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Summary of influenza in 2009 in the Northern Territory

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Abstract

Aims

This paper aims to summarise the 2009 influenza season in the Northern Territory, including a comparison of regional rates (both within the NT and with national figures), a description of the H3N2 epidemic and an assessment of the impact of the local pandemic response.

Method

Laboratory-confirmed influenza rates were calculated using data from the NT Notifiable Diseases System and hospitalization data from the national web-based NetEpi system. Assumptions were made for further estimates. Data concerning influenza-like illness were extracted from the Emergency Department Syndromic Surveillance System and the Territory Influenza Surveillance System. Testing data were obtained from the Royal Darwin Hospital.

Results

There were 1,584 cases of notified pandemic (H1N1) 2009 influenza, a rate of 677 per 100,000 which was 4 times the national rate. The NT rate ratio of Indigenous/non-Indigenous for the notified pandemic strain was 4.9 and for hospitalisation of pandemic cases, 12.3. There were 6 deaths. There were an estimated 15,900 cases of the pandemic strain with an overall estimated attack rate of 7.2% (3.3% in non-Indigenous and 16.3% in Indigenous people). Alice Springs Rural region had the highest rate. There were also 331 cases of H3N2 strain notified (with 1 death), almost exclusively in the Top End and preceding the pandemic strain.

Conclusions

The NT recorded notification and hospitalisation rates of pandemic (H1N1) 2009 influenza that were higher than the corresponding national rates. The Indigenous population was disproportionately affected by this pandemic. Public health interventions in the CONTAIN phase appeared to have impacted on transmission.

Keywords; pandemic, influenza, Northern Territory.

Contents

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Introduction

The impact of the pandemic (H1N1) 2009 influenza in the Northern Territory (NT) has been previously discussed in issues of the *Disease Control Bulletin* and elsewhere.[1,2,3] Nevertheless, with both the 2009 season and the H1N1 pandemic vaccination campaign well behind us, we can now further analyse the influenza statistics in the NT for 2009. In particular, there have been some questions which remain unanswered, such as;

- Given that the number of laboratory-confirmed cases was influenced by the amount of testing being done and that testing decreased over time, can we estimate how many total cases of pandemic (H1N1) 2009 influenza there were in the NT?
- How did the NT rates of confirmed cases for the pandemic compare to the rest of Australia and how might we explain the difference?
- Did attack rates vary geographically and what were the rates in remote communities?
- What was the impact of the CONTAIN strategy in the NT?
- What was the epidemiology of the A/H3N2 strain in the NT?

Some of these questions are difficult to answer with any certainty and may lead to yet more questions about the nature of the H1N1 pandemic; however 2009 was such an unusual year with respect to the epidemiology of influenza and its clinical and public health impact that further analysis is warranted. Moreover, we can now draw together all the information sources to allow a better understanding of both the data and the epidemiology. This paper seeks to use available surveillance and testing data to summarise the epidemiology of the pandemic and other influenza strains in the NT in 2009, as compared with national statistics and at different phases of disease control.

Methods

The pandemic (H1N1) 2009 influenza strain

Rates were calculated using laboratory-confirmed data from the NT Notifiable Diseases System (NTNDS) and hospital admission data from the national web-based surveillance system used during the pandemic (NetEpi). National rates were those reported in the Australian Influenza Surveillance Report.[4] The NT population data used for rate calculation were estimated resident population (ERP) data for 2008 prepared by the NT Department of Health and Families.[5]

The total number of pandemic influenza cases in the NT was estimated using the following method. The total number of influenza-like illness (ILI) cases during each week of the pandemic was estimated by multiplying the number of ILI cases from the GP sentinel system

Assumptions in the calculations of total H1N1 cases

- The sample of GPs (6) is representative of the NT GP population.
- Number of consultations seen per GP full time equivalent=125.
- Number of FTE GPs or equivalent primary health care providers in the NT=165.[a7]
- The ratio of the number of ILIs in Indigenous people presenting to primary health care compared to non-Indigenous= 2.7[b].
- The positivity rate of testing through the RDH laboratory represented the rate in the NT. It remained constant from 30 August until 30 September (no data available after the end of August).
- The proportion of ILI sufferers who attend their GP was 0.7 between 28 May and 17 June, 0.6 between 18 June and 7 July, 0.5 between 9 July and 29 July, 0.4 between 30 July and 19 August and 0.3 between 20 August and 30 September.

[a] This is the number of FTE GPs published on the Primary Health Care Research and Information website,[7] plus a figure (50) which was estimated by the NT GP Network staff of the shortfall in the Indigenous remote communities.

[b] This is the Indigenous/non-Indigenous ratio of the number of influenza tests processed in the RDH laboratory (June-September). The assumption is that the ratio in ILI presentations would be equivalent.

seen per full-time equivalent (FTE) GP by the estimated number of FTE GPs (or primary care equivalent) in the NT. To this was added the number of excess ILI cases (above the baseline) seen in the hospitals' Emergency Departments (EDs). This was then multiplied by the positivity rate (proportion of tests positive for pandemic influenza) calculated from testing data from Royal Darwin Hospital (RDH) for that week. Finally, this number (the total number of pandemic cases seen in primary care or ED) was scaled up according to estimates of the proportion of influenza cases who would have sought medical care. It was thought that this was likely to have been 70% at the beginning of the pandemic and 30% at the end and is based on estimates made in the UK.[6]

Given that GPs who contribute to the sentinel GP surveillance system see mainly non-Indigenous patients and that all systems suggest that the rate of both ILI and pandemic influenza was higher in the Indigenous population,[2] an adjustment to allow for this was necessary. The total ILI figure was therefore adjusted on the basis that the Indigenous ILI rate was greater than the non-Indigenous by a factor equal to the ratio of influenza testing rates at RDH for the 12 peak weeks of the pandemic (2.7). That is, the Indigenous/non-Indigenous testing rate ratio was assumed to be equivalent to the ILI rate ratio on the grounds that a test was an indication of an ILI. Testing data from the non-NT government laboratories were not available. The assumptions are summarised in the Box.

The numbers of pandemic cases were estimated per week and summed over the 18 weeks from the beginning of June to the end of September. The Indigenous and non-Indigenous attack rates were calculated by extrapolating the total number of cases against the ERP using the known rate ratio between Indigenous and non-Indigenous laboratory confirmed cases (4.9 unadjusted).

Geographic distribution

The rates of laboratory-confirmed cases for each epidemiological district and statistical subdivision were calculated using the 2008 ERP[5] and the 2006 census data respectively.[8] Community specific rates were calculated using the 2006 Census QuickStats.[8] Communities were chosen if they had at least 1 case of confirmed pandemic (H1N1) 2009 influenza, their population was available on the website and was at least 150. Rates were plotted against the median date of the pandemic (or the date of the "middle" case) for each community using STATA statistical software (version 11).

The A/H3N2 influenza strain

Counts of laboratory-confirmed A/H3N2 influenza cases were derived from the NTNDS, analysed using Business Objects reporting software (version XI) and plotted on graphs using Excel 2003. Age and Indigenous specific rates were calculated using 2008 ERP.[5]

Impact of the CONTAIN strategy

To examine the impact of the CONTAIN strategy, the outputs of NT surveillance systems were plotted together. These included laboratory-confirmed cases notified under the NTNDS, the total influenza tests done at RDH, the numbers of ILI in the RDH ED and the rates of ILI in sentinel GPs. These were compared with the timing of the implementation of the CONTAIN phase strategies.

Results

Pandemic (H1N1)2009 Influenza strain

There were 2,084 cases of influenza notified in 2009, all but 6 of which were influenza A (Table 1). A significant proportion (5.5%) were influenza A not further defined while there were 49 H1N1 cases (2.3%) which were not strain-typed.

The rate of laboratory-confirmed pandemic (H1N1) 2009 influenza in the NT was 677 per 100,000 which was just under 4 times the national rate of 172.1 per 100,000. The NT non-Indigenous rate of 311 per 100,000 was 1.81 times the national rate and the Indigenous rate of 1522 per 100,000 was 8.84 times, while the NT rate ratio of Indigenous/non-Indigenous was 4.9. The non-Indigenous hospitalisation rate was 1.7 times the national rate of 22.8. Interestingly the rate in females was 1.22 higher than that of males. The non-Indigenous hospitalisation rate

of 38 per 100,000 and Indigenous rate of 468 per 100,000 gave an NT rate ratio of 12.3.[2] Six people, 4 males and 2 females with laboratory confirmed pandemic (H1N1) 2009 influenza died. Of these 4 were Indigenous and 1 was post-partum.

We estimate that there were 15,900 cases of pandemic influenza in the NT during June-September 2009 giving an overall attack rate of 7.2%. Assuming the Indigenous/non-Indigenous rate ratio calculated with laboratory-confirmed cases was the same as in the population, then there were 10,800 Indigenous cases and 5,100 non-Indigenous. The attack rate in the non-Indigenous population was 3.3% and in the Indigenous population 16.3%.

Geographic distribution

Analysis by epidemiological district revealed that Alice Springs Rural district had the highest rate of pandemic (H1N1) 2009 influenza (Table 2). Interestingly the regions with the highest rates in the non-Indigenous population where Katherine and East Arnhem while the highest in the Indigenous population were Alice Springs urban and rural. Analysis by statistical sub-division revealed that the Tiwi Islands had the highest rate (Figure 1).

A look at the rates of laboratory confirmed pandemic influenza in remote communities suggests that there was a marked spread in rates which was independent of the stage of the pandemic. Figure 2 shows the rates of confirmed cases plotted against the median date for the pandemic in each of 63 communities, each dot representing a community. There is, as expected, a statistically significant relationship between rates and time (p=0.017), reflecting the reduction in testing that went on during the PROTECT phase. However, this relationship becomes non-significant (p=0.054) if the communities with epidemics after the middle of August (by which time testing was at a minimum) together with an early outlier are excluded. In addition, the heterogeneity (spread) of the rates at all stages of the pandemic is obvious in Figure 2 and the reduction in rates over time is perhaps not as marked as expected. This suggests that testing rates may not have decreased until several weeks after the beginning of the PROTECT phase.

Table 1. Laboratory confirmed cases of influenza notified in the Northern Territory, 2009

Type	Subtype	Strain	n	%
A	H1N1	Mexico/223/2009	1,584	76.0
	H1N1		48	2.3
	H3N2		331	15.9
		Not further specified	115	5.5
B			6	0.3
Total			2,084	100.0

Table 2. Rates of laboratory-confirmed Pandemic (H1N1) 2009 Influenza by district per 100,000.

	Indigenous	Non-Indigenous	All
Alice Springs Rural	2651	304	2195
Alice Springs Urban	2843	235	786
Barkly	1435	135	978
Darwin Rural	1034	376	851
Darwin Urban	1183	304	401
East Arnhem	380	535	437
Katherine	1753	449	1147
All districts	1522	311	677

There is evidence looking at community specific epidemic curves (data not published) that, despite the high rates in some Indigenous communities, the public health response in the CONTAIN phase was successful in some communities in slowing down the spread and, in at least 1 community, of stopping the spread altogether. Importantly, there were no deaths in any NT remote Indigenous communities. The epidemic curves for each NT region are illustrated (Figure 3).

The A/H3N2 influenza strain

There were 331 cases of A/H3N2 influenza notified in 2009. The epidemic occurred in late May 2009 after the declaration of the DELAY phase of the pandemic and it was almost

Figure 1. Map showing the rates in pandemic (H1N1) 2009 influenza by statistical subdivision. Inset shows the rates in the statistical sub-divisions of Darwin City and surrounds

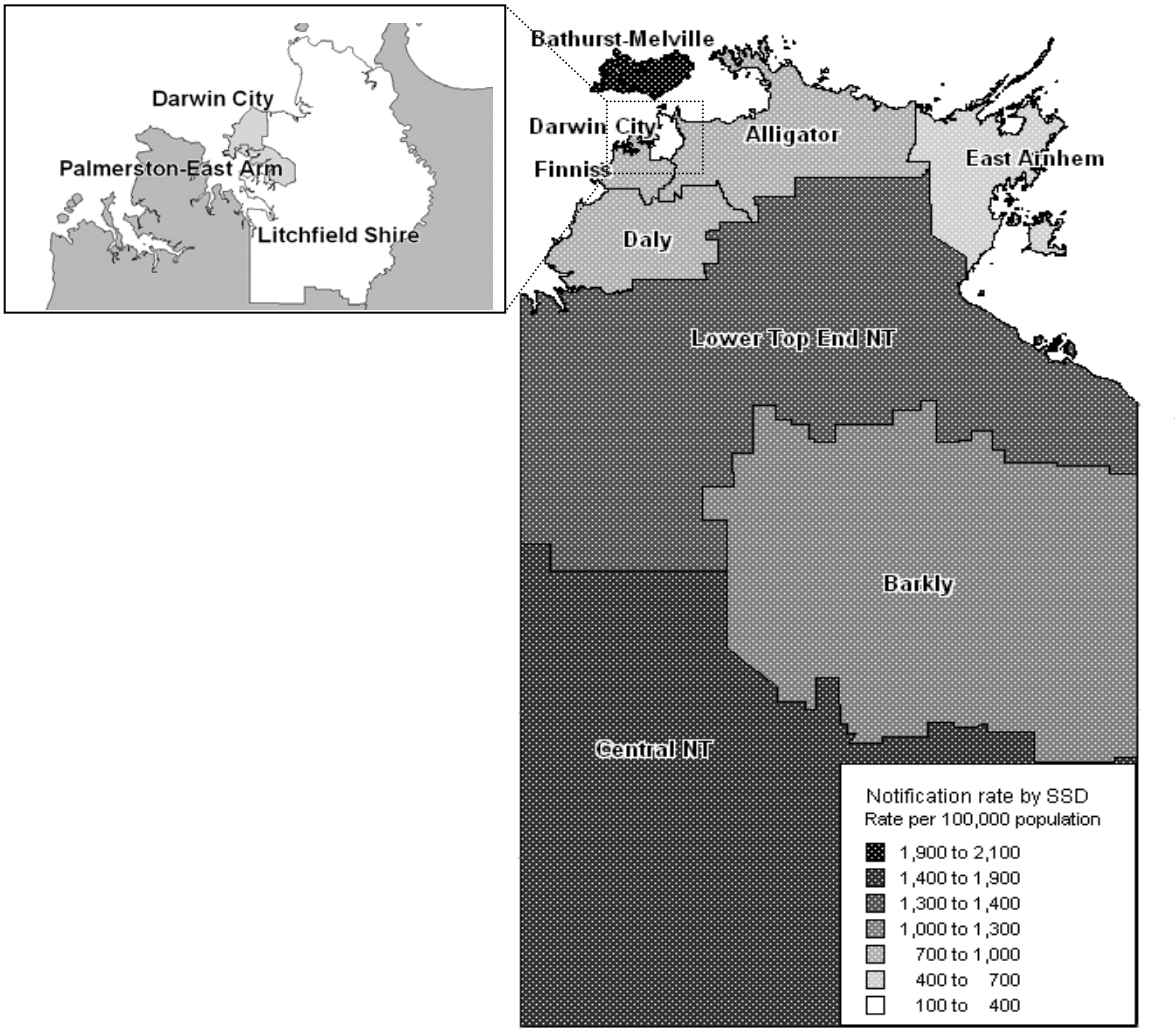
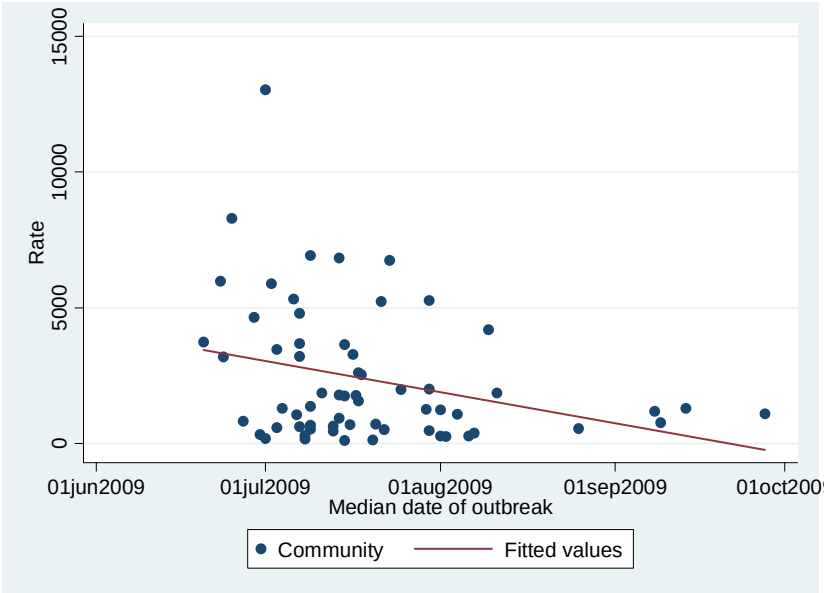


Figure 2. Scatter plot illustrating the relationship between the rate of pandemic (H1N1) 2009 influenza (per 100,000) and the median date of the outbreak 63 major NT communities and towns



exclusively confined to the Top End (Figure 4). The epidemic peaked in the week commencing 28 May in Darwin, 9 July in East Arnhem and 23 July in Katherine and in most cases preceded the H1N1 pandemic strain. This epidemic was also seen in other parts of the country (but not Central Australia) and often followed the H1N1 pandemic strain.[9]

In the Indigenous population the peak incidence was in the very young and the very old, while in the non-Indigenous the age-groups most affected were 5-24 year old age-groups (Figure 5). There was no difference in rates between the sexes (data not shown). There were 22 confirmed cases admitted to hospital representing 8.2% of all those with known hospitalisation status. One laboratory-confirmed case died of complications from the illness.

The impact of the CONTAIN phase

Figure 6 reveals that at the end of the CONTAIN phase (22 May – 16 June) ILI counts in the RDH Emergency Department, GP sentinel rates of ILI, confirmed influenza case numbers and the number of tests being requested were all falling. This suggests that the strategies on the CONTAIN phase, those of isolation, quarantining, extensive contact tracing, prophylactic antivirals and school closures, were successful in reducing the spread of the disease in the community.

Discussion

It is of interest that the NT had such high rates of notified pandemic (H1N1) 2009 influenza, almost 4 times the national rate compared to the rest of Australia. Even the non-Indigenous rate, both for laboratory-confirmed H1N1 pandemic strain and hospitalisations, was almost twice that of the national average. Testing data are incomplete but there is some evidence that the NT had a higher testing rate which might explain the higher number of confirmed cases, but even so, this would not explain the higher hospitalisation rate.

The higher rate in the Indigenous population might be due to several factors. It has been recognised that there was a degree of prior natural immunity to the pandemic strain in the Australian population, in particular in those over 60 years of age.[10] It might be that the Indigenous population had less of this natural immunity to the H1N1 pandemic strain due to remoteness and younger age. It is also possible that transmission was greater in the Indigenous setting due to an increased risk of contact between infectious cases and susceptible individuals; the so-called “force of infection”. This increase might be due to differences in infrastructure, housing, social and cultural interactions, education and hygiene measures that are often presented as explanations for the well documented higher rates of communicable

Figure 3. Cases of Pandemic (H1N1) 2009 Influenza by district of residence; NT 2009.

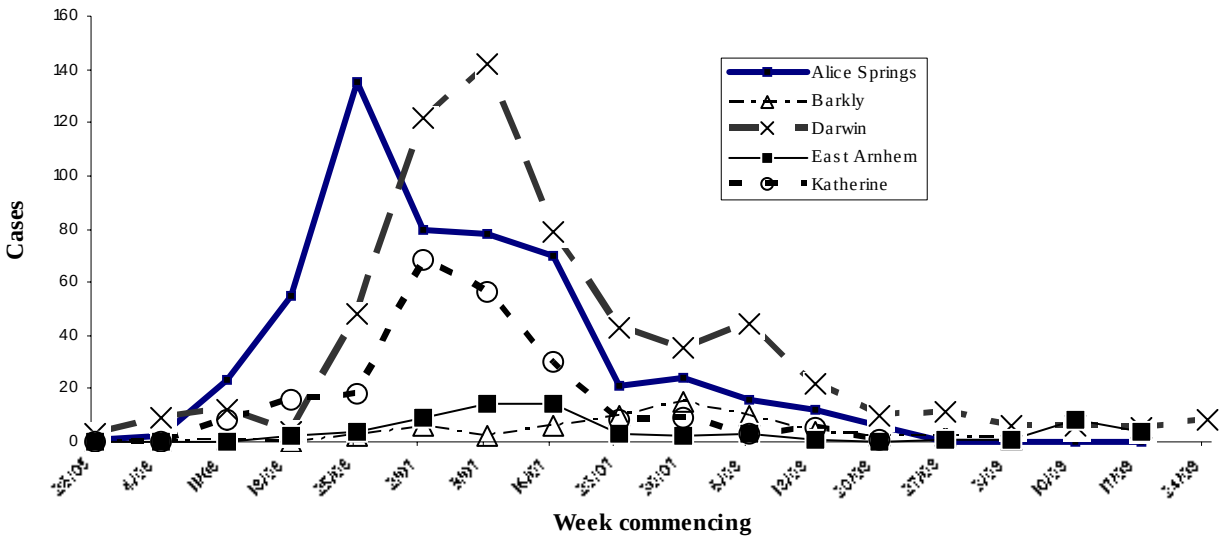


Figure 4. Cases of A/H3N2 influenza by district of residence; NT 2009

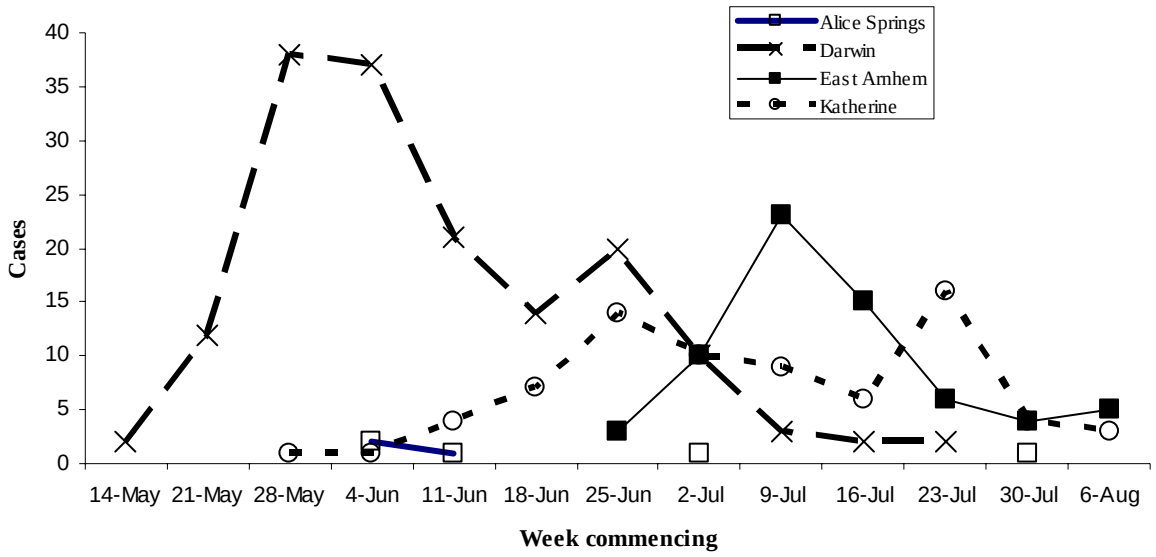
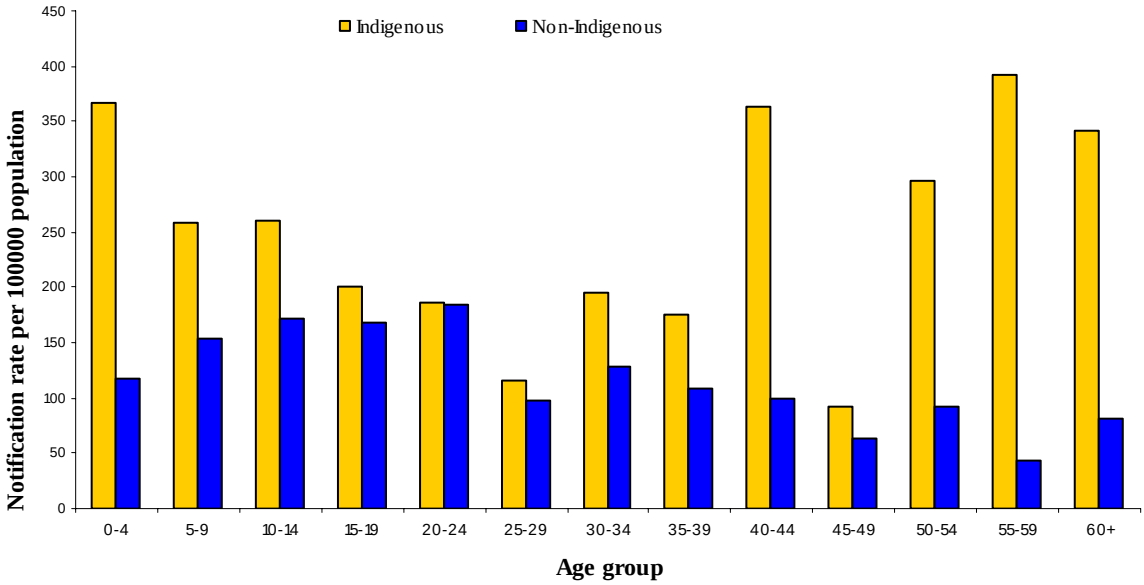


Figure 5. Rates of A/H3N2 influenza in the NT by age and indigenous status; 2009



diseases in the Indigenous setting. Given that 30% of the NT population is Indigenous, it is plausible that the force of infection in the non-Indigenous NT population, and therefore its incidence, is higher that that of other jurisdictions by virtue of its contact with the Indigenous population.

Whether the rate ratio between the 2 populations for laboratory-confirmed pandemic (H1N1) 2009 influenza was in reality as high as the measured 4.9 we can only speculate. While the limited testing data available did reveal a higher rate of testing in the Indigenous population, it was not higher per case detected and therefore cannot necessarily explain the difference.

Any attempt to estimate the total numbers of pandemic influenza cases in this setting is fraught with problems of unprovable assumptions. Neither confidence intervals nor a range of values were cited in the text because statistical uncertainty in this setting is dwarfed by the uncertainty relating to other non-statistical estimates rendering any range of values meaningless. The number hypothesized is a midpoint estimate based on a number of assumptions (see Box) and should be interpreted with caution. Attempts at this exercise have been made elsewhere with similar provisos.[6]

Even though the syndromic surveillance systems recorded levels higher than the usual seasonal influenza, the estimated total is likely to be an underestimate of the true figures.

Geographic comparisons of rates of laboratory-confirmed pandemic (H1N1) 2009 influenza need to be interpreted in the context of timing. After the middle of June testing was discouraged and, while it is difficult to measure changes in testing behaviour in the face of an evolving epidemic, rates of confirmed influenza in remote communities did decrease over time as is illustrated in Figure 2. This may partly explain the high rates in Central Australia and the Tiwi Islands where the pandemic spread first. However, the heterogeneity of the rates across NT communities persisted, and testing was still promoted after mid-June in those communities yet to be affected. Hence, while the low rates in the Indigenous population in East Arnhem might be explained by the lateness of the arrival of the pandemic in that region, the fact that the non-Indigenous population in East Arnhem had high rates relative to non-Indigenous rates elsewhere suggests that testing was still taking place. Hence it might be that remote East Arnhem rates were genuinely low.

The H3N2 influenza cases of May and June were indeed unusual. The total number of cases of H3N2 in the 2009 epidemic was in fact 70% higher than the previous highest annual figure for all influenza (199 in 2008). Of course, this is likely to have been biased by the amount of testing which took place because it coincided with the DELAY phase of the pandemic. Nevertheless, it is not likely that the H3N2 epidemic would have gone undetected without the pandemic being called, given that there were high rates in infants, the group which usually gets tested and heralds a seasonal epidemic. Notwithstanding the point that attendances for ILI to GPs or EDs might have increased due to anxiety about the H1N1 pandemic strain, there was an increase detected in syndromic surveillance systems during the H3N2 epidemic.

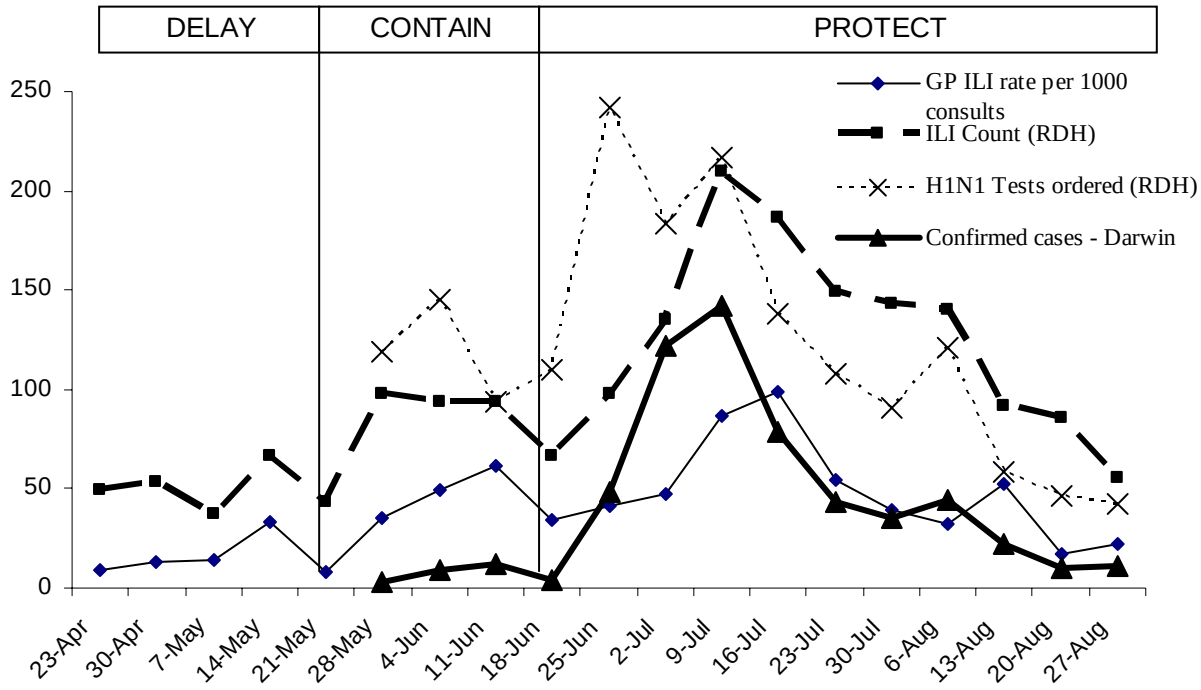
Prior to 2009, May and June were historically the 2 months with the lowest number of laboratory-confirmed cases. This is despite the fact that the NT often sees a spike in influenza cases in March and April. We can only speculate why 2009 May and June conditions should favour transmission of an influenza strain (most likely the A/H2N3/Brisbane/10/2007 strain) which had circulated in previous years.

It is important to recognise that the evidence presented here suggests that the public health response to the pandemic did have an impact on the spread, particularly in the Top End. How the rates would have gone had the CONTAIN phase persisted for several more weeks is hypothetical, but it suggests that, should a more virulent but no more transmissible virus emerge in the future, then public health responses of this nature will make a measurable impact on transmission. An analysis of the data with discussion about the population-specific effective reproduction numbers is planned.

Conclusion

In summary, the 2009 influenza season could be described as follows.

Figure 6. ILI rates, testing numbers and confirmed cases by phase of the pandemic and week commencing illustrating the fall in influenza cases during the latter part of the CONTAIN phase



- There was an H3N2 epidemic in late May to late June (historically the time period of lowest reported notified influenza cases), exclusively in the Darwin, Katherine and East Arnhem regions.
- For pandemic (H1N1) 2009 influenza-
  - The notification rate of confirmed cases was nearly 4 times higher in the NT than nationally and although NT testing rates may have been higher this does not explain the higher documented NT hospitalisation rate.
  - The NT non-Indigenous notification rate was 1.8 times the national rate and Indigenous rate 8.84 times with hospitalisation rates being 1.7 and 22.8, respectively.
  - There were 6 deaths, 4 of whom were Indigenous but no deaths in any NT remote Indigenous communities.
  - The estimated number of cases was 15,900 with an estimated attack rate in the non-Indigenous population of 3.3% and in the Indigenous population 16.3%.
  - There was a large degree of heterogeneity in the attack rates in the Indigenous communities.
  - The pandemic started simultaneously in Darwin and Alice Springs regions in early June but peaked earlier in Alice Springs at the end of June and tailed off more quickly. The Katherine region followed a similar pattern, peaking in early July between the peaks of Alice Springs and Darwin regions. The Barkly region had a biphasic pandemic. There was an early peak coinciding with that of the Katherine region but cases then tailed off until late July when there was a second larger peak. East Arnhem region peaked soon after the Darwin region but had a second peak in early September.
  - Public health interventions in the CONTAIN phase appear to have had a measurable impact on transmission.

Acknowledgements

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References

1. Flint S, Su J-Y, Scott L, Krause V. The early experience of pandemic (H1N1) 2009 influenza in the Northern Territory, Australia. *The Northern Territory Disease Control Bulletin* 2009;16 (2):1-8.
2. Markey P, Su J-Y. The epidemiology of pandemic (H1N1) 2009 influenza in the Northern Territory, June-September 2009. *The Northern Territory Disease Control Bulletin* 2009;16(3):1-7.
3. Flint SM, Davis JS, Su J, Oliver-Landry EP, Rogers BA, Goldstein A, et al. Disproportionate impact of pandemic (H1N1) 2009 influenza on Indigenous people in the Top End of Australia's Northern Territory. *Med J Aust* [Internet] 2010 [cited 2010 April 22] Available from http://www.mja.com.au/public/issues/192_10_170510/fli10103_fm.html.
4. Australian Government Department of Health and Ageing. *Australian Influenza Surveillance Report.* [Internet] 2010;8:9 [cited 2010 March 20] Available from: http://www.healthemergency.gov.au/internet/healthemergency/publishing.nsf/Content/ozflu2010.htm.
5. Health Gains and Planning. *Northern Territory resident Population Estimates by Age, Sex, Indigenous Status and Health Districts* (1971-2008). 2010 Darwin. NT Department of Health and Families.
6. Health Protection Agency. *Method used to estimate new pandemic (H1N1) 2009 influenza cases in England in the week 3 August to 9 August 2009.* [Internet] [cited 2010 March 20] Available from: http://www.hpa.org.uk/web/HPAwebFile/HPAweb_C/1250150839845.
7. Primary Health Care Research and Information Service. Key Division of General Practice characteristics, 2007-2008. [Internet] [cited 2010 March 24] Available from: http://www.phcris.org.au/products/asd/keycharacteristic/KeyDGPstatistics.xls
8. Australian Bureau of Statistics. Canberra. Census Data [Internet] 2010. [cited 2010 March 20. Available from: http://abs.gov.au/websitedbs/D3310114.nsf/home/Census+data.
9. Australian Government Department of Health and Ageing. *Australian Influenza Surveillance Report.* [Internet] 2009;12:1-4 [cited 2010 March 20] Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-ozflu-31-7-09.htm.
10. Greenberg ME, Lai MH, Hartel GF, Wichems CH, Gittleson C, Bennet J, et al. Response after one dose of a monovalent influenza A (H1N1) 2009 vaccine – Preliminary report. *N Engl J Med* 2009;361(25):2405-13.

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More on Flu:
News on the seasonal influenza vaccine 2010

Andre Wattiax, CDC Darwin

As influenza viruses often mutate and change, there is a need to update the influenza vaccine yearly. Every year the seasonal influenza vaccine contains 3 different strains predicted to be the most commonly circulating. A global system of surveillance called Global Influenza Surveillance Network (GISN) exists to monitor virus activity throughout the world. Based on reports from the GISN, consultations take place twice a year, one for the southern hemisphere and one for the northern hemisphere, to inform the World Health Organization (WHO) recommendations on the composition of the vaccine for that particular season.

This year WHO recommends that influenza vaccines for the southern hemisphere contain:

- a A/California/7/2009 (H1N1)-like virus [this covers the pandemic (H1N1) 2009 influenza strain];
- a A/Perth/16/2009 (H3N2)-like virus; and
- a B/Brisbane/60/2008-like virus.

This year's influenza season in Australia is marked by 2 major issues. Firstly, it follows last year's emergence of the pandemic (H1N1) 2009 influenza strain and the related vaccination effort which began in September 2009 with a vaccine produced to protect against this new strain. Secondly, the group eligible for FREE seasonal influenza vaccine in Australia has been expanded to reflect the recommendations contained in the *Australian Immunisation Handbook 9th edition.*

This change means that people at increased risk of complications from influenza infection will now all have access to FREE seasonal influenza vaccination. In 2010, people eligible for Commonwealth-funded seasonal influenza vaccine include:

- Pregnant women;
- Indigenous people over 15 years of age (previously over 50 years of age);
- Children aged 6 months to 10 years who receive long term aspirin:
- All people over 65 years of age; and
- All people 6 months of age and over with medical conditions predisposing to severe influenza.

Information about medical conditions predisposing to severe influenza can be found in the influenza section of the *Australian Immunisation Handbook 9th edition* (pp. 190-191) and include:

Heart problems including

- rheumatic heart disease priority 1 & 2 patients
- cyanotic congenital heart disease
- coronary artery disease
- congestive cardiac failure.

Chronic lung/breathing problems including

- severe asthma
- suppurative lung disease
- bronchiectasis
- cystic fibrosis
- chronic obstructive pulmonary disease
- chronic emphysema.

Chronic illness requiring medical follow-up or hospitalisation in the preceding year including

- diabetes mellitus
- chronic metabolic diseases
- chronic renal failure
- haemoglobinopathies
- impaired immunity including drug-induced immune impairment.

Chronic neurological problems including

- multiple sclerosis
- spinal cord injuries
- seizure and neuromuscular disorders.

People with lowered immunity including

- HIV
- malignancy
- chronic steroid use.

People not eligible for the FREE seasonal influenza vaccine can purchase the vaccine at their own cost. The seasonal influenza vaccine can be safely received by people who had the

Panvax® vaccine (the vaccine for the pandemic (H1N1) 2009 influenza strain) and it will protect them against the 2 other strains predicted to be commonly circulating this influenza season.

Additionally, all Australians over 6 months of age can still receive the FREE Panvax® vaccine. We are still actively promoting the FREE Panvax® vaccine to all adults and children over 6 months of age who are not eligible for the FREE seasonal influenza vaccine as the pandemic (H1N1) 2009 influenza virus is likely to be the predominant strain this year.

Information to healthcare providers has been issued to update them on the 2010 seasonal flu vaccine and the new 'at risk' groups as well as to encourage them to offer Panvax® to those not eligible for the FREE 2010 seasonal flu vaccine.

Do not hesitate to contact the Centre for Disease Control if you have any queries.
Darwin (08) 8922 8044
Katherine (08) 8973 9049
Tennant Creek (08) 8962 4259
Alice Springs (08) 8951 7540
Nhulunbuy (08) 8987 0357

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Top End TB Outbreak Update
A Household Connection – What's been happening?

Liz Stephenson, CDC Nhulunbuy

Abstract

In 2008 an outbreak of tuberculosis (TB) in a Northern Territory indigenous community occurred, where over a 10 month period 9 cases were notified, 8 cases from 1 house.

The 2 main public health actions taken in response to this outbreak were;

1) contact tracing, where contacts were identified and initial screening revealed 5 active TB cases and 17 contacts were found to have latent TB infection (LTBI) of whom 15 started on preventive treatment

2) a community/camp screen was carried out where 267 individuals were identified for screening and 253 have received a Mantoux test and/ or a chest X- ray (CXR) and clinical review and 1 TB case was diagnosed. Directly observed therapy was given to active TB cases to assist them to complete curative TB treatment and also for those being treated for LTBI to prevent progression to disease.

Ongoing follow-up in the community will continue for 1 more year.

Keywords: tuberculosis, Indigenous, outbreaks, households.

Introduction

A tuberculosis (TB) outbreak in 2008 in a Northern Territory (NT) Top End community has required a co-ordinated and ongoing response. In the 10 years from 1998 to 2007, this community of approximately 1000 Aboriginal people had 3 cases of TB diagnosed, with the most recent case in 2005.[1] In 2008, a total of 9 cases were notified in this Top End community, 8 cases from 1 house and 1 case from a different household in a separate camp. In 2009 a 10th case of TB was diagnosed from the community but from a different camp again.

Background

The 2 main public health actions taken in response to this TB outbreak were:

1. Contact tracing for the 3 initial TB cases.

   This action resulted in:

   - 23 additional household members identified as contacts
   - 5 of the 23 members identified as further cases of TB
   - 15 of the 17 members identified as Mantoux positive and diagnosed with latent TB infection (LTBI) were commenced on latent TB infection (LTBI) treatment.

2. A community screen as recommended in the *Guidelines for the Control of Tuberculosis in the Northern Territory*[2] when secondary cases are detected in routine contact tracing or when 2 or more cases of active TB are diagnosed within 1 year in a community.

   This action resulted in:

   - an initial Community Screen being undertaken in June 2008
   - 267 individuals identified for follow up with a Mantoux test, or CXR and clinical review[1]
   - no further cases of TB identified
   - TB and germ theory education in Yolngu language provided by Aboriginal Resource Development (ARDS) Staff to the community
   - follow up screenings as per *Guidelines.*[2.]

Follow-up Community Screening

Follow-up community screens including CXRs and clinical reviews as per *Guidelines*[2] have been provided in November 2008, April 2009, September 2009 and December 2009, by visiting Centre for Disease Control (CDC) staff, a visiting radiographer from Darwin, in collaboration with the community health centre (CHC) staff and the community.

The number in the community to be screened, according to the camp/community list currently stands at 267, of whom:

- 174 (65%) individuals have had at least 1 CXR and clinical review by a CDC medical officer
- 196 (73%) have been discharged as their Mantoux tests were negative (less than 10mm in adults, less than 5mm in children) as per *Guidelines* [2]
- 23 (9%) have been discharged after CXRs and clinical reviews by a CDC Medical Officer
- 12 (4.5%) are currently over-due for a CXR and clinical review
- 16 (6%) are due for a CXR and clinical review in 2010
- 4 (1.5%) have commenced or are considering a 9 months course of LTBI treatment
- 14 (5%) Mantoux tests are still outstanding, as these individuals are currently unable to be located in the NT, some had Mantoux tests administered but were not found to have the result measured and recorded.

TB contact tracing including Mantoux testing and then CXRs and clinical reviews for all Mantoux positive cases was also provided to contacts of Case 9 (12 positive Mantoux tests from 47 performed) and Case 10 (9 positive Mantoux tests from 64 performed).

The index cases and their contacts

All 8 individuals from the 1 household who were diagnosed with TB have completed their directly observed therapy (DOT) TB medications, as per the *Guidelines*[2] with 100% compliance and are all currently receiving yearly CXRs and clinical reviews.

The 15 Mantoux positive household contacts have all completed their 9 months of LTBI treatment by DOT. The compliance/completion rates range from 12 who were 100% compliant to 3 whose compliance ranged from 75% to 90%. Of these 15 contacts, 8 family members have been discharged from follow-up after CXR and clinical reviews. The other 7 require further CXRs and clinical reviews to complete their follow-up. Some have moved to other communities in the NT.

CHC staff were also offered screening where 9 of 15 were found to be Mantoux positive, 5 have had CXRs and clinical reviews and have been discharged and 4 are currently due for CXRs and clinical reviews.

Another community in the Top End of the NT where the 4th TB case in this outbreak stayed for several months has also been receiving regular CDC follow-up.

Contact tracing results from this second community include:

- 85 contacts were identified by CHC staff and the patient's family
- 8 (9%) contacts were found to be Mantoux positive (3 previous positives and 5 positive on contact tracing) have been assessed for LTBI treatment. All are continuing to have follow-up with CXR and clinical reviews in CDC Nhulunbuy as they either declined or defaulted on LTBI treatment
- 70 (82%) were Mantoux negative and therefore discharged from follow-up
- 7 (8%) still require Mantoux testing. CHC

and CDC staff are still trying to locate them for contact tracing follow up.

The next visit by CDC staff to the community where the outbreak occurred will be in July 2010. This visit will provide CXRs and clinical reviews for those who are due or over-due and will include those on the community screen follow-up list, the cases and their contacts, as well as CHC staff. Cases 9 and 10 and their Mantoux positive contacts will also be reviewed with CXRs.

Acknowledgements

Thank you to the community members, CHC staff, the Radiology Department at Royal Darwin Hospital, ARDS and CDC staff (RN's, doctors and AHW's) for their help in the ongoing management of this TB outbreak.

References

1. Coleman K, Stephenson E, Krause V. Top End TB Outbreak. *NT Dis. Control Bull* 2008; 15(4):8-10.
2. Centre for Disease Control, Department of Health and Families. Guidelines for the Control of Tuberculosis in the Northern Territory. 2008. http://www.health.nt.gov.au/library/scripts/objectifyMedia.aspx?file=pdf/25/05.pdf&siteID=1&str_title=Tuberculosis%20Control%20Guidelines.pdf accessed 30/03/2009.

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The meaning of STI/BBV prevention through Web 2.0 applications

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"The continued growth of the internet as a communication medium has had major implications for the transmission and prevention of sexually transmitted infections"

Rietmeijer and MacFarlane[1]

Abstract

This discussion paper outlines the use of Web 2.0 internet technology and its potential in the practice of behavioural interventions for the prevention of sexually transmitted infections/ blood borne virus.

Web 2.0 is a web application that allows for interactive information sharing, user centred page design and collaboration with other online users allowing the sharing of the web page or its results through personal networks (e.g. Facebook, Twitter, Youtube, Flickr, and MySpace).

This technology could prove to be a useful tool for a growing population group that communicates and networks with other online users, forming relationships that eventually translate into face to face interaction.

Keywords; internet, sexually transmitted infections, social networking, disease prevention.

Introduction

Millions of people are engaging in different ways on the internet every day. The digital divide is shrinking. Internet technology transcends time zones, borders, languages, age groups and nationality. Cyberspace has become the environment of choice for communication, information sharing, networking, making friends and being part of a virtual community. This also provides the opportunity for members of this community to "hook up" in person and for the potential spread of sexually transmitted infection and blood borne virus (STI/BBV) transmission.[1] However it can also provide an opportunity for the sharing of disease prevention information.

According to Rietmeijer and MacFarlane[1], "the interface between the internet and STI/BBV transmission and prevention strategies can be looked at from 3 perspectives:

- the internet as a risk environment, that is, a place where prospective, potentially STI-infected, sex partners can be recruited;
- the internet as a venue where public health prevention interventions aimed at STIs and BBV prevention can be placed; and
- the internet as an increasingly important work

environment for all STI prevention disciplines".[2]

With growing internet access, it is timely to consider development and testing of online interventions. These interventions can target adolescents, women and men with messages about STI/BBV prevention and ways to stay safe if seeking sex partners online. Further research is required to know more about the health risks of people who engage in sexual contact with an internet partner and the context of these sexual encounters, especially in the Northern Territory.

What is Web 2.0?

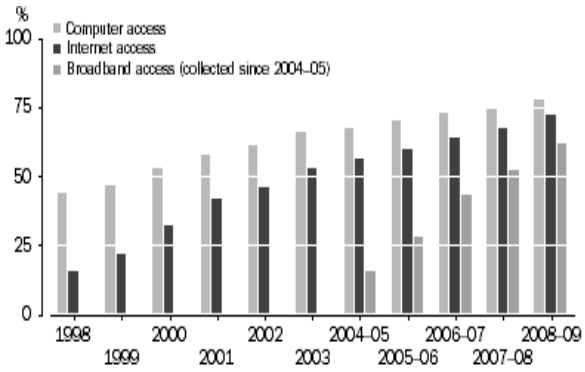
Most people today have heard of *Facebook*, *Twitter, Myspace*, *Wiki* and *Youtube*. These are examples of social networking sites that are built using Web 2.0 technology. Web 2.0 is interactive, user driven and removes the need for a moderator in order to post comments but still requires the moderator to manage the page content. All interaction is documented and other users are notified of changes in real time.

The principle behind Web 2.0 is that it is driven by a community of users open to anyone and everyone. It attracts people who appreciate 'collective knowledge' and those who enjoy interactive participation with others online. There is instant gratification and anonymity which is a well sought after element in communication today, especially amongst generation Y users.

An example of using this technology for health promotion is as follows:

> The topic 'condom use' is posted by a health professional. This information could be where to find condoms in Darwin, what STIs condoms can prevent etc. This information is then read by other online users and can be commented on. This in turn creates an online discussion. Challenges and barriers that people face when using condoms can be addressed and the moderator can then benefit from a better understanding of our target group's knowledge, attitudes and practices which can be qualitatively analysed.

Figure 1. Household computer or internet access, proportion of all households-1998 to 2008-09.



Despite this freedom, the responsibility of online users in posting valid messages, media and information is monitored by other online users and the creator of the web page. The moderator and other users are able to report abuse (to the owners of the website), delete irrelevant content, false information and block a user that doesn't obey the rules and regulations of the website. One important security feature is the history log that keeps track of who was online and what changes were made. A popular example of this is *Wikipedia* which is a combination of a *Wiki* and an online encyclopaedia.

How many and who are online?

According the Australian Bureau of Statistics,[3] from 2008 – 2009, 72% of Australian households had home internet access to a computer (Figure 1).[3] The number of households with a broadband internet connection is estimated at 5 million. The 2009 Children's Participation in Cultural and Leisure Activities survey reported that of the 2.7 million children aged 5 to 14 years, 79% used the internet. Although disparities in access remain, even teens in low-income communities have regular access to the internet and social networking sites at libraries and community centres/youth drop in centres.

The internet has become an important aspect of learning and communication for children and young adults. A survey of 180 young Territorians (2009) on ways/methods for teens and young adults to find information about programs targeting them, showed 25% of the respondents chose internet social networking sites as a method of communication.[4]

From Web 1.0 to Web 2.0 and STI/BBV prevention

Web 1.0 websites are static pages instead of dynamic user generated content. This shift from Web 1.0 to Web 2.0 is a direct result of the change in behaviour of those who use the World Wide Web. In order to effectively communicate with the population as a public health agency, it is important to use the same tools that our target group are using. Therefore, there is a need for internet-based prevention services to better understand this evolving medium and subsequently shift towards a user driven environment.

Population health interventions need to move on into the next generation of online interventions by developing collaborations with social networking sites, and creating profiles and programs on these sites. Identifying and collaborating with existing resources on these sites may prove an efficient and effective way of reaching out to adolescents and young adults.

The interactivity of Web 2.0 and the power of media based health communication (audio, video, video blogs) allow a fully automated cost efficient STI/BBV intervention. However, the full potential of these interventions has not been reached. Further research is required to explore ways to promote STI/BBV prevention services to the population at risk.

The Sexual Health and Blood Borne Virus unit is working on applications that serve an informative purpose for professionals and the public, "a web product that is rich in information, appealing and is spread through the user's personal networks e.g. friends, family, colleagues etc., and allows for discussions and feedback".[5]

Theoretical applications of behavioural interventions through Web 2.0 social networking sites[6]

When using Web 2.0 social networking sites, a roll out of health promotion messages for behavioural interventions must make use of sound theoretical applications such as the Health Belief Model, Social Cognitive Theory and the Theory of Planned Behaviour in order to initiate its users into the continuum of behaviour change. Here are a few examples:

Social cognitive theory

Example: "Sexual Health" is a *Facebook* page from Indonesia, started by sexual health physicians. This *Facebook* page is constantly updated with information from sexual health journals, practical steps in having safe sex, accessing sexual health clinics and answering questions from the public.

Additionally, more specific information can be displayed e.g. clinic location, hours, directions, public transportation, pictures of clinic staff, services and policies. Local events can be highlighted such as upcoming health fairs and clinic tours.[6]

Health belief model

Example: "HIV Is No Picnic" is a "STOP AIDS" project in San Francisco that can inspire a similar campaign on a Web 2.0 social networking site with the following advertisements.

A series of 4 advertisements from the project's "Positive Force Program" were designed to counter a perception among HIV negative men that contracting HIV is no longer something to be so concerned about due to the advancement of anti-retroviral therapy and its availability and accessibility. Because of this perception, men in the target group can be persuaded to not use protection with their partners.

Each advertisement features an HIV+ man describing the impact of a specific side effect of HIV disease or anti-retroviral medication. e.g. "It might seem like diarrhoea is no biggie. That's probably because you never had it like I have. How about being terrified to go anywhere because you might crap your pants? Don't get me wrong. I'm really glad to be alive, but HIV is NO PICNIC. I don't care how good the sex is or how hot the guy is, nothing is worth what I'm going through now".[6]

Theory of planned behaviour

Example: An initial roll out of a project such as condom usage or examples from the first 2 theories is posted on the web page. From the first postings the number of online users using the web page will increase and many others become fans of the web page and follow the dialogue.

From this discussion, the moderator can inform the online community with messages that testing and using condoms are the norm, with the help of social marketing techniques.[6]

Conclusion

There are challenges and risks that come with implementing health promotion through Web 2.0 internet technology. Common challenges include:

- Using new technology and the potential for viruses/spam.
- Designating staff time to update the site and monitor the content.
- Generating new and creative resources in order to be able to constantly update the web page.
- Marketing the web page to teens and convincing them to join the network and designing an evaluation program to measure impact.

STI/BBV prevention relies heavily on behavior change theories as part of its intervention. Web 2.0 supports the process by allowing real time analysis of knowledge, attitudes and practices whilst increasing awareness of risks in transmission. It could potentially enhance safe sex practices through education and by allowing users to engage anonymously with others in a comfortable learning environment.

References

1. Rietmeijer C, MacFarlane M. Web 2.0 and beyond: risks for sexually transmitted infections and opportunities for prevention. *Curr Opin Infect Dis.* 2009; 22:67-71.
2. Rietmeijer C, Bull SS. Risks and benefits of the Internet for populations at risk for sexually transmitted infections (STIs). Results of an STI clinic survey. *Sex Transm Dis.* 2002 Vol. 30(1):15-19.
3. Australian Bureau of Statistics 8146.0 – Household use of information technology, Australia, 2008-09, Canberra. Released 16/12/2009. Multipurpose household survey and the 2009 Children's participation in Cultural and Leisure Activities. *http://abs.gov.au/ausstats/abs@.nsf/mf/8146.0/.*
4. The Youth Minister's Round Table of Young Territorians: "The Youth Engagement and Awareness Project" 2009. http://www.youth.nt.gov.au/pdf/Round%20Table/2009/4%20%20YEAPT%20FINAL1.pdf.
5. Collins A. The CDC National Prevention Information Network (NPIN). www.cdcnpin.org.
6. Aral SO, Douglas JM and Lipshutz JA. Behavioural Interventions for prevention and control of sexually transmitted diseases. New York, NY: Springer, c2007.

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An analysis of injury patterns following road traffic collisions in the Northern Territory

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Abstract

Aims

To describe the pattern and trends over time of injury in patients requiring hospital admission following road traffic crashes in the NT.

Methods

A retrospective descriptive analysis of hospitalization data from 1999 to 2007. ICD10-AM codes were used to extract admissions due to road traffic crashes and to classify injuries according to their nature (soft tissue, fractures, visceral, central nervous system (CNS), neurovascular, burn or other) and road user type.

Results

There were 4,172 admissions with an overall age standardized rate of 224 per 100,000 population per year (95% confidence interval (95%CI) 217-232). The predominant primary injury was fracture (46.0%), followed by soft tissue (28.3%), CNS (8.9%) and visceral (5.7%). Burns and neurovascular injuries were uncommon (each <2%). CNS injuries accounted for 57% of in-hospital deaths.

Cyclists were less likely to sustain visceral injuries (RR 0.45, 95%CI 0.32-0.64). Motorcyclists were less likely to sustain CNS injury (RR 0.63, 95%CI 0.52-0.77) but more likely to sustain burns (RR 5.3, 95%CI 4.2-6.8). Pedestrians were more likely to sustain visceral injuries (RR 1.4, 95%CI 1.2-1.6).

Admission rates for Indigenous Australians were higher than the general population (incidence risk ratio 1.28, 95%CI 1.20-1.37).

Conclusion

Soft tissue injuries and fractures account for three quarters of hospital admissions following road traffic crashes in the NT, but CNS injury was associated with the highest risk of in-hospital death. We also report a recent increase in incidence of burn injuries, particularly in motorcyclists, suggesting a potential topic for future road safety messages.

Keywords: road traffic, injury pattern, Northern Territory.

Introduction

In comparison with the other states and territories, the Northern Territory (NT) is disproportionately affected by road traffic crashes. The most recent (2006/07) age-adjusted NT mortality rate for transportation accidents is 23.6 per 100,000 population, 2.7 times the national rate and substantially higher than that of any other jurisdiction.[1] Rates of hospital admission following traffic crashes in the NT are less starkly disproportionate, being only 1.4 times the national rate, but still the highest in the country.[2]

A major report was released in 2006 considering in detail the causal factors underpinning this remarkable excess of road fatalities and serious injuries in the NT.[3] Alcohol and speed were identified as key contributors to road traffic crashes in the Territory. In half the fatalities reviewed and 13% of the cases of serious injury no seatbelt was worn.

This study focuses on patterns of injury in those admitted to hospital following road traffic crashes in the NT. National level data shows that head and chest injuries are the most common serious injury among motor vehicle occupants, in contrast with head and lower limb injuries in pedestrians and shoulder and upper limb injuries in motorcyclists and pedal cyclists.[2] However, the NT experience of road traffic crashes – summarized briefly above – is sufficiently different from the rest of Australia that these patterns may not hold, particularly given the previously documented comparatively low rates of seatbelt use.[3] This study also reports patterns of injury in more detail than has been reported elsewhere, considering not only the body part injured but the type of injury and correlating this with key explanatory variables.

Patients and methods

Data source, and derivation of study dataset

Data on hospital admissions to any public hospital in the NT following injury between 1 January 1999 and 31 December 2007 were obtained from computerized records of the Department of Health and Families (DHF), NT Government. Given that no private hospitals are known to directly admit trauma patients, these data should completely capture hospital admissions resulting from road trauma within the Territory. Injury admissions were selected as those with an ICD-10-AM 'S' or 'T' code as a primary diagnosis. The start date was chosen as the first year for which ICD-10-AM was used in discharge coding and the end date as the last year for which discharge coding was complete.

Records comprised basic demographic information (hospital record number, sex, date of birth, Indigenous status), admission characteristics (time and date of admission, hospital name, type of admission (e.g. elective, emergency), discharge date and date of death where appropriate). This initial dataset was then refined as follows:

Secondary diagnostic codes within each record were searched for 'V' codes (signifying a transport accident). Records without a 'V' code were discarded as being for injury admissions not due to a transport accident.

The 'V'-code was used to determine whether the accident took place in a 'traffic' or 'non-traffic' setting, according to the ICD-10 definition. These were cross-checked with 'Y' place codes; where place was recorded as a street, highway or parking lot the accident was considered to be a 'traffic' accident regardless of the 'V'-code. Only those accidents occurring in a traffic setting (i.e. on a public road) were included in this analysis.

Only the initial admission following a transport accident was used in this analysis. Thus 'elective' type admissions were discarded. Subsequent 'emergency' admissions were also discarded if they were likely to be a follow-up admission related to the initial crash (assumed if the admission was <28 days after the initial admission, and examined by hand if the admission was between 28 and 365 days after). This method is based on that used by the Australian Institute of Health and Welfare for their reports,[2] but does not exclude hospitalizations in which the person died, or where another external cause code preceded the V-code.

Statistical Analysis

Raw ICD injury codes were classified into an injury type ("superficial, muscle and soft-tissue injury", "fracture", "central nervous system [CNS] injury", "peripheral neurovascular injury", "visceral injury", "burn injury" or "amputation, crush or other injury"). Note that this in this context, burn includes friction burns. These categories loosely mirror the ICD-10 coding system: each ICD-10 injury code or group of codes was hand-mapped to an appropriate category. A primary injury type was assigned to each admission based on the primary diagnosis code. Each additional injury diagnosis was also translated into an injury type.

Variables describing the role of the admitted person in the crash (e.g. driver, pedestrian) and the nature of the crash (e.g. pedestrian "versus" car, non-collision car crash) was also derived directly from the 'V'-code. Unusual entries in these variables were manually reviewed and corrected if an error was obvious, otherwise the coded entry was assumed to be correct.

Age standardized rates were calculated by the direct method to the 2001 Australian standard population using 2008 Northern Territory population data provided by the Health Gains Unit of the Northern Territory Department of Health and Families. Adjusted incident rate ratios were derived using a Poisson regression model and Poisson regression was also used to determine the significance of trends over time.

Correspondence analysis was used as an exploratory technique to identify patterns in injury type by potential explanatory variables (e.g. occupant type). Any patterns so identified were then submitted to further univariate analysis with relative risks and confidence intervals reported.

Statistical analysis was with STATA version 11 (Statcorp, Texas, USA). Two-sided P-values of <0.05 were considered significant.

Results

Basic demographics

There were 4,172 hospital admissions following road traffic crashes in the NT during the study period, giving an age-adjusted rate of 224 admissions per 100,000 estimated resident population (ERP) per year (95% CI 217-232) for the study period overall. In-hospital mortality was 56 patients (1.3%). Pedestrians comprised 14 of these 56 patients (25%), despite only constituting 11% (451 admissions) of the overall dataset. The relative risk of death for a pedestrian requiring hospital admission after a traffic crash compared with other road user types was 2.9 (95% CI 1.6-5.4), although the absolute risk of death was small (3.0%).

When examined by year, admissions rose from an age-adjusted rate of 182 per 100,000 ERP per year (95% CI 162-201) in 1999 to 226 per 100,000 ERP per year (95% CI 205-248) in 2007. This trend was statistically significant (p=0.047) using a Poisson model with year and age group as independent variables and with exposure estimated using age-stratified Northern Territory population data. Within each year there was marked seasonality with peak admissions during the dry season (May-October, characterised by a marked increase in visitors to the region and road traffic in general). Further demographic characteristics are summarized in Table 1.

Table 1. Demographic characteristics of patients admitted to hospital following a road traffic accident in the Northern Territory 1999-2007

Characteristic	N (%) n=4,172	
Male gender	2,860	(68.5%)
Age group		
0-14 years	532	(12.8%)
15-29 years	1,601	(38.4%)
30-44 years	1,219	(29.2%)
45-59 years	582	(14.0%)
60+ years	238	(5.7%)
Indigenous	1,451	(34.8%)
Occupant type - Collision type*		
Motor vehicle occupant	2,210	(53%)
- *non-collision accident*	1,292	58%
- *versus motor vehicle*	463	21%
- *versus fixed object*	369	17%
Cyclist	479	(11.5%)
- *non-collision accident*	293	61%
- *versus motor vehicle*	72	15%
- *versus fixed object*	30	6.2%
Motorcyclist	683	(16%)
- *non-collision accident*	389	57%
- *versus motor vehicle*	114	17%
- *versus fixed object*	80	12%
Pedestrian	465	(11%)
- *versus motor vehicle*	421	91%
Unknown	335	(8.0%)

* Only collision types occurring with a relative frequency greater than 5% are reported.

Primary injury (injury responsible for admission)

Fractures and soft tissue injuries together constituted 74% of the injuries responsible for admission. This proportion remained remarkably constant across road user types, ranging from 71% in motor vehicle occupants to 82% in cyclists. The next most common primary injuries were CNS injury (8.9%), amputation, crush or other injury (8.8%) and visceral injury (5.7%). Burns were uncommon (1.2%), except in motorcyclists where they represented 4.4% of admissions (Table 2).

The 3 most common primary diagnoses in the 56 patients who died during their admissions were CNS injury 32 (57%), fracture 11 (20%), and visceral injury 8 (14%). The relative risk of in-hospital death for a person admitted with a primary diagnosis of CNS injury compared with other primary diagnoses was 14 (95% CI 8.1-22), with an absolute risk of in-hospital death of 8.6%.

All injuries

Overall, 56% of those admitted had multiple types of injury, with substantial variation according to the primary injury diagnosis. At

Table 2. Primary injury class by occupant type for patients admitted to hospital following a road traffic accident in the Northern Territory 1999-2007

Injury class*	Motor vehicle	Motorcyclist	Cyclist	Pedestrian	Unknown	Overall
Sup., mm., soft tiss.	711 (32%)	158 (23%)	127 (27%)	98 (21%)	87 (26%)	1,181 (28%)
Fracture	862 (39%)	379 (55%)	265 (55%)	248 (53%)	164 (49%)	1,918 (46%)
CNS	222 (10%)	37 (5.4%)	40 (8.3%)	43 (9.3%)	31 (9.2%)	373 (8.9%)
Peripheral neurovasc.	30 (1.4%)	6 (0.9%)		6 (1.3%)		48 (1.2%)
Visceral	139 (6.3%)	33 (7.1%)	14 (4.2%)	33 (7.1%)	14 (4.2%)	237 (5.7%)
Burn	7 (0.3%)	30 (4.4%)	4 (0.8%)			48 (1.2%)
Amp., crush, or other	239 (11%)				33 (9.9%)	367 (8.8%)

* Sup., mm., soft tiss. = superficial, muscle or soft-tissue injury. CNS = central nervous system injury. Neurovasc. = neurovascular. Amp. = amputation

Cells with a frequency < 4 have been omitted to preserve confidentiality. Where this has occurred frequencies in the 'unknown' or 'other' category will also be omitted.

one end of the spectrum only 30% of those with soft tissue injury had additional injury types, whereas additional injury types were common (>80%) for those admitted for visceral, central nervous system or neurovascular injury. Among these additional injuries, the most common were superficial, soft tissue or muscular injuries: 1,606 (38%) patients with non-soft tissue primary injuries had an additional superficial, soft tissue or muscle injury. The next most common additional injury type was fracture, with 472 (11%) of admissions having a fracture diagnosis in addition to a non-fracture primary injury. Conversely, the most common additional injuries associated with a primary injury class of soft tissue injury are fractures (28%) and concussion (20%).

Primary injury by occupant type

Examining the primary injury by road user type suggested that cyclists were less likely to sustain visceral injuries (relative risk 0.45, 95% CI0.32 to 0.64). Motorcyclists were less likely to sustain central nervous system injury (RR 0.63, 95% CI 0.52 to 0.77) but more likely to sustains burns (RR 5.3, 95% CI 4.2 to 6.8). Pedestrians were more likely to sustain visceral injuries (RR 1.4, 95% CI 1.2 to 1.6).

Trends

The increase in hospital admission rates between 1999 and 2007 appears largely due to an increase in admission for soft tissue injuries over the same period. The only other injury class showing a statistically significant increase in admission rates over time is burns, but absolute numbers remain small (Figures 1 and 2). As motorcyclists are the road-user group most at risk of burn injuries,, age-adjusted hospital admission rates over time for motorcyclists were also examined. They rose from 27 per 100,000 ERP (95% CI 20-34) in 1999 to 38 per 100,000 ERP (95% CI 30-46) in 2007 ($p=0.044$ for Poisson regression model including all data 1999-2007).

Injury patterns in Indigenous Australians

Indigenous Australians are slightly overrepresented, representing 34.8% of the injury admissions but only 30% of the NT population ($p<0.01$, one sample test of proportion). They comprised 38% (21 cases) of the in-hospital deaths. When examined over time as a population-specific rate, the admission rate following road traffic accidents for the Indigenous population is higher than the non-Indigenous population, varying from an excess of 15 admissions per 100,000 population per year in 2005 to an excess of 95 admissions per 100,000 population per year in 2003. Using a Poisson model as described previously with year, age group and indigenous status as independent variables only age group and Indigenous status were significant, with an incidence risk ratio of 1.28 (95% CI 1.20-1.37) for Indigenous people being admitted after a road traffic accident versus non-Indigenous.

Figure 1. Age-adjusted hospital admission rate following road traffic accidents in the Northern Territory from 1999-2007, overall and stratified by injury type

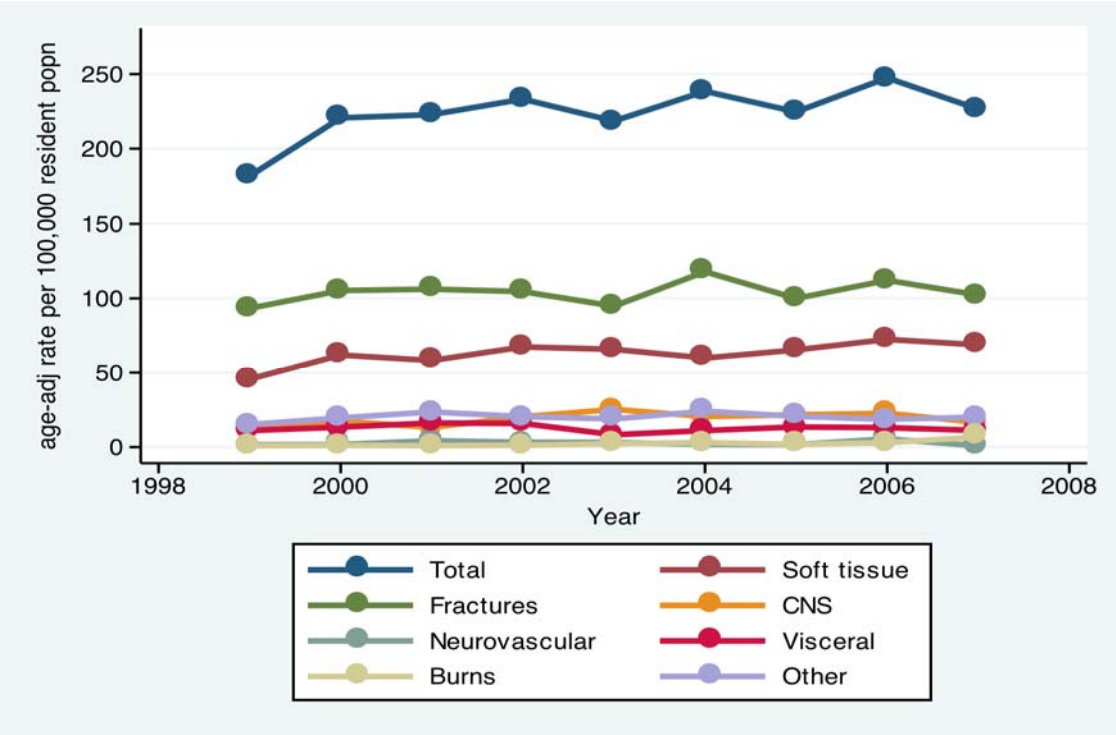
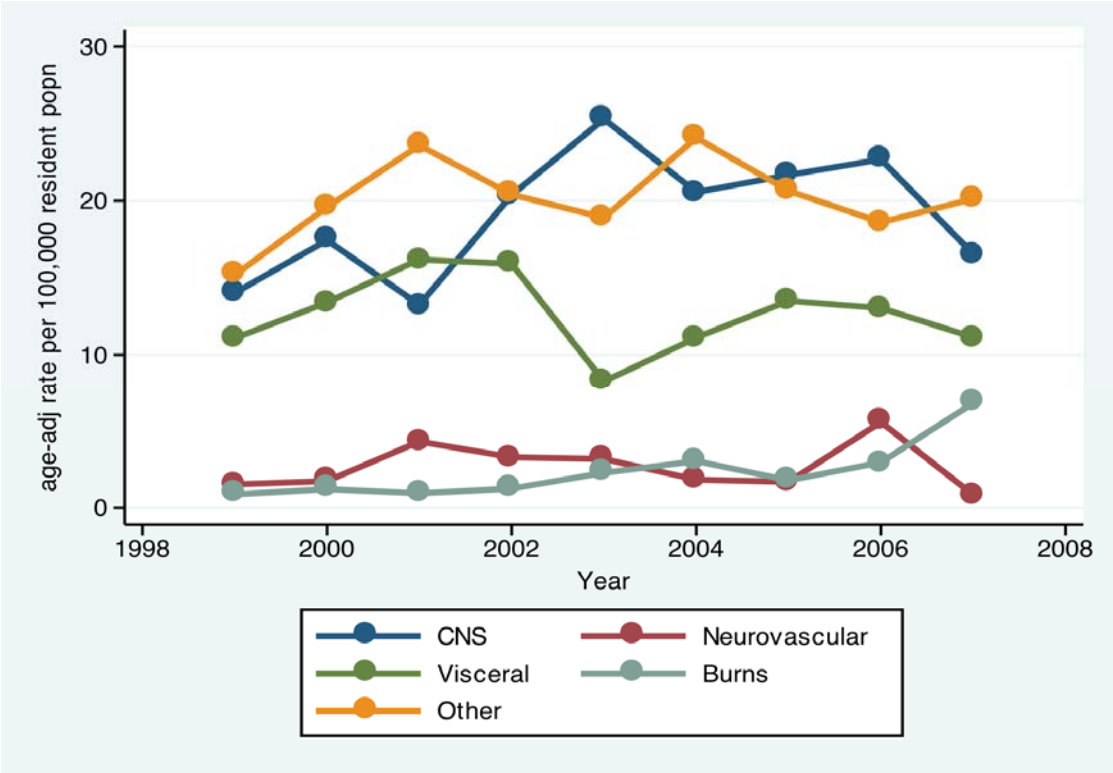


Figure 2. Age-adjusted hospital admission rate following road traffic accidents in the Northern Territory from 1999-2007 showing the lowest incidence injury types in more detail.



There are subtle differences in injury patterns between Indigenous and non-Indigenous admissions. Indigenous occupants of motor vehicles are more likely to admitted with visceral injuries (RR 1.24, 95% CI 1.04-1.49) but less likely to be admitted with CNS injuries (RR 0.79, 95% CI 0.66-0.94). Indigenous cyclists are less likely to be admitted with fractures (RR 0.69, 95% CI 0.54-0.88). However, absolute numbers are small – for example, in the number of Indigenous cyclists admitted after a crash over the study period was 103.

Discussion

Overall, an increase over time in admission rates following road traffic crashes was observed. However, its significance and magnitude are debatable. An examination of Figure 1 suggests that 1999 recorded an abnormally low hospital admission rate; repeating the analysis when 1999 is excluded from the model reveals that the trend is no longer significant. The trend in soft tissue injuries likewise becomes non-significant when 1999 data are excluded. It is noteworthy that 1999 was the first year in which diagnoses were coded using ICD-10-AM and the lower rates may represent a period of adjustment. Certainly when data on mortality following road traffic crashes is reviewed there is no major corresponding mortality dip (49 fatalities in 1999, compared with 51 in 2000) suggesting that there may not have been a true decrease in hospital admissions.[4] Direct comparison with morbidity data from the police-administered Vehicle Accident Database would provide additional corroboration, however this work has not yet been done.

Of concern is a recent increase in the number of patients admitted for burns, most of whom are motorcyclists. Serious burn injuries are known for their high morbidity and have been shown in a previous NT-based study to consume a disproportionately high amount of health-care resources per admission.[5] At this stage the increase is only seen in 2007 data, and the trend becomes non-significant (p=0.076) when these are excluded from the analysis. As to reasons why 2007 should have recorded a substantial increase in burn injuries, it is worth noting that the road user group most at risk of admission for burn injuries was motorcyclists, with a relative risk of 5.5. Motorcycle use has increased substantially in recent years: between 2003 and 2008 the number of registered motorcycles in the NT increased by 52%, as compared with passenger vehicle registrations which increased by 14% over the same period.[6] Admissions for motorcycle crashes was also observed to increase by approximately 40% over the same interval, providing one possible explanation, although a re-analysis that includes 2008 data will be essential in confirming the trend. Use of adequate protective clothing in a tropical climate is one road safety message which may assist in reducing the burden of burns injuries.

Of importance to health planners is the utilisation of healthcare resources by primary injury type. A detailed consideration of this is beyond the scope of this article, however, it is worth noting that soft tissue injury and fractures formed the primary diagnosis for three quarters of all hospital admissions following road traffic accidents. In their 2005 report, You *et al* found that fractures and soft tissue injuries together accounted for 58% of total injury type admissions (not just road traffic-related) and 54% of the total cost.[5] The You report taken together with these data nonetheless suggests that managing soft tissue injuries and fractures would consume the majority of health-care resources related to road traffic accident admissions. In other words, substantial quality and cost gains may be made by even incremental improvements in optimizing and streamlining the care of patients with these injuries.

Transport-related injuries in the Indigenous population have been addressed in detail already by Harrison and Berry[7]; our findings of an increased incidence rate ratio of 1.3 for hospital admission following a road traffic accident in this group are consistent with their report. However, hospital admissions alone understate the problem: in 2007, 53% of those who died on NT roads were Indigenous.[4] Coupled with this study's finding that only 37.5% of the in-hospital deaths were in Indigenous people, this suggests an excess traffic accident related mortality in the Indigenous population, most likely prior to arrival in hospital. I would hypothesis that one or other of two factors – distance to hospital care and severity of sustained injuries – may provide an explanation, although further study will be required to confirm this.

In conclusion, we have found that soft tissue injuries and fractures account for three quarters of hospital admissions following road traffic accidents in the NT, but that CNS injury was associated with the highest risk of in-hospital death. The number of burn injuries, particularly in motorcyclists, in 2007 was disproportionately high, suggesting another potential topic for future road safety messages.

Acknowledgements

I thank Karen Dempsey for her invaluable assistance and provision of STATA files to aid the extraction and cleaning of data. I also thank Dr Steven Skov for his insight into the issues surrounding road traffic injuries in the NT and assistance preparing this manuscript.

References

1. Henley G and Harrison JE. Injury deaths, Australia 2004–05. Injury research and statistics series no 51. Cat. no. INJCAT 127. Adelaide: AIHW; 2009.
2. Henley G and Harrison JE. Serious injury due to land transport accidents, Australia 2006–07. Injury research and statistics series no. 53. Cat. no. INJCAT 129. Canberra: AIHW; 2009.
3. NT Road Safety Taskforce. Safer Road Use: A Territory Imperative. Darwin: Northern Territory Government; 2006.
4. Northern Territory Transport Group. Northern Territory Crash Statistics. 2010 [cited 2010 26 February 2010]; Available from: http://www.roadsafety.nt.gov.au/transport/safety/road/stats/index.shtml.
5. You JQ & Guthridge SL, Mortality, Morbidity and Health Care Costs of Injury in the Northern Territory 1991- 2001. Darwin: Department of Health and Community Services; 2005.
6. Australian Bureau of Statistics. Motor Vehicle Census, Australia, 31 March 2008. Cat. no. 9309.0. Canberra: ABS; 2008.
7. Harrison JE and Berry JG. Injury of Aboriginal and Torres Strait Islander people due to transport, 2001–02 to 2005–06. Injury research and statistics series number 46. Cat. no. INJCAT 120. Adelaide: AIHW; 2008.

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Editorial

Steven Skov, CDC Darwin

This study provides the first analysis of the types of injuries suffered by victims of road crashes in the NT. It unfortunately again demonstrates the very high rates of crash injuries. The author refers to work under way to allow for comparison with the Vehicle Accident Database. This work, being undertaken by Karen Dempsey of the Health Gains Planning Unit, will allow for a more sophisticated analysis of the relationship between crash characteristics (eg place, involvement of alcohol, speed, wearing of seatbelts) and the nature and severity of injuries from hospital data. This will hopefully better inform road crash prevention initiatives in the NT.

Global warming – rising sea surface temperatures - a longer box jellyfish (*Chironex fleckeri)* stinger season for the Northern Territory?

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Abstract

That large numbers of Chironex fleckeri appear in Northern Australian waters on a seasonal basis has long been known by local Indigenous people, however, the exact nature of this yearly phenomenon and the factors influencing it are still not fully understood. Sea surface temperature (SST) appears to be a determining factor signaling the "arrival" of C. fleckeri each year. Anthropogenic climate change modeling predicts global rises in SST. Rises in SST may result in an earlier "arrival" of C. fleckeri during the dry season, possibly necessitating extension of the official Northern Territory stinger season to commence in September rather than the current October through to June.

This short report presents data to support this hypothesis and provides a rationale for policy makers to consider changing the timing of the stinger season.

Keywords: Chironex fleckeri, box-jellyfish, marine stings, climate change, sea surface temperature.

Being stung by a box jellyfish (*Chironex fleckeri)* represents an extremely painful or even life-threatening risk for those swimming in Australia's tropical coastal waters.

Large numbers of *Chironex fleckeri* appear in Northern Australian waters on a seasonal basis usually beginning in about October each year (Grey, D. 1978, unpublished data). However the exact nature of this yearly phenomenon, its development, geographical extent, timing and contributing factors require further elucidation.[1,2] Sea surface temperature (SST) appears to be a major determining factor signaling the "arrival" of *C. fleckeri* each year[3,4] as well as an important influence over the duration of the "stinger season" in coastal waters.[3,4,5] It is predicted that global climate change will increase SST[6] and indeed a rise has been evident since the 1970's for the northern Tropics (Figure 1).

In Darwin the official annual stinger season has been recognized as between 1 October to 1 June, each year (8 months). This was determined on the basis of monthly "sting counts" data from the 1970s.

Figure 1 Annual SST anomalies for Tropical Australia[7]

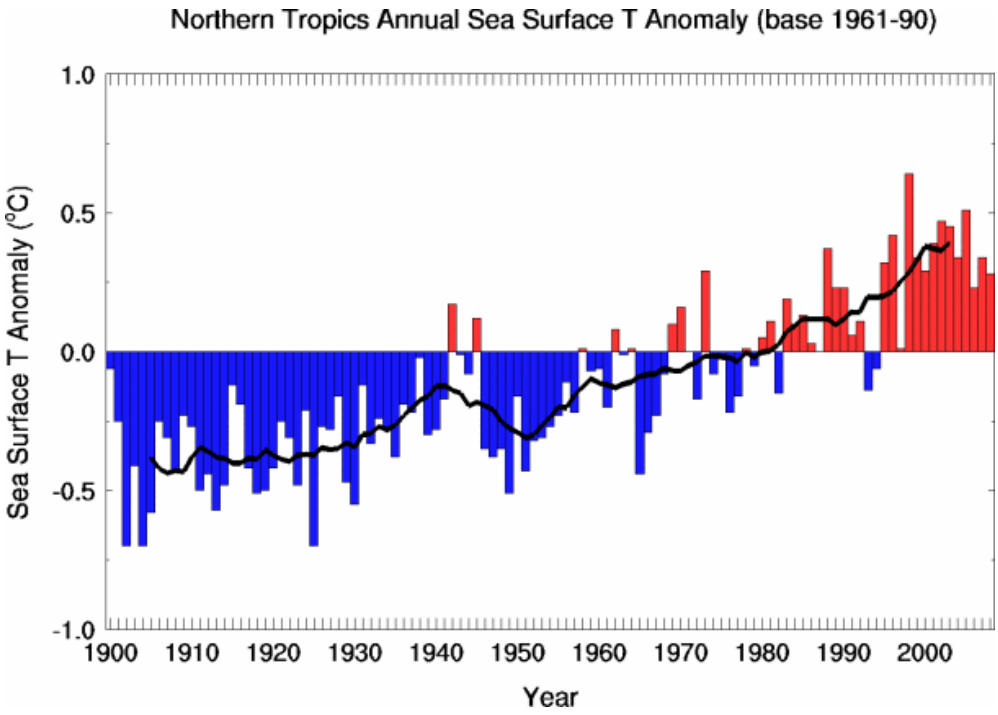
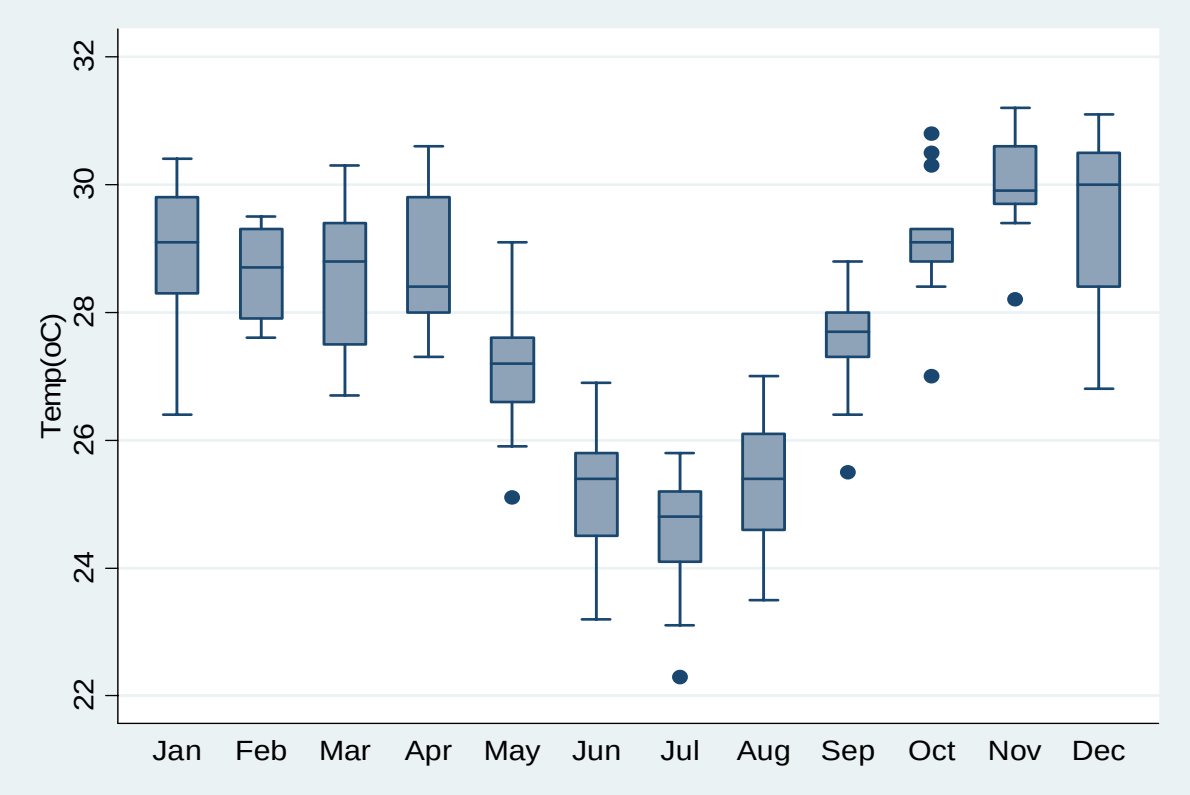


Figure 2. Average daily minimum sea surface temperature for each month during study period. Median, 25th and 75th percentiles shown as the box, with upper and lower limit bars (whiskers) and outliers as dots[7].



Historical data from trawling (sea netting) in Darwin harbour (12.3°S), between 1971-1978 revealed that the medusae arrive only when the SST rises above 26°C[4], which occurs by September on average (Figure 2).[7]

Hartwick's work in Queensland found that box jellyfish reproduction does not commence until water temperatures rise above 25° C.[8] The annual "arrival" of *C. fleckeri* has also been attributed to the wet season freshwater discharge which renders coastal waters highly enriched with organic material which is required to nourish the juvenile medusae.[9] However, as heavy rains usually do not occur in the Northern Territory (NT) until November-December,[10] the medusa arrive before the wet season commences, creating speculation as to whether wet season freshwater discharge does have a causal relationship with their arrival.[11]

Stings have occurred in every month of the year in Darwin[12,13], with few reported in June or July. A prospective study of confirmed *C. fleckeri* stings between 1991-2004 in the NT found that of the 225 reported stings, 208 (92%) occurred within the official season.[12] In north Queensland *C. fleckeri* stings have yet to be reported in June or July.[13] Water temperatures off Cairns average 26° C between April to May, and drop to 24° C from June to August.[14]

For the present study, data concerning stings in the Darwin region from the 1991-2004 NT prospective box jellyfish study were further analysed to study the relationship between SST and the occurrence of stings in NT waters. Results of this analysis reveal a strong positive correlation between monthly numbers of confirmed *C. fleckeri* stings and average daily minimum SST in Darwin harbour, R^2=0.327 p<0.001 (Figure 3).

When comparing these results with actual average daily minimum SSTs for Darwin harbour for the study period (1991-2004) (Figure 1), of note is the relative coolness of water temperature during June, July and August, the majority of recordings below 26° C, while May and September are between 26-28° C. Analysis on the exact SST (Darwin harbour) at the time of *C. fleckeri* sting - recorded as sting

Figure 3. Monthly *Chironex fleckeri* stings and average minimum daily sea surface temperature, Darwin harbour, April 1991 to May 2004[12]

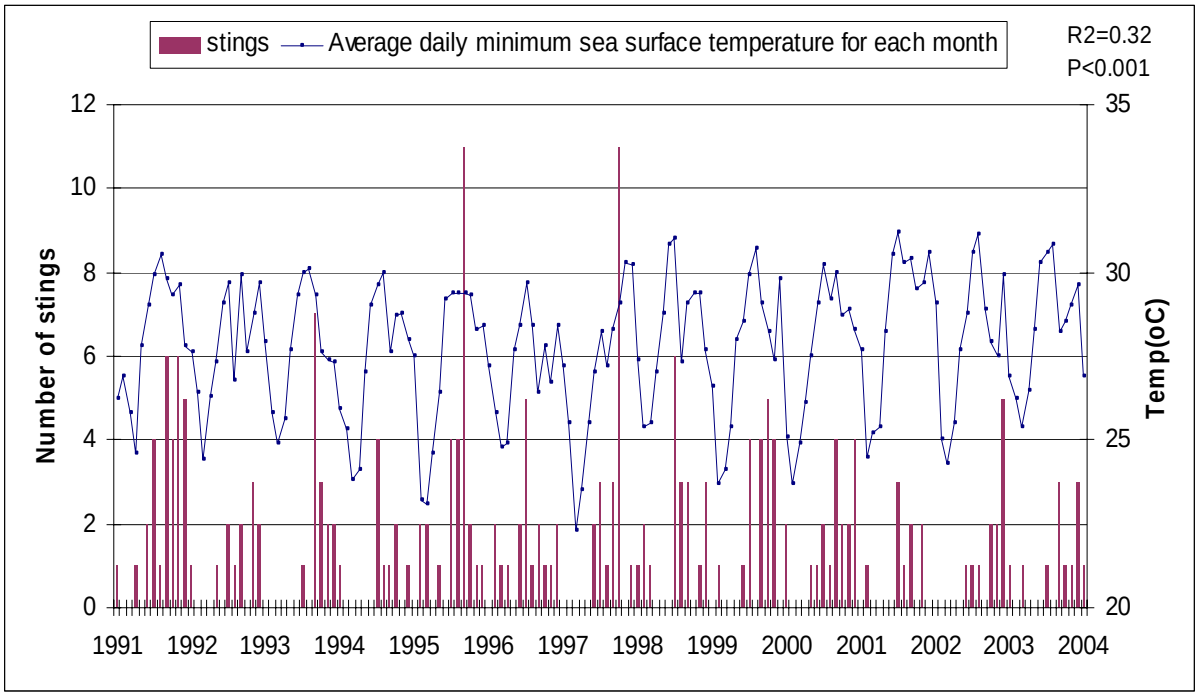
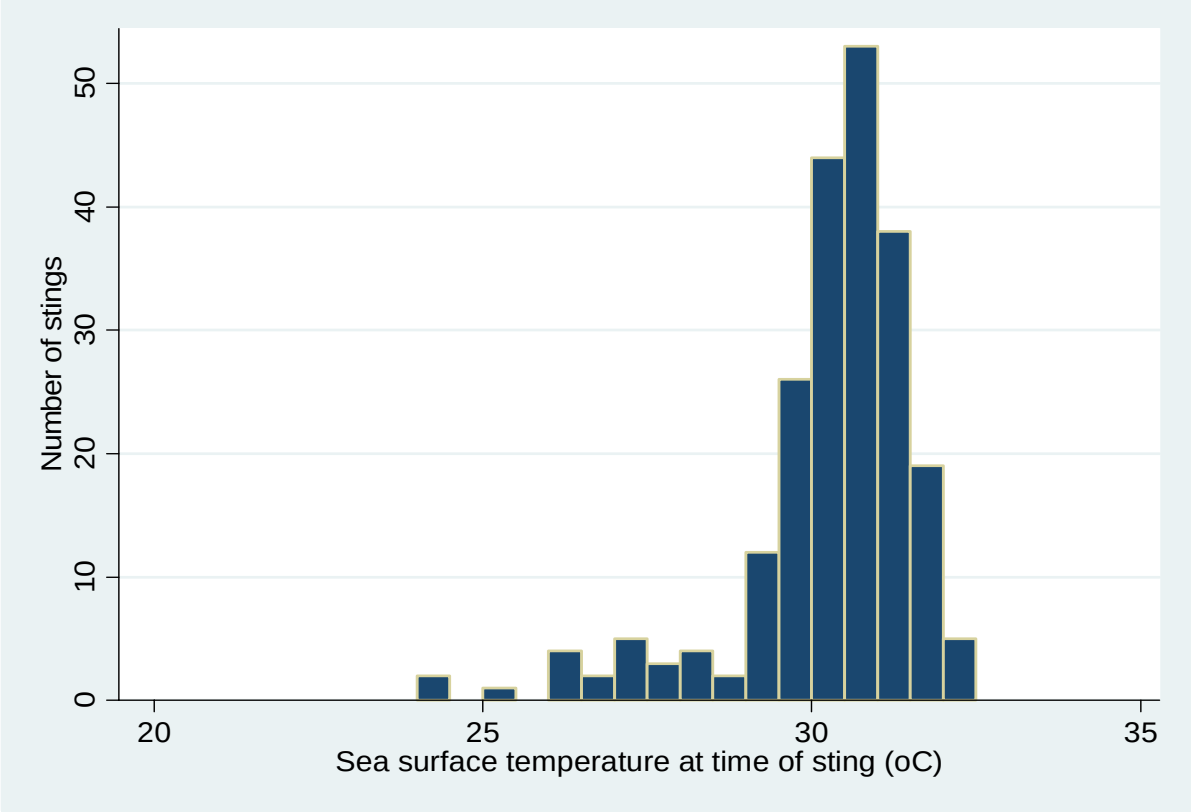


Figure 4. Monthly *Chironex fleckeri* stings by SST (0.5^{0}C increments) for Darwin harbour at time of sting April 1991 to May 2004.[7, 12]



time when interviewed in the emergency department of Royal Darwin Hospital, revealed that fewer stings occurred when SST was below 29°C, 23/220 (10%) (Figure 4).[7]

Extrapolating from this, should the monthly median SST increase by 0.5°C in Darwin harbour, then SST for the entire month of September SSTs will cross a potential 26°C threshold, with most days over 28°C (Figure 2). These changes in SST may be associated with an earlier arrival of *C. fleckeri* in NT waters, which might necessitate the inclusion of September in the official stinger season.

This report presents data which strengthens the link between SST, the arrival of *C. fleckeri* and the occurrence of stings in the NT. It suggests that SST may be used as a surrogate measure for the multiple environmental cues which determine the annual arrival and duration of the medusae in NT waters. A recognition of this link and the observation that SST appear to be rising would provide a rationale for policy makers to consider changing the stinger season duration prospectively rather than waiting for an increase in monthly sting counts. Potentially life threatening stings might thereby be prevented.

This may necessitate an extension of the Northern Territory stinger season to include September, and in time, even May, as the calendar month heralding the approaching *C. fleckeri* stinger season.

Acknowledgements

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References

1. Williamson JA, Fenner PJ, Burnett JW, Rifkin JF, editors. Venomous and poisonous marine animals - a medical and biological handbook. Sydney: University of New South Wales Press; 1996.
2. Goggin L, Gershwin L-A, Fenner P, Seymour J, Carrette T. Stinging Jellyfish in tropical Australia-current state of knowledge. In: CRC Reef Research Centre, editor. Townsville: CRC Reef Research Centre; 2004. p. 1-6.
3. Baker JT. Task Force Report: *Chironex fleckeri*, Southcott. Townsville: Queensland government, Department of Health 1984.
4. Grey DL. Observations on the seasonal abundance of *Chironex fleckeri Southcott* in the Northern Territory. Darwin: Government of the Northern Territory: Department of Primary Production, Production DoP;1984.
5. Holmes JL. Marine stingers in far north Queensland. *Australas J Dermatol*. 1996 May;37 Suppl 1:S23-6.
6. McMichael AJ, Woodruff RE, Hales S. Climate change and human health: present and future risks. *Lancet*. 2006 Mar 11;367(9513):859-69.
7. Australian Bureau of Meteorology. Australian Bureau of Meteorology Website. [website] 2008 [6-11-08]; Available from: http://www.bom.gov.au.
8. Hartwick RF. Distributional ecology and behaviour of the early life stages of the box-jellyfish *Chironex fleckeri*. *Hydrobiologia*. 1991;216/217:181-88.
9. Grey DL. Sea wasps (*Scyphozoa: Cubomedusae*) in the Northern Territory. *Northern Territory Naturalist.* 1978;1:4-6.
10. Australian Bureau of Meteorology. Australian Bureau of Meteorology Website. [website] Darwin: Australian Bureau of Meteorology; 2009 [cited 2009 18 September, 2009]; Available from: {Australian Bureau of Meteorology, 2008 #66}.
11. Gordon M, Hatcher C, Seymour J. Growth and age determination of the tropical Australian cubozoan *Chiropsalmus sp*. *Hydrobiologia*. 2004 Nov 15;530-31:339-45.
12. Currie BJ, Jacups SP. Prospective study of *Chironex fleckeri* and other box jellyfish stings in the "Top End" of Australia's Northern Territory. *Med J Aust.* 2005 Dec 5-19;183(11-12):631-6.
13. Fenner PJ, Harrison SL. Irukandji and *Chironex fleckeri* jellyfish envenomation in tropical Australia. *Wilderness Environ Med*. 2000 Winter;11(4):233-40.
14. Weather zone. Cairns weather patterns. Weather zone; 2009 [cited 2008]; Available from: http://www.weatherzone.com.au/.

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Interceptions of *Aedes aegypti* and *Aedes albopictus* in the port of Darwin, NT, Australia, 25 January and 5 February 2010

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Abstract

There were 2 exotic mosquito interceptions at a port facility in Darwin NT on the 25 January and 5 February 2010. Aedes aegypti larvae and pupae were collected live from a tank container offloaded from a vessel from Timor Lesté. The receptacle and all other receptacles in the port facility were treated with residual insecticide and the area fogged to kill any possible importations of adult mosquitoes.

Follow up surveillance collected Aedes albopictus males in a Biogents trap near the first importation site. Further surveys and elimination measures were undertaken. There have been no further detections of any adults or larvae of these 2 species.

The importations have revealed a new mode of transport of exotic mosquitoes into the NT and indicate further inspection, surveillance and treatment requirements to maintain the NT free of dengue mosquitoes.

Keywords: Aedes aegypti, Aedes albopictus, mosquito, eliminate, dengue vectors.

Introduction

The Northern Territory (NT) is one of the very few regions of the tropical world that has a history of the presence of either of the dengue vectors, *Aedes aegypti* and *Aedes albopictus* and is now free of these vectors. However, the Darwin port areas are particularly vulnerable to the importation of exotic mosquito vectors of dengue originating from overseas ports, with numerous recorded incidents of risk importations,[1,4] with the last recorded in 2007.[5]

Recently, there have been 2 successive exotic mosquito interceptions at the Perkins port facility in Darwin on the 25 January and 5 February 2010. The responses to both of these interception events have been guided by the protocols outlined by the National Arbovirus and Malaria Advisory Committee (NAMAC).[6] This report describes the interceptions and the responses to them.

Detection, Elimination and Surveillance

Detection 1 – Aedes aegypti

On the 25 January 2010, the Australian Quarantine Inspection Services (AQIS) collected live *Ae. aegypti* larvae and pupae from cargo on the *M.V. Kathryn Bay* that routinely voyages between Darwin and Singapore via Dili. The cargo in question was offloaded onto Perkins international wharf in Darwin one week prior to the detection. The mosquitoes were detected in a water holding section of an access hatch (Figure. 1) on top of a large fuel tank (Figure. 2), which was deck cargo on the vessel. Medical Entomology (ME) of the NT Department of Health and Families (DHF) assessment was that the previously laid and recently flooded mosquito eggs had probably hatched soon after the vessel arrived in Darwin. The presence of pupal skins indicated that live adults had possibly flown from the cargo.

Figure 1: Non draining access hatch

Sample Identification – Aedes aegypti

After initial identification as *Ae. aegypti*, AQIS forwarded the juvenile mosquitoes to ME for confirmation on the day of collection. The sample was immediately confirmed as *Ae. aegypti*, with 53 x 4th larval instars, 2 x 3rd larval instars and 3 x pupae. All of the pupae were males and 2 were well developed with scales visible through the pupal skin. Larvae and pupae were identified using taxonomic keys.[7] The AQIS officer who collected the sample indicated that a number of pupal skins were present, but were not collected.

Elimination and Survey Procedures – Aedes aegypti

On the same day of the detection, the inspecting AQIS officer and the ME exotic vector surveillance officer treated all the inside surfaces of the tank-container access hatches with a chlorine/detergent mix (~12% active chlorine) applied by a pump sprayer.[8] Perkins personnel had already applied chlorine to the hatch after the sample collection, but it was not certain that all inner surfaces of the hatch had been treated. A residual spray of the synthetic pyrethroid lambda-cyhalothrin was also applied to the area behind the Perkins Quarantine wash down bay by pressure spray. This was where the water recycling system, including retention pits and tanks is located and was the closest potential breeding and harbourage site for any adult mosquitoes that may have emerged from the access hatch breeding site.

On the evening of the detection, ME conducted an Ultra Low Volume (ULV) fogging application of bioresmethrin to the Perkins international shipping area and the general Perkins shipping and engineering yards, together with the adjacent area to the south at the Frances Bay Marine premises (Figure. 3). The wind directed the fog to the east towards the sea. Approval for fogging was given by the Chief Health Officer (CHO). All Perkins personnel were requested to vacate areas potentially exposed to the fog.

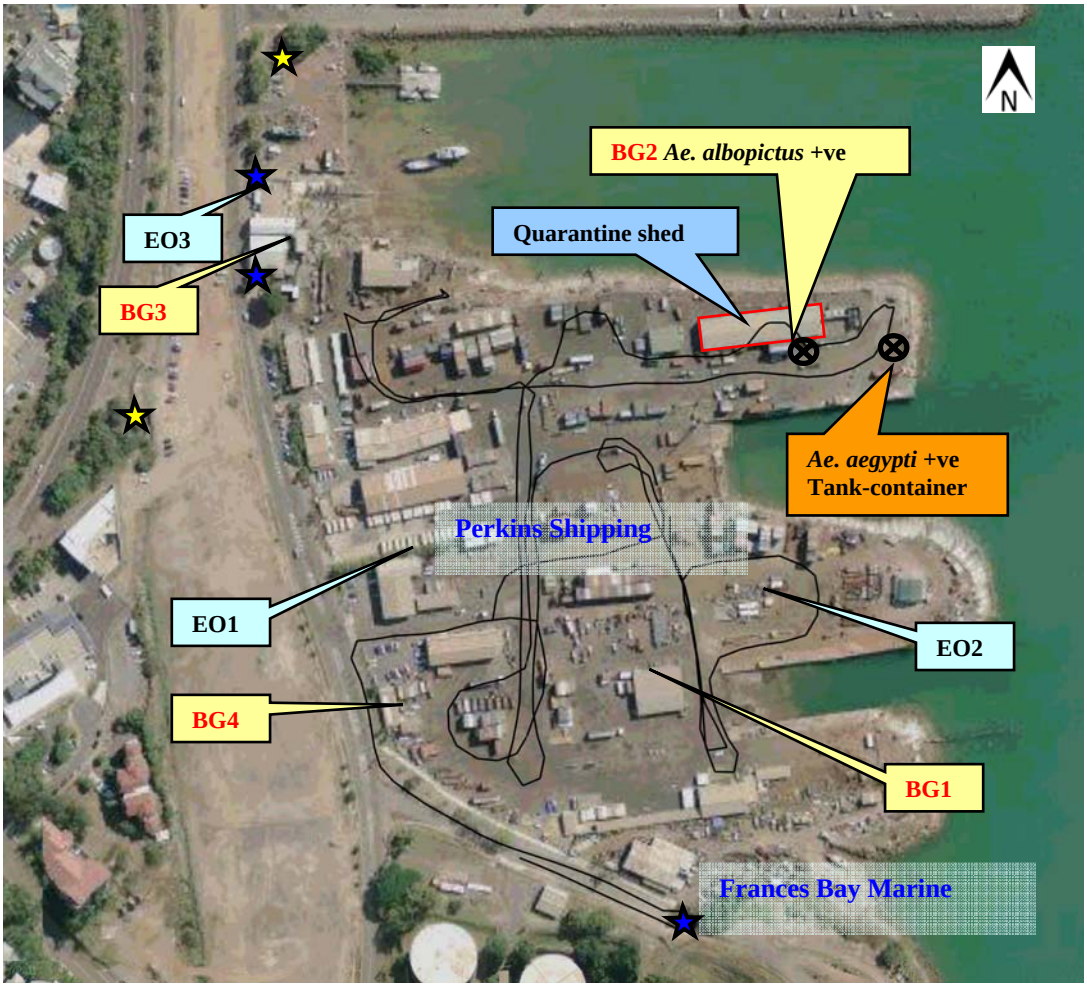
Figure 2. ***Ae. aegypti*** **positive tank-container, Perkins shipping 25 January 2010**

The positive tank-container was 1 of 3 tanks transported on the *M.V. Kathryn Bay*. The 2 other tank-containers were located within the Perkins yard on 27 January. One of these was identical to the positive tank-container, but did not have water pooling in the hatch section. The inside surfaces of the dry hatches were sprayed with a chlorine/detergent mix to kill any possible eggs present. The remaining tank-container did not have access hatches located on top of the tank.

On 3 February, a receptacle treatment round was conducted by applying alpha-cypermethrin (synthetic pyrethroid) by pressure sprayer to all actual and possible water holding receptacles within the entire Perkins yard. This procedure included sampling receptacles for larval mosquitoes. Recent trials testing alpha-cypermethrin and lambda-cyhalothrin found that both successfully prevented mosquito breeding in receptacles for up to at least 10 weeks, with evidence that alpha-cypermethrin provided better knock down for harbouring adults than lambda-cyhalothrin.[9] In the NT, the Australian Pesticides and Veterinary Medicines Authority provided an interpretation that alpha-cypermethrin was able to be used in accordance with the label rates for the treatment of surfaces to kill adult mosquitoes

Prior to this interception, ME and AQIS had conducted joint receptacle surveys on 13 November 2009 for exotic mosquitoes at risk locations within the 400m quarantine zone in the Darwin port area. This incorporated the treatment of actual and potential breeding receptacles with S-methoprene pellets to prevent the emergence of adult mosquitoes. These applications of S-methoprene were assessed as still active (up to and including the first week of February 2010), when reared *Aedes tremulus* larvae from a marked-as-treated tyre either perished upon pupation or did not survive to the pupal stage. Many earthmoving truck tyres were identified as potential breeding sites during this survey and removal or drilling on multiple surfaces to allow for complete drainage was recommended.

Figure 3: Exotic mosquito interception locations 25 January - 5 February; enhanced adult surveillance locations; fog route 25 January, and sites of human landing collections 8 February 2010



Enhanced Surveillance Response – Aedes aegypti

Ovitraps

ME and AQIS constantly maintain 8 ovitraps (egg traps) within Perkins and the adjacent premises of Frances Bay Marine. AQIS also has a sentinel tyre trap located in the Quarantine shed at Perkins.

On 28 January 2010, 3 extra ovitraps were set and serviced weekly for 4 weeks (Figure. 3).

Adult traps

On 28 January 2010, 4 Biogents® sentinel (BG) traps were deployed. (Figure. 3). The BG traps were baited with CO_2 gas delivered through a regulator attached to a D size gas bottle and these were run continuously for 1 week. The catch bags were collected and reset after ~24 hrs for weekdays and around 72 hrs over the weekend. After the initial week, BG catch bags were collected once per week for 3 more weeks until 5 March.

Detection 2 – Aedes albopictus

A collection of 3 *Aedes albopictus* males was made on 5 February in one of the BG traps set for the detection of additional *Ae. aegypti*. One male was positively identified using a taxonomic key,[7] while the remaining 2 were probably *Ae. albopictus* with considerable scale damage. The 2 rubbed males have been sent for DNA analysis.

The positive trap was located in the Quarantine shed on the international wharf at Perkins, and is adjacent to where overseas vessels berth (Figure 3, BG trap 2). The last vessel to have berthed there was the *M.V. Arafura Endeavour* on 29 January. Since this vessel voyages along the same route as the *M.V. Kathryn Bay*, the *Ae. albopictus* originated either from Singapore or Timor Lesté. It is possible that the *Ae. albopictus* adults could have harboured in shipping containers or on the vessel. On docking or opening of the shipping container for inspection, the mosquitoes could have dispersed to the BG trap (Figure 4).

Figure 4. Quarantine shed with opened shipping container and baggage being inspected. BG trap 2 (circled).

Elimination and Survey Procedures – Aedes albopictus

On the same day as the *Ae. albopictus* detection (5 February), a receptacle survey was conducted within the vicinity of the international wharf to detect any new, or previously overlooked potential mosquito breeding sites. Potential sites were located in the wash down bay channel and sump, and the wash down bay recycled water holding tanks. These areas were treated with alpha-cypermethrin and S-methoprene briquettes. It was noted during this survey that the residual pyrethroid (alpha-cypermethrin) treatment for the previous *Ae. aegypti* interception to earthmoving tyres near the international area contained numerous *Culex quinquefasciatus* mosquitoes dead on the water, indicating good knock down by the insecticide for harbouring adult mosquitoes.

The wash down bay area on the international wharf was targeted for further scrutiny a few days later. This site had water pooling in a steel covered drain channel. On Monday 8 February, the steel covers were removed with a forklift to reveal that the drainage channels were partially blocked with damp silt, about 5 cm deep. The previous pooled water observed on 5 February was no longer present and no larvae or adults were present in the drain. The silt was removed by Perkins the same day and was disposed of according to quarantine procedures. The only remaining water was located in the sump at the end of the drain channels, which then lead to the water recycling system.

The water recycling system behind the wash down bay was assessed in terms of chlorination procedures and system flow dynamics. Some overflow pipes from the holding tanks needed sealing and screening to prevent adult mosquito entry. The access pits also need screening, since the water returning into the system via these pits may sit for extended periods with no further chlorine treatment.

The Perkins yard and Frances Bay Marine premises were fogged on the evening of the *Ae. albopictus* detection.

Additional receptacle mosquito breeding surveys were conducted between 8 and 17 February to the north and south of Perkins, including the Fisherman's Wharf, the 'Duck Ponds' marina, Pearl Marine Engineering, and Frances Bay Marine. The remaining tank-containers from the *M.V Arafura Endeavour* were located, with 3 of these reported to be on the vessel back to Singapore and the remainder held within the Perkins yard. These were inspected and were not holding water. This was due to the inclusion of drainage holes in the design of the access hatches, some of which do not have lids. However, it was noted that these drainage holes were capable of being blocked by accumulations of soil, grass and leaves.

Enhanced Surveillance Response – Aedes albopictus

Ovitraps

The 3 additional ovitraps that were set on 28 January (Figure 3) were continued for an

additional week until 5 March. Routinely placed ovitraps continue to be operated as normal.

Adult traps
Four BG traps were operated continuously for 1 week from 5 February and serviced daily. After 12 February, 2 BG traps were operational for 4 days continuously per week for 4 weeks for the surveillance of further exotic adult mosquitoes.

Human landing collections
Adult mosquito catches using human bait subjects for landing collections were conducted at 5 locations in the late afternoon on 8 February. The collection sites were on the western perimeter of Perkins, Frances Bay Marine, Fisherman's pontoon, and west on the escarpment leading to Darwin City (Figure 3). Collection sites were amongst dense vegetation or in harbourage areas in close proximity to areas of vegetation.

Results

Aedes aegypti

No *Ae. aegypti* adults or larvae were recovered from any of the surveillance methods used after the first interception on 25 January. However, adults specimens of *Aedes katherinensis*, an NT endemic mosquito not recorded in Darwin city for 30 years, were collected at Perkins engineering in BG trap 3 on 1 February, with larvae found in a vehicle tyre in an area ~ 400m north of Perkins on 11 February.

Aedes albopictus

No further *Ae. albopictus* adults or larvae were collected subsequent to the detection on 5 February.

Discussion

It does not appear to be a deliberate design to make the lower section of the access hatches 'water tight', and on inspection of numerous other tank-containers, the incorporation of drainage holes in the design of the lower component of the hatch is common. Any tanks having the water holding design need drainage holes, to allow for complete drainage of any water or spilled fuel.

The *Ae. aegypti* importation within the tank-container access hatch was assessed as a low risk importation, because of the developmental stages present, the small numbers, and the sex ratios, with possibly only 3 to 5 adults emerging and with a high probability that the emergences were all males. In general, with *Aedes* receptacle breeding mosquitoes, males emerge first from any single breeding cohort. The collection of mostly 4^{th} instars, with only a few male pupae supports an assessment that the adult emergences had only just begun when the breeding site was detected.

The risk level for the *Ae. albopictus* interception was considered to be higher than that for *Ae. aegypti*, but only at a moderate level, because the adult stage was involved, and the fact that the source of the adults and the potential numbers of females involved, if any, has not been clearly established.

The most probable explanation for their arrival is that there was a water-holding receptacle on or offloaded as cargo from the *M.V. Arafura Endeavour*, which contained larvae and pupae. A few males escaped, while any remaining larvae and pupae were probably inadvertently destroyed or left on the vessel when it departed.

The alternative explanation of a few harbouring adults on board the vessel or in an offloaded shipping container dispersing to the BG is considered less likely, as the adults were all males. Harbouring adults in normal circumstances would be expected to be mainly females.

The results of the surveillance following the elimination responses indicate that the probability of an establishment of either *Ae. aegypti* or *Ae. albopictus* in Darwin port from these interceptions is now very low.

This is the first time BG traps baited with CO_2 have been deployed by ME and AQIS in surveillance for risk importation events in Darwin port facilities. The current routine adult trap (a modified Encephalitis Virus Surveillance trap) is located ~270m from the unloading area of the international wharf. With the recent addition of the BG baited CO_2 traps as a routine monitoring method at locations in close

proximity to the unloading and inspection areas of Darwin port, the probability of detections of exotic adult mosquitoes is now likely to increase. The detection of the *Ae. albopictus* males and of the endemic *Ae. katherinensis* demonstrates the effectiveness of the CO_2 baited BG traps.

Recommendations

Tank –container access hatches
Any tanks having the water holding design need drainage holes or a larger gap at the base of the hatch flush with the tank surface to allow for complete drainage of any water or spilt fuel.

Recycled water storage tanks
The over flow pipes from the holding tanks behind the wash down bay at Perkins international wharf should be screened and any gaps in pipe connections sealed. The inspection hatches on top of the tanks need to be repaired or replaced to ensure they are completely sealed.

Water recycling pits
Although the water in the main holding tank is kept at 5 ppm of chlorine, it is possible for chlorine in the water recovery pits to be diluted by rainwater runoff or be reduced to low levels when there are periods of infrequent use. Chlorine could then fall to a non-lethal level for larval mosquitoes.

The pits associated with the recycled water tanks behind the wash down bay need to be appropriately screened to prevent adult mosquito entry.

Wash down bay
The drainage channels that contained silt need to be routinely cleared to prevent water pooling. The bottom of the channel should be inspected and re-concreted if necessary so that a slope is created, which will allow water to effectively drain out of the channels and into the sump.

Wash down bay sump
Water pools in the wash down bay sump and, because the outlet pipe is ~ 5 cm above the level of the sump, can also pool for a distance along the drain channels. The channels and sump should be re-concreted to allow for more complete drainage of the water, or the outlet pipe could be lowered and screened.

Routine adult mosquito surveillance
At least 1 CO_2 baited BG trap should be maintained in the Perkins quarantine area for at least a 4 day collection period each week. There should be consideration to run this trap over a 7 day period with twice weekly catch collections. Regularly running a BG trap in this area increases the probability for exotic mosquito detection in close proximity to docking, offloading and inspection areas receiving overseas cargo vessels or any vessels originating from north Queensland where *Ae. aegypti* or *Ae. albopictus* occur.

Acknowledgements

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References

1. Shortus M, Whelan PI 2006a. Interim Report to the National Arbovirus and Malaria Advisory Committee on the detection of exotic mosquitoes in tyres at Perkins Shipping, Darwin, Northern Territory on 12 May 2006. *Northern Territory Disease Control Bulletin*. 13:2:29-32.
2. Lamche G, Whelan P, Espinoza H. 2004. Exotic mosquitoes detected in tyres at East Arm Wharf, Darwin NT, 1 December 2003. *The Northern Territory Disease Control Bulletin*. 11:1:4-6.
3. Whelan PI, Hayes G, Tucker G, Carter J, Wilson A and Haigh B. 2001a. The detection of exotic mosquitoes in the Northern Territory of Australia. *Arbovirus Research in Australia*, 8:395-404.
4. Nguyen H, Whelan PI. 2007. Detection and elimination of *Aedes albopictus* on cable drums at Perkins Shipping, Darwin, NT. *Northern Territory Disease Control Bulletin*. 14:3:39-41 5.
5. Whelan PI, Russell RC, Hayes G, Tucker G and Goodwin G. 2001b. Exotic Aedes mosquitoes: Onshore detection and elimination in Darwin, Northern Territory, Australia. Communicable Disease Intelligence. 25:4:283-285.
6. National Arbovirus Malaria Advisory Committee, vector sub-committee, November 2006 (Draft). Proposed protocol for action when a 'risk importation' or introduced exotic mosquito is detected.

7. Huang YM. 1979, Medical entomology studies - XI. *Contributions of the American Entomological Institute*. 15:6:12-26.
8. Shortus M, Whelan PI. 2006b. Recommended interim water receptacle treatment for exotic mosquitoes on international foreign fishing vessels arriving in Australia. *Mosquito Bites in the Asia Pacific Region. Mosquito Control Association of Australia*. 1:2.
9. Pettit WJ, Whelan PI, McDonnell J, Jacups SP (submitted *JAMCA*). Efficacy of alpha-cypermethrin and lambda-cyhalothrin applications in tires to prevent *Aedes* mosquito-breeding.

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Salt marsh mosquito larval control in Leanyer coastal wetland, Northern Territory

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Abstract

A coastal wetland with important larval habitats for Aedes vigilax (Skuse), the northern salt marsh mosquito is located adjacent to the northern suburbs of Darwin. This species is a vector for Ross River virus and Barmah Forest virus, as well as an appreciable human pest. To improve aerial larval control, we identified the most important vegetation categories and climatic/seasonal aspects associated with aerial control operations in this wetland after inundation with tide, rain and tide and rain combined.

The analyses showed that the Schoenoplectus/ mangrove areas require most of the control after inundation by tide only (30.1%), and also extensive control when tides and rain are coinciding (18.2%). Tide-affected reticulate vegetation requires extensive control after inundation by rain only (44.7%), and when tide and rain inundation coincide (38.0%). The analyses further showed that most of the control needs to be carried out between September and January, with a control peak in November and December.

To maximise the efficiency of aerial salt marsh mosquito control operations in northern Australia, aerial control efforts should concentrate on Schoenoplectus/mangrove and tide-affected reticulate areas, especially between September and January.

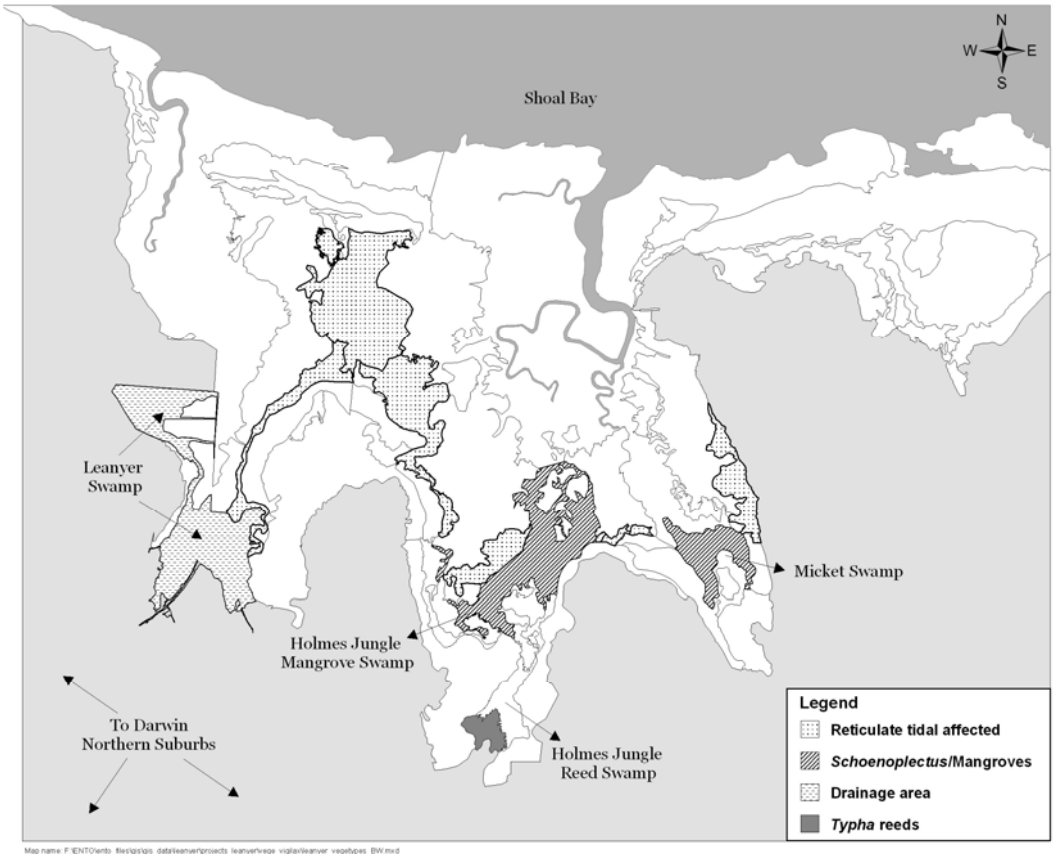
Keywords; mosquito, mosquito control, Aedes vigilax, Schoenoplectus/mangrove, wetlands.

Introduction

The Darwin area experiences a monsoon climate, with most rainfall occurring between November and April. A number of the northern Darwin residential suburbs are located close to an extensive coastal reed and upper mangrove wetland, which experiences seasonal tidal inundation. This wetland provides suitable breeding conditions for the northern salt marsh mosquito *Aedes vigilax*.[1] This mosquito species is not only a human pest species[2,3] but is also a major vector for Ross River virus and Barmah Forest virus in the Northern Territory (NT).[4,5] Thus, control of *Ae. vigilax* is of great importance for disease prevention and nuisance reduction in the NT.

Medical Entomology of the Department of Health and Families conducts integrated mosquito control for *Ae. vigilax* breeding in this wetland.[6] This paper outlines the vegetation categories requiring most of the control effort for *Ae. vigilax* larvae, and shows in which months most of the control needs to be carried out. By identifying the most important

Figure 1. Darwin's northern suburbs swamps; Leanyer, Holmes Jungle, Micket Creek and Shoal Bay



vegetation categories and seasonal conditions associated with *Ae. vigilax* breeding, survey time can be reduced and aerial salt marsh mosquito larval control can be greatly improved.

Methods

Geographic Information System (GIS) analysis

Aerial larval mosquito survey and control operations have been conducted in the various vegetation categories in Leanyer, Holmes Jungle, Micket Creek and Shoal Bay swamps, within a 5km radius of Darwin's northern suburbs margin (Figure 1). We determined the vegetation categories (as defined by the NT herbarium)[7] most associated with Ae. vigilax aerial control from July 2000 to June 2007. Detailed methodology can been found in recent publications.[8,9,10]

Data analyses

To compare the area sprayed for each vegetation category over the study period, a generalised linear model using a binominal distribution with logit link was applied to the aerial control data. Control areas were separated into tide only inundation (Model 1), rain only inundation (Model 2), and rain and tide inundation coinciding (Model 3). The free draining lower mangroves vegetation category was used as the reference vegetation, where little breeding is expected, and March was used as the reference month, when the least breeding is expected. The results were displayed as odds ratios, indicating the likelihood of areas needing to be sprayed compared to the reference vegetation and the likelihood of when control would most likely need to occur.

Results

For the tide only model (1), greatest control efforts were directed towards the *Schoenoplectus*/mangrove (i) vegetation category (OR = 46.8) (Table 1). The *Schoenoplectus*/mangroves are mainly

Table 1. Most important vegetation categories controlled for *Ae. Vigilax* for tide, rain, and rain and tide coinciding, July 2000 to June 2007

	Tide only (1)		Rain only (2)		Tide & rain (3)	
Vegetation category	**OR**	***P*-value**	**OR**	***P*-value**	**OR**	***P*-value**
Drainage area (k)	14.24	0.001	35.74	0.001	41.3	0.001
Reticulate - tide affected (b)	19.45	0.001	100.3	0.001	74.4	0.001
Schoenoplectus /mangroves (i)	46.81	0.001	39.37	0.001	71.6	0.001
Typha (g)	16.50	0.001	124.7	0.001	59.5	0.001
Lower mangroves (a)	1.00		1.00		1.00	

Table 2. Vegetation categories and areas controlled (%) in the northern coastal Darwin wetlands July 2000 to June 2007

Vegetation categories	Total area (ha)	Tide* (% controlled) (Model 1)	Rain** (% controlled) (Model 2)	T&R*** (% controlled) (Model 3)
Lower mangroves	908.35	6.8	2.3	2.6
Reticulate tide affected	190.7	26.7	44.7	38.0
Reticulate (non tidal)	64.03	1.9	10.1	6.2
Brackish grassland	135.11	3.2	2.4	2.5
Grassland floodplains	263.38	2.4	6.5	3.9
Eleocharis	65.18	8.8	7.2	8.4
Typha	9.18	1.1	2.6	1.5
Schoenoplectus	24.85	5.5	3.0	3.7
Schoenoplectus/ mangroves	94.64	30.1	9.0	18.2
Dune/ interdune depressions with mangroves^	188.65	0.7	1.1	0.8
Drainage area	90.45	9.4	7.9	10.3
Mudflat	650.66	3.1	3.0	3.2
#Other	453.61	0.3	0.3	0.8

*Tide – Control for tide-only.
**Rain – Control for rain- only
***T&R – Control for rain and tide coinciding
- Other includes: monsoon rainforest, open woodland, sewage pond area, *Pandanus*, paperbark, freshwater sedge areas
^ - Beach chernier with tidally influenced interdune depressions

comprised of *Avicennia marina* and *Schoenoplectus litoralis.* For the rain only model (2), control efforts were highest for the tall *Typha* reeds (g) (OR = 124.7), followed by the tidal affected reticulate areas (b) (OR = 100.3) (Table 2). The tidal affected reticulate areas are mostly comprised of *Xerochloa imberbis* and *Sporobolus virginicus* grasses. However, the largest area of control (44.69%) for rain only was carried out in the tide affected reticulate vegetation (b), with only 2.64% carried out in the *Typha* vegetation category (g), indicating that the majority of control is required in the tide affected reticulate areas, which constitute a much larger area (Table 2). For the rain and tide coinciding (model 3), control efforts were most strongly associated with the tidal affected reticulate areas (OR = 74.4), closely followed by the *Schoenoplectus*/ mangrove vegetation (i) (OR = 71.6) (Table 1). The highest area of control (38.0%) for rain and tides coinciding was carried out in the reticulate tidal affected (b) category (Table 2).

Months

The analyses of control efforts indicated that most of the control was carried out between September and January, with a control peak in December (OR = 54.60) (Table 3).

Table 3. Aerial control carried out by month for all vegetation categories July 2000 to June 2007

Month	OR	P-value	Highest average tide per month (m)
Jan	21.31	0.001	7.7
Feb	5.14	0.001	7.8
Mar	1.00		7.8
Apr	2.94	0.001	7.7
May	2.95	0.001	7.6
Jun	1.86	0.001	7.5
Jul	1.25	NS	7.5
Aug	5.53	0.001	7.6
Sep	10.16	0.001	7.6
Oct	24.94	0.001	7.7
Nov	44.16	0.001	7.7
Dec	54.60	0.001	7.6

Discussion

Our results indicate that after tide only inundation, the *Schoenoplectus/*mangrove vegetation category requires most of the aerial larval control for *Ae. vigilax* in the Darwin coastal wetland, with additional control required when tide and rain events coincide. The reason is that the *Schoenoplectus/*mangroves are partly inundated by relatively small monthly high tides >7.5m in the early dry season that trigger *Ae. vigilax* breeding. Higher tides in the late dry season and early wet season further increase inundation, and thus *Ae. vigilax* breeding, in this vegetation category. Due to the regular flooding-desiccation cycle in this area, repeated and extensive larval control is required throughout the late dry and early wet season until heavy rains seasonally flood the whole area, and oviposition sites are no longer available. Considering the relatively large area of *Schoenoplectus/*mangroves in close proximity to the northern urban areas of Darwin, it is of high importance as an *Ae. vigilax* breeding habitat, and is responsible for many of the public complaints regarding mosquitoes (Figure 2).

Further results from this study show that the tide-affected reticulate vegetation requires extensive control after inundation by rain, and inundation by tide and rain coinciding. This is because high tides >7.7m in October and November, together with early wet season rains, lead to repeated inundation of this vegetation category. After inundation by tides or rain, water remains pooling with no fish predation, and thus mosquito breeding occurs at high densities until the area is seasonally flooded by heavy wet season rains.

For inundation by rain only, significantly more *Ae. vigilax* larval control was conducted in the *Typha* reeds compared to other vegetation categories. The *Typha* reeds area in the wetland is relatively small compared to other vegetation categories, and thus, control of this area is less important than for the large areas, such as the *Schoenoplectus/*mangroves or the tide-affected reticulate areas.

Aerial larval control varies between the wet and dry seasons, with most of the larval control

Figure 2. Public complaints for pest levels of *Aedes vigilax* in Darwin's northern suburbs August 1983-2008, 278 complaints (dots may represent multiple complaints at the same location).

required between September and January, with a marked peak in November and December. Larval control after tidal inundation is less important during the wet season when the wetland is seasonally flooded, making it unavailable for *Ae. vigilax* oviposition. However, once the wet season flooded areas are dry in the mid-dry season, increasing tides again inundate potential *Ae. vigilax* breeding habitat, and *Ae. vigilax* breeding increases, with more breeding following early wet season rain in November and December.

In summary, our results demonstrate the benefits gained by identifying vegetation categories and seasonal aspects most associated with aerial salt marsh mosquito larval control operations in the coastal wetlands adjacent to Darwin's northern suburbs. Maximum efficiency can be achieved by focussing surveys and aerial control on the extensive areas of *Schoenoplectus/*mangroves

and tide-affected reticulate vegetation. Additionally, to further reduce the egg laying capacity of *Ae. vigilax* in succeeding flooded areas, early seasonal hatches of *Ae. vigilax* need to be diligently controlled. While repetitive control is required in the *Schoenoplectus/* mangrove areas in the mid dry season to early wet season after each inundation, control in the tide-affected reticulate areas is only required in the late dry season when tides are high enough to reach those areas.

The present practice of extensive helicopter surveys for *Ae. vigilax* breeding could be reduced by pre-emptive sprays of the *Schoenoplectus/*mangrove and the tide-affected reticulate areas based on breeding initiated tide or rain events. To further reduce the initial survey time for *Ae. vigilax* larval habitats, a small number of indicator sites could be used in these vegetation categories to verify control requirements. Additionally, the 2 main productive vegetation categories could be earmarked for engineering rectification measures or avoidance in the urban or development planning process. These results may be applied to other coastal areas in northern Australia.

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References

1. Whelan PI. Integrated mosquito control in Darwin. *Arbo Res Aust*. 1989;5:178-85.
2. Marks EN, editor. An atlas of common Queensland mosquitoes- with a guide to common Queensland biting midges by E J Reye. Mimeo ed. St Lucia: Univ Qd. Bksp; 1967.
3. Webb CE, Russell RC. Towards management of mosquitoes at Homebush Bay, Sydney, Australia. I. Seasonal activity and relative abundance of adults of *Aedes vigilax*, *Culex sitiens*, and other salt-marsh species, 1993-94 through 1997-98. *J Am Mosq Control Assoc.* 1999 Jun;15(2):242-9.
4. Jacups SP, Whelan PI, Currie BJ. Ross River virus and Barmah Forest virus infections: a review of history, ecology, and predictive models, with implications for tropical northern Australia. *Vector Borne Zoonotic Dis.* 2008 Summer;8 (2):283-98.
5. Russell RC. Mosquito-borne arboviruses in Australia: the current scene and implications of climate change for human health. *Int J Parasitol*. 1998 Jun;28(6):955-69.
6. Whelan PI. Mosquito control in the Leanyer Swamp. Northern Territory *Communicable Diseases Bulletin*. 2007;14(2):19-20.
7. Northern Territory Government. Herbarium. Department of Natural Resources, Environment, *The Arts and Sport*; 2008.
8. Jacups SP, Kurucz N, Whelan PI, Carter JM. A comparison of *Aedes vigilax* larval population densities and associated vegetation categories in a coastal wetland, Northern Territory, Australia. *Journal of Vector Ecology*. 2009;34(2), 311-16.
9. Kurucz N, Whelan PI, Carter JM, Jacups SP. A geospacial evaluation of *Aedes vigilax* larval control efforts across a coastal wetland, Northern Territory, Australia. *Journal of Vector Ecology*. 2009;34(2):317-23.
10. Kurucz N, Whelan PI, Carter J, Jacups SP. Vegetation parameters as indicators for salt marsh mosquito larval control in coastal swamps in northern Australia. *Arbo Res Aust* 2009;10:84-90.

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Centre for Disease Control

March 2010

Scabies

What is scabies?

Scabies is a skin condition caused by a microscopic mite called *Sarcoptes scabiei*. The mites burrow under the skin and the females lay eggs. The itch results from the inflammatory response to mite excreta and other components.

It is a common problem in many remote Aboriginal communities within the Northern Territory where in some areas up to 50% of children and 25% of adults are affected.

What are the symptoms?

The first time someone is infected symptoms do not appear until 2-6 weeks after exposure. If someone has been infected previously, symptoms usually take 1-4 days to appear.

A red lumpy rash appears. Rarely little burrow markings about 10mm long can also be seen. In adults the rash is usually around the buttocks, wrist and ankles, and between the fingers and toes. It also commonly occurs in the folds of the skin around the armpits, elbows and genitals. In young children the rash may be from head to toe with early pustule formation on the hands and feet. The rash is very itchy, often much more so at night.

Are there any complications?

Scratching of the affected area often causes secondary infection with *Staphylococcus* and *Streptococcus* bacteria. Streptococcal infections can be associated with kidney infections (glomerulonephritis) and rheumatic fever so early antibiotic treatment for skin infection is recommended.

How is it spread?

The scabies mite is spread from person to person by direct physical contact. Contact must be prolonged – a quick handshake or hug will not spread it. Although the scabies mite does not live long outside the human body it can also be spread by clothes and linen that have been used by a person with scabies if they have been worn or used immediately before. People with untreated crusted scabies can be 'core transmitters' of scabies in communities and health care facilities. Scabies will continue to be spread until all mites and eggs are destroyed.

A similar illness occurs in dogs, however the mite that causes dog scabies is different from that which causes human scabies.

Who is most at risk?

Scabies occurs worldwide, however people living in crowded conditions with poor hygiene and malnutrition are most at risk.

How is scabies treated?

For the individual

There are currently a number of creams or lotions for the treatment of scabies for adults and children available. These include:

- 5% Permethrin (Lyclear)
- Benzyl Benzoate (Ascabiol, Benzemul)

CENTRE FOR DISEASE CONTROL

For babies less than 2 months old:

Crotamiton cream (Eurax)

Application of the treatment varies depending on which one is used, so it is important to read the instructions carefully.

The person who is infected should first have a shower or bath to soften the skin. The treatment should then be applied to the skin as per the instructions and left on for the recommended period of time before washing it off. While the treatment is on the skin a complete set of new clothes should be worn.

It is recommended that treatment be repeated after 1 week.

Tingling and itching may still be present for 1 to 2 weeks after treatment.

For others in the house

For the treatment to be successful all members of the household and other close contacts should be treated at the same time as the infected person. Contacts may be incubating scabies at the time of treatment and therefore not show any symptoms.

For the household

All clothing, towels and linen need to be washed in hot soapy water and left to dry in the sun.

Mattresses and pillows should be put out in a shaded position in the late afternoon, sprayed with surface spray containing pyrethroid according to the directions, left overnight, then put in sun full day the next day. Curtains, chair covers and carpets may also need to be sprayed with surface spray.

How is scabies prevented?

Early diagnosis and prompt treatment helps to prevent the spread of scabies.

Healthy Skin Programs are being conducted by many communities, for further information about this contact your nearest health centre.

What is Crusted (Norwegian) scabies?

While most people are infested with about 10 to 15 mites, in crusted scabies, there is a proliferation of mites and people are infested with thousands of mites. Sometimes this happens because a person's immune system is not working well due to other illness. However, in many cases in the NT there are no clear underlying immune problems.

Crusted scabies does not look like scabies. The rash appears as scaling, thickening and crusting of the skin. Often this appears on buttocks, elbows and arms.

Mild cases of crusted scabies can be treated in the community with creams, lotions and oral ivermectin. Severe cases will require admission to hospital.

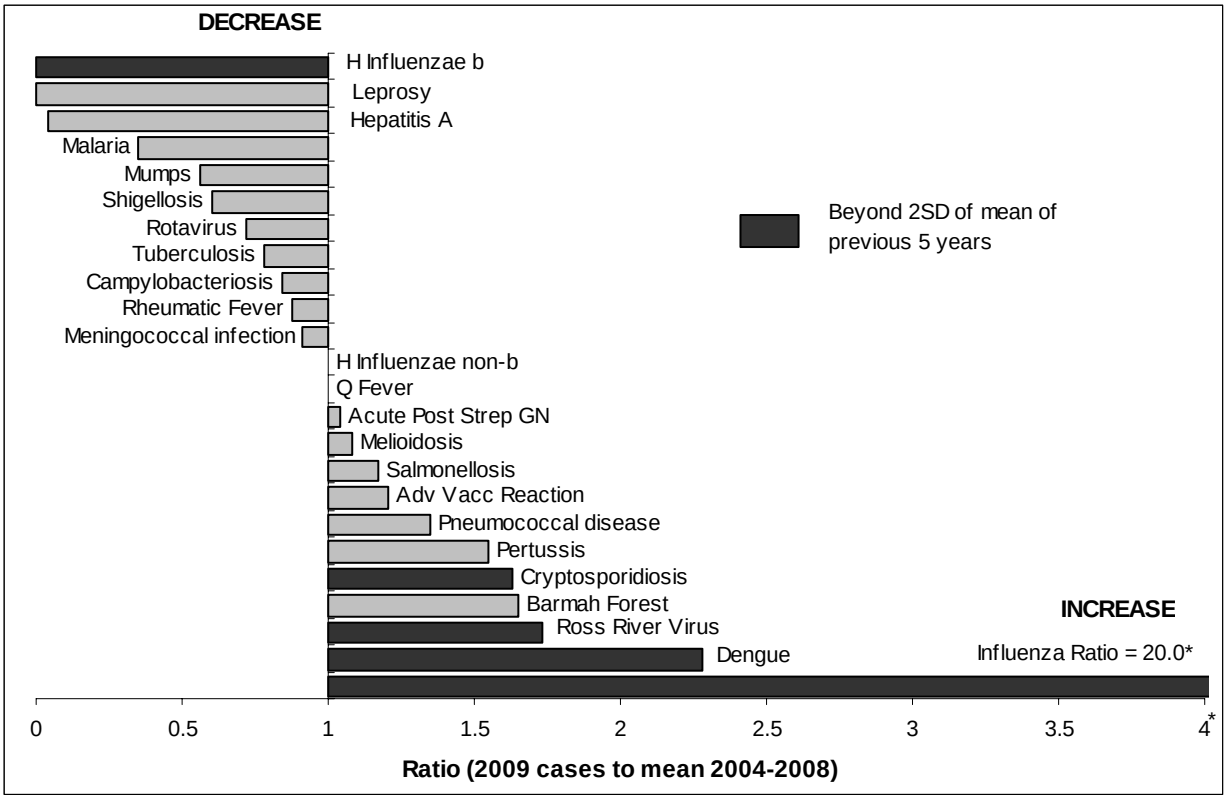
For more information contact the Centre for Disease Control in your region

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

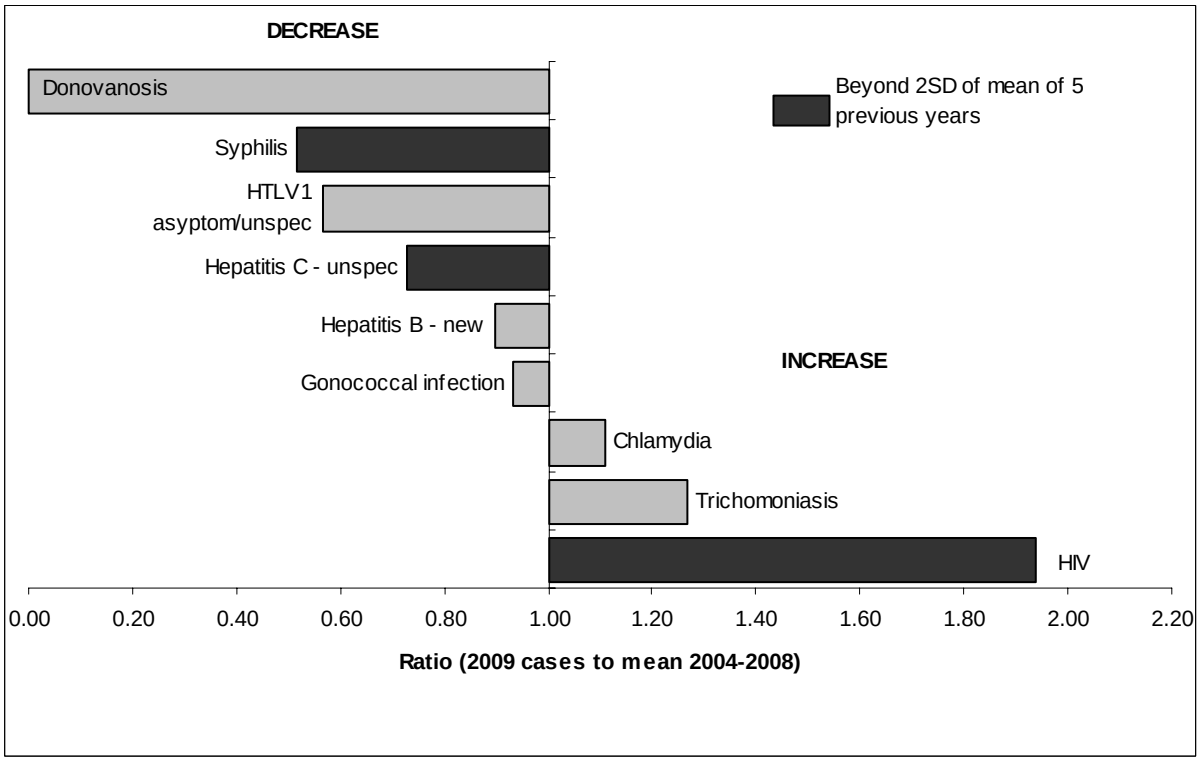
NT NOTIFICATIONS OF DISEASES BY ONSET DATE & DISTRICTS
1 January — 31 December 2009 & 2008

	Alice Springs		Barkly		Darwin		East Arnhem		Darwin		NT	
	2009	2008	2009	2008	2009	2008	2009	2008	2009	2008	2009	2008
Acute Post Strep Glomerulonephritis	5	9	1	4	16	20	13	3	5	2	40	38
Adverse Vaccine Reaction	6	9	1	4	30	28	6	3	6	1	49	45
Amoebiasis	0	0	0	0	0	2	0	0	0	0	0	2
Arbovirus not otherwise specified	0	0	0	0	2	0	0	0	0	0	2	0
Barmah Forest	14	12	6	0	85	56	8	4	9	3	122	75
Campylobacteriosis	58	62	10	8	129	161	1	8	18	18	216	257
Chickenpox	6	26	2	7	49	43	16	37	18	2	91	115
Chlamydia	777	904	56	56	956	913	168	184	209	231	2166	2288
Chlamydial conjunctivitis	116	14	13	0	10	3	0	0	3	5	142	22
Cryptosporidiosis	49	47	4	4	69	37	16	7	18	7	156	102
Dengue	0	2	0	0	40	20	2	1	0	0	42	23
Donovanosis	0	1	0	0	0	0	0	0	0	0	0	1
Gonococcal conjunctivitis	2	0	0	0	1	2	1	0	0	0	4	2
Gonococcal infection	903	781	46	78	268	318	96	110	237	263	1550	1550
Hepatitis A	1	1	0	0	0	2	0	0	0	0	1	3
Hepatitis B - chronic	27	63	3	1	45	72	71	71	16	20	162	227
Hepatitis B - new	0	1	2	1	3	4	1	0	1	2	7	8
Hepatitis B - unspecified	53	47	9	2	80	104	1	3	22	32	165	188
Hepatitis C - chronic	0	0	0	0	0	0	0	2	0	0	0	2
Hepatitis C - new	0	2	0	0	3	3	1	1	2	0	6	6
Hepatitis C - unspecified	27	33	3	2	120	154	5	8	15	10	170	207
Hepatitis D	0	1	0	0	0	0	0	0	0	0	0	1
Hepatitis E	0	0	0	0	0	3	0	0	0	0	0	3
***H Influenzae* b**	0	0	0	0	0	2	0	0	0	0	0	2
***H Influenzae* non-b**	5	2	0	1	2	1	1	0	0	0	8	4
HIV	1	1	0	0	18	13	0	0	0	1	19	15
HTLV1 asyptomatic/unspecified	44	79	1	0	1	2	0	1	0	1	46	83
HUS	0	0	0	0	0	1	0	0	0	0	0	1
Influenza	620	65	62	24	965	98	158	4	279	8	2084	199
Kunjin Virus	0	0	0	0	1	0	0	0	0	0	1	0
Legionellosis	0	0	0	0	3	1	2	0	1	0	6	1
Leprosy	0	0	0	0	0	1	0	0	0	0	0	1
Leptospirosis	0	0	0	0	3	0	0	0	1	1	4	1
Listeriosis	0	0	0	0	1	0	0	0	0	0	1	0
Malaria	1	1	0	0	12	16	1	2	0	0	14	19
Measles	0	0	0	0	1	0	0	0	0	3	1	3
Melioidosis	0	0	0	0	24	17	3	1	3	5	30	23
Meningococcal infection	3	1	0	1	5	6	0	1	0	0	8	9
Mumps	1	34	9	13	3	2	0	1	1	2	14	52
MVE	0	0	0	0	2	1	0	0	0	0	2	1
Pertussis	24	35	10	14	166	373	14	2	9	54	223	478
Pneumococcal disease	46	28	6	4	28	24	5	3	8	1	93	60
Q Fever	3	3	0	0	0	0	0	0	0	0	3	3
Rheumatic Fever	23	12	0	2	8	26	11	2	13	6	55	48
Ross River Virus	56	31	26	4	297	195	28	13	37	19	444	262
Rotavirus	122	44	2	15	85	97	12	19	35	25	256	200
Salmonellosis	74	77	21	22	322	311	32	28	69	56	518	494
Shigellosis	54	89	7	14	16	46	10	13	8	15	95	177
STEC/VTEC	1	0	0	0	0	0	0	0	0	0	1	0
Syphilis	46	93	2	6	59	88	9	14	22	53	138	254
Syphilis congenital	1	1	0	0	2	0	0	0	0	0	3	1
Trichomoniasis	567	854	77	62	571	599	247	305	308	386	1770	2206
Tuberculosis	4	3	0	0	20	17	1	11	3	2	28	33
Typhoid	0	0	0	0	0	1	0	0	0	0	0	1
Typhus	0	0	0	0	0	1	0	0	0	0	0	1
Varicella unspecified	1	0	0	0	2	2	0	0	0	0	3	2
Vibrio food poisoning	0	0	0	0	1	1	0	0	0	0	1	1
Yersiniosis	0	0	0	0	4	1	0	0	0	0	4	1
Zoster	25	13	6	4	68	81	7	2	13	6	119	106
Total	**3,766**	**3,481**	**385**	**353**	**4,596**	**3,969**	**947**	**864**	**1,389**	**1,240**	**11083**	**9907**

Ratio of the number of notifications in 2009 to the mean 2004-2008: selected diseases



Ratio of the number of notifications in 2009 to the mean 2004-2008: sexually transmitted diseases



Comments on notifications p 43

Haemophilus influenzae type b

2009 was the first year since 1998 during which there were no *Haemophilus influenzae* type b (Hib) cases notified. The 5 year mean is 2 cases per annum. This is a welcome result but with evidence of continuing carriage of Hib in the indigenous community and recent changes to the vaccine schedule, it is important that we continue to closely monitor this disease.

Cryptosporidiosis

A 62.5% increase of cryptosporidium notifications for 2009 (156 vs. expected 96 FYM) is noted. On average 64% of cryptosporidium cases occur during the months January to June. In 2009 notifications for this period doubled (117 vs. expected 62) and increased to 75% of the total. While the increase was NT-wide it was most obvious in the Top End where the majority of cases occur. No outbreaks or particular sources were identified. Children under 2 years of age made up 79% of 2009 notifications with 37% of these children resident in Darwin.

Ross River Virus

The NT RRV figures were increased due to unusually high numbers of cases in January to May and September to November. The high wet season cases from January to April were largely due to cases from the Darwin urban area in the Darwin region, with additional contributions made by higher than usual numbers in the Darwin rural area and the Alice Springs urban area. The Darwin area cases were due to early and high wet season rain in November and December 2008 leading to high salt marsh vector numbers and following cases in January and February 2009 and the high tides in the September to November 2009 period leading to high salt marsh mosquitoes during September to December 2009, despite near record amounts of aerial salt marsh swamp spraying near Darwin by Medical Entomology.

The Alice cases were largely due to high wet season rain November-December 2008 and a peak in common banded mosquitoes from Ilparpa swamp in January 2009. An unusual feature of this year has been the continuation of cases in the Darwin urban area in the April-June period which is unusual and cannot be explained readily by vector numbers at that time.

Dengue Fever

The dengue cases recorded by the Northern Territory were all contracted overseas and are due to the trend of increasing dengue transmission in South East Asia and particularly our near neighbours including Timor Leste. There are no dengue vectors present in the NT, so dengue transmission in the Northern Territory at this time is not possible.

Influenza

Influenza notifications were 20 times the 5 year mean in 2009 and influenced by the H1N1 swine flu pandemic. This in part reflects more testing for influenza compared to a normal influenza season. See article page 1.

Syphilis

The decrease was a continuation of an existing decreasing trend noted in the last 5 years. It is believed to be the result of effective case detection and management, rather than due to decreased testing.

Hep C unspecified

This was consistent with an existing decreasing trend seen in the last 3 years. It is unlikely to be due to decreased testing, judging by available testing data. The specific reasons for this decrease are not known.

HIV

Compared with statistics from previous years, there was an increase in persons contracting HIV during overseas travel. The majority of cases were infected through heterosexual contact, similar to the previous report.

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Immunisation coverage for children aged 12-<15 months at 31 December 2009

Region	Number in District	% DTP	% Polio	% Hib	% Hep B	% Fully vaccinated
Darwin	289	87.2%	86.9%	90.7%	86.9%	85.1%
Winnellie PO Bag	106	87.7%	87.7%	94.3%	87.7%	86.8%
Palmerston/Rural	217	93.1%	93.1%	93.5%	93.1%	91.2%
Katherine	98	90.8%	90.8%	96.9%	90.8%	89.8%
Barkly	18	77.8%	77.8%	77.8%	77.8%	72.2%
Alice Springs	126	90.5%	90.5%	92.1%	90.5%	88.9%
Alice Springs PO Bag	64	93.8%	93.8%	93.8%	93.8%	89.1%
East Arnhem	56	91.1%	91.1%	96.4%	91.1%	91.1%
NT	974	89.8%	89.7%	92.8%	89.7%	88.0%
NT Indigenous	384	87.0%	87.0%	93.2%	86.7%	85.4%
NT Non-Indigenous	590	91.7%	91.5%	92.5%	91.7%	89.7%
Australia Indigenous	3,389	85.0%	84.9%	85.9%	84.9%	84.1%
Australia Non Indigenous	72,699	92.6%	92.6%	92.3%	92.1%	92.0%
Australia Total	76,088	92.3%	92.2%	92.1%	91.8%	91.6%

Immunisation coverage for children aged 24-<27 months at 31 December 2009

Region	Number in District	% DTP	% Polio	% Hib	% Hep B	% MMR	% Fully vaccinated
Darwin	267	92.1%	91.8%	89.5%	91.0%	91.4%	86.9%
Winnellie PO Bag	77	97.4%	97.4%	97.4%	97.4%	97.4%	97.4%
Palm/Rural	237	95.8%	95.8%	93.7%	95.4%	94.9%	92.0%
Katherine	82	98.8%	98.8%	95.1%	98.8%	98.8%	95.1%
Barkly	24	100.0 %	100.0%	100.0%	91.7%	100.0%	91.7%
Alice Springs	119	88.2%	89.1%	84.9%	88.2%	89.1%	84.9%
Alice Springs PO Bag	54	96.3%	96.3%	94.4%	96.3%	98.1%	94.4%
East Arnhem	51	96.1%	96.1%	98.0%	96.1%	98.0%	96.1%
NT	911	94.3%	94.3%	92.2%	93.6%	94.2%	90.7%
Indigenous	358	95.3%	95.3%	91.9%	95.0%	95.3%	91.3%
Non-Indigenous	553	93.7%	93.7%	92.4%	92.8%	93.5%	90.2%
Australia Ind	3,442	93.7%	93.6%	90.0%	92.8%	93.1%	87.1%
Australia Non Ind	72,754	94.8%	94.7%	93.6%	93.7%	93.7%	91.1%
Aus Total	76,196	94.7%	94.7%	93.4%	93.7%	93.7%	91.0%

Immunisation coverage for children aged 60-<63 months at 31 December 2009

Region	Number in District	% DTP	% Polio	% MMR	% Fully vaccinated
Darwin	227	73.6%	73.6%	73.6%	72.2%
Winnellie PO Bag	87	97.7%	97.7%	97.7%	97.7%
Palm/Rural	190	77.9%	77.9%	76.8%	76.3%
Katherine	90	90.0%	88.9%	91.1%	88.9%
Barkly	20	100.0%	100.0%	100.0%	100.0%
Alice Springs	119	82.4%	82.4%	81.5%	80.7%
Alice Springs PO Bag	36	88.9%	88.9%	88.9%	88.9%
East Arnhem	48	85.4%	85.4%	85.4%	85.4%
NT	817	82.3%	82.1%	82.0%	81.2%
Indigenous	327	86.9%	86.9%	86.9%	86.2%
Non-Indigenous	490	79.2%	79.0%	78.8%	77.8%
Australia Ind	3,060	79.0%	79.0%	79.5%	78.2%
Australia Non Ind	66,345	83.5%	83.4%	83.3%	82.8%
Aus Total	69,405	83.3%	83.2%	83.1%	82.6%

Immunisation coverage 31 December 2009

Immunisation coverage rates for Northern Territory (NT) children by regions based on Medicare address postcode as estimated by the Australian Childhood Immunisation Register are shown on page 45.

Background information to interpret coverage

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

The cohort of children assessed at 12 to <15 months of age on 31 December 2009 were born between 1 September 2008 and 31 December 2008 inclusive. To be considered fully vaccinated, these children must have received 3 valid doses of vaccines containing diphtheria, tetanus, pertussis, and poliomyelitis antigens, either 2 doses of PRP-OMP Hib or 3 doses of another Hib vaccine, and 2 doses of hepatitis B vaccine (not including the birth dose) (latest doses due at 6 months of age). All vaccinations must have been administered by 12 months of age.

The cohort of children assessed at 24 to <27 months of age on 31 December 2009 were born between 1 September 2007 and 31 December 2007 inclusive. To be considered fully vaccinated, these children must have received 3 valid doses of vaccines containing diphtheria, tetanus, pertussis, and poliomyelitis antigens, either 3 doses of PRP-OMP Hib or 4 doses of another Hib vaccine, and 2 doses of hepatitis B vaccine (not including the birth dose) and 1 dose of measles, mumps, rubella vaccine (latest doses due at 12 months of age). All vaccinations must have been administered by 24 months of age.

The cohort of children assessed at 60 to <63 months of age on 31 December 2009 were born between 1 September 2004 and 31 December 2004 inclusive. To be considered fully vaccinated, these children must have received 4 valid doses of vaccines containing diphtheria, tetanus, pertussis antigens, 4 doses of poliomyelitis vaccine and 2 valid doses of measles, mumps, rubella vaccine (latest doses due at 4 years of age). All vaccinations must have been administered by 60 months (5 years) of age.

Interpretation

Immunisation coverage in NT children was below the national average across all cohorts. Immunisation coverage in Indigenous children in the NT was higher across all cohorts compared to the national coverage of Indigenous children. Indigenous NT children had higher coverage than non-Indigenous NT children in the 24 to <27 months and 60 to <63 months cohorts but below non-Indigenous NT children in the 12 to <15 months cohort.

Immunisation coverage for NT children as a whole at 60 to <63 months of age (81.2%) remains lower than the younger cohorts, and this is a concern across Australia, with the national average for this cohort being 82.6%. For Indigenous NT children, immunisation coverage is higher for the middle cohort (ie. 91.3% at 24 to <27 months) but lower for the younger and older age groups (ie. 85.4% at 12 to <15 months and 86.2% at 60 to <63 months).

NT Malaria notifications October- December 2009

Merv Fairley, CDC, Darwin

There were 2 notifications of malaria received in the fourth quarter of 2009. The following table provides details about where the infection was thought to be acquired, the infecting agent and whether chemoprophylaxis was used.

Number of cases	Origin of infection	Reason exposed	Agent	Chemoprophylaxis
1	East Timor	Resident	*P. vivax*	No
1	Indonesia	National	*P. vivax*	No

Disease Control staff updates

Sexual Health

Darwin

Cathy Pell is returning to Sydney after 2 years working in the Top End. **Nathan Ryder** is the new Sexual Health Physician and commenced at the beginning of April.

Tilly Todhunter Sexual Health Nurse, Clinic 34 has left and has been replaced by **Kirsten Thompson.**

Anguree Jansen van Rensburg Administration Officer/Receptionist, Clinic 34, has also left with **Michelle Banks** replacing her.

Prue Boylan has transferred from RDH to take up the new position of Accreditation Officer for Clinic 34.

Patrick May commenced on 22 March 2010 as the Remote Sexual Health Coordinator in Darwin. Patrick has recently returned to Darwin after working in Cairns.

Immunisation

Darwin

Bruce Maley has completed his contract with the H1N1 pandemic influenza vaccination program and returned to work at RDH. (He returns as a new father to baby Max – congratulations!

Linda Pitts, AGV Course Coordinator has commenced 6 months Long Service Leave. **Ursula (Uschi) Janssen,** most recently serving as the H1N1 Vaccine program Coordinator will act as the course co-ordinator in Linda's absence.

TB/Leprosy

Darwin

Laura Edwards, recently from Sydney**,** has commenced in the TB/Leprosy Unit in Darwin as the Public Health Physician Trainee.

Vanessa Johnston has joined the TB / Leprosy Unit as a Medical Officer on a part-time basis. She will be helping with the TB Clinic and working on several projects with the Darwin Refugee Health Service. She has previously worked on a malaria research program in Africa and with resettling refugee communities in Melbourne and is a post-doctoral scholar at Menzies School of Health Research.

CDC Darwin

Mark McMillian has left the Northern Territory to return to Adelaide to work and spend time with family.

Christine Quirke joins CDC as the Senior Policy and Coordination Officer. Christine has been working in DHF Acute Care Policy and Services Development for the past 3 years and is a long term DHF employee.

Kristy Sporn, Business Manager has joined the team for 4 months.

Alice Springs

Robbie Charles, Sexual Health Aboriginal Health Worker has left, to go to university in Geelong.

Michelle Callard H1N1 nurse has also left Alice Springs to take up an infection control position in Batemans Bay, New South Wales.

Lauren Coelli has left her position as a remote Sexual Health Nurse, to do a stint in the Top End before moving back to Victoria to get married.

Jodi Pipes has resigned as the remote Sexual Health Co-ordinator and has moved back to Canada for family reasons.

East Arnhem

Michael Williams, AHW has transferred to Katherine to work in CDC.

Katherine

Margaret Cooper has "retired from the Territory" after 21 years living in and working in the NT. Margaret has moved to Canberra and is working in Communicable Diseases.

Margaret commenced work as a registered nurse in Tennant Creek on 19/10/1988. She then moved to Katherine Centre for Disease Control (CDC) unit where she has worked in the area of tuberculosis control as well as contributing in other areas of CDC for the past 18 years.

During those 18 years Margaret, along with her team at CDC, worked tirelessly and professionally to provide TB control measures to the Katherine Region. This often saw Margaret in communities raising awareness about TB, becoming a trusted healthcare provider and confidant to patients and their families.

Throughout the years she has been educating staff e.g. RNs, AHWs and new doctors about TB control and leprosy. She has contributed to the fight against TB and seen the fruits of her labour in reduced rates of TB in Katherine over the years.

During her time she has been part of outbreak investigations for measles, pertussis, salmonella, meningococcal disease and TB, of course, to name just a few conditions. She contributed to intense disaster responses for Katherine floods and community floods and to local disease control preparedness for conditions such as SARS and more recently pandemic H1N1.

Margaret, through her many years of service has provided excellent clinical service and follow-up and has shown understanding towards her patients and their families. She has trained other healthcare professionals in the ways of public health and contributed greatly to communicable disease control in the Katherine area. Margaret has been a very generous contributor to the CDC team and will be missed.