Australian Government
Department of Health and Ageing

Communicable Diseases Intelligence

Volume 37 Number 1 Quarterly report March 2013

ISSN 1445-4866 Online

Communicable Diseases Intelligence aims to diseminate information on the epidemiology and control of communicable diseases in Australia. *Communicable Diseases Intelligence* invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence. Instructions for authors can be found in *Commun Dis Intell* 2013;37(1):93–96.

Communicable Diseases Intelligence contributes to the work of the Communicable Diseases Network Australia (http://www.health.gov.au/cdna)

Website

http://www.health.gov.au/cdi

Subscriptions and contacts

Communicable Diseases Intelligence is produced every quarter by:
Health Emergency Management Branch
Office of Health Protection
Australian Government Department of Health and Ageing
GPO Box 9848, (MDP 6)
CANBERRA ACT 2601;
Telephone: +61 2 6289 2751
Facsimile: +61 2 6289 2700
Email: cdi.editor@health.gov.au

This journal is indexed by *Index Medicus* and Medline.

Disclaimer

Annual reports

Arboviral diseases and malaria in Australia, 2010-11: Annual report of the National Arbovirus and Malaria Advisory Committee

Katrina Knope, Peter Whelan, David Smith, Cheryl Johansen, Rod Moran, Stephen Doggett, Angus Sly, Michaela Hobby, Nina Kurucz, Phil Wright, Jay Nicholson and the National Arbovirus and Malaria Advisory Committee

Abstract

The National Notifiable Diseases Surveillance System (NNDSS) received notification of 9,291 cases of disease transmitted by mosquitoes during the 2010–11 season (1 July 2010 to 30 June 2011). The alphaviruses Barmah Forest virus and Ross River virus accounted for 7,515 (81%) of these. There were 133 notifications of dengue virus infection acquired in Australia and 1,133 cases that were acquired overseas, while for 10 cases, the place of acquisition was unknown. The number of overseas acquired cases of dengue continues to rise each year, and these are most frequently acquired in Indonesia. Sentinel chicken, mosquito surveillance, viral detection in mosquitoes and climate modelling are used to provide early warning of arboviral disease activity in Australia. In early 2011, sentinel chickens in south eastern Australia widely seroconverted to flaviviruses. In 2010–11, there were 16 confirmed human cases of Murray Valley encephalitis acquired in Australia. There was one human case of Kunjin virus infection. There were 7 notifications of locally-acquired malaria in Australia and 407 notifications of overseas-acquired malaria during the 2010–11 season. *Commun Dis Intell* 2013;37:E1–E20.

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 2010 to 30 June 2011. It includes notified cases of disease caused by the alphaviruses (Barmah Forest virus, BFV, chikungunya virus, CHIKV and Ross River virus, RRV), flaviviruses (dengue virus, DENV, Murray Valley encephalitis virus, MVEV, Kunjin virus, KUNV, Japanese encephalitis virus, JEV and yellow fever virus) and malaria. Both locally acquired and overseas acquired cases are described. Vector, climate and vertebrate surveillance measures for arboviruses (in particular for MVEV) conducted by states and territories and at the border are also 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 the Committee have expertise in 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 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.

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 notification of communicable diseases and authorises the exchange of health information between the Commonwealth and the States and Territories. The NHS Act 2007 provides for the establishment of the National Notifiable Diseases List, which specifies the diseases about which personal information can be exchanged between the States and Territories and the Commonwealth. State and territory health departments transfer these notifications regularly to the NNDSS. The primary

responsibility for public health action resulting from a notification resides with state and territory health departments.

This report presents data extracted from NNDSS during June 2012 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 receive 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 2010 NNDSS Annual report. Case definitions for the diseases included in this report are available at 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, MVEV and yellow fever virus);
- 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 chikungunya virus infection as a separate infection. Prior to this, CHIKV infections were notified under the disease category arbovirus NEC and the Northern Territory continues this practice. All notifications of CHIKV infection under arbovirus NEC were counted under CHIKV infection in this report.

Crude notification rates or counts for the 2010–11 season were compared with rates or counts for that disease over the previous five 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 2010–11 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 (ABS) estimated resident populations for Australia and each state or territory at June 2011.[1] 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 (2011 population applied to the 2010–11 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 2010–11 season, additional information was collected to enable these mixed infections to be reported as one case for the purpose of this report. In 2010–11, this resulted in five 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 New South Wales Ministry of Health, and laboratory services are contracted to Institute of Clinical Pathology and Medical Research (ICPMR), Pathology West at Westmead Hospital. Mosquito trapping occurs from mid-spring to mid-autumn (November to April), and mosquitoes are collected weekly for species identification and quantification, and processed for isolation of arboviruses. Data on the Southern Oscillation Index, rainfall and temperature obtained from 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.

Northern Territory

Surveillance consists of routine year round once per month sentinel chicken surveillance and *ad hoc* virus isolation from mosquitoes during the high risk months of February to June inclusive. The program is combined and coordinated by the Northern Territory Department of Primary Industries, Fisheries and Mines (DPIFM) and the Northern Territory Department of Health, with support from volunteers. Laboratory support is provided by Berrimah laboratories DPIFM. 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. Routine mosquito trapping consists of 22 trapping sites throughout the Darwin urban area, six in Nhulunbuy, three in Tennant Creek, five in Katherine, three in Alyangula on Groote Eylandt, and six in Alice Springs. In Alice Springs it is a cooperative program between Alice Springs Environmental Health, and the Medical Entomology unit in Darwin. Climate information from 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 state-wide surveillance for MVEV in vertebrate hosts, and does not maintain sentinel chicken flocks. Queensland is introducing direct mosquito virus surveillance using honey trap saliva technology. Mosquito monitoring is performed by local councils, under the Joint Strategic Mosquito Management Framework and is funded by Queensland Health. 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 Queensland Institute of Medical Research.

South Australia

Across South Australia, mosquito surveillance and control activities are conducted through a partnership between South Australia Health, the University of South Australia, Local Government and Biosecurity South Australia. The program is coordinated by the South Australian Department for Health and Ageing 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 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 Sorrell Council region (which includes mosquito breeding areas, is fairly populous, and is close to Hobart) during during high risk periods from 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 from November to April. Flocks of 20 chickens are located at a range of locations mostly along the Murray, and are bled weekly from mid-October to April, and flocks are replaced annually. Six councils undertake mosquito surveillance, four traps are placed in each area and mosquitoes are collected weekly and sent live to DPI for identification, enumeration and virus isolation. The Victorian Arbovirus Taskforce examines the risk of outbreaks of MVEV using meteorological surveillance data such as the Southern Oscillation Index and rainfall deciles using Forbes and Nicholls models.[2,3]

Western Australia

The University of Western Australia's Arbovirus Surveillance and Research Laboratory (ASRL) is funded by the Health Department of Western Australia to coordinate sentinel chicken program and mosquito surveillance, and provide confirmatory serological testing for other sentinel chicken programs in Australia. Thirty 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 (March to April) when MVEV 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.

Arbovirus research laboratories in Australia

CSIRO

Commonwealth Scientific and industrial Research Organisation (CSIRO)
CSIRO Australian Animal Health Laboratory
Private Bag 24 (5 Portarlington Road)
Geelong Victoria 3220
Switchboard: (03) 5227 5000

New South Wales

Institute of Clinical Pathology and
Medical Research
Westmead Hospital
Locked Bag 9001
Westmead, New South Wales 2145

Phone: (02) 9845 6255
(Vector surveillance, sentinel animal testing, human serology)

Northern Territory

Berrimah Veterinary Laboratory
39 Kessells Rd
Coopers Plains
PO Box 594 Archerfield Qld 4108
Phone: (07) 3274 9151

Queensland

Queensland Health Forensic and Scientific Services
Northern Territory Department of Primary Industries, Fisheries and Mines (DPIFM)
Makagon Rd, Berrimah, Darwin
Northern Territory 0828
Phone (08) 8999 2065

Victoria

Department of Primary Industries
Attwood Centre
475 Mickleham Road
Attwood Victoria 3049
Phone: (03) 9217 4200

Victorian Infectious Diseases Reference Laboratory (Human)
10 Wrecklyn St
North Melbourne Victoria 3051
Phone: (03) 9342 2600

Western Australia

PathWest Laboratory Medicine WA
Division of Microbiology and Infectious Diseases (Human)
Hospital Avenue
Nedlands Western Australia 6009
Phone: (08) 9346 3122

Arbovirus Surveillance and Research Laboratory

Discipline of Microbiology and Immunology (animal/vector)
School of Pathology and Laboratory Medicine
The University of Western Australia
35 Stirling Highway
Crawley Western Australia 6009
Phone: (08) 9346 2212

Results

During the 2010–11 season, there were 9,291 notifications of diseases transmitted by mosquitoes (Table 1). This represented an 18% increase from the mean of 7,894 notifications for the previous five years.

Alphaviruses

In Australia, the most frequently detected 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 mosquito that breed in diverse environments (freshwater habitats, coastal regions, salt marshes, floodwaters, established wetlands and urban areas).[4] RRV and BFV occur exclusively in the Australasian region.[5]

The alphavirus CHIKV does not occur in Australia, but 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.[6] 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 *Aedes aegypti*, which occurs in northern Queensland and *Aedes albopictus* which is found on Cocos Island, Christmas Island and the Torres Strait Islands.[7] Other Australian mosquito species have been shown to be competent vectors of CHIKV in the laboratory.[8]

Ross River virus infections

There were 5,653 notifications of RRV infection during the 2010–11 season, representing a rate of 25.0 per 100,000 population, compared with a 5 year mean of 23.0 per 100,000 (Table 1). Queensland reported the largest number of cases (n=1,397), while the highest rate was in the Northern Territory.

Rates of RRV in South Australia trebled from 23.8 per 100,000 (n=391) in 2009–10 to 69.7 per 100,000 in 2010–11 (n=1,154) and were more than 4 times the five year mean of 17.1 per 100,000 (n=274.2 cases) (Figure 1). Rates in Victoria increased to 23.7 per 100,000 (n=1,334) from 6.4 per 100,000 in 2009–10 (n=353) and were more than 6 times the five year mean of 3.8 per 100,000 (n=204).

RRV was most commonly reported among middle-aged adults, peaking in the 35–54 year age-groups (RRV was most commonly reported among middle-aged adults, peaking in the 35–54 year age-groups (Figure 2). As in previous years, a little over half of all cases (54%) were female. The overall rate of RRV

Table 1: Number of notified cases of mosquito-borne disease and rate, Australia, 2010-11 and 5 year mean, by disease and state or territory

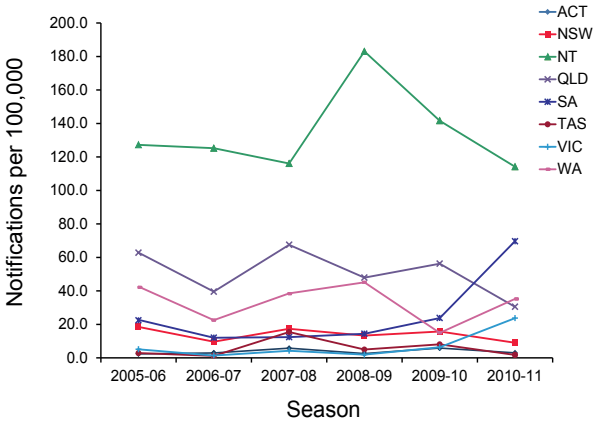
		ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
Arbovirus NEC	Cases 2010–11	0	1	0	5	0	0	16	0	22
	5 year mean cases	0	0.6	1	9.4	0	0	5.2	0	16.2
	Rate 2010–11	-	-	-	-	-	-	-	-	-
	5 year mean rate	-	-	-	-	-	-	-	-	-
Barmah Forest virus infection	Cases 2010–11	3	427	63	885	134	4	223	123	1862
	5 year mean cases	5.4	486.8	97.8	915.4	73.6	1.7	26.8	143.6	1,750.4
	Rate 2010–11	0.8	5.8	27.3	19.3	8.1	0.8	4.0	5.2	8.2
	5 year mean rate	1.6	7.0	44.4	21.3	4.6	0.2	0.5	6.6	8.2
Chikungunya virus infection	Cases 2010–11	NN	15	8	7	2	2	18	11	63
	5 year mean cases	NN	3.6	0.0	1.6	0.6	0.0	4.0	3.2	13.0
	Rate 2010–11	-	-	-	-	-	-	-	-	-
	5 year mean rate	-	-	-	-	-	-	-	-	-
Dengue virus infection	Cases 2010–11	12	204	32	309	28	5	141	528	1259
	5 year mean cases	9.2	103.4	23.8	316.0	18.8	2.8	22.2	98.2	594.4
	Rate 2010–11	-	-	-	-	-	-	-	-	-
	5 year mean rate	-	-	-	-	-	-	-	-	-
Japanese encephalitis virus infection	Cases 2010–11	0	0	0	0	0	0	0	0	0
	5 year mean cases	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.2
	Rate 2010–11	-	-	-	-	-	-	-	-	-
	5 year mean rate	-	-	-	-	-	-	-	-	-
Kunjin virus infection	Cases 2010–11	0	0	1	0	0	0	0	0	1
	5 year mean cases	0.0	0.0	0.4	0.7	0.0	0.0	0.2	0.3	1.8
	Rate 2010–11	-	-	-	-	-	-	-	-	-
	5 year mean rate	-	-	-	-	-	-	-	-	-
Malaria	Cases 2010–11	3	112	15	135	4	7	78	60	414
	5 year mean cases	9.6	109.8	30.4	188.2	26.4	12.6	105.6	88	570.6
	Rate 2010–11	-	-	-	-	-	-	-	-	-
	5 year mean rate	-	-	-	-	-	-	-	-	-
Murray Valley encephalitis virus infection	Cases 2010–11	0	2	2	0	2	0	0	9	15
	5 year mean cases	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	Rate 2010–11	-	-	-	-	-	-	-	-	-
	5 year mean rate	-	-	-	-	-	-	-	-	-
Ross River virus infection	Cases 2010–11	11	658	263	1,397	1,154	9	1,334	827	5,653
	5 year mean cases	13.6	1,047.0	306.4	2,359.2	274.2	32.6	204.0	709.4	4,946.4
	Rate 2010–11	3.0	9.0	114.2	30.5	69.7	1.8	23.7	35.2	25.0
	5 year mean rate	3.9	14.9	138.6	54.8	17.1	6.5	3.8	32.7	23.0
Yellow fever	Cases 2010–11	0	0	0	2	0	0	0	0	2
	5 year mean cases	0	0	0	0	0	0	0	0	0
	Rate 2010–11	-	-	-	-	-	-	-	-	-
	5 year mean rate	-	-	-	-	-	-	-	-	-
Total 2010–11		29	1,419	384	2,740	1,324	27	1,810	1,558	9,291

* Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004. Arbovirus (NEC) replaced Flavivirus (NEC) from 2008.

NEC Not elsewhere classified

Notification rates for diseases that were primarily acquired overseas or for rare diseases (n<20 in 2010-11) are not presented.

Figure 1: Rates of Ross River virus infection, Australia, July 2005 to June 2011, by year and state or territory



in females was 26.7 per 100,000, while in males the rate was 23.2 per 100,000). As in previous years, a little over half of all cases (54%) were female. The overall rate of RRV in females was 26.7 per 100,000, while in males the rate was 23.2 per 100,000.

As in previous years, there was a marked seasonal trend in RRV notifications, with the largest number diagnosed in January (n=900), February (n=1,184) and March (n=964). This was an earlier start to the season than in previous years where the peak months have been February to April (Figure 3).

Barmah Forest virus infections

There were 1,862 notifications of BFV infections during the 2010–11 season, representing a rate of 5.2 per 100,000, which has decreased from the mean of 8.2 per 100,000 for the previous five years (Table 1). Queensland reported the largest number of notifications of BFV infection (n=885) while the highest rate was reported in the Northern Territory (27.3 per 100,000).

Figure 2: Rates of Ross River virus infection, Australia, 2010-11, by age-group and sex

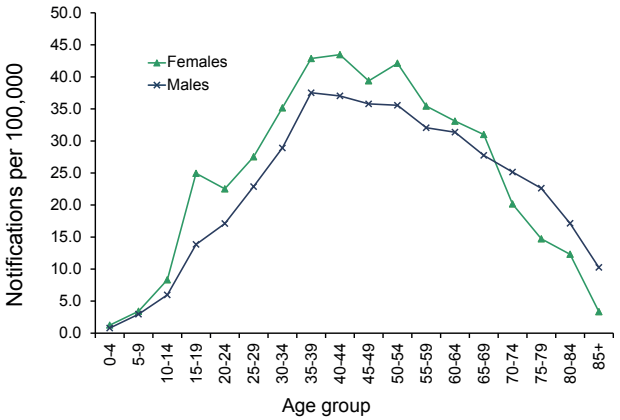
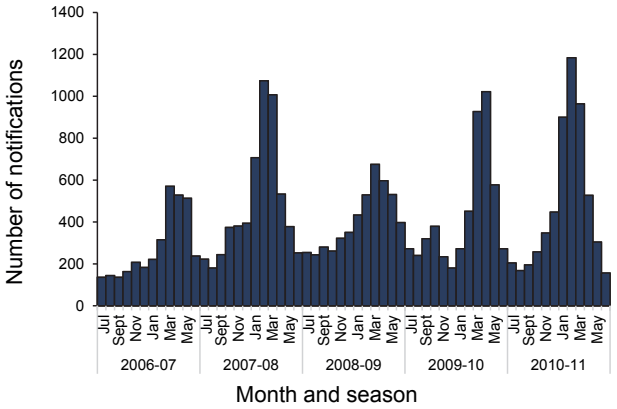


Figure 3: Number of notified cases of Ross River virus infection, Australia, July 2006 to June 2011, by month, year and state or territory



In South Australia, the 2010–11 notification rate was four times the five year mean, while in Victoria, the notification rate was six times the five year mean.

BFV infections were most commonly reported among middle aged adults, peaking in the 45-64 year-old age-groups, and 50% of cases were male (Figure 5).

In 2010–11, infections were most frequently diagnosed in January (n=326), February (n=238) and March (n=273), and this was an earlier than average start to the season compared with the previous five years when cases were most frequently reported between February and April (Figure 6). While BFV notifications showed a clear seasonal trend, this trend is less marked than for RRV infections. The higher than expected numbers of BFV notifications in winter is possibly an artefact, reflecting the possibility of false positive IgM diagnoses.).

Figure 4: Rates of Barmah Forest virus infection, Australia, July 2005 to June 2011, by year and state or territory

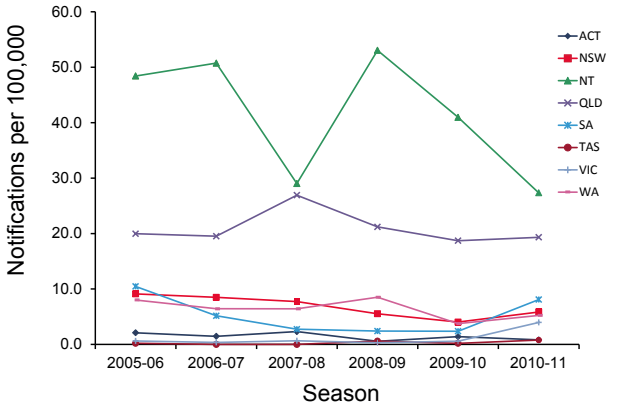


Figure 5: Rates of Barmah Forest virus infection, 2010-11, Australia, by age-group and sex

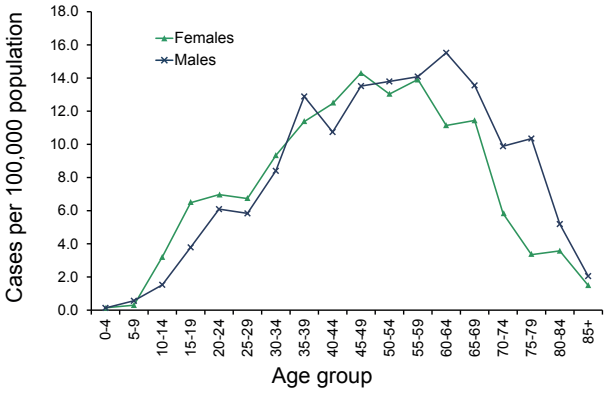


Figure 6: Number of notified cases of Barmah Forest virus infection, Australia, July 2006 to June 2011, by month, year and state or territory

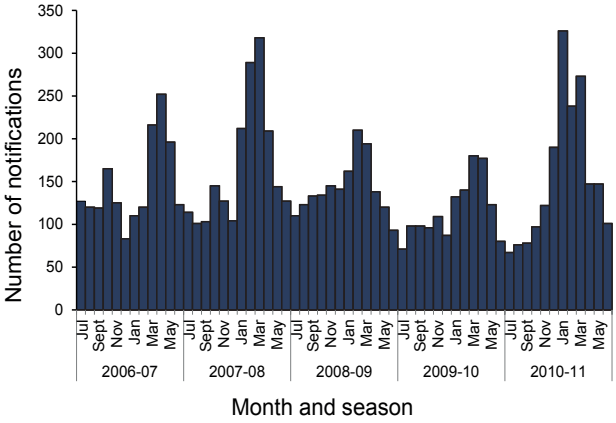
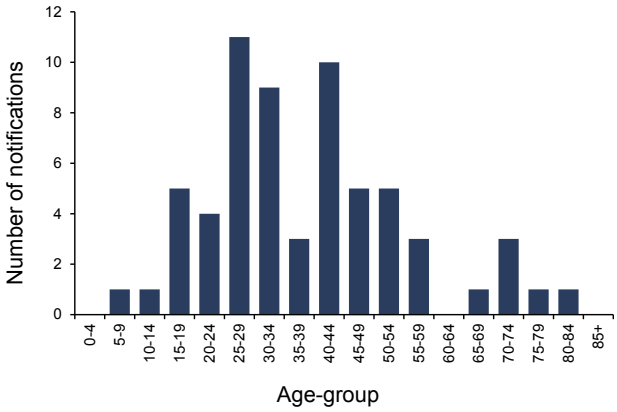


Figure 7: Number of notified cases of chikungunya virus infection, Australia, 2010-11, by age-group



In 2010–11, infections were most frequently diagnosed in January (n=326), February (n=238) and March (n=273), and this was an earlier than average start to the season compared with the previous five years when cases were most frequently reported between February and April (Figure 6). While BFV notifications showed a clear seasonal trend, this trend is less marked than for RRV infections. The higher than expected numbers of BFV notifications in winter is possibly an artefact, reflecting the possibility of false positive IgM diagnoses.

Chikungunya virus infection

CHIKV infection is a notifiable disease in all jurisdictions other than the Australian Capital Territory. There were 63 notifications of CHIKV infection during the 2010–11 season compared with a 5 year mean of 14 cases. All cases were acquired overseas, with complete information supplied on the country of acquisition for 55 of these. The most frequently reported countries of acquisition were Indonesia (32 cases) and India (11 cases). CHIKV infection was most frequently notified for young and middle aged adults, with the largest number of cases in the 25-29 year age-group (Figure 7).

Flaviviruses

This section provides information on flavivirus infection notified to NNDSS including DENV, MVEV, the Kunjin strain of West Nile virus (KUNV) and JEV. Other flavivirus infections may be notified under the Arbovirus (NEC) category.

DENV has four serotypes. 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 type. *Ae. aegypti* is the major vector of dengue in Australia.

Infection with MVEV, KUNV or JEV is usually asymptomatic or produces a non-specific illness. A small percentage of cases progress to encephalomyelitis of variable severity. *Culex annulirostris* is the major vector of MVEV, JEV and KUNV. No specific treatment is available for these diseases and care is largely supportive. A vaccine is available to prevent JEV infection,[9] but there is no vaccine for DENV, MVEV or KUNV infection.

Dengue virus infection

There were 1,276 notified cases of DENV infection during the 2010–11 season. Of these, 133 were acquired in Australia, and 1,133 acquired the infection while overseas (Table 2). For the remaining 10 cases, no information on place of acquisition was available.

Locally-acquired dengue virus infection

There were 133 notifications of DENV infection acquired in Australia during 2010–11 representing a rate of 0.6 per 100,000 population, compared with 37 locally-acquired cases in 2009–10.

Local transmission of dengue in Australia is normally restricted to areas of northern Queensland where the key mosquito vector, *Ae. aegypti* is present.[10] 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.[11]

In 2010–11, 131 cases of dengue were acquired in Queensland, with 128 cases known to have either resided or travelled in North Queensland, while for the remainder the specific region of travel within Queensland could not be confirmed. Most cases were linked with known outbreaks (124/131). In 2010–11, Queensland Health reported 8 outbreaks of dengue in North Queensland, with 123 cases (100 DENV-2, 13 DENV-4 and 10 DENV-1).*[12] The largest outbreaks were in Townsville between 30 June and 17 December 2010 (31 cases, DENV-2), Cairns between July and September 2010 (19 cases, DENV-2) and Innisfail and Cairns between 16 January and 5 March 2011 (47 cases, DENV-2).[12] Each of these outbreaks was linked to single importations in infected travellers. Despite frequent outbreaks, mosquito and infection control measures undertaken by public health authorities and by residents have ensured that dengue has not become endemic in North Queensland.

In 2010–11, one case of dengue in the Northern Territory was thought to have been acquired near Darwin airport where the case worked in an industrial area.[13] The case was likely to have been bitten

* These numbers are based on data from Queensland Health as at 3 June 2011, and variance between that dataset and the data extracted from NNDSS in June 2012 is not unexpected (123 outbreak cases reported by Queensland Health versus 124 in NNDSS).

Table 2: Number of notified cases of dengue virus infection, Australia, 1 July 2005 to 30 June 2011, by year, state or territory and place of acquisition

Place of acquisition	Year	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Total
Locally-acquired†	2005–06	0	3	0	43	0	0	1	0	47
	2006–07	0	6	1	48	0	0	0	0	55
	2007–08	0	5	0	27	3	0	0	0	35
	2008–09	0	5	0	1006	1	0	3	1	1016
	2009–10	0	3	0	33	0	0	1	0	37
	2010–11	0	2	1	126	0	0	3	1	133
Overseas-acquired	2005–06	7	53	16	30	10	0	12	21	149
	2006–07	2	65	14	59	12	0	8	27	187
	2007–08	4	100	25	78	30	4	15	94	350
	2008–09	14	168	27	115	25	6	18	120	493
	2009–10	19	121	36	125	11	4	50	227	593
	2010–11	10	219	30	181	27	5	139	522	1133
Unknown	2005–06	0	0	0	2	0	0	0	0	2
	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	4	0	0	1	0	5
	2009–10	0	2	0	1	0	0	1	0	4
	2010–11	2	1	1	2	1	0	0	3	10
Total	2005–06	7	56	16	75	10	0	13	21	198
	2006–07	2	71	15	112	12	0	9	27	248
	2007–08	4	105	25	109	35	4	15	94	391
	2008–09	14	173	27	1125	26	6	22	121	1514
	2009–10	19	126	36	159	11	4	52	227	634
	2010–11	12	222	32	309	28	5	142	526	1276

† Locally-acquired cases are acquired in Australia and not necessarily in the states or territory from which they are reported. Under the cross border notification protocol, cases are notified by their state or territory of residence, not the state or territory where the disease was diagnosed or acquired.

by an infected mosquito that was carried by a military aircraft that had returned from Bali just prior to the likely date of acquisition and had unloaded and parked less than 2km from the case's place of work.[13] There was one case of health-care associated dengue in Western Australia in 2010; a physician in Perth sustained a needlestick injury whilst taking blood, five days prior to symptom onset.[14]

Overseas-acquired dengue virus infection

There were 1,133 notifications of dengue virus infection acquired overseas during the 2010–11 season (Table 2), more than three times the 5 year mean for overseas-acquired infection (354.4). All jurisdictions reported increased numbers of notifications of overseas-acquired DENV infection from 2005–06, with the largest increases compared with the 5 year mean being in Victoria (6.7 times the 5 year mean) and Western Australia (5.3 times the 5 year mean).

The country of acquisition was available for 99.8% (n=1,131) of overseas-acquired cases. The median age of locally acquired cases and overseas-acquired cases was the same (38 years), and for both categories, 53% of cases were male. Indonesia was the country of acquisition for 66% of cases (n=747), and these were of all four dengue serotypes. The infecting DENV serotype was determined for 50% (n=570) of overseas-acquired dengue cases. DENV-1 (n=236) was the most frequently reported serotype.

The median age of locally acquired cases and overseas-acquired cases was the same (38 years), and for both categories, 53% of cases were male.

Japanese encephalitis virus infections

There were no cases of JEV infection notified to NNDSS in Australia during 2010–11. The last imported case was during the 2008–09 season and the last locally-acquired case was in 1998.[15,16]

Kunjin virus infection

There was one human case of KUNV infection reported in Australia during 2010–11. The case was a 60 year-old man from the Northern Territory who was IgM positive for KUNV and negative for MVEV, BFV and RRV. The infection was acquired in the Barkly region. The case was non encephalitic, and recovered.

Murray Valley encephalitis

In 2010–11, 15 cases of MVEV infection in Australia were notified to the NNDSS, compared with an average of 1.4 cases per annum for the previous five years. There was also a confirmed case in a Canadian resident who was diagnosed in Canada after being exposed in the Northern Territory and was thus not notified to NNDSS (Table 4). Two of the 16 confirmed cases died.

All cases in 2010–11 were acquired in Australia, and 13 of the 16 confirmed cases were acquired in areas where regular enzootic viral activity is reported (the Pilbara and Kimberley regions of Western Australia, and the northern two thirds of the Northern Territory), or where epizootic disease activity is not unexpected (the Midwest and Goldfields region of WA) (Table 4). Three confirmed cases were reported from south-eastern Australia where epizootic disease

Table 3: Overseas-acquired cases of dengue virus infection, Australia, 2010-11, 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	747	66	190	91	104	50	312
Thailand	123	11	7	24	11	2	79
India	41	4	12	1	0	0	28
Philippines	40	4	2	2	7	2	27
Vietnam	36	3	9	6	1	1	19
East Timor	22	2	1	5	0	1	15
Papua new Guinea	17	2	2	3	1	0	11
Malaysia	16	1	3	2	2	1	8
Cambodia	13	1	1	2	2	0	8
Sri Lanka	11	1	2	0	1	0	8
Other countries	65	6	7	10	1	0	47
Unknown country	2	0	0	1	0	0	1
Total	1133	100	236	147	130	57	563

Table 4: Confirmed cases of Murray Valley encephalitis infection acquired in Australia, 2010-11 (N=16), by region of infection

State/territory and region of infection	State/territory of residence	Month of onset	Sex	Age (years)	Comment
Western NSW	NSW	March	F	63	Non-encephalitic. Recovered.
Berri, SA	SA	March	M	47	Encephalitis. Recovered with residual disease.
Mannum, SA	SA	March	M	27	Encephalitis. Died.
Katherine region, NT	NSW	May	F	63	Encephalitis. Recovered.
Barkly region, NT	NT	March	M	33	Encephalitis. Recovered.
Barkly region, NT	NT	March	M	1	Encephalitis. Recovered.
Darwin/Katherine region, NT	Overseas (Canada)‡	May	F	19	Encephalitis. Died.
Kimberley, WA	WA	April	M	29	Encephalitis. Recovered with residual disease.
Midwest/Goldfields, WA	WA	April	M	25	Encephalitis. Recovered.
Pilbara, WA	WA	April	M	25	Encephalitis. Recovered with residual disease
Midwest, WA	WA	March	F	41	Encephalitis. Recovered with residual disease.
Midwest/Pilbara, WA	WA	March	M	61	Encephalitis. Died
Pilbara, WA	WA	March	F	50	Non-encephalitic. Recovered.
Kimberley, WA	WA	May	F	2	Encephalitis. Recovered with residual disease.
Pilbara, WA	WA	May	F	67	Encephalitis. Recovered with residual disease.
Pilbara, WA	WA	May	F	1*	Encephalitis. Recovered.

* 23 months of age

‡ This case in a Canadian resident was diagnosed in Canada and thus not notified to NNDSS and not included in Table 1, which listed only notified cases.

activity is rare or unknown (2 in South Australia and 1 in New South Wales). All cases had an onset date between March and May 2011.

Three further possible cases could not be laboratory confirmed and were not reported to the NNDSS. These were a possible case in a 39 year-old man who was hospitalised in Western Australia with clinically-diagnosed MVEV infection based on a consistent exposure history, clinical picture, and magnetic resonance imaging, a possible case in an asymptomatic family member of the confirmed case in New South Wales who was identified through active case finding, and a possible fatal case in a 69 year-old from north-western Victoria.[17,18]

Outbreaks of arboviral disease in horses in 2010–11

The outbreaks of MVEV infection in humans occurred in the context of widespread evidence of seroconversion in sentinel chickens to flaviviruses and outbreaks of arboviral disease causing neurological and muscular disease in horses due to both KUNV and RRV. Between January and June 2011, there were 982 clinically apparent cases of arboviral disease in horses and 91 horses died.[19] The first horse cases were investigated from early February, and new reports reached a peak in March and April and declined in mid-May.[20] Cases were widely distributed across Victoria and New South Wales, and were also reported from southeastern parts of South Australia and Queensland, and the southwestern areas of Western Australia.[20] In New South Wales, South Australia and Western Australia the majority of cases were due to KUNV infection, while in Victoria, RRV infections comprised more than half of all confirmed infections.[20] The KUNV cases in horses were due to a KUNV variant ($WNV_{NSW2011}$) which is likely to have been derived from previously described KUNV in Australia, rather than of exotic origin. The variant strain was found to be more neuroinvasive in mouse studies than KUNV.[21]

Yellow Fever

Two cases of yellow fever (YF) were notified in 2010–11, both from Queensland. The cases had recently returned from travel to YF endemic areas (one from Colombia and the other from Ghana), were IgM positive, had a clinically-compatible illness

and had received yellow fever vaccine in the previous 3 months. The cases met the CDNA national surveillance case definition. However, treating clinicians and the public health units considered that on the balance of probabilities, both were likely to have been vaccine related but that the possibility that they were true cases could not be excluded. A revised CDNA case definition for yellow fever which came into effect on 1 January 2013 will exclude vaccine-associated cases in future.

Vertebrate, vector and climate surveillance programs for flaviviruses in 2010-11

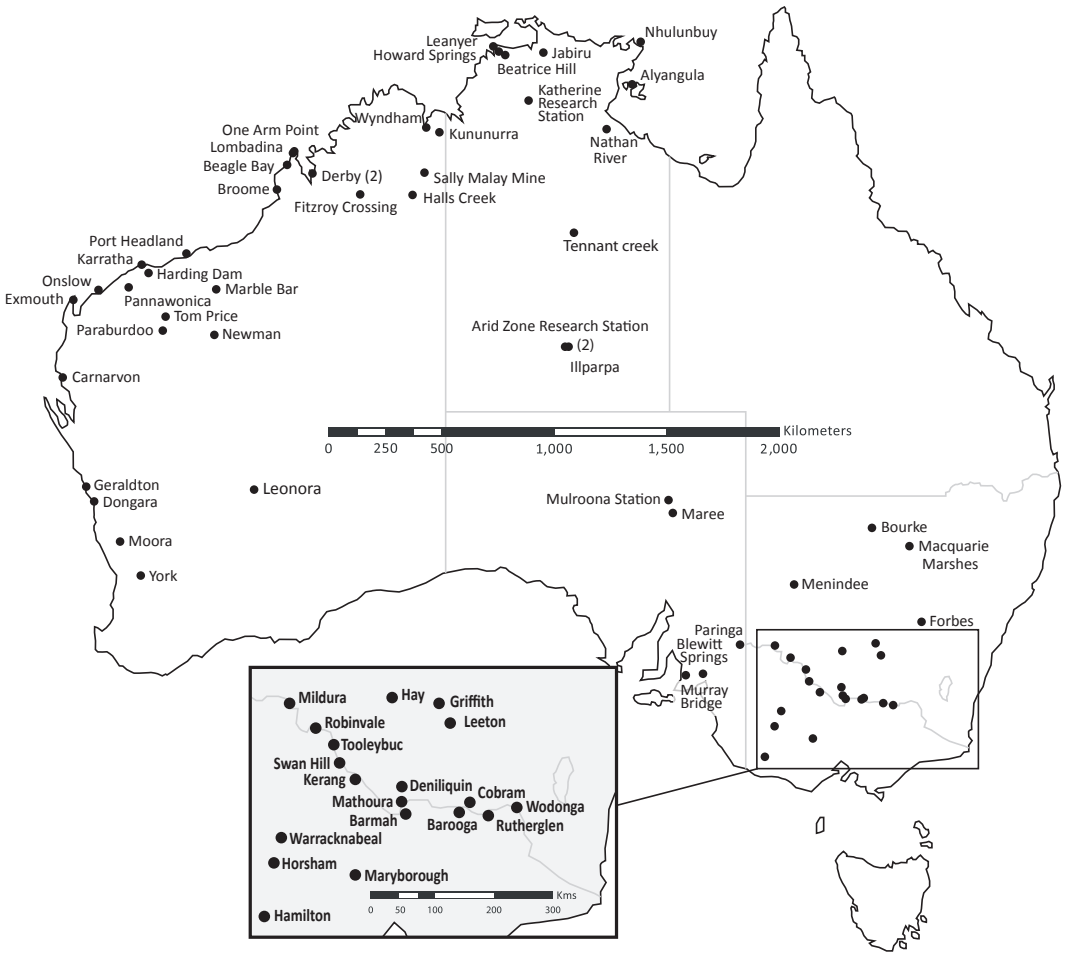
The sentinel chicken program is designed to detect flavivirus activity. In 2010–11, sentinel chicken flocks were located in the Northern Territory, New South Wales, South Australia, Victoria and Western Australia. The program aims to provide early warning of the endemic arboviruses MVEV and KUNV, as well as exotic arboviruses such as JEV.[22] 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 distributed geographically to detect flavivirus activity and to provide a timely and accurate indication of risk to people (Map).[23]

New South Wales

The climatic conditions leading up to the 2010-2011 arboviral season for the New South Wales inland were of above, to well above average rainfall for the entire second half of 2010, plus above average rainfall along the Murray River valley for the first quarter of 2011. Both the Forbes' and Nicholls' MVEV climatic models indicated probable activity in southeastern Australia for the 2010-2011 season.[2] The elevated precipitation levels led to increased vector production with over 200,000 mosquitoes trapped at inland localities, being over six times that of the previous season. Two collections from Griffith

Map: Location of sentinel chicken flocks, Australia, 2010-11



yielded over 10,000 mosquitoes per trap. A considerable number of arboviruses were isolated from the collected mosquitoes, with 102 arboviral isolates (7 BFV, 13 RRV, 71 Sindbis Virus, 2 Edge Hill visus, 2 Kokobera virus and 7 KUNV).

For the coast, climatic conditions were mostly similar to those inland although the north coast had average rainfall through the late summer and temperatures became cool from March onwards, which resulted in reduced mosquito numbers. Overall mosquito collections were well below previous years, due largely to very small numbers of *Aedes vigilax*, the major coastal vector. As a result, arboviral activity was lower than in previous years. There was a total of seven isolates, including 4 BFV and 3 RRV.

The 2010–11 season began on 1 November 2010 with the first bleed and ended on 29 April 2011. For 2010–2011, a total of eight flocks each containing up to 15 Isa Brown pullets was deployed, with one flock each at Bourke, Deniliquin, Forbes, Griffith, Leeton, Macquarie Marshes, Menindee and Moama (near Mathoura) (Map).

The New South Wales sentinel chicken program was approved by the South West Area Health Service 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 veterinarian (usually the Director of Animal Care at Westmead) must inspect all new flock locations prior to deployment to ensure animal housing is adequate. 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 2003–2004 New South Wales Arbovirus Surveillance Program Annual Report.[24]

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

The 2010–11 season began with 118 pullets and five deaths were recorded during the program. A total of 2,300 samples were received from the eight flocks in New South Wales over the six-month period in 2010–2011. This represented 4,600 Enzyme-Linked ImmunoSorbant Assay (ELISA) tests (excluding controls and quality assurance samples), with each specimen being tested for MVEV and KUNV antibodies.

During the 2010–11 season, MVEV was first detected in February 2011 in the Macquarie Marshes and Bourke, and Kunjin in March 2011 in the Macquarie Marshes, Bourke and Forbes (Table 5).

Northern Territory

The sentinel chicken program in the Northern Territory is part of a national program involving Western Australia, New South Wales and Victoria and is designed to detect flavivirus activity (including the endemic arboviruses MVEV and KUNV, as well as exotic arboviruses such as JEV.[22] The current program in the Northern Territory commenced in January 1992 and replaced an earlier program run by Australian Quarantine Inspection Service (AQIS). Sentinel chicken flocks in the Northern Territory are maintained, bled and tested for flaviviruses in a combined program between the Department of Health, the virology laboratories of DPIFM and volunteers.

Sentinel chicken flocks are presently located at Leanyer, Howard Springs, Coastal Plains Research Station (CPRS), Katherine, Nhulunbuy, Tennant Creek, Jabiru, Alice Springs (two flocks), Nathan River and Alyangula. DPIFM officers or volunteers usually bleed flocks once a month and the samples are tested for MVEV and KUNV.

Table 5: Seroconversions to Murray Valley encephalitis virus and Kunjin virus in sentinel chicken flocks, New South Wales, 2010-11

Site	Seroconversions			First Positive Date	Last Positive Date
	MVEV	KUNV	Total		
Bourke	6	1	7	21 February 2011	21 March 2011
Forbes	1	2	3	17 March 2011	23 March 2011
Leeton	1	4	5	13 March 2011	17 April 2011
Macquarie marshes	2	3	5	21 February 2011	6 March 2011
Moama	0	1	1	6 April 2011	6 April 2011
Total	10	11	21		

Sentinel chickens are well located to detect flavivirus activity near the principal towns of the Northern Territory and hence provide timely and accurate indication of risk to people in those towns.

In the 2010–11 season, MVEV activity was detected in the Leanyer flock in April 2011, in the Adelaide River flock in May and June 2011, in the Tennant Creek flock in December 2010 and March 2011 and in the two Alice Springs flocks in April 2011 (Table 6).

KUNV activity was detected 22 times, and was present in all flocks between July 2010 and June 2011, except in the Alyangula and Alice Springs flocks (Table 6).

South Australia

Over the course of the 2010–11 summer period, South Australia was affected by the La Niña weather pattern which resulted in increased rainfall, elevated river levels and high levels of mosquito activity. Increased arboviral activity in both animals and humans was reported. In South Australia, notifications of mosquito-borne disease increased to the highest levels on record. For the first time since 1974, two cases of locally acquired MVEV infection were reported, with one resulting in the death of a 27 year old male.

Data obtained by the South Australian Health Department identified various regional and metropolitan locations as potential sources of infection, with a significant number of notifications being received from residents and visitors to the Riverland and Murraylands where mosquito-borne disease is endemic. Across South Australia, mosquito surveillance and control activities are conducted in partnership between the South Australian Health Department, University of South Australia, Local Government and Biosecurity SA. In response to predicted and emergent risks, seasonal mosquito monitoring and control activities were significantly expanded.

In South Australia in 2010–11, sentinel chicken flocks were established at Maree, Mulroona Station, Murray Bridge, Paringa, Blewitt Springs, and were screened between 10 November 2010 and 9 May 2011. The only seroconversion detected was in Paringa in April 2011.

In response to surveillance intelligence and disease notification data, the South Australian Health Department issued a number of health warnings and aggressively promoted the 'Fight the Bite' arbovirus prevention campaign. With the support of the South Australian Health Department, arbovirus prevention activities and mosquito control programs at the local government level were intensified, particularly in high risk areas.

Victoria

Flocks of chickens have been placed at ten locations (20 per flock) throughout the Murray River region in Victoria since the 1974 outbreak and act as an early warning system for possible human infection with flaviviruses. The chickens are bled weekly over the summer months (usually mid-October to April) and tested at the Department of Primary Industries.

Table 6: Seroconversions to Murray Valley encephalitis virus and Kunjin virus in sentinel chicken flocks, Northern Territory, 2010-11

Site	Seroconversions			First positive date	Last positive date
	MVEV	KUNV	Total§		
Howards Springs	0	3	4	29 Sep 2010	27 May 2011
Leanyer	1	6	7	21 July 2010	20 April 2011
Beatrice Hill	2	7	11	8 July 2010	2 June 2011
Jabiru	0	9	9	31 January 2011	13 June 2011
Nhulunbuy	0	2	4	25 July 2010	3 April 2011
Alyangula	0	0	2	25 August 2010	25 August 2010
Katherine Research Station	0	4	4	5 April 2011	28 May 2011
Nathan River	0	7	8	16 February 2011	4 May 2011
Tennant Creek	6	3	16	14 December 2010	8 May 2011
Arid Zone Research Station	2	0	3	14 April 2011	14 April 2011
Ilparpa	4	0	6	13 April 2011	13 April 2011
Total	15	41	74		

§ Includes seroconversions to flavivirus unspecified

In September 2010, parts of northern Victoria were affected by flood events following a number of years of drought conditions. There was also the possibility of future flooding events and increased humidity across Victoria during the 2010–11 summer. Given the predicted increase in mosquito breeding, the sentinel chicken program was brought forward with flocks placed on-site two weeks earlier than usual on 19 October 2010.

In January 2011, a major flood event occurred throughout the western and north western parts of the state. In response, additional adult mosquito and sentinel chicken surveillance was established in Hamilton, Horsham, Warracknabeal, Dimboola, Castlemaine and Bendigo (Map). This consisted of weekly adult mosquito trapping at two sites per council and weekly bleeding of the sentinel chickens. The chickens were on private properties, mainly show birds.

In February 2011, surveillance programs in the Murray Valley area detected the presence of MVEV in sentinel chickens. Antibodies to MVEV were first detected in the sentinel chicken flocks during week six of 2011 (beginning 6 February 2011). The flocks continued to seroconvert to flaviviruses through to week 18 (beginning 1 May 2011). MVEV activity was confirmed in chickens from Barmah, Bendigo, Cobram, Kerang, Mildura, Robinvale, Swan Hill, Toolamba and Tooleybuc (Table 7). Sixty-nine of the 260 sentinel chickens tested positive for flavivirus antibodies; 47 of these were MVEV specific.

The last detection of the virus was in Kerang in bloods collected on 9 May 2011 (Table 7).

Western Australia

The flavivirus sentinel chicken program in Western Australia is undertaken by the Arbovirus Surveillance and Research Laboratory (ASRL) at The University of Western Australia, on behalf of the Western Australian Department of Health. Many state and local government authorities and community volunteers also take part in the program. Thirty 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 (Map). Blood samples are collected from the chickens by environmental health officers or trained volunteers at fortnightly intervals. Samples are transported to the ASRL where they are tested for antibodies to flaviviruses using an epitope blocking ELISA.[25]

Rainfall prior to commencement of the 2010–11 wet season was generally well above average in northern Western Australia. Ex-Tropical Cyclone Abele created record rainfall in the Gascoyne and surrounding districts in mid-December 2010. The monsoon

Table 7: Seroconversions to Murray Valley encephalitis virus and Kunjin virus in sentinel chicken flocks, Victoria, 2010-11*

Site	Total number of birds that seroconverted	First Positive Date	Last Positive Date
Mildura	14	7 February 2011	7 March 2011
Mildura (new flock)	1	9 May 2011	9 May 2011
Robinvale #	12	13 February 2011	1 March 2011
Tooleybuc	6	7 February 2011	4 April 2011
Tooleybuc (new flock)	3	28 March 2011	18 April 2011
Swan Hill	1	7 March 2011	7 March 2011
Kerang	11	15 February 2011	15 March 2011
Kerang (new flock)	3	27 April 2011	9 May 2011
Barmah	11	8 February 2011	2 May 2011
Cobram	6	20 February 2011	1 May 2011
Rutherglen	Nil	n/a	n/a
Wodonga	5	23 March 2011	4 May 2011
Toolamba	8	16 February 2011	5 May 2011
Bendigo	4	2 March 2011	16 March 2011
Hamilton	Nil	n/a	n/a
Horsham	Nil	n/a	n/a
Total	85		

* 20 chickens at each site. Results include a combination of flavivirus unspecified and Murray Valley encephalitis virus.

remained very active in northern Western Australia between December and April, and the north of the state was affected by Tropical Cyclone Dianne and Tropical Cyclone Carlos. Above average rainfall was recorded across the state, with extensive flooding in many areas. Tropical Cyclone Errol brought more heavy rainfall to the Kimberley region in mid-April, and a cloud band in mid-May brought substantial rainfall to the Pilbara region. Cool, wet conditions occurred in the western Pilbara in June.

A total of 3,672 serum samples from 30 flocks were tested for antibodies to flaviviruses during 2010–11.[26] Seroconversions to flaviviruses were detected 237 (6.5%) samples compared with 16/3,941 samples (0.4%) in 2009–10.[27] Two MVEV seroconversions detected at Derby in July and one KUNV infection at Fitzroy Crossing in August and Exmouth in September were associated with continuing activity from the 2009–10 season.

The first activity associated with the 2010–11 wet season occurred in February 2011, when KUNV infections were detected in sentinel chickens at Fitzroy Crossing in the west Kimberley region and MVEV infections were detected at Wyndham (North East Kimberley), Halls Creek (South East Kimberley), Beagle Bay (West Kimberley) and Tom Price (Pilbara). Very high levels of flavivirus activity were subsequently detected throughout the Kimberley, Pilbara and Gascoyne regions in March, and the activity continued in April (Kimberley and Pilbara), May (Kimberley, Pilbara, Midwest/Wheatbelt and Goldfields) and June (Pilbara). Overall there were 226 seroconversions to MVEV and 14 KUNV infections, including seven dual MVEV/KUNV infections. The detections of MVEV activity at Leonora in the Goldfields and Dongara in the Midwest/Wheatbelt are the first in these regions since the extensive southerly activity of MVEV in 2000, and the overall level of flavivirus activity was similar to the levels seen in 2000.[28] The majority of sentinel chicken flocks required replacement with new chickens during the course of the season.

The Western Australian Department of Health issued five media statements. The first was issued on 24 September 2010 following continued detections of MVEV antibodies in sentinel chickens in the Kimberley region, and KUNV antibodies in chickens in the Pilbara and Gascoyne regions. The second was issued on 25 February 2011 after KUNV infections were detected in sentinel chickens in the Kimberley region for the first time in the 2010–11 wet season. The third media release was issued on 25 March 2011 after widespread detections of MVEV infections in sentinel chickens in the Kimberley, Pilbara and Gascoyne regions, the first evidence of MVEV activity in Western Australia for the 2010–11 season. The fourth media release was issued on 12 April 2011 after the diagnosis of a case of MVEV in Carnarvon and continued detections of antibodies to MVEV and KUNV in sentinel chickens in the Kimberley, Pilbara and Gascoyne regions. A fifth media warning was issued on the 16 May 2011 due to new detections of MVEV and KUNV infections in sentinel chickens in the Midwest/Wheatbelt and Goldfields regions, and six cases of MVEV infection including one fatality.

Tasmania

No viruses were isolated in 2010–11 in mosquitoes trapped during *ad hoc* collections undertaken in 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 arboviral diseases are then made nationally notifiable. An unspecified category is particularly important for the flaviruses, because it is recognised that some infections cannot be attributed to a single flavivirus.

There were 22 notifications of arbovirus NEC in 2010–11, compared with a five-year average of 16 cases (range 4 to 33 cases). Most of these notifications relate to infections that had been acquired overseas (n=17). In 2010–11, 16 notifications were of flavivirus (unspecified), with the remainder due to the flaviviruses Kokobera (n=3) and Stratford (n=1) and unknown arboviruses (n=2).

Malaria

Malaria is a serious acute febrile illness that is normally transmitted from person to person through the bite of an infected mosquito. It is caused by a protozoan parasite in the genus *Plasmodium* that includes five species that infect humans, *Plasmodium vivax*, *Plasmodium falciparum*, *Plasmodium malariae*, *Plasmodium ovale* and *Plasmodium knowlesi*.[29,30]

There were 414 notifications of malaria during the season 2010–11 (Table 1), a 27 per cent decrease compared with the mean of 570.6 notifications per year during the previous five years, consistent with the steady decline in the number of notifications since the 2004–05 season. The decline has been in both overseas acquired and locally acquired cases.

There were 7 notified locally-acquired cases associated with an outbreak of *P. falciparum* in Saibai and Duan Islands in the Torres Strait during March and April 2011.[31] The last outbreak of locally-acquired malaria on the Australian mainland occurred in North Queensland during 2002.[32]

Malaria was most frequently reported amongst people aged 25-29 years, with 57 notified cases. Similar to previous years, the majority of cases were in males (72%, n=299). Cases were from all jurisdictions. No deaths from malaria were reported during the 2010–11 season.

The infecting species was reported for 96% of notifications during the season 2010–11. *P. falciparum* and *P. vivax* were the predominant infecting species (Table 8). In 2010–11, no cases were infected with *P. knowlesi*.

Complete information about the country of acquisition was available for 341 (82%) malaria cases. Papua New Guinea was the most frequently reported place of acquisition, with 26% of cases (108/414), followed by India with 14% cases (58/414) (Figure 8).

P. vivax infections were commonly associated with travel to Asia or pacific nations (82%, 146/179). *P. falciparum* infections were frequently associated with travel to sub-Saharan, Central, West, Southern and East Africa (44%, 85/192), and only three *P. vivax* infections (2%) were associated with travel to these regions.

Table 8: Malaria cases, 2010-11, by *Plasmodium* species

Malaria species	Number of cases	%of all cases
Plasmodium falciparum	186	45%
Plasmodium vivax	175	42%
Plasmodium ovale	18	4%
Plasmodium malariae	14	1%
P. falciparum and P. vivax	4	<1%
P. falciparum and P. ovale	2	0%
Plasmodium spp.	15	4%
Total	414	100%

Other surveillance and research activities

Exotic mosquito detections at the border

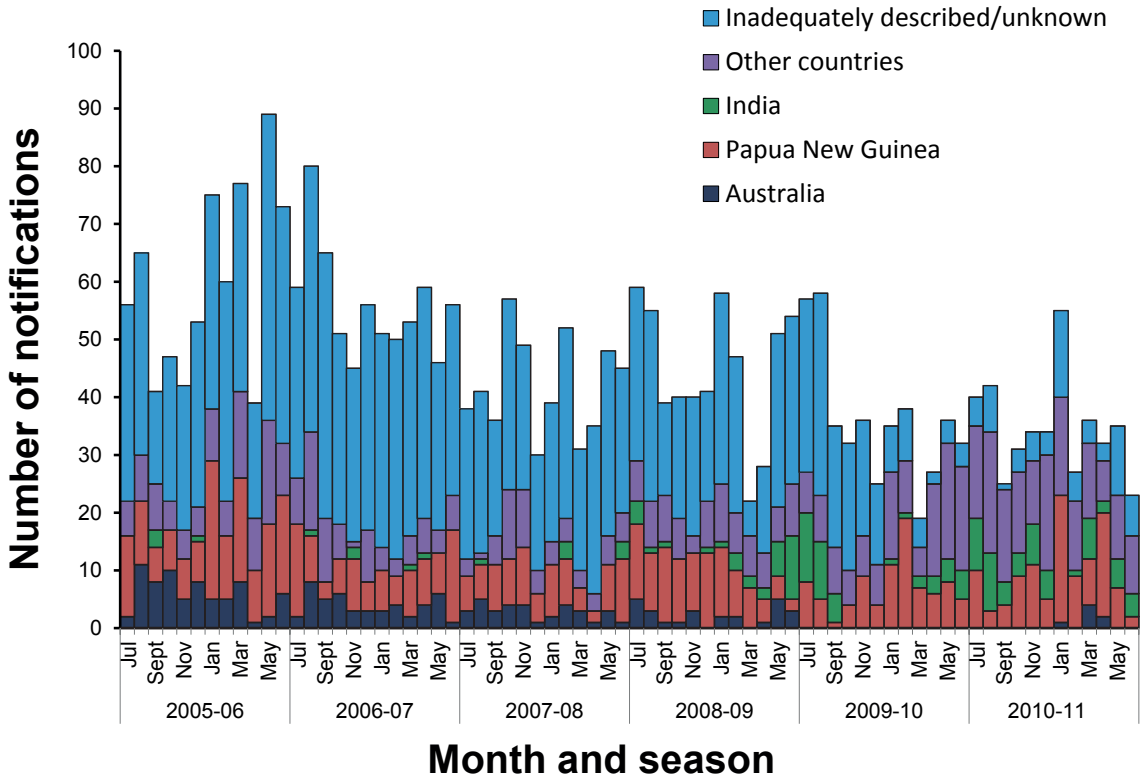
Between July 2010 and June 2011 there were five exotic mosquito detections made by the Department of Agriculture, Fisheries and Forestry (DAFF) at the Australian border compared with eight for the 2009–10 period. Two detections were made via routine quarantine inspection of imported cargo while the remaining three detections were made

Table 9: Exotic mosquito detections at the border, Australia, 2010-11

Date	Location	Species	Method of Detection	Source / Origin	Action/ Mitigation	Surveillance Results
July 2010	Port Hedland Seaport	*Ae. aegypti* (5 larvae)	Inspection	Pre-fabricated steel from China	Chlorination of goods, increased trapping	No further exotic mosquitoes detected
Dec 2010	Darwin Seaport	*Ae. albopictus* (3 larvae)	Inspection	Wooden reel of steel wire	Chlorination of goods, increased trapping	No further exotic mosquitoes detected
Jan 2011	Darwin Seaport	*Ae. aegypti* (3 adults)	CO_2 baited BG trap	Unknown/ unable to identify source	ULV fogging, receptacle treatment surveys, increased trapping	No further exotic mosquitoes detected
Jan 2011*	Thursday Island Seaport	*Ae. albopictus* (larvae)	Sentinel tyre trap	Spread from surrounding Torres Strait Islands	Port area included in control activities already being performed by QLD Health on the island.	No further exotic mosquitoes detected
Mar 2011	Darwin Seaport	*Ae. albopictus* (1 adult)	CO_2 baited BG trap	Unknown/ unable to identify source	ULV fogging, receptacle treatment surveys, increased trapping	No further exotic mosquitoes detected

* *Ae. albopictus* was collected on Thursday Island in December 2010 by QLD Health. However this detection was made on the opposite side of Thursday Island to that made by DAFF.

Figure 8: Number of notified cases of malaria, Australia, July 2005 to June 2011, by month, year and place of acquisition



through routine vector monitoring at international ports (Table 9). No further exotic mosquitoes were collected following the initial detections with the exception of *Ae. albopictus* on Thursday Island. Control activities are ongoing.

Torres Strait Ae. 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.[33] This mosquito is capable of transmitting dengue and CHIKV, 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 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. *Ae. albopictus* was detected on Thursday Island for the first time during a Nov-Dec 2010 field trip (one larval sample) and there were further detections on subsequent trips. On Horn Island, small populations of *Ae. albopictus* continue to occur, despite control efforts. There were no detections of *Ae. albopictus* on the mainland in 2010–11.

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 2010 to 30 June 2011, and activities undertaken by health authorities in response to human cases, as well as evidence of virus activity. Sentinel animal and vector monitoring continue to be an important part of the early warning system for arbovirus activity in Australia.

Rates and counts of RRV infection and counts of dengue fever were notably increased compared with historical totals. There were more than three times as many overseas–acquired dengue cases during the 2010–11 season as the average number during the previous five years; two thirds of these cases were in travellers returning to Australia from Indonesia.

The number and proportion of dengue cases that are 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 the increase.[34] Viraemic returning travellers or visitors from overseas present a risk of starting a local outbreak in North Queensland. 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.[35]

The risk of dengue becoming established 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. There has not been a large outbreak (>100 cases) of dengue in Australia since the 2008–09 season, when there was an outbreak of DENV-3 with 915 cases in Cairns that lasted for 31 weeks.[12] In 2011, Queensland Health released the Queensland Dengue Management Plan 2010–2015 which outlines current best practice in dengue management for the four levels of dengue activity; ongoing prevention, response to sporadic cases, outbreak response and multiple outbreaks.[36]

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. Control efforts through the Torres Strait *Ae. albopictus* Elimination and Control Program are vital to prevent incursions on the mainland. In mid-2011, small populations of *Ae. albopictus* continued to be maintained on Horn Island despite control efforts. Since that time, the program has been demonstrably successful at reducing *Ae. albopictus* numbers in the *cordon sanitaire* to levels where elimination is now a real possibility.

Researchers at the University of Queensland and international collaborators are trialling a novel biological control agent for mosquito-borne diseases such as dengue fever. The mosquito vectors are infected with a naturally occurring intracellular bacterium *Wolbachia pipientis* wMel strain which inhibits dengue transmission while conferring only small fitness cost to the mosquito.[37] In January 2011, researchers commenced a field trial with wMel Wolbachia-infected mosquitoes in January 2011 at Gordonvale and Yorkeys Knob in North Queensland with strong community support. Ongoing monitoring shows that Wolbachia is still present in almost 100% of all Ae. aegypti mosquitoes in these sites.[38] Based on the success of these trials, this program is being expanded to other countries (Indonesia, Vietnam and Brazil) where dengue is endemic.

Over the spring and summer of 2010–11 the southeast of Australia experienced unusually wet weather and flooding resulting in increased mosquito and wild bird populations. In South Australia and Victoria in the 2010–11 season, large increases in reported cases and rates of BFV and RRV were noted along with evidence of MVEV activity. There were two confirmed MVEV cases in South Australia. These increases occurred concurrently with outbreaks of arboviral disease in horses, particularly RRV and Kunjin. While KUNV infections were widely diagnosed in horses in southern Australia in early 2011, there were no human cases except for one case in the Northern Territory in April. The Victorian Infectious Diseases Reference Laboratory conducted real-time opportunistic serological surveillance for MVEV on referred human sera from the Murray River between February and May 2011. No individuals born after 1974 had antibodies to MVEV and the seroprevalence was comparable to background rates.[39] Differences in laboratory diagnostic practices between human and veterinary health may in part account for marked difference between case numbers in humans and animals in 2011, and there is a need to ensure the equivalence of case definitions and laboratory practices for the confirmation of zoonotic arboviruses in Australia.

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 Murray Valley encephalitis virus in Australia, emphasising a One-Health approach, along with guidance for public health units as part of the CDNA Series of National Guidelines (SoNGs). This work is in progress.

While the two cases of yellow fever reported from Queensland in 2010–11 met the CDNA national surveillance case definition, both were likely to have been vaccine related. A revised CDNA case definition for yellow fever which came into effect on 1 January 2013 excludes vaccine-associated cases. Under the revised case definition, laboratory evidence provided by serology is required to be in the absence of recent vaccination (in the previous 3 weeks for seroconversion and in the previous three months for a single IgM to yellow fever in the absence of IgM to other flaviviruses).

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 2010 NNDSS annual report.[40] 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 assume a clinically compatible illness. A case can still be notified when clinical illness may not be consistent with

the diagnosis of alphavirus infection. Cross-reacting IgM between RRV and BFV is a known issue, and from 1 January 2013, revised case definitions for RRV and BFV were implemented to address this. Under the revised case definition, a diagnosis for BFV based on IgM with requires the absence of IgM to RRV, and vice versa for the diagnosis of RRV. Alternatively, diagnosis can be based on IgM in the presence of IgG to that same virus. 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. 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.

Acknowledgements

NAMAC members are (in alphabetical order): Bart Currie, Peter Daniels, Stephen Doggett, Jenny Firman, Katrina Knope, Ann Koehler, Rogan Lee, Mike Lindsay, John Mackenzie, Rodney Moran, Scott Ritchie, Richard Russell, Christine Selvey, Angus Sly, David Smith, Peter Whelan, Craig Williams, Jennifer Wall and Phil Wright (Secretariat).

The data on which this report is based is the work of many people. We thank the public health laboratories, State and Territory communicable disease control units and public health units and staff in State and Territory arbovirus surveillance and monitoring programmes.

Author details

Katrina Knope[1]
Peter Whelan[2]
David Smith[3]
Cheryl Johansen[4]
Rod Moran[5]
Stephen Doggett[6]
Angus Sly[7]
Michaela Hobby[8]
Phil Wright[1]
Jay Nicholson[4]
The National Arbovirus and Malaria Advisory Committee (see acknowledgement)

1. Zoonoses, Foodborne and Emerging Infectious Diseases Section
   Health Emergency Management Branch
   Office of Health Protection
   Department of Health and Ageing
   Canberra ACT
2. Medical Entomology, Centre for Disease Control
   Health Protection Division, Northern Territory Department of Health
   Building 19, Royal Darwin Hospital. Casuarina NT 0811
3. Division of Microbiology and Infectious Diseases; PathWest QEII Medical Centre.
   School of Pathology and Laboratory Medicine, Faculty of Medicine, Dentistry and Health Sciences, University of Western Australia
4. Arbovirus Surveillance and Research Laboratory
   School of Pathology and Laboratory Medicine
   Faculty of Medicine, Dentistry and Health Sciences
   University of Western Australia, M502
5. No affiliation (retired)
6. New South Wales Arbovirus Surveillance & Mosquito Monitoring Program
   Department of Medical Entomology
   Institute for Clinical Pathology and Medical Research
   Westmead Hospital, Sydney NSW
7. Operational Science Program (OSP)
   Department of Agriculture, Fisheries and Forestry
   Border Compliance Division
   42-44 Qantas Drive, Eagle Farm, Brisbane, QLD 4009
8. Health Protection, Public Health, SA Health Government of South Australia

Correspondence:

Katrina Knope
Zoonoses, Foodborne and Emerging Infectious Diseases Section
Health Emergency Management Branch
Office of health Protection
Department of Health and Ageing
Phone: +61 2 6289 2751
katrina.knope@health.gov.au

References

1. Australian Bureau of Statistics. 3101.0 Australia Demographic Statistics, June 2011. Available from http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/3101.0Jun%202011.
2. Forbes J. Murray Valley encephalitis 1974. also The epidemic variance since 1914 and predisposing rainfall patterns. Sydney; 1978.
3. Nicholls N. A method for predicting Murray Valley encephalitis in south-east Australia using the Southern Oscillation. *Aust J Exp Bioi Mod Sei* 1986;64:587-594.
4. Russell RC, Dwyer DE. Arboviruses associated with human disease in Australia. *Microbes Infect* 2000;2(14):1693-1704.
5. Mackenzie JS, Lindsay MD, Coelen RJ, Broom AK, Hall RA, Smith DW. Arboviruses causing human disease in the Australasian zoogeographic region. *Arch Virol* 1994;136(3-4):447-467.
6. Parida MM, Santhosh SR, Dash PK, Lakshmana Rao PV. Rapid and Real-time Assays for Detection and Quantification of Chikungunya Virus. *Future Virol* 2008;3(2):179-192.
7. Harrington S, Lindsay MD, Douglas A. Christmas Island and Cocos (Keeling) Islands, Indian Ocean: Mosquito fauna and mosquito-borne disease risk assessment and management recommendations. Final report of investigations undertaken in 2007-08: Public Health Division, Western Australian Department of Health; 2009.
8. van den Hurk AF H-MS, Pyke AT, Smith GA, Mackenzie JS, Vector competence of Australian mosquitoes for chikungunya virus. *Vector Borne Zoonotic Dis* 2010;10(5):489-495.

9. Australian Technical Advisory Group on Immunisation, Australian Government Department of Health and Ageing. *The Australian Immunisation Handbook*. 9th edn. Canberra: Australian Government; 2008.
10. Hanna JN, Ritchie SA, Richards AR, Humphreys JL, Montgomery BL, Ehlers GJ, et al. Dengue in north Queensland, 2005-2008. *Commun Dis Intell* 2009;33(2):198-203.
11. Queensland Health. Dengue Fever management plan for North Queensland 2005-2010 Cairns, Queensland: Tropical Public Health Unit Network, Queensland Health; 2005.
12. Ritchie S. Outbreaks of dengue in North Queensland. Unpublished data 2012.
13. Whelan P, Nguyen H, Hajkowicz K, Davis J, Smith D, Pyke A, et al. Evidence in Australia for a Case of Airport Dengue. *PLoS Negl Trop Dis* 2012;6(9):e1619.
14. Clark B, Molton JS, Habib T, Williams DT, Weston EL, Smith DW, Dengue virus infection in Australia following occupational exposure: A reflection of increasing numbers of imported cases. *J Clin Virol* 2012;54(4):376-377.
15. Fitzsimmons GJ, Wright P, Johansen CA, Whelan PI. Arboviral diseases and malaria in Australia, 2008-09: annual report of the National Arbovirus and Malaria Advisory Committee. *Commun Dis Intell* 2010;34(3):225-240.
16. Hanna JN, Ritchie SA, Phillips DA, Lee JM, Hills SL, van den Hurk AF, et al. Japanese encephalitis in north Queensland, Australia, 1998. *Med J Aust* 1999;170(11):533-536.
17. Speers DJ, Flexman J, Blyth OC, Rooban N, Raby E, Ramaseshan G, Benson S, and Smith DW. Clinical and Radiological Predictors of Outcome for Murray Valley Encephalitis, Am J Trop Med Hyg 2013: 12-0379.
18. Knox J, Cowan RU, Doyle JS, Ligtermoet MK, Archer JS, Burrow JNC, Tong SYC, Murray Valley encephalitis: a review of clinical features, diagnosis and treatment. Medical Journal of Australia 2012.
19. Roche S WR, Garner M, East I, Paskin R, Moloney B, Carr M, Kirkland P. Descriptive overview of the 2011 epidemic of arboviral disease in horses in Australia. *Aust Vet* J 2013;91(1-2):5-13.
20. Roche S, Garner, Graeme, Paskin, Roger, Paskin, Equine Arbovirus Working Group Epidemiology Subgroup,. Epidemiological Report on Equine Arboviral Events February - August 2011 for Animal Health Committee 30 September 2011. Unpublished report.
21. Frost MJ, Zhang J, Edmonds JH, Prow NA , Gu X, Davis R, Hornitzky C, Arzey KE, Finlaison D, Hick P, Read A, Hobson-Peters J, May FJ, Doggett SL, Haniotis J, Russell RC, Hall RA, Khromykh AA, Kirkland PD. Characterization of Virulent West Nile Virus Kunjin Strain, Australia, 2011. *Emerg Infect Dis*;18(5):792–800.
22. Broom AK, Azuolas J, Hueston L, Mackenzie JS, Melville L, Smith DW, et al. Australian encephalitis: Sentinel Chicken Surveillance Programme. *Commun Dis Intell*;25(3):157-160.
23. Broom AK. Sentinel Chicken Surveillance Program in Australia, July 2002 to June 2003. *Commun Dis Intell* 2003;27(3):367-369.
24. Doggett S, Clancy L, Haniotis J, Russell RC, Hueston L, Marchetti M, Dwyer D. The New South Wales Arbovirus Surveillance & Mosquito Monitoring Program. 2003 – 2004 Annual Report. Department of Medical Entomology, Westmead. 23pp.; 2004.
25. Hall RA, Broom AK, Hartnett AC, Howard MJ, Mackenzie JS. Immunodominant epitopes on the NS1 protein of MVE and KUN viruses serve as targets for a blocking ELISA to detect virus-specific antibodies in sentinel animal serum. *J Virol Methods* 1995;51(2-3):201-210.
26. Johansen C, Nicholson J, Power S, Cashen C, Wong S, McFall S, Wallace M, Burley M, Smith D, Shellam G. The University of Western Australia Arbovirus Surveillance and Research Laboratory Annual Report: 2010-2011. Discipline of Microbiology and Immunology, The University of Western Australia; 2010, 84p. 2011.
27. Wright P, Fitzsimmons GJ, Johansen CA, Whelan PI, National Arbovirus and Malaria Advisory Committee. Arboviral diseases and malaria in Australia, 2009-10: annual report of the National Arbovirus and Malaria Advisory Committee. *Commun Dis Intell* 2012;36(1):70-81.
28. Cordova SP, Smith DW, Broom AK, Lindsay MD, Dowse GK, Beers MY. Murray Valley encephalitis in Western Australia in 2000, with evidence of southerly spread. *Commun Dis Intell* 2000;24(12):368-372.
29. Heymann D, editor. *Control of Communicable Diseases Manual*. 18th edn. Washington: American Public Health Association; 2004.
30. Cox-Singh J, Davis TM, Lee KS, Shamsul SS, Matusop A, Ratnam S, et al. *Plasmodium knowlesi* malaria in humans is widely distributed and potentially life threatening. *Clin Infect Dis* 2008;46(2):165-171.
31. Preston-Thomas A, Gair RW, Hosking KA, Devine GJ, Donohue SD. An outbreak of *Plasmodium falciparum* malaria in the Torres Strait *Commun Dis Intell* 2012;26(2).
32. Hanna JN, Ritchie SA, Eisen DP, Cooper RD, Brookes DL, Montgomery BL. An outbreak of *Plasmodium* vivax malaria in Far North Queensland, 2002. *Med J Aust* 2004;180(1):24-28.
33. Ritchie SA, Moore P, Carruthers M, Williams C, Montgomery B, Foley P, et al. Discovery of a widespread infestation of *Aedes albopictus* in the Torres Strait, Australia. *J Am Mosq Control Assoc* 2006;22(3):358-365.
34. Knope K, National Arbovirus and Malaria Advisory Committee, Giele C. Increasing notifications of dengue related to overseas travel, 1991 to 2012. *Commun Dis Intell* 2013; Early release ahead of vol 37 (1).
35. Australian Government Department of Foreign Affairs and Trade. Smartraveller: The Australian Government's travel advisory and consular assistance service. Accessed on 24 December 2012. Available from: http:\\www.smartraveller.gov.au
36. Queensland Health. Queensland Dengue Management Plan 2010-2015. In; 2011.
37. Walker T, Johnson PH, Moreira LA, Iturbe-Ormaetxe I, Frentiu FD, McMeniman CJ, et al. The wMel *Wolbachia* strain blocks dengue and invades caged *Aedes aegypti* populations. *Nature* 2011;476(7361):450-453.
38. Hoffmann AA, Montgomery BL, Popovici J, Iturbe-Ormaetxe I, Johnson PH, Muzzi F, et al. Successful establishment of *Wolbachia* in Aedes populations to suppress dengue transmission. *Nature* 2011;476(7361):454-457.
39. Joseph S Doyle SN, Jennifer A Leydon, Rodney J Moran and Michael G Catton. Opportunistic serological surveillance for Murray Valley encephalitis virus in Victoria, February–May 2011. *Med J Aust* 2012;197(3):150.
40. NNDSS Annual Report Writing Group. Australia's notifiable disease status, 2010: annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell* 2012;36(1).

Annual reports

Annual Immunisation Coverage Report, 2010

Brynley Hull, Aditi Dey, Rob Menzies, Peter McIntyre

Abstract

This, the fourth annual immunisation coverage report, documents trends during 2010 for a range of standard measures derived from Australian Childhood Immunisation Register (ACIR) data. These include coverage at standard age milestones and for individual vaccines included on the National Immunisation Program (NIP). For the first time, coverage from other sources for adolescents and the elderly are included.

The proportion of children 'fully vaccinated' at 12, 24 and 60 months of age was 91.6%, 92.1% and 89.1% respectively. For vaccines available on the NIP but not currently assessed for 'fully immunised' status or for eligibility for incentive payments (rotavirus and pneumococcal at 12 months and meningococcal C and varicella at 24 months) coverage varied. Although pneumococcal vaccine had similar coverage at 12 months to other vaccines, coverage was lower for rotavirus at 12 months (84.7%) and varicella at 24 months (83.0%). Overall coverage at 24 months of age exceeded that at 12 months of age nationally and for most jurisdictions, but as receipt of varicella vaccine at 18 months is excluded from calculations, this represents delayed immunisation, with some contribution from immunisation incentives. The 'fully immunised' coverage estimates for immunisations due by 60 months increased substantially in 2009, reaching almost 90% in 2010, probably related to completed immunisation by 60 months of age being introduced in 2009 as a requirement for GP incentive payments. As previously documented, vaccines recommended for Indigenous children only (hepatitis A and pneumococcal polysaccharide vaccine) had suboptimal coverage at around 57%. Delayed receipt of vaccines by Indigenous children at the 60-month milestone age improved from 56% to 62% but the disparity in on-time vaccination between Indigenous and non-Indigenous children at earlier age milestones did not improve. Coverage data for human papillomavirus (HPV) from the national HPV register are consistent with high coverage in the school-based program (73%) but were lower for the catch-up program for women outside school (30-38%). Coverage estimates for vaccines on the NIP from 65 years of age were comparable with other developed countries.

Keywords: immunisation coverage, immunisation delay, small area coverage reporting, human papilloma virus vaccine coverage

Introduction

This is the fourth Annual Immunisation Coverage Report, following the first on 2007 data published in 2009.[1] This series of annual reports was established to consolidate the various forms of regular coverage reports and ad-hoc publications produced by the National Centre For Immunisation Research and Surveillance (NCIRS) using ACIR data, highlighting important trends and significant issues over the preceding 12 months.[1-16] It follows the format of the previous reports, providing a detailed summary for 2010 that includes vaccination coverage at standard milestone ages. It also includes coverage for vaccines not included in standard coverage assessments, timeliness of vaccination, coverage for Indigenous children and data for small geographic areas on vaccination coverage and prevalence of conscientious objectors. This report includes data on adolescents for the first time and the elderly age groups not on the ACIR, from previously published sources. Readers are referred to the first report for a more detailed explanation of the background to this series of annual reports and the range of analyses presented.[1] This report uses the longstanding international practice of reporting coverage at key milestone ages, to measure coverage against national targets and to track trends over time. No new vaccines were introduced to the NIP during 2010. However, in late 2009 in the Northern Territory, the 10-valent pneumococcal conjugate vaccine at 2, 4, 6 and 12 months of age replaced the 7-valent conjugate (7vPCV) and 23-valent polysaccharide (23vPPV) vaccines.

Incentives for vaccination and reporting to the ACIR

The Australian Government, through the Department of Health and Ageing, advises the ACIR whether calculation of coverage of the new vaccines/antigens should be included in the completed schedule assessment for eligibility for payments to parents or immunisation providers. The ACIR makes information payments (up to $6) to all immunisation

providers and general practitioners (GPs). Payments to GPs for the provision of data to the ACIR have been in place since its inception in 1996. In the 2008-09 Budget, the Australian Government announced that one of the components of the General Practice Immunisation Incentives Scheme (GPII), the GPII Service Incentive Payment (SIP), would stop from 1 October 2008. SIP payments of $18.50 were made to GPs for reporting a vaccination which completed a schedule point on the NIP, at 6, 12, 18 months and 60 months.[17] However, the GPII Outcomes Payments,* which paid practices that achieved 90 per cent or greater levels for full immunisation, were maintained. The vaccines/antigens required for full immunisation in assessment for the outcomes payment in 2010 were the same as in recent years, ie. diphtheria, *Haemophilus influenzae* type b (Hib), hepatitis B, measles, mumps, pertussis, polio, rubella and tetanus. Vaccines included on the NIP in 2010 but not part of the completed schedule assessment for provider payments were: meningococcal C vaccine (Men C); 7vPCV; and rotavirus vaccine. Varicella vaccine was also not included for coverage assessment but eligible providers received an information and SIP payment (up to October 2008)* for reporting, as varicella vaccine is currently the only vaccine required for completion of the 18-month schedule point. While the ACIR records vaccines given only to Indigenous children in Queensland, Northern Territory, Western Australia and South Australia (hepatitis A and 23vPPV and vaccines not included in the NIP such as BCG, reporting of these vaccines does not attract a GPII payment.[1]

Table 1 shows the Australian National Immunisation Program Schedule for 2010.

In 2004-05, the means test to qualify for the Maternity Immunisation Allowance† (MIA was removed. This payment, of $233 per child in 2008, was intended to provide motivation both to complete immunisation and for parents to prompt their provider to notify any outstanding reports to the ACIR before the child reaches 24 months of age. In the 2008-09 budget, in addition to the changes mentioned above, it was announced that the MIA payment would be paid in 2 equal amounts of $167, with eligibility for the 2nd payment assessed between 48 and 60 months of age. This came into effect in January 2009, through a change in the National Due and Overdue Rules for Childhood Immunisation for all children born from 1 January 2005 onwards. They now state that a child is due for their 48-month old vaccinations at 48 months and overdue at 49 months of age, instead of overdue at 60 months of age.

Methods

The Australian Childhood Immunisation Register

The ACIR was established on 1 January, 1996, by incorporating demographic data from Medicare

* GPII Outcomes Payments will cease on 30 June 2013.

† The MIA ceased in 2012 and was replaced from 1 July 2012 by a new immunisation check for one year olds to supplement the existing focus on immunisation at two and five years of age. Eligibility for the Family Tax Benefit Part A supplement will require that children are assessed as fully immunised.

Table 1: Australian National Immunisation Program Schedule for children in 2010[29]

Age	Vaccine									
Birth	Hep B									
2 months	Hep B	DTPa	Hib	Polio				7vPCV		Rotavirus
4 months	Hep B	DTPa	Hib	Polio				7vPCV		Rotavirus
6 months	Hep B	DTPa	Hib‡	Polio				7vPCV		Rotavirus§
12 months			Hib		MMR		Hep A‖		Men C	
18 months						VZV	Hep A‖ ¶	23vPPV‖		
24 months							Hep A¶	23vPPV¶		
60 months		DTPa		Polio	MMR					

‡ 3rd dose of Hib vaccine at 6 months is dependent on vaccine brand used in state or territory

§ 3rd dose of rotavirus vaccine at 6 months is dependent on vaccine brand used in state or territory

‖ Aboriginal and Torres Strait Islander children in Western Australia and the Northern Territory

¶ Aboriginal and Torres Strait Islander children in Queensland and South Australia

on all enrolled children under the age of 7 years.[5] Participation in the ACIR is opt-out hence it constitutes a nearly complete population register, as approximately 99% of children are registered with Medicare by 12 months of age.[5] Children not enrolled with Medicare can also be added to the ACIR via a supplementary number. Since 2001, immunisations given overseas may be recorded if a provider endorses their validity. Data are transferred to the ACIR when a recognised immunisation provider supplies details of an eligible immunisation either through the internet using the Medicare Australia website or by submitting paper encounter forms which are scanned at a central location. The existence of medical contraindications and conscientious objection to immunisation we are also recorded on the ACIR. All vaccination records for a child remain on the register indefinitely, but no new immunisation encounter records are added after the 7th birthday.

Immunisations recorded on the Register must be administered in accordance with the guidelines issued by the National Health and Medical Research Council as stated in *The Australian Immunisation Handbook*.[18] Notifications falling outside these guidelines or duplicate notifications prompt an enquiry with the provider and, if their validity cannot be established, they are rejected.

Measuring immunisation coverage using the Australian Childhood Immunisation Register

The cohort method has been used for calculating coverage at the population level (national and state/territory)[19] since the ACIR's inception. Cohort immunisation status is assessed at 12 months of age (for vaccines due at 6 months), 24 months of age (for vaccines due at 12 months), and 60 months of age (for vaccines due at 48 months). A minimum 3-month lag period is allowed for late notification of immunisations to the Register, but only immunisations given on or before a child's 1st, 2nd or 5th respective birthdays are considered.[19] If a child's records indicate receipt of the last dose of a vaccine that requires more than 1 dose to complete the series, it is assumed that earlier vaccinations in the sequence have been given. This assumption has been shown to be valid.[7,8]

Three-month birth cohorts are used for time trend analyses, while 12-month wide cohorts are used for other analyses in this report such as for small area coverage analysis and mapping of coverage estimates. A minimum 3-month lag is allowed for late notifications. These cohorts are children born between 1 January and 31 December 2009 for the 12-month milestone age; children born between 1 January and 31 December 2008 for the 24-month milestone age; and children born between 1 January and 31 December 2005 for the 60-month milestone age.

The proportion of children designated as 'fully immunised' is calculated using the number of children completely immunised with the vaccines of interest by the designated age as the numerator, and the total number of Medicare-registered children in the age cohort as the denominator. 'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of a 3rd dose of a diphtheria (D), tetanus (T), and acellular pertussis containing (P) vaccine(DTPa), a 3rd dose of polio vaccine, a 2nd or 3rd dose of a PRP-OMP containing (Hib) vaccine or a 3rd dose of any other Hib vaccine, and a 2nd or 3rd dose of a Comvax hepatitis B vaccine or a 3rd dose of dose of any other hepatitis B vaccine. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of a 3rd or 4th dose of a DTPa vaccine, a 3rd dose of polio vaccine, a 3rd or 4th dose of a PRP-OMP containing Hib vaccine or a 4th dose of any other Hib vaccine, a 3rd or 4th dose of Comvax hepatitis B vaccine or a 4th dose of any other hepatitis B vaccine, and a 1st 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 a 4th or 5th dose of a DTPa vaccine, a 4th dose of polio vaccine, and 2nd dose of a measles, mumps and rubella-containing (MMR) vaccine.

Immunisation coverage estimates were also calculated for individual NIP vaccines, including the 6 NIP vaccines not routinely reported in *Communicable Diseases Intelligence (*CDI). They were: a 3rd dose of 7vPCV and 2nd or 3rd dose of rotavirus vaccine by 12 months of age; a 1st dose of varicella vaccine and a 1st dose of meningococcal C vaccine by 24 months of age; a 2nd dose of hepatitis A vaccine in Indigenous children by 30 or 36 months of age; and a dose of 23vPPV vaccine in Indigenous children by 36 months of age.

Changes to immunisation policy and changes to the 'fully immunised' coverage algorithms have had an impact on vaccination coverage presented in this report. From the September 2009 coverage assessment date onwards, changes were made in the coverage calculation algorithms that tightened the rules regarding receipt of Hib and hepatitis B vaccines for children aged 12 months of age to lead to more accurate measures of coverage in Australia for these two vaccines. Prior to September 2009, if a child had a record on the ACIR of a 2nd or 3rd dose of any child Hib vaccine then they were considered 'fully vaccinated' for this vaccine. From September 2009, a child needed a record on the ACIR of a 3rd dose of any child Hib vaccine or a 2nd dose of either PedvaxHIB or Comvax to be assessed as 'fully vaccinated' for this vaccine. Prior to September 2009, if a child had a record on the ACIR of a 2nd or 3rd dose of any child hepatitis B vaccine then they were considered

'fully vaccinated' for this vaccine. From September 2009, a child needed a record on the ACIR of a 3rd dose of any child hepatitis B vaccine or a 2nd dose of either Engerix B (paediatric), Comvax, or HBVAX II (paediatric) to be assessed as 'fully vaccinated' for this vaccine. In October 2009, a recommendation was made by the Australian Technical Advisory Group on Immunisation that the 4th dose of DTPa containing vaccine can be given from 42 months of age instead of the previously recommended 48 months of age. In March 2009, another recommendation was made by the Australian Technical Advisory Group on Immunisation to parents and immunisation providers to consider bringing the first dose of DTPa forward to 6 weeks of age to provide earlier protection against pertussis infection. In January 2009, changes were made to the overdue rules so that children were classified as overdue for pre-school boosters at 49 months instead of the previous 60 months of age. This applied to parental and provider incentive payments. In December 2007, the coverage algorithm for immunisations due at 48 months of age was changed to assess children at 60 months, not 72 months of age.

Timeliness

Age-appropriate immunisation was defined as receipt of a scheduled vaccine dose within 30 days of the recommended age. For example, a child who received the 1st dose of DTPa (due at 60 days of age) when he or she was more than 90 days of age was classified as not age-appropriately immunised (i.e. late for the dose). For descriptive purposes, we categorised the outcome measure for each dose as either vaccine dose 'no delay' (age-appropriately immunised), 'delay of between 1 to 6 months', or 'delay greater than 6 months'. Doses received 'too early' (greater than 30 days prior to when it was due), and doses never administered or recorded were excluded. Timeliness is measured in 12-month birth cohorts. Children included in the timeliness analysis were assessed at 1-2 years after doses were due, to allow time for late vaccinations to be recorded. Therefore, cohorts assessed for timeliness are not the same as those assessed for coverage milestones. The interval between doses was not evaluated. Timeliness of different vaccines and doses was also compared by plotting the cumulative percentage receiving each vaccine dose by age, with the proportion ever immunised set as 100%.

Remoteness status

The area of residence of children was defined as accessible or remote using the Accessibility/Remoteness Index of Australia (ARIA), which was developed by the then Department of Health and Aged Care, and proposed as the national standard measure of remoteness for inclusion in the Australian Bureau of Statistics (ABS) 2001 census.[20] For the timeliness analysis, we defined the two ARIA categories with most restricted access to services as 'remote' (approximately 2.6% of the Australian population) and all other areas as 'accessible'.

Indigenous status

Indigenous status on the ACIR is recorded as 'Indigenous', 'non-Indigenous' or 'unknown', as reported by the child's carer to Medicare, or by the immunisation provider to the ACIR. For this report we considered two categories of children: 'Indigenous' and 'non-Indigenous', children with unknown Indigenous status were presumed to be 'non-Indigenous'. Coverage estimate time trends are presented from 2004 only, due to poor rates of reporting Indigenous status prior to then.[21]

Small area coverage

Coverage was calculated for ABS-defined Statistical Subdivisions (SSD), chosen because each is small enough to show differences within jurisdictions but not too small to render maps unreadable.[22] Maps were created using version 10 of the MapInfo mapping software and the ABS Census Boundary Information.[23] As postcode is the only geographical indicator available from the ACIR, the ABS Postal Area to Statistical Local Area (SLA) Concordance 2006 was used to match ACIR postcodes to SSDs, in order to create a SSD field for each child in the relevant study cohorts.[24]

Conscientious objection / No vaccine recorded

A child must be registered with Medicare before the parent(s) can lodge a conscientious objection to immunisation. Parents can also object to immunisation but refuse to lodge any official objection to the ACIR. We used the percentage of children with no vaccines recorded on the ACIR as a proxy measure of the number of these children.[16] Some children with no vaccines recorded on the ACIR will be officially registered as conscientious objectors to vaccination and some will not be registered as such. Registered conscientious objectors are eligible for parent incentive payments even if they are unvaccinated. Proportions of conscientious objectors and children with no vaccines recorded by region were calculated from the cohort of children registered with Medicare, and born between 1 January 2003 and 31 December 2009. At the time of data extraction on 31 March 2011, they were between 12 and 72 months of age. We chose this cohort when calculating proportions so that children under the age of 12 months were not included, to allow sufficient time for registration of objection and exclude infants late for vaccination.

Human papillomavirus vaccine coverage

The HPV vaccination program is listed on the NIP Schedule, funded under the Immunise Australia Program and delivered to girls through an ongoing school-based program usually in the first year of secondary school. From 2007 to 2009 there was a time-limited catch-up program delivered through schools, general practices and community immunisation services. Immunisation for HPV is achieved with a course of 3 doses of vaccine, over a 6 month period. Data on the National HPV Vaccination Program is provided by the National HPV Vaccination Program Register. The purpose of this legislated register is to monitor and evaluate the vaccination program and is operated by the Victorian Cytology Service. The World Health Organization (WHO) proposes using 15 years as the reference age for HPV vaccination coverage for the purposes of international comparison. Data on HPV coverage was obtained from the Immunise Australia website.[25]

Coverage in the elderly

Influenza and pneumococcal (23vPPV) vaccination coverage estimates in the elderly were taken from the 2009 Adult Vaccination Survey.[26] This telephone survey forms part of the evaluations of two vaccine programs for older Australia. The first is the National Influenza Vaccination Program for Older Australians, which commenced in 1999; the second is the National Pneumococcal Vaccination Program for Older Australians which commenced on 1 January 2005. This was a survey of 10,231 Australians aged 18 years or older, conducted during November to December 2009. Participants in the survey were asked about their recent experience of influenza and pneumococcal vaccination, and about their medical history and socio-demographic status.

Results

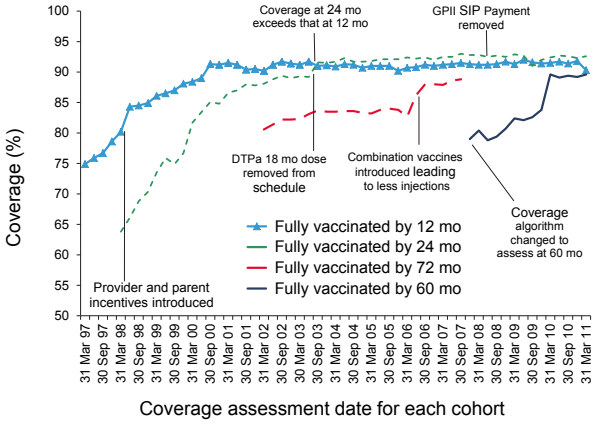
Coverage estimates

Overall

Coverage estimates in 2010 for full-year birth cohorts at the 3 milestone ages of 12 months, 24 months and 60 months are provided in Tables 2, 3 and 4. Nationally, 'fully immunised' coverage and coverage for all individual vaccines for the 12-month and 24-month age groups exceed the 1993 Immunise Australia Program's target of 90%. Recorded national coverage for the 60-month age group is marginally below the target, at 89% for all vaccines, and lower in some jurisdictions.

Figure 1 shows time trends in 'fully immunised' childhood vaccination coverage in Australia, assessed at 12 months, 24 months, and at 60 months of age, for 3-month cohorts born from 1 January 1996 to 31 December 2009. The proportion 'fully immunised' at 12 months of age increased steadily from 75% for the 1st cohort in 1997 to 91.8% by 31 December 2010. At the 24-month milestone, 'fully immunised' coverage estimates also increased steadily from 64% for the 1st cohort to 92.3% by December 2010. 'Fully immunised' coverage estimates assessed at 72 months of age for vaccines due at 48 months were first reported in CDI in 2002, and increased steadily from 80.6% in early 2002 to 87.3% in late 2007, including a noticeable increase in June 2006, corresponding with the introduction of combination vaccines. However, from the beginning of 2008, when the assessment age was changed from 72 months to 60 months, 'fully immunised' coverage was substantially lower at 80.7% in December 2008, related to delayed immunisation. However, during 2009 and 2010, coverage for this age group rose substantially.

Figure 1: Trends in 'fully immunised' vaccination coverage, Australia, 1997 to 2010, by age cohort



Coverage calculated at 60 months was unchanged during the latter half of 2010 at 89%.

Coverage estimates for the 24-month age group increased substantially and suddenly in September 2003 to 91.6%, following the removal from the immunisation schedule of the 4th dose of DTPa (due at 18 months of age) from this quarter onwards. Coverage estimates for the 12-month age group have remained steady over the past 10 years, fluctuating around the 91% level.

There is a clear trend of increasing vaccination coverage over time for all age groups assessed, with the 2 youngest age cohorts having the highest coverage.

Table 2: Percentage of children in 2010 immunised at 12 months of age, by vaccine and state or territory*

Vaccine	Jurisdiction								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	4,978	97,303	3,853	61,963	19,563	6,448	71,581	31,105	296,794
Diphtheria, tetanus, pertussis (%)	94.6	92.2	90.9	92.3	92.2	92.3	92.9	90.8	92.3
Poliomyelitis (%)	94.5	92.2	90.9	92.2	92.2	92.3	92.8	90.8	92.2
Haemophilus influenzae type b (%)	94.4	92.0	91.3	92.1	92.0	92.2	92.6	90.6	92.1
Hepatitis B (%)	93.7	91.8	90.7	91.9	91.7	92.1	92.3	90.3	91.8
Rotavirus (%)	88.0	86.4	81.6	83.2	84.6	86.1	83.7	85.6	84.7
7vPCV (%)	93.6	91.5	89.4	91.7	91.5	91.6	92.1	89.8	91.5
Fully immunised (%)	93.6	91.7	90.2	91.7	91.6	92.0	92.1	90.1	91.6
Fully immunised (incl rotavirus & 7vPCV) (%)	86.7	84.1	78.4	86.1	87.3	84.0	86.5	83.0	85.2

* For the birth cohort born in 2009

Table 3: Percentage of children in 2010 immunised at 24 months of age, by vaccine and state or territory*

Vaccine	Jurisdiction								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	4,923	98,729	3,711	63,062	19,779	6,609	72,248	31,499	300,560
Diphtheria, tetanus, pertussis (%)	96.0	94.6	95.1	94.5	94.5	95.3	95.4	93.4	94.7
Poliomyelitis (%)	95.9	94.6	95.0	94.4	94.5	95.3	95.4	93.3	94.6
Haemophilus influenzae type b (%)	95.6	94.8	94.0	94.2	94.2	95.3	95.1	93.2	94.6
Hepatitis B (%)	95.2	94.1	94.6	93.9	94.0	95.1	94.7	92.5	94.1
Measles, mumps, rubella (%)	94.8	93.8	95.0	93.9	93.8	94.9	94.6	92.5	93.9
Varicella (%)	87.5	82.1	84.9	86.4	81.9	81.7	82.7	79.4	83.0
MenC (%)	94.4	93.4	94.9	93.5	93.7	94.8	94.3	91.9	93.6
Fully immunised (%)	93.3	92.1	92.4	92.2	92.1	93.6	92.9	90.0	92.1
Fully immunised (incl varicella & MenC) (%)	85.6	80.0	82.5	84.8	80.2	80.2	80.9	77.2	81.1

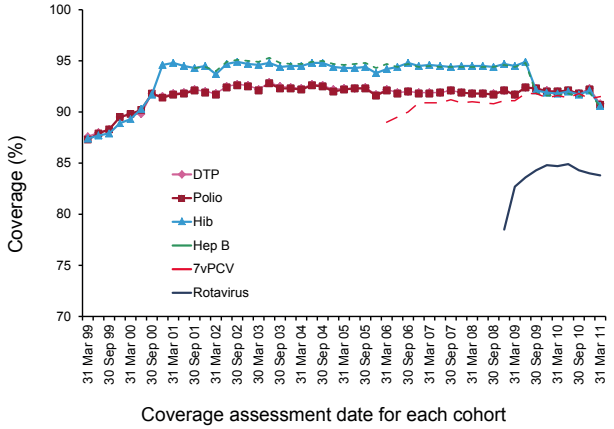
* For the birth cohort born in 2008

Table 4: Percentage of children in 2010 immunised at 60 months of age, by vaccine and state or territory*

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	4,628	93,742	3,576	60,695	18,699	6,263	68,128	29,601	285,332
Diphtheria, tetanus, pertussis (%)	91.3	89.5	87.5	90.3	87.6	91.9	91.2	86.6	89.7
Poliomyelitis (%)	91.5	89.4	87.4	90.3	87.6	91.9	91.3	86.7	89.7
Measles, mumps, rubella (%)	91.1	89.3	87.1	90.2	87.4	92.0	91.1	86.5	89.6
Fully immunised (%)	90.7	88.9	86.6	89.7	87.0	91.5	90.7	85.9	89.1

* For the birth cohort born in 2005

Figure 2: Trends in vaccination coverage estimates for individual vaccines at 12 months of age (DTPa, polio, hepatitis B and Hib)*

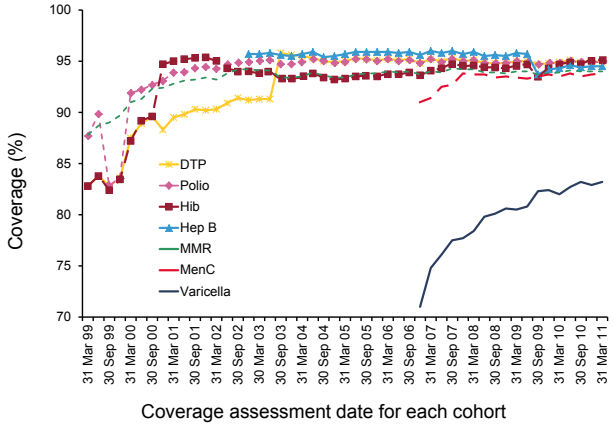


Source: Australian Childhood Immunisation Register.

By 3-month birth cohorts born between 1 January 1999 and 31 December 2009. Coverage assessment date was 12 months after the last birth date of each cohort.

* 3rd dose of DTP and polio, 2nd or 3rd dose of Hib and Hep B

Figure 3: Trends in vaccination coverage estimates for individual vaccines at 24 months of age (DTPa, polio, hepatitis B, Hib and MMR)*



Source: Australian Childhood Immunisation Register.

By 3-month birth cohorts born between 1 January 1998 and 31 December 2008. Coverage assessment date was 24 months after the last birth date of each cohort.

* 3rd or 4th dose of DTP, 3rd dose of polio, 3rd or 4th dose of Hib, 2nd or 3rd dose of Hep B, and 1 dose of MMR

Individual vaccines

DTPa and polio coverage at 12 months of age remained relatively stable from the latter part of 2001 to 2010 (Figure 2).

Prior to the change in algorithm to measure coverage that occurred in the latter half of 2009, coverage for the Hib and hepatitis B vaccines at 12 months of age become similar to those for DTPa and polio in the last two cohorts of 2009 and all of 2010 (Figure 2). Coverage for 7vPCV rose steadily from below 90% in mid-2007 to be just below that for all other vaccines due at this age at around 92%, except for rotavirus vaccine. Rotavirus vaccine coverage rose steeply from late 2008 from below 70% to almost 85% in late 2010.

For most of the study period, at 24 months of age, hepatitis B coverage was higher than for all other vaccines, at just under 95%, due to the different coverage algorithm described above (Figure 3). Coverage was lowest for MMR and Hib, the only vaccines that have a 12-month dose used in

Table 5: Vaccination coverage estimates by age, vaccine and Indigenous status, 2010

Vaccine	Milestone age	Indigenous	Non-Indigenous
DTPa	12 mths*	85.5	92.6
	24 mths†	94.1	94.7
	60 mths‡	85.7	89.9
Polio	12 mths*	85.5	92.5
	24 mths†	94.0	94.7
	60 mths‡	85.7	89.9
Hib	12 mths*	83.5	92.0
	24 mths†	94.5	94.8
	60 mths‡	N/A§	N/A§
Hep B	12 mths*	85.5	92.1
	24 mths†	96.9	95.4
	60 mths‡	N/A§	N/A§
MMR	12 mths*	N/A§	N/A§
	24 mths†	94.4	93.9
	60 mths‡	86.2	89.7
Varicella	12 mths*	N/A§	N/A§
	24 mths†	82.2	83.0
	60 mths‡	N/A§	N/A§
Meningoc-occal C	12 mths*	N/A§	N/A§
	24 mths†	93.9	93.5
	60 mths‡	N/A§	N/A§
7vPCV	12 mths*	85.2	91.8
	24 mths†	N/A§	N/A§
	60 mths‡	N/A§	N/A§
Rotavirus	12 mths*	71.7	85.1
	24 mths†	N/A§	N/A§
	60 mths‡	N/A§	N/A§

* Birth cohort born 1 January 2009 – 31 December 2009
† Birth cohort born 1 January 2008 – 31 December 2008
‡ Birth cohort born 1 January 2005 – 31 December 2005
§ Not included in coverage estimates for that group

Table 6: Percentage of children fully immunised at 12 months, 24 months and 60 months of age, by Indigenous status and state or territory, 2010

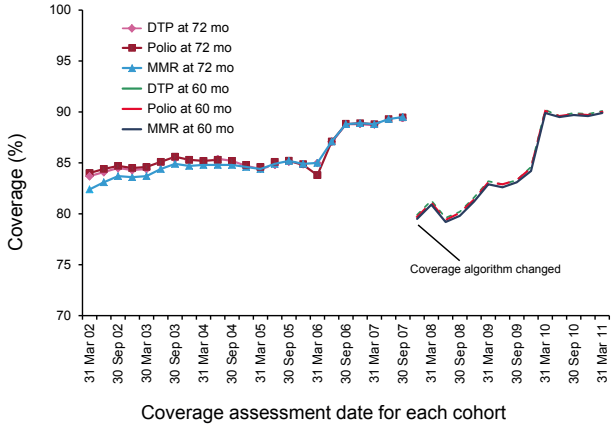
	States and Territories								
	ACT	NSW	VIC	QLD	SA	WA	TAS	NT	Australia
12 months – fully immunised (%)*									
Indigenous	87.1	87.2	84.6	85.9	79.3	80.6	89.1	87.7	85.4
Non-Indigenous	94.2	92.0	92.5	92.4	92.2	90.9	92.4	92.6	92.1
12 months – fully immunised (including rotavirus & 7vPCV) (%)									
Indigenous	73.1	76.2	77.4	76.0	74.8	69.2	79.4	68.2	74.3
Non-Indigenous	87.0	84.5	86.7	87.0	87.8	83.9	84.4	86.0	85.8
24 months – fully immunised (%)†									
Indigenous	97.9	91.5	92.5	92.6	90.0	87.3	93.1	93.5	91.6
Non-Indigenous	93.8	92.4	93.3	92.5	92.5	90.6	93.8	92.2	92.5
24 months – fully immunised (including varicella & MenC) (%)									
Indigenous	89.3	78.4	76.6	82.4	77.0	72.7	77.1	84.7	79.5
Non-Indigenous	85.9	80.2	81.2	85.2	80.6	77.7	80.5	81.5	81.4
60 months – fully immunised (%)‡									
Indigenous	86.6	84.4	86.4	86.9	79.2	80.8	90.0	89.4	85.3
Non-Indigenous	90.7	89.1	90.8	89.9	87.3	86.2	91.6	84.6	89.3

* 'Fully immunised' - 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, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of all other hepatitis B vaccines.

† 'Fully immunised' - 3 or 4 doses of a DTPa-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP-containing 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' - 4 or 5 doses of a DTPa-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

Figure 4: Trends in vaccination coverage estimates for individual vaccines (DTPa, polio, and MMR)* at 60 months (72 months prior to December 2007)



Source: Australian Childhood Immunisation Register.

By 3-month birth cohorts born between 1 January 1996 and 31 December 2005. Coverage assessment date was 72 months after the last birth date of each cohort up to December 2007 and then 60 months after the last birth date of each cohort.

* 4th dose of DTP and polio, 2nd dose of MMR.

calculations. The overall coverage estimates for 24-month olds are approaching 95% for all vaccines except varicella.

There was a marked increase in coverage for individual vaccines at 60 months of age following the change in the due/overdue rules in January 2009, with coverage increasing to levels similar to when coverage was assessed at 72 months of age (Figure 4).

Coverage estimates for Indigenous children

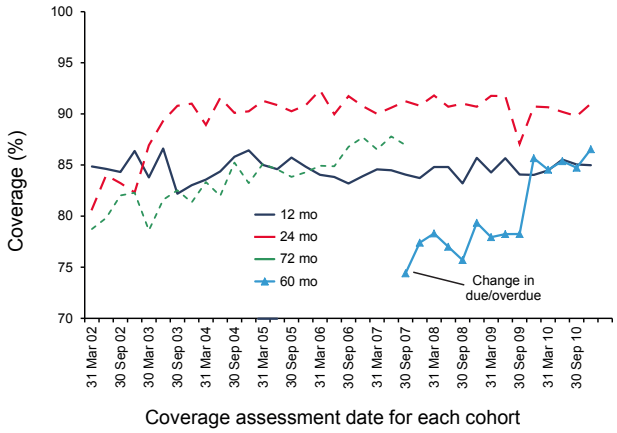
Immunisation coverage is lower for Indigenous children than non-Indigenous at the 12-month, 24-month and 60-month age milestones for most vaccines, with the difference being greatest at 12 months of age (Table 5). The difference in coverage at 12 months of age has been relatively consistent for the past 7 years. The coverage differential between Indigenous and non-Indigenous children for individual vaccines varies, with coverage at 24 months of

age for most vaccines being almost identical for both groups and greater among Indigenous children for hepatitis B, MMR and meningococcal C vaccines.

The proportion of children 'fully immunised' by 24 months of age has consistently remained higher than at 12 months and 60 months of age (Figure 5). For non-Indigenous children, coverage at 60 months of age increased following the change in due/overdue rules. Coverage in 2010 was the same at 12 months and 60 months of age.

At age 12 months, the overall proportion of Indigenous children fully vaccinated was 85.4%, compared with 92.1% for non-Indigenous children (Table 6). Although coverage was lower among Indigenous children in all jurisdictions, the extent of the difference varied, reaching a 14 percentage point differential in South Australia and a 10 percentage point differential in Western Australia. However, by age 24 months, the coverage disparity between Indigenous and non-Indigenous children had

Figure 5: Trends in 'fully immunised' vaccination coverage for Indigenous children in Australia, 2004 to 2010, by age cohorts



greatly reduced nationally to be less than 1 percentage point lower for Indigenous children, ranging from 4 percentage points higher in the Australian Capital Territory to 3.3 percentage points lower in Western Australia.

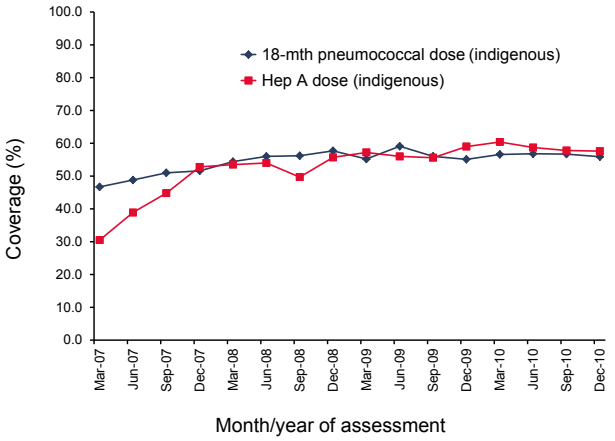
At 60 months of age, the proportion of Indigenous children recorded as being 'fully vaccinated' was lower than that at 24 months of age. At the national level, the coverage for Indigenous and non-Indigenous children was 85.3% and 89.3%, respectively. There was dramatic variation between individual jurisdictions, ranging from coverage 8.1% lower in Indigenous children in South Australia to 4.8% higher in the Northern Territory, compared to non-Indigenous children (Table 6) in 2010.

Coverage for National Immunisation Program vaccines not routinely reported elsewhere

7vPCV and Rotavirus

7vPCV was first added to the NIP in January 2005. Since coverage was first calculated for this vaccine in early 2006, it has remained high, with a slight increase from 89% to 91.5% (Figure 2). Coverage is greater than the 1993 Immunise Australia Program target of 90% in all jurisdictions except for Western Australia and Tasmania where it approaches 90% (Table 2).

Figure 6: Trends in coverage for hepatitis A* and pneumococcal polysaccharide (23vPPV) vaccines for Indigenous children, 2007 to 2010



* Two doses assessed at 30 months for WA and NT. 2 doses assessed at 36 months for QLD and SA.

Rotavirus vaccine was added to the NIP in July 2007, thus coverage for 2 or 3 doses (depending on vaccine) at 12 months of age could be calculated only from the December 2008 quarter. Rotavirus coverage was lower nationally (Figure 2), and had greater variation between jurisdictions compared to other vaccines given at 2, 4 and 6 months, which may be due to the strict upper age limits for this vaccine. Reported coverage for 2 or 3 doses (Rotarix® versus Rotateq®) of rotavirus vaccine at 12 months of age varied from 83.2% in Queensland (Rotateq®) to 86.4% and 88% in New South Wales and the Australian Capital Territory (both Rotarix®) respectively (Table 2).

Meningococcal C and Varicella

Meningococcal C vaccine was added to the NIP in January 2003. Since coverage was first calculated for this vaccine in early 2006, it has remained at high levels, with an increase over 2 years from 88% to almost 94% (Figure 3). There was little variation by

jurisdiction, with all jurisdictions experiencing coverage levels greater than 91% and some, Victoria and Western Australia, closely approaching 95% (Table 3).

Varicella vaccine was added to the NIP in November 2005. Reported coverage for this vaccine has consistently been 10–15 percentage points lower than that for all the other vaccines assessed at 24-month milestone, being 83% for the latest assessment (Figure 3). This is probably partly due to the shorter time varicella has been on the NIP and the age of administration (18 months). The 18-month schedule point was historically associated with lower coverage when there was an 18-month pertussis booster prior to 2003, there was a gap when no vaccine was administered at 18 months over 2 years and only one vaccine is given. Reported varicella vaccine coverage also shows considerable variation by jurisdiction from 79.4% in Tasmania to 87.5% in the Australian Capital Territory (Table 3). Data are also available from the ACIR on the numbers of reports from GPs stating that children born since May 2004 have natural immunity to varicella and do not require varicella vaccination. Reports of natural immunity to varicella total greater than 20,000 since May 2004 (not shown), corresponding to approximately 1.1% of the cohort. It is likely that there is underreporting of presumed natural immunity by GPs but this is unlikely to fully account for lower varicella coverage.

Table 7: Vaccination coverage* for hepatitis A (Indigenous only) and 23vPPV (Indigenous only) by state/territory

State/territory	Vaccine type	
	Hep A†	23vPPV‡
NT	81.5 (84.2)	69.6
QLD	53.2 (61.4)	55.0
SA	31.5 (49.7)	39.7
WA	60.1 (64.7)	57.1
AUST	57.6 (65.6)	56.7‡

* For the last 3-month cohort assessable in 2010

† Indigenous only: 2 doses by 30 months of age for WA and NT (1 dose by 18 months of age), 2 doses by 36 months of age for QLD and SA (1 dose by 24 months of age)

‡ Indigenous only: 1 dose by 36 months of age for Northern Territory, Queensland, South Australia and Western Australia only

Hepatitis A and 23vPPV

Hepatitis A vaccine was available in Australia prior to the development of the ACIR in 1996 and has been included on the NIP for Indigenous children in the Northern Territory, South Australia, Western Australia, and in Queensland since November 2005, but was used earlier than this in North Queensland. Since March 2007, coverage of 2 doses of hepatitis A vaccine by 30 months of age in Western Australia and the Northern Territory and 36 months of age in Queensland and South Australia for Indigenous children has increased from below 20% to 58% in December 2010 (Figure 6). An additional 8% of children had received 1 dose of hepatitis A vaccine by 18 or 24 months of age, increasing national coverage for at least one dose of hepatitis A vaccine to 66% in Indigenous children (Table 7). There is a variation in reported hepatitis A vaccine coverage by jurisdiction, from a low of 31.5% in South Australia to a high of 81.5% in the Northern Territory (Table 7).

The 23vPPV has been recommended for Indigenous children in the same 4 jurisdictions (the Northern Territory, South Australia, Western Australia, and Queensland) as a booster at 18–24 months of age since 2001; coverage has gradually increased from 47% in December 2006 to 56% in December 2010 (Figure 6). There is a large variation in 23vPPV coverage by jurisdiction from a low of 39.7% in South Australia to a high of 69.6% in the Northern Territory (Table 7).

Table 8: Vaccination delay, by Indigenous and remoteness status for the cohort of children born in 2008, Australia

Vaccine dose	Indigenous status	Remoteness	1–6 mths delay %	> 6 mths delay %
DTPa3	Indigenous	Accessible	30	8
		Remote	34	7
	Non-Indigenous	Accessible	17	2
		Remote	16	2
MMR1	Indigenous	Accessible	33	5
		Remote	33	4
	Non-Indigenous	Accessible	25	2
		Remote	24	2

Table 9: Vaccination coverage for dose 3 of HPV vaccine for girls aged 15 years, 2009

State/territory	Vaccine type
	HPV
NSW	73.0
Vic	76.1
Qld	71.1
WA	64.7
SA	72.4
Tas	63.7
ACT	79.6
NT	76.1
Australia	70.8

Source: Human papillomavirus vaccination coverage data Australian Government. Department of Health and Ageing, April 2011, Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/immunise-hpv

Includes only eligible consumers and vaccinations given as part of the National HPV Vaccination Program

Includes valid doses and too close doses for Clinically Complete Consumers

Population is Estimated Resident Population provided by the Australian Bureau of Statistics - Cat 3101.0, Australian Demographic Statistics, Tables 51 to 59: Estimated Resident Population By Single year of Age by State and Territory, published June 2010 for 2010.

Age is age as at date of Estimated Resident Population estimate

Coverage is calculated as doses administered and reported to the National HPV Program Register (NHPVPR) / estimated resident population expressed as a percentage

Excludes consumers who do not wish their details to be recorded on the HPV Register.

Table 10: Estimated seasonal influenza and pneumococcal vaccination status, persons aged 65 year or older, 2009

State/territory	Vaccine type	
	Influenza †	Pneumococcal (23vPPV) ‡
NSW	72.7	53.5
Vic	75.0	55.5
Qld	74.6	54.6
WA	72.9	51.8
SA	81.3	57.6
Tas	77.5	55.4
ACT	78.0	53.3
NT	69.3*	47.8*
Australia	74.6	54.4

Source: Australian Institute of Health and Welfare. 2009 Adult Vaccination Survey – Summary results. Canberra, March 2011. http://www.aihw.gov.au/publication-detail/?id=10737418409&tab=2 Published with permission from the Australian Institute of Health and Welfare.

* Estimate has 25% or greater relative standard error

† Vaccine received in the past 12 months

‡ Vaccine received in the past 5 years

Timeliness of immunisation

Timeliness has been examined for vaccines requiring both multiple doses (DTPa, 7vPCV and MMR) and a single dose (Men C) at 12 and 24 months of age.

Since 2004, the proportion of children with timely receipt of the 3rd dose of DTP vaccine has remained at 88% (not shown). Across the 5-year period, 2004–2008, timely receipt of 1 dose of MMR rose only 3 percentage points, although estimated coverage by 24 months of age remained stable at almost 94% (not shown).

As demonstrated in previous studies, the proportion with vaccination delay increased with older age (Figure 7). The greatest proportion with any delay was seen with the second dose of MMR vaccine with 65% of doses given late and almost 24% given more than 6 months late. This is a considerable improvement over the 2009 report where the corresponding figures were 80% and 35%.

For the 3rd dose of DTPa, there was greater delay for Indigenous children than non-Indigenous children, with a 21% differential of on-time vaccination at 6 months of age (Figure 8). The same pattern was found for timeliness of the 1st dose of MMR, but with a smaller differential of 11% (Figure 9). Although Indigenous children had only slightly lower coverage than non-Indigenous children by 24 months of age, they were more likely to have delayed vaccination.

Vaccination was delayed by more than one month for 30-34% of Indigenous children and 16-25% of non-Indigenous children (Table 8). The proportion with long delays (i.e. greater than 6 months) was 2–4 times higher in Indigenous children than in non-Indigenous children, with no great differences between accessible and remote areas or vaccines. Delays of 1-6 months were also more frequent for Indigenous children, although less marked, especially for the first dose of MMR. The proportion with short delays was greater among Indigenous children residing in remote areas than in accessible areas for the 3rd dose of DTP vaccine (34% versus 30%), but not for the 1st dose of MMR.

Vaccination delay for Indigenous children by jurisdiction was measured for 7vPCV, with greater delays in Western Australia (47.4%) and South Australia (42.6%) (Figure 10). The proportion of South Australian Indigenous children with long delays in receipt of the 3rd dose of 7vPCV vaccine decreased

from the previous report in 2009 (from 7.7% to 5.8%) but increased in Indigenous children from the Northern Territory (5.2% to 6.9%). There were no important differences in vaccination delay for non-Indigenous children by jurisdiction (not shown).

In contrast to younger ages, analysis of timeliness of immunisation for a vaccines at 48 months of age, the 2nd dose of MMR, showed a small difference in delay in receiving this vaccine for non-Indigenous children and Indigenous children, with only a 3.0% differential at 4 years and 3 months of age (Figure 11). However, timeliness for both groups was improved from the previous report in 2009.

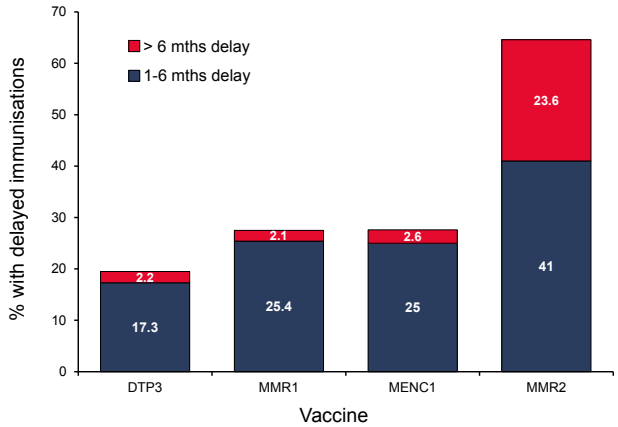
In response to the current pertussis epidemic and to provide early protection to young infants, it was recommended by the Australian Technical Advisory Group on Immunisation (ATAGI) in March 2009 and promoted in New South Wales and Tasmania that immunisation providers give the first dose of DTPa vaccine at 6 weeks of age instead of 8 weeks of age. Prior to this very few children received the vaccine dose at less than 8 weeks of age but for New South Wales and Tasmania the percentage rose over the 2 years with more than 60% of children receiving the dose prior to 8 weeks of age in December 2010 (Figure 12). In late 2010 this percentage also began to increase in other jurisdictions but not to the same extent as in New South Wales and Tasmania.

Small area coverage

Immunisation coverage in Australia in 2010 varies substantially within jurisdictions, with some areas substantially below the national averages, potentially putting them at risk of outbreaks (Figures 13-15). In particular, there are many areas with coverage at 60 months of age below 85% (Figure 15).

The proportion of children whose parents recorded as conscientious objectors and with no vaccines recorded are presented by SSD in Figures 16 and 17, respectively. No vaccines recorded may represent either non-immunisation (parents refusing any vaccines) or, and probably much less commonly, non-reporting by a provider. The percentage of children with no vaccines recorded nationally (3.0%) is greater than those recorded as conscientious objectors (1.7%). The map of the proportion of conscientious objectors to immunisation shows pockets of high levels of objection within jurisdictions in 2009, particularly in coastal areas of south

Figure 7: Vaccination delay - cohorts born in 2008 (DTPa3, MMR1, MENC1) and 2004 (MMR2)



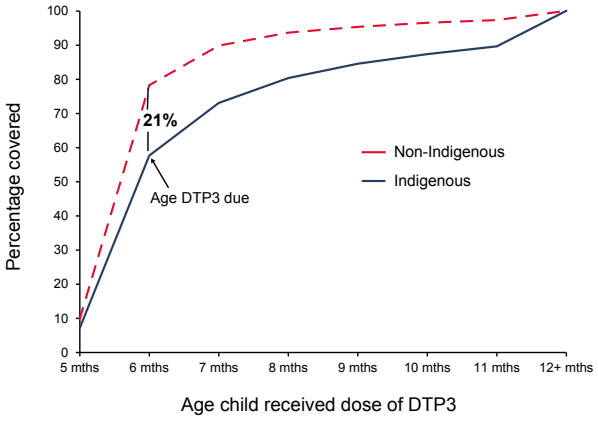
DTP3 = 3rd dose of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine

MMR1 = 1st dose of a measles, mumps and rubella vaccine

MENC1 = 1st dose of a meningococcal C vaccine

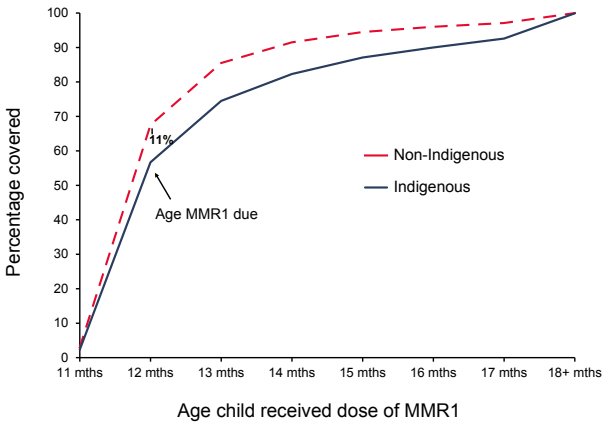
MMR2 = 2nd dose of a measles, mumps and rubella vaccine

Figure 8: Timeliness of the 3rd dose of DTP vaccine (DTPa3) by Indigenous status – cohort born in 2008*



* Percentage covered = number of children who received vaccine dose at particular ages/the total number of children who received the vaccine dose

Figure 9: Timeliness of the 1st dose of MMR vaccine (MMR1) by Indigenous status – cohort born in 2008*



* Percentage covered = number of children who received vaccine dose at particular ages/the total number of children who received the vaccine dose

east Queensland, northern New South Wales, the Mount Lofty Ranges region in South Australia and south west Western Australia, which also appear with low coverage in Figures 13-16.

The map of the proportion of children with no vaccines recorded (Figure 17) shows some additional areas not evident from, but usually adjacent to, maps of official conscientious objection. Children with no vaccines recorded and children who have parents who register as a conscientious objector are not mutually exclusive groups. Only 30% of children with no vaccines recorded were registered conscientious objectors, whilst 45% of conscientious objectors have vaccines recorded on the ACIR (not shown). Areas with low or normal levels of conscientious objection and/or low levels of no vaccines recorded but low coverage may reflect access issues rather than issues of ideology.

Provider type

GPs administer the large majority of immunisations in Australia; the proportion given by GPs has increased over the past 11 years by almost 5% (not shown) (Figure 18). Local government clinics also administer a substantial proportion of immunisations, especially in some jurisdictions. The only other category of provider administering major numbers of immunisations nationally is community health centres. Regional differences are marked, with immunisations almost entirely administered by GPs in some jurisdictions (New South Wales, Queensland, South Australia, Tasmania and Western Australia), while in others a majority are given by local government (Victoria) and community health clinics (the Northern Territory).

Human papillomavirus vaccine coverage

Vaccination coverage for dose 3 of the HPV vaccine for girls aged 15 years in 2009 is shown in Table 9. For Australia, almost 71% of girls completed a full course of the vaccine. Coverage varied by jurisdiction from a low of 63.7% in Tasmania to a high of 79.6% in the Australian Capital Territory. Coverage in all age groups was higher for earlier doses, as high as 84% for the first dose in girls aged 14 to 15 (Figure 19). Coverage was higher in the younger age groups than the older age groups with only 30% of girls aged 20-26 years and 38% of girls aged 18 to 19 years vaccinated for the third dose of HPV vaccine.

Vaccines for the elderly (pneumococcal and influenza)

In 2009, influenza (in previous 12 months) and pneumococcal polysaccharide (23vPPV) (in previous 5 years) vaccine coverage in the elderly

Figure 10: Vaccination delay for Indigenous children for the 3rd dose of 7vPCV by jurisdiction, cohort born in 2008

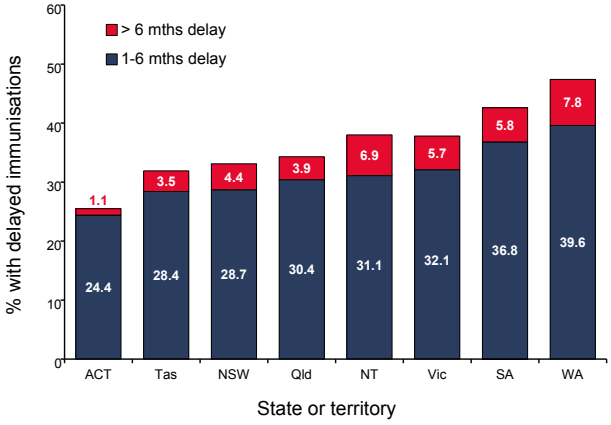
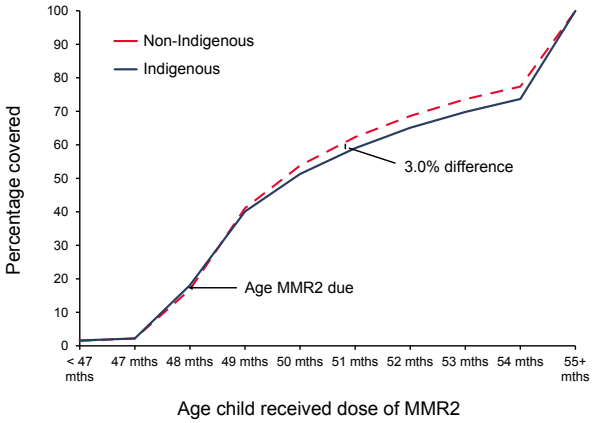


Figure 11: Timeliness of the 2nd dose of MMR vaccine (MMR2) by Indigenous status – cohort born in 2004*



* Percentage covered = number of children who received vaccine dose at particular ages/the total number of children who received the vaccine dose

Figure 12: The percentage of children who received their 1st dose of DTPa/Hexa vaccine at age 6 < 8 weeks, by jurisdiction and month of receipt, January 2009 – December 2010

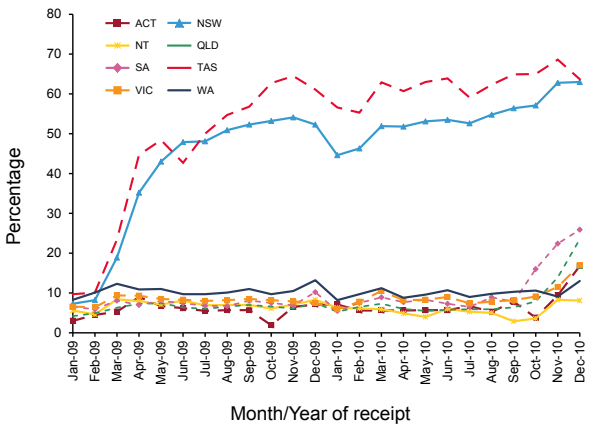


Figure 13: 'Fully immunised' coverage at 12 months of age Australia, by Statistical Sub-Division, 2010

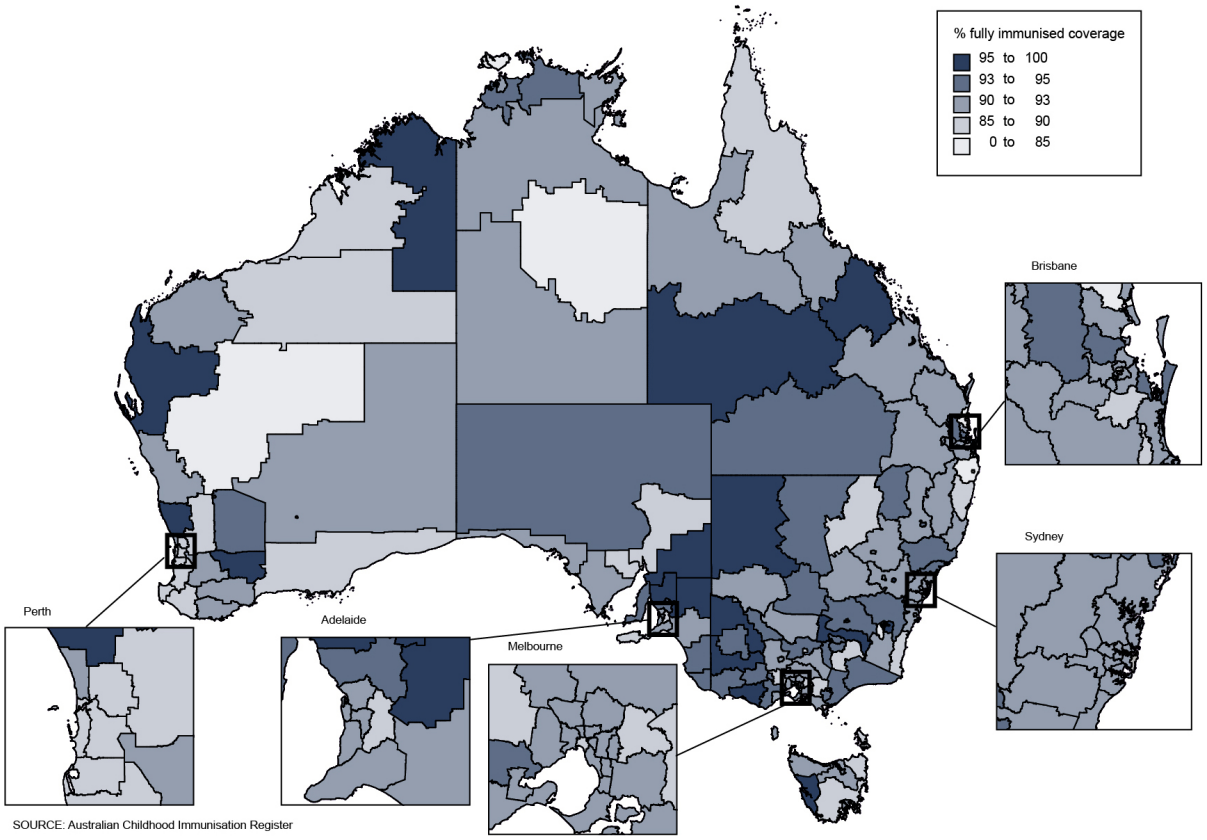


Figure 14: 'Fully immunised' coverage at 24 months of age Australia, by Statistical Sub-Division, 2010

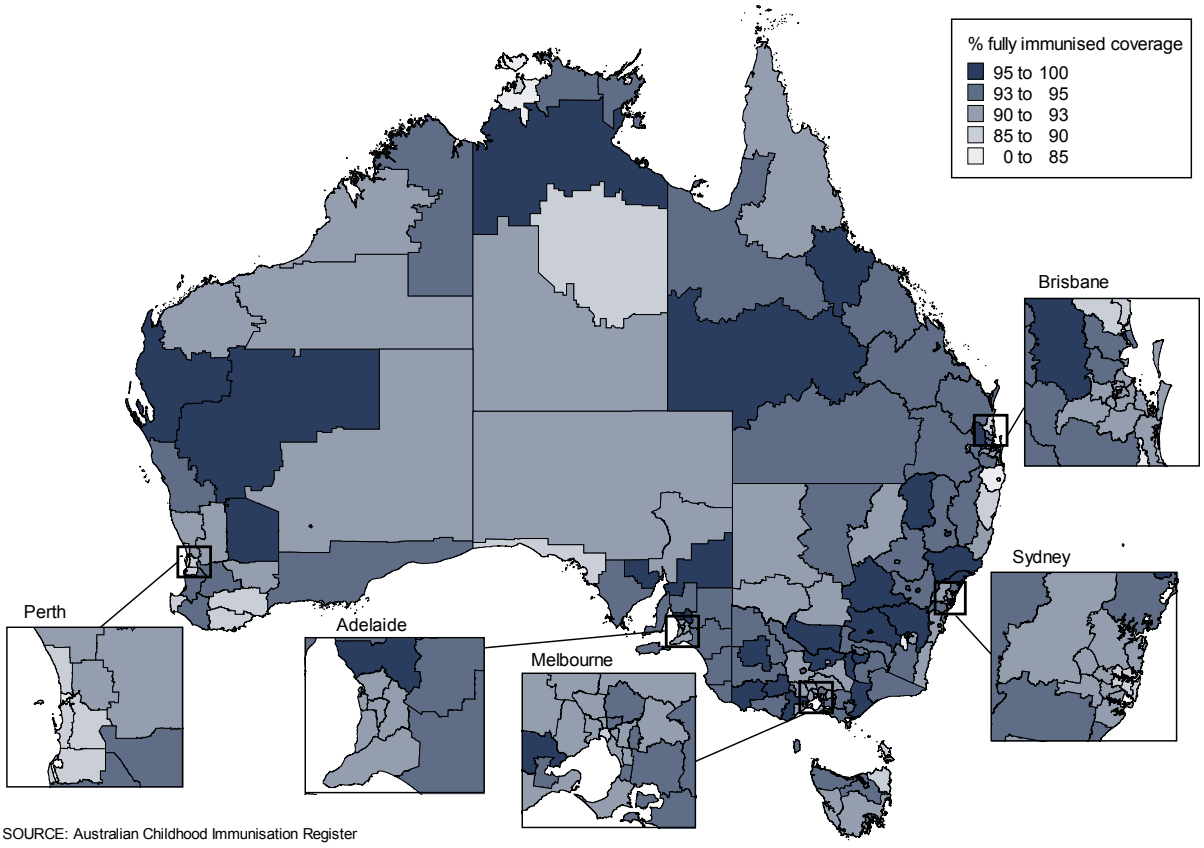


Figure 15: 'Fully immunised' coverage at 5 years of age Australia, by Statistical Sub-Division, 2010

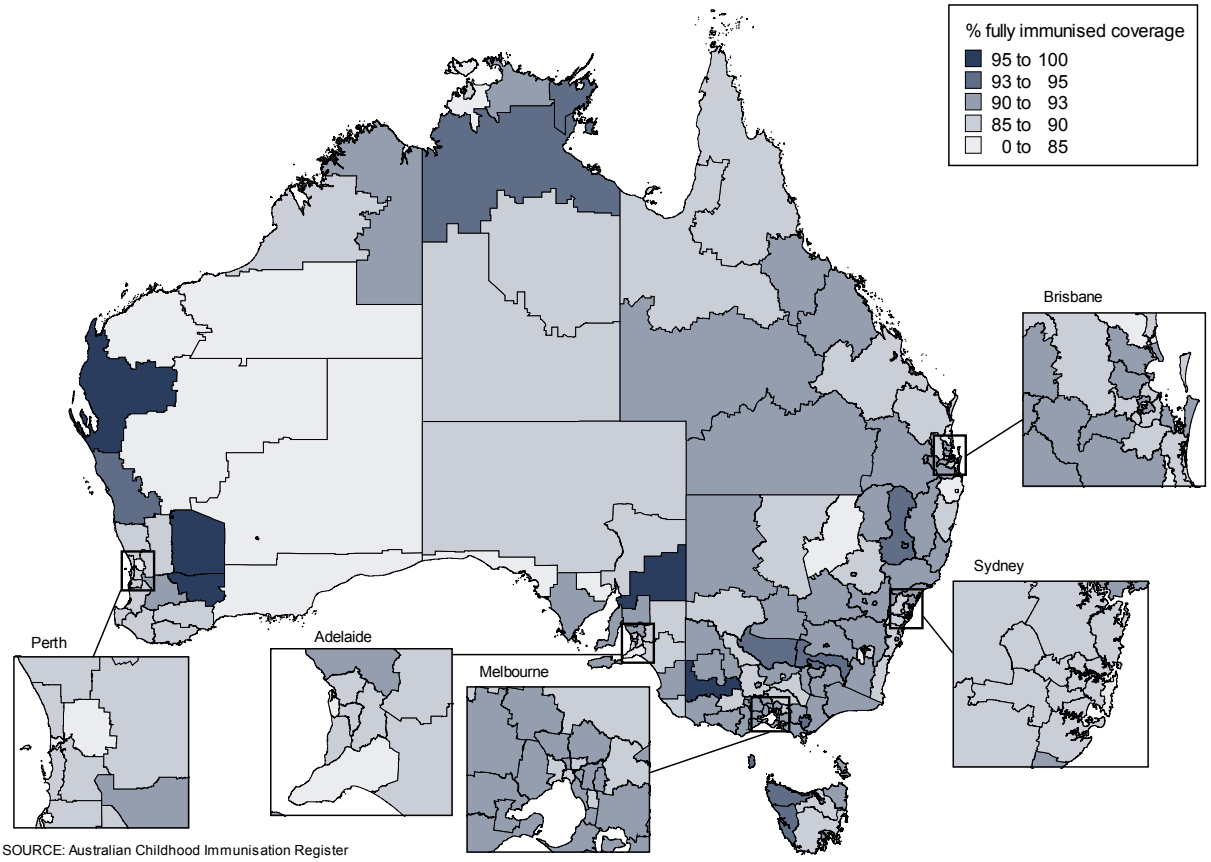


Figure 16: Proportion of official conscientious objectors to immunisation, Australia, 2010 (cohort born Jan 2004 – Dec 2009)

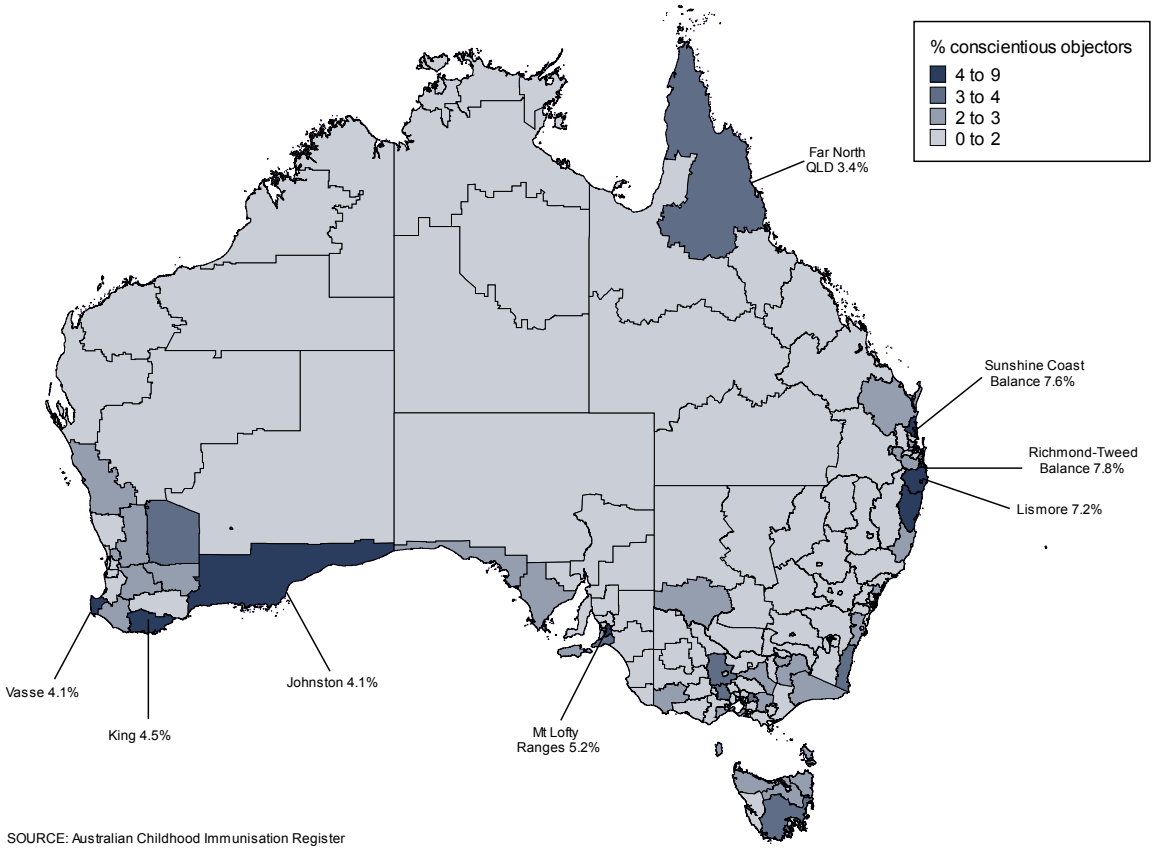


Figure 17: Proportion of children with no vaccines recorded on the ACIR, Australia, 2010 (cohort born Jan 2004 – Dec 2009)

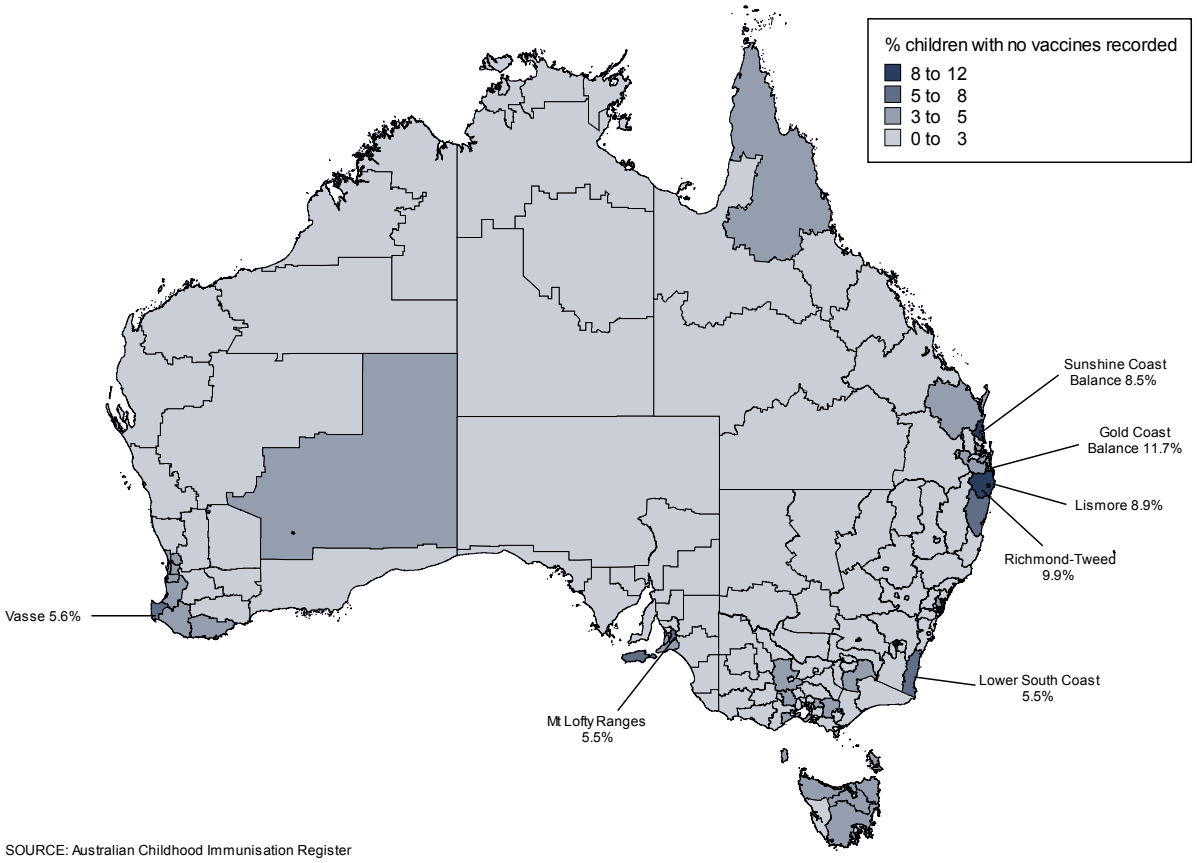
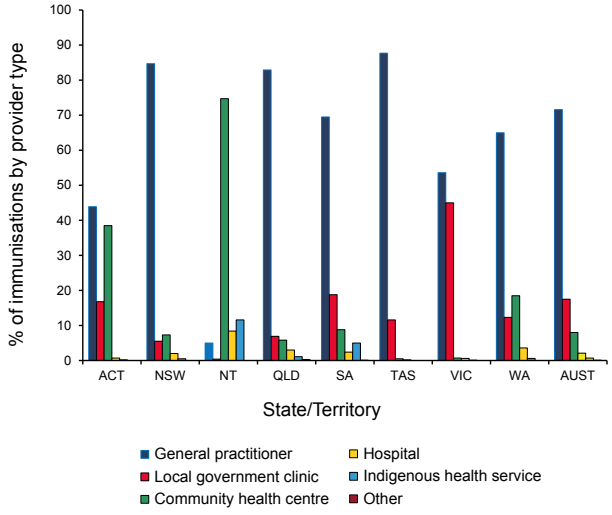


Figure 18: Proportion of immunisations on the ACIR given by various provider types, by state or territory, 2010



were highest in South Australia and Tasmania and lowest in the Northern Territory and Western Australia (Table 10).

Discussion

These data show that 1993 Immunise Australia Program coverage targets (90%) have been reached for children both 12 and 24 months of age. However, this is not the case for children 60 months of age where coverage, whilst much improved, is below the target in some jurisdictions.

"Fully immunised" coverage at 24 months of age marginally exceeds that at 12 months of age, and this is likely related to the exclusion of varicella vaccine at 18 months from calculation of 'fully vaccinated', the absence of any other vaccines administered between those ages, and the impact of immunisation incentives. Coverage for vaccines due at 48 months of age improved considerably during 2009 and 2010 approaching 90% for all 4 cohorts in 2010. This increase is due to improved timeliness of vaccination, and is probably related to the change to the overdue rules in January 2009, where children became overdue for their pre-school boosters at 49 months of age instead of the previous 60 months.

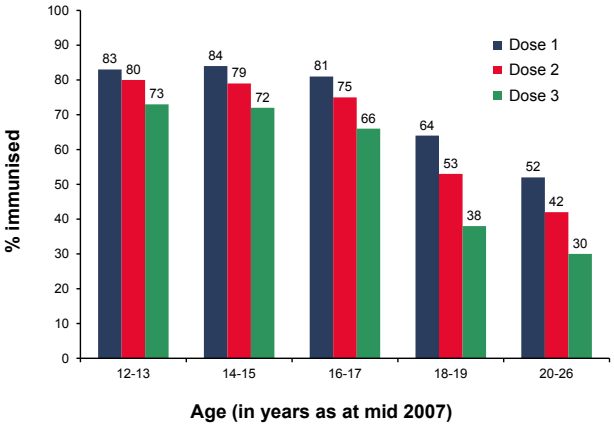
This change had an impact on eligibility for child care benefits for parents and outcome payments for providers. It was accompanied by a letter from Medicare Australia advising parents of the change, and the follow-up of overdue children by local health authorities. It is unlikely that the splitting of the Maternity Immunisation Allowance at that time could have had an impact in these data, as it applies only to children turning 48 months from 2011 onwards. Parents of older children would have received the full Maternity Immunisation Allowance payment at 24 months of age and were therefore not eligible for another payment at 48 months.

There is earlier evidence that immunisation incentives to providers positively impacts on coverage estimates.[15] However, the initial analyses in this report provides no evidence of a reduction in coverage associated with the removal of SIP payments in October 2008, while coverage at 60 months has increased following the due and overdue rule changes. More analysis is required to examine the impact of these changes in more detail.

A number of vaccines that are included in the NIP are not included when calculating 'fully immunised' status or in eligibility for incentive payments. Coverage estimates for 7vPCV and meningococcal C vaccines are comparable with estimates for vaccines that are included in 'fully vaccinated' calculations, but estimates for varicella and rotavirus are still substantially lower. During 2010, there were only slight changes in coverage for varicella (from 82% to 83%) and rotavirus vaccine (from 85% to 84%). For rotavirus vaccines, strict upper age limits for administration may explain lower coverage, whilst varicella is the only vaccine due at 18 months, and this milestone was historically problematic and lapsed for a two year period (2003 to 2005). The implications also vary. In the case of rotavirus vaccine, coverage of 80% or greater has been associated with substantial herd immunity and decreases in rotavirus hospitalisations in Australia and elsewhere.[27] By contrast, modelling studies suggest that low coverage with varicella vaccine may result in a shift of disease to older age groups with higher disease severity.[28]

Coverage for vaccines recommended for Indigenous children only (i.e. hepatitis A and 23vPPV), remained sub-optimal during 2010. The extent of under-reporting to the ACIR for these vaccines is unknown but likely to be more than for 'universal' vaccines, given the lack of incentive payments for notification to the ACIR. However, lower coverage for vaccines targeted at Indigenous people has been a relatively consistent finding using a range of different methods for both children and adults.[14,30] Both a lack of provider knowledge about the recommendations for high risk groups, and poor identification of Indigenous children by immunisation

Figure 19: HPV vaccination coverage by dose number, females vaccinated between April 2007-December 2009, Australia



Source: Human papillomavirus vaccination coverage data Australian Government. Department of Health and Ageing, April 2011. Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/immunise-hpv

12-13 years – School program

14-15 years – School catch up

16-17 years – School catch up

18-19 years – GP/community

20-26 years - GP/community

Includes only eligible consumers and vaccinations given as part of the National HPV Vaccination Program

Includes valid doses and too close doses for Clinically Complete Consumers

Population is Estimated Resident Population provided by the Australian Bureau of Statistics as at 30/06/2007

Age is age as at date of Estimated Resident Population estimate

Coverage is calculated as doses administered and reported to the NHVPR/estimated resident population expressed as a percentage

Excludes consumers who do not wish their details to be recorded on the HPV Register

Data is cumulative so covers all people over a three year period within each cohort (April 2007-December 2009)

Published with permission from the Australian Government Department of Health and Ageing.

providers are likely to be important contributing factors. Differences in schedules between jurisdictions may also be a contributing factor. During 2010, coverage for both vaccines was still higher in the Northern Territory and Western Australia, which give the vaccines 6 months younger (hepatitis A, 12 and 18 months, 23vPPV 18 months), than in Queensland and South Australia (18 and 24, and 24 months). The presence of other vaccines on the schedule at the same age may assist achieving higher coverage, particularly at 12 months and less so at 18 months of age. Failure to receive a 2nd dose by 8% of children also contributed to the low coverage for hepatitis A vaccine. However, a protective antibody response after one dose is expected from a majority of children.[31]

Although coverage data reveal that most children eventually complete the scheduled vaccination

series by the 24-month milestone, many still do not do so in a timely manner. Vaccination delay in 2010 as measured in this report for vaccines assessed at 12 and 24 months of age has improved only marginally. However, timeliness cannot be measured in the most recent cohort, as time must be allowed for late vaccination to be received. An improvement in coverage seen at 60 months of age is reflected in much improved timeliness calculations in this age group for 2010. However, coverage 12 months after the due date of this vaccine is still <85%. Poorer timeliness in Indigenous children has been noted previously in infants. Timeliness has improved markedly at 60 months of age for both Indigenous and non-Indigenous children. However, as coverage and timeliness of vaccines assessed at 60 months of age has improved, a not previously seen disparity in timeliness between Indigenous and non-Indigenous children has emerged, as improvements in non-Indigenous children were not fully reproduced in Indigenous children. Delayed vaccination is a concern, especially for diseases where multiple vaccine doses are required for protection and the disease risk among young infants is significant (e.g. pertussis). Immunisation at the earliest appropriate age should be a public health goal for countries such as Australia where high levels of vaccine coverage at milestone ages have been achieved.

The ACIR has shown the rapid uptake of new vaccines and consistently high coverage for all vaccines, unlike some other developed countries.[32,33] In comparison with similar countries, reported coverage at 12 months of age is higher, and with almost 2% of children not vaccinated due to parental objection, targeting of on time vaccination is required to significantly improve the current levels of >91% 'fully immunised' at 12 months of age.[33] The reporting of national small area coverage data has not been noted elsewhere. Areas of low coverage have been identified in many remote areas and areas containing higher proportions of conscientious objectors. Vaccination timeliness has been reported elsewhere but not routinely.[9]

Coverage data for HPV from the national HPV register reflect a successful school-based program with lower coverage for the catch-up program.[34,35]

Coverage estimates for the elderly are comparable with other developed countries.[36,37]

Unfortunately, coverage data are not available for Indigenous adolescents. For adults, data are only available from the Aboriginal and Torres Strait Islander Health Survey, last conducted in 2004-05.[38]

Data provided in this report reflect continuing successful delivery of the NIP in Australia, while identifying some areas for improvement. Coverage for varicella and rotavirus vaccines are below those for other vaccines, and is low in some small geographic areas. Timeliness of vaccination could be improved, particularly for Indigenous infants, and coverage for vaccines recommended only for Indigenous infants is lower than for other vaccines. It was recently announced that varicella and other NIP vaccines (meningococcal C and pneumococcal conjugate vaccines) will be included in coverage assessments for 'fully immunised', and thereby in eligibility for provider and parent incentives, from 2012.[29] It will be important to evaluate the impact of this change in coming years and given the encouraging improvements in timely coverage seen with the changes to reimbursement introduced in 2009 for the 48-month milestone, this promises to have a favourable impact especially for varicella vaccine where high coverage is crucial to long-term outcomes of the program.

Author details

Brynely Hull
Aditi Dey
Rob Menzies
Peter McIntyre

National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases The Children's Hospital at Westmead and University of Sydney Locked Bag 4001 Westmead, NSW 2145

References

1. Hull B, Deeks S, Menzies R, et al. Immunisation coverage annual report, 2007. *Commun Dis Intell*. 2009;33:170-87.
2. Hull B, Mahajan D, Dey A, et al. Immunisation coverage annual report, 2008. *Commun Dis Intell*. 2010;34:241-58.
3. Hull B, Dey A, Mahajan D, et al. Immunisation coverage annual report, 2009. *Commun Dis Intell*. 2011;35:132-48.
4. Hull B, Lawrence G, MacIntyre CR, et al. Immunisation coverage: Australia 2001. Canberra: Commonwealth Department of Health and Ageing; 2002.
5. Hull BP, McIntyre PB, Heath TC, et al. Measuring immunisation coverage in Australia. A review of the Australian Childhood Immunisation Register. *Aust Fam Physician*. 1999;28:55-60.
6. Hull BP, Lawrence GL, MacIntyre CR, et al. Immunisation coverage in Australia corrected for under-reporting to the Australian Childhood Immunisation Register. *Aust NZ Public Health*. 2003;27:533-8.
7. Hull BP, McIntyre PB. Immunisation coverage reporting through the Australian Childhood Immunisation Register - an evaluation of the third-dose assumption. *Aust NZ Public Health*. 2000;24:17-21.
8. Hull BP, Lawrence GL, MacIntyre CR, et al. Estimating immunisation coverage: is the 'third dose assumption' still valid? *Commun Dis Intell*. 2003;27:357-61.
9. Hull BP, McIntyre PB. Timeliness of childhood immunisation in Australia. *Vaccine*. 2006;24:4403-8.
10. Hull BP, McIntyre PB. What do we know about 7vPCV coverage in Aboriginal and Torres Strait Islander children? *Commun Dis Intell*. 2004;28:238-43.

11. Hull BP, McIntyre PB, Couzos S. Evaluation of immunisation coverage for Aboriginal and Torres Strait Islander children using the Australian Childhood Immunisation Register. *Aust NZ Public Health*. 2004;28:47-52.
12. Hull BP, Lawrence GL, MacIntyre CR, et al. Is low immunisation coverage in inner urban areas of Australia due to low uptake or poor notification? *Aust Fam Physician*. 2003;32:1041-3.
13. Hull BP, McIntyre PB, Sayer GP. Factors associated with low uptake of measles and pertussis vaccines - an ecologic study based on the Australian Childhood Immunisation Register. *Aust NZ Public Health*. 2001;25:405-10.
14. Hull BP, Deeks S, Menzies R, et al. What do we know about 7vPCV coverage in Aboriginal and Torres Strait islander children? A 2007 update. *Commun Dis Intell*. 2008;32:257-60.
15. Lawrence GL, MacIntyre CR, Hull BP, et al. Effectiveness of the linkage of child care and maternity payments to childhood immunisation. *Vaccine*. 2004;22:2345-50.
16. Lawrence GL, Hull BP, MacIntyre CR, et al. Reasons for incomplete immunisation among Australian children. A national survey of parents. *Aust Fam Physician*. 2004;33:568-71.
17. Australian Government Department of Human Services Medicare Australia. General Practice Immunisation Incentives (GPII) Scheme. 2007. Available from: http://www.medicare.gov.au/provider/incentives/gpii/index.jsp#N100D3
18. National Health and Medical Research Council. *The Australian immunisation handbook*. Canberra: Australian Government Department of Health and Ageing; 2013.
19. O'Brien ED, Sam GA, Mead C. Methodology for measuring Australia's childhood immunisation coverage. *Commun Dis Intell*. 1998;22:36-7.
20. Department of Health and Aged Care. Measuring Remoteness: Accessibility/Remoteness Index of Australia (ARIA). Occasional Papers, New Series No.14. Canberra: Department of Health and Aged Care; 2001.
21. Rank C, Menzies RI. How reliable are Australian Childhood Immunisation Register coverage estimates for indigenous children? An assessment of data quality and coverage. *Commun Dis Intell*. 2007;31:283-7.
22. Australian Bureau of Statistics. Australian Standard Geographical Classification (ASGC), 2001. Cat. no. 1216.0. Canberra: ABS; 2001.
23. MapInfo. MapInfo version 10.0 [computer program]. 7 ed. New York: MapInfo Corporation; 2009.
24. Australian Bureau of Statistics. Statistical Subdivision from Postal Area 2006 Concordance. Canberra: ABS; 2007. Available from: http://www.abs.gov.au/AUSSTATS/abs@.nsf/39433889d406eeb9ca2570610019e9a5/5942283858e38743ca25730c00009f2e!OpenDocument.
25. Department of Health and Ageing. Immunise Australia. Human Papillomavirus (HPV). Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/immunise-hpv
26. Australian Institute of Health and Welfare. 2009 Adult Vaccination Survey – Summary results. Cat. No. PHE 135.. Canberra. 2011.
27. Buttery JP, Lambert SB, Grimwood K, et al. Reduction in rotavirus-associated acute gastroenteritis following introduction of rotavirus vaccine into Australia's National Childhood vaccine schedule. *Pediatr Infect Dis*. 2011 Jan;30:S25-9.
28. Brisson M, Edmunds W, Gay N, et al. Modelling the impact of immunization on the epidemiology of varicella zoster virus. *Epidemiol Infect*. 2000;125:651-69.
29. Department of Health and Ageing. Immunise Australia Program. Available from: http://www.health.gov.au/internet/immunise/publishing.nsf/Content/home
30. Menzies R, Turnour C, Chiu C, et al. Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia 2003 to 2006. *Commun Dis Intell*. 2008;32 Suppl:S2-S67.
31. Plotkin S, Orenstein WA, Offit PA. Vaccines 5th Edition: Elsevier; 2008.
32. Centre of Disease Control and Prevention. National, state, and local area vaccination coverage among children aged 19-35 months - United States, 2008. MMWR. 2009;58:921-6.
33. Health Protection Agency. NHS Immunisation Statistics, England 2008-09 Report. The Health and Social Care Information Centre; 2009.
34. Ward KF, Menzies RI, Quinn HE, Campbell-Lloyd S. School-based vaccination in NSW. *NSW Public Health Bull*. 2010;21(9-10):237-42.
35. Brotherton J, Gertig D, Chappell G, Rowlands L, Saville M. Catching up with the catch-up: HPV vaccination coverage data for Australian women aged 18-26 years from the National HPV Vaccination Program Register. *Commun Dis Intell*. 201;35(2):197-201.
36. The NHS Information Centre, Public Health Indicators and Population Statistics Team. NHS Immunisation Statistics England 2009-10. Available from: http://www.ic.nhs.uk/webfiles/publications/003_Health_Lifestyles/immstats0910/Immunisations_Bulletin_2009-10.pdf
37. CDC. Prevention and control of influenza: recommendations of the Advisory Committee on Immunization Practices [ACIP]. *MMWR* 2010;59[No. RR-08].
38. Australian Bureau of Statistics. National Aboriginal and Torres Strait Islander Health Survey, 2004-05. Cat. No. 4715.0. Canberra. 2006.

Tuberculosis In Australia: Bacteriologically confirmed Cases And Drug Resistance, 2010

A report of the Australian Mycobacterium Reference Laboratory Network

Richard Lumb, Ivan Bastian, Robyn Carter, Peter Jelfs, Terillee Keehner, Aina Sievers

Abstract

The Australian Mycobacterium Reference Laboratory Network collects and analyses laboratory data on new cases of disease caused by the *Mycobacterium tuberculosis* complex. In 2010, a total of 1,051 cases were identified by bacteriology; an annual reporting rate of 4.7 cases per 100,000 population. Twelve children aged less than 10 years had bacteriologically-confirmed tuberculosis. Results of *in vitro* drug susceptibility testing were available for 1,050 isolates for isoniazid (INH), rifampicin (RIF), ethambutol (EMB), and pyrazinamide (PYZ). A total of 126 (12%) isolates of *M. tuberculosis* were resistant to at least one of these anti-tuberculosis agents. Resistance to at least INH and RIF (defined as multi-drug resistance, MDR) was detected in 37 (3.5%) isolates, including three Australians with extensive travel in high burden TB countries; 33 were from the respiratory tract (sputum n=28, bronchoscopy n=5). Nineteen (65.5%) of the MDR-TB-positive sputum specimens were smear-positive, as were single samples from bronchoscopy and urine. Sixteen patients with MDR-TB were from the Torres Strait Protected Zone. If these Papa New Guinea nationals are excluded from the analysis, the underlying MDR-TB rate in Australian isolates was 2.0%. One case of extensively drug-resistant TB (defined as MDR-TB with additional resistance to a fluoroquinolone and an injectable agent) was detected in 2010.

Keywords: *Mycobacterium tuberculosis*, *Mycobacterium* bovis, laboratory diagnosis, tuberculosis, drug resistance

Introduction

Australia continues to record one of the lowest notification rates (5–6 cases per 100,000 population of tuberculosis (TB) in the world. For non-indigenous persons born in Australia, the notification rate is around 0.7 per 100,000 population.[1] In both 2008 and 2009, more than 85% of notified cases occurred in the overseas-born population. The current epidemiology of TB in Australia is largely a direct effect of the global TB situation with overseas-born persons contributing to a steadily increasing number and proportion of notifications since 2002. In 2007 and 2008, treatment success was attained in 95% of cases, well above the national target of 90%.[2]

Nationals from Papua New Guinea who are able to access Australian health care facilities in the Torres Strait Protected Zone (TSPZ)continue to influence Australian laboratory data. In 2008 and 2009, there were 40 notifications of TB among this patient group reported to NNDSS. Drug susceptibility testing was performed for 30 patients in 2009. Eleven of the 30 patients had MDR-TB and a further three had mono-resistance to rifampicin.[1,3]

Since 1991, the National Notifiable Diseases Surveillance System (NNDSS) has provided statistics on TB notifications reported to public health authorities in Australia's states and territories. The Australian Tuberculosis Reporting Scheme has been conducted by the Australian Mycobacterium Reference Laboratory Network (AMRLN) since 1986. Statistics compiled by the AMRLN relate to cases of bacteriologically-confirmed tuberculosis whereas NNDSS data also includes cases that have been identified on the basis of clinical and epidemiological information, or on non-bacteriological laboratory investigations. This report describes the bacteriologically-confirmed TB diagnoses for the year 2010. This report should be considered in conjunction with the National TB Advisory Committee (NTAC) TB notification report.[4]

Methods

These data are based on clinical specimens that were culture-positive for *Mycobacterium tuberculosis* complex (MTBC). Almost all isolates of MTBC were referred to one of the five laboratories comprising the AMRLN for species identification and drug susceptibility testing. Comparable methodologies are used in the reference laboratories. Relapse cases, as defined by the *National Strategic Plan for TB Control in Australia Beyond 2000* prepared by the NTAC,[2] were included in the laboratory data as laboratories are generally unable to differentiate relapse cases from new cases. Data include temporary visitors to Australia, asylum seekers and persons detained in Australia in correctional services facilities. For each new bacteriologically-confirmed

case, the following information was collected where available: demography: (patient identifier, age, sex, HIV status and state of residence); specimen: (type, site of collection, date of collection and microscopy result); isolate: (*Mycobacterium* species and results of drug susceptibility testing); nucleic acid amplification testing results; and for drug resistant isolates: (patient country of origin, and history of previous TB treatment to determine whether resistance was initial or acquired). Data from contributing laboratories were submitted in standard format to the AMRLN coordinator for collation and analysis. Duplicate entries (indicated by identical patient identifier and date of birth) were deleted prior to analysis. Rates were calculated using mid-year estimates of the population for 2010 supplied by the Australian Bureau of Statistics.[5] For each case, the nature of the first clinical specimen that yielded an isolate of MTBC was used to record the nominal site of disease. Culture-positive specimens collected at bronchoscopy or by gastric lavage were counted as pulmonary disease. Patients with isolates recovered from multiple sites were counted as pulmonary disease (the most important category for public health purposes) if a sputum, bronchoscopy, or lung biopsy specimen was culture positive. Drug resistance among new cases (a proxy for primary resistance) was defined as the presence of resistant isolates of *M. tuberculosis* in patients who, in response to direct questioning, report that they had not received any prior anti-TB treatment (for more than one month) and, in countries where adequate documentation is available, for whom there is no evidence of such a history.[6] Drug resistance among previously treated cases (a proxy for acquired resistance) is defined as the presence of resistant isolates of *M. tuberculosis* in cases who, in response to direct questioning, report having been treated for one month or more, or, in countries where adequate documentation is available, for whom there is evidence of such a history.[6] For 2009 onwards, the AMRLN has been requested by NTAC to provide laboratory data on bacteriologically confirmed isolation of *Mycobacterium bovis* (bacille Calmette Guérin) (BCG).

Results

There were 1,051 bacteriologically-confirmed cases of tuberculosis in 2010 (Figure 1), representing an annual rate of 4.7 cases per 100,000 population. State-specific reporting rates varied from 1.8 (Tasmania) to 10.0 (Northern Territory) cases per 100,000 population (Table 1).

Causative organism

Almost all isolates were identified as *M. tuberculosis* (n=1,045), the remaining isolates being *Mycobacterium africanum* (n=2), *M. bovis* (n=2), *Mycobacterium orygis* (formerly known as the "Oryx" bacillus; n=1) and a mixed *M. tuberculosis* complex/*Mycobacterium avium* isolation (n=1). In addition, a total of 18 *M. bovis* (BCG) were isolated from clinical samples.

Distribution by sex, age and site of disease

Complete information for gender and age was available for 1,047 patients. Of the 1,047 MTBC isolates, 457 (44%) were from females, 589 (56%) were from males, and sex was not recorded for 1 case. The site of disease was dependent upon age and sex. The overall male:female ratio was 1.3:1. For respiratory isolates, the male:female ratio was 1.4:1. For TB lymphadenitis, the female:male ratio was 1.4:1. For males, there were two distinct peaks in age–group specific rates of bacteriologically-confirmed TB; 14.1 cases of TB per 100,000 population at 25 to 29 years and a second peak in elderly males aged

Table 1: Bacteriologically confirmed cases of tuberculosis in Australia, 2000 and 2008 to 2010, and rate per 100,000 population by State or Territory

State or territory	2010		2009*		2008*		2000*	
	n	Rate	n	Rate	n	Rate	n	Rate
New South Wales†	370	4.9	409	5.5	327	4.5	307	4.5
Victoria	344	6.2	331	6.1	299	5.6	231	4.8
Queensland	166	3.7	153	3.4	111	2.6	76	2.1
Western Australia	87	3.8	87	3.9	72	3.3	63	3.3
South Australia	52	3.2	51	3.1	49	3.1	41	2.7
Tasmania	9	1.8	7	1.4	3	0.6	2	0.4
Northern Territory	23	10.0	24	10.9	25	11.4	45	23.0
Total	1051	4.7	1,062	4.9	886	4.1	765	4.0

* Data from previous reports of the Australian Mycobacterium Reference Laboratory Network

† Data from the Australian Capital Territory are included with those from New South Wales.

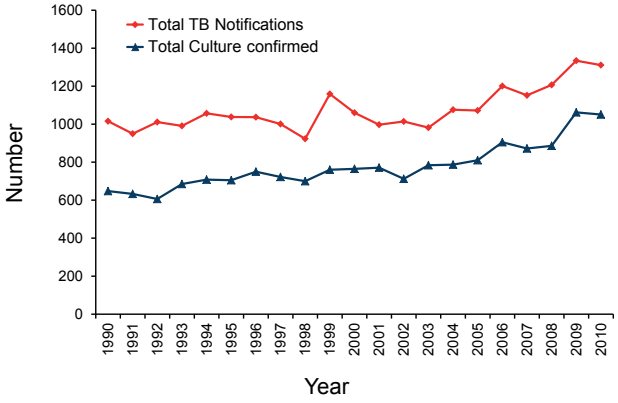
more than 75 years (greater than 9 cases of TB per 100,000 population). The age distribution of female cases was similar with 12.5 and 7.3 bacteriologically-confirmed TB cases per 100,000 population in the 25 to 29 and 80 to 84 year age groups, respectively. The median age for patients with bacteriologically-confirmed disease was 32 years for both males and females. The predominant culture-positive respiratory specimen was sputum (n=505), a further 137 specimens were obtained from bronchoscopy, 10 were aspirates, and 3 from lung biopsies. Fifty-five pleural specimens (44 fluid, 11 biopsy/tissue) were culture positive. The most commonly encountered extrapulmonary culture-positive specimen was lymph tissue (n=184) followed by pleural (n=55), bone/joint (n=32), genitourinary tract (n=24), and peritoneal (n=23) specimens.

Six children aged under 10 years (male n=2, female n=4) had bacteriologically-confirmed tuberculosis (sputum n=3, gastric aspirate n=2, bronchoscopy n=1). None of the specimens were smear positive and all were fully susceptible to first line anti-TB drugs. An additional 6 children (males n=5) from TSPZ were bacteriologically confirmed for TB (an isolate each from lymph node, brain tissue, leg aspirate, urine, sputum, gastric aspirate). Of these, 4/6 specimens were smear positive including sputum and brain tissue. Drug resistance was prevalent amongst cases in children from the TSPZ, including MDR (n=2) and resistance to streptomycin and isoniazid (n=2).

Association with HIV

The AMRLN database recorded the HIV status of only 91 (8.6%) patients. One patient was identified as being HIV-seropositive.

Figure 1: Tuberculosis notifications and laboratory data, 1990 to 2010, by year



Microscopy

Microscopy was available for 1,010 of the bacteriologically-confirmed cases in 2010. Microscopy was not performed on 33 specimens and no result was provided for the remaining 8 specimens. The majority of samples without a microscopy performed were from extrapulmonary sites. Smears were positive in 267 of 505 (52.9%) sputum and 37 of 137 (27.0%) bronchoscopy specimens respectively (Table 2). Of 55 pleural specimens (11 biopsy and 44 fluids) that were culture-positive for *M. tuberculosis* and reported a microscopy result, 4 fluids and 1 biopsy were smear-positive. Lymph node specimens were smear-positive in only 38 of 184 (20.6%) of cases.

Drug susceptibility testing

Results of *in vitro* drug susceptibility testing were available for all but one isolate (1,050/1,051) for isoniazid (INH), rifampicin (RIF), ethambutol (EMB), and pyrazinamide (PYZ). A total of 126 (12.0%) *M. tuberculosis* isolates were resistant to at least one of these. Resistance to at least INH and RIF (defined as MDR-TB) was detected in 37 (3.5%) isolates. All of the MDR-TB isolates were *M. tuberculosis* (Table 3). Of the 37 MDR-TB isolates, 33 were from the respiratory tract (sputum n=28, bronchoscopy n=5). Isolates of MDR-TB were obtained from the following non-respiratory sites: lymph node (n=2), a thigh aspirate and a urine specimen.

Sixteen patients with MDR-TB were from the TSPZ, and these patients access health services in the outer TSPZ. MDR-TB was also isolated from patients born in the Philippines (n=4), India (n=3, China (n=2), Vietnam (n=3), Myanmar (n=2) with a single case each from Ethiopia, Papua New Guinea, South Korea, and unknown. Three Australians were identified with MDR-TB, all had a history of extensive travel in high-burden countries including the Philippines and South Africa. In the past three years (2005-07), the impact of MDR-TB cases from the TSPZ have lifted the proportion of MDR-TB cases above the 0.5 – 2.0% range (Figure 2).

In 2010, 3.5% of all isolates were MDR-TB, but only 2.0% when the Papua New Guinea TSPZ isolates were excluded. When the TSPZ isolates were excluded, 21 MDR-TB cases were documented from patients living in Australia. Of these, 14 were sputum, 5 were bronchoscopy specimens, and two were from lymph nodes. Ten of the sputum specimens were smear positive, as was a single bronchial washing.

The revised definition of extensively drug resistant resistant-TB (XDR-TB) is an isolate that has resistance to at least INH and RIF (MDR-TB) plus

Table 2: Site of specimens smear- and culture-positive for *Mycobacterium tuberculosis* complex, 2010

	N*	Smear positive	%
Sputum	505	267	52.9
Bronchoscopy	137	37	27.0
Lymph node	184	38	20.6
Pleural	55	5#	9.1
Genito-urinary	24	†	
Bone/Joint	32	†	
Peritoneal	23	†	
Skin	5	†	
CSF	10	†	

* Based on specimens that reported a microscopy result and excludes (i) microscopy not performed or (ii) result unknown

\# One pleural biopsy and four pleural fluids were smear positive

† Percentage of specimens smear positive not calculated due to the small number of cases

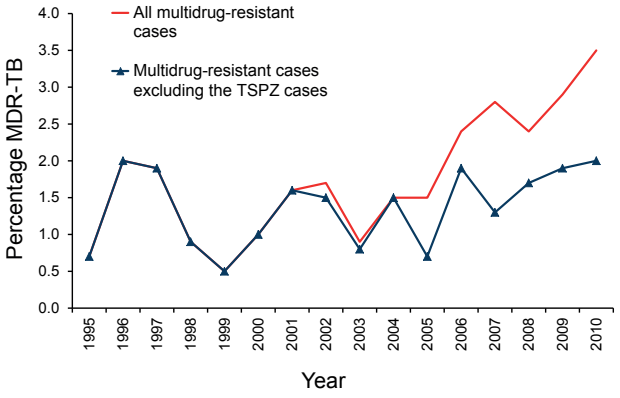
Table 3: Drug resistance patterns in multi-drug resistant strains of tuberculosis, Australia 1995 to 2010

Resistance pattern (standard drugs)*	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010
H+R only	3	10	6	2	2	3	8	8	4	7	5	16	16	10	21	18
H+R+E	1	1	1	1	1	1	1	1	2	2	3	1	2	3	1	1
H+R+Z	1	4	5	2	1	3	3	1	1	1	1	0	5	3	7	15
H+R+E+Z	0	0	2	1	0	1	0	2	0	2	3	5	1	5	2	3
XDR-TB	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	1
TOTAL (%)	5 (0.7)	15 (2.0)	14 (1.9)	6 (0.9)	4 (0.5)	8 (1.0)	12 (1.6)	12 (1.7)	7 (0.9)	12 (1.5)	12 (1.5)	22 (2.4)	24 (2.8)	21 (2.4)	31 (2.9)	37 (3.5)

* the streptomycin result was not considered for this table

H = isoniazid, R = rifampicin, E = ethambutol, Z = pyrazinamide

Figure 2: Percentage MDR-TB in Australia; all cases excluding the TSPZ zone



additional resistance to a fluoroquinolone and an injectable (kanamycin, amikacin, capreomycin).[10] In 2010, there was a single case of XDR-TB in a 33 year old male where the isolate was resistant to amikacin and ciprofloxacin (but was susceptible to moxifloxacin). One other MDR-TB isolate also had resistance to a quinolone.

Mono-resistance to isoniazid (INH) was detected in 49 isolates; mono-resistance to RIF (n=3), EMB (n=1), and PYZ (n=4) was also detected (Table 4). One hundred and ten isolates demonstrated resistance to INH at a concentration of 0.1 mg/L. Of these, 62 (56.3%) demonstrated resistance to INH at the higher level of 0.4 mg/L. Among MDR-TB strains, 15/37 (40.5%) demonstrated INH resistance at the higher concentration (0.4 mg/L). Forty-seven (37.3%) of 126 of specimens culture-positive for drug resistant strains, including 38 of 88 (43.2%) sputum or bronchoscopy specimens, were smear-positive for acid-fast bacilli. The single *M. bovis* isolate which is inherently resistant to pyrazinamide, was not included in the above analyses.

Results of testing for streptomycin (STR) were available for 345 of 1,050 (32.9%) isolates with 54 demonstrating resistance to at least STR; 8 had mono-resistance, 19 were resistant to STR and INH, and 25 of 31 MDR-TB strains reporting a S-result were also STR-resistant.

New or previously treated cases, and country of birth

Of the 126 *M. tuberculosis* isolates resistant to at least one of the standard drugs, 61 (48.4%) were from new cases, 7 (5.5%) from previously treated cases, and no information was available on the remaining 58 cases. Nine were Australian-born, 116 were overseas- born, and the country of birth of one case was unknown. The 116 overseas-born persons with drug resistant disease were from 20 countries; most frequently from Papua New Guinea (including TSPZ) (n=23), India (n=14), Vietnam (n=13), China (n=10), Philippines (n=8), and Myanmar (n=6).

Table 4: Drug resistance profiles, 2010

	2010
ANY RESISTANCE	
S	54
H	110
R	40
E	7
Z	26
MONO RESISTANCE	**(65)**
S	8
H	49
R	3
E	1
Z	4
MDR-TB	**(37)**
HR	4
HRZ	7
HREZ	1
SHR	15
SHRE	1
SHRZ	7
SHREZ	2
XDR-TB	**(1)**
HRZ + resistance to amkican, capreomycin, ciprofloxacin	1
POLY RESISTANCE	**(24)**
SH	19
SHE	1
SHEZ	1
Total isolates	1,051
Total isolates and DST completed	1,050
Fully susceptible	924

Isolation of *M. bovis* (BCG)

There were 18 isolations of *M. bovis* (BCG) in 2010. Thirteen were cultured from males (4 aged ≤ 5 years) and from 5 females (4 aged ≤ 5 years). Seven isolations were from the vaccination site or axilla, and all were children aged ≤ 5 years. Nine males (age range: 60 to 86 years) had *M. bovis* (BCG) isolated from urine. In addition, *M. bovis* (BCG) was isolated from an ankle biopsy from a two-year old female, and also buttock pus from a 22 year old female.

Discussion

The detection of 1,051 laboratory-confirmed cases of TB in 2010 representing 4.7 cases per 100,000 population is slightly lower than the 4.9 cases per 100,000 population reported in 2009.[3] Previously, the incidence of bacteriologically confirmed TB was between 3.5 and 4.4 cases per 100,000 population (see previous AMRLN reports) but has now risen above the upper limit twice in the past two years. As expected, the number of cases notified to the NNDSS was higher than for bacteriologically confirmed TB.[3] The most frequent reasons postulated for the extra cases reported in the NNDSS database include: diagnosis of childhood and extrapulmonary TB based on clinical, radiological and epidemiological information, and submission of extrapulmonary samples in formalin precluding bacteriological investigations. There were 1,311 notifications of tuberculosis in 2010 compared with 1,051 (80.2%) cases confirmed bacteriologically.[4] In the past decade, the proportion of notifications confirmed by culture has stayed within a range of 70-80% bacteriological confirmations (see previous AMRLN reports).

The number of isolates with resistance to any drug (including streptomycin) was 126 (12.0%), a reduction from the peak of 15.9% recorded in 2009.[3] Mono-resistance to INH remains the most frequently encountered mono-resistance profile in Australia. Resistance to INH at higher MIC levels was observed in 56.3% of isolates. High level INH resistance is associated with mutations in *katG* whilst low level INH resistance is associated with mutations in *inhA* and *ethA*.[7,8,9] Strains with low-level INH resistance may have cross-resistance to ethionamide, a structural analogue of INH.[8,9] Ethionamide is a second-line drug used commonly in standard regimens for treatment of drug-resistant TB.[10]

MDR-TB remains at a low but concerning level. Since the AMRLN began preparing annual reports in 1985, the proportion of patients with MDR-TB has stayed within a band of 0.5-2.0%, but since 2000, the influence of MDR-TB cases occurring in people moving within the TSPZ has pushed the percentage above 2%. In 2010, when the 16 TSPZ isolates were included, the MDR-TB rate was 3.5%, the highest level since records began in 1985. When the TSPZ isolates were excluded, the proportion of MDR-TB isolates was 2.0%. In 2010, there were no TSPZ patients with mono-rifampicin resistance compared with three patients in 2009.

For the first time since 2004, an XDR-TB isolate was reported. The isolate from the smear-positive patient was resistant to STR, INH, RIF, EMB, PYZ, amikacin, capreomycin, ofloxacin, and ethionamide. Since 1995, 257 cases of MDR-TB have been documented, and of these, only 2 (0.8%) have been confirmed using current internationally-recognised breakpoints for drug susceptibility testing (DST) as XDR-TB (see previous AMRLN reports).

The emergence of "totally drug-resistant TB" (TDR-TB) in Iran and now India was the culmination of *M. tuberculosis* acquiring mutations associated with resistance to ever more classes of anti-TB agents.[11] The Indian report described four cases from the Hinduja hospital where resistance was documented for all first-line (INH, RIF, EMB, PYZ, STR) and second-line (kanamycin, capreomycin, amikacin, levofloxacin, moxifloxacin, ethionamide and para-amino-salicylic acid, PAS) anti-TB drugs.[12] Erratic treatment by multiple doctors in private practice was held responsible for the emergence of TDR-TB. There are now 14 TDR-TB cases in the Hinduja hospital. In a recent editorial, one of the authors involved in reporting the Indian cases wrote a scathing account of the global failure to utilise anti-TB drugs in a manner that did not generate drug resistance.[13] Exposure to the wrong drugs and/or to the wrong regimen, and/or to the wrong doses had created a selective pressure for the stepwise accumulation of mutations associated with resistance.

The definition and reliable detection of TDR-TB is confounded by some laboratory issues. Although drug susceptibility testing of the injectables and fluoroquinolones is now well established and shown to provide reliable consistent results; that cannot be said for PYZ, ethionamide, and PAS.[14] Critical concentrations for kanamycin and PAS were not described in the 2008 WHO policy guidance document on DST for second-line anti-tuberculosis drugs, and were provided in a guidance update released at a Global Laboratory Initiative meeting in April 2012.[15] The critical concentration (MGIT 960) for kanamycin is 2.5μg/ml and 4.0 μg/ml for PAS. The critical concentration for levofloxacin has been reduced from 2.0 μg/ml to 1.5 μg/ml, and moxifloxacin has been revised upwards from 0.25 μg/ml to 0.5 μg/ml and also tested simultaneously at 2.0 μg/ml. There is still no critical concentration for cycloserine in liquid culture. Drug susceptibility testing for ciprofloxacin is no longer recommended.[15]

The 2010 laboratory data raise a biosafety issue and a public health issue. Firstly, 27% of bronchoscopy specimens were smear-positive. Bronchoscopic samples are superior to sputa (which may not always be obtainable) so some sputum-smear-negative but bronchoscopy-smear-positive cases will always be expected. Nonetheless, performing bronchoscopy on sputum smear-positive cases subjects them to a needless invasive procedure and exposes healthcare workers to *M. tuberculosis*. Bronchoscopists are therefore encouraged whenever possible to ensure that patients are confirmed as sputum-smear-negative before proceeding to bronchoscopy. Secondly, 65.5%

of the MDR-TB-positive sputum specimens in 2010 were smear-positive and therefore infectious. The state tuberculosis services, in collaboration with the laboratories, medical practitioners, public health authorities, migrant groups and other stakeholders, must attempt to minimise the interval between symptom onset and MDR-TB diagnosis and management, and hence curtail the infectivity of MDR-TB patients.

The much anticipated merging of the AMRLN and NNDSS databases has experienced further delays due to information technology limitations and transitions in various states. A combined database will not be available before 2012 dataset at the earliest. In the interim, Australia must continue to provide a combined prevalence of drug resistance and remains unable to provide comprehensive data to the World Health Organization global reports sub-classifying drug-resistance between new cases and previously-treated patients.

Acknowledgements

The Australian Mycobacterium Reference Laboratory Network comprises the Mycobacterium Reference Laboratories at the following facilities:

- SA Pathology, Adelaide, South Australia.
- Queensland Health Pathology Services, Herston Hospitals Complex, Herston, Queensland.
- Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria.
- PathWest Laboratory Medicine WA – QEIIMC, Hospital Avenue, Nedlands, Western Australia.
- Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales.
- Additional information and support from Ms Amanda Christensen, Dr Ral Antic, Dr Vicki Krause, Ms Lynne Brown, Dr Anastasios Konstantinos, and Dr Justin Waring is gratefully acknowledged.

Author details

Richard Lumb[1,2]
Ivan Bastian[1,2]
Robyn Carter[2]
Peter Jelfs[2]
Terillee Keehner[2]
Aina Sievers[2]

1. Microbiology and Infectious Diseases, SA Pathology,Adelaide, South Australia
2. Australian Mycobacterium Reference Laboratory Network

Corresponding author: Mr Richard Lumb, Mycobacterium Reference Laboratory and WHO Supranational TB Reference Laboratory, Microbiology and Infectious Diseases, SA Pathology, Adelaide, South Australia, PO Box 14, Rundle, Mall, Adelaide, South Australia 5000. Telephone: +61 8 8222 3579, Facsimile: +61 8 8222 3543, Email: richard.lumb2@health.sa.gov.au

References

1. Barry C, Waring J, Stapledon R, Konstantinos A and the National Tuberculosis Advisory Committee, for the Communicable Diseases Network Australia. Tuberculosis notifications in Australia 2008 and 2009. *Commun Dis Intell* 2012;36(1):82–94.
2. National Tuberculosis Advisory Committee. National strategic plan for TB control in Australia beyond 2000. Australian Government Department of Health and Ageing [Internet]; [cited 2002] Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-other-tb_plan.htm/$FILE/tbstrat_plan.pdf
3. Lumb R, Bastian I, Carter R, Jelfs P, Keehner T, Sievers A. Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2008 and 2009. *Commun Dis Intell* 2011(2):154-161.
4. NNDSS Annual Report Writing Group. Australia's notifiable disease status, 2010: annual report of the National Notifiable Diseases Surveillance System. *Commun Dis Intell*. 2012;36(1).
5. Australian Bureau of Statistics. Cat no. 3101.0 Australia Demographic Statistics [Internet], June 2010. Available from: http://www.abs.gov.au/ausstats/abs@.nsf/mf/3101.0/
6. World Health Organization. Anti-tuberculosis drug resistance in the world. [Internet] 2008;4. Available from http://whqlibdoc.who.int/hq/2008/WHO_HTM_TB_2008.394_eng.pdf
7. Zhang Y, Heym B, AllenB, Young D, Cole S. The catalase-peroxidase gene and isoniazid resistance of *Mycobacterium tuberculosis*. *Nature* 1992;358:591-593.
8. Banerjee A, Dubnau E, Quemard, et al. inhA, a gene encoding a target for isoniazid and ethionamide in *Mycobacterium tuberculosis*. *Science* 1994;263:227-230.
9. Morlock GP, Metchock B, Sikes D, Crawford JT, Cooksey R. ethA, inhA and katG loci of ethionamide-resistant clinical *Mycobacterium tuberculosis* isolates. *Antimicrob Agents Chemother* 2003;47(12): 3799-3805.
10. World Health Organisation. Guidelines for the programmatic management of drug-resistant tuberculosis. [Internet] 2011 update. Available from http://whqlibdoc.who.int/publications/2011/9789241501583_eng.pdf
11. World Health Organisation. "Totally Drug-Resistant" tuberculosis: a WHO consultation on the diagnostic definition and treatment options. [Internet] 21-22 March 2012. [Accessed 14th August 2012]. Available from http://www.who.int/tb/challenges/xdr/xdrconsultation/en/
12. Udwadia ZF, Amale RA, Ajbani KK, Rodrigues C. Totally drug-resistant tuberculosis in India. *Clin Infect Dis* 2012; 54:579-581.
13. Udwadia ZF. Totally drug-resistant tuberculosis in India: Who let the djinn out? *Respirology* 2012; 17: 741-742.
14. World Health Organization. Policy guidance on drug-susceptibility testing (DST) of second-line antituberculosis drugs. Geneva; World Health Organization. [Internet] Available from http://www.who.int/tb/publications/2008/whohtmtb_2008_392/en/index.html
15. Global Laboratory Initiative. 4th Annual GLI Meeting/ Consultation of the SRL network/Early implementers meeting on Xpert MTB/RIF roll out, April 2012, Annecy, France. [Internet] Summary of outcomes from WHO Expert Group Meeting on DST for anti-TB drugs. Available from http://www.stoptb.org/wg/gli/meetingarchive.asp

Original articles

ß-lactamases in *Salmonella enterica* isolated in Australia

Gino Micalizzi

Abstract

Understanding the antibiotic susceptibility of *Salmonella enterica* is important both from a clinical treatment and a public health perspective. The emergence of extended spectrum ß-lactamases (ESßLs) and AmpC ß-lactamases in *S. enterica* is important, as this will limit treatment options and could provide a strain with a significant selective advantage. The aim of the study was to screen isolates of *S. enterica*, including isolates that had previously shown antibiotic resistance, to gauge the extent of ß-lactamase activity in *S. enterica* in Australia. Phenotypic detection involved screening in accordance with Clinical and Laboratory Standards Institute double disk synergy test guidelines and assessing susceptibility to cefoxitin. Presumptive positives were then screened using a MAST® AmpC & ESßL detection set. *S. enterica* isolates that were consecutively received in the laboratory (n=624), or had previously exhibited some antibiotic resistance (n=351), were screened for ß-lactamase activity. None of the isolates in the second group were included in the first. ß-lactamase activity was detected in nine of the consecutively received isolates; one with demonstrated ESßL activity and eight others with demonstrated AmpC ß-lactamase. ß-lactamase activity was detected in 16 of the isolates that had previously demonstrated some antibiotic resistance; three with demonstrated ESßL activity and 13 others with demonstrated AmpC ß-lactamase activity. *S. enterica* serovar Stanley is a serovar that is frequently acquired overseas and this serovar had the highest proportion of isolates that demonstrated ß-Lactamase activity in consecutively sampled isolates (4.95%), reflecting the emergence of an epidemic clone within South East Asia. While antibiotic resistance is being detected in *Salmonella* isolates, the data indicates that there is limited awareness of, or screening for, ß-lactamases in *S. enterica*. This study will help to overcome these deficiencies and provide some baseline surveillance data against which future trends can be measured. *Commun Dis Intell* 2013;37(1):47–51.

Keywords: extended spectrum ß-lactamase, AmpC ß-lactamase, *Salmonella enterica*, Queensland, Australia, antibiotic, resistance, surveillance

Introduction

Salmonella enterica belongs to the family *Enterobacteriaceae* and is one of the main causes of foodborne illness worldwide. The most common form of disease caused by non-typhoidal *Salmonella* is gastroenteritis or salmonellosis. Extra-intestinal infection resulting in bacteraemia occurs in approximately 5 per cent of cases. Other invasive diseases such as meningitis, osteomyelitis and pneumonia are more likely in high risk groups, such as the very young or the immuno-compromised.[1,2]

To date, over 2,500 different *S. enterica* serovars have been identified.[3] In Australia, approximately 150 of these are responsible for the majority of human infections, caused mainly by the consumption of contaminated food or drink.[2,3] In Australia, 9,249 laboratory-confirmed human cases were notified to the National Enteric Pathogens Surveillance Scheme in 2009, at a rate of 42.1 cases per 100,000 population, with 11 per cent of these infections known to have been acquired overseas.[2] Internationally, *Salmonella* serotyping provides the primary epidemiological information and data required for the surveillance and control of salmonellosis. The global distribution of serovars varies greatly and information about whether a certain serovar or strain exhibits antibiotic resistance is of public health importance to other countries.[3] This epidemiological information is critical for developing appropriately targeted interventions to control the emergence and dissemination of a resistant strain.

Whilst intestinal infections are usually self-limiting, antimicrobial treatment of non-typhoidal *Salmonella* is required with invasive disease or in high risk patients.[3] The highest age-specific incidence of *Salmonella* infection in 2009 was 300 cases per 100,000 population in children aged 0–1 year.[4] Consequently, treatment is often required in these higher risk patients. Third generation cephalosporins are in many cases the drugs of choice for treatment, particularly in infants to whom fluoroquinolones should not be administered due to concerns regarding toxicity.[5] Therefore, the emergence of resistance in *S. enterica* to this group of antibiotics will severely restrict treatment options.

There are two main classes of ß-lactamases that confer resistance to 3rd generation cephalosporins that are being reported in *S. enterica*.[5] The first group are extended-spectrum beta-lactamases (ESßLs), which belong to the Molecular Class A ß-lactamase enzymes.[6,7] First described in *Klebsiella sp* (SHV) and *Escherichia coli* (TEM), an evolving group of over 600 different ESßLs have been reported, mainly arising from point mutations of the original SHV and TEM genes with many emerging in *S. enterica*.[6,7,8] More recently, a new rapidly proliferating group of ESßL enzymes called CTX-M that are preferentially active against cefotaxime have arisen, found mainly in strains of *S.* Typhimurium and *E. coli*.[6,8] The second group are AmpC ß-lactamases, which were first reported in 1988 and belong to molecular class C ß-lactamase enzymes and 50 variants have since been described.[7,9] These enzymes are derived from chromosomal genes in organisms such as *Enterobacter cloacae* and *Citrobacter* sp that can be transferred via mobile genetic elements to other *Enterobacteriaceae* that do not possess these genes, such as *S. enterica*.[7,9]

Routine antimicrobial susceptibility testing (AST) may not detect the presence of an ESßL or AmpC ß-lactamase as the high breakpoints used in AST and the differing levels of activity against various cephalosporins can make detection difficult.[10] The inappropriate use of cephalosporins to treat invasive infections caused by ESßL or AmpC ß-lactamase producing *Enterobacteriaceae* such as *S. enterica* have been associated with increased rates of morbidity and mortality.[11] This is a particular problem in health care environments where antibiotic selective pressure is conducive to the acquisition of these resistant genes.[10] This risk extends beyond patients. Infection through exposure with carriage of ESßL producing *Salmonella* sp has been reported among health care workers.[12]

In the last decade, the emergence of ESßLs and AmpC ß-lactamases in *S. enterica* populations have been reported from many countries.[5,10,13–18] Whilst AST is carried out when required for individual patient treatment and by reference laboratories as an epidemiological tool, little is known of the prevalence of ESßLs or AmpC ß-lactamases within *S. enterica* isolated in Australia.

The main aim of this study was to determine if *S. enterica* isolated in Australia had phenotypically detectable ESßL or AmpC ß-lactamase resistance and whether there were differences among serovars, thereby providing the initial baseline data against which to measure future trends.

Methods

Isolates

The study included a selection of isolates from two separate groups, all of which were human clinical isolates received by the *Salmonella* Reference Laboratory in Queensland from diagnostic laboratories between 2000 and 2009. Isolates were serologically classified according to the White-Kauffmann-LeMinor scheme, as recommended by the World Health Organization Collaborating Centre for Reference and Research on *Salmonella*.

The first group included isolates from six different serovars consecutively received by the laboratory (n=624). These included *S. enterica* serovar Typhimurium (n=100), *S.* Virchow (n=111), *S.* Aberdeen (n=97), *S.* Enteritidis (n=114), *S.* Weltevreden (n=101) and *S.* Stanley (n=101).

The second group consisted of *S.* Typhimurium (n=298) and *S.* Virchow (n=53) isolates that had exhibited resistance to at least one of the following antibiotics (µg/ml): ampicillin 32; streptomycin 5; tetracycline 20; chloramphenicol 10; sulphathiazole 550; trimethoprim 50; kanamycin 10; nalidixic acid 50; spectinomycin 50; gentamicin 2.5; ciprofloxacin 0.06 or cefotaxime 1.0 as part of epidemiological typing. The screening of these isolates for this study was thought to improve the likelihood of detecting ß-lactamase resistance, which was expected to be very low. None of the isolates from the second group were included in the first group.

Phenotypic method

Isolates were screened as per Clinical and Laboratory Standards Institute (CLSI) recommendations for screening and confirmatory tests for ESßLs in *Klebsiella pneumoniae, Klebsiella oxytoca, E. coli* and *Proteusmirabilis* (M100-S19).[19,20] The screen is based on demonstrating synergy with cephalosporin (ceftazidime, CAZ, 30 µg and cefotaxime, CTX, 30 µg) and with cephalosporin in combination with clavulanic acid (CAZ/CLA 30/10 µg and CTX/CLA 30/10 µg).[19,20] Cefoxitin 30 µg (FOX 30 µg) was included in the screen as AmpC ß-lactamase can also demonstrate activity against cephamycins.[16,20,21] In organisms that harbour both an ESßL and AmpC ß-lactamase, the induction of AmpC ß-lactamases by clavulanic acid can potentially mask the positive synergistic reaction that confirms ESßL activity.[10,20] This concern is particularly pertinent as ESßLs and AmpC ß-lactamases have reportedly been found in conjunction in *S. enterica*.[22] To overcome this problem isolates that exhibited resistance to CAZ, CTX or

FOX were further tested to differentiate and distinguish between the two mechanisms of resistance. Currently, no recommendations exist for ESßL detection and reporting in the presence of AmpC ß-lactamase.[19,20]

MAST® (D68C) has developed an antibiotic detection set incorporating four discs that contain a combination of cefpodoxime with either and both an AmpC ß-lactamase and ESßL inhibitor to enable the differentiation of both mechanisms of resistance.

Controls included were *E. coli* ATCC 25922 as the negative control, *K. pneumoniae* ATCC 700603 as the ESßL positive control and *E. cloacae* NCTC 13406 as the AmpC ß-lactamase positive control.

Results

Nine of the 624 consecutively isolated serovars screened demonstrated ß-lactamase activity. Phenotypic ESßL activity was detected in one of these isolates and phenotypic AmpC ß-lactamase activity was detected in eight others (Table 1).

Table 1: ESßL and AmpC ß-lactamase phenotypes in isolates

Serovar	Number of isolates	Number of ESβLs	Number of AmpC	Total
S. Typhimurium	100	0	0	0
S. Virchow	111	0	1	1
S. Aberdeen	97	1	0	1
S. Enteritidis	114	0	2	2
S. Weltevreden	101	0	0	0
S. Stanley	101	0	5	5
Total	624	1	8	9

Table 2: Resistance profiles of ß-lactamase positive *Salmonella* Typhimurium and *S.* Virchow isolates that had previously demonstrated antibiotic resistance

Number of positive / total	ß-lactamase class	Antibiotic resistance profiles
S. Typhimurium	ESβL	Amp, S, T, C, Su, Tm, K, Sp, G, Cp, Cf
n=11 / 298 (3.7%)	ESβL	Amp, Tm, G, Cf
	AmpC	Amp, Cf
	AmpC	Amp, S, Su, Cf
	AmpC	Amp, Cf
	AmpC	Amp, T, C, Tm, Na, Sp, Cp, Cf
	AmpC	Amp, S, T, Cf
	AmpC	Amp, Cf
	AmpC	Amp, Cf
	AmpC	Amp, Cf
	AmpC	Amp, Cf
S. Virchow	ESβL	Amp, G, Cf
n= 5 / 53 (9.4%)	AmpC	Amp, Cf
	AmpC	Amp, Cf
	AmpC	Amp, Cf
	AmpC	Amp, Cf

Amp ampicillin (32 µg/ml); S streptomycin (25 µg/ml); T tetracycline (20 µg/ml); C chloramphenicol (10 µg/ml);Su sulphathiazole (550 µg/ml); Tm trimethoprim (50 µg/ml); K kanamycin (10 µg/ml); Na nalidixic acid (50 µg/ml);Sp spectinomycin (50 µg/ml); G gentamicin (2.5 µg/ml); Cp ciprofloxacin (2.0 µg/ml); Cf cefotaxime (1)

Eleven of the 298 *S*. Typhimurium isolates that had previously demonstrated antibiotic resistance were ß-lactamase positive, with two demonstrating ESßL activity and nine possessing AmpC ß-lactamase. (Table 2). Five of the 53 *S*. Virchow isolates that had previously demonstrated antibiotic resistance demonstrated ß-lactamase activity. One of these isolates demonstrated ESßL activity and four AmpC ß-lactamase activity (Table 2).

The antibiograms for each of the ß-lactamase positive *S*. Typhimurium and *S*. Virchow isolates are shown in Table 2.

Discussion

This study demonstrates the presence of ß-lactamase activity in some isolates of *S. enterica* in Queensland and therefore antibiotic treatment failure is a distinct possibility. This may not apply to the rest of Australia as geographical distribution of salmonellosis and serovars varies greatly between states and territories.[1,2] Specifically, of the isolates that had previously demonstrated some antibiotic resistance, 21 produced AmpC ß-lactamase and four were ESßL producers. AmpC ß-lactamase was observed more frequently than ESßL in *S. enterica* indicating that resistance to 3rd generation cephalosporins is more likely to be a result of an AmpC ß-lactamase rather than an ESßL. In this study, there was no evidence of the coexistence of both an ESßL and AmpC ß-lactamase in the same *S. enterica* isolate, which has been found elsewhere.[22]

The results of this study suggest an epidemiological association between certain *Salmonella* serovars and ß-lactamase activity. The serovars with the highest proportions of isolates demonstrating AmpC ß-lactamase activity were *S*. Enteritidis (1.75%) and *S*. Stanley (4.95%). These serovars are predominately associated with overseas-acquired infections particularly in Indonesia and Thailand.[2,4] The high proportion of *S*. Stanley isolates demonstrating AmpC ß-lactamase activity reflects the recently emerged epidemic clone seen in South East Asia, particularly Thailand and Taiwan. Not surprisingly, 3 cases reported overseas travel specifically to Thailand prior to onset of illness.[15]

Laboratories should remain vigilant regarding the possibility of ß-lactamase activity when overseas travel has been reported, particularly if the isolate demonstrates antibiotic resistance to any ß-lactam antibiotics. It is also imperative from a clinical perspective that the case's travel history be reported in clinical notes. From a public health perspective, the introduction of these exotic serovars into Australia is a concern, as the exchange of genetic material occurs readily across species of the same genera. This could provide a native strain with a significant selective advantage, resulting in the rapid dissemination of the strain into environmental niches as seen with *S*. Newport multidrug-resistant-AmpC overseas.[1,18]

Among isolates that had previously demonstrated resistance as part of epidemiological typing, ß-Lactamase activity contributed to 3.7% of the resistance observed in *S*. Typhimurium isolates and a much higher 9.4% in *S*. Virchow isolates. This is of concern because *S*. Virchow continually demonstrates a higher rate of invasiveness (8.7%) compared with other serovars.[2] Overall, multiple antibiotic resistance was more evident with ESßLs than with AmpC ß-lactamases, as all 3 positive ESßL isolates demonstrated resistance not only to ß-lactams but to a wide range of antibiotics. By contrast, only three (3/13) of the positive AmpC ß-lactamase isolates demonstrated resistance to antibiotics other than ß-lactams. This would suggest that the coexistence of multiple resistance mechanisms is more likely to occur with strains that demonstrate ESßL activity than with AmpC ß-lactamase. Both of these antibiograms should alert laboratories to the potential existence of ß-lactamase activity in *S. enterica* and should be further investigated.

Laboratory capacity to detect ß-lactamase activity in *S. enterica* will also be of public health benefit as antibiotic resistance profiling has in the past proven to be a valuable tool in linking cases in outbreak investigations.[2] Whilst none of the ß-lactamase-producing isolates in this study can be directly associated with a defined outbreak, outbreaks caused by ß-lactamase-producing *S. enterica* have occurred overseas.[10,12] The probability of such an event occurring in Australia will increase as the dissemination of ß-lactamase resistance in *S. enterica* continues.

It is apparent from the results of this study that many laboratories still face significant challenges when detecting ß-lactamases, as only three (3/25) of the isolates in which ß-lactamase activity was detected in this study submitted were reported as exhibiting ESßL and or AmpC ß-lactamase activity. Whilst the CLSI guidelines have been evaluated as applying quite well to *S. enterica*, the low incidence of resistance observed at the time of validation concluded that the inclusion of *S. enterica* was not warranted and so the guidelines specifically cover only *K. pneumoniae, K. oxytoca, E. coli* and *P. mirabilis*.[10] The evidence presented in this study would support the suitability of this detection methodology for *S. enterica* and warrant its inclusion in the guidelines. The need to improve the awareness of ß-lactamase activity in *S. enterica* worldwide has been recognised by the World Health Organization Global Foodborne Infections Network. It now incorporates guidelines for ß-lactamase confirmation requirements as part of their Salm-Surv External Quality Assurance System annual proficiency test.

In conclusion, whilst surveillance in itself does not control resistance, accurate surveillance data informs the clinical use of antimicrobials and provides a powerful tool to prevent the spread of resistance. The results of this study provide the first surveillance data of ß-lactamase activity in *S. enterica* in Australia. This will provide some baseline surveillance data against which future trends can be measured, and against which to gauge the dissemination of ß-lactamases in *S. enterica* within Australia in the coming years.

Acknowledgements

I acknowledge the assistance provided by the Microbiological Diagnostic Unit and National Enteric Pathogens Surveillance Scheme in Melbourne.

Author details

Gino Micalizzi, *Salmonella* Reference Laboratory, Public Health Microbiology, Forensic and Scientific Services, Deaprtment of Health, Coopers Plains, Queensland, P.O. Box 594, Archerfield. QLD. 4108. Telephone: +61 7 3274 9126. Facsimile: +61 7 3274 9175. Email: Gino_Micalizzi@health.qld.gov.au

References

1. Davos D. A Compendium of Laboratory Diagnostic Methods for Common and Unusual Enteric Pathogens—an Australian persective. Chapter 10, *Salmonella*. 2005:119–135.
2. Powling J. National Enteric Pathogens Surveillance Scheme: Human Annual Report 2009.
3. Hendriksen RS, Vieira AR, Karlsmose S, Lo Fo Wong DM, Jensen AB, Wegener HC, et al. Global monitoring of *Salmonella* serovar distribution from the World Health Organization Global Foodborne Infections Network Country Data Bank: results of quality assured laboratories from 2001 to 2007. *Foodborne Pathog Dis* 2011;8(8):887–900.
4. Bright A. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet Network, 2009. *Commun Dis Intell* 2010;34(No. 4):375–483.
5. Su L-H, Chiu C-H, Chu C, Ou JT. Antimicrobial resistance in nontyphoid *Salmonella* serotypes: A global challenge. *Clin Infect Dis* 2004;39(4):546–551.
6. Bush K, Jacoby GA. Updated functional classification of ß-lactamases. *Antimicrob Agents Chemother* 2010;54(3):969–976.
7. Bush K. New ß-lactamases in gram-negative bacteria: diversity and impact on the selection of antimicrobial therapy. *Clin Infect Dis* 2001;32(7):1085–1089.
8. Bradford PA. Extended-spectrum ß-lactamases in the 21st Century: characterization, epidemiology, and detection of this important resistance threat. *Clin Microbiol Rev* 2001;14(4):933–951.
9. Thomson KS. Controversies about extended-spectrum and AmpC beta-lactamases. *Emerg Infect Dis* 2001;7(2):333–336.
10. Paterson DL, Bonomo RA. Extended-spectrum ß-lactamases: a clinical update. *Clin Microbiol Rev* 2005;18(4):657–686.
11. Helms M, Vastrup P, Gerner-Smidt P, Molbak K. Excess mortality associated with antimicrobial drug-resistant *Salmonella* Typhimurium. *Emerg Infect Dis* 2002;8(5):490–495.
12. Hammami A, Arlet G, Ben Redjeb S, Grimont F, Ben Hassen A, Rekik A, et al. Nosocomial outbreak of acute gastroenteritis in a neonatal intensive care unit in Tunisia caused by multiply drug resistant *Salmonella* wien producing SHV-2 beta-lactamase. *Eur J Clin Microbiol Infect Dis* 1991;10(8):641–646.
13. Batchelor M, Hopkins K, Threlfall EJ, Clifton-Hadley FA, Stallwood AD, Davies RH, et al. blaCTX-M genes in clinical *Salmonella* isolates recovered from humans in England and Wales from 1992 to 2003. *Antimicrob Agents Chemother* 2005;49(4):1319–1322.
14. Coque TM, Baquero F, Canton R. Increasing prevalence of ESBL-producing Enterobacteriaceae in Europe. *Euro Surveill* 2008;13(47).
15. Huang KY, Hong YW, Wang MH, Chu C, Su LH, Chiou CS, et al. Molecular epidemiology and antimicrobial susceptibility of *Salmonella enterica* serotype Stanley isolates in Taiwan. *J Microbiol Immunol Infect* 2007;40(5):411–418.
16. Kruger T, Szabo D, Keddy KH, Deeley K, Marsh JW, Hujer AM, et al. Infections with Nontyphoidal *Salmonella* species producing TEM-63 or a novel TEM enzyme, TEM-131, in South Africa. *Antimicrob Agents Chemother* 2004;48(11):4263–4270.
17. Tassios PT, Gazouli M, Tzelepi E, Milch H, Kozlova N, Sidorenko S, et al. Spread of a *Salmonella* Typhimurium clone resistant to expanded-spectrum cephalosporins in three European Countries. *J Clin Microbiol* 1999;37(11):3774–3777.
18. Whichard JM, Gay K, Stevenson JE, Joyce KJ, Cooper KL, Omondi M, et al. Human *Salmonella* and concurrent decreased susceptibility to quinolones and extended-spectrum cephalosporins. *Emerg Infect Dis* 2007;13(11):1681–1688.
19. Clinical and Laboratory Standards Institute. Performance Standard for Antimicrobial Susceptibility Testing; Nineteenth Informational Supplement. CLSI document M100-S21. Clinical and Laboratory Standards Institute. P.A.Wayne. 2009.
20. Clinical and Laboratory Standards Institute. Performance Standard for Antimicrobial Disc Susceptibility Tests; Approved Standards – Tenth Edition, CLSI document M02-10. Clinical and Laboratory Standards Institute. P.A.Wayne. 2009.
21. Livermore DM, Brown DFJ. Detection of ß-lactamase-mediated resistance. *J Antimicrob Chemother* 2001;48(Suppl 1):59–64.
22. Hanson ND, Moland ES, Hossain A, Neville SA, Gosbell IB, Thomson KS. Unusual *Salmonella enterica* serotype Typhimurium isolate producing CMY-7, SHV-9 and OXA-30 beta-lactamases. *J Antimicrob Chemother* 2002;49(6):1011–1014.

Short reports

Paralytic Shellfish Poisoning in South Eastern Tasmania

Alison Turnbull, Rosalind Harrison, Scott McKeown

Keywords: Public health, environmental health, toxicology, paralytic shellfish poisoning, biotoxins

Introduction

Paralytic shellfish poisoning (PSP) is a public health risk worldwide, resulting from the consumption of seafood that has bioaccumulated toxins produced by microalgae.[1] Bivalve shellfish (such as mussels, clams, oysters and scallops) pose a particularly high risk as they feed by filtration, providing a method of toxin accumulation. Algae responsible for causing PSP include several species of the genus *Alexandrium, Pyrodinium bahamense,* and *Gymnodinium catenatum*. Paralytic shellfish toxins (PST) have been recorded in shellfish from Victoria, South Australia, New South Wales and Tasmania (data from state shellfish regulatory programs).

G. catenatum was introduced into Tasmanian waters around 1973.[2] Recurrent blooms form in the Huon River, D'Entrecasteaux Channel, and the Derwent River in autumn and occasionally spring, particularly after calm weather and when water temperatures are above 14°C. PST are commonly referred to as saxitoxins.[3] Saxitoxins bind with high affinity to sodium channels in mammals, blocking nerve and muscle cell membranes. Thus cell action potentials are slowed or suppressed.[1] The binding is reversible.

Diagnosis of PSP is generally based on the onset of neurological signs and symptoms after recent shellfish consumption. Symptoms begin anywhere from 15 minutes to 12 hours after consuming contaminated shellfish, although usually within 2 hours.[1,4] Symptoms begin with tingling and numbness around the mouth and face, progressing to the extremities. This is followed by dizziness, nausea, headache, vomiting, vertigo, a floating sensation, weakness, and muscular incoordination. In severe cases paralysis, difficulty in breathing leading to respiratory failure, and even death can occur.[1,4]

Clinical record

A male fish farm worker developed paraesthesia in his hands, feet, face and tongue approximately 20 minutes after ingesting 12 fresh cooked wild mussels, *Mytilus galloprovincialis,* on 6 April 2011. The mussels were collected from the side of commercial fish cages in Port Esperance, south eastern Tasmania. Informed consent has been obtained to publish this case data.

The patient presented to a regional health centre and was seen by a general practitioner within 3 hours of ingesting the suspect mussels. He was experiencing additional symptoms of clumsiness, limb muscle weakness and vertigo. He had not experienced any difficulties breathing and had an oxygen saturation of 97% on room air as measured by transcutaneous pulse oximetry. His speech was slurred. Haemodynamic observations and physical examination were otherwise normal. The patient was transferred to the Royal Hobart Hospital with a presumptive clinical diagnosis of PSP.

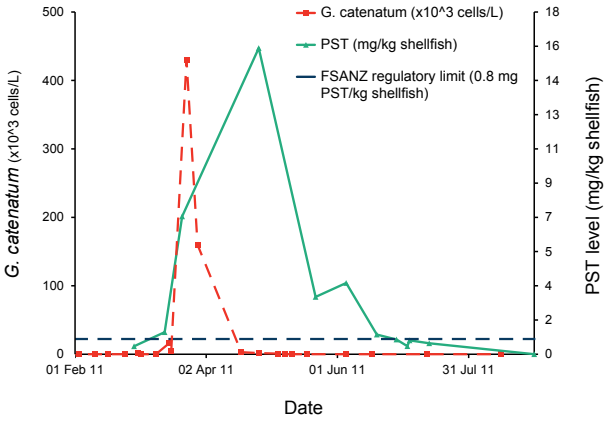
The patient was assessed in the Emergency Department 7½ hours after exposure. He was experiencing an additional symptom of a floating sensation in his limbs. There had been no further progression of his other symptoms. His haemodynamic observations remained normal. He had a peak expiratory flow rate of 210 L/min and his arterial oxygen saturation was 96% on room air. His examination findings included reduced lung air entry and wheeze bilaterally, diplopia with gaze to his left, mildly reduced power bilaterally in flexion of his elbows and fingers, and signs of cerebellar dysfunction (bilateral hypermetria and dysdiadochokinesis). Haematology and biochemistry testing showed no significant abnormality. His chest x-ray was reported as normal. A diagnosis was made of probable PSP but unfortunately no urinary sample was taken for saxitoxin testing. He was observed in the Emergency Department for 8 hours during which his symptoms improved. He was admitted to the medical ward for continued observation overnight and was discharged 31 hours after ingestion.

Discussion

At the time the patient presented, a toxic bloom of *G. catenatum* was present in the Huon Estuary, D'Entrecasteaux Channel and Port Esperance (Figure 1), resulting in high levels of PST in shellfish

Figure 1: Location of the *G. catenatum* bloom in south eastern Tasmania, March to April 2011

Figure 2: Cell counts of *G. catenatum* and corresponding levels of PST in Port Esperance, 1 February to 30 August 2011



from these areas. A public health warning against eating the shellfish due to the presence of PST had been issued on 25 March 2011.

G. catenatum was detected in Port Esperance in early March 2011 (Figure 2). Cell counts rapidly rose above 5 x 10^3 cells per litre (the harvest closure trigger) to 430 x 10^3 cells per litre on 29 March 2011, a week prior to the case presenting. Toxicity in the shellfish (analysed by mouse bioassay) followed the cell counts (Figure 2), exceeding the Australian maximum level[5] of 0.8 milligrams (mg) PST per kilogram (kg) of shellfish meat, by up to 20 times.

To confirm the diagnosis, the US Centers for Disease Control and Prevention recommend laboratory analysis for saxitoxins in either the urine or the seafood.[6] Individual sensitivity to PST varies considerably: the lowest reported doses causing mild symptoms of PSP are between 120 and 304 micrograms (μg) PST per person; and the lowest reported doses associated with severe intoxications or fatalities are between 456 and 1060 μg PST per person.[1,7] In the case presented in this paper, it is probable that the PST intake was between 1218 and 2688 μg (the 12 mussels consumed are estimated to be 168 grams of meat and PST concentrations around the time of illness ranged 7.25 to 16 mg/kg). The patient's symptoms were consistent with moderately severe poisoning.[3,8] Although confirmatory testing was not performed, toxin levels in the nearby commercial shellfish farm, microalgae in the water and a clinically compatible history support the diagnosis of a probable case of PSP.[6]

No previous PSP cases in Tasmania have been clinically confirmed. Only a few anecdotal cases were reported following the consumption of mussels during extensive blooms of *G. catenatum* in 1986 and 1993,[9,10] despite maximum levels of 340 mg PST per kg being detected in shellfish (over 400 times the Australian maximum level). It is possible that the local toxin profile in Tasmania is contributing to the lack of recorded illness. *G. catenatum* in Tasmania contains a high proportion of C-toxins, a group of analogues with considerably lower toxicity than saxitoxin.[11]

Whilst commercial fisheries are covered by shellfish quality assurance programs in each Australian state, recreational harvesting is an unmanaged public health risk. Campaigns to increase public health awareness during bloom periods need to be well considered and targeted. This case highlights that public health departments around Australia should be aware of the risks from recreational harvesting. Clinicians should be encouraged to perform urinary saxitoxin testing when patients present to the emergency department with clinical symptoms suggestive of PSP.

Author details

1. Ms Alison R Turnbull, Manager, Tasmanian Shellfish Quality Assurance Program, Public and Environmental Health Service, Department of Health and Human Services, GPO Box 125, Hobart, Tasmania 7001, Ph (03) 6222 7717 Fax (03) 6222 7692 Email alison.turnbull@dhhs.tas.gov.au
2. Ms Rosalind A Harrison, Senior Scientific Officer (Toxicology), Public and Environmental Health Service, Department of Health and Human Services

3. Dr Scott G McKeown, Public Health Medicine Trainee, Public and Environmental Health Service, Department of Health and Human Services

References

1. Food and Agriculture Organization of the United Nations. *Marine Biotoxins. Food and Nutrition Paper 80*; 2004.
2. Hallegraeff GM, McCausland MA, Brown RK. Early warning of toxic dinoflagellate blooms of *Gymnodinium catenatum* in southern Tasmanian waters. *J Plankton Res.* 1995;17:1163-1176.
3. Scientific Opinion of the Panel on Contaminants in the Food Chain. Marine Biotoxins in Shellfish – Saxitoxin Group. *The EFSA J.* 2009;1019:1-76.
4. Department of Health and Human Services. Centers for Disease Control and Prevention. USA: Marine Toxins, 12 October 2005. Available from: http://www.cdc.gov/ncidod/dbmd/diseaseinfo/marinetoxins_g.htm
5. Australia New Zealand Food Standards Code. Available from: http://www.foodstandards.gov.au/foodstandards/foodstandardscode.cfm. Accessed 3 February 2012.
6. Department of Health and Human Services, Centers for Disease Control and Prevention. USA: Case Definition: Saxitoxin. Reviewed 22 February 2006. Updated 11 March 2005. Department of Health and Human Services. Available from: http://emergency.cdc.gov/agent/saxitoxin/casedef.asp
7. Australia New Zealand Food Authority. Shellfish toxins in food. A toxicological review and risk assessment. Technical Report Series no.14;2001.
8. FAO/IOC/WHO. Report of the Joint FAO/IOC/WHO *ad hoc* expert consultation on Biotoxins in Bivalve Molluscs. Food and Agriculture Organization of the United Nations/Intergovernmental Oceanographic Commission of UNESCO/World Health Organization, Oslo 2004;31.
9. Department of Health and Human Services, Public and Environmental Health Service. Biotoxin Management Plan for the Tasmanian Shellfish Quality Assurance Program, Tasmania 2012.
10. Hallegraeff GM, Stanley SO, Bolch CJ, Blackburn SI. *Gymnodinium catenatum* blooms and shellfish toxicity in southern Tasmania, Australia. In: Nemoto T, Anderson DM, Okaichi T, editors. Red Tides: biology, environmental science, and toxicology. Elsevier Science Publishing Co. Inc 1989;77-80.
11. Oshima Y, Blackburn SI, Hallegraeff GM. Comparative study on paralytic shellfish toxin profiles of the dinoflagellate *Gymnodinium catenatum* from three different countries. *Mar Biol* 1993;116:471-476.

Increasing notifications of dengue in Australia related to overseas travel, 1991 to 2012

Katrina Knope, the National Arbovirus and Malaria Advisory Committee, Carolein Giele

Abstract

Dengue is an important cause of illness in travellers returning to Australia. The risk of local transmission from imported cases is of particular concern, with several large and explosive outbreaks recorded in recent years in north Queensland in areas where the mosquito vector of dengue is present. The number and proportion of dengue cases that are overseas-acquired is increasing. The number of overseas cases in 2010 and 2011 had increased by 298% and 155% respectively compared with the 5 year mean. The number of overseas acquired cases in 2012 is likely to be the largest on record, with an average of 144 cases per month during the first 7 months of the year. More than half of all dengue cases with a known country of acquisition between 1999 and July 2012 were acquired in Indonesia. In Western Australia in 2010 and 2011, more than 80% of cases acquired in Indonesia were acquired in Bali and the trend has continued into 2012.[1] While the frequency of travel by Australians to Indonesia has steadily increased since 2000, this does not completely explain the increased number of dengue cases in returning travellers. The relative risk of dengue in travellers returning from Indonesia between 2000 and 2011 compared with all other destinations was 8.3 (95% confidence interval 7.9–8.9). *Commun Dis Intell* 2013;37(1):55-59.

Introduction

Dengue is an important cause of illness in travellers returning to Australia. The number and proportion of cases that are known to have been acquired overseas has been increasing in recent years from 156 cases in the 2005–06 season, to 581 cases in the 2009–10 season.[2]

The risk of local transmission from imported cases is of particular concern, with several large and explosive outbreaks recorded in recent years (Figure 1). At present, the risk of local transmission is restricted to urban areas of Queensland where *Aedes aegypti*, a mosquito vector for dengue virus, is well established. There is a risk that dengue could become endemic there. Infected travellers returning to north Queensland and those transiting through the region to other states and territories can present the risk of starting local outbreaks.

Dengue was considered endemic in north Queensland up to the end of World War II, and the last outbreaks outside north Queensland (Brisbane and northern New South Wales) also occurred during that period.[3] There is the potential for *Ae. aegypti* incursions into the northern areas of Western Australia and the Northern Territory. The historical range of this mosquito species and of dengue transmission suggests a larger area of potential transmission risk on both the east and west coasts.[4] Recently, *Aedes albopictus* has become established in the Torres Strait and is a known vector of dengue virus.[5] An incursion to the mainland would have the potential to greatly extend the range of dengue in Australia.

Imported cases of dengue (and therefore local outbreaks) comprises all four serotypes. Infection with one serotype probably confers lifelong immunity. Antibodies cause an enhanced immune response to infection with a second serotype, increasing the risk of dengue haemorrhagic fever and dengue shock syndrome.[6]

Data sources and methods

In Australia, dengue is diagnosed and notified according to nationally agreed surveillance and laboratory case definitions.[7,8] Case definitions can change over time, and this should be considered in the interpretation of dengue data. Notifications are compiled nationally in the National Notifiable Diseases Surveillance System (NNDSS). The National Arbovirus and Malaria Advisory Committee (NAMAC) publishes annual reports on arboviral diseases.[9]

Information on short term resident departures collected by Australian Customs and Border Protection, and published by the Australia Bureau of Statistics was used to estimate the number of Australians travelling to destinations that are frequently reported as the place of acquisition for dengue cases.[10] Departure statistics relate to the number of movements of travellers rather than the number of travellers (i.e. multiple movements of individual persons during a given reference period are each counted separately) and are based on a combination of enumeration and estimation from samples. This dataset may underestimate the

denominator for overseas acquired dengue cases because it does not include visitors or migrants coming to Australia.

Numbers and rates of infection were calculated using Microsoft Excel, and incidence rate ratio analyses using the country of acquisition as a risk factor were conducted using Stata SE version 10.0.

Overseas acquired cases 1991–1999

Imported cases of dengue are estimated to account for 23% of all notifications between 1991 and 1999. Place of acquisition was not routinely recorded in NNDSS data prior to 1999. However, observed peaks in notifications (Figure 1) can be explained by reported outbreaks of locally-acquired infection in north Queensland rather than increases in the number of imported cases. Significant local outbreaks that occurred during this period were an outbreak of dengue virus serotype 2 (DENV2) in 1992–93 in Townsville and Charters Towers, with 900 cases;[11–13] outbreaks of DENV2 in 1996–97 in the Torres Strait and Cairns with 208 cases; and an outbreak of DENV3 in 1997–99 in Cairns,[11,14] Mossman and Port Douglas with 498 cases.[11,15] These large outbreaks and other smaller outbreaks during the period account for 77% (1,649/2,139) of reported cases.[11]

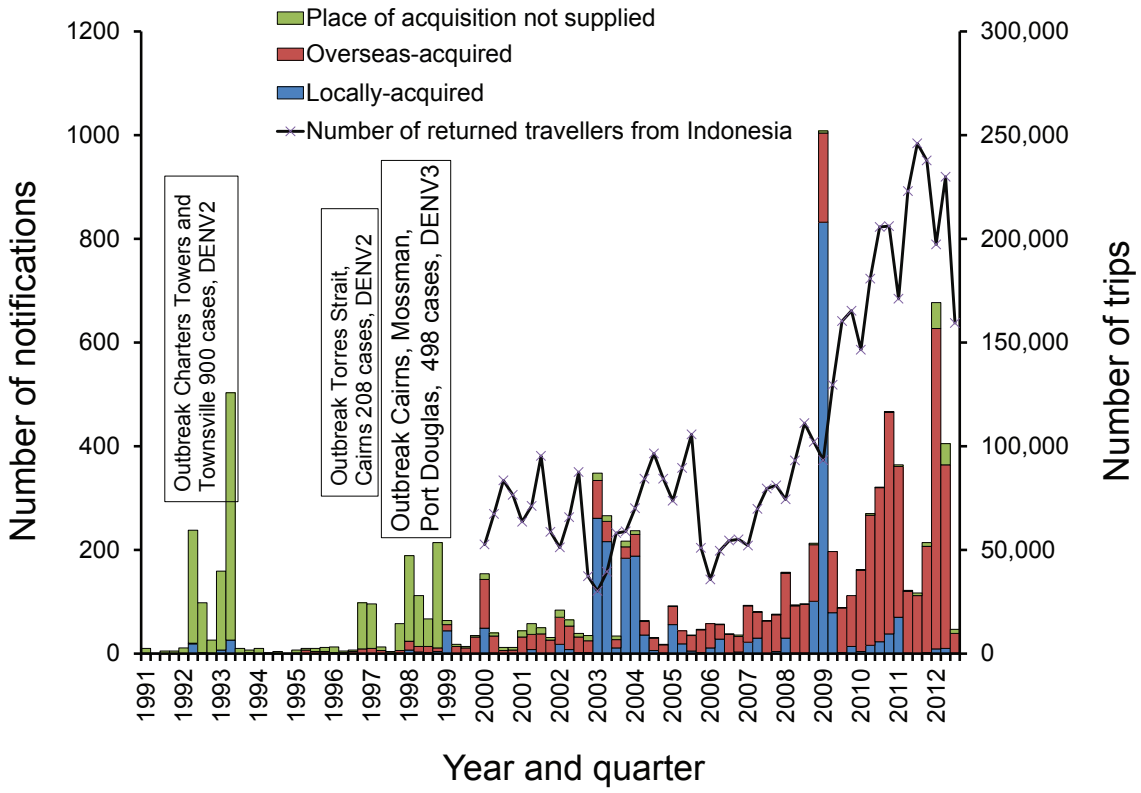
Overseas acquired cases 2000–2012

More than half of all dengue cases in Australia since 2000 (64%, 4,965/7,693) were related to overseas travel (Figure 1). The number and proportion of cases that are overseas-acquired is increasing. In 2010, there were 1,132 overseas-acquired cases (comprising 93% of all dengue notifications in 2010), and 727 cases in 2011 (comprising 89% of all dengue notifications in 2011) (Table 1). This compares with a mean of 284.6 cases per year between 2005 and 2009, when overseas-acquired cases comprised only 46% of all dengue notifications. The number of overseas acquired cases in 2012 is likely to be the largest on record, with an average of 144 cases per month during the first 7 months of the year.

The largest number of overseas-acquired dengue cases in 2010–2011 was reported by Western Australia (820 cases comprising 44% of overseas-acquired cases in 2010–11). The largest increases in the number of overseas-acquired cases were in Victoria and Western Australia, with ratios of 7.8 each for 2010 cases compared with the 5 year mean and 6.8 and 5.0 respectively for 2011 compared with the 5 year mean (Table).

Overseas-acquired dengue infections were of all four serotypes. Between 2005 and July 2012, serotypes 1 and 2 were the most commonly reported.

Figure 1: Dengue in Australia,* January 1991 to July 2012, by place of acquisition and number of returned travellers from Indonesia



* Outbreaks of dengue in North Queensland with >100 cases prior to the recording of place of acquisition in NNDSS are shown

Rates and countries of acquisition between 1999 and July 2012

Indonesia was the most frequently reported country of acquisition among overseas acquired cases of dengue in Australia between 1999 and July 2012, with overseas travel there accounting for 52% (2,306/4,403) of cases with a known country of acquisition during those years. Most cases related to travel in Indonesia occurred between 2010 and July 2012 (1,834 cases). More than half of all cases related to travel to Indonesia were from Western Australia (56%, 1,295/2,306), and in 2010 and 2011, more than 80% of these were among travellers returning from Bali (Carolein Giele, personal communication, place of acquisition for overseas acquired cases of dengue in Western Australia, 2012). This trend has continued into 2012, with 83% of cases between January and September 2012 in Western Australia associated with Indonesia, and of these, 96% reported travel to Bali.[1]

Nationally, other frequently reported places of acquisition were Thailand and East Timor, together accounting for 18% of overseas-acquired cases during the period. Complete information on country of acquisition was supplied for 87% (4,403/5,034) of overseas acquired cases.

The rates of Australians acquiring dengue overseas has varied from year to year, ranging from 2.5 per 100,000 trips in 2004 to a high of 15.9 per 100,000 in 2010 (Figure 2). Rates of infection among returned travellers from key destinations varied between 2000 and 2011, with a peak in rates among returned travellers from Papua New Guinea in 2002–2003, and a dramatic increase in rates among returned travellers from Indonesia peaking in 2010. Whilst rates of dengue among returned travellers from East Timor have exceeded that for every country in every year except Indonesia in 2010 (Figure 2, inset), rates have decreased in recent years (between 6 and 36 cases per year between 2007 and 2011, compared with 100 cases in 2000).

Risk of acquiring dengue in Indonesia

The popularity of Indonesia as a destination for Australians continues to rise. In 2011, there were 9 times as many travellers to Indonesia as in 2000 (Figure 1), while overall overseas travel was not quite double that in 2000.[10] Cases of dengue acquired in Indonesia are most frequently diagnosed between December and April, whilst the peak season of travel to Indonesia is between June and October 2012.

While the frequency of travel by Australians to Indonesia has steadily increased since 2000, this does not completely explain the increased number of dengue cases in returning travellers. The relative risk (RR) of dengue in travellers returning from Indonesia compared with all other destinations was 8.3 (95% confidence interval (CI) 7.9 to 8.9) while for travellers returning from Thailand, the RR was 1.8 (95% CI 1.76 to 2.0). The risk of a traveller acquiring dengue in Indonesia varies from year to year and appears to have increased overall between 2000 and 2011, peaking in 2010.

The incidence of dengue in Indonesia increased from 24.4 cases per 100,000 in 2003 to 66 cases per 100,000 in 2009.[16] Between 2005 and 2009, Bali had the fourth highest incidence of the Indonesian provinces, and ranged between 109 and 193 cases per 100,000.[17]

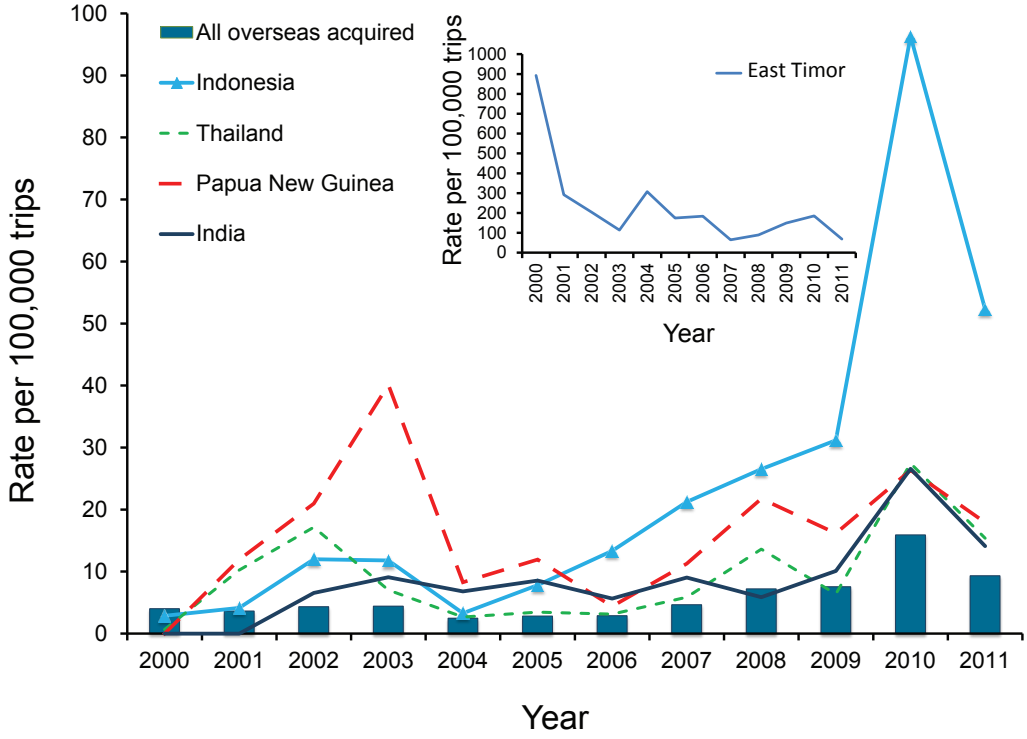
Age and sex of overseas acquired cases 2005 to July 2012

Overseas-acquired infections were most commonly reported among young and middle-aged adults

Table: Number of overseas-acquired cases of dengue, Australia, 2010 and 2011 compared with the five year average, by state or territory of residence

State or territory	Percentage of cases 2005–2011 with information on place of acquisition	Number of overseas-acquired cases				Ratio 2010/5 year average	Ratio 2011/5 year average
		2010	2011	Jan to Jul 2012	5 year average (2005 to 2009)		
ACT	100.0%	15	15	0	7	2.1	2.1
NSW	99.6%	224	135	156	89.2	2.5	1.5
NT	99.4%	40	25	63	20.8	1.9	1.2
Qld	99.1%	202	118	159	69.6	2.9	1.7
SA	89.2%	24	10	0	16.6	1.4	0.6
Tas	100.0%	7	3	5	2.2	3.2	1.4
Vic	96.6%	118	103	173	15.2	7.8	6.8
WA	99.9%	502	318	454	64	7.8	5.0
Total	99.0%	1,132	727	1,010	284.6	4.0	2.6

Figure 2: Rates of dengue virus infection in returning travellers by selected destinations of travel, and inset, rates of dengue virus infection in returning travellers from East Timor



reflecting the frequency of travel among these age groups. The median age of cases was 39, and 52% of cases were male.

Discussion

The increasing numbers of overseas-acquired cases of dengue in Australia are occurring in the context of endemic disease and periodic epidemics in many countries in South East Asia, including Indonesia, where incidence peaks in January and February and reported incidence doubled between 2003 and 2006.[18]

The source countries for overseas-acquired cases can be expected to change over time due to changing patterns of travel and local disease epidemiology in the source country. Papua New Guinea and East Timor have declined as important sources of dengue among travellers in recent years. In north Queensland in recent years, cases linked to Papua New Guinea predominated between 1999 and 2003 (51%) whilst between 2004 and 2008 Papua New Guinea was the source of only 12% of notifications.[19]

Improved diagnostic techniques, in particular the availability of the rapid NS1 antigen detection kit in recent years, have improved detection and would have contributed to the observed increase in reported numbers of overseas-acquired dengue in Australia.

The frequency of travel by Australians to Indonesia has increased sharply in recent years, particularly from Western Australia where the largest percentage increase in overseas-acquired cases is being reported. However, the increase in travel does not completely explain the increased number of cases of dengue in returning travellers from Indonesia.

Differences in the risk of acquiring dengue in Indonesia from year to year may be due to changes in the epidemiology of dengue in the country, particularly in Bali, or changes in traveller behaviour. Further research to determine the ecology and epidemiology of dengue in Bali, together with behaviour of Australian travellers, are urgently needed to improve our understanding of the changing risk of acquiring dengue in Indonesia.

Travellers to South East Asia need to take appropriate precautions to avoid being bitten by mosquitoes.[20] These include ensuring sleeping accommodation is free of mosquitoes by closing window screens, using insecticide sprays indoors or using bed nets, wearing light-coloured, long-sleeved clothing in urban or residential areas to minimise skin exposure to day-biting mosquitoes and using an appropriate mosquito repellent on exposed skin.

Acknowledgements

We are indebted to the private and public health laboratories and health departments who produce and collect the data on which this report is based.

Members of the National Arbovirus and Malaria Advisory Committee are (in alphabetical order): Bart

Currie, Peter Dagg, Peter Daniels, Stephen Doggett, Jenny Firman, Katrina Knope, Ann Koehler, Rogan Lee, Mike Lindsay, John Mackenzie, Scott Ritchie, Richard Russell, Christine Selvey, David Smith, Peter Whelan, Craig Williams. Jennifer Wall and Phil Wright (Secretariat).

Author details

Katrina Knope[1]
the National Arbovirus and Malaria Advisory Committee (see acknowledgement)
Carolein Giele[2]

1. Office of Health Protection, Department of Health and Ageing, Canberra, Australian Capital Territory
2. Department of Health, Western Australia, Subiaco, Western Australia

References

1. Department of Health Western Australia. Bali travel behind spike in dengue fever. *Disease WAtch* 2012;16.
2. Wright P, Fitzsimmons GJ, Johansen CA, Whelan PI, National Arbovirus and Malaria Advisory Committee. Arboviral diseases and malaria in Australia, 2009–10: annual report of the National Arbovirus and Malaria Advisory Committee. *Commun Dis Intell* 2012;36(1):70–81.
3. McBride WJH. Dengue fever: is it endemic in Australia? *Intern Med J* 2010;40(4):247–249.
4. Russell RC, Currie BJ, Lindsay MD, Mackenzie JS, Ritchie SA, Whelan PI. Dengue and climate change in Australia: predictions for the future should incorporate knowledge from the past. *Med J Aust* 2009;190(5):265–268.
5. Moore PR, Johnson PH, Smith GA, Ritchie SA, Van Den Hurk AF. Infection and dissemination of dengue virus type 2 in *Aedes aegypti, Aedes albopictus,* and *Aedes scutellaris* from the Torres Strait, Australia. *J Am Mosquito Control Assoc* 2007;23(4):383–388.
6. Morens DM. Antibody-dependent enhancement of infection and the pathogenesis of viral disease. *Clin Infect Dis* 1994;19(3):500–512.
7. Australian National Notifiable Diseases Surveillance Case Definitions. Accessed on 15 May 2012. Available from: http://www.health.gov.au/casedefinitions
8. Laboratory case definitions for diagnosis of communicable diseases. Accessed on 14 August 2012. Available from: http://www.health.gov.au/internet/main/publishing.nsf/content/Laboratory%20case%20definitions-1
9. National Arbovirus and Malaria Advisory Committee annual reports. Available from: http://www.health.gov.au/internet/main/publishing.nsf/content/cda-arboanrep.htm
10. Australian Bureau of Statistics. Overseas Arrivals and Departures, Australia, Table 9: Short-term Movement, Resident Departures – Selected Destinations: Original. Australian Bureau of Statistics; Canberra: 2012. ABS catalogue no.3401.0
11. Hanna JN, Ritchie SA. Outbreaks of dengue in north Queensland, 1990–2008. *Commun Dis Intell* 2009;33(1):32–33.
12. Streatfield R SD, Bielby G, Sheridan J, Pearce M, Phillips D. Dengue serotype 2 epidemic, Townsville,1992–93. *Commun Dis Intell* 1993;17:330–332.
13. McBride WJH MH, LaBrooy JT, Wronski I. The 1993 dengue 2 epidemic in Charters Towers, North Queensland: clinical features and public health impact. *Epidemiol Infect* 1998;121(1):151–156.
14. Hanna JN RS, Merritt AD, van den Hurk AF, Phillips DA, Serafin IL, et al. Two contiguous outbreaks of dengue type 2 in north Queensland. *Med J Aust* 1998;168(5):221–225.
15. Hanna JN RS, Phillips DA, Serafin IL, Hills SL, van den Hurk AF, et al. An epidemic of dengue 3 in Far North Queensland, 1997–1999. *Med J Aust* 2001;174(4):178–182.
16. World Health Organization. Dataset – Dengue in SEARO member countries 2010, 2011. Geneva; World Health Organization: 2012.
17. Demam berdarah dengue. *Bull Jendela Epidemiologi* 2010;3.
18. Situation update of dengue in the SEA Region, 2010. Available from: http://www.searo.who.int/LinkFiles/Dengue_Dengue_update_SEA_2010.pdf
19. Hanna JN RS. An apparent recent decline in importations of dengue from Papua New Guinea into north Queensland. *Commun Dis Intell* 2009;33(1):34–35.
20. Australian Government Department of Foreign Affairs and Trade. Smartraveller: The Australian Government's travel advisory and consular assistance service. Accessed on 24 December 2012. Available from: http://www.smartraveller.gov.au/

Surveillance summaries

Surveillance systems reported in *CDI*, 2013

This article describes the surveillance schemes that are routinely reported on in *Communicable Diseases Intelligence* (*CDI*).

Communicable disease surveillance in Australia operates at the national, state and local levels. Primary responsibility for public health action lies with the state and territory health departments. The role of communicable disease surveillance at a national level includes:

- detecting outbreaks and identifying national trends;
- providing guidance for policy development and resource allocation at the national level;
- monitoring the need for and impact of national disease control programs;
- coordinating a response to national or multi-jurisdictional outbreaks;
- describing the epidemiology of rare diseases that occur infrequently at state and territory levels;
- meeting various international reporting requirements, such as providing disease statistics to the World Health Organization; and
- supporting quarantine activities, which are the responsibility of the Australian government.

State and territory health departments collect notifications of communicable diseases under their public health legislation. In September 2007, the *National Health Security Act 2007* (*National Health Security Act , No 174*) received royal assent. This Act provides the legislative basis for and authorises the exchange of health information, including personal information, between jurisdictions and the Commonwealth. The Act provides for the establishment of the National Notifiable Diseases List (NNDL), which specifies the diseases about which personal information can be provided. The National Health Security Agreement, which was drafted in 2007 and signed by Health Ministers in April 2008, establishes the operational arrangements to formalise and enhance existing surveillance and reporting systems, an important objective of the Act. States and territories voluntarily forward de-identified data on a nationally agreed group of communicable diseases to the Department of Health and Ageing (DoHA) for the purposes of national communicable disease surveillance.

Surveillance has been defined by the World Health Organization as the 'continuing scrutiny of all aspects of the occurrence and spread of disease that are pertinent to effective control.' It is characterised by 'methods distinguished by their practicability, uniformity, and frequently by their rapidity, rather than complete accuracy.[1] Although some surveillance schemes aim for complete case ascertainment, others include only a proportion of all cases of the conditions under surveillance, and these samples are subject to systematic and other biases. Results generated from surveillance schemes must be interpreted with caution, particularly when comparing results between schemes, between different geographical areas or jurisdictions and over time. Surveillance data may also differ from data on communicable diseases gathered in other settings.

The major features of the surveillance schemes for which *CDI* publishes regular reports are described below.

Other surveillance schemes for which *CDI* publishes annual reports include tuberculosis notifications (*Commun Dis Intell* 2012;36(1):82–94), the Australian Mycobacterium Reference Laboratory Network (*Commun Dis Intell* 2011;35(2):154–161), invasive pneumococcal disease surveillance (*Commun Dis Intell* 2012;36(2):E151–E165), the National Arbovirus and Malaria Advisory Committee (*Commun Dis Intell* 2013;37(1):E1–E20), and the Australian Rotavirus Surveillance Program (*Commun Dis Intell* 2011;35(4):281–287).

Australian Childhood Immunisation Register

Accurate information on the immunisation status of children is needed at the community level for program management and targeted immunisation efforts. A population-based immunisation register can provide this need. The Australian Childhood Immunisation Register (ACIR) commenced operation on 1 January 1996 and is now an important component of the *Immunise Australia Program*. It is administered and operated by Medicare Australia. The Register was established by transferring data on all children under the age of 7 years enrolled with Medicare to the ACIR. This constitutes a nearly complete population register, as approximately 99% of children are registered with Medicare by 12 months of age. Children who are not enrolled in Medicare are added to the Register when a recog-

nised immunisation provider supplies details of an eligible immunisation. Immunisations are generally notified to Medicare Australia either by electronic means, the Internet or by paper ACIR notification forms. Immunisations recorded on the Register must have been given in accordance with the guidelines for immunisation determined by the National Health and Medical Research Council.

From the data finally entered onto the ACIR, Medicare Australia provides regular quarterly coverage reports at the national and state level. Coverage for these reports is calculated using the cohort method previously described (*Commun Dis Intell* 1998;22:36–37). With this method, a cohort of children is defined by date of birth in 3-month groups. This birth cohort has the immunisation status of its members assessed at the 3 key milestones of 12 months, 24 months and 5 years of age. Analysis of coverage is undertaken 3 months after the due date for completion of each milestone, so that time is available for processing notifications and the impact on coverage estimates of delayed notification to the ACIR is minimised. Only children enrolled with Medicare are included, in order to minimise inaccuracies in coverage estimates due to duplicate records.

Medicare Australia coverage reports for the 3 milestones are published in *CDI* each quarter. Coverage estimates are provided for each state and territory and Australia as a whole and for each individual vaccine assessed at each milestone. Changes in 'fully immunised' coverage from the previous quarter are also included in the tables.

A commentary on ACIR immunisation coverage estimates is included with the tables in each issue and graphs are used to provide trends in immunisation coverage.

An Immunisation Coverage Report is also published in *CDI* on an annual basis and provides more detailed data on immunisation coverage for all recommended vaccines by age group which are funded by the Immunise Australia Program, timeliness of immunisation, small area coverage estimates and data on conscientious objection to immunisation.

Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) is a continuing program to monitor antimicrobial resistance in *Neisseria gonorrhoeae* and includes the reference laboratories in all states and territories. These laboratories report data on sensitivity to an agreed core group of antimicrobial agents on a quarterly basis and provide an expanded analysis as an annual report in *CDI (Commun Dis Intell* 2012;36(2):E166–E173). The antibiotics that are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. A major purpose of the AGSP is to help define standard protocols for antibiotic treatment of gonococcal infection. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates, it is usual to reconsider the inclusion of that agent in current treatment schedules. Additional data are also provided on other antibiotics from time to time. At present, all laboratories also test isolates for the presence of high level resistance to the tetracyclines and intermittent surveys of azithromycin resistance are conducted. Comparability of data is achieved by means of a standardised system of minimal inhibitory concentration (MIC) testing and a program-specific quality assurance process.

Australian Meningococcal Surveillance Programme

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on laboratory-confirmed cases confirmed either by culture or by non-culture techniques. Culture-positive cases where *Neisseria meningitidis* is grown from a normally sterile site or skin, and non-culture based diagnoses, derived from the results of nucleic acid amplification assays and serological techniques are defined as invasive meningococcal disease (IMD) according to the Public Health Laboratory Network definitions.

Data are reported annually and quarterly in *CDI*. Data in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup where known. A full analysis of laboratory-confirmed cases of IMD, including phenotyping and antibiotic susceptibility data are published annually (*Commun Dis Intell* 2012;36(3):E251–E262).

Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit (APSU) is an active surveillance mechanism for prospective, national identification and study of children aged <15 years, newly diagnosed with uncommon conditions including rare infectious and vaccine preventable diseases, genetic disorders, child mental health problems, and rare injuries. Up to 16 different conditions are studied simultaneously. APSU relies on monthly reporting by ~1,400 paediatricians and other child health clinicians and over 80% of clinicians respond via e-mail. Clinicians reporting cases are asked to provide details about demographics, diagnosis, treatments and short-term outcomes. All negative and positive reports are logged into a database and the report card return rate has been maintained at over 90% over the last 20 years.

Communicable diseases currently under surveillance include: acute flaccid paralysis (to identify potential cases of poliovirus infection); congenital cytomegalovirus infection; congenital rubella; perinatal exposure to HIV, and HIV infection; neonatal herpes simplex virus infection; neonatal varicella, congenital varicella, severe complications of varicella and juvenile onset recurrent respiratory papillomatosis.

After demonstrating feasibility in 2007, APSU has conducted seasonal surveillance for severe complications of influenza each year. In 2009 APSU contributed to the national surveillance effort during the Influenza H1N1 09 pandemic.

The activities of the APSU are funded in part by the Australian Government Department of Health and Ageing, and NHMRC Practitioner Fellowship No: 1021480 (E Elliott). The Faculty of Medicine, The University of Sydney, and the Royal Australasian College of Physicians, Division of Paediatrics and Child Health, and the Kids Research Institute, Sydney Children's Hospitals Network provide in-kind support. APSU publishes an annual report *(Commun Dis Intell* 2012;36(3):E263–E276). For further information please contact the APSU Director, Professor Elizabeth Elliott on telephone: +61 2 9845 3005, facsimile +61 2 9845 3082 or email: apsu@chw.edu.au; Internet: http://www.apsu.org.au

Australian National Creutzfeldt-Jakob Disease Registry

Surveillance for CJD in Australia is conducted through the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR). CJD has been listed as a notifiable disease in all Australian states and territories. The ANCJDR is under contract to the Commonwealth to identify and investigate all suspect cases of transmissible spongiform encephalopathies (TSE) in Australia. An annual update is published in *CDI (Commun Dis Intell* 2012;36(2):E174–E179).

Australian Sentinel Practice Research Network

The Discipline of General Practice at the University of Adelaide operates the Australian Sentinel Practices Research Network (ASPREN). ASPREN is a national network of general practitioners who report presentations of defined medical conditions each week. The main aims of ASPREN are to provide an indicator of disease burden and distribution in the community and to be an early indicator of pandemic influenza.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2013, 4 conditions are being monitored; all of which are related to communicable diseases. These are influenza like illness (ILI), gastroenteritis, chickenpox and shingles.

Laboratory testing of ILI cases was implemented in 2010, allowing for viral testing of 25% of ILI patients for a range of respiratory viruses including influenza A, influenza B and H1N1(2009).

There are currently 210 general practitioners registered with the network from all jurisdictions. Fifty eight per cent of these are in metropolitan areas, 32% in rural and 10% in remote areas of Australia. Approximately 15,000 consultations are recorded by these GPs each week.

Data for communicable diseases are published in *CDI* each quarter. Data are presented in graphical format with the rate reported as the number of conditions per 1,000 consultations per week. The conditions are defined as:

Influenza-like illness – record once only per patient

Must have the following: fever, cough and fatigue.

Gastroenteritis – record once only per patient

Three or more loose stools, and/or 2 vomits in a 24 hour period excluding cases who have a known cause, for example bowel disease, alcohol, pregnancy.

Chickenpox – record once only per patient

An acute, generalised viral disease with a sudden onset of slight fever, mild constitutional symptoms and a skin eruption which is maculopapular for a few hours, vesicular for three to 4 days and leaves a granular scab.

Shingles – record once only per patient

Recurrence, recrudescence or re-activation of chickenpox infection. Vesicles with any erythematous base restricted to skin areas supplied by sensory nerves of a single or associated group of dorsal root ganglia. Lesions may appear in crops in irregular fashion along nerve pathways, are usually unilateral, deeper seated and more closely aggregated than those of chickenpox.

Note: Those conditions which show 'record once only per patient' are to have each occurrence of the condition recorded on 1 occasion no matter how many patient contacts are made for this episode of illness. If the condition recurs at a later date it can be recorded/counted again.

HIV and AIDS surveillance

National surveillance for HIV and AIDS is coordinated by the Kirby Institute, in collaboration with state and territory health authorities, the Australian Government Department of Health and Ageing, the Australian Institute of Health and Welfare and other collaborating networks in surveillance for HIV, viral hepatitis and sexually transmissible infections.

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

Currently, 2 tables presenting the number of new diagnoses of HIV infection, AIDS and deaths following AIDS are published in each issue of *CDI*. The tabulations are based on data available 3 months after the end of the reporting period, to allow for reporting delay and to incorporate newly available information.

An annual surveillance report, *HIV, viral hepatitis and sexually transmissible infections in Australia Annual Surveillance Report* has been published by the Kirby Institute since 1997. The Annual Surveillance Report, available through http://www.kirby.unsw.edu.au, provides a comprehensive analysis and interpretation of surveillance data on HIV, viral hepatitis and sexually transmissible infections in Australia. The report *Bloodborne viral and sexually transmitted infections in Aboriginal and Torres Strait Islander people: Surveillance and Evaluation Report* has been published from 2007, as an accompanying document to the annual surveillance report. The Surveillance and Evaluation Report provides detailed analysis and interpretation of the occurrence of these infections in Aboriginal and Torres Strait Islander communities in Australia.

National Influenza Surveillance Scheme

Australian influenza activity and severity in the community are monitored using a number of indicators and surveillance schemes:

- Notifications of laboratory-confirmed influenza are reported from all Australian states and territories and included in the National Notifiable Diseases Surveillance System.
- Community level ILI is monitored through two sentinel systems, Flutracking, a weekly online survey integrating syndromic information with participant influenza immunity status; and data from the National Health Call Centre Network.
- Reports on general practice ILI consultations are provided through the Australian Sentinel Practice Research Network and the Victorian Sentinel General Practice Scheme. Additionally, data on ILI presentations to hospital emergency departments are collected from sentinel hospitals sites in Western Australia and New South Wales.
- Hospitalised cases of laboratory-confirmed influenza are reported through the Influenza Complications Alert Network (FluCAN); and severe complications in children are monitored by the APSU.
- Information on influenza subtypes and positivity are provided by sentinel laboratories, including the National Influenza Centre laboratories and some state public health laboratories. Additional virology and antiviral resistance data are also provided from the World Health Organization Collaborating Centre for Reference and Research on Influenza.
- During the influenza season, data from each of these surveillance systems are compiled and published fortnightly in the Australian Influenza Report, which is available generally from May to October on the department's web site. These reports include the above data as well as additional mortality and international surveillance data.

Annual reports on the National Influenza Surveillance Scheme are published in the *CDI* each year (*Commun Dis Intell* 2010;34(1):8–22).

National Notifiable Diseases Surveillance System

National compilations of notifiable diseases have been published intermittently in a number of publications since 1917.[2] The National Notifiable Diseases Surveillance System (NNDSS) was established in 1990 under the auspices of the Communicable Diseases Network Australia (CDNA).

Sixty-five communicable diseases agreed upon nationally are reported to NNDSS, although not all 65 are notifiable in each jurisdiction. Data are sent electronically from states and territories daily (business days only in some jurisdictions). The system is complemented by other surveillance systems, which provide information on various diseases, including four that are not reported to NNDSS (AIDS, HIV, and the classical and variant forms of Creutzfeldt-Jakob disease).

The NNDSS core dataset includes data fields for a unique record reference number; notifying state or territory, disease code, age, sex, Indigenous status, postcode of residence, date of onset of the disease, death, date of report to the state or territory health department and outbreak reference (to identify cases linked to an outbreak). Where relevant, information on the species, serogroups/subtypes and phage types of organisms isolated, and on the vaccination status of the case is collected. Data quality is monitored by DoHA and the National Surveillance Committee (NSC) and there is a continual process of improving the national consistency of communicable disease surveillance.

While not included in the core national dataset, enhanced surveillance information for some diseases (hepatitis B [newly acquired], hepatitis C [newly acquired], invasive pneumococcal disease and tuberculosis) is obtained from states and territories.

Aggregated data are presented on the department's internet site under *Communicable Diseases Surveillance* and updated daily (http://www.health.gov.au/nndssdata). A summary report and data table are also published on the internet each fortnight (http://www.health.gov.au/cdnareport).

Data are published in CDI each quarter and in an annual report. The reports include numbers of notifications for each disease by state and territory, and totals for Australia for the current period, the year to date, and for the corresponding period of the previous year. The national total for each disease is compared with the average number of notifications over the previous 5 years in the same period. A commentary on the notification data is included with the tables in each issue of CDI and graphs are used to illustrate important aspects of the data.

OzFoodNet: enhanced foodborne disease surveillance

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally in the investigation of foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease.

OzFoodNet reports quarterly on investigations of gastroenteritis outbreaks and clusters of disease potentially related to food. Annual reports have been produced and published in *CDI* (*Commun Dis Intell* 2012;36(3):E213–E241) since 2001. Data are reported from all Australian jurisdictions.

Sentinel Chicken Surveillance Programme

The Sentinel Chicken Surveillance Programme is used to provide an early warning of increased flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis virus (MVEV) and Kunjin virus. MVEV can cause the disease Murray Valley encephalitis (formerly known as Australian encephalitis), a potentially fatal disease in humans. Encephalitis occurs less frequently in cases of Kunjin virus infection and these encephalitis cases have a lower rate of sequelae.

These viruses are enzootic in parts of the north-east Kimberley region of Western Australia and the Top End of the Northern Territory but are epizootic in other areas of the Kimberley, Pilbara, Gascoyne Murchison and Mid-west regions of Western Australia, in north Queensland and in Central Australia. MVEV is also responsible for occasional epidemics of encephalitis in eastern Australia. Since 1974, a number of sentinel chicken flocks have been established in Australia to provide an early warning of increased MVEV activity. These programs are supported by individual state health departments. Each state has a contingency plan that will be implemented if one or more chickens in a flock seroconverts to MVEV. Further details are provided in the Annual Report of the National Arbovirus and Malaria Advisory Committee (*Commun Dis Intell* 2013;37(1):E1–E20).

References

1. Last JM. A dictionary of epidemiology. New York: Oxford University Press, 1988.
2. Hall R. Notifiable diseases surveillance, 1917 to 1991. *Commun Dis Intell* 1993;226–236. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-annlrpt-oz_dis19_91.htm Accessed March 2012.

Policy and guidelines

The BCG vaccine: information and recommendations for use in Australia

National Tuberculosis Advisory Committee Update October 2012

Summary points

The *BCG vaccine: information and recommendations for use in Australia* (March 2006) has been updated to incorporate the most recent trends in annual national tuberculosis (TB) surveillance data.

Australia continues to meet international epidemiological criteria that limit Bacille Calmette Guérin (BCG) vaccination to selected high risk groups in countries with a low incidence of TB.

No significant change has been made to existing recommendations for BCG vaccination in Australia.

Key recommendations

BCG vaccination is not recommended for general use in the Australian population or for most health care workers (HCWs).

BCG vaccination is contraindicated in HIV infected persons.

BCG vaccination is recommended for:

1. Aboriginal and Torres Strait Islander neonates in communities with a high incidence of TB;
2. Neonates and children 5 years of age and under who will be travelling to or living in countries or areas with a high prevalence of TB for extended periods;
3. Neonates born to parents with leprosy or a family history of leprosy.

BCG vaccination may be considered in the following:

1. Children over 5 years of age who will be travelling to or living in countries or areas with a high prevalence of TB for extended periods;
2. HCWs who may be at high risk of exposure to drug resistant TB.

State and territory TB control units should be consulted with regard to their BCG vaccination guidelines.

Executive summary

This report provides an update on the role of Bacille Calmette Guérin (BCG) vaccination in tuberculosis (TB) control and prevention in Australia. While no significant change has been made to current recommendations it was considered important to review the 2006 document in the context of the ongoing epidemiological situation and concerns about benefit versus risk.

Annual TB surveillance data for Australia in the past decade confirm ongoing low rates of disease in the general population. Most disease (80%–90%) is limited to people from high prevalence countries. In the Australian-born population, rates remain very low, particularly in children less than 5 years of age, who are an important marker of good TB control. While TB rates are higher in some Aboriginal and Torres Strait Islander communities than for non-Indigenous Australian-born people, actual case numbers are still small. Drug resistant TB is being carefully monitored based on international concerns, but the rate remains low with cases predominantly 'imported'. The impact on disease rates from HIV infection remains minimal.

The control of TB in all countries primarily relies on early detection and treatment of infectious cases to minimise transmission to the community. In Australia, this is supported by the secondary strategy of targeting those most at risk from latent TB infection for preventive therapy. BCG vaccination has a very limited role in the control and prevention of TB in low prevalence settings. Its use is limited to neonates and infants considered at high risk of exposure to TB for whom access to early detection and treatment of TB is potentially problematic. The benefit of vaccination in older age groups is less clear and it is no longer recommended as a routine measure in most health care workers (HCWs).

The National TB Advisory Committee recognises the different BCG vaccination policies that exist within and between countries and this reflects the controversial history of BCG vaccine in terms of its effectiveness. The advice provided aims to enhance uniformity between the states and territories based on the best available evidence and guiding principles for the use of BCG vaccine in low prevalence countries.

Introduction

Mass BCG vaccination in populations with a low prevalence of tuberculosis is no longer considered necessary.[1] Rather, such an intervention should be directed at well-defined, high-risk groups, principally because of its direct effect in reducing the serious consequences from primary infection. The indirect population effect of mass vaccination in terms of reducing the number of infectious cases, and hence limiting future transmission to the uninfected population, is considered to be minimal in low prevalence countries.[2]

In Australia, the broad-based BCG vaccination program originated at a time when the epidemiology of TB was quite different. Initially in 1948, vaccination targeted health care workers, Aboriginal people and close contacts of active cases, especially children. In the 1950s the program was expanded to include all Australian school children except those from New South Wales and the Australian Capital Territory. This policy was discontinued in the mid-1980s (1991 in the Northern Territory) in favour of a more selective approach. The change occurred because of the low prevalence of TB in our community and concerns about the balance between the benefits and the risks.

Prior to 1975, Sweden vaccinated all newborns and is one of the few countries to have closely studied the implications of this policy. TB notification data from 1975 to 2004 indicate that the observed incidence of TB in unvaccinated Swedish children from a low risk background remains low, and importantly, the risk of serious TB is still rare.[3,4]

Further, the similarities in TB disease trends between Australia and countries where universal BCG vaccination has never been practised (United States of America, Netherlands)[5] suggest that the incidence of TB in a community is determined by the combined effect of all TB control measures rather than BCG vaccination alone.

BCG vaccination does not prevent the transmission of TB to an individual. The direct effect of the vaccine (for which it was introduced) appears to be in limiting the spread of primary infection from an infected individual. Varying reports suggest levels of protection anywhere from 0% to 80%.[6–9] The differences possibly relate to the use of different BCG vaccine strains, methodological factors, the influence of environmental mycobacteria and age, and immune or genetic factors.[10]

Two meta-analyses have been helpful in summarising the variable findings from several studies on BCG vaccine efficacy.[11,12] The key conclusions were that it is about 50% effective in preventing TB disease and that the most important protective benefits are in minimising the risk of death, meningitis and disseminated disease in neonates and young children.

Although the use of BCG vaccine in health workers has declined considerably, there has been renewed interest related to multi-drug resistant (MDR) TB.[9] The benefit of BCG vaccination over tuberculin skin testing (TST) screening may be enhanced for the health worker in such a setting.[13–15] It offers some protection irrespective of antimicrobial susceptibility of the infecting organism, whereas the benefit of preventive therapy is unproven in those infected with an MDR strain.[16,17] This dilemma highlights the importance of appropriate infection control measures in health care settings.

Epidemiology

The annual incidence of TB in Australia has remained relatively stable since the mid-1980s. From 2005 to 2009 there was little change, with rates between 5.28 and 5.95 cases per 100,000 population.[18] These low rates have been maintained despite the increased level of immigration from high TB burden countries.

Overseas-born persons account for approximately 85% of notifications in Australia. Over the last decade overall incidence rates in this group have increased from 15.5 to 21.0 per 100,000. By contrast, the overall rate in Australian-born people is approximately 1 per 100,000. In 2008, the rate for Aboriginal and

Torres Strait Islander peoples was 5.9 per 100,000 with the highest rates detected in the Northern Territory (25 per 100,000) and Queensland (6.2 per 100,000). However, case numbers are low.[18]

Review of data from 2005 to 2009 shows that pulmonary cases represented an average of 57% of all cases. Sputum microscopy positive cases, which are the main source of transmission in a community, accounted for 22.8% (expected 40%–60%) of the total pulmonary cases.[18] Even if this is an underestimate due to notification factors, the rate is still very low at approximately 1 per 100,000.

Children under 5 years of age are a key indicator of the level of transmission of infection in the community. From 2005 to 2009 the proportion of cases in this age group was an average of 1.8% of the total compared with 1.97% in the preceding 5 years. The rate remains steady at about 1 per million. Of the serious forms of TB in this age group, for the same period there were only 5 cases of TB meningitis notified in Australia, equivalent to less than 1 per 20 million general population per year.[18]

These outcomes meet the International Union Against TB and Lung Disease (IUATLD) criteria for low prevalence countries, in determining BCG policy, which are:

- average annual notification rate of pulmonary sputum smear positive cases of 5 per 100,000 or less in the preceding 3 years; or
- average annual notification rate of TB meningitis in children under 5 years of less than 1 case per 10 million general population; or
- an average annual risk of TB infection of 0.1% or less.[1]

Risk groups

Significant debate continues on the role of BCG vaccination within certain groups that are classified as high risk. The Canadian and United States of America guidelines recommend that the use of BCG vaccine be limited to neonates and infants considered at high risk of exposure to TB, in whom access to early detection and treatment of TB infection is problematic, or where TB control efforts have had limited impact.[9,19] A recent Dutch study highlighted the high numbers of neonates in a low prevalence setting who would need to be vaccinated to prevent one case, and safety data in Canadian First Nations Children cautioned that the risk of BCG disseminated infection should be carefully considered.[19,20]

Aboriginal and Torres Strait Islander peoples

Aboriginal and Torres Strait Islander peoples from some communities are at greater risk of developing active TB than non-Indigenous Australian-born people. This likely reflects socioeconomic, nutritional and health factors.[21–23] Rates of TB in Aboriginal and Torres Strait Islander peoples remain higher than in non-Indigenous Australians but these rates have decreased and are not uniform. In 2007 there were only 35 notifications of TB in Ingidenous Australians, 60% of these being from the Northern Territory and 20% from Queensland.[18]

The recommendation that at-risk Aboriginal and Torres Strait Islander neonates be vaccinated with BCG shortly after birth is based on the premise that in high risk populations, infants and children have a greater potential for exposure to an active case of TB. Infection in this age group has a significantly higher risk for producing severe manifestations of TB, including meningitis, rapid dissemination and death. However with such low numbers, the safety of BCG vaccine is a key consideration. Data from vaccinated Canadian First Nations children suggest that they are at higher risk of disseminated BCG infection than children elsewhere, in turn suggesting that they may be less immunocompetent.[19] This risk needs to be carefully considered in the BCG vaccination of Aboriginal and Torres Strait Islander neonates from communities deemed to be at high risk of infection.

Migrants

The most important factor contributing to the epidemiology of TB in Australia has been the increased migration from countries with a high incidence of TB. Rates of TB in these people remain similar to those of their country of origin, particularly in the first 5 years after arrival.[24]

The overall rate of TB for non-Indigenous children born in Australia remains very low. While the rates are higher in overseas-born children the actual numbers reported are small.[18] Further, data from Australian prevalence surveys indicate that the rate of TB infection in children born in Australia to overseas-born parents is as low as that of children of Australian-born parents.[25–28]

Hence, BCG vaccination is not routinely recommended in neonates and infants of migrant parents but rather should be based on a careful assessment of the individual situation. For example, neonates of parents from high incidence countries staying for extended periods in their parents' country of origin should be considered for BCG vaccination. The optimal approach is to advise a TST after such travel if household exposure is suspected or known

and recommend preventive treatment as appropriate. If close contact with an infectious case did occur, current World Health Organization policy recommends the use of preventive therapy in those less than 5 years of age, even if vaccinated.[29]

Health care workers

HCWs in Australia are at low risk of being exposed to patients with active TB. As such, the use of BCG vaccination for HCWs is no longer recommended as the primary means of protection.

The role of BCG vaccination in HCWs is unclear and the uncertainty has led to divergent policies in the Australian states and territories and overseas. The main issues are the lack of evidence supporting a protective benefit from BCG in adults and the fact that it renders future interpretation of the post-exposure TST imprecise.[9]

For most HCWs, BCG vaccination is not indicated but should be considered in those who may be at high risk of exposure to drug resistant organisms, e.g. the HCW moving to an overseas country to work in an area with a known or suspected drug resistance problem.

The preferred strategy advocated to control TB in HCWs is TST to identify new infection and the use of preventive therapy.

The TST policy is theoretically sound but weakened by the reluctance of many HCWs to comply with the recommended measures. Further, with the emergence of multi-drug resistance, the benefit of preventive treatment for infected contacts is uncertain.[16,17] Although the number of cases reported to date in Australia is small, multi-drug resistant TB is nevertheless a major concern because of the lower cure rate, higher mortality and potential implications for exposed HCWs.

Irrespective of the HCW strategy, it is important to ensure that both the individual and the institution in which they are working are adequately informed about TB and that appropriate infection control measures are in place to minimise the risk of transmission.

Overseas travel

The number of cases of TB reported in Australians who have travelled or lived in high prevalence countries for significant periods is small.

Vaccination is not considered necessary for those undertaking brief holidays to well known tourist destinations. However, for neonates and children 5 years of age and under who will be staying for an extended period in countries where the incidence of TB is high, vaccination is recommended. Each individual's situation needs to be carefully assessed. The protective benefit of vaccination in older age groups is less certain.[9] BCG should be given 2 to 3 months prior to departure.

Other groups

Based on overseas experience, there are additional groups in our community that may be at increased risk of TB. These include the homeless, prison residents and injecting drug users. BCG vaccination is not recommended for these persons.

Although BCG vaccine is considered a TB vaccine, it may also be effective against leprosy and is still recommended in some countries for neonates of leprosy patients.[10,30] In Australia, occasional cases of leprosy are reported, mainly in migrants from leprosy endemic countries but also sporadically in Indigenous communities.[31]

Recommendations

BCG vaccination is not recommended for general use in the Australian population or for most HCWs.

BCG vaccination is contraindicated in HIV infected persons.

BCG is recommended for:

1. Aboriginal and Torres Strait Islander neonates in communities with a high incidence of TB;
2. Neonates and children 5 years of age and under who will be travelling to or living in countries or areas with a high prevalence of TB for extended periods; and
3. Neonates born to parents with leprosy or a family history of leprosy.

In addition to these recommendations, BCG may be considered for the following:

1. Children over 5 years of age who will be travelling to or living in countries or areas with a high prevalence of TB for extended periods; and
2. HCWs who may be at high risk of exposure to drug resistant cases.

State and territory TB control units should be consulted with regard to their BCG vaccination guidelines.

Important notes

All individuals should have a TST prior to BCG vaccination except infants less than 6 months of age with no history of TB contact.

BCG should not be given to an individual with a tuberculin reading of 5 mm or more.

BCG vaccine should not be administered unless consent has been obtained following a full explanation of the benefits and risks associated with vaccination.

BCG revaccination is not recommended, regardless of TST reaction (TST reaction size is not a correlate of BCG vaccine efficacy).

Contraindications

The use of BCG vaccine is contraindicated in the following:

- persons immuno-compromised by HIV infection, corticosteroids or other immuno-suppressive agents and malignancies involving bone marrow or lymphoid systems (because of the risk of disseminated BCG infection);
- individuals with any serious illness including the malnourished;
- individuals with generalised septic skin diseases and skin conditions such as eczema, dermatitis and psoriasis;
- pregnant women—BCG has not been shown to cause foetal damage but the use of a live vaccine in pregnancy is generally contraindicated; and
- individuals who have previously had tuberculosis or a large tuberculin (TST) reaction.

BCG vaccination should be deferred for the following:

- individuals with a significant febrile illness (administer 1 month from the time of recovery);
- neonates with a birth weight less than 2.5 kg or in those who may be relatively undernourished. It should not be offered to neonates of mothers who are HIV positive;
- individuals with a high risk of HIV infection until HIV infection is excluded; and
- a 4 week interval should be allowed following administration of another live parenteral (injectable) vaccine unless given concurrently e.g. measles-mumps-rubella, yellow fever, varicella. There are no restrictions on the timing of BCG vaccine in relation to oral live vaccines, e.g. rotavirus, oral typhoid vaccines.

NB: Care should be taken in those with a history of keloid scarring or an increased risk of developing it e.g. Aboriginal and Torres Strait Islander peoples, and Melanesians. The likelihood of this occurring can be minimised if the injection is given into the skin over the region of the deltoid muscle insertion.

It is recommended that a list of exclusion criteria be given to the patient to allow self-exclusion with complete anonymity regarding the specific risk factor.

Vaccination

BCG vaccine

- BCG vaccine* is a suspension of living organisms of an attenuated strain of *Mycobacterium bovis*. It is available as a freeze-dried powder for intradermal use in a 100-dose vial and should be stored at 2°C to 8°C with protection from light. Exposure to heat and light both before and after reconstitution may result in a loss of potency. The expiry date should be checked prior to administration.
- BCG vaccine is reconstituted using 1.0 ml of the Diluted Sauton SSI (solvent) supplied. It should be gently and thoroughly mixed then used strictly within a 4–6 hour period. Store at 2°C–8°C.
- As BCG vaccine does not contain a bacteriostatic agent, extreme care is required to avoid contamination. A new 26–27-gauge needle and 1 ml syringe should be used for each dose and the remaining vaccine discarded as per procedures recommended for biohazardous substances.
- Providing a strictly aseptic technique is adhered to in accordance with approved infection control guidelines, the use of a multi-dose vial is an accepted practice.

BCG vaccination procedure

The National Health and Medical Research Council recommends that administration of BCG vaccine be carried out by an accredited health care worker to limit the risk of adverse events.

The BCG dose is:

- adults and children over 12 months – 0.1 ml
- infants 12 months and under – 0.05 ml

Vaccination should be deferred in premature or small-for-dates babies less than 2.5 kg.

- A TST should be done prior to vaccination except in infants less than 6 months (exclude history of

* The manufacture of BCG vaccine in Australia has been discontinued. The Sanofi Pasteur BCG vaccine (Toronto, Ontario, Canada) has been approved for use in Australia by the Therapeutic Goods Administration, however, was recalled in June 2012. Sanofi Pasteur is supplying BCG vaccine SSI, manutactured by Statens Serum Institut in Denmark, as a substitute. Product information provided above relates to BCG vaccine SSI. The indication and dosage of the BCG vaccine. Supply of the Sanofi Pasteur BCG vaccine is expected to resume by early 2014.

TB contact). BCG can be administered to those with a reaction size less than 5 mm providing no contraindications exist.

- The site of injection into the skin is very important in order to minimise the risk of keloid formation. The position normally recommended is at the level of insertion of the deltoid muscle into the humerus. While it can be given into the middle third of the antero-lateral aspect of the thigh, many prefer not to for cosmetic reasons.
- The injection must be given strictly intradermally—needle bevel uppermost, until its opening is just visible through the epidermis.
- A blanched weal should be raised. If little resistance is felt, then this may mean that the needle is in the subcutaneous tissue and therefore should be withdrawn. The injection should then be given at an alternative site. Inadvertent subcutaneous injection is likely to cause an excessive reaction.

BCG vaccination reaction

Initially, a small red papule forms within a 2–3 week period followed by softening and ulceration. Healing usually occurs after several weeks with a resultant small scar. An accelerated reaction begins within 24–48 hours with induration followed by pustule formation in 5–7 days and healing within 10–15 days.

BCG vaccination aftercare

Information, both verbal and written, on what to expect and how to care for the resultant local reaction, should be provided to the vaccinee or carer. The importance of promptly reporting any suspected problems should be stressed.

Adverse effects

Serious complications from BCG vaccination including anaphylactoid reactions are rare.[32–34]

Adverse effects include:

- regional lymphadenitis – this is the most common adverse reaction;
- subcutaneous abscess;
- accelerated local reactions;
- osteitis;
- keloid scarring; and
- disseminated infection.

Correct assessment and technique is essential to minimise these risks.

Immuno-compromised individuals can develop disseminated infection from BCG, e.g. malnourished children and HIV positive persons.

Some adverse reactions may require anti-tuberculous treatment.

Adverse events following vaccination should be notified to the relevant state health authority.

BCG revaccination

In many developing countries, systematic revaccination has been accepted practice because of doubts about the persistence capacity of the vaccine when given in the early neonatal period.[35] However, such an approach is not supported by scientific evidence.

The effectiveness of repeat BCG to the individual remains in question.[36–38] Previously, the finding of a negative TST response was considered to indicate the need for revaccination. It was argued that revaccination may increase the rate of tuberculin conversion and result in more sustained reactivity over time. However, the tuberculin response is not a correlate of the protective benefit derived from BCG vaccination and there is no evidence that a waning of tuberculin sensitivity with time equates to a loss of TB specific immunity.[9,39]

Based on the information available, BCG revaccination is not recommended for any person.[39]

BCG vaccine alternative

BCG remains the only available vaccine for TB. However, it only offers partial and variable protection to the uninfected for a relatively short period.

Several new vaccine candidates (pre and post exposure) are under investigation. These include recombinant vaccines, sub-unit vaccines and DNA-based vaccines. Novel T-cell adjuvants are also being tested with experimental sub-unit vaccines.[38–42] The improved safety of the latter over live-attenuated vaccines offers a potential benefit to HIV-infected persons.

The relatively short-lived efficacy of BCG for only 10–20 years appears accepted.[43] A vaccine that both has the ability to boost immunity in those vaccinated in childhood to protect against the risk from primary infection, or if already infected, prevent reactivation of latent infection would be a substantial advance in the control of TB globally.

References

1. International Union Against Tuberculosis and Lung Disease. Criteria for discontinuation of vaccination programmes using Bacille Calmette-Guérin (BCG) in countries with a low prevalence of tuberculosis. *Tuber Lung Dis* 1994;75:179–181
2. Styblo K. Epidemiology of Tuberculosis. Selected Papers, 24. Royal Netherlands Tuberculosis Association, 1991.
3. Romanus V, Svensson A, Hallander HO. The impact of changing BCG coverage on tuberculosis incidence in Swedish born children between 1969 and 1989. *Tuber Lung Dis* 1992;73,150–161.
4. Romanus V. Surveillance report. Selective BCG vaccination in a country with low incidence of tuberculosis. *Euro Surveill* 2006;11(3).
5. Zwerling et al. The BCG World Atlas: A Database of Global BCG vaccination Policies and Practices. *PLoS Med* 2011;8(3): e1001012 doi: 10.1371/journal.pmed. 1001012
6. Clemens JD, Chuong JJ, Feinstein AR. The BCG controversy: a methodological and statistical reappraisal. *JAMA* 1983;249:2362–2369.
7. Smith PG. Case-control studies of the efficacy of BCG against tuberculosis. In: *International Union Against Tuberculosis*, ed. Proceedings of the XXVIth IUAT World Conference on Tuberculosis and Respiratory Diseases. Singapore: Professional Postgraduate Services International, 1987;73–79.
8. Tripathy SP. Fifteen-year follow-up of the Indian BCG prevention trial. In: *International Union Against Tuberculosis*, ed. Proceedings of the XXVIth IUAT World Conference on Tuberculosis and Respiratory Diseases. Singapore: Professional Postgraduate Services International, 1987;69–72.
9. The role of BCG vaccine in the prevention and control of tuberculosis in the United States. A joint statement by the Advisory Council for the Elimination of Tuberculosis and the Advisory Committee on Immunization Practices. *MMWR Recomm Rep* 1996;45 (RR–4):1–18.
10. Fine PE, Carneiro IA, Milstien JB, Clements CJ. Issues relating to the use of BCG in immunization programmes. A discussion document. Department of Vaccines and Biologicals, World Health Organization; Geneva: 1999.
11. Colditz GA, Brewer TF, Berkey CS, Wilson ME, Burdick E, Fineberg HV, *et al*. Efficacy of BCG vaccine in the prevention of tuberculosis. Meta-analysis of the published literature. *JAMA* 1994;271:698–702.
12. Rodrigues LC, Diwan VK, Wheeler JG. Protective effect of BCG against tuberculous meningitis and miliary tuberculosis: a meta-analysis. *Int J Epidemiol* 1993;22:1154–1158.
13. Stevens JP, Daniel TM. Bacille Calmette-Guérin immunization of health care workers exposed to multidrug-resistant tuberculosis: a decision analysis. *Tuber Lung Dis* 1996;77:315–321.
14. Marcus AM, Rose DN, Sacks HS, Schechter CB. BCG vaccination to prevent tuberculosis in health care workers: a decision analysis. *Prev Med* 1997;26:201–207.
15. Greenberg PD, Lax KG, Schechter CB. Tuberculosis in house staff. A decision analysis comparing the tuberculin screening strategy with the BCG vaccination. *Am Rev Respir Dis* 1991;143:490–495.
16. Stapledon RA, Lumb R, Lim IS. *Chemoprophylaxis and BCG in contacts of multidrug resistant tuberculosis*. Chapter 14, 213–224. In: Bastian I, Portaels F, eds. *Multidrug-resistant tuberculosis*. Kluwer Academic Publishers. The Netherlands 2000.
17. Management of persons exposed to multidrug-resistant tuberculosis. *MMWR Recomm Rep* 1992;41 (RR–11):61–71.
18. Office of Health Protection, National Notifiable Diseases Surveillance System, Australian Government Department of Health and Ageing, 2011.
19. An Advisory Committee Statement. National Advisory Committee on Immunization. Statement on Bacille Calmette Guérin (BCG) Vaccine. *Canada Commun Dis Rep* 2004;30:ACS 5.
20. Altes HK, Dijkstra F, Lugnèr A, Cobelens F, Wallinga J. Targeted BCG vaccination against severe tuberculosis in low prevalence settings: epidemiologic and economic assessment. *Epidemiology* 2009;20(4):562–568.
21. Krause VL. Tuberculosis in the young: focusing on those at risk. *Med J Aust* 1998;168:100–101.
22. Beilby J, Reed J, Baker J, Wilson K, Sansbury M, Antic R, et al. Tuberculosis surveillance in the South Australian Aboriginal community. *Med J Aust* 1990;153:149–155.
23. Plant AJ, Krause VL, Condon JR, Kerr C. Aborigines and tuberculosis: why they are at risk. *Aust J Public Health* 1995;19:487–491
24. Barry C, Konstantinos A, *et al*. Tuberculosis notifications in Australia, 2007. *Commun Dis Intell* 2009;33(3):304–315.
25. Johnson PD, Carlin JB, Bennett CM, Phelan PD, Starr M, Hulls J, *et al*. Prevalence of tuberculosis infection in Melbourne secondary school students. *Med J Aust* 1998;168:106–110.
26. Alperstein G, Morgan KR, Fett MJ, Nossar V, Stewart GJ. Prevalence of tuberculosis infection among primary school-entry children in Sydney. *Aust N Z J Public Health* 1996;20:123–128.
27. Markey P, Barclay L, Krause V. NT Mantoux school screening 1991–2000. *Northern Territory Disease Control Bulletin* 2002;9:6–9.
28. Broomell K, Antic R, Stapledon R. A decade of tuberculosis control in SA. [Abstract]. Program and Abstracts. The 2nd National Tuberculosis Conference: Australia's regional role in tuberculosis control. 1997 Nov 17–18:38. Sydney: The Public Health Association of Australia, 1997.
29. Guidance for national tuberculosis programmes on the management of tuberculosis in children. WHO/HTM/TB/2006.371. WHO/FCH/CAH/2006.7
30. Setia MS. The role of BCG in prevention of leprosy: a meta-analysis. *Lancet Infect Dis* 2006;6(3):162–170.
31. NNDSS Annual Report Writing Group. Australia's notifiable disease status, 2008: annual report of the National Notifiable Diseases Surveillance System – Other bacterial infections. *Commun Dis Intell* 2010;34(3):214.
32. Lotte A, Wasz-Hockert O, Poisson N, Engbaek H, Landmann H, Quast U, *et al*. Second IUATLD study on complications induced by intradermal BCG-vaccination. *Bull Int Union Tuberc Lung Dis* 1988;63:47–59.
33. Lotte A, Wasz-Hockett O, Poisson N, Dumitrescu N, Verron M. Complications induced by BCG vaccination; retrospective study. *Bull Int Union Tuberc* 1980;55:58–67.

34. Turnbull FM, McIntyre PB, Achat HM, Wang H, Stapledon R, Gold M, *et al*. National study of adverse reactions after vaccination with Bacille Calmette-Guérin. *Clin Infect Dis* 2002;34:447–453.

35. Lugosi L. Theoretical and methodological aspects of BCG vaccine from the discovery of Calmette and Guérin to molecular biology. A review. *Tuber Lung Dis* 1992;73:252–261.

36. Randomised controlled trial of single BCG, repeated BCG, or combined BCG and killed *Mycobacterium leprae* vaccine for prevention of leprosy and tuberculosis in Malawi. *Lancet* 1996;348:17–24.

37. Tala-Heikkila MM, Tuominen JE, Tala EO. Bacillus Calmette-Guérin revaccination questionable with low tuberculosis incidence. *Am J Respir Crit Care Med* 1998;157:1324–1327.

38. Rodrigues LC, Pereira SM, Cunha SS, Genser B, Ichihara MY, de Brito SC, *et al*. Effect of BCG revaccination on incidence of tuberculosis in school-aged children in Brazil: the BCG-REVAC cluster-randomised trial. *Lancet* 2005;366:1290–1295.

39. Global tuberculosis programme and global programme on vaccines: Statement on BCG revaccination for the prevention of tuberculosis. *Wkly Epidemiol Rec* 1995;70:229–231

40. Griffin JF, Chinn DN, Rodgers CR, Mackintosh CG. Optimal models to evaluate the protective efficacy of tuberculosis vaccines. *Tuber (Edinb)* 2001;81:133–139.

41. Orme IM. Progress in the development of new vaccines against tuberculosis. *Int J Tuberc Lung Dis* 1997;1:95–100.

42. Doherty TM, Andersen P. Vaccines for tuberculosis: novel concepts and recent progress. *Clin Microbiol Rev* 2005;18:687–702.

43. Sterne JA, Rodrigues LC, Guedes IN. Does the efficacy of BCG decline with time since vaccination? *Int J Tuberc Lung Dis* 1998;2:200–207.

Recommended composition of the northern hemisphere influenza vaccine for the 2013-14 season

On 21 February 2013, the World Health Organization (WHO) recommended that vaccines for the 2013-14 northern hemisphere influenza season contain the following:

A (H1N1): an A/California/7/2009 - like virus.

A (H3N2): a virus antigenically like the cell-propagated prototype virus A/Victoria/361/2011. It is recommended that A/Texas/50/2012 is used as the A(H3N2) vaccine component because of antigenic changes in earlier A/Victoria/361/2011-like vaccine viruses resulting from adaptation to propagation in eggs.

B: a B/Massachusetts/2/2012 - like virus (Yamagata lineage)

It is recommended that quadrivalent vaccines containing two influenza B viruses contain the above three viruses plus a B/Brisbane/60/2008-like virus (Victoria lineage).

For further information please see the WHO web site.

The composition of the Australian 2013 influenza vaccine was announced in October 2012. For further information please see the TGA web site.

Quarterly reports

OzFoodNet quarterly report, 1 April to 30 June 2012

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. In each Australian state and territory, OzFoodNet epidemiologists investigate outbreaks of enteric infection. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, which occurred in Australia between 1 April and 30 June 2012.

Data were received from OzFoodNet epidemiologists in all Australian states and territories. The data in this report are provisional and subject to change, as the results of outbreak investigations can take some months to finalise.

During the 2nd quarter of 2012, OzFoodNet sites reported 501 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports may be delayed, meaning that these figures under-represent the true burden of enteric disease outbreaks. In total, these outbreaks affected 8,585 people, of whom 186 were hospitalised. There were 19 deaths reported during these outbreaks. The majority of outbreaks (79%, n=394) were due to person-to-person transmission (Foodborne and suspected foodborne disease outbreaks), with 56% (221/394) of these occurring in residential aged care facilities.

Foodborne and suspected foodborne disease outbreaks

There were 31 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission. These outbreaks affected 382 people, resulted in 26 hospitalisations and no deaths. This compares with 51 outbreaks in the 1st quarter of 2012[1] and a 5 year mean of 33 outbreaks for the 2nd quarter between 2007 and 2011.

Salmonella enterica serotypes were identified as the aetiological agent in 17 outbreaks (55%) during this quarter (14 *S.* Typhimurium, Appendix 1). Of the remaining outbreaks, 3 (10%) were due to scombroid poisoning, 2 (6%) were due to *Campylobacter*, and 1 each due to norovirus, *Staphylococcus aureus* and an unspecified viral agent. In 5 outbreaks (16%), the aetiological agent was unknown.

Thirteen outbreaks (42% of foodborne or suspected foodborne outbreaks) reported in this quarter were associated with food prepared in restaurants (Table 2).

To investigate these outbreaks, sites conducted 5 cohort studies, 1 case control study and collected descriptive case series data for 22 investigations, while for 3 outbreaks, no individual patient data were collected. The evidence used to implicate food vehicles included analytical evidence in 2 outbreaks, microbiological evidence in 3 outbreaks and both analytical and microbiological evidence in 3 outbreaks. Descriptive evidence alone was obtained in 23 outbreak investigations.

Table 1: Outbreaks and clusters of gastrointestinal illness reported by OzFoodNet, 1 April to 30 June 2012 by mode of transmission

Transmission Mode	Number of outbreaks and clusters	Per cent of Total
Foodborne and suspected foodborne	31	6%
Waterborne and suspected waterborne	4	1%
Person-to-person	394	79%
Animal-to-person	2	<1%
Unknown (Salmonella cluster)	18	4%
Unknown (Other pathogen cluster)	3	1%
Unknown	49	10%
Total	501	100%*

* Percentages do not add up due to rounding.

The following jurisdictional summaries describe key outbreaks and public health actions that occurred in this quarter.

Australian Capital Territory

There were 2 reported outbreaks of foodborne or suspected foodborne illness during the quarter, of which 1 was due to *S.* Typhimurium and the other due to *Campylobacter*.

Description of key outbreaks

An increase in *Salmonella* notifications led to the detection of an outbreak of 20 cases (2 hospitalised) of *S.* Typhimurium phage type (PT) 135a multi-locus variable number of tandem repeats analysis (MLVA) profile 03-13-11-10-523 linked to a café. A case control study was undertaken, with univariate analysis showing that eating eggs benedict was significantly associated with illness (OR 63, 95% CI 6.1, 2771.7 p <0.001). An inspection of the café did not identify any serious food safety breaches however, the outbreak strain was identified in swabs of a fridge door and a cool room door handle. Traceback identified a New South Wales-based egg grading facility as the likely eggs supplier for the café. The facility was not linked to any previous outbreaks.[2]

Routine follow-up of a case of co-infection with *Salmonella* and *Campylobacter* identified an outbreak of 7 cases of gastrointestinal illness (2 confirmed *Campylobacter* cases and the mixed *Campylobacter/Salmonella* infection) associated with the consumption of home prepared chicken liver paté. No remaining paté was available for microbiological testing. It is suspected that some of the livers used in the paté were not cooked thoroughly.

Table 2: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet, 1 April to 30 June 2012 by food preparation setting

Food Preparation Setting	Outbreaks
Restaurant	13
Private residence	6
Commercial caterer	3
National franchised fast food	1
Aged care	1
Institution	1
Fast food restaurant	1
School	1
Bakery	1
Takeaway venue	1
Other	1
Unknown	1
Total	31

New South Wales

There were 13 reported outbreaks of foodborne or suspected foodborne illness during the quarter, of which 6 were due to *S.* Typhimurium and 1 each due to *S. aureus*, scombroid poisoning and a suspected viral agent. For the others no pathogen could be identified.

Description of key outbreaks

The following 3 outbreaks were linked to the same egg farm that was linked to an outbreak reported in the previous quarterly report.[1] The New South Wales Food Authority (NSWFA) issued an Improvement Notice to the owner of the egg farm requiring increased cleaning and improvements within the grading facility.

A public health unit follow-up of *Salmonella* cases identified an outbreak of *S.* Typhimurium MLVA profile 03-09-09-12-523 (historically associated with PT 170). Three family groups ate together at the same restaurant with 12 of 15 people becoming ill after consuming deep fried ice-cream. This restaurant was investigated in March 2012 following an outbreak of the same strain associated with the consumption of a raw egg dessert. The NSWFA issued a warning letter after the March outbreak highlighting the risk of illness associated with the use of raw and undercooked eggs. Traceback of the eggs during the March outbreak identified the egg farm described above.

Investigators were notified of an outbreak of *S.* Typhimurium MLVA profile 03-09-09-12-523 (historically associated with PT 170) involving 5 cases from a group of 49 who had eaten at the same restaurant as the outbreak above, but on a separate date. All those who were ill had eaten deep fried ice-cream. The outbreak strain was isolated from a sample of uncooked deep fried ice-cream taken from the premises. As a result of this outbreak, the NSWFA issued a Prohibition Order, prohibiting the use of raw and minimally cooked eggs/egg products.

Follow-up of a cluster of *S.* Typhimurium MLVA profile 03-09-09-12-523 (historically associated with PT 170) cases, identified 27 cases linked to a single bakery. This bakery was also supplied with eggs from the egg farm implicated in the restaurant outbreaks above. A NSWFA inspection of the bakery resulted in the outbreak strain being isolated from swabs/samples of re-usable piping bags, machine nozzles and freshly whipped cream. The business is now using only disposable piping bags and no further cases linked to this bakery have been reported since the NSWFA intervention.

Public Health authorities were notified of 22 individuals with gastrointestinal illness after attending a

sports championship event. The 22 ill were from a group of 40 people who ate the same meal. Of the 35 people interviewed, only the 22 who were ill had eaten fried rice. *Staphylococcus aureus* was grown from one stool specimen and *S. aureus* toxin was detected in another.

A Public Health Unit investigated an outbreak of *Salmonella* Typhimurium MLVA profile 03-10-07-13-523 (historically associated with PT 170) associated with a Vietnamese bakery. A total of 12 confirmed cases of *Salmonella* (11 cases MLVA profile 03-10-07-13-523 and 1 case MLVA profile 03-09-08-14-523) and 2 suspected cases were identified as part of the outbreak. All cases reported consuming a variety of rolls and sandwiches containing meat and/or salad items. The NSWFA investigation revealed that food for the bakery was prepared at a private residence and transported to the shop for sale. Foods were not held at an appropriate temperature and the premises was lacking an appropriate sanitiser. Sampled foods were found to have unacceptable growth of coliforms but no *Salmonella* was detected. The NSWFA prohibited the sale of all goods with the exception of bread until a subsequent inspection was passed where the defects had been corrected.

A Public Health Unit investigated an outbreak of *S.* Typhimurium MLVA profile 03-09-07-12-523 (historically associated with PT 170) in a family who ate at a restaurant. Three of 5 family members became ill after sharing a 'Tasting Platter of Desserts' which included a raw egg ice-cream. The restaurant used eggs from a local unlicensed egg producer and following an inspection of the egg farm, swabs of the egg shed and chicken faeces tested positive for *Salmonella* (*S.* Typhimurium MLVA profile 05-15-14-00-490).

Northern Territory

There were no reported outbreaks of foodborne or suspected foodborne illness during the quarter.

Queensland

There were 4 reported outbreaks of foodborne or suspected foodborne illness during the quarter, of which 2 were due to *S.* Typhimurium, and 1 each due to *Salmonella* sub species I and scombroid poisoning.

South Australia

There were 3 outbreaks of foodborne and suspected foodborne illness investigated during the quarter. One was due to *S.* Typhimurium and for the others no pathogen could be identified.

Description of key outbreak

Investigators were notified of an outbreak of *S.* Typhimurium PT 44 at 2 separate private functions catered by the same commercial caterer. Twenty-seven of 63 attendees (6 confirmed *S.* Typhimurium PT 44 cases) at the first function and 23 of 58 attendees (7 confirmed *S.* Typhimurium PT 44 cases) at the second function reported illness. An analytical study identified multiple foods with an association between illness and consumption; including chicken dishes, veal parmigiana and salads. Leftover food samples of some items were collected from the second function and beef lasagne was positive for *S.* Typhimurium PT 44. Illness is likely to have resulted from cross contamination of food due to poor hygiene and inadequate food handling procedures. Action was taken by the local council and state authorities to address a number of food handling issues identified during the investigation.

Tasmania

There was 1 reported outbreak of foodborne illness during the quarter, due to *S.* Typhimurium.

Investigators were notified of an outbreak of *S.* Typhimurium PT 135 (11 confirmed and 33 suspected cases) on board a vessel anchored in Tasmanian waters. Meals were served on board the vessel, and 2 food items that contained raw eggs were served at the time of the outbreak (mayonnaise and tartare sauce). These items were not available for sampling at the time the outbreak was reported, and all other environmental and food samples taken during the site inspection were negative for *Salmonella*. The vessel has stopped serving the dishes containing raw egg.

Victoria

There were 6 reported outbreaks of foodborne or suspected foodborne illness during the quarter, of which 2 were due to *S.* Typhimurium, and 1 each due to norovirus, *S.* Newport, *Salmonella* sub species I and scombroid poisoning.

Description of key outbreaks

Investigators were notified of an outbreak of *Salmonella* sub species I (ser 4,5,12:i:-) PT 193 affecting 14 people (8 confirmed *Salmonella*) following a party at a private residence. Four leftover food samples (ice-cream cake made with raw eggs, leftover eggs from the same batch used to make the cake, a tuna rice salad and a cheesecake) were positive for the outbreak strain of *Salmonella*. It is suspected that the rice salad and cheesecake were cross contaminated with the ice-cream cake during serving and or storage.

Investigators were notified of an outbreak of salmonellosis involving 12 cases (4 confirmed *S.* Typhimurium PT 170) at an aged care facility. There was a statistically significant association between consumption of vitamised/minced meals and illness (RR 8.2, 95% CI 3.1 – 21.9; p<0.001). A review of food preparation and processing procedures revealed that the equipment used to vitamise/mince meals was also used to process raw ingredients (e.g. cake batters containing raw eggs). It is suspected that this outbreak may have been caused by cross contamination due to the use of inadequately cleaned and sanitised vitamising equipment between uses.

Western Australia

There were 2 reported outbreaks of foodborne or suspected foodborne illness during the quarter, of which 1 was due to *S.* Typhimurium and the other due to *Campylobacter*.

Investigators were notified of an outbreak of *S.* Typhimurium pulsed-field gel electrophoresis (PFGE) pattern 0436 PT 135a involving 4 cases (2 hospitalised) who had eaten separately at the same café. One additional case reported that they may have visited this café during their incubation period but this was not confirmed. This café is part of a franchise chain; an additional four outbreaks associated with this chain have been reported in the last five years. An environmental investigation of the café found food handling and cleanliness deficiencies. Food samples collected during the inspection were negative for *Salmonella* and the source of the infection was not determined.

Comments

The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission, and in this quarter, 79% of outbreaks (n=394) were transmitted via this route. The number of foodborne outbreaks this quarter (n=31) is a decrease from the previous quarter and is consistent with the 5-year mean (2007-2011). *S.* Typhimurium continues to be a leading cause of foodborne outbreaks in Australia, with 58% (14/24) of outbreaks this quarter with a known *Salmonella* aetiology being due to this serotype.

Foodborne disease outbreak investigations this quarter have highlighted a range of high risk practices, many occurring in food service settings. Thirteen foodborne or suspected foodborne disease outbreaks were associated with foods prepared in a restaurant, while a further 3 were associated with foods prepared by caterers. Catering for large groups presents particular challenges in adequately controlling the temperature of stored foods and in preventing cross contamination between raw and cooked foods. There may often be inadequate facilities for the safe storage and handling of large quantities of food at the location where it is to be served.

Almost half (8/17) of the foodborne or suspected foodborne outbreaks where *Salmonella* was implicated as the responsible agent were associated with raw or undercooked egg products (raw egg dressings, raw/undercooked egg desserts). To address this continuing issue, members of the OzFoodNet Egg Working Group are working towards describing the national epidemiology of egg-associated outbreaks. Jurisdictional food safety authorities are focussing on communication and education in relation to the use of raw egg products in commercial settings.

A limitation of the outbreak data provided by OzFoodNet sites for this report was the potential for variation in the categorisation of the features of outbreaks depending on circumstances and investigator interpretation. Changes in the number of foodborne outbreaks should be interpreted with caution due to the small number each quarter.

Acknowledgements

OzFoodNet thanks the investigators in the public health units and state and territory departments of health, as well as public health laboratories, local government environmental health officers and food safety agencies who provided data used in this report. We would particularly like to thank reference laboratories for conducting sub-typing of *Salmonella*, *Listeria monocytogenes* and other enteric pathogens and for their continuing work and advice during the quarter.

OzFoodNet contributors to this report include (in alphabetical order): Robert Bell (QLD), Barry Combs (WA), Emily Fearnley (SA), Gerard Fitzsimmons (DoHA), Neil Franklin (NSW), Catriona Furlong (NSW), Robyn Gibbs (WA), Joy Gregory (VIC), Michelle Green (NT and TAS), Karin Lalor (VIC), Robyn Leader (DoHA), Cameron Moffatt (ACT), Sally Munnoch (Hunter New England), Jennie Musto (NSW), Amy Parry (SA), Nevada Pingault (WA), Timothy Sloan-Gardner (DoHA), Russell Stafford (QLD), and Kate Ward (NSW).

Correspondence:

Timothy Sloan-Gardner, Office of Health Protection, Australian Government Department of Health and Ageing, GPO Box 9848, MDP 14, Canberra, ACT 2601. Telephone: +61 2 6289 2777. Facsimile: +61 2 6289 2600. Email: ozfoodnet@health.gov.au

Reference

1. OzFoodNet Working Group. OzFoodNet quarterly report, 1 January to 31 March 2012. *Commun Dis Intell* 2012;36(4):E353-359.
2. Moffatt CR, Appuhamy R, Kaye A, Carswell A, Denehy D. An outbreak of *Salmonella* Typhimurium phage type 135a gastroenteritis linked to eggs served at an Australian Capital Territory cafe. *Commun Dis Intell* 2012;36(3):E281-285.

Appendix 1: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites, 1 April to 30 June 2012 (n=31)

State	Month	Setting Prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
ACT	April	Restaurant	*S.* Typhimurium PT 135a (MLVA profile 03-13-11-10-523)	20	2	AM	Eggs Benedict
ACT	May	Private residence	Campylobacter	7	0	D	Chicken liver pate
NSW	April	Private residence	Viral Suspected	19	0	A	Home-made cake
NSW	April	Restaurant	Unknown	3	0	D	Unknown
NSW	April	Restaurant	*S.* Typhimurium MLVA profile 03-09-09-12-523 (historically associated with PT 170)	5	1	D	Deep fried ice-cream
NSW	April	Take-away	*S.* Typhimurium MLVA profile 03-10-07-13-523 (historically associated with PT 170)	14	0	D	Vietnamese bakery goods
NSW	May	Bakery	*S.* Typhimurium MLVA profile 03-09-09-12-523 (historically associated with PT 170)	27	Unknown	M	Multiple bakery goods
NSW	May	Commercial caterer	Unknown	5	0	D	Unknown
NSW	May	Private residence	Scombroid poisoning	3	0	D	Tuna
NSW	May	Restaurant	*S.* Typhimurium MLVA profile 03-09-09-12-523 (historically associated with PT 170)	12	0	M	Deep fried ice-cream
NSW	May	Institution	*S.* Typhimurium MLVA profile 03-14-09-14-523	3	0	D	Unknown
NSW	June	Commercial caterer	*Staphylococcus. aureus*	22	6	AM	Suspected fried rice
NSW	June	National franchised fast food	Unknown	7	0	D	Unknown
NSW	June	Restaurant	Unknown	35	0	D	Unknown
NSW	June	Restaurant	*S.* Typhimurium MLVA profile 03-09-07-12-523 (historically associated with PT 170)	3	0	D	Ice-cream containing raw egg
QLD	April	Restaurant	*S.* Typhimurium PT 193 MLVA profile 03-13-14-10-524	3	0	D	Unknown
QLD	May	Private residence	Scombroid poisoning	4	0	D	Suspected seafood meal
QLD	May	Unknown	*Salmonella* sub species I ser 4,5,12:-:1,2 MLVA profile 03-14-00-00-490	10	3	D	Unknown
QLD	June	Restaurant	*S.* Typhimurium MLVA profiles 03-12-15-09-524 and 03-12-16-09-524	2	2	D	French Toast
SA	May	Commercial caterer	*S.* Typhimurium PT 44	50	2	AM	Suspected cross contamination
SA	May	Restaurant	Unknown	3	0	D	Unknown

Appendix 1 *continued*: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites, 1 April to 30 June 2012 (n=31)

State	Month	Setting Prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
SA	May	Fast food restaurant	Unknown	4	0	D	Unknown
TAS	April	Other	*S.* Typhimurium PT 135	44	2	D	Suspect raw egg mayonnaise and/or tartare sauce
VIC	April	Private residence	*Salmonella* sub species I ser 4,5,12:i:- PT 193	14	2	M	Raw egg ice-cream cake
VIC	April	Private residence	*S.* Typhimurium PT 4	4	4	D	Raw egg smoothies
VIC	April	Restaurant	Norovirus	27	0	D	Multiple foods
VIC	April	Aged care	*S.* Typhimurium PT 170	12	1	A	Vitamised meals
VIC	May	School	Scombroid poisoning	4	0	D	Tuna
VIC	June	Restaurant	*S.* Newport	10	0	D	Kebabs
WA	April	Restaurant	*S.* Typhimurium PFGE pattern 0436, PT 135a	4	1	D	Unknown
WA	May	Restaurant	*Campylobacter*	4	0	D	Suspected chicken liver pate

A Analytical epidemiological association between illness and 1 or more foods.
D Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission.
M Microbiological confirmation of agent in the suspected vehicle and cases.
MLVA Multi-locus variable number tandem repeat analysis.
PFGE Pulsed-field gel electrophoresis.
PT Phage type.

Additional Reports

National Notifiable Diseases Surveillance System tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 45,254 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 October and 31 December 2012 (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 except South Australia

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 October to 31 December 2012, by date of diagnosis*

Disease	State or territory								Total 3rd quarter 2012	Total 2nd quarter 2012	Total 3rd quarter 2011	Last 5 years mean 3rd quarter	Ratio††	Year to date 2012	Last 5 years YTD mean
	ACT	NSW	NT	QLD	SA	TAS	VIC	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	3	0	3	0	1	0.4	7.5	3	0.4
Hepatitis B (newly acquired)†	0	8	1	16	3	1	10	9	48	48	52	57.0	0.8	184	243.6
Hepatitis B (unspecified)‡	25	512	39	186	72	19	302	217	1,372	1,755	1,571	1,628.4	0.8	6,358	6,754.8
Hepatitis C (newly acquired)†	1	15	0	NN	16	5	36	40	113	103	87	94.6	1.2	437	389.0
Hepatitis C (unspecified)‡	31	736	68	605	74	63	500	277	2,354	2,471	2,318	2,606.4	0.9	9,643	10,847.8
Hepatitis D	0	2	0	2	1	0	1	0	6	5	4	6.8	0.9	28	35.8
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.8
Campylobacteriosis	101	NN	36	1,018	444	267	928	528	3,322	3,686	4,367	4,559.8	0.7	15,078	16,656.8
Cryptosporidiosis	1	136	14	92	12	5	77	59	396	332	412	466.4	0.8	3,094	2,544.2
Haemolytic uraemic syndrome	0	0	0	1	0	0	3	0	4	3	4	5.4	0.7	18	17.2
Hepatitis A	0	15	0	7	2	1	15	2	42	39	40	83.6	0.5	163	283.6
Hepatitis E	0	4	0	1	0	0	2	0	7	5	9	5.6	1.3	35	34.6
Listeriosis	0	12	0	2	0	1	7	3	25	18	22	17.0	1.5	92	70.2
STEC, VTEC§	0	3	0	12	11	1	4	1	32	19	36	34.2	0.9	110	101.2
Salmonellosis	59	825	78	664	154	71	657	272	2,780	2,024	2,907	2,508.6	1.1	11,217	10,289.8
Shigellosis	2	32	19	23	3	2	31	15	127	106	140	150.0	0.8	544	617.0
Typhoid Fever	0	12	0	5	1	0	6	7	31	15	31	23.8	1.3	122	108.0
Quarantinable diseases															
Cholera	0	0	0	0	0	0	3	0	3	2	1	1.4	2.1	10	5.0
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	2.0

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2012, by date of diagnosis*

Disease	State or territory								Total 3rd quarter 2012	Total 2nd quarter 2012	Total 3rd quarter 2011	Last 5 years mean 3rd quarter	Ratio††	Year to date 2012	Last 5 years YTD mean
	ACT	NSW	NT	QLD	SA	TAS	VIC	WA							
Sexually transmissible infections															
Chlamydial infection§§**	304	4,916	542	4,529	1,041	406	1,231	2,781	15,750	19,979	19,608	16,003.8	1.0	78,616	65,700.0
Donovanosis	0	0	0	0	0	0	0	0	0	1	0	0.0	0.0	1	1.4
Gonococcal infection§§	26	960	351	652	78	14	460	530	3,071	3,090	3,202	2,298.0	1.3	13,232	9,188.0
Syphilis – congenital	0	0	0	0	0	0	0	0	0	0	1	1.4	0.0	1	5.2
Syphilis < 2 years§§	4	48	3	70	22	2	85	24	258	393	307	299.4	0.9	1,415	1,302.8
Syphilis > 2 years or unspecified duration‡§§	5	29	15	50	NN	6	136	20	261	316	320	320.4	0.8	1,196	1,333.8
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.8
Haemophilus influenzae type b	0	0	0	2	0	0	1	0	3	5	2	4.4	0.7	15	19.8
Influenza (laboratory confirmed)	40	479	66	789	859	35	547	220	3,035	32,877	2,905	2,012.6	1.5	44,525	23,887.4
Measles	0	20	1	0	3	0	0	5	29	129	50	14.4	2.0	199	88.8
Mumps	0	11	0	10	4	1	7	4	37	48	42	86.8	0.4	192	257.0
Pertussis	168	1,143	38	2,158	218	453	1,015	456	5,649	5,458	10,336	8,144.6	0.7	23,677	24,478.2
Pneumococcal disease (invasive)	7	120	10	63	23	9	82	43	357	719	386	348.6	1.0	1,811	1,635.8
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Rubella	0	0	0	2	0	0	3	0	5	8	8	6.0	0.8	35	39.8
Rubella Congenital	0	0	0	0	0	0	0	0	0	1	0	0.0	0.0	1	0.4
Tetanus	0	1	0	1	0	0	1	0	3	2	0	0.2	15.0	6	3.0
Varicella zoster (Chickenpox)	3	NN	28	44	120	4	199	88	486	597	651	586.0	0.8	1,939	1,811.8
Varicella zoster (Shingles)	18	NN	39	17	443	49	278	279	1,123	1,069	1,068	733.6	1.5	4,397	2,718.4
Varicella zoster (Unspecified)	35	NN	0	1,208	93	26	705	292	2,359	2,051	2,177	1,692.2	1.4	8,512	6,068.8
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	8	0	0	0	0	8	2	8	5.4	1.5	16	16.8
Barmah Forest virus infection	1	101	42	374	22	0	11	86	637	279	345	368.4	1.7	1,701	1,718.6
Dengue virus infection	7	50	3	44	8	1	38	35	186	193	213	216.4	0.9	1,481	863.2
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	1	0.2

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 October to 31 December 2012, by date of diagnosis*

Disease	State or territory								Total 3rd quarter 2012	Total 2nd quarter 2012	Total 3rd quarter 2011	Last 5 years mean 3rd quarter	Ratio††	Year to date 2012	Last 5 years YTD mean
	ACT	NSW	NT	QLD	SA	TAS	VIC	WA							
Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	1	0.4	0.0	0	1.6
Malaria	3	16	7	26	2	1	12	17	84	99	110	112.6	0.7	325	479.0
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	1	0.2	0.0	1	4.4
Ross River virus infection	2	103	44	330	41	0	56	103	679	483	747	926.6	0.7	4,651	4,956.0
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.4
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Brucellosis	0	1	0	6	1	0	0	0	8	9	9	8.0	1.0	25	34.8
Leptospirosis	0	2	0	2	1	0	1	2	8	11	21	22.4	0.4	111	141.2
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	1	0	1	1	0	21	3	27	14	26	23.0	1.2	65	81.2
Q fever	0	21	2	31	0	0	1	1	56	80	97	91.8	0.6	318	361.0
Tularaemia	0	0	0	0	0	0	0	0	0	0	1	1.0	0.0	0	2.0
Other bacterial infections															
Legionellosis	1	12	0	19	9	3	17	28	89	97	100	83.8	1.1	367	305.2
Leprosy	0	0	0	0	0	0	1	0	1	1	3	3.0	0.3	3	9.8
Meningococcal disease (invasive)‡‡	0	9	0	12	5	2	7	3	38	83	49	62.4	0.6	222	264.2
Tuberculosis	3	96	7	39	21	2	126	48	342	302	390	384.2	0.9	1,202	1,282.8
Total	**847**	**10,451**	**1,453**	**13,121**	**3,808**	**1,450**	**7,626**	**6,498**	**45,254**	**79,027**	**55,186**			**237,395**	

* Date of diagnosis is the date of symptom onset, or if this is not available, the earliest of the specimen collection date, the notification date (when the health professional signed the form or laboratory issued the results), or the notification receive date (when notification of the disease was received by the health authority). Hepatitis B and C and tuberculosis were analysed by date of notification.

† Newly-acquired hepatitis includes cases where the infection was determined to have been acquired within 24 months prior to diagnosis.

‡ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined.

§ Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli*.

‖ In the national case definitions for chlamydial, gonnoccocal and syphilis infection, the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode.

** Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for in South Australia, where only genital tract specimens are reported, and the Northern Territory and Western Australia where ocular specimens are excluded, and Western Australia also excludes perinatal infection.

†† Ratios of the current quarter to the mean of the last 5 quarters (ratios for Varicella are based on 4 years data)

‡‡ Only invasive meningococcal disease is nationally notifiable, but New South Wales, the Australia Capital Territory and South Australia also report conjunctival cases.

NN Not notifiable

NEC Not elsewhere classified

Table 3. Notification rates of diseases, 1 October to 31 December 2012, by date of diagnosis and state or territory (Annualised rate per 100,000 population)[*†]

Disease	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	0.2	0.0	0.1
Hepatitis B (newly acquired)[‡]	0.0	0.4	1.7	1.4	0.7	0.8	0.7	1.5	0.8
Hepatitis B (unspecified)[§]	27.4	28.0	67.7	16.2	17.4	14.9	21.5	36.9	24.3
Hepatitis C (newly acquired)[‡]	1.1	0.8	0.0	NN	3.9	3.9	2.6	6.8	2.5
Hepatitis C (unspecified) [§]	33.9	40.3	118.1	52.8	17.9	49.4	35.6	47.2	41.6
Hepatitis D	0.0	0.1	0.0	0.2	0.2	0.0	0.1	0.0	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	110.5	NN	62.5	88.9	107.2	209.2	66.0	89.9	86.8
Cryptosporidiosis	1.1	7.4	24.3	8.0	2.9	3.9	5.5	10.0	7.0
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.1	0.0	0.0	0.2	0.0	0.1
Hepatitis A	0.0	0.8	0.0	0.6	0.5	0.8	1.1	0.3	0.7
Hepatitis E	0.0	0.2	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Listeriosis	0.0	0.7	0.0	0.2	0.0	0.8	0.5	0.5	0.4
STEC, VTEC[‖]	0.0	0.2	0.0	1.0	2.7	0.8	0.3	0.2	0.6
Salmonellosis	64.5	45.2	135.4	58.0	37.2	55.6	46.8	46.3	49.2
Shigellosis	2.2	1.8	33.0	2.0	0.7	1.6	2.2	2.6	2.2
Typhoid Fever	0.0	0.7	0.0	0.4	0.2	0.0	0.4	1.2	0.5
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.1
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
Sexually transmissible infections									
Chlamydial infection[¶**]	332.6	269.3	941.1	395.5	251.4	318.1	87.6	473.5	278.5
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection[¶]	28.4	52.6	609.5	56.9	18.8	11.0	32.7	90.2	54.3
Syphilis - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years[¶]	4.4	2.6	5.2	6.1	5.3	1.6	6.0	4.1	4.6
Syphilis > 2 years or unspecified duration[¶]	5.5	1.6	26.0	4.4	NN	4.7	9.7	3.4	5.0
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	0.0	0.2	0.0	0.0	0.1	0.0	0.1
Influenza (laboratory confirmed)	43.8	26.2	114.6	68.9	207.5	27.4	38.9	37.5	53.7
Measles	0.0	1.1	1.7	0.0	0.7	0.0	0.0	0.9	0.5
Mumps	0.0	0.6	0.0	0.9	1.0	0.8	0.5	0.7	0.7

Table 3 *continued*: Notification rates of diseases, 1 October to 31 December 2012, by date of diagnosis and state or territory (Annualised rate per 100,000 population)*†

Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Pertussis	183.8	62.6	66.0	188.5	52.6	354.9	72.2	77.6	99.9
Pneumococcal disease (invasive)	7.7	6.6	17.4	5.5	5.6	7.1	5.8	7.3	6.3
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.0	0.0	0.2	0.0	0.0	0.2	0.0	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	0.1	0.0	0.0	0.1	0.0	0.1
Varicella zoster (Chickenpox)	3.3	NN	48.6	3.8	29.0	3.1	14.2	15.0	12.7
Varicella zoster (Shingles)	19.7	NN	67.7	1.5	107.0	38.4	19.8	47.5	29.3
Varicella zoster (Unspecified)	38.3	NN	0.0	105.5	22.5	20.4	50.2	49.7	61.6
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.1
Barmah Forest virus infection	1.1	5.5	72.9	32.7	5.3	0.0	0.8	14.6	11.3
Dengue virus infection	7.7	2.7	5.2	3.8	1.9	0.8	2.7	6.0	3.3
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	3.3	0.9	12.2	2.3	0.5	0.8	0.9	2.9	1.5
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	2.2	5.6	76.4	28.8	9.9	0.0	4.0	17.5	12.0
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.5	0.2	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.1	0.0	0.2	0.2	0.0	0.1	0.3	0.1
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.1	0.0	0.1	0.2	0.0	1.5	0.5	0.5
Q fever	0.0	1.2	3.5	2.7	0.0	0.0	0.1	0.2	1.0
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	1.1	0.7	0.0	1.7	2.2	2.4	1.2	4.8	1.6
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Meningococcal disease (invasive)††	0.0	0.5	0.0	1.0	1.2	1.6	0.5	0.5	0.7
Tuberculosis	3.3	5.3	12.2	3.4	5.1	1.6	9.0	8.2	6.0

* Date of diagnosis is the date of symptom onset, or if this is not available, the earliest of the specimen collection date, the notification date (when the health professional signed the form or laboratory issued the results) or the notification receive date (when notification of the disease was received by the health authority). Hepatitis B and C and tuberculosis were analysed by date of notification.

† Rate per 100,000 population. The annualisation factor was 4.0.

‡ Newly-acquired hepatitis and syphilis includes cases where the infection was determined to have been acquired within 24 months prior to diagnosis.

§ Unspecified hepatitis includes cases where the duration of infection could not be determined.

|| Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli*.

¶ In the national case definitions for chlamydial, gonnoccocal and syphilis infection, the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode.

** Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for in South Australia, where only genital tract specimens are reported, and the Northern Territory and Western Australia where ocular specimens are excluded, and Western Australia also excludes perinatal infection.

†† Only invasive meningococcal disease is nationally notifiable, but New South Wales, the Australia Capital Territory and South Australia also report conjunctival cases.

NN Not notifiable

NEC Not elsewhere classified

Gonococcal Surveillance Australia: Quarter 3, 2012

Monica M Lahra

The Prince of Wales Hospital, Randwick, NSW, 2031 for The Australian Gonococcal Surveillance Programme

Introduction

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various states and territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin which are current or potential agents used for the treatment of gonorrhoea. Azithromycin testing is now performed by all states and territories as it has a role as part of a dual therapy regimen in the treatment of gonorrhoea. When in *vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates from a general population, it is usual to consider the removal of that agent from the list of recommended treatments.[1] Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as tetracycline-resistant *Neisseria gonorrhoeae* (TRNG). Tetracyclines are however not a recommended therapy for gonorrhoea in Australia. These data are reported in the AGSP Annual Report. Comparability of data is achieved by means of a standardised system of testing and a programme-specific quality assurance process. Due to the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. Since quarter 2, 2012 these data have been presented quarterly in tabulated form, as well as in the AGSP Annual Report. Tables for quarters 1, 2 and 3 2012 have been included in this report to complete presentation of the AGSP quarterly data in this format for 2012.

Comments and Notes

Penicillin resistant *N. gonorrhoeae* are defined as those isolates with an minimum inhibitory concentration (MIC) to penicillin equal to or greater than 1.0 mg/L. Total penicillin resistance includes penicillinase producing *N. gonorrhoea* (PPNG) and chromosomally mediated resistance to penicillin (CMRP).

Quinolone resistant *N. gonorrhoeae* are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L, and azithromycin resistance as those isolates with an MIC to azithromycin equal to or greater than 1.0 mg/L. In the Northern Territory, the number of isolates for which results were available in the 3rd quarter 2012 was lower than in the previous two quarters as data from Alice Springs was not available. Penicillin and ciprofloxacin resistance increased to 8.5% in this quarter in the 47 *N. gonorrhoeae* isolates tested in the Northern Territory.

Regarding ceftriaxone, isolates with MIC values in the range 0.06-0.125 mg/L are reported as having decreased susceptibility. There has not been an isolate reported in Australia with an MIC >0.125mgL. In Figure 1 the AGSP data for 2011 Quarters 1, 2 and 3, and for the same period in 2012 is presented by ceftriaxone MIC value to enable monitoring of the shift in MIC values in N. *gonorrhoeae* isolates over time, in addition to reporting the proportion in the category of decreased susceptibility. A decrease in proportion of isolates with a ceftriaxone MIC value of ≤ 0.008mgL is evident in 2012 compared

Table 1: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to ciprofloxacin, azithromycin and penicillin, Australia, 1 January to 31 March 2012, by state or territory

State or Territory	Number of isolates tested	Decreased Susceptibility		Resistance					
		Ceftriaxone		Ciprofloxacin		Azithromycin		Penicillin	
		No.	%	No.	%	No.	%	No.	%
Australian Capital Territory	13	0	0.0	8	61.5	0	0.0	3	23.1
New South Wales	447	17	3.8	121	27.1	4	0.9	119	26.6
Northern Territory	77	0	0.0	1	1.3	1	1.3	1	1.3
Queensland	205	3	1.5	35	17.1	2	1.0	44	21.5
South Australia	27	0	0.0	8	29.6	8	29.6	12	44.4
Tasmania	1	1	100.0	1	100.0	0	0.0	0	0.0
Victoria	312	21	6.7	166	53.2	14	4.5	178	57.1
Western Australia	130	2	1.5	29	22.3	1	0.8	21	16.2
Australia	1,212	44	3.6	369	30.4	30	2.5	378	31.2

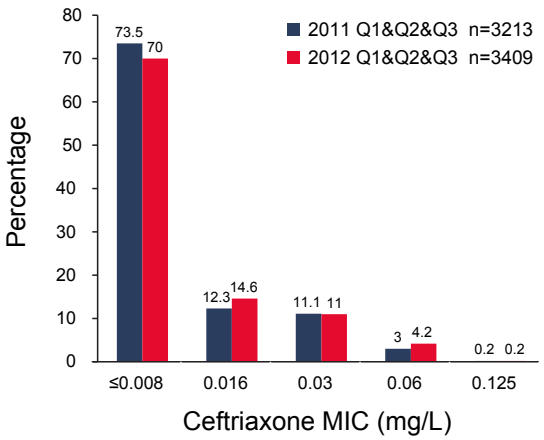
Table 2: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to ciprofloxacin, azithromycin and penicillin, Australia, 1 April to 30 June 2012, by state or territory

State or Territory	Number of isolates tested	Decreased Susceptibility		Resistance					
		Ceftriaxone		Ciprofloxacin		Azithromycin		Penicillin	
		No.	%	No.	%	No.	%	No.	%
Australian Capital Territory	9	0	0.0	4	44.4	0	0.0	3	33.3
New South Wales	421	16	3.8	115	27.3	0	0.0	105	24.9
Northern Territory	82	0	0.0	0	0.0	0	0.0	1	1.2
Queensland	174	8	4.6	26	14.9	2	1.1	48	27.6
South Australia	44	0	0.0	7	15.9	0	0.0	7	15.9
Tasmania	4	0	0.0	1	25.0	0	0.0	3	75.0
Victoria	355	21	5.9	149	42.0	108	30.4	202	56.9
Western Australia	119	1	0.8	30	25.2	1	0.8	26	21.8
Australia	1208	46	3.8	332	27.5	111	9.2	395	32.7

Table 3: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to ciprofloxacin, azithromycin and penicillin, Australia, 1 July to 30 September 2012, by state or territory

State or Territory	Number of isolates tested	Decreased Susceptibility		Resistance					
		Ceftriaxone		Ciprofloxacin		Azithromycin		Penicillin	
		No.	%	No.	%	No.	%	No.	%
Australian Capital Territory	15	1	6.7	1	6.7	0	0.0	0	0.0
New South Wales	442	20	4.5	146	33.0	3	0.7	126	28.5
Northern Territory	47	0	0.0	4	8.5	0	0.0	4	8.5
Queensland	159	2	1.3	25	15.7	3	1.9	42	26.4
South Australia	41	1	2.4	16	39.0	0	0.0	19	46.3
Tasmania	4	0	0.0	2	50.0	0	0.0	2	50.0
Victoria	279	36	12.9	120	43.0	7	2.5	146	52.3
Western Australia	136	3	2.2	34	25.0	0	0.0	37	27.2
Australia	1123	63	5.6	348	31.0	13	1.2	376	33.5

Figure 1: Distribution of ceftriaxone MIC values in gonococcal isolates tested in the AGSP, Quarters 1, 2 and 3: January to September, 2011 and 2012



with 2011, with increases in the higher MIC values demonstrating a right shift over these periods which will continue to be monitored.

Reference

1. Management of sexually transmitted diseases. World Health Organization [Internet] 1997. [Revised1997:p.37]. Available from: http://whqlibdoc.who.int/hq/1997/WHO_GPA_TEM_94.1_Rev.1.pdf

Australian Childhood Immunisation Coverage

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 2012. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, pneumococcal conjugate, 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 a 3rd dose of a DTPa vaccine, a 3rd dose of polio vaccine, a 2nd or 3rd dose of a polyribosylribitol phosphate–outer membrane protein (PRP-OMP) containing *Haemophilus influenzae* type b (Hib) vaccine or a 3rd dose of any other Hib vaccine, and a 2nd or 3rd dose of a Comvax hepatitis B vaccine or a 3rd dose of dose of any other hepatitis B vaccine. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of a 3rd or 4th dose of a DTPa vaccine, a 3rd dose of polio vaccine, a 3rd or 4th dose of a PRP-OMP containing Hib vaccine or a 4th dose of any other Hib vaccine, a 3rd or 4th dose of Comvax hepatitis B vaccine or a 4th dose of any other hepatitis B vaccine, and a 1st 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 a 4th or 5th dose of a DTPa vaccine, a 4th dose of polio vaccine, and 2nd dose of an MMR-containing vaccine.

A full description of the basic methodology used can be found in CDI 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: brynleyh.hull@health.nsw.gov.au

Results

The percentage of children 'fully immunised' by 12 months of age for Australia decreased marginally from the previous quarter by 0.2 of a percentage point to 91.8% (Table 1). There were no important changes in coverage for any individual vaccines due at 12 months of age or by jurisdiction.

The percentage of children 'fully immunised' by 24 months of age for Australia decreased marginally from the previous quarter by 0.2 of a percentage point to 92.8% (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 increased from the previous quarter by 1.2 percentage points to 91.9% (Table 3). This continues the upward trend in coverage for this age milestone. There were important increases in coverage for all individual vaccines due at 60 months of age for most jurisdictions.

Table 1. Percentage of children immunised at 1 year of age for the birth cohort 1 July to 30 September 2011; preliminary results by vaccine and state or territory, assessment date 31 December 2012

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Australia
Total number of children	1,338	24,735	973	15,406	5,024	1,543	18,508	8,191	75,718
Diphtheria, tetanus, pertussis (%)	93.1	91.6	90.9	92.6	92.0	93.6	92.9	90.8	92.1
Poliomyelitis (%)	92.9	91.5	90.9	92.6	92.0	93.5	92.8	90.7	92.0
Haemophilus influenzae type b (%)	93.0	91.5	90.8	92.5	92.0	93.3	92.7	90.6	92.0
Hepatitis B (%)	92.5	91.3	90.5	92.2	91.7	93.1	92.4	90.2	91.7
Fully immunised (%)	92.5	91.2	90.5	92.2	91.6	93.1	92.3	90.0	91.6
Change in fully immunised since last quarter (%)	-0.6	-0.4	-2.3	+0.0	-0.4	+0.8	-0.0	-0.3	-0.2

Table 2. Percentage of children immunised at 2 years of age for the birth cohort 1 July to 30 September 2010; preliminary results by disease and state or territory, assessment date 31 December 2012*

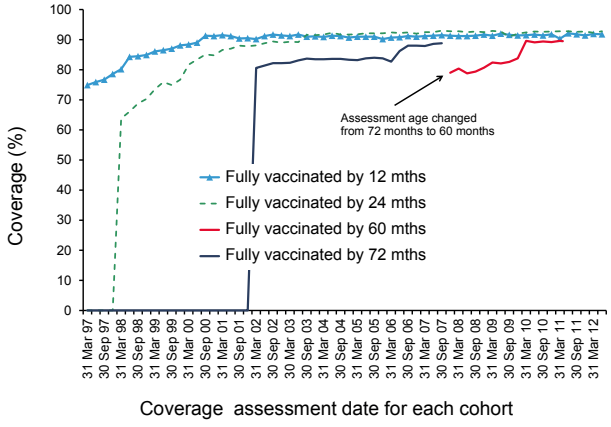
Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,354	24,914	932	15,797	4,922	1,512	18,500	8,174	76,105
Diphtheria, tetanus, pertussis (%)	95.8	94.8	95.2	94.8	94.3	95.3	95.9	93.3	94.9
Poliomyelitis (%)	95.7	94.7	95.2	94.8	94.2	95.3	95.8	93.3	94.8
Haemophilus influenzae type b (%)	95.9	95.1	95.3	94.8	94.2	95.6	95.8	93.5	95.0
Measles, mumps, rubella (%)	94.6	93.7	94.7	94.1	93.3	95.0	94.8	92.6	94.0
Hepatitis B (%)	94.8	94.3	94.6	94.3	93.9	95.3	95.3	92.5	94.4
Fully immunised (%)	93.1	92.3	93.6	92.8	92.0	93.9	93.5	90.8	92.6
Change in fully immunised since last quarter (%)	-0.3	-0.5	-0.2	+0.2	-1.1	+0.0	-0.0	+0.1	-0.2

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2011;35(1):49.

Table 3. Percentage of children immunised at 6 years of age for the birth cohort 1 July to 30 September 2007; preliminary results by disease and state or territory, assessment date 31 December 2012

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,316	25,441	878	16,620	5,165	1,733	19,023	8,440	78,616
Diphtheria, tetanus, pertussis (%)	92.8	92.1	91.1	92.5	92.0	93.7	93.6	90.3	92.4
Poliomyelitis (%)	92.7	92.0	91.1	92.5	92.0	93.5	93.5	90.4	92.3
Measles, mumps, rubella (%)	92.3	92.0	91.0	92.4	91.9	94.1	93.6	90.3	92.3
Fully immunised (%)	92.0	91.7	90.6	92.0	91.5	93.4	93.1	89.8	91.9
Change in fully immunised since last quarter (%)	-0.5	+1.0	+1.0	+1.5	+2.4	+0.5	+1.3	+0.8	+1.2

Figure: Trends in vaccination coverage, Australia, 1997 to 30 September 2012, by age cohort



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 5 years of age is now higher for the first time than coverage at 12 months of age.

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 and Ageing, 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 influenza-like illness (ILI) patients for a range of respiratory viruses including influenza A, influenza B and influenza pandemic A(H1N1) 2009.

The list of conditions reported is reviewed annually by the ASPREN management committee. In 2012, 4 conditions were being monitored. These were ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions for these conditions are described in Surveillance systems reported in CDI, published in *Commun Dis Intell* 2013;37(1):60.

Reporting period 1 July to 30 September 2012

Sentinel practices contributing to ASPREN were located in all 8 jurisdictions in Australia. A total of 236 general practitioners contributed data to ASPREN

Figure 1: Consultation rates for influenza-like illness, ASPREN, 2011 to 2012, by week of report

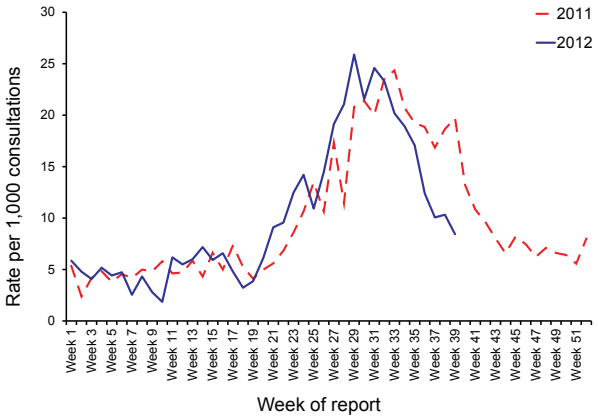
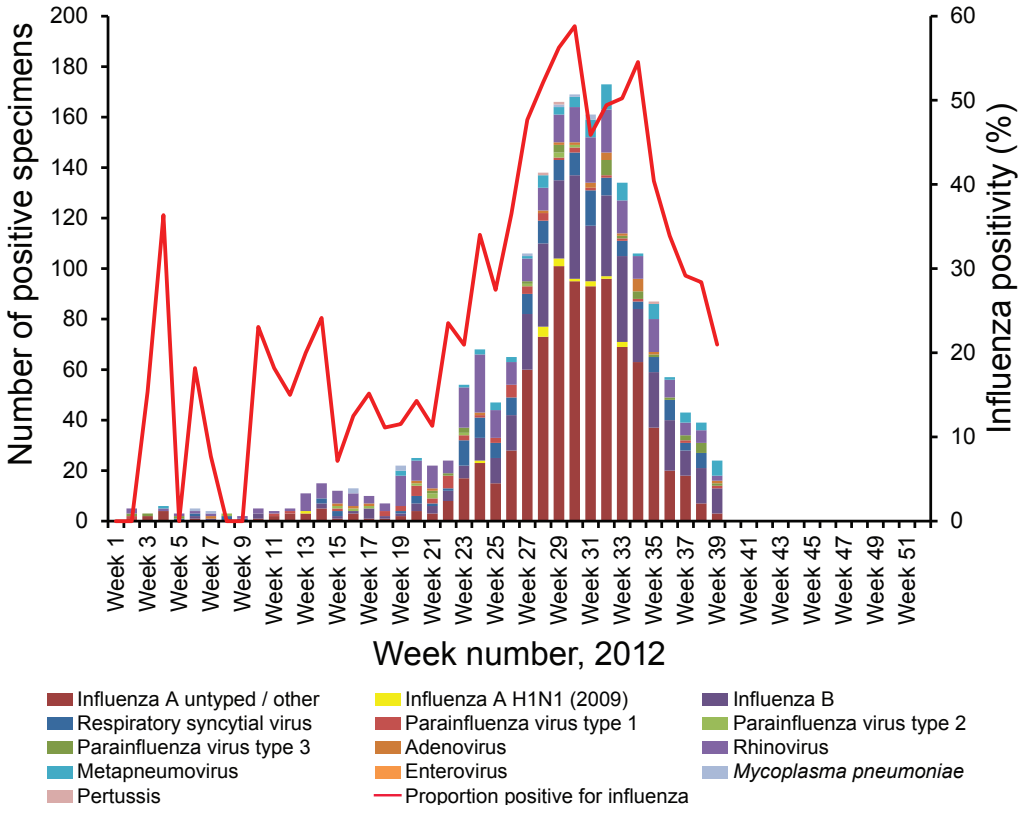


Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January to 30 September 2012, by week of report



in the third quarter of 2012. Each week an average of 194 general practitioners provided information to ASPREN at an average of 20,235 (range 15,070–23,235) consultations per week and an average of 429 (range 241–571) notifications per week.

ILI rates reported from 1 July to 30 September 2012 averaged 17 cases per 1,000 consultations (range 7–26 cases per 1,000 consultations). This was lower than the same reporting period in 2011, which averaged 19 cases per 1,000 consultations (range 11–24 cases per 1,000 consultations) (Figure 1).

ILI swab testing continued during 2012. From the beginning of 2012 to the end of week 39, 1,251 cases of influenza had been detected, the majority of these being influenza A (untyped) (28% of all swabs performed), influenza B (12% of all swabs performed) and the remainder H1N1(2009) (0.5% of all swabs performed) (Figure 2).

During this reporting period, consultation rates for gastroenteritis averaged 4 cases per 1,000 consultations (range 3–5 cases per 1,000 consultations, Figure 3). This was lower than the same reporting period in 2011 where the average was 5 cases per 1,000 consultations (range 4–7 cases per 1,000 consultations).

Varicella infections were reported at a lower rate for the third quarter of 2012 compared with the same period in 2011. From 1 July to 30 September 2012, recorded rates for chickenpox averaged 0.2 cases per 1,000 consultations (range 0–0.6 cases per 1,000 consultations, Figure 4).

In the third quarter of 2012, reported rates for shingles averaged 0.6 cases per 1,000 consultations (range 0.3–0.9 cases per 1,000 consultations, Figure 5), slightly lower than the same reporting period in 2011 where the average shingles rate was 0.8 case per 1,000 consultations (range 0.5–1.3 cases per 1,000 consultations).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 2011 to 2012, by week of report

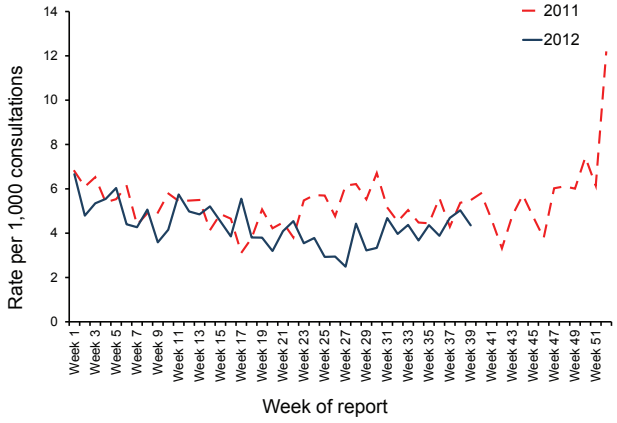


Figure 4: Consultation rates for chickenpox, ASPREN, 2011 to 2012, by week of report

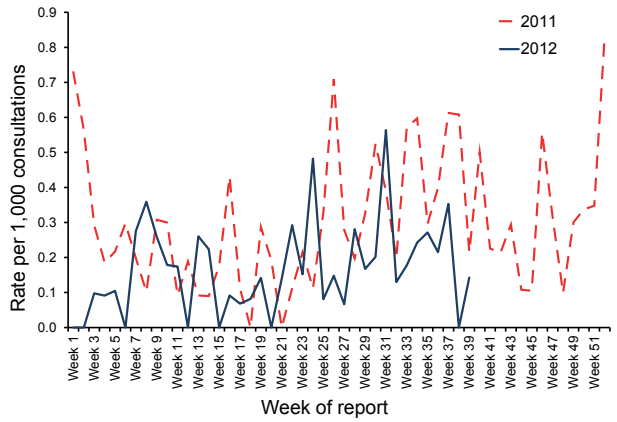
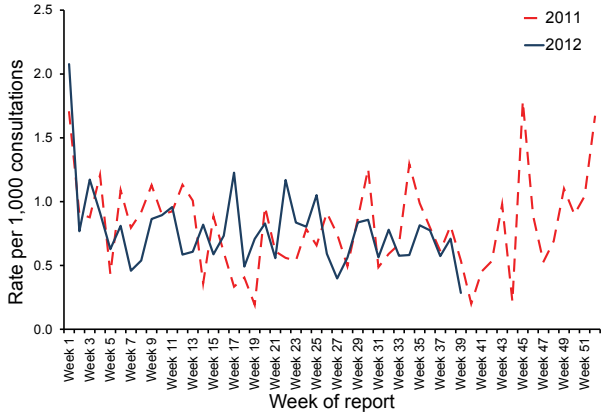


Figure 5: Consultation rates for shingles, ASPREN, 2011 to 2012, by week of report



HIV and AIDS surveillance

Introduction

National surveillance for HIV disease is coordinated by the Kirby Institute, in collaboration with state and territory health authorities and the Australian Government Department of Health and Ageing. 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). Cases of AIDS are notified through the state and territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Tabulations of diagnoses of HIV infection and AIDS 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 and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in '*HIV, viral hepatitis and sexually transmissible infections in Australia, Annual Surveillance Report*'. The reports are available from the Kirby Institute, CFI Building, Cnr Boundary and West Streets, Darlinghurst NSW 2010. Internet: http://www.kirby.unsw.edu.au/ Telephone: +61 2 9385 0900. Facsimile: +61 2 9385 0920. For more information see *Commun Dis Intell* 2012;36(1):123.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 January to 31 March 2012, are included in this report (Tables 1 and 2).

Table 1: Number of new diagnoses of HIV infection and AIDS, and deaths following AIDS occurring in the period 1 January to 31 March 2012, by sex and state or territory of diagnosis

		State or territory								Totals for Australia			
	Sex	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	This period 2012	This period 2011	YTD 2012	YTD 2011
HIV diagnoses	Female	0	6	3	11	2	0	8	10	40	25	40	25
	Male	0	86	3	58	6	3	78	29	263	263	263	263
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	0	92	6	69	8	3	86	39	303	288	303	288
AIDS diagnoses	Female	0	0	1	0	0	0	0	0	1	5	1	5
	Male	0	1	1	3	0	2	4	6	17	26	17	26
	Total	0	1	2	3	0	2	4	6	18	31	18	31
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	1	0	1
	Male	0	2	0	0	0	0	0	0	2	6	2	6
	Total	0	2	0	0	0	0	0	0	2	7	2	7

* Totals include people whose sex was reported as transgender.

Table 2: Cumulative diagnoses of HIV infection and AIDS, since the introduction of HIV antibody testing in 1985 and deaths following AIDS since 1981 to 31 March 2012, by sex and state or territory of diagnosis

		State or territory								
	Sex	ACT	NSW	NT	QLD	SA	TAS	VIC	WA	Australia
HIV diagnoses	Female	40	1,081	35	436	149	26	541	343	2,651
	Male	302	15,279	174	3,703	1,159	155	6,629	1,575	28,976
	Not reported	0	227	0	0	0	0	22	0	249
	Total*	342	16,622	209	4,148	1,309	181	7,217	1,925	31,953
AIDS diagnoses	Female	10	289	7	81	33	4	136	51	611
	Male	95	5,673	54	1,120	428	60	2,256	481	10,167
	Total*	105	5,981	61	1,203	462	64	2,405	534	10,815
AIDS deaths	Female	7	144	1	44	20	2	67	30	315
	Male	73	3,626	33	687	281	34	1,472	301	6,507
	Total*	80	3,781	34	733	301	36	1,548	332	6,845

* Totals include 77 HIV diagnoses, 37 AIDS diagnoses and 23 deaths in people whose sex was reported as transgender.

Administration

Communicable Diseases Intelligence
INSTRUCTIONS FOR AUTHORS

Communicable Diseases Intelligence (CDI) is published quarterly (March, June, September and December) by the Health Emergency Management Branch, Office of Health Protection, Australian Government Department of Health and Ageing.

The aim of CDI is to disseminate information on the epidemiology of communicable disease in Australia, including surveillance, prevention and control.

The objectives of CDI are:

- to report on surveillance of communicable diseases of relevance to Australia;
- to publish other articles relevant to communicable disease epidemiology in Australia; and
- to provide information on other activities relevant to the surveillance, prevention and control of communicable disease in Australia.

CDI invites contributions dealing with any aspect of communicable disease epidemiology, surveillance, prevention or control in Australia. Submissions can be in the form of original articles, short reports, or letters to the editor.

CDI publish guidelines and position papers from the Communicable Diseases Network Australia (CDNA) and its expert sub-committees and may invite guest editorials and review articles on occasion.

Manuscripts for submission

Manuscripts submitted to CDI must be offered exclusively to the journal. All manuscripts should be accompanied by a covering letter that should include:

- a list of all authors;
- confirmation that the manuscript content (in part or in full) has not been submitted or published elsewhere; and
- whether the manuscript is being submitted as an article, short report, surveillance summary, outbreak report or case report.

In addition, manuscripts should include a title page that should contain the following information:

- title (e.g. Prof, Dr, Ms, Miss, Mrs, Mr), full name including middle initial, position held, and institution at the time the article was produced, of each author;
- name of corresponding author, including current postal address, telephone, and email; and
- word count of the main text and of the abstract.

On receipt of a manuscript, authors will be sent a brief acknowledgment. Accepted manuscripts are edited for style and clarity and final proofs are returned to the corresponding author for checking prior to printing.

Authorship

Authorship should be based on substantial contribution to the article. Each author should have participated sufficiently to take public responsibility for the article. Others contributing to the work should be recognised in the acknowledgments.

Types of manuscript

Original Articles

The text of articles must be structured to contain an abstract, introduction, methods, results, discussion, acknowledgments and references. Manuscripts submitted as articles must be 3,000 words or less and will be peer-reviewed.

Original articles may be submitted at any time and will be included in an issue once their review and revision has been completed. Articles may be published ahead of the scheduled issue, in the "early release" format.

Systematic reviews submitted to CDI will be expected to conform to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. Refer to: http://www.prisma-statement.org/

Short reports

Short reports may be submitted for peer review or for publication without peer review, depending on the content. Articles of particular relevance for rapid dissemination (such as timely outbreak reports)

may be fast-tracked for early release prior to the next issue of CDI. Please discuss your requirements with the editorial team. Short reports may include an unstructured abstract. Types of short reports include:

Surveillance summaries

A report of 1,000 words or less that briefly reports on changes in the local epidemiology of a communicable disease, changes in surveillance systems, or new interventions, such as introducing vaccination in an at-risk group. Surveillance summaries should provide a brief description of the setting and a discussion of the significance of the events, changes or interventions.

Outbreak reports

Reports of communicable disease outbreaks of 500 to 1,000 words will be considered for publication based on their public health significance. Reports should include details of the investigation, including results of interventions and the significance of the outbreak for public health practice. More comprehensive reports on outbreaks should be submitted as articles.

Case reports

Brief reports of 500 to 1,000 words on unique cases of communicable disease will be considered based on their public health significance. Authors must note the instructions on the protection of patient's right to privacy (see Ethics committee approvals and patients' right to privacy below). Some discussion of the significance of the case for communicable disease control should be included.

Letters to the Editor

The editorial team welcome comments on articles published in CDI in the form of letters to the Editor. Letters should normally be less than 500 words, include no more than a single chart and less than six references.

Peer review process

Articles provisionally accepted for publication undergo a peer review process and articles may be rejected without peer review. Short reports may be submitted for peer review, or may be reviewed at the discretion of the Editor. Articles will be subject to review by two experts in the field and short reports by one or two reviewers.

When submitting your manuscript, you may specify reviewers who are qualified to referee the work, who are not close colleagues and who would not have a conflict of interest. Suggestions regarding reviewers will be considered, however, the editorial team have the final decision as to who to invite to review a particular article.

Authors may be asked to revise articles as a result of the review process before the final decision about publication is made by the Editor. Revised articles are to be returned with a covering letter addressing each comment made by each reviewer.

Annual reports and quarterly reports are not subject to peer review.

Document preparation

Articles and reports must be written in clear, comprehensible English. Authors should pay particular attention to the style guides, web accessibility requirements and table and figure formatting requirements provided on these pages.

Articles are only accepted in electronic form, in Microsoft Word and Microsoft Excel. Graphics may be provided in a range of other formats (see section below on illustrations). In addition:

- Arial font is preferred but if not available use Times New Roman.
- Abstracts should not exceed 250 words. Do not cite references in abstracts.
- Structured abstracts are acceptable for original articles only.
- Include up to 10 keywords.
- Avoid too many abbreviations.
- Do not use numbered paragraphs.
- Do not use page numbering.
- Do not use headers or footers.

Final manuscripts should not include any field codes such as automatic numbering for references. Electronic referencing software (e.g. Endnote) field codes should be embedded before submission of the final version.

Manuscripts should be submitted with a one or two sentence summary of the article.

A list of styles and conventions for CDI can be found here (link through to CDI style guide).

Tables

Tables and table headings should be located within the body of the manuscript and all tables should be referred to within the results section.

Information in tables should not be duplicated in the text.

Headings should be brief.

Simplify the information as much as possible, keeping the number of columns to a minimum.

Separate rows or columns are to be used for each information type (e.g. percentage and number should be in separate columns rather than having one in parentheses in the same column).

If abbreviations are used these should be explained in a footnote.

Footnotes should use the following symbols in sequence:

* † ‡ § || ¶ ** †† ‡‡

Do not use borders, or blank rows or blank columns for spacing.

A short summary of each table should be included to satisfy government accessibility requirements (see section on accessibility requirements).

Figures and illustrations

Figures and illustrations, including headings, should be provided in the body of the manuscript and should be referred to within the results section. They should also be provided as a separate file.

Examples of each of the following can be found in the on-line version of Instructions to authors at: http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-pubs-cdi-auth_inst.htm

A short summary should be included to satisfy government accessibility requirements (see accessibility requirements).

Figures

Use Microsoft Excel for Windows.

Each figure should be created on a separate worksheet rather than as an object in the datasheet (use the 'as new sheet' option for chart location).

The numerical data used to create each figure must be included on a separate worksheet.

Worksheets should be appropriately titled to distinguish each graph.

Do not include the graph heading on the Excel worksheet.

Further information on formatting figures is provided in the formatting for figures in CDI section.

Illustrations

Illustrations or flow charts can be included if required.

Images should preferably be at least 300 dpi.

Electronic copies of computer-generated illustrations should preferably be saved in a vector image program such as Adobe Illustrator but other similar graphic software is acceptable. Files should be saved in one of the following graphic formats (in preferential order):

- AI,
- TIFF,
- EPS, or
- GIF.

Use a sans serif font for figures (e.g. arial). Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

Photographs

Photographs may be submitted if required.

Photos need to be at least 300 dpi.

Electronic copies should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats (in preferential order):

- PSD,
- TIFF,
- EPS,
- AI, or
- JPEG (JPG).

Maps

Electronic copies of maps should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats (in preferential order):

- PSD,
- TIFF,
- EPS, or
- GIF.

Thermal maps created by mapping programs such as MapInfo or Arc GIS should be saved at 300 dpi and in one of the following graphic formats (in preferential order):

- TIFF,
- EPS, or
- JPEG (JPG).

Authors should aim for maximum levels of contrast between shaded areas. Use a sans serif font for text. Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

Web Accessibility Requirements

The Australian Government is now required to meet new web site accessibility guidelines, the Web Content Accessibility Guidelines version 2.0 (WCAG 2.0). These guidelines include the need for alternate methods of presenting the information depicted in images - including figures and maps - for vision impaired people using text readers. Complex tables also present challenges for text readers.

Articles and reports should be submitted with:

- a short summary of any tables
- a long text description of any figures
- a long text description of any maps, flowcharts, or other images. For thermal maps showing disease rates by statistical location, a data table may be a preferred alternative.

Samples of descriptors for tables and figures can be found here (link through to examples page)

Further information about WCAG 2.0 is available from the Australian Government Information Management Office (http://agimo.gov.au/)

References

References should be identified consecutively in the text by the use of superscript numbers without brackets. Any punctuation should precede the reference indicators.

The accuracy of references is the responsibility of authors. Use the Vancouver reference style (see International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. Ann Intern Med 1997;1126:36–47 available from: http://www.nlm.nih.gov/bsd/uniform_requirements.html) and abbreviate journal names as in Medline (e.g. Commun Dis Intell). The Medline journal database is available from: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=journals. Include the surnames and initials of all authors (or only the first six authors, et al, if there are more than six). Cite the first and last page numbers in full, and specify the type of reference (e.g. a letter, an editorial, an abstract, or supplement).

Cite personal communications and unpublished papers in the text, not in the reference list, with the exception of material that has been accepted for publication (in press). Obtain written permission from people cited, and include their title, position and affiliation.

More detail about reference styles and examples is included on the CDI referencing style page.

Ethics committee approvals and patients' rights to privacy

All investigations on human subjects must include a statement that the subjects gave their written informed consent, unless data collection was covered by public health legislation or similar studies have been considered by a relevant ethics committee and a decision made that its approval was not required. The name of the ethics committee that gave approval for the study should be included in the text. Alternatively, if approval is not required a statement to this effect should appear in the manuscript.

When informed consent has been obtained this should be included in the text.

Ethical approval and patient consent may also be required for case reports. Identifying details about patients should be omitted if they are not essential, but data should never be altered or falsified in an attempt to attain anonymity.

Copyright

All authors are asked to transfer copyright to the Commonwealth before publication. A copyright form will be sent to the corresponding author. All authors are required to sign the copyright release. The Commonwealth copyright will be rescinded if the article is not accepted for publication.

Submission of manuscripts

Manuscripts should be provided electronically by email to: cdi.editor@health.gov.au

Please contact the editorial team at cdi.editor@health.gov.au if you require any further information.

Revised 21 January 2013.