisingly robust biogeographic separation between isolates from Australia and Asia. To date, there have been no confirmed autochthonous melioidosis cases in Australia caused by an Asian isolate; likewise, no autochthonous cases in Asia have been identified as Australian in origin. Here, we used comparative genomic analysis of 455 *B. pseudomallei* genomes to confirm the unprecedented presence of an Asian clone, sequence type (ST) 562, in Darwin, northern Australia. First observed in 2005, the incidence of melioidosis cases attributable to ST-562 infection has steadily risen, and it is now a common Darwin strain. Intriguingly, Australian ST-562 appears to be geographically restricted to a single locale and is genetically less diverse than other common STs from this region, indicating a recent introduction of this clone into northern Australia. Detailed genomic and epidemiological investigation of new clinical and environmental *B. pseudomallei* isolates in the Darwin region, and ST-562 isolates from Asia, will be critical for understanding the origin, distribution and dissemination of this emerging clone in northern Australia.

Epidemiological investigation of an outbreak of cutaneous sporotrichosis, Northern Territory, Australia

McGuinness S, Boyd R, Kidd S, McLeod C, Krause V, Ralph A

BMC Infectious Diseases (2016) 16:16 DOI 10.1186/s12879-016-1338-0

Background: An outbreak of cutaneous sporotrichosis occurred in the Darwin region of the Northern Territory (NT) in 2014. We aimed to determine the source and risk factors associated with the outbreak and describe the clinical spectrum of cases seen.

Methods: Epidemiological investigation of cases of cutaneous sporotrichosis identified through the Royal Darwin Hospital was undertaken to investigate risk factors and potential sources of infection. Data were collected through chart review and individual patient interviews. Environmental investigation followed identification of a common risk factor.

Results: Nine confirmed cases of cutaneous sporotrichosis caused by *Sporothrix schenckii* were identified with onset of symptoms between April and July 2014. Patients were aged 29 to 70 years and seven were male (78 %). Two strains of *S. schenckii* were identified, neither of which have been previously documented. One common risk factor was identified: all patients were occupational or recreational gardeners, with each reporting exposure to mulching hay, originating from a single NT farm. Local environmental health officers visited the farm and the owners confirmed that the implicated hay had been stored over the monsoon season and had been affected by rain. Storage of hay over the wet season was a new practice.

Conclusions: This constitutes the third reported outbreak of *S. schenckii* sporotrichosis attributable to contaminated hay in Australia and the first outbreak of sporotrichosis in the NT. This outbreak prompted public health interventions, including distribution of information to general practitioners, farmers and suppliers in the Top End. Media reporting led to the identification and treatment of an additional case. Local practitioners should remain alert to the possibility of further occurrences of sporotrichosis.

A cluster-randomised controlled trial integrating a community-based water, sanitation and hygiene programme, with mass distribution of albendazole to reduce intestinal parasites in Timor-Leste: the WASH for WORMS research protocol

Nery S, McCarthy J, Traub R, Andrews R, Black J, Gray D, Weking E, Atkinson J, Campbell S, Francis N, Vallely A, Williams G, Clements A

BMJ Open. 2015 Dec 30;5(12):e009293. doi: 10.1136/bmjopen-2015-009293

Introduction: There is limited evidence demonstrating the benefits of community-based water, sanitation and hygiene (WASH)

programmes on infections with soil-transmitted helminths (STH) and intestinal protozoa. Our study aims to contribute to that evidence base by investigating the effectiveness of combining 2 complementary approaches for control of STH: periodic mass administration of albendazole, and delivery of a community-based WASH programme.

Methods and analysis: WASH for WORMS is a cluster-randomised controlled trial to test the hypothesis that a community-based WASH intervention integrated with periodic mass distribution of albendazole will be more effective in reducing infections with STH and protozoa than mass deworming alone. All 18 participating rural communities in Timor-Leste receive mass chemotherapy every 6 months. Half the communities also receive the community-based WASH programme. Primary outcomes are the cumulative incidence of infection with STH. Secondary outcomes include the prevalence of protozoa; intensity of infection with STH; as well as morbidity indicators (anaemia, stunting and wasting). Each of the trial outcomes will be compared between control and intervention communities. End points will be measured 2 years after the first albendazole distribution; and midpoints are measured at 6 months intervals (12 months for haemoglobin and anthropometric indexes). Mixed-methods research will also be conducted in order to identify barriers and enablers associated with the acceptability and uptake of the WASH programme.

Ethics and dissemination: Ethics approval was obtained from the human ethics committees at the University of Queensland, Australian National University, Timorese Ministry of Health, and University of Melbourne. The results of the trial will be published in peer-reviewed journals presented at national and international conferences, and disseminated to relevant stakeholders in health and WASH programmes. This study is funded by a Partnership for Better Health-Project grant from the National Health and Research Council (NHMRC), Australia.

The epidemiology of tuberculosis in children in Australia, 2003–2012

Teo S, Tay E, Douglas P, Krause V, Graham S

Med J Aust 2015; 203 (11): 440. doi: 10.5694/mja15.00717

Objective: To describe the burden of and trends in paediatric tuberculosis (TB) in Australia between 2003 and 2012.

Design: A retrospective analysis of TB data from the National Notifiable Diseases Surveillance System (NNDSS) on TB in children (under 15 years of age) during the 10–year period, 2003–2012.

Results: TB notifications in Australia during the study period included 538 children (range, 37–66 cases per year), representing 4.6% of the total TB case load during the period (range, 3.8%–5.8% each year). Place of birth was recorded for 524 patients (97.4%); of these, 230 (43.9%) were born in Australia, 294 (56.1%) overseas. The average annual notification rate was 1.31 (95% CI, 1.20–1.43) cases per 100 000 child population. The rate was higher for overseas-born than for Australian-born children (9.57 [95% CI, 8.51–10.73] v 0.61 [95% CI, 0.53–0.69] cases per 100 000 children. The overall rate was highest among those aged 0–4 years. The annual notification rate was three times higher for Indigenous children than for non-Indigenous Australian-born children. Of 427 patients (79.4% of total) for whom the method of case detection was recorded, 37.0% were detected by contact screening, 8.7% by post-arrival immigration screening, and 54.3% by passive case detection. Pulmonary TB was the most common diagnostic classification (64.7% of patients). The most common risk factors were close contact with a TB case and recent residence in a country with a high incidence of TB. Treatment outcomes were satisfactory; 89.4% of children had completed treatment or were cured.

Conclusions: The burden of paediatric TB in Australia is low but has not changed over the past decade. The highest rates are among children born overseas, emphasising the important role of immigration screening as Australia aspires to eliminate TB.

Investigation into High Barmah Forest Virus Disease Case Numbers Reported in the Northern Territory, Australia in 2012 -2013

Kurucz N, Markey P, Draper A, Melville L, Weir R, Davis S, Warchot A, Boyd R, Stokeld D

Vector Borne Zoonotic Dis. 2016 Jan 20

Between October 2012 and October 2013, unprecedented high numbers of Barmah Forest virus (BFV) disease cases were reported in the Northern Territory (NT). An investigation was launched by the NT Department of Health in cooperation with the Department of Primary Industry and Fisheries and the Department of Land Resource Management to investigate possible causes for this phenomenon. The investigation included virus isolations from mosquitoes collected in Darwin urban areas, BFV antibody testing in peri-urban small mammals and a human BFV disease case series investigation of recent cases. No BFV was isolated from the 4641 mosquitoes tested, none of the mammals tested positive for BFV antibodies, and the high BFV disease case numbers did not correlate with the relatively low mosquito vector numbers trapped in 2012-2013. It was estimated that up to 89% of the 79 human cases investigated did not have an acute arboviral illness and therefore had tested falsely positive. An Alere PanBio BFV immunoglobulin M enzyme-linked immunosorbent assay test kit is generally used to test for BFV, with the BFV disease case definition based on immunoglobulin M positives only. Other jurisdictions in Australia also reported high numbers of BFV disease cases, with the majority of the cases suspected to be false positives. Therefore, current testing methods need to be revised to reflect the true numbers of BFV disease cases occurring in Australia and to provide correct diagnoses for patients.

Early influences on developmental outcomes among children, at age 5, in Australia's Northern Territory

Gutheridge S, Li L, Silburn S, Li S, McKenzie J, Lynch J

Early Childhood Research Quarterly·January 2016 DOI: 10.1016/j.ecresq.2015.12.008

Redressing developmental and school learning inequalities among children requires an understanding of the factors that influence development across population groups. This study utilized the 2009 Australian Early Development Census (AEDC) to explore the association of perinatal health and socio-demographic factors with early development of children in the Northern Territory of Australia. The study cohort included 1110 Aboriginal and 812 non-Aboriginal children, most aged 5 years, whose developmental status was assessed during their first year of full-time school enrolment. Individual-level information was probabilistically linked across three administrative datasets. Logistic regression models were used to estimate the association (odds ratio (OR)) between early life characteristics of children and teacher-rated vulnerability on one or more of five domains of development. The crude OR for developmental vulnerability was much greater for Aboriginal than non-Aboriginal children (OR: 6.93, 95% CI: 5.62–8.56). After adjustment for other variables, the increased risk of developmental vulnerability for Aboriginal children was substantially moderated (OR: 1.68, 95% CI: 1.21 –2.32). Influential factors in the adjusted model included: English as a second language (OR: 3.11, 95% CI: 2.27–4.26), gestational age at birth of 34–36 weeks (OR: 2.08, 95% CI: 1.27–3.39) and living in a very remote area (OR: 1.68, 95% CI: 1.19–2.37). There was a gradient in the strength of the association with the level of primary caregiver's education. An additional risk, for Aboriginal children only, was not having attended a day care or pre-school program (OR: 1.43, 95% CI: 1.01–2.04). The study demonstrates the emerging capacity for linkage of data across administrative datasets to inform our understanding of the extent to which multiple factors in early-life operate in their association with children's early development. Our findings are of particular relevance to initiatives to improve outcomes for Aboriginal children by demonstrating that potentially modifiable health and socio-economic factors account for almost all of the difference in developmental vulnerabilities observed between Aboriginal and non-Aboriginal children.

Comparing risks of cerebral palsy in births between Australian Indigenous and non-Indigenous mothers

Blair E, Watson L, O'Kearney E, D'Antoine H, Delacy M on behalf of the Australian Cerebral Palsy Register Group

Developmental Medicine and Child Neurology, DOI: 10.1111/dmcn.13005 1

Aim: To compare proportions of live births subsequently described as having cerebral palsy (CP), the distributions of associated impairments, and the causes of postneonatal CP between Aboriginal and Torres Strait Islander (Indigenous) and non-Indigenous populations in Australia.

Method: Data from statutory birth records and CP registers for the 1996 to 2005 birth cohort in Queensland, Western Australia, and the Northern Territory were stratified by Indigenous status and whether the CP was acquired pre/perinatally or postneonatally. Relative risks associated with Indigenous status were estimated and the distributions of causes of postneonatal CP compared.

Results: Indigenous births had a relative risk of 4.9 (95% confidence interval [CI] 3.0–7.9) for postneonatal CP but only of 1.42 (95% CI 1.2–1.7) for pre/perinatal CP. Almost half of postneonatal CP in Indigenous infants resulted from infection, whereas for non-Indigenous infants the most frequent cause was cerebrovascular accident. The impairments of Indigenous CP and of postneonatally acquired CP tended to be more numerous and more severe.

Interpretation: Indigenous children are at significantly greater risk of CP, particularly postneonatal CP. The predominant cause of postneonatal CP in non-Indigenous children has shifted to cerebrovascular accident over time; however, infections followed by head injury are still the most frequent causes in Indigenous infants.

Factors Associated with Routine Dental Attendance among Aboriginal Australians

Amarasena N, Kapellas K, Skilton M, Maple-Brown L, Brown A, Bartold M, O'Dea K, Celermajer D, Jamieson L

J Health Care Poor Underserved 27(1), February 2016 Supplement,pp. 67-80 | 10.1353/hpu.2016.0040

Objectives. To determine factors associated with routine dental attendance in Aboriginal Australians.

Methods. Data of 271 Aboriginal adults residing in Australia's Northern Territory were used. Routine dental attendance was defined as last visiting a dentist less than one year ago or visiting a dentist for a check-up. Both bivariate and multivariable analytical techniques were used.

Results. While 27% visited a dentist in the past year, 29% of these visited for a check-up. In bivariate analysis, being female, low psychological distress, and low clinical attachment loss (CAL) were associated with visiting a dentist within last year. Being aged younger than 39 years, male, no oral health impairment, being caries-free, low CAL, and low apolipoprotein B were associated with visiting for a check-up. Clinical attachment loss remained associated with visiting a dentist less than one year ago while being younger than 39 years and having no oral health impairment remained associated with usually visiting for a check-up in multivariable analysis.

Conclusions. Younger age, no oral health impairment, and low CAL were associated with routine dental attendance among Indigenous Australians.

Whole genome sequencing reveals extensive community-level transmission of group A Streptococcus in remote communities.

Bowen A, Harris T, Holt D, Giffard P, Carapetis J, Campbell P, McVernon J, Tong S

Epidemiol Infect. 2016 Feb 1:1-8

Impetigo is common in remote Indigenous children of northern Australia, with the primary driver in this context being *Streptococcus pyogenes* [or group A Streptococcus (GAS)]. To reduce the high burden of impetigo, the transmission dynamics of GAS must be more clearly elucidated. We performed whole genome sequencing on 31 GAS isolates collected in a single community from children in 11 households with ⩾2 GAS-infected children. We aimed to determine whether transmission was occurring principally within households or across the community. The 31 isolates were represented by nine multilocus sequence types and isolates within each sequence type differed from one another by only 0-3 single nucleotide polymorphisms. There was evidence of extensive transmission both within households and across the community. Our findings suggest that strategies to reduce the burden of impetigo in this setting will need to extend beyond individual households, and incorporate multi-faceted, community-wide approaches.

Editorial

Scabies and Global Control of Neglected Tropical Diseases

Currie B

N Engl J Med 2015; 373:2371-2372 December 10, 2015DOI: 10.1056/NEJMe1511805

In 1991, Taplin et al. published a seminal study from Panama showing that mass drug administration of the then newly available permethrin cream dramatically reduced the high rates of endemic scabies in island communities.[1] In this issue of the *Journal*, Romani and colleagues report the results of the Skin Health Intervention Fiji Trial (SHIFT),[2] which confirm the efficacy of mass administration of topical permethrin for scabies control. However, in their elegant randomized study involving island communities in Fiji, they also found that mass administration of oral ivermectin had superior effectiveness for scabies control. This study is timely, because scabies was added to the World Health Organization (WHO) list of neglected tropical diseases in 2013 and is estimated to affect more than 130 million people globally at any time.[3]

The effect of scabies extends beyond itching and sleep disturbance. In many resource-poor settings and especially in tropical regions, scabies is a major underlying cause of high rates of bacterial skin infections and their consequences. Impetigo due to *Streptococcus pyogenes* or *Staphylococcus aureus* (increasingly including community-acquired methicillin-resistant *S. aureus*) confers a predisposition to both septic sequelae (including fatal invasive infections) and the nonsuppurative streptococcal sequelae of glomerulonephritis and potentially of rheumatic heart disease.[4] It is important to note that successful mass drug administration for scabies control has led to substantial decreases in impetigo; in SHIFT, the prevalence of impetigo declined from 24.6% at baseline to 8.0% at 12 months in the group that received mass administration of ivermectin.

The classification of scabies as a neglected tropical disease and the acknowledgment that it is an important communicable disease in resource-poor countries pave the way for support for and coordination of research efforts and development of scabies control programs to be embedded in the expanding framework for the control of neglected tropical diseases.[5,6] However, although the WHO initially classified scabies as a neglected tropical disease, it has not developed a formal program for scabies control, whereas 17 other neglected tropical diseases have road maps extending beyond 2015 that include detailed funding models, research priorities, and elucidation of disease-specific control measures that are based on organism biology, epidemiology, and availability of effective antimicrobial agents.[7]

Mass drug administration is central to global control of some neglected tropical diseases, including mass administration of albendazole and mebendazole for soil-transmitted helminth infections (ascariasis, hookworm infection, and trichuriasis), mass administration of azithromycin for trachoma and yaws, and mass administration of ivermectin for onchocerciasis and lymphatic filariasis. The considerable success of mass drug administration for the control of neglected tropical diseases to date has resulted from strong, coordinated public–private partnerships, with pharmaceutical industries donating billions of treatments and philanthropic foundations, governments in developed

countries, and banking institutions providing large investments.[7] There is optimism that the momentum for the control of neglected tropical diseases that has been built during the 15-year era of the United Nations Millennium Development Goals will be maintained in the post-2015 era of the Sustainable Development Goals; for instance, there is a realistic possibility that trachoma, lymphatic filariasis, and yaws will be eliminated before 2030. However, the inextricable link between neglected tropical diseases and development issues such as housing, sanitation, hygiene, nutrition, education, and maternal health cautions against making unreservedly optimistic predictions about the global effect of mass drug administration. Disruptions in even the most well-planned and well-funded programs occur in the context of regional conflicts,[1] and disastrous health consequences can result from substantial population displacement.

Treatment of only persons with scabies and their contacts is unlikely to lead to scabies control at the community level. SHIFT — together with lessons learned from the past decade of research, planning, and field experience in developing other programs for mass drug administration for control of neglected tropical diseases — will underpin the development of a formal global strategy for scabies control.[5-8]

SHIFT shows that, although the administration of topical permethrin and oral ivermectin may have similar efficacy in individual persons with scabies,[9] the practical aspects of oral therapy translate to superior effectiveness on a large scale. Unhatched scabies eggs are refractory to ivermectin. Approximately 7 to 14 days after the initial dose of ivermectin was administered, a second dose of ivermectin was given only to persons with clinical scabies, but the authors suggest that a more pragmatic approach may be to provide both doses to everyone in the community. The importance of this second dose in the mass administration of ivermectin for scabies control remains to be evaluated, as do the diagnostic and epidemiologic algorithms that determine when to initiate a program of mass drug administration, the time interval between interventions, and when to cease providing the interventions. Issues related to ivermectin use during pregnancy and in small children and the management of crusted scabies require further assessment.[10] Scabies moves with people, and population mobility across regions must be factored into program delivery models to prevent rapid reintroduction of infection.

Ultimately, the success and sustainability of programs for the control and elimination of scabies and other neglected tropical diseases will depend on coordination with and integration into existing clinical and public health programs.

References

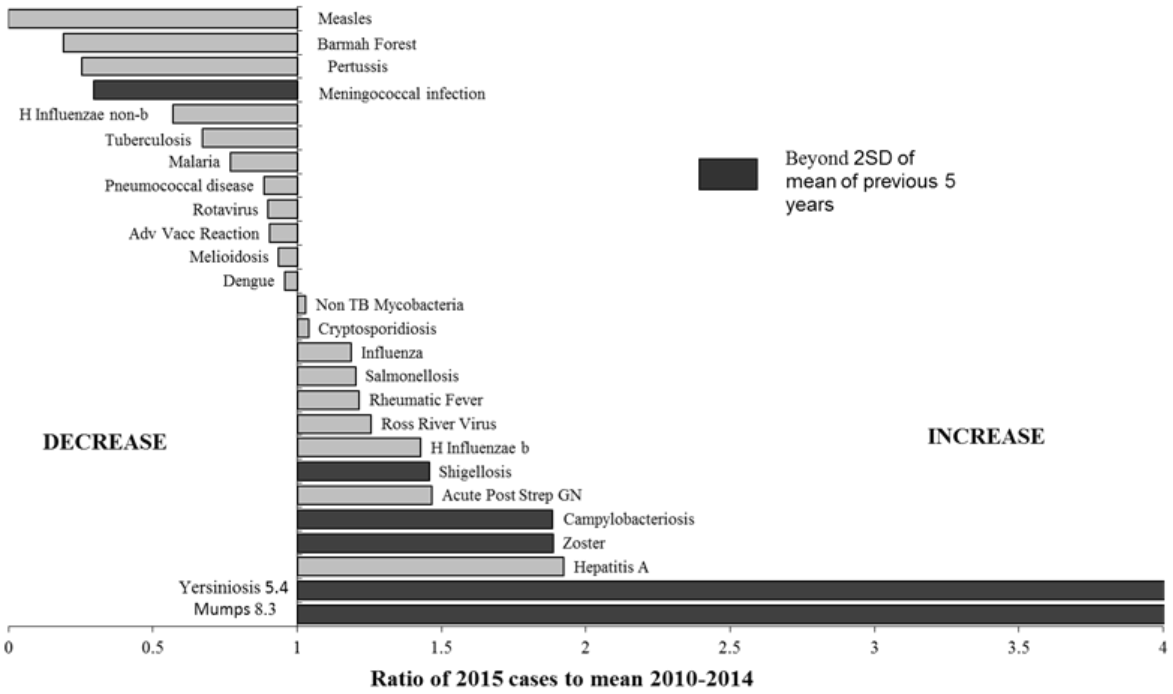
1. Taplin D, Porcelain SL, Meinking TL, et al. Community control of scabies: a model based on use of permethrin cream. *Lancet* 1991;337:1016-1018.
2. Romani L, Whitfeld MJ, Koroivueta J, et al. Mass drug administration for scabies control in a population with endemic disease. *N Engl J Med* 2015;373:2305-2313.
3. 3 Scabies. Geneva: World Health Organization, 2015 http://www.who.int/lymphatic_filariasis/epidemiology/scabies/en.
4. Romani L, Steer AC, Whitfeld MJ, Kaldor JM. Prevalence of scabies and impetigo worldwide: a systematic review. *Lancet Infect Dis* 2015;15:960-967.
5. Hotez PJ, Molyneux DH, Fenwick A, et al. Control of neglected tropical diseases. *N Engl J Med* 2007;357:1018-1027.
6. Engelman D, Kiang K, Chosidow O, et al. Toward the global control of human scabies: introducing the International Alliance for the Control of Scabies. *PLoS Negl Trop Dis* 2013;7:e2167-e2167.
7. Investing to overcome the global impact of neglected tropical diseases: third WHO report on neglected tropical diseases. Geneva: World Health Organization, 2015 http://apps.who.int/iris/bitstream/10665/152781/1/9789241564861_eng.pdf.
8. Marks M, Mitjà O, Vestergaard LS, et al. Challenges and key research questions for yaws eradication. *Lancet Infect Dis* 2015;15:1220-1225.
9. Chosidow O. Scabies. *N Engl J Med* 2006;354:1718-1727.
10. Currie BJ, McCarthy JS. Permethrin and ivermectin for scabies. *N Engl J Med* 2010;362:717-725.

****

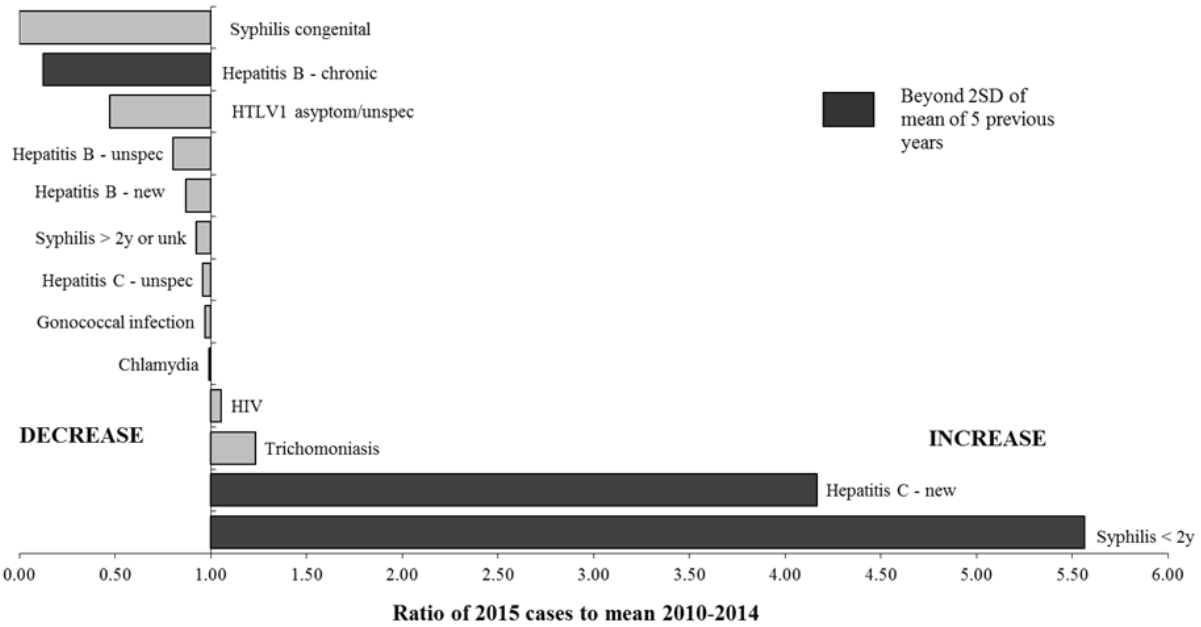
NT NOTIFICATIONS OF DISEASES BY ONSET DATE & DISTRICTS
1 January—31 December 2014 and 2015

	Alice Springs		Barkly		Darwin		East Arnhem		Katherine		NT	
	2014	2015	2014	2015	2014	2015	2014	2015	2014	2015	2014	2015
Acute post strep glomerulonephritis	18	15	12	7	18	15	13	12	15	5	76	54
Adverse vaccine reaction	10	15	0	3	29	27	4	1	5	5	48	51
Amoebiasis	0	0	0	0	3	1	0	0	2	0	5	1
Barmah Forest virus	0	0	1	0	26	25	2	0	1	1	30	26
Brucellosis	0	0	0	0	1	1	0	0	0	0	1	1
Campylobacteriosis	64	61	4	6	187	270	16	31	27	21	298	389
Chickenpox	15	28	0	0	55	73	5	10	29	10	104	121
Chikungunya	0	0	0	0	0	7	0	0	0	0	0	7
Chlamydia	796	785	111	50	1562	1406	188	192	396	381	3053	2814
Chlamydial conjunctivitis	11	2	5	0	8	7	0	1	5	0	29	10
CJD	0	0	0	0	1	1	0	0	0	0	1	1
Cryptosporidiosis	21	33	2	8	43	65	10	12	14	10	90	128
Dengue	1	2	0	0	62	54	2	1	3	4	68	61
Food/water borne disease	0	0	0	0	0	14	0	0	0	0	0	14
Gastro - related cases	13	2	0	0	0	6	0	0	0	0	13	8
Gonococcal conjunctivitis	1	1	0	0	0	2	0	0	1	2	2	5
Gonococcal infection	770	949	90	69	470	404	92	145	362	302	1784	1869
Gonococcal neonatal ophthalmia	1	1	0	0	0	1	0	0	1	0	2	2
Group A strep invasive	20	18	3	7	23	25	5	6	9	4	60	60
Hepatitis A	0	0	0	0	3	5	0	0	0	0	3	5
Hepatitis B - chronic	37	3	0	0	19	2	9	6	0	2	65	13
Hepatitis B - new	0	1	0	0	2	3	2	0	0	0	4	4
Hepatitis B - unspecified	32	43	5	1	119	101	3	10	4	3	163	158
Hepatitis C - new	1	1	0	0	1	4	0	0	0	0	2	5
Hepatitis C - unspecified	35	38	2	1	149	159	3	3	13	11	202	212
Hepatitis D	0	0	0	0	1	0	0	0	0	0	1	0
H Influenzae b	1	1	0	0	0	1	0	0	0	0	1	2
H Influenzae non-b	6	3	0	0	3	2	0	0	2	0	11	5
HIV	1	1	1	0	10	17	0	0	2	1	14	19
HTLV1 asymptomatic/unspecified	30	23	2	1	3	5	0	0	1	2	36	31
Haemolytic uraemic syndrome	1	0	0	0	0	0	0	0	0	0	1	0
Influenza	321	134	28	8	369	401	86	78	56	87	860	708
Legionellosis	0	1	0	0	6	4	1	1	0	2	7	8
Leprosy	0	0	0	0	0	1	0	0	0	0	0	1
Leptospirosis	0	0	0	0	1	5	0	0	1	1	2	6
Lymphogranuloma venereum	0	0	0	0	1	0	0	0	0	0	1	0
Listeriosis	0	1	0	0	2	0	0	1	0	0	2	2
Malaria	0	1	0	0	10	12	1	0	0	1	11	14
Measles	1	0	0	0	51	0	0	0	2	0	54	0
Melioidosis	2	0	1	0	51	50	9	11	3	6	66	67
Meningococcal infection	0	0	0	0	4	0	0	1	0	0	4	1
Mumps	1	3	0	1	0	1	0	0	0	10	1	15
MVE	0	0	0	1	0	0	0	0	0	1	0	2
Non TB mycobacteria	0	0	0	0	1	6	0	0	1	1	2	7
Pertussis	27	9	1	0	50	50	4	1	2	2	84	62
Pneumococcal disease	22	34	3	2	18	21	0	5	3	6	46	68
Q fever	0	1	0	0	0	0	0	0	1	0	1	1
Rheumatic fever	33	39	2	1	45	33	14	31	21	16	115	120
Ross River virus	23	16	7	3	336	319	21	16	37	22	424	376
Rotavirus	54	80	10	4	24	76	2	22	15	7	105	189
Salmonellosis	89	74	8	11	312	381	22	30	59	68	490	564
Shigellosis	23	34	3	2	48	40	17	52	13	16	104	144
Syphilis < 2y	36	86	1	4	12	34	1	4	22	79	72	207
Syphilis > 2y or unknown	31	27	1	1	23	25	1	4	17	16	73	73
Syphilis congenital	2	0	0	0	0	0	0	0	1	0	3	0
Trichomoniasis	992	1053	211	135	1163	1216	563	567	647	608	3576	3579
Tuberculosis	4	2	0	0	23	20	0	1	3	4	30	27
Typhoid	0	0	0	0	1	0	0	0	0	0	1	0
Typhus	0	0	0	0	1	1	0	0	0	0	1	1
Varicella - unspecified	0	2	0	0	1	1	1	0	7	0	9	3
Vibrio food poisoning	0	0	0	0	2	0	0	1	0	0	2	1
Vibrio invasive	0	0	0	0	0	0	1	1	0	0	1	1
Yersiniosis	2	3	0	0	7	9	0	0	0	2	9	14
Zoster	51	37	3	1	166	289	12	29	29	33	261	389
Total	**3,599**	**3,663**	**517**	**327**	**5,526**	**5,698**	**1,110**	**1,286**	**1,832**	**1,752**	**12584**	**12726**

Ratio of the number of notifications in 2015 to the 5 year mean (2010–14): selected diseases



Ratio of the number of notifications in 2015 to the 5 year mean (2010–14): sexually transmitted diseases



Comments on notifications

Campylobacter, yersiniosis and shigellosis

In September 2013, the major private pathology laboratory servicing the NT introduced a faecal multiplex PCR test. The increased sensitivity of this test accounts for most of the increase in campylobacteriosis, yersinosis and shigellosis seen in 2014 and 2015.

Of the 270 campylobacteriosis notifications received in the Darwin CDC office in 2015, 141 (52%) were PCR positive but culture negative, suggesting they would not have been notified prior to the new testing regimen. In addition there were 2 outbreaks of shigellosis in remote Indigenous communities in early 2015 that may have contributed to the increase in notifications.

Hepatitis C

All cases were sporadic. All except one most likely acquired the infection through recent injecting drug use.

Syphilis<2yrs

The increase was mostly due to an ongoing outbreak affecting the Indigenous population in remote NT districts (except Darwin remote district), which started in July 2014. In addition, there was an increase in cases diagnosed in men who have sex with men in the Darwin urban area. The majority of these cases were non-Indigenous.

Meningococcal infection

There was only 1 case of meningococcal disease notified in 2016—the lowest number since electronic records began in 1990. The 5 year mean (2010 to 2014) is 3.4 and the mean figure 2000-09 was 9 cases per year. The decline in cases can only be partly attributed to the introduction of the meningococcal C vaccine in 2003 because the majority of cases in the NT have been caused by B group meningococci. Interestingly, cases caused by group B have also been in decline elsewhere in Australia but cases caused by meningococcal W have been increasing over the past 2 to 3 years in some jurisdictions but not in the NT. Increased vigilance for group W disease will remain important in the future.

Mumps

There were 15 cases of mumps in 2015 compared with a total of 9 cases over the last 5 years. Nearly all of these cases were linked to a large outbreak in WA of several hundred cases. There has been one community-wide response to the outbreak implemented in the Katherine region which resulted in no further cases in that community.

Zoster

There were 389 cases of zoster notified in 2015 which is almost 1.9 times the 5 year mean. The extent to which the increase is due to the availability and uptake of the PCR test is yet to be determined.

****

Immunisation coverage for children aged 12–<15 months at 31 December 2015

Region	Number in district	%DTP	%Polio	%HIB	%Hep	%Pneumo	% Fully vaccinated
Darwin	275	92.0%	92.0%	91.6%	93.1%	91.6%	90.9%
Winnellie PO Bag	53	92.5%	92.5%	92.5%	92.5%	92.5%	92.5%
Palm/Rural	251	92.8%	92.8%	92.8%	94.0%	93.6%	92.8%
Katherine	90	96.7%	96.7%	96.7%	97.8%	97.8%	96.7%
Barkly	18	100.0%	100.0%	100.0%	100.0%	88.9%	88.9%
Alice Springs	124	91.9%	91.9%	91.9%	93.5%	91.9%	91.9%
Alice Springs PO Bag	44	95.5%	95.5%	95.5%	95.5%	95.5%	95.5%
East Arnhem	34	97.1%	97.1%	97.1%	97.1%	97.1%	97.1%
NT	889	93.3%	93.3%	93.1%	94.3%	93.3%	92.7%
Non-Indigenous (NT)	576	92.4%	92.4%	92.2%	93.4%	92.0%	91.7%
Indigenous (NT)	313	94.9%	94.9%	94.9%	95.8%	95.5%	94.6%
Australia	78259	93.8%	93.8%	93.5%	93.6%	93.4%	93.0%

Immunisation coverage for children aged 24–<27 months at 31 December 2015

Region	Number in district	%DTP	%Polio	%HIB	%Hep	%MMR	%MenC	%Varicella	%Fully vaccinated
Darwin	282	96.5%	96.1%	95.7%	96.5%	94.3%	95.4%	94.3%	92.6%
Winnellie PO Bag	52	96.2%	96.2%	96.2%	96.2%	94.2%	96.2%	92.3%	92.3%
Palm/Rural	252	96.0%	96.0%	93.3%	96.0%	90.9%	92.9%	90.1%	88.5%
Katherine	71	97.2%	97.2%	95.8%	97.2%	91.5%	95.8%	90.1%	88.7%
Barkly	30	86.7%	86.7%	83.3%	86.7%	73.3%	83.3%	73.3%	73.3%
Alice Springs	120	93.3%	93.3%	92.5%	95.0%	90.0%	94.2%	90.0%	88.3%
Alice Springs PO Bag	40	95.0%	95.0%	95.0%	95.0%	90.0%	92.5%	87.5%	87.5%
East Arnhem	30	86.7%	86.7%	86.7%	86.7%	83.3%	86.7%	86.7%	80.0%
NT	877	95.2%	95.1%	93.8%	95.4%	91.2%	93.7%	90.8%	89.2%
Non-Indigenous (NT)	559	95.2%	95.0%	93.4%	95.2%	91.8%	93.9%	91.6%	89.6%
Indigenous (NT)	318	95.3%	95.3%	94.7%	95.9%	90.3%	93.4%	89.3%	88.4%
Australia	77243	95.5%	95.5%	94.6%	95.3%	91.9%	94.4%	92.0%	90.3%

Immunisation coverage for children aged 60–<63 months at 31 December 2015

	Number in district	%DTP	%Polio	%MMR	%Fully vaccinated
Darwin	242	94.2%	94.2%	94.6%	93.4%
Winnellie PO Bag	63	98.4%	98.4%	98.4%	98.4%
Palm/Rural	247	92.7%	92.7%	92.3%	91.9%
Katherine	67	100%	100.0%	100.0%	100.0%
Barkly	24	91.7%	91.7%	91.7%	91.7%
Alice Springs	130	87.7%	88.5%	89.2%	87.7%
Alice Springs PO Bag	39	92.3%	92.3%	92.3%	92.3%
East Arnhem	56	96.4%	96.4%	94.6%	94.6%
NT	868	93.5%	93.7%	93.7%	93.0%
Non-Indig (NT)	534	91.8%	91.9%	91.8%	90.8%
Indig (NT)	334	96.4%	96.4%	96.7%	96.4%
Australia	79098	93.7%	93.7%	93.7%	93.2%

Immunisation coverage at 31 December 2015

Charles Strebor, CDC, Darwin

Immunisation coverage rates for Northern Territory (NT) children by regions based on Medicare address postcode as estimated by the Australian Childhood Immunisation Register are shown on page 52.

Background information to interpret coverage

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

The cohort of children assessed at 12 to <15 months of age on 31 December 2015 were born between 1 June 2014 and 30 September 2014 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 2015 were born between 1 June 2013 and 30 September 2013 inclusive. To be considered fully vaccinated, these children must have also received meningococcal C vaccination (given at the 12 month schedule point), dose 2 of measles, mumps, rubella (MMR) and dose 1 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 inclusion of these 3 additional immunisations has caused a drop in the reported coverage rates which are measured at 2 years of age. The primary reason for the decrease is that uptake and/or reporting of the 18 month dose of MMRV is lower than for other vaccines. It is expected that these children will catch-up by their 4th birthday when they present for their next scheduled vaccination, if not before. Over time parents and providers will become more familiar with the due date for MMRV and timely coverage should improve.

The cohort of children assessed at 60 to <63 months of age on 31 December 2015 were born between 1 June 2010 and 30 September 2010 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

The vaccination coverage rates for children in the NT are comparable with the national average for all age cohorts: 12 to <15 months cohort (NT 92.7%, National 93.0%); 24 to <27 months cohort (NT 89.2%, National 90.3 %), and for the 60 to <63 months cohort (NT 93.0%, National 93.2%).

Indigenous children were more likely (Indigenous 94.6 %, non-Indigenous 91.7%) to be fully immunised than non-Indigenous children in the 12 to <15 month cohort and less likely to be immunised in the 24 to <27 cohort (Indigenous 88.4%, non-Indigenous 89.6%) but more likely to be fully immunised in the 60 to <63 cohort (Indigenous 96.4%, non-Indigenous 90.8%).

Further information about the Australian Childhood Immunisation Register coverage may be found at: http://ncirs.edu.au/immunisation/coverage/index.php

****

Disease Control staff updates January—March 2016

Top End

Welcome to the new medical officers to CDC Darwin. Drs **Jin Yang**, and **Prashanti Manchikanti** are working with CDC on a 6 monthly rotation as General Practice (GP) Registrars and Dr **Afshan Bhagat** will be on a 12 month rotation. Drs **Sarah Barklay, Sarah Smith** and **Michael Tong** completed their 6 month rotation as GP Registrars in December. Dr **Kristen Overton** has replaced Dr **Sarah McGuiness** as the Infectious Diseases Registrar on a 6 month placement at CDC.

Technical Officer **Jaana Wenham** left Medical Entomology (ME) in February and has moved to Melbourne. Senior Technical Officer **Jane Carter** will go on extended 12 months leave to work with the Northern Australia Quarantine Services. Two new Technical Officers, **Tomoko Okazaki** and **Megan Hoskins** have recently commenced working with ME.

Kaylene Prince, Public Health Nurse (PHN) in the Immunisation Unit has moved to Sydney after 15 months working with the Darwin CDC team. 2 immunisation data entry officers, **Kylie** and **Chloe Pelizzo**, have started on 3 month contracts from March until the end of June.

Kelly Hosking, Remote PHN Sexual Health, has temporarily transferred to the Primary Health Care Branch to work as the Continuous Quality Improvement Facilitator, Maningrida for 6 months. **Peter Nihill** and **Leo Gregor** have joined the Top End Remote Sexual Health Team as PHNs. Peter has extensive experience in remote primary health care and public health programs. Leo has most recently been working in Wadeye Clinic and has a background in Nursing, Education and the Arts.

Brendan Johnson, PHN, after 4 years of work with the Darwin Trachoma team and actively contributing to community screens and treatment has taken on a position in the Preventable Chronic Disease Program.

Central Australia

Kate Wales transferred from the TB to Clinic 34 as the PHN Hepatitis, backfilling for **Leanne O'Connor** who started maternity leave in January. Leanne has since given birth to a gorgeous baby girl, Elsie.

Daniel Miller, PHN Trachoma, moved to Canberra in January. **Jessica Harries** who was backfilling for Paula Wines transferred into Daniel's position.

Emma Hunt joined the Trachoma Team as a PHN in January 2016 to backfill Paula Wines until July 2016.

Abby Wagner started as a Receptionist/ Administrative Support Officer in January 2016 replacing **Rebecca Creeper** who transferred to the Department of Children and Families.

Dr Belinda Greenwood-Smith has taken on the role as Co-ordinator, Alice Springs and Barkly. She comes to CDC after working for 2 years as a Senior Rural Medical Practitioner for the Central Australian Health Service Primary Care, where she worked both in the Alice Springs Correctional Centre and in the remote community of Titjikala. She will continue to work at Titjikala on a 0.2 part-time basis. She has clinical skills in chronic disease management, Blood Borne Virus and Alcohol and Other Drug work and hopes to support the CDC in extending its reach into the prison system.

Photo: Dr Belinda Greenwood-Smith, Coordinator for Alice Springs and Barkly CDC

****