



# Communicable Diseases Intelligence

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## Editorial

**INFLUENZA SURVEILLANCE IN AUSTRALIA**

Kate Pennington, Christina Bareja, Sheena G Sullivan, Lucinda J Franklin, Jane Raupach

This issue of *Communicable Diseases Intelligence* contains a brief overview of the influenza surveillance systems in Australia as well as an original article on influenza coverage among pregnant Indigenous women in the Northern Territory and a short report on the timing of influenza vaccination in an Australian community-based surveillance system. The overview describes the systems based on the aspect of influenza activity that they represent, as well as their respective strengths and limitations in describing the epidemiology of influenza in Australia.

Influenza infection can manifest in a variety of ways, from mild to severe illness or even death. Although most people are susceptible to infection, individuals at the extremes of the age spectrum and populations with comorbidities tend to be the most vulnerable to more severe illness and complications. Globally, it is estimated that seasonal influenza affects 5% to 15% of the population annually, resulting in between 250,000 and 500,000 deaths.<sup>1,2</sup> Although rare, the emergence of a pandemic can result in large numbers of infections due to population susceptibility, with varying proportions of severity. For example the 1918 influenza pandemic caused an estimated 20–50 million deaths worldwide, whereas subsequent pandemics have resulted in many fewer deaths despite a large proportion of the world's population being susceptible to infection.<sup>2</sup> Economically, influenza is associated with both direct and indirect costs through health care costs and productivity losses.

The public health significance of influenza is derived from the rate with which the virus undergoes antigenic change, allowing it to evade immune recognition,<sup>3</sup> resulting in ongoing variability in population susceptibility and disease severity. Minor antigenic changes occur regularly in a process known as antigenic drift. This is the reason for annual, seasonal epidemics and also the reason why the World Health Organization (WHO) reviews the composition of influenza vaccines in the lead up to each hemisphere's season. In Australia's temperate climates, seasonal epidemics occur mainly during winter while in tropical and sub-tropical localities, influenza exhibits more complex, less clearly defined patterns.<sup>4</sup> Less frequently, the viruses undergo an abrupt antigenic shift causing the emergence of an antigenically

distinct virus, which is generally unrecognised by the population and tends to have more extreme outcomes, such as pandemics.

Influenza immunisation remains the most important intervention in preventing or attenuating influenza infection and mortality.<sup>5</sup> To keep pace with antigenic drift, continued protection requires annual vaccination with vaccine containing the most recent and closely matched virus strains to those circulating in the community. The Australian Government funds annual influenza vaccination under the National Immunisation Program to mitigate the impact of influenza on populations most vulnerable to severe disease: persons aged 65 years or over; persons with medical conditions placing them at risk of the more serious complications of influenza; pregnant women; and Indigenous Australians aged 6 to 59 months or 15 years or over.<sup>6</sup> Additionally, annual influenza vaccination is recommended for any persons aged 6 months or older to reduce the likelihood of becoming ill with influenza.<sup>5</sup>

The broad aim of public health surveillance is to ensure the systematic collection, analysis, interpretation, and dissemination of data regarding a health issue for use in public health action.<sup>7</sup> Effective and functional nationally representative surveillance data are critical for estimating the impact of influenza across the population. These data enable evidence-based decisions for public health action, the planning of appropriate control and intervention measures, and the effective allocation of resources.<sup>8</sup> Influenza surveillance is made complex by the non-specific disease presentation, including asymptomatic infections, and the volume of cases. It is not feasible, nor necessary, to identify every influenza infection. Moreover, no single system can adequately capture the variations in severity, circulating strains and population susceptibility.

In Australia, the National Influenza Surveillance Scheme (the Scheme) began in 1994.<sup>9</sup> Over time, the surveillance systems incorporated into the Scheme have evolved but have continued to focus on ensuring an understanding of influenza incidence, severity and virology. The 2007 and 2009 influenza seasons tested Australia's influenza surveillance systems, especially in terms of disease severity measurement capability and data collec-

tion sustainability. Influenza activity is currently captured through a collection of representative and stable surveillance systems that incorporate both syndromic and laboratory confirmed influenza infection identified in the community, general practices, hospitals and among deaths.

The National Influenza Surveillance Committee, a sub-committee of the Communicable Diseases Network Australia,<sup>10</sup> plays an important role in ensuring the influenza surveillance systems in Australia are effective, including improving performance, integration and interpretation, as well as the scalability of the systems in response to a pandemic. During the influenza season, the Australian Government Department of Health compiles data from each of the surveillance systems contributing to the Scheme, as well as international surveillance data, and publishes the *Australian Influenza Surveillance Report*<sup>11</sup> on its web site each fortnight. These reports are regularly utilised by Australia's international counterparts and data are used to inform the WHO's fortnightly assessment of regional and global influenza activity.<sup>12,13</sup> A more detailed description and analysis of the Scheme, including surveillance systems that function outside the Scheme, is provided in the paper *A Summary of Influenza Surveillance Systems in Australia, 2015*,<sup>14</sup> available on the Australian Government Department of Health web site.

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## Original article

# INFECTIOUS DISEASES NOTIFICATION PRACTICES, VICTORIA 2013

Katherine B Gibney, Lucinda J Franklin, Nicola Stephens

## Abstract

**Introduction:** Infectious disease notification practices in Victoria were reviewed to identify areas for potential improvement.

**Methods:** Confirmed or probable cases of certain infectious diseases required to be notified to the Department of Health and Human Services (DHHS) Victoria in 2013, excluding elevated blood lead, foodborne or water-borne illness with 2 or more related cases and chlamydial infection, were analysed according to: notification source of doctor ± laboratory vs. laboratory-only; routine follow-up by public health staff for selected conditions vs. not routine; priority for Indigenous status reporting for 18 priority conditions with a target of ≥95% completeness vs. other conditions with a target of ≥80% completeness; and urgency of notification (conditions requiring immediate [same day] notification vs. conditions requiring notification within 5 days).

**Results:** Almost half (49%) the 34,893 confirmed and probable cases were notified by laboratory report alone. Indigenous status was complete for 48% of cases. Indigenous status was more likely to be completed for conditions with active vs. no active follow-up (RR 1.88 (95% CI 1.84–1.92)) and priority conditions for Indigenous status reporting vs. other conditions (RR 1.62 (95% CI 1.59–1.66)). Among conditions without active follow-up, doctor-notified cases had more complete Indigenous status reporting than laboratory-only notified cases (86% vs. 6%, RR 15.06 (95% CI 14.15–16.03)). Fewer notifications requiring same day notification were received within the legislated time frame (59%) than notifications required to be notified within 5 days (90%).

**Discussion:** DHHS Victoria handles a large volume of infectious disease notifications. Incomplete Indigenous status reporting, particularly for conditions without active follow-up, and delayed notification of conditions requiring immediate attention warrant attention. These findings will be used to improve notification practices in Victoria. *Commun Dis Intell* 2016;40(3):E317–E325.

**Keywords:** public health surveillance; public health practice; disease notification; communicable disease control; Indigenous population; Victoria

## Introduction

Infectious disease surveillance data are used to monitor disease epidemiology, detect and manage disease outbreaks, inform the need for public health interventions and monitor the impact of these interventions. In Victoria, the *Public Health and Wellbeing Act 2008* requires doctors and laboratories to notify the Department of Health and Human Services (DHHS) when certain infectious diseases are diagnosed or suspected. Seventy-two conditions are specified in the Public Health and Wellbeing Regulations 2009 as requiring notification; all except elevated blood lead levels are infectious diseases or complications of infectious diseases. Twenty-four notifiable conditions are classified as ‘Group A’ conditions\* and require immediate (same day) notification by telephone on initial diagnosis, whether presumptive or confirmed, followed by written notification within 5 days. This allows immediate public health action, for example providing prophylactic antibiotics to people who have had contact with a case with invasive meningococcal disease. The remaining 48 conditions require notification within 5 days of initial diagnosis. In Victoria, notifications are received centrally and entered into the State’s notifiable diseases database, the Public Health Event Surveillance System (PHESS), an electronic platform introduced in 2012, with 2013 being the first full year of use. Although PHESS has capacity to receive electronic notifications directly,<sup>1</sup> in 2013 all clinical and laboratory notifications were entered manually. Active case follow-up by DHHS staff is undertaken for all Group A conditions and selected other conditions based on the need for additional (enhanced) data, to inform public health action. There is no active follow-up for the remaining conditions. Responsibility for public health response to these notifications lies with the DHHS. Additionally, for the purposes of national

\* Group A conditions: Anthrax, botulism, chikungunya, cholera, diphtheria, food or water borne illness (2 or more related cases), haemolytic uraemic syndrome, *Haemophilus influenzae* type b, hepatitis A, Japanese encephalitis, legionellosis, measles, meningococcal disease (invasive), Middle East respiratory syndrome coronavirus, Murray Valley encephalitis, paratyphoid, plague, poliomyelitis, rabies, severe acute respiratory syndrome, smallpox, tularaemia, typhoid, viral haemorrhagic fevers, yellow fever.

surveillance of infectious diseases, de-identified data regarding confirmed and probable cases are forwarded daily to the National Notifiable Diseases Surveillance System (NNDSS) for a nationally agreed set of 65 communicable diseases.<sup>2</sup>

This paper represents an audit of notifications received in 2013 by DHHS Victoria into PHESS. Such audits have been performed every 1–3 years since 2004<sup>3–7</sup> to inform Victorian public health staff and notifiers of notification practices in Victoria and identify notifier and system factors that need improvement. Findings of this audit will be used to optimise the utility and efficiency of disease notification in Victoria.

## Methods

All notifications received by DHHS in 2013 were entered into PHESS and all notifications were included in this analysis, excluding the conditions of elevated blood lead, foodborne and water-borne illness with 2 or more related cases as these are not a single pathogen and are notified by certain institutions only, and chlamydial infection for which the notification process was under review during 2013. De-identified case notification data were extracted from PHESS in April 2014. Cases were reported and analysed according to the following classifications: ‘confirmed’ and ‘probable’ cases met nationally agreed case definitions;<sup>8</sup> ‘rejected’ cases did not meet the national case definition; ‘suspected’ cases had not been assessed against the national case definition; ‘at-risk’ cases included contacts of known cases; and ‘not notifiable’ cases were residents of another Australian jurisdiction and were therefore counted in that jurisdiction. Fields relating to the notified case included event identification, disease-group, condition, onset date, sex, age, Aboriginal and Torres Strait Islander (Indigenous) status and postcode of residence. Notification details included the notifier, date of specimen collection (for laboratory notifications), date the notification was authorised by the notifying doctor or positive result was authorised by the notifying laboratory (signature date), and date the notification was received by DHHS (notification received date).

Case classification, number of notifications per case, and notification source (doctor, laboratory, or both) was described for all notifications. All other analyses, including data completeness and time to notification, were restricted to confirmed and probable cases. The Communicable Diseases Network Australia (CDNA) has set a target for Indigenous status reporting of  $\geq 95\%$  for 18 priority conditions and  $\geq 80\%$  for all other conditions.<sup>2</sup> Confirmed and probable notifications were benchmarked against these targets.

Notification outcomes for different groups, including cases notified by a laboratory but not a medical practitioner (laboratory-only notified cases) with cases notified by a medical practitioner  $\pm$  laboratory (doctor-notified cases); follow-up by public health staff, which is routine for all notified cases of Group A conditions and selected Group B, C and D conditions, vs. not routine; and priority for Indigenous status reporting for 18 priority conditions vs. all other conditions, were compared using chi-square tests and relative risks (RR) and 95% confidence intervals (95%CI) were generated. A *P*-value  $< 0.05$  was considered statistically significant.

Time to notification was calculated as the number of days between the earliest signature date and the earliest notification received date for each notified case. Cases with missing signature date or a delay of more than 365 days were excluded from the time to notification analysis. Median delay to notification and proportion of cases notified within the legislated time frame of 0 days for Group A conditions or within 5 days for Group B, C and D conditions were reported.

Data were analysed using Stata version 13.1. This project was an audit of disease notifications made under state legislation and was not subject to human research ethics committee review.

## Results

A total of 94,592 notifications were received by the department relating to 39,389 cases of notifiable infectious diseases that met the inclusion criteria. Of these, 33,436/39,389 (85%) cases were classified as confirmed and 1,457 (4%) probable. The remaining cases were classified as rejected (1,885 cases, 5%), at-risk (1,477 cases, 4%), not notifiable (1,103 cases, 3%), and suspected (31 cases, 0.08%). Varicella zoster infection, pertussis and dengue made up 98% of the 1,457 probable cases, with psittacosis, legionellosis, HIV (newly acquired), meningococcal infection and rubella also having cases classified as probable. The majority of the 1,477 cases classified as at-risk were tuberculosis (1,327 cases, 90%), followed by typhoid (86 cases, 6%), and paratyphoid (56 cases, 4%).

Of the total 94,592 notifications, 48,913 (52%) were from primary laboratories, 21,417 (23%) from reference laboratories, and 22,681 (24%) from medical practitioners. Seventy-eight notifications were laboratory results where the testing laboratory was not identified, and 1,503 notifications were generated by public health staff at DHHS or other public health units. Of the included 39,389 cases, 40% were notified on a single occasion, with a median of 2 and a maximum of 64 notifications per case (interquartile range 1–3 notifications per

case). Multiple notifications for a single case could result from notification by both clinician and laboratory (according to the legislative requirement); notification by more than 1 clinician; and/or multiple laboratory tests, which sometimes resulted in a high number of notifications for a single case.

Almost half the 34,893 cases classified as confirmed or probable were attributable to 3 diseases: *Campylobacter* infection (5,898 cases, 17%), influenza (5,833 cases, 17%), and varicella zoster infection (5,084 cases, 15%). More confirmed or probable cases were notified in 2013 than during the preceding decade (2003–2012) for cryptosporidiosis, dengue, gonococcal infection, hepatitis D, HIV – unspecified duration, Q fever, salmonellosis, syphilis – infectious (primary, secondary and early latent less than 2 years duration), syphilis – late (more than 2 years or unknown duration), and typhoid (Table 1). More confirmed and probable cases of chikungunya (notifiable from 2005) and varicella zoster infection (notifiable from 2008) were notified in 2013 than in previous years.

**Table 1: Conditions for which more confirmed and probable notifications were received in 2013 than for any single year in the preceding decade, 2003 to 2012**

Condition	Notified cases	
	2013	Range 2003–2012
Chikungunya virus infection*	30	0–17
Cryptosporidiosis	1,261	215–1,142
Dengue virus infection	407	6–326
Gonococcal infection	2,992	922–2,438
Hepatitis D	23	4–16
HIV – unspecified duration	208	112–183
Q fever	50	16–35
Salmonellosis	2,944	1,160–2,743
Syphilis – infectious	655	55–467
Syphilis – late	572	293–537
Typhoid	46	12–41
<b>Varicella zoster infection†</b>		
Chickenpox	871	222–738
Shingles	1,209	168–1,111
Unspecified	3,004	146–2,626

\* Notifiable from 2005 – comparative period 2005–2012.

† Notifiable from 2008 – comparative period 2008–2012.

Among the 34,893 confirmed and probable cases, 49% were notified by the laboratory alone, 45% by both medical practitioner and laboratory, and

6% by medical practitioner alone. The remaining 97 (0.3%) cases were identified through other means including active surveillance by DHHS staff and public health units. Four conditions were notified by both laboratory and doctor in all confirmed and probable cases—newly acquired HIV infection (110 cases), leprosy (3 cases), cholera (1 case) and congenital rubella (1 case) (Figure). More than 80% of confirmed and probable cases were notified by both laboratory and doctor for chikungunya, HIV – unspecified duration, listeriosis, meningococcal disease, paratyphoid, rubella, shigellosis, syphilis – infectious, tuberculosis and typhoid.

Medical practitioners made 22,681 separate notifications relating to 19,047 cases. Of these, 17,594/19,047 (92%) doctor-notified cases were confirmed or probable. The most common methods of initial notification for medical practitioners were facsimile (50%), web and e-notification (23%), and post (19%) (Table 2). Medical practitioners were more likely to first notify Group A conditions by telephone than Group B, C or D conditions (51% vs. 5%, RR 10.5 (95% CI 9.1–12.2)).

**Table 2: Method of first notification of doctor-notified cases, Victoria, 2013, by disease group\***

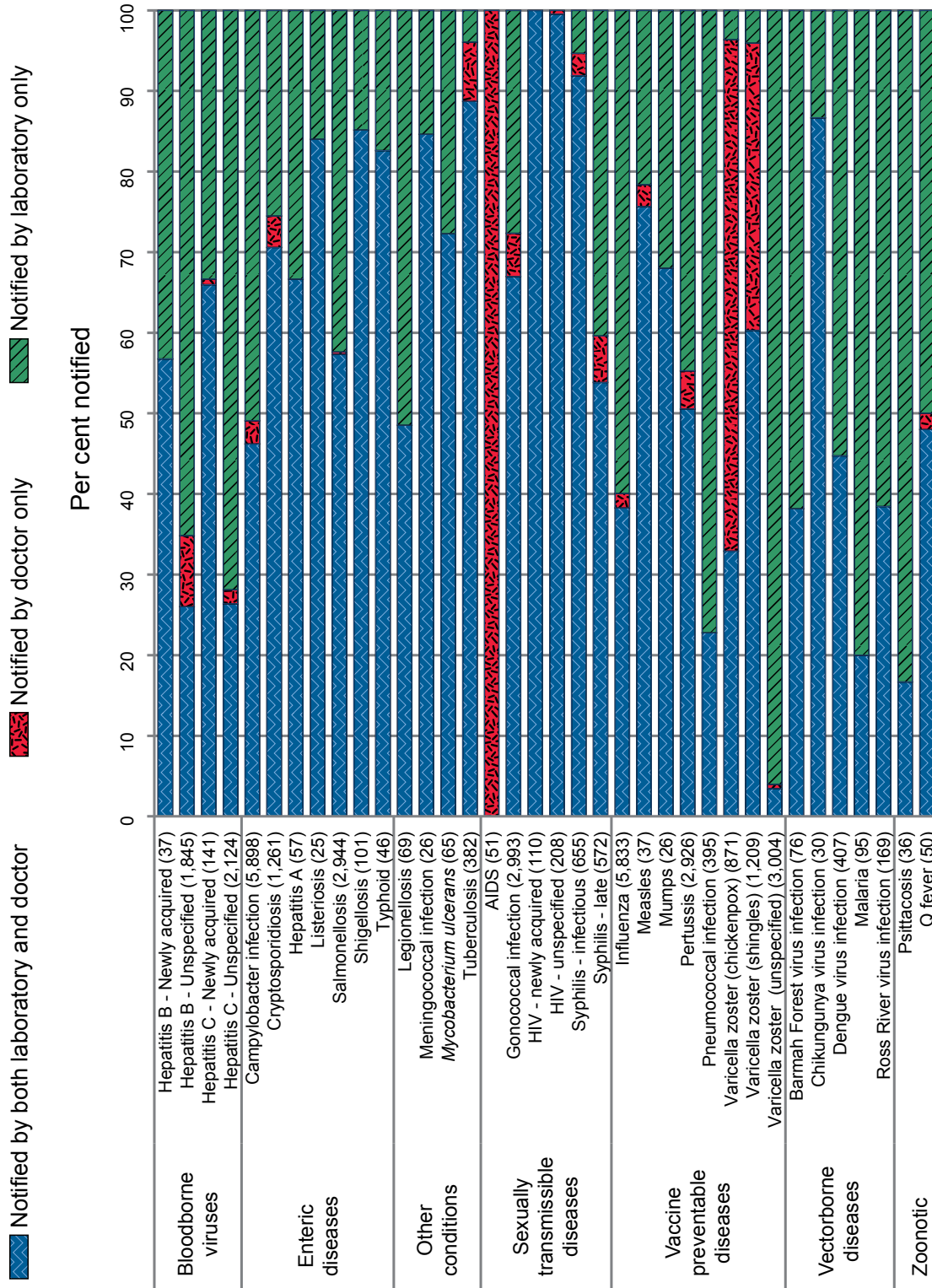
Method of notification	Group A		Groups B, C, D		All	
	n	%	n	%	n	%
Facsimile	49	23	8,704	50	8,753	50
Web / e-notification	34	16	3,933	23	3,967	23
Post	12	6	3,349	19	3,361	19
Telephone	107	51	847	5	954	5
Other	3	1	364	2	367	2
Unknown	4	2	188	1	192	1
Total	209		17,385		17,594	

\* Confirmed and probable cases only, excludes chlamydial infection and foodborne or water-borne illness.

Group A conditions require immediate notification by telephone followed by written notification; groups B, C, and D conditions require written notification within 5 days of initial diagnosis

Of the 70,408 separate notifications received from laboratories, 63,711 (90%) related to confirmed or probable cases. Sixty per cent of the 32,850 confirmed or probable cases notified by laboratories were notified using a single laboratory notification, 19% had two, 8% had three and 12% had 4 or more separate laboratory notifications per laboratory-notified case.

Figure: Method of notification for confirmed and probable cases notified to the Victorian Department of Health and Human Services, 2013\*



\* Number of confirmed and probable cases notified in 2013 in parentheses. The following conditions had fewer than 25 confirmed and probable cases notified: hepatitis D (23 cases), *Mycobacterium* infection (non-tuberculosis) (22), paratyphoid (19), Shiga toxin and vero toxin-producing *Escherichia coli* (12), hepatitis E (9), leptospirosis (9), Creutzfeldt-Jakob disease (7), rubella (5), *Haemophilus influenzae* type B (4), leprosy (3), botulism (2), haemolytic uraemic syndrome (2), cholera (1), rubella – congenital (1), tetanus (1).



Age, sex and postcode were complete in  $\geq 99.5\%$  of confirmed and probable cases notified. Country of birth was reported in 41% of cases; more often among cases notified by a doctor than by laboratory-report alone (75% vs. 6%, RR 11.7 (95%CI 11.0–12.4),  $P < 0.001$ ).

Indigenous status was complete in only 48% of confirmed and probable cases. Conditions with routine active follow-up by DHHS public health staff were more likely to have Indigenous status reported than those without active follow-up (83% vs. 44%, RR 1.88 (95% CI 1.84–1.92),  $P < 0.001$ ). This difference in Indigenous status completeness was less marked among conditions notified by a doctor (92% with active follow-up vs. 86% with no active follow-up, RR 1.07 (95% CI 1.05–1.08),  $P < 0.001$ ) than among laboratory-only notifications (63% vs. 6%, RR 10.97 (95% CI 10.13–11.89),  $P < 0.001$ ). Among conditions without routine active follow-up, doctor-notified cases were more likely to have Indigenous status reported than laboratory-only notified cases (86% vs. 6%, RR 15.06 (95%CI 14.15–16.03),  $P < 0.001$ ) (Table 3).

Notifications were received for 15 of the 18 priority conditions for Indigenous status data completeness identified by CDNA.<sup>2</sup> Among these, Indigenous status completeness ranged from 58% for gonococcal infection to  $\geq 95\%$  for hepatitis A, hepatitis B (newly acquired), HIV, leprosy and tuberculosis (Table 4). These priority conditions for Indigenous status reporting were more likely to have Indigenous status completed than other conditions (71% vs. 44%, RR 1.62 (95%CI 1.59–1.66),  $P < 0.001$ ). Indigenous status was complete for 89% of notified priority condition cases for which active follow-up by DHHS public health staff is routine compared with 58% for gonococcal infection, which is the only priority condition without routine active follow-up.

**Table 4: Completeness of Indigenous status reporting for priority diseases,\* Victoria, 2013**

Priority condition	Cases notified	Indigenous status complete %
Dengue virus (locally acquired)	0	–
Donovanosis	0	–
Gonococcal infection <sup>†</sup>	2,992	58
<i>Haemophilus influenzae</i> type b	4	75
Hepatitis A	57	96
Hepatitis B (newly acquired)	37	95
Hepatitis C (newly acquired)	141	64
HIV	369	95
Leprosy	3	100
Measles	37	92
Meningococcal disease (invasive)	26	81
Pertussis <5 years	227	79
Pneumococcal disease <5 years	38	89
Pneumococcal disease $\geq 50$ years	235	89
Shigellosis	101	89
Syphilis – congenital	0	–
Syphilis - infectious	655	86
Tuberculosis	382	100
All priority conditions	5,304	71
Other (non-priority) conditions <sup>‡</sup>	29,589	44

\* Target for priority diseases is  $\geq 95\%$  Indigenous status complete and  $\geq 80\%$  for all other diseases.

† Gonococcal infection is the only priority condition for Indigenous reporting that is not routinely followed up by the Department of Health and Human Services staff.

‡ All other notifiable conditions not listed above as priority conditions.

**Table 3: Completeness of Indigenous status reporting for conditions with and without active follow-up, by notifier**

	All notifications			Doctor notified			Lab-only notified			RR*	(95% CI)	P-value
	n	N	%	n	N	%	n	N	%			
<b>Conditions with active follow-up of all notified cases</b>												
Group A	232	293	79	172	209	82	60	84	71	1.15	0.99–1.34	0.038
Group B, C, D	2,464	2,936	84	1,961	2,121	92	503	815	62	1.50	1.42–1.58	<0.001
<b>Conditions without active follow-up of all notified cases<sup>†</sup></b>												
Group B, C, D	14,054	31,664	44	13,118	15,264	86	936	16,400	6	15.06	14.15–16.03	<0.001
All conditions	16,750	34,893	48	15,251	17,594	87	1,499	17,299	9	10.00	9.53–10.50	<0.001

\* Relative risk for having Indigenous status complete if notified by a doctor vs. laboratory only.

† Barmah Forest virus infection, campylobacteriosis, cryptosporidiosis  $\geq 6$  months of age, gonococcal infection (laboratory notified), hepatitis B (unspecified duration), hepatitis C (unspecified duration), influenza, non-tuberculosis mycobacterium infection (excluding *Mycobacterium ulcerans*), pertussis (aged  $\geq 5$  years), invasive pneumococcal infection (aged 5–49 years), Ross River virus infection, salmonellosis, syphilis – late (laboratory notified), and varicella zoster infection

Group A conditions require immediate notification by telephone followed by written notification; groups B, C, and D require written notification within 5 days of initial diagnosis.

The median time to notification for confirmed and probable Group A conditions was zero days (range 0 to 52 days), with 59% of notifications received on the same day as the signature date and within the legislated time frame for notification (Table 5). For Group B, C and D conditions the median delay from signature date to notification was 1 day, with 90% of cases notified within the legislated time frame of 5 days from the signature date. Among medical practitioners, 100% of Group A conditions were notified within the legislated time frame (same day as diagnosis) when notified by web or e-notification; 79% when notified by telephone and 50% when notified by fax (Table 5). For Group B, C and D conditions notified by medical practitioners,  $\geq 97\%$  were notified within the legislated time frame (within 5 days of diagnosis) when notified by web or e-notification, telephone or facsimile, and 77% when notified by post.

## Discussion

A major finding of this audit was the low proportion of notified cases with completed Indigenous status. Reporting Indigenous status in health data is essential in order to quantify health disparities between Indigenous and non-Indigenous Australians, inform policy development and service delivery planning, and measure the effectiveness of interventions against targets of improved Indigenous health.<sup>9</sup> In 2011, CDNA set national targets for data completeness of Indigenous status at  $\geq 95\%$  for 18 priority conditions and  $\geq 80\%$  for all other notifiable conditions.<sup>2</sup> In Victoria in 2013, Indigenous status was complete for 71% of the priority diseases and 42% of other diseases. The proportion of all confirmed and probable cases with complete Indigenous status was 48% in 2013, similar to previous Victorian reports of 45% to 51% from 2004 to 2011.<sup>3-5,7</sup> Overall, 48% of cases in the NNDSS in 2013 had Indigenous status reported, ranging from 18% in New South Wales to  $>90\%$  in the Northern Territory, South Australia, and Western Australia.<sup>2</sup> Similarly, ethnicity was reported for 49% of cases notified to the US National Notifiable Diseases Surveillance System from 2006 to 2010.<sup>10</sup> When restricted to doctor-notified confirmed and probable cases in Victoria, the proportion with complete Indigenous status was 87% in 2013, a slight improvement compared with 80% to 84% from 2006 to 2011.<sup>3,5-7</sup> Despite awareness of the issue, there has not been substantial progress in improving completeness of Indigenous status reporting in Victoria. In this study we have provided more detailed analysis of Indigenous status reporting, highlighting higher completion rates among doctor notified cases, conditions with active follow-up, and priority diseases, in order to highlight areas that require attention and potential strategies for improvement.

In particular, more needs to be done to meet the CDNA targets for Indigenous status reporting for gonococcal infection, which is the only priority condition for which active case follow-up of laboratory notifications is not routine in Victoria. Indigenous status was complete for 83% of cases with active follow-up; therefore re-instituting routine active case follow-up for laboratory-notified

**Table 5: Proportion of cases notified within 0 days, 1–5 days, and >5 days of signature date, by condition group, Victoria, 2013**

	Number of cases*	Days to notification %		
		0	1–5	>5
<b>Group A</b>				
All cases	285	59	30	11
<b>Notifier</b>				
Both doctor and laboratory	201	62	29	9
Laboratory only	82	50	35	15
Doctor only	2	100	0	0
<b>Method of doctor notifications (if known)</b>				
Facsimile	18	50	44	6
Web / e-notification	10	100	0	0
Post	2	0	0	100
Telephone	77	79	16	5
Other†	3	67	33	0
<b>Groups B, C and D</b>				
All cases	31,779	33	56	10
<b>Notifier</b>				
Both doctor and laboratory	14,659	38	53	9
Laboratory only	15,250	27	61	12
Doctor only	1,870	51	40	9
<b>Method of doctor notifications (if known)</b>				
Facsimile	6,702	64	32	4
Post	1,090	8	69	23
Web / e-notification	1,610	79	18	3
Telephone	326	86	14	0.6
Other‡	80	59	34	7
Total	30,325	47	48	5

\* Confirmed and probable cases only; elevated blood lead, chlamydial infection and food-borne or water-borne illness excluded. Excludes notified cases where signature date was missing, or the date difference between 'signature date' and 'date notified' was greater than 365 days or less than 0 days (assuming transcription errors by notifier or data entry errors).

† Number of days between earliest signature date and the earliest notification received date.

‡ All other methods of notification.

Group A conditions require immediate notification by telephone followed by written notification; groups B, C, and D require written notification within 5 days of initial diagnosis.

cases of gonococcal infection is likely to improve completeness of Indigenous status reporting for gonococcal infection to >80%.

Ideally, Indigenous status would be ascertained at the time of notification. This requires educating clinician-notifiers of the importance of completing the Indigenous status field on the notification form. As Indigenous status was complete for 87% of doctor-notified cases in 2013, there is some scope for improvement as a result of clinician education. A DHHS communication strategy in 2009 aimed to increase the proportion of notified cases for which a notification was received from a doctor. This contributed to a temporary increase in this proportion to 58% in 2009,<sup>11</sup> but by 2013 this had fallen back to the baseline of 50%, indicating only modest gains in Indigenous status ascertainment are likely to be achieved through clinician education and that such education needs to be ongoing to maintain these gains. Inclusion of an Indigenous status identifier on laboratory request forms has potential to do more to improve ascertainment,<sup>12</sup> particularly for laboratory-only notified cases without routine follow-up such as gonococcal infection. Although this can be encouraged through clinician-education, changes to legislation and regulations requiring inclusion of Indigenous status on pathology request slips could prove more effective.<sup>12</sup> This requirement would also improve Indigenous status ascertainment in other datasets such as cancer registries. Regardless of the method used to improve completeness of Indigenous status, individuals should retain the right to withhold their Indigenous status through use of the 'declined to answer' response.

Another potential approach is to undertake record linkage with other data sets to improve Indigenous status reporting completeness. In response to poor completeness of Indigenous status identified in previous audits of Victorian notification practices, a data-linkage pilot study was performed that aimed to improve Indigenous status reporting for 3 of the nominated priority conditions for Indigenous reporting completeness.<sup>13</sup> Data from newly acquired hepatitis B and C and gonococcal infection cases notified in Victoria in 2009–10 were linked with Victorian hospitalisation data (1997–2011). Among the 82% of cases able to be linked, the proportion with missing Indigenous status decreased from 62% for hepatitis B, 68% for hepatitis C, and 33% for gonococcal infection to less than 0.2% for all conditions. Importantly, this resulted in a 2–4 fold increase in notification incidence among Indigenous Victorians for each of these conditions.<sup>13</sup> Although the pilot data-linkage study illustrated potential use of other Victorian Government datasets to improve completeness of Indigenous status for data analysis and reporting,

it was a retrospective study that did not update or correct the Indigenous status field in PHESS. The use of record linkage to update the Indigenous status field in PHESS raises ethical and privacy issues as people have the right to withhold their Indigenous status for some or all health service interactions. At present, these ethical and privacy issues prevent updating the Indigenous status field in PHESS using information already contained in PHESS, related to an individual's previous disease notification(s), or other health-related data sources. However, such record linkage is routine in certain countries, indicating these issues may not be insurmountable. For example, a National Health Index (NHI) number is assigned to individuals accessing health and disability support services in New Zealand. The NHI holds various demographic and health data, including self-reported ethnicity. The NHI is included in the national notifiable communicable diseases database (EpiSurv), which facilitates record linkage with the New Zealand Health Information Service.<sup>14</sup>

In Victoria, the *Public Health and Wellbeing Act 2008* requires both doctors and laboratories to notify all infectious diseases scheduled in the Public Health and Wellbeing Regulations 2009. In 2013, only 45% of confirmed and probable cases had both medical practitioner and laboratory notifications, similar to our findings for 2004 to 2011 (43% to 52%).<sup>4–7</sup> A 2008 survey of 152 Victorian medical practitioners identified the most common reasons for not notifying as: 1) assumption that the laboratory would notify; 2) belief that doctors notify confirmed, not suspected cases; and 3) notification was time consuming process.<sup>11,15</sup> The proportion of notifications received by laboratory alone increased from 38% in 2011 to 49% in 2013.<sup>7</sup> In comparison, in the proportion of notifications made by laboratory alone was estimated to be 4% in South Australia, 33% in Western Australia, and ≥95% in all other Australian jurisdictions in 2013.<sup>16</sup> This highlights the variability of surveillance practices in different Australian jurisdictions and potential issues with comparing notification data between jurisdictions. Unlike Victoria, in New South Wales, the Northern Territory, Queensland and Tasmania certain high-incidence conditions (e.g. chlamydial genital infection) require notification from the laboratory but not the doctor and in each of these jurisdictions laboratory only notifications account for ≥98% of all notified cases. The value of requiring dual notification by laboratories and clinicians for all notifiable conditions is currently under review in Victoria. If doctor notifications were not required for all conditions, the notification burden on clinicians and workload of DHHS surveillance staff would be reduced without impacting case ascertainment or timeliness of notification for high incidence diseases which require laboratory

confirmation. However, the trade-off associated with reliance on laboratory only notifications is the potential loss of certain clinical, demographic and epidemiological information which can enable DHHS to identify sources of exposure and implement strategies to prevent further cases. For example, cases notified by a doctor were 12 times more likely to have completed country of birth compared to laboratory only notifications. For several conditions, additional data are collected by public health officers during routine case follow-up with the treating doctor and/or case through telephone contact or a request to complete an enhanced surveillance form (ESF). To expedite this, DHHS are trialling a system for selected conditions whereby doctors making web notifications are immediately directed to the appropriate ESF so that enhanced data are collected at the time of notification. Active case follow-up also provides an opportunity to collect missing notification data. Among conditions with routine active case follow-up, the difference in completeness of reporting of Indigenous status between doctor notified and laboratory-only notified cases (RR 1.88) were considerably less marked than among conditions with no routine active follow-up (RR 15.06). This suggests that for conditions with routine case follow-up, Indigenous status and other missing demographic information can be collected during case follow-up for laboratory-only notified cases.

As several high-incidence conditions are currently not routinely followed up, alternate ways to obtain data relevant to notified cases need to be considered. The modernisation of surveillance in Australia through formalised data linkages with existing datasets has been identified as a national surveillance strategic priority,<sup>17</sup> while development of secure and reliable record linkage has been identified in surveillance strategies in Australian and international jurisdictions.<sup>18,19</sup> It might be possible to obtain demographic data, including Indigenous status, postcode of residence and country of birth from electronic medical records if this information was automatically included on electronically generated pathology request slips and notification forms.<sup>12</sup> This would result in more complete data without the need for medical practitioners to separately notify each diagnosed case. Linkage of case notification data with extracts from other government databases has potential to be more easily achieved. In New South Wales and Western Australia, linkage of the Australia Childhood Immunisation Register data with state-based disease notification data has been successfully piloted for a 17-year birth cohort (more than 2 million children) to improve vaccination status reporting.<sup>20</sup> This allows identification of vaccine failures and population-based assessment of vaccine effectiveness and can be used to evaluate and inform the

Australian Immunisation Program. Updating PHESS records regarding vaccination status using data obtained via record linkage is unlikely to raise the same ethical and privacy concerns as Indigenous status fields.

Electronic laboratory reporting (ELR), the automated transmission of laboratory results from laboratories to public health units, is recognised to improve notification timeliness and accuracy and therefore public health response capacity.<sup>21–23</sup> PHESS is a customised version of a commercial product known as Maven Enhanced Disease Surveillance System (Maven EDSS), developed by Consilience Software, Austin Texas USA. In 2014, Maven EDSS was used in 7 US states, 5 US cities (including New York City) and New South Wales – the most populous Australian state with 32% of the national population.<sup>1</sup> The use of ELR is expanding in New South Wales, with 4 laboratories commencing ELR in 2013 and additional laboratories added subsequently.<sup>24</sup> Electronic laboratory notifications from some laboratories are received directly into the New South Wales surveillance system, the Notifiable Conditions Information Management System (NCIMS). As yet, the Victorian PHESS database does not receive laboratory reports electronically. However, a pilot is underway for ELR from a Victorian public health laboratory with plans to expand this to other Victorian laboratories. As more than 90% of notified cases include a laboratory notification, this has the potential to reduce notification delay as well as reducing data entry workload and errors within DHHS.

DHHS Victoria continues to receive and respond to a high number of notifications of communicable diseases. In 2013, fewer than half the notified cases had Indigenous status completed, although higher ascertainment was achieved for doctor-notified cases, priority conditions for Indigenous reporting, and conditions with active follow-up. An increasing proportion of cases were notified by laboratory alone in Victoria. This is in keeping with national trends, with the potential consequence of incomplete demographic and risk factor data for notified cases. Possible actions to ensure adequate data quality and completeness in this context include prioritisation of data fields and diseases for which data completeness is necessary; education and support of doctors to ensure appropriate and timely notification; automation of systems to pre-populate laboratory request slips and notification forms with relevant demographic data; and development of ELR and data linkage capacity. Notifying doctors should be reminded of the requirement for immediate notification by telephone for Group A conditions to facilitate rapid public health response and prevention of further cases. DHHS Victoria will continue to work with notifiers and data cus-

todians on these issues to ensure timely, complete and efficient notification to inform and monitor public health actions.

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# THE AUSTRALIAN MASTER OF APPLIED EPIDEMIOLOGY PROGRAM: LOOKING BACK, MOVING FORWARD

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## Abstract

The Master of Applied Epidemiology Program is Australia's Field Epidemiology Training Program. It was established in 1991 and is run out of the National Centre for Population Health (NCEPH) at the Australian National University. The Program has a strong track record in using field-based training to produce competent applied epidemiologists who have contributed to public health in Australia and globally. A new funding model for the program was implemented in 2012, backed by funds from field placement partners and NCEPH. In this paper we review the program's origins and achievements, discuss the ongoing needs of the program and outline a vision for the future. *Commun Dis Intell* 2016;40(3):E326–E333.

Keywords: epidemiology; university; program; funding

## Introduction

Recent infectious disease emergencies, including the outbreaks of Ebola virus disease (EVD) in West Africa, Middle East respiratory syndrome coronavirus (MERS-CoV) in Saudi Arabia and later South Korea, and hepatitis A virus infections linked to imported berries in Australia, are reminders of the importance of competent field epidemiologists for emergency response, both in Australia and globally. We therefore thought it timely to review Australia's Field Epidemiology Training Program (FETP), the Master of Applied Epidemiology (MAE) Program, its origins, the changes initiated in response to recent funding cuts and where it is headed in the future.

## The history of the Master of Applied Epidemiology Program

Field or applied epidemiology is the use of epidemiological methods and principles to study and understand real-world public health problems and produce evidence-based and actionable recommendations, frequently within a limited timeframe.<sup>1,2</sup> It is sometimes referred to as 'shoe leather epidemiology' in recognition that much of the investigation and response involves getting out of the office or laboratory and into the field where the problem is occurring and evolving.<sup>3</sup>

The MAE Program was established in 1991 in response to a recognised gap in field epidemiology training in Australia. The driving force behind its establishment was Professor Bob Douglas, the then director of the National Centre for Epidemiology and Population Health (NCEPH) at the Australian National University. At the time, the program was funded by the Commonwealth Department of Health and was supported by the Communicable Diseases Network Australia (CDNA).<sup>3</sup> The United States Centers for Disease Control and Prevention (CDC) provided assistance to develop the curriculum, based on the highly successful American Epidemic Intelligence Service program.

Under the original model, MAE scholars received a generous stipend while completing 3 months of coursework at NCEPH (spread across the 2 year timeframe), and 21 months spent in a field placement, typically a state, territory or Commonwealth health department. The curriculum included training on how to: 1) establish and evaluate public health surveillance systems, 2) analyse surveillance and other data, 3) investigate outbreaks, and 4) conduct epidemiological studies to inform the development and implementation of policies and programs. Scholars applied this knowledge and skills in their field placements to real-world public health problems. At the conclusion of the course, scholars submitted a bound volume summarising their 2 years of fieldwork, which was examined by 2 experienced field epidemiologists during an oral examination. Scholars recruited into the MAE Program came from medical, nursing, veterinary and science backgrounds. Further description of the MAE training model and its relevance for strengthening capacity in public health has been described elsewhere.<sup>4</sup>

The first cohort of 8 MAE scholars established notifiable disease surveillance systems in several jurisdictions and investigated various outbreaks of disease. The value of the national collaboration by MAE scholars was quickly demonstrated when they worked with CDNA partners to investigate a large multi-state outbreak of norovirus gastroenteritis associated with the consumption of orange juice served on domestic airlines.<sup>5</sup> An estimated 25,000 people were ill as a result of this outbreak. Subsequent years saw MAE scholars investigate outbreaks of emerging infectious diseases, such as melioidosis in Darwin,<sup>6</sup> respiratory illness in horses

and 2 horse handlers in Queensland, subsequently identified as Hendra virus;<sup>7</sup> haemolytic uraemic syndrome due to consumption of mettwurst salami in South Australia;<sup>8</sup> and the largest outbreak of Legionnaires' disease in Australia at the Melbourne Aquarium;<sup>9</sup> along with many others. Other notable projects included both the establishment and later, the evaluation of the National Notifiable Diseases Surveillance System (NNDSS),<sup>10</sup> as well as projects in non-communicable diseases such as perinatal outcomes in Indigenous infants,<sup>11</sup> investigating the link between maternal trauma and cerebral palsy<sup>12</sup> and a mortality survey in the Democratic Republic of Congo.<sup>13</sup>

Training Indigenous scholars was a priority for the program when the inaugural Indigenous MAE cohort commenced in 1998, to address the notable disparity of Indigenous public health workers who did not have a professional qualification in public health.<sup>14</sup> (In this paper 'Indigenous' is used to refer to anyone who identifies as Aboriginal and/or Torres Strait Islander.) The Indigenous stream of the MAE Program continued until 2002 with cohorts of between 4 and 8 scholars each year, to be replaced by 2 positions for Indigenous scholars within each cohort from 2003 onwards.

Given the nature of communicable diseases, it was important for the MAE Program to be engaged globally. In 1997, the MAE Program was a founding member and provided the inaugural Chair of the Training Programs in Epidemiology and Public Health Interventions Network (TEPHINET)—a network of 55 field-based epidemiology training programs from around the world that aims to strengthen public health capacity in applied epidemiology and public health practice.<sup>15</sup> Program staff also designed, planned and helped implement FETPs in India (1999), China (2001), Malaysia (2003) and Vietnam (2009), and contributed to the development of the monitoring framework of the revised International Health Regulations (2005).<sup>16</sup> In addition, MAE scholars have responded to international public health emergencies, starting with the severe acute respiratory syndrome outbreak in 2003 when 9 students, graduates and staff were deployed across South-East Asia and to Geneva. Scholars and staff have also supported responses to H5N1 avian influenza in 2004 to 2005, and the H1N1 influenza pandemic in 2009. For the latter, scholars and staff contributed over 1,100 person days in investigation and control efforts at the local, national and international levels.<sup>17</sup>

### Funding challenges, bold initiatives

The MAE Program was originally funded by the Australian Government Department of Health, and subsequently through the Public Health

Education and Research Program (PHERP).<sup>18</sup> The PHERP program ceased in 2009 and consequently funding through this source ceased<sup>19</sup> with the last group of scholars graduating in 2011.

However, 2 initiatives proposed in 2011 ensured that the MAE Program could continue to operate as a key source of training competent field epidemiologists in Australia. One was commitments from field placement partners around Australia to fund and host MAE scholars, and the other was the willingness of NCEPH to implement a novel funding model and underwrite initial staffing and other program costs.

### The current program

The MAE Program remains a 2-year training program that emphasises '*learning at work, from work, for work*' and '*learning-by-collaborative problem-solving*'.<sup>4</sup> A comparison of the previous and current forms of the MAE Program is presented in Table 1. Importantly, the structure and competency areas covered by the program remain almost identical. The major change relates to the funding. Field placements now provide the full cost of hosting a scholar either by paying the scholar's full stipend, with the expectation that the scholar will dedicate all of their time to MAE requirements, or a salary if the scholar is already an employee or recruited to be an employee, e.g. by a jurisdictional health department. In the latter case the employee will be given negotiated time off to complete the MAE requirements while still employed in the salaried position. From the NCEPH side, program costs are sourced through the [Research Training Scheme accessible to Universities](https://www.education.gov.au/research-training-scheme) (<https://www.education.gov.au/research-training-scheme>). To facilitate this process, the University reclassified the degree from a coursework to a research degree. Consequently the MAE is now more in line with a traditional Masters in Philosophy program, where MAE scholars prepare a thesis and are enrolled in 5 coursework subjects each of which includes formal assessments. These coursework subjects are also open to graduate students enrolled in other degrees.

The current model has proven very successful. After a modest start in 2012 with 4 full-time and 4 part-time scholars, subsequent MAE cohorts have increased in size with the 2015 cohort having 13 scholars—one of the largest in the program's history, including 2 Australian scholars based in overseas placements. The Program has a memorandum of understanding with the Australian Government Department of Health, which recognises it as the national field epidemiology training program. In addition, the program remains an active member of TEPHINET.

**Table 1. Comparison of the original Master of Applied Epidemiology degree and the re-invigorated Master of Philosophy in Applied Epidemiology degree at Australian National University before and after 2011**

Element	Master of Applied Epidemiology	Master of Philosophy in Applied Epidemiology
Years of operation	1991–2011	2012–onward
Funding source	Majority of program costs funded by the Australian Government Department of Health, some staff costs funded by NCEPH and partial scholarship cost covered by field placement	Program costs covered by the Research Training Scheme, scholarship (or salary) cost covered by field placement, travel to course block covered by field placement
A scholar's stipend	\$32,000–36,000 tax-free annually	\$50,000 tax-free annually
Course work	Three months of course work in 4 intensive course blocks over the 2 years. Initial course block of 4 weeks duration covered introductory epidemiology concepts.	Five course work modules taught in 3 two-week intensive course blocks and via online teaching sessions: (1) outbreak investigation, (2) public health surveillance, (3) analysis of public health data, (4) applied epidemiology research methods, and (5) issues in applied epidemiology.
Duration	Program was initially for 24 months, but was reduced to 21 months in 2007, consistent with the timing of university semesters	22 months
Assessment	Bound volume with at least 4 field projects, followed by an oral exam.	Assessment for all 5 course work subjects, as well a thesis comprising at least 4 field projects followed by an oral viva.
Additional	–	Program allows for new arrangements, including part-time scholars, and scholars already employed by the field placement.

There are some advantages of the current model. Delivery of the MAE curriculum via courses open to other graduate students means that MAE scholars are more integrated into NCEPH, and more students are able to acquire the skills of applied epidemiology. Some of these non-MAE students have subsequently enrolled in the MAE Program. The greater investment by field placements in MAE scholars has allowed them to have a greater input into the selection of candidates and to strengthen organisational capacity by enrolling their own employees into the program.

All MAE scholars are required to publish at least one of their projects and to present their work at a national or international conference. The Box provides examples of papers published and conference presentations by scholars while Table 2 outlines the variety of placements and the number of scholars in each cohort since 2012. Of note, there continues to be a focus on Indigenous health, with 2 Indigenous scholars enrolled in the program since the new model was implemented. Moreover, several field placements have a dedicated focus on Indigenous health issues including the Indigenous Offender Health Research Capacity Building Group at the Kirby Institute and the Indigenous Health Division of the Australian Government Department of Health.

### The future of the Master of Applied Epidemiology Program

The MAE Program is an example of a successful government research institution partnership. It has contributed to the advancement of public health in Australia in 3 main areas: workforce development, applied research informing evidence-based policy, and surge capacity during public health emergencies. We briefly discuss the program's track record in each of these areas and outline plans and options for the future.

Since its inception, the program has graduated 187 individuals of whom more than 15% are Indigenous. MAE graduates have made significant contributions to public health, both in Australia and internationally, including holding senior roles in state and territory health departments, national and international organisations such as the World Health Organization (in Headquarters and at various regional and country offices), as well as in university schools of public health and non-government organisations. To support workforce needs, we aim to train and graduate a minimum of 10 scholars annually for at least the next 5 years. In the context of contemporary national and global public health threats, communicable disease surveillance and control will remain the core business of the MAE Program in the short-term, with the majority of placements in this area. However, the principles



**Box: List of selected Master of Philosophy in Applied Epidemiology projects completed by scholars since 2012 and published in peer review journals or presented at national or international conferences**

- Human rabies immunoglobulin usage in Australia, 2010 to 2013<sup>20</sup>
- Associations between antimicrobial susceptibility patterns of *Shigella* isolates and suspected country of acquisition – Victoria, Australia, 2008–2012<sup>21</sup>
- Very high incidence of invasive group A streptococcal disease across Northern Territory populations<sup>22</sup>
- High levels of lead solder in drinking water tanks, Tasmania, 2013<sup>23</sup>
- An outbreak of norovirus genotype II associated with New South Wales oysters<sup>24</sup>
- Outbreak of influenza A(H1N1) virus in a remote Aboriginal community post-pandemic: implications for pandemic planning and health service policy<sup>25</sup>
- Exploring a proposed World Health Organization method to determine thresholds for seasonal influenza surveillance<sup>26</sup>
- Estimating the measles effective reproduction number in Australia from routine notification data<sup>27</sup>
- Re-thinking traditional adverse event following immunisation surveillance: lessons from Australia's successful experience with intussusception surveillance following the 2007 introduction of rotavirus vaccines<sup>28</sup>
- Evaluating the effectiveness of the human papillomavirus vaccine among Indigenous women in Australia<sup>29</sup>
- Are we ready for rubella elimination? A review of rubella and congenital rubella syndrome (CRS) notification and hospitalisation data – Australia, 2008–2012<sup>30</sup>
- Trends in testing for chlamydial infection in the ACT, 2003 to 2012<sup>31</sup>
- *Salmonella* Typhimurium phage type 44: A Victorian outbreak and review of MLVA patterns<sup>32</sup>
- Is the National Notifiable Surveillance System an effective surveillance system for flu?<sup>33</sup>

**Table 2: A summary of field placements for the Master of Epidemiology Program, Australian National University, 2012 to 2016**

Field placement type	Placement name (number of scholars completed and in progress 2012 to 2016)
Commonwealth Government departments	Office of Health Protection, Department of Health (3 completed, 2 in progress)
	Indigenous Health Division, Department of Health (1 completed, 3 in progress)
	Therapeutic Goods Administration, Department of Health (1 in progress)
State or territory health departments and regional public health units	Victorian Department of Health and Human Services (DHHS) (1 completed, 1 in progress, plus 2 completed and 1 in progress in shared placement with the Victorian Infectious Diseases Reference Laboratory, 1 in progress with Murdoch Children's Research Institute and 1 in progress with the Microbiological Diagnostic Unit)
	Centre for Disease Control, Department of Health Northern Territory (1 completed, 2 in progress)
	Western Australian Communicable Disease Control Directorate, Government of Western Australia Department of Health (1 completed and 1 in progress, shared with PathWest Laboratory; 1 in progress shared with Telethon Kids Institute)
	Communicable Disease Control Branch, SA Health (1 completed)
	Department of Health and Human Services, Tasmanian Government (1 completed, 1 in progress)
	Health Protection Branch, New South Wales Ministry of Health (1 completed, 1 in progress)
	Health Improvement Branch, ACT Health (1 in progress)

**Table 2 cont'd: A summary of field placements for the Master of Epidemiology Program, Australian National University, 2012 to 2016**

Field placement type	Placement name (number of scholars completed and in progress 2012 to 2016)
State or territory health departments and regional public health units, <i>cont'd</i>	Health Protection Service, ACT Health (1 completed, 1 in progress)
	Hunter New England Population Health, New South Wales Ministry of Health (1 completed, 1 in progress)
	Kimberly Population Health Unit, Western Australian Country Health Service (1 completed)
	Queensland Health (1 completed in shared placement with Queensland Children's Medical Research Institute, 1 in progress)
Public health laboratories	Victorian Infectious Diseases Reference Laboratory (2 completed and 1 in progress, in shared placement Victorian DHHS)
	Microbiological Diagnostic Unit (1 in progress in shared placement with Victorian DHHS)
	PathWest Laboratory (1 completed and 1 in progress, both in shared placements with Western Australian Communicable Disease Control Directorate)
National surveillance centres/research institutes/ non-government organisations/other	Médécins Sans Frontières (1 in progress, based in India)
	National Centre for Immunisation Research and Surveillance (3 completed and 2 in progress)
	The Kirby Institute (1 completed, partially funded by Leonard Broome Scholarship)
	Australian Institute of Aboriginal and Torres Strait Islander Studies (1 in progress)
	Murdoch Children's Research Institute (1 in progress based in Lao People's Democratic Republic, 1 in progress in shared placement with Victorian DHHS)
	Queensland Children's Medical Research Institute (1 completed in shared placement with Queensland Health)
	Telethon Kids Institute (1 in progress, in shared placement with Western Australian Communicable Disease Control Directorate)
	National Aboriginal Controlled Community Health Organisation (1 completed)
	Healthdirect Australia (1 in progress)
	National Critical Care Trauma Response Centre (1 in progress)

and practice of field epidemiology are applicable to other areas of public health and we will continue to expand placements beyond communicable diseases. Indigenous health will remain central to the MAE Program and we aim to have at least 1 Indigenous scholar graduate per year, as well as at least 2 other field placements with a primary focus on Indigenous health. We plan to strengthen links with both government and non-government organisations in this area, as well as exploring further options to increase the pool of Indigenous applicants. This may include developing bridging courses and alternative entry pathways for Indigenous candidates from non-traditional academic backgrounds.

During their time on the program, MAE scholars have investigated over 300 outbreaks, established and evaluated national and local surveillance systems and have published more than 200 papers in peer reviewed journals. This body of work has contributed to evidence-based actions, policies, programs and practice in Australia and internationally. As the funding for the MAE Program now comes from a diverse range of field placement organisations it is imperative to determine how the program can continue to effectively meet the needs of field placement partners and the broader public health community to promote, protect and restore health. To this end we are currently evalu-

ating the MAE Program with an emphasis on the contributions of students and staff to public health in Australia and globally.

This evaluation includes analysing MAE outputs to document systematically how and where MAE projects have contributed to public health, including factors that have facilitated or hampered this process. We are also examining whether the traditional field epidemiology curriculum, originally designed to strengthen the surveillance and control of communicable diseases, best equips scholars to contribute effectively to their field placements. From a technical standpoint, field epidemiology is becoming increasingly challenged by rapid advances in many areas. These include developments in diagnosis, analysis, prevention and management such as whole genome sequencing; increased availability of large administrative data sets; data-linkage and the use of novel sources of data for disease surveillance. For this reason, MAE scholars must now be competent to work in a high-tech environment whilst still acquiring the investigative competencies needed for 'shoe leather' epidemiology. Furthermore, to conduct and transform research into effective public health actions and policies, scholars must also grasp and apply principles of systems-thinking when exploring causality and when seeking to influence policy-makers. Through this evaluation we hope to inform decisions on revising or fine-tuning the curriculum and strengthen learning, teaching and training techniques to ensure that the MAE Program focuses on those areas where our scholars, in collaboration with Program partners, will have the greatest impact within and beyond Australia's borders. We will also review administrative aspects of the current Program, such as how field placements and supervisors can be better supported to host MAE scholars, and the relative advantages or disadvantages of the 2 models of funding (employee and scholar).

MAE scholars constitute an important surge workforce during national and international public health emergencies. This was demonstrated most recently during the 2014 to 2015 EVD outbreak, when almost all scholars were involved in surveillance activities for EVD at either the jurisdictional or national levels, and 3 scholars and 1 staff member were deployed to West Africa to assist with the public health response. It is our intention that MAE scholars continue to provide this service at both a national and global level, a resource that now extends beyond scholars currently enrolled in the program via the [Australian Response MAE \(ARM\) network](http://www.arm.org.au/) (<http://www.arm.org.au/>). Established by 3 MAE alumni after Typhoon Haiyan in the Philippines in 2013, the ARM network is built on the alumni of the MAE Program<sup>34</sup> and functions

as a focal point for identification, selection and referral of Australian public health practitioners for deployment to public health emergencies through the World Health Organization's Global Outbreak Alert and Response Network (GOARN), RedR, or other agencies.<sup>35</sup> The ARM network is open to all public health epidemiologists and other public health specialists, not solely MAE alumni. Since its establishment, ARM has conducted training workshops on EVD and on measles, and facilitated the deployment of Australian practitioners (including a significant number of MAE alumni) to assist with responses to EVD in West Africa.

No single country in isolation can respond effectively to the escalating public health threats and challenges resulting from globalisation. The work and orientation of the MAE Program must be contextualised within a global health framework. The MAE Program will continue to work with other FETPs in the region and globally, including supporting the development of an adapted model of the MAE in the Pacific. This work aligns with regional health priorities of the Australian government and other national partners. Targeting work towards government priorities as well as forming collaborative research partnerships with international organisations will also help our continuing efforts to identify alternative funding sources that will ensure the ongoing expansion and long-term sustainability of the MAE Program.

## Conclusions

In 2016, the MAE Program celebrates 25 years as Australia's FETP. Australia requires competent field epidemiologists to detect and respond effectively to ongoing and emerging threats and challenges to public health in the realms of communicable, non-communicable and other diseases. Indeed the International Health Regulations (2005) require that member states have the core capacities to detect and respond to public health events of international concern, and an FETP is an essential component of building and maintaining this capacity. With the ongoing support of field placement partners and NCEPH, the MAE Program has emerged from recent funding cuts in a strong position. Indications are that it will adapt continuously to the changing national and global context and provide another 25 years of training and service to Australia and the global community.

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# MULTIDRUG-RESISTANT TUBERCULOSIS IN THE NORTHERN TERRITORY: A 10-YEAR RETROSPECTIVE CASE SERIES

Daniel Judge, Vicki L Krause

## Abstract

**Background and objective:** To describe the clinical characteristics, risk factors, diagnostic modalities, treatments, subsequent outcomes and complications of Multidrug-resistant tuberculosis (MDR-TB) cases residing in the Northern Territory.

**Methods:** A retrospective case series was conducted of all patients treated for MDR-TB in the Northern Territory between 1 January 2004 and 31 December 2013. This is the first study to analyse data relating to the subset of MDR-TB cases treated in the Northern Territory. Cases were identified by the Northern Territory Centre for Disease Control (NT CDC): the public health unit responsible for the management of tuberculosis in the Northern Territory. Outcome measures included patient demographics, diagnostics, HIV status, treatment methods, outcomes, and complications.

**Results and conclusions:** Six MDR-TB cases were treated in the Northern Territory; 5 of these were notified by the NT CDC during the study period (1.5% of all Northern Territory TB notifications). The median age of all 6 patients was 31 years (range 21 to 50 years), sex distribution was equal and all were born overseas. Country of birth in a World Health Organization (WHO) high burden MDR-TB country and previous treatment were most highly correlated with a current diagnosis of MDR-TB. Access to rapid drug susceptibility testing reduced the time to effective therapy from 45 to 27 days. Five patients met criteria for the WHO outcome term 'treatment success'. The median length of treatment for the 5 patients treated in Australia was 623 days (537 to 730 days). Side effects to therapy were common and serious. The incidence of MDR-TB in the Northern Territory is similar to other Australian states. Rapid drug susceptibility testing reduces the time to effective therapy. Treatment regimens are complex, toxic and have serious resource implications for health care providers. Successful treatment outcomes are possible with coordinated TB control programs. *Commun Dis Intell* 2016;40(3):E334–E339.

**Keywords:** tuberculosis, multidrug resistance, Northern Territory

## Introduction

Treatment of multidrug-resistant tuberculosis (MDR-TB), defined as resistance to isoniazid and rifampicin, is longer and requires more expensive and more toxic drugs,<sup>1</sup> than fully susceptible disease. Nearly 20 years after the World Health Organization (WHO) declared TB a global health emergency, major progress has been made towards targets for diagnosis and treatment of TB.<sup>1,2</sup> Advancement towards targets for MDR-TB control has been less successful.<sup>1</sup>

A review of the published literature shows the incidence of MDR-TB notifications varies over time and across Australian states. A 10-year review of Victorian data found that MDR-TB accounted for up to 2.2% of all TB notifications between 1998 and 2007.<sup>3</sup> Western Australian MDR-TB notifications over a 15-year period to 2012 accounted for 1.2% of all TB cases.<sup>4</sup> Higher rates of MDR-TB are reported from the Torres Strait Protected Zone (TSPZ), with 26% of isolates from the TSPZ defined as MDR-TB in one Queensland study.<sup>5</sup> Yearly reports from the Australian Mycobacterium Reference Laboratory Network between 1985 and 2013 show that the proportion of isolates from patients with MDR-TB has stayed within a band of 0.5 to 2.4% nationally, excluding those from the TSPZ.<sup>6,7</sup>

The WHO estimates that the cost of treating MDR-TB is 100 times that of susceptible TB and treatment success globally is attained in only 48% of MDR-TB cases.<sup>2</sup> This has wide reaching repercussions for public health planning and may have important implications for hospitals treating even a single case of MDR-TB.<sup>8</sup>

Here we describe the characteristics, risk factors, diagnostics, treatments, subsequent outcomes and complications of MDR-TB cases residing in the Northern Territory, for all or part of their therapy in the 10 years to 2013.

## Methods

All patients treated for MDR-TB in the Northern Territory between 1 January 2004 and 31 December 2013 were included in the study. Formal drug

susceptibility testing demonstrating drug resistance was undertaken at the Victorian Infectious Diseases Reference Laboratory.

Data obtained from the Northern Territory Notifiable Disease System (NTNDS) included: patient demographic information; known previous TB infection and anti-tuberculosis therapy; risk factors for MDR-TB including country of birth or residence in a high MDR-TB burden country and contact with an MDR-TB case; diagnostic information including formal drug susceptibility testing (DST) and molecular methods; HIV status; and treatment methods, outcomes, and complications. Descriptive statistical analysis for these data points was undertaken using Microsoft Excel. Treatment outcome was defined as successful in accordance with WHO guidelines adopted from Laserson et al (Table 1).<sup>9</sup>

The study received ethical approval from the Human Research Ethics Committee, part of the Menzies School of Health Research.

## Results

Review of the NTNDS revealed 6 patients with laboratory-confirmed MDR-TB who received treatment in the Northern Territory during the study period. Data included 1 individual previously notified in Victoria and subsequently treated in the Northern Territory. The Northern Territory Centre for Disease Control (NT CDC) notified 5 cases of MDR-TB, representing 1.5% (total of 343 TB notifications) of all cases notified over the 10-year period to 2013.

The median age of the 6 patients was 31 years with a range of 21 to 50 years and sex distribution was equal with 3 male and 3 female patients. All patients were born overseas and all countries of origin were defined as WHO high burden TB countries, as well as high MDR-TB burden countries (Table 2).<sup>1,2</sup> Country of origin was therefore the most highly associated risk factor with a diagnosis of MDR-TB. The next most common risk factor was previous diagnosis of TB +/- exposure to treatment and this included 3 patients with a laboratory-confirmed or suspected diagnosis of TB in their past. Of these, 2 had documentation of previous exposure to rifampicin and isoniazid as part of an appropriate treatment regimen (Table 3). Treatment adherence documentation varied. However, 2 of the cases were suspected to have been non-adherent with therapy. Only 1 case had resided in a high MDR-TB country other than country of birth and there were no exposures to known MDR-TB cases. All of the cases identified as MDR-TB were tested for human immunodeficiency virus (HIV) and all were negative.

The period between arrival in Australia and notification of TB was less than 2 years in 4 of the patients. The remaining 2 patients were notified at 10 and 19 years post arrival dates. Two of the patients were permanent residents of Australia. Of the remaining 4, 2 held working visas in Australia, 1 patient was seeking asylum and 1 individual was an unauthorised fisherperson. Three of the cases were identified after self-presentation with symptomatic disease, 2 cases were identified as a part of routine screening in detention, and 1 case was identified as a result of a health care undertaking required for an Australian visa.

**Table 1: Treatment outcome definitions for multidrug-resistant tuberculosis patients**

<b>Cure</b>	Treatment completed as recommended by the national policy without evidence of failure AND 3 or more consecutive cultures taken at least 30 days apart are negative after the intensive phase.
<b>Treatment completed</b>	Treatment completed as recommended by the national policy without evidence of failure BUT no record that 3 or more consecutive cultures taken at least 30 days apart are negative after the intensive phase.
<b>Treatment failed</b>	Treatment terminated or need for permanent regimen change of at least 2 anti-tuberculosis drugs because of: <ul style="list-style-type: none"> <li>• lack of conversion by the end of the intensive phase; or</li> <li>• bacteriological reversion in the continuation phase after conversion to negative; or</li> <li>• evidence of additional acquired resistance to fluoroquinolones or second-line injectable drugs; or</li> <li>• adverse drug reactions.</li> </ul>
<b>Died</b>	A patient who dies for any reason during the course of treatment.
<b>Lost to follow-up</b>	A patient whose treatment was interrupted for 2 consecutive months or more.
<b>Not evaluated</b>	A patient for whom no treatment outcome is assigned. (This includes cases "transferred out" to another treatment unit and whose treatment outcome is unknown.)
<b>Treatment success</b>	The sum of Cured and Treatment completed.

\* World Health Organization guidelines adapted from Laserson 2005<sup>8</sup>

**Table 2: Demographics of multidrug-resistant tuberculosis cases undergoing treatment in the Northern Territory, 2004 to 2013**

Case	Year of diagnosis	State or territory of notification	Sex	Age	Country of birth	Visa status
1	2004	NT	Female	36	South Africa	Working visa
2	2006	NT	Male	50	Indonesia	Illegal fisherperson
3	2009	NT	Female	33	Vietnam	Permanent resident
4	2010	NT	Male	26	Bulgaria	Permanent resident
5	2010	Victoria	Female	29	Burma	Working visa
6	2012	NT	Male	21	Afghanistan	Illegal arrival

**Table 3: Risk factors for multidrug-resistant tuberculosis cases, Northern Territory, 2004 to 2013**

Case	Country of birth in a high-burden MDR-TB country	Previous diagnosis of tuberculosis +/- exposure to treatment	Suspected non-adherence or inappropriate tuberculosis therapy	Exposure to a known MDR-TB case	Residence in areas of high MDR-TB prevalence (other than country of birth)	HIV status
1	Yes	No	No	No	No	Negative
2	Yes	Yes	Yes	No	No	Negative
3	Yes	No	No	No	No	Negative
4	Yes	Yes*	Yes	No	No	Negative
5	Yes	Yes	No	No	Yes	Negative
6	Yes	No	No	No	No	Negative

\* Empiric treatment in Australia for possible fully susceptible tuberculosis as no culture/susceptibility testing was available.

Adapted from the World Health Organization 2008 guidelines

In addition to formal drug susceptibility testing on all cases, nucleic acid amplification testing (NAAT) was available for 2 of the patients and identified the presence of *Mycobacterium tuberculosis* DNA and *rpoB* gene mutations, a surrogate for rifampicin resistance, in both specimens. Both patients were commenced on second line agents at initiation of intensive phase therapy in the context of suspected rifampicin resistance. The median delay to effective treatment with second line therapies for all patients was 47 days. Those who underwent NAAT testing had a reduced median delay to 29 days. The delay to effective therapy was defined as the time from diagnostic specimen collection to the commencement of a second line treatment regimen. Four patients had pulmonary tuberculosis only, 2 of whom were sputum smear positive. The 2 extra-pulmonary cases included 1 diagnosis of disease limited to the terminal ileum and 1 case of axillary TB lymphadenitis (Table 4). Drug susceptibility testing identified 3 cases of streptomycin resistance (streptomycin was not used at any time in any of these 3 cases) and 1 case of pyrazinamide

resistance (the case isolate was not identified as *Mycobacterium bovis*). Resistance to other second line treatment agents was not demonstrated.

Five patients met criteria for the WHO cumulative outcome term 'treatment success' (either cure or treatment completed outcome categories as per Table 1). One case was classified as 'not evaluated' due to a transfer out to a resource limited setting overseas. Data at 1 and 5 years post treatment were limited, but no known cases of reactivation have been identified.

The median length of treatment for the 5 patients who completed therapy in Australia was 623 days with a range of treatment lengths from 537 to 730 days. The entire treatment period was completed in the Northern Territory in only 2 instances. One case was transferred out (deported) at 7 months of therapy having completed only 214 days of treatment. The remainder of the cases had treatment coordinated by multiple Australian jurisdictional tuberculosis control units. Diagnostic, treatment composition, and compliance data were incomplete



**Table 4: Site of disease, smear positivity and diagnostic modality for multidrug-resistant tuberculosis cases**

Case	Pulmonary disease	Smear result	Site of extra-pulmonary disease	Identification of MDR-TB by diagnostic modality	
				PCR	DST
1	Yes	Negative	NA	Not undertaken	Yes
2	Yes	Positive	NA	Not undertaken	Yes
3	No	NA	Terminal Ileum	Not undertaken	Yes
4	No	NA	Axillary lymph node	Yes	Yes
5	Yes	Positive	NA	Yes	Yes
6	Yes	Negative	NA	Not undertaken	Yes

MDR-TB Multidrug-resistant tuberculosis.

PCR Polymerase chain reaction.

DST Drug susceptibility testing.

as a result of the transfer of patients and it is noted that on at least 1 occasion a significant interruption to therapy complicated this process.

The treatment of MDR-TB in the Northern Territory involves directly observed therapy. Adherence data were excellent (approaching 100%) for all cases during the period of treatment coordinated by the NT CDC. Contact tracing identified 25 at risk individuals for appropriate follow-up.

All 6 cases commenced an injectable therapy (4 intravenous amikacin and 2 intramuscular streptomycin) in the first instance. One case developed a significant adverse outcome acutely (Table 5) and 4 cases on amikacin required permanent vascular access (peripherally inserted central catheter). All cases were treated with a later generation fluoroquinolone (moxifloxacin) and half received a thioamide as part of their regimen (Table 6). Two cases required hospitalisation with a combined total of 248 inpatient days. Adverse reactions to second line treatments were noted in 4 of the 6 cases (Table 5).

## Discussion

MDR-TB accounted for 1.5 % of all TB notifications in the Northern Territory over the decade to 2013. With respect to Australian and global MDR disease burden, this figure was lower than may have been expected in the context of the Northern Territory's position geographically and politically, with 3 immigration detention centres accommodating asylum seekers and alleged illegal fisherpersons (also referred to as unauthorised persons). The nationalities of unauthorised persons reviewed by the NT CDC are represented in the WHO defined 27 countries of high MDR-TB disease burden.<sup>1,2</sup> No significant difference in dis-

**Table 5: Side effects by case and drug implicated**

Case	Implicated drug	Side effect
1	NA	NA
2	Isoniazid or moxifloxacin	Stevens-Johnson syndrome
3	Amikacin	Ototoxicity
4	Amikacin	Ototoxicity
5	Prothionamide	Nausea
6	NA	NA

**Table 6: Multidrug-resistant tuberculosis definitive treatment regimen**

Drug	Number of cases employing drug for all or part of treatment regimen
Isoniazid	1/6
Rifampicin	0/6
Rifabutin	1/6
Ethambutol	6/6
Pyrazinamide	5/6
Moxifloxacin	6/6
Prothionamide	3/6
Amikacin*	5/6
Streptomycin	2/6
Para-aminosalicylic acid	1/6

ease burden is identified between the results of this study and national data.<sup>3,4</sup> The regular transfer of unauthorised persons between detention centres and subsequent notifications interstate may confound results leading to lower than anticipated case numbers.

Distribution of disease between sexes was even. While our numbers are small this finding is contrary to Toungousova et al 2002,<sup>10</sup> who identified a trend towards women being at higher risk of carrying MDR-TB strains. The median age of diagnosis (31 years) likely reflects the age of expected and unauthorised arrivals to Australia.

Country of birth being a high MDR-TB burden country was the most frequently reported risk factor for a diagnosis of MDR-TB, in keeping with current reports.<sup>1,4</sup> All of the cases treated for MDR-TB in the Northern Territory were born in 1 of the WHO defined 27 high MDR-TB burden countries. One case had resided in a WHO defined high burden TB or MDR-TB country other than their country of birth, prior to diagnosis. Primary transmission of MDR-TB was suspected in half of the cases with the other 3 previously treated for presumed susceptible disease. Previous treatment documentation and adherence varied and initial DST results were unknown. The importance of obtaining an isolate for culture and formal drug susceptibility cannot be overstated in the setting of the emergence of drug resistance. Acquired resistance was considered likely in these 3 cases.

One case considered as possible acquired resistance received an initial supervised and then a subsequent unsupervised treatment in Australia. As there were no links to any other Australian MDR-TB cases this case represents an episode of possible acquired resistance in Australia or a missed primary MDR-TB that was not adequately treated, as an isolate for susceptibility testing was not available.

It is felt that all but possibly 1 of the cases brought latent MDR-TB from their country of birth or residence overseas. It is noted that 4 of the 6 cases were identified within 2 years of arrival. Half of the cases were identified within 2 years of arrival by routine screening of individuals in detention, or health care undertakings. Analysis of enhanced data collected on all national MDR-TB cases will be useful in guiding future policy.

The WHO recommends rapid drug susceptibility testing of isoniazid and rifampicin or of rifampicin alone over conventional testing or no testing at time of diagnosis, subject to available resources.<sup>2</sup> A rapid test is defined as that yielding diagnostic and resistance results within 2 days. Only molecular tests can detect resistance so rapidly, of which 2 technologies: line probe assay and Xpert® MTB/RIF, are currently recommended by WHO.<sup>11</sup> Molecular testing data were available for cases from 2010 onwards. Xpert® was diagnostic on MDR-TB specimens subsequently identified by culture (1 sputum, 1 lymph node

tissue sample). Detection of *rpoB* gene mutation, as a surrogate marker for rifampicin resistance, correctly identified both cases of MDR-TB. Diagnosis in the Northern Territory has been based on either diagnostic modality result returning positive in the first instance, with DST taking precedence over NAAT if results are discordant in the same sample.

The positive predictive value of any test will decrease with the decline in the prevalence of the disease in question, an important consideration for the use of molecular testing for resistance in the low prevalence MDR-TB Australian population. Inappropriate treatment with toxic, less effective second-line therapy in patients with susceptible disease is concerning, but specificity on newer generation Xpert® assays are very promising (99.8%).<sup>9</sup>

There are significant disease control implications for the rapid determination of drug resistance, ensuring successful treatment of the patient and preventing further spread of the drug-resistant isolate.<sup>12</sup> One individual diagnosed with smear positive pulmonary disease was diagnosed with MDR-TB on NAAT, allowing appropriate treatment and infection control mechanisms to be employed earlier. This was evidenced in the 18-day reduction in delay to appropriate treatment for patients investigated with NAAT on clinical isolates.

Significant variation in treatment regimens was noted among cases. The attempt to tailor individual treatments is likely to go part of the way to explaining this observation. However, expert consensus with regard to regimen composition, dose, and duration has historically been lacking worldwide and continues to evolve. To work to provide the best standard of care, the Northern Territory has an MDR-TB steering committee that meets to initially assess each case and decide on management and then meets as needed or at least 3 monthly for continued follow-up. Four cases were treated with amikacin necessitating permanent intravascular access. Three of those cases experienced complications specifically related to this drug (Table 6). In effect, only 1 case successfully completed the WHO recommended 8 month intensive phase with a parenteral agent.<sup>8</sup> The increased toxicity of second-line anti-tuberculosis regimens is also evident in the observation that 4 patients experienced significant adverse drug effects including hepatitis, ototoxicity and Stevens-Johnson syndrome. The increased complexity and toxicity of treatment regimens necessitates more frequent reviews and closer clinical and laboratory monitoring for toxicity and drug levels in some instances. There are parallel increases in resource consumption

through inpatient admissions, multidisciplinary specialist input, vascular access and the extended treatment duration associated with this diagnosis.

Contact tracing identified 25 individuals for review and follow-up. These numbers are very manageable, likely as a result of the low number of smear positive individuals, and in those cases seeking asylum or identified as unauthorised fisherpersons, by the early active case finding carried out once in Australia and attendant respiratory isolation. The NT CDC does not advocate routine prophylaxis for infected contacts of MDR-TB patients because of the lack of an agreed-on treatment regimen and paucity of outcome data to support this approach. Rather chest x-ray programs and patient and healthcare provider education are employed for surveillance for those contacts considered to be at increased risk of infection with MDR-TB.

‘To fulfil her/his public health responsibility, as well as responsibility to the individual patient, the provider must prescribe an appropriate treatment regimen, monitor adherence to the regimen and, when necessary, address factors leading to interruption or discontinuation of treatment’.<sup>13</sup> Coordination of directly observed treatment programs for patients in this study was complicated by multiple routine transfers of patients in detention, with 1 case experiencing a lengthy interruption in appropriate therapy as a result of transfer between care providers within Australia. A 2nd case was deported and lost to follow-up at 7 months. This preceded completion of the 8 month recommended intensive treatment phase and was 13 months short of the total treatment duration outlined in the most recent WHO MDR-TB guidelines.<sup>8</sup> Successful treatment outcome was defined in all cases as treatment completed rather than cure (Table 1) as definitive sputum smear results were unavailable.

The movement of patients between TB control programs, as well as deportation, represents a potential obstacle to the coordinated treatment and follow-up of MDR-TB patients. The paucity of treatment outcome data in this study is attributed to the regular transfer of patients.

This study is the first to investigate demographic details, diagnostic modalities, treatment regimens, and outcomes associated with MDR-TB in the Northern Territory. Treatment outcomes were known and successful for all but the deported case at the completion of treatment and at 12 months follow-up. It is anticipated that data will be incorporated into a future national study that will offer greater insight into the Australian experience with MDR-TB. Such research is required to address

the important gaps in knowledge with regard to: optimising combinations of drug regimens and treatment duration; treatment of paediatric MDR-TB; effective chemoprophylaxis for contacts of MDR-TB cases and strategies to avoid or therapies to relieve adverse reactions to second-line anti-tuberculosis drugs.<sup>8</sup>

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# INFLUENZA VACCINATION COVERAGE AMONG PREGNANT INDIGENOUS WOMEN IN THE NORTHERN TERRITORY OF AUSTRALIA

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## Abstract

Pregnant Aboriginal and Torres Strait Islander women are at particular risk of severe illness and high attack rates of influenza infection. In Australia, routine seasonal influenza vaccination is currently strongly recommended for all pregnant women and women planning pregnancy, and is provided free of charge for all pregnant women. We sought to determine vaccination coverage, describe the trends and characteristics associated with influenza vaccine uptake and determine the validity of self-reported influenza vaccination in a population of Indigenous pregnant women who were participants of a vaccine trial, prior to and during the 2009 H1N1 influenza pandemic. Vaccine coverage over the study period was 16% (35/214), increasing from 2.2% (3/136) in the period preceding the pandemic (2006–2009) to 41% (32/78) in the intra-pandemic period (2009–2010). Self-report was not a reliable estimate of verified vaccination status in the pre-pandemic period ( $\kappa=0.38$ ) but was reliable in the intra-pandemic period ( $\kappa=0.91$ ). None of the socio-demographic characteristics that we examined were associated with vaccine uptake. Whilst the increase in maternal influenza coverage rates are encouraging and indicate a willingness of pregnant Indigenous women to be vaccinated, the majority of women remained unvaccinated. Activities to improve influenza vaccination coverage for Indigenous pregnant women and monitor vaccine uptake remain a priority. *Commun Dis Intell* 2016;40(3):E340–E346.

**Keywords:** Aboriginal and Torres Strait Islander peoples; influenza; Northern Territory; vaccination coverage

## Introduction

Influenza is responsible for considerable morbidity within Australia each year.<sup>1</sup> The annual (seasonal) influenza vaccine is recommended for all persons more than 6 months of age who have risk factors for influenza infection. This 'at risk' group includes all women who are pregnant during an influenza season as well as all Indigenous Australians 15 years of age or over, both reflecting the disproportionate influenza-related morbidity and mortality observed in these 2 groups.<sup>2</sup>

Influenza infection during pregnancy places the health of both the mother and the fetus at risk, which has been demonstrated in numerous settings worldwide.<sup>3,4</sup> Influenza infection during pregnancy is associated with adverse maternal and neonatal outcomes including preterm labour and delivery, pneumonia, hospitalisation and death.<sup>5</sup> Influenza-attributable morbidity and mortality is of particular concern in women who will be pregnant in the second or third trimester during the influenza season.<sup>6</sup>

Aboriginal and Torres Strait Islander people experience elevated rates and severity of influenza infection compared with their non-Indigenous counterparts. Pneumonia is the most common communicable disease contributor to premature mortality in the Indigenous population, with children under 5 years of age and adults over 25 years of age most at risk.<sup>7</sup> Data from the 2009 H1N1 pandemic indicated that among Indigenous Australian adults residing in the Northern Territory rates of notification, hospital admission and Intensive Care Unit admission were 3.5, 12 and 5 times higher, respectively, than for non-Indigenous adults.<sup>8</sup>

A higher prevalence of comorbidities is 1 factor that has been associated with the increased risk of severe influenza infection among Indigenous Australians.<sup>7,9</sup> These include cardiac disease, chronic respiratory conditions, chronic obstructive pulmonary disease, severe asthma, diabetes mellitus and chronic renal failure.<sup>10,11</sup> Socio-demographic factors include a high level of mobility between communities, overcrowded living conditions, poverty, and poorly constructed and maintained housing.<sup>8,9,12</sup> All are recognised contributors to the spread of communicable diseases such as influenza.<sup>8,9</sup>

A comprehensive influenza vaccination program targeting Indigenous Australians commenced in 1999.<sup>13</sup> Indigenous Australians aged 50 years or more and those aged 15–49 years with at least 1 risk factor for complicated influenza disease are eligible for free influenza vaccination.<sup>14</sup> Since 2007, the recommendation is that all Indigenous adults receive the vaccination, but the vaccine is not free for those under 50 years of age who don't have a risk factor.<sup>2,7</sup>

In Australia, routine seasonal influenza vaccination is currently strongly recommended for all pregnant women and women planning pregnancy, and is deemed safe to be administered at any stage of pregnancy.<sup>2</sup> The annual influenza vaccine is provided free in the Northern Territory for all pregnant women.<sup>15</sup>

Despite evidence for the risk of influenza during pregnancy and the safety and efficacy of influenza vaccination in pregnancy,<sup>16</sup> coverage estimates of influenza vaccine during pregnancy in Australia remains low (23% to 27%).<sup>17,18</sup> Two recent studies into the determinants of influenza vaccination in Australia have determined that a high proportion of women would accept vaccination (68% to 74%) if recommended by their health care provider.<sup>17,18</sup> We have not identified any previous studies of influenza vaccine uptake in pregnancy among Indigenous Australians.

## Methods

This cross-sectional study was nested within a randomised controlled trial (RCT), which aimed to determine the effectiveness of maternal 23-valent pneumococcal polysaccharide vaccine in preventing ear disease in Indigenous infants residing in the Northern Territory.<sup>19</sup> Participants identified as Aboriginal or Torres Strait Islander; were aged between 17 and 39 years; had a current, uncomplicated singleton pregnancy; intended to deliver their infant at the Royal Darwin Hospital or Alice Springs Hospital; lived in 1 of 2 regional (Darwin and Alice Springs) or 4 remote participating communities; intended to live in the study area until 7 months post-partum; and were between 30 and 36 weeks gestation (inclusive at the time of enrolment). Enrolment occurred from August 2006 to January 2011.

Data were collected through face-to-face interviews with participants. In addition, we undertook systematic review of medical records, documenting all medical clinic attendances (including hospitalisations) within the previous 5 years, using a structured data collection form. Influenza vaccine uptake was ascertained through self-report and from documented receipt of influenza vaccination in medical records. We also validated influenza vaccination history through the Northern Territory Centre for Disease Control adult immunisation database. Comorbid medical conditions and various socio-demographic characteristics of participants (maternal and gestational age, parity, education, tobacco use and household occupancy) were recorded.

Vaccine coverage was calculated annually for the 12-month period prior to the participant delivery

date. These data were compared with influenza notification rates in the Northern Territory for the same period, calculated from the National Notifiable Diseases Surveillance System website.<sup>20</sup> The inter-rater agreement between self-reported influenza vaccination during pregnancy and documented vaccine receipt in participant medical records in the 12-month period prior to the interview was calculated. The validity of self-reported vaccination was compared for the pre-pandemic and intra-pandemic H1N1 influenza periods. The latter was defined as the release of the World Health Organization first Global Alert and Response declaration of influenza A(H1N1) on 24 April 2009. Whilst data on documented vaccination coverage were obtained for all participants, self-report of influenza vaccination during pregnancy was only assessed from 25 September 2007. Therefore, the period 25 September 2007 to 24 April 2009 was compared with the period 25 April 2009 to 4 May 2010, the date the final vaccine study participant was interviewed (at that time the pandemic was ongoing<sup>21</sup>).

Data analyses were carried out using Stata software, version 11 (StataCorp, LP, College Station, TX). Wilcoxon rank sum test and Pearson's chi-square test for independence were used to compare groups for continuous and categorical variables, respectively. Cohen's kappa indices were calculated to assess the inter-rater agreement between self-report and true coverage. Kappa values above 0.80 were considered as almost perfect agreement.<sup>22</sup>

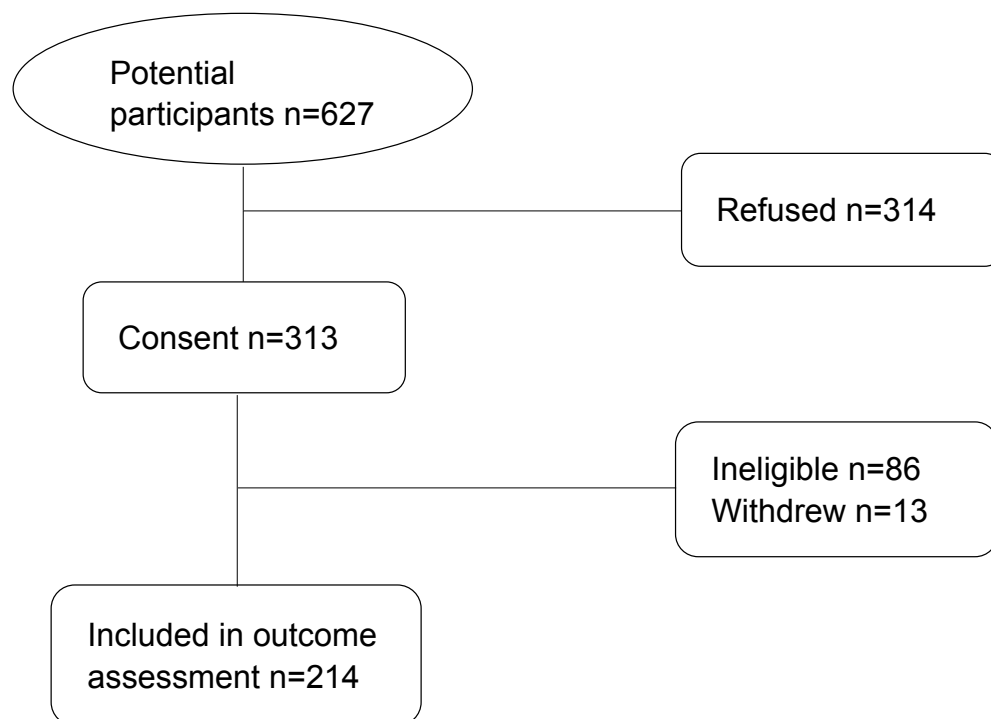
## Ethics approval

Participants provided written informed consent to participate in this study. This study (and consent procedure) was granted ethics approval by the Human Research Ethics Committee of the Northern Territory Department and Health and Families and the Menzies School of Health Research (05/52).

## Results

### Characteristics of participants

There were 627 pregnant Indigenous women who were invited to participate in the RCT, of whom 313 (50%) consented (Figure 1). Over the 4-year period, the consent rate for the vaccine study remained at approximately 50% of all approached participants (Figure 2). Whilst 86 women were subsequently deemed ineligible, a further 13 participants withdrew leaving 214 participants who completed the antenatal and post-partum assessments that contributed to this report.

**Figure 1: Recruitment and retention of study participants**

There was no significant difference in maternal age, gestational age and parity between the 214 eligible participants and 86 ineligible participants (Table 1). Among the 214 eligible participants, 49 (23%) participants had at least 1 medical risk factor for influenza with asthma ( $n=27$ , 13%) being the most common (Table 2).

### Maternal influenza vaccine coverage

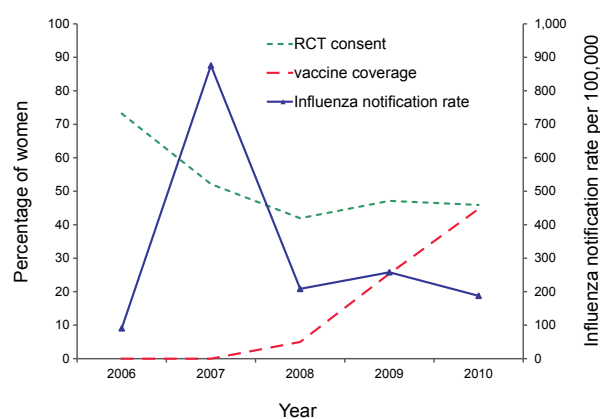
We ascertained influenza vaccination status from all 214 participants, among whom 16% ( $n=35$ ) had been vaccinated within the 12 months preceding their due date. Of the 35 women with documented influenza vaccination, 2 (6%) were vaccinated prior to becoming pregnant, 9 (26%) during their first trimester of pregnancy, 13 (37%) in their 2nd trimester and 11 (31%) in their 3rd trimester.

Influenza vaccination coverage during the pre-pandemic period was 2.2% (3/136). The 3 women vaccinated during this period received their vaccines in January 2008 ( $n=1$ ) or April 2008 ( $n=2$ ). Vaccine uptake then increased more than 10-fold to 41% (32/78) during the subsequent intra-pandemic period. Over this same period, from April 2009, there was a significant increase in the number of influenza notifications in the Northern Territory, which coincided with the H1N1 influenza pandemic (Figure 2).<sup>20</sup>

### Validity of self-reported influenza vaccination

Vaccination status was verified against self-report in a subset of 138 of the 214 women in the study group. Data from 76 women were excluded as these participants completed an earlier version of the eligibility assessment where self-reported vaccination was not assessed. Overall, self-report of influenza vaccination was a reliable estimate of documented

**Figure 2: Rate of laboratory-confirmed influenza notifications and influenza vaccine coverage of pregnant Indigenous women, Northern Territory, April 2006 to June 2010, by year**



Source: Communicable Disease Control, Northern Territory Department of Health

**Table 1: Socio-demographic characteristics of participants**

Variable	Consenting participants (n = 300)				Bivariate analysis ( $\chi^2$ or Wilcoxon rank sum test) P value
	Eligible for vaccine study (n=214)		Ineligible for vaccine study (n=86)		
<b>Maternal age (years)</b>					
Median (IQR)	24	(21–28)	25	(21–31)	0.37
Range	17–39		17–38		
<b>Gestational age* (weeks)</b>					
Median (IQR)	29	(24–32)	30	(26–33)	0.73
Range	6–36		14–39		
<b>Primiparity</b>					
Yes	73	(34)	25	(34)	0.84
No	141	(66)	48	(66)	

\* Gestational age at time of eligibility assessment.

IQR Interquartile range.

**Table 2: Clinic-documented medical conditions of eligible participants (n=214)**

Category	n	%
Cardiac disease	9	4.2
Chronic respiratory conditions	1	0.5
Chronic obstructive pulmonary disease and chronic emphysema	0	
Asthma	27	13.0
Diabetes mellitus	15	7.0
Chronic metabolic diseases	0	
Chronic renal failure	2	0.9
Haemoglobinopathies	0	
Impaired immunity	0	
Chronic neurological conditions	0	
History of long-term aspirin therapy as a child	0	
Total (1 or more medical risk factor)	49	23.0

**Table 3: Validity of self-report of maternal influenza vaccination in pregnant Indigenous women, Northern Territory, September 2007 to May 2010\***

Period	Self-report		True coverage		$\kappa$	Sens.	Spec.	PPV	NPV
	N	%	N	%					
Pre-pandemic (n=71)	2	2.8	3	4.2	0.38	0.33	0.99	0.50	0.97
Intra-pandemic (n=67)	27	40.0	28	42.0	0.91	0.93	0.97	0.96	0.95
Total (n=138)	29	21.0	31	22.0	0.87	0.87	0.98	0.93	0.96

\*  $\kappa$  denotes kappa index, Sens. denotes sensitivity, Spec. denotes specificity, PPV denotes positive predictive value and NPV denotes negative predictive value.

vaccine receipt ( $\kappa=0.87$ ) (Table 3). However, in the pre-pandemic period, confirmed vaccination coverage of the subset was only 4.2% (3/71) and the positive predictive value of self-reported vaccination was 0.5, meaning that for every 2 women who reported being vaccinated; only 1 had actually been vaccinated. The validity of self-report during this period was low ( $\kappa=0.38$ ).

In the intra-pandemic period, documented vaccine coverage of the subset was 42% (28/67) and the positive predictive value of self-reported vaccination was 0.96. The validity of self-report was significantly higher in this period than the pre-pandemic period ( $\kappa=0.91$ ). Self-report of influenza vaccination underestimated documented coverage in both the pre-pandemic and intra-pandemic periods but the absolute difference was very small, 1.4% and 2.0% respectively.

### Socio-demographic characteristics associated with vaccination

None of the socio-demographics characteristics that we investigated were associated with the likelihood of influenza vaccination: maternal age (OR 0.39,  $P$  value 0.38), parity (OR 0.97 95% CI 0.42 to 2.2,  $P$  value 0.38), medical condition (OR 1.0, 95%CI 0.36 to 2.5,  $P$  value 0.99), education

(OR 2.4, 95%CI 0.67 to 13,  $P$  value 0.16), tobacco use (OR 1.0, 95%CI 0.45 to 2.2,  $P$  value 0.99) or overcrowded living conditions (OR 1.5, 95%CI 0.66 to 3.5,  $P$  value 0.31) (Table 4).

### Discussion

In our study population, uptake of influenza vaccine coverage during the pre-pandemic period was negligible (2%). There was a substantial increase in vaccine uptake that coincided both with the declaration of the global alert for influenza H1N1 and the local increase in influenza notifications. Whilst the improvement in the vaccine uptake to 41% was encouraging the coverage rate remained sub-optimal. The majority of pregnant women remained unvaccinated and susceptible to the complication of influenza during pregnancy.

The 10-fold increase in vaccine coverage between the pre- and intra-pandemic periods provided suggests that given the right conditions, women will choose to receive the vaccine. While it was beyond the scope of this study to ask women their reasons for choosing to be vaccinated or not, a review of the literature shows a number of key initiatives that took place in Australia during that period may have contributed to this increase in coverage. The first World Health Organization

**Table 4: Association between socio-demographic characteristics and documented influenza vaccination status of pregnant Indigenous women, Northern Territory, 2006 to 2010**

Variable	Pregnant women n=214				Bivariate analysis ( $\chi^2$ or Wilcoxon rank sum test)	
	Vaccinated		Not vaccinated		$P$ value	OR (95% CI) difference of averages (years)
	n	%	n	%		
<b>Maternal age</b>						
Median	24		24		0.38	0.39
<b>Primiparity</b>						
Yes	12	16.0	62	84.0	0.97	0.98 (0.42–2.2)
No	23	16.0	117	84.0		
<b>Presence of one or more medical risk factor</b>						
Yes	8	16.0	41	84.0	0.99	1.0 (0.36–2.5)
No	27	16.0	138	84.0		
<b>Highest year of education</b>						
$\geq 10$	32	18.0	144	82.0	0.16	2.4 (0.67–13)
$< 10$	3	8.6	32	91.0		
<b>Tobacco use</b>						
Yes	16	16.0	82	84.0	0.99	1.0 (0.45–2.2)
No	19	16.0	97	84.0		
<b>Children in house &lt;5 years of age</b>						
Yes	23	19.0	101	81.0	0.31	1.5 (0.66–3.5)
No	12	13.0	78	87.0		



global pandemic alert on 24 April 2009 set in motion the Australian Health Management Plan for Pandemic Influenza.<sup>23</sup> A key component of this infection control strategy was an extensive public health campaign, raising awareness among medical practitioners and the general public of the risks of pandemic H1N1 influenza infection. This campaign was accompanied by rapid manufacture, the Therapeutic Goods Administration approval and funding of a monovalent (September 2009) and then trivalent pandemic influenza vaccine (November 2009).<sup>24,25</sup>

We found that self-reported influenza vaccination status to be a reliable indicator for documented receipt of the vaccine for the whole study period and the intra-pandemic period. With only 3 women vaccinated in the pre-pandemic period, the correlation was less reliable at this time. There were no socio-demographic characteristics of participants that were significantly associated with receipt of the inactivated influenza vaccine.

There are a number of limitations that may impact on the generalisability of our findings. Participants had agreed to take part in a vaccine trial, which may mean that study participants were more likely to be accepting of vaccines compared with the wider population. In addition, participants were generally more educated and had less chronic illness than the Indigenous female population of the Northern Territory.<sup>26,27</sup> Given these factors, we expect our calculation of vaccine coverage may have overestimated true coverage of pregnant Indigenous women in the Northern Territory. Given a relatively small sample of women, our within cohort comparison of the specific factors that we examined may have been underpowered to exclude an association with vaccine uptake.

Whilst acknowledging the limitations of the study, we highlight several findings that require public health action. Relatively low uptake in both the pre- and intra-pandemic period demonstrates a need for more effective, targeted activities to improve influenza vaccine uptake during pregnancy among Indigenous Australians specifically but possibly among all pregnant women in Australia. The heightened public awareness of influenza post-pandemic offers an opportunity to continue to promote maternal influenza vaccination. However, strategies to improve uptake must be accompanied by comprehensive monitoring of coverage.

National telephone surveys to determine vaccine coverage are unlikely to reach a significant proportion of pregnant Indigenous women, who may have low access to landlines.<sup>27</sup> The proposed whole of life immunisation register may be a feasible option

for monitoring vaccine uptake in pregnancy and would also allow for the evaluation of vaccination programs across Australia.<sup>28,29</sup>

Research within Australia and overseas into attitudes and behaviours of pregnant women and their health care providers, including obstetricians, midwives and general practitioners, has demonstrated that health care provider recommendation is the most significant influence on maternal influenza vaccine uptake. Studies in Canada and the United States of America have found that pregnant women whose health care providers recommend the vaccine were 32 (95% CI: 10-100) and 57 (95% CI: 37-86) times more likely to receive the vaccine, respectively, than women who did not receive an offer or encouragement of vaccination from their health care providers.<sup>30,31</sup> Within Australia, women who had received a recommendation to have the vaccine were 20 times (95% CI, 10.9–36.9) more likely to have been vaccinated.<sup>18</sup> Within Australia, the greatest barrier to vaccination was reported to be concerns about vaccine safety.<sup>17</sup>

There may well be other predictors of vaccine uptake among pregnant Indigenous women in Australia but, to our knowledge, these are yet to be comprehensively investigated. In our view, such work should not only involve exploration of socio-demographic predictors of uptake but also qualitative research into the attitudes and behaviours of pregnant women and their health care providers.

## Conclusion

The provision of inactivated influenza vaccination during pregnancy is considered the safest and most effective way of protecting women and their babies from the complications of influenza during pregnancy.<sup>32</sup> Our data suggests that pregnant Indigenous women are prepared to be vaccinated; however, activities to promote improved coverage and understand barriers in our setting are required. In addition, routine monitoring of vaccination in pregnancy is also required to ensure uptake in this high-risk group reflects national recommendations.

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# A BRIEF OVERVIEW OF INFLUENZA SURVEILLANCE SYSTEMS IN AUSTRALIA, 2015

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## Introduction

The World Health Organization (WHO) has estimated that worldwide 5% to 15% of the population is affected by influenza each year, with between three and 5 million cases of severe illness and about 250,000 to 500,000 deaths.<sup>1</sup> In Australia, it has been estimated that the disease is associated with 366 respiratory and 1,400 all-cause deaths,<sup>2</sup> 18,000 hospitalisations and over 300,000 general practice consultations<sup>3</sup> each year. The morbidity, mortality and consequent economic burden of influenza epidemics vary annually. Although typically falling within the winter months in Australia, the onset and severity of annual epidemics varies. Therefore, robust surveillance is needed to guide prevention and controls efforts.

In Australia, the National Influenza Surveillance Scheme<sup>4</sup> (the Scheme) began in 1994 and its objectives are to:

- ensure the early detection of influenza epidemics;
- trigger public health prevention and control activities;
- characterise the epidemic, especially identification of risk groups and disease severity;
- estimate the impact of the epidemic;
- characterise the circulating viruses to inform vaccine virus selection and assess the effectiveness of influenza vaccines and antiviral medications; and
- ensure flexibility to enable adaptability for responding to additional surveillance requirements during a pandemic or particularly severe season.

The Scheme is currently guided by the *Enhanced Influenza Surveillance Framework for Australia* (unpublished) developed by the Communicable Diseases Network Australia (CDNA) after the moderately severe 2007 influenza season. Ongoing monitoring and enhancement of the Framework is co-ordinated by the National Influenza Surveillance Committee, a subcommittee of CDNA. The Scheme is supported by a number of government and other surveillance systems which are combined to enable monitoring of influenza incidence, severity, transmission and virology. These systems capture influenza activity in the community, general practice (GP) clinics, emergency departments and hospitals, as well as influenza-associated mortality.

This paper provides a brief overview of the range of influenza surveillance systems that formed the Scheme in 2015 and describes their respective strengths and limitations in describing the epidemiology of influenza. The Scheme is coordinated by the Australian Government Department of Health (DoH). Influenza activity monitored through its systems is reported in the Australian Influenza Surveillance Report, which is published fortnightly on the DoH web site during the influenza season, and an annual surveillance report, which is published in the *Communicable Diseases Intelligence* journal.<sup>5</sup> For a more detailed description and analysis of the Scheme, including surveillance systems that function outside of the Scheme, readers are referred to the paper *A Summary of Influenza Surveillance Systems in Australia, 2015*,<sup>6</sup> which is available on the DoH web site.

## National notifiable diseases

Under state and territory public health legislation, notifications of laboratory-confirmed influenza are initially made to jurisdictional health authorities by laboratories and, in some states, medical practitioners. These data are forwarded to the National Notifiable Diseases Surveillance System (NNDSS) on a daily basis and are the primary source of national influenza activity data. Aggregated data are available online and more detailed data can be requested from CDNA. An agreed surveillance case definition and core data specifications ensure national consistency in case counting and quality of data by person, place and time.

The system is considered acceptable, simple and valuable by stakeholder groups.<sup>7</sup> The quality and completeness of these data are affected by a range of factors including healthcare seeking behaviours of patients, clinician testing propensity, notification practices and case follow-up by jurisdictional health departments. The impact of these factors on the notified fraction (the cases notified as a proportion of all cases occurring in the community) is likely to vary over time and across jurisdictions, making year-on-year comparisons difficult.<sup>8</sup>

## Community self-report surveillance

Influenza-like illness (ILI) is widely used as a surrogate measure for influenza infection. Definitions vary, but typically include fever, cough, fatigue, sore throat or some combination of these symptoms.<sup>9,10</sup>

Two self-report based systems in Australia monitor ILI in the community: Flutracking and the National Health Call Centre Network (NHCCN).

Flutracking, established in 2006, is an online health surveillance system in which volunteer participants are surveyed weekly, via email. Surveillance is conducted during the influenza season to capture ILI episodes self-reported by participants or nominated household members.<sup>11,12</sup> The information collected includes specific symptoms, absence from normal duties, medical consultation, clinical or laboratory diagnosis of influenza and influenza vaccination status. During 2015 there were about 27,000 participants, with over 23,000 completing the survey each week.

The NHCCN has provided free, 24-hour health triage advice and information services by telephone since 2007. The network services all states and territories, except Victoria and Queensland. Registered nurses use electronic decision support software to provide advice to roughly 640,000 callers per annum. Data collected include demographic details of the patients, presenting issue, diagnosis and final triage disposition. Selected diagnoses are used to monitor ILI. Since 2009, NHCCN data were routinely provided to DoH; however, due to system changes and associated incompatibilities, data transmissions could no longer be received after mid-2015.

Flutracking surveillance of ILI was used during the 2009 H1N1 influenza pandemic to demonstrate that community attack rates were no higher than most other years and suggested that much of the increase in influenza notifications was due to an increased health care seeking behaviour coupled with increased testing of those patients.<sup>13</sup>

Whilst ILI surveillance is only a surrogate indicator for influenza, as it is based on a non-specific set of symptoms that may be caused by a number of respiratory pathogens, ILI activity tends to correlate well with laboratory confirmed influenza reports.<sup>14</sup> Although there are some discrepancies between the NHCCN and Flutracking ILI surveillance systems, such as differing ILI case definitions, and geographic and demographic representativeness, which limit direct comparison and interpretations; as they have been collected in a relatively consistent manner over a number of seasons, they do offer reference to ILI activity at the community level.

### General practice sentinel surveillance

General practice based sentinel surveillance systems capture data on medically-attended ILI and influenza activity trends. The largest GP-based ILI surveillance system in Australia is the Australian Sentinel Practices Research Network (ASPREN). Established in 1991, ASPREN collects de-identified information on ILI and other conditions seen in

general practice. All patients presenting with ILI at participating practices are enumerated, and, since 2010 samples have been collected from around 20% of these patients for laboratory testing for a range of respiratory pathogens, including influenza. GPs submit data using a web-based form, paper form or a data extraction tool that utilises practice management software to extract information on ILI cases, including demographics, vaccination status, and total number of consultations. ASPREN aims to achieve a participant rate of one GP per 200,000 population in urban settings and one GP per 50,000 population in rural and remote settings.

Victoria and Western Australia manage separate systems: the Victorian Sentinel Practice Influenza Network, established in 1993 with swab testing since 2007; and the Sentinel Practitioners Network of Western Australia, based on a system originally established in 2000. More than 70% of ILI patients in these 2 systems are swabbed for laboratory confirmation.

All 3 systems collect information from swabbed patients, including vaccination status and high risk conditions, to enable calculation of vaccine effectiveness. A current limitation in enhancing the representativeness of vaccine effectiveness calculations through data pooling across the 3 systems relates to their differing participation targets, laboratory testing practices and data collection methods.

### Emergency department surveillance

Emergency department (ED) surveillance systems for influenza that inform the national Scheme operate in New South Wales, the Northern Territory and Western Australia. Additionally, data from Queensland and South Australia's ED surveillance systems are monitored to inform local influenza activity trends.<sup>7</sup> Like GP ILI surveillance, ED surveillance is an indicator of the ILI burden in the community, severity of a season and may capture groups in the community that are under-represented in GP surveillance, especially the very young.<sup>15,16</sup>

ED surveillance in New South Wales commenced in 2003 and includes 59 urban and rural hospitals. Influenza is monitored using provisional diagnosis codes recorded by either an International Classification of Diseases 9th or 10th revision (ICD-9 or ICD-10)<sup>17,18</sup> code or a Systematized Nomenclature of Medicine – Clinical Terminology<sup>19</sup> concept identifier. Although not necessarily laboratory confirmed, these presentations correlate well with laboratory-confirmed influenza reports.<sup>20</sup> Incidents of related conditions including pneumonia, respiratory illness and fever or unspecified infections, are also monitored. Statistical signals trigger when indicators exceed expected thresholds.

In the Northern Territory, ED surveillance commenced in 2007 and is conducted across the Royal Darwin, Gove District, Katherine District, Tennant Creek and Alice Springs hospitals. These hospitals use the same information system from which the data are transmitted nightly to a data warehouse. Business intelligence software is then used to analyse information on presenting complaints and discharge diagnoses. The presenting complaints included in the ILI definition are: 'febrile illness', 'cough', 'respiratory infection' and 'viral illness'. Trends are analysed using CuSum techniques to determine activity changes for each hospital site.

Western Australia uses the Emergency Department Information System for ED surveillance in 9 public Perth metropolitan EDs and one regional hospital ED. Data on respiratory viral presentations (upper respiratory tract infection and viraemia) are extracted weekly. These diagnoses were chosen as they best correlated with notification and laboratory data for influenza. Respiratory viral presentation data are also used to monitor the number and rate of ILI hospital admissions through EDs.

In its current form, ED surveillance in Australia has limited capacity to build a nation-wide picture of ILI activity. Each jurisdiction bases their definition of ILI on different presentation codes, and may have a different method of data collection or abstraction. Some jurisdictions (Australian Capital Territory, Tasmania and Victoria) do not carry out ED surveillance, limiting representativeness. Year-on-year comparisons can be hindered by upgrades to hospital information systems and the absence of reliable denominator data. Harmonisation of the diagnostic case definitions used and the methods of data extraction could enable pooling of data and comparison of activity among jurisdictions, including those currently not formally included in the national Scheme.

## Hospital surveillance

Surveillance for hospitalised cases of influenza is useful for gauging the severity of a season and measuring the burden placed on health services. Three main hospital based systems operate as part of the national Scheme: the Influenza Complications Alert Network (FluCAN), Queensland EpiLog and the Australian Paediatric Surveillance Unit (APSU). Additionally, data from New South Wales and Western Australia's hospital admission surveillance systems are monitored to inform local influenza activity trends.<sup>6</sup> While a field for hospitalisation is included in the NNDSS dataset, these data are currently not easily captured or of sufficient completeness for routine analysis.

FluCAN was established in 2009 and provides national, sentinel, hospital-based surveillance for severe influenza.<sup>21,22</sup> In 2015 there were 17 par-

ticipating hospitals that represented 12% of national hospital bed capacity. FluCAN also includes information about paediatric patients from 2 paediatric hospital sites, with data on paediatric patients also collected from 4 of the community-based hospital sites. Extensive information on all laboratory-confirmed influenza-positive patients admitted to participating sites is collected, including demographics, comorbidities, vaccination status, intensive care unit admission and mortality. The collection of vaccination status and comorbidities also permit the calculation of influenza vaccine effectiveness estimates against hospitalisation and can provide information on nosocomial influenza infections.

In 2009, Queensland introduced EpiLog; a system of near-real-time surveillance of public hospital admissions for ILI. Patients admitted with influenza are identified through the linkage of laboratory test results with admissions data. These data include patients diagnosed with influenza prior to admission, but do not capture patients admitted to private hospitals.

The APSU has monitored children (<15 years) hospitalised with severe complications of influenza since 2008. Data are reported by paediatricians and other child health clinicians, who report demographics, diagnosis, treatments and short-term outcomes.

Enhanced surveillance of hospitalised cases provides useful information on the severity of an influenza season and its burden on hospitals. FluCAN, with its use of standard case definitions, facilitates uniform national reporting of hospital data, with influenza status confirmed by polymerase chain reaction (PCR) testing. In addition, during epidemics with high severity or other significance, 2 additional systems have historically been accessed to provide additional information: the Australian New Zealand Intensive Care Society and the Paediatric Active Enhanced Disease Surveillance system.

The current limitations to hospital data, include the lack of denominator (i.e. source population) data to calculate incidence rates. Additionally, while Queensland and Western Australia have the capacity to track patients through the public hospital system, it is currently not easy to track a patient's journey from community care (e.g. GP consultations) into the hospital system. This information would facilitate routine estimation of the risk of hospitalisation among patients with confirmed clinical disease.

## Mortality surveillance

Influenza-related mortality surveillance also provides an important indicator of the severity of a season. Three main sources of national influenza

mortality data are utilised: notified laboratory confirmed influenza deaths, official coding of influenza related deaths from national vital statistics reporting, and estimates of excess mortality associated with influenza epidemics using time series analysis.

The NNDSS is able to record deaths associated with a laboratory-confirmed case of influenza. While these data are not easily captured at the time of notification, to improve the completeness of the died status field of notified cases, a range of variably applied methods have been employed by jurisdictional health departments. These methods include: cross-matching of notifications with local death registration data; reporting by doctors; linkage to hospitalisation records; and reporting of deaths detected by sentinel hospital surveillance systems to jurisdictional health departments. Current limitations to these methods include: the variability in the methods used to improve the completeness of the died status field; the timeliness of which the information is available; and the potential discrepancies in the methods applied to determine the relatedness of a death to an influenza notification.

National death data compiled by the Australian Bureau of Statistics and the National Death Index report coded or all-cause death registrations, but these data are not timely enough for public health response or reporting throughout the influenza season. However, retrospective analysis of mortality data can provide an estimate for the severity of an influenza season, and can be used to validate real-time analyses.

Timely death registration data and analyses are reported through the New South Wales Ministry of Health's Influenza Surveillance Reports.<sup>23</sup> Although jurisdictionally based, these data can be utilised to inform mortality trends through comparisons of influenza and pneumonia deaths to previous years' data and trends.

### Laboratory surveillance

Laboratory-based surveillance provides information on the extent and characteristics of circulating influenza. Some types of laboratory surveillance are useful for developing baselines and thresholds to indicate the start and end of a season as well as inform severity assessment, while others are used to monitor antigenic drift, antiviral drug susceptibility and inform vaccine effectiveness.

National influenza centres (NICs) are part of the WHO Global Influenza Surveillance and Response System, a network tasked with monitoring changes in influenza viruses with the aim of informing influenza vaccine composition. NICs collect virus

specimens, perform preliminary analysis (usually by reverse transcription PCR (RT-PCR) and ship representative and unusual clinical specimens and isolated viruses to WHO collaborating centres for advanced antigenic and genetic analysis. Australia has 3 NICs; PathWest Laboratory Medicine (Perth, WA), the Victorian Infectious Diseases Reference Laboratory (Melbourne, Victoria) and Pathology West (Sydney, NSW); and 1 collaborating centre (Melbourne, Victoria).

The WHO Collaborating Centre receives influenza virus samples from NICs and other public and private health laboratories around Australia for virus characterisation. Viruses undergo various assays to assess antigenic and genetic drift, as well as sensitivity to antiviral drugs. These data are reported weekly to the DoH.

The proportion of requested respiratory tests positive for influenza provides a further indicator of influenza activity. This method is less biased than simply counting positive cases (as in the NNDSS notified cases), as it provides a denominator for controlling annual fluctuations in testing behaviours. 'Laboratory per cent positive' data are reported as part of the national Scheme by the NICs, and Tasmanian laboratories.

The timing, severity and economic burden of influenza seasons depends on the dominant circulating strain, so there is a compelling need to consider the A subtypes and B lineages separately. Many laboratories now use RT-PCR to confirm influenza infection. However few provide A subtypes and only the WHO collaborating centre, PathWest and Pathology West are able to provide lineage of type B viruses. Thus there is variable determination of subtypes or lineages between jurisdictions, which is a limitation of laboratory surveillance.

### Conclusion

Australia's National Influenza Surveillance Scheme generally provides timely syndromic and laboratory surveillance of influenza from the community through to hospitalisation and death, but each system has its own inherent limitations and no system is completely accurate. Therefore, limitations of the Scheme's component surveillance systems must be taken into consideration when interpreting their outputs, and conclusions are best based on a considered assessment of all the indicators. Overall, the components of the Scheme combine to meet the stated goals of the system in informing control measures to lessen the burden of influenza in the Australian community and ensure that decision makers have access to the best available and timely information on which to base their decisions.

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## Short reports

# TIMING OF INFLUENZA VACCINATION IN AN AUSTRALIAN COMMUNITY-BASED SURVEILLANCE SYSTEM, 2010–2014

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In Australia, the National Immunisation Program provides influenza vaccine free to people at high-risk of severe influenza including the elderly, anyone with an underlying medical condition, pregnant women, Indigenous Australians and nursing homes residents. This program aims to deliver the seasonal influenza vaccine to these groups before the onset of the influenza season, typically between March and May of each year. The timing of vaccination is important for a number of reasons.

For individual protection, the vaccine needs to be given before the influenza virus is circulating. Based on recent European research, some high-risk groups might be better vaccinated late in the pre-season because immunity may wane over the course of a single season.<sup>1–3</sup>

For estimates of influenza vaccine effectiveness (VE) derived from health facility-based surveillance data (using the test-negative study design), knowing when someone was vaccinated is important for determining whether they can be expected to have developed immunity as a result of vaccination. Such estimates typically exclude or consider unvaccinated those people vaccinated within 10 to 14 days of attendance at the health facility because vaccine-induced immunity takes this long to develop.<sup>4</sup> Avoiding this potential misascertainment of immunisation status is challenging in recruitment sites that are different from where people receive the vaccine, such as hospital-based studies, as the vaccination date may not be easily verified.<sup>4–9</sup>

The whole-of-life vaccine registry<sup>10</sup> could provide data on the timing of vaccination for adult groups targeted by the Australian National Immunisation Program. However, full implementation may take several years. We aimed to assess whether adults ( $\geq 18$  years of age) were vaccinated before the onset of the influenza season and whether misclassification bias was likely to be a concern for estimating VE in Australia by using data from 2 existing national influenza surveillance systems for 2010 to 2014.

We plotted the onset of illness for patients with influenza admitted to hospitals participating

in the Influenza Complications Alert Network (FluCAN),<sup>11</sup> an Australia-wide hospital-based sentinel surveillance system, against the uptake of influenza vaccine throughout each season among participants of Flutracking,<sup>12</sup> an online national community influenza-like illness (ILI) surveillance system.

Flutracking participants were recruited by a combination of emailed invitations via organisation email networks, government and commercial workplaces, promotional activities in the media, and increasingly through participants inviting friends over recent years.<sup>13</sup> The cumulative proportion of vaccinated participants who reported being vaccinated was plotted by week for each season to document vaccine uptake. Vaccinated participants needed to have responded to at least 1 weekly online survey by the end of the influenza season defined as a period of 24 to 26 weeks between April and October in each year.

In hospitals participating in FluCAN, data are collected on hospitalised patients with confirmed influenza including the date of their illness onset and their vaccination status. The number of vaccinated hospitalised patients with confirmed influenza by week of onset of illness in each season was plotted as a measure of severe influenza activity.

We separately graphed those aged 18–64 years and those aged 65 years or over as the provision of free vaccine to the elderly may influence the timing of vaccination. Except for Indigenous status, Flutracking does not collect data on other medical and demographic factors that influence eligibility for free government supplied vaccine. Although the group of patients aged 18–64 years is expected to be mostly made of people ineligible for free vaccine, it may include those who are eligible such as the few Indigenous participants as well as pregnant women or those with underlying medical conditions.

Ethical approval for Flutracking was obtained from the Hunter New England Human Research Ethics Committee. Ethics committees of the Australian National University and all participating hospitals approved FluCAN.



The number of Flutracking participants who reported being vaccinated has almost doubled since 2010 for those aged 18–64 years and increased by more than 3 and a half times for those aged 65 years or over (Table 1). By the beginning of June (week 22; Figure 1; Table 2), the majority of Flutracking respondents aged 65 years or over who received the influenza vaccine at any time during the season had already been vaccinated (range 70% to 91%). In 3 of the 5 seasons, slightly less of the younger age group (Figure 2; Table 2) had been vaccinated at this point (range 67% to 86%). Between 2% and 11% of patients admitted to hospital for a severe respiratory illness during each season had developed an ILI by the start of

June. In 2012, a higher proportion of patients were admitted earlier in the season compared with other years.

From the beginning of May (week 18–19) to the beginning of June (week 22), vaccine uptake among those aged 65 years or over increased from 46% to 70% in 2010, from 60% to 86% in 2011, 60% to 84% in 2012, 77% to 91% in 2013, and 63% to 89% in 2014.

These data suggest that the majority of people are vaccinated before the onset of the influenza season, at least for these 5 seasons that exhibit the typical winter period of transmission. In turn, this suggests that misascertainment bias is unlikely to be a major concern for hospital-based vaccine effectiveness studies in Australia<sup>14–18</sup> and that unusually early season onset (or delayed vaccine availability) would be needed to compromise the current tim-

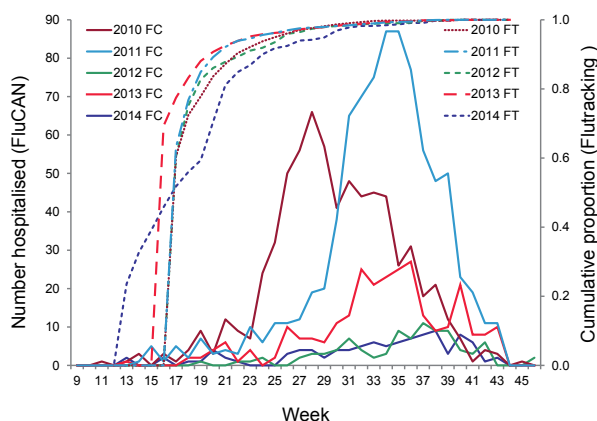
**Table 1: Number of vaccinated Flutracking respondents who completed at least one online survey during the year and patients admitted to FluCAN-participating hospitals with an influenza-like illness, 2010 to 2014, by age group and year**

Year	Vaccinated Flutracking respondents		Vaccinated patients admitted to hospitals participating in FluCAN	
	18–64 years	≥65 years	18–64 years	≥65 years
2010	5,716	450	122	100
2011	6,334	617	90	93
2012	7,272	915	329	684
2013	9,032	1,290	193	276
2014	9,786	1,617	490	837

**Table 2: Proportion of vaccinated Flutracking respondents who reported being vaccinated by week 22 in each season, 2010 to 2014, by age group**

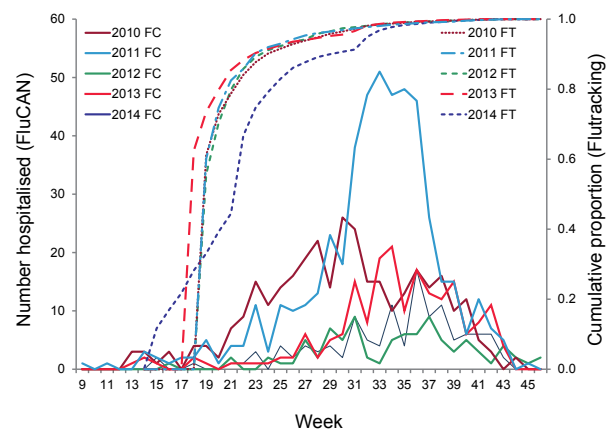
Year	Age group	
	18–64 years %	≥65 years %
2010	67	70
2011	86	86
2012	84	84
2013	88	91
2014	86	89

**Figure 1: The onset of illness for hospitalised patients aged ≥65 years with confirmed influenza and cumulative proportion of vaccinated Flutracking respondents aged ≥65 years who reported being vaccinated, 2010 to 2014, by week**



FC = FluCAN; FT = Flutracking

**Figure 2: The onset of illness for hospitalised patients aged 18–64 years with confirmed influenza and cumulative proportion of vaccinated Flutracking respondents aged 18–64 years who reported being vaccinated, 2010 to 2014, by week**



FC = FluCAN; FT = Flutracking

ing of the delivery of the vaccine program for most recipients. In Australia, the influenza vaccine usually becomes available in March, and we note that a significant proportion of the Flutracking sample had received vaccine by April–May, when Flutracking surveillance typically commences. It is possible that Flutracking participants may receive influenza vaccination earlier than the general population given that the weekly online survey reminds them about the vaccine by asking them if they have been vaccinated in the prior week. However, the uptake of vaccine among Flutracking participants who were vaccinated is consistent with an earlier Australian study that found that vaccine coverage estimated from pre-season pneumonia cases (in whom influenza was an unlikely cause) were similar to mid-season test-negative controls for those aged 65 years or over, those aged less than 65 years with a medical condition and those aged less than 65 years without a medical condition.<sup>19</sup>

Flutracking data for those aged 65 years or over are also consistent with periodic nationally representative vaccination coverage surveys by the Australian Institute of Health and Welfare (AIHW) in 2002, 2003, 2004, and 2009, which found that between 70% and 78% of the elderly were vaccinated by the end of April and 86% to 92% by the end of May.<sup>20–23</sup> A minority of Flutracking participants aged 65 years or over were vaccinated between the beginning of May and the start of June, indicating that most are vaccinated earlier in the season. Exactly how early is not known as Flutracking surveillance only commences in April. As noted, this is important for the elderly who may benefit from receiving the vaccine closer to the onset of the influenza season because of possible waning immunity.

AIHW vaccination coverage estimates for younger age groups tend to be lower than recorded by Flutracking, presumably related to selection biases associated with Flutracking such as much higher education levels of participants compared with the general population.<sup>24</sup> The Flutracking subset for those aged 18–64 years may also be biased by over-representation of groups eligible or ineligible for free government vaccine.

The consistency of Flutracking data with representative studies does suggest that it might be able to provide general information on when influenza vaccines are being given and an indication of the magnitude of misascertainment of immunisation status for hospital-based studies of influenza vaccine effectiveness. Early vaccine coverage estimates, whether through community or hospital-based systems, may be useful for identifying community concerns about vaccine safety and for triggering public health investigations to explore decreases

in coverage. For instance, negative public reaction to vaccination after the 2009 H1N1 influenza pandemic<sup>25</sup> led to slow uptake of the seasonal trivalent influenza vaccine in the following year (as suggested by the uptake of the vaccine in 2010 in Figures 1 and 2). Until an adult vaccine registry is established, these two surveillance systems can provide data on vaccine uptake that is not currently available from any other source. However, these data must be carefully appraised each season given the increasing but non-random community participation in Flutracking and the biases inherent to each system: FluCAN mostly relies on patient recall for the date of vaccination and Flutracking is a sample of online volunteers that have different socio-demographic features to the general Australian population.

## Conflicts of interest

None

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BC designed the study and, with AC, performed the analysis and drafted the manuscript. SC provided Flutracking data. All authors provided input into the analysis and interpretation of results.

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# Policy and guidelines

## DEFINING A TUBERCULOSIS CLUSTER OR OUTBREAK

Justin Denholm, Chris Coulter, Ivan Bastian and the National Tuberculosis Advisory Committee

### Executive summary

Transmission of tuberculosis (TB) in an Australian context is a relatively uncommon event. However, episodes of transmission do occur, and may have a large significance in a low-incidence region. Defining when transmission has occurred is not straightforward in a variety of circumstances, but may have significant epidemiologic, public health and political implications. This paper, therefore, will review approaches to determining when transmission has occurred, and offer standardised Australian policy for classification of possible transmission events, including ‘clusters’ and ‘outbreaks’.

### Key definitions:

- A ‘cluster’ of TB cases will be any 2 or more active cases with identified epidemiological links and the same genotype of *Mycobacterium tuberculosis* as defined by the method used.
- A ‘probable cluster’ will be any 2 or more active cases with identified epidemiological links where genotyping is not feasible (e.g. the case is not confirmed by culture) or the genetic variability between *M. tuberculosis* isolates recovered from cases is minimal, defined as no more than 1 locus variance for mycobacterial interspersed repetitive unit-variable number tandem repeat typing or as advised by expert analysis for whole genome sequencing.
- A ‘possible cluster’ will be any 2 or more active cases with the same genotype as defined by the method used where temporal and geo-spatial association is plausible but no direct epidemiological link is identified.
- An ‘outbreak’ will be defined as a cluster that includes 3 or more active cases with evidence of serial transmission.

### Introduction

The World Health Organization’s *Framework towards TB elimination in low incidence countries* highlights the importance of detailed understanding of epidemiology and transmission in local contexts.<sup>1</sup> The *Framework* emphasises the need to develop tailored public health interven-

tions in response to this information, particularly for ‘containment of local outbreaks’ in high-risk groups. However, no standardised inter-jurisdictional definition of an ‘outbreak’ is offered. Standardised definitions of terms for considering transmission within Australia is an important step towards an improved understanding of local disease epidemiology. Adopting uniform terminology across Australian state and territory jurisdictions would allow for better considerations of national epidemic descriptions, as well as comparison between and within regions. Such considerations are of considerable importance for TB service planning into the future, and in particular, allow detailed consideration of which of various approaches may be more likely to be effective in a given region or population. For example, 2 suburbs may have the same TB incidence but very different rates of transmission, and contexts with high clustering rates will benefit more from appropriately targeted strategies.

### Epidemiologic contact tracing

Historically, epidemiologic and contact tracing investigations have formed the basis of the evaluation of transmission of TB in most settings. In particular, the identification of household and other close contacts of known cases of active pulmonary TB through active case finding has been key to describing patterns of risk in many contexts.<sup>2,3</sup> This approach, still very much in use in all Australian jurisdictions, seeks to identify individuals with a history of significant contact with infectious tuberculosis in order to both find additional cases of TB and allow for chemoprophylaxis where TB infection has occurred.<sup>4,5</sup> However, while a history of close contact is associated with an increased risk of TB disease, 2 cases of active TB with known contact may not conclusively establish that transmission has occurred. For example, individuals with TB may have had opportunities for contact with multiple cases of TB in the past, some unrecognised, and it may not be clear which contact has led to infection.<sup>6,7</sup> Conversely, 2 cases of TB without known contact may be linked, as transmission through minor or casual contact is less common but recognised.<sup>8</sup> Therefore, in many circumstances, additional laboratory methods can be employed to further consider the degree to which 2 TB cases may be related.

## Laboratory approaches

Fundamentally, laboratory approaches to evaluating potential transmission events seek to evaluate the degree to which 2 (or more) clinical isolates of *M. tuberculosis* are related. While some methods for the identification of TB do not discriminate between isolates (such as microscopy or diagnostic polymerase chain reaction), others provide genomic detail, which may be used to demonstrate similarity or differences between isolates. As summarised in a recent comprehensive review, various methods provide a range of degrees of resolution, from pulsed-field gel electrophoresis, to spoligotyping IS6110-based approaches, mycobacterial interspersed repetitive unit-variable number tandem repeat (MIRU-VNTR) and whole genome sequencing (WGS) (in increasing order of discriminatory power).<sup>9</sup> A demonstration that 2 isolates are significantly different may occur using any method, but increasing confidence in concluding that isolates are clonally related can be provided with methods offering more resolution. Drug susceptibility testing patterns may also allow isolates to be distinguished, particularly where common genomic profiles are present.

In practice, Australian mycobacterium reference laboratories (MRL) in different jurisdictions all utilise MIRU-VNTR typing and some are increasingly employing WGS. The discriminatory power of a given method can be determined not only by the method but the strain of organism, the time over which transmission has occurred, the presence of mixed strain infection and the section of genome examined. Beijing family strains are well recognised to show restricted variability with conventional MIRU-VNTR typing; increased discrimination can be achieved by examining additional hypervariable VNTR loci,<sup>10</sup> which are not part of the panel usually used by Australian MRLs. Even whole genome sequencing seldom covers the whole genome and certain repetitive sequences are often excluded from analysis.<sup>11</sup>

## Review of existing published definitions

Clustering definitions from the United States Centers for Disease Control and Prevention (CDC) are based in the first instance on laboratory data; that is, questions of whether 2 cases of TB are linked are considered subsequent to the identification of genomically indistinguishable isolates.<sup>12</sup> Where 2 or more identical isolates are identified, they are referred to as 'clustered'. Epidemiological considerations are then employed to classify the strength of connection between 2 cases with clustered isolates, with links grouped as 'identified', 'possible' or 'none identified' based on disease characteristics and contact patterns.

Epidemiological and molecular publications on TB transmission have offered a variety of different approaches to defining related terms. In some high incidence settings, clusters may be defined on the basis of epidemiological connection alone (for instance, disease in individuals with shared membership in a household) or spatial proximity.<sup>13</sup> Others have adopted definitions of genomic relatedness to define clusters, sometimes with little epidemiological data beyond date and location of diagnosis.<sup>14</sup> Examples can also be found of laboratory evaluation of isolate similarity by non-genomic methods, such as comparison of strain drug-susceptibility test results.<sup>15</sup> In a review of TB outbreak investigations, the US CDC defined a TB outbreak as  $\geq 3$  epidemiologically linked and genomically matched cases,<sup>16</sup> but such an approach adds little further to the definition of cluster unless there is evidence of serial transmission.

## Special challenges

### Genomic linkage without local epidemiology

Where circulating international strains are common, cases in Australia may be identified where identical strains occur without known local contact. While connection between these cases (such as may have occurred prior to migration) is possible, the focus on these guidelines is on transmission within Australia. Therefore, definitions will concentrate on a requirement for local epidemiological contact; i.e. where local transmission is plausible based on geospatial and temporal association.

### Cases without culture confirmation

While the majority of cases of TB in Australia are confirmed by culture, a proportion are not. This may be due to the site of disease (e.g. pericardial TB or TB uveitis) or related to patient characteristics, particularly young age, where a substantial proportion of paediatric cases are not culture confirmed. While epidemiologic links may be very strong in such situations, such as an Australian-born child with no other history of TB exposure other than a parent recently diagnosed, the absence of genotypic confirmation may still leave some uncertainty regarding the potential transmission event.

### Evolution of genotype

The mutation rate of *M. tuberculosis* is low, but incompletely defined. It is accepted that changes in genetic composition occur with time, and it is theoretically possible for mutation to occur around the time of transmission. In such a circumstance, closely related but non-identical strains could be truly clustered. However, such events appear uncommon where MIRU-VNTR (24 loci) test-

ing is employed. Defining a genotyped cluster as sharing identical 24 loci MIRU-VNTR type has been employed in an Australian context<sup>17</sup> but published<sup>18,19</sup> and unpublished observations indicate that isolates recovered from cases with strong epidemiological links can occasionally show a single locus variance and this genomic clustering can be confirmed by use of a second typing method.

Two isolates of *M. tuberculosis* are judged to be the same by WGS if they differ by no more than 5 single nucleotide polymorphisms.<sup>19</sup> It is estimated that molecular evolution would anticipate 0.3–0.5 SNP differences per genome per annum,<sup>19</sup> but these 'molecular clocks' have broad confidence intervals and are not regular<sup>20</sup> and greater than 5 SNP differences to the index case may occur following sequential transmission over many years. In addition, estimates of mutational rate may differ for different phylogenetic lineages.<sup>21</sup> Current evidence indicates that strains with more than 12 SNP differences are very unlikely to be related; where there are 6–12 SNP differences transmission is possible.<sup>19</sup> Use of such definitions has recently been endorsed in a large multi-centre European/North American study.<sup>22</sup>

As whole genome sequencing is increasingly adopted, definitions regarding the degree of genetic change permissible within a cluster will be expected to be reassessed. For the purposes of this standardised Australian position paper, contemporary criteria as proposed by Walker<sup>19</sup> and supported by Pankhurst<sup>22</sup> shall be adopted.

### Time course of tuberculosis transmission

Finally, a general issue in TB transmission evaluation is the protracted time that may occur between exposure and development of subsequent disease. This means that any evidence of transmission in a given environment will have the possibility of change over time; that is, even years following potential exposure there remains the chance of additional cases of TB becoming evident. Accordingly, it is proposed that no time considerations be included in definitions related to TB transmission.

### Recommendations

Assessment of clusters defined by genomic data and possible transmission pathways within these clusters requires a close collaboration between laboratory specialists, clinicians and epidemiologists taking into account such factors as described above and new scientific information in a rapidly evolving field of study.

A 'cluster' of TB cases will be defined as any 2 or more cases with identified epidemiological links and the same laboratory (genomic and drug susceptibility) profiles. The capacity to define strains as being genetically the same is dependent on the method used and may be subject to change where a more discriminatory method is sequentially adopted. The term 'probable cluster' will be reserved for cases epidemiologically linked without genomic identification of organism (e.g. case not confirmed by culture) or where genotype is not indistinguishable but very closely related as discussed above for MIRU-VNTR typing and WGS. 'Possible cluster' will be reserved for the scenario where the genotype is the same but no epidemiological links are demonstrated but geospatial and temporal association is plausible. Where epidemiology or genomic testing demonstrates linkage is not possible, clustering is excluded. This may occur if case history is incompatible with transmission (for example, 2 cases with extra-pulmonary disease only, or cases not residing in the same state or country during a period of potential transmissibility) or if isolates are shown to be not clonally related.

The term 'outbreak' is not one typically defined in literature relating to TB, in part due to the lengthy latency periods, which may occur following exposure. However, it is felt that a working definition of an outbreak would be useful in an Australian setting, particularly given that identification of an outbreak may signal a need for increased resources applied to a given region or situation. We would suggest that the relevant features of a TB outbreak would be evidence of ongoing community transmission of a genotypic strain of TB, indicating that additional public health measures may be required for prevention of future cases. It is proposed, then, that an 'outbreak' will be defined as a cluster that includes 3 or more cases with evidence of serial transmission; that is, where at least 2 members of the cluster have transmitted disease. While a cluster may occur in a household setting, an outbreak is most unlikely.

It is important to note that these definitions are based only on active disease; cases of TB that result only in the probable acquisition of latent tuberculosis infection (LTBI) are neither clusters nor an outbreak, unless they progress to active disease in future. There are several reasons for this. Firstly, the absence of an isolate in LTBI means that acquisition from a given source is always to a degree, uncertain. Secondly, as a public health evaluation, the identification of recently acquired LTBI allows the use of chemoprophylaxis to prevent the development of active disease. Therefore,

inclusion of cases of LTBI within these definitions would not accurately reflect the public health focus of epidemiological surveillance.

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# INFECTION CONTROL GUIDELINES FOR THE MANAGEMENT OF PATIENTS WITH SUSPECTED OR CONFIRMED PULMONARY TUBERCULOSIS IN HEALTHCARE SETTINGS

Chris Coulter and the National Tuberculosis Advisory Committee

## Introduction

Tuberculosis is caused by the bacterium *Mycobacterium tuberculosis*. Globally, *M. tuberculosis* is responsible for an estimated 9.6 million new tuberculosis (TB) cases per annum with 1.5 million deaths estimated in 2014.<sup>1</sup> In Australia, there are around 1,200 to 1,400 cases of TB each year.<sup>2</sup> Worldwide, TB incidence is slowly declining and it is estimated that 43 million lives have been saved between 2000 and 2014.<sup>1</sup> TB disease can occur in pulmonary and extrapulmonary sites. Persons with extrapulmonary disease are usually not infectious unless the TB disease is located in the larynx or the oral cavity or if the extrapulmonary disease includes an open abscess or lesion where drainage fluid may be aerosolised.<sup>3</sup> Laryngeal tuberculosis should be considered as having the same or greater risk of transmission as smear positive pulmonary tuberculosis.

The risk of transmission in healthcare settings is increased when healthcare workers and patients come in contact with persons who:

- have unsuspected pulmonary TB;
- are not receiving adequate treatment; and, or
- who have not been isolated from others.<sup>4</sup>

These guidelines provide recommendations for healthcare workers to manage patients who are confirmed or suspected of having pulmonary TB.

## Transmission

TB is spread via inhalation of small particle aerosols (airborne route). When a person with pulmonary TB coughs, sings, laughs or sneezes, *M. tuberculosis* is generated and carried in droplet nuclei particles that are approximately 1–5  $\mu\text{m}$  in size.<sup>4</sup> Depending on the environment, tubercle bacilli can remain suspended in the air for prolonged periods and air currents can carry them throughout a room or building.<sup>4</sup>

Infectivity is directly related to the magnitude of viable organism load in respiratory secretions. The risk of transmission increases with duration of

exposure. Household members are at greatest risk of acquiring TB from an index case of pulmonary tuberculosis. In healthcare settings, the duration is often considered significant after eight accumulative hours of exposure have occurred but this is not an absolute cut off for decision making. Intensity of smear positivity, mechanical factors (e.g. aerosol generating procedures) and host vulnerability must all be taken into account. *M. tuberculosis* is transmitted only through air containing microdroplets of TB organisms. It is not transmitted by touching surfaces such as bed linen, toilet seats, shaking hands etc.

## Importance of early detection

The most effective measure to control TB in a healthcare setting is early detection. By having a high level of vigilance for TB, appropriate isolation can occur at an early stage. The early flags for TB, as listed in the Series of National Guidelines for TB<sup>5</sup> are:

- a chronic cough, sometimes accompanied by haemoptysis;
- fever and night sweats;
- loss of weight; and
- feeling generally tired and unwell.

Clinical suspicion of TB should be high in any person with exposure risk factors and a respiratory infection unresponsive to standard treatments or an unexplained non-respiratory illness. This particularly includes:

- new arrivals and recently returned travellers from high incidence countries;
- contacts of an active case within the past 5 years;
- those with a history of previous TB treatment;
- Indigenous Australians in localised areas (e.g. as occurs in parts of the Northern Territory and Queensland);
- patients with HIV or other immuno-compromised states; and
- elderly Australians.



It is important that clinicians specifically request that the laboratory stains for acid fast bacilli and performs TB culture. Rapid molecular tests should be utilised where clinically appropriate. Where multidrug-resistant TB is suspected, an Xpert MTB/RIF assay should be requested as this can detect the presence of rifampicin resistance as well as the presence of *M. tuberculosis* directly from sputum and some extrapulmonary specimens (including cerebrospinal fluid).

### Transmission based precautions

Transmission based precautions are additional work practices used in situations where standard precautions alone may be insufficient to prevent infections.<sup>4</sup> They are based on the use of personal protective equipment (PPE) appropriate to the mode of disease transmission and should always be used in conjunction with standard precautions.

### When to use airborne precautions

Airborne precautions are a subset of transmission based precautions and are used to prevent transmission of microorganisms that remain infectious over time and distance when suspended in the air. These agents may be inhaled by susceptible individuals who have not had face-to-face contact with (or been in the same room as) the infectious individual.<sup>4</sup>

Airborne precautions are indicated for all patients where pulmonary TB is suspected or proven. Patients with HIV TB co-infection may not have typical symptoms: pulmonary TB should be considered in the differential diagnosis of HIV positive patients epidemiologically at risk of TB (e.g. from higher burden TB countries) with respiratory symptoms or undiagnosed systemic illness.

At the point of first contact with a medical service, ideally designed engineering controls may not be available. In this setting the following is recommended:

- place the patient in an area that can be contained (i.e. a single room);
- ask the patient to wear a surgical mask when not in a single room or if air from the single room recirculates to other areas of the building, until advised to remove it by attending staff;
- if not wearing a surgical mask, cough etiquette should be used (covering mouth when coughing using disposable tissues, or hand followed by hand hygiene); and
- the door to the single room remains closed.<sup>4</sup>

Airborne precautions should also be used if any procedure involving aerosolisation is to be performed and tuberculosis is a diagnostic possibility.

Airborne precautions are not necessary for persons with extrapulmonary (where there is no evidence of pulmonary TB as well) and latent TB infection.

Non-tuberculous mycobacteria are not transmissible person to person. However, recent data suggesting the possibility of person to person transmission of *Mycobacterium abscessus* complex strains between patients with cystic fibrosis (CF) in the United Kingdom is acknowledged.<sup>6</sup> Until more information is available for other settings, infection control requirements for CF patients with *M. abscessus* complex isolates should be determined by local experts involved in CF care and infection control.

### Accommodation

Ideally patients with pulmonary TB should be accommodated in negative pressure rooms or Type 5 (respiratory isolation) rooms that are equipped with environmental controls to reduce the risk of transmission of airborne diseases. If this is not possible then the patient should be placed in a single room with en suite from which the air does not circulate to other areas.<sup>4</sup>

### Environmental controls

Environmental controls consist of engineering technologies that are designed to prevent the spread and reduce the concentration of infectious TB droplet nuclei in the air.<sup>4</sup> These engineering strategies include ventilation and high-efficiency particulate air (HEPA) filtration.<sup>7</sup>

The Australian standard is that all hospitals, irrespective of their size, should have at least one Type 5 (respiratory isolation) room and should aim to provide between 1% and 3% of all available beds for respiratory isolation.<sup>8</sup>

The final estimates of the number of rooms required for infection control purposes including the containment of TB should be made in consultation with clinicians, engineers, architects and the infection control committee.<sup>8</sup> Infection surveillance data collected for more than 12 months will assist in determining peak needs and marked seasonal variations recognising that other diseases may also require respiratory isolation.<sup>8</sup>

### Type 5 (respiratory isolation room) air-handling requirements

The supply and exhaust of Type 5 (respiratory isolation) rooms should provide a negative pressure, relative to the corridor and adjacent areas. To obtain the negative pressure, the exhaust flow rate should be a minimum of 10% greater than the supply air with all doors and openings closed.<sup>8</sup>

For a new building, air from Type 5 (respiratory isolation) rooms ideally should not be reticulated via, or to, any other ventilation system, i.e. it should be a single pass system. Air from these rooms should be exhausted directly to the outside of the building. The discharge points should be located as far as possible from air-intakes, persons and animals. It is recommended that the discharge point be positioned above the roof and at such a height and velocity that exhausted air is unlikely to re-enter the building or its ventilation system.<sup>8</sup>

Alternatively, where existing facilities do not allow external exhausting, air that is to be re-circulated should be directed through HEPA filters.<sup>8</sup> The door to the room should remain closed at all times. For Type 5 (respiratory isolation) rooms, air change rates greater than or equal to 12 air changes per hour with a minimum of 2 air changes per hour of outside air, whichever results in the greater air quantity, should be achievable when the filters have reached their maximum pressure drop.<sup>8</sup>

For further information on Type 5 (respiratory isolation) rooms and facility requirements please refer to Standards Australia, HB 260: Hospital acquired infections-Engineering down the risks.<sup>8</sup>

### Non-conventional settings

In non-conventional facility-based and congregate settings without a central ventilation system, natural ventilation can be useful.<sup>7</sup> Natural ventilation relies on open doors and windows to bring in air from the outside. When using natural ventilation, facility staff should be aware of the direction of airflow. If the air direction is known, staff should sit near the fresh air source and clients should sit near the exhaust location.<sup>7</sup>

### Prioritising type 5 (respiratory isolation) rooms

On occasions, certain patients may need to be prioritised for Type 5 isolation, including patients with other respiratory infectious diseases. Where there are two or more patients with TB, prioritisation should be given to smear positive over smear negative, drug resistance over pan susceptible, confirmed untreated smear positive over suspected. Decisions on prioritisation based on a combination of these parameters should be made in consultation with the local infection control service and the local TB service.

### Specimen collection

It is very important for healthcare workers to use infection control precautions to control the spread of tubercle bacilli during specimen collection

procedures and any other procedures that may cause persons who have pulmonary TB disease to cough.<sup>9</sup>

All cough-inducing and aerosol-generating procedures e.g. induced sputum, nasopharyngeal aspiration should be performed using environmental controls such as in a sputum induction booth/room or a Type 5 (respiratory isolation) room. Patients should be left in the booth/room or Type 5 (respiratory isolation) room until coughing subsides.<sup>4</sup>

Sputum collection from ambulant patients can occur outdoors away from others. Pathology providers or clinicians should provide specific instructions to patients on how to collect a good sputum sample in a safe manner.<sup>10</sup> Private enclosed spaces, e.g. toilets, specimen collection centres, are not adequately ventilated and are potentially dangerous locations for specimen collection.

Another patient or healthcare worker should not be allowed to enter the booth or the Type 5 (respiratory isolation) room until enough time has passed for a sufficient number of air changes to occur for adequate removal of *M. tuberculosis* contaminated air.<sup>7</sup> Consult with your facility plant engineers to determine the air changes per hour for each airborne infection isolation room.<sup>7</sup>

### Personal protective equipment

All staff should wear a correctly fitted P2/N95 respirator mask\* prior to entering the patient-care area when an airborne transmissible infectious agent is known or suspected.<sup>4</sup> If the patient is ventilated, a filter must be present on the expiratory circuit.<sup>7</sup> Standard Precautions are to be adhered to in addition to transmission-based airborne precautions.

### Masks and respirators

Surgical masks are designed to stop droplet nuclei from being generated from exhaled respiratory particles by the person wearing them when they breathe, talk, cough, or sneeze. In the absence of a surgical mask or effective cough etiquette, droplet nuclei form when larger droplets desiccate in the ambient environment following expulsion by cough or other circumstances as mentioned. Persons who are suspected or confirmed of having infectious TB may be given a surgical mask to wear to prevent them from expelling infectious

\* P2 is an Australian and New Zealand classification, and N95 North American. Both devices are correctly referred to as particulate respirators. They filter >95% of airborne particles. Due to their appearance they are commonly called "masks."

droplet nuclei<sup>2</sup> when they are outside of a negative pressure room. It is unnecessary for a patient to wear a P2/N95 mask.<sup>7</sup>

Masks (P2/N95) are designed to protect healthcare workers and other individuals from inhaling droplet nuclei. This can protect these individuals from becoming infected with *M. tuberculosis* when in contact with a person with infectious TB.<sup>4</sup>

In order for a P2/N95 mask to offer the maximum desired protection it is essential that the wearer is properly fitted and trained in its safe use. Healthcare facilities should ensure that they have a respiratory protection program that regularly evaluates the risk to which healthcare workers are exposed and determines which employees are required to undertake fit testing.<sup>4</sup>

Considerations when using a P2/N95 mask include:

- masks should not be touched while being worn;
- masks should be changed when they become moist;
- masks should never be reapplied after they have been removed;
- masks should not be left dangling around the neck; and
- hand hygiene should be performed upon touching or disposing of a used mask.<sup>4</sup>

Healthcare workers who have facial hair (including a 1–2 day beard growth) must be aware that an adequate seal cannot be guaranteed between the P2/N95 mask and the wearer's face.<sup>4</sup>

### Fit checking

Healthcare workers must perform fit checks every time they put on a P2/N95 mask to ensure it is properly applied. No clinical activity should be undertaken until a satisfactory fit has been achieved.<sup>4</sup> Fit checks ensure the mask is sealed over the bridge of the nose and mouth and that there are no gaps between the mask and face. Healthcare workers must be informed about how to perform a fit check.<sup>2</sup>

The procedure for fit checking includes:

- placement of the mask on the face;
- placement of the headband or ties over the head and at the base of the neck;
- compressing the mask to ensure a seal across the face, cheeks and the bridge of the nose;

- checking the positive pressure seal of the mask by gently exhaling. If air escapes, the mask needs to be adjusted; and
- checking the negative pressure seal of the mask by gently inhaling. If the mask is not drawn in towards the face, or air leaks around the face seal, readjust the respirator and repeat process, or check for defects in the respirator.<sup>4</sup>

The manufacturer's instructions for fit checking of individual brands and types of a P2/N95 mask should be referred to at all times.<sup>4</sup>

### Fit testing

Fit testing is a qualitative or quantitative method that is used to evaluate the fit of a specific make, model and size of mask on an individual<sup>12</sup> and to ensure that it is worn correctly. It also provides an opportunity to ensure healthcare workers are properly trained in the correct use of the mask.<sup>4</sup>

The National Health and Medical Research Council *Australian Guidelines for the Prevention and Control of Infection in Healthcare, 2010*<sup>11</sup> state a risk management approach should be applied and that fit testing should be performed at the commencement of employment for employees who will be working in clinical areas where there is a significant risk of exposure to infectious agents transmitted via the airborne route: assessment of the significance of risk will involve consideration of the location and activities to be undertaken. In the context of tuberculosis, a risk assessment should pay particular attention to factors which heighten:

- a. the risk of transmission – duration of anticipated exposure, smear status, aerosol generating procedures, pre-test probability of TB as a cause of undiagnosed respiratory infection; and
- b. the consequences of transmission – antimicrobial resistance, host impairment of healthcare worker.

The optimal frequency of fit-testing has not been determined although the Australian standard AS1715:2009 recommends annual testing. Re-testing may be indicated if there is a change in facial features of the wearer, or a change in the availability of a model or size of the initially assigned P2 mask.<sup>12</sup> Fit testing should be considered if a seal cannot be obtained or easily recognised with a given model of P2/N95 mask even if the overall risk is considered to be low. There is no published evidence to indicate that nosocomial transmission of TB occurs less frequency when fit testing is implemented compared with when it is not.

## Transfer of patients

If transfer of the patient outside the negative pressure room is necessary, e.g. to attend radiology, the patient should be asked to wear a correctly fitted surgical mask while they are being transferred and to follow respiratory hygiene and cough etiquette.<sup>4</sup> It is unnecessary for a patient to wear a P2/N95 mask.<sup>7</sup> The majority of young children are not infectious, and therefore, would not need a mask; however this decision should be done in consultation with a TB specialist.

## Visitors

Close household contacts should be assessed for active tuberculosis prior to visiting the facility. Children should be discouraged from visiting infectious patients. Close household contacts should wear the same PPE as hospital staff during patient visits. People who are vulnerable for disease following TB infection e.g. preschool children and the immunosuppressed, should not visit. Exceptional circumstances may include breast feeding and each situation should be considered individually. Visitors other than close household contacts should be discouraged from visiting. If visiting, they should be counselled about their risk and they should wear a P2/N95 mask with good fit characteristics. Instruction should be given on how to perform a fit check.<sup>7</sup> This should include a demonstration of donning, removing and disposing of PPE as required, as well as hand hygiene.<sup>7</sup>

## Cleaning

*M. tuberculosis* is usually transmitted only through air, not by surface contact.<sup>4</sup> Routine environmental cleaning with a facility's standard cleaning product should be sufficient for cleaning the room. The room door must remain closed and negative airflow maintained after patient discharge until all air in the room has been replaced; this will vary based on the number of room air changes per hour. Consult facility plant engineers to determine the air changes per hour for each airborne infection isolation room.<sup>7</sup>

Staff responsible for cleaning the room will need to use appropriate PPE including a P2/N95 mask while performing cleaning if this occurs before the required number of air changes have occurred. Once the room has been thoroughly cleaned and a sufficient number of air changes have occurred the room may be used for subsequent patients.

## Bronchoscopy

Bronchoscopy can result in the transmission of *M. tuberculosis* either through the airborne route or

via a contaminated bronchoscope.<sup>13,14</sup> If active TB is suspected or part of a differential diagnosis, then sputum collection spontaneously or by induction is a preferred test before bronchoscopy. In the case of confirmed TB, bronchoscopy should be postponed, if at all possible, until treatment has rendered the patient noninfectious.

Bronchoscopy suites should be under negative pressure and have the same minimum number of air exchange and air exhaust provisions as a Type 5 isolation room.<sup>8</sup> If it is necessary to perform bronchoscopy, this should be the last procedure of the day otherwise sufficient time should be allowed for adequate air exchange prior to the next procedure. Meticulous and detailed cleaning and high level disinfection by staff properly trained in bronchoscope reprocessing is the best defence against transmission of mycobacterial infection by flexible bronchoscopy. Australian guidelines for cleaning and microbiological monitoring of bronchoscopes should be followed.<sup>15</sup>

## Cessation of respiratory isolation precautions

It is recommended that patients with suspected or confirmed pulmonary TB who are admitted to hospital, should remain isolated in a negative pressure room with airborne precautions applied until criteria are met. In principle these criteria should include:

- a reduction in or absence of cough;
- reduced smear burden or smear negativity;
- assured treatment by direct observation; and
- an appropriate discharge plan.<sup>5</sup>

If drug resistance is suspected then cases should remain in isolation with airborne precautions in place until susceptibility results are confirmed. If sputum remains smear positive, a decision about hospital discharge should be made in consultation with a specialist physician with experience in managing TB and taking into account the social circumstances at home, such as the potential to expose new contacts and the presence of children under 5 years of age.

Patients with pulmonary TB who are managed at home should be isolated until assessed as being at minimal risk of transmitting infection. Adequate social support and supervised therapy is essential in the home environment to maintain home isolation. Assessment of other family members should be undertaken as a matter of priority to determine their status and also the possible need for preventive therapy in any children under 5 years of age with no initial evidence of infection. The patient

and family must also be provided with appropriate education and counselling about minimising the risk of transmission of infection; cough hygiene, avoiding new contacts and restricting movements away from home.

## Cohorting

It is not recommended that patients with TB are cohorted as there is a risk of cross transmission of different strains between patients. This is of particular concern where strains with drug resistance, including multidrug resistant tuberculosis may be present.

## Bacille Calmette-Guérin vaccination

Generally, bacilli Calmette-Guérin (BCG) vaccination is not recommended for healthcare workers although may be considered where there is a high risk of exposure to drug resistant tuberculosis and BCG vaccination is not otherwise contraindicated.<sup>16</sup> BCG vaccination should be given in accordance with the most current edition of *The Australian Immunisation Handbook*<sup>17</sup> (<http://www.immunise.health.gov.au/>)

## Management of healthcare workers and students with tuberculosis

If a healthcare worker or student is diagnosed as having infectious TB and was infectious while at work the healthcare facility should consider convening an expert incident management team to:

- determine the infectiousness of the healthcare worker/student;
- determine the dates the healthcare worker/student was in the facility and infectious;
- determine the areas of the facility that the healthcare worker/student was during the infectious period;
- determine if staff and/or patients need to be contact traced; and
- if required, designate responsibility for contact tracing and screening.

Issues relating to healthcare workers and tuberculosis are addressed in greater detail in the following National Tuberculosis Advisory Committee *Guideline: Management of Tuberculosis Risk in Health Care Workers in Australia* (currently unpublished).

## Contact tracing in hospitals

Contact tracing in hospitals should be undertaken in accordance with legislative requirements and should be in conjunction with the appropri-

ate tuberculosis control unit (state or regional). Facilities should ensure that roles and responsibilities between themselves and TB control units are clearly defined in regards to contact tracing and screening within the facility.

This should include designating:

- an appointed position or unit within the facility to be the designated contact for confirmed tuberculosis case notification. These notifications may come directly from the laboratory, from the treating team or via the tuberculosis control unit;
- an appointed position or unit to be responsible for collating a list of contacts including staff, patients and visitors;
- the responsibility for assessing the contacts;
- the responsibility for conducting contact screening of identified contacts as appropriate; and
- a mechanism for documenting and reporting the outcomes of a contact screening investigation.

## Glossary of terms

<b>Fit checking</b>	A procedure that the healthcare provider must perform each time a P2/N95 respirator is worn to ensure it fits the wearer's face correctly to provide adequate respiratory protection. The healthcare provider must receive training on how to perform a seal-check correctly.
<b>Fit testing</b>	A qualitative or quantitative method to evaluate the fit of a specific make, model and size of respirator on an individual.
<b>HEPA filter</b>	High efficiency particulate air filter with an efficiency of 99.97% in the removal of airborne particles 0.3 microns or larger in diameter.
<b>Latent TB infection (LTBI)</b>	Refers to the condition when a person is infected with tubercle bacilli but has not developed TB disease. Persons with LTBI carry the organism that causes TB but do not have TB disease symptoms and they cannot spread TB to others.
<b>Non-tuberculous mycobacteria</b>	Mycobacteria that do not cause TB disease and are not usually spread from person to person; one example is <i>Mycobacterium avium</i> complex.

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# REVISED SURVEILLANCE CASE DEFINITIONS

This report provides the revised surveillance case definitions approved by the Communicable Diseases Network Australia (CDNA) since 1 July 2016.

The Case Definitions Working Group (CDWG) is a subcommittee of the CDNA and comprises members representing all states and territories, the Australian Government Department of Health, the Public Health Laboratory Network, OzFoodNet, the Kirby Institute, the National Centre for Immunisation Research and Surveillance and other communicable disease experts. CDWG develops and revises surveillance case definitions for all diseases reported to the National Notifiable Diseases Surveillance System. Surveillance case definitions incorporate laboratory, clinical and epidemiological elements as appropriate.

The following case definition has been reviewed by CDWG and endorsed by CDNA.

The Shiga toxin-producing *Escherichia coli* case definition was implemented on 1 July 2016 and supersedes any previous versions.

## Shiga toxin-producing *Escherichia coli* (STEC)

### Reporting

Only confirmed cases should be notified.

#### Confirmed case

A confirmed case requires laboratory definitive evidence only.

#### Laboratory definitive evidence

1. Isolation of Shiga toxigenic *Escherichia coli* from faeces

OR

2. Detection of the gene(s) encoding the Shiga toxins (stx1 and/or stx2) in faeces or from a clinical isolate of *Escherichia coli*.

Note: Where STEC is isolated or detected in the context of haemolytic uraemic syndrome (HUS), it should be notified as STEC and HUS.

<p><b>Summary of changes to STEC surveillance case definition</b></p>	<p><b>Title and throughout</b> Removal of vero toxin-producing <i>Escherichia coli</i> (VTEC).</p> <p><b>Laboratory definitive evidence</b> Removal of 'isolation of Shiga toxin or vero toxin from a clinical isolate of <i>Escherichia coli</i>'. Replacement of 'raw bloody diarrhoea' with 'faeces' for detection of genes encoding Shiga toxins.</p>
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## Annual report

# CREUTZFELDT-JAKOB DISEASE SURVEILLANCE IN AUSTRALIA: UPDATE TO DECEMBER 2015

Genevieve M Klug, Alison Boyd, Shannon Sarros, Christiane Stehmann, Marion Simpson, Catriona McLean, Colin L Masters, Steven J Collins

### Abstract

Nation-wide surveillance of human transmissible spongiform encephalopathies (also known as prion diseases), the most common being Creutzfeldt-Jakob disease, is performed by the Australian National Creutzfeldt-Jakob Disease Registry, based at the University of Melbourne. Prospective surveillance has been undertaken since 1993 and over this dynamic period in transmissible spongiform encephalopathy research and understanding, the unit has evolved and adapted to changes in surveillance practices and requirements concomitant with the delineation of new disease subtypes, improvements in diagnostic capabilities and the overall heightened awareness of prion diseases in the health care setting. In 2015, routine national surveillance continued and this brief report provides an update of the cumulative surveillance data collected by the Australian National Creutzfeldt-Jakob Disease Registry prospectively from 1993 to December 2015, and retrospectively to 1970. *Commun Dis Intell* 2016;40(3):E368–E376.

**Keywords:** Creutzfeldt-Jakob disease, prion disease, transmissible spongiform encephalopathy, disease surveillance

### Introduction

In 1993, the Allars' inquiry<sup>1</sup> into the use of cadaver-derived pituitary hormones under The Australian Human Pituitary Hormone Program and the association with 4 medically acquired (iatrogenic) Creutzfeldt-Jakob disease (CJD) deaths recommended broadening of the responsibilities of the nascent Australian surveillance unit while monitoring for further cases of iatrogenic CJD in Australia. The Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) was established in October 1993 at the University of Melbourne. The monitoring of further Australian iatrogenic CJD cases related to cadaveric pituitary hormone treatment for infertility or short stature and contaminated dura mater grafts remains one of the core objectives of the ANCJDR. However, the ANCJDR's activities have evolved to encompass the surveillance of all types of CJD, including sporadic, genetic and variant CJD and other transmis-

sible spongiform encephalopathies (TSE) or prion diseases such as Gerstmann-Sträussler-Scheinker syndrome and fatal familial insomnia.

As described previously,<sup>2</sup> human prion disease can arise sporadically or from genetic or iatrogenic aetiologies. Detailed evaluation of each suspected case added to the register is undertaken to determine whether a case can be excluded from suspicion or classified as a definite, probable or possible prion disease case according to World Health Organization (WHO) diagnostic criteria.<sup>3</sup> CJD was made a notifiable disease in all states and territories of Australia as of June 2006. Most initial notifications to the ANCJDR arise through diagnostic testing available through the Registry and this occurs prior to health department notification.

The global incidence of CJD is commonly reported to be 1 case per million per year but in most countries with long-standing surveillance systems in place such as France and Switzerland, annual incidence rates have been consistently reported above this quoted figure.<sup>4</sup> Incidence rates as high as 2.4 to 2.6 cases per million per year have been reported.<sup>4</sup> Temporally, human prion disease incidence rates have increased in most countries, including Australia, as surveillance mechanisms evolved and diagnostic testing capabilities improved, in parallel with a generally greater awareness of this rare disease in the health care setting.

In 2015, national surveillance of prion disease continued, influenced positively by the restoration of routine autopsy services in New South Wales and Queensland. This has led to increased case classifications in 2015 and overall, a return to more usual annual incidence rates of prion disease in Australia. In this report, updated surveillance figures to 31 December 2015 are provided for all retrospective (to 1970) and prospective (from 1993) cases ascertained, including discussion on case notifications, classifications and overall incidence.

### Methods

Patients with a suspected human prion disease are prospectively notified to the ANCJDR predominantly through referral for diagnostic cer-



cerebrospinal fluid (CSF) 14-3-3 protein detection. Other mechanisms include or have included personal communications from clinicians, families, hospitals and CJD-related groups, as well as health record searches through hospitals or health departments. Once notified to the ANCJDR, referrals are assessed and if the suspicion of prion disease is supported, the case will be added to the register as a formally notified suspected case for continued investigation with the aim of exclusion or classification according to WHO diagnostic criteria. Investigation of register cases can be prolonged as the ANCJDR requires next-of-kin consent to access and compile the appropriate clinical information from various health information sources for comprehensive evaluation. Response times can vary as the information can be extensive or sources numerous. Medico-demographic questionnaires are offered and forwarded to families if they are willing to contribute, providing valuable information for analysis and evaluation.

The classification of register cases remains as 'incomplete' until all known available information is gathered and reviewed or a definitive result from neuropathological assessment is obtained. Cases may be excluded from the register on the basis of neuropathological examination or after thorough clinical evaluation. A 'definite' classification requires brain tissue examination, including immunohistochemically and 'probable' and 'possible' cases are reliant on specific clinical profile and diagnostic test outcomes being met as previously described.<sup>3</sup> In this report, the total number of confirmed prion disease cases includes those that have been classified as definite or probable cases during 2015.

In conjunction with the ANCJDR's surveillance responsibilities, the registry provides diagnostic platforms for ante- and post-mortem diagnostic testing for human prion diseases. The testing of CSF for the presence of a family of low molecular weight proteins called '14-3-3' is performed weekly by the ANCJDR. This test, first introduced in 1997, has been readily utilised by the health community and referrals have increased substantially since its introduction to more than 400 referrals each year. As described previously, the test provides an increasingly larger proportion of initial notifications of suspected human prion disease to the ANCJDR each year. The ANCJDR also undertakes Western blot analysis for misfolded, protease-resistant prion protein in tonsil and brain tissue from biopsies or autopsies to supplement immunohistochemical assessment. Previously, the ANCJDR performed prion protein gene testing as appropriate. However from 1 September 2015, this service was ceased

and is now undertaken by external, independent providers. The ANCJDR actively promotes all diagnostic tests so that these options are available to clinicians and families to achieve the most accurate diagnosis and classification of persons suspected to have prion disease.

Annual human prion disease incidence rates are calculated using direct age-standardisation, based on the Australian Bureau of Statistics 1970 to 2015 estimated resident population for Australia and for each state and territory<sup>5-13</sup> and standardised to 2000 population estimates.<sup>14</sup> Population based rates of post-mortem examination in suspected human prion disease were calculated using the Australian Bureau of Statistics 1993 to 2015 estimated resident population for specific states and territories.<sup>5-12</sup> Health information is collected through a combination of public health and surveillance responsibilities, based on the national notification of communicable diseases. ANCJDR surveillance activities for the period reported were approved by The University of Melbourne Human Research Ethics Committee.

Statistical analysis (Log-Rank test) was performed using Stata (Intercooled Stata 7, Stata Corporation, College Station, TX).

## Results

Sixty-six persons with suspected human prion disease were added to the CJD surveillance register in 2015. Cases were initially notified via request for CSF 14-3-3 protein testing (53 cases), the CJD Support Group network (6 cases), personal communication from clinicians (4 cases), a coronial referral (1 case), funeral director communication (1 case) and the Victorian Brain Bank Network (1 case). The proportions of the initial notification sources of the 66 cases are consistent with those in previous years and the overall trends for all register cases (Table 1).

Of the 66 cases that were added to register in 2015, 3 cases were known to the ANCJDR prior to 2015 via the CSF 14-3-3 protein test. At the time of referral for diagnostic CSF testing, these 3 cases were not added to the register due to a low level of suspicion for prion disease after assessment. Further information ascertained in 2015 increased the likelihood of prion disease resulting in formal notification and addition of the cases to the register. The number of case additions to the register in 2015 is lower than the previous year (76 cases) but consistent with the previous 10-year average for the years 2004 to 2014 (66 cases).

By state and territory, only modest fluctuations in the number of suspected case notifications com-

**Table 1: Source of initial notification of suspected prion disease cases ascertained between 1993 and 2015**

Method	Register cases* (%)	Cases removed from the register† (%)	Overall
CSF 14-3-3 protein test request (Since September 1997)	54.4	50.4	52.8
<b>Personal communications</b>			
Neurologists	13.0	12.0	12.6
Neurologists (mail-out reply cards)	2.4	1.7	2.2
Neuropathologists	7.6	8.6	8.0
Neuropathologists (mail-out reply cards)	0.6		0.3
Pituitary Hormones Task Force	1.7	3.0	2.2
Family	2.8	2.4	2.7
Funeral directors	0.1		0.1
Molecular biologist	0.1		0.1
Hospital	0.5	1.4	0.9
<b>Hospital and health department searches</b>			
Death certificates	9.0	5.3	7.5
Hospital medical records	3.0	7.5	4.7
Health department search/state morbidity data	1.3	3.4	2.1
Direct health department notification	1.5	0.3	1.0
CJD Support Group	0.7	0.4	0.6
Combined CSF/genetic test request	0.3	0.9	0.5
Genetic test request	0.3	1.6	0.8
CJD Counselling Service	0.2	0.6	0.3
Victorian Brain Bank network	0.2	0.1	0.2
Coroner's post-mortem request	0.1	0.4	0.2
Press	0.1		0.1
UK Surveillance Unit	0.1		0.1
	100.0	100.0	100.0

\* Registry cases; includes all cases currently on the register as classified cases or cases still under investigation.

† Cases removed by the registry; includes all suspected cases excluded from the register after detailed investigation including neuropathological investigation.

CSF Cerebrospinal fluid.

pared with the previous year were observed in 2015 (Figure 1). Since 2012, the number of suspected case notifications from Western Australia was lower than the 1993–2014 long-term average (8 cases per year). This trend continued in 2015, although not as noticeably as in the previous 3 years.

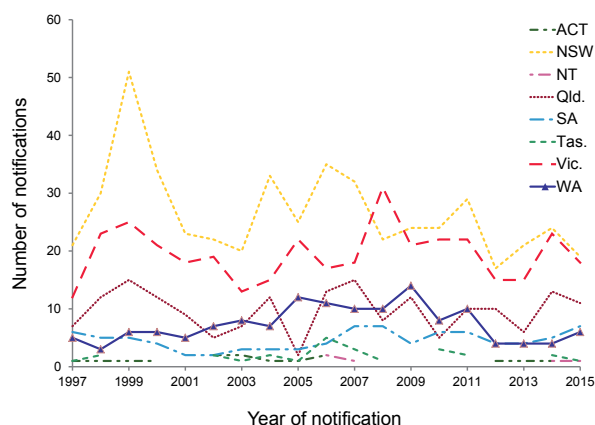
As of 31 December 2015, the majority of the 66 suspected cases added to the register in 2015 were classified as incomplete (43 cases). Eight cases were excluded by either detailed clinical follow-up (1 case) or neuropathological examination (7 cases); 12 cases were classified as definite and 2 as probable prion disease. The remaining suspect case added to the register in 2015 was initially treated in Australia; however, the patient subsequently returned overseas and was therefore

unable to be investigated further. This person was thereby excluded from the overall analysis of Australian prion disease cases.

Excluding the prion disease-related post-mortem rate in 2015, wherein figures are still provisional, the average proportion of suspected prion disease cases on the register and who died between 1993 and 2014 and underwent post-mortem examination is 61%. Over this period, this proportion has steadily increased from 38% in 1993 to a peak of 80% in 2008. Since 2008, the proportion has stabilised at around 65%.

Based on the Australian population, the average crude rate of prion disease-related post-mortems between 1993 and 2015 is 1.4 post-mortems per

**Figure 1: Prospective notifications of suspected prion disease cases to the Australian National Creutzfeldt-Jakob Disease Registry, 1997 to 2015, by state or territory and year**



million per year (range, 0.6 to 2.0), which is considerable given prion disease is particularly rare. By state and territory and for the same period, the lowest rates of suspected prion disease post-mortems performed annually were in the Australian Capital Territory, Tasmania and the Northern Territory (0.7, 1.0 and 0.9 per million per year, respectively) while the highest rates were in Victoria and New South Wales (1.6 per million per year). Despite the smaller populations in Tasmania, the Northern Territory and the Australian Capital Territory, the post-mortem rates are not substantially lower than the rates of more populous states and provide a level of confidence that suspected case deaths in these states and territories have a similar likelihood of undergoing post-mortem examination.

In New South Wales and Victoria, there has been an overall temporal increase in post-mortem rates between 1993 and 2015 (Figure 2a, 2b). Previously, the rate of prion disease-related post-mortems in New South Wales was reported to have declined sharply in 2014, which was related to the deferral of analyses by neuropathological laboratory services during this time. As anticipated, upon completion of these analyses in 2015, post-mortem rates for 2014 returned to an expected level in New South Wales.

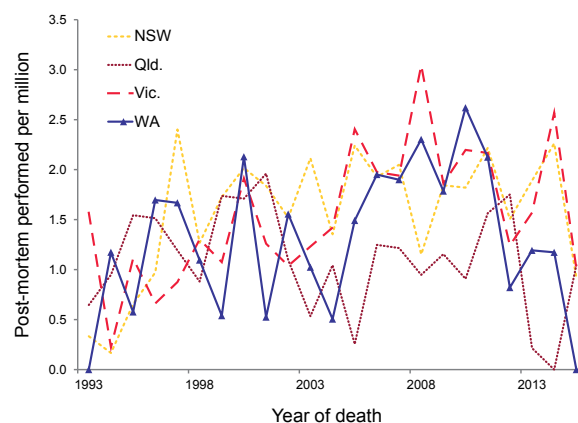
In Queensland, South Australia and Western Australia, variability in post-mortem rates has been observed, especially in recent years. In Queensland, the post-mortem rates in 2013 and 2014 were substantially diminished (0.2 and 0.0 post-mortems per million per year respectively) compared with the long-term average of 1.2 post-mortems per million per year between 1993 and 2012. This was directly related to changes to routine autopsy services in this State during 2013 and 2014. In 2015, 5 post-mortems were completed and

the post-mortem rate returned to expected levels (1.0 post-mortem per million per year) (Figure 2a). In South Australia and Western Australia, a sustained decrease in the post-mortem examination rate has been observed since 2010–2011. In both states, there were a number of suspected prion disease deaths in 2014 and 2015, where neuropathological examination remains pending. Once finalised, the post-mortem rates for these years is predicted to return to an expected level but will not change the lower rates in 2012 and 2013.

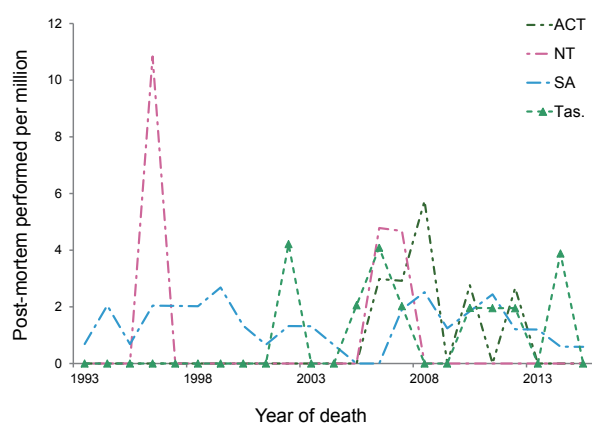
As of 31 December 2015, there were 1,092 cases on the register with 817 of these being classified as probable or definite prion disease cases. An additional definite iatrogenic case who was treated

**Figure 2: Rates of post-mortem examination\* in prion disease suspected case deaths per million population, by state and territory and year†**

a



b



\* Post-mortem examination rates were calculated using the Australian Bureau of Statistics 1993 to 2015 estimated resident population for Australia for each state and territory.

† Cases with neuropathology examination results pending are not included in the analyses.

in Australia, and died in the United Kingdom is included in Table 2. However this case is not classified as an Australian case due to the location at death and is thereby excluded from the overall statistical analysis of Australian prion disease cases. Since the start of surveillance, 699 suspected prion disease cases have been excluded from the register after detailed follow-up, with 21 of these being excluded in 2015 (16 after neuropathological examination).

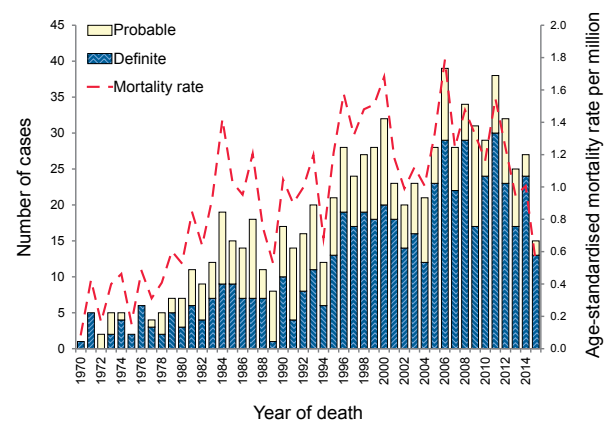
In 2015, 28 cases were re-classified from incomplete to definite prion disease and 8 cases to probable prion disease and there were no further cases of possible prion disease classified. The total number of possible cases remains at 15 of which 14 were sporadic and 1 iatrogenic CJD (Table 2). Of the 259 incomplete cases, 142 are presently alive. In 2015, the total number of incomplete cases (259) under evaluation was only marginally higher than the number in 2014 (251 cases) but still remains significantly higher than the number in 2012 (214 cases) and 2013 (216 cases).

Age-standardised mortality rates show that the rate of human prion disease mortality in Australia during the period of 1970 to 2015 is generally increasing, with the exception of 2015, where case evaluation is pending for the majority of deaths (Figure 3) and incidence is therefore provisional. In 2015, the age-adjusted mortality rate was 0.5 deaths per million per year and this would be expected to increase after further investigation and classification of incomplete cases. The mean annual age-adjusted mortality rate during the period from 1970 to 2014 was 1.0 death per million (range, 0.1 to 1.8). For the prospective surveillance period of 1993 to 2014, the mean annual rate is 1.2 deaths per million (range, 0.7 to 1.8). By state and territory, the majority of regions in Australia have a mean age-adjusted mortality rate above 1 case per million per year between 1993 and 2014 (range, 1.0 to 1.5). The exceptions are Tasmania

and the Northern Territory both with 0.7 deaths per million per year. Restriction of the surveillance data to the period between 2003 and 2014 allows comparisons between states and territories during a time-frame of relatively consistent surveillance practices, diagnostic capabilities and utility with the exception of MRI diagnostics (Table 3). During this period, Tasmania, the Northern Territory and Queensland have lower than expected mean mortality rates, while Western Australia and Victoria have the highest prion disease mortality in Australia.

The proportions of human prion disease aetiologies represented on the register have remained similar to previous years (Figure 4). Previously we have reported that the annual number of genetic prion disease cases had declined in recent years<sup>2</sup> although this changed with the classification of 6 confirmed

**Figure 3: Number of definite and probable prion disease cases and age-standardised mortality rate,\* Australia, 1970 to 2015, by classification and year**



\* Age-standardised mortality rates were calculated using the Australian Bureau of Statistics 2000 estimated resident population for Australia.

**Table 2: Classification of Australian National Creutzfeldt-Jakob Disease Register cases, Australia, 1970 to 2015**

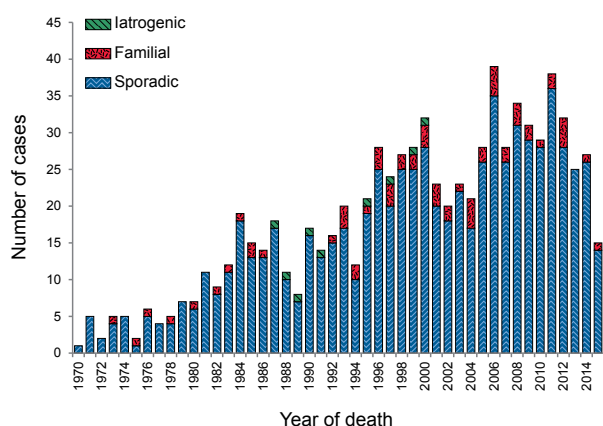
Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total
Definite	490	51	5*	0	0	546
Probable	256	12	4	0	0	272
Possible	14	0	1	0	0	15
Incomplete					259 <sup>†</sup>	259
Total	760	63	10	0	259	1,092

\* Includes 1 definite iatrogenic case who received pituitary hormone treatment in Australia but disease onset and death occurred while a resident of the United Kingdom. This case is not included in statistical analysis since morbidity and mortality did not occur within Australia.

† Includes 142 living cases.

genetic prion disease cases during 2013 and 3 in 2015. Overall, the vast majority of human prion disease cases are sporadic (91%) while genetic and iatrogenic cases represent 8% and 1% respectively, of all definite and probable cases.

**Figure 4: Definite and probable human prion disease cases, 1970 to 2015, by aetiology and year**



Based on 817 definite and probable human prion disease cases, 54% were female. Similar proportions for gender exist for all human prion disease aetiologies. Median ages at death for the overall case group or by specific aetiology are largely unchanged from the previous reporting period. Sixty-seven years is the median age at death for all cases overall and only a single year difference

between males (66 years) and females (67 years). For sporadic cases, 67 years is the median age at death both overall and for both males and females. For genetic prion disease, there is a 4 year age difference between males (58 years) and females (62 years) and overall the median age of death from genetic prion disease is 61 years. As there have been no further iatrogenic cases identified since the last reporting period at 31 December 2014, there has been no change to the previously reported median age at death for iatrogenic cases.<sup>2</sup>

Duration of illness is typically short for human prion disease, especially sporadic CJD, with the median length of illness duration for all cases combined being 4 months. By aetiology, median duration was found to be 3.7 months for sporadic cases (range, 0.9 to 60 months), 6.3 months for iatrogenic cases (range, 2 to 25 months) and 6 months for genetic cases (range, 1.3 to 192 months). Within 6 months of disease onset, 70% of all prion disease cases were deceased. By aetiology, 72% of sporadic, 51% of genetic and 56% of iatrogenic human prion disease were deceased 6 months after the onset of symptoms. Survival is significantly shorter in sporadic CJD than the genetic form ( $P < 0.0001$  by Log Rank Test).

Between 1 January and 31 December 2015, no variant CJD or further iatrogenic prion disease cases were identified in Australia. The most recent human-derived pituitary gonadotrophin-related CJD death occurred in 1991, while the most recent Lyodura-related CJD death occurred in 2000.

**Table 3: Prion disease deaths and age-adjusted mortality rates, 2003 to 2015, by year and state or territory**

Year	03	04	05	06	07	08	09	10	11	12	13	14	15*	Total	Mean age-adjusted mortality rate† (deaths/million/year)
ACT		1		1		2		1			1			6	1.3
NSW	7	11	10	12	10	6	11	5	14	7	11	11	6	121	1.3
NT				2	1								1	4	0.8
Qld	3			7	2	4	4	2	5	6	3		5	41	0.6
SA	1	2	1	1	3	5	2	4	4	2	2	1	2	30	1.3
Tas.			1	2					1	1		2		7	0.9
Vic.	9	5	11	10	6	13	9	13	9	13	6	11	1	116	1.7
WA	3	2	5	4	6	4	5	4	5	3	2	2		45	1.5
Aus.	23	21	28	39	28	34	31	29	38	32	25	27	15	370	1.3

\* Provisional figures.

† Age-standardised mortality rates (2003-2014) were calculated using the Australian Bureau of Statistics 2000 estimated resident population for Australian states and territories.

## Discussion

In 2015, the number of suspected prion disease notifications was consistent with the long-term average for the previous 10 years of surveillance (2004 to 2014). This was in contrast to 2012 and 2013, when reduced numbers of notifications were attributed to several possible factors including the temporary changes to the Queensland suspected prion disease autopsy service, changes to the approach to adding cases to the register for investigation by the ANCJDR and natural fluctuations.

By state and territory, only modest fluctuations in the number of suspected case notifications compared with the previous year were observed in 2015. The number of notifications of suspected cases in Western Australia in 2015 continued to be lower than the numbers observed prior to 2012, but not as significantly as the previous 3 years. Sizeable relative fluctuations are not surprising with annual CJD notifications given the small absolute case numbers involved. However, it should be noted that since 2009, notifications have been consistently declining in Western Australia. Previous evidence that elevated CSF referrals correspond with elevated suspected prion disease notifications<sup>15</sup> led to speculation that lower CSF referrals may be influencing this downward trend in suspected case notifications. CSF referrals from Western Australia have increased annually since the test's introduction in 1997 to a peak level in 2012. Since 2012, referrals appeared to be trending downward but overall were consistent with pre-2012 levels. The exception was in 2014 where there was a marked decline in CSF referrals. This may explain the lower notifications of suspected cases in 2014 although it does not explain the lower suspect case notifications that have been observed for the remaining years with lower notifications since 2012. As previously discussed, Western Australian health services are relied upon to manage case investigations following notifications and manage autopsy referrals. Changes to the role of the ANCJDR in Western Australia during these years may limit the ANCJDR's capacity to ascertain the true level of clinical suspicion for CJD, which may have contributed to a reduced number of formal notifications and subsequently, confirmed cases reported by the ANCJDR. The ANCJDR in partnership with the Western Australian Department of Health will continue working towards optimal prion disease ascertainment in this State.

The proportion of prion disease-related post-mortems being performed in suspected prion disease cases remains high (61% of all case deaths between 1993 and 2014). This contrasts with the findings of an Australian healthcare setting survey where the national hospital post-mortem rate was

12% in 2002 to 2003<sup>16</sup> and more recently, a major Australian tertiary centre audit of hospital autopsy data was published and described an autopsy rate of 6.6% in 2011 to 2013.<sup>17</sup> The high suspected prion disease-related post-mortem proportion underpins the high and consistent number of confirmed Australian human prion disease cases recorded over the more recent time period and provides confident understanding of the cause of death in suspected cases ultimately determined as non-prion disease.

In recent years, changes to the routine autopsy services in both New South Wales and Queensland have impacted on the number and timing of post-mortems being completed. In January 2013, the Queensland autopsy service experienced difficulties with a reliable on-call service to perform brain-only autopsies greatly impacting the ability to achieve TSE post-mortem examinations. While the difficulties were temporary, the practical interruption remained in place until September 2014 and as a result, no autopsies were performed in Queensland in 2014. This contributed to significantly lower figures in Queensland compared with the 2 years prior, where 7 to 8 autopsies were completed per year. The routine service is now operational through the Royal Brisbane Hospital and in 2015, 5 post-mortem examinations were completed.

In New South Wales, the closure of the neuropathology laboratory for refurbishment extended the time required for reporting during 2013 and 2014, although this appears to have had little effect on formal suspected case notifications and CSF referrals for 14-3-3 testing during these years. Furthermore, incidence has remained consistent with levels prior to the laboratory closure. As expected, post-mortem rates slowed in 2014 due to reporting delays. These figures have returned to an expected level now that the laboratory is fully operational and there has been a concerted effort to finalise outstanding investigations during 2015.

The number of cases classified as definite and probable prion disease in 2015 (36 cases) was higher than the long-term average classified annually (28 cases) between 2004 and 2014. In comparison with the previous reporting period, more definite cases were classified in 2015 as expected due to the completion of outstanding post-mortem examinations. This has contributed to prion disease incidence in Australia re-aligning with previously observed levels, rather than diminishing. In 2015, the total number of incomplete cases under evaluation was only marginally higher than the number in 2014 but still remains significantly higher than the annual number prior to 2014. Although the high number of incomplete cases is not unprecedented, it does highlight the imbalance of new suspected

cases with fully evaluated cases with an outcome. In 2015, there have been signs of improvement to this imbalance despite the overall high number of incomplete cases. Compared with the longer-term average (2004 to 2014), an equivalent number of cases have been added to and removed from the register in 2015. Furthermore, the number of definite and probable cases classified during 2015 was 28% higher than the long-term average. This was in contrast to 2014, where increased numbers of cases were added to the register (compared with the long-term average) yet fewer cases were classified as either definite or probable prion disease and fewer were removed from the register as non-prion disease cases. This was predominantly attributable to the alteration of routine autopsy services in Queensland and New South Wales respectively during 2013 and 2014. In 2015, the resumption of routine autopsy services in New South Wales and Queensland led to a greater number of suspect cases classified as confirmed TSE or non-TSE within the current reporting period. Continued effort will be made to evaluate incomplete cases in 2016 to minimise the inflation of the incomplete case group.

## Acknowledgements

The ANCDJR wishes to thank families, as well as medical practitioners and associated staff for their generous support of Australian CJD surveillance. The ANCDJR also thanks Dr Handan Wand, Dr Matthew Law and Professor John Kaldor (The Kirby Centre at the University of New South Wales) for their expert ad hoc epidemiological and statistical support, as well as the CJD Support Group Network for their assistance in surveillance activities.

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# SURVEILLANCE OF ADVERSE EVENTS FOLLOWING IMMUNISATION IN AUSTRALIA ANNUAL REPORT, 2014

Aditi Dey, Han Wang, Helen E Quinn, Richard Hill, Kristine K Macartney

## Abstract

This report summarises Australian passive surveillance data for adverse events following immunisation (AEFI) for 2014 reported to the Therapeutic Goods Administration for 2014 and describes reporting trends over the 15-year period 1 January 2000 to 31 December 2014. There were 3,087 AEFI records for vaccines administered in 2014; an annual AEFI reporting rate of 13.2 per 100,000 population. There was a decline of 5% in the overall AEFI reporting rate in 2014 compared with 2013. This decline in reported adverse events in 2014 compared with the previous year was mainly attributable to fewer reports following the human papillomavirus (HPV) vaccine as it was the 2nd year of the extension of the National HPV Vaccination Program to males. AEFI reporting rates for most vaccines were lower in 2014 compared with 2013. The most commonly reported reactions were injection site reaction (27%), pyrexia (18%), rash (16%), vomiting (9%), headache (7%), and syncope (5%). The majority of AEFI reports described non-serious events while 7% (n=211) were classified as serious. There were 5 deaths reported with no clear causal relationship with vaccination found. *Commun Dis Intell* 2016;40(3):E377–E390.

Keywords: AEFI, adverse events, vaccines, surveillance, immunisation, vaccine

## Introduction

This report summarises national passive surveillance data for adverse events following immunisation (AEFI) reported to the Therapeutic Goods Administration (TGA) by 28 February 2015. The report focuses on AEFI reported for vaccines administered during 2014 and trends in AEFI reporting over the 15-year period 1 January 2000–31 December 2014.

An adverse event following immunisation is defined as any untoward medical occurrence that follows immunisation and that does not necessarily have a causal relationship with the usage of the vaccine.<sup>1</sup> The adverse event may be any unfavourable or unintended sign, abnormal laboratory finding, symptom or disease.<sup>1</sup>

Thus, AEFI may be caused by a vaccine(s) or may be coincidental. Adverse events may also include conditions that occur following the incorrect handling and/or administration of a vaccine(s). The post-marketing surveillance of AEFI is particularly important to detect signals of rare, late onset or unexpected events, which are difficult to detect in pre-registration vaccine trials.

Reports summarising national AEFI surveillance data have been published regularly since 2003.<sup>2–13</sup> Trends in reported adverse events following immunisation are heavily influenced by changes to vaccine funding and availability provided through the National Immunisation Program (NIP). These changes impact on the interpretation of trend data and have been described in detail in previous reports published regularly since 2003.<sup>2–13</sup> Table 1 shows the chronological listing of the changes.

Recent changes that impact on AEFI surveillance data presented in this report are:

- On 31 December 2013, the secondary school Year 7 hepatitis B vaccine catch-up program ceased.
- From January 2014, the hepatitis B vaccine was recommended to at-risk groups: household contacts and sexual partners of people living with hepatitis B; people who inject drugs or are on opioid substitution therapy; people living with hepatitis C; men who have sex with men; people living with HIV and prisoners and remandees.
- In February 2013, the National Human Papillomavirus Vaccination Program (quadrivalent HPV vaccine Gardasil®, CSL Biotherapies/Merck & Co. Inc.) was extended to males aged 12–13 years through the school-based program, including a 2-year catch-up program for males aged 14–15 years until the end of 2014.
- On 14 August 2013, TGA included Bexsero® (4CMenB) on the Australian Register of Therapeutic Goods.<sup>14</sup> The vaccine is registered for use in people  $\geq 2$  months of age for the prevention of invasive disease caused by serogroup B meningococci.<sup>14,15</sup> It is available through purchase on the private market.<sup>14,15</sup> This vaccine is not funded under the NIP.<sup>15</sup>

**Table 1: Changes to the Australian Standard Vaccination Schedule (2005–2014)<sup>2–14</sup>**

Year	Intervention
2014	4vHPV catch-up program for males aged 14–15 years
2013	<p>From 1 February 2013, 4vHPV was extended to males aged 12–13 years, delivered through a school-based program, with a catch-up program for males aged 14–15 years in 2013 and 2014.</p> <p>From July 2013, the 2nd dose of MMR vaccine, previously given at 4 years, was brought forward to 18 months of age and delivered as a combination MMRV vaccine.</p> <p>From July 2013, combined <i>Haemophilus influenzae</i> type b (Hib) and meningococcal serogroup C (MenC) vaccine, Menitorix®, was funded for infants aged 12 months. This combination vaccine replaced the single dose of monovalent meningococcal C conjugate vaccine (MenCCV) and booster dose of monovalent Hib vaccine previously scheduled at 12 months of age.</p> <p>At the end of December 2013, the secondary school Year 7 hepatitis B vaccine catch-up program ceased, as all younger age cohorts were eligible for infant immunisation under the NIP (commenced 2000).</p>
2012	From 1 October 2012, a 4th dose of Prevenar 13®, (13vPCV, a 13-valent pneumococcal conjugate vaccine) was listed on the National Immunisation Program (NIP) for Indigenous children, aged 12–18 months, residing in Queensland, South Australia, Western Australia and the Northern Territory. This replaced the booster dose of Pneumovax23®, (23vPPV, a 23-valent pneumococcal polysaccharide vaccine) administered between 18 and 24 months of age for Indigenous children from these jurisdictions.
2011	<p>From 1 July 2011, Prevenar 13® replaced Prevenar® on the NIP for children at 2, 4 and 6 months of age in all states and territories except the Northern Territory, which adopted 13vPCV from 1 October 2011.</p> <p>1 October 2011 to 30 September 2012 – all children aged between 12 and 35 months who had completed a primary pneumococcal vaccination course with 7vPCV, were eligible to receive a free supplementary dose of Prevenar 13®</p> <p>On 25 March 2011, TGA issued a recall of Batch N3336 of the 23 valent pneumococcal polysaccharide vaccine 23vPPV, Pneumovax® 23. April 2011: health professionals were advised not to administer a 2nd or subsequent dose of Pneumovax 23 vaccine. December 2011 - Revised recommendations regarding which patients should be re-vaccinated under the NIP were provided.</p>
2010	<p>Annual vaccination with seasonal trivalent influenza vaccine (TIV, containing 3 influenza strains: A/H1N1, A/H3N2 and B) was funded under the NIP for people aged ≥6 months with medical risk factors (previously subsidised through the Pharmaceutical Benefits Scheme) and all Indigenous people aged ≥15 years (previously all Indigenous adults ≥50 years and 15–49 years with medical risk factors).</p> <p>On 23 April 2010, the use of the 2010 seasonal TIV in children &lt;5 years of age was suspended by Australia's Chief Medical Officer due to an increased number of reports of fever and febrile convulsions post vaccination. A subsequent investigation identified that Fluvax® and Fluvax junior® (CSL Biotherapies), but neither of the other 2 available brands registered for use in young children, were associated with an unacceptably high risk of febrile convulsions. The recommendation to resume the use of seasonal influenza vaccine in children aged 6 months to 5 years, using brands other than Fluvax® and Fluvax junior®, was made in August 2010.</p>
2009	<p>By late 2009, all states and territories were using the single hexavalent DTPa-IPV-Hib-HepB (Infanrix hexa®) vaccine for all children at 2, 4 and 6 months of age, due to an international shortage of <i>Haemophilus influenzae</i> type b (Hib) (PedvaxHib® [monovalent] and Comvax® [Hib-HepB]) vaccines.</p> <p>Pandemic H1N1 2009 influenza vaccine (Panvax®) was rolled out across Australia from 30 September 2009 for people aged ≥10 years. From December 2009, the pandemic vaccine was made available to children aged 6 months to 10 years.</p>
2008	<p>Western Australia commenced a seasonal influenza vaccination program for all children aged 6 months to &lt;5 years (born after 1 April 2003).</p> <p>In March 2008, Queensland, South Australia and Victoria changed from using 2 combination vaccines (quadrivalent DTPa-IPV and Hib-HepB) to the single hexavalent DTPa-IPV-HepB-Hib vaccine.</p>
2007	<p>From April 2007, funded immunisation against human papillomavirus for all Australian girls aged 12–13 years delivered through a school-based program from April 2007, with a temporary catch-up program through schools or primary care providers for females aged 13–26 years until December 2009.</p> <p>From July 2007, universal funded immunisation against rotavirus at 2 and 4 months of age (Rotarix®) or at 2, 4 and 6 months of age (Rotateq®).</p>
2005	<p>From January 2005, universal funded infant 7-valent pneumococcal conjugate vaccine (7vPCV) program replaced the previous targeted childhood program, with a catch-up program for children aged &lt;2 years.</p> <p>Universal 23vPPV for adults aged ≥65 years replaced previous subsidy through the Pharmaceutical Benefits Scheme.</p> <p>From November 2005, universal funded immunisation against varicella at 18 months of age with a school-based catch-up program for children at 10–13 years of age not previously vaccinated and without a history of varicella infection (no funded catch-up for children 2–10 years of age).</p> <p>Inactivated polio vaccine was funded to replace the oral polio vaccine, in combination vaccines.</p>

A glossary of the abbreviations of the vaccines is included at the end of this report to assist readers.

## Methods

AEFI are notified to the TGA by state and territory health departments, health professionals, vaccine companies and members of the public.<sup>16,17</sup> All reports are assessed using internationally consistent criteria<sup>18</sup> and entered into the Australian Adverse Drug Reactions System (ADRS) database. The TGA medical officers review all serious reports for drugs and vaccines. Reports are used in data mining and signal detection activities. Where there is insufficient information in a report to determine causality for a serious adverse event the TGA will contact the reporter on up to 3 occasions to elicit further information.

### Adverse events following immunisation data

De-identified information on all AEFI reported to the TGA from 1 January 2000 to 31 December 2014 and stored in the ADRS database, were released to the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) in March 2015. Readers are referred to previous AEFI surveillance reports for a description of the surveillance system.<sup>2,5</sup>

Records\* contained in the ADRS database were eligible for inclusion in the analysis if a vaccine was recorded as 'suspected'<sup>†</sup> of involvement in the reported adverse event and *either*

- the vaccination occurred between 1 January 2000 and 31 December 2014, *or*
- for records where the vaccination date was not recorded, the date of onset of symptoms or signs that occurred between 1 January 2000 and 31 December 2014.

### Study definitions of adverse events following immunisation

AEFI were defined as 'serious' or 'non-serious' based on information in the report sent to the TGA and criteria similar to those used by the World Health Organization<sup>18</sup> and the US Vaccine Adverse Events Reporting System.<sup>19</sup> In this report, an AEFI is defined as 'serious' if it meets one or

more of the following criteria: (1) results in death; (2) is life-threatening; (3) requires inpatient hospitalisation or prolongation of existing hospitalisation; (4) results in persistent or significant disability/incapacity; (5) is a congenital anomaly/birth defect or; (6) is a medically important event or reaction.

Typically, each record lists several reaction terms that are symptoms, signs and/or diagnoses that have been coded by TGA staff from the reporter's description into standardised terms using the Medical Dictionary for Regulatory Activities (MedDRA®).<sup>20,21</sup>

In reports published previously, in order to analyse the data, MedDRA® coding terms were grouped to create a set of reaction categories that were broadly analogous to the reactions listed in previous *Australian Immunisation Handbooks*.<sup>16,17</sup> However, the methodological framework of reporting of adverse events have been reviewed by NCIRS in collaboration with TGA and a revised format for AEFI analyses using MedDRA preferred terms (PTs) was adopted.<sup>22</sup> For this report, MedDRA PTs are used for data analysis. Grouping of reactions using PTs is more comparable with data from other countries and internationally accepted.<sup>23,24,25</sup> In conjunction with the more recent national vaccine-specific reporting form,<sup>26</sup> the use of PTs allow better reflection of post-marketing surveillance data on vaccines in Australia.

### Data analysis

All data analyses were performed using SAS software version 9.3.<sup>27</sup> Average annual population-based reporting rates were calculated for each state and territory and by age group using 2014 population estimates obtained from the Australian Bureau of Statistics.<sup>28</sup> All rates are presented as average annual rates per 100,000 population. Reporting rates per 100,000 administered doses were estimated where information was available on the number of doses administered. This was done for vaccines funded through the NIP for children aged <7 years. The number of administered doses of each of the childhood vaccines was obtained from the Australian Childhood Immunisation Register (ACIR), a national population-based register of approximately 99% of children aged under 7 years.<sup>29</sup>

### Notes on interpretation

Caution is required when interpreting the data presented in this report. Due to reporting delays and late onset of some AEFI, the data are considered preliminary, particularly for the 4th quarter of 2014. Data published in previous reports for 2000 to 2013

\* The term 'AEFI record' is used throughout this report because a single AEFI notification/report to the Office of Product review can generate more than 1 record in the ADRS database. This may occur if there is a time sequence of separate adverse reactions in a single patient, such as systemic and local reactions.

† Vaccines are classified as 'suspected' if the report contains sufficient information to be valid and the relationship between reported reactions and the vaccine is deemed at least possible.

may differ from that presented in this report for the same period because this report has been updated to include delayed notifications to the TGA that were not included in prior publications. Data can also differ because reports may be updated and recoded when follow-up information is received or when vaccine-specific analyses are conducted.

The information collated in the ADRS database is intended primarily for signal detection and hypothesis generation. While reporting rates can be estimated using appropriate denominators, they cannot be interpreted as incidence rates due to under-reporting and biased reporting of suspected events, and the variable quality and completeness of information provided in individual notifications.<sup>2-13,30</sup>

It is important to note that this report is based on vaccine information and MedDRA preferred terms collated in the ADRS database and not on comprehensive clinical notes or case reviews. The reported symptoms, signs and diagnoses in each AEFI record in the ADRS database are temporally associated with vaccination but are not necessarily causally associated with a vaccine or vaccines.

### Comparison with online Database of Adverse Events Notifications

In August 2012, the TGA made available to the public on its website a searchable database, the Database of Adverse Event Notifications (DAEN) that contains reports of all adverse event reports for medicines and vaccines.<sup>31</sup> The data in this report have not been downloaded from DAEN. This annual report uses data sent to NCIRS from the ADRS database by TGA in March 2015, and includes more detailed data than are provided by DAEN. The numbers published in this report may be different to the numbers in the DAEN database, due to different dates of data extraction and amendment to reports where further information has become available. In addition, this report provides several features that are not available from the DAEN database, including long-term trends and population and dose-based reporting rates, put in the context of changes in vaccine policy and use, and reporting practices.

## Results

The ADRS database included a total of 3,087 records where the date of vaccination (or onset of adverse event, if vaccination date was not reported) was between 1 January and 31 December 2014.

In 2014, 82% of AEFI (n=2,521) were reported to the TGA via states and territories, while the rest were reported directly to the TGA by healthcare

professionals (12% n=355), members of the public (4% n=119), vaccine companies (3% n=88) and hospitals (1% n=43).

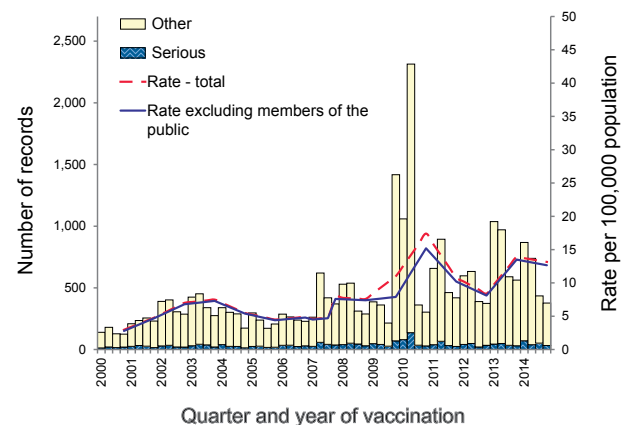
### Reporting trends

The overall reporting rate for 2014 was 13.2 per 100,000 population compared with 13.9 per 100,000 in 2013. The highest peak was observed in 2010 (17.4 per 100,000) predominantly due to reports in children following vaccination with the pandemic and 2010 seasonal trivalent influenza vaccines.<sup>11</sup>

The vast majority of reported events in 2014 (from all reporter types) were of a non-serious nature similar to the previous years (Figure 1).<sup>9,10</sup> Figures 2a, 2b and 2c demonstrate marked variations in reporting levels in association with previous changes to the NIP from 2000 onwards. The decrease in reports in 2014 was predominantly due to a decline in reports following HPV vaccines in adolescents, and cessation of the hepatitis B program in schools (Figure 2c).

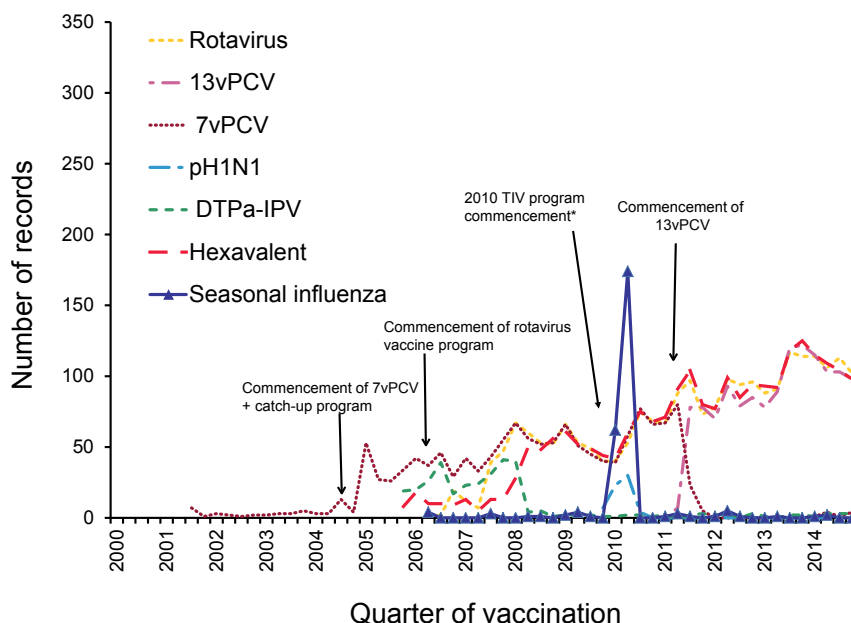
A seasonal pattern of AEFI reporting was apparent in 2014 as in previous years, with the highest number of AEFI notifications for vaccinations administered in the 1st half of the year (Figure 1). This corresponds with the months when influenza vaccine was given and older Australians received 23vPPV (March to June). However, more AEFI reports following influenza vaccine were received in each of the last 5 years than years prior to 2009 (pre-pandemic era) (Figure 2c).

**Figure 1: Adverse events following immunisation, ADRS database, 2000 to 2014, by quarter and year of vaccination**



For reports where the date of vaccination was not recorded, the date of onset or date event was reported to the Therapeutic Goods Administration was used as a proxy for vaccination date.

**Figure 2a: Adverse events following immunisation for children aged <1 year, ADRS database, 2000 to 2014, by quarter and year of vaccination**

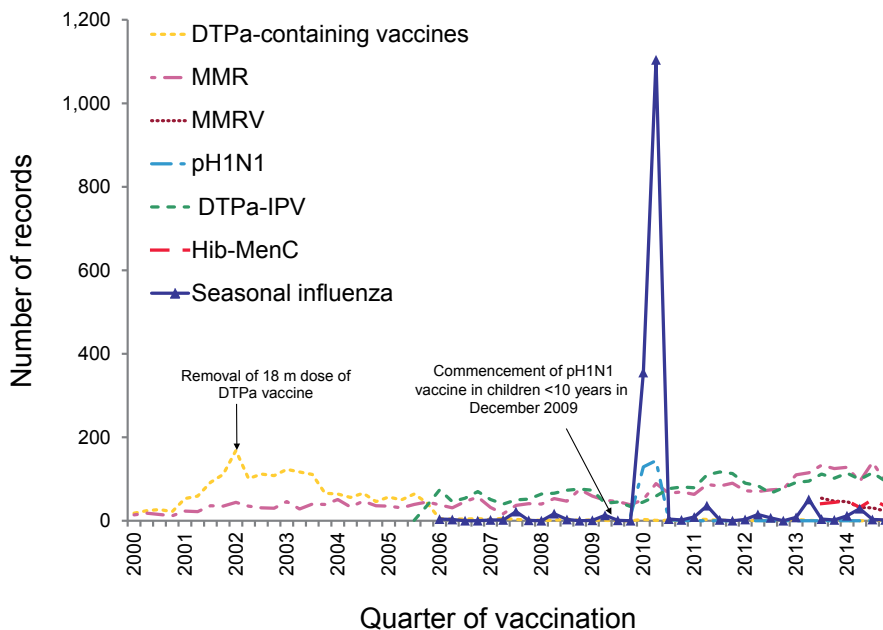


\* Safety signal for fever and febrile convulsion found to be due to Seqirus, formerly bioCSL Fluvax 2010 TIV in children.

DTPa-IPV and DTPa-IPV-HepB-Hib (hexavalent) vaccines were introduced into the National Immunisation Program schedule in November 2005; rotavirus (RotaTeq® and Rotarix®) vaccines on 1 July 2007; pH1N1 influenza vaccine for children 6 months to 10 years on December 2009; seasonal trivalent influenza vaccine in 2010, which was an extension of existing adult and Indigenous programs to at-risk populations; and the 13-valent pneumococcal conjugate vaccine (13vPCV) on 1 July 2011 (Table 1).

For reports where the date of vaccination was not recorded, the date of onset or date event was reported to the Therapeutic Goods Administration was used as a proxy for vaccination date.

**Figure 2b: Adverse events following immunisation for children aged 1 to <7 years in frequently reported vaccines, ADRS database, 2000 to 2014, by quarter and year of vaccination**



\* Safety signal for fever and febrile convulsion found to be due to Seqirus, formerly bioCSL, Fluvax 2010 TIV in children.

DTPa-IPV was introduced into the National Immunisation Program schedule in November 2005 replacing DTPa and OPV; seasonal trivalent influenza vaccine in 2010, which was an extension of existing adult and Indigenous programs to at-risk populations; MMRV and HibMenC vaccines on July 2013, and HPV program extended to boys in February 2013 (Table 1).

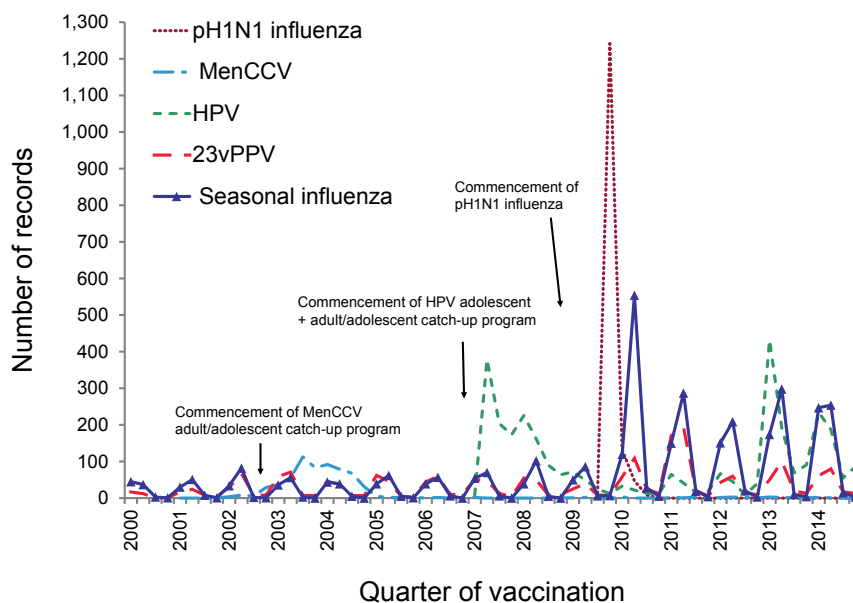
For reports where the date of vaccination was not recorded, the date of onset or date event was reported to the Therapeutic Goods Administration was used as a proxy for vaccination date.

**Age distribution**

The highest population-based AEFI reporting rate per 100,000 population occurred in infants under 1 year of age, the age group that received the high-

est number of vaccines (Figure 3). Compared with 2013, AEFI reporting rates in children decreased in the 1–<2 years age group from 132.1 to 117.3. A decline was also observed in the 7–<20 years age group from 26.6 to 19.7 (Figure 3).

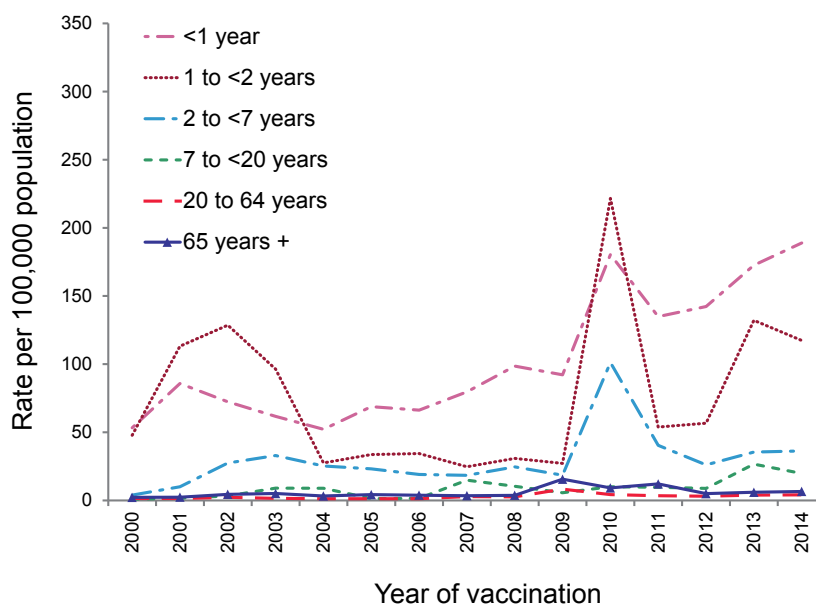
**Figure 2c. Adverse events following immunisation for people aged ≥7 years in frequently reported vaccines, ADRS database, 2000–2014, by quarter of vaccination**



MenCCV was introduced into the National Immunisation Program schedule on 1 January 2003; pH1N1 influenza vaccine for children 6 months to 10 years on December 2009; pH1N1 vaccination for those ≥10 years commenced on 30 September 2009; seasonal trivalent influenza vaccine in 2010, which was an extension of existing adult and Indigenous programs to at-risk populations; and HPV program extended to boys in February 2013 (Table 1).

For reports where the date of vaccination was not recorded, the date of onset or date event was reported to the Therapeutic Goods Administration was used as a proxy for vaccination date.

**Figure 3: Reporting rates of adverse events following immunisation per 100,000 population, ADRS database, 2000 to 2014, by age group and year of vaccination**



For reports where the date of vaccination was not recorded, the date of onset or date event was reported to the Therapeutic Goods Administration was used as a proxy for vaccination date.

Reporting rates per 100,000 doses decreased overall and for most individual vaccines in 2014 compared with 2013 (Table 2). For children under 7 years of age, rates for varicella and MenC should be interpreted with caution since these monovalent vaccines were replaced by combination vaccines in July 2013 and hence very few doses were given during 2014.

### Geographical distribution

Population-based reporting patterns varied between states and territories during 2014 (Table 3) as in previous years.<sup>2,-13</sup> Reporting rates decreased in most jurisdictions in 2014 compared with 2013 except in Victoria and South Australia, which experienced a slight increase.

**Table 2: Vaccine types listed as 'suspected' in records of adverse events following immunisation by age groups (<7, 7–17, 18–64 and ≥65 years), ADRS database, 2014**

Vaccines*	AEFI records† (n)	Vaccine doses	Reporting rate per 100,000 doses§			
			2014		2013	
			Rate	95% CI	Rate	95% CI
<b>&lt;7 years</b>						
DTPa-containing vaccines	894	1,169,168	76.5	71.6–81.6	75.2	70.3–80.3
Hexavalent (DTPa-IPV-HepB-Hib)	461	866,828	53.2	48.5–58.3	53.7	49.0–58.9
DTPa-IPV	433	302,340	143.2	130.3–157.4	136.7	123.8–150.7
Measles-mumps-rubella	480	594,553	80.7	73.8–88.3	83.6	76.4–91.2
Pneumococcal conjugate – PCV	450	880,999	51.1	46.6–56.0	52.8	48.1–57.9
Rotavirus vaccine	442	716,984	61.6	56.2–67.7	77.2	70.0–85.0
Meningococcal C conjugate	13	10,476	124.1	72.1–213.7	57.9	47.5–70.0
Measles-mumps-rubella-varicella	138	301,203	45.8	38.8–54.1	75.1	61.3–91.1
<i>Haemophilus influenzae</i> type b	5	12,943	38.6	16.1–92.8	56.2	45.5–68.6
Hib–MenC	180	295,170	61.0	52.7–70.6	73.7	59.4–90.3
Seasonal influenza	49	n/a	–	–	–	–
Varicella	11	11,586	94.9	52.6–171.4	37.4	28.5–48.1
Total (<7 years)	1,485	4,002,987	37.1	35.3–39.0	36.1	34.3–38.1
<b>7–17 years</b>						
HPV	556	n/a	–	–	–	–
Hepatitis B	4	n/a	–	–	–	–
dTpa	216	n/a	–	–	–	–
Varicella	112	n/a	–	–	–	–
Seasonal influenza	32	n/a	–	–	–	–
Total (7–17 years)	729	n/a	–	–	–	–
<b>18–64 years</b>						
Seasonal influenza	374	n/a	–	–	10.5	9.4–11.7
dTpa	53	n/a	–	–	–	–
23vPPV	39	n/a	–	–	42.3	31.9–54.9
Total (18–64 years)	582	n/a	–	–	11.8	10.7–13.0
<b>≥65 years</b>						
Seasonal influenza	113	n/a	–	–	4.3	3.5–5.2
23vPPV	120	n/a	–	–	35.9	29.6–43.1
dTpa	6	n/a	–	–	–	–
Total (≥65 years)	226	n/a	–	–	8.3	7.2–9.5

\* Records where at least 1 of the vaccines shown in the table was suspected of involvement in the reported adverse event.

† Number of adverse events following immunisation records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 31 December 2014. More than 1 vaccine may be coded as 'suspected' if several were administered at the same time.

‡ Number of vaccine doses recorded on the Australian Childhood Immunisation Register and administered between 1 January and 31 December 2014.

§ The estimated reporting rate per 100,000 vaccine doses recorded.

n/a Not applicable.

**Vaccines**

There were 3,087 AEFI records received in 2014 (Table 4). The percentage of records where only 1 vaccine was reported as being the suspected vaccine differed by vaccine administered, typically varying according to whether multiple vaccines were routinely co-administered for the patient's age. There were slight variations in the numbers with events defined as 'serious', which have remained low as in previous years.

The most frequently reported individual vaccine was seasonal influenza vaccine with 589 records (19%), followed by HPV vaccine with 571 records (18.5%), MMR (n=523; 17%), hexavalent DTPa-IPV-HepB-Hib (n=467; 15%) and rotavirus vaccine (n=446; 14%) (Table 4).

For HPV vaccine, of the 571 AEFI reports, 57% were reported in males and 43% in females (Figure 4). HPV vaccine was the only suspected vaccine in 334 records (58.5%).

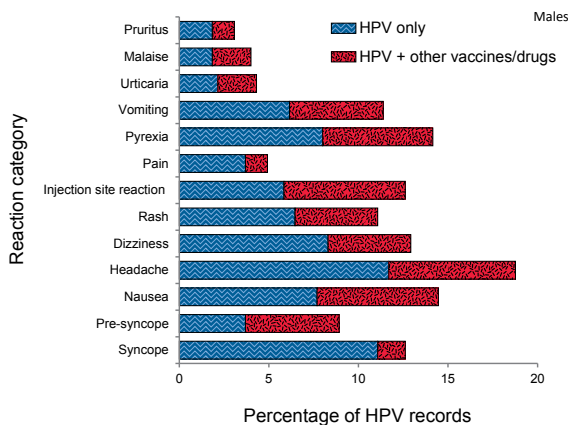
**Table 3: Adverse events following immunisation records, ADRS database, 1 January to 31 December 2014, by state or territory**

State or territory	AEFI records n	%	Annual reporting rate per 100,000 population*			
			'Serious' <sup>†</sup>	Aged <7 years	Overall rate	95% confidence interval
Australian Capital Territory	164	5.3	1.3	8.0	42.5	36.5–49.5
New South Wales	518	16.8	0.7	2.4	6.9	6.3–7.5
Northern Territory	52	1.7	2.0	7.3	21.2	16.2–27.8
Queensland	574	18.6	0.5	5.8	12.2	11.2–13.2
South Australia	280	9.1	0.8	6.8	16.6	14.8–18.7
Tasmania	82	2.7	0.2	6.0	15.9	12.8–19.8
Victoria	1212	39.3	1.6	12.3	20.8	19.6–22.0
Western Australia	205	6.6	0.7	4.4	8.0	6.9–9.1
Total	3,087	100.0	0.9	6.3	13.2	12.7–13.6

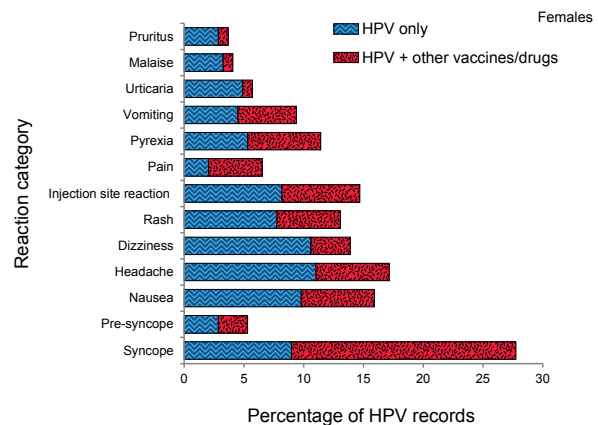
\* Average annual rates per 100,000 population calculated using Australian Bureau of Statistics mid-2014 population estimates.  
 † Adverse events following immunisation records defined as 'serious' (i.e. recovery with sequelae, hospitalisation, life-threatening or death).

**Figure 4: Most frequently reported adverse events following immunisation with human papillomavirus vaccine,\* 2014, by number of vaccines suspected of involvement in the reported adverse event**

**Males**



**Females**



\* Per cent of 325 adverse events following immunisation records (human papillomavirus males) and 245 records (human papillomavirus females) where the vaccine was listed as suspected of involvement in the reported adverse event following immunisation.

Source: Adverse Drug Reactions Reporting System database, Therapeutic Goods Administration.



**Table 4: Vaccine types listed as ‘suspected’ in records of adverse events following immunisation, ADRS database, 2014**

Suspected vaccine type	AEFI records		One suspected vaccine only <sup>†</sup>		‘Serious’ <sup>§</sup>		<7 years <sup>  </sup>		≥7 years <sup>  </sup>	
	n	%	n	% <sup>¶</sup>	n	% <sup>¶</sup>	n	% <sup>¶</sup>	n	% <sup>¶</sup>
Influenza	589	19.1	495	84.0	32	5.4	49	8.3	541	91.7
HPV	571	18.5	334	58.5	31	5.4	2	0.4	569	99.6
MMR	523	16.9	103	19.7	35	6.7	480	91.8	38	7.3
DTPa-IPV-HepB-Hib	467	15.1	36	7.7	49	10.5	461	98.7	3	0.6
Rotavirus	446	14.4	61	13.7	56	12.6	442	99.1	1	0.2
13vPCV	445	14.4	13	2.9	49	11.0	439	98.7	6	1.3
DTPa-IPV	443	14.3	203	45.8	14	3.2	433	97.7	8	1.8
dTpa	281	9.1	131	46.6	9	3.2	3	1.1	278	98.9
23vPPV	185	6.0	116	62.7	6	3.2	7	3.8	178	96.2
Hib-MenC	181	5.9	9	5.0	22	12.2	180	99.4	1	0.6
MMRV	140	4.5	119	85.0	20	14.3	138	98.6	2	1.4
Varicella	138	4.5	26	18.8	2	1.4	11	8.0	124	89.9
Meningococcal B	66	2.1	64	97.0	2	3.0	39	59.1	24	36.4
Hepatitis B	61	2.0	42	68.9	1	1.6	9	14.8	34	55.7
dT	22	0.7	18	81.8	2	9.1	0	0.0	22	100.0
Hepatitis A-Typhoid	20	0.6	11	55.0	0	0.0	1	5.0	19	95.0
BCG	19	0.6	18	94.7	1	5.3	17	89.5	1	5.3
Yellow fever	18	0.6	11	61.1	0	0.0	0	0.0	17	94.4
Hepatitis A	17	0.6	5	29.4	1	5.9	8	47.1	9	52.9
MenCCV	17	0.6	3	17.6	1	5.9	13	76.5	4	23.5
Typhoid	15	0.5	6	40.0	3	20.0	2	13.3	13	86.7
Rabies	12	0.4	9	75.0	0	0.0	0	0.0	12	100.0
Q fever	11	0.4	11	100.0	1	9.1	0	0.0	11	100.0
Zoster	9	0.3	8	88.9	1	11.1	0	0.0	9	100.0
Hib	7	0.2	0	0.0	0	0.0	5	71.4	2	28.6
Hepatitis A + B	6	0.2	4	66.7	1	16.7	0	0.0	6	100.0
Cholera	5	0.2	4	80.0	0	0.0	0	0.0	5	100.0
Japanese encephalitis	3	0.1	1	33.3	0	0.0	0	0.0	3	100.0
Tetanus	1	0.0	0	0.0	0	0.0	0	0.0	1	100.0
Total**	3,087	100.0	18,73	60.7	211	6.8	1,485	48.1	1,537	49.8

\* Abbreviations of vaccine names are defined in the Appendix.

† Adverse events following immunisation (AEFI) records where only 1 vaccine was suspected of involvement in a reported adverse event.

‡ Causality ratings were assigned to AEFI records using criteria described previously.<sup>2,3</sup>

§ ‘Serious’ is defined in the Methods section.

|| Includes only AEFI records where an age or date of birth has been reported.

¶ Percentages are calculated for the number of AEFI records where the vaccine was suspected of involvement in the AEFI.

\*\* Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and an AEFI record may list more than 1 vaccine.

## Reactions

In 2014, there was a total of 6,810 events reported for 3,087 AEFI records. Out of the 3087 records, the most frequently reported adverse events were injection site reactions (ISRs) (n=832; 27%), pyrexia (n=558; 18%), rash (n=484; 16%), vomiting (n=289;

9%), headache (n=219; 7%), nausea (n=193; 6%), extensive swelling of vaccinated limb (n=177; 6%) and syncope (n=154; 5%) (Table 5, Figure 5). Some of the other reactions of interest were convulsions (n=85; 3%), hypotonic-hyporesponsive episode (n=51; 1.7%), intussusception (n=16; 0.5%) and

Guillain-Barré syndrome (n=5; 0.2%) (Table 5). Anaphylaxis (n=20) was reported for less than 1% of AEFI records in 2014.

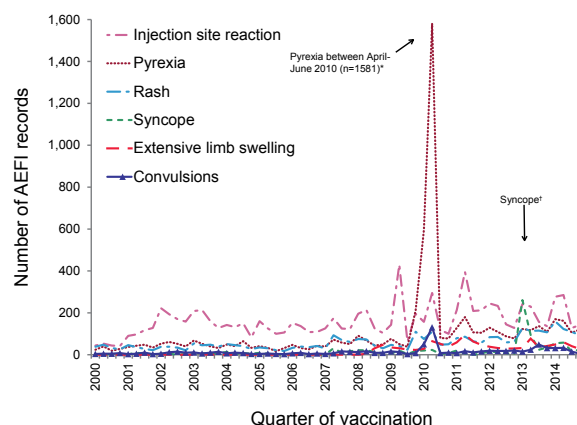
The number of reports for each reaction has changed over time (Figure 5). The variation in reporting of ISRs is related to changes in the immunisation schedule for vaccines that are known to have higher rates of ISR, including DTPa-containing vaccines, MenCCV, 23vPPV and HPV vaccine.<sup>2-13,32,33</sup> Increases in reports of fever were largely associated with time periods when new vaccines were added to the NIP in the reporting period, such as 7vPCV and HPV; the extension of seasonal influenza vaccine on the NIP to include persons <65 years at high risk of influenza in 2010; 13vPCV replacing 7vPCV in July 2011; and the extension of HPV to males in 2013.

For HPV vaccine, the spectrum of reactions was similar in boys and girls in this reporting period, however there were more cases in females of syncope (62% in females versus 38% in males) (Figure 4)

### Severity

The majority of reported events in 2014 were defined as 'non-serious' and only 7% (n=211) were defined as 'serious'. This was similar to the proportions of serious AEFI in previous years.<sup>9,11,12</sup>

**Figure 5: Selected frequently of reported adverse events following immunisation, ADRS database, 2000 to 2014, by quarter and year of vaccination**



\* Associated with administration of bioCSL Fluvax 2010 TIV and associated stimulated reporting.

† The peak in syncope coincided with the enhanced human papillomavirus surveillance program in which there was stimulated reporting of syncope for the first 6 months of 2013.

For reports where the date of vaccination was not recorded, the date of onset or date event was reported to the Therapeutic Goods Administration was used as a proxy for vaccination date. Also, grouping for reactions are different for this report though these reactions have been mapped back to 2000 as mentioned in the Methods section.

**Table 5: Selected reported adverse events and reactions of interest\* classified by MedDRA Preferred Terms in records of adverse events following immunisation, ADRS database, 2014**

MedDRA preferred terms adverse events	AEFI records N	Only reaction reported†		'Serious'‡		<7 years§		≥7 years§	
		n	%	n	%	n	%	n	%
Injection site reaction¶	832	360	43.3	8	1.0	397	47.7	422	50.7
Pyrexia	558	14	2.5	47	8.4	318	57.0	230	41.2
Rash**	484	185	38.2	29	6.0	325	67.1	153	31.6
Vomiting	289	30	10.4	32	11.1	166	57.4	120	41.5
Headache	219	4	1.8	9	4.1	8	3.7	209	95.4
Nausea	193	3	1.6	4	2.1	7	3.6	180	93.3
Extensive limb swelling	177	73	41.2	3	1.7	106	59.9	67	37.9
Syncope	154	117	76.0	8	5.2	17	11.0	135	87.7
Diarrhoea	153	11	7.2	16	10.5	114	74.5	39	25.5
Lethargy	145	0	0.0	11	7.6	62	42.8	82	56.6
Dizziness	142	11	7.7	4	2.8	1	0.7	136	95.8
Urticaria	139	57	41.0	6	4.3	72	51.8	65	46.8
Irritability	127	5	3.9	15	11.8	124	97.6	2	1.6
Pain	126	5	4.0	6	4.8	19	15.1	105	83.3
Malaise	105	1	1.0	4	3.8	11	10.5	92	87.6
Pallor	94	2	2.1	9	9.6	48	51.1	46	48.9
Erythema	85	15	17.6	1	1.2	39	45.9	46	54.1
Convulsions††	85	60	70.6	27	31.8	83	97.6	2	2.4

**Table 5 continued: Selected reported adverse events and reactions of interest\* classified by MedDRA Preferred Terms in records of adverse events following immunisation, ADRS database, 2014**

MedDRA preferred terms adverse events	AEFI records	Only reaction reported†		'Serious'‡		<7 years§		≥7 years§	
	N	n	%	n	%	n	%	n	%
Myalgia	82	2	2.4	3	3.7	4	4.9	77	93.9
Pruritus	79	6	7.6	1	1.3	18	22.8	60	75.9
Decreased appetite	71	0	0.0	4	5.6	48	67.6	22	31.0
Presyncope	70	41	58.6	1	1.4	6	8.6	63	90.0
Abdominal pain	68	2	2.9	8	11.8	31	45.6	37	54.4
Fatigue	61	0	0.0	3	4.9	4	6.6	55	90.2
Cough	54	2	3.7	4	7.4	26	48.1	28	51.9
Paraesthesia	54	1	1.9	1	1.9	1	1.9	52	96.3
Chills	51	0	0.0	1	2.0	3	5.9	48	94.1
Hypotonic-hyporesponsive episode	51	34	66.7	10	19.6	50	98.0	0	0.0
Arthralgia	47	3	6.4	1	2.1	2	4.3	45	95.7
Somnolence	42	1	2.4	3	7.1	29	69.0	13	31.0
Dyspnoea	39	0	0.0	5	12.8	7	17.9	31	79.5
Hyperhidrosis	35	0	0.0	1	2.9	5	14.3	35	100.0
Oropharyngeal pain	30	1	3.3	0	0.0	6	20.0	24	80.0
Rhinorrhoea	28	0	0.0	3	10.7	20	71.4	8	28.6
Hypoaesthesia	27	2	7.4	1	3.7	0	0.0	27	100.0
Tachycardia	24	0	0.0	7	29.2	10	41.7	13	54.2
Haematochezia	22	6	27.3	3	13.6	21	95.5	1	4.5
Anaphylactic reaction	20	16	80.0	20	100.0	6	28.6	12	57.1
Tremor	16	2	12.5	0	0.0	4	25.0	11	68.8
Intussusception	16	13	81.3	7	43.8	16	100.0	0	0.0
Chest discomfort	16	0	0.0	1	6.3	0	0.0	15	93.8
Lymphadenitis	6	2	33.3	0	0.0	0	0.0	6	100.0
Guillain-Barré syndrome	5	3	60.0	3	60.0	0	0.0	3	60.0

\* Selected reported adverse events reported during 1 January to 31 December 2014. For injection site reaction, rash and convulsions, Preferred Terms (PTs) were grouped as described below. A complete list of adverse reactions as classified by individual Preferred Terms is available on request.

† Adverse events following immunisation (AEFI) records where only one reaction was reported.

‡ 'Serious' is defined in the Methods section.

§ Includes only AEFI records where an age or date of birth has been reported.

|| Percentages relate to the number of AEFI records in which the specific reaction term was listed.

¶ Injection site reaction includes the following MedDRA PTs: injection site reaction, injection site swelling, injection site pain, injection site mass, injection site erythema, injection site cellulitis, injection site rash, injection site induration, injection site abscess, injection site pruritus, injection site nodule, injected limb mobility decreased, injection site urticaria, injection site inflammation, injection site bruising, injection site infection, and injection site warmth.

\*\* Rash includes the following MedDRA PTs: rash, rash generalised, rash erythematous, rash pruritic, rash maculo-papular, rash macular, rash vesicular, rash papular, rash morbilliform, and rash pustular.

†† Convulsion includes the following MedDRA PTs: febrile convulsion, and convulsion, grand mal convulsion, and partial seizures.

Five deaths were recorded as temporally associated with receipt of vaccines in 2014:

- A 77-year-old male immunised with a seasonal influenza vaccine died 9 hours later from sudden cardiac arrest. He had left ventricular dysfunction and a medical history of hypertension.
- A 58-year-old male had an infected leg wound prior to vaccination with diphtheria and tetanus vaccine and seasonal influenza vaccine. He developed acute disseminated myeloencephalitis, which progressed over 6 weeks leading to death. Symptom onset date was 5 days after vaccination.

- A 2-month-old female infant who had received Infanrix hexa®, Prevenar 13® and Rotateq® died 4 days following immunisation in hospital. *Bordetella pertussis* DNA was detected from the epiglottis on post-mortem.
- A 1-year-old male child in the terminal stages of spinal muscular atrophy type 1 died 7 days following vaccination with measles-mumps-rubella (Priorix®), seasonal influenza (Vaxigrip Junior®) and Hib–MenC (Menitorix®) vaccines.
- A 2-month old male infant died 2 days following immunisation with Infanrix hexa®, Prevenar 13® and Rotateq®. He had underlying congenital heart disease (atrio-ventricular septal defect and aortic arch repair with post-operative complications).

All deaths were investigated by the TGA and no clear causal relationship with vaccination was found.

## Discussion

This report uses a similar methodology of analysis used in the previous 2013 annual report. As per the previous report, this method allows for clearer reporting of adverse events using MedDRA PTs, as used in the DAEN. This change in methodology needs to be taken into account when comparing with data from pre-2013 annual reports on specific reaction terms and categories.

In 2014, there was an overall decline in the AEFI reporting rate. The decline was likely due to it being the second year of the extension of National HPV Vaccination Program to males. There is usually an increase in reporting of adverse events when a program is newly rolled out. Historical data have shown that initial high levels of AEFI reporting occur each time a new vaccine is introduced, as immunisation providers are more likely to report milder, less serious AEFIs for vaccines with which they are not familiar, which is then followed by a reduction and stabilisation of reporting over time. Of note, during 2013 and 2014 the TGA, together with states and territories, closely monitored adverse events reported following HPV vaccination as the program was extended to males, including via enhanced surveillance using rapid reporting from school-based programs.<sup>34</sup>

Furthermore, in 2014, the drop in the number of adverse events could partially be attributed to ceasing the school-based hepatitis B vaccination program by the end of 2013 and therefore only 4 adverse events for hepatitis B vaccine were reported for this cohort of children. In addition, there were very few reports of adverse events following admin-

istration of monovalent vaccines such as varicella, MenC and Hib in this reporting period. This was anticipated as the combined Hib–MenC vaccine replaced the respective monovalent MenC and Hib vaccines in July 2013. Also, from July 2013, the 2nd dose of MMR vaccine was brought forward to 18 months of age and delivered as a combination MMRV vaccine.

Overall in Australia, injection site reaction, pyrexia and rash were the most commonly reported reactions in 2014. Vaccines such as DTPa-containing vaccines, MMR, rotavirus, Hib–MenC and pneumococcal conjugate (PCV13) had higher reporting rates than other vaccines for children aged under 7 years in the current reporting period. However, these rates were not significantly higher than the previous reporting period.

## Conclusion

The total number of reported AEFI in 2014 decreased compared with 2013. The majority of AEFIs reported to the TGA were mild transient events. The data reported here are consistent with an overall high level of safety for vaccines included in the NIP schedule.

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## Abbreviations of vaccine types

7vPCV	7-valent pneumococcal conjugate vaccine
13vPCV	13-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
BCG	Bacille Calmette-Guérin (i.e. tuberculosis)
dT	diphtheria-tetanus – adolescent and adult formulation
DTPa	diphtheria-tetanus-pertussis (acellular) – paediatric formulation
dTpa	diphtheria-tetanus-pertussis (acellular) – adolescent and adult formulation
DTPa-IPV	combined diphtheria-tetanus-pertussis (acellular) and inactivated poliovirus (quadrivalent)
DTPa-IPV-HepB-Hib	combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus, hepatitis B and <i>Haemophilus influenzae</i> type b vaccine (hexavalent)
HepB	hepatitis B
Hib	<i>Haemophilus influenzae</i> type b
Hib-HepB	combined <i>Haemophilus influenzae</i> type b and hepatitis B
Hib-MenC	combined <i>Haemophilus influenzae</i> type b and meningococcal C conjugate vaccine
HPV	human papillomavirus
MenCCV	meningococcal C conjugate vaccine
MMR	measles-mumps-rubella
MMRV	measles-mumps-rubella-varicella
pH1N1	pandemic H1N1 influenza 2009

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# PAEDIATRIC ACTIVE ENHANCED DISEASE SURVEILLANCE INAUGURAL ANNUAL REPORT, 2014

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## Abstract

**Introduction:** The Paediatric Active Enhanced Disease Surveillance (PAEDS) network is a hospital-based active surveillance system employing prospective case ascertainment of selected uncommon vaccine preventable diseases and potential adverse events following immunisation (AEFI). PAEDS enhances other Australian surveillance systems by providing prospective detailed clinical and laboratory data for the same child.

**Methods:** Specialist surveillance nurses screen hospital admissions, emergency department records, laboratory and other data, to prospectively identify hospitalised children aged under 15 years in 5 paediatric tertiary referral hospitals in New South Wales, Victoria, South Australia, Western Australia and Queensland. Standardised protocols and case definitions are used across all sites. Conditions under surveillance include vaccine preventable diseases: acute flaccid paralysis, varicella, pandemic and seasonal influenza and pertussis, and potential AEFIs: febrile seizures and intussusception. PAEDS also conducts surveillance for acute childhood encephalitis.

**Results:** Since August 2007, PAEDS has recruited a total of 6,227 hospitalised cases in total, for all conditions. From January to December 2014, there were 1,220 cases recruited across all conditions. Key outcomes include: enhanced acute flaccid paralysis surveillance to reach World Health Organization targets; supporting varicella and influenza vaccination in children; confirmation of a known low risk of febrile seizures following the 1st dose of measles-mumps-rubella vaccine but no increased risk of febrile seizures after measles-mumps-rubella-varicella vaccine, and a slightly increased risk of developing intussusception 1–7 days after rotavirus vaccination in infants aged less than 3 months. Acute childhood encephalitis data facilitated rapid investigation and response to the enterovirus 71 outbreak in 2013–2014.

**Conclusions:** PAEDS provides unique policy-relevant data. This is the first of planned PAEDS annual reports to *Communicable Diseases Intelligence*. *Commun Dis Intell* 2016;40(3):E391–E400.

**Keywords:** hospital-based; surveillance; immunisation

## Introduction

In 2007, the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, the Australian Paediatric Surveillance Unit (APSU) and a network of experts in immunisation and infectious diseases joined forces to develop a hospital-based active surveillance system: Paediatric Active Enhanced Disease Surveillance (PAEDS).<sup>1</sup> PAEDS provides important data on vaccine preventable diseases (VPDs) and adverse events following immunisation (AEFI), which are sufficiently severe to result in hospitalisation or emergency department presentation for select conditions, and difficult to adequately capture through passive surveillance mechanisms. PAEDS enables timely, prospective case identification and ascertainment, collection of detailed clinical data, medical and vaccination history, and biological samples from the same child.<sup>1</sup> For conditions where longer term outcomes are relevant, patients may be followed up after discharge from hospital.

Initially, 4 hospitals participated: The Children's Hospital at Westmead, Sydney, New South Wales; the Royal Children's Hospital, Melbourne, Victoria; the Women's and Children's Hospital, Adelaide, South Australia; and the Princess Margaret Hospital, Perth, Western Australia. In 2013, the Royal Children's Hospital, Brisbane, Queensland, joined PAEDS; this hospital moved and amalgamated in 2014 and is now the Lady Cilento Children's Hospital. Each of the 5 participating states' health departments now also contribute funding to support activities and PAEDS currently produces monthly data reports for all funding bodies and collaborators. The 5 paediatric hospitals have an estimated 148,920 admissions per annum (Table 1), representing approximately 72% of all admissions (~204,431) to tertiary hospitals providing specialist paediatric services in Australia.<sup>2</sup>

PAEDS is a separate surveillance mechanism from the APSU which relies on passive reporting from paediatricians.

In August 2007, PAEDS began surveillance of 4 conditions, including 2 VPDs and 2 AEFIs of public health and clinical interest: acute flaccid paralysis (AFP), hospitalised varicella, intussusception (IS) and seizures in infants (Table 2). PAEDS

also conducted active prospective surveillance for febrile seizures (FS) following measles-containing vaccines from 2013 to 2014, under funding by the Australian Government Department of Health as part of the vaccine safety plan for the introduction of measles-mumps-rubella-varicella (MMRV) vaccine to the National Immunisation Program (NIP). MMRV vaccine was associated with an increased risk of FS when used as the 1st dose of a measles-containing vaccine in the United States of America.<sup>3</sup> A retrospective review of FS (from January 2012 to April 2013) to investigate the risk of FS post-MMR (dose 1) and varicella vaccine was also conducted.<sup>4</sup>

PAEDS also conducted surveillance for children aged under 15 years with laboratory proven influenza during the influenza pandemic who were hospitalised during the period June to October 2009. This was funded by a National Health and Medical Research Council grant (no.633028) and supplemented by additional funding from the NSW Ministry of Health, enabling recruitment of influenza cases at 2 additional hospitals in New South Wales: John Hunter Children's Hospital, Newcastle and the Sydney Children's Hospital, Randwick.<sup>7</sup> The protocol and data collection forms were developed quickly by adapting the existing APSU protocol.<sup>9</sup>

**Table 1: Total hospital admissions and emergency department presentations for the 5 hospitals participating in Paediatric Active Enhanced Disease Surveillance in 2014**

Hospital	Hospital admissions	Emergency department presentations
Children's Hospital at Westmead, Sydney	32,149	55,049
Royal Children's Hospital, Melbourne	45,548	83,970
Women's and Children's Hospital, Adelaide	21,101	46,289
Princess Margaret Hospital, Perth	28,910	70,834
Lady Cilento Children's Hospital, Brisbane	21,212	26,773
Total	148,920	282,915

**Table 2: Paediatric Active Enhanced Disease Surveillance conditions under surveillance, case definitions and rationale, 2007–2014**

Condition and case definition	Rationale
<p>Acute flaccid paralysis (AFP) (2007 – ongoing)</p> <p><i>Case definition:</i></p> <p>Any child aged up to 15 years and presenting with acute flaccid paralysis: onset of flaccid paralysis in one or more limbs or acute onset of bulbar paralysis.</p>	<p>The World Health Organization requires active national surveillance for cases of AFP in children aged &lt;15 years in order to monitor for potential cases of paralytic poliomyelitis. Because of long-standing problems in obtaining adequate reporting and stool collection rates (at least 1/100,000 AFP cases in children &lt;15 years of age and collection of 2 stool specimens within 14 days of onset of paralysis in all identified cases), AFP was considered as a priority condition for inclusion in Paediatric Active Enhanced Disease Surveillance (PAEDS). PAEDS collects ~77% of all AFP cases identified annually in Australia.</p>
<p>Intussusception (2007 – ongoing)</p> <p><i>Case definition:</i></p> <p>Any child aged &lt;24 months presenting with a diagnosis of acute intussusception confirmed on air/liquid contrast enema or surgery (i.e. based on Level 1 of Diagnostic Certainty using the Brighton Collaboration clinical case definition). Includes hospitalised or emergency department only.<sup>5</sup></p> <p>From May 2013 the case definition age changed to &lt;9 months.</p>	<p>Intussusception is the most common cause of bowel obstruction in infants and young children and was associated with a previous rotavirus vaccine withdrawn from the United States of America in 1999. Timely, active and systematic surveillance of intussusception cases has been important to identify any temporal association with the 'new generation' rotavirus vaccines funded under the National Immunisation Program (NIP) from July 2007. Surveillance also aims to describe the epidemiology, aetiology and severity of intussusception.</p>
<p>Varicella and zoster hospitalisations (2007 – ongoing)</p> <p><i>Case definition:</i></p> <p>Any child aged 1 month to &lt;15 years hospitalised for varicella-zoster virus infection with or without complications.</p>	<p>Varicella vaccination was funded under the NIP from late 2005. Complications of varicella requiring hospitalisation provide a measure of disease burden and severity. Ongoing surveillance may show trends in both varicella and herpes zoster related to the varicella vaccination program and allow vaccine effectiveness estimations. The timely collection of vesicle samples and genetic subtyping of varicella-zoster virus allows for identification of vaccine failures in immunised children and genotypes associated with severe complications.</p>



**Table 2 continued: Paediatric Active Enhanced Disease Surveillance conditions under surveillance, case definitions and rationale, 2007–2014**

Condition and case definition	Rationale
<p>Seizures (August 2007–2008)</p> <p><i>Case definition:</i></p> <p>Any child aged 1 to &lt;8 months who presents with seizures and meets the following criteria: first seizure presentation AND there is no identifying trauma (e.g. head injury) AND the hospital stay is 4 hours or more.</p>	<p>Infants presenting with seizures in the first 8 months of life are of interest because seizures are a recognised potential serious adverse event following vaccination. Surveillance for infantile seizures provides an opportunity to describe the temporal relationship between seizures and recent vaccination.</p> <p>This surveillance was discontinued in 2008, in part due to the difficulty of applying the case definition in young infants, in whom the presentation of seizures can be complex to diagnose.</p>
<p>Febrile seizures following measles-containing vaccines (May 2013 – June 2014)</p> <p><i>Case definition:</i></p> <p>Any child aged &lt;5 years who presents with a seizure that fulfils the Brighton Collaboration case definition for a seizure AND occurs within 48 hours of an inactivated vaccine and/or 14 days of a live attenuated vaccine AND is associated with fever documented either by a parent and/or health provider.</p>	<p>Use of measles-mumps-rubella-varicella (MMRV) combination vaccine as the first dose of measles-containing vaccine in the United States of America was found to double the risk of fever and febrile seizures in children aged 12–23 months in the 5–12 days after vaccination (when compared with children who received MMR and varicella vaccines as separate injections).<sup>3</sup> In July 2013, MMRV vaccine was included on the NIP as the 2nd dose of measles-containing vaccine. Surveillance (retrospective and prospective) for febrile seizures following MMR, varicella and then MMRV vaccine was conducted to determine the risk of febrile seizures occurring after each vaccine as used under the Australian NIP.</p>
<p>Pertussis (2012 – ongoing)</p> <p><i>Case definition:</i></p> <p>Any child aged birth to 15 years (ineligible as of 15th birthday) admitted to hospital with laboratory-confirmed pertussis.</p>	<p>Despite immunisation coverage approaching 90% (for the 3 primary doses of diphtheria-tetanus-pertussis vaccine in pre-school children), pertussis continues to cause significant morbidity and mortality in Australian children.<sup>6</sup> The aims of this surveillance are to determine the burden of disease from hospitalised pertussis, with special emphasis on the duration of hospitalisation, use of intensive care, death and disability. The contribution of comorbidities to the severity of pertussis and possible sources of infection will also be examined. This surveillance data will assist in optimising pertussis prevention strategies.</p>
<p>Influenza – pandemic (June–October 2009)</p> <p><i>Case definition:</i></p> <p>Any child aged &lt;15 years at the time of diagnosis of influenza confirmed by laboratory testing, and admitted to hospital.</p>	<p>Children may suffer severe complications from influenza, including encephalopathy, myocarditis and rhabdomyolysis. Timely detailed data describing pre-existing risk factors, presentation, clinical course and outcome in children hospitalised with influenza, including H1N1-09, were lacking. Such data were needed to inform vaccination policy and clinical practice, as well as to assess the effectiveness of outbreak response measures.</p>
<p>Influenza – FluCAN (April–October each year. Commenced 2014)</p> <p><i>Case definition:</i></p> <p>Any hospitalised child aged &lt;18 years who presents with suspected influenza (respiratory symptoms +/- fever) who is positive for influenza by polymerase chain reaction.</p>	<p>The emergence of H1N1-09 influenza in 2009 demonstrated the importance of enhanced surveillance in children.<sup>7</sup> PAEDS provides unique timely sentinel data from 2 sites (Sydney and Perth) on influenza hospitalisations including complications and deaths, which can be used to inform public health response and policy. The data on children supplements adult influenza surveillance data collected by the other 15 sites under the FluCAN network. Information on influenza test negative (control) patients with acute respiratory illness is also collected and allows calculation of vaccine effectiveness to be performed.</p>
<p>Acute childhood encephalitis (2013 – ongoing)</p> <p><i>Case definition:</i></p> <p>Any child aged &lt;15 years AND hospitalised with acute encephalopathy AND who has <i>one or more</i> of the following: fever, seizures, focal neurological findings, at least one abnormality of cerebrospinal fluid, or EEG/neuroimaging findings consistent with infection-related encephalitis.</p>	<p>Encephalitis is a critical condition that requires hospitalisation and is considered a marker syndrome for emerging infectious diseases. It is most often caused by viruses (including those that are or potentially will be vaccine preventable). It can also be immune-mediated, and uncommonly can be associated with vaccine receipt. Although a potentially preventable cause of mortality and morbidity in children, there are limited epidemiologic data on encephalitis. PAEDS is uniquely placed to undertake active, syndromic surveillance with the additional capacity to collect biological specimens and enrol participants into comprehensive follow-up studies to improve understanding of long-term neuropsychological sequelae.<sup>8</sup></p>

From 2014, active prospective surveillance for influenza has been resumed at 2 PAEDS sites (Sydney and Perth) in collaboration with the Influenza Complications Alert Network (FluCAN) surveillance system, established in multiple adult and general hospitals.<sup>10,11</sup> Surveillance for acute childhood encephalitis also commenced in 2014 following a successful pilot study in New South Wales in 2013.<sup>8</sup>

In this report we summarise data collected by PAEDS between 2007 and 2014, with emphasis on the impacts and outcomes of surveillance and their potential usefulness to inform clinical practice and policy. We also provide a detailed report of surveil-

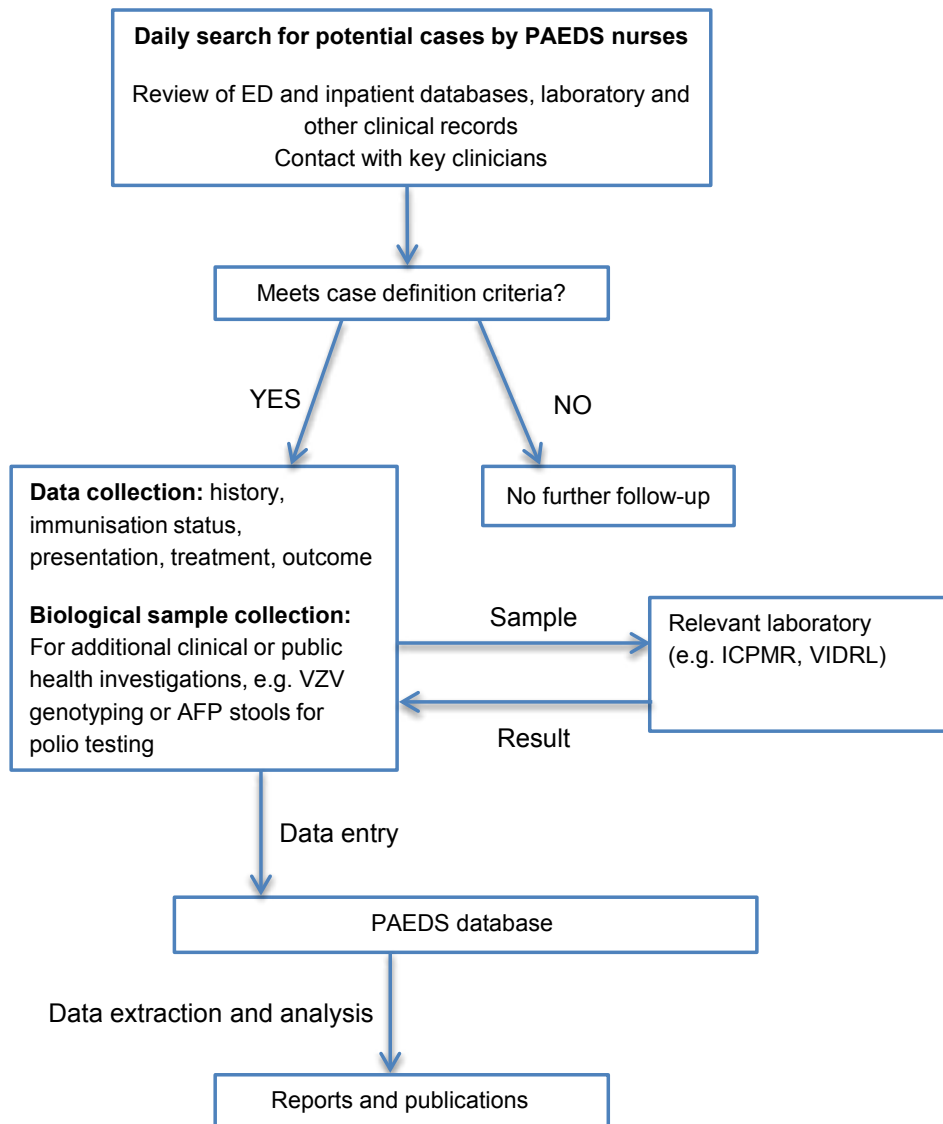
lance data for the year 2014, with a view to providing annual surveillance reports in *Communicable Diseases Intelligence* each year.

## Methods

### Active case ascertainment

Under PAEDS, specialist surveillance nurses in each hospital identified children aged less than 15 years diagnosed with the target conditions as defined in Table 2, by reviewing admission and emergency department databases and clinical records, laboratory results and/or infection control logs (Figure). Relationship-building and networking with medical and nursing staff in each hospital enhances prospective case identification.

**Figure: Overview of Paediatric Active Enhanced Disease Surveillance methods in the participating 5 sites\***



\* Participating sites are 5 sites: Children's Hospital at Westmead (Sydney), Royal Children's Hospital (Melbourne), Women's and Children's Hospital (Adelaide), Princess Margaret Hospital (Perth), Lady Cilento Children's Hospital (Brisbane)

Ethics permission was obtained from the Human Research Ethics Committees at each of the 5 hospitals. The initial model was based on consent being obtained from parents or guardians, after which detailed data were extracted from the clinical record, with data collection enhanced by interviewing the family.<sup>1</sup> In 2014, PAEDS moved to a 'no consent' model using de-identified data. By early 2015 all sites obtained ethics approval for reporting on de-identified data from clinical records, without the need to obtain written consent; families are provided with information sheets and written consent is still sought where information not collected in the medical record as part of best clinical practice is required from the family.

To check for completeness of case ascertainment, PAEDS nurses at each site conduct regular retrospective audits of medical records by searching for primary and secondary International Statistical Classification of Diseases and Related Health Problems, 10th revision, Australian Modification (ICD-10-AM) codes describing the relevant conditions (e.g. K56.1 for intussusception and BO1/BO2 and subcategories for varicella infection). Cases ascertained through the medical records audits were compared with the cases ascertained by PAEDS for the same period. Any additional cases identified by the ICD-10-AM audit process were retrospectively recruited into PAEDS.

### Collection of biological samples

Surveillance nurses facilitated collection of 2 stool samples within 14 days of onset of paralysis from children hospitalised with AFP. These samples were sent to the Australian National Enterovirus Reference Laboratory in Melbourne for identifi-

cation of enteroviruses.<sup>12</sup> Residual samples from vesicle scrapings obtained from children admitted for varicella or herpes zoster were collected and sent to the Institute for Clinical Pathology and Medical Research at Westmead Hospital in Sydney for genotyping of varicella-zoster virus. Stool samples from children with IS were analysed in local diagnostic laboratories for the presence of rotavirus (including vaccine-derived types), adenovirus and enterovirus. Residual specimens from children hospitalised with acute encephalitis were also collected and tested for unknown pathogens. Laboratory results for cases of influenza, pertussis and encephalitis were also collected and recorded in the PAEDS database.

### Data management and communication

Originally, a purpose-built Microsoft Access database was developed by APSU and deployed to participating hospitals. Since 2013, PAEDS adopted the database 'WebSpirit',<sup>13</sup> which enables online data entry by surveillance nurses at each site. Data are held securely and exported on a regular basis by staff at the PAEDS coordinating centre for clinical review, quality checks, analysis and reporting. Communication is facilitated by joint monthly teleconferences of all PAEDS investigators and nurses, as well as monthly nurse teleconferences. Detailed review of protocols and study outcomes occurs at an annual face-to-face meeting, which also facilitates planning for the introduction of new conditions into PAEDS.

### Results

From August 2007 to December 2014, PAEDS collected data on 6,227 cases of the conditions under surveillance (Table 3). Data on an additional

**Table 3: Cases ascertained through the Paediatric Active Enhanced Disease Surveillance system, August 2007 to December 2014**

Condition	Period of surveillance	Cases ascertained (n)
Acute flaccid paralysis	August 2007 – December 2014	299
Intussusception	August 2007 – December 2014	562
Varicella	August 2007 – December 2014	300
Seizures in infants aged 1–9 months	August 2007 – October 2008	126
Febrile seizures*	May 2013 – June 2014 (prospective)	1,701
	January 2012 – April 2013 (retrospective)	2,013
Pandemic influenza	June 2009 – September 2009	601 total (529 pandemic H1N1)
Pertussis	January 2012 – December 2014	201
Acute childhood encephalitis†	May 2013 – December 2014	140
Influenza (FluCAN)‡	April 2014 – October 2014	284§
Total for all conditions		6,227

\* Retrospective surveillance for febrile seizures was conducted using hospital discharge data (ICD-10-AM coding).<sup>4</sup>

† Children's Hospital at Westmead (Sydney) from 1 May 2013 and all Paediatric Active Enhanced Disease Surveillance sites progressively from 1 January 2014.

‡ Children's Hospital at Westmead (Sydney) and Princess Margaret Hospital (Perth) sites only.

§ 284 hospitalised control cases were also recruited.

284 control cases (influenza test-negative acute respiratory illness cases) were collected under FluCAN surveillance. Key results and impacts of surveillance for all conditions for 2007 to 2014 are summarised in Table 4.

### Surveillance results for 2014

Seven conditions were under surveillance during 2014, including 4 vaccine preventable diseases (AFP, varicella, pertussis and influenza [2 sites,

collaboration with FluCAN]); 2 potential AEFIs (IS and febrile seizures); and another serious disease of childhood, encephalitis. Table 5 shows case numbers for all conditions for 2014 and provides details of auditing and assessment of cases in relationship to ICD-coded hospital discharge data for select conditions. Following the move to operate under a waiver of consent framework, data on cases identified from ICD audit only have also been eligible for inclusion.

**Table 4: Key Paediatric Active Enhanced Disease Surveillance results and impacts, 2007 to 2014**

Condition	Results and impacts
Acute flaccid paralysis	<p>Cases reported to the Polio Expert Panel* for review; at least 1 stool sample collected in 72% of cases†</p> <p>The World Health Organization (WHO) surveillance target reached<sup>12</sup></p> <p>Surveillance contributes to Australia fulfilling polio-free status, as certified by the WHO</p> <p>Paediatric Active Enhanced Disease Surveillance (PAEDS) contributed cases to the WHO surveillance effort for Guillain-Barré syndrome (identified as a potential adverse event following immunisation following pandemic influenza vaccination)<sup>14</sup></p>
Varicella and zoster	<p>Number of hospitalised varicella cases has reduced since the introduction of vaccination onto the National Immunisation Program (NIP)<sup>13</sup></p> <p>Most hospitalised cases not vaccinated against varicella<sup>15</sup></p> <p>Varicella-zoster virus genotyping conducted to monitor for presence of wild and vaccine type strains<sup>15</sup></p>
Intussusception	<p>First global study demonstrating that infants aged &lt;3 months had a slightly increased risk of developing intussusception 1–7 days after the 1st dose of the new rotavirus vaccines.<sup>16</sup> Results confirmed by additional Australian and global studies.<sup>17</sup></p> <p>Informed ongoing risk–benefit analysis for vaccine program, and information for parents and providers on rotavirus vaccine safety developed</p> <p>Ongoing surveillance contributes to maintaining public confidence in rotavirus vaccines</p>
Febrile seizures	<p>Analysis showed known low risk of febrile seizures post measles-mumps-rubella dose 1, but no increased risk of febrile seizures post monovalent varicella vaccine</p> <p>Preliminary analysis to 2013–2014 shows no increased risk of febrile seizures for measles-mumps-rubella-varicella (MMRV) under the NIP (where MMRV is used as the 2nd measles-containing vaccine dose)<sup>18</sup></p> <p>Affirmed safety profile of MMRV as used under the Australian NIP</p>
Pandemic influenza (2009 only)	<p>Approximately 30% of children admitted to hospital with pandemic influenza were previously healthy, while the remainder had a chronic disorder that predisposed them to infection</p> <p>Only 17% of children who had a chronic disorder making them more vulnerable to influenza infection had been vaccinated against influenza<sup>7</sup></p> <p>Named in the National Health and Medical Research Council's 10 of the Best Projects for 2013 (grant number: 633028 under the 2009 Urgent Call for Research on H1N1 Influenza 09 to Inform Public Policy)</p>
Influenza (in collaboration with FluCAN)	<p>Inclusion of paediatric cases in FluCAN from 2014 (n=401 hospitalised cases, 284 from 2 PAEDS sites)</p> <p>Demonstrated good vaccine effectiveness against paediatric influenza hospitalisation<sup>19</sup></p> <p>Demonstrated low vaccine uptake (among control subjects) suggests need to improve influenza immunisation program</p>
Acute childhood encephalitis	<p>Pilot surveillance and protocol development helped to inform comprehensive guidelines for the investigation and management of encephalitis in Australia and New Zealand<sup>20</sup></p> <p>Facilitated rapid investigation and response to enterovirus-71 outbreak and emergence of parechovirus disease in 2013–14, incorporating cases captured by PAEDS surveillance<sup>21,22,23</sup></p>

\* The Polio Expert Panel is a subcommittee of the Communicable Diseases Network Australia. Results of acute flaccid paralysis surveillance are published annually in *Communicable Diseases Intelligence*.

† Although the World Health Organization requires 2 stool samples within 2 weeks of paralysis and at least 24 hours apart, this target is rarely reached in developing countries.<sup>24</sup>

### Influenza

In 2014, 284 paediatric cases of influenza and 284 controls were identified at the Children's Hospital at Westmead (Sydney) and Princess Margaret Hospital (Perth) sites and contributed to FluCAN surveillance. Of these 284 cases, 22 (7.8%) were admitted to the intensive care unit. There were 125 (44%) children who had chronic conditions predisposing them to influenza infection, but only 16 (6.5%) of these had received at least 1 dose of influenza vaccine in the 2014 influenza season.

### Acute flaccid paralysis

The 46 cases of AFP identified in 2014 (rate 44/100,000 children aged <15 years per annum) met the World Health Organization (WHO) AFP surveillance target. At least 1 stool sample was collected within 2 weeks of onset of paralysis for 33 cases (72%), and 2 stool samples were collected for 24 (52%) cases. The most common diagnoses associated with AFP were transverse myelitis (24%) and Guillain-Barré syndrome (39%).

### Intussusception

Of the 52 cases of IS identified in 2014, 12 (23%) had received a rotavirus vaccine in the previous 21 days. Of these 12 children, 3 had IS after the 1st dose of vaccine, 3 after the 2nd dose, and 6 after

the 3rd dose. Two of the 12 children required surgery to correct IS, 6 resolved with air enema and 4 resolved spontaneously. Among all 52 cases of IS, 7 (13.5%) children required surgery, 32 (62%) resolved with an air enema and in 13 (25.0%) cases the IS resolved spontaneously.

### Varicella

Among the 49 cases of varicella, vesicular fluid or vesicle scraping samples were obtained from 25 (51%) cases; in many children sampling was difficult as vesicles had crusted over by the time the child was admitted and approached by the PAEDS nurse. Of the 49 children, 22 (45%) were eligible for NIP-funded varicella vaccination but only 14 had been vaccinated.

### Pertussis

There were 49 children hospitalised with laboratory-confirmed pertussis in 2014. Detailed clinical data on all cases and their contacts and vaccination histories were collected. Seven children required admission to the paediatric intensive care unit. Approximately half (n=25) were under 3 months of age.

### Febrile seizures

In 2014 (January–June), 647 cases of febrile seizures were captured by PAEDS. Active surveil-

**Table 5: Cases recruited to Paediatric Active Enhanced Disease Surveillance in 2014 by condition, number and methods of case ascertainment**

Condition	Case identification methods			Total recruited cases (Surveillance and ICD-10 audit combined)
	Total cases captured via active surveillance	Number captured by PAEDS only, not ICD-coded	Number recruited retrospectively following ICD-10 audit	
Acute flaccid paralysis*	44	15	2	46
Intussusception	43	3	9	52
Varicella-zoster virus	41	8	8	49
Pertussis	45	3	4	49
Febrile seizures†	641	7	6	647
Acute childhood encephalitis‡	93	93	ND	93
Influenza§	284	284	ND	284
Total	1,191	36	29	1,220

ND = not done

\* Acute flaccid paralysis numbers may differ from that published in the Australian Paediatric Surveillance Unit and/or Australian National Enterovirus Reference Laboratory reports due to differences in surveillance systems.

† Febrile seizure surveillance period January – June 2014; Children's Hospital at Westmead (Sydney) audited only, 164 total cases of which 7 were Paediatric Active Enhanced Disease Surveillance (PAEDS) only and 6 added by audit.

‡ Acute childhood encephalitis ICD-10-AM audit incomplete at time of report.

§ Influenza – an additional 284 control cases were recruited at Children's Hospital at Westmead (Sydney) and Princess Margaret Hospital (Perth). These may include some cases separately reported to the Australian Paediatric Surveillance Unit from other sites. No ICD-10-AM audit was carried out on this condition.

lance for this condition concluded on 30 June 2014. Between 1 May 2013 and 30 June 2014, prospective surveillance identified 1,701 FS episodes in 1,471 children aged 0 to <5 years. Of these, 1,335 had only 1 FS and 136 (11%) had 2 or more episodes in the study period. Five hundred and seventy (39%) children with an FS had received MMRV vaccine at any time. PAEDS analysis of the risk of FS in various time periods up to 30 days post MMRV vaccine, using self-controlled case-series analysis, showed no vaccine-associated increase in risk.<sup>18</sup>

### *Acute childhood encephalitis*

Between May 2013 and December 2014, the surveillance identified 140 cases of suspected childhood encephalitis. An analysis of the pilot phase has shown that PAEDS performs very well in detecting cases of childhood encephalitis and has the capacity to identify cases associated with epidemic infectious diseases.<sup>21–23</sup> Approximately 3-quarters of eligible children have been recruited to follow-up studies and over half have had biological specimens salvaged for future analysis. The study is revealing key differences in the clinical features of infectious encephalitis when compared with immune-mediated encephalitis.

## Discussion

PAEDS has provided novel and unique data on hospitalisations due to selected uncommon serious childhood conditions, particularly VPDs and potential AEFI, over the last 7 years. Active case finding by specialist surveillance nurses, and collection of detailed clinical and laboratory data in the same child is unique to PAEDS.<sup>1</sup> This surveillance approach provides a rich and timely source of data that is comprehensive in nature and allows for the collection of demographic details, family history, clinical characteristics, outcome data and analysis of biological specimens, all matched to each individual patient. Such data are not available from other systems. Importantly, our detailed case ascertainment and reporting serves to enrich data collected under other systems. Comparison of PAEDS-ascertained cases with regular audits of hospital discharge data using relevant ICD-10-AM codes is conducted as part of quality assurance processes. These comparisons have shown that case ascertainment yields through PAEDS are high, and more timely than auditing medical records. PAEDS also provides additional cases not otherwise ICD-coded for the condition of interest.

PAEDS surveillance for AFP significantly enhanced surveillance conducted via the APSU and the Australian National Enterovirus Reference

Laboratory and has enabled Australia to meet the WHO AFP surveillance targets for the last 7 years.<sup>12,24</sup> Achieving the WHO stool collection target of 2 stool samples within 2 weeks remains challenging in the context of a modern health system where a non-polio AFP diagnosis is rapidly available.<sup>24</sup> However, PAEDS nurses facilitated collection of at least 1 stool sample in 72% of AFP cases ascertained in 2014.<sup>25</sup>

PAEDS surveillance suggested an excess of IS cases in infants 1–7 days after receipt of the 1st dose of either of the new rotavirus vaccines currently used in Australia, the first study worldwide to describe this link.<sup>16</sup> These data informed vaccination policy and practice, stimulated additional studies and resulted in the development of educational materials for parents and vaccine providers.<sup>17</sup> Analysis of the more than 500 IS cases for which PAEDS holds detailed clinical data is underway to compare the clinical characteristics of vaccine proximate cases with non-vaccine proximate cases.

The number of hospitalised cases of varicella-zoster virus has reduced with increased uptake of varicella vaccination.<sup>15</sup> Nevertheless, the majority of children (71%) hospitalised due to varicella-zoster virus infection were not vaccinated for varicella, despite being eligible under the NIP. These data support the continuation of the population-based funded varicella vaccination program in Australia, and current efforts to increase varicella vaccine coverage, such as via the inclusion of MMRV vaccine onto the NIP.

PAEDS also conducted a high intensity, short-term study of FS following measles-containing vaccines, to support the vaccine safety plan for the introduction of MMRV onto the NIP. Retrospective and prospective surveillance identified data on more than 3,700 FS presentations and, using vaccine data from the Australian Childhood Immunisation Register, we were able to analyse the risk of FS following MMR, varicella and MMRV vaccines.<sup>4</sup> The absence of an increased risk of FS following MMRV vaccine supports ongoing use of this vaccine as the 2nd dose of measles-containing vaccine at 18 months of age under the NIP.

PAEDS has the capacity to rapidly respond to disease outbreaks as shown by surveillance for influenza during the H1N1-09 pandemic,<sup>7</sup> contributions towards enterovirus 71<sup>22</sup> and parechovirus<sup>21,23</sup> outbreak investigations and, from 2014, PAEDS continues to contribute paediatric data to the influenza surveillance efforts in Australia through the collaboration with FluCAN.<sup>9</sup> PAEDS data highlights the need for improved uptake of influenza vaccination in children, particularly those who have predisposing chronic conditions.<sup>19</sup>

PAEDS reliably collects demographic details such as ethnicity, enabling potential analysis of subgroups of children with greater susceptibility to severe disease and missed opportunities for disease prevention, including missed or late immunisation. PAEDS collects laboratory data that is directly linked to clinical details and vaccination history for the same child, enabling the description of relationships between genetic subtypes and disease severity or vaccine failures. Such data are important to support development of immunisation policy and for maintaining consumer and provider confidence in the NIP. However, collection of biological samples can be challenging for a range of reasons. For example, a child might be admitted after varicella vesicles have crusted over and taking a sample of vesicle fluid is not possible, or a patient with AFP may be unable to produce a stool sample within the prescribed time period and before they are discharged from hospital.

Currently, PAEDS operates in 5 tertiary paediatric hospitals based in large metropolitan centres, limiting surveillance coverage to populations served by these hospitals. Despite this, we estimate that approximately 70% of all paediatric admissions to tertiary paediatric services are covered by PAEDS. Further expansion, especially to hospitals in northern Australia which serve Aboriginal and Torres Strait Islander populations, would enhance coverage in these vulnerable populations. Not all tertiary paediatric hospitals in New South Wales and Victoria participate in PAEDS and coverage could be significantly enhanced by including these hospitals.

PAEDS is an important capacity building initiative to enhance existing public health surveillance for VPDs and AEFIs, with the overarching aim of improving child health outcomes. This unique surveillance platform also has the potential to be used for other urgent or research focused studies, for which active surveillance is optimal. More information on PAEDS is available on the [PAEDS web site](http://www.paeds.edu.au) (www.paeds.edu.au).

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# ARBOVIRAL DISEASES AND MALARIA IN AUSTRALIA, 2013–14: ANNUAL REPORT OF THE NATIONAL ARBOVIRUS AND MALARIA ADVISORY COMMITTEE

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## Abstract

This report describes the epidemiology of mosquito-borne diseases of public health importance in Australia during the 2013–14 season (1 July 2013 to 30 June 2014) and includes data from human notifications, sentinel chicken, vector and virus surveillance programs. The National Notifiable Diseases Surveillance System received notifications for 8,898 cases of disease transmitted by mosquitoes during the 2013–14 season. The Australasian alphaviruses Barmah Forest virus and Ross River virus accounted for 6,372 (72%) total notifications. However, over-diagnosis and possible false positive diagnostic test results for these 2 infections mean that the true burden of infection is likely overestimated, and as a consequence, the case definitions have been amended. There were 94 notifications of imported chikungunya virus infection and 13 cases of imported Zika virus infection. There were 212 notifications of dengue virus infection acquired in Australia and 1,795 cases acquired overseas, with an additional 14 cases for which the place of acquisition was unknown. Imported cases of dengue were most frequently acquired in Indonesia (51%). No cases of locally-acquired malaria were notified during the 2013–14 season, though there were 373 notifications of overseas-acquired malaria. In 2013–14, arbovirus and mosquito surveillance programs were conducted in most jurisdictions. Surveillance for exotic mosquitoes at international ports of entry continues to be a vital part of preventing the spread of vectors of mosquito-borne diseases such as dengue to new areas of Australia, with 13 detections of exotic mosquitoes at the ports of entry in 2013–14. *Commun Dis Intell* 2016;40(3):E401–E436.

**Keywords:** arbovirus; Barmah Forest virus, chikungunya, dengue, disease surveillance, epidemiology, flavivirus, Kunjin virus, Japanese encephalitis, West Nile virus, malaria, mosquito-borne, mosquitoes, Murray Valley encephalitis virus, Ross River virus, yellow fever, West Nile virus

## Introduction

This report describes the epidemiology of mosquito-borne diseases of public health impor-

tance in Australia during the period 1 July 2013 to 30 June 2014. It includes a summary of notified cases of disease caused by the alphaviruses Barmah Forest virus (BFV), chikungunya virus (CHIKV) and Ross River virus (RRV); the flaviviruses dengue virus (DENV), Murray Valley encephalitis virus (MVEV), West Nile virus (WNV) and the Kunjin lineage of West Nile virus (KUNV), Japanese encephalitis virus (JEV) and yellow fever virus (YFV); and malaria. Both locally acquired and overseas acquired cases are described. Vector, climate and sentinel chicken surveillance measures for arboviruses conducted by states and territories, and also at the international first ports of entry are described.

The National Arbovirus and Malaria Advisory Committee (NAMAC) provides expert technical advice on arboviruses and malaria to the Australian Health Protection Principal Committee through the Communicable Diseases Network Australia (CDNA). Members of NAMAC have expertise in virus and disease surveillance, epidemiology, virology, vector ecology, vector and disease control and quarantine, and represent agencies with a substantial interest in this area. NAMAC makes recommendations about surveillance and reporting systems, strategic approaches for disease and vector management and control, and laboratory support and outlines research priorities. NAMAC assists in the prevention, detection, management and control of outbreaks of arboviruses or malaria and provides advice on the risk posed to Australia by these viruses or exotic vectors that may be imported from overseas. NAMAC members participate in or advise outbreak management teams as required.

## Methods

Human cases of arbovirus infection and malaria are monitored using the National Notifiable Diseases Surveillance System (NNDSS). All Australian states and territories require doctors and/or pathology laboratories to notify cases of infectious diseases that are important to public health. The *National Health Security Act 2007* (NHS Act 2007) provides the legislative basis for the national notification of communicable diseases

and authorises the exchange of health information between the Commonwealth and the states and territories. The NHS Act 2007 provides for the establishment of the National Notifiable Diseases List, which specifies the diseases about which personal information can be exchanged between the states and territories and the Commonwealth. State and territory health departments transfer these notifications regularly to the NNDSS. The primary responsibility for public health action resulting from a notification resides with state and territory health departments.

This report presents case data from a snap-shot of NNDSS taken during July 2015 and analysed by date of diagnosis. This derived field is the onset date, or where the date of onset was not known, for vectorborne diseases, it is the earliest of the specimen collection date, the notification date, or the notification received date. Since the data are from a snap-shot, numbers in this report may vary slightly from those reported elsewhere due to changes in diagnostic validation or classification. Data were verified with state and territory public health surveillance managers. Detailed notes on the interpretation of NNDSS are available in the 2014 NNDSS annual report.<sup>1</sup> Case definitions for the diseases included in this report are available on the [Australian Government Department of Health web site](http://www.health.gov.au/casedefinitions) (<http://www.health.gov.au/casedefinitions>). The report includes information on the following nationally notifiable pathogens that are transmitted by mosquitoes:

- alphaviruses (BFV, RRV, and CHIKV);
- flaviviruses (DENV, JEV, WNV/KUNV, MVEV, YFV and unspecified, including Zika virus (ZIKV)); and
- malaria.

CHIKV infection was made nationally notifiable in 2015, though a national case definition was implemented from 2010. Prior to this, CHIKV infections were notified under the disease category arbovirus NEC, and all notifications have now been included under CHIKV in NNDSS.

Data were analysed by financial year to reflect the seasonal cycle of arboviral activity in most areas of Australia. Crude notification rates or counts for the 2013–14 season were compared with those recorded over the previous 5 years. Notification rates were not calculated for diseases that are primarily acquired overseas because resident populations are not an appropriate denominator. Rates are not provided for rare diseases ( $n < 20$  notifications for the year) because these rates typically have large standard errors and therefore cannot be meaningfully compared across time or geographical location.

Notification rates were calculated using the Australian Bureau of Statistics (ABS) estimated resident populations for Australia and each state or territory at June 2013.<sup>2</sup> Population data are supplied as an estimate for calendar years; for this report, the population for the second half of the financial year was applied to that year (2014 population applied to the 2013–14 financial year). Additional spatial analyses were performed using the ABS Statistical Area level 3 classifications,<sup>3</sup> and using ABS defined ratios to allocate notifications by their postcode of residence to a statistical area. Analyses were conducted using Microsoft Excel® and Stata SE version 13. The nonparametric test for trend in Stata was used to analyse trends in notifications over time where relevant, using  $P < 0.05$  to indicate a significant trend. Maps were produced using Arc GIS (ESRI).

Additional information on the details of some notifications were obtained from state and territory public health surveillance managers. Data on sentinel chicken surveillance, vector (including detection of exotic mosquitoes at International ports of entry, hereafter referred to as the border) and virus surveillance are also reported.

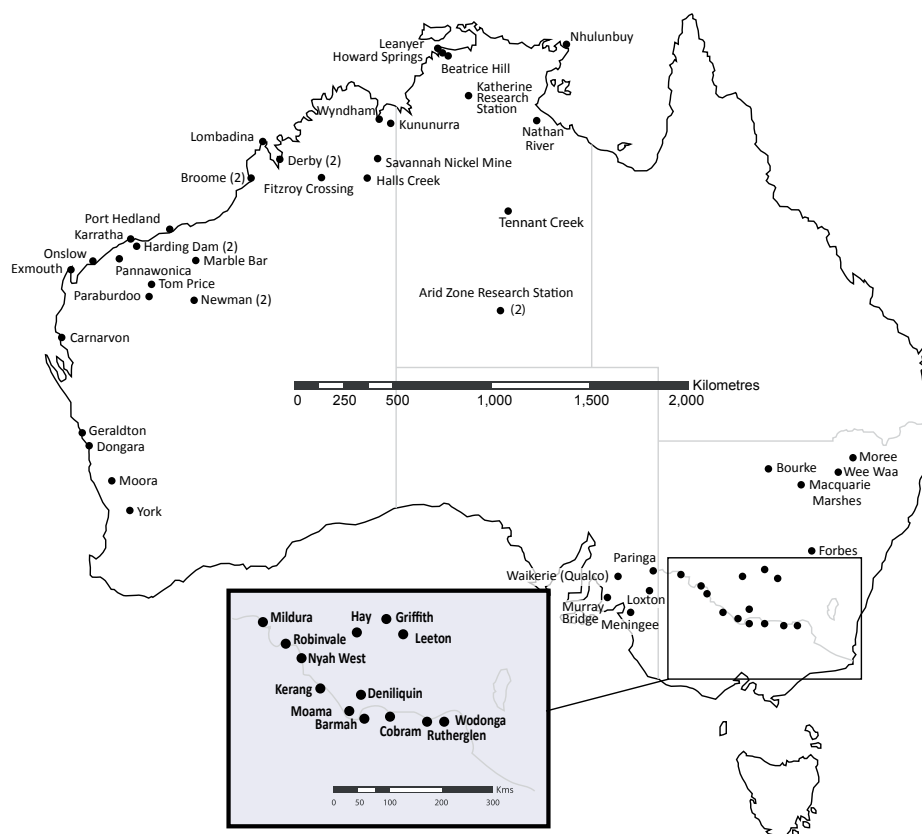
### Vertebrate, vector and climate surveillance in states and territories

Sentinel chicken flavivirus surveillance programs aim to provide early warning of the endemic arboviruses MVEV and KUNV as well as exotic flaviviruses such as JEV.<sup>4</sup> Public health messaging or other response measures can be implemented in response to surveillance signals. Public health messaging may advise at-risk residents or target groups such as campers or fishermen of the need to take added precautions to avoid mosquito bites. Sentinel chicken flocks are an important component of the early warning system in several jurisdictions, and these are located geographically to detect flavivirus activity and provide a timely and accurate indication of the risk of transmission to people (Map 1).<sup>5</sup> Detailed descriptions of the sentinel chicken, vector and virus surveillance programs, as well as contact details for jurisdictional arbovirus reference or research laboratories are included in the Appendix.

## Results

During the 2013–14 season, there were 8,898 notifications of mosquito-borne diseases in humans (Table 1). This represented a 3% increase from the mean of 8,628.4 notifications for the previous 5 years.

Map 1: Location of sentinel chicken sites, Australia, 2013–14



## Alphaviruses

In Australia, the most frequently notified viruses in the genus *Alphavirus* are RRV and BFV. RRV and BFV occur exclusively in the Australasian region.<sup>6</sup> Infection with RRV or BFV can cause illness characterised by fever, rash and polyarthrits. These viruses are transmitted by numerous species of mosquitoes that breed in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).<sup>7</sup> However, there are known problems with the unreliability of serological tests that diagnose infection on the basis of IgM only and with the case definitions that allow for confirmation based on these tests, leading to over diagnosis particularly during the off-season.<sup>8</sup> Importantly, the case definitions have been reviewed by the Case Definitions Working Group of CDNA, and these changes were implemented on 1 January 2016.<sup>9</sup>

Local transmission of the alphavirus CHIKV has not occurred in Australia, but the infection is regularly reported in travellers returning from overseas. The illness is characterised by an abrupt onset of fever, rash and severe joint pain. The acute disease lasts 1 to 10 days, but convalescence may include prolonged joint swelling and pain lasting months. Haemorrhagic manifestations may occur occasionally.<sup>10</sup> Humans are amplification hosts for

CHIKV and other vertebrates are not required for transmission to occur. There is the potential for transmission of CHIKV in areas where a suitable mosquito vector exists. Internationally, CHIKV is most commonly transmitted by *Aedes aegypti* and *Ae. albopictus*. In Australia, *Ae. aegypti* is present in parts of Northern, Central and South West Queensland and *Ae. albopictus*, which is found on Cocos Island, Christmas Island and in some areas of the Torres Strait Islands.<sup>11</sup> Other Australian mosquito species have been shown to be competent vectors of CHIKV in the laboratory,<sup>12</sup> but any role in field transmission is likely to be minor compared with either *Ae. aegypti* or *Ae. albopictus*.<sup>13</sup>

### Barmah Forest virus infections

There were 1,803 notifications of BFV infections during the 2013–14 season, representing a rate of 7.7 per 100,000 population, a decrease from the mean of 2,060.6 cases (9.1 per 100,000) for the previous 5 years (Table 1, Figure 1). Queensland reported the largest number of notifications of BFV infection (n=1,115) while the highest rate was reported in the Northern Territory (52.7 per 100,000 population) (Figure 2). Rates in 2013–14 were below the 5-year mean for all states and territories. It is important to note that seasonal trends vary between and within states and territories

**Table 1: Number of notified human cases, notification rate\* and 5-year mean for mosquito-borne disease, Australia, 2013–14, by disease and state or territory**

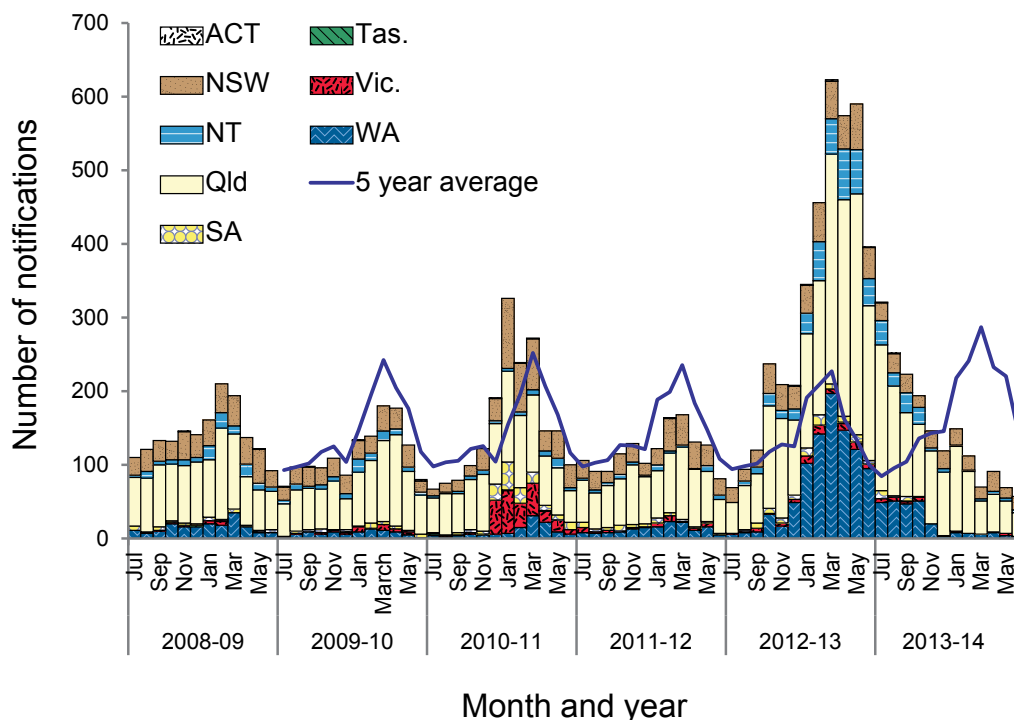
		ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust
Barmah Forest virus infection	Cases 2013–14	2	254	129	1,115	20	1	25	257	1,803
	5-year mean cases	3.2	381.2	136.4	1,099.2	69.0	2.0	76.4	293.2	2,060.6
	Rate 2013–14	0.5	3.4	52.7	23.6	1.2	0.2	0.4	10.0	7.7
	5-year mean rate	0.9	5.2	58.0	24.1	4.2	0.4	1.4	12.0	9.1
Chikungunya virus infection	Cases 2013–14	0	22	2	8	5	0	20	37	94
	5-year mean cases	0.0	9.6	2.8	5.0	2.8	0.6	14.4	13.0	48.2
	Rate 2013–14	–	–	–	–	–	–	–	–	–
	5-year mean rate	–	–	–	–	–	–	–	–	–
Dengue virus infection	Cases 2013–14	20	421	69	461	82	23	414	531	2,021
	5-year mean cases	15.6	206.4	40.2	448.2	31.8	6.4	159.6	352.2	1,260.4
	Rate 2013–14	–	–	–	–	–	–	–	–	–
	5-year mean rate	–	–	–	–	–	–	–	–	–
Flavivirus unspecified†	Cases 2013–14	0	4	0	27	0	0	1	0	32
	5-year mean cases	0.0	0.2	0.2	3.6	0.2	0.0	5.8	0.0	10.0
	Rate 2013–14	–	–	–	–	–	–	–	–	–
	5-year mean rate	–	–	–	–	–	–	–	–	–
Japanese encephalitis virus infection	Cases 2013–14	0	0	0	2	0	0	0	0	2
	5-year mean cases	0.0	0.2	0.0	0.2	0.2	0.0	0.0	0.2	0.8
	Rate 2013–14	–	–	–	–	–	–	–	–	–
	5-year mean rate	–	–	–	–	–	–	–	–	–
West Nile virus/Kunjin virus infection	Cases 2013–14	0	0	0	2	0	0	1	0	3
	5-year mean cases	0.0	0.2	0.6	0.6	0.0	0.0	0.0	0.0	1.4
	Rate 2013–14	–	–	–	–	–	–	–	–	–
	5-year mean rate	–	–	–	–	–	–	–	–	–
Malaria	Cases 2013–14	8	105	17	88	8	5	84	58	373
	5-year mean cases	11.8	92.4	17.2	133.0	13.2	7.6	90.8	67.6	433.6
	Rate 2013–14	–	–	–	–	–	–	–	–	–
	5-year mean rate	–	–	–	–	–	–	–	–	–
Murray Valley encephalitis virus infection	Cases 2013–14	0	0	0	0	0	0	0	0	0
	5-year mean cases	0.0	0.6	0.6	0.4	0.4	0.0	0.0	2.2	4.2
	Rate 2013–14	–	–	–	–	–	–	–	–	–
	5-year mean rate	–	–	–	–	–	–	–	–	–
Ross River virus infection	Cases 2013–14	5	509	434	1,845	111	19	161	1,485	4,569
	5-year mean cases	10.6	766.4	286.4	1,904.0	436.0	19.8	451.0	934.6	4,808.8
	Rate 2013–14	1.3	6.8	177.4	39.1	6.6	3.7	2.8	57.9	19.5
	5-year mean rate	2.9	10.6	124.1	42.0	26.4	3.9	8.0	39.2	21.4
Yellow fever	Cases 2013–14	0	0	0	0	0	0	0	0	0
	5-year mean cases	0	0	0	0.3	0	0	0	0	0
	Rate 2013–14	–	–	–	–	–	–	–	–	–
	5-year mean rate	–	–	–	–	–	–	–	–	–
Total 2013–14		35	1,315	651	3,549	226	48	706	2,368	8,898

\* Rates are not provided for diseases with less than 20 cases, or for diseases predominantly acquired overseas.

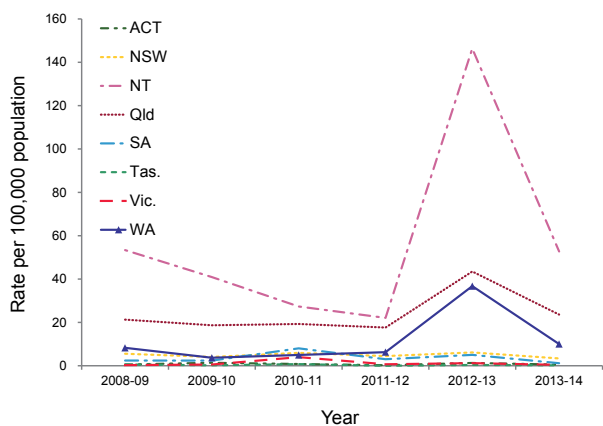
† Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004. Arbovirus (NEC) replaced Flavivirus (NEC) from 2008. Flavivirus (unspecified) replaced arbovirus (NEC) from 14 January 2015.

NEC Not elsewhere classified.

**Figure 1: Notifications of Barmah Forest virus infection, Australia, 1 July 2008 to 30 June 2014, by month and year, and state or territory**



**Figure 2: Notification rate for Barmah Forest virus infection, Australia, 1 July 2008 to 30 June 2014, by year and state or territory**



according to differences in mosquito vectors, hosts and climate. In addition, comparisons between regions are likely to be influenced by accuracy of case-ascertainment, which may vary between jurisdictions because of some differences in reporting criteria and the quality of diagnostic tests used, with false positive IgMs a long term issue.

Rates of BFV in 2013–14 by Statistical Area Level 3 were highest in Litchfield, surrounding Darwin, (133 per 100,000), Innisfail in Queensland (85 per 100,000) and Nambour-Pomona on the Sunshine Coast, Queensland (81 per 100,000) (Map 2). Rates were lower in 2013–14 than in the previ-

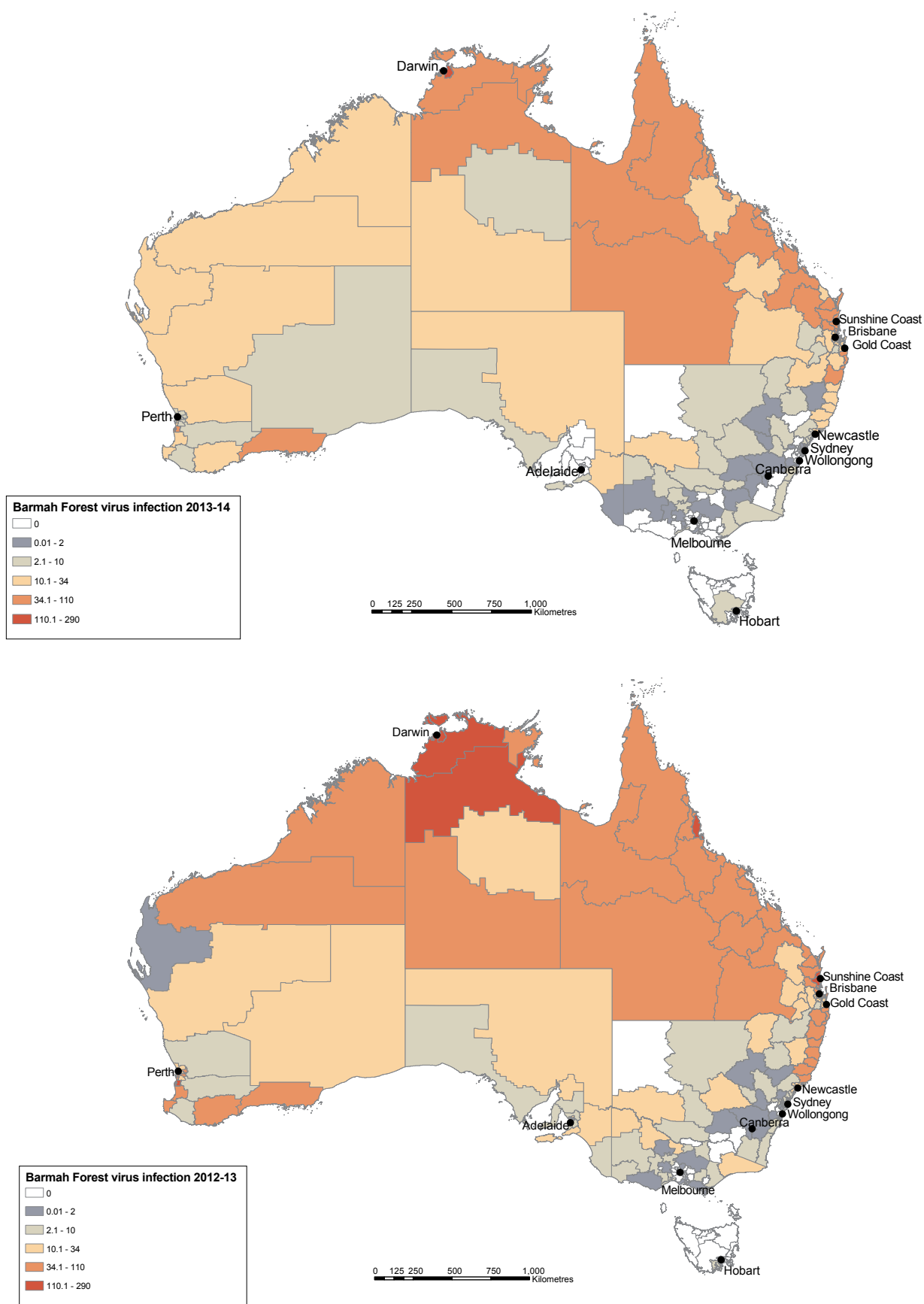
ous year in almost all Statistical Areas with some exceptions, including Burnett (west of Bundaberg and Maryborough, Queensland) and Gascoyne (Western Australia).

In 2013–14, BFV notifications were most common among adults, with notification rates peaking in the 35–59 years age groups for women and 40–54 years age groups for men (Figure 3). There was a secondary peak in younger females in the age groups between 15 and 34 years, similar to that observed in 2012–13. In 2013–14, 42% of cases were male, which was similar to 2012–13 (41%) but lower than the 5 years prior to that (51% to 53%).

**Figure 3: Notification rate for Barmah Forest virus infection, Australia, 2013–14, by age group and sex (n=1,803)**



Map 2: Notification rates for Barmah Forest virus infection, 2013–14, and 2012–13, by Statistical Area Level 3



BFV infections are unexpected outside of the warmer months when suitable mosquito vectors are abundant. In 2013–14, infections were most frequently notified between July and January. This was due to the continuation of an epidemic of false positive IgM diagnoses that was reported previously, and which began in October 2012 and was associated with inaccuracies with the commercial BFV serological test kits (Figure 1).

**Ross River virus infections**

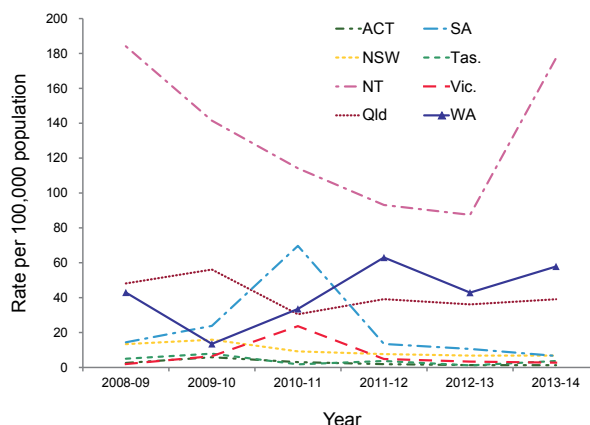
There were 4,569 notifications of RRV infection during the 2013–14 season, representing a rate of 19.5 per 100,000 population, compared with a 5-year mean of 4,808.8 notifications (21.4 per 100,000) (Table 1, Figure 4). Queensland reported the largest number of cases (n=1,845), while the highest rate was in the Northern Territory (177.4 per 100,000).

Rates of RRV were 1.4 and 1.5 times the 5-year mean in the Northern Territory and Western Australia respectively (Figure 5). Rates of RRV in 2013–14 were highest in Litchfield, surrounding Darwin (501 per 100,000), Esperance (238 per 100,000) and the Kimberley (215 per 100,000), and rates were higher across much of Western and Northern Australia than in 2012–13 including in Litchfield (surrounding Darwin), Katherine (Northern Territory), the Kimberley and the Pilbara (Western Australia), Noosa and Nambour-Pomona (Queensland) (Map 3).

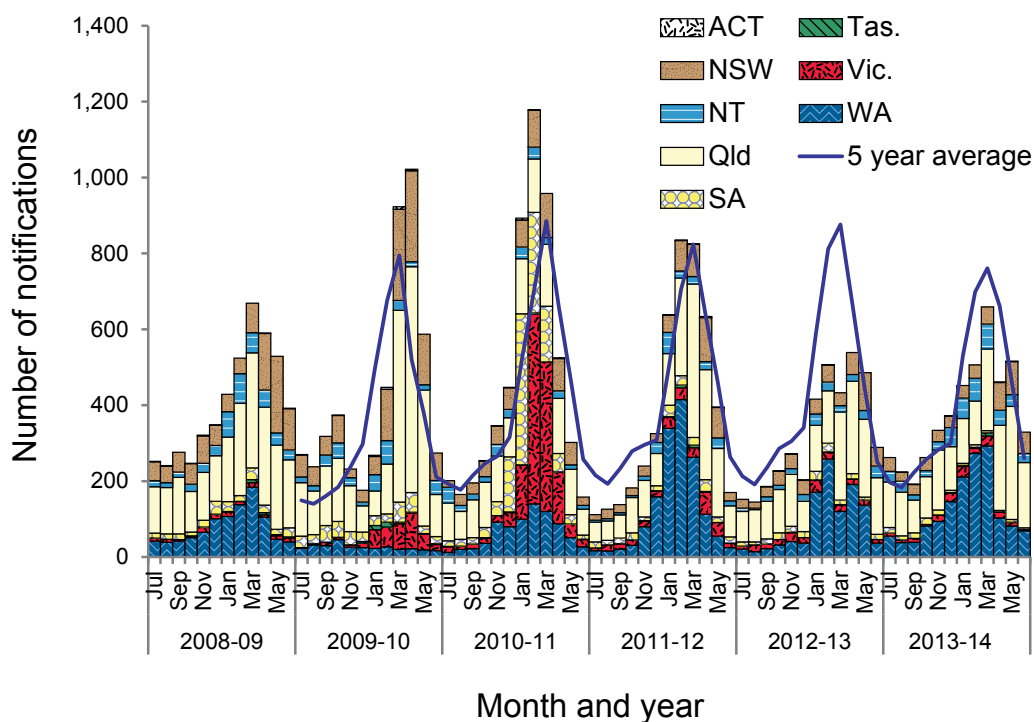
RRV was most commonly reported among adults, with notification rates peaking in the 35–49 years age groups (Figure 6). In 2013–14, 47% of notifications were in males, similar to previous years.

As in previous years, there was a marked seasonal trend in RRV notifications, with the largest number notified between February and May (Figure 4). It is important to note that as for BFV, seasonal trends vary between and within states and territories according to differences in mosquito vectors, hosts and climate. In addition, as for BFV, comparisons between regions are likely to be influenced by accuracy of case-ascertainment, which may vary between jurisdictions because of some differences

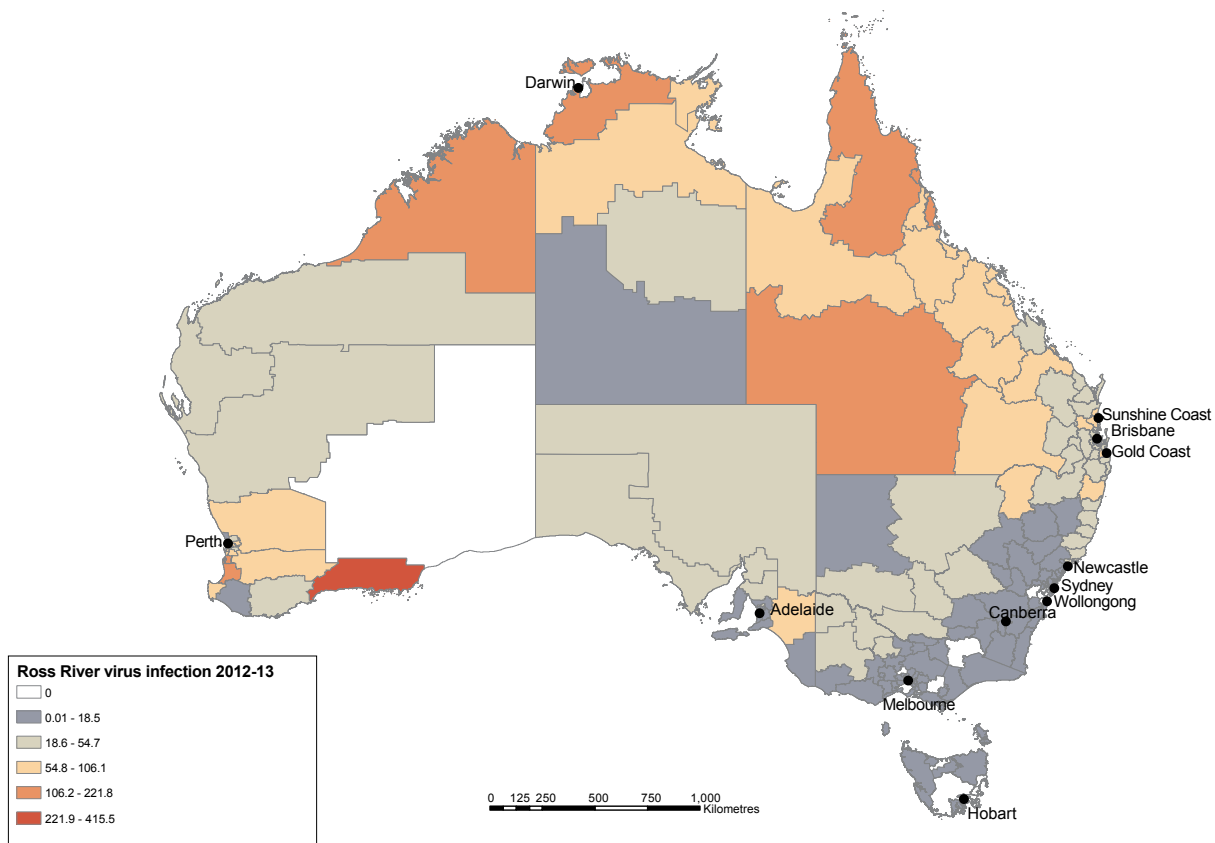
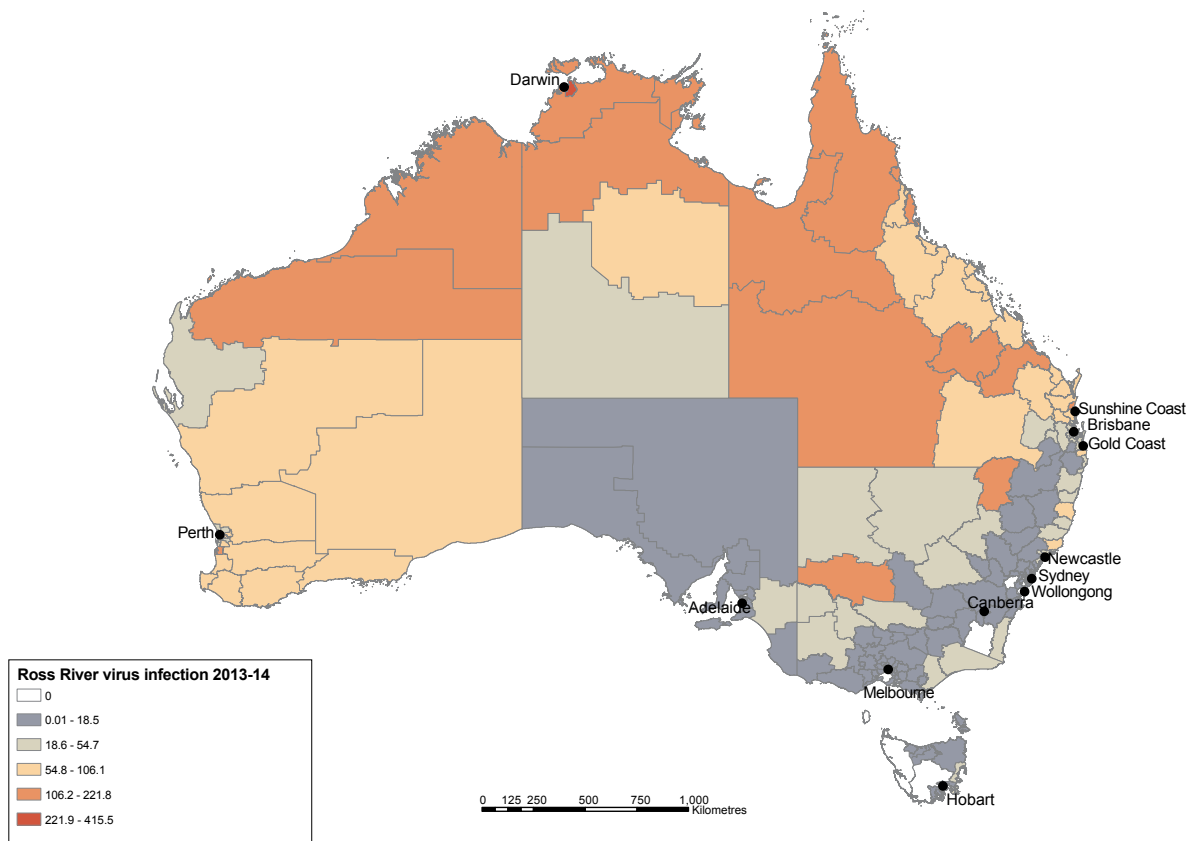
**Figure 5: Notification rate for Ross River virus infection, Australia, 1 July 2008 to 30 June 2014, by year and state or territory**



**Figure 4: Notifications of Ross River virus infection, 1 July 2008 to 30 June 2014, by month and year and state or territory**



Map 3: Notification rates for Ross River virus infection, 2013–14 and 2012–13, by Statistical Area Level 3





in reporting criteria and the quality of diagnostic tests used, with false positive IgM diagnoses a long term issue.<sup>8,14</sup>

**Chikungunya virus infection**

There were 94 notifications of CHIKV infection during the 2013–14 season compared with a 5–year mean of 48.2 cases, and similar to the 96 cases in 2012–13 (Table 1, Table 2, Figure 7) when the largest number ever were reported. All cases were

acquired overseas, with specific information supplied on the country or region of acquisition for 78% (73/94) of these cases while the remainder were reported as overseas-acquired, but the specific country was not known (Table 2). For cases with a known country of acquisition, the most frequently reported countries of acquisition in 2013–14 were Indonesia (47 cases, 64%) and India (10 cases, 14%). Outbreaks of chikungunya were reported from multiple countries in the South Pacific during 2013–14,<sup>15</sup> but there were only 9 importations from the region (7 from Tonga and 2 from Papua New Guinea). An outbreak in Tonga was first reported in April 2014 on PacNet, the Pacific Public Health Surveillance Network early warning system.<sup>16</sup>

**Figure 6: Notification rate for Ross River virus infection, Australia, 2013–14, by age group and sex (n=4,568)\***



\* Sex for 1 notification was not available and this notification is excluded.

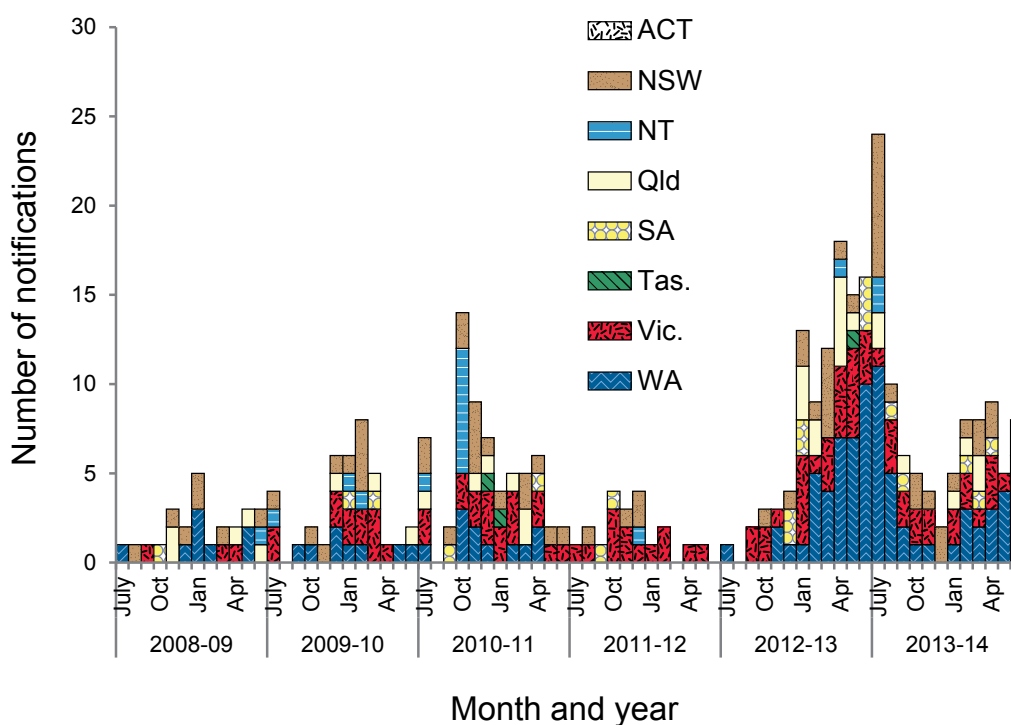
CHIKV infection was most frequently notified among young and middle aged adults (Figure 8). The median age was 46 years and 45% per cent of cases were male.

**Flaviviruses**

This section provides information on several flaviviruses notified to NNDSS including DENV, MVEV, WNV/KUNV and JEV. Other flaviviruses, including ZIKV may be notified under the flavivirus (unspecified) category.

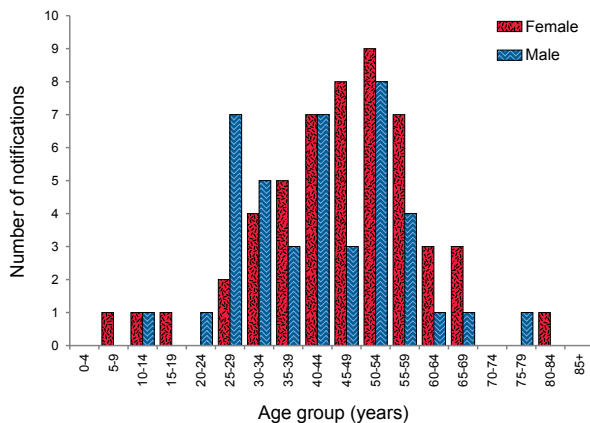
Four serotypes of dengue virus have been described and all 4 are reported in imported cases to varying degrees each year, some of which may result in local

**Figure 7: Notifications of chikungunya virus infection, Australia, 2013–14, by month and year and state or territory**



**Table 2: Notifications of chikungunya virus infection, Australia, by year and country or region of acquisition**

Country or region of acquisition	2009–10	2010–11	2011–12	2012–13	2013–14
Indonesia	7	32	2	34	47
India	14	11	6	2	10
Tonga	0	0	0	0	7
Philippines	1	0	2	2	3
Papua New Guinea	0	2	0	13	2
Singapore	0	1	0	0	2
Thailand	0	2	3	2	1
Nepal	0	0	0	0	1
Other countries/regions	14	13	5	4	0
Overseas-country unknown	1	2	2	39	21
Total	37	63	20	96	94

**Figure 8: Notifications of chikungunya virus infection, Australia, 2013–14, by age and sex**

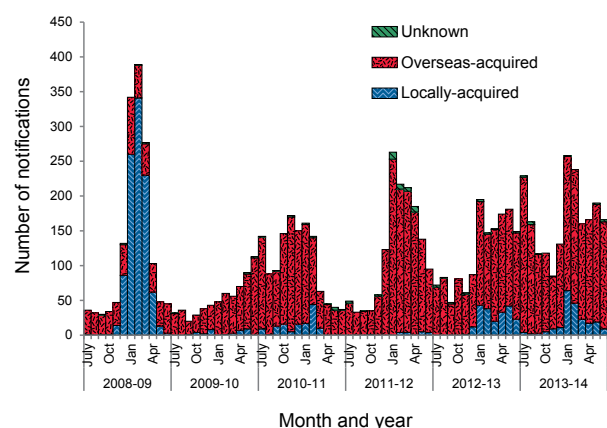
outbreaks. The clinical illness is characterised by mild to severe febrile illness with fever, headache, muscle or joint pain and sometimes a rash. A minority of cases progress to severe dengue with haemorrhage and shock, more commonly where, in a second or subsequent infection, a person is infected with a different DENV serotype to the first infection. Local transmission of dengue in Australia is restricted to areas of northern Queensland where the key mosquito vector, *Ae. aegypti* is present in sufficient numbers and with human populations of sufficient density.<sup>17</sup> Dengue is not endemic in north Queensland, but local transmission can occur upon introduction of the virus to the mosquito vector by a viraemic tourist or a resident returning from a dengue-affected area overseas.<sup>18</sup>

Infection with MVEV, KUNV or JEV is usually asymptomatic or produces a non-specific illness, but a small percentage of cases progress to encephalomyelitis of variable severity. *Cx. annulirostris* is the major vector of MVEV, KUNV and JEV. No specific treatment is available for these

diseases and care is largely supportive. A vaccine is available to prevent JEV infection (available for residents in areas of Queensland where there is a risk of acquiring JEV and for long term travellers to endemic areas),<sup>19</sup> but there are no vaccines currently available for DENV, MVEV or KUNV. YFV does not occur in Australia, but travellers to affected areas overseas need to be aware of the risks and vaccination requirements, and there is the potential for transmission in the areas of north Queensland where the vector *Ae. aegypti* is present.

### Dengue virus infection

There were 2,021 notifications of DENV infection during the 2013–14 season. Of these, 212 cases were acquired in Australia, while the majority (1,795 cases) acquired the infection overseas (Table 3, Figure 9). For the remaining 14 cases, no information on place of acquisition was supplied.

**Figure 9: Notifications of dengue virus infection, Australia, 1 July 2008 to 30 June 2014, by month, year and place of acquisition**

**Table 3: Notifications of dengue virus infection, Australia, 1 July 2008 to 30 June 2014, by year, state or territory and place of acquisition**

Place of acquisition	Year	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Locally-acquired*	2008–09	0	5	0	1,003	1	0	3	0	1,012
	2009–10	0	2	0	33	0	0	0	0	35
	2010–11	0	2	1	125	0	0	2	1	131
	2011–12	0	1	0	16	0	0	1	0	18
	2012–13	0	0	0	206	2	0	4	0	212
	2013–14	0	2	0	202	2	0	5	1	212
Overseas-acquired	2008–09	14	169	27	115	26	6	19	121	497
	2009–10	19	121	36	126	11	4	52	226	595
	2010–11	4	222	29	181	28	5	140	525	1,134
	2011–12	11	240	69	209	44	9	246	561	1,389
	2012–13	12	257	38	216	47	8	299	325	1,202
	2013–14	14	417	69	259	80	23	403	530	1,795
Unknown	2008–09	0	0	0	5	0	0	1	0	6
	2009–10	0	3	0	1	0	0	1	0	5
	2010–11	8	2	1	2	0	0	0	1	14
	2011–12	6	2	0	0	0	0	28	0	36
	2012–13	4	6	0	3	0	0	2	1	16
	2013–14	6	2	0	0	0	0	6	0	14
Total	2008–09	14	174	27	1,123	27	6	23	121	1,515
	2009–10	19	126	36	160	11	4	53	226	635
	2010–11	12	226	31	308	28	5	142	527	1,279
	2011–12	17	243	69	225	44	9	275	561	1,443
	2012–13	16	263	38	425	49	8	305	326	1,430
	2013–14	20	421	69	461	82	23	414	531	2,021

\* Locally-acquired cases are acquired in Australia and not necessarily in the state or territory from which they are reported. Under the cross-border notification protocol, cases are notified by their state or territory of residence where this differs from the diagnosing state or territory.

In 2013–14, the median age of cases was 39 years (range 0 to 85 years), and 51% (n=1,023) of cases were male.

#### Locally-acquired dengue virus infection

The 212 notified cases of DENV infection acquired in Australia during 2013–14 was the same number as that notified in 2012–13. Of these, 202 were reported by Queensland and 10 from other states.

In Queensland, a single case of locally-acquired dengue is considered to be an outbreak. Five dengue outbreaks were identified by Queensland Health in the 2013–14 season, all located in the north of the state. A total of 206 dengue notifications were known to have been associated with these outbreaks, with cases in each outbreak ranging from 8 to 135 (note: data extracted from the Queensland notifiable disease system; these numbers do not match exactly with the 202 reported

from NNDSS due to differences in the dates used for data extraction). Four of the 5 outbreaks were serotype 1, including the largest. The remaining outbreak was serotype 3, which had 12 associated notifications. From 2010 to 2014, dengue serotype 1 has been the identified serotype in nearly 60% of dengue outbreaks in Queensland and 73% of all locally-acquired dengue notifications that were typed. In 2013–14, 57% of locally-acquired dengue notifications were typed.

Eight notifications of locally-acquired dengue from other states were listed in NNDSS as being acquired in Queensland. Two other locally-acquired cases were reported that were not associated with outbreaks in Queensland:

- a case that was acquired in or near Point Sampson, Western Australia from an unknown source. Extensive investigations did not find any evidence of a local vector and there were no

further cases. This was the first locally-acquired case in Western Australia since the 1940s, and was thought most likely to have resulted from the importation of an infected mosquito on cargo or luggage, which bit the patient, but did not survive to lay eggs.<sup>20</sup>

- a laboratory-acquired infection in New South Wales.

#### Overseas-acquired dengue virus infection

There were 1,795 notifications of DENV infection acquired overseas during the 2013–14 season (Table 3), 1.9 times the 5-year mean of overseas-acquired infections (963.4). All states and territories reported increased numbers of overseas-acquired DENV infection compared with the long-term average. The ratio of notifications in 2013–14 compared with the 5-year mean ranged from 1.2 in the Australian Capital Territory to 3.6 in Tasmania.

A specific country or region of acquisition was supplied for 89% (1,602/1,795) of cases listed as overseas-acquired (Table 4). Indonesia was the country of acquisition for more than half of the overseas acquired cases for which a specific country or region was available (51%, n=817). The infecting DENV serotype was determined for 46% (n=820) of overseas-acquired dengue cases (an increase from 42% in 2012–13, and 23% in 2011–12). DENV 1 (n=432) was the most frequently reported serotype in 2013–14 for overseas-acquired cases (Table 4).

#### *Flavivirus (unspecified)*

This disease category enables the capture and epidemiological analysis of emerging infections within this very broad disease group. Emerging diseases can be made nationally notifiable if required, according to the Protocol for making a change to the National Notifiable Diseases List in Australia, which is available on the Department of Health website. An unspecified category is particularly important for the flaviviruses, because it is recognised that some infections cannot be attributed to a single flavivirus.

There were 32 notifications of flavivirus (unspecified) in 2013–14, 3.2 times the 5-year mean of 10.0 notifications. Thirteen of these notifications were for Zika virus (ZIKV) infection acquired in the Pacific Islands countries or territories; the Cook Islands (12 cases) and Samoa (1 case) (Table 5). Outbreaks of ZIKV in the Pacific Islands were first reported in Yap State Micronesia in 2007,<sup>21</sup> and then on PacNet<sup>16</sup> in February 2013 in the Cook Islands, and later New Caledonia and French Polynesia. These outbreaks continued to mid-2014.

The largest number of notifications were from Queensland (n=27). In Queensland, an extensive panel of flaviviruses is used for testing. Flaviviruses may be more prevalent particularly in the north of the State, so patients may be more likely to be exposed to more than 1 flavivirus, and these 2 factors could increase the probability of cross-reacting antibodies (Dr Sonya Bennett, Queensland Health, personal communication) resulting in more notifications of flavivirus (unspecified).

#### *Japanese encephalitis virus infections*

There were 2 notifications of JEV infection in Australia during 2013–14. Both cases were notified by Queensland:

- a 70-year-old man who acquired the infection in the Philippines, after travelling between January and July 2013. The case had a non-encephalitic illness, and recovered fully;
- a 47-year-old male who acquired the infection in Taiwan, after travelling for a total of 37 days in June and July 2013. The case had a non-encephalitic illness, and recovered fully.

#### *West Nile virus/Kunjin virus infection*

This category includes all WNV infections, including KUNV, which is an Australian lineage and has not been isolated from anywhere except on the Australian mainland and Torres Strait, and other WNV infections that are acquired overseas. While infection with KUNV is probably not uncommon in northern Australia, clinical KUNV cases are rare in Australia.<sup>22</sup>

There were 3 notifications of WNV/KUNV infection in Australia in 2013–14 compared with an average of 1.4 cases per year during the past 5 years.

The cases in 2013–14 were:

- a 49-year-old man who acquired the infection in Djibouti and notified by Victoria;
- a 26-year-old man who acquired the infection in Papua New Guinea and notified by Queensland;
- a 38-year-old man who acquired the infection in Timor-Leste and notified by Queensland.

#### *Murray Valley encephalitis virus infection*

There were no notifications of MVEV infection in Australia in 2013–14. MVEV infection is a rare disease in Australia, with an average of 4.2 cases per year during the past 5 years.

Table 4: Overseas-acquired cases of dengue virus infection, Australia, 2013–14, by serotype and country of acquisition

Country or region	Total number	Percentage of cases*	Serotype 1	Serotype 1 and 3	Serotype 1 and 4	Serotype 2	Serotype 3	Serotype 4	Unknown/ untyped
Indonesia	817	51	259	1	0	52	61	24	420
Thailand	170	11	26	0	0	17	7	5	115
Fiji	109	7	3	0	0	10	26	0	70
Malaysia	66	4	11	0	0	14	1	3	37
Philippines	63	4	8	0	0	11	2	8	34
Timor-Leste	60	4	21	0	0	0	4	0	35
India	47	3	3	0	0	5	1	1	37
Vanuatu	35	2	2	0	1	0	12	1	19
Sri Lanka	31	2	5	0	0	0	0	2	24
Vietnam	23	1	0	0	0	2	0	4	17
South-East Asia, nfd	22	1	2	0	0	1	0	0	19
Cambodia	16	1	1	0	0	0	3	0	12
Singapore	14	1	6	0	0	1	0	1	6
Papua New Guinea	14	1	0	0	0	2	3	0	9
Bangladesh	12	1	0	0	0	1	1	0	10
Nauru	11	1	0	0	2	0	5	0	4
Myanmar, The Republic of the Union of	8	0	2	0	0	0	1	0	5
Tonga	8	0	0	0	0	0	1	0	7
North Africa, nfd	6	0	1	0	0	2	0	1	2
French Polynesia	5	0	1	0	0	0	0	0	4
Pakistan	5	0	0	0	0	0	1	0	4
Solomon Islands	5	0	0	0	0	0	0	0	5
Kiribati	5	0	0	0	0	0	0	0	5
Kenya	3	0	0	0	0	0	0	0	3
Colombia	3	0	1	0	0	0	0	0	2
Tanzania	3	0	0	0	0	2	0	0	1
Other countries†	41	3	4	0	2	3	2	2	28
Overseas-country unknown	193		76	0	0	32	23	21	41
Total	1,795		432	1	5	155	154	73	975

\* The denominator excludes cases with place of acquisition 'Overseas-country unknown'. Percentages do not add up due to rounding.

† Each country with less than 3 cases.

nfd Not further defined.

Table 5: Notifications of flavivirus (unspecified), Australia, 2013–14

Virus species	Country of acquisition	State or territory	Month	Confirmation status
Kokobera	Place of acquisition unknown	Qld	Aug	Confirmed
Kokobera	Australia	Qld	Apr	Confirmed
Unspecified	Thailand	Qld	July	Confirmed
Unspecified	Indonesia	Qld	July	Confirmed
Unspecified	Place of acquisition unknown	Qld	July	Confirmed
Unspecified	Place of acquisition unknown	Qld	Aug	Confirmed
Unspecified	Place of acquisition unknown	Qld	Aug	Confirmed
Unspecified	Place of acquisition unknown	Qld	Aug	Confirmed
Unspecified	Vietnam	Qld	Oct	Confirmed
Unspecified	Papua New Guinea	Qld	Nov	Confirmed
Unspecified	India	Qld	Dec	Probable
Unspecified	Indonesia	Qld	Jan	Confirmed
Unspecified	Vanuatu	Qld	Feb	Confirmed
Unspecified	Fiji	Qld	Feb	Confirmed
Unspecified	Cook Islands	Qld	Mar	Confirmed
Unspecified	Philippines	Qld	Mar	Confirmed
Unspecified	Sub-Saharan Africa, nfd	Qld	Apr	Confirmed
Unspecified	Indonesia	Qld	May	Confirmed
Unspecified	Central and West Africa, nfd	Qld	May	Confirmed
Zika	Cook Islands	NSW	Apr	Confirmed
Zika	Cook Islands	NSW	Mar	Confirmed
Zika	Cook Islands	NSW	Mar	Confirmed
Zika	Cook Islands	NSW	Apr	Confirmed
Zika	Cook Islands	Qld	Mar	Confirmed
Zika	Cook Islands	Qld	Mar	Confirmed
Zika	Cook Islands	Qld	Mar	Confirmed
Zika	Cook Islands	Qld	Mar	Confirmed
Zika	Cook Islands	Qld	Mar	Confirmed
Zika	Cook Islands	Qld	Apr	Confirmed
Zika	Cook Islands	Qld	Apr	Confirmed
Zika	Cook Islands	Vic.	Apr	Confirmed
Zika	Samoa	Qld	Feb	Probable

nfd Not further defined.

### Yellow fever

There were no notifications of yellow fever in 2013–14. The only previous notifications of yellow fever were in 2011, and while the notifications met the surveillance case definition at the time, they were thought to be vaccine-associated. The surveillance case definition has since been revised to exclude vaccine associated cases.

### Malaria

Malaria is a serious acute febrile illness that is transmitted from person to person through the bite of an infected mosquito of the genus *Anopheles*.

It is caused by a protozoan parasite in the genus *Plasmodium* that includes 5 species that infect humans: *Plasmodium vivax*, *Plasmodium falciparum*, *Plasmodium malariae*, *Plasmodium ovale* and *Plasmodium knowlesi*.<sup>23,24</sup>

Australia is free of endemic malaria, but suitable vectors are present in northern Australia, and the area remains malaria-receptive. Malaria in Australia is therefore a disease associated with residing or travelling overseas in areas with endemic transmission. A case series in the Northern Territory showed that malaria cases were reported in travellers returning from endemic areas, but also reflected current events

such as military operations and increased refugee arrivals from malaria endemic areas. The last cases acquired on mainland Australia were during an outbreak in north Queensland in 2002.<sup>25</sup> Limited transmission occurs occasionally in the Torres Strait following importation. The most recent locally-acquired cases of malaria in Australia were a single case in 2013 acquired on Saibai Island in the Torres Strait and 7 locally-acquired cases in the Torres Strait in 2011.

There were 373 notifications of malaria during 2013–14 (Table 1, Figure 10), a 14% decrease compared with the mean of 433.6 notifications during the past 5 years. This was consistent with the trend of significant decline in the number of notifications since 2004–05 (test for trend,  $P=0.001$ ) (Figure 11), and consistent with the steady decline in malaria incidence globally between 2000 and 2015.<sup>26</sup> There were no locally-acquired cases of malaria in Australia in 2013–14, and complete information on the overseas country or region of acquisition was supplied for 92% of cases (343/373). India was the most frequently reported place of acquisition (15%, 56/373), followed by the Sudan (12%, 43/373) (Table 6). Malaria was most frequently reported among people aged 25–29 years, with 67 notified cases in this age group (Figure 12). Similar to previous years, the majority of cases were male

(72%,  $n=246$ ), and males predominated in every age group except in those aged under 5 years and those aged 80–84 years.

The infecting species was reported for 98% (366/373) of notifications during 2013–14. *P. falciparum* and *P. vivax* were the predominant species (Table 6). No cases were infected with *P. knowlesi*. *P. vivax* infections were commonly associated with travel to Asia or Pacific nations while *P. falciparum* infections were frequently associated with travel to the Middle East, Africa and Papua New Guinea.

Figure 11: Notifications of malaria, Australia, 1991–92 to 2013–14

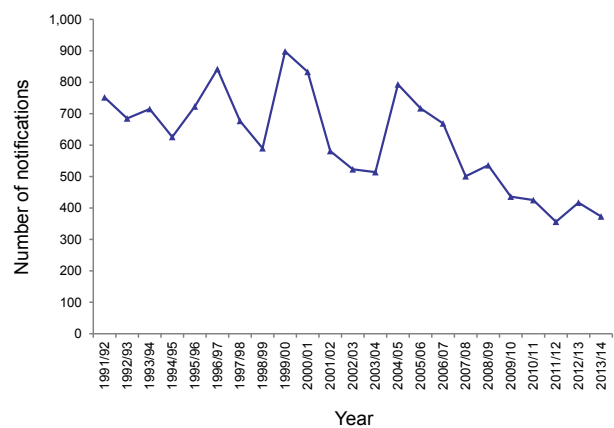
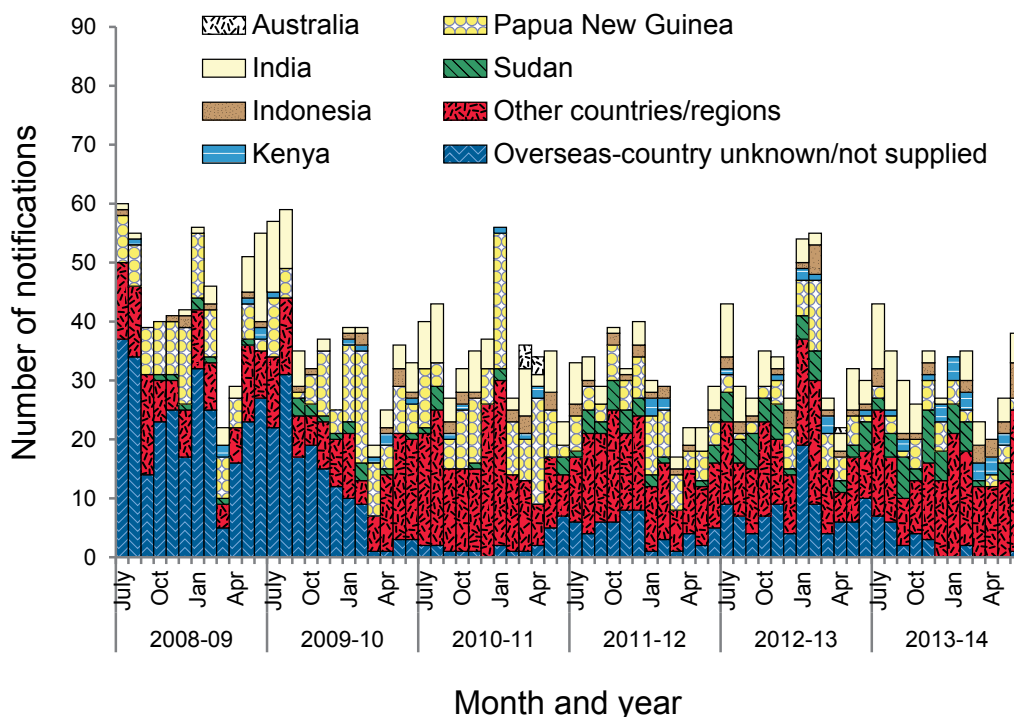


Figure 10: Notifications of malaria, Australia, 1 July 2008 to 30 June 2014, by month, year and place of acquisition\*



Note: 'Other countries/regions' each had less than 20 notified cases in 2013–14.

Table 6: Cases of malaria, Australia, 2013–14, by *Plasmodium* species and country or region of acquisition

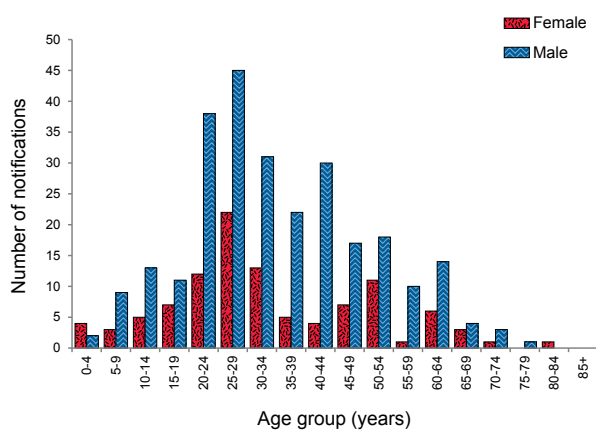
Country or region of acquisition	<i>Plasmodium falciparum</i>	<i>Plasmodium malariae</i>	<i>Plasmodium ovale</i>	<i>Plasmodium vivax</i>	Mixed species infections	<i>Plasmodium</i> spp	Total	% of all cases
India	2	0	0	51	2	1	56	15
Sudan	31	1	7	2	1	1	43	12
Papua New Guinea	10	1	1	22	0	0	34	9
Indonesia	7	1	3	11	1	0	23	6
Kenya	16	3	2	0	0	1	22	6
Ghana	14	0	1	1	1	0	17	5
Uganda	11	1	1	4	0	0	17	5
Sierra Leone	13	0	2	0	0	0	15	4
Nigeria	10	0	1	0	0	1	12	3
Tanzania	11	0	0	0	0	0	11	3
Pakistan	0	0	0	8	0	1	9	2
Sub-Saharan Africa, nfd	8	1	0	0	0	0	9	2
Zambia	4	0	3	0	0	0	7	2
Mozambique	3	0	2	0	0	0	5	1
Solomon Islands	0	0	0	5	0	0	5	1
Burundi	4	0	0	0	0	0	4	1
Congo, Republic of	4	0	0	0	0	0	4	1
Southern and East Africa, nfd	3	0	0	0	0	1	4	1
Zimbabwe	4	0	0	0	0	0	4	1
Other countries/regions*	22	2	3	14	0	1	42	11
Australia	0	1	0	0	0	0	1	0
Overseas-country unknown	16	0	3	8	2	0	29	8
Total	193	11	29	126	7	7	373	100
% of all cases	52	3	8	34	2	2	100	

\* Each with less than 4 cases.

nfd Not further defined.



**Figure 12: Notifications of malaria, Australia, 2013–14, by age group and sex**



### Sentinel chicken, arbovirus detections in mosquitoes and mosquito abundance monitoring

#### New South Wales

The season began with 150 pullets and a total of 2,871 samples was received from the 10 flocks in New South Wales over the 6-month period in 2013–14 (Map 1). This represented 5,742 enzyme-linked immunosorbent assay (ELISA) tests (excluding controls and quality assurance samples), with each specimen being tested for MVEV and KUNV antibodies. There were 4 seroconversions; 1 KUNV from Forbes (bleed taken 11 February 2014), 1 KUNV from Griffith (12 February 2014), 1 KUNV from Leeton (30 March 2014), and 1 MVEV from Deniliquin (31 March 2014).

For 2013–14 the climatic conditions leading up to the season for the inland were of well below average rainfall for the last 6 months of 2013. In contrast, rainfall was above average for most of the inland during the first 6 months of 2014. The Forbes hypothesis<sup>27</sup> was not suggestive of a potential MVEV epidemic for the 2013–14 season, however the Nichols' theory<sup>28</sup> was not exclusive of possible activity. The dry conditions produced fewer mosquito numbers with a total trapped of around 100,000, being about 30,000 down from the previous season. Human notifications were below normal; particularly from the inland where alphavirus notifications (RRV and BFV combined) were close to half the long term average.

For the coast, weather patterns were mostly similar to the inland, however, the dry conditions continued for the north coast into the first three months of 2014, and mosquito numbers were below average. Coastal disease notifications of RRV and BFV were 27% below the long-term average.

Further detail can be found in New South Wales Arbovirus Surveillance Program annual reports, available on the [NSW Health web site](http://medent.usyd.edu.au/arbovirus/information/publications.htm) (<http://medent.usyd.edu.au/arbovirus/information/publications.htm>)

#### Northern Territory

In 2013–14, there were 433 laboratory confirmed cases of RRV in the Northern Territory, which was the highest notification rate since 1990–91. Most (n=345) cases were recorded in the Darwin region, and occurred between December and May, with 26 cases also reported in July. It is uncertain how many notified cases were false positive diagnoses and the high number of cases did not coincide with high numbers of *Ae. vigilax* or *Cx. annulirostris*, except for in July, when *Ae. vigilax* numbers were elevated, and January, when *Cx. annulirostris* peaked. In the Darwin region there were 136 cases reported in Darwin urban, 111 in rural Darwin (Litchfield Shire) and 63 in Palmerston. This represents a rate (cases per 100,000 population) of 163 in Darwin urban (population: 83,304), 197 in Palmerston (population: 31,996) and 530 in rural Darwin (population: 20,935). Population figures are based on Australian Bureau of Statistics figures from June 2013.

In the regions, 21 RRV disease cases were recorded in the East Arnhem region, 40 in the Katherine region, 6 in the Barkly and 14 in the Alice Springs region.

In the 2013–14 season, Northern Territory sentinel chickens seroconverted to MVEV in April in the Katherine region, to KUNV in May in the Darwin, Katherine and Barkly regions and again to KUNV in July in the Darwin region. No MVEV or KUNV disease cases were reported in the Northern Territory in 2013–14. In parallel with the sentinel chicken surveillance program, the flavivirus surveillance trial using honey bait cards (or FTA cards) was continued. None of the cards tested positive for flaviviruses.

The dengue mosquito *Ae. aegypti*, was detected in Tennant Creek in late 2011. This triggered a coordinated and intensive program, consisting of 8 rounds of property by property surveys and treatment of all receptacles to eliminate this exotic mosquito. The dengue mosquito elimination program in Tennant Creek was successfully completed on 30 April 2014. Further details are available from the Northern Territory Medical Entomology annual reports, available on the [Northern Territory government web site](http://www.health.nt.gov.au/Medical_Entomology/index.aspx) ([http://www.health.nt.gov.au/Medical\\_Entomology/index.aspx](http://www.health.nt.gov.au/Medical_Entomology/index.aspx)).

## Queensland

### Torres Strait *Aedes albopictus* Prevention and Control Program

The exotic Asian tiger mosquito, *Ae. albopictus* was first found on the outer islands of Torres Strait in April 2005.<sup>29</sup>

This mosquito is a competent vector of a number of arboviruses including DENV and CHIKV, and represents a serious nuisance biting mosquito. Since 2005, the Australian Government has funded Queensland Health for a mosquito elimination program in the Torres Strait. The initial aim of the program was to eliminate *Ae. albopictus* from the Torres Strait islands but this was revised in May 2008 to a *cordon sanitaire* approach (a barrier designed to prevent spread) focused on Thursday and Horn islands. Harbourage treatment on Horn and Thursday islands remained the focus of the program. Whilst this provided good control of *Ae. albopictus*, relatively high numbers of *Ae. aegypti* persisted, particularly on Thursday Island.

Harbourage treatment with synthetic pyrethroids remains the key component of the *Ae. albopictus* suppression strategy in the Torres Strait and has proven successful at reducing numbers of mosquitoes collected and also preventing establishment on the mainland. While these intervention techniques are proving very effective at controlling *Ae. albopictus*, the continued presence of *Ae. aegypti* in relatively high numbers on Thursday Island remains a cause for concern. Vector control teams inspected the majority of premises on Thursday Island during the reporting period (up to 850 properties per trip with a total of 6 trips conducted) and undertook source reduction to address this.

Human-bait sweep-net sampling did not detect *Ae. albopictus* at any of the sites across the Northern Peninsula area. However, a total of 1,249 potential breeding sites were identified and treated during yard inspections in Seisia, New Mapoon and Bamaga, and at least 320 larval samples were collected from water-holding containers in these communities.

### North Queensland

Ongoing container-inhabiting mosquito surveillance in Cairns and Townsville by public health units through a network of traps in various suburbs did not detect *Ae. albopictus* in either location during the reporting period.

The sugar-baited FTA card based arbovirus surveillance conducted in the Northern Peninsula Area of Cape York by the Australian Government Department of Agriculture did not detect JEV during the reporting period.

### Central Queensland

Surveillance across Rockhampton using Biogents, gravid *Aedes* traps (GATs) and ovitraps in early 2014 confirmed the presence of *Ae. aegypti* across a number of urban locations. Notably, the exotic species *Cx. gelidus* was identified in the Yeppoon area for the first time in May/June 2014. Larval surveys/GATs did not detect *Ae. aegypti* in Childers or Apple Tree Creek in February 2014. However, *Ae. aegypti* was again observed in Gin Gin in the greater Bundaberg region.

### Southern Queensland

GATs deployed in the South Burnett towns of Murgon, Wondai, Kumbia, Nanango, Kingaroy and Blackbutt only detected *Ae. aegypti* in Wondai.

*Ae. aegypti* were detected in both Roma and Charleville during a trial of ovitraps and GAT traps in south-west Queensland. All 3 sites in Charleville and 2 of 3 in Roma detected *Ae. aegypti*. In Roma, *Ae. aegypti* were only collected in GATs while *Ae. aegypti* were present in both GATs and ovitraps in Charleville. The novel urban surveillance program using ovitraps and GATs in the Brisbane local government area did not detect *Ae. aegypti* or *Ae. albopictus*. The trial of sugar-baited FTA card virus surveillance across Gold Coast City, Brisbane City and Sunshine Coast Regional councils demonstrated the utility of this virus detection system for councils who monitor mosquito populations at peri-urban sites. RRV or BFV were not detected at any locations.

In South East Queensland, the 2013–14 season was similar to the previous season, with a very dry first half. Large but discrete rain events were observed in summer and autumn, but overall rainfall remained very low. Total rainfall at Brisbane Airport from 1 July to 30 June was 549 mm, just 46% of long term average. Whilst February is normally the wettest month of the year, Brisbane Airport recorded 15 mm, only 9% of the average rainfall and, most unusually, many saltmarshes had completely dried out by mid-March. This may explain the huge number of *Ae. vigilax* that hatched across the region after widespread rain in late March, a phenomenon that has been observed previously after saltmarshes become dry. A mild autumn prolonged the activity of mosquitoes into early

winter, and most local governments with aerial spraying programs observed sufficient hatching of saltmarsh mosquitoes after the mid-June tide peak to unusually conduct an aerial treatment in June. The dry season ensured that *Ae. vigilax* was the dominant species in coastal areas, but *Cx. annulirostris* and a few of other freshwater species were active later in the season.

### South Australia

The mosquito populations along the River Murray during the season exhibited 2 distinct patterns associated with geographic location of the trap sites. Traps located north of Mannum in the Mid-Murray council were typified by low mosquito numbers over the majority of the season. A slight increase in mosquito numbers was observed in samples from this group of adult traps retrieved in March, with numbers then dropping off in April. The composition of the mosquito community varied across upper river councils. The mosquito species *Anopheles annulipes*, *Coquillettidia linealis*, *Cx. annulirostris*, *Cx. molestus* and *Cx. quinquefasciatus* all formed significant components of the mosquito community in at least 1 of the upper river councils. Spring peaks in mosquito populations were observed in 2 of the 3 upper river councils. These were distinguished from previous seasons by the virtual absence of the Southern Salt Marsh mosquito *Ae. camptorhynchus*, a species that typically overwinters as larvae and emerges in spring. In the northern councils, some locally rare mosquitoes were also recorded this season including *Ae. eidsvoldensis*, *Mansonia uniformis*, *Ae. alternans*, *Ae. sagax*, and *Ae. vittiger*.

The mosquito populations at Mannum and to the south of this town retained distinct spring peaks of *Ae. camptorhynchus* through to November with some areas also experiencing a late season flush in mosquito numbers attributed to heavy February rainfall.

Overall, a total of 46,713 adult mosquitoes were collected from the 35 regular monitoring sites in the season. The total number of mosquitoes caught this season represented an overall increase of 45% compared with the previous season's total mosquito catch. However, this increase was not uniform across all councils. In the 3 northernmost councils there was a decrease in the total mosquitoes caught across the councils by 55% while in the 3 southernmost councils there was an increase of 64% on the total number of mosquitoes caught in the previous

season (although Alexandrina council actually experienced a decline of around 25%). The overall mosquito catch within the Mid-Murray Council was around the same as the previous season.

The University of South Australia (Uni SA) also conducted mosquito surveillance trapping at 6 locations on 16 occasions from September 2013 to April 2014 for the City of Salisbury in the Adelaide northern metropolitan suburbs of Globe Derby Park and St Kilda during the season. In this region, the mosquito season can be characterised by 2 distinct features. Firstly, there was a peak in *Ae. camptorhynchus* abundance in March 2014. This was likely triggered by the high rainfall in February–March 2014. Secondly, *Ae. vigilax* numbers in the late summer and autumn of 2014 continued to remain low, but overall numbers showed a slight increase compared with the previous season. *Ae. vigilax* numbers peaked at approximately 98 mosquitoes per trap in early March 2014. This peak was possibly constrained by high tides not occurring until mid-March to April 2014.

The *Ae. camptorhynchus* abundance pattern during spring was similar to that during 2012–13 although slightly higher in numbers compared with the previous season. However, as previously mentioned, a distinct peak of the species was observed in March 2014 in response to heavy rainfall in late February 2014. *Ae. vigilax* abundance has been lower over the last 3 monitoring seasons compared to the previous years, indicating that the improved and complementary larval control activities of South Australian Department of Health and Ageing (SA Health) and Uni SA in the area have been successful in reducing the mosquito numbers.

No sentinel chicken seroconversions for MVEV or KUNV were recorded during the 2013–2014 season.

This season, sugar-baited FTA cards were trialled by Uni SA in the traditional Encephalitis Vector Survey CO<sub>2</sub> baited traps set within each client council, and in a number of additional regional and metropolitan locations. In addition to this research, 3 passive CO<sub>2</sub> baited box traps were deployed for the first time along the River Murray at Renmark, Mannum and Murray Bridge. Arboviruses were detected in a number of regional and metropolitan locations between January and March 2014 (Table 7).

Table 7: Virus and sentinel chicken surveillance in Australia for selected regions, by surveillance method and virus genus, 2013–14

State or territory	Region	Flaviviruses			Alphaviruses		
		Number positive or seroconverted/number tested*	First positive date	Last positive date	Number positive or seroconverted/number tested*	First positive date	Last positive date
<b>Sentinel chickens</b>							
NSW	Bourke	0/157			N/A		
NSW	Deniliquin	1 MVEV /331			N/A		
NSW	Forbes	1 KUNV/330			N/A		
NSW	Griffith	1 KUNV/315			N/A		
NSW	Hay	0/345			N/A		
NSW	Leeton	1 KUNV / 328			N/A		
NSW	Macquarie Marshes	0/212			N/A		
NSW	Moama	0/255			N/A		
NSW	Moree	0/300			N/A		
NSW	Wee Waa	0/298			N/A		
NT	Darwin region	4/279	7 May 2014	30 Jul 2014	N/A		
NT	East Arnhem region	0/53			N/A		
NT	Katherine region	3/110	2 Apr 2014	6 May 2014	N/A		
NT	Barkly region	2/45	13 May 2014	13 May 2014	N/A		
NT	Alice Springs region	0/95			N/A		
SA	Paringa	0/35			N/A		
SA	Loxton	0/35			N/A		
SA	Waikerie (Qualco)	0/35			N/A		
SA	Murray Bridge	0/35			N/A		
SA	Meningie	0/35			N/A		
Vic.	Mooroopna	0/144			N/A		
Vic.	Mildura	0/444			N/A		
Vic.	Robinvale	0/265			N/A		
Vic.	Nyah West	0/428			N/A		
Vic.	Kerang	0/411			N/A		
Vic.	Barmah	0/381			N/A		
Vic.	Cobram	0/495			N/A		
Vic.	Wodonga	0/307			N/A		

Table 7 cont'd: Virus and sentinel chicken surveillance in Australia for selected regions, by surveillance method and virus genus, 2013–14

State or territory	Region	Flaviviruses			Alphaviruses		
		Number positive or seroconverted/number tested*	First positive date	Last positive date	Number positive or seroconverted/number tested*	First positive date	Last positive date
WA	Wyndham	0/130			N/A		
WA	Kununurra	0/148			N/A		
WA	Savannah Nickel Mine	0/119			N/A		
WA	Halls Creek	0/255			N/A		
WA	Fitzroy Crossing	0/141			N/A		
WA	Derby	1/489	16 May 2014	16 May 2014	N/A		
WA	Lombadina	0/45			N/A		
WA	Broome	1/58	26 Sept 2013	26 Sept 2013	N/A		
WA	Roebuck Plains	9/156	25 July 2013	28 May 2014	N/A		
WA	Port Hedland	0/67			N/A		
WA	Karratha	0/245			N/A		
WA	Harding Dam	0/568			N/A		
WA	Marble Bar	0/95			N/A		
WA	Pannawonica	0/228			N/A		
WA	Tom Price	0/203			N/A		
WA	Paraburdoo	0/237			N/A		
WA	Onslow	0/154			N/A		
WA	Ophthalmia Dam	4/222	22 May 2014	17 June 2014	N/A		
WA	Newman	0/238			N/A		
WA	Exmouth	0/262			N/A		
WA	Carnarvon	0/222			N/A		
WA	Moora	0/156			N/A		
WA	Geraldton	0/206			N/A		
WA	Dongara	0/174			N/A		
WA	York	0/80			N/A		

Table 7 cont'd: Virus and sentinel chicken surveillance in Australia for selected regions, by surveillance method and virus genus, 2013–14

State or territory	Region	Flaviviruses			Alphaviruses		
		Number positive or seroconverted/number tested*	First positive date	Last positive date	Number positive or seroconverted/number tested*	First positive date	Last positive date
<b>Sugar-baited FTA cards</b>							
NSW	Ballina				1 RRV /8,328		
NSW	Bankstown				1 BFV & 1 RRV /2,443		
NSW	Coffs Harbour				1 RRV /2,448		
NSW	Georges River				4 BFV, 5 RRV /9,932		
NSW	Griffith				1 BFV, 3 RRV /28,622		
NSW	Leeton				2 BFV /11,935		
NSW	Port Macquarie				1 BFV, 5 RRV /3,938		
NSW	Port Stephens				2 RRV /19,126		
NSW	Tweed Heads				4 RRV /10,030		
NT	Darwin region	0/80			N/A		
Qld	Northern Peninsula Area†	Nil					
SA	Goolwa	Stratford	Jan	Jan	BFV RRV	Feb	Feb
SA	Goolwa North						
SA	Mypolonga	Stratford	Jan	Jan	BFV RRV	Feb	Feb
SA	Adelaide Hills (Mylor-Hahndorf)	Stratford	Jan	Jan			
SA	Tailem Bend				BFV	Feb	Feb
SA	Paringa				BFV	Feb	Feb
SA	Renmark				BFV RRV	Jan	Feb
SA	Murray Bridge				RRV	Mar	Mar
SA	Globe Derby Park				RRV	Feb	Feb
SA	Bedford Park				RRV	Jan	Jan
SA	Port Adelaide				BFV RRV	Jan	Feb
SA	Thebarton				RRV	Feb	Feb
SA	Richmond				RRV	Feb	Feb
SA	Enfield				BFV	Feb	Feb

Table 7 cont'd: Virus and sentinel chicken surveillance in Australia for selected regions, by surveillance method and virus genus, 2013–14

State or territory	Region	Flaviviruses			Alphaviruses		
		Number positive or seroconverted/number tested*	First positive date	Last positive date	Number positive or seroconverted/number tested*	First positive date	Last positive date
<b>Virus isolation/polymerase chain reaction detection from mosquitoes</b>							
NSW	Blacktown	0/1,470			3 RRV /1,470		
NSW	Georges River	11 STRV /9,932			3 BFV & 6 RRV /9,932		
NSW	Gosford	0/2,378			1 BFV /2,378		
NSW	Griffith	1 STRV /28,622			1 RRV /28,622		
NSW	Leeton	0/11,935			1 RRV /11,935		
NSW	Port Stephens	2 EHV /19,126			2 BFV & 17 RRV /19,126		
NT	Darwin region	N/A					
Qld	Northern Peninsula Area, Cape York JEV surveillance†	Nil					
Vic.	Inland North West	0/1,924			0/1,924		
Vic.	Inland North East	0/2,347			0/2,347		
Vic.	Gippsland – Lake Wellington	0/2,285			1 RRV/2,285		
WA	Kununurra	0/8,690			0/8,690		
WA	Wyndham	3 KUNV/5,810			2 SINV/5,810		
WA	Roebourne area	0/3,458			0/3,458		
WA	Mt Magnet	0/31			0/31		
WA	Cue	0/242			0/242		
WA	Peel region	0/24,142			1 RRV & 6 BFV/24,142		
WA	Leschenault region	0/17,130			6 RRV & 2 BFV/17,130		
WA	Capel-Busselton region	0/18,269			11 RRV & 1 BFV/18,269		

\* For virus detections/isolations, the number tested is the number of individual mosquitoes or chickens tested, unless otherwise noted. The number tested is not always known.

† Surveillance only conducted from 28 January to 22 April 2014.

Note: Sentinel chickens are not screened for antibodies to Alphaviruses

### Victoria

Through the routine sentinel chicken program, weekly blood samples were tested from the 9 flocks between November 2013 and April 2014. No seroconversions for flaviviruses were detected during the season, which involved testing of 3,319 samples.

Mosquito monitoring in Victoria was conducted through the Victorian Arbovirus Disease Control Program by 10 Local Government Areas. Across the standard mosquito monitoring program, 31,433 mosquitoes were collected between November and April and submitted for species identification and arbovirus detection. Mosquito abundance at inland sites was low throughout the season, except in the North West (including Kerang and Mildura) where following above average rainfall in summer and autumn, moderate numbers of *Cx. australicus* and *Cx. annulirostris* were detected. *Cx. annulirostris* was the dominant species at approximately half (13 of 23) of inland sites, accounting for between 24% to 66% of collections. Other species that dominated catches included *Cx. australicus*, *Cx. quinquefasciatus*, *Ae. notoscriptus* and *Ae. bancroftianus*.

Coastal mosquito populations are monitored in the Gippsland and Bellarine Peninsula areas, with the Wellington Shire Council participating in the standardised mosquito monitoring program with weekly submissions. In Gippsland, mosquito abundance was highest in spring and early summer with moderate to high numbers of *Ae. camptorhynchus* detected (Table 8 shows the definition of 'High' and other numerical categories). Mosquito abundance peaked in early January 2013 with very high levels detected. A reduction in mosquito abundance was detected for the remainder of the season, until mid-April, where high numbers were recorded.

Virus isolation was conducted on over 7,000 pools of mosquito samples (a total of 48,095 mosquitoes). A single RRV isolate was cultured from a pool of *Ae. camptorhynchus* collected in Gippsland in November 2013. The RRV isolate was phylogenetically related to the well-documented, eastern Australian RRV lineage.<sup>30</sup>

### Western Australia

Above average rainfall was observed in northern parts of Western Australia between October 2013 and February 2014. Tropical Cyclones Alessia and Christine in particular influenced rainfall patterns in November and December 2013.

Numerous sites in the Kimberley, Pilbara and Gascoyne observed their wettest January on record. Monsoonal activity was weaker than usual in northern Western Australia in March, however, typical rainfall patterns returned in April and May 2014. In the south-west of Western Australia, rainfall was above average at the commencement of the season, and then declined to below or very much below average from October 2013 to April 2014, particularly during the summer period. Temperatures were generally warmer than average for most of the season. Tides impacted saltmarsh breeding sites with the exception of summer months when they had less impact than predicted.

The level of flavivirus activity in sentinel chickens in northern Western Australia in 2013–14 was low.<sup>31</sup> Seroconversions were detected in 15 of the 4,798 samples tested (0.3%), which was above the level of activity in 2012–13,<sup>32</sup> but still low. Low level activity associated with the end of the 2012–13 season was detected in the West Kimberley region and continued to October 2013. Flavivirus activity commenced late in the 2013–14 season, following generally above average rainfall between November 2013 and February 2014, followed by 2 months of very much below average or average rainfall in March and April in the Kimberley region. The first seroconversion for the season occurred in mid-May, when a KUNV seroconversion was detected in the Derby flock in the West Kimberley region. In the same month, antibodies to KUNV were detected at Ophthalmia Dam in the Pilbara region, followed by seroconversions to KUNV (3), MVEV (2) and an unknown flavivirus infection at Roebuck Plains, in the West Kimberley region. Flavivirus activity continued in June in the Ophthalmia Dam chicken flock. Overall, 11 flavivirus seroconversions were detected in sentinel chickens in the 2013–14 season in Western Australia, and the majority (63.6%) were due to KUNV infection. Activity of MVEV was only detected at Roebuck Plains in the West Kimberley region. In addition, KUNV was the only flavivirus isolated from mosquitoes collected in the Northeast Kimberley region in April 2014. The Western Australian Department of Health initially released a media alert in early May reminding travellers and residents to take precautions against mosquito bites following late season flooding in the Pilbara and Gascoyne regions. Detection of antibodies to flaviviruses in sentinel chickens triggered a second media release in mid-June. No human cases of MVE or KUNV disease were reported in the 2013–14 season in Western Australia.



Table 8: Key mosquito vector abundance in selected regions of Australia in 2013–14, by species, state or territory, region and month

Species	State or territory	Region/ locality	Month														
			Jul	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun			
<b>Saltwater</b>																	
<i>Aedes vigilax</i>	NSW	North Coast	-	-	-	-	-	-	LOW	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH	MED	LOW
<i>Ae. vigilax</i>	NSW	Mid-North Coast	-	-	-	-	-	-	LOW	HIGH	HIGH	HIGH	HIGH	HIGH	MED	LOW	LOW
<i>Ae. vigilax</i>	NSW	Central Coast	-	-	-	-	-	-	MED	HIGH	HIGH	HIGH	MED	MED	LOW	LOW	LOW
<i>Ae. vigilax</i>	NSW	Sydney – Georges River	-	-	-	-	-	-	HIGH	HIGH	HIGH	HIGH	MED	LOW	LOW	LOW	LOW
<i>Ae. vigilax</i>	NSW	Sydney – Homebush	-	-	-	-	-	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH	MED	LOW	LOW	LOW
<i>Ae. vigilax</i>	NSW	Sydney – Western	-	-	-	-	-	-	LOW	MED	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<i>Ae. vigilax</i>	NT	Darwin region	HIGH	LOW	MED	MED	HIGH	HIGH	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<i>Ae. vigilax</i>	NT	East Arnhem region	N/A	LOW	LOW	LOW	N/A	HIGH	HIGH	HIGH	HIGH	HIGH	N/A	N/A	LOW	LOW	LOW
<i>Ae. vigilax</i>	Qld	Brisbane inland–Indooroopilly Island	-	-	-	LOW	LOW	LOW	MED	LOW	MED	LOW	LOW	LOW	LOW	LOW	LOW
<i>Ae. vigilax</i>	Qld	Brisbane coastal–Bracken Ridge	LOW	LOW	LOW	MED	HIGH	HIGH	MED	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH	LOW	LOW
<i>Ae. vigilax</i>	Qld	Brisbane coastal–Virginia	LOW	LOW	MED	HIGH	HIGH	HIGH	HIGH	VERY HIGH	VERY HIGH	VERY HIGH	VERY HIGH	VERY HIGH	VERY HIGH	VERY HIGH	HIGH
<i>Ae. vigilax</i>	Qld	Brisbane coastal–Albion	-	-	-	LOW	LOW	LOW	MED	HIGH	HIGH	HIGH	LOW	HIGH	HIGH	LOW	LOW
<i>Ae. vigilax</i>	Qld	Brisbane coastal–Hemmant	-	-	LOW	MED	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH	HIGH
<i>Ae. vigilax</i>	Qld	Brisbane coastal–Lota	-	-	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	MED	LOW	HIGH	LOW	LOW
<i>Ae. camptorhynchus</i>	SA (SK1,SK2)	St Kilda	-	-	LOW	HIGH	HIGH	HIGH	LOW	HIGH	LOW	LOW	LOW	HIGH	MED	LOW	LOW
<i>Ae. camptorhynchus</i>	SA (GD1,GD2, GD6)	Globe Derby Park	-	-	HIGH	HIGH	HIGH	MED	MED	LOW	LOW	LOW	LOW	HIGH	HIGH	LOW	LOW
<i>Culex molestus</i>	SA (A3,A4)	Goolwa	-	-	HIGH	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<i>Ae. camptorhynchus</i>	Vic.	Gippsland / Lake Wellington	-	-	HIGH	LOW	LOW	HIGH	HIGH	HIGH	HIGH	HIGH	LOW	LOW	MED	LOW	MED

Table 8 cont'd: Key mosquito vector abundance in selected regions of Australia in 2013–14, by species, state or territory, region and month

Species	State or territory	Region/ locality	Month															
			Jul	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun				
<i>Ae. vigilax</i>	WA	Kununurra	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
<i>Ae. vigilax</i>	WA	Wyndham	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
<i>Culex sitiens</i>	WA	Wyndham	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
<i>Ae. vigilax</i>	WA	Roebourne area	-	-	-	LOW	-	-	-	-	-	-	-	-	-	-	-	-
<i>Ae. camptorhynchus</i>	WA	Peel region	MED	HIGH	HIGH	HIGH	MED	MED	LOW	LOW	LOW	LOW	LOW	LOW	LOW	HIGH	HIGH	MED
<i>Ae. vigilax</i>	WA	Peel region	Nil	Nil	Nil	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<i>Ae. camptorhynchus</i>	WA	Leschenault region	LOW	HIGH	HIGH	HIGH	HIGH	MED	MED	LOW	LOW	LOW	LOW	LOW	LOW	LOW	HIGH	HIGH
<i>Ae. vigilax</i>	WA	Leschenault region	Nil	Nil	Nil	Nil	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<i>Ae. camptorhynchus</i>	WA	Capel-Busselton region	MED	HIGH	HIGH	VERY HIGH	VERY HIGH	HIGH	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<b>Freshwater</b>																		
<i>Culex annulirostris</i>	NSW	Inland – Riverina	-	-	-	-	HIGH	HIGH	HIGH	VERY HIGH	VERY HIGH	VERY HIGH	HIGH	LOW	LOW	LOW	LOW	LOW
<i>Cx. annulirostris</i>	NSW	Inland – Murray region	-	-	-	-	LOW	LOW	LOW	HIGH	HIGH	HIGH	LOW	LOW	LOW	LOW	LOW	LOW
<i>Cx. annulirostris</i>	NSW	Inland, West & Nth West	-	-	-	-	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<i>Cx. annulirostris</i>	NT	Darwin region	MED	MED	LOW	LOW	LOW	LOW	HIGH	HIGH	HIGH	MED	LOW	LOW	LOW	MED	MED	MED
<i>Cx. annulirostris</i>	NT	East Arnhem region	N/A	LOW	MED	MED	N/A	N/A	HIGH	HIGH	HIGH	HIGH	LOW	LOW	LOW	LOW	LOW	MED
<i>Cx. annulirostris</i>	NT	Katherine region	N/A	N/A	N/A	N/A	N/A	N/A	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<i>Cx. annulirostris</i>	NT	Barkly region	N/A	N/A	N/A	N/A	N/A	N/A	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	N/A
<i>Cx. annulirostris</i>	NT	Alice Springs region	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<i>Ae. camptorhynchus</i>	SA	Wellington	-	-	VERY HIGH	HIGH	HIGH	HIGH	MED	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<i>Ae. camptorhynchus</i>	SA	Tallem Bend	-	-	MED	LOW	MED	MED	HIGH	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<i>Ae. camptorhynchus</i>	SA	Murray Bridge	-	-	HIGH	MED	HIGH	HIGH	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW
<i>Ae. camptorhynchus</i>	SA	Mannum	-	-	HIGH	MED	MED	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	MED	LOW	LOW
<i>Ae. camptorhynchus</i>	SA	Meningie	-	-	VERY HIGH	HIGH	HIGH	MED	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	HIGH

Table 8 cont'd: Key mosquito vector abundance in selected regions of Australia in 2013–14, by species, state or territory, region and month

Species	State or territory	Region/ locality	Month														
			Jul	Aug	Sept	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun			
<i>Culex molestus</i>	SA	Swan Reach	-	-	LOW	LOW	LOW	LOW	LOW	LOW	HIGH	LOW	LOW	LOW	LOW	-	-
<i>Cx. annulirostris</i>	SA	Blanchetown	-	-	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	-	-
<i>Anopheles annulipes</i>	SA	Morgan	-	-	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	-	-
<i>Cx. annulirostris</i>	SA	Waikerie	-	-	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	-	-
<i>Cx. molestus</i>	SA	Kingston on Murray	-	-	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	-	-
<i>Cx. annulirostris</i>	SA	Loxton	-	-	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	-	-
<i>An. annulipes</i>	SA	Berri	-	-	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	-	-
<i>Cx. annulirostris</i>	SA	Renmark/Paringa	-	-	LOW	HIGH	LOW	LOW	LOW	LOW	LOW	LOW	LOW	MED	LOW	-	-
<i>Cx. annulirostris</i>	Vic.	North West	-	-	-	-	-	LOW	LOW	LOW	LOW	LOW	LOW	MED	MED	-	-
<i>Cx. annulirostris</i>	Vic.	North East	-	-	-	-	-	LOW	LOW	LOW	LOW	LOW	LOW	LOW	LOW	-	-
<i>Cx. annulirostris</i>	WA	Kununurra	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
<i>Cx. palpalis</i>	WA	Kununurra	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
<i>Aedes normanensis</i>	WA	Kununurra	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
<i>Cx. annulirostris</i>	WA	Wyndham	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
<i>Culex palpalis</i>	WA	Wyndham	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
<i>Ae. normanensis</i>	WA	Wyndham	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
<i>Cx. annulirostris</i>	WA	Roebourne area	-	-	-	LOW	-	-	-	-	-	LOW	-	-	-	-	-
<i>Ae. normanensis</i>	WA	Roebourne area	-	-	-	Nil	-	-	-	-	-	LOW	-	-	-	-	-
<i>Cx. annulirostris</i>	WA	Mt Magnet	-	-	-	-	-	-	-	-	-	-	-	Nil	-	-	-
<i>Cx. annulirostris</i>	WA	Cue	-	-	-	-	-	-	-	-	-	-	-	-	LOW	-	-

Calculated as an average for traps across the region and rated as:

LOW (<50)	MEDIUM (50–100)	HIGH (101–1,000)	VERY HIGH (1,001–10,000)	EXTREME (>10,000)
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\* Trap fail

- Denotes no data collected.

In the south-west of Western Australia, vector abundance was initially high and then declined to low abundance in summer and autumn, likely due to reduced impact of high tides and very low rainfall.<sup>31</sup> The first arbovirus detection for the season was RRV in the Peel region and at Capel in early October 2013, prompting the Western Australian Department of Health to issue a media release advising residents and travellers of the increased risk of mosquito-borne disease. The minimum infection rate for RRV was greatest when it reached 7.7 per 1,000 mosquitoes<sup>33</sup> at Capel on 10 October 2013. RRV was also detected in the Leschenault region and at Busselton later in the season, and detections continued through to mid-February 2014. The first detection of BFV was in the Peel region in late October 2013, and this virus was also subsequently detected in the Leschenault region and Capel. The minimum infection rate for BFV peaked at 8.0 per 1,000 mosquitoes in early February in the Leschenault region. The majority of alphavirus detections were from *Ae. camptorhynchus* (74%). Detections of RRV in mosquitoes occurred during the time that roughly 60% of human cases were notified, and human cases occurred when vector abundance was low. It was recently suggested that the large number of notified RRV cases that occur outside the peak risk season may be due to issues with the superseded case definition, the low positive predictive value of IgM positive only tests in the off-season and inconsistencies between notification methodologies of different testing laboratories.<sup>8</sup>

Further detail can be found in the [Western Australian annual reports](http://ww2.health.wa.gov.au/~media/Files/Corporate/general%20documents/Mosquitoes/PDF/Arbovirus-AnnRpt-2013-14.ashx) (<http://ww2.health.wa.gov.au/~media/Files/Corporate/general%20documents/Mosquitoes/PDF/Arbovirus-AnnRpt-2013-14.ashx>)

### Tasmania

No viruses were isolated in 2013–14 from mosquitoes trapped during ad hoc collections undertaken in the Sorrell Council region.

### Exotic mosquito detections at the border

Between July 2013 and June 2014 there were 13 exotic mosquito detections made by the Australian Government Department of Agriculture and Water Resources at the Australian border (Table 9). This represents an increase compared with the 2012–13 period where there were 7 exotic mosquito detections. This increase was due to an increase in the number of exotic

mosquito detections at international airports. Four detections were made via inspection of imported cargo while the remaining 9 detections resulted from routine vector monitoring activities performed at international ports. The 4 exotic mosquito detections associated with imported cargo reinforce that imported used tyres and exposed machinery remain a high risk pathway for the introduction of exotic mosquitoes. The 2 *Ae. albopictus* detections in Darwin in November and December 2013 occurred a week apart however, the detections were made at different port areas and were not deemed to be related (i.e. 2 separate introductions). There was a significant increase in the detections of exotic mosquitoes, particularly *Ae. aegypti* at international airports in southern Australia during this period. Perth, Adelaide and Melbourne International Airports all experienced exotic detections within the baggage handling areas. Initial DNA analyses concluded the *Ae. aegypti* mosquitoes detected at the airports did not originate from Queensland populations and likely originated from a common origin in South East Asia. Extensive treatments and enhanced surveillance were conducted in response to these detections involving the relevant state health jurisdiction, the airport authority and the Australian Government Department of Agriculture and Water Resources. Pathway analysis is underway and the Department of Agriculture and Water Resources, in conjunction with the Australian Government Department of Health, is progressing enhanced emergency measures and on-board verification of aircraft disinsection in response to these detections at international airports. NAMAC has also established a working group to develop national best practice guidelines and response protocols for managing exotic mosquito detections / incursions.

### Discussion

NAMAC contributes to a One-Health approach to the control of arboviral disease and malaria by uniting experts from a range of fields to provide strategic advice on the epidemiology, surveillance and management of these diseases. This report describes the epidemiology of arboviral diseases and malaria for the season 1 July 2013 to 30 June 2014, activities undertaken by health authorities in response to human cases, and evidence of virus activity. Sentinel chicken and vector monitoring continue to be an important part of the early warning system for arboviruses in Australia.

Table 9: Exotic mosquito detections at the border, Australia, 2013–14

Date	Species	Location	Method of detection	Source / origin	Action/ mitigation	Surveillance results
Aug 2013	<i>Ae. albopictus</i> (larvae)	Perth / Fremantle (Port)	Cargo inspection	Imported used tyres from Japan	Tyres chlorinated and fumigated. Increased trapping and ground surveillance conducted.	No further exotic mosquitoes detected.
Aug 2013	<i>Ae. albopictus</i> (larvae)	Darwin (East Arm wharf)	Cargo inspection	Machinery parts imported in an open top container from Indonesia	Ultra low volume fogging, receptacle treatment surveys and continued increased trapping. Imported goods fumigated	No further exotic mosquitoes detected.
Nov 2013	<i>Ae. albopictus</i> (1 adult)	Darwin (Port)	CO <sub>2</sub> baited Biogents trap	Unknown/unable to identify source	Ultra low volume fogging, receptacle treatment surveys and continued increased trapping.	No further exotic mosquitoes detected.
Dec 2013	<i>Ae. albopictus</i> (1 adult)	Darwin (East Arm wharf)	CO <sub>2</sub> baited Biogents trap	Unknown/unable to identify source	Ultra low volume fogging, receptacle treatment surveys and continued increased trapping.	No further exotic mosquitoes detected.
Dec 2013	<i>Ae. aegypti</i> (larvae)	Townsville (Port)	Cargo inspection	Imported used oversize tyres from Papua New Guinea	Tyres chlorinated and fumigated. Early intervention meant no further action required	No further exotic mosquitoes detected.
Feb 2014	<i>Ae. aegypti</i> (larva)	Brisbane (Port)	Cargo inspection	Used machinery from Papua New Guinea	Chlorination of water and fumigation of imported goods. Further actions not deemed necessary.	No further exotic mosquitoes detected.
Feb 2014	<i>Ae. aegypti</i> (adults and larvae)	Perth (Airport)	Ovitrap, Biogents traps and ground surveys	DNA suggests SE Asian origin	Thermal fogging, residual harbourage treatments, receptacle treatment surveys and increased trapping.	Sporadic detections continued to July 2014.
Mar 2014	<i>Ae. aegypti</i> (adult, pupae and larvae)	Adelaide (Airport)	Ovitrap, Biogents trap and ground surveys	DNA suggests SE Asian origin	Ultra low volume fogging, residual harbourage treatments, receptacle treatment surveys and increased trapping.	No further exotic mosquitoes detected.
Mar 2014	<i>Ae. aegypti</i> (adult, pupae and larvae)	Melbourne (Airport)	Ground surveys,	DNA suggests SE Asian origin	Thermal fogging, residual harbourage treatments, receptacle treatment surveys and increased trapping.	No further exotic mosquitoes detected.
Apr 2014	<i>Ae. aegypti</i> (1 adult)	Perth (Airport)	Octenol baited Biogents trap	DNA suggests SE Asian origin	Residual harbourage treatments, receptacle treatment surveys and increased trapping.	Sporadic detections continued to July 2014.
Apr 2014	<i>Ae. albopictus</i> (1 adult)	Perth (Airport)	Sticky ovitrap	Unknown/unable to identify source.	Thermal fogging, breeding site surveys and increased trapping.	Sporadic detections continued to July 2014.
May 2014	<i>Ae. aegypti</i> (1 adult)	Perth (Airport)	Octenol baited Biogents trap	DNA suggests SE Asian origin	Thermal fogging, residual harbourage treatments, receptacle treatment surveys and increased trapping.	One further detection in July 2014
May 2014	<i>Cx. gelidus</i> (1 adult)	Perth (Airport)	Sticky ovitrap	Unknown/unable to identify source.	Thermal fogging, residual harbourage treatments, receptacle treatment surveys and increased trapping.	One further detection in July 2014

In 2013–14, the number of notifications of BFV infection and the population rates declined markedly compared with the previous year, following the recognition of the ‘epidemic’ of false positive IgM diagnoses that was reported previously, and which began in October 2012. On recommendation from NAMAC, the Case Definitions Working Group of CDNA undertook a review of surveillance case definition for BFV infection and for RRV infection. Under the revised case definition, a single IgM positive result will no longer constitute laboratory evidence for infection, and where a single result is IgM and IgG positive, it may be notified as a probable case. A confirmed case will require IgG seroconversion or a significant increase in IgG antibody level (e.g. 4-fold or greater rise in titre). There is currently no plan to undertake a retrospective revision of notifications to apply the revised case definitions because there is insufficient information on the diagnosis method available in NNDSS. Therefore, the historical data prior to the change of case definition will continue to be considered unreliable. The new case definition was implemented on 1 January 2016.

There were only a small number of ZIKV infections reported in Australia in 2013–14. All were acquired in Pacific Island countries (12/13 in the Cook Islands). These infections were not thought to be cause for serious public health concern at the time, due to the high rate of asymptomatic infection, and that symptomatic cases were generally mild, notwithstanding the reports of a possible association with Guillain-Barré syndrome.<sup>34</sup> Subsequent to the 2013–14 season, ZIKV spread rapidly through many countries in the Americas after being first confirmed in Brazil in May 2015.<sup>35,36</sup> The virus was thought to have been introduced to Brazil during the August 2014 World Sprint Championship canoe race, held in Rio de Janeiro, which attracted participants from 4 Pacific Island nations, including French Polynesia, with active ZIKV transmission.<sup>37</sup> An increase in microcephaly in Brazil with geographical and temporal links to ZIKV was reported in November 2015, and the World Health Organization declared the clusters of microcephaly and neurological disorders a Public Health Event of International Concern on 1 February 2016.<sup>38</sup> There is strong scientific consensus that the virus can be transmitted *in utero* and can cause severe birth defects such as microcephaly,<sup>39</sup> and that it can cause Guillain-Barré syndrome.<sup>40,41</sup>

During 2013–14, there was a sharp increase in notifications of CHIKV infection, with nearly twice as many notifications as the 5-year mean. Indonesia continues to be the major source country for CHIKV infections in Australia. Recent widespread emergence and re-emergence of CHIKV, DENV and ZIKV in the south Pacific have had

serious impacts for local populations. CHIKV infection was first reported in the Pacific Islands in February 2011 in New Caledonia, and in 2013 and 2014, it emerged in Papua New Guinea, New Caledonia, Yap State, Tonga, American Samoa, Tokelau, Samoa, and in French Polynesia where an outbreak affected up to 25% of the local population. There is no evidence of any local transmission of CHIKV in Australia to date, but these outbreaks in the South Pacific have been cause for particular concern in areas of Queensland where there is a risk of local transmission. In July 2014, Queensland Health released the Queensland Chikungunya Management Plan 2014–2019, available on the [Queensland Health web site](https://www.health.qld.gov.au/publications/clinical-practice/guidelines-procedures/diseases-infection/governance/chikungunya-management-plan.pdf) (<https://www.health.qld.gov.au/publications/clinical-practice/guidelines-procedures/diseases-infection/governance/chikungunya-management-plan.pdf>).

With a number of detections of *Ae. aegypti* and *Ae. albopictus* at international airports around Australia during the year, there is the threat of establishment of these vectors of dengue and chikungunya. There is also the risk of isolated cases where transient incursions of infected mosquitoes occur, as seen in Western Australia in 2013–14, and previously reported in the Northern Territory in 2010.<sup>42</sup> Used tyres and exposed machinery continue to be a high risk pathway for the introduction of exotic mosquitoes. The NAMAC guidelines under development to manage exotic mosquito incursions will be an important tool to ensure the use of best practice around the country.

The prevention of incursion of DENV vectors into densely populated areas of South-East Queensland where imported DENV cases are regularly notified, is a continuing priority in Queensland. Despite regular seasonal outbreaks relating to transmission from imported cases, mosquito and infection control measures undertaken by public health authorities and by residents have ensured that DENV has not become endemic in north Queensland. The Queensland Dengue Management Plan 2010–15 provides clear guidance on ongoing prevention, sporadic case response and outbreak management.<sup>18</sup>

The number of imported cases of dengue in Australia continues to increase each year, reflecting the continuing increase in dengue in important source countries such as Indonesia, and elsewhere in South East Asia. While there is progress towards development of a dengue vaccine, efficacy in prevention of infection by the most promising candidate is disappointing, and the results on whether it can prevent hospitalisations with severe dengue are mixed.<sup>43</sup> Along with the failure of traditional prevention through vector control in endemic countries, this highlights the need for development

and application of novel strategies such as the use of *Wolbachia* to prevent transmission of dengue in mosquitoes infected with the bacterium.<sup>44</sup>

Continued vigilance and the involvement of all relevant sectors enable the rapid detection of and early response to the threat of arboviral disease and malaria in Australia. The expert advice provided by NAMAC to the Australian Health Protection Principal Committee, CDNA and health departments has a vital role in mitigating mosquito-borne disease threats. Into the future, NAMAC strives for a reduction in the number of arbovirus cases in Australia, a strengthened disease prediction capacity to allow planning for response, and to retain, build and disseminate expertise and knowledge pertaining to mosquito-borne diseases.

## Appendix

### Australian Capital Territory

There were no vertebrate, vector and climate surveillance programs in the Australian Capital Territory.

### New South Wales

Surveillance mechanisms include mosquito monitoring, virus isolation from mosquitoes and sentinel chicken surveillance. The New South Wales Arbovirus Surveillance and Vector Monitoring Program is funded and coordinated by the NSW Ministry of Health (NSW Health), and laboratory services are contracted to the Institute of Clinical Pathology and Medical Research, Pathology West at Westmead Hospital. Mosquito trapping occurs from mid-spring to mid-autumn (November to April), and mosquitoes are collected weekly for species identification and quantification, and processed for isolation of arboviruses. Data on the Southern Oscillation Index, rainfall and temperature obtained from the Bureau of Meteorology are used by members of the program to predict mosquito-breeding capabilities and potential arboviral activity, while climatic data are used to predict MVEV outbreaks. Sentinel chickens are operated along with mosquito monitoring and isolation at inland locations of major population centres at risk of MVEV, while along the coast where MVEV does not occur, only mosquito monitoring and viral isolation are undertaken.

The NSW Chicken Sentinel Program was approved by the Western Sydney Local Health Network Animal Ethics Committee. This approval requires that the chicken handlers undergo training to ensure the chickens are cared for appropriately and that blood sampling is conducted in a manner that minimises trauma to the chickens. The chickens

are cared for and bled by local council staff and members of the public. Laboratory staff members are responsible for training the chicken handlers. A veterinarian (usually the Director of Animal Care at Westmead) must inspect all new flock locations prior to deployment to ensure animal housing is adequate. Existing flocks are inspected approximately every 2 years. The health of each flock is reported weekly, and is independently monitored by the Animal Ethics Committee via the Director of Animal Care. Full details of the bleeding method and laboratory testing regimen were detailed in the 2003–04 NSW Arbovirus Surveillance Program annual report.<sup>45</sup>

The results of chicken serology are disseminated via email to the relevant government groups as determined by NSW Health and are placed on the NSW Arbovirus Surveillance website. Confirmed positives are notified by telephone to NSW Health and CDNA.

### Northern Territory

Sentinel chicken flocks in the Northern Territory are maintained, bled and tested for MVEV and KUNV in a combined program between the Northern Territory Department of Health, the virology laboratories of the Northern Territory Department of Primary Industries and Fisheries and volunteers.

Surveillance consists of monthly routine sentinel chicken surveillance during the high risk period for MVEV, with flocks located in Leanyer (Darwin), Howard Springs, Coastal Plains Research Station at Beatrice Hill (Darwin region), Katherine, Nhulunbuy, Nathan River, Tennant Creek and Alice Springs. When chickens from a flock show antibodies to MVEV during a prime risk period, a media warning is issued for the general region. These warnings advise Northern Territory residents and visitors of the need to take added precautions to avoid mosquito bites. In 2013–14, sentinel chickens were bled between December 2013 and August 2014.

In addition, ad hoc virus isolation from mosquitoes is carried out when MVEV or KUNV disease cases are reported. The Northern Territory Mosquito Borne Disease Control Program assists regional authorities with mosquito monitoring and provides some funding for direct mosquito control. In 2013–14, routine adult mosquito trapping consisted of 14 trapping sites throughout the Darwin urban area. In other Northern Territory regions, adult mosquito trapping is carried out in liaison with Environmental Health and mining companies, with 6 traps located in Nhulunbuy, 3 in Alyangula on Groote Eylandt, 4 in Katherine, 3 in Tennant

Creek and 6 in Alice Springs. Climate information from the Bureau of Meteorology is used in conjunction with chicken and vector surveillance. Rainfall patterns, daily rainfall records and rain threshold models are used to assist in predicting mosquito and virus activity.

### Queensland

Mosquito monitoring is performed by some local councils, primarily for salt water and fresh water mosquitoes. Some councils perform surveillance for container-inhabiting mosquitoes in domestic and commercial premises as part of a joint Queensland Health and local government initiative. This surveillance comprises various methods including the use of Biogents traps, GATs, ovitraps and larval survey.

Evaluation of ovitraps and GATs in the south western towns of Charleville and Roma was undertaken to determine the water retention capacity of various CIM surveillance tools and to ascertain the most appropriate system for the region. These towns were selected as *Ae. aegypti* had previously been detected in both locations. Each town had a set of 4 traps (GAT, standard ovitrap, double ovitrap and large ovitrap) placed at 3 locations. The relevant local government set the traps and collected the data. The trial commenced in February and continued to April 2014.

Also of note, a novel urban surveillance program using ovitraps and GATs was deployed for the first time in the Brisbane metropolitan region across 200 sites. Eggs collected in ovitraps were identified using real time polymerase chain reaction (RT-PCR) by Queensland Health Forensic and Scientific Services (van den Hurk et al., unpublished data).

The Torres Strait *Aedes albopictus* prevention and control program conducted by Cairns Public Health Unit targets mosquito habitat to minimise the threat of a mainland *Ae. albopictus* incursion from Torres Strait region. This is an ongoing program with recurrent funding from the Australian Government Department of Health. As part of the program, selected *Ae. albopictus* harbourage sites were treated with residual pyrethroid insecticide at high risk locations on both Thursday and Horn islands, the main population and transport hubs in Torres Strait. Lethal tyre traps were deployed near sea cargo depots on Thursday and Horn islands and the airport on Horn Island to control gravid container inhabiting mosquitoes.

On the mainland, human-bait sweep-net sampling was conducted on at least 60 selected suitable sites across the 5 Northern Peninsula Area com-

munities; Seisia, New Mapoon, Bamaga, Injinoo and Umagico. House-to-house yard inspections for larval sampling were also conducted in Seisia, New Mapoon and Bamaga.

The Cairns office of the Australian Government Department of Agriculture carried out sugar-baited FTA card based arbovirus surveillance utilising passive box traps in the Northern Peninsula Area of Cape York mainly targeting JEV during the high risk period of January to May.

A Mosquito and Arbovirus Research Committee-funded project evaluated a sugar-based virus surveillance system using passive box traps in peri-urban locations across south-east Queensland. Passive box traps containing sugar feeding stations with FTA cards were deployed at 2 locations in each of Brisbane City, Sunshine Coast Regional and Gold Coast City council areas between December 2013 and March 2014. Cards were analysed by real-time TaqMan RT-PCR (van den Hurk et al. 2014) for the presence of RRV and BFV.

### South Australia

Across South Australia, mosquito management activities are conducted in partnership between SA Health, the Uni SA, and local government. The program is focused on the Riverland and Murraylands areas where arbovirus is endemic, and extends to a range of coastal areas in regional and metropolitan localities of the State. SA Health funds half of local government costs for mosquito surveillance and control on public land through the South Australian Mosquito Management Subsidy.

The Uni SA's Mosquitoes and Public Health Research Group conducted mosquito surveillance trapping at 35 locations on 11 occasions from September 2013 to April 2014 for 7 South Australian local councils along the River Murray (Renmark Paringa Council, Berri Barmera Council, the District Council of Loxton Waikerie, the Mid-Murray Council, the Rural City of Murray Bridge, the Coorong District Council and Alexandrina Council).

The South Australian Sentinel Surveillance Program (SASSP) operated from September 2013 to March 2014. The SASSP consists of 5 backyard flocks of 5 chickens located along the River Murray in South Australia in Paringa, Loxton, Waikerie (Qualco), Murray Bridge and Meningie.

### Tasmania

No state-wide systematic mosquito abundance, virus isolation or sentinel chicken surveillance activities are undertaken due to the relatively



low risk of arbovirus transmission in the State. However, mosquito collections are undertaken ad hoc in Sorell Council region, (which includes mosquito breeding areas, is fairly populous, and is close to Hobart). This is undertaken during high risk periods over January to March when tidal inundation floods salt marsh habitat, thereby leading to egg hatching and subsequent increased abundance of the main local vector, *Ae. camptorhynchus*. These samples are sent to Westmead Hospital for species identification and viral isolation.

### Victoria

The Victorian Department of Human Services contracts the Victorian Department of Economic Development, Jobs, Transport and Resources to conduct sentinel chicken surveillance, mosquito species identification and arbovirus detection during the arbovirus season from November to April. The routine sentinel chicken monitoring program involves the weekly collection of blood samples from 20 chickens located at each of 9 sites in northern Victoria along the Murray River or in the surrounding region. This program has been in place in Victoria since the 1974 MVEV outbreak and acts as an early warning system for possible human infections with flaviviruses. Flocks are replaced annually. Seven councils undertake mosquito surveillance as part of the routine mosquito monitoring program, which involves the weekly trapping of mosquitoes at 4 sites within each area. Six councils are located along the Murray and Goulburn River, one is a coastal site in Gippsland. Collections are also received from 3 additional councils located on the Murray River, Bellarine Peninsula and Melbourne. Mosquitoes are sent on cold storage to the Victorian Department of Economic Development, Jobs, Transport and Resources for identification, enumeration and virus isolation. The Victorian Arbovirus Taskforce examines the risk of outbreaks of MVEV using meteorological surveillance data such as the Southern Oscillation Index and rainfall deciles, and Indian Ocean Dipole using respectively the Forbes,<sup>27</sup> and Nicholls<sup>28</sup> and Bennett models.

### Western Australia

During 2013–14 the University of Western Australia Arbovirus Surveillance and Research Laboratory (ASRL) was funded by the Western Australian Department of Health to coordinate the sentinel chicken program and mosquito surveillance, and to provide confirmatory serological testing for other sentinel chicken programs in Australia, as required. The flavivirus sentinel chicken program in Western Australia was undertaken by the ASRL at The University of Western Australia, on behalf of the Western Australian Department of Health.

The sentinel chicken surveillance program was approved by The University of Western Australia Animal Ethics Committee. Many state and local government authorities and community volunteers also took part in the program. Twenty-seven sentinel chicken flocks (of up to 12 chickens) were located at major towns and communities in the Kimberley, Pilbara, Gascoyne, Mid West and Wheatbelt regions of Western Australia (Map 1). The Western Australian flavivirus sentinel chicken program operated all year around. Blood samples were collected from the chickens by environmental health officers or trained volunteers at fortnightly intervals during the peak flavivirus risk season (December to June). At other times, monthly samples were collected unless prolonged flavivirus activity warranted continued fortnightly sampling. Samples were transported to ASRL where they were tested for antibodies to flaviviruses using an epitope blocking ELISA.<sup>46</sup>

To supplement information provided by the flavivirus sentinel chicken program, adult mosquitoes were collected by the ASRL from the north-east Kimberley region of northern Western Australia in April 2014. In addition, the Western Australian Department of Health collected adult mosquitoes in the Pilbara region in October 2013 and January 2014 and the Murchison region in March 2014. These mosquitoes were identified to species and processed for virus isolation to investigate vector species and virus infection rates. In the south-west of Western Australia, adult mosquitoes were collected by the ASRL on a regular basis in the Peel, Leschenault and Capel-Busselton regions for surveillance of RRV and BFV. In the 2013–14 season, mosquito homogenates from these regions were tested by both virus isolation and RT-PCR.

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## Quarterly report

# NATIONAL NOTIFIABLE DISEASES SURVEILLANCE SYSTEM, 1 APRIL TO 30 JUNE 2016

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 67,081 notifications to the National Notifiable Diseases Surveillance System (NNDSS) between 1 April and 30 June 2016 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

**Table 1: Reporting of notifiable diseases by jurisdiction**

Disease	Data received from:
<b>Bloodborne diseases</b>	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
<b>Gastrointestinal diseases</b>	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
Paratyphoid	All jurisdictions
Shiga toxin/verotoxin-producing <i>Escherichia coli</i>	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid fever	All jurisdictions
<b>Quarantinable diseases</b>	
Avian influenza in humans	All jurisdictions
Cholera	All jurisdictions
Middle East respiratory syndrome coronavirus	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
<b>Sexually transmissible infections</b>	
Chlamydia	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions
Syphilis - congenital	All jurisdictions

Table 1 continued: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
<b>Vaccine preventable diseases</b>	
Diphtheria	All jurisdictions
<i>Haemophilus influenzae</i> type b	All jurisdictions
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease – invasive	All jurisdictions
Poliovirus infection	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Varicella zoster (chickenpox)	All jurisdictions except New South Wales
Varicella zoster (shingles)	All jurisdictions except New South Wales
Varicella zoster (unspecified)	All jurisdictions except New South Wales
<b>Vectorborne diseases</b>	
Barmah Forest virus infection	All jurisdictions
Chikungunya virus infection	All jurisdictions except Australian Capital Territory
Dengue virus infection	All jurisdictions
Flavivirus infection (unspecified)	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
<b>Zoonoses</b>	
Anthrax	All jurisdictions
Australian bat lyssavirus infection	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus infection (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
<b>Other bacterial infections</b>	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection – invasive	All jurisdictions
Tuberculosis	All jurisdictions

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2016, by date of diagnosis\*

Disease	State or territory								Total 2nd quarter 2016	Total 1st quarter 2016	Total 2nd quarter 2015	Last 5 years mean 2nd quarter	Ratio	Year to date 2016	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
<b>Bloodborne diseases</b>															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Hepatitis B (newly acquired)†	0	2	0	13	0	0	17	9	41	38	38	41.4	1.0	83	89.8
Hepatitis B (unspecified)†	22	666	32	284	78	13	537	244	1,573	1,501	1,583.2	1.2	1.2	3,458	3,166.6
Hepatitis C (newly acquired)†	4	3	1	0	8	4	26	29	89	104	107.6	0.7	0.7	164	222.0
Hepatitis C (unspecified)†	48	1,081	61	703	126	52	664	327	3,018	2,452	2,456.2	1.2	1.2	6,084	4,938.0
Hepatitis D	0	4	2	1	2	0	3	0	15	12	13.4	0.9	0.9	27	25.8
<b>Gastrointestinal diseases</b>															
Botulism	0	0	0	0	0	0	0	0	0	1	1	0.8	0.0	0	1.4
Campylobacteriosis	125	NN	111	1,625	608	207	1,752	759	5,876	5,006	4,031.8	1.3	1.3	11,109	8,732.4
Cryptosporidiosis	10	311	109	685	103	5	205	71	2,299	971	830.2	1.8	1.8	3,812	2,065.8
Haemolytic uraemic syndrome	0	0	0	0	0	1	0	0	4	4	3.6	0.3	0.3	5	9.2
Hepatitis A	0	9	0	4	2	0	6	2	53	28	36.2	0.6	0.6	78	102.4
Hepatitis E	0	1	0	3	0	0	0	1	15	6	10.4	0.5	0.5	20	23.2
Listeriosis	0	12	0	5	0	1	4	1	27	18	19.0	1.2	1.2	50	41.4
Paratyphoid	0	4	0	2	0	1	5	1	33	12	16.4	0.8	0.8	46	44.4
STEC <sup>s</sup>	0	8	0	3	31	0	7	6	60	35	27.0	2.0	2.0	115	60.4
Salmonellosis	78	1,067	245	1,209	461	75	929	451	6,488	4,044	3,252.2	1.4	1.4	11,059	8,117.2
Shigellosis	2	88	40	30	11	2	154	21	348	244	157.2	2.2	2.2	712	379.8
Typhoid fever	0	4	0	6	1	0	6	2	37	29	24.8	0.8	0.8	56	79.0
<b>Quarantinable diseases</b>															
Avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0	0	0.0
Cholera	0	0	0	0	0	0	0	0	0	1	2.2	0.0	0.0	0	2.6
Middle East respiratory syndrome coronavirus	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0.0	0	0.4

Table 2 continued: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2016, by date of diagnosis\*

Disease	State or territory								Total 2nd quarter 2016	Total 1st quarter 2016	Total 2nd quarter 2015	Last 5 years mean 2nd quarter	Ratio	Year to date 2016	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
<b>Sexually transmissible infections</b>															
Chlamydia <sup>¶¶</sup>	362	6,672	663	5,794	1,418	446	1,154	3,077	19,586	18,620	16,560	20,160.0	1.0	38,238	42,224.6
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Gonococcal infection <sup>¶¶</sup>	39	1,818	437	938	332	23	1,751	834	6,172	5,584	4,560	3,800.2	1.6	11,782	7,669.8
Syphilis <2 years duration <sup>¶¶</sup>	5	186	41	160	21	1	271	68	753	776	631	450.2	1.7	1,541	896.6
Syphilis >2 years or unspecified duration <sup>¶¶</sup>	7	89	10	69	26	5	219	13	438	471	487	415.8	1.1	911	819.6
Syphilis – congenital	0	0	0	0	0	0	0	0	0	0	1	0.6	0.0	0	1.4
<b>Vaccine preventable diseases</b>															
Diphtheria	0	0	0	1	0	0	0	0	1	1	0	0.8	1.3	2	1.2
<i>Haemophilus influenzae</i> type b	0	1	0	1	1	0	2	0	5	4	4	4.8	1.0	9	7.6
Influenza (laboratory confirmed)	71	2,633	52	2,139	492	73	1,041	767	7,268	5,772	9,757	5,661.4	1.3	13,065	8,589.0
Measles	0	2	0	2	2	3	10	0	19	37	19	33.8	0.6	56	93.8
Mumps	0	12	18	11	5	1	4	137	188	331	99	61.2	3.1	519	115.4
Pertussis	99	2,014	54	463	271	9	653	236	3,799	5,766	4,180	4,456.6	0.9	9,666	10,051.6
Pneumococcal disease – invasive	7	146	10	77	33	17	111	39	440	177	401	459.8	1.0	622	676.6
Poliovirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	5	0	1	0	0	3	0	9	3	4	7.4	1.2	12	16.8
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Tetanus	0	0	0	1	0	0	0	0	1	3	1	0.8	1.3	4	2.0
Varicella zoster (chickenpox)	15	NN	14	88	79	22	235	137	590	589	548	470.8	1.3	1,181	908.8
Varicella zoster (shingles)	63	NN	97	18	547	78	520	370	1,693	1,767	1,601	1,251.8	1.4	3,475	2,518.2
Varicella zoster (unspecified)	51	NN	3	1,836	41	36	1,430	369	3,766	3,786	3,272	2,567.8	1.5	7,564	5,154.6
<b>Vectorborne diseases</b>															
Barmah Forest virus infection	0	9	1	86	0	0	0	3	99	141	187	539.0	0.2	240	1,202.0
Chikungunya virus infection	0	1	1	1	3	0	5	2	13	18	29	22.4	0.6	31	47.8
Dengue virus infection	7	122	38	135	29	15	139	179	664	729	427	398.2	1.7	1,400	986.4
Flavivirus infection (unspecified)	0	2	1	10	0	1	2	1	17	52	2	4.2	4.0	70	8.6
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	1.0
Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	0.4
Malaria	1	13	2	21	3	0	19	11	70	70	48	76.4	0.9	141	174.0
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	1	1.6	0.0	0	3.6
Ross River virus infection	2	147	42	883	12	2	39	106	1,233	1,697	2,001	1,360.8	0.9	2,935	4,156.0



Table 2 continued: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2016, by date of diagnosis

Disease	State or territory								Total 2nd quarter 2016	Total 1st quarter 2016	Total 2nd quarter 2015	Last 5 years mean 2nd quarter	Ratio	Year to date 2016	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
<b>Zoonoses</b>															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Australian bat lyssavirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Brucellosis	0	2	0	1	0	0	0	0	0	3	2	5.2	11.8	5	11.8
Leptospirosis	0	3	0	32	0	0	0	2	37	50	17	35.4	81.8	87	81.8
Lyssavirus infection (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	1	0	0	0	0	0	0	1	2	2	11.4	22.4	3	22.4
Q fever	1	37	0	52	3	0	8	4	105	141	144	113.4	232.2	250	232.2
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
<b>Other bacterial infections</b>															
Legionellosis	1	36	0	11	7	5	17	22	99	83	100	103.4	194.4	182	194.4
Leptosy	0	1	0	2	0	0	1	0	4	4	3	3.4	4.8	8	4.8
Meningococcal infection – invasive**	0	15	1	8	6	0	15	1	46	43	48	47.8	84.4	91	84.4
Tuberculosis	8	99	9	42	17	2	86	28	291	320	304	290.6	603.6	611	603.6
<b>Total</b>	<b>1,028</b>	<b>17,421</b>	<b>2,100</b>	<b>17,747</b>	<b>4,873</b>	<b>1,109</b>	<b>12,050</b>	<b>8,357</b>	<b>64,164</b>	<b>67,018</b>	<b>59,951</b>			<b>131,579</b>	

\* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.

† Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland reports hepatitis C newly acquired under hepatitis unspecified.

‡ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.

§ Infection with Shiga toxin/verotoxin-producing *Escherichia coli*.

|| Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens.

¶ The national case definitions for chlamydia, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

\*\* Only invasive meningococcal disease is nationally notifiable. However, New South Wales and the Australian Capital Territory also report conjunctival cases.

NN Not notifiable

NEC Not elsewhere classified

Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment in the cumulative figure from the previous period.

**Table 3: Notification rates of diseases, 1 April to 30 June 2016, by state or territory. (Annualised rate per 100,000 population)\*,†**

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
<b>Bloodborne diseases</b>									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)‡	0.0	0.1	0.0	1.1	0.0	0.0	1.1	1.4	0.7
Hepatitis B (unspecified)§	22.5	35.0	52.4	23.8	18.4	10.1	36.2	37.7	31.5
Hepatitis C (newly acquired)‡	4.1	0.2	1.6	0.0	1.9	3.1	1.8	4.5	1.3
Hepatitis C (unspecified)§	49.1	56.7	99.8	58.8	29.7	40.3	44.7	50.5	51.5
Hepatitis D	0.0	0.2	3.3	0.1	0.5	0.0	0.2	0.0	0.2
<b>Gastrointestinal diseases</b>									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	127.9	NN	181.6	136.0	143.1	160.3	118.0	117.2	128.3
Cryptosporidiosis	10.2	16.3	178.3	57.3	24.2	3.9	13.8	11.0	25.2
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.0	0.0	0.8	0.0	0.0	0.0
Hepatitis A	0.0	0.5	0.0	0.3	0.5	0.0	0.4	0.3	0.4
Hepatitis E	0.0	0.1	0.0	0.3	0.0	0.0	0.0	0.2	0.1
Listeriosis	0.0	0.6	0.0	0.4	0.0	0.8	0.3	0.2	0.4
Paratyphoid	0.0	0.2	0.0	0.2	0.0	0.8	0.3	0.2	0.2
STEC <sup>  </sup>	0.0	0.4	0.0	0.3	7.3	0.0	0.5	0.9	0.9
Salmonellosis	79.8	56.0	400.8	101.2	108.5	58.1	62.5	69.6	75.9
Shigellosis	2.0	4.6	65.4	2.5	2.6	1.5	10.4	3.2	5.9
Typhoid fever	0.0	0.2	0.0	0.5	0.2	0.0	0.4	0.3	0.3
<b>Quarantinable diseases</b>									
Avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Middle East respiratory syndrome coronavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
<b>Sexually transmitted infections</b>									
Chlamydia <sup>  ,**</sup>	370.3	350.3	1,084.7	484.8	333.8	345.3	77.7	475.1	329.4
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	39.9	95.4	714.9	78.5	78.2	17.8	117.9	128.8	103.8
Syphilis < 2 years duration**	5.1	9.8	67.1	13.4	4.9	0.8	18.2	10.5	12.7
Syphilis > 2 years or unspecified duration <sup>§,**</sup>	7.2	4.7	16.4	5.8	6.1	3.9	14.7	2.0	7.4
Syphilis – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
<b>Vaccine preventable diseases</b>									
Diphtheria	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
<i>Haemophilus influenzae</i> type b	0.0	0.1	0.0	0.1	0.2	0.0	0.1	0.0	0.1
Influenza (laboratory confirmed)	72.6	138.2	85.1	179.0	115.8	56.5	70.1	118.4	122.2
Measles	0.0	0.1	0.0	0.2	0.5	2.3	0.7	0.0	0.3
Mumps	0.0	0.6	29.4	0.9	1.2	0.8	0.3	21.2	3.2
Pertussis	101.3	105.7	88.3	38.7	63.8	7.0	44.0	36.4	63.9
Pneumococcal disease – invasive	7.2	7.7	16.4	6.4	7.8	13.2	7.5	6.0	7.4
Poliovirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.3	0.0	0.1	0.0	0.0	0.2	0.0	0.2

**Table 3 continued: Notification rates of diseases, 1 April to 30 June 2016, by state or territory. (Annualised rate per 100,000 population)\*,†**

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	15.3	NN	22.9	7.4	18.6	17.0	15.8	21.2	14.6
Varicella zoster (shingles)	64.4	NN	158.7	1.5	128.8	60.4	35.0	57.1	41.9
Varicella zoster (unspecified)	52.2	NN	4.9	153.6	9.7	27.9	96.3	57.0	93.2
<b>Vectorborne diseases</b>									
Barmah Forest virus infection	0.0	0.5	1.6	7.2	0.0	0.0	0.0	0.5	1.7
Chikungunya virus infection	0.0	0.1	1.6	0.1	0.7	0.0	0.3	0.3	0.2
Dengue virus infection	7.2	6.4	62.2	11.3	6.8	11.6	9.4	27.6	11.2
Flavivirus infection (unspecified)	0.0	0.1	1.6	0.8	0.0	0.8	0.1	0.2	0.3
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.0	0.7	3.3	1.8	0.7	0.0	1.3	1.7	1.2
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	2.0	7.7	68.7	73.9	2.8	1.5	2.6	16.4	20.7
<b>Zoonoses</b>									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.2	0.0	2.7	0.0	0.0	0.0	0.3	0.6
Lyssavirus infection (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Q fever	1.0	1.9	0.0	4.4	0.7	0.0	0.5	0.6	1.8
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
<b>Other bacterial diseases</b>									
Legionellosis	1.0	1.9	0.0	0.9	1.6	3.9	1.1	3.4	1.7
Leprosy	0.0	0.1	0.0	0.2	0.0	0.0	0.1	0.0	0.1
Meningococcal infection – invasive <sup>††</sup>	0.0	0.8	1.6	0.7	1.4	0.0	1.0	0.2	0.8
Tuberculosis	8.2	5.2	14.7	3.5	4.0	1.5	5.8	4.3	4.9

\* The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.

† Rate per 100,000 of population. Annualisation Factor was 4.0

‡ Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland reports hepatitis C newly acquired under hepatitis C unspecified.

§ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.

|| Infection with Shiga toxin/verotoxin-producing *Escherichia coli*.

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens.

\*\* The national case definitions for chlamydia, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

†† Only invasive meningococcal disease is nationally notifiable. However, New South Wales and the Australian Capital Territory also report conjunctival cases.

NEC Not elsewhere classified.

NN Not notifiable.

# AUSTRALIAN CHILDHOOD IMMUNISATION COVERAGE, 1 JANUARY TO 31 DECEMBER COHORT, ASSESSED AS AT 31 MARCH 2016

Alexandra Hendry for the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

## Introduction

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) provides commentary on the trends in ACIR data. For further information please contact NCIRS at: telephone +61 2 9845 1423, email: alexandra.hendry@health.nsw.gov.au

Tables 1, 2 and 3 provide the latest rolling annualised quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR) for all children.

The data show the percentage of all children 'fully immunised' at 12 months, 24 months and 60 months of age, for four 3-month birth cohorts of children assessed at the stated ages between 1 January 2015 and 31 December 2015 using ACIR data up to 31 March 2016. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, and is outlined in more detail below.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of *Haemophilus B* conjugate (PRP-OMP) containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, and 3 doses of 13-valent pneumococcal conjugate vaccine. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 doses of a DTP-containing vaccine, 3 doses of polio vac-

cine, 3 or 4 doses of PRP-OMP Hib, Infanrix Hexa or Hiberix vaccine (3 doses only of Infanrix Hexa or Hiberix if given after 11.5 months of age), or 4 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, 2 doses of a measles-mumps-rubella-containing (MMR) vaccine, 1 dose of meningococcal C vaccine, and 1 dose of varicella vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

A full description of the basic methodology used can be found in *Commun Dis Intell* 1998;22(3):36–37.

## Results

The rolling annualised percentage of all children 'fully immunised' by 12 months of age for Australia increased marginally from the previous report by 0.4 of a percentage point to 92.7% (Table 1). All jurisdictions experienced small increases in the percentage of children 'fully immunised' by 12 months of age. For individual vaccines due by 12 months of age all jurisdictions achieved coverage greater than 92%.

The rolling annualised percentage of all children 'fully immunised' by 24 months of age for Australia increased by 0.8 percentage points for the 2nd consecutive report to reach 90.1% (Table 2). All jurisdictions experienced increases of more than 0.6 percentage points in fully immunised coverage for this age group. Coverage for individual vaccines due by 24 months of age remains above 93% in all

**Table 1. Percentage of children immunised at 12 months of age for the birth cohort 1 January to 31 December 2014, preliminary results, by disease and state or territory; assessment date 31 March 2016**

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	5,698	99,224	3,695	62,926	20,295	5,846	77,189	34,555	309,428
Diphtheria, tetanus, pertussis (%)	95.0	93.6	93.5	93.4	93.4	93.3	93.7	93.2	93.5
Poliomyelitis (%)	95.0	93.5	93.5	93.4	93.4	93.2	93.7	93.2	93.5
<i>Haemophilus influenzae</i> type b (%)	94.5	93.3	93.4	93.2	93.2	93.2	93.3	93.0	93.3
Hepatitis B (%)	94.8	93.3	93.8	93.3	93.2	93.2	93.4	92.9	93.3
Pneumococcal	94.6	93.2	93.5	93.1	93.1	93.2	93.3	92.9	93.2
Fully immunised (%)	94.0	92.7	93.0	92.8	92.7	92.9	92.7	92.4	92.7

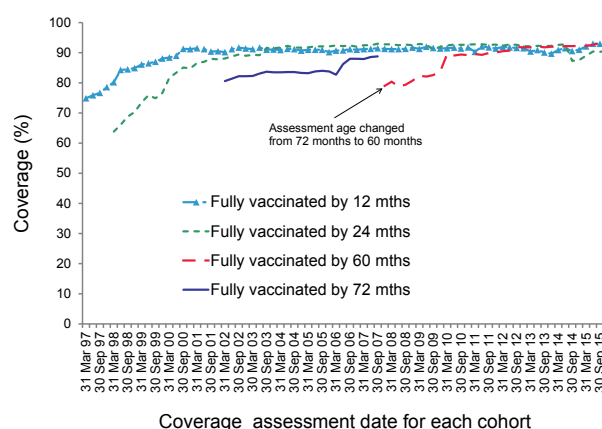
jurisdictions, except for varicella and the MMR vaccine. Coverage for these antigens at 24 months of age have however, continued to improve with varicella coverage increasing from the previous report by 0.3 of a percentage point to 92.1% and MMR increasing by 0.6 of a percentage point to 91.8%.

The rolling annualised percentage of all children ‘fully immunised’ by 60 months of age for Australia increased from the previous report by only 0.1 of a percentage point to 92.7% (Table 3). Coverage for individual vaccines due by 60 months of age remains greater than 91% in all jurisdictions.

The Figure shows the trends in vaccination coverage from the 1st ACIR-derived published coverage estimates in 1997 to the current estimates. Overall, there is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007). Coverage by 24 months did fall below the 12 and 60 month coverage estimates following the change in the 24 month coverage assessment algorithm to include meningococcal dose 1, varicella dose 1, and MMR dose 2 instead of MMR dose 1. However, ‘fully immunised’ coverage by 24 months has been steadily increasing since this change and as at 31 December 2015 reached 90.8%. In the last quarter of 2015 there have been marginal decreases in the coverage estimates by 12 and 60 months to 92.8% for both milestones.

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases is supported by the Australian Government Department of Health, the NSW Ministry of Health and The Children’s Hospital at Westmead. The opinions expressed in this paper are those of the authors, and do not necessarily represent the views of these agencies.

**Figure: Trends in vaccination coverage, Australia, 1997 to 31 December 2015, by age cohorts**



**Table 2. Percentage of children immunised at 24 months of age for the birth cohort 1 January to 31 December 2013, preliminary results, by disease and state or territory; assessment date 31 March 2016**

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	5,632	99,150	3,560	62,164	19,764	5,950	76,598	33,989	306,807
Diphtheria, tetanus, pertussis (%)	96.9	95.5	95.3	95.6	95.6	95.7	96.1	95.6	95.7
Poliomyelitis (%)	96.8	95.5	95.3	95.6	95.5	95.7	96.1	95.6	95.7
<i>Haemophilus influenzae</i> type b (%)	95.9	94.6	94.3	94.9	94.5	94.5	95.1	94.5	94.8
Measles, mumps, rubella (%)	93.4	91.8	91.2	92.2	91.6	92.0	92.1	90.8	91.8
Hepatitis B (%)	96.6	95.3	95.5	95.4	95.3	95.5	95.8	95.2	95.5
Meningococcal C (%)	95.5	94.5	94.4	94.8	93.8	94.6	94.7	93.6	94.5
Varicella (%)	93.9	92.1	90.3	92.2	91.9	91.7	92.6	91.1	92.1
Fully immunised (%)	91.7	89.9	88.5	90.9	89.2	89.7	90.5	88.7	90.1

**Table 3. Percentage of children immunised at 60 months of age for the birth cohort 1 January to 31 December 2010, preliminary results, by disease and state or territory; assessment date 31 March 2016**

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	5,442	100,916	3,490	64,983	20,184	6,130	76,176	34,257	311,578
Diphtheria, tetanus, pertussis (%)	94.3	93.8	93.1	93.1	92.4	94.4	93.7	91.9	93.3
Poliomyelitis (%)	94.2	93.8	93.1	93.1	92.4	94.3	93.7	91.9	93.3
Measles, mumps, rubella (%)	94.2	93.8	93.3	93.0	92.4	94.4	93.7	91.9	93.3
Fully immunised (%)	93.7	93.2	92.1	92.5	91.7	93.7	93.1	91.2	92.7

# AUSTRALIAN MENINGOCOCCAL SURVEILLANCE PROGRAMME, 1 JANUARY TO 31 MARCH 2016

Monica M Lahra, Rodney P Enriquez for the Australian Meningococcal Surveillance Programme

## Introduction

The reference laboratories of the Australian Meningococcal Surveillance Programme (AMSP) report data on the number of cases confirmed by laboratory testing using culture and by non-culture based techniques. Culture positive cases, where *Neisseria meningitidis* is grown from a normally sterile site or skin lesions, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in quarterly reports are restricted to a description of the number of cases by jurisdiction and serogroup, where known. Some minor corrections to data in the Table may be made in subsequent reports if additional data are received. A full analysis of laboratory confirmed cases of IMD in each calendar year is

contained in the AMSP annual reports published in *Communicable Diseases Intelligence*. For more information see *Commun Dis Intell* 2016;40(1):E13.

## Results

Laboratory confirmed cases of invasive meningococcal disease for the period 1 January to 31 March 2016 are shown in the Table.

## Author details

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2. School of Medical Sciences, Faculty of Medicine, The University of New South Wales, New South Wales

**Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2016, by serogroup and state or territory**

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD
Australian Capital Territory	2016	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2015	0	0	0	0	0	0	0	0	0	0	0	0	0	0
New South Wales	2016	0	0	5	5	1	1	1	1	4	4	2	2	13	13
	2015	0	0	3	3	1	1	0	0	1	1	0	0	5	5
Northern Territory	2016	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2015	0	0	1	1	0	0	0	0	0	0	0	0	1	1
Queensland	2016	0	0	3	3	0	0	4	4	2	2	2	2	11	11
	2015	0	0	5	5	0	0	1	1	0	0	0	0	6	6
South Australia	2016	0	0	6	6	0	0	0	0	0	0	0	0	6	6
	2015	0	0	2	2	0	0	0	0	0	0	0	0	2	2
Tasmania	2016	0	0	0	0	0	0	0	0	2	2	0	0	2	2
	2015	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Victoria	2016	0	0	3	3	1	1	0	0	6	6	0	0	10	10
	2015	0	0	8	8	0	0	0	0	1	1	0	0	9	9
Western Australia	2016	0	0	1	1	0	0	0	0	2	2	0	0	3	3
	2015	0	0	2	2	0	0	0	0	0	0	0	0	2	2
Total	2016	0	0	18	18	2	2	5	5	16	16	4	4	45	45
	2015	0	0	21	21	1	1	1	1	2	2	0	0	25	25

# AUSTRALIAN MENINGOCOCCAL SURVEILLANCE PROGRAMME, 1 APRIL TO 30 JUNE 2016

Monica M Lahra, Rodney P Enriquez for the Australian Meningococcal Surveillance Programme

## Introduction

The reference laboratories of the National Neisseria Network, Australia report laboratory data on invasive meningococcal disease (IMD) cases confirmed by laboratory testing using culture and non-culture based techniques for the Australian Meningococcal Surveillance Programme. Culture positive cases, where *Neisseria meningitidis* is grown from a normally sterile site or skin lesions, and non-culture based diagnoses, derived from results of nucleic acid amplification testing and serological techniques, are defined as IMD according to Public Health Laboratory Network definitions. Data contained in quarterly reports are usually restricted to a description of the numbers of cases by jurisdiction and serogroup, where known.

## Results

Of note in this quarter 2016 is the number and proportion of IMD caused by serogroup W. In the years 2007 to 2011 the proportion of IMD caused by serogroup W in Australia ranged from 1.8% to 4.5%, and increased to 8.6% to 9.9% in 2013 to 2014. In 2015, this increased markedly to 31/81 (21.4%) of the IMD in Australia. In 2015, 25/31 serogroup W IMD strains were genotyped, and 81% were

sequence type (ST)-11, and had the *porA* antigen encoding gene type P1.5,2, the same genotype as the hypervirulent serogroup W strain reported in the United Kingdom and South America since 2009. Nationally enhanced surveillance strategies, including whole genome sequencing and phylogenetic inference, has been applied to the recent emergence in Australia of *N. meningitidis* serogroup W in Australia.

Some minor corrections to data in the Table below may be made in subsequent reports if additional data are received. A full analysis of laboratory confirmed cases of IMD in each calendar year is contained in the AMSP annual report published in *Communicable Diseases Intelligence*. For more information see *Commun Dis Intell* 2016;40(1):E13.

## Author details

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1. Neisseria Reference Laboratory and World Health Organisation Collaborating Centre for STD, Sydney. Department of Microbiology, South Eastern Area Laboratory Services, The Prince of Wales Hospital, Randwick, NSW
2. School of Medical Sciences, Faculty of Medicine, The University of New South Wales, New South Wales

**Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2016, by serogroup and state or territory**

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD
Australian Capital Territory	2016	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2015	0	0	1	1	0	0	0	0	0	0	0	0	1	1
New South Wales	2016	0	0	2	7	0	1	3	4	5	9	0	2	10	23
	2015	0	0	9	12	0	1	2	2	1	2	1	1	13	18
Northern Territory	2016	0	0	2	2	0	0	0	0	0	0	0	0	2	2
	2015	0	0	1	1	0	0	0	0	0	0	0	0	1	1
Queensland	2016	0	0	2	5	0	0	2	6	3	5	2	2	9	18
	2015	0	0	4	9	0	0	0	1	0	0	1	1	5	11
South Australia	2016	0	0	5	11	0	0	0	0	0	0	0	0	5	11
	2015	0	0	7	9	0	0	0	0	0	0	0	0	7	9
Tasmania	2016	0	0	0	0	0	0	0	0	0	2	0	0	0	2
	2015	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Victoria	2016	0	0	4	7	0	1	2	2	9	15	0	0	15	25
	2015	0	0	8	16	0	0	2	2	5	6	0	0	15	24
Western Australia	2016	0	0	1	2	0	0	0	0	1	3	0	0	2	5
	2015	0	0	2	4	0	0	1	1	0	0	0	0	3	5
Total	2016	0	0	16	34	0	2	7	12	18	34	2	4	43	86
	2015	0	0	32	52	0	1	5	6	6	8	2	2	45	69

# AUSTRALIAN SENTINEL PRACTICES RESEARCH NETWORK, 1 APRIL TO 30 JUNE 2016

Monique B-N Chilver, Daniel Blakeley, Nigel P Stocks for the Australian Sentinel Practices Research Network

## Introduction

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is funded by the Australian Government Department of Health, owned and operated by the Royal Australian College of General Practitioners and directed through the Discipline of General Practice at the University of Adelaide.

The network consists of general practitioners and nurse practitioners, Australia wide, who report syndromic presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can inform public health officials of the epidemiology of pandemic threats in the early stages of a pandemic, as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Reporters currently submit data via automated data extraction from patient records, web-based data collection or paper form.

In 2010, virological surveillance was established allowing ASPREN practitioners to collect nasal swab samples for laboratory viral testing of a proportion of influenza-like illness (ILI) patients for a range of respiratory viruses including influenza A and influenza B. In 2016, practitioners are instructed to swab 20% of all patients presenting with an ILI.

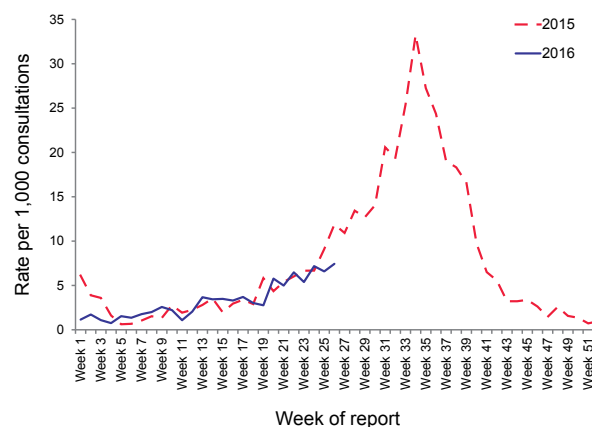
The list of conditions reported is reviewed annually by the ASPREN management committee. In 2016, 4 conditions are being monitored. They include ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, published in *Commun Dis Intell* 2016;40(1):E14.

## Results

Sentinel practices contributing to ASPREN were located in all 8 states and territories in Australia. A total of 240 general practitioners regularly contributed data to ASPREN in the 2nd quarter of 2016. Each week an average of 217 general practitioners provided information to ASPREN at an average of 17,481 (range 16,128 to 18,770) consultations per week and an average of 153 (range 118 to 202) notifications per week (all conditions).

ILI rates reported from 1 April to 30 June 2016 averaged 4.9 cases per 1,000 consultations (range 2.8 to 7.5 cases per 1,000 consultations) weighted / 5.5 cases per 1,000 consultations (range 3.0 to 6.6 cases per 1,000 consultations) unweighted. This was similar to the rates in the same reporting period in 2015, which averaged 5.4 cases per 1,000 consultations (range 2.0 to 11.9 cases per 1,000 consultations, Figure 1) weighted / 5.5 cases per 1,000 consultations (range 2.2 to 10.5 cases per 1,000 consultations, Figure 1) unweighted. ILI rates started to increase at the end of the reporting period with rates in week 26 being 7.4 ILI cases per 1,000 consultations weighted / 6.4 ILI cases per 1,000 consultations unweighted.

**Figure 1: Consultation rates for influenza-like illness, ASPREN, 2015 and 1 January to 30 June 2016, by week of report**



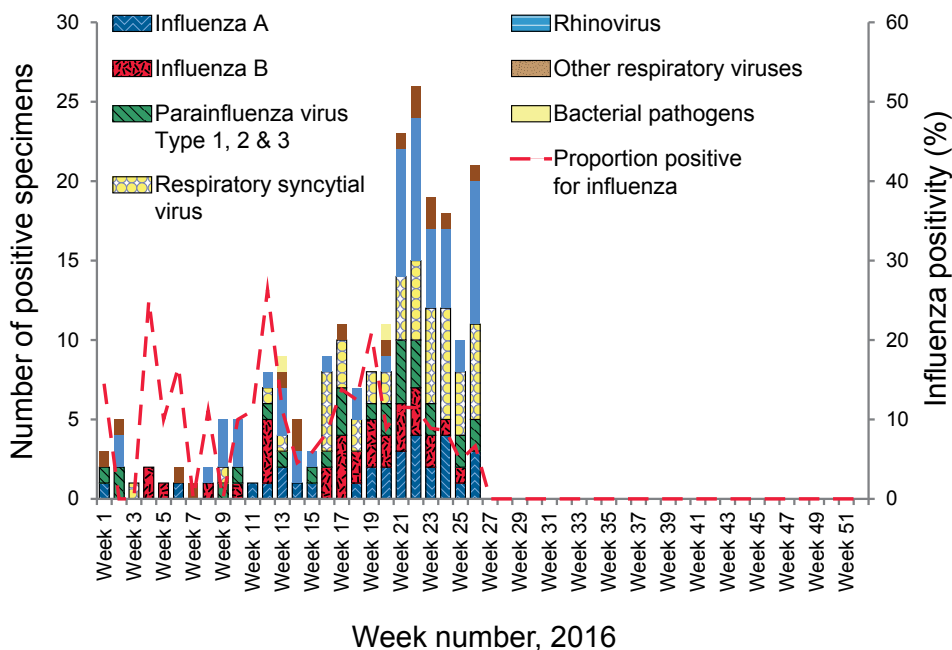
The ASPREN ILI swab testing program continued in 2016 with 487 tests being undertaken from 1 April to 30 June. The most commonly reported virus during this reporting period was respiratory syncytial virus (9.4% of all swabs performed, Figure 2), with the 2nd most common virus being rhinovirus (9.2% of all swabs performed).

From the beginning of 2016 to the end of week 26, 62 cases of influenza were detected with 32 of these typed as influenza B (5.2% of all swabs performed) and the remaining 30 being influenza A (4.9% of all swabs performed) (Figure 2).

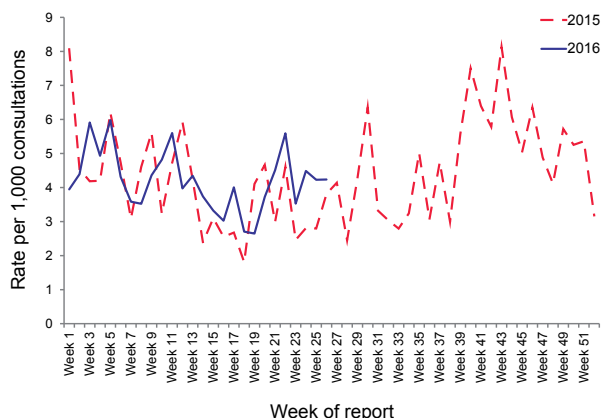
During this reporting period, consultation rates for gastroenteritis averaged 3.8 cases per 1,000 consultations (range 2.7 to 5.6 cases per 1,000, Figure 3). This was slightly higher than the rate in



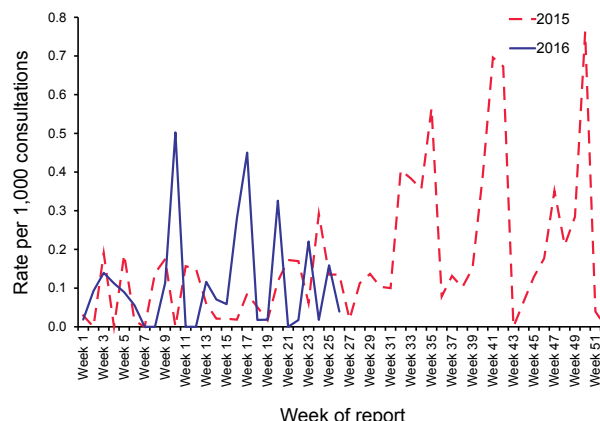
**Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January to 30 June 2016, by week of report**



**Figure 3: Consultation rates for gastroenteritis, ASPREN, 2015 and 1 January to 30 June 2016, by week of report**



**Figure 4: Consultation rates for chickenpox, ASPREN, 2015 and 1 January to 30 June 2016, by week of report**

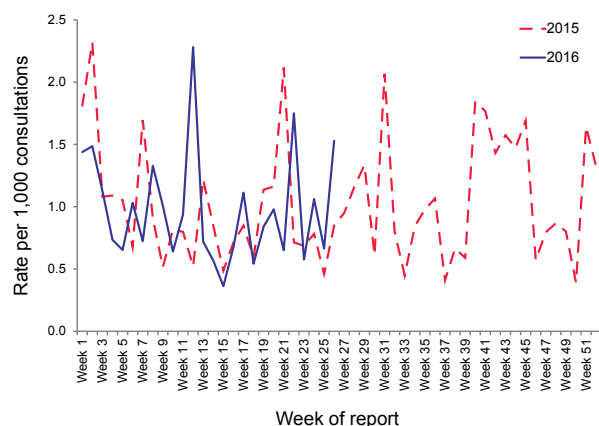


the same reporting period in 2015 where the average was 3.1 cases per 1,000 consultations (range 1.8 to 4.7 cases per 1,000).

Varicella infections were reported at a similar rate for the 2nd quarter of 2016 compared with the same period in 2015. From 1 April to 30 June 2016, recorded rates for chickenpox averaged 0.1 cases per 1,000 consultations (range 0.0 to 0.5 cases per 1,000 consultations, Figure 4).

In the 2nd quarter of 2016, reported rates for shingles averaged 0.9 cases per 1,000 consultations (range 0.4 to 1.8 cases per 1,000 consultations, Figure 5). This was similar to the rates in the same reporting period in 2015 where the average shingles rate was 0.9 cases per 1,000 consultations (range 0.5 to 2.1 cases per 1,000 consultations).

**Figure 5: Consultation rates for shingles, ASPREN, 2015 and 1 January to 30 June 2016, by week of report**



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