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Editorial

Polio anywhere is a risk everywhere

David N Durrheim, Anthony Adams

The 2014 diagnosis of poliomyelitis-like illness in a middle-aged Victorian man on his return to Australia after working in the Horn of Africa, highlights the possibility of poliovirus being imported into any country while the virus circulates anywhere.[1] This possibility is also illustrated by Australia's last confirmed incursion of poliovirus. In 2007 a Melbourne university student was confirmed with polio virus infection after returning from a visit to Pakistan.[2] Thus the article by Martin et al in this issue of CDI is a timely reminder of the importance of: maintaining high childhood immunisation coverage; encouraging travellers into and out of Australia to check their immunisation status against polio; and ensuring strong surveillance throughout the health system for polio-like illness.[3,4]

Progress by the Pan American Health Organization during the 1980s to interrupt indigenous wild poliovirus transmission in the Western Hemisphere led to the ambitious global initiative to eradicate poliomyelitis.[5] This was endorsed by the World Health Assembly in 1988 and in the past quarter of a century progress has been remarkable, with the annual poliomyelitis incidence reduced by more than 99%. Only 3 countries (Nigeria, Pakistan and Afghanistan) have never interrupted indigenous poliovirus transmission. The Western Pacific Region was declared polio-free on 29 October 2000 and in March 2014, following the amazing efforts of India to interrupt transmission, the South-East Asian Region has joined the Americas (1994), Western Pacific and European Regions (2002) in being certified as having successfully interrupted poliovirus transmission.[6,7]

There is no evidence that wild serotype 2 and 3 polioviruses are still circulating anywhere in the world. In 2013 all wild poliomyelitis cases were due to serotype 1. Unfortunately 63 cases of vaccine-derived poliovirus type 2 occurred in 7 countries during 2013 and this has resulted in a decision to replace the trivalent oral vaccine with a bivalent oral vaccine containing serotypes 1 and 3 virus and at least a single dose of inactivated trivalent vaccine as insurance.[8,9]

Major hurdles must still be overcome to complete the job of eradication. In 2012, 223 polio cases were confirmed in 5 countries, while in 2013 this increased to 407 cases in 8 countries.[8] Persistent safe havens for poliovirus in northern Nigerian states and in the border areas between Pakistan and Afghanistan have repeatedly seeded virus to other countries, both neighbouring countries and further afield. Perpetual underperformance of immunisation programs has been complicated by insurgency, targeted killing of polioworkers and diabolical political folly.[10] Some countries that have received imported poliovirus have risen successfully to meet the considerable challenge and expense of stamping out the importation. In China, for example, this required an extensive response with 5 mass campaigns, 44 million doses of oral poliovirus vaccine administered and a direct cost of US$26 million in outbreak response activities.[11] However, the frail nature of health services in other seeded countries and/or their political instability has resulted in polio again establishing a foothold.[12]

The urgency for achieving eradication was recognised by the World Health Organization this year with the declaration that the international spread of wild poliovirus is a public health emergency of international concern. This was accompanied by strong recommendations that Pakistan, Syria, and Cameroon, countries that have recently exported poliovirus to other countries, ensure that their residents and long-term visitors are fully vaccinated against polio before travelling internationally, and that this be recorded in an International Certificate of Vaccination.[13]

There is an unavoidable obligation on all governments to finish the job of polio eradication for all children worldwide and future generations of children. It would be a travesty if the gains achieved over the past 26 years, through massive financial and human resource investment, were squandered. The domino effect of failing to achieve the polio eradication goal would have a profound detrimental effect on other global health initiatives, including measles and rubella elimination. One huge final effort is required to once and for all rid the world of this infectious scourge.

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Original articles

Australia's polio risk

Nicolee Martin, Beverley J Paterson, David N Durrheim

Abstract

Australia, like all polio-free countries and regions, remains at risk of a wild poliovirus importation until polio is eradicated globally. The most probable route of importation will be through a traveller arriving in Australia either by air or sea from a polio-endemic or re-infected country. While the overall risk of an imported wild poliovirus infection leading to transmission within Australia is assessed as being low, some areas of the country have been identified as at increased risk. Local areas with relatively high arrivals from polio endemic countries, areas of low vaccination coverage and the potential for transmission to occur when these 2 factors are combined, were identified by this review as Australia's main polio risk. The risk of an importation event leading to locally acquired cases is mitigated by generally high polio vaccination coverage in Australia. This high coverage extends to residents of the Torres Strait Islands who are in close proximity to Papua New Guinea, a country identified as at high risk of poliovirus transmission should an importation occur. In 2012, all states and territories had vaccination coverage of greater than 90% at 1 year of age and all exceeded 93% at 2 years of age. Population immunity to wild poliovirus type 1, which remains the major cause of paralysis globally, has been estimated at 82%. This is sufficient to prevent outbreaks of this type in Australia. Of the 211 eligible non-polio acute flaccid paralysis (AFP) cases classified between 2008 and 2011, 91% (193) were vaccinated against polio at least once. High quality surveillance for AFP, which is supplemented by sentinel enterovirus and environmental surveillance activities, gives confidence that an imported case would be detected and appropriate public health action would ensue. *Commun Dis Intell* 2014;38(2):E107–E113.

Keywords: polio, risk assessment, epidemiology, acute flaccid paralysis

Introduction

Australia, as part of the World Health Organization's (WHO) Western Pacific Region, was certified polio-free during the Kyoto meeting in Japan on 29 October 2000. Poliomyelitis is a notifiable condition throughout Australia and laboratory investigation is recommended for cases of any age with a clinical suspicion of poliomyelitis. In 2007, a single imported poliomyelitis case was detected in a university student from Pakistan residing in Melbourne, Australia. While there were no secondary cases, the public health outbreak response was extensive and included the tracing of airline passengers; household; medical clinic and hospital contacts; isolation of the case; home quarantine of close contacts; and vaccination of at risk contacts. This imported case of poliomyelitis in an adult who had reportedly been fully vaccinated as a child, served to highlight the potential for importation in individuals of any age, even with a prior history of vaccination.[1]

Globally, polio is still endemic in 3 countries, Afghanistan, Pakistan and Nigeria, down from 125 countries in 1988, yet the threat of resurgence remains. In addition to cases in all endemic countries, WHO had reported confirmed wild poliovirus infections in Kenya and Somalia at the time of this analysis in 2013.[2] In 2005, an outbreak in this region led to over 700 cases.[2] Polio-free countries and WHO regions remain at risk while poliovirus continues to circulate. Recent examples include the outbreak in the WHO European Region (polio-free since 2002), associated with an importation from India to Tajikistan in 2010, which ultimately resulted in 457 cases, including 29 deaths across 4 countries.[3] Closer to home, the Western Pacific Region experienced an outbreak in China in 2011 caused by an imported wild poliovirus type 1 from Pakistan, which resulted in 21 cases including 2 deaths.[4]

Australia, like all polio-free countries and regions, will remain at risk of a wild poliovirus importation until polio is eradicated globally. As Australia does not share borders with polio affected countries, the main risk will occur through importation from an endemic or re-infected country by a traveller arriving either by air or sea. Papua New Guinea (PNG), one of Australia's closest neighbours, is classified by the WHO as a country at high risk of transmission following an importation of wild poliovirus.[5]

A comprehensive national risk assessment has been undertaken to identify areas at highest risk of transmission should an importation of wild poliovirus occur in Australia. This assessment focuses on 4 main areas: population immunity; program delivery; importation threats; and surveillance.

Data sources and methods

The Australian polio risk assessment was originally conducted by the Office of Health Protection in the Australian Government Department of Health, and reported at the 18th Meeting of the Regional Certification Commission for the Certification of the Eradication of Poliomyelitis in the Western Pacific Region in November 2012. This assessment applies to the risk of polio transmission based on the data available at the time of analysis in early 2013.

Poliomyelitis was notifiable in all Australian states and territories by 1922 and these notification data have been captured by the National Notifiable Diseases Surveillance System since its inception in 1991. There is a national case definition for polio, which was revised in 2010–2011 and implemented in July 2011.

Information on vaccination coverage was extracted from publicly accessible reports published by the National Health Performance Authority,[6] the Australian Childhood Immunisation Register (ACIR) and unpublished ACIR data sourced through the Australian Government Department of Health. Immunisation coverage for residents of the Torres Strait Islands was estimated through coverage data available for postcode 4875, which covers the following islands in the Torres Strait: Badu, Boigu, Coconut, Dauan, Erub, Horn, Kubin Village, Mabuiag, Moa, Murray, Saibai, Stephens, Thursday, Warraber, Yam and Yorke. Population immunity was identified from a national serosurvey of poliovirus immunity in Australia conducted between 1996 and 1999, prior to the cessation of oral polio vaccine use in 2005.[7]

Overseas Arrivals and Departures (OAD) data refers to the arrival and departure of Australian residents or overseas visitors, through Australian air and sea ports, as recorded on incoming or outgoing passenger cards. OAD data describe the number of movements of travellers rather than the number of travellers. Unpublished data on arrivals in Australia from selected countries of residence was sourced from the Department of Immigration and Border Protection (DIBP) for Table 1. These data are obtained from visa information for settler arrivals, and from incoming passenger cards (IPCs) for visitors or temporary entrants and returning residents. Country of residence is self-reported in response to the IPC question 'Your country of residence =' for visitors or temporary entrants and from 'The country where you spent most time abroad=' for returning residents. These statistics exclude the movements of operational air and ships' crew, transit passengers who pass through Australia but are not cleared for entry, passengers on pleasure cruises commencing and finishing in Australia, and unauthorised arrivals such as irregular maritime arrivals (IMAs). For purposes of confidentiality, DIBP did not provide small numbers (less than 5).

Settlement by the Australian Bureau of Statistics Statistical Division information in Table 2 were sourced from DIBP's online Settlement Reporting Facility,[8] which allows customised reports to be generated on statistical data related to permanent arrivals in Australia. These data represent the last address known to DIBP of permanent arrivals of all migration streams from the following selected countries of birth (Afghanistan, Angola, Chad, the Democratic Republic of Congo, Nigeria, Pakistan, Kenya and Somalia) and may not accurately reflect the actual current address of the settler.

Table 1: Arrivals in Australia, 2011–12, by selected country of residence and category of traveller

Country of residence	Settler arrival	Long term resident return	Long term visitor arrival	Short term resident return	Short term visitor arrival	Grand total*
Pakistan	1,684	417	4,540	14,734	9,545	30,919
Afghanistan	293	66	152	3,740	331	4,581
Chad	–	–	–	20	199	226
Congo, Democratic Republic of	19	–	–	107	18	148
Nigeria	70	29	266	1,308	1,601	3,274
Angola	–	5	12	475	149	641
Kenya	643	235	775	9,356	2,967	13,976
Somalia	16	25	7	384	120	552
Grand total*	2,730	779	5,756	30,124	14,929	54,318

* Row and column totals may not equal grand totals due to removal of small numbers.

Source: DIBP unpublished overseas arrivals and departures data

Table 2. Settlers in Australia where country of birth was reported as Afghanistan, Pakistan, Nigeria, Kenya, Somalia, Chad, Angola, or the Democratic Republic of Congo, 2011–12,[8] by Statistical Division and financial year of arrival

Statistical Division	Permanent arrivals 2011–2012	Proportion of total arrivals
Adelaide	966	10
Canberra	148	2
Darwin	74	1
Greater Hobart	45	0
Melbourne	2,561	28
Perth	1,049	11
Sydney	1,720	19
Brisbane	834	9
All others	1,863	20
Total	9,260	100

Settlement statistics were cross referenced against Local Government Areas (LGA) with low vaccination coverage. Data on LGAs with low coverage were provided by the Australian Government Department of Health and sourced from unpublished ACIR data. An LGA with low coverage is defined as one in which the proportion of children assessed between April 2011 and March 2012 as being fully vaccinated according to ACIR guidelines was less than 85%. LGAs are only included in this assessment where there are 25 or more children. LGAs are included if coverage was assessed as less than 85% at any of the 3 age points (1, 2 or 5 years).

Unpublished acute flaccid paralysis (AFP) surveillance data were provided by the National Enterovirus Reference Laboratory, which coordinates Australia's polio surveillance program. Information on risk groups and threat assessments were identified through document review and personal communication with key informants from DIBP, the Department of Foreign Affairs and Trade (DFAT) including the former AusAID, and the Department of Defence.

Results

Population immunity (susceptibility assessment)

Vaccination coverage for polio in Australia is generally high. This includes residents of the Torres Strait Islands (TSIs) that are geographically in close proximity to PNG and who have contact with residents of PNG through the movement of peoples for traditional purposes under the Torres Strait Islander Treaty.[9] In 2012, all states and territories including the TSIs had vaccination coverage for all children of greater than 90% (range 91%–94%) at 12 months of age resulting in a national coverage of 92% and coverage of 93% in the TSIs. This coverage had improved by 2 years of age with all states and territories having coverage of 93% or more (range 93%–96%), resulting in a national coverage of 95%, and 97% in the TSIs. Coverage at 2 years of age is particularly important as it reflects the children at this age having received all 3 recommended doses of the primary course of inactivated polio vaccine (IPV). By 5 years of age coverage had declined from the peak at 2 years to a national coverage of 91% with all states and territories and the TSIs being at least 89%. Trends over the last 6 years show no real change in coverage between 2007 and 2012 at 1 year of age but an improved coverage at 5 years of age from 84% in 2007 to 91% in 2012 (unpublished data).

Vaccination coverage varies widely across Australia by geographical area, age group and Indigenous status. In 2011–12, areas of low vaccination coverage (≤ 85%) were identified across the 61 newly established Medicare Locals where the proportion of children aged 1 year who were fully immunised against all assessable vaccines, including IPV, ranged from 85% to 94%. Those fully immunised at 2 years ranged from 89% to 96% and those fully immunised at 5 years ranged from 84% to 95%. This pattern of wide ranging coverage is reflected amongst Aboriginal and Torres Strait Islander children who, while exceeding or equalling the highest range of coverage for all children, had a substantially lower bottom range at all 3 age points. Coverage amongst this group is highest and closest to that for all children at the 2 year age point.[6]

Coverage can be further broken down for all children by the smaller local Statistical Area Level 3, of which there are 333 in Australia. In 2011–12 there were 32 statistical areas where the percentage of children fully immunised was 85% or lower for at least 1 age group representing a total of 76,769 children who were not fully immunised.[6]

A national serosurvey of poliovirus immunity in Australia, conducted between 1996 and 1999, indicated that herd immunity was likely to be sufficient to prevent generalised outbreaks due to type 1 and type 2 poliovirus at 82% and 88% respectively. However, this may not be the case for type 3 for which immunity was measured at 74%.[7] There was considerable variation by age, with peak immunity amongst children aged 2–4 years.[7] Results of a second serosurvey of polio immunity in Australia, conducted in 2012–13, are expected in 2014.

Of the 211 eligible non-polio AFP classified between 2008 and 2011, 91% (193) were vaccinated against polio at least once (noting that the AFP questionnaires provide the last date of vaccination and not the complete vaccination history) (unpublished data).

Program delivery assessment

Australia has a modern, sophisticated health care system with access to quality laboratory diagnosis. The Australian Government funds a National Immunisation Program, which includes 3 primary doses and 1 booster dose of inactivated polio vaccine, which is free to all Australian children. Australia has a polio importation plan that was endorsed by the Australian Health Protection Principal Committee in December 2008. The plan is currently under review.

The vast majority of Australians have access to safe water and adequate sewerage systems. To prevent disease, a community requires a clean, adequate and reliable supply of water, a functional sewerage system and reliable electricity supply. The majority of Australians live in urban or regional areas with access to these essential services.[10,11] While the performance of essential service providers varies across Australia, cities and large towns generally monitor the quality of drinking water and have reticulated sewerage systems where waste is collected and treated at central treatment plants. In rural and remote areas there is increased reliance on local or individual household systems such as generators, septic tanks and drinking water sourced from bores and rainwater tanks.[10]

Crowded living conditions and lack of maintenance can impact on the quality of water and system effectiveness.[10,11] These conditions are known to facilitate the spread of many infectious diseases, including faecal-oral infections.[12] Exposure to crowded living conditions, which places pressure on household infrastructure such as septic tanks, sewerage pipes and washing facilities, disproportionately affects Aboriginal and Torres Strait Islander people, with rates of 'overcrowding', as defined by the Productivity Commission, amongst this population almost 5 times more common than for non-Indigenous people.[10,11]

Importation threats

In 2012, there were 3 remaining polio endemic countries in which polio transmission has never been interrupted: Afghanistan, Pakistan and Nigeria. There were 3 countries in which transmission was considered to have been re-established: Angola, Chad and the Democratic Republic of the Congo (DRC). In 2013, cases were reported in the 3 endemic countries and in Somalia and Kenya.[13] Angola, Chad and the DRC have now been poliovirus free for 12 months.

The likelihood of Australian residents travelling to the current polio endemic or re-infected countries for tourism is low given that most of these last reserves of polio are in areas of conflict and political unrest, or are uncommon destinations for Australian travellers. Advice is available on the Department of Foreign Affairs and Trade Smartraveller web site encouraging all travellers to these countries to be up to date with polio immunisation.[14]

Australian Government agencies such DFAT have internal health policies as part of their overseas conditions of service that recommend all employees and their dependents have country specific vaccinations, including polio vaccination, prior to travel on posting (personal communication Dr Kerry McMekin, DFAT). Australian Defence Force personnel are provided with all routine immunisations and undergo a targeted review of country specific immunisation requirements prior to deployment overseas. Personnel deploying to a polio endemic area are given a booster dose of polio vaccine if the last dose was more than 10 years prior, in accordance with the *Australian Immunisation Handbook*[15] and the Australian Defence Force Publication 1.2.2.1 Immunisation Procedures (email correspondence, Director Military Medicine, Department of Defence).

The following groups of people arriving in Australia from endemic or re-infected countries could potentially be a source of importation:

- Australian residents returning from visiting a polio infected country;
- Australian defence force personnel returning to Australia after active service;
- government and non-government employees returning to Australia;
- migrants;
- asylum seekers including irregular maritime arrivals;
- international students; and
- short term visitors.

In the 2011–12 financial year there were approximately 54,000 arrivals from countries with endemic or re-established polio transmission or recent outbreaks, including from Pakistan (30,919), Afghanistan (4,581), Nigeria (3,274), Angola (642), Chad (226), the DRC (149), Kenya (13,976) and Somalia (552). These numbers include international students, migrants and offshore humanitar-

ian entrants from these countries in the settler, long and short term visitor arrival statistics and returning Australian residents who nominated these countries as the place where they spent the majority of their time while overseas (Table 1). There are also a number of people who enter the country as IMAs. Of the above countries information on arrivals is publicly available only for Afghanistan and Pakistan. IMAs from Afghanistan represented 42% (7,672) of all IMAs from 2009 to 2012 and less than 5% (818) of IMAs were from Pakistan.[16]

Definitive information on where migrants settle once arriving in Australia is difficult to obtain. Available data indicate that the majority settle in the most populous states, with Perth, Sydney and Melbourne having the highest proportion of overseas-born residents.[17] This also appears to be true for settlers who nominated one of the 8 endemic or polio infected countries listed above as their country of birth. Data from the DIBP indicate that of those settlers arriving in the 2011–12 financial year, 80% (7,397 people) had a last known address in one of the capital cities, with Melbourne and Sydney having the highest proportion at 28% and 19% respectively (Table 2).[8] Of these arrivals, 13% (n=1,216) provided their last address in a LGA of low vaccination coverage, represented by 31 LGAs with approximately 3,400 un– or under-immunised children (unpublished data).[8]

There is also the potential for escape of a wild poliovirus from the laboratory to the community. While the probability of this occurring is low, the potential consequences of such transmission will become increasingly serious as polio-free countries increase and immunisation decreases or stops. Australia has complied with the requirements of phase one of the *Global action plan for laboratory containment of wild polioviruses*[18] by completing the laboratory survey and inventory phase, and maintaining a national inventory of laboratories holding wild-type poliovirus and potentially polio infectious materials since 2002. The inventory, which initially consisted of 16 laboratories across Australia, in 2012 had decreased to two: the Victorian Infectious Diseases Reference Laboratory and the Therapeutic Goods Administration. It is reviewed annually to ensure that samples have either been destroyed or are appropriately contained at biosafety level two (physical containment level 2). The Australian/New Zealand Standard for Safety in Laboratories, Part 3: Microbiological Safety and Containment (AS/NZS 2243.3:2010)[20] currently requires that 'All laboratories retaining wild poliovirus infectious materials or potential wild poliovirus infectious materials should be listed on the inventory held by the national government', however, there is no mechanism in place to enforce this. A permit from the Australian Quarantine and Inspection Service is required to import human viruses such as poliovirus.

Surveillance assessment

Australia has a well-established surveillance system for poliomyelitis based on the WHO recommended AFP surveillance and supplemented in recent years by sentinel environmental and enterovirus surveillance. A 2012 review of polio surveillance in Australia found that high immunisation coverage, sensitive polio surveillance and an effective polio response plan would ensure that any imported poliomyelitis would rapidly be contained in Australia.[20]

Australia has met the non-polio AFP rate clinical indicator for the last 5 years consecutively (2008–2012) and 9 times since surveillance began in 1995. Australia has never met the virological surveillance indicator for adequate stool specimen collection (5-year mean of 34%).[21]

The Enterovirus Reference Laboratory Network of Australia was established in 2008 primarily as a means of detecting poliovirus amongst untyped enteroviruses from clinical specimens. Further typing targets those from cases with a history of neurological symptoms potentially related to a poliovirus infection such as meningitis. Sentinel environmental surveillance was established in 2010 at 3 sites in New South Wales targeted as areas with low vaccination coverage, high volumes of tourism and a large proportion of international students. These additional virological surveillance systems have not detected any wild polioviruses, providing additional evidence that there is no wild poliovirus circulating undetected in Australia.[21]

Discussion

Vaccination coverage for polio is high in Australian children. Discrepancies between coverage for Aboriginal and Torres Strait Islander children and coverage for all children are minimised by 2 years of age. There are no major differences in polio vaccine coverage or immunity by state or territory and vaccination coverage in the TSIs is higher than the national overall coverage at all age points. The first national serosurvey for polio indicated herd immunity sufficient to prevent outbreaks of type 1 and type 2 poliovirus.

There are areas of low coverage identified amongst the general population. Improving vaccination coverage in areas of low coverage is the subject of an agreement between states and territories and the Australian Government. Most of these low coverage communities are in areas with high

quality water and sanitation services. The risk to remote Aboriginal and Torres Strait Islander communities is mitigated by their remoteness and consequent lower likelihood of poliovirus introduction through international travel or visitors from endemic regions, and generally high vaccination coverage.

Despite inadequate stool specimen collection rates, surveillance quality is high and there is reasonable confidence that polio surveillance in combination with the health care system would identify polio cases should they occur.[20] The importance of collecting stool specimens as part of the routine clinical work up in the differential diagnosis of acute neurological presentations in which AFP is evident cannot be underestimated.

The potential for importation of a wild poliovirus exists in Australia with a large number of arrivals from polio endemic or re-infected countries. The majority of new permanent arrivals from these countries settle in the major capital cities, especially Sydney and Melbourne.

In 2011–12, 13% of these settlers had a last known address in a LGA known to have low vaccination coverage.

Conclusion

While the overall risk of an imported wild poliovirus leading to transmission within Australia is assessed as being low, the consequences of such an event are very serious. Every possible effort should be made to mitigate against the risk of polio transmission following possible importation of poliovirus into Australia. Geographical areas, which have a relatively higher risk through lower vaccination coverage and relatively high volume of arrivals from polio endemic countries, have been identified. Ensuring the AFP surveillance system is sensitive enough to detect an imported wild poliovirus to allow timely follow-up of all cases in which poliovirus infection is suspected, supplemented by environmental and enterovirus surveillance, is essential. High vaccination coverage, not just nationally, but at all sub-national levels, must be maintained and identification of immunity gaps reviewed using the latest population-wide serosurvey results. Consideration should also be given to ways in which new arrivals from high risk countries may be immunised to reduce the risk of introduction and spread in these potentially susceptible communities. Travel advisories need to be continually reviewed and updated with information about countries with active transmission of polio and recommending that all visitors are up-to-date with polio vaccinations prior to travel. Finally, as the global goal of eradication approaches, regular review of Australia's importation and subsequent transmission risk is increasingly important.

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Ross River virus infection surveillance in the Greater Perth Metropolitan area – has there been an increase in cases in the winter months?

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Abstract

An increase in off-season (June to September) Ross River virus (RRV) notifications from the greater Perth metropolitan area was observed from 2006 to 2009. We investigated the increase to determine whether it is likely to have reflected a true increase in off-season cases. A single positive RRV IgM test result is sufficient for RRV notification but where follow-up testing was performed, the positive predictive value of an IgM test where IgG was negative was very low in the off-season and also in the season when using the only commercially available test kit. The increase in off-season notifications was not associated with an increase in off-season testing. Some Perth laboratories use more stringent notification criteria than the nationally agreed RRV case definition, and the geographical distribution of samples tested varies between laboratories. Our findings make a strong case to change the nationally agreed case definition for RRV to not accept a single IgM positive test result as laboratory definitive evidence where the IgG is negative. Our study also identified a range of challenges in interpreting changes in seasonal patterns and geographical distribution of RRV. Any such observed changes should be investigated through further data analysis and/or mosquito trapping and testing in order to assess validity. *Commun Dis Intell* 2014;38(2):E114–E121.

Keywords: Ross River virus, surveillance, notifications, serology

Introduction

Ross River virus (RRV) is a mosquito-borne alphavirus infection that occurs throughout Australia and the South Pacific.[1,2] It causes an acute illness characterised by joint pain, arthritis, fatigue and malaise, often accompanied by fever and a rash. While not life-threatening, RRV can cause significant morbidity, with symptoms lasting up to 6 months.[1–3]

RRV is a nationally notifiable disease. Laboratories and treating doctors are required to report to the Western Australian Department of Health (WA Health), all cases of RRV infection meeting the nationally agreed case definition.[4] In Western Australia, notifications are recorded in the Western Australian Notifiable Infectious Diseases Database (WANIDD). The serological component of the case definition is: RRV-IgG seroconversion or a fourfold rise in RRV-IgG between acute and convalescent samples; detection of RRV IgG and IgM in a single sample; or detection of RRV-IgM without IgG when there is no detectable IgM to Barmah Forest virus (BFV).

From 2006 onwards, all laboratories in Western Australia were legislatively required to directly notify cases. Prior to that date, notifiable diseases were notified by doctors based on clinical and/or laboratory diagnoses, and in the early 2000s by the only public sector testing laboratory (PathWest Laboratory Medicine WA) and some private laboratories.

Local governments and the Environmental Health Directorate of WA Health undertake enhanced surveillance of RRV cases notified by treating doctors, to identify the most likely place of exposure and date of onset of symptoms. This information is recorded in the Mosquito Borne Disease Control (MBDC) database.

In the south-west of Western Australia, RRV typically causes outbreaks of varying sizes between October and May. In 2006 it was observed that the number of RRV notifications reported during the off-season (June to September) were higher than expected in the Perth Metropolitan region and the Peel region immediately south of Perth (Figure 1).

In Western Australia, RRV notifications are used to identify areas of high RRV activity to enable additional mosquito control activity and public warnings. Notification data are complemented by mosquito surveillance in areas of historically high activity over summer. A change in the distribution of RRV cases to include the off-season in the south-west is potentially very important as it could flag changes in the ecology of vector mosquitoes and animal hosts. This has implications for human health and for risk mitigation activities such as mosquito control.

This study examined the notification data and laboratory testing data, assessing whether the notifications reflected a real increase in off-season RRV cases or an artefact of testing and/or notification practices.

Methods

This was a descriptive study.

Notification rates

RRV notifications for the Perth Metropolitan and Peel regions by laboratory, month and year of onset from 1 January 1990 to 30 June 2012 were retrieved from WANIDD. Population data at Statistical Local Area level were obtained from the Australian Bureau of Statistics and aggregated into MBDC regions. Notification rates per 100,000 population were calculated by dividing the number of notifications in that time period by the estimated population for that year and multiplying by 100,000, and were annualised by multiplying the rate by 12 divided by the number of months.

Enhanced surveillance data

Notification data by month and year of onset and region of acquisition as documented by enhanced surveillance were retrieved from the MBDC database for the years 2006 to 2009 inclusive. The enhanced surveillance data were compared with the notification data to assess whether having further information about the onset date and likely place of acquisition of infection changed the proportion of off-season notifications in the Perth Metropolitan and Peel regions.

Laboratory testing

Laboratory testing data for the Perth Metropolitan and Peel regions for the period from 1 January 2002 to 31 August 2012 were obtained from PathWest Laboratory Medicine WA (PathWest) and St John of God Pathology (SJGP). PathWest is the state reference laboratory. It uses an in-house immunofluorescence assay (IFA) for the detection of IgM antibodies,[5] and an in-house haemagglutination inhibition (HI) test.[6] The latter detects both IgG and IgM together and is less sensitive than the IFA for detection of IgM.[6] The in-house assays have been validated according to guidelines published by the National Pathology Accreditation Advisory Council, and have been approved for diagnostic use by the National Association of Testing Authorities. The sensitivity and specificity of these tests is not known. Both tests are routinely performed on samples referred for RRV diagnosis. All but one laboratory (which refers their samples to PathWest), including SJGP, use a commercial enzyme immunoassay (EIA); Panbio® RRV IgM ELISA and Panbio® RRV IgG ELISA (Alere, Sinnamon Park, Queensland Australia). The sensitivity and specificity of these tests was estimated in South Australia by testing samples taken in 1996 and 1997 using an HI test as the comparator. The sensitivity of the PanBio ELISA kits was estimated to be 98.5% and 84.6% and the specificity 96.5% and 97.6% for IgM and IgG respectively.[7] These are the only commercial tests currently available in Australia. In response to a doctor's request for RRV serology, all private laboratories test separately for IgM and IgG antibodies.

PathWest routinely requests second samples where the sample is IgM positive within 2 weeks of onset of illness independent of the HI test result. This allows detection of seroconversion or a significant rise in IgG using the HI tests to confirm acute infection. SJGP requests second samples where the initial IgM is positive but the IgG is negative in order to test for seroconversion. The EIA tests do not quantify results and cannot be routinely used to detect rises in IgG, but will detect seroconversion.

A more detailed analysis of positive IgM results was carried out for patients from the Perth Metropolitan and Peel regions. Patients who had results for more than 1 sample were identified and the results were used to assess the interpretation of the first IgM positive sample for each patient. (Table 1).

Notification practices of the laboratories

The three private laboratories (SJGP and Laboratory B and C) and PathWest who, combined, notify the majority of RRV cases, were contacted to ask about their notification practices for RRV.

Table 1: Classification of Ross River virus IgM positive test results following follow-up testing

Classification	PathWest	Private
True positive	Seroconversion from negative IgG (HI <40) to positive OR HI ≥40 and a fourfold or greater rise in HI titre between acute and convalescent samples OR HI ≥40 on first and second samples	IgM with IgG seroconversion OR IgM and IgG positive on initial and a later sample
False positive	HI ≤40 on second sample seven or more days after the first test OR IgM becomes negative within 6 months of the first test OR patient is known to have past Ross River virus infection	IgG negative on repeat testing seven or more days after the first positive IgM test OR IgM becomes negative within 6 months of the first test

The positive predictive value of the test was calculated by the formula: true positive/(true positive + false positive).

Data analysis

Data were analysed for Perth Metropolitan and Peel regions. Comparisons between means were performed using Independent t tests. Analysis was undertaken using Microsoft Excel and SPSS version 21 software.

Results

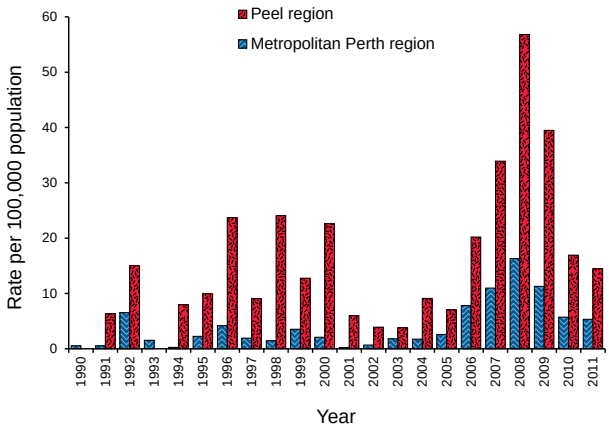
Notification rates

There were 6,337 RRV notifications from Perth Metropolitan and Peel regions from 1 January 1990 to 30 June 2012. The population in the Perth Metropolitan region in 2012 was 1.6 million people and in the Peel region was 236,000. The notification rates in the off-season in Perth Metropolitan and Peel regions were higher in the years between 2006 and 2009 compared to the other years (mean of 11.6 vs 2.4 for the Perth Metropolitan region; 37.6 vs 10.6 for the Peel region ($P<0.05$)), but there was no significant difference in the notification rates during the season for the 2006 to 2009 period compared with the other years (mean of 23.4 vs 18.3 for the Perth Metropolitan region; 88.7 vs 59.6 for the Peel region ($P>0.05$)) (Figure 1 and Figure 2).

Enhanced surveillance

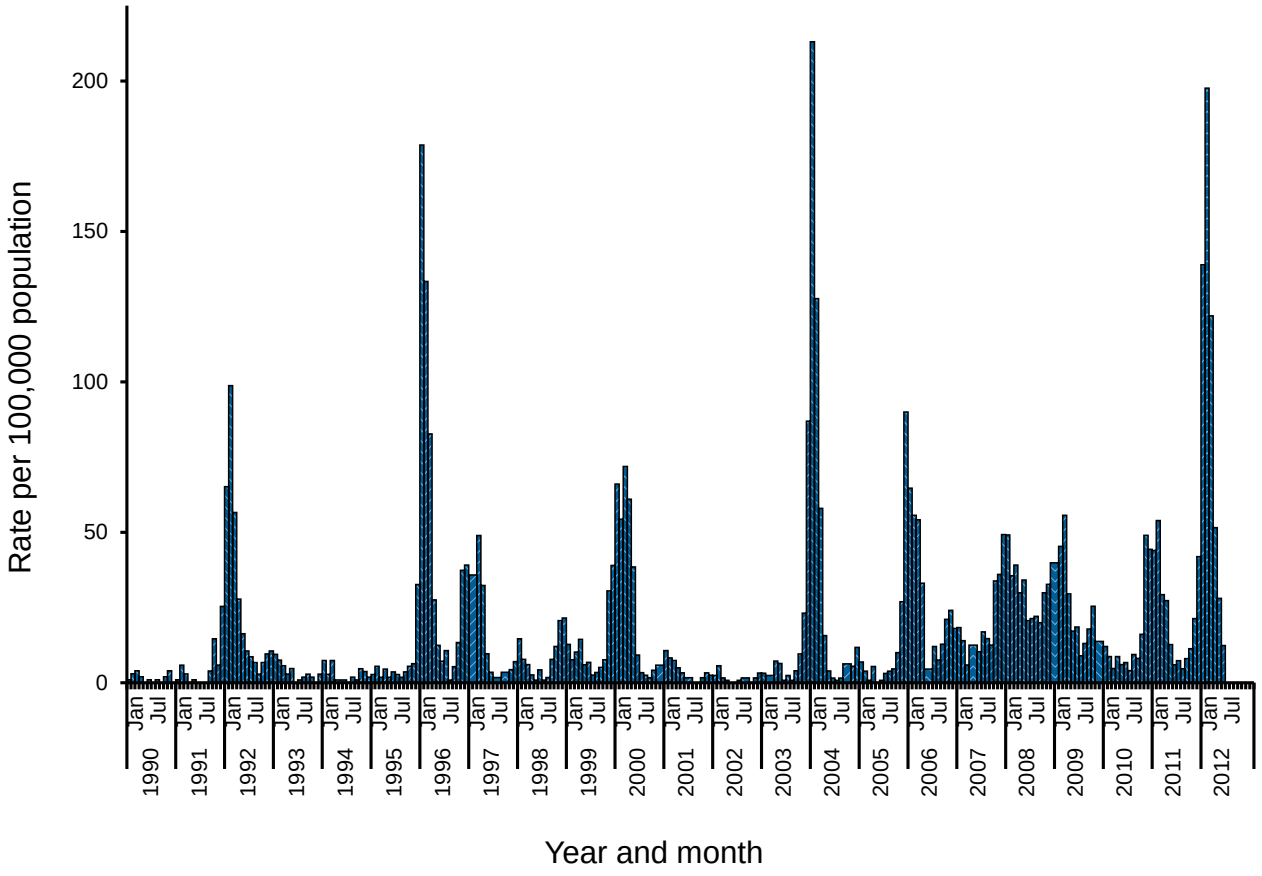
From 2006 to 2009 inclusive, 322 cases of RRV with onset in the off-season in the two regions, were notified on WANIDD. Of these, 67 (20.8%) had enhanced surveillance from which it could be ascertained that 44/67 cases (65.7%) occurred

Figure 2: Notification rates for off-season Ross River virus infections,* Perth Metropolitan and Peel regions, by onset year



* Ross River virus infection notifications occurring in the months of June to September inclusive.

Figure 1: Notification rate Ross River, Perth Metropolitan and Peel regions, by month and year



in the Perth Metropolitan or Peel region in June to September, whilst 23 were reclassified as either seasonal exposure (10), non-Perth Metropolitan/Peel exposure (9) or both (4). Of the 1,416 RRV cases that were notified on WANIDD as seasonal in the two regions from 2006–2009, 455 (32%) had enhanced surveillance. Seventeen (3.7%) of these were reclassified as off-season using enhanced surveillance data; and 5 cases originally classified as non-metropolitan were determined by enhanced surveillance to be in Perth Metropolitan or Peel.

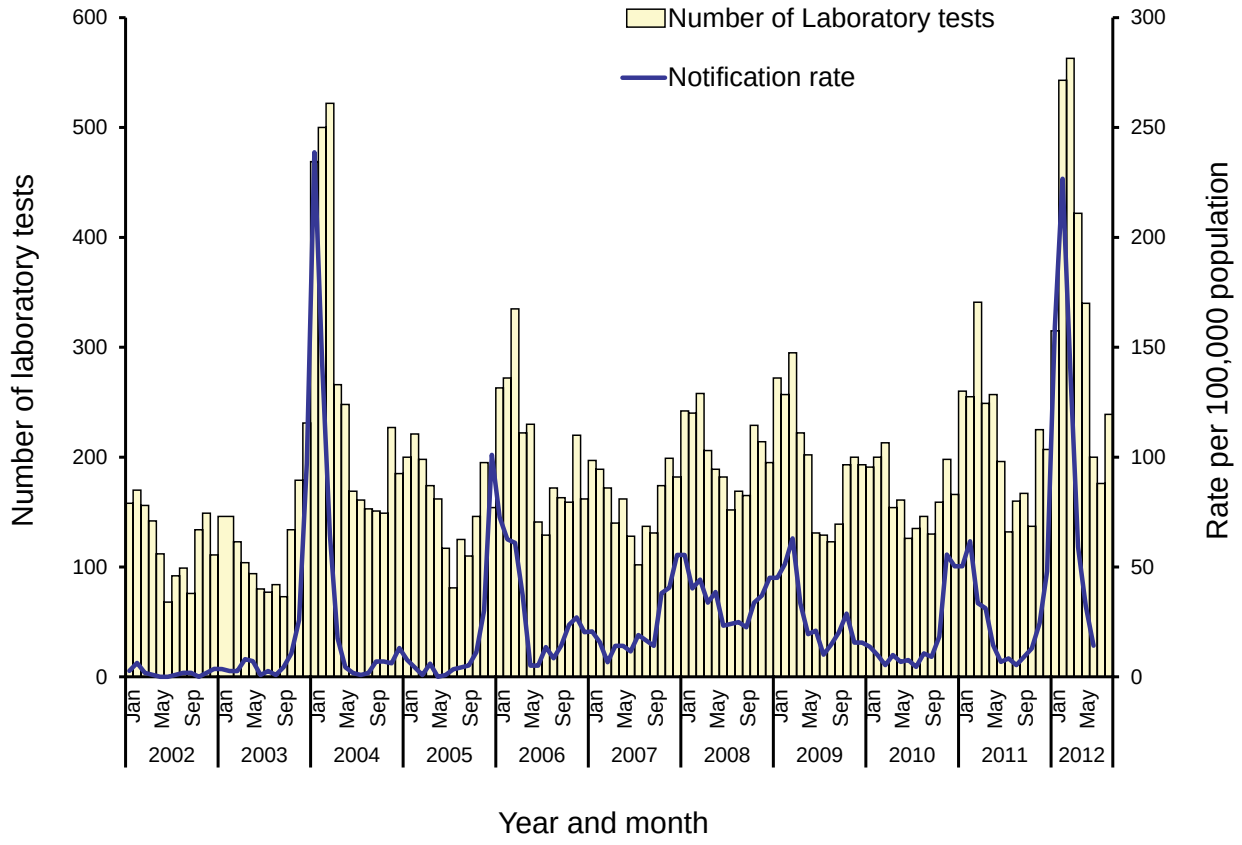
Laboratory testing

Between 1 January 2002 and 30 June 2012, PathWest notified 7.6% of all RRV notifications in the Perth Metropolitan and 14.2% from the Peel region, while 19.7% and 4.3% respectively came from SJGP. While peak periods of testing occurred during the season, testing continued at high numbers during the off-season (Figure 3). A similar pattern of testing occurred for Peel (data not shown). The mean number of off-season RRV tests did not differ significantly between the years of higher off-season notifications (2006–2009) compared with the remainder (573.3 per year vs 484.6 per year, $P=0.25$). Off-season testing increased from 2004 onwards compared with 2002 and 2003 (mean 569.0 per year vs 324.5 per year, $P<0.001$).

In the two regions of interest, the proportion of IgM tests that were positive showed some seasonal variation with peaks in the summer months, while a higher proportion of tests were positive in the off-season from 2007 to 2009, particularly from SJGP (Figure 4). During the off-seasons from 2002 to 2011, the proportion of positive tests from SJGP ranged from 0.4% in 2002 to 15% in 2009. For PathWest the proportion of positive test results ranged from 0% in 2003 to 6% in 2007.

Between 1 January 2002 and 31 August 2012, 8,428 RRV IFA IgM tests were performed at PathWest from the two regions; 1,979 during the off-season and 6,449 during the season. Of these, 142 (7.2%) were positive during the off-season and 1,044 (16.1%) during the season. Sixty-six patients tested during the off-season and 563 tested during the season had more than 1 sample at intervals suitable for patient classification and IgM assessment (Table 2).

Figure 3: Total number of IgM tests performed by PathWest and St John of God Pathology and notification rate for Ross River virus infection, Metropolitan Perth, January 2002 to June 2012, by month and year of specimen collection date



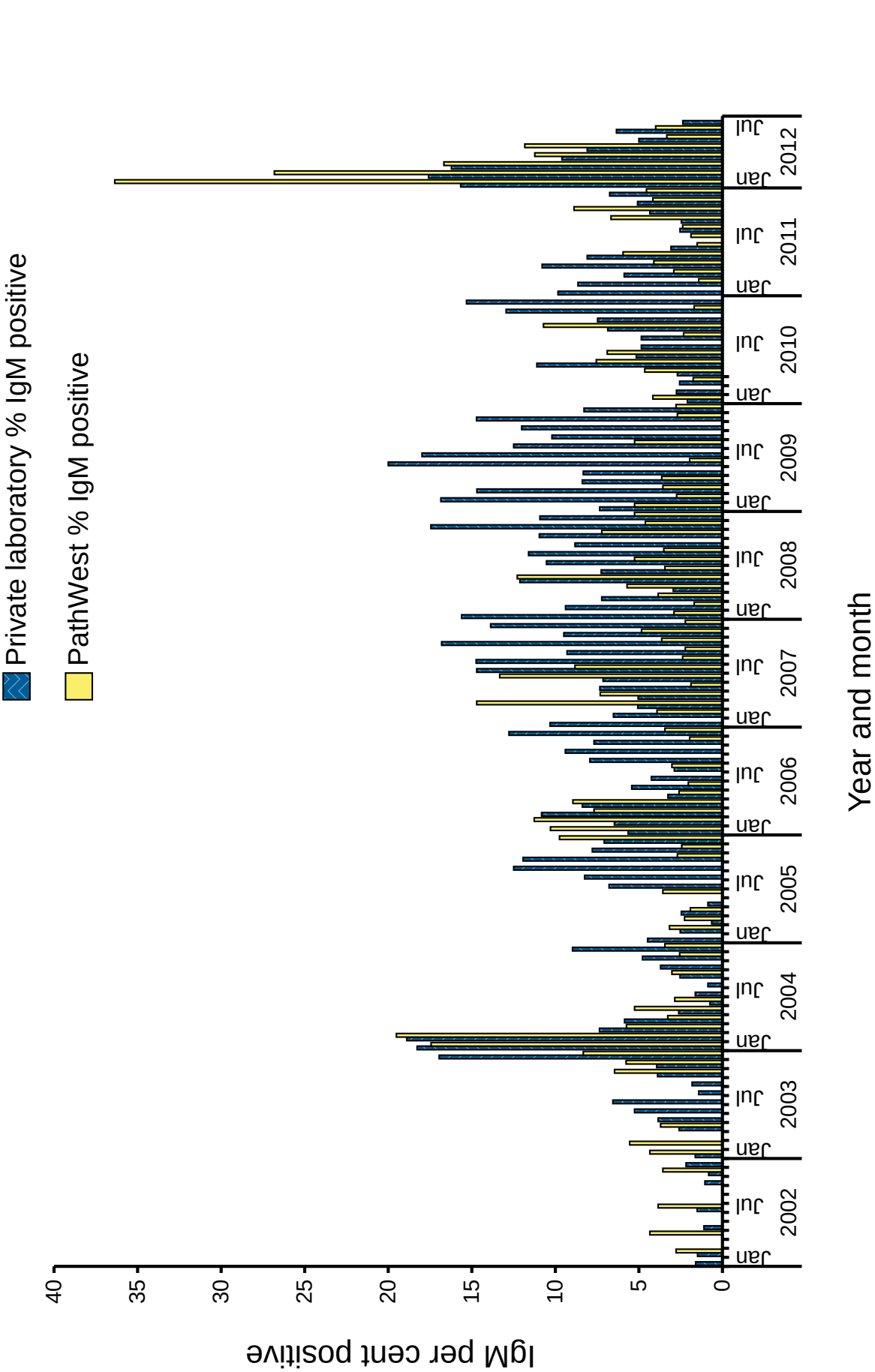


Figure 4: Per cent of Ross River virus IgM tests that were positive, Perth Metropolitan region, January 2002 to June 2012, by month, year of specimen collection date and laboratory

During the off-season the positive predictive value (PPV) for an IgM positive, HI<40 result was 39.3%, and for an IgM positive, HI≥40 result the PPV was 84.2%. During the season the PPV for IgM positive, HI<40 result was 81.1%, and for an IgM positive, HI≥40 result the PPV was 92.7%.

During the off-season, 25 (17.4%) of the 142 PathWest RRV test results meeting the case definition for notification were IgM positive, HI<40. During the season, 177 (17.0 %) of the 1044 PathWest RRV test results meeting the case definition for notification were IgM positive, HI<40.

Between 1 January 2002 and 31 of August 2012, 19,177 RRV EIA IgM tests were performed at SGJP for the two regions; 4,569 during the off-season and 14,608 during the season. Of these, 197 (4.3%) were positive during the off-season and 1,062 (7.3%) during the season. Eighty-one patients tested during the off-season and 366 tested during the season had more than 1 sample at intervals suitable for patient classification and IgM assessment (Table 3).

During the off-season the PPV for an IgM positive, IgG negative result was 4.0%, and 60.0% for an IgM positive, IgG positive result. During the season the PPV for IgM positive, IgG negative result was 24.6%, and 60.7% for an IgM positive, IgG positive result the PPV.

During the off-season, 150 (76.1%) of the 197 SJGP RRV test results meeting the case definition for notification were IgM positive, IgG negative. During the season, 689 (64.9%) of the 1,062 SJGP test results meeting the case definition for notification were IgM positive, IgG negative.

Of the patients from PathWest classified as having genuine IgM, 54.4% had serological evidence of acute infection (either seroconversion or rising HI titres), the remaining having stable HI titres.

Notification practices of the laboratories

SJGP and PathWest contributed data to the study while the others did not, but all pathology laboratories doing their own RRV testing provided information about their notification practices.

PathWest notifies all IgM positive test results. For the period 2006 to 2009, PathWest notified 16.6% of notifications from Perth Metropolitan and Peel regions in the off-season and 30.3% of the notifications for the remaining months.

All private laboratories in Western Australia use the commercial EIA assay, but notification practices varied.

SJGP does not always notify IgM positive test results amongst individuals with known autoimmune disease or viral infections known to cause false positive test results (around one-third of positive IgM results). The approach was variously applied over time. For the period 2006 to 2009, SJGP notified 18.2% of the notifications from the Perth Metropolitan and Peel regions in the off-season and 14.5% of the notifications for the remaining months.

Table 2. Classification of positive IgM tests using immunofluorescence assay and haemagglutination inhibition tests where follow-up testing was performed, 1 January 2002 to 31 August 2012

	Season		Off-season	
Initial test result	IgM positive, HI<40	IgM positive, HI≥40	IgM positive, HI<40	IgM positive, HI≥40
True positive	202	291	11	32
False positive	47	23	17	6
Total	249	314	28	38

Table 3: Classification of positive IgM enzyme immunoassay tests where follow-up testing was performed, 2002 to 31 August 2012

	Season		Off-season	
Initial test result	IgM positive, IgG negative	IgM positive, IgG positive	IgM positive, IgG negative	IgM positive, IgG negative
True positive	82	17	3	3
False positive	252	11	71	2
Total	334	28	74	5

Private laboratory B notifies positive IgM test results only when there is a positive IgG test result either at the time of the initial IgM test or upon seroconversion. For the period 2006 to 2009, this laboratory notified 10.1% of notifications from the Perth Metropolitan and Peel regions in the off-season and 11.2% of notifications for the remaining months.

Private laboratory C notifies all positive IgM test results. For the period 2006 to 2009, this laboratory notified 40.8% of notifications from the Perth Metropolitan and Peel regions in the off-season and 36% of notifications for the remaining months.

Discussion

While we were unable to explain the increase in off-season notifications during 2006 to 2009, we identified a number of challenges in interpreting RRV notification data, particularly in the off-season.

We found that where follow-up tests were performed on patients with their first positive IgM during the off-season, the PPV of an IgM positive test in the absence of IgG was very low regardless of the IgM test used. During the season, the PPV value for the IFA alone rose to 81.1%, while for the EIA it remained low.

Patients who had follow-up serology within the acceptable timeframe may not represent all patients undergoing testing. Furthermore, some patients may have been incorrectly classified, such as those with delayed seroconversion. Despite these limitations, it is clear that detection of IgM in the absence of IgG using the commercial EIA test should be interpreted with caution as there is a high chance that it is a false positive. Similarly the IFA-IgM test alone cannot be reliably used to indicate acute infection during the off-season.

If the HI titre on the initial test is ≥40 the PPV for the IFA/HI is over 80% regardless of the season. For the commercial EIA the PPV of an IgM and IgG positive test was around 60% regardless of the season, which may be acceptable for surveillance purposes, but should be interpreted cautiously for patient diagnosis.

Our findings are consistent with a study that found that 45% of patients with RRV IgM but not IgG failed to seroconvert on follow-up testing.[8] A similar problem has been described with the EIA for IgM to the closely related BFV from the same manufacturer.[9]

Based on these findings, there is a very strong case to remove from the RRV case definition the possibility of laboratory confirmation for RRV IgM positive/ IgG negative test results. This would substantially reduce the number of notifications from the private laboratories; at least for those who notify according to the agreed case definition; but very few genuine positives will be lost. Removing the IFA-IgM only positives detected during the season would remove up to 14% of the genuine cases from PathWest.

RRV IgM antibodies usually appear within a few days of illness onset, so the presence of IgM antibody in sera is considered to be indicative of acute or recent infection.[6] However, in most cases of RRV infection, IgM antibodies remain in sera for 1 year at least.[6,10] Therefore even if IgM is present it may not mean infection or reinfection occurred in the season of sampling, unless it is accompanied by seroconversion or a rise in IgG titre. The latter cannot be easily determined using the EIA tests. Patients with RRV may not present in the acute phase, but rather some time later when their symptoms persist, which may be during the off-season. If the date of symptom onset is not available, as in the majority of cases, the specimen date will be interpreted as the onset date and the cases misclassified as off-season cases.

We also identified variations in reporting practice for single IgM positive results between laboratories and at different times. Some laboratories don't notify these cases, despite them meeting the RRV case definition. Therefore the PPV of notifications from different laboratories differs. This is not problematic if there is consistency in notification practices over time, and if there is no geographical variation in coverage by the different laboratories. However, there are differences in the regional coverage of the laboratories, making the comparison of notification rates between different regions difficult.

Testing data from the two laboratories did not reveal increased RRV testing in the off-season in the years 2006 to 2009. The higher rate of positive results during this time may reflect an actual increase in RRV incidence, but this would usually be accompanied by increased testing. From these data, there is no clear explanation for the increase in off-season notifications in those years.

RRV symptoms can last for up to 6 months.[6] Therefore people with persisting symptoms may be tested after the acute phase. Without enhanced surveillance, a notification of a positive RRV test could be wrongly classified as occurring in the off-season as pathology laboratories do not routinely provide the date of symptom onset. Where enhanced surveillance was undertaken, there were notifications that were thus misclassified in both

the off-season and during the season. This is likely to be due to the long duration of symptoms and the fact that laboratory notifications report the residential address of the case rather than their place of acquisition. Based on our data, if enhanced surveillance was uniformly performed for off-season and seasonal RRV notifications, more RRV notifications in the off-season would be reclassified as seasonal than vice versa.

The change in legislation in Western Australia requiring laboratory notification for notifiable diseases that was introduced in 2006 may explain the observed increase in winter cases from 2006 to 2009. However, as this increase did not continue between 2010 and 2012 it is difficult to make this interpretation until a few more years of notification data become available.

We identified a number of challenges in interpreting RRV notification data. Much of this appears to be due to the low PPV of the RRV IgM test that is used as the basis for most notifications. At peak risk times and in higher risk geographical areas, the PPV of the IgM test results is likely to be sufficiently high to enable identification of the timing and distribution of RRV activity, particularly if the IgM is accompanied by RRV-IgG. Furthermore, the long-term persistence of IgM is likely to be contributing to the over-notification of apparent off-season cases where patients were actually infected during the preceding season. Therefore only large changes in disease notification rates are likely to be able to be interpreted reliably based in IgM results alone.

Finally, enhanced surveillance should remain an important tool for the accurate understanding of RRV epidemiology, particularly when notification data suggests a change in the distribution of RRV, either in time or in place, or when there are unexpected increases in notifications.

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Annual report

Arboviral diseases and malaria in Australia, 2011–12: Annual report of the National Arbovirus and Malaria Advisory Committee

Katrina E Knope, Stephen L Doggett, Nina Kurucz, Rebecca Feldman, Cheryl A Johansen, Jay Nicholson, Angus Sly, Michaela Hobby, Debra El Saadi, Mike Muller, Cassie C Jansen, Odwell M Muzari and the National Arbovirus and Malaria Advisory Committee

Abstract

The National Notifiable Diseases Surveillance System received notifications for 7,875 cases of disease transmitted by mosquitoes during the 2011–12 season (1 July 2011 to 30 June 2012). The alphaviruses Barmah Forest virus and Ross River virus accounted for 6,036 (77%) of these. There were 18 notifications of dengue virus infection acquired in Australia and 1,390 cases that were acquired overseas, while for 38 cases, the place of acquisition was unknown. Imported cases of dengue in Australia were most frequently acquired in Indonesia. There were 20 imported cases of chikungunya virus. There were no notifications of locally-acquired malaria in Australia during the 2011–12 season. There were 314 notifications of overseas-acquired malaria and 41 notifications where the place of acquisition was unknown. Sentinel chicken, mosquito surveillance, viral detection in mosquitoes and climate modelling are used to provide early warning of arboviral disease activity in Australia. In 2011–12, sentinel chicken programs for the detection of flavivirus activity were conducted in most states with the risk of arboviral transmission. Other surveillance activities to detect the presence of arboviruses in mosquitoes or mosquito saliva or for surveying mosquito abundance included honey-baited trap surveillance, surveys of household containers that may provide suitable habitat for the dengue vector, *Aedes aegypti*, and carbon dioxide baited traps. Surveillance for exotic mosquitoes at the border continues to be a vital part of preventing the spread of mosquito-borne diseases to new areas of Australia. *Commun Dis Intell* 2014;38(2):E122–E142.

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

Introduction

This report describes the epidemiology of mosquito-borne diseases of public health importance in Australia during the season 1 July 2011 to 30 June 2012. It includes 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), the Kunjin strain 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 animal surveillance measures for arboviruses (in particular for MVEV) conducted by states and territories, and also at the border 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 (AHPPC) through the Communicable Diseases Network of Australia (CDNA). Members of NAMAC have expertise in virus and disease surveillance, epidemiology, virology, vector ecology, vector 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, laboratory support, development of national guidelines and response plans and research priorities. NAMAC assists in the detection, management and control of real or potential outbreaks of arboviruses or malaria and provides advice on the risk of these diseases or exotic vectors being imported from overseas. NAMAC members participate in 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 including several arboviruses and malaria. The *National Health Security Act 2007* (NHS Act 2007) provides the legislative basis for the national noti-

fication 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 data extracted from NNDSS during December 2013 and analysed by date of diagnosis. This derived field 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 received date. The data are from a 'snap-shot', thus numbers in this report may vary slightly from those reported elsewhere. Data in the snap-shot were confirmed with state and territory public health surveillance managers. Detailed notes on the interpretation of NNDSS are available in the 2011 NNDSS annual report.[1] 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). The report includes information on the following pathogens transmitted by mosquitoes, all which are nationally notifiable except CHIKV:

- alphaviruses (BFV, RRV, and CHIKV);
- flaviviruses (DENV, JEV, KUNV, MVEV and YFV);
- arboviruses not elsewhere classified (NEC); and
- malaria.

Whilst CHIKV infection is not currently nationally notifiable, a national case definition was implemented from 2010, and NNDSS allows the collection of notifications of CHIKV infection as a separate infection. Prior to this, CHIKV infections were notified under the disease category arbovirus NEC. All notifications of CHIKV infection under arbovirus NEC were counted under CHIKV infection instead of under arbovirus NEC.

Crude notification rates or counts for the 2011–12 season were compared with rates or counts for that disease 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 for these diseases. Rates are not provided for rare diseases ($n<20$ in the 2011–12 season), because these rates tend to have very large standard errors and therefore cannot be meaningfully compared across time or geographical location.

Notification rates were calculated using the Australian Bureau of Statistics estimated resident populations for Australia and each state or territory at June 2012.[2] 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 (2012 population applied to the 2011–12 financial year).

Due to a limitation of surveillance systems, Queensland notifies mixed infections of malaria as a separate notification for each infecting organism. For the 2011–12 season, additional information was collected to enable these mixed infections to be reported as 1 case for the purpose of this report. In 2011–12, this resulted in 2 fewer notifications than if the adjustment was not made.

Additional information on the details of some notifications was obtained from state and territory public health surveillance managers. Data on sentinel animals and mosquito surveillance, control measures and detections of exotic mosquito vectors at the border were supplied by relevant agencies.

Vertebrate, vector and climate surveillance in states and territories

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 (SOI), rainfall and temperature obtained from the Bureau of Meteorology (BOM) 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 animals 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.

Northern Territory

Surveillance consists of routine year round sentinel chicken surveillance with monthly bleeds and *ad hoc* virus isolation from mosquitoes when MVEV

or KUNV cases are reported. The program is combined and coordinated by the Northern Territory Department of Primary Industries and Fisheries (DPIF) and the Northern Territory Department of Health, with support from volunteers. The Northern Territory Mosquito Borne Disease Control Program assists regional authorities with mosquito monitoring and provides some funding for direct mosquito control in some major towns. In 2011–12, routine adult mosquito trapping consisted of 21 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, three in Tennant Creek, four in Katherine, three in Alyangula on Groote Eylandt, and six in Alice Springs. Climate information from the BOM is used in conjunction with animal and vector surveillance. Rainfall patterns, daily rainfall records and rain threshold models are used to assist in predicting mosquito and virus activity.

Queensland

Queensland does not currently conduct statewide surveillance for MVEV in vertebrate hosts, and does not maintain sentinel chicken flocks. Queensland commenced an arbovirus surveillance trial using honey trap saliva technology in 2012, with the aim of evaluating its effectiveness as a sustainable method for arbovirus surveillance in Queensland. Mosquito monitoring is performed by some local councils, primarily for salt water and fresh water mosquitoes. Some councils implemented surveillance for container breeding mosquitoes in domestic and commercial premises as part of a joint Queensland Health and local government initiative. Opportunistic virus isolations from mosquitoes or animals have been carried out by the University of Queensland, the Tropical Public Health Unit network within Queensland Health and the Queensland Institute of Medical Research.

South Australia

Across South Australia, mosquito surveillance and control activities are conducted in partnership between South Australia Health, the University of South Australia, local government and Biosecurity South Australia. The program is coordinated by the South Australia Health and consists of mosquito trapping in the Riverland and areas in the mid-north of the state and virus isolation when required. Seasonal monitoring of mosquito population is undertaken along the Murray River; live collections for virus isolation are sampled in response to high vector numbers and samples are sent to Westmead Hospital for testing.

Tasmania

No state-wide systematic mosquito abundance, virus isolation or sentinel animal surveillance activities are undertaken due to the relatively low risk of arbovirus transmission in the state. However, mosquito collections are undertaken in Sorell Council region, (which includes mosquito breeding areas, is fairly populous, and is close to Hobart) 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, *Aedes camptorhynchus*. These are sent to Westmead Hospital for species identification and viral isolation.

Victoria

The Victorian Department of Health contracts the Victorian Department of Primary Industries to conduct sentinel chicken surveillance during the arbovirus season from November to April. The standard 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, usually from November to April. This program has been in place in Victoria since the 1974 outbreak and acts as an early warning system for possible human infections with flaviviruses. The samples are tested at the Department of Environment and Primary Industries. Flocks are replaced annually. Eight councils undertake mosquito surveillance as part of the standard mosquito monitoring program, with 4 traps placed in each area. Six councils are located along the Murray River, one is a coastal site and the other is within metropolitan Melbourne. The mosquitoes are collected weekly and sent on cold storage to the Department of Environment and Primary Industries for identification, enumeration and virus isolation. Additional mosquito surveillance and identification also occurs in the Geelong area. The Victorian Arbovirus Taskforce examines the risk of outbreaks of MVEV using meteorological surveillance data such as the SOI and rainfall deciles, and Indian Ocean Dipole using respectively the Forbes,[3] and Nicholls[4] and Bennett models.

Western Australia

The University of Western Australia Arbovirus Surveillance and Research Laboratory (ASRL) is funded by 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. Twenty-eight sentinel chicken flocks of up to 12 chickens are located at major towns and communities in the

Kimberley, Pilbara, Gascoyne, Goldfields, Midwest and Central Coastal regions of Western Australia. Blood samples are collected from the chickens at fortnightly intervals during the peak MVEV risk season (December to June). At other times, monthly samples are collected unless prolonged flavivirus activity warrants continued fortnightly sampling. An annual program of mosquito trapping is undertaken towards the end of the wet season when MVEV activity is active over a 3–4 week period. This provides important information on size and species composition of mosquito populations, vector species and virus infection rates.

Results

During the 2011–12 season, there were 7,875 notifications of diseases transmitted by mosquitoes (Table 1). This represented a 2% decrease from the mean of 8,031 notifications for the previous 5 years.

Alphaviruses

In Australia, the most frequently notified viruses in the genus Alphavirus are RRV and BFV. Infection with RRV or BFV can cause illness characterised by fever, rash and polyarthritis. 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).[5] RRV and BFV occur exclusively in the Australasian region.[6]

Local transmission of the alphavirus CHIKV does not occur in Australia, but the infection is regularly reported in travellers returning from overseas. Illness is characterised by an abrupt onset of fever, rash and severe joint pain. The acute disease lasts one to 10 days, but convalescence may include prolonged joint swelling and pain lasting months. Haemorrhagic manifestations may occur occasionally.[7] Humans are amplification hosts for CHIKV, and other vertebrates are not required for high levels of transmission to occur. Internationally, CHIKV is most commonly transmitted by *Ae. aegypti*, which occurs in northern Queensland and *Ae. albopictus,* which is found on Cocos Island, Christmas Island and in some areas of the Torres Strait Islands.[8] Other Australian mosquito species have been shown to be competent vectors of CHIKV in the laboratory.[9]

Ross River virus infections

There were 4,617 notifications of RRV infection during the 2011–12 season, representing a rate of 20.3 per 100,000 population, compared with a 5-year mean of 22.5 per 100,000 (Table 1). Queensland reported the largest number of cases (n=1,788), while the highest rate was in the Northern Territory.

Rates of RRV in the Northern Territory continued to decrease from previous years, and in South Australia and Victoria, where large increases were reported in 2010—11,[10] rates returned to previously reported levels (Figure 1). Rates in Western Australia increased to 63 per 100,000 (n=1,533) from 35.2 per 100,000 in 2010–11 (n=827) and were more than double the 5 year mean of 29.6 per 100,000 (n=661.6).

Figure 1: Notification rate for Ross River virus infection, Australia, July 2006 to June 2013, by year and state or territory

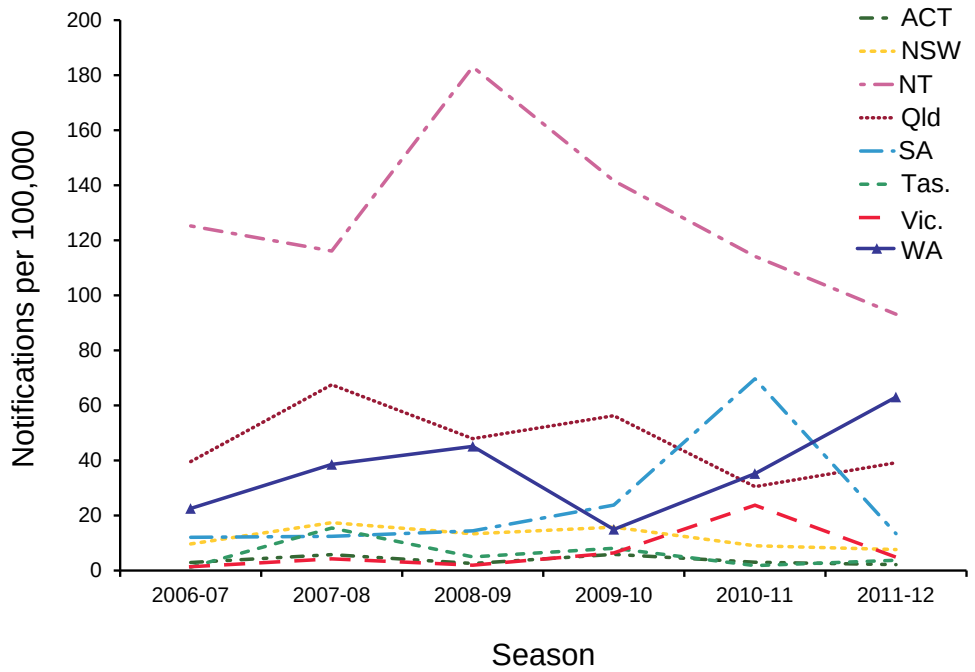


Table 1: Number of notified cases, notification rate* and 5 year mean for mosquito-borne disease, Australia, 2011–12, by disease and state or territory

		ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Arbovirus NEC†	Cases 2011–12	0	0	0	7	1	0	6	0	14
	5 year mean cases	0.0	0.6	0.0	6.6	0.0	0.0	5.8	0.0	13.0
	Rate 2011–12	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Barmah Forest virus infection	Cases 2011–12	1	318	52	806	50	0	39	153	1,419
	5 year mean cases	4.6	450.2	90.0	928.8	67.4	1.6	65.2	131.4	1,739.2
	Rate 2011–12	0.3	4.4	22.1	17.7	3.0	0.0	0.7	6.3	6.2
	5 year mean rate	1.3	6.3	40.2	21.1	4.2	0.3	1.2	5.9	8.0
Chikungunya virus infection	Cases 2011–12	0	4	1	0	2	0	13	0	20
	5 year mean cases	NN	6.8	2.6	2.8	1.0	0.4	7.4	5.4	26.4
	Rate 2011–12	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Dengue virus infection	Cases 2011–12	17	245	69	225	44	9	276	561	1,446
	5 year mean cases	10.2	140.2	26.8	361.8	22.4	3.8	48.6	199.0	812.8
	Rate 2011–12	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Japanese encephalitis virus infection	Cases 2011–12	0	0	0	1	0	0	0	0	1
	5 year mean cases	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.2
	Rate 2011–12	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Kunjin virus infection	Cases 2011–12	0	1	0	0	0	0	0	0	1
	5 year mean cases	0.0	0.0	0.6	0.6	0.0	0.0	0.2	0.0	1.4
	Rate 2011–12	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Malaria	Cases 2011–12	9	71	15	120	5	10	82	43	355
	5 year mean cases	11.6	107.4	24.6	160.6	20.0	9.4	100.4	80.0	514.0
	Rate 2011–12	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Murray Valley encephalitis virus infection	Cases 2011–12	0	1	0	1	0	0	0	0	2
	5 year mean cases	0.0	0.6	0.6	0.2	0.4	0.0	0.0	2.4	4.2
	Rate 2011–12	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Ross River virus infection	Cases 2011–12	8	556	219	1,788	222	19	272	1,533	4,617
	5 year mean cases	14.2	931.2	305.4	2,124.0	433.8	31.8	418.2	661.6	4,920.2
	Rate 2011–12	2.1	7.6	93.1	39.2	13.4	3.7	4.8	63.0	20.3
	5 year mean rate	4.0	13.1	136.4	48.3	26.7	6.3	7.7	29.6	22.5
Yellow fever	Cases 2011–12	0	0	0	0	0	0	0	0	0
	5 year mean cases	0.0	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.4
	Rate 2011–12	–	–	–	–	–	–	–	–	–
	5 year mean rate	–	–	–	–	–	–	–	–	–
Total 2011–12		35	1,197	356	2,948	324	38	688	2,290	7,875

* 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.

NEC Not elsewhere classified.

NN Not notifiable.

RRV was most commonly reported amongst middle-aged adults, with notification rates peaking in the 35–54 year age groups (Figure 2). As in previous years, a little more than half of all cases (54%) were female. The overall rate of RRV in females was 21.9 per 100,000, while in males the rate was 18.7 per 100,000.

As in previous years, there was a marked seasonal trend in RRV notifications, with the largest number notified between January and April (Figure 3). It is important to note that seasonal trends vary between and within states and territories according to differences in mosquito vectors, hosts and climate.

Figure 2: Notification rate for Ross River virus infection, Australia, 2011–12, by age group and sex*

* Age for 1 notification was not available.

Barmah Forest virus infections

There were 1,419 notifications of BFV infections during the 2011–12 season, representing a rate of 6.2 per 100,000 population, a decrease from the mean of 8.0 per 100,000 for the previous 5 years (Table 1). Queensland reported the largest number of notifications of BFV infection (n=806) while the highest rate was reported in the Northern Territory (22.1 per 100,000 population). Rates in 2011–12 were similar to or below the 5-year mean for all states and territories (Figure 4).

BFV notifications were most commonly reported amongst middle aged adults, with notification rates peaking in the 40–64 year age range (Figure 5). Similar to previous years, 52% of cases were male.

Figure 3: Notifications of Ross River virus infection, July 2007 to June 2012, by month

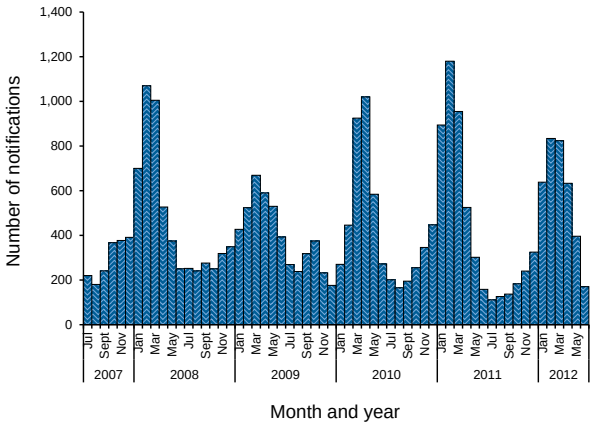


Figure 4: Notification rate for Barmah Forest virus infection, Australia, July 2006 to June 2012, by year and state or territory

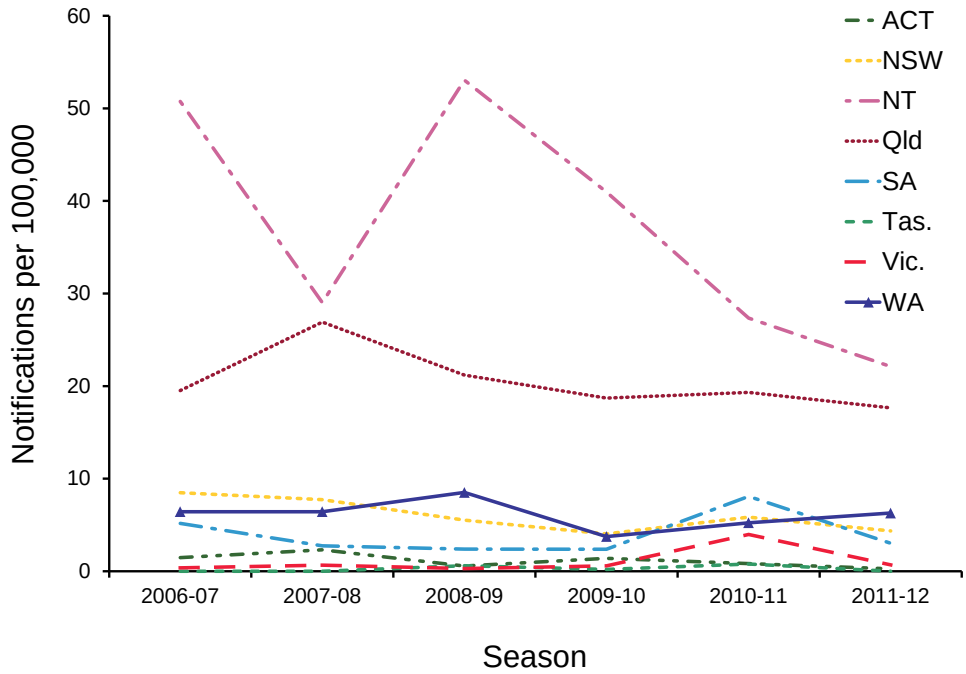
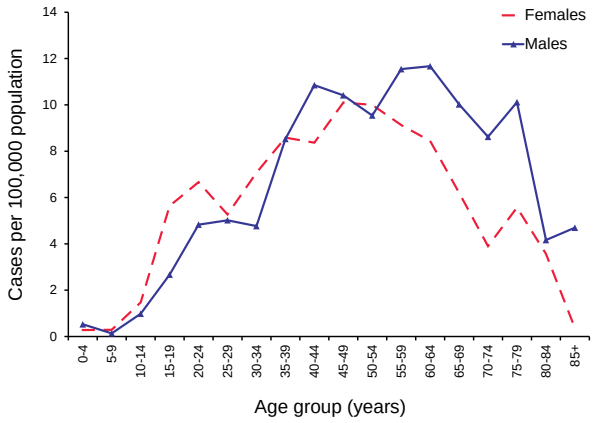
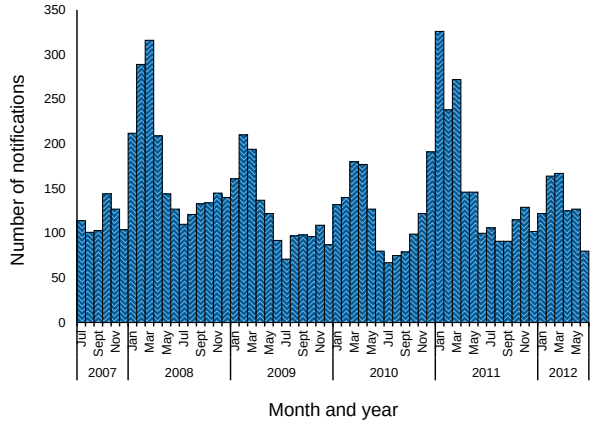


Figure 5: Notification rate for Barmah Forest virus infection, Australia, 2011–12, by age group and sex



In 2011–12, infections were most frequently notified between February and March (Figure 6), and while BFV notifications showed a seasonal trend, this trend was less marked than for RRV infections. The higher than expected numbers of BFV notifications in the cooler months is possibly an artefact, reflecting the possibility of false positive IgM diagnoses. Subsequent to the 2011–12 season, in October 2012, the number of BFV notifications began to increase dramatically, and marked the start of an epidemic of notifications due to false positive IgM diagnoses.

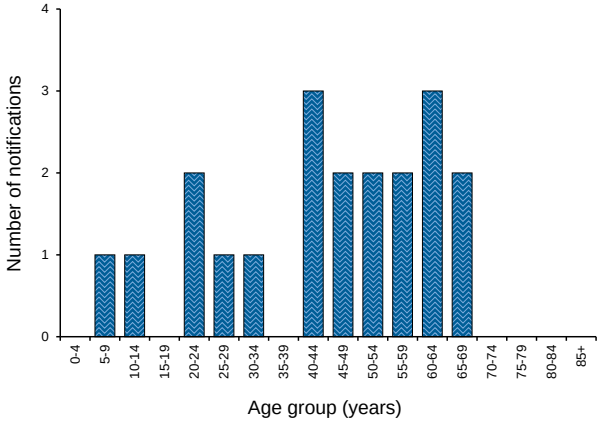
Figure 6: Notifications of Barmah Forest virus infection, Australia, July 2007 to June 2012, by month and year



Chikungunya virus infection

CHIKV infection is a notifiable disease in all jurisdictions other than the Australian Capital Territory, where there are plans to make the infection notifiable in the future (Ranil Appuhamy, ACT Health personal communication). There were 20 notifications of CHIKV infection during the 2011–12 season compared with a 5–year mean of 26.4 cases. All cases were acquired overseas, with complete information supplied on the country of acquisition for 15 of these cases. The most frequently reported countries of acquisition were India (6 cases), Thailand (2 cases) and Indonesia (2 cases). For 4 notifications, the specific country of acquisition could not be determined (for example, due to visiting multiple countries within their incubation period), and 1 case was lost to follow-up so the place of acquisition could not be determined. CHIKV infection was most frequently notified amongst young and middle aged adults (Figure 7).

Figure 7: Notifications of chikungunya virus infection, Australia, 2011–12, by age group



Flaviviruses

This section provides information on several flaviviruses notified to NNDSS including DENV, MVEV, KUNV and JEV. Other flaviviruses may be notified under the Arbovirus (NEC) category.

Dengue virus has 4 serotypes, all of which are notified in imported cases to varying degrees each year, and may be involved in local outbreaks. The clinical illness is characterised by mild to severe febrile illness with fever, headache, muscle/joint pain and sometimes a rash. A minority of cases progress to severe dengue with haemorrhage and shock, more commonly on subsequent infection with a different DENV serotype.

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. *Culex 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 children in

affected areas of Queensland and for long term travellers to endemic areas)[11] but there are no vaccines currently available for DENV, MVEV or KUNV infection.

Dengue virus infection

There were 1,446 notifications of DENV infection during the 2011–12 season. Of these, 18 cases were acquired in Australia, and 1,390 cases acquired the infection while overseas (Table 2). For the remaining 38 cases, no information on place of acquisition was supplied. In 2011–12, the median age of cases was 38 years (range 0–86 years), and 48% (n=704) of cases were male.

Locally-acquired dengue virus infection

The 18 notified cases of DENV infection acquired in Australia during 2011–12 (9 DENV-1, 2 DENV-2 and 7 untyped) was an 87% decrease compared with the 134 locally-acquired cases in 2010–11, and was the lowest number reported since the 2000–01 season when there were 10 cases.

Local transmission of dengue in Australia is normally restricted to areas of northern Queensland where the key mosquito vector, *Ae. aegypti* is present.[12] 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.[13]

In 2011–12, 16 cases of locally-acquired DENV were notified by Queensland and one each by New South Wales and Victoria. All but one of these cases resided or travelled in north Queensland and those reported by Queensland were linked with one of the 3 known outbreaks (2 in Townsville and

Table 2: Notifications of dengue virus infection, Australia, 1 July 2006 to 30 June 2012, by year, state or territory and place of acquisition

Place of acquisition	Year	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Locally-acquired*	2006–07	0	6	1	48	0	0	0	0	55
	2007–08	0	5	0	26	3	0	0	0	34
	2008–09	0	5	0	1,003	1	0	4	1	1,014
	2009–10	0	3	0	33	0	0	1	0	37
	2010–11	0	2	1	126	0	0	3	2	134
	2011–12	0	1	0	16	0	0	1	0	18
Overseas-acquired	2006–07	2	65	14	58	12	0	9	27	187
	2007–08	4	100	25	78	30	4	15	94	350
	2008–09	14	169	27	115	25	6	18	120	494
	2009–10	19	121	36	125	11	4	51	226	593
	2010–11	4	221	29	180	27	5	139	524	1,129
	2011–12	11	240	69	209	44	9	247	561	1,390
Unknown	2006–07	0	0	0	5	0	0	1	0	6
	2007–08	0	0	0	4	2	0	0	0	6
	2008–09	0	0	0	5	0	0	1	0	6
	2009–10	0	2	0	1	0	0	1	0	4
	2010–11	8	2	1	2	1	0	0	1	15
	2011–12	6	4	0	0	0	0	28	0	38
Total	2006–07	2	71	15	111	12	0	10	27	248
	2007–08	4	105	25	108	35	4	15	94	390
	2008–09	14	174	27	1,123	26	6	23	121	1,514
	2009–10	19	126	36	159	11	4	53	226	634
	2010–11	12	225	31	308	28	5	142	527	1,278
	2011–12	17	245	69	225	44	9	276	561	1,446

* 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.

1 in Cairns) during the reporting period. One of the outbreaks continued post 30 June 2012, and data on cases after 2011–12 are not included in this report. The prevention of incursion of dengue vectors into densely populated areas of South-East Queensland where imported dengue cases are regularly notified, is a continuing priority in Queensland. Despite frequent outbreaks relating to transmission from imported cases, mosquito and infection control measures undertaken by public health authorities and by residents have ensured that dengue has not become endemic in north Queensland.

Overseas-acquired dengue virus infection

There were 1,390 notifications of dengue virus infection acquired overseas during the 2011–12 season (Table 2), 2.5 times the 5-year mean of overseas-acquired infections (550.6). All states and territories reported increased numbers of notified cases of overseas-acquired DENV infection compared with 2006–07, and the ratio compared with the 5-year mean ranged between 1.3 in the Australian Capital Territory, to 5.3 in Victoria.

The country of acquisition was available for all but two of the overseas-acquired cases (n=1,388) (Figure 8). Indonesia was the country of acquisition for more than half of all overseas acquired cases (64.2%, n=893). The infecting DENV serotype was determined for 23.1% (n=330) of overseas-acquired dengue cases (down from 50% in 2010–11 and 64% in 2009–10). DENV 2 (n=205) was the most frequently reported serotype in 2011–12 (Figure 8, Table 3).

Japanese encephalitis virus infections

There was 1 notification of JEV infection in Australia during 2011–12. The case was a 16-year-old girl who acquired the infection in the Philippines and was diagnosed in February 2012. The last locally-acquired case was in 1998.[14]

Kunjin virus infection

There was 1 human case of KUNV infection notified in Australia during 2011–12. The case was a 44-year-old woman from the South Coast of New South Wales, who was diagnosed in December 2011. This is thought to have been the 1st case ever to have been acquired in a coastal area of the state.

Murray Valley encephalitis

In 2011–12, two cases of MVEV infection in Australia were notified, compared with an average of 4.2 cases for the previous 5 years.

Figure 8: Notifications of dengue virus infection, Australia, July 2006 to June 2012, by month, year and place of acquisition

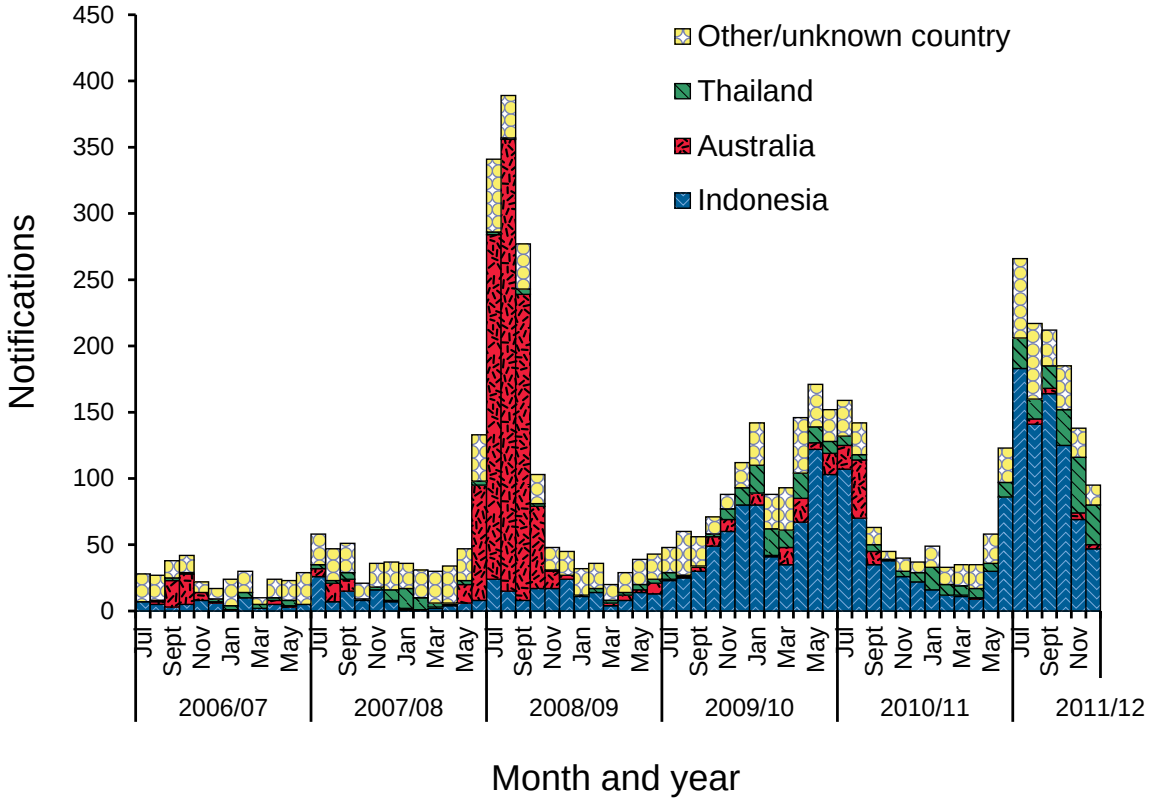


Table 3: Overseas acquired cases of dengue virus infection, Australia, 2011–12, by serotype and country of acquisition

Country	Total number of cases	Percentage of cases*	Dengue virus serotype				
			DENV 1	DENV 2	DENV 3	DENV 4	Untyped
Indonesia	893	64.3	30	168	11	8	676
Thailand	210	15.1	15	16	17	3	159
East Timor	55	4.0	1	1	13	0	40
The Philippines	45	3.2	3	0	1	1	40
India	35	2.5	1	2	1	0	31
Fiji	25	1.8	5	0	0	0	20
Malaysia	23	1.7	3	4	1	0	15
Vietnam	16	1.2	0	3	1	0	12
Sri Lanka	14	1.0	2	1	0	0	11
Papua New Guinea	10	0.7	1	2	2	0	5
Bangladesh	9	0.6	1	2	0	0	6
Cambodia	7	0.5	0	1	0	0	6
Kiribati	7	0.5	2	0	0	0	5
South East Asia	4	0.3	0	0	0	0	4
Pakistan	3	0.2	0	1	0	0	2
Other countries†	32	2.3	0	2	0	1	29
Unknown country	2	0.0	0	2	0	0	0
Total	1,390	100.0	65	205	47	13	1,061

* Excludes cases where the specific country was unknown. Percentages do not add up due to rounding.

† Each country with less than 2 cases.

The 1st case was a 25-year-old woman who was diagnosed in New South Wales in December 2011. The 2nd case was a 14-year-old boy who acquired the infection in Papua New Guinea and was diagnosed in Queensland in February 2012.

Yellow fever

There were no notifications of yellow fever in 2011–12.

Vertebrate, vector and climate surveillance programs for flaviviruses in 2011–12

The sentinel chicken program is designed to detect flavivirus activity. In 2011–12, sentinel chicken flocks were located in the Northern Territory, New South Wales, South Australia, Victoria and Western Australia. The programs aim to provide early warning of the endemic arboviruses MVEV and KUNV, as well as exotic arboviruses such as JEV.[15] Public health messaging or other response measures can be implemented when chickens from a flock seroconvert to a flavivirus of interest. Public health messaging may advise residents or target groups such as campers or fishermen of the need to take added precautions to avoid mosquito bites.

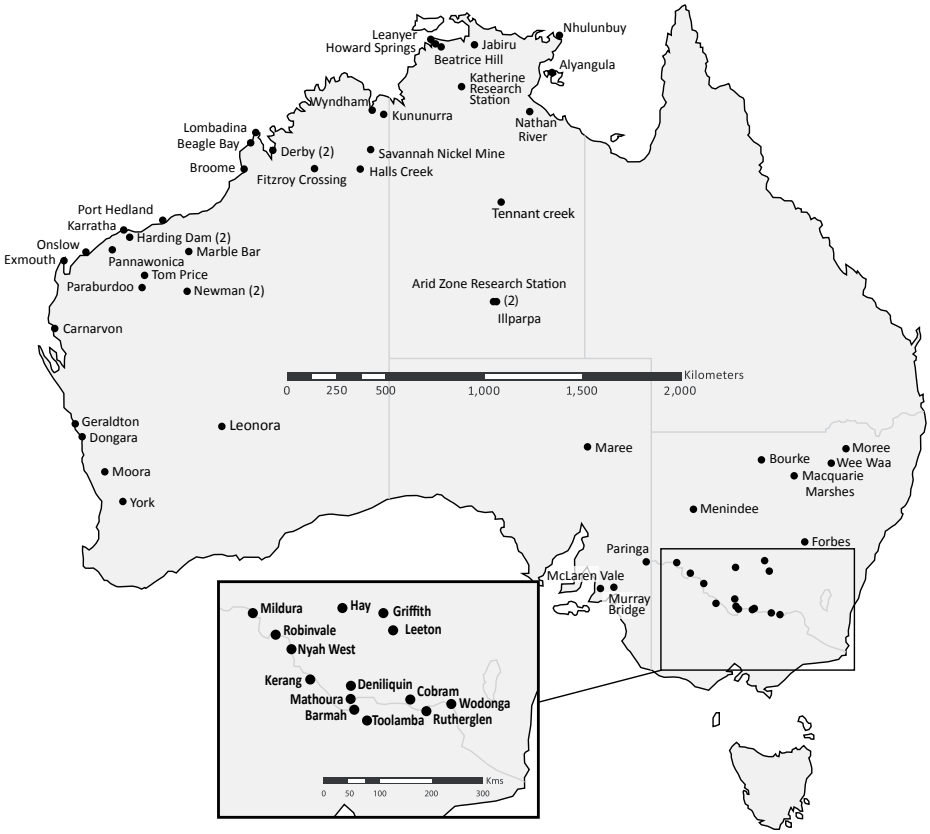
Sentinel chickens are replaced at least annually and more frequently if birds die or large proportions seroconvert. Flocks are well located geographically to detect flavivirus activity and to provide a timely and accurate indication of risk to people (Map).[16]

New South Wales

The 2011–12 sentinel chicken program began on 27 October 2011 with the first bleed and ended on 30 April 2012. A total of 11 flocks each containing up to 15 Isa Brown pullets were deployed, with 1 flock each at Bourke, Deniliquin, Forbes, Griffith, Hay, Leeton, Macquarie Marshes, Menindee, Moama (near Mathoura), Moree and Wee Waa (Map).

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 are responsible for training the chicken handlers. A veterinar-

Map: Location of sentinel chicken sites, Australia, 2011–12



ian (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.[17]

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.

The season began with 164 pullets and 6 deaths were recorded during the program. A total of 2,660 samples were received from the 11 flocks in New South Wales over the 6-month period in 2011–12. This represented 5,320 enzyme-linked immunosorbent assay (ELISA) tests (excluding controls and quality assurance samples), with each specimen being tested for MVEV and KUNV antibodies.

During the 2011–12 season, MVEV and KUNV were first detected on 4 December 2011 at Hay and Forbes respectively (Table 4). There were a number of other seroconversions to MVEV, but no further KUNV detections.

Northern Territory

The current Northern Territory sentinel chicken program commenced in January 1992 and replaced an earlier program run by the Australian Quarantine Inspection Service. Sentinel chicken flocks in the Northern Territory are maintained, bled and analysed for flaviviruses in a combined program between the Department of Health, the virology laboratories of DPIF and volunteers.

Sentinel chicken flocks are located at Leanyer (Darwin), Howard Springs, Coastal Plains Research Station (CPRS), Jabiru, Alyangula, Katherine, Nhulunbuy, Nathan River, Tennant Creek, Jabiru,and Alice Springs (2 flocks), Nathan River and Alyangula. DPIF officers or volunteers usually bleed flocks once a month and the samples are tested for MVEV and KUNV. When chickens from a flock show antibodies to MVEV during a prime risk period, a media warning is issued for the general area or the region for

Table 4: Seroconversions to Murray Valley encephalitis virus and Kunjin virus in sentinel chicken flocks, New South Wales, 2011–12

Site	Seroconversions			First positive date	Last positive date
	MVEV	KUNV	Total		
Forbes	0	1	1	30 April 2012	30 April 2012
Hay	2	0	2	4 December 2011	11 December 2011
Leeton	8	0	8	12 December 2011	12 December 2011
Macquarie Marshes	3	0	3	13 December 2011	13 December 2011
Moama	1	0	1	7 December 2011	7 December 2011
Total	14	1	15	4 December 2011	30 April 2012

the risk period. These warnings advise residents and visitors of the need to take added precautions to avoid mosquito bites.

In February 2012, the Northern Territory sentinel chicken program was revised, and the Jabiru, the Ilparpa Swamp and the Alyangula flocks were terminated. The Jabiru flock was terminated due to difficulty in regular bleeding. The Alyangula flock was terminated, as no seroconversions to MVEV have occurred, most likely due to the ecology in the area not being favourable for MVEV activity. The Ilparpa Swamp flock was terminated due to sufficient data being available from the flock. In addition, it was decided to only bleed the chickens during the highest MVEV risk period between December and June inclusive.

In the 2011–12 season, MVEV activity was only detected in the Katherine region, with seroconversions in Katherine in January and in Nathan River in January and February 2012. KUNV activity was detected in Nathan River in January 2012 and in the Nhulunbuy flock in the East Arnhem region in April 2012 and in Nathan River in January 2012.

Preliminary results from the new experimental mosquito honey-baited card arbovirus surveillance system trial carried out between February and June 2012 in the Darwin area, in collaboration with Queensland Health, James Cook University and the Berrimah Veterinary Laboratory, showed there was additional KUNV activity in the Darwin region in February and March. This new system is thus more sensitive than sentinel chickens and is envisaged to replace the current Northern Territory sentinel chicken program in at least some locations in the future.

Queensland

Queensland does not have a single system for mosquito surveillance due to its large geography and mix of tropical and sub-tropical climate. The prevalence of mosquito-borne diseases such as DENV, RRV, BFV and malaria, along with the presence of vectors such as *Ae. aegypti* and *Ae. albopictus* in specific areas of Queensland pose a particular challenge for surveillance activities.

During 2011–12, a range of mosquito and arbovirus surveillance and/or control activities were carried out in Queensland. This included targeting mosquitoes inhabiting domestic containers in southern and central Queensland; *Ae. albopictus* elimination activities on Horn and Thursday Islands; surveillance and control of *Ae. aegypti* in north Queensland; mosquito surveillance activities on Saibai and Dauan Islands following a malaria outbreak in early 2011;[18] salt water and fresh water mosquito surveillance and control activities across Queensland; and targeted arbovirus monitoring using honey-baited surveillance technology in Mt Isa and Emerald.

During 2011–12, a large survey of Brisbane houses (n=2,381) was led by the Queensland Institute of Medical Research, in collaboration with Queensland Health and the Brisbane City Council to identify mosquito species inhabiting domestic containers. No *Ae. aegypti* were detected at any Brisbane premise. *Ae. notoscriptus,* a likely vector of RRV and BFV, was found in 949 premises.

Household surveys for container inhabiting mosquitoes were also undertaken at approximately 2,300 premises in 13 towns in regional central and southern Queensland during the reporting period, with *Ae. aegypti* found in six of the 13 towns. *Ae. aegypti* had been detected in all of these towns in previous years. Of note, an extensive 473 house survey for *Ae. aegypti* was undertaken in the town of Gin Gin following confirmation of an imported dengue case. Mosquito larvae were found at approximately 40% of premises and *Ae. aegypti* was detected at 11 properties across approximately 6 neighbourhood blocks. No local transmission of dengue occurred. Similarly, in May 2012, *Ae. aegypti* was detected at several residences nearby to a notified overseas acquired dengue case and again no local transmission occurred.

An unusually low level of activity by saltmarsh mosquitoes in the south-east corner of the State continued throughout the 2011–12 season. In general, the number of aerial programs required targeting *Ae. vigilax* was well below expectations, and started later in the season and finished early. This may have been attributable to higher than average rainfall over the preceding 18 months, keeping larval habitat continuously wet, rather than subject to the usual cycles of wetting and drying, which favours saltmarsh mosquito production.

For similar reasons, an interesting spectrum of freshwater species was more abundant than usual in the south-east. In addition to *Culex annulirostris*, some less common species, which were also abundant, included *Ae. aculeatus, Ae. burpengaryensis* and *Ae. lineatopennis*, suggesting the persistence of the eggs of these species over long periods.

In coastal Central Queensland, there were also relatively low numbers of *Ae. vigilax*. But in the Central Highlands, there was medium to high rainfall and consequently very high mosquito numbers. Abundant species included *Cx. annulirostris, Ae. lineatopennis, Ae. vittiger, Ae. alternans* and *Coquillettidia xanthogaster*. One spectacular overnight collection at the sewage treatment plant at Emerald in March collected approximately 30,000 mosquitoes. *Austrosimulium pestilens* was also active across Central Queensland, following good summer rains. A number of coastal local governments sent staff and equipment to assist inland mosquito control after floods in March.

A new honey-baited arbovirus surveillance tool based on the collection and testing of mosquito saliva was trialled in 2 sites, Mt Isa and Emerald, in Queensland between February and May 2012 to compare the sensitivity of this novel system with sentinel chicken surveillance.[19] In Emerald, KUNV was detected in 8 traps between February and March and RRV was detected once each in February and April. In March, BFV, KUNV and RRV were detected in Mt Isa, with an additional detection of RRV in April. The honey-baited surveillance tool will be used more broadly in 2012–13 to survey areas where MVEV viral activity could be present.

Following a falciparum malaria outbreak in the Torres Strait islands of Saibai and Dauan in early 2011, surveillance of overnight landing rate counts and hourly indoor and outdoor mosquito collections were conducted on Saibai Island. Carbon-dioxide-baited United States Centers for Disease Control and Prevention (CDC) light traps were also used to compare vector activity across the island. More than 2,000 samples of the potential malaria vectors *Anopheles farauti* and *Anopheles hilli* were collected and host-seeking behaviours were observed.

South Australia

The Mosquitoes and Public Health Research Group at the University of South Australia (UniSA) provided contracted mosquito surveillance and spot control services approximately monthly (11 trips in total) to 7 local governments along the Murray River in South Australia from September 2011 to April 2012. UniSA also provided mosquito surveillance and control services for 2 northern metropolitan councils, the City of Salisbury and the City of Port Adelaide Enfield for the 2011–12 season. South Australia Health funds half of all local government costs for mosquito surveillance and control on public land through the South Australian Mosquito Management Subsidy.

In the north of metropolitan Adelaide, *Ae. camptorhynchus* and *Ae. vigilax* numbers were low compared with the previous 2 seasons. The mosquito populations along the Murray River during the season exhibited 2 distinct patterns associated with geographic location. Traps north of Mannum in the Mid Murray Council were typified by low numbers through most of the season with a sudden increase around the end of March into April of primarily *Cx. annulirostris*. These areas also lacked any significant number of the spring mosquito *Ae. camptorhynchus* at any time in the season. In the northern river Murray councils, increased numbers of *Ae. eidsvoldensis* adults were recorded and an increased number of *Ae. alternans* larvae were observed. The mosquito populations at Mannum and to the south of this town, retained distinct spring peaks of *Ae. camptorhynchus* through to December.

Throughout the 2011–12 season, sentinel chicken surveillance was conducted opportunistically in South Australia with 2 seroconversions to KUNV at Marree in a remote area of the State in December 2011. One of these had previously been seronegative in May 2011, while the other was a new introduction to the flock, had not been previously bled, and was of unknown origin.

Victoria

A winter sentinel surveillance program was in place between April and October 2011 in response to increased arboviral activity during 2010–11. Sentinel chicken flocks in Barmah, Kerang and Mildura were bled and tested fortnightly for flaviviruses. Fortnightly mosquito trapping was also conducted at these 3 winter sentinel chicken flock locations. Across the winter sentinel monitoring program, 384 serum samples were tested for general flavivirus antibodies during the period of July to October 2011. There was no evidence of seroconversion. In addition, no arboviruses were detected in the trapped mosquitoes.

The 9 standard seasonal sites were located at Mildura, Robinvale, Nyah West, Kerang, Barmah, Toolamba, Cobram, Rutherglen and Wodonga. The standard 2011–12 monitoring period was brought forward to mid-October in eight of the 9 flock sites.

The standard sentinel chicken monitoring program tested 4,249 serum samples for antibodies to flaviviruses using a defined epitope blocking ELISA. KUNV activity was detected in 2 chickens; one from the Barmah flock during week 14 (beginning 2 April) and one from a chicken in the Nyah West flock during week 17 (beginning 23 April 2012). The Barmah flock seroconversion was confirmed by The Elizabeth Macarthur Agricultural Institute, NSW Department of Primary Industries. The Nyah West flock site chicken died before sufficient blood could be collected for additional testing.

No flaviviruses were isolated in trapped mosquitoes (70,290 sent for testing) during the 2011–12 season.

Western Australia

The flavivirus sentinel chicken program in Western Australia is undertaken by the ASRL at The University of Western Australia, on behalf of the Western Australian Department of Health. The sentinel chicken surveillance program is approved by The University of Western Australia Animal Ethics Committee. Many state and local government authorities and community volunteers also take part in the program. Twenty-eight sentinel chicken flocks (of up to 12 chickens) are located at major towns and communities in the Kimberley, Pilbara, Gascoyne, Goldfields, Midwest and Wheatbelt regions of Western Australia (Map). Blood samples are collected from the chickens by environmental health officers or trained volunteers at fortnightly intervals. Samples are transported to ASRL where they are tested for antibodies to flaviviruses using an epitope blocking ELISA.[20]

Central parts of Western Australia experienced wetter than normal conditions prior to the commencement of the 2011–12 wet season. November 2011 was the second wettest on record in Western Australia with thunderstorm activity creating heavy rainfall through the Pilbara, Gascoyne and northern Goldfields. The combination of an active monsoonal trough, Tropical Cyclone Heidi crossing the Pilbara coast and ex-Tropical Cyclone Iggy resulted in parts of the west Kimberley, Pilbara and north Gascoyne recording their wettest January on record. Monsoonal activity during the middle of March and Tropical Cyclone Lua crossing the Pilbara coast resulted in much of the Kimberley and Pilbara experiencing above average rainfall during the month.

A total of 4,185 serum samples from 28 flocks were tested for antibodies to flaviviruses during 2011–12.[21, 22] Seroconversions to flaviviruses were detected in 225 (5.3%) samples. Seroconversions to MVEV detected at Paraburdoo (3 samples), Fitzroy Crossing (1 sample) and Karratha (1 sample) in July, Roebuck Plains (1 sample) and Ophthalmia Dam (1 sample) in August and Roebuck Plains (2 samples), Port Hedland (4 samples) and Marble Bar (7 samples) in September were associated with activity continuing from the 2010–11 season.

The 1st activity associated with the 2011–12 wet season occurred in November 2011, when MVEV (1 sample) and KUNV (1 sample) infections were detected in sentinel chickens at Kununurra in the north-east Kimberley region and 2 KUNV infections were detected at Moora, in the Wheatbelt. This was the earliest start to the flavivirus season in more than 10 years. High levels of flavivirus activity were subsequently detected throughout the Kimberley, Pilbara and Midwest regions in December. The activity continued in January (Kimberley, Pilbara and Midwest/Wheatbelt), February and March (Kimberley, Pilbara and Midwest), April (Kimberley, Pilbara and Gascoyne), May (Kimberley, Pilbara and Midwest), and June (Kimberley). Overall, there were 195 seroconversions to MVEV (including 3 dual MVEV/KUNV infections) and 31 KUNV infections (including the dual MVEV/KUNV infections). The overall level of flavivirus activity was slightly lower than the very high levels seen in 2000 and 2011.[10, 23] The majority of sentinel chicken flocks required replacement with new chickens during the course of the season, some on multiple occasions. No human cases of MVE were diagnosed in Western Australia during the 2011–12 season (Dr David Smith, PathWest Laboratory Medicine Western Australia, personal communication).

The Western Australia Department of Health issued 3 media statements. The 1st was issued on 24 August 2011 following continued detections of MVEV antibodies in sentinel chickens in the Kimberley and Pilbara regions associated with the 2010–11 wet season. The 2nd was issued on 16 December 2011 after MVEV and KUNV infections were detected in sentinel chickens in the Kimberley and Wheatbelt regions for the 1st time in the 2011–12 wet season. The 3rd media release was issued on 26 March 2012 after widespread detections of MVEV and KUNV infections in sentinel chickens in the Kimberley, Pilbara, Gascoyne, Midwest and Wheatbelt regions.

Tasmania

No viruses were isolated in 2011–12 from mosquitoes trapped during ad hoc collections undertaken in the Sorrell Council region.

Arbovirus infection (NEC)

This disease category enables the capture and epidemiological analysis of emerging infections within this very broad disease group. Emerging diseases are then made nationally notifiable. 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 14 notifications of arbovirus NEC in 2011–12, similar to the 5-year average of 13 cases (range 4–22 cases). Half of these notifications relate to infections that were known to have been acquired overseas (n=7). In 2011–12, 5 notifications were for an unspecified arbovirus, 6 notifications were for an unspecified flavivirus, with the remainder due to the flaviviruses Kokobera (n=2) and Alfuy (n=1).

The largest number of notifications were from Queensland (n=7) and Victoria (n=6). In Queensland, an extensive panel of flaviviruses is used for testing, and 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 arbovirus NEC.

Malaria

Malaria is a serious acute febrile illness that is normally 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*.[24,25]

There were 355 notifications of malaria during the season 2011–12 (Table 1), a 30.9% decrease compared with the mean of 514 notifications during the past 5 years and, consistent with the steady decline in the number of notifications since the 2004–05 season. Most infections were known to have been acquired overseas (88%, n=314), while the place of acquisition for the remainder was reported as unknown or not stated, but none were known to have been locally-acquired.

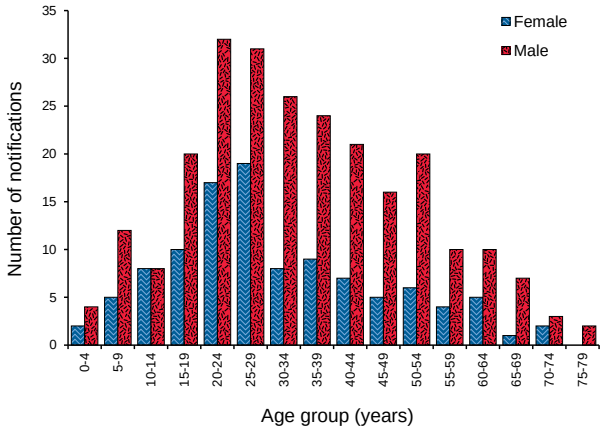
Malaria was most frequently reported amongst people aged 20–29 years, with 99 notified cases (Figure 9). Similar to previous years, the majority of cases were male (69.3%, n=246), and males predominated in every age group. Cases were from all jurisdictions.

The infecting species was reported for 97.7% of notifications during the season 2011–12. *P. falciparum* and *P. vivax* were the predominant infecting species (Table 5). In 2011–12, no cases were infected with *P. knowlesi*.

Complete information about the country of acquisition was available for 87.0% (n=309) of malaria cases. Papua New Guinea was the most frequently reported place of acquisition for cases with a country of acquisition specified (21.7%, 67/309), followed by India (11.7%, 36/309) (Figure 10).

P. vivax infections were commonly associated with travel to Asia or Pacific nations (96%, 106/110). *P. falciparum* infections were frequently associ-

Figure 9: Notifications of malaria infection, Australia, 2011–12, by age group and sex*



* Sex was not available for 1 case, and this case is not included here.

Table 5: Cases of malaria, Australia, 2011–12, by *Plasmodium* species

Malaria species	Number of cases	% of all cases
Plasmodium falciparum	206	58.0
Plasmodium vivax	123	34.6
Plasmodium ovale	9	2.5
Plasmodium malariae	6	1.7
Plasmodium falciparum and *P. malariae*	3	0.8
Plasmodium spp.	8	2.3
Total	355	100.0

ated with travel to the Middle East and Africa (79%, 139/176), and only 3 *P. vivax* infections (2.7%) were associated with travel to African and Middle East regions.

Other surveillance and research activities

Exotic mosquito detections at the border

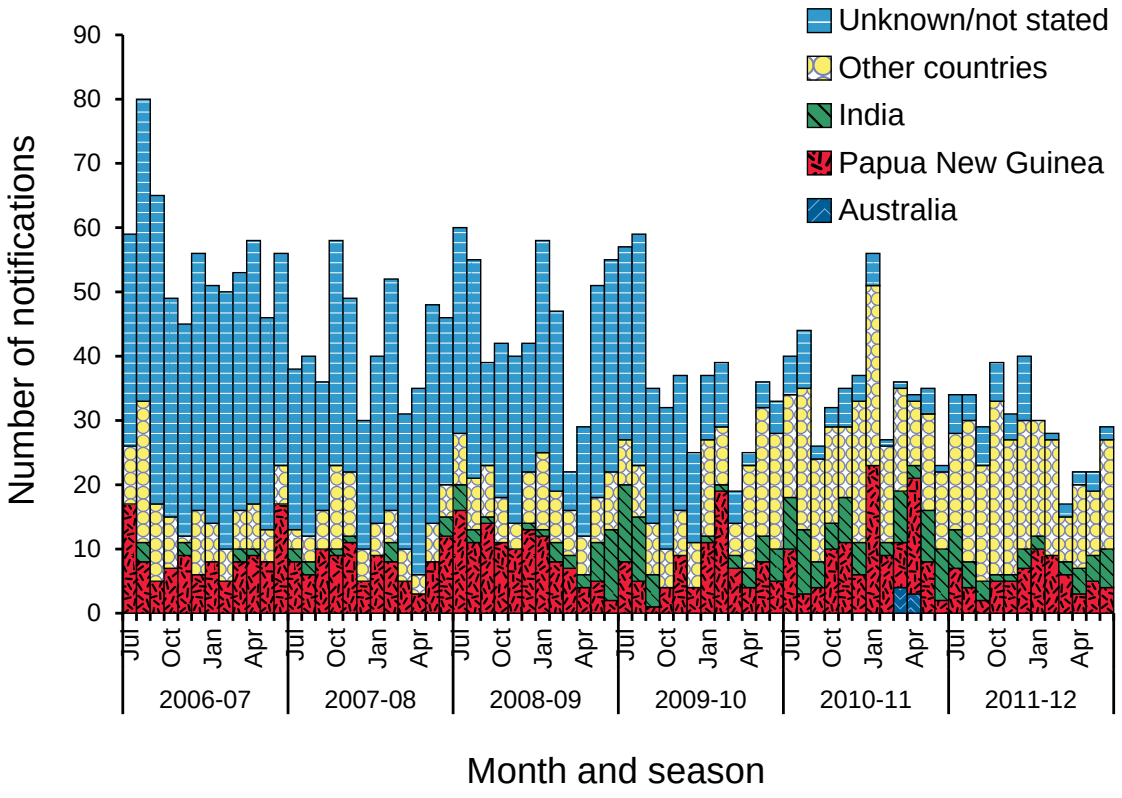
Between July 2011 and June 2012 there were 5 exotic mosquito detections made by the Department of Agriculture (formally the Department of Agriculture, Forestry and Fisheries) at the Australian border (Table 6). This is similar to the 2010–11 period where there were also 5 exotic mosquito detections. Two detections were made via inspection of international vessels and a single detection was made via routine inspection of imported cargo. The remaining 2 detections were made through vector monitoring activities performed at international ports. No further exotic mosquitoes were collected following the initial detection.

Torres Strait Aedes albopictus *Elimination and Control Program*

The Asian tiger mosquito, *Ae. albopictus*, which was previously exotic to Australia, was found on the outer islands of Torres Strait in April 2005.[26] This mosquito is capable of transmitting dengue and chikungunya, as well as becoming a new serious pest mosquito. Since 2005, the Australian Government has provided funding to Queensland Health towards a mosquito elimination program in the Torres Strait. The initial aim of the program was to eliminate *Ae. albopictus* from the Torres Strait islands. However, as elimination was subsequently not considered to be possible, the development and implementation of a program based on the *cordon sanitaire* approach (a barrier designed to prevent a disease or other undesirable condition from spreading) around Thursday and Horn islands was initiated in May 2008 in an attempt to prevent the spread of *Ae. albopictus* further south.

The main focus of the program in 2011–12 was to suppress *Ae. albopictus* populations and possibly eliminate the species from Horn Island and Thursday Island, both regarded as the gateway to the mainland of Australia due to their strategic

Figure 10: Notifications of malaria, Australia, July 2006 to June 2012, by month, year and place of acquisition*



* Thirty-four cases between 2006–07 and 2008–09 were listed as being acquired in Australia, however this was due to a default value for place of acquisition. The country of acquisition was set to unknown for this report.

Table 6: Exotic mosquito detections at the border, Australia, 2011–12

Date	Species	Location	Method of detection	Source / origin	Action/ mitigation	Surveillance results
July 2011	*Ae. albopictus* (larvae)	Cairns	Inspection	Larvae found in water within storage recesses inside vessel hold. Vessel from Irian Jaya.	Water in the hold treated. Mosquito harbourage treatments performed in the surrounding port area and additional trapping. Storage recess modified to exclude water pooling.	No further exotic mosquitoes detected.
Aug 2011	*Ae. albopictus* (adult)	Cairns	Inspection	Adult mosquitoes observed flying inside vessel hold. Vessel from Irian Jaya. No mosquito breeding observed in the vessel hold.	Targeted trapping performed on board the vessel and in the hold. Increased surveillance and trapping.	No further exotic mosquitoes detected.
Jan 2012	*Ae. aegypti* (1 adult)	Darwin	CO_2 baited BG trap.	Unknown/unable to identify source.	Ultra low volume fogging, receptacle treatment surveys, increased trapping.	No further exotic mosquitoes detected.
Feb 2012	*Ae. albopictus* (larvae and adults)	Townsville	Inspection	New oversize tyres from Papua New Guinea.	Tyres treated. Ultra low volume fogging, mosquito harbourage treatments, receptacle treatment surveys and increased trapping.	No further exotic mosquitoes detected.
June 2012	*Ae. Albopictus* (1 adult)	Townsville	CO_2 baited BG trap.	Detection coincided with the importation of new oversize tyres from Papua New Guinea but no mosquitoes found associated with the imported tyres.	Ultra low volume fogging, mosquito harbourage treatments, receptacle treatment surveys and increased trapping.	No further exotic mosquitoes detected.

location and transport networks. Consistent monitoring of mosquito densities and distribution on the 2 islands showed up to a 60-fold decline in numbers of adult *Ae. albopictus* after intensive intervention that included residual pyrethroid treatment of vegetated peri-domestic harbourage sites. The control operation also included repeated house-to-house yard inspections on at least 800 properties for removal or treatment of water-holding receptacles. At least 3,000 receptacles were inspected and treated on each field visit. The yard inspections also constituted part of the mosquito surveys and *Aedes* larval samples were collected from all positive receptacles for identification. A decline in the Breteau Index of up to 10-fold was recorded for *Ae. albopictus* on the 2 islands during the wet seasons of the reporting period, demonstrating a dramatic impact of the control program.

Surveillance activities for early detection of *Ae. albopictus* incursions in the high-risk zones of Cairns and the Northern Peninsula Area (Bamaga, Seisia, New Mapoon, Injinoo and Umajico) throughout the 2011–12 wet season did not find any *Ae. albopictus*.

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 2011 to 30 June 2012, activities undertaken by health authorities in response to human cases, and evidence of virus activity. Sentinel animal and

vector monitoring continue to be an important part of the early warning system for arboviruses in Australia.

The number of dengue notifications was notably increased compared with historical totals; there were 1.7 times as many dengue cases during the 2011–12 season as during the previous 5 years, due to an increase in the number of overseas-acquired cases. For all other diseases, notification counts and rates were similar to or below the 5-year means.

The number and proportion of dengue cases that were overseas acquired has increased in recent years, and for cases acquired in Indonesia, (which comprises most of the increase), the increase in the frequency of travel by Australians to Indonesia does not completely explain this increase.[27] Viraemic returning travellers (or visitors from overseas) present a risk of starting a local outbreak in North Queensland, and travellers should minimise the risk of infection by avoiding being bitten by mosquitoes through the use of personal prevention measures. Travellers are encouraged to consider the information available on the Smartraveller travel health website and to seek a doctor's advice prior to travel.[28]

The risk of dengue becoming endemic in North Queensland following an imported case remains a major concern. Public health authorities conduct extensive control efforts in partnership with residents in order to control the outbreaks that occur every season. The most recent large outbreak of dengue in Australia was in the 2008–09 season, when there was an outbreak of DENV3 in Cairns that lasted for 31 weeks, with 915 cases.[29] Subsequent to this reporting period (in 2012–13 and 2013–14) there have been significant outbreaks, but each comprised less than 150 notified cases. The Queensland Dengue Management Plan 2010–15[13] outlines current best practice in dengue management for the 4 levels of dengue activity; ongoing prevention, response to sporadic cases, outbreak response, and multiple outbreaks.

During the 2011–12 season, there were a small number of imported cases of CHIKV in Australia, but no local transmission. Health authorities are alert to any changes in the number of notified cases in Australia and in the region, and to the possibility of local transmission, particularly in North Queensland where competent mosquito vectors occur in suitable environments near susceptible populations.[30] While *Ae. aegypti* and *Ae. albopictus* are the principal vectors for CHIKV, laboratory studies suggest the possibility of spread by some Australian mosquito species.[31] CHIKV transmission in Australia would have significant population health implications.

The national surveillance case definitions for RRV, BFV and CHIKV require laboratory definitive evidence. One option for laboratory definitive evidence is virus-specific IgM alone, in the absence of IgM to other alphaviruses. These case definitions may introduce the possibility of false positive diagnoses, where the pre-test probability of infection is low (i.e. where the infection is rare, such as RRV or BFV in metropolitan areas). This has been particularly recognised as a problem for BFV notifications in recent years, and the laboratory case definition (on which the surveillance case definition is based) is currently under review by the Public Health Laboratory Network (PHLN). In Victoria, BFV diagnoses by IgM alone, but without a compatible exposure history (such as metropolitan Melbourne cases who have not travelled to rural areas) are followed up, and a 2nd blood sample is requested from patients to demonstrate seroconversion (Rebecca Feldman, Victorian Department of Health, personal communication). Consequently, an epidemic of false positive BFV from October 2012 in a number of Australian states was not observed in Victoria.

Since 2005, *Ae. albopictus* has become established on the majority of islands in the Torres Strait. The risk of dengue transmission in central and southern Queensland and other jurisdictions would be substantially increased if this vector became established on the mainland, and control efforts through the Torres Strait *Ae. albopictus* Elimination and Control Program are vital to prevent incursions to the mainland. In mid-2011, small populations of *Ae. albopictus* continued to persist on Horn Island despite control efforts, however since that time, the program has been demonstrably successful at reducing *Ae. albopictus* numbers in the *cordon sanitaire* to levels where eradication is now a real possibility. The 60-fold decline in the number of adult *Ae. albopictus* on the 2 islands following intensive intervention, and a 10-fold decline in the Breteaux index demonstrates this impact.

In response to the MVEV outbreak between March and May 2011, the AHPPC requested that NAMAC prepare a framework for the surveillance, prevention and control of MVEV in Australia, emphasising a One-Health approach, along with guidance for public health units as part of CDNA Series of National Guidelines (SoNGs). The SoNGs document and the Framework were endorsed by AHPPC on 14 November 2013.

The limitations of surveillance data used in this report are referred to in detailed notes on the interpretation of NNDSS, which is available in the 2011 NNDSS annual report.[1] A specific limitation of the data used in this report relates to the virological testing, which is required to

distinguish alphavirus disease from other causes of arthritis. The alphavirus infections notified to NNDSS each season are based on laboratory definitive evidence only and assumes a clinically compatible illness. A case can still be notified when clinical illness may not be consistent with the diagnosis of alphavirus infection. From 1 January 2013, revised case definitions for RRV and BFV were implemented, whereby an IgM-only diagnosis for one of these was required to be in the absence of IgM to the other. However, there remains the issue of whether IgM only is an appropriate diagnostic method for these viruses. At the time of writing, the laboratory case definition for BFV was under review by the PHLN. Another limitation on the findings of this report relates to place of acquisition of infection for infections that are commonly acquired overseas, in terms of completeness and consistency of coding. Information on place of acquisition is particularly important for the arboviruses that do not commonly occur in Australia, because it facilitates the monitoring of increased importations from particular areas, and allows the detection of any local transmission. The National Surveillance Committee is currently undertaking a project to standardise coding of place of acquisition between jurisdictions.

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 AHPPC, 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.

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Influenza epidemiology, vaccine coverage and vaccine effectiveness in sentinel Australian hospitals in 2013: the Influenza Complications Alert Network

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Abstract

The National Influenza Program aims to reduce serious morbidity and mortality from influenza by providing public funding for vaccination to at-risk groups. The Influenza Complications Alert Network (FluCAN) is a sentinel hospital-based surveillance program that operates at 14 sites in all states and territories in Australia. This report summarises the epidemiology of hospitalisations with confirmed influenza, estimates vaccine coverage and influenza vaccine protection against hospitalisation with influenza during the 2013 influenza season. In this observational study, cases were defined as patients admitted to one of the sentinel hospitals, with influenza confirmed by nucleic acid testing. Controls were patients who had acute respiratory illnesses who were test-negative for influenza. Vaccine effectiveness was estimated as 1 minus the odds ratio of vaccination in case patients compared with control patients, after adjusting for known confounders. During the period 5 April to 31 October 2013, 631 patients were admitted with confirmed influenza at the 14 FluCAN sentinel hospitals. Of these, 31% were more than 65 years of age, 9.5% were Indigenous Australians, 4.3% were pregnant and 77% had chronic co-morbidities. Influenza B was detected in 30% of patients. Vaccination coverage was estimated at 81% in patients more than 65 years of age but only 49% in patients aged less than 65 years with chronic comorbidities. Vaccination effectiveness against hospitalisation with influenza was estimated at 50% (95% confidence interval: 33%, 63%, $P<0.001$). We detected a significant number of hospital admissions with confirmed influenza in a national observational study. Vaccine coverage was incomplete in at-risk groups, particularly non-elderly patients with medical comorbidities. Our results suggest that the seasonal influenza vaccine was moderately protective against hospitalisation with influenza in the 2013 season. *Commun Dis Intell* 2014;38(2):E143–E149.

Keywords: influenza; vaccine effectiveness

Introduction

Influenza vaccination is recommended in Australia for high risk groups, including the elderly, patients with chronic comorbidities, pregnant women and Indigenous Australians.[1] The National Immunisation Program, which provides public funding for influenza and other vaccinations, aims to reduce serious morbidity and mortality from influenza. Clinical trials have shown that the influenza vaccine is moderately protective against influenza[2] but fewer studies have examined its effectiveness in reducing serious complications, as the proportion of cases requiring hospitalisation is low. However, because infection with influenza virus is relatively widespread and estimated to affect 5%–10% of the population, the incidence of hospitalisation from influenza is of public health significance.

The Influenza Complications Alert Network (FluCAN) was established in 2009 primarily to provide timely surveillance to public health authorities nationally on hospitalisations with confirmed influenza.[3] In this report, we describe the epidemiology of hospitalisation with confirmed influenza, estimate vaccine coverage in hospitalised patients with acute respiratory illnesses but without influenza, and estimate influenza vaccine protection against hospitalisation with influenza during the 2013 influenza season.

Methods

FluCAN is a national hospital-based sentinel surveillance system.[3] For the 2 most recent influenza seasons including 2013, the participating sites have been The Alfred Hospital (Vic.), Royal Melbourne Hospital (Vic.), Canberra Hospital (ACT), Monash Medical Centre (Vic.), Geelong Hospital (Vic.), Royal Perth Hospital (WA), Royal Adelaide Hospital (SA), Royal Hobart Hospital (Tas.), Mater Hospital (Qld), Princess Alexandra Hospital (Qld), Cairns Base Hospital (Qld), Alice Springs Hospital (NT), Westmead Hospital (NSW), and John Hunter Hospital (NSW). Ethical approval

has been obtained at all participating sites, at Monash University and the Australian National University.

Definitions

An influenza case was defined as a patient admitted to hospital with influenza confirmed by nucleic acid testing (NAT). Surveillance was conducted from 5 April to 31 October 2013. Test negative controls (one for each case where available) were the next tested patient with acute respiratory symptoms who was negative for influenza by NAT. Admission or transfer to intensive care unit (ICU) included patients managed in a high dependency unit. The onset date was defined as the date of admission except for patients where the date of the test was more than 7 days after admission, where the onset date was the date of the test. Admissions that are listed as influenza A include both untyped and seasonal strains, and may include infections involving the pandemic H1N1/09 strain if not specifically typed.

Estimation of vaccination coverage and effectiveness

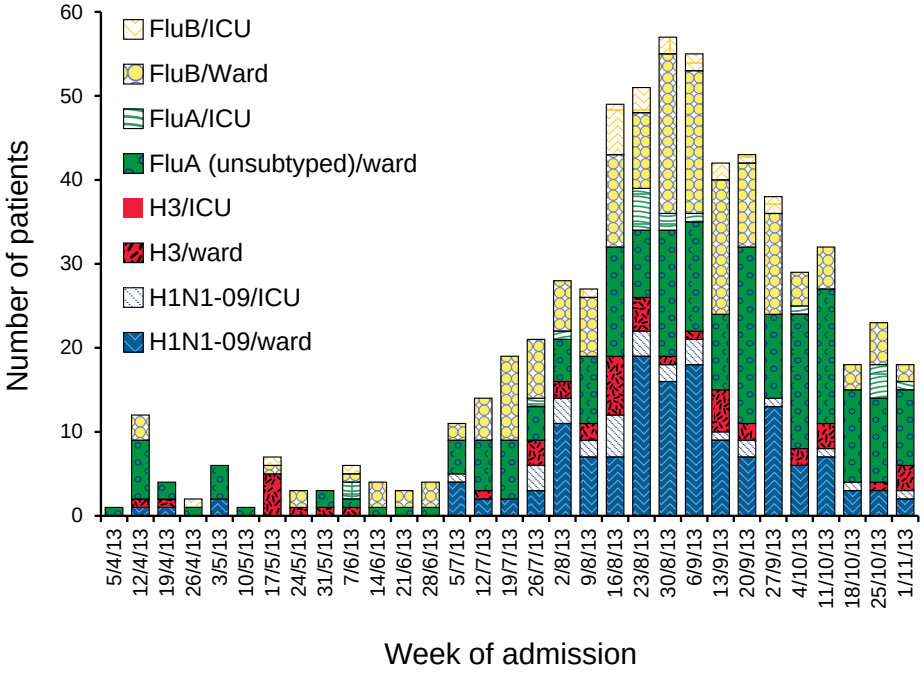
Vaccination coverage was estimated from patients admitted with influenza-like illness but who were negative for influenza by NAT. Patients were defined as being vaccinated if they reported receiving the 2013 trivalent seasonal vaccine more than 2 weeks prior to presentation (as documented in the medical record or from self-report). In Australia, only unadjuvanted vaccines are available under the National Immunisation Program although 1 adjuvanted vaccine is approved for use. Nasally administered live attenuated and quadrivalent influenza vaccines are not available in Australia.

We used an incidence density test negative design to estimate vaccine effectiveness, where controls are selected from patients without influenza contemporaneous to a case.[4–6] Vaccine effectiveness was estimated as 1 minus the odds ratio of vaccination in case patients compared with test negative control patients using methods previously described.[7,8] A multi-variable model was constructed from factors known to be associated with both vaccination and risk of illness, and therefore were regarded as potential confounders.

Results

During the period 5 April to 31 October 2013, 631 patients were admitted with confirmed influenza infection at the 14 FluCAN sentinel hospitals. In most jurisdictions, the peak number of hospitalised cases varied between late August and late October 2013 (Figure 1). The majority of cases were due to influenza A, but 30% were due to influenza B; however, the proportion due to influenza B varied from 2 of 50 cases (4%) at Alice Springs Hospital to 27 of 47 cases (57%) at Geelong Hospital.

Figure 1: Date of admission for patients hospitalised with confirmed influenza



Source: FluCAN sentinel hospitals

Of these 631 patients, 200 (32%) were more than 65 years of age, 60 (9.5%) were Indigenous Australians, 27 (4.3%) were pregnant and 488 (77%) had chronic co-morbidities (Table 1). Of the 514 patients (81%) of patients where influenza vaccination status was ascertained, 199 (31%) had been vaccinated. Factors associated with admission with confirmed influenza, compared with admission with non-influenza controls are detailed in Table 2. As there was no community-based control group, factors associated with hospital admission could not be quantified, but it was noted that 77%

Table 1: Demographics, risk factors and outcomes in hospitalised patients with confirmed influenza

	2012		2013	
	Number	Proportion	Number	Proportion
Demographics				
Number of patients	1,231		631	
Age group				
<18 years	148	12.0	32	5.1
18–<40 years	229	18.6	139	22.0
40–<65 years	281	22.8	260	41.2
65–<80 years	307	24.9	131	20.8
≥80 years	266	21.6	69	10.9
Male	614	49.9	314	49.8
Indigenous	99	8.0	60	9.5
State or territory				
ACT	105	8.5	35	5.5
NSW	84	6.8	125	19.8
NT	83	6.7	50	7.9
Qld	167	13.6	29	4.6
SA	200	16.3	109	17.3
Tas.	99	8.0	30	4.8
Vic.	390	31.7	202	32.0
WA	103	8.4	51	8.1
Influenza strain				
H1N1/09	12	1.0	167	26.5
Flu A (unknown/seasonal)	1,006	81.7	277	43.9
Flu B	213	17.3	187	29.6
Risk factors				
Pregnant	39	3.2	27	4.3
Nursing home resident	68	5.5	18	2.8
Medical co-morbidities	944	76.7	488	77.3
Chronic respiratory disease	446	36.2	226	35.7
Diabetes	260	21.1	136	21.6
Chronic liver disease	38	3.1	38	6.0
Immunosuppressed	217	17.6	155	24.6
Chronic cardiac disease	353	28.7	183	29.0
Chronic neurological disease	175	14.2	94	14.8
Chronic renal disease	116	9.4	76	12.0
Severity and outcome				
Initial admitting ward				
General ward	1,123	91.2	561	88.6
High dependency or intensive care unit	108	8.8	69	10.9
In-hospital mortality	40/1,157	3.5	20/621	3.2

Table 2: Estimated vaccine coverage in influenza cases and test negative controls

	Confirmed influenza		Test negative acute respiratory illness	
	n/N	%	n/N	%
All patients	199/514	38.7	283/450	62.9
Age >65 years	100/157	63.7	178/221	80.5
Medical comorbidities	95/143	66.4	172/214	80.4
No medical comorbidities	5/14	35.7	6/7	85.7
Age <65 years	99/357	27.7	105/229	45.9
Medical comorbidities	86/252	34.1	92/187	49.2
No medical comorbidities	13/105	12.4	13/42	31.0

of patients had medical comorbidities. The most commonly reported co-morbidities were respiratory disease, cardiac disease, immunosuppression and diabetes.

Clinical course, severity and outcome

In 609 patients with confirmed influenza where the duration of symptoms was known, the median duration of symptoms prior to admission was 3 days (interquartile range (IQR) 2, 5 days). In patients with confirmed influenza, 347 (54%) received oseltamivir. Of these, 142 patients received oseltamivir within 48 hours of symptom onset. The duration of hospital stay was similar for patients that did not receive antivirals (median 4 days, IQR 2, 8 days), received antivirals within 48 hours of symptom onset (4 days, IQR 2, 8 days) and who received antivirals more than 48 hours after symptom onset (5 days, IQR 3, 10 days).

Of all cases, 69 (11%) were initially admitted to ICU and a further 33 patients were subsequently transferred to ICU after initial admission to a general ward. In a multivariate model stratified by hospital site, more than 65 years of age and pregnancy were negatively associated with ICU admission in hospitalised patients with confirmed influenza, while the presence of medical comorbidities was positively associated with ICU admission (Table 3). Indigenous patients were more likely to be admitted to ICU, but this difference was not statistically significant. In patients where influenza vaccination status was ascertained, influenza vaccination was negatively associated with ICU admission (odds ratio 0.61, 95% CI: 0.24, 1.12, n=490) but this difference was not statistically significant.

Of the 621 patients where hospital mortality status was documented, 20 patients died, of which 10 patients died in intensive care. Ten (50%) of these patients were more than 65 years of age, 19 (95%) had medical comorbidities and 3 (15%) were Indigenous Australians. Significant medical comorbidities in patients who died following admission with confirmed influenza were recorded as chronic cardiac disease (n=10), chronic respiratory disease (n=9), immunosuppression (n=8), diabetes (n=4) and renal disease (n=3).

Table 3: Factors associated with admission to intensive care in patients hospitalised with confirmed influenza

Variable	Odds ratio (95% CI)	*P* value
Age >65 years	0.49 (0.29, 0.84)	0.01
Medical comorbidities	1.89 (1.02, 3.50)	0.042
Pregnancy	0.20 (0.04, 0.89)	0.034
Indigenous Australian	2.05 (0.68, 6.19)	0.206
Influenza type		
Influenza A	1 (referent)	–
Influenza B	1.08 (0.66, 1.77)	0.747

Vaccine coverage and vaccine effectiveness

During this same period, 594 control patients were enrolled; vaccination status was ascertained in 450 (76%) control patients. In test negative controls during the season, vaccination coverage was estimated at 81% (178/221) and 66% (264/401) in the elderly and those with medical co-morbidities respectively (Table 2).

The effectiveness of the 2013 trivalent seasonal influenza vaccine in reducing the risk of hospitalisation with influenza was estimated at 50% (95% CI: 33%, 63%, $P<0.001$) in the 2013 influenza season (Table 4). Vaccine effectiveness was estimated to be lower in elderly patients and in those with medical co-morbidities (Figure 2).

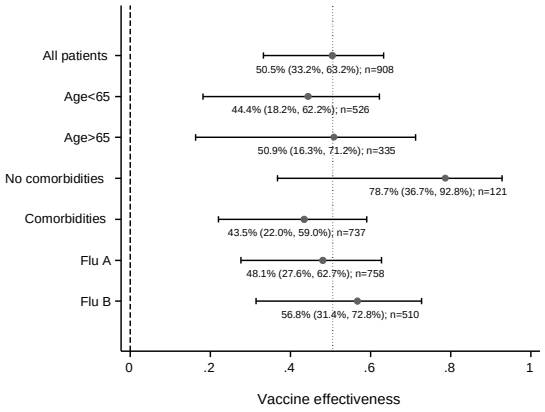
Discussion

In 2013, we recorded more than 600 admissions to the 14 participating hospitals, representing half those detected in 2012. Virological surveillance of circulating strains suggested that all 3 lineages

Table 4: Factors associated with hospitalisation with influenza compared with admission associated with non-influenza acute respiratory illnesses

Variable	Crude odds ratio	P	Adjusted odds ratio	P
Age ≥ 65 years	0.49 (0.38, 0.62)	<0.001	0.62 (0.45, 0.86)	0.003
Medical comorbidities	0.49 (0.36, 0.68)	<0.001	0.69 (0.46, 1.04)	0.076
Influenza vaccination	0.39 (0.30, 0.51)	<0.001	0.50 (0.37, 0.67)	<0.001
Pregnancy	3.02 (1.39, 6.58)	0.005	3.36 (1.09, 10.34)	0.035
Indigenous	1.08 (0.61, 1.90)	0.80	1.08 (0.50, 2.32)	0.84

Figure 2: Estimated vaccine effectiveness against hospitalisation for all patients, in specified subgroups and against infection with influenza subtypes



Numbers under bars represent point estimate (with 95% CI) and number of patients in analysis.

(A/H1N1/09, A/H3N2 and B/Yamagata-lineage) were detected in varying proportions in different states and territories, but vaccine match to circulating strains was good.[9] As the hospitals represented in this network represent approximately 12% of the national hospital bed capacity, the cases detected here are likely to represent approximately 5,400 admissions nationally. Compared with 2012, the 2013 season was later (peaking in September in 2013, compared with July in 2012), younger age groups were represented in a higher proportion of patients (<65 years: 68% vs 53%), and a higher proportion were due to influenza B (30% vs 17%). There was a similar number of patients in the 40–65 year age group but a decrease in all other age groups. It should be noted that the relative number of cases between jurisdictions does not reflect true influenza activity, due to differences in the number and size of sentinel hospitals in each jurisdiction.

The World Health Organization recommends surveillance for severe acute respiratory illness. Hospital-based surveillance programs similar to FluCAN are operating in many countries.[10–15] By providing information on influenza severity, hospital-based surveillance complements community– and primary care-based surveillance systems, which provide information on the extent of spread.

Influenza vaccine coverage has been estimated infrequently in hospitalised patients in Australia.[16] In 2012, we estimated vaccine coverage in 2 separate groups of patients: patients with pneumonia prior to the influenza season, and patients during the influenza season who had tested negative for influenza. The results in these groups were consistent with each other, and are similar to the vaccine coverage estimated in 2013. Self-reported vaccination status has been shown to slightly overestimate true influenza vaccination status.[16–18] Community-based estimates of influenza vaccine coverage, last reported in 2009, have shown similar results that have been consistent over time since 2002.[19]

The test negative study design to estimate vaccine effectiveness is becoming increasingly accepted and is ideally suited to being incorporated into surveillance systems. Two recent papers have examined the validity of this design. Foppa et al found that the estimates would be robust to most assumptions, but bias may be introduced by differences in health care-seeking behaviour amongst cases and non-cases by vaccine status, strong viral interference, or modification of the probability of symptomatic illness by vaccine status.[20] De Serres et al compared estimates from per protocol analyses of 4 randomised controlled trials of live attenuated influenza vaccine, with estimates based on the test negative design, and found minimal bias.[21] However, these studies have primarily considered vaccine effectiveness in the primary care setting.

Several studies, many of them embedded in surveillance systems, are able to provide regular estimates of vaccine effectiveness against hospitalisation. We previously reported vaccine effectiveness of 37%–48% in the 2010 and 2011 seasons, and of 41% in the 2012 season.[7,8] Estimates from other hospital-based studies have ranged widely from 30% to 71%, in part reflecting smaller sample

sizes than in community-based studies.[22–25] These results reinforce previous findings that vaccination coverage in non-elderly patients with comorbidities is relatively low. Further work is required to explore reasons for poor vaccination uptake, whether related to poor awareness in patients or healthcare providers.

There are several limitations to this study. Incomplete case ascertainment is likely due to the lack of use of influenza laboratory tests, despite the diagnosis of influenza having implications for infection control and antiviral use in hospitals. Delayed presentations or secondary bacterial pneumonia may be associated with false negative influenza tests as the influenza infection may be cleared at the time of presentation. There may also be unmeasured confounding of the association between vaccination and admission with influenza, a bias that has plagued studies of influenza mortality.[26] Although previous studies have suggested that self-reported influenza vaccination status only slightly overestimates vaccination coverage, we have not validated this in our population.[16–18]

In summary, we detected a smaller number of hospital admissions with confirmed influenza in a national observational study in 2013 compared with 2012. Vaccine coverage was incomplete in at-risk groups, particularly non-elderly patients with medical comorbidities. Our results suggest that the 2013 seasonal influenza vaccine was moderately protective against hospitalisation with influenza.

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Quarterly report

National Notifiable Diseases Surveillance System, 1 January to 31 March 2014

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 57,805 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 January to 31 March 2014 (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:
Bloodborne diseases	
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
Gastrointestinal diseases	
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
STEC, VTEC*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	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
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis - congenital	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions

Table 1 *continued*: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	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
Vectorborne diseases	
Arbovirus infection (NEC)	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	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
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (STEC/VTEC).

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2014, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2014	Total 4th quarter 2013	Total 1st quarter 2013	Last 5 years mean 1st quarter	Ratio	Year to date 2014	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
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	11	1	17	4	1	16	7	57	40	47	55.8	1.0	57	55.8
Hepatitis B (unspecified)‡	24	668	43	235	82	17	399	162	1,630	1,675	1,590	1,705.6	1.0	1,630	1,705.6
Hepatitis C (newly acquired)†	2	8	1	0	12	3	30	39	95	109	117	110.2	0.9	95	110.2
Hepatitis C (unspecified)‡	43	885	48	649	82	61	497	226	2,491	2,532	2,432	2,614.8	1.0	2,491	2,614.8
Hepatitis D	0	1	0	5	0	0	4	0	10	13	15	9.8	1.0	10	9.8
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	2	0.6	0.0	0	0.6
Campylobacteriosis	139	NN	75	1,380	360	253	1,953	606	4,766	4,400	3,481	4,303.0	1.1	4,766	4,303.0
Cryptosporidiosis	22	145	34	271	71	5	195	113	856	567	1,727	1,439.8	0.6	856	1,439.8
Haemolytic uraemic syndrome	0	3	0	1	2	1	2	0	9	6	4	4.4	2.0	9	4.4
Hepatitis A	2	27	1	16	5	0	26	8	85	33	69	65.4	1.3	85	65.4
Hepatitis E	0	6	0	1	0	0	2	0	9	8	15	14.4	0.6	9	14.4
Listeriosis	0	7	0	2	1	0	9	3	22	13	29	27.2	0.8	22	27.2
STEC, VTEC§	0	17	0	4	15	0	4	1	41	29	43	35.8	1.1	41	35.8
Salmonellosis	58	1,505	110	1,565	339	104	1,171	345	5,197	3,307	4,130	4,051.6	1.3	5,197	4,051.6
Shigellosis	10	77	23	45	11	0	125	25	316	161	144	174.4	1.8	316	174.4
Typhoid	0	14	0	11	3	0	13	3	44	35	68	49.6	0.9	44	49.6
Quarantinable diseases															
Cholera	0	0	0	0	0	0	1	0	1	2	0	0.6	1.7	1	0.6
Highly pathogenic avian influenza in humans	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

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2014, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2014	Total 4th quarter 2013	Total 1st quarter 2013	Last 5 years mean 1st quarter	Ratio	Year to date 2014	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection[‖,¶]	297	6,136	717	5,440	1,372	440	5,064	3,032	22,498	19,565	21,033	19,803.0	1.1	22,498	19,803.0
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Gonococcal infection[¶]	50	1,240	450	763	215	19	903	595	4,235	3,486	3,837	3,025.4	1.4	4,235	3,025.4
Syphilis – congenital	0	0	0	0	0	0	0	0	0	1	1	1.4	0.0	0	1.4
Syphilis < 2 years duration[¶]	5	153	6	96	6	1	174	18	459	425	447	362.2	1.3	459	362.2
Syphilis > 2 years or unspecified duration[‡,¶]	8	88	14	87	36	7	178	18	436	375	386	338.6	1.3	436	338.6
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	1	1	0.2	0.0	0	0.2
Haemophilus influenzae type b	0	0	1	2	0	0	0	0	3	4	3	3.8	0.8	3	3.8
Influenza (laboratory confirmed)	47	721	247	1,321	487	50	568	380	3,821	6,127	2,331	1,441.2	2.7	3,821	1,441.2
Measles	1	49	47	15	10	0	34	17	173	85	10	38.0	4.6	173	38.0
Mumps	0	30	1	16	4	2	4	6	63	40	72	45.0	1.4	63	45.0
Pertussis	46	482	17	542	110	32	750	348	2,327	3,071	3,620	7,168.8	0.3	2,327	7,168.8
Pneumococcal disease (invasive)	3	69	11	29	18	3	51	29	213	324	212	215.8	1.0	213	215.8
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	3	0	1	1	0	0	1	6	5	1	12.2	0.5	6	12.2
Rubella – congenital	0	0	0	0	0	0	0	0	0	1	0	0.0	0.0	0	0.0
Tetanus	0	1	0	0	0	0	0	0	1	0	3	1.8	0.6	1	1.8
Varicella zoster (chickenpox)	15	NN	36	44	73	5	211	84	468	648	374	372.6	1.3	468	372.6
Varicella zoster (shingles)	10	NN	57	16	501	67	378	350	1,379	1,348	1,206	968.0	1.4	1,379	968.0
Varicella zoster (unspecified)	50	NN	1	1,500	55	41	816	315	2,778	2,686	2,250	1,913.8	1.5	2,778	1,913.8
Vectorborne diseases															
Arbovirus infection (NEC)	0	2	0	13	0	0	0	0	15	4	5	3.4	4.4	15	3.4
Barmah Forest virus infection	0	57	7	244	0	0	2	21	331	442	1,423	746.4	0.4	331	746.4
Dengue virus infection	4	136	26	217	17	5	92	156	653	315	495	544.8	1.2	653	544.8
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Kunjin virus infection**	0	0	0	1	0	0	0	0	1	3	0	0.6	1.7	1	0.6
Malaria	2	28	3	24	2	0	19	13	91	82	138	110.8	0.8	91	110.8
Murray Valley encephalitis virus infection**	0	0	0	0	0	0	0	0	0	0	0	2.2	0.0	0	2.2
Ross River virus infection	0	113	188	448	18	12	70	776	1,625	939	1,352	1,987.6	0.8	1,625	1,987.6

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2014, by date of diagnosis

Disease	State or territory								Total 1st quarter 2014	Total 4th quarter 2013	Total 1st quarter 2013	Last 5 years mean 1st quarter	Ratio	Year to date 2014	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	1	0.2	0.0	0	0.2
Brucellosis	0	0	0	5	0	0	1	0	6	4	5	7.2	0.8	6	7.2
Leptospirosis	0	4	1	21	0	0	0	0	26	22	15	54.2	0.5	26	54.2
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	4	0	1	0	0	4	2	11	15	7	14.0	0.8	11	14.0
Q fever	0	38	1	79	2	0	6	2	128	119	103	92.8	1.4	128	92.8
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.3	0.0	0	0.3
Other bacterial infections															
Legionellosis	0	19	4	14	11	1	15	23	87	118	98	81.4	1.1	87	81.4
Leprosy	0	1	0	0	0	0	0	2	3	4	2	1.2	2.5	3	1.2
Meningococcal infection††	0	4	2	9	4	0	4	3	26	32	39	44.4	0.6	26	44.4
Tuberculosis	7	105	5	46	11	2	101	36	313	312	315	317.8	1.0	313	317.8
Total	845	12,857	2,178	15,196	3,940	1,132	13,892	7,765	57,805	53,533	53,698			57,805	

* 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. From 1 July 2013 case definition changed to exclude all ocular infections.

¶ The national case definitions for chlamydial, 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).

** In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

†† 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 January to 31 March 2014, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
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	0.6	1.7	1.5	1.0	0.8	1.1	1.1	1.0
Hepatitis B (unspecified)§	25.8	37.0	73.1	20.7	20.1	13.6	28.5	26.4	28.9
Hepatitis C (newly acquired)‡	2.1	0.4	1.7	0.0	2.9	2.4	2.1	6.3	1.7
Hepatitis C (unspecified)§	46.2	49.0	81.6	57.2	20.1	48.7	35.5	36.8	44.1
Hepatitis D	0.0	0.1	0.0	0.4	0.0	0.0	0.3	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	149.4	NN	127.5	121.5	88.3	202.2	139.5	98.6	124.3
Cryptosporidiosis	23.6	8.0	57.8	23.9	17.4	4.0	13.9	18.4	15.2
Haemolytic uraemic syndrome	0.0	0.2	0.0	0.1	0.5	0.8	0.1	0.0	0.2
Hepatitis A	2.1	1.5	1.7	1.4	1.2	0.0	1.9	1.3	1.5
Hepatitis E	0.0	0.3	0.0	0.1	0.0	0.0	0.1	0.0	0.2
Listeriosis	0.0	0.4	0.0	0.2	0.2	0.0	0.6	0.5	0.4
STEC,VTEC‖	0.0	0.9	0.0	0.4	3.7	0.0	0.3	0.2	0.7
Salmonellosis	62.3	83.3	187.0	137.8	83.2	83.1	83.7	56.1	92.1
Shigellosis	10.7	4.3	39.1	4.0	2.7	0.0	8.9	4.1	5.6
Typhoid fever	0.0	0.8	0.0	1.0	0.7	0.0	0.9	0.5	0.8
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Human pathogenic avian influenza in humans	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
Sexually transmitted infections									
Chlamydial infection¶,**	319.2	339.5	1,218.8	479.1	336.7	351.6	361.8	493.2	398.7
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	53.7	68.6	764.9	67.2	52.8	15.2	64.5	96.8	75.1
Syphilis – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years duration**	5.4	8.5	10.2	8.5	1.5	0.8	12.4	2.9	8.1
Syphilis > 2 years or unspecified duration§,**	8.6	4.9	23.8	7.7	8.8	5.6	12.7	2.9	7.7
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	1.7	0.2	0.0	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)	50.5	39.9	419.9	116.3	119.5	40.0	40.6	61.8	67.7
Measles	1.1	2.7	79.9	1.3	2.5	0.0	2.4	2.8	3.1
Mumps	0.0	1.7	1.7	1.4	1.0	1.6	0.3	1.0	1.1
Pertussis	49.4	26.7	28.9	47.7	27.0	25.6	53.6	56.6	41.2
Pneumococcal disease (invasive)	3.2	3.8	18.7	2.6	4.4	2.4	3.6	4.7	3.8
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.2	0.0	0.1	0.2	0.0	0.0	0.2	0.1
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 *continued*: Notification rates of diseases, 1 January to 31 March 2014, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Varicella zoster (chickenpox)	16.1	NN	61.2	3.9	17.9	4.0	15.1	13.7	12.2
Varicella zoster (shingles)	10.7	NN	96.9	1.4	122.9	53.5	27.0	56.9	36.0
Varicella zoster (unspecified)	53.7	NN	1.7	132.1	13.5	32.8	58.3	51.2	72.4
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.1	0.0	1.1	0.0	0.0	0.0	0.0	0.3
Barmah Forest virus infection	0.0	3.2	11.9	21.5	0.0	0.0	0.1	3.4	5.9
Dengue virus infection	4.3	7.5	44.2	19.1	4.2	4.0	6.6	25.4	11.6
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	0.1	0.0	0.0	0.0	0.0	0.0
Malaria	2.1	1.5	5.1	2.1	0.5	0.0	1.4	2.1	1.6
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	0.0	6.3	319.6	39.5	4.4	9.6	5.0	126.2	28.8
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.4	0.0	0.0	0.1	0.0	0.1
Leptospirosis	0.0	0.2	1.7	1.8	0.0	0.0	0.0	0.0	0.5
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.2	0.0	0.1	0.0	0.0	0.3	0.3	0.2
Q fever	0.0	2.1	1.7	7.0	0.5	0.0	0.4	0.3	2.3
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	0.0	1.1	6.8	1.2	2.7	0.8	1.1	3.7	1.5
Leprosy	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.3	0.1
Meningococcal infection‡‡	0.0	0.2	3.4	0.8	1.0	0.0	0.3	0.5	0.5
Tuberculosis	7.5	5.8	8.5	4.1	2.7	1.6	7.2	5.9	5.5

* 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. From 1 July 2013 case definition changed to exclude all ocular infections.

** The national case definitions for chlamydial, 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).

†† In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

‡‡ 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 July to 30 September cohort, assessed as at 31 December 2013

Brynley P Hull for the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

Introduction

Tables 1, 2 and 3 provide the latest quarterly report on childhood immunisation coverage from the Australian Childhood Immunisation Register (ACIR).

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months, for 3-month birth cohorts of children at the stated ages between July and September 2013 using ACIR data as at 31 December 2013. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, varicella, and meningococcal C conjugate vaccines, 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 PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of all other hepatitis B vaccines 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 or 4 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP Hib vaccine or 4 doses of any other Hib vaccine, 3 or 4 doses of Comvax hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 or 5 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.

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 1435, email: brynley.hull@health.nsw.gov.au

Results

The percentage of children 'fully immunised' by 12 months of age for Australia decreased from the previous quarter by 0.8 of a percentage point to 90.1% (Table 1). This decrease is likely due to the recent inclusion of 13-valent pneumococcal conjugate vaccine in the coverage calculation algorithm for 'fully immunised' at 12 months of age. Except for the Australian Capital Territory, all jurisdictions experienced decreases in coverage for all individual vaccines due at 12 months of age, ranging from 0.1 of a percentage point to 2.4 percentage points.

Table 1. Percentage of children immunised at 12 months of age for the birth cohort 1 July to 30 September 2012, preliminary results, by disease and state or territory; assessment date 31 December 2013

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Number of children	1,451	25,558	961	16,036	5,171	1,498	19,142	8,441	78,258
Diphtheria, tetanus, pertussis (%)	94.1	90.4	90.6	91.8	90.6	90.2	91.6	90.8	91.1
Poliomyelitis (%)	94.1	90.2	90.5	91.7	90.4	89.9	91.5	90.8	91.0
Haemophilus influenzae type b (%)	94.1	90.1	90.6	91.6	90.4	89.5	91.2	90.5	90.8
Hepatitis B (%)	93.8	89.8	90.3	91.4	90.1	89.6	91.1	90.2	90.6
Pneumococcal	93.8	90.2	90.4	91.4	90.4	90.3	91.2	90.2	90.8
Fully immunised (%)	93.3	89.5	90.0	91.1	89.7	89.2	90.5	89.5	90.1
Change in fully immunised since last quarter (%)	+0.6	-0.5	-2.4	-0.4	-1.5	-2.4	-0.8	-1.2	-0.8

The percentage of children 'fully immunised' by 24 months of age for Australia decreased marginally from the previous quarter by 0.1 of a percentage point to 92.3% (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

The percentage of children 'fully immunised' by 60 months of age for Australia decreased marginally from the previous quarter by 0.3 of a percentage point to 92.1% (Table 3). There were no important changes in coverage for any individual vaccines due at 60 months of age or by jurisdiction.

The Figure shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. 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 at 24 months is still higher than coverage at 12 months of age.

Figure: Trends in vaccination coverage, Australia, 1997 to 30 September 2013, by age cohorts

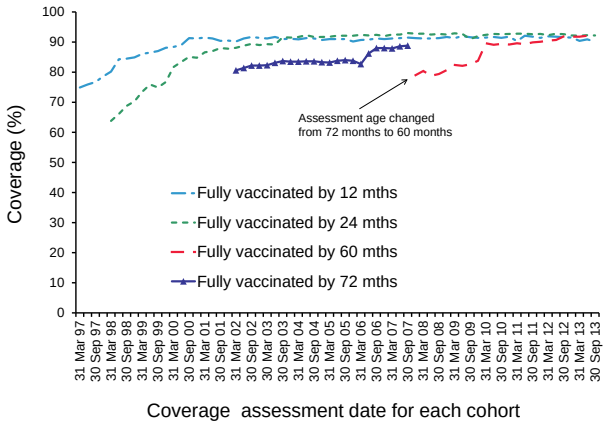

Table 2. Percentage of children immunised at 24 months of age for the birth cohort 1 July to 30 September 2011, preliminary results, by disease and state or territory; assessment date 31 December 2013*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,377	24,948	944	15,473	5,026	1,527	18,730	8,407	76,432
Diphtheria, tetanus, pertussis (%)	95.4	94.5	94.4	94.8	94.9	95.9	95.5	93.1	94.7
Poliomyelitis (%)	95.4	94.4	94.3	94.8	94.9	95.9	95.4	93.1	94.7
Haemophilus influenzae type b (%)	95.9	95.0	95.1	94.9	95.2	96.1	95.6	93.4	95.0
Measles, mumps, rubella (%)	94.6	93.2	93.8	94.0	93.8	94.9	94.3	92.2	93.6
Hepatitis B (%)	95.2	94.1	94.0	94.5	94.6	95.5	95.0	92.4	94.3
Fully immunised (%)	93.6	91.8	92.5	92.8	92.3	93.6	92.9	90.1	92.2
Change in fully immunised since last quarter (%)	+1.5	0.0	-2.4	-0.1	0.0	0.0	0.0	-1.0	-0.1

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2013;37(1):E89.

Table 3. Percentage of children immunised at 60 months of age for the birth cohort 1 July to 30 September 2008, preliminary results, by disease and state or territory; assessment date 31 December 2013

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,360	25,797	897	16,644	5,265	1,660	19,345	8,547	79,515
Diphtheria, tetanus, pertussis (%)	91.5	92.6	92.9	92.3	91.2	93.6	92.9	90.3	92.3
Poliomyelitis (%)	91.4	92.5	92.9	92.3	91.1	93.3	92.9	90.2	92.2
Measles, mumps, rubella (%)	91.1	92.4	92.8	92.4	91.3	93.6	92.8	90.1	92.2
Fully immunised (%)	90.9	92.1	92.2	91.9	90.8	92.8	92.4	89.5	91.8
Change in fully immunised since last quarter (%)	-2.1	-0.2	+1.8	-0.2	-1.1	-0.4	-0.4	-0.8	-0.3

Australian Gonococcal Surveillance Programme, 1 January to 31 March 2014

Monica M Lahra for the Australian Gonococcal Surveillance Programme

Introduction

The Australian National Neisseria Network (NNN), which comprises reference laboratories in each state and territory, report data quarterly on sensitivity to an agreed group of antimicrobial agents for the Australian Gonococcal Surveillance Programme (AGSP). The antibiotics routinely tested and reported each quarter are penicillin, ceftriaxone, azithromycin and ciprofloxacin, which are current or potential agents used for the treatment of gonorrhoea. Azithromycin testing has been recently introduced by all states and territories as it has a role as part of a dual therapy regimen with ceftriaxone in the treatment of gonorrhoea in the majority of Australia. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. In certain remote regions of the Northern Territory and Western Australia antimicrobial resistance rates are low and an oral treatment regimen comprising amoxycillin, probenecid and azithromycin is recommended for use. For this reason, for the Northern Territory, data from Darwin are presented separately as Northern Territory – urban, and Northern Territory rural and remote for the rest of the Northern Territory. In Western Australia, data from regions classified as remote (Kimberley, Pilbara and Goldfields), are separated from urban and rural data. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatments.[1] Additional data are also provided on other antibiotics from time to time. These data are reported in the AGSP annual report. The AGSP has a program-specific quality assurance process. The AGSP data are presented quarterly in tabulated form (Table 1), as well as in the AGSP annual report.

Results

A summary of the proportion of isolates with decreased susceptibility to ceftriaxone, and proportion resistant to penicillin, ciprofloxacin and azithromycin are shown in Table 1.

Penicillin resistant *Neisseria gonorrhoeae* are defined as those isolates with a minimum inhibitory concentration (MIC) to penicillin equal to or greater than 1.0 mg/L. Total penicillin resistance includes penicillinase producing *N. gonorrhoeae* (PPNG) and *N. gonorrhoeae* that have chromosom-

Table 1: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to ciprofloxacin, azithromycin and penicillin, Australia, 1 January to 31 March 2014, by state or territory

State or territory	Number of isolates tested	Decreased susceptibility		Resistance					
		Ceftriaxone		Ciprofloxacin		Azithromycin		Penicillin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	31	2	6.5	18	58.0	3	9.7	2	6.5
New South Wales	466	41	8.8	208	45.0	13	2.8	234	50.0
Queensland	166	11	6.6	45	27.0	4	2.4	32	19.0
South Australia	76	2	2.6	31	41.0	1	1.3	12	16.0
Tasmania	13	0	0.0	3	23.0	1	7.7	4	31.0
Victoria	446	37	8.3	191	43.0	3	0.7	100	22.0
Northern Territory/urban	28	1	3.6	6	21.0	0	0.0	5	18.0
Northern Territory/rural and remote	40	0	0.0	3	7.5	0	0.0	2	5.0
Western Australia/urban and rural	106	1	0.9	38	35.8	4	3.8	35	33.0
Western Australia/remote	37	1	2.7	4	11.0	0	0.0	4	11.0
Australia	1,409	96	6.8	547	39.0	29	2.1	430	31.0

ally mediated resistance to penicillin (CMRP). In areas classified as remote, in the Northern Territory and Western Australia, a treatment regimen based on oral amoxycillin, probenecid and azithromycin is used. Penicillin resistance in remote Northern Territory was reported in 2 of 40 strains tested and 4 of 37 strains tested from remote areas of Western Australia. A low number of cultures are collected in these remote regions, due in part to increasing use of nucleic acid amplification testing (NAAT). In Western Australia, the introduction of a targeted NAAT, developed by the NNN to detect PPNG, is in use to enhance surveillance.[2,3]

Ciprofloxacin resistance includes isolates with a MIC to ciprofloxacin equal to or greater than 0.06 mg/L.

Azithromycin resistance is defined as a MIC to azithromycin equal to or greater than 1.0 mg/L. In 2013, gonococcal strains with azithromycin high level resistance were reported from Victoria and Queensland.[4] There were no isolates reported in Australia with high level resistance with an azithromycin (MIC value >256 mg/L) in this quarter, 2014.

Ceftriaxone MIC values in the range 0.06–0.125 mg/L have been reported in the category decreased susceptibility since 2005. To date there has not been an isolate reported in Australia with a ceftriaxone MIC value >0.125 mg/L.

In the 1st quarter of 2014 there was a decrease in the proportion of *N. gonorrhoeae* isolates with decreased susceptibility to ceftriaxone, predominantly from New South Wales and Victoria, when compared with the same quarter in 2013; and in the annual data for 2013.[4] When compared with the 1st quarter of 2013, there was a decrease from 9.7% to 6.8% in the proportion of *N. gonorrhoeae* isolates with decreased susceptibility to ceftriaxone nationally, but this is more than double that reported in the 3rd quarters of 2011 and 2012 (2.7%–3.5%).

The highest proportions of isolates with decreased susceptibility to ceftriaxone were reported from the eastern states: Victoria, New South Wales and Queensland. In New South Wales there were 41 strains with decreased susceptibility to ceftriaxone and of those, 40/41 (98%) were multi-drug-resistant (MDR); 33/41 (80%) were from males; and 13/41 (32%) were isolated from extra genital sites (rectal and pharyngeal). In Victoria, there were 37 strains with decreased susceptibility to ceftriaxone and, of those, 31/37 (83%) were MDR strains, 37/37 (100%) were from males, and 23/37 (62%) were isolated from extra genital sites (rectal and pharyngeal). In contrast, there were no gonococci with decreased susceptibility to ceftriaxone reported from the remote Northern Territory, or Tasmania and low numbers were reported from Western Australia.

The proportion of strains with decreased susceptibility to ceftriaxone is of increasing concern in Australia and overseas, as this is phenotypic of the genotype with the key mutations that are the precursor to ceftriaxone resistance.[5] There are recent reports of ceftriaxone 500 mg treatment failures from Victoria and New South Wales. These patients had pharyngeal infections where the gonococcal strains had ceftriaxone MIC values in the range 0.03–0.06 mg/L.[6,7] Until 2014 there had not been an isolate reported in Australia with a ceftriaxone MIC value >0.125 mg/L.[4] In late December 2013 there was a new MDR gonococcal strain with a ceftriaxone MIC of 0.5 mg/L, the highest ever reported in Australia (unpublished data from the NNN). To date there has been no evidence of spread of this strain in the 1st quarter of 2014.

The category of ceftriaxone decreased susceptibility includes the MIC values 0.06 and 0.125 mg/L. The right shift in the distribution of ceftriaxone MIC values over recent years (Table 2), is statistically significant with a sustained increase in the proportion of strains with an MIC value of 0.06 mg/L (2011–2012: [P=0.02, 95% CI: 1.04 –1.62], and 2012–2013 [P<0.0001, 95% CI: 1.70–2.38]). In 2010, the proportion of strains with ceftriaxone decreased susceptibility was higher than that reported in 2011. This proportion has subsequently increased as described. The proportion of strains with a ceftriaxone MIC 0.125 mg/L has also increased from 0.1% in 2010 and 2011, to 0.3% in 2012 to 0.6% in 2013. These differences were not significant which may be attributable to the low number of strains in this MIC category.[4] In the 1st quarter of 2014 there were lower proportions of strains at both 0.06 and 0.125 mg/L.

Table 2: Percentage of gonococcal isolates with decreased susceptibility to ceftriaxone MIC 0.06–0.125 mg/L, Australia, 2010 to 2013, and 1 January to 31 March 2014, by state or territory

Ceftriaxone MIC mg/L	2010	2011	2012	2013	1 January to 31 March 2014
0.06	4.6	3.2	4.1	8.2	6.4
0.125	0.1	0.1	0.3	0.6	0.4

In response to concerns over the increasing proportions of *N. gonorrhoeae* strains with decreased susceptibility to ceftriaxone, dual therapy (ceftriaxone plus azithromycin) is recommended as a strategy to temper development of more widespread resistance.[8] Patients with infections in extra genital sites, where the isolate has decreased susceptibility to ceftriaxone, are recommended to have a test of cure cultures collected.

Continued surveillance to monitor *N. gonorrhoeae* with elevated MIC values coupled with sentinel site surveillance in high risk populations remains critically important to inform our therapeutic strategies and to detect instances of treatment failure.

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Australian Meningococcal Surveillance Programme quarterly report, 1 January to 31 March 2014

Monica M Lahra for the Australian Meningococcal Surveillance Programme

Introduction

The reference laboratories of the Australian Meningococcal Surveillance Programme 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 annual reports of the programme published in *Communicable Diseases Intelligence*. For more information see *Commun Dis Intell* 2014;38(1):E97.

Results

Laboratory confirmed cases of invasive meningococcal disease for the period 1 January to 31 March 2014 are shown in the Table.

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2014, 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	2014	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2013	0	0	0	0	0	0	0	0	0	0	0	0	0	0
New South Wales	2014	0	0	3	3	0	0	0	0	1	1	0	0	4	4
	2013	0	0	4	4	2	2	0	0	0	0	0	0	6	6
Northern Territory	2014	0	0	2	2	0	0	0	0	0	0	0	0	2	2
	2013	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Queensland	2014	0	0	7	7	0	0	0	0	1	1	0	0	8	8
	2013	0	0	7	7	0	0	1	1	0	0	0	0	8	8
South Australia	2014	0	0	5	5	0	0	0	0	0	0	0	0	5	5
	2013	0	0	4	4	0	0	0	0	1	1	0	0	5	5
Tasmania	2014	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2013	0	0	1	1	0	0	0	0	0	0	0	0	1	1
Victoria	2014	0	0	3	3	0	0	0	0	1	1	0	0	4	4
	2013	0	0	6	6	1	1	0	0	0	0	0	0	7	7
Western Australia	2014	0	0	1	1	2	2	0	0	0	0	0	0	3	3
	2013	0	0	7	7	0	0	0	0	0	0	0	0	7	7
Total	2014	0	0	21	21	2	2	0	0	3	3	0	0	26	26
	2013	0	0	29	29	3	3	1	1	1	1	0	0	34	34

Australian Sentinel Practices Research Network, 1 January to 31 March 2013

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 who report 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 alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Electronic, web-based data collection was established in 2006.

Since 2010, ASPREN GPs have been collecting nasal swab samples for laboratory testing, allowing for viral testing of 25% of patients with influenza-like illness (ILI) for a range of respiratory viruses including influenza A and B.

The list of conditions reported is reviewed annually by the ASPREN management committee. In 2013, four conditions were being monitored. They included 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* 2013;37(1):62.

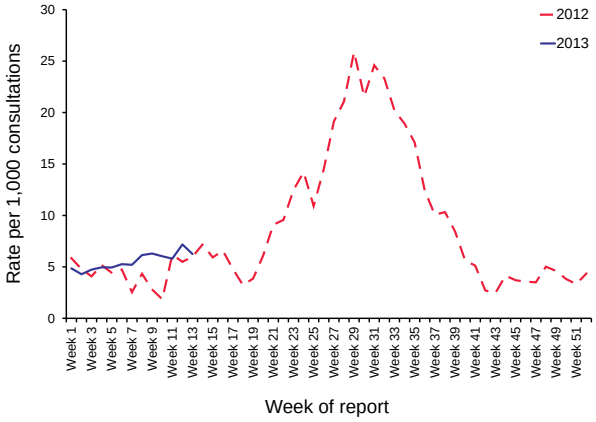
Results

Sentinel practices contributing to ASPREN were located in all 8 states and territories in Australia. A total of 247 general practitioners contributed data to ASPREN in the 1st quarter of 2013. Each week an average of 220 general practitioners provided information to ASPREN at an average of 20,996 (range 14,216–23,505) consultations per week and an average of 288 (range 183–350) notifications per week.

ILI rates reported from 1 January to 31 March 2013 averaged 6 cases per 1,000 consultations (range 4–7 cases per 1,000 consultations). This was higher compared with rates during the same reporting period in 2012, which averaged 4 cases per 1,000 consultations (range 2–6 cases per 1,000 consultations, Figure 1).

The 2013 ILI data is weighted by state to avoid over or under-representation of states in the calculation of the national notification incidence. Weekly observations within each state were weighted according to population estimates from the 2012 census.

Figure 1: Consultation rates for influenza-like illness, ASPREN, 2012 and 1 January to 31 March 2013, by week of report



ILI swab testing continued in 2013. The most commonly reported virus during this reporting period was rhinovirus (19% of all swabs performed, Figure 2), with the second most common virus being influenza A (untyped) (11% of all swabs performed).

From the beginning of 2013 to the end of week 13, 34 cases of influenza were detected comprising of influenza A (untyped) (11% of all swabs performed) and influenza B (3% of all swabs performed) (Figure 2).

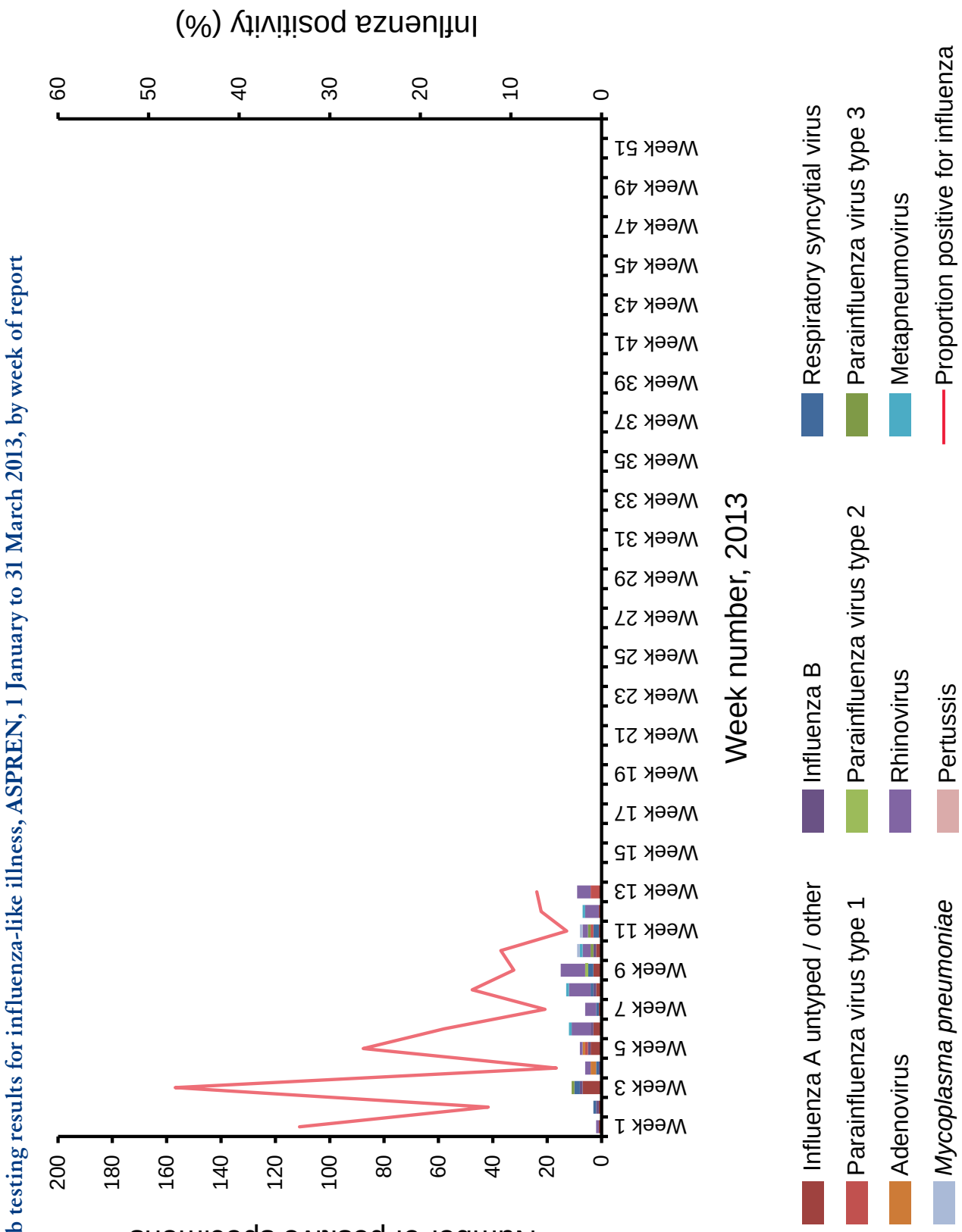
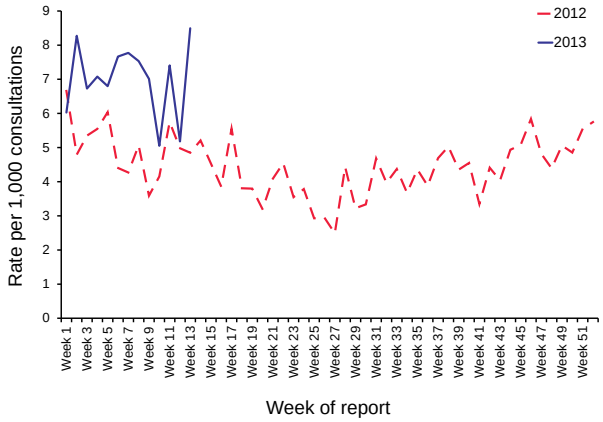


Figure 2: Swab testing results for influenza-like illness, ASPREN, 1 January to 31 March 2013, by week of report

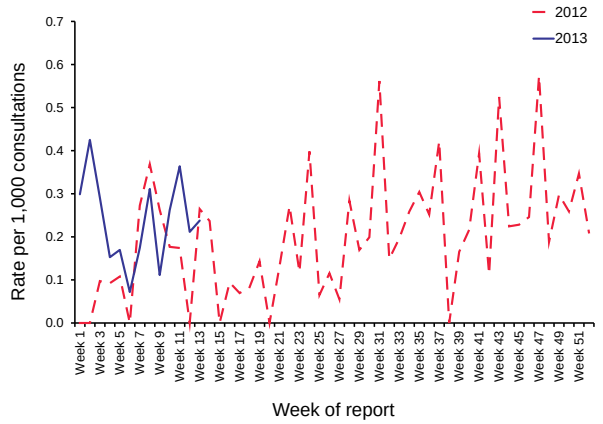
During this reporting period, consultation rates for gastroenteritis averaged 7 cases per 1,000 consultations (range 5–8 cases per 1,000 consultations, Figure 3). This was higher compared with rates during the same reporting period in 2012 where the average was 5 cases per 1,000 consultations (range 4–7 cases per 1,000 consultations).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 2012 and 1 January to 31 March 2013, by week of report



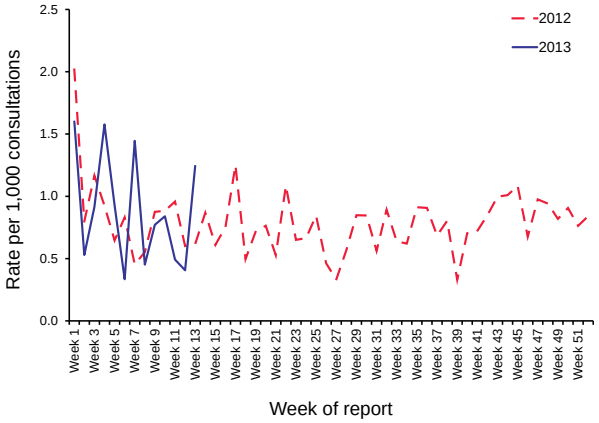
Varicella infections were reported at a higher rate for the 1st quarter of 2013 compared with the same period in 2012. From 1 January to 31 March 2013, recorded rates for chickenpox averaged 0.24 cases per 1,000 consultations (range 0.07–0.42 cases per 1,000 consultations, Figure 4).

Figure 4: Consultation rates for chickenpox, ASPREN, 2012 and 1 January to 31 March 2013, by week of report



In the 1st quarter of 2013, reported rates for shingles averaged 0.89 cases per 1,000 consultations (range 0.33–1.60 cases per 1,000 consultations, Figure 5). This was slightly higher compared with the same reporting period in 2012 where the average shingles rate was 0.87 case per 1,000 consultations (range 0.55–2.02 cases per 1,000 consultations).

Figure 5: Consultation rates for shingles, ASPREN, 2012 and 1 January to 31 March 2013, by week of report



HIV surveillance, 1 April to 30 June 2013

The Kirby Institute

Introduction

National surveillance for HIV infection is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Diagnoses of HIV infection are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of newly diagnosed HIV infections are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection is published in the quarterly Australian HIV Surveillance Report, and annually in the *HIV, Viral Hepatitis and Sexually Transmissible Infections in Australia, Annual Surveillance Report*. The reports are available from the Kirby Institute, University of New South Wales, SYDNEY NSW 2052. Internet: www.kirby.unsw.edu.au Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see *Commun Dis Intell* 2014;38(1):E96–E97.

Results

Newly diagnosed cases of HIV infection reported for 1 April to 30 June 2013, are shown in Tables 1 and 2).

Table 1: Number of new diagnoses of HIV infection, 1 April to 30 June 2013, by sex and state or territory of diagnosis

Sex	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total 2nd qrt 2013	Total 2nd qrt 2012	YTD 2013	YTD 2012
Female	2	9	3	8	5	0	6	9	42	40	82	84
Male	6	106	2	43	9	0	92	18	276	229	529	525
Not reported	0	0	0	0	0	0	0	0	0	0	0	0
Total*	8	115	5	51	14	0	98	27	318	270	611	610

* Totals include people whose sex was reported as transgender.

Table 2: Cumulative number of new diagnoses of HIV infection since the introduction of HIV antibody testing, 1985 to 30 June 2013, by sex and state or territory

Sex	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Female	44	1,148	43	475	167	27	584	375	2,863
Male	343	16,629	197	4,005	1,231	173	7,182	1,705	31,465
Not reported	0	227	0	0	0	0	22	0	249
Total*	387	18,044	240	4,489	1,399	200	7,816	2,087	34,662

* Totals include people whose sex was reported as transgender.

HIV surveillance, 1 July to 30 September 2013

The Kirby Institute

Introduction

National surveillance for HIV infection is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Diagnoses of HIV infection are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

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Results

Newly diagnosed cases of HIV infection reported for 1 July to 30 September 2013, are shown in Tables 1 and 2).

Table 1: Number of new diagnoses of HIV infection, 1 July to 30 September 2013, by sex and state or territory of diagnosis

Sex	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total 3rd qrt 2013	Total 3rd qrt 2012	YTD 2013	YTD 2012
Female	0	7	2	7	2	0	8	4	30	42	112	126
Male	5	91	6	57	13	2	92	22	288	285	817	810
Not reported	0	0	0	0	0	0	0	0	0	0	0	0
Total*	5	99	8	64	15	2	100	26	319	327	930	937

* Totals include people whose sex was reported as transgender.

Table 2: Cumulative number of new diagnoses of HIV infection since the introduction of HIV antibody testing, 1985 to 30 September 2013, by sex and state or territory

Sex	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Female	44	1,155	45	482	169	27	592	379	2,893
Male	348	16,720	203	4,062	1,244	175	7,274	1,727	31,753
Not reported	0	227	0	0	0	0	22	0	249
Total*	392	18,143	248	4,553	1,414	202	7,916	2,113	34,981

* Totals include people whose sex was reported as transgender.

HIV surveillance, 1 October to 31 December 2013

The Kirby Institute

Introduction

National surveillance for HIV infection is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health. Cases of HIV infection are notified to the National HIV Registry on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). Diagnoses of HIV infection are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of newly diagnosed HIV infections are based on data available 3 months after the end of the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection is published in the quarterly Australian HIV Surveillance Report, and annually in the *HIV, Viral Hepatitis and Sexually Transmissible Infections in Australia, Annual Surveillance Report*. The reports are available from the Kirby Institute, University of New South Wales, SYDNEY NSW 2052. Internet: www.kirby.unsw.edu.au Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see *Commun Dis Intell* 2014;38(1):E96–E97.

Results

Newly diagnosed cases of HIV infection reported for 1 October to 31 December 2013, are shown in Tables 1 and 2).

Table 1: Number of new diagnoses of HIV infection, 1 October to 31 December 2013, by sex and state or territory of diagnosis

Sex	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total 4th qrt 2013	Total 4th qrt 2012	YTD 2013	YTD 2012
Female	2	12	3	9	6	2	9	6	49	31	161	157
Male	5	81	2	45	20	4	74	24	255	284	1,072	1,094
Not reported	0	0	0	0	0	0	0	0	0	0	0	0
Total*	7	95	5	54	26	6	83	30	306	316	1,236	1,253

* Totals include people whose sex was reported as transgender.

Table 2: Cumulative number of new diagnoses of HIV infection since the introduction of HIV antibody testing, 1985 to 31 December 2013, by sex and state or territory

Sex	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Female	46	1,167	48	491	175	29	601	385	2,942
Male	353	16,801	205	4,107	1,264	179	7,348	1,751	32,008
Not reported	0	227	0	0	0	0	22	0	249
Total*	399	18,238	253	4,607	1,440	208	7,999	2,143	35,287

* Totals include people whose sex was reported as transgender.

Invasive pneumococcal disease surveillance Australia, 1 January to 31 March 2014

Rachel de Kluyver for the Enhanced Invasive Pneumococcal Disease Surveillance Working Group

Introduction

Invasive pneumococcal disease (IPD) is caused by the bacterium *Streptococcus pneumoniae* and results in illnesses such as pneumonia, bacteraemia and meningitis. There are currently more than 90 serotypes recognised worldwide, approximately half of which are found in Australia where IPD has been a nationally notifiable disease since 2001. The Communicable Diseases Network Australia established the Enhanced Invasive Pneumococcal Disease Surveillance Working Group in 2000 to assist in developing and implementing a nationally standardised approach to the enhanced surveillance of IPD in Australia. This quarterly report documents trends in notified cases of IPD occurring in Australia in the 1st quarter of 2014.

Notification data are collected by all Australian states and territories under jurisdictional public health legislation and are forwarded to the Commonwealth under the *National Health Security Act 2007*. Notified cases are collated nationally in the National Notifiable Diseases Surveillance System (NNDSS). The data in this report are provisional and subject to change as laboratory results and additional case information become available. The data are analysed by diagnosis date, which is the onset date or where the onset date was not known, the earliest of the specimen collection date, the notification date, and the notification receive date. Data for this report were extracted on 7 May 2014. Crude rates were calculated using the Australian Bureau of Statistics estimated resident populations for Australia at 30 June of each year.

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk and older Australians. The 7-valent pneumococcal conjugate vaccine (7vPCV) was added to the National Immunisation Program (NIP) schedule for Indigenous and medically at-risk children in 2001 and for all children up to 2 years of age in 2005. The 13-valent pneumococcal conjugate vaccine (13vPCV) replaced the 7vPCV in the childhood immunisation program from July 2011. The 23-valent pneumococcal polysaccharide vaccine (23vPPV) was added to the NIP schedule for Aboriginal and Torres Strait Islander peoples aged 50 years or over in 1999 and for non-Indigenous Australians aged 65 years or over from January 2005.

Results

There were 209 cases of IPD reported to the NNDSS in the 1st quarter of 2014 (Table). The number of cases notified in the reporting period fell 4% from the 1st quarter of 2013 (n=217). Similar to many infectious diseases, the number of cases of IPD is highest in the cooler months. This trend was observed in all analyses included in this report.

Overall, Indigenous status was reported for 86% (n=179) of cases, ranging from 67% of cases reported by Victoria to 100% of cases reported by the Australian Capital Territory, the Northern Territory, South Australia and Tasmania. New South Wales and Victoria conduct targeted follow-up notified cases of IPD aged 5 years or under and 50 years or over for core and enhanced data, whereas follow-up of all cases is undertaken in other states and territories. Of cases reported with a valid Indigenous status, Aboriginal and Torres Strait peoples accounted for 24% (n=43) of all cases notified in the quarter (Table).

Serotype information was available for 90% (n=189) of all cases reported in the quarter, ranging from 87% of cases reported by Queensland and Victoria, to 100% of cases reported by the Australian Capital Territory, the Northern Territory and Tasmania. There was 1 case reported in the quarter that was deemed by the reference laboratory as non-typable. For figures in this report, cases deemed non-typable are included in the 'Serotype not specified' category with respect to vaccine serotype group.

During the quarter, notified case numbers were highest in the under 5 years age group (n=29), followed by the 65–69 years age group (n=18). This age distribution was evident in cases reported as non-Indigenous Australian (Figure 1). However in cases reported as Indigenous, the most prevalent age groups were the under 5 years (n=8) followed by the 40–44 years age group (n=6). In this report, 3 age groups have been selected for focused analyses. These age groups align with groups that carry the greatest burden of disease and for which the NIP is targeted.

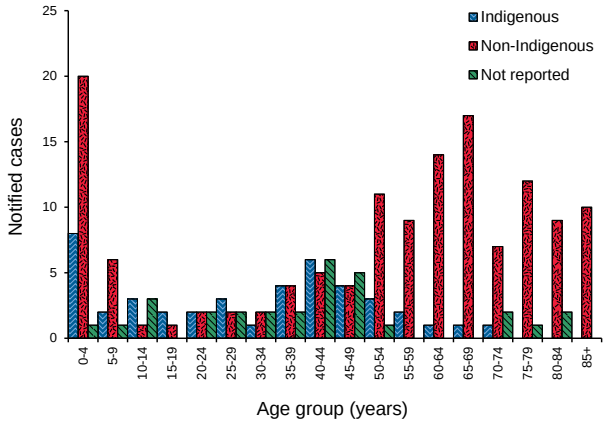
Table: Notified cases of invasive pneumococcal disease, Australia, 1 January to 31 March 2014, by Indigenous status, serotype completeness and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	1st qrt 2014	4th qrt 2013	1st qrt 2013	Year to date 2014
Indigenous	0	5	10	7	3	1	0	17	43	38	33	43
Non-Indigenous	3	49	1	20	15	2	35	11	136	272	157	136
Not stated/ unknown	0	9	0	3	0	0	17	1	30	25	27	30
Total	3	63	11	30	18	3	52	29	209	335	217	209
Indigenous status completeness* (%)	100	86	100	90	100	100	67	97	86			-
Serotype completeness† (%)	100	89	100	87	94	100	87	97	90			-

* Indigenous status completeness is defined as the reporting of a known Indigenous status, excluding the reporting of not stated or unknown Indigenous status.

† Serotype completeness is the proportion of all cases of invasive pneumococcal disease that were reported with a serotype or reported as non-typable. Serotype incompleteness may include when no isolate was available as diagnosis was by polymerase chain reaction and no molecular typing was attempted or was not possible due to insufficient genetic material; the isolate was not referred to the reference laboratory or was not viable; typing was pending at the time of reporting, or no serotype was reported by the notifying jurisdiction to the National Notifiable Diseases Surveillance System.

Figure 1: Notifications of invasive pneumococcal disease, Australia, 1 January to 31 March 2014, by Indigenous status and age group



Invasive pneumococcal disease in children aged less than 5 years

In the 1st quarter of 2014, 14% (n=29) of notified cases were aged less than 5 years. This was a decrease on the number of cases reported in the previous quarter (n=43) and similar to the number reported during the same period of 2013 (n=23) (Figure 2).

The majority of cases (97%, n=28) aged less than 5 years were reported with serotype information. Of these, 36% (n=10) were reported with a serotype included in the 7vPCV or the 13vPCV.

Notified cases aged less than 5 years with disease caused by the 6 additional serotypes targeted by the 13vPCV increased steadily over the period 2007 to 2011, particularly those caused by serotype 19A (Figure 3). However, cases of this type have decreased since the 4th quarter of 2011, reflecting the introduction of the 13vPCV on the universal childhood immunisation program in mid-2011. In the 1st quarter of 2014, there were 3 cases aged less than 5 years with disease due to serotype 19A and 4 cases due to serotype 3. Similar to the 1st quarter of 2013, no cases in this age group were reported with disease caused by serotypes 1, 5 or 6A.

Invasive pneumococcal disease in Indigenous Australians aged 50 years or over

In the 1st quarter of 2014, 4% (n=8) of notified cases were reported as Indigenous Australians aged 50 years or over (Figure 4). This was consistent with the same period in previous years having the lowest number of notifications overall and was equal to the number reported during the same period in 2013 (n=8). For 2010 to 2012, the annual rate of IPD in this group has tended to increase. An outbreak of disease caused by serotype 1 in Central Australia that commenced in late 2010 contributed, in part, to this increase.[1] During 2013, the annual rate fell to 63 per 100,000 population, a 23% decrease from the peak rate in 2012 (82 per 100,000 population).

All but one of the cases notified in the 1st quarter of 2014 was reported with serotype information. Of cases with serotype, three were reported with disease due to serotypes targeted by the 23vPPV; the remaining reported disease due to a non-vaccine serotype (n=4).

Figure 2: Notifications and rates of invasive pneumococcal disease in those aged less than 5 years, Australia, 2003 to 31 March 2014, by vaccine serotype group

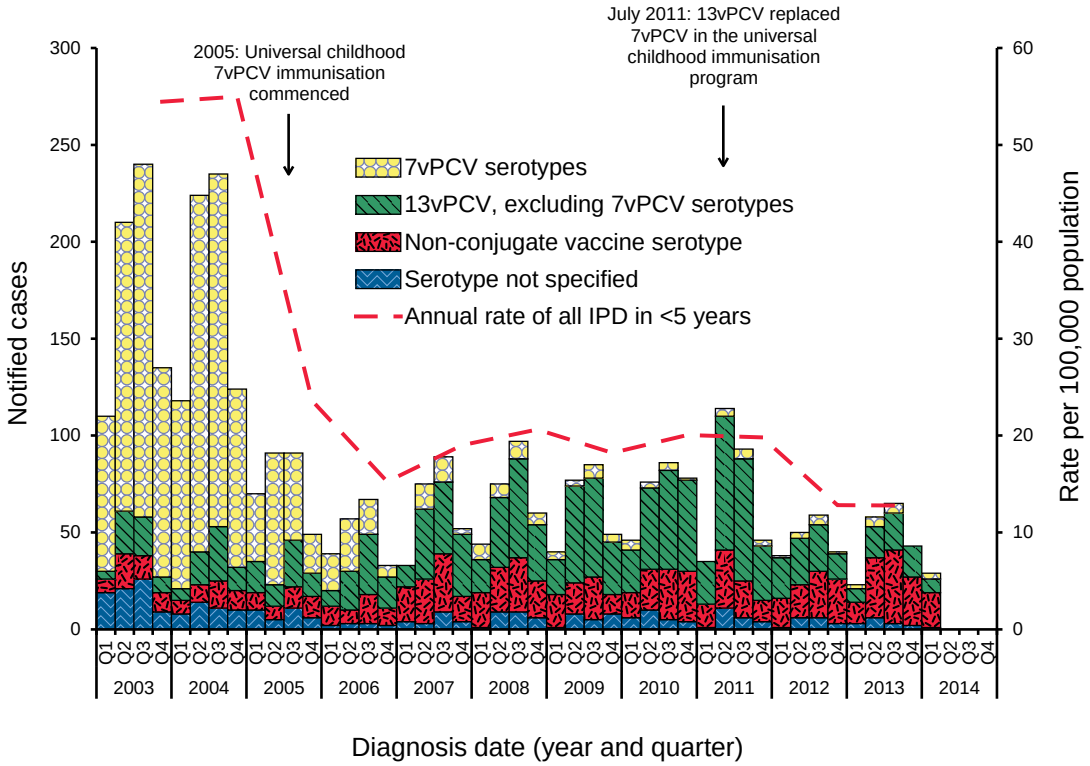


Figure 3: Notifications of invasive pneumococcal disease caused by serotypes targeted by the 13-valent pneumococcal conjugate vaccine (excluding those targeted by 7-valent pneumococcal conjugate vaccine) and rates of all invasive pneumococcal disease, aged less than 5 years, Australia, 2003 to 31 March 2014

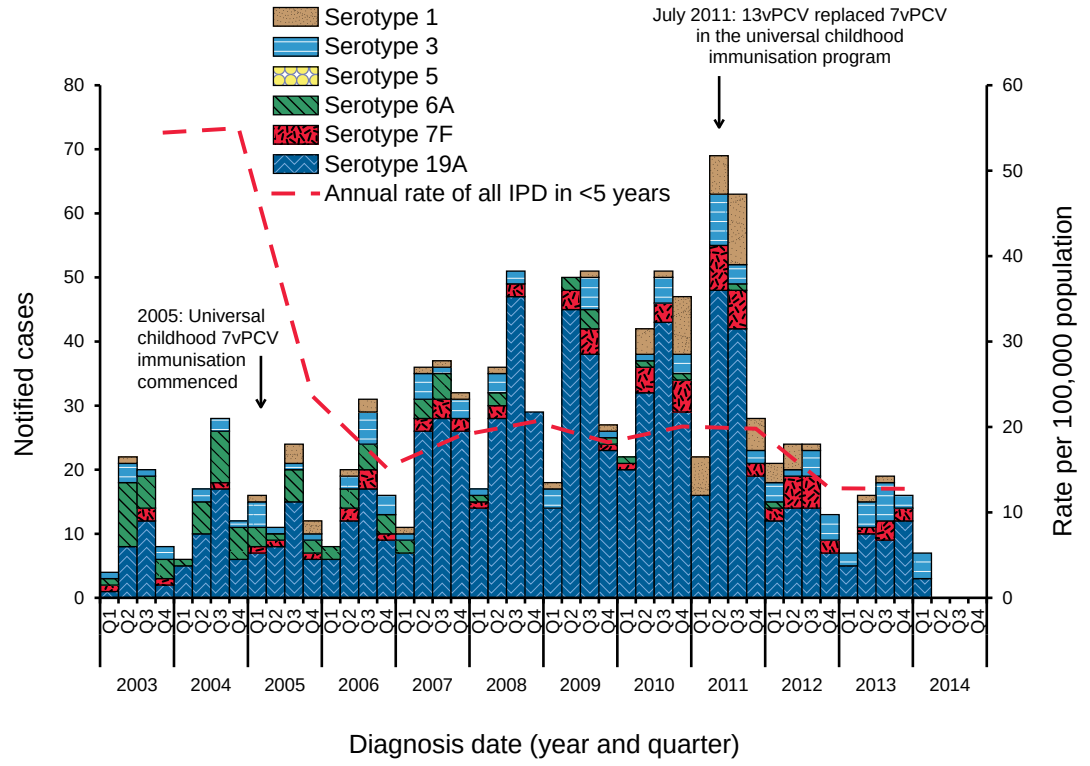
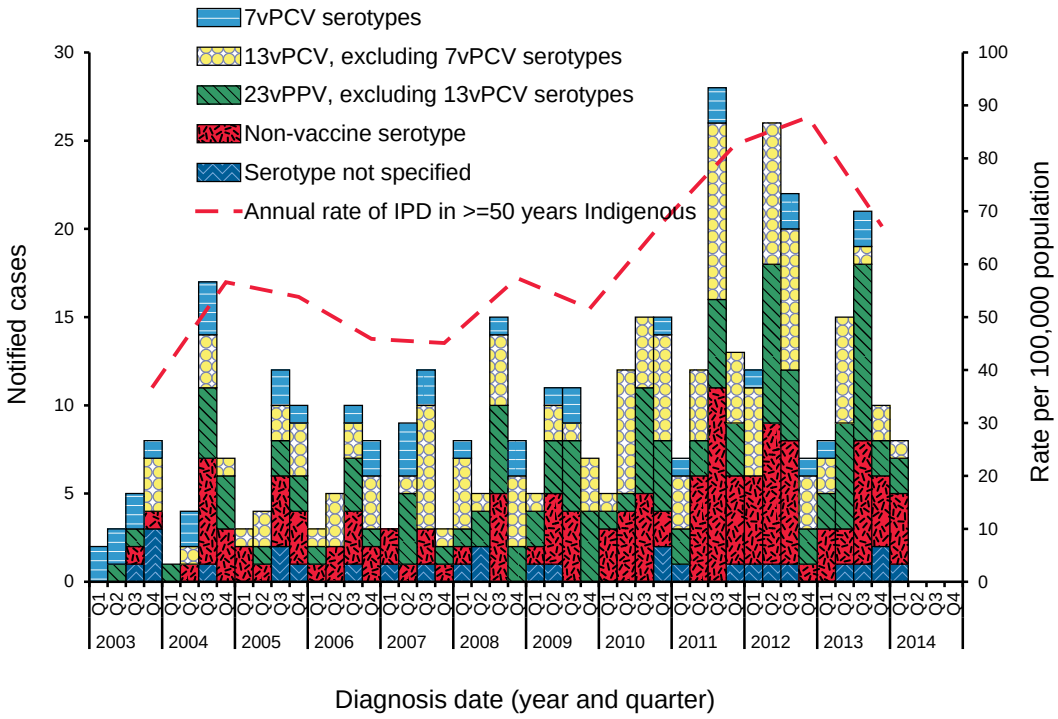


Figure 4: Notifications and rates of invasive pneumococcal disease in Indigenous Australians aged 50 years or over, Australia, 2003 to 31 March 2014, by vaccine serotype group



In 1999 23vPPV immunisation commenced for Indigenous Australians aged 50 years or over.

Invasive pneumococcal disease in non-Indigenous Australians aged 65 years or over

In the 1st quarter of 2014, 26% (n=55) of notified cases were reported as non-Indigenous Australians aged 65 years or over. This was a large decrease in the number of cases reported in the previous quarter (n=122) and was less than the number reported during the same period of 2013 (n=64) (Figure 5). During 2013, the annual rate fell to 16 per 100,000 population, an 11% decrease from the rate in 2012 (18 per 100,000 population).

The majority of cases (89%, n=49) reported in this quarter were reported with serotype information. Of these cases, 49% (n=24) were reported with a serotype targeted by the 23vPPV. While the burden of disease in this age group has remained relatively stable, the profile of serotypes causing disease has changed over time. Disease due to serotypes targeted by the 7vPCV has reduced substantially in this age group, which is likely to be due to herd immunity impacts from the childhood immunisation program.

Conclusion

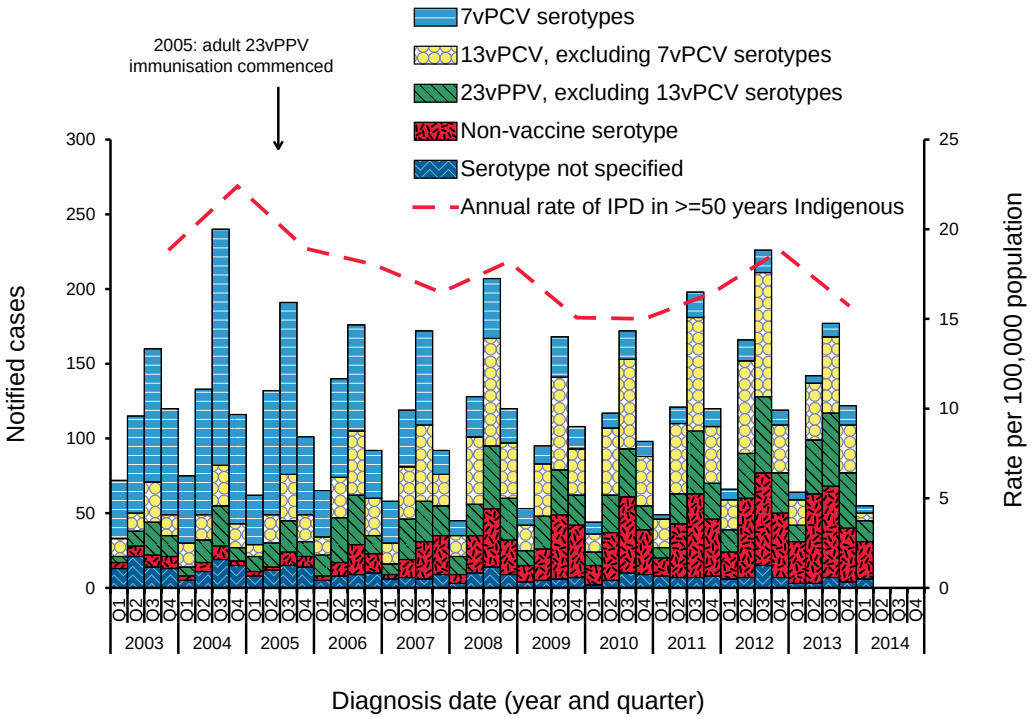
The number of notified cases of IPD in the 1st quarter of 2014 was a 38% decrease on the previous quarter. The total number of cases for the quarter was a 30% increase on the number of cases reported for the same period in 2013. Nationally, the pattern of disease has not changed from the 4th quarter of 2013. Specifically, disease due to the serotypes targeted by the 13vPCV has continued to decline since the 13vPCV replaced the 7vPCV in the childhood immunisation program from July 2011. Compared with the previous quarter, IPD associated with non-vaccine serotypes has remained stable in all groups targeted for IPD vaccination. The rising trend in notified cases of IPD in Indigenous Australians aged 50 years or over reversed considerably, whereas disease in non-Indigenous Australians aged 65 years or over has remained relatively stable but the profile of serotypes causing disease has diversified.

Acknowledgements

Report compiled by Dr Rachel de Kluyver on behalf of the Enhanced Invasive Pneumococcal Disease Surveillance Working Group.

Enhanced Invasive Pneumococcal Disease Surveillance Working Group contributors to this report include (in alphabetical order): David Coleman (Tas.), Heather Cook (NT), Rachel de Kluyver (Health), Lucinda Franklin (Vic.), Carolien Giele (WA), Robin Gilmour (NSW), Michelle Green (Tas.), Geoff Hogg (Microbiological

Figure 5: Notifications and rates of invasive pneumococcal disease in non-Indigenous Australians aged 65 years or over, Australia, 2003 to 31 March 2014, by vaccine serotype group



Diagnostic Unit, University of Melbourne), Vicki Krause (NT), Rob Menzies (NCIRS), Shahin Oftadeh (Centre for Infectious Diseases and Microbiology- Public Health, Westmead Hospital), Sue Reid (ACT), Stacey Rowe (Vic.), Vitali Sintchenko (Centre for Infectious Diseases and Microbiology- Public Health, Westmead Hospital), Helen Smith (Queensland Health Forensic and Scientific Services), Janet Strachan (Microbiological Diagnostic Unit, University of Melbourne), Cindy Toms (Health), Hannah Vogt (SA), Angela Wakefield (Qld).

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Reference

1. Centre for Disease Control Northern Territory. Comments on notifications. *Northern Territory Disease Control Bulletin*. 2012;19(1):29.

Policy and guidelines

REVISED SURVEILLANCE CASE DEFINITIONS

This report provides the revised Surveillance case definitions approved by the Communicable Diseases Network Australia (CDNA) since 1 January 2014.

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.

This case definition will be implemented on 1 July 2014 and supersede any previous versions.

Haemophilus influenzae serotype b infection - invasive

(Effective 1 July 2014)

Reporting

Only confirmed cases should be notified.

Confirmed case

A confirmed case requires laboratory definitive evidence only.

Laboratory definitive evidence

Isolation or detection of *Haemophilus influenzae* type b (Hib) from a normally sterile site where typing has been confirmed at a jurisdictional or regional reference laboratory.

Haemophilus influenzae serotype b infection – invasive changes

Laboratory definitive evidence

after 'Isolation' ADD 'or detection' and CHANGE 'at an approved reference laboratory' to 'at a jurisdictional or regional reference laboratory'

DELETE 'OR Detection of Hib antigen in cerebrospinal fluid when other laboratory parameters are consistent with meningitis'.

Administration

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Communicable Diseases Intelligence (CDI) is currently expanding its base of reviewers. We are seeking reviewers who can provide constructive analysis of papers thus enhancing the overall reputation and quality of CDI.

As a reviewer you will be asked to evaluate the quality, relevance and merit of submitted papers. Comments should be as complete and detailed as possible and contain clear opinions about strengths, weaknesses, relevance, importance to the field, and if relevant, how the paper might be improved. Reviewers will usually be asked to provide their review within two weeks.

Reviewers are not paid but the role as a reviewer can be added to your CV and is a matter of prestige. For some colleges and faculties, peer review is an activity that counts towards continuing professional development points

All peer-reviewers for each year are acknowledged in the December issue of CDI.

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