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Influenza in the Northern Territory—Highlights from laboratory-confirmed influenza in 2015 and 5 year trends

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Abstract

The Centre for Disease Control (CDC) monitors influenza in the community using a combination of different surveillance systems, but the main source of epidemiological data is with laboratory-confirmed notifications. The CDC collects extra information on these notifications including vaccination status, hospitalisation status and pregnancy status.

The flu season in 2015 started in late July coincident with the national flu season but there was a persistence of activity until early December particularly in the remote areas of the Top End. The season was 'moderate' with influenza B predominating early and A/H3N2 later in the season. The season was more severe than usual in the non-Indigenous population and less severe in the Indigenous population but rates in the Indigenous population were still 1.78 times higher than non-Indigenous rates and hospitalisation rates 3.5 times higher. There were 4 deaths. The Odds Ratio for getting laboratory-confirmed influenza while pregnant compared to not pregnant was 1.49.

The effectiveness of the new federally-funded influenza vaccination program for Indigenous children 6 months to under 5 years of age was able to be assessed using the screening method. Based on the 45 cases in Indigenous children 6 months to under 5, the estimate of vaccine effectiveness for 1 or 2 doses of vaccine was 45% (95% CI: -5–71) but should be interpreted with caution.

Key Words: Influenza; vaccine effectiveness; trends; Indigenous; pregnancy; hospitalisation rates.

Introduction

Since the H1N1 influenza pandemic of 2009 there has been increased interest in enhancing surveillance methods for influenza. This has been driven not only by the need to better assess the impact of seasonal flu on the population but also by the need to prepare for the next pandemic, which is likely to have a greater impact than that of 2009.

Contents

Influenza in the Northern Territory—Highlights from laboratory-confirmed influenza in 2015 and 5 year trends..	1
Childhood Vaccination Schedule	7
Imported Zika virus infection in a woman returning from Fiji.....	8
A case of borderline lepromatous, multibacillary leprosy	12
Leprosy (Hansen's Disease) fact sheet.....	17
The use of Short Message Service (SMS) to follow up cases of <i>campylobacter</i> gastroenteritis in Darwin, Northern Territory	19
Northern Territory malaria notifications January–March 2016	26
Abstracts/summaries from peer reviewed published articles related to the Northern Territory	27
Northern Territory notifications of diseases by onset date and districts	28
Graphs of selected diseases and sexually transmitted infections.....	29
Comments on notifications.....	30
Fact sheet and guideline update April–June 2016	30
Mumps fact sheet	31
Mumps information for general practitioners.....	33
Immunisation coverage	35
Disease control staff updates April–June 2016	37

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At the Northern Territory (NT) Centre for Disease Control (CDC) we now have several arms to our influenza surveillance system. Apart from the laboratory-confirmed notifications through the notifiable disease system and the GP sentinel surveillance (ASPREN), we also undertake Emergency Department (ED) syndromic surveillance to detect sudden rises of influenza-like illness presentations and in addition we monitor monthly respiratory mortality data (through death certification) to detect rises in mortality. We also participate in national flu surveillance systems such as Flutracking, an on-line weekly symptom survey, and FluCan, a sentinel hospital surveillance system to which Alice Springs Hospital contributes.

While these additions to our flu monitoring system have been valuable, the main source of information concerning flu epidemiology is still with the notification of laboratory-confirmed cases through the NT Notifiable Diseases System (NTNDS). The advent of nucleic acid testing (PCR) and the increase in the amount of testing at the clinical interface has meant that more than 500 cases are notified every year, allowing for a more detailed description of the epidemiology than was achievable prior to 2009.

In the NT, surveillance staff follow up lab-confirmed cases of flu to ensure that core data fields are completed. These include data on hospitalisation, influenza vaccination history for the previous 12 months and outcome data (whether the case survived or died). Pregnancy status is also included as a non-core data item. Here we report on the epidemiology of the flu in 2015 in comparison with previous years. We also report on the effectiveness of the NT flu vaccine program for Indigenous children following the introduction in 2015 of the annual nationwide federally funded influenza vaccine program for all Indigenous children from 6 months to under 5 years of age¹ (see NT Childhood Vaccination Schedule page 7). In its inaugural year the NT achieved coverage of 70% of 1 dose of influenza vaccine to this Indigenous age group (personal communication Rosalind Webby).

Methods

An extract of the laboratory-confirmed influenza notifications from the years 2010–2015 was retrieved from the NTNDS and analysed using STATA. The NTNDS includes all influenza cases who were tested within the NT, even if they were

resident interstate or overseas; the non-NT resident cases (about 5% of the total) were included in the analysis because it was assumed they acquired their infection locally, however they were excluded from rate calculations and comparisons.

For analysis of Indigenous status, those with Indigenous status unknown (2.7% of the total) were not included. For the analysis of influenza subtype, cases which tested positive for influenza A but negative for A/H1N1 were presumed to be A/H3N2. Those with type A that were not further classified were distributed to either H1N1 or H3N2 according to the distribution of known subtypes in the same stratum (fortnight). However with type B influenza the numbers that were strain -typed were very small so the Bs were analysed as 1 group. Population data were obtained from the NT population file at the Health Gain's website,² with the 2015 population data obtained by extrapolating the 2013 and 2014 data.

Vaccine efficacy was calculated for the under 5 year age group using the screening method with vaccine effectiveness given as:

$$VE=1-\frac{PCV}{1-PCV} \times \frac{1-PPV}{PPV}$$

where PCV is the proportion of cases vaccinated and PPV is the proportion of the population vaccinated, or coverage rate which was estimated using CDC immunisation data. Confidence intervals were calculated using the immediate commands in STATA.

Results

There were 707 cases of influenza notified by laboratories in 2015, with all but 36 (5.1%) being NT residents. The total was 103 less than in 2014 but still the second highest number of notifications reported since the pandemic year of 2009 when 2079 were notified. Data completion in 2015 was excellent with 97.3% Indigenous identification complete and all but 1 case having the "Died" field complete. Pregnancy status was entered for all but 12 (6.3%) of those 190 women of child-bearing age. Hospitalisation status was entered in all but 4 cases. Vaccine information was recorded for 85% of cases.

Figure 1 illustrates the flu seasons for 2014 and 2015 comparing the Top End with the Centre and exemplifies the wide variation in year-to-year flu

activity. In the Top End, 2014 was characterised by a large wet season flu epidemic in January to March followed by a weak dry season epidemic coinciding with the winter flu season elsewhere in Australia. In contrast the winter flu season in the Centre in 2014 was very severe. In 2015, there was no wet season epidemic at all and in both the Top End and Centre the flu season started in the last week of July commensurate with the flu season elsewhere, and continued until the last week of November. The flu season did not reach the remote areas of the NT until late in the year (September – November) and, although the rates were higher in remote areas compared with urban the flu season, was less intense and more sporadic in remote areas than previous years.

The 2015 flu types and subtypes are summarised in Table 1. About 50% of all notified flu cases were typed as B and 38 (93%) of the 41 (12%) type Bs that underwent strain analysis were of the Victorian lineage (B/Brisbane/60/2008) and the remainder were Yamagata lineage (B/Phuket/3073/2013). The proportion of cases that were H3N2 increased throughout the season and towards the end of the year a small number of cases of H1N1 were notified (Figure 2).

In 2015 the notification rate in the non-Indigenous population was 214 per 100,000, the highest since the 2009 pandemic. The Indigenous rate, at 382

Table 1. Laboratory confirmed influenza cases by type, subtype and lineage, 2015

Type	Cases	% of type	% of total	Type total
A/H1N1	25	7.0	3.5	
A/H3N2	152	42.8	21.5	
A Unspecified	178	50.1	25.2	
Total A		100.0	50.2	355
B/Victoria	38	10.9	5.4	
B/Yamagata	3	0.9	0.4	
B Unspecified	309	88.3	43.7	
Total B		100.0	49.5	350
Both A and B			0.3	2
Total			100.0	707

per 100,000, was 1.78 times that of the non-Indigenous population but this rate ratio was the lowest since 2009. During most of the season, July–September, the Indigenous and non-Indigenous rates were very similar but later in the year there was spread to remote communities resulting in a significantly higher annual rate in the Indigenous population. Age-specific rates in the Indigenous and non-Indigenous populations were similar in the age-groups 10–29 years but diverged in the younger and older age-groups (Figure 3).

Notable trends in the age-specific rates in 2015 compared with the previous year was the large rise in the rates in non-Indigenous 5–14 year olds

Figure 1. Cases of influenza by week commencing date; Top versus Centre; 2014–15

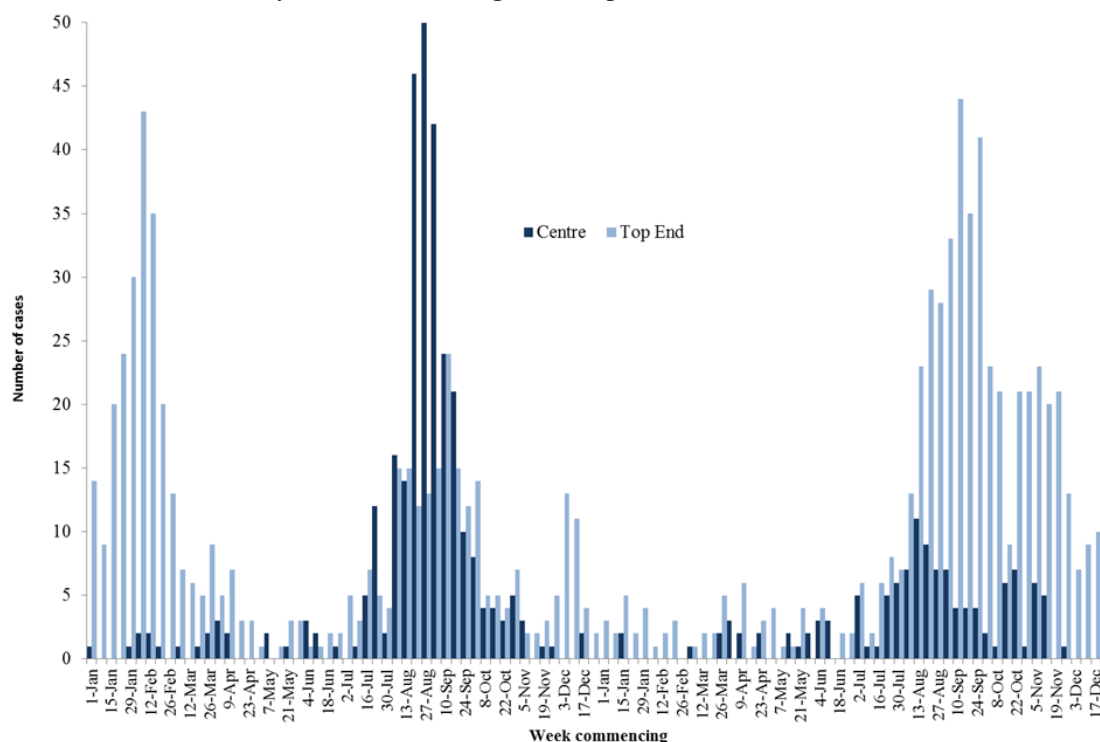
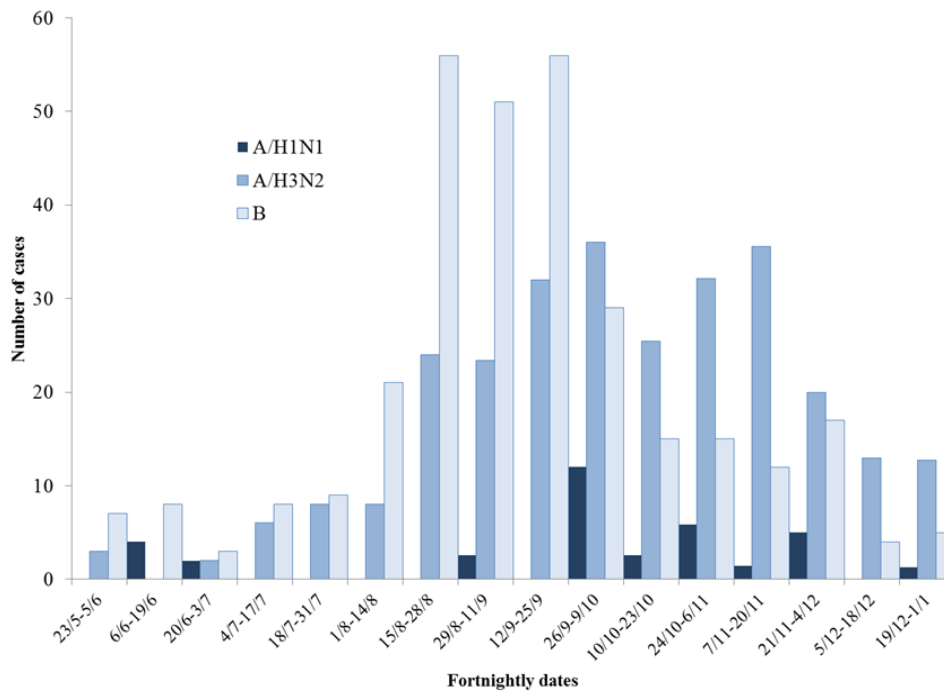
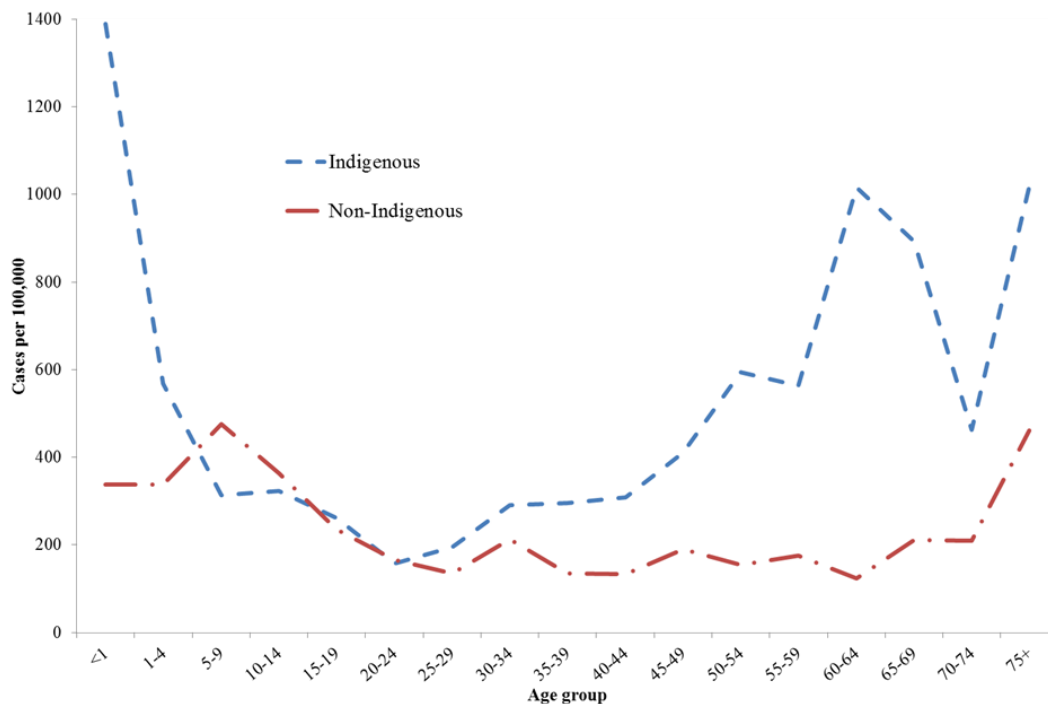


Figure 2. Fortnightly cases of influenza by subtype from late May 2015**Figure 3. Rates of laboratory confirmed influenza by age-group and Indigenous status; 2015**

which went from 127 per 100,000 in 2014 to 432 in 2015. In addition, the rates in Indigenous infants under 1 year of age fell from 2906 to 1523 per 100,000 and in the Indigenous people over 50 years from 1237 to 637 per 100,000 (Table 2). However compared with the previous 5 years, the proportion of cases in the under 1 year Indigenous population was not significantly less than in the years 2010–14 (7.8% compared with 8.8%; difference of proportion 0.97%; 95%CI: -2.4–4.4%). The proportion in the 1–4 year age

category in 2015 was 12.8% in 2015 compared to 10.7% in the years 2010–14.

There were 254 notified flu cases who were admitted to hospital in 2015, this was approximately equal to the mean number in 2010–14 (268) and is consistent with 2015 being a 'usual' or moderate year. Non-Indigenous admissions were 31% higher than the 5 year mean, while Indigenous admissions were 25% lower. Nevertheless the Indigenous hospitalisation

Table 2. Rates of laboratory confirmed influenza by year, age-group and Indigenous status 2010–15 (cases per 100,000)

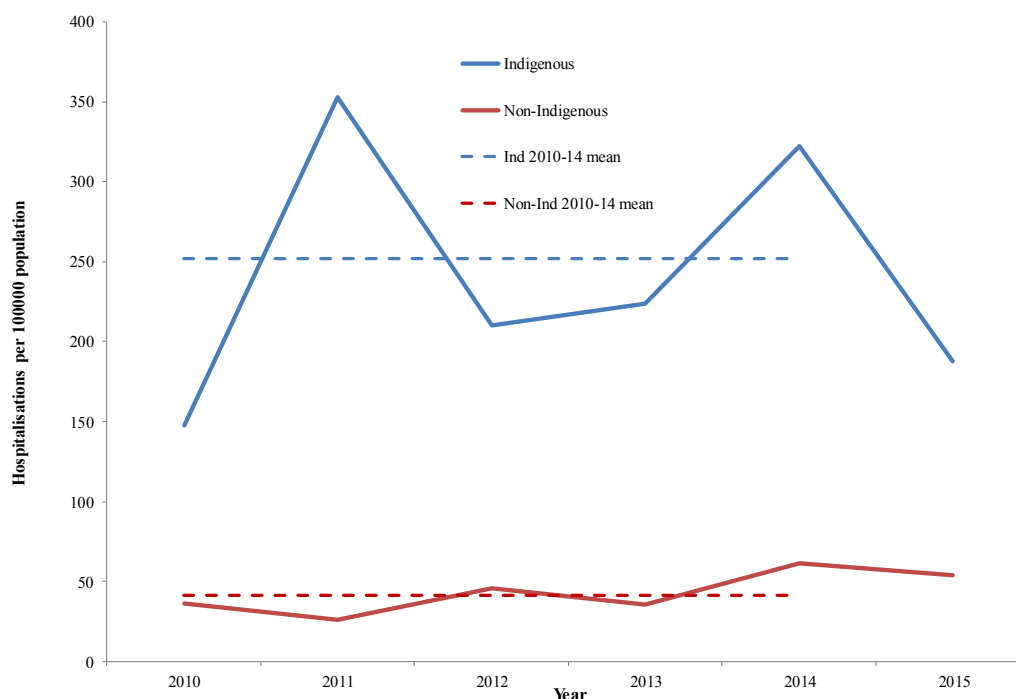
Indigenous						
Age	2010	2011	2012	2013	2014	2015
<1	1429	2832	1440	1313	2906	1524
1–4	471	860	566	467	657	623
5–14	406	417	217	216	479	334
15–49	295	534	191	311	493	258
50+	455	918	424	918	1238	637
All	381	635	284	406	656	383
Non-Indigenous						
Age	2010	2011	2012	2013	2014	2015
<1	181	45	261	127	210	333
1–4	90	113	185	212	315	333
5–14	174	105	156	107	127	432
15–49	137	98	130	91	167	170
50+	92	77	137	115	247	184
All	128	94	140	106	192	215

rate was still 3.5 times that of the non-Indigenous. Among the notified flu cases there was a total of 4 deaths (1 Indigenous) compared to the 5 year mean of 2.8 deaths per year. The trend in hospitalisations is illustrated in Figure 4.

There were 190 cases of influenza in women of child-bearing age (12–50 years) of whom the pregnancy status was determined in 178 (93.7%). Of those with known status, 18 (10.1%) were pregnant, with 9 being in the 3rd trimester or post-partum. Among those cases who were pregnant 5 had type A (either H3N2 or not subtyped) while 13 were type B. About 7% of women of child-bearing age are pregnant during a calendar year. This meant that the Odds Ratio for getting lab-confirmed flu if pregnant compared to not pregnant was 1.49 (95%CI: 0.86–2.4).

Of those who had vaccine status recorded, 54% of Indigenous cases and 14% of non-Indigenous cases were immunised against the flu. Analysis by subtype revealed that 16% of H1N1 subtype, 34% of H3N2 subtype and 31% of type B were immunised.

Lack of accurate estimates of vaccine coverage in the adult population and the inability to account for confounders (such as comorbidities) precluded estimation of vaccine effectiveness in the adult population. Analysis was limited in the younger age-group due to small numbers. Among the 45 Indigenous cases between 6 months and 5 years of age, 25 (55.5%; 95%CI 40.0–70.4) had had at least 1 vaccine (9 had 1 vaccine, 16 had 2). Based on this figure and vaccine coverage data from the NT Immunisation Register, the vaccine effectiveness

Figure 4. Hospitalisation rates from laboratory confirmed cases of influenza per 100,000 population, 2010–15, by Indigenous status, showing mean rate for 2010–14

in this age-group with either 1 or 2 doses was 45% (95%CI -5–71).

Discussion

Overall, analysis of the laboratory notified data revealed that the 2015 flu season was a moderate one. It was probably a more severe season than usual for the non-Indigenous population, in particular school-aged children, and a milder one for the Indigenous population. There was no Top End March–April season as is sometimes seen; the actual season started in mid-July, coinciding with the season in other states, and had a long ‘tail’ which extended to the end of November mainly due to late spread into remote Top End regions.

Laboratory confirmed notifications are always susceptible to the vagaries of testing behaviour. That is to say, true variations in flu incidence from year to year, between urban and rural areas, between hospitalised and non-hospitalised cases and even between Indigenous and non-Indigenous populations will be confounded by differences in the proportion of flu cases who get tested.

Nevertheless, it is probably reasonable to assume that, apart from pandemic years, testing behaviour will change only slowly, so trends in notified cases are likely to be valid. Likewise, testing for influenza on admission to hospital with a flu-like illness is likely to be less biased than in other settings.

In 2015, the influenza vaccine funded by the National Immunisation Program was the trivalent vaccine which contained the B/Phuket-like strain from the Yamagata lineage. However, from the beginning of the season the proportion of B viruses from the Victorian lineage (B/Brisbane-like strain) gradually increased at the national level, causing concern that vaccine effectiveness (VE) may be compromised. Not many type B specimens from the NT were strain typed in 2015 but of those sent off, the majority were from the Victorian lineage. Even though paucity of data did not allow us to estimate adult VE using 2015 data, it is interesting to note that in the NT the proportion of cases vaccinated in those who acquired A/H3N2 and B were similar (34% and 31% respectively) suggesting a similar VE, while the proportion vaccinated in those who had H1N1 was lower (16%), suggesting a higher VE.

Estimates of vaccine effectiveness were only performed in the 6 month to under 5 years Indigenous age-group because this cohort was for the first time eligible and targeted for funded vaccine in 2015 and robust coverage data were available. Analysis of this age group was also less likely to be affected by confounders such as comorbidities which might bias effectiveness estimates in other age-groups. The moderate vaccine effectiveness figure (45%), suggests the infant vaccine program is yet to have a sustained effect. Children under 9 years who are receiving their first ever influenza vaccine are recommended to have 2 vaccines, 1 month apart. In this analysis we accepted children with 1 or 2 vaccines as being immunised. In addition, even though the influenza rates in the Indigenous under 5 year olds were lower than 2014 (Table 1), the proportion of Indigenous cases aged under 5 years in 2015 was about the same as it had been in the years 2010–14 (20.6% in 2015 v 19.9% in 2010–14; $p=0.60$). Nevertheless, the small numbers of cases, the possibility of confounding from other factors and the fact that 1 vaccine was counted as immunised means that the results should be interpreted cautiously.

The lower Indigenous/non-Indigenous rate ratio in 2015 for both lab-confirmed cases (1.78) and hospitalisations of confirmed flu (3.5) compared with previous years 2010–2014 (3.6 and 6.1 respectively) may reflect a higher immunisation coverage in the Indigenous population (as would be expected from a focused influenza vaccination campaign for this group) or may simply be a property of this particular flu season, with its predominance of urban centres and type B virus. This nevertheless holds some promise that the gap between Indigenous and non-Indigenous flu morbidity might be closing.

References

1. Centre for Disease Control. Childhood vaccination schedule. NT Department of Health; March 2016 [cited 2016 Jun 28]. Available from: http://www.health.nt.gov.au/Centre_for_Disease_Control/Immunisation/index.aspx
2. Health District Population Data. Health Gains Planning, Department of Health [cited 2016 Jun 28]. Available from: <http://internal.health.nt.gov.au/healthstatistics/library/Pages/default.aspx>

Childhood Vaccination Schedule

Effective from
March 2016

	Hepatitis B	Rotavirus	Diphtheria Tetanus Pertussis Hepatitis B Polio Haemophilus influenzae type b	Conjugate Pneumococcal (13/PCV)	Haemophilus influenzae type b Meningococcal C	Measles Mumps Rubella	Hepatitis A	Diphtheria Tetanus Pertussis	Measles Mumps Rubella Varicella	Diphtheria Tetanus Pertussis Polio	Human Papillomavirus	Varicella	Polysaccharide Pneumococcal	Adult Diphtheria Tetanus Pertussis	Flu Quadrant Junior IMI	Flu Tera IMI
	Engerix [®] -B 0.5ml IMI	Rotarix [®] 1.5ml ORAL	INFANRIX [®] hexa 0.5ml IMI	Prevenar [®] 13 [®] 0.5ml IMI	Mentorix [®] 0.5ml IMI	M-M-R [®] II or Phionix [®] 0.5ml IMI	VAQTA [®] 0.5ml IMI	Tripace [®] 0.5ml IMI or Infanrix [®] 0.5ml IMI	Phionix-Terra [®] 0.5ml SC or ProQuad [®] 0.5ml SC	Infanrix [®] IPV 0.5ml IMI	Gardasil [®] 0.5ml IMI	Varivax [®] 0.5ml SC or Varitrix [®] 0.5ml SC	Pneumovax23 [®] 0.5ml IMI	Boostrix [®] 0.5ml IMI	Flu Quadrant Junior IMI	Flu Tera IMI
Birth ●	✓														<3 years	>=3 years
6 weeks		✓	✓	✓												
4 months		✓	✓	✓												
6 months		✓	✓	✓												
12 months					✓	✓	□	□	✓							
18 months									✓							
4 years										✓						
5 years											✓					
12 years												✓				
13 years																
15 years																

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 1 st 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



Imported Zika virus infection in a woman returning from Fiji

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Abstract

In February 2016, Zika virus (ZIKV) was declared a 'Public Health Emergency of International Concern,' largely due to the observed correlation with microcephaly and other foetal abnormalities and the growing numbers of countries declaring epidemics. In the Australian context, awareness of ZIKV has become increasingly important for both healthcare professionals and the public, with numerous countries in our Region reporting current transmission and 60 imported cases now reported. Here we review a recent case of ZIKV diagnosed in Darwin, Northern Territory (NT), Australia in a traveller returned from Fiji, only the second confirmed case in the NT. We include a review of the epidemiology and current clinical challenges, in particular those presented in managing women of reproductive age at risk of Zika infection.

Keywords: Zika; Fiji; Australia; pregnancy; women of reproductive age.

Case Report

We present a 37 year old woman living in Darwin, Northern Territory (NT), Australia who in March 2016 travelled to Fiji to visit her husband. Background history included ulcerative colitis treated with azathioprine and previous dengue fever.

On day 7 of her trip, she developed an erythematous, circular rash on her thighs that on day 8 developed into a 'sunburn-like' rash over her torso, abdomen and arms. The rash was followed by arthralgia with swollen, painful and stiff hands, wrists and feet. Symptoms peaked on day 3 of her illness, the day she returned to Australia, and symptoms completely resolved by day 6 of her illness at which time she was reviewed. She had been noted to have had possible mild and transient conjunctivitis, but denied fevers or any other associated symptoms.

A β -HCG (pregnancy test) was negative at review, albeit only 4 days after her return. Basic blood tests were largely unremarkable, with a C-reactive protein of only 9.3mg/L (<5.0mg/L), white cell count $4.4 \times 10^9/L$ (4.0 - $11.0 \times 10^9/L$), platelets $275 \times 10^9/L$ (150 - $450 \times 10^9/L$) and normal liver function tests apart from an alanine transaminase of 27U/L. Dengue NS1 antigen/IgM and chikungunya IgM were negative. ZIKV RT-PCR on serum on day 4 and 5 of illness were negative, as was ZIKV IgM on day 4 of illness. However ZIKV IgM on day 5 returned a positive titre of 1:40, with a subsequent convalescent IgM at day 17 of 1:320. ZIKV was confirmed by a positive urine PCR on day 4 of illness (see Chart).

Discussion

ZIKV is a mosquito-borne *Flavivirus* closely related to dengue virus and is a notifiable

Chart. Testing history for ZIKV in Northern Territory patient

Test	Day of Zika virus illness																	
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Urine				ZIKV RT-PCR Positive														
Serum				ZIKV RT-PCR Negative	ZIKV RT-PCR Negative													
				ZIKV IgM Negative	ZIKV IgM Positive 1:40													ZIKV IgM Positive 1:320

disease in the NT. It was first described in 1947, but has been showing increasing incidence worldwide since the first human epidemic was described in the Yap Islands of Micronesia in 2007.¹ Between 1 January 2007 and 6 April 2016, ZIKV transmission has been documented in 62 countries and territories, including 18 countries in the Western Pacific Region (WPR) of WHO. Of these WPR countries, 9 (American Samoa, Fiji, Marshall Islands, Micronesia, Papua New Guinea, Philippines, Samoa, Tonga and Vietnam) have reported mosquito-borne transmission of ZIKV in 2016, with 1 case of reported sexual transmission occurring in New Zealand.² By May 2016, 60 confirmed cases of imported ZIKV have been reported in Australians returning from international travel, with a higher incidence occurring during rainy seasons (November–April).³ The threat of further Australian cases remains high with ongoing circulation of virus at close proximity to our shores.

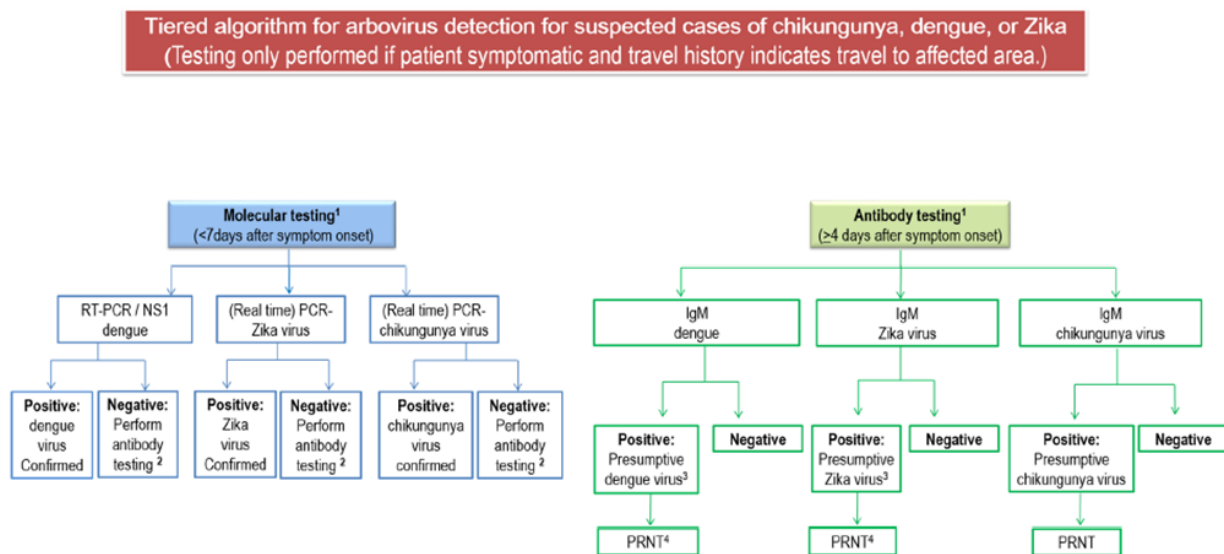
The dengue mosquito, *Aedes aegypti* and the Asian Tiger mosquito, *Aedes albopictus* are thought to be the predominant vectors of ZIKV transmission linked to recent outbreaks, although *Ae. hensilli* and *Ae. polynesiensis* were the likely vectors in the Yap and French Polynesia outbreaks.^{4,5} In Australia, *Ae. aegypti* is only present in Northern Queensland, while *Ae. albopictus* is restricted to the Torres Strait. The Queensland state government has a dengue vector monitoring and control program, which has been highly successful in preventing dengue disease from becoming endemic.⁶ Just as enhanced surveillance and control are carried out in response to notification of dengue cases, it will now also be carried out in response to overseas-acquired Zika cases in Queensland.

Within the NT, *Ae. aegypti* mosquitoes disappeared in the 1950s. There have been recent transient incursions of *Ae. aegypti* mosquitoes in Tennant Creek in 2004–2006, in Groote Eylandt in 2006–2008 and again in Tennant Creek in 2011–2014 that led to rigorous and successful elimination programs. There were no cases of dengue transmission in the NT from these 3 periods of transient incursions. In the past 12 months there have been increasing numbers of *Ae. aegypti* mosquitoes detected throughout Australia at international airports including 4 separate episodes of *Ae. aegypti*

detection at Darwin International Airport, the last on 17 January. All have necessitated a further escalation of surveillance and control activities, with no reported cases of dengue transmission within the NT. The last seaport incursions of *Ae. albopictus* were documented in 2013.^{7,8}

Sexual transmission of ZIKV has now been reported in 10 countries without the presence of local vectors. ZIKV has been isolated in high titres in semen, with 1 case report of a viral load roughly 100,000 times that of blood or urine.⁹ In this NT case, in addition to the risk of mosquito-borne transmission there was also a potential for sexual transmission despite her partner being asymptomatic. Saliva, breast milk and blood products for transfusion have also all been found to harbour ZIKV, however it is uncertain whether these have yet transmitted infection. A case of possible transmission by monkey bite in Indonesia was described in 2015 in a patient returned to the NT.¹⁰

With an incubation period of 3–14 days, ZIKV should be suspected in anyone with a compatible illness who has travelled to a country with local or recent transmission in the previous 2 weeks. It is important to also take a thorough history of possible sexual or other exposure. One of the difficulties is often in differentiation of the clinical features of ZIKV and other viruses such as dengue and chikungunya that are often co-endemic in the Pacific. Compared with dengue, ZIKV is usually less severe, with around 81% of infections thought to be asymptomatic. The most common feature is of a macular or papular rash, followed by fever (usually 37.5°C–38.5°C), arthritis or arthralgia, non-purulent conjunctivitis, myalgia, headache, retro-orbital pain, asthenia and less commonly oedema and vomiting.¹ Laboratory findings are often non-specific, with a usually normal C-reactive protein and platelet count, and possible mild neutropaenia or mildly elevated alanine transaminase. In this case, there was no fever or arthritis despite significant small-joint arthralgia and the rash and joint pain were brief, resolving within 6 days of onset. Reports worldwide suggest it is very rare for the virus to cause a serious illness requiring hospitalisation, or to lead to haemorrhage or shock. Of growing concern has been the risk of post-infectious Guillain-Barré Syndrome (GBS) and

Figure. Tiered algorithm¹³

meningoencephalitis, with a suspected attack rate of approximately 0.24 cases of GBS per 1000 ZIKV infections.¹¹ This compares to an estimated attack rate of GBS for *Campylobacter* infection of approximately 1 per 1058 infections.¹²

For diagnosis, ZIKV RT-PCR should be performed on serum if <7 days from onset of symptoms, and RT-PCR on urine if ≤ 14 days with urine testing reportedly more sensitive (see Chart and Figure).¹³ A positive PCR is confirmatory of ZIKV infection. However a negative RT-PCR cannot exclude ZIKV, so if ≥ 4 days after symptom onset a serum ZIKV IgM should also be requested. A positive IgM should then be confirmed either by a serum ZIKV Plaque Reduction Neutralisation Test (PRNT), or convalescent serology, depending on the laboratory. The specificity of PRNT is particularly useful in differentiating between ZIKV and other flaviviruses in areas of endemicity, as there is the potential for cross-reactivity leading to false positive results in those previously infected by viruses such as dengue. Testing of semen by RT-PCR should be considered if the diagnosis remains uncertain, particularly as positive semen PCR has been reported up to 62 days after confirmed infection. Saliva testing may also increase sensitivity, particularly useful in neonates and children with its ease of collection, however it should be noted that saliva does not remain positive any longer than serum.

Of the greatest concern is the now widely accepted correlation between ZIKV infection and foetal abnormalities due to congenital transmission of ZIKV. ZIKV has been detected in amniotic fluid, placental and miscarriage products of mothers with clinical syndromes consistent with ZIKV¹⁴ and in the cerebrospinal fluid of infants with microcephaly.¹⁵ A retrospective analysis of the 2013–2014 ZIKV outbreak in French Polynesia suggested microcephaly may affect around 1% of foetuses with maternal infection in the first trimester,¹⁶ a rate similar to that seen in the recent Brazil outbreak. While mostly associated with the 1st trimester, abnormalities have also been described in the 2nd and 3rd trimesters. Microcephaly with typical features has been the most reported neurological sequelae, however other associations include intrauterine growth restriction, ventricular calcifications, other cerebral and brainstem abnormalities, abnormal amniotic fluid volume, abnormal cerebral or umbilical artery flow, ocular abnormalities, arthrogryposis and even foetal death.¹⁷

Based on these concerns, in April the United States CDC published *Interim Guidance for Health Care Providers Caring for Women of Reproductive Age with Possible Zika Virus Exposure*.¹⁸ The Australian Department of Foreign Affairs and Trade¹⁹ lists Fiji as one of a number of countries to which women who are pregnant or actively seeking pregnancy should avoid travelling and the list is continuously

being updated with the changing epidemiology. Women attempting conception who do decide to travel to an area with active ZIKV transmission are encouraged to optimise prevention methods, including personal protective clothing, repellent and window screens and undergo vector control measures such as removal of potential mosquito breeding grounds. They should also be adequately counselled by a medical professional prior to departure on the risks so they can make informed decisions where possible.

For women of child-bearing age with confirmed ZIKV disease, the USA CDC recommends waiting at least 8 weeks after symptom onset to attempt conception to prevent intrauterine transmission and for men with ZIKV disease to wait at least 6 months after symptom onset to attempt conception to avoid sexual transmission.²⁰ Women and men with possible exposure to ZIKV without a consistent illness should also wait at least 8 weeks after exposure to attempt conception.

Pregnant women with possible exposure in the 8 weeks prior to conception should be offered serological testing within 2–12 weeks of exposure, whether or not there was clinical disease. If ZIKV is confirmed baseline ultrasonography should be performed and repeated every 4 weeks if initially normal. The Royal Australian and New Zealand College of Obstetricians and Gynaecologists have published guidelines on further management if foetal abnormalities are detected.²¹

At this stage Zika remains a rapidly evolving illness both in regard to its epidemiology and in its clinical consequences. Medical professionals in the NT, particularly those managing the returned traveller, will need to stay abreast of the most recent recommendations as more and more becomes known about this increasingly prevalent virus.

For more information see the Australian Government Department of Health Website: <http://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-zika.htm>

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A case of borderline lepromatous, multibacillary leprosy

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Abstract

Multidrug therapy (MDT) for Mycobacterium leprae consists of treatment with clofazimine, rifampicin and dapsone. However adverse reactions can make treatment troublesome. This is a case report of a 54 year old female from the Philippines with borderline lepromatous, multibacillary leprosy who developed treatment induced haemolytic anaemia, in the absence of G6PD deficiency and a Type 1 (reversal or upgrading) reaction.

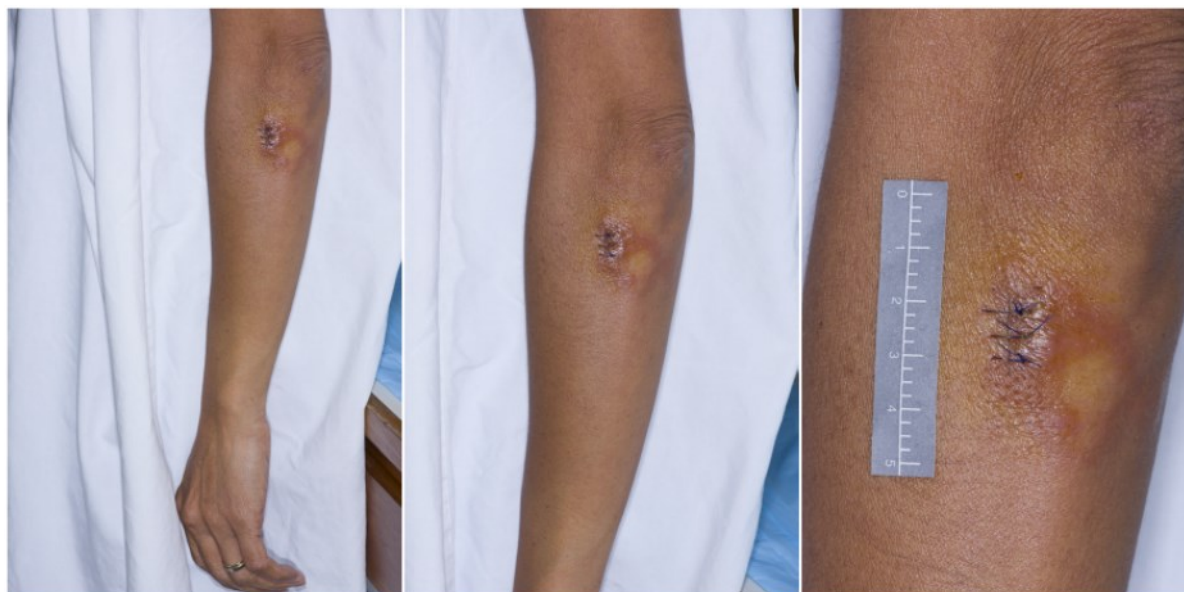
Key words: leprosy; multibacillary; leprostatic agents; drug-related side effects and adverse reactions.

Case report

A 54 year old female, who migrated from the Philippines over 20 years ago, presented to a dermatology outpatient clinic with a 3 month history of rash. The patient described a painful red skin lesion distal to the left elbow that had been gradually increasing in size. Altered sensation on the sole of her left foot was also reported. No associated cough, haemoptysis, fevers, night sweats or weight loss were reported. Inspection of the skin revealed a 4cm dusky red plaque and erythematous and hypopigmented areas with sharp borders on the extensor surface of the left forearm (Figure).

Examination revealed decreased light touch and pin prick sensation and alopecia over the affected area. There were bilateral enlarged median, ulnar, lateral popliteal and posterior tibial nerves. A skin biopsy was performed which demonstrated a nodular dermatitis with perivascular infiltrates consisting predominately of macrophages. Ziehl–Neelsen and Fite stains revealed numerous acid fast bacilli (AFB) classified as bacterial index (BI) $\geq 4+$ and *Mycobacterium leprae* DNA was detected by polymerase chain reaction (PCR). Nasal swabs were AFB smear negative and PCR negative for *M. leprae*. A chest radiograph and Mantoux test were performed to exclude co-existent active or latent tuberculosis infection. Pre-treatment electrolytes, full blood count, renal and liver function tests were also performed and were within normal limits. The patient was tested for glucose-6-phosphate dehydrogenase (G6PD) deficiency prior to administering dapsone and no deficiency was found.

As per *Guidelines for the Control of Leprosy in the Northern Territory*¹ the patient was commenced on self-administered dapsone 100mg daily and clofazimine 50mg daily with directly observed monthly doses of clofazimine 300mg and rifampicin 600mg. This combination of drugs is referred to as multi-drug treatment (MDT). Patients are routinely reviewed when

Figure: Skin lesion on the left forearm**Table 1: Haematological investigations**

Investigation	Result
Haemoglobin (115-165 g/L)	97 g/L ↓
Reticulocytes (20-100x10 ⁹ /L)	110 x 10 ⁹ /L ↑
Glucose-6-phosphate dehydrogenase	Normal
Direct antiglobulin test	Negative
Haptoglobin (0.36-1.95g/L)	0.17g/L ↓
Lactate dehydrogenase (<250 unit/L)	125 unit/L
Vitamin B12 (147-612pmol/L)	1325pmol/L ↑
Folate (10.9-43nmol/L)	30.4 nmol/L

given their monthly medications and electrolyte, full blood count, renal and liver function blood tests are taken to monitor for adverse effects. At our patient's first review, a significant drop in haemoglobin from 125 to 97g/L was identified, however the patient was asymptomatic. The test was repeated and confirmed to be an accurate result, and investigations to determine the cause of anaemia were performed as listed in Table 1. The results showed normal haematinics (B12, folate and iron) and were indicative of low level haemolysis in the absence of glucose-6-phosphate dehydrogenase (G6PD) deficiency. Repeat haemoglobin 1 week later was stable at 103g/L. Given the patient was asymptomatic and the haemoglobin was stable, the team decided to

continue current treatment with close monitoring. Ongoing blood tests demonstrate that the haemoglobin is slowly increasing (Table 2).

Treatment was further complicated at the 3 month review when the patient reported intermittent left hand numbness and occasionally weakness, with persistent numbness in the sole of the left foot. Additionally intermittent periorbital, left arm and left leg oedema had been noted. Examination confirmed new decreased sensation to pinprick and light touch on the plantar surface of the left foot. There was normal motor function in the upper and lower limbs. Bilateral enlarged median, ulnar, lateral popliteal and posterior tibial nerves were unchanged. The symptoms of sensory change and oedema, and examination findings were most consistent with a Type 1 (reversal or upgrading) reaction with neuritis. Accordingly the patient was commenced on prednisone 1mg/kg. Given the potential requirement for a prolonged course of high dose steroids, testing for opportunistic infections including hepatitis B and *Strongyloides* was undertaken. The prednisone remained at 1mg/kg for 4 weeks until complete resolution of the Type 1 reaction symptoms was noted and was then weaned. The patient experienced sleep and mood disturbances while on the prednisone but was able to continue the medication.

Table 2: Haemoglobin trend over time

	Pre-Treatment	Week 7	Week 8	Week 12	Week 16	Week 23
Haemoglobin g/L	125	97	103	106	105	110

Currently the patient remains on MDT with no symptoms of anaemia. The prednisone was slowly weaned over 3 months and ceased with no recrudescence of the Type 1 reaction. The patient's greatest concern is darkening of her face and skin due to cutaneous pigmentation from the clofazimine. She remains fully compliant with treatment and continues to be reviewed monthly. The patient has been educated and instructed to seek medical attention if any symptoms concerning for neuritis or anaemia occur inbetween appointments. The aim will be to continue MDT for a total of 24 months in line with the NT Guidelines.¹

Discussion

Leprosy is an infectious disease caused by *M. leprae*, which is a slow-growing intracellular bacillus that infiltrates the skin, the peripheral nerves, respiratory mucosa and the eyes.^{2,3} Leprosy rates in Australia are low (less than 1 case per 1 million population) and the disease predominantly occurs in immigrants from leprosy endemic areas and Indigenous Australians.⁴ Worldwide leprosy currently affects about 1.15 million people, with the majority of cases originating in India, Brazil, Indonesia, Nigeria, Ethiopia and Bangladesh.⁵

M. leprae is thought to be transmitted via droplets from the nose and mouth during close and frequent contact with affected individuals. However less than 1% of the population that comes into contact with *M. leprae* will develop the disease.⁶ The incubation period between infection and appearance of leprosy varies between 2 to 10 years and may be longer.

Worldwide there are 2 systems that are used to classify leprosy. The first, most comprehensive and accurate, is the Ridley-Jopling classification system that combines clinical, histopathological and immunological criteria to identify 5 leprosy forms:

- Tuberculoid (TT)
- Borderline tuberculoid (BT)
- Mid-borderline (BB)
- Borderline lepromatous (BL)
- Lepromatous (LL)

In this classification, there is a progression from the mildest TT to the disseminated form LL.¹

The second system is the WHO classification system. This is based on the number of skin lesions and identifies 2 forms: paucibacillary (<6 skin lesions) and multibacillary (≥ 6 skin lesions).³ The WHO system was developed mainly for use in low resource endemic areas where slit skin smears are not available, to allow for classification by general health workers in the field to achieve timely and appropriate treatment.¹ Now, often terminology from both classification systems is used.

The hallmark of leprosy is a broad clinical spectrum of pathology determined by the host's immune response. Patients with paucibacillary leprosy (towards the TT end of the spectrum) mount a vigorous T-helper 1 (Th1) cell-mediated immune response in skin and nerves. Although it limits the number of bacilli and lesions, this strong immune response leads to prominent peripheral nerve damage.⁷ In stark comparison, patients with multibacillary leprosy (towards the LL end of the spectrum) exhibit cellular unresponsiveness to *M. leprae* antigens. Multibacillary leprosy is associated with a T-helper 2 (Th2) immune response and high mycobacterial loads in the skin and nerves, and more diffuse skin involvement.⁷ However most patients, including our patient, display a clinical picture between the 2 polar forms and are classified as having either borderline tuberculoid (BT), borderline (BB) or borderline lepromatous (BL) leprosy. These borderline forms are considered to be highly unstable and represent poorly understood immunoregulatory responses.¹

The immune response to *M. leprae* fluctuates and can give rise to acute episodes of clinical inflammation during the chronic course of disease. Immunologic reactions occur in over 50% of patients, particular those with borderline disease. The 'unstable' immunologic state makes these patients more prone to the 2 main types of immunologic reactions.⁶ Type 1, reversal or upgrading reactions, as seen in our patient, are delayed-type IV hypersensitivity reactions and manifest as neuritis and increased inflammation of pre-existing skin lesions. Type 1 reactions are most common in the BB, BT and TT forms of leprosy.¹ Type 2 reactions or erythema nodosum leprosum (ENL) are a systemic response to immune complex deposition and present with multiple tender

nodules, fevers, arthritis, iritis and neuritis.⁸ This type of reaction is more common in the BL and LL forms of leprosy.

The simplification of the leprosy drug treatment regimen to MDT and promotion of free treatment to patients with leprosy since 1995 has resulted in more than 11 million people diagnosed with leprosy being cured.³ MDT has provided simple yet highly effective treatment for all types of leprosy. For multibacillary leprosy the standard WHO regimen consists of rifampicin 600 mg once a month, dapsone 100 mg daily and clofazimine 50 mg daily for 12 months. The recommended regimen for paucibacillary leprosy is rifampicin 600 mg once a month for 6 months and dapsone 100 mg daily for 6 months.³ The most important indicator for the effectiveness of MDT is the rate of relapse following successful completion of a full course of treatment. The information available to WHO, from a number of control programs, shows that the relapse rate is very low (0.1% per year for paucibacillary and 0.06% per year for multibacillary leprosy on the average).⁹ Our patient, had borderline lepromatous (BL), multibacillary leprosy and because of a high BI ($\geq 4+$) the recommended treatment is 3 drug MDT for 24 months as per the NT CDC Leprosy Guidelines.¹ The duration of treatment for 24 months is affordable in the NT and is recommended for cases with a high BI to achieve the lowest possibility of relapse.

According to the WHO, MDT is considered generally safe given the low dosages used for leprosy treatment.³ However this drug regimen may be accompanied by rare but undesirable side effects. The following reactions have been reported in patients while taking dapsone: haemolysis, methaemoglobinaemia, gastrointestinal problems, neuropsychiatric complications, peripheral neuropathy, cutaneous disorders, dapsone hypersensitivity syndrome (DHS) and agranulocytosis. DHS is characterised by fever, skin rash, eosinophilia, lymphadenopathy, hepatic, pulmonary and other systemic manifestations.¹⁰ The main adverse effects of clofazimine are cutaneous pigmentation, xeroderma, photosensitivity, gastrointestinal problems, and lower limb oedema. The principal side effects of rifampicin are icterus, painful hepatomegaly, altered liver function tests, intra-hepatic cholestasis,

gastrointestinal symptoms, cutaneous manifestations, acne lesions, eosinophilia, leucopenia, haemolysis, anaemia, thrombocytopenia, agranulocytosis and a flu-like syndrome.^{11,12} NT CDC Guidelines recommend reviewing patients weekly until full adherence and understanding of the medications is achieved, which also allows for assessment of any rare but potentially serious side effects, and then a 4 weekly cycle of review can be implemented.¹

Dapsone, a sulfone, inhibits bacterial synthesis of dihydrofolic acid.¹² It is an agent used for infections such as leprosy, malaria and pneumocystis. It also has anti-inflammatory and immunomodulatory properties that may be useful in various skin diseases such as lichen planus and dermatitis herpetiformis. Dapsone is the most likely component of MDT to be associated with adverse events. Dapsone was the cause for cessation of MDT in over 55% of individuals in a Brazilian study.¹³ Haemolytic anaemia is a well-known complication of treatment with dapsone due to oxidant haemolysis caused by its metabolite hydroxylamine. Dapsone induced haemolytic anaemia can be severe and potentially fatal in a patient with glucose-6-phosphate dehydrogenase (G6PD) deficiency.¹² Therefore screening for G6PD deficiency, as done for our patient, is recommended before the drug is started. Although dapsone-induced haemolytic anaemia is mostly reported in patients with G6PD deficiency, there are reports of dapsone induced haemolytic anaemia in the literature in patients with normal G6PD levels.^{14,15,16,17} Assessment for evidence of anaemia or haemolysis for leprosy patients receiving dapsone should be included in their routine follow up management. As our patient is experiencing only low levels of haemolysis secondary to dapsone, it is possible to continue treatment. It is important to be aware of the potential side effects of treatment, such that appropriate monitoring is undertaken and serious side effects are recognised early.

Conclusion

Leprosy is a rare but curable condition and this case highlights many useful learning points. Our patient was diagnosed relatively quickly, as on average patients report 2 years of symptoms prior to diagnosis. Any damage caused by the

disease before curative antibiotics are administered is usually not reversible. This highlights the importance of early recognition of skin and neurological pathology that can point to the diagnosis. Once the diagnosis of leprosy is suspected, the Guidelines¹ provide important information to assist with confirming the diagnosis, classifying the form of disease, formulating a treatment regimen and monitoring for immunologic reactions and side effects, such that cure is achieved and disability is minimised.

As occurred in our patient, clinicians should be aware of the potential for Type 1 or Type 2 reactions, as they can lead to irreversible nerve damage and significant ongoing disability. The management of these often involves prolonged immunosuppression, and screening for opportunistic infections prior to commencement of immunosuppressive medications is important. Likewise, drug side effects including haemolytic anaemia can be serious and sometimes fatal. The NT CDC suggests that patients with leprosy should be seen weekly at the beginning of treatment, so that adverse side effects can be detected as early as possible and proper measures can be taken.¹ This is important for patient safety, but also enhances motivation and understanding of treatment. The ultimate goal is the global elimination of leprosy.

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Leprosy (Hansen's Disease)

Leprosy is an infection caused by the bacteria, *Mycobacterium leprae*. The bacteria look very similar to *Mycobacterium tuberculosis* but leprosy is a very different disease from tuberculosis. *M. leprae* often affects the nerves of the hands, feet and face, and also the skin. There is often much fear and misunderstanding about leprosy because it can cause disabilities, however it is not very contagious and it is easily treatable with antibiotics. Leprosy is curable and treatment provided in the early stages prevents or minimizes permanent damage to the skin, nerves, limbs and eyes.

Distribution

Approximately 219 000 new cases of leprosy were reported worldwide during 2011, occurring mainly in Africa, Asia and South America. Control of leprosy has improved significantly over the last 20 years due to national campaigns in many countries around the world. In Australia leprosy is rare and found mainly in Northern Australian Aboriginal people and migrants from overseas countries in Asia, the Pacific and Africa where leprosy is more common.

Infectivity

Leprosy is not a very contagious infection. It is probably transmitted by droplets from the nose and mouth when people are in close and frequent contact with an infectious person. The great majority of people who come in contact with untreated leprosy are unlikely to become infected. In fact, it is close family contacts who are most at risk of catching the infection. Infectious cases become non-infectious soon after starting regular treatment.

Types of Leprosy

Manifestations of leprosy are determined by a person's immune response to the disease. If the infected person has little

resistance the bacteria multiply and this end of the spectrum of disease is called multibacillary leprosy (previously referred to as lepromatous leprosy). If the infected person has a high level of resistance, most of the bacteria are destroyed and this end of the spectrum of disease is called paucibacillary leprosy (previously referred to as tuberculoid leprosy).

Diagnosis

The diagnosis of leprosy is often delayed because it is not considered, especially in countries like Australia where it is rare. Some people with leprosy may have a close family member with the disease, but often people do not know the source of their disease.

A discoloured skin patch, often, but not always without sensation, may be the first sign of leprosy. A doctor or nurse will ask about and look for numbness in the hands or feet, swollen nerves, eye problems, wounds or deformities on the hands or feet or skin changes that might indicate leprosy. A doctor or nurse may make a tiny cut in the skin to take a small sample of fluid under the skin to send to a laboratory for testing. If the leprosy bacteria, *M. leprae* are detected in the sample or other biopsy specimen, then leprosy is diagnosed.

If a person suspects he/she has leprosy, advice can be sought from the Centre for Disease Control (TB/Leprosy Unit), Building 4, Royal Darwin Hospital phone 89228804 or from any Centre for Disease Control in Nhulunbuy, Katherine, Tennant Creek or Alice Springs. People who live remotely may consult the Remote Medical Officers who regularly visit many of the rural community care centres in the Northern Territory. Discussion with GPs or Infectious Disease physicians may also be appropriate.

www.nt.gov.au/health

Leprosy

Treatment of the Infection

M. leprae bacteria can be completely cured with multidrug therapy (MDT). MDT means taking 2 or 3 special antibiotics (rifampicin, dapsone and sometimes clofazimine) for between 6 months and 2 years, depending on the type of leprosy. After only a few doses of MDT people with leprosy are no longer infectious to others, but to cure their disease they need to take all the antibiotics as prescribed by their doctor. People with leprosy usually do not need to stay in hospital for treatment. Treatment is free.

Treatment and care for deformities and disabilities

Leprosy can often damage nerves and cause deformities, especially if the diagnosis of the disease is delayed. Unfortunately the damage that results, often to the hands or feet cannot be cured with the antibiotics: these are the scars of leprosy. Occupational therapists and physiotherapists can help people take special care of their hands and feet to avoid developing further problems. Reconstructive surgery can be done for people with a range of deformities and disabilities from leprosy making it possible for them to live independent and productive lives.

Remember:

People with leprosy are not very contagious.

The leprosy infection can be treated with special antibiotics.

People with leprosy can live normally in a family, socialise and be employed.

There is no need to segregate people with leprosy.

Control

Leprosy is becoming less common around the world. Screening programs in the past have resulted in early detection of leprosy. Effective treatment programs with MDT therapy have reduced transmission of the disease. People living in the same house as a person with leprosy should be examined and followed up by a doctor or nurse.

Further information

For more information contact the TB Clinic in your region

Alice Springs 8951 7548

Darwin 8922 8804

Katherine 8973 9049

Nhulunbuy 8987 0282

Tennant Creek 8962 4259

or

<http://www.nt.gov.au/health/cdc>

The use of short message service (SMS) to follow up cases of *Campylobacter* gastroenteritis in Darwin, Northern Territory

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Abstract

One of the most common known causes of foodborne gastroenteritis in Australia is *Campylobacter* bacterium. A trial was conducted via the OzFoodNet epidemiologist to determine the usefulness of sending short message service (SMS) messages to people notified to the Northern Territory (NT) Centre for Disease Control (CDC) with campylobacteriosis in order to detect clusters of disease and estimate the incidence of overseas acquired infection.

An SMS was sent to 97 people to ask whether they ate restaurant or takeaway foods or travelled overseas before they were sick.

The response rate was 49% (48/97). Of the 48 respondents 23 (48%) reported eating from a food business before they were sick but no outbreaks or clusters were detected. Almost half (20/48, 42%) of respondents reported overseas travel in the week before they were sick, with the majority (13/20) reporting travel to Bali, Indonesia. Females were 1.8 times (95% CI 1.2-2.6) more likely to respond than males ($p < 0.05$). Parents/guardians of children under 18 years of age were 1.6 times (95% CI 1.0-2.4, $p = 0.09$) more likely to respond to an SMS than adult cases although this was not statistically significant. The median time delay to receive a response from parents/guardians was 13 minutes compared to 29 minutes from adult cases although this difference was also not statistically significant ($p = 0.09$).

SMS was effective in estimating overseas acquisition of campylobacteriosis but no outbreaks were detected by this method. It did prove to be a low-cost method of follow-up, placed a minimum resource burden on the CDC staff and is an accepted method of communicating with the public. SMS should be considered in the future to gather information in a timely fashion on people notified to the NT

CDC with potential communicable diseases or risks and to disseminate information rapidly.

Keywords: *Campylobacter*; short message service (SMS); gastroenteritis; notifiable conditions; OzFoodNet; foodborne illness; Northern Territory.

Introduction

The most common known causes of foodborne gastroenteritis in Australia are norovirus, pathogenic *Escherichia coli*, *Campylobacter* spp. and non-typhoidal *Salmonella* bacterium.¹ *Campylobacter* infection is notifiable in all Australian states and territories except New South Wales (NSW). In 2014, campylobacteriosis was the most frequently notified foodborne infection in Australia, with a rate of 124.9 cases per 100,000 population (19,933 cases).² In the Northern Territory (NT), campylobacteriosis is the second most frequently notified enteric disease after salmonellosis.³ In 2014 there were 294 notifications of campylobacteriosis in the NT which was a 42% increase in notifications compared to the previous year.³ The rate of campylobacteriosis in the NT in 2014 was 120 cases per 100,000 compared to the national rate of 125 cases per 100,000.²

Campylobacteriosis has an incubation period of 1-10 days (usually 2-5 days) and typically manifests as diarrhoea (sometimes bloody), abdominal pain, fever, malaise nausea and sometimes vomiting with symptoms persisting for several days to 2 weeks. Sequelae can include reactive arthritis in approximately 1% of cases and Guillain-Barré Syndrome which occurs in approximately 0.1% of cases.⁴

It is recognised that poultry meat is the primary source of *Campylobacter* infection in humans.⁵ A survey undertaken by Food Standards Australia New Zealand (FSANZ) in 2007-2008 on the incidence and concentration of

Campylobacter and *Salmonella* in raw chicken observed that 84.3% of post-processing carcass rinse samples (n=1,104) were positive for *Campylobacter* spp.⁶ Other sources of *Campylobacter* infection include other undercooked meats and unpasteurised milk. People can also become ill through the ingestion of food or water that has been contaminated by the faeces of animals, particularly kittens, puppies, birds and other farm animals or through close contact with these animals. Person to person transmission is rare.⁴

At NT CDC, campylobacteriosis cases are not routinely investigated. On the other hand people notified with salmonellosis in the NT are telephoned by trained public health staff at CDC and a standardised questionnaire is delivered in order to determine a person's likely source of infection and to attempt to detect outbreaks or clusters of disease. In the instance of salmonellosis, the incubation period is usually 12–36 hours and infection is through contact with food, water or an environment that has been contaminated with the faeces of a wide range of animals including poultry, reptiles, amphibians, swine, cattle, rodents and other pets.⁷ In most cases it is possible to identify a risk factor or meal that may have caused the illness.

Following up every case of campylobacteriosis in the same manner is not as efficient. With the high probability of poultry meat being the source of infection,⁵ it is difficult to identify specific meals that may lead to infection, particularly given the 1–10 day incubation period of *Campylobacter* bacterium⁴ and the increasing rate of consumption of chicken in Australia which sees the average Australian eating chicken multiple times per week.⁸ In addition, the limited human resources available in a typical public health unit requires staff to undertake activities that yield maximum returns. Currently there are limited means of detecting clusters or outbreaks of campylobacteriosis in the NT and no means of routinely detecting overseas acquisition using routine surveillance data.

Campylobacteriosis notifications have increased in the NT in 2013 and 2014 (Figure 1).⁹ In September 2013, the largest private pathology provider in the NT introduced a multiplex faecal polymerase chain reaction (PCR) test¹⁰ which is more sensitive in detecting *Campylobacter* than traditional culture methods. Whereas culture detects only viable organisms, PCR testing detects the nucleic acid of both culturally viable as well as non-viable organisms.

Figure 1. Campylobacteriosis notifications in the Northern Territory, 2005–2014

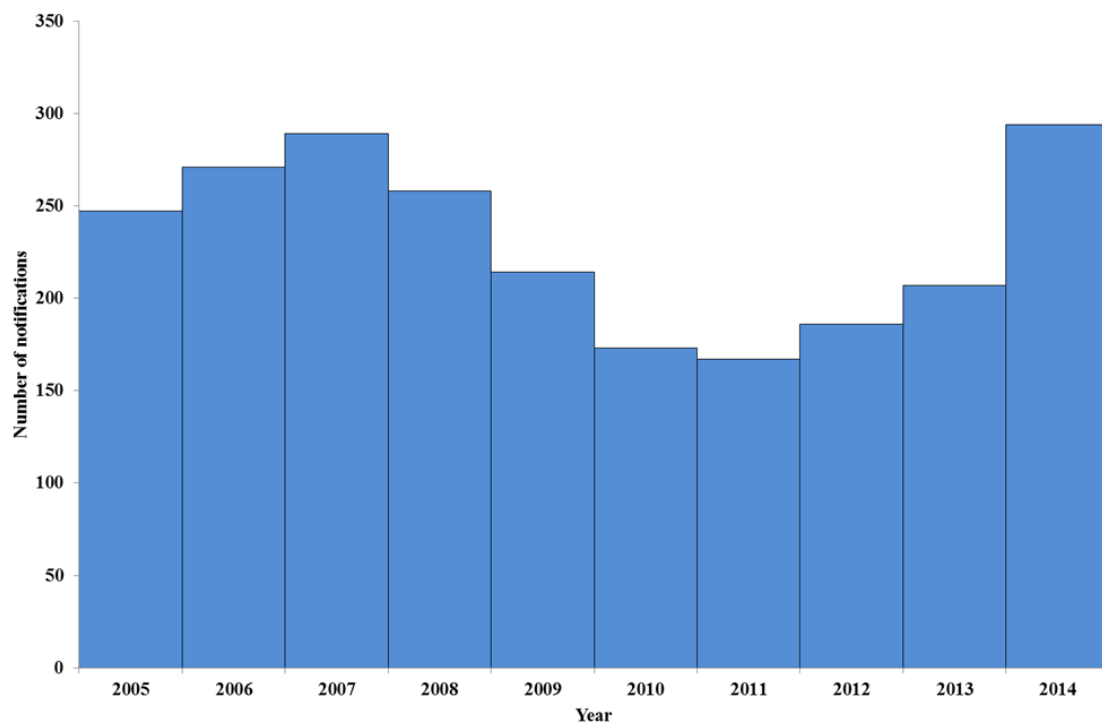
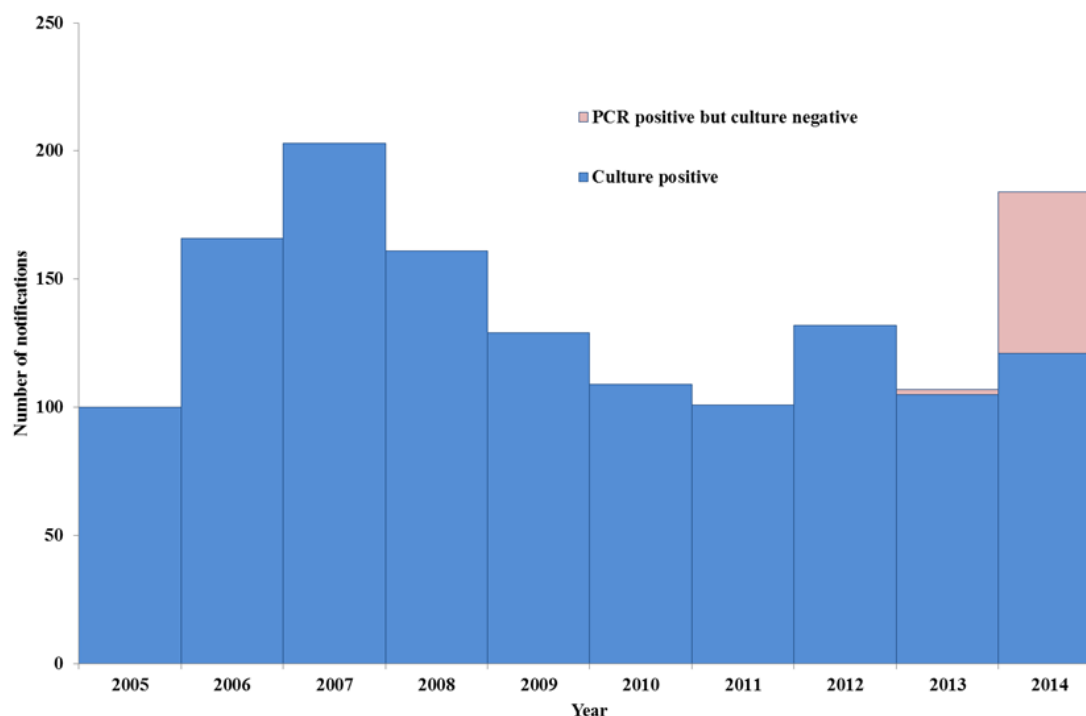


Figure 2. Campylobacteriosis notifications in the Darwin region by laboratory diagnosis method, 2005–2014



Based on surveillance data,⁹ it is estimated that 34% of campylobacteriosis notifications in the Darwin region in 2014 were PCR positive but culture negative (Figure 2) and this appears to account for most of the increase in notifications. The increase represents campylobacteriosis infections that would likely not have been detected prior to the introduction of the new faecal multiplex PCR test. An increase in notifications has not yet led to an increase in the detection of outbreaks.

Short message service (SMS) has been used effectively to follow up contacts of people notified with sexually transmitted infections (STIs),¹¹ people suffering from alcohol dependency¹² and people notified with malarial infection.¹³ SMS was recently trialled as a method of following up salmonellosis notifications in NSW and a 46% response rate was observed (Franklin N, 2015, unpublished data). Recently in the NT, the use of mobile phone messaging was shown to be appropriate and effective in following up Indigenous participants in a clinical trial with 90% of the Indigenous participants owning a mobile phone.¹⁴ These studies all identify the use of SMS as a potential low cost means for public health units to communicate with the public that is accepted by users and not resource intensive.

The NT OzFoodNet epidemiologist position is based in the Centre for Disease Control (CDC) Darwin and funded by the Australian Government with the main aim to improve food safety. The position works to enhance enteric disease surveillance in the NT and to conduct foodborne and non-foodborne gastrointestinal illness investigations to increase our knowledge about the causes of foodborne illness. This paper looks at the use of SMS messaging as a communication medium to collect data and investigate campylobacteriosis notifications.

Aims

The overall purpose of this project was to determine whether SMS is a useful and feasible tool for following up campylobacteriosis notifications in the NT.

The main research questions were:

- 1) Is the use of SMS to follow up campylobacteriosis notifications useful in terms of detecting clusters or outbreaks?
- 2) Is the use of SMS to follow up campylobacteriosis notifications useful in terms of measuring the incidence of imported infections?
- 3) Is SMS an appropriate communication medium for the CDC to communicate with

people notified with gastrointestinal or indeed other disease?

- 4) What, if any additional burden is placed on the NT CDC when using SMS as a follow up tool for following up campylobacteriosis notifications?

Methods

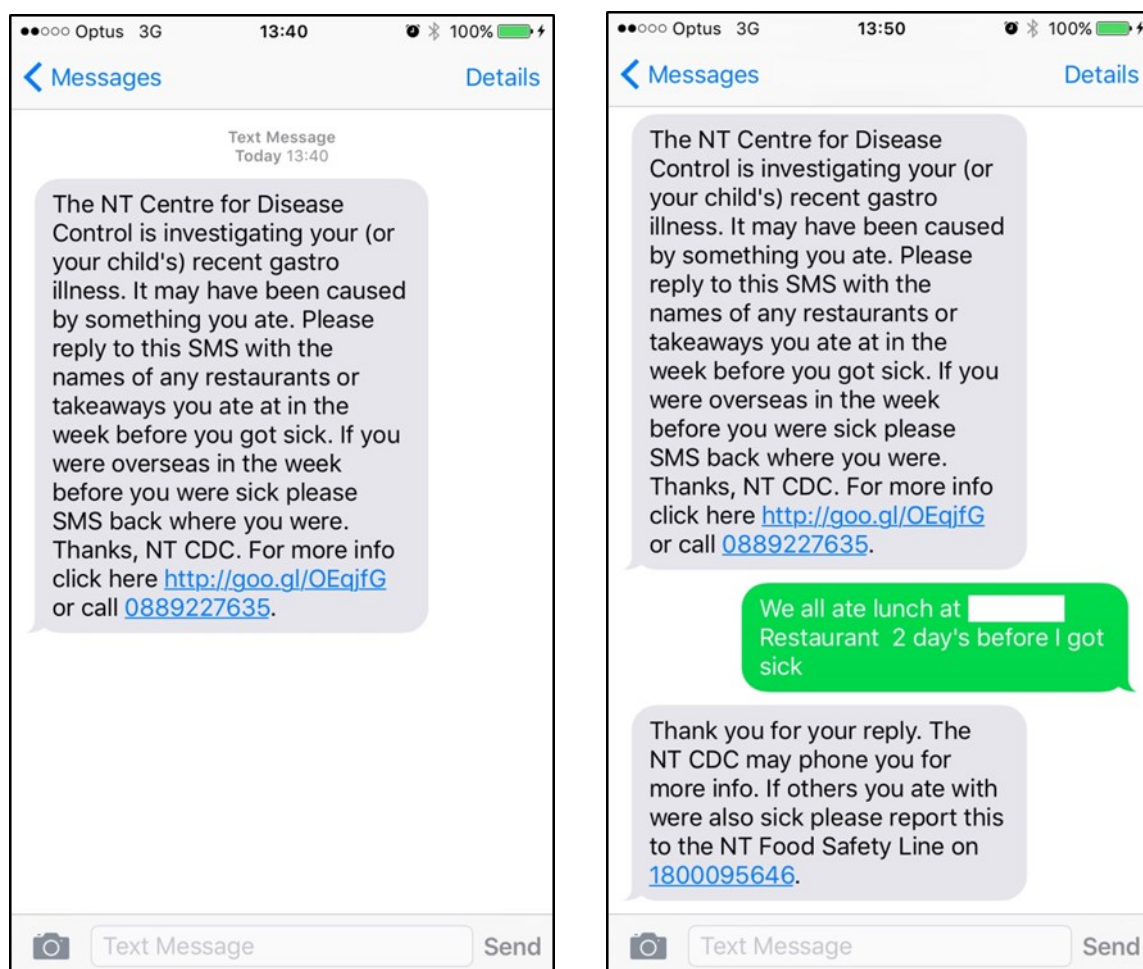
The study population was campylobacteriosis cases aged 5 years and over and notified to the Northern Territory Notifiable Disease Surveillance system (NTNDS) in the Darwin region between 1 November 2015 and 30 April 2016. Exclusion criteria were not having a mobile telephone number recorded, or a residential address in remote areas of the Darwin region (beyond the cities of Darwin, Palmerston and the Darwin rural area).

Cases were sent an SMS to either the number that was recorded on their pathology results form, the mobile number recorded in their NT

Health Department Hospital Record or the mobile phone number on file at their general practitioner. The SMS (Figure 3) was sent using the Telstra Integrated Messaging Service (TIMS) which is an internet based system for sending messages to mobile phones. The SMS asked cases or parents to reply with details of any restaurants or takeaways visited or overseas travel that occurred in the week prior to onset of symptoms. The SMS also contained a hyperlink to the NT CDC gastroenteritis factsheet.¹⁵

When cases replied to the SMS, an email with the contents of the reply was instantly generated by TIMS and sent to the OzFoodNet epidemiologist. The contents of the reply SMS, including details of exposures, travel and the date and time of the reply were entered into a Microsoft Excel spreadsheet. When cases replied to the SMS by voice call, the details provided were summarised and entered into the same spreadsheet. This line-listing of exposure histories was then merged with the case's

Figure 3. Screenshot of standard SMS messages sent to campylobacteriosis cases notified to the Darwin CDC between November 2015 and April 2016.



corresponding entry in the NTNDS, including any mention of overseas travel. Descriptive and comparative analysis including comparisons of medians and relative risks were performed using Intercooled Stata™ 13.1 and p-values calculated at the 5% significance level.

Any person who replied to the initial SMS and mentioned a takeaway or restaurant was sent a second SMS (as per Figure 3) informing them that the CDC may contact them for further information.

Thresholds for cluster investigation were established if the same branch of a national fast food chain was mentioned 3 times within a 2 week period, or if the same non-franchise food business was reported twice in a 2 week period. These thresholds mirror those used to investigate potential clusters of salmonellosis in the NT. Potential clusters identified via this system were to be investigated as per current local protocols.

Any campylobacteriosis cases who mentioned overseas travel in their response had this information recorded in the NTNDS.

Ethics

The collection of information on individuals notified with a notifiable disease falls under the auspices of the NT Notifiable Diseases Act.¹⁷ However, given that this study comprises a project that will form part of a master's thesis, ethics approval was obtained from the Human Research Ethics Committee of the Northern Territory Department of Health and Menzies School of Health Research and the Australian National University (ANU) Human Research Ethics Committee.

Results

Between 1 November 2016 and 30 April 2016, there were 139 notifications of campylobacteriosis in the Darwin region, of which 108 were aged 5 years or over. Of these cases 7 were immediately excluded as they did not have mobile telephones. A further 4 cases were excluded as they resided in remote localities. In total, 97 cases were sent an SMS and 48 replied (total response rate 49%). Demographics are summarised in Table 1 and Table 2 summarises responses to the SMS.

Table 1. Demographics of cases that were sent an SMS (n=97)

		No. of SMS Sent	Response
Age Group	5-17 years	14	10 (71%)
	18-54 years	67	32 (48%)
	≥ 55 years	16	6 (38%)
Respondent type	Parent/guardian	14	10 (71%)
	Adult case	83	38 (46%)
Gender of case	Male	58	22 (38%)
	Female	39	26 (67%)
Indigenous status	Indigenous	5	2 (40%)
	Non-Indigenous	74	34 (46%)

Table 2. Summary of responses to SMS (n=48)

	Numbers responding
Replied by SMS	30/48 (63%)
Replied by telephone call	18/48 (37%)
Reported overseas travel	20/48 (42%)
Reported travel to Bali, Indonesia	13/48 (27%)
Reported eating at a commercial food business	23/48 (48%)
National fast food chain	5/48 (10%)
Other takeaway	8/48 (17%)
Restaurant	6/48 (13%)
Café	4/48 (8%)

On days when patients were notified with campylobacteriosis, it is estimated that less than 5 minutes was spent on the activities required to run this surveillance project which included sending the original SMS, sending a reply SMS if required, entering and coding data collected and updating the NTNDS. When cases replied by phone, a conversation took place that enabled further, more specific public health information to be conveyed. However, the phone call still typically was less than 10 minutes duration and resulted in a specific campylobacteriosis factsheet¹⁸ being emailed to the case. Sending and receiving SMS using the TIMS service was free.

No clusters of disease or outbreaks were detected based on the 48 responses received. In the same time period there were no clusters or outbreaks detected by traditional laboratory based surveillance methods.

Almost half (42%) of those who responded reported overseas travel with 13/20 (62%) of these reporting travel to Bali, Indonesia. Two persons reported travel to Cambodia as well as 2 to Vietnam. There were single cases that had travelled to Bangladesh, India and the Philippines, respectively.

Females were 1.8 times (95% CI 1.2–2.6, $p < 0.05$) more likely to respond to an SMS than males. The median time delay to receive a response to an SMS was 19 minutes for females and 33 minutes for males but this difference was not statistically significant ($p = 0.65$).

Parents/guardians of children under 18 years of age were 1.6 times (95% CI 1.0–2.4, $p = 0.09$) more likely to respond to an SMS than adult cases although this was not statistically significant. The median delay between a person going to their doctor and being sent an SMS was 4 days whether people replied to the SMS or not. The median time delay to receive a response from parents/guardians was 13 minutes compared to 29 minutes from adult cases although this difference again was not statistically significant ($p = 0.09$).

Discussion

This study shows that SMS is a rapid and low-cost method for following up notifications of campylobacteriosis and does not place a heavy additional burden on public health unit staff.

However, the use of SMS did not detect any potential clusters or point source outbreaks of campylobacteriosis. This could be because SMS was not sufficiently suitable as it does not collect enough detail on potential exposures or it could, more likely, be a reflection that no outbreaks actually occurred during the study period as none were detected by traditional laboratory based surveillance either. It could also be because less than half of people who were sent an SMS replied and this did not provide sufficient numbers to detect an outbreak.

Establishing thresholds for investigating clusters associated with food businesses was difficult particularly as there is currently no reliable food frequency information available in the NT or Australia. An industry survey in 2014 found that 45% of Australians eat from a fast food restaurant every 4 weeks, with 42% visiting McDonalds, 29% visiting Subway, 23% visiting KFC and 16% visiting Hungry Jack's.¹⁶ The cluster investigation thresholds established for this project were arbitrary and consistent with those used to investigate clusters of salmonellosis in the NT.

The NT OzFoodNet site maintains a register of outbreaks that are investigated in the NT. Between 2003 and 2015 there were only 6 outbreaks in the NT where *Campylobacter* was identified as the aetiological agent and only 1 of these was a suspected foodborne outbreak. This sole suspected foodborne outbreak was among military personnel who had become ill after consuming food in Indonesia.

A high number of respondents reported overseas travel (20/48, 42%) as a likely source of infection which means that the annual proportion of campylobacteriosis in Darwin acquired overseas is likely to be even higher than that ascertained from SMS during the study period. It is possible however that people who reported overseas travel in the week before the onset of symptoms could have still spent a portion of their incubation period in Australia but this was not reported due to their preference to reply only with details of their overseas travel. Likewise, it is possible that cases could be biased in their nomination of restaurants and takeaways attended in the week before they became ill by only nominating the ones they suspected were the cause of their illness. It is also common for members of the public to suspect the last meal they ate before becoming sick as the cause of the illness which again introduces a bias. If this meal occurred within the minimum incubation period for an infectious agent there is no way for this to be ascertained by an SMS reply. The incubation period of up to 10 days for campylobacteriosis means that up to 30 separate meals could have been the potential source of infection. The fact that no clusters were detected could also suggest that the majority of those people who did not report

overseas travel (80%) likely acquired their infection due to consumption of poultry in the home which would be a reasonable assumption in light of the high chicken consumption rates reported in Australia.⁸

The finding however that parents/guardians and females were more likely to respond to the SMS and in a more timely manner has great implications for improved public health practice. For notifiable diseases in the NT that potentially require follow up or outbreak investigation and often affect children such as salmonellosis, cryptosporidiosis or chicken pox, SMS could provide an efficient primary follow-up method followed by a telephone call if no response is received or if insufficient information is gathered in the first instance. Similarly, for diseases such as dengue where a place of acquisition needs to be ascertained, or during a measles outbreak where vaccination status among contacts needs to be ascertained, an SMS could be effective as a primary contact method with a telephone call serving as a secondary method.

The approximately 50% response rate means that SMS should not be used as a complete follow up tool but could be used as an initial tool in situations where public health resources are scarce or when simple hypothesis generating is required such as may be required during an increase in notifications preceding an outbreak. SMS could also be potentially utilised to send links to online questionnaires that could instantly record information that could be later downloaded and analysed.

Conclusion and recommendations

As laboratory detection methods improve, public health units are required to investigate new and improved ways to collect enhanced data and investigate outbreaks and clusters of disease. SMS is easy to use and a low-cost method to follow up people notified to NT CDC with notifiable conditions. Women and parents/ guardians are more likely to respond to SMS and in a more timely fashion. In the future, it is recommended that SMS is used to engage this demographic, particularly if human resources are limited.

SMS is only useful if a limited amount of information is required in response and if the information required is well defined. As a result, it does not appear to be any more effective at detecting outbreaks of campylobacteriosis than traditional surveillance, but could be useful for follow up of other diseases. SMS is an effective method for estimating the proportion of imported disease in the NT. The proportion of campylobacteriosis in Darwin is likely higher than 21% and tourists departing Darwin should be provided with more information about eating safely while on overseas holidays.

In instances when information needs to be disseminated and no response sought, SMS should be used in the first instance as most adults own a mobile phone and phones are increasingly internet connected. The result is that information can be disseminated rapidly to a large number of people. It is recommended that NT CDC consider the use of SMS for dissemination of information and factsheets to the public.

Acknowledgments

Mrs Mary Verus, Data Manager, Surveillance CDC Darwin.

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Northern Territory malaria notifications January–March 2016

Liz Stephenson, Centre for Disease Control, Darwin

There were 3 cases of malaria notified in the 1st quarter of 2016. 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	Indonesia West Papua	Visiting student	<i>P. falciparum</i>	No	Darwin
1	Indonesia West Papua	Visiting student	<i>P. vivax</i>	No	Darwin
1	Papua New Guinea	Expatriate visiting relatives	<i>P. falciparum</i>	No	East Arnhem

Abstracts/summaries from peer reviewed published articles related to the Northern Territory

Infectious and congenital syphilis notifications associated with an ongoing outbreak in northern Australia. Short report.

Bright A, Dups J

CDI (2016) 40(1)E7-E10 <http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-cdi4001b.htm>

A summary of the above article is provided below by Matthew Thalany, Head Sexual Health and Blood Borne Virus Unit, CDC

The March issue of the *Communicable Diseases Intelligence (CDI)* journal carries an interesting 'Short Report' that describes the ongoing syphilis outbreak spanning the 3 jurisdictions of Northern Australia with 790 cases at the time of the report and 7 cases of congenital syphilis.

The *CDI* report tells us that the outbreak commenced in northern Queensland in 2011. It traversed the western boundary and sporadic cases started arriving in the Northern Territory (NT) in 2013. By July 2014 the NT had declared an outbreak, which moved on to the Kimberley in Western Australia in 2015. All 3 jurisdictions are now contending with the outbreak. A Multi-Jurisdictional Syphilis Outbreak (MJSO) Response Group was formed in 2015. This Short Report comes from that Group.

The epidemiology of the 790 outbreak cases reveals that it is mainly young Indigenous people aged between 15–29 years who are affected, 45% female, 55% male.

Although not part of the report, the MJSO response group has, through its work, identified that some of the challenges to outbreak control which include:

- Difficult-to-reach target population (highly transient and mobile young people)
- Lack of dedicated budget and resources to run outbreak control
- High turnover of staff in primary care in remote and rural communities

- Lack of knowledge about syphilis and its treatment among health professionals
- Difficulty of engagement with local communities to support community screening and other outbreak control measures due to stigma and shame of a taboo subject and disease

The MJSO Response Group reports to the Communicable Diseases Network of Australia (CDNA) and advocates a national response to this multi-jurisdictional problem.

Why are men less tested for sexually transmitted infections in remote Australian Indigenous communities? A mixed-methods study

Su JY, Belton S, Ryder N

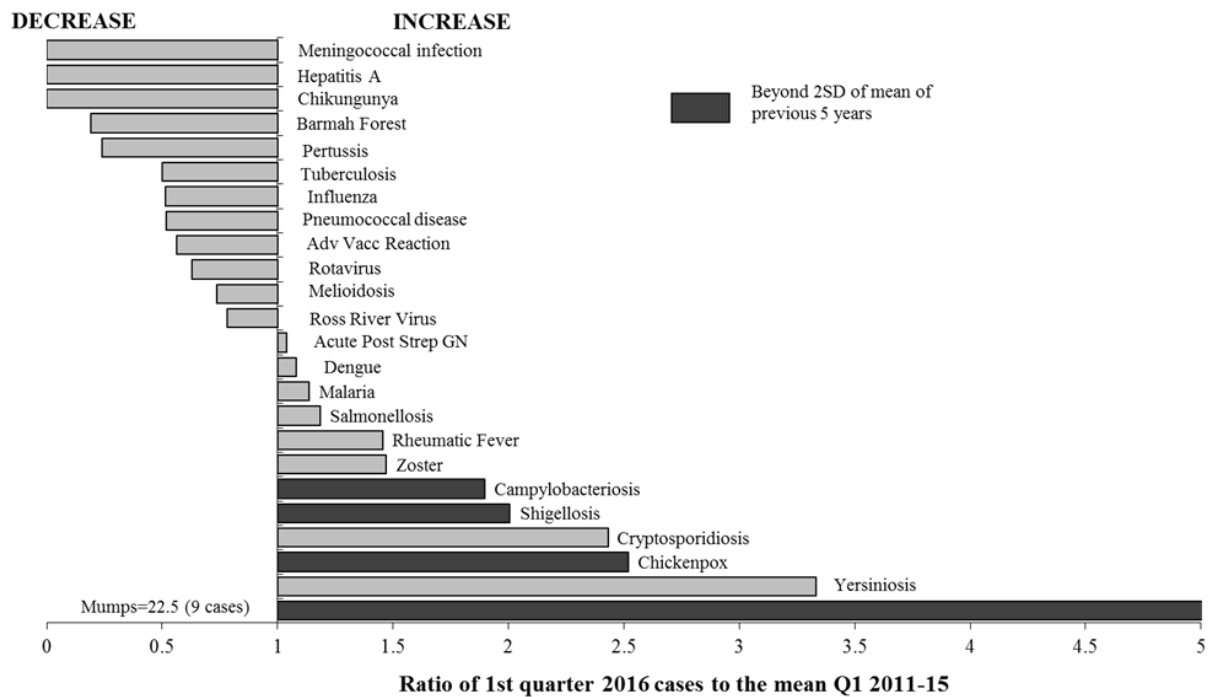
Culture, Health and Sexuality 2016 DOI: 10.1080/13691058.2016.1175028

Gender disparities in testing rates for sexually transmitted infections (STIs) have been identified as one potential factor sustaining high rates of STIs and repeat infections in the Northern Territory of Australia, especially in remote Indigenous communities. The study aimed to investigate the reasons for these disparities utilising a mixed-method study design. We conducted an audit on client information at a remote community health clinic, focus-group discussions with young men in the same community and interviews with experienced remote area clinicians. The clinic audit found a significantly higher proportion of female residents of the community than males visited the clinic (72.8 versus 55.3%, $p < 0.005$). Women were also more likely to be tested for STIs than men when visiting the clinic (49.7 versus 40.3%, $p = 0.015$). Major barriers to men's seeking STI testing included a sense of shame from being seen visiting the clinic by women, men's lack of understanding of STIs and the need for testing, and inadequate access to male clinicians. Increasing men's access to healthcare and STI testing requires offering testing at a gender-sensitive and separate locations, and community-based sexual health promotion to increase knowledge of STIs.

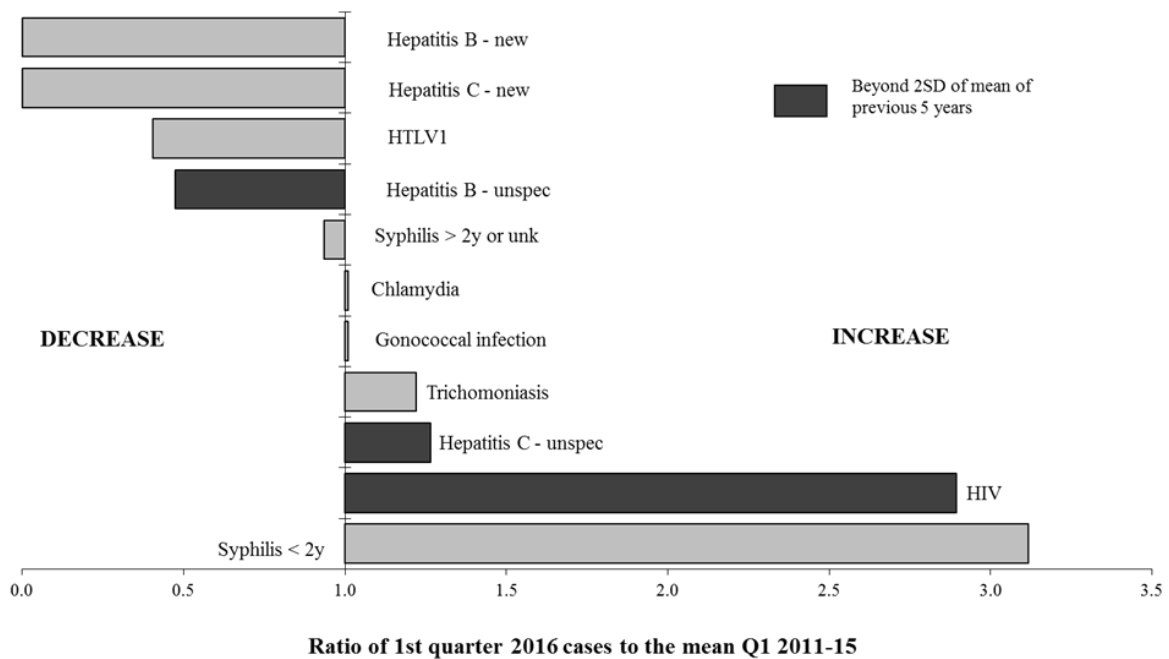
NT NOTIFICATIONS OF DISEASES BY ONSET DATE & DISTRICTS
January–March 2015 and 2016

	Alice Springs		Barkly		Darwin		East Arnhem		Katherine		NT	
	2016	2015	2016	2015	2016	2015	2016	2015	2016	2015	2016	2015
Acute post strep glomerulonephritis	4	8	0	3	2	4	1	4	4	2	11	21
Adverse vaccine reaction	1	3	0	1	6	3	0	0	1	2	8	9
Barmah Forest virus	0	0	0	0	5	12	2	0	0	0	7	12
Campylobacteriosis	29	12	2	0	74	68	6	12	13	8	124	100
Chickenpox	6	5	1	0	51	4	1	6	5	1	64	16
Chikungunya	0	0	0	0	0	3	0	0	0	0	0	3
Chlamydia	233	243	16	13	354	346	69	57	64	112	736	771
Chlamydial conjunctivitis	0	1	0	0	5	2	0	0	0	0	5	3
CJD	0	0	0	0	0	1	0	0	0	0	0	1
Crusted scabies	0	n/a	0	n/a	4	n/a	0	n/a	1	n/a	5	n/a
Cryptosporidiosis	40	4	9	4	69	12	4	6	5	2	127	28
Dengue	5	2	0	0	27	18	0	0	0	3	32	23
Food/water borne disease	0	0	0	0	3	0	0	0	0	0	3	0
Gastro - related cases	0	1	0	0	0	6	0	0	0	0	0	7
Gonococcal conjunctivitis	3	1	0	0	1	0	0	0	0	0	4	1
Gonococcal infection	276	268	25	17	114	112	50	36	52	87	517	520
Gonococcal neonatal ophthalmia	0	1	0	0	0	0	0	0	0	0	0	1
Group A strep invasive	7	6	4	1	5	4	1	1	2	1	19	13
Hepatitis A	0	0	0	0	0	2	0	0	0	0	0	2
Hepatitis B - chronic	0	2	0	0	0	1	1	4	0	1	1	8
Hepatitis B - new	0	0	0	0	0	1	0	0	0	0	0	1
Hepatitis B - unspecified	2	19	0	1	22	32	0	2	0	3	24	57
Hepatitis C - new	0	0	0	0	0	1	0	0	0	0	0	1
Hepatitis C - unspecified	10	12	1	0	55	37	0	0	1	2	67	51
H Influenzae b	1	0	0	0	0	1	0	0	0	0	1	1
HIV	1	0	1	0	9	3	0	0	0	0	11	3
HTLV1 asymptomatic/unspecified	2	10	0	1	3	1	0	0	1	1	6	13
Influenza	4	5	0	0	56	38	6	0	12	2	78	45
Legionellosis	0	0	0	0	0	1	0	0	0	1	0	2
Leprosy	0	0	0	0	0	0	1	0	0	0	1	0
Leptospirosis	0	0	0	0	0	1	0	0	0	0	0	1
Listeriosis	0	0	0	0	0	0	0	1	0	0	0	1
Malaria	0	0	0	0	4	2	1	0	0	0	5	2
Melioidosis	0	0	0	0	19	27	4	5	4	2	27	34
Mumps	2	0	3	0	1	0	0	0	3	0	9	0
MVE	0	0	0	1	0	0	0	0	0	0	0	1
Non TB Mycobacteria	0	0	0	0	1	2	0	0	0	0	1	2
Pertussis	1	1	0	0	11	12	0	0	0	1	12	14
Pneumococcal disease	4	5	1	0	4	4	0	1	0	3	9	13
Q Fever	0	1	0	0	0	0	0	0	0	0	0	1
Rheumatic fever	8	7	1	0	9	5	11	3	1	2	30	17
Ross River virus	3	7	1	1	77	150	5	9	11	17	97	184
Rotavirus	10	3	0	0	2	35	1	0	1	0	14	38
Salmonellosis	29	20	10	4	89	88	9	6	18	24	155	142
Shigellosis	31	16	8	0	23	14	10	24	9	2	81	56
Syphilis < 2y	18	18	0	0	13	6	1	0	11	15	43	39
Syphilis > 2y or unknown	3	18	0	1	9	6	2	0	3	11	17	36
Trichomoniasis	237	276	44	23	337	290	169	137	124	182	911	908
Tuberculosis	0	0	0	0	4	5	0	0	0	1	4	6
Typhoid	0	0	0	0	1	0	0	0	0	0	1	0
Typhus	0	0	0	0	0	1	0	0	0	0	0	1
Varicella - unspecified	2	1	0	0	0	0	0	0	0	0	2	1
Vibrio invasive	0	0	0	0	3	0	0	0	0	0	3	0
Yersiniosis	2	2	0	0	2	1	0	0	0	1	4	4
Zoster	17	8	1	1	63	72	7	12	2	4	90	97
Total:	991	986	128	72	1,533	1,434	362	326	347	493	3366	3311

Ratio of the number of notifications in the 1st quarter 2016 to the 5 year mean (2011–15): selected diseases



Ratio of the number of notifications in the 1st quarter 2016 to the 5 year mean (2011–15): sexually transmitted diseases



Comments on notifications

Hepatitis C

There were 67 cases of hepatitis C notified in the first quarter compared with expected 53. There was no evidence of increased spread of hepatitis C. The increase in notifications might have been due to new treatments becoming available which has encouraged those at risk or those previously diagnosed elsewhere to come forward for testing.

HIV

There were 11 cases of HIV notified in the first quarter compared with the expected 3.8 based on the 5 year mean. The increase was due to a combination of locally acquired cases and some acquired overseas.

Hepatitis B

There were 24 cases of hepatitis B notified during the first quarter compared with the 'expected' number of 51 based on the 5 year mean. An increasing proportion of the population is now immune due to the expansion of the vaccination program. A downward trend is therefore the true expectation as the people in the high risk groups are identified and notified in recent years.

Mumps

There were 9 cases on mumps in the first quarter. These cases were connected with the large outbreak that has been ongoing in Western Australia. It is likely that more cases will present over the next few months.

Chickenpox

There were 64 cases of chickenpox in the first quarter; 2.5 times the 5 year mean of 25. The increased rate of PCR testing of chickenpox cases may explain this increase. It is hoped that in the near future this will be confirmed by the availability of testing data.

Campylobacteriosis and shigellosis

In the first quarter of 2016, there were 121 cases of campylobacteriosis notified compared to the 5 year mean of 65 cases, a 90% increase. There were 81 shigellosis notifications compared to the 5 year mean of 40, a 100% increase. A large proportion of these increases can be attributed to the introduction of PCR testing which is much more sensitive. Recent data from the Darwin region indicates that at least 20% of campylobacteriosis cases in Darwin acquired their infection overseas.

Fact sheet and guideline update April–June 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

Below are the fact sheets that are new* or updated over April to June 2016.

- Diphtheria
- Legionella
- Mumps

- *Mumps information for General Practitioners

Below is an updated guideline which can be found at:

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

- NT Guidelines for the Management of Sexually Transmitted Infections in the Primary Health Care Setting



Centre for Disease Control

June 2016

Mumps

What is mumps?

Mumps is an infectious disease caused by the mumps virus. It was once very common in children, but due to vaccination it is now very uncommon.

How is it spread?

Mumps is spread through infected saliva or mucus from the mouth or nose. Spread occurs by coughing, sneezing, sharing utensils with others or touching surfaces contaminated with the virus.

What are the symptoms?

The symptoms generally develop 12 to 25 days after infection with the usual time being 16 to 18 days.

Common symptoms of mumps are fever, loss of appetite, tiredness and headaches, followed by swelling and tenderness of one or more salivary glands. The parotid salivary glands, located in the cheek at the jaw line below the ears, are most commonly affected.

About a third of infected people do not show any symptoms at all. People infected after puberty usually have more severe disease.

Complications of mumps are uncommon but can include;

- Hearing loss
- Inflammation of the brain (encephalitis)
- Inflammation of the covering of the brain and spinal cord (meningitis)
- Inflammation of the testicles (orchitis)
- Inflammation of the ovaries (oophoritis)
- Spontaneous abortion

Sterility (inability to have children) in males following orchitis is however extremely rare.

What is the infectious period?

A person can be infectious 7 days before the swelling until 9 days after the swelling of the salivary glands. People are most infectious from 2 days before until 4 days after the onset of symptoms.

Who is at risk?

Mumps can affect any age group, but is more severe in those post-puberty. Vaccination or one episode of the disease usually produces long-term immunity.

What is the treatment?

There is no specific treatment for mumps. Simple analgesics can help reduce pain and fever. Rest and drinking plenty of fluids is important. Warm or cold packs to the swollen glands may help.

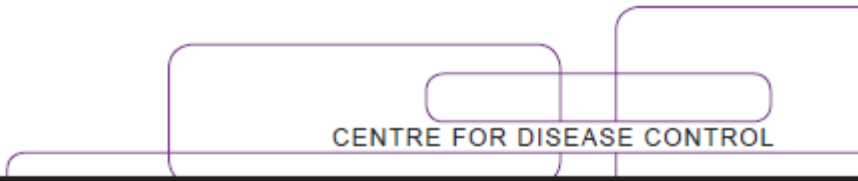
How can mumps be prevented?

Mumps can be prevented by immunisation. Mumps containing vaccine prevents most cases of mumps and decreases the severity of the illness if acquired.

The measles, mumps, rubella (MMR) vaccine is part of the routine childhood immunisation schedule at 12 months of age and MMRV (measles, mumps, rubella, varicella) vaccine is given at 18 months of age.

People born during or after 1966 should ensure they have received 2 doses of a mumps containing vaccine.

MMR and MMRV vaccines contain a live attenuated virus and should not be given during pregnancy or to women contemplating pregnancy. Pregnancy should be avoided for 28 days after vaccination.



CENTRE FOR DISEASE CONTROL

How can it be controlled?

People with mumps should stay away from work, school and childcare for 9 days following the onset of swelling of the salivary glands or until the swelling goes down, whichever occurs first.

Laboratories, doctors, school principals and directors of child care centres are required to report all cases of mumps to the local Centre for Disease Control.

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

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

or

<http://www.nt.gov.au/health/cdc>



Northern
Territory
Government



Centre for Disease Control

June 2016

Mumps Information for General Practitioners

It is important to recognise the early symptoms and signs of mumps to prevent further spread of this highly contagious illness in the community. The recent outbreak in Western Australia has seen approximately 9% of cases hospitalised and 6% of cases develop the complication of orchitis. The recent increase in cases has mainly been in Indigenous Australians.

What are the important diagnostic features?

Mumps frequently has a non-specific prodrome including malaise, anorexia, low grade fever and myalgia leading to acute onset of uni or bilateral, tender, self-limited swelling of the parotid or other salivary gland. Careful history taking to determine the susceptibility, exposure and symptoms can assist diagnosis. Here is a list of the important features of mumps:

Susceptibility

Those susceptible include:

- Anyone who is born after 1965 who is not fully vaccinated with 2 MMR (Measles-Mumps-Rubella) vaccines. Some people from overseas may have received the measles vaccine without the mumps component. In Australia, mumps vaccine became available in the early 1980s. Two doses of MMR vaccine were given after 1994.
- Babies 6-12 months when the maternal antibodies have declined but babies have not yet been immunised.
- Anyone who received their first dose of MMR prior to 12 months of age and has not received 2 subsequent doses of MMR containing vaccine after 12 months
- Anyone who is living with someone who has been diagnosed with mumps.

- People who are immunocompromised are also 'at risk' at any age, even if immunised.

Mumps can occur in those who are fully immunised but MMR vaccination has a protective effect on the severity of mumps disease.

Two doses of the vaccine are 88% (range: 66-95%) effective at preventing mumps; one dose is 78% (range: 49%-92%) effective.

Exposure

To acquire mumps a susceptible individual must be exposed to an infectious case although asymptomatic cases do occur. Mumps generally does not circulate in the NT, although recent cases have occurred in Katherine and Central Australia regions. People generally develop symptoms of the disease after 12-25 days with the usual timeframe being 16 to 18 days after having been exposed to an infectious person.

Individuals are usually infectious up to 7 days before and up to 9 days after onset of parotitis.

Approximately one third of infected people are asymptomatic.

A prodrome

The prodrome is usually non-specific, consisting of low-grade fever, malaise, headache, myalgias and anorexia. These symptoms or rarely lower respiratory symptoms in young children are the only manifestations in up to 50% of cases. The prodrome usually occurs approximately 48 hours before classical symptoms of parotid or other salivary gland tenderness and/or swelling occur.

Parotitis

Parotitis occurs in approximately 65-70% of symptomatic cases and is caused by direct infection of the ductal epithelium with associated local inflammation. This is usually

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Mumps information for General Practitioners

preceded by local parotid tenderness and occasionally earache. Parotid enlargement of the contralateral gland occurs in approximately 90% of cases but this may be delayed by several days and the swelling can last for up to 10 days.

Complications

Mumps can have some serious complications that need to be considered. Complications are reduced in those who have been vaccinated. These include:

- **Orchitis:** Epididymo-orchitis occurs in up to 15-20% of post-pubescent males and is characterized by high fevers, severe testicular pain with erythema and swelling of the scrotum.
- **Oophoritis:** Occurs in up to 7% of post-pubescent girls.
- **Aseptic meningitis:** Is the most frequent extrasalivary complication of mumps but is usually asymptomatic (up to 50%) with only approximately 4-6% showing clinical signs.

Less common neurologic complications

These include:

- Encephalitis, deafness, Guillain-Barre syndrome, transverse myelitis and facial palsy.

Less common end-organ syndromes linked to mumps

- Thyroiditis, myocardial involvement, pancreatitis, interstitial nephritis and arthritis.

What is the public health response to a suspect mumps case?

The best test for mumps is a PCR on a throat or buccal swab. Notify Centre for Disease Control (CDC) immediately on 8922 8044 that mumps is suspected and tests have been taken.

Isolate the case at home until at least 9 days after the onset of swelling of the salivary glands, or until the swelling goes down, whichever is sooner, at which time the case is no longer considered infectious.

It is best to avoid alerting schools and child care until the case can be confirmed. CDC will assist in alerting appropriate institutions and contacts.

Ensure patients are up to date with the mumps vaccination. Babies are due at 12 and 18 months of age (MMR or MMRV). All adults born after 1966 should have had 2 doses of mumps containing vaccine (now given as MMR).

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

Alice Springs	8951 7540
Darwin	8922 8044
Katherine	8973 9049
Nhulunbuy	8987 0357
Tennant Creek	8962 4259
or	
http://www.nt.gov.au/health/cdc	

Immunisation coverage for children aged 12–<15 months at 31 March 2016

Region	Number in district	%DTP	%Polio	%HIB	%Hep	%Pneumo	%Fully vaccinated
Darwin	304	93.8%	93.8%	93.8%	93.8%	94.1%	93.8%
Winnellie PO Bag	61	95.1%	95.1%	95.1%	96.7%	93.4%	93.4%
Palmerston/Rural	267	93.6%	93.3%	92.9%	93.6%	94.0%	92.9%
Katherine	68	98.5%	98.5%	98.5%	98.5%	98.5%	98.5%
Barkly	26	92.3%	92.3%	92.3%	92.3%	88.5%	88.5%
Alice Springs	106	91.5%	91.5%	91.5%	90.6%	91.5%	90.6%
Alice Springs PO Bag	46	97.8%	97.8%	97.8%	97.8%	97.8%	97.8%
East Arnhem	30	96.7%	96.7%	96.7%	96.7%	96.7%	96.7%
NT	908	94.2%	94.1%	93.9%	94.2%	94.2%	93.6%
Non-Indig (NT)	618	94.5%	94.3%	94.2%	94.3%	94.7%	94.0%
Indigenous (NT)	290	93.4%	93.4%	93.4%	93.8%	93.1%	92.8%
Australia	76564	93.7%	93.7%	93.3%	93.6%	93.4%	92.8%

Immunisation coverage for children aged 24–<27 months at 31 March 2016

Region	Number in district	%DTP	%Polio	%HIB	%Hep	%MMR	%MenC	%Varicella	%Fully vaccinated
Darwin	270	95.6%	95.6%	94.1%	95.6%	88.9%	94.1%	88.5%	86.7%
Winnellie PO Bag	48	95.8%	95.8%	93.8%	95.8%	95.8%	93.8%	95.8%	93.8%
Palmerston/Rural	263	96.6%	96.6%	95.8%	97.7%	94.7%	95.8%	93.5%	92.4%
Katherine	64	98.4%	98.4%	98.4%	98.4%	95.3%	98.4%	92.2%	92.2%
Barkly	25	92.0%	92.0%	92.0%	92.0%	84.0%	92.0%	76.0%	76.0%
Alice Springs	109	99.1%	99.1%	97.2%	99.1%	97.2%	97.2%	95.4%	93.6%
Alice Springs PO Bag	33	93.9%	93.9%	93.9%	93.9%	87.9%	93.9%	90.9%	87.9%
East Arnhem	31	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%
NT	843	96.6%	96.6%	95.5%	96.9%	92.9%	95.5%	91.8%	90.4%
Non-Indigenous (NT)	559	96.2%	96.2%	95.2%	96.6%	93.9%	95.9%	93.2%	91.9%
Indigenous (NT)	284	97.2%	97.2%	96.1%	97.5%	90.8%	94.7%	89.1%	87.3%
Australia	75798	95.9%	95.9%	95.0%	95.7%	92.4%	94.8%	92.4%	90.8%

Immunisation coverage for children aged 60–<63 months at 31 March 2016

Region	Number in district	%DTP	%Polio	%MMR	%Fully vaccinated
Darwin	265	95.5%	89.4%	89.4%	87.2%
Winnellie PO Bag	64	93.8%	89.1%	89.1%	87.5%
Palm/Rural	254	98.0%	95.7%	95.7%	94.5%
Katherine	78	98.7%	96.2%	92.3%	91.0%
Barkly	14	100.0%	85.7%	92.9%	85.7%
Alice Springs	132	95.5%	92.4%	93.9%	92.4%
Alice Springs PO Bag	35	97.1%	94.3%	94.3%	94.3%
East Arnhem	36	97.2%	88.9%	91.7%	88.9%
NT	878	96.6%	92.4%	92.5%	90.8%
Non-Indigenous (NT)	571	95.6%	90.7%	91.9%	89.3%
Indigenous (NT)	307	98.4%	95.4%	93.5%	93.5%
Australia	75722	96.7%	93.5%	93.5%	92.8%

Immunisation coverage at 31 March 2016

Charles Strebtor, CDC, Darwin

Background information to interpret coverage

Children were assigned to regions based on the postcode taken from their Medicare address listed in the Australian Childhood Immunisation Register (ACIR). Children with a PO Box address listed are counted among that PO Box postcode, and not assigned to a region. Winnellie PO Bag is postcode 0822, which includes most Darwin Rural District communities, some East Arnhem District communities and some residents of the Darwin 'rural area' who collect mail from the Virginia store or Bees Creek. Alice Springs PO Bag is postcode 0872, which includes Alice Springs District, Nganampa and Ngaanyatjarra communities.

The cohort of children assessed at 12 to <15 months of age on 31 March 2016 were born between 1 September 2014 and 31 December 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 March 2016 were born between 1 September 2013 and 31 December 2013 inclusive. To be considered fully vaccinated, these children must have received meningococcal C vaccination (given at the 12 month schedule point), and a second dose of measles, mumps, rubella (MMR) and the first dose of the varicella vaccination (given in combination as MMRV at the 18 months schedule point). All vaccinations must have been administered by 24 months of age.

The cohort of children assessed at 60 to <63 months of age on 31 March 2016 were born between 1 September 2010 and 31 December 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

Immunisation coverage rates for NT children by regions as estimated by the ACIR are shown on page 35.

The vaccination coverage rates for children in the NT are comparable with the national average for all age cohorts: 12 <15 months cohort (NT 93.6%, National 92.8%); 24 to <27 months cohort (NT 90.4%, National 90.8 %); and for the 60 to <63 months cohort (NT 90.80%, National 92.8%).

Indigenous children were less likely to be fully immunised than non-Indigenous children in the 12 to <15 month cohort (Indigenous 92.8%, non -Indigenous 94.0%) and in the 24 to <27 month cohort (Indigenous 87.3%, non-Indigenous 91.9%) but more likely to be fully immunised in the 60 to <63 month cohort (Indigenous 93.6%, non-Indigenous 89.3%).

The inclusion of the 3 additional immunisations for the 24 to <27 month cohort 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 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.

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 April–June 2016

Top End

Christian James (see photo) has commenced a 6 month contract as Nursing Advisor Public Health to fill the position vacated by **Lesley Scott**. He comes to CDC after working at RHD Australia for the last 2 years. He has worked as a nurse in remote and urban centres across the NT, and brings skills in innovation, technology and contemporary public health. **Lesley Scott** is now working part-time as a Nursing Project Consultant for CDC.

Photo: Christian James



Jiunn-Yih Su has resigned as the Project Officer at the Sexual Health and Blood Borne Virus Unit and is moving to Perth. **Linda Garton**, Darwin Remote Sexual Health Manager, has been successful in winning Jiunn-Yih's position and will be moving to the position on 1/8/16. Welcome to **Padaila Mudu** who is the new Administration Officer at Clinic 34.

Central Australia

Aboriginal Health Practitioner **Daniel Williams** has joined the Remote Sexual Health team. **Anne Correy** has re-joined the team as a Remote Sexual Health Nurse.

Kate Wales, Public Health Nurse Hepatitis started maternity leave in June and **Helen Tindall** Public Health Nurse Tuberculosis has backfilled Kate's position. **Jacqui Arnold**, Public Health Nurse Trachoma, has backfilled Helen's position.

NT Guidelines for the Management of Sexually Transmitted Infections in the Primary Health Care Setting

This guideline is designed to be used as a comprehensive reference guide for sexual health service delivery for primary health care clinicians. All clinicians should refer to the above guidelines for more detailed information in STI/BBV testing and management. You will find the Guideline at: http://www.health.nt.gov.au/Centre_for_Disease_Control/Publications/CDC_Protocols/index.aspx

Save the date
Northern Territory Centre for Disease Control
2016 Conference
Communicating messages:
Thinking inside the culture
Michael Long Learning and Leadership Centre
6-8 September, Darwin



Northern
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MAKE SURE YOUR CHILDREN
ARE **PROTECTED** AGAINST THE

FLU



FREE flu (influenza) vaccine available for
Indigenous children aged 6 months to <5 years

NOW

- Flu is an infection that spreads easily by coughing and sneezing.
- The flu vaccine is the safest and best way to fight the spread of flu.
- It is strongly recommended that you get a flu vaccine for yourself and your child every year.

The flu vaccine is available **FREE** from your local health centre

For more information contact your nearest Centre for Disease Control

Darwin 8922 8044 • Katherine 8973 9049 • Tennant Creek 8962 4259
Alice Springs 8951 7549 • Nhulunbuy 8987 0357

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