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Annual reports

Annual report of the Australian National Enterovirus Reference Laboratory 2012

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Abstract

In 2012 no cases of poliomyelitis were reported through clinical surveillance in Australia, and poliovirus was not detected through virological surveillance. Australia conducts surveillance for cases of acute flaccid paralysis (AFP) in children less than 15 years as the main mechanism to monitor its polio-free status in accordance with World Health Organization (WHO) recommendations. Cases of AFP in children are notified to the Australian Paediatric Surveillance Unit or the Paediatric Active Enhanced Disease Surveillance System. In 2012 Australia reported 1.2 non-polio AFP cases per 100,000 children, meeting the WHO performance criterion for a sensitive system for the fifth year in a row. However the faecal specimen collection rate from AFP cases was 29%, which was well below the WHO target of 80%. Virological surveillance for poliovirus consists of two components. Firstly, the Enterovirus Reference Laboratory Network of Australia (ERLNA) reports on the typing of enteroviruses detected in or isolated from clinical specimens. Secondly, environmental surveillance is conducted at sentinel sites. These surveillance systems are co-ordinated by the National Enterovirus Reference Laboratory (NERL).

Keywords: poliovirus, acute flaccid paralysis, surveillance, enterovirus, poliomyelitis, eradication, vaccination

Introduction

Australia, along with the World Health Organization (WHO) Western Pacific Region, was declared polio-free in 2000 and has established clinical and virological surveillance schemes to monitor its polio-free status. Clinical surveillance follows the WHO recommendation of investigating cases of acute flaccid paralysis (AFP) in children less than 15 years of age. AFP cases are ascertained either by clinicians notifying the Australian Paediatric Surveillance Unit (APSU) via a monthly report card or through the Paediatric Active Enhanced Disease Surveillance System (PAEDS). PAEDS involves ward based nurses reviewing hospital records and enrolling AFP patients with the consent of a parent or guardian at four sentinel tertiary paediatric hospitals.[1,2] The WHO recommends that two faecal specimens be collected for virological investigation at least 24 hours apart and within 14 days of the onset of paralysis from cases of AFP in order to exclude poliovirus as the causative agent. It is a requirement of the WHO polio eradication program that specimens are tested in a WHO accredited laboratory, which for Australia is the National Enterovirus Reference Laboratory (NERL), formerly the National Polio Reference Laboratory (NPRL) at the Victorian Infectious Diseases Reference Laboratory (VIDRL). The clinical and laboratory data from AFP cases in children is reviewed by the Polio Expert Panel (PEP) and reported to the WHO as evidence of Australia's continued polio-free status.

Enterovirus and environmental surveillance programs were established as virological surveillance for poliovirus to complement the clinical surveillance program focussed on AFP cases in children. Enteroviruses other than poliovirus have been associated with AFP, and poliovirus infection may manifest clinically without paralysis. The Enterovirus Reference Laboratory Network of Australia (ERLNA) was established in 2009. Public diagnostic virology laboratories report their enterovirus typing results from clinical specimens to exclude poliovirus and establish the epidemiology of non-polio enteroviruses in Australia. WHO supports environmental surveillance as a sensitive means of detecting poliovirus through the testing of sewage samples. In December 2012, Egypt reported the detection of wild poliovirus type 1 in 2 sewage samples collected in Cairo.[3] Genetic sequencing identified Pakistan as the source of the viruses.

The certification of India as being polio-free in January 2012 was a significant achievement for the global polio eradication program, reducing the number of endemic countries to three; Afghanistan, Nigeria and Pakistan.[4] Furthermore, the reporting of 223 polio cases globally in 2012 represented the lowest number since the eradication program started in 1988.[5] Nevertheless, it is important to maintain high polio vaccine coverage and sensitive surveillance systems for AFP cases in children until global eradication is achieved. As an example, China along with other countries of the Western Pacific Region, was declared polio-free in 2000. However, an outbreak in Xinjiang province in China due to a wild poliovirus type 1 importation from Pakistan caused 21 cases of polio before the country was declared polio-free once again in October 2012.[4] A weekly situ-

ation report of polio cases worldwide is available at the WHO website http://www.polioeradication.org/Dataandmonitoring/Poliothisweek.aspx

This report summarises the polio surveillance program in Australia for 2012 encompassing clinical surveillance for AFP cases in children and virological surveillance for poliovirus.

Methods

AFP Surveillance

Paediatricians reviewing a patient less than 15 years of age presenting with AFP, or clinicians reviewing a patient of any age suspected of poliomyelitis, are requested to notify the NERL (telephone 03-9342 2607, email polio@mh.org.au). Paediatricians also notify the AFP case to the APSU (http://www.apsu.org.au/) via a monthly report card. Upon receipt of the notification, the AFP National Surveillance Co-ordinator based at VIDRL forwards a clinical questionnaire for the clinician to complete. Alternatively, AFP cases are ascertained by PAEDS nursing staff from medical records and are enrolled in the surveillance program with parental or guardian consent.

WHO classifies specimens as being adequate for virological investigation when two faecal specimens are collected more than 24 hours apart (due to intermittent virus shedding), and the specimens are collected within 14 days of the onset of paralysis (while the virus titre remains high). The faecal specimens are tested free of charge by the NERL.

The PEP, convened by the Department of Health (DoH), reviews the clinical and laboratory data for all notified cases of AFP, irrespective of whether they are an eligible or ineligible case. An eligible case is an Australian child under 15 years of age with AFP (including Guillain-Barrè syndrome and transverse myelitis) or an Australian of any age with suspected polio. Ineligible cases include patients aged 15 years or older, overseas residents and cases notified in error or later determined not to be AFP.

The PEP classifies cases of AFP as:

- Poliomyelitis due to wild poliovirus, vaccine-derived poliovirus (VDPV) or vaccine associated paralytic poliomyelitis (VAPP);
- Polio compatible if there is insufficient evidence to exclude poliomyelitis;
- Non-polio AFP; or
- Non-AFP.

A follow-up questionnaire is sent to notifying clinicians if the PEP requires more information regarding the AFP case before a final classification can be made. After each PEP meeting the Australian AFP case classifications are forwarded to WHO for inclusion in the global AFP surveillance data published in the Weekly Epidemiological Record (available at http://www.who.int/wer/en/). Ineligible cases are not reported to WHO.

The WHO AFP surveillance performance indicator for a polio non-endemic country is one case of non-polio AFP per 100,000 children aged less than 15 years. For Australia in 2012, this equated to 43 cases per year, based on the Australian Bureau of Statistics data released in December 2011. An AFP surveillance scheme that satisfies the surveillance performance indicator is deemed sufficiently sensitive to detect a wild poliovirus importation in children of that country. The WHO surveillance performance indicator for laboratory testing is that at least 80% of notified AFP cases have adequate faecal specimens collected and tested in a WHO accredited laboratory.

At the end of each calendar year a number of AFP notifications remain pending where insufficient clinical and laboratory data were made available to the PEP. The PEP classifies the remaining AFP notifications as "polio compatible-zero evidence" if a final review reveals no evidence of clustering amongst the cases.

Virus Culture

Upon receipt at the NERL, faecal specimens are treated with Minimum Essential Medium containing Hank's salts, chloroform (9.1% v/v) and foetal bovine serum (2%). The suspension is clarified and the supernatant inoculated onto a series of mammalian cell lines. Two WHO recommended cell lines are used for the isolation of poliovirus, L20B (a transgenic mouse epithelial cell line expressing the human poliovirus receptor, CD155) and RD-A (human rhabdomyosarcoma).[6,7] Diagnostic laboratories in Australia are encouraged to refer enteroviruses of unknown serotype to the NERL for further characterisation as poliovirus infection can lead to clinical presentations without paralysis such as aseptic meningitis.

Two WHO real time reverse transcription polymerase chain reaction (RT-PCR) tests are used to determine whether a poliovirus is a wild strain, oral poliomyelitis vaccine (OPV) strain (Sabin-like) or a vaccine-derived poliovirus (VDPV), in a process known as intratypic differentiation.[8] The NERL sequences the complete poliovirus viral protein 1 (VP1) genomic region, which contains a major neutralizing antibody binding site. The VP1 genomic sequence provides valuable biological information, including the number of mutations within a signifi-

cant region of the OPV virus strain and it enables phylogenetic analysis of wild poliovirus to rapidly determine the likely source of the virus, as utilised in the 2007 wild poliovirus importation.[9]

Enterovirus Surveillance

The ERLNA was established primarily as a means of detecting imported poliovirus amongst untyped enteroviruses from clinical specimens. The network consists of 10 public sector diagnostic virology laboratories in the Australian Capital Territory (Canberra Hospital), New South Wales (Royal Prince Alfred Hospital), Queensland (Queensland Health and Scientific Services), South Australia (Flinders Medical Centre, Institute of Medical and Veterinary Science), Tasmania (Royal Hobart Hospital), Victoria (Royal Children's Hospital, VIDRL) and Western Australia (Queen Elizabeth II Medical Centre, Princess Margaret Hospital for Children).

The NERL encourages members of the ERLNA to perform their own enterovirus typing. It has advised members of the ERLNA on enterovirus detection, supplied laboratory and computer analysis protocols and performed tests in parallel with other laboratories for quality assurance purposes. The NERL receives untyped enteroviruses from three laboratories for typing on a regular basis. The other laboratories perform their own enterovirus typing and report the results to the NERL for inclusion in the national enterovirus database.

The NERL screens clinical specimens for enteroviruses using a semi-nested RT-PCR directed to highly conserved sequence in the 5' non-translated region (NTR).[10] Enterovirus typing is primarily performed by amplifying a fragment of the VP1 genomic region according to a published method,[11] but the complete nucleotide sequence of VP1 is required to type some enteroviruses. The enterovirus typing RT-PCR is directed to a region of sequence divergence that allows differentiation between enterovirus genomes. As a consequence, the enterovirus sequence based typing assay is not as sensitive as the pan-enterovirus detection assay. This can result in an enterovirus being detected by pan-enterovirus RT-PCR in a clinical specimen without subsequent identification by the VP1 enterovirus typing assay.

Environmental surveillance

The laboratory cell culture protocol implemented by the NERL for environmental surveillance is based on a two-phase separation procedure published by WHO and further advice was obtained from the Enterovirus Laboratory at the National Public Health Institute,[12] Finland, a Global Specialised Laboratory in the WHO Polio Laboratory Network. In brief, 800 mL of sewage is collected prior to any biological or chemical treatment and referred to the NERL within 24 hours. At the laboratory 500 mL of the sample is centrifuged and the supernatant vigorously shaken at 4°C with dextran, polyethylene glycol and sodium chloride. The mixture is incubated overnight at 4°C in a separating funnel and the lower organic phase is collected the next day and used to re-suspend any pellet stored after the initial centrifugation. The final solution is clarified as for a faecal specimen and inoculated onto the L20B and RD-A cell lines and observed microscopically for cytopathic effect. The sewage extracts are tested in parallel by cell culture and a pan-enterovirus RT-PCR. The pan-enterovirus RT-PCR is a validated in-house test and is utilised to confirm the cell culture results as not all human enteroviruses can infect the RD-A cell line. All enterovirus isolates from cell culture and positive detections by RT-PCR were investigated to determine the virus type by nucleic acid sequencing.

Results

Classification of AFP cases

A total of 77 notifications of AFP in children less than 15 years of age were received in 2012 (Table 1). The PEP classified 51 cases as non-polio AFP with onset of paralysis in 2012. This equated to a non-polio AFP rate of 1.2 cases per 100,000 children less than 15 years of age, exceeding the WHO AFP surveillance performance criterion for a polio-free country of one case of non-polio AFP per 100,000 children (Table 2, Figure 1).

In 2012, one AFP case reviewed by the PEP had a differential diagnosis of Guillain-Barré syndrome and anterior horn cell disease due to an enteroviral infection. One faecal specimen, collected eight days after the onset of paralysis, was reported as no enterovirus isolated by cell culture by the NERL. The patient had not travelled overseas in the three months preceding the onset of symptoms. The PEP was not able to exclude polio based on the available clinical evidence and classified the case as "polio compatible" (Table 1). No further clinical information or laboratory specimens were received from one other AFP notification and the PEP classified the case as "polio compatible – zero evidence" to indicate the fact that it was a notification only with no further evidence to support a clinical diagnosis of polio.

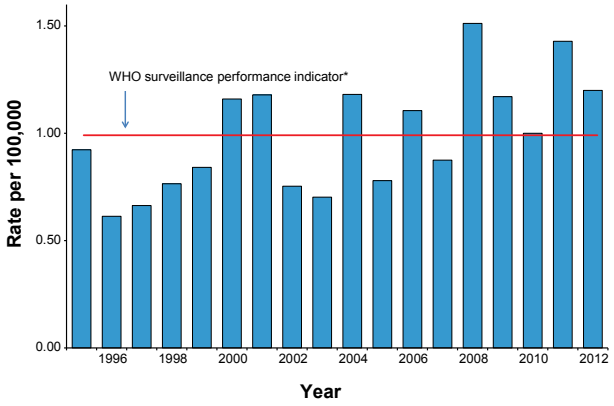
Thirteen AFP cases were notified by more than one clinician and were regarded as duplicate notifications (Table 1). Eight AFP notifications did not meet the criteria for an eligible case. These involved either patients greater than 14 years of age, cases with symptom onset prior to 2012, or cases that were later reported as non-AFP. Three cases involving patients older than 14 years of age were all classified

by the PEP as non-polio AFP. However, they were not reported to the WHO as the global polio surveillance program focuses on AFP in children less than 15 years of age as the age group being at high risk of poliovirus infection.

Notification of AFP cases by state and territory

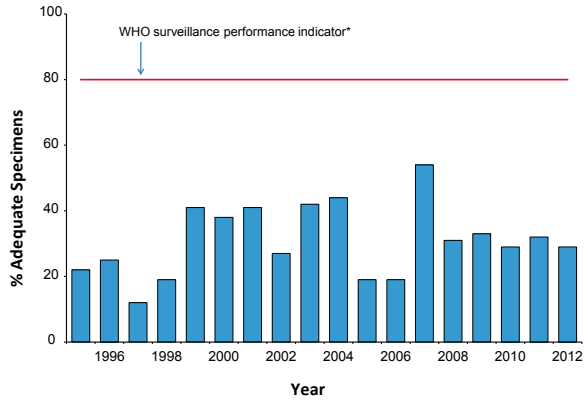
In 2012, eligible AFP cases were notified from all jurisdictions in Australia except the Australian Capital Territory and Tasmania (Table 1). The non-polio AFP rates for eligible cases per jurisdiction exceeded the WHO AFP surveillance performance indicator of one case per 100,000 children in New South Wales, Northern Territory, South Australia, Victoria and Western Australia. Queensland was the only more populous state not to have achieved the WHO surveillance performance indicator.

Figure 1: Non-polio AFP rate classsified by the PEP, 1995 to 2012



* The WHO AFP surveillance performance indicator for a polio non-endemic country is one case per 100,000 children <15 years of age.

Figure 2: Percentage of AFP cases with adequate faecal specimens, 1995 to 2012



* The criterion for the WHO surveillance performance indicator is the collection of two faecal specimens more than 24 hours apart and within 14 days of the onset of paralysis from 80% of classified non-polio AFP cases.

Faecal collection from AFP cases

In 2012, a total of 64 faecal specimens from 37 of the 51 eligible cases were tested at the NERL (Table 3). No poliovirus was isolated from any of the specimens. The non-polio enteroviruses, coxsackievirus A7, coxsackievirus A16, coxsackievirus B5 and enterovirus 71 subgenogroup C2 were reported from four AFP cases in 2012. Diagnoses were transverse myelitis for the first two cases, Guillain-Barré syndrome for the third and acute disseminated encephalomyelitis for the last. Fifteen (29%) of the eligible cases had adequate specimens collected in 2012, while another 12 (24%) cases had only one specimen collected within the optimal period. This compares to the further WHO AFP surveillance criterion that 80% of the eligible AFP cases should have adequate specimens collected, a result that Australia has never achieved nationally (Figure 2). At the jurisdictional level, Queensland was the only state to reach the WHO target in 2012, with adequate specimens collected from all five cases classified (100%).

Enterovirus and environmental surveillance

No poliovirus was detected by enterovirus or environmental surveillance in 2012. The ERLNA typed 277 non-polio enteroviruses with coxsackievirus A6 and echovirus 18 amongst the most frequent detections in Australia during the year (Table 4).

Four collections from each of the three sentinel sites (Armidale, Byron Bay and Newcastle) were tested by RT-PCR, and virus isolation. Twelve collections (four from each site) were tested by cell culture and RT-PCR. All 12 samples were positive by pan-enterovirus RT-PCR and non-polio enterovirus was isolated in cell culture from eight samples. Four samples positive by pan-enterovirus RT-PCR could not be typed due to low virus titres.

Regional reference laboratory activities

Several activities were performed as a Polio Regional Reference Laboratory in 2012. Specimens from AFP cases were referred from Brunei Darussalam (2 cases), Pacific Island countries (6 cases) and Papua New Guinea (9 cases). No poliovirus was isolated from any of the specimens but non-polio enteroviruses were reported from one case from the Pacific Islands and 5 cases from Papua New Guinea.

Six poliovirus type 2 and eight poliovirus type 3 isolates were referred from AFP cases in the Philippines for intratypic differentiation and all were character-

Table 1: Notification of AFP cases in Australia, 2012, by state or territory

State/ Territory	Estimated population aged <15 years*	Expected number of AFP cases†	Total number of AFP cases	Ineligible notifications	Duplicate notifications	Polio compatible-zero evidence	Polio compatible	Pending	Eligible cases with final classification by PEP	Non-polio AFP rate per 100,000 children
ACT	67,397	0.5	0	0	0	0	0	0	0	0.0
NSW	1,358,279	14	21	1	2	0	1	1	16	1.1
NT	52,749	0.5	2	0	0	0	0	0	2	4.0
QLD	909,482	9	14	2	4	1	0	2	5	0.6
SA	293,392	3	5	0	0	0	0	0	5	1.7
TAS	97,694	1	1	1	0	0	0	0	0	0.0
VIC	1,027,417	10	28	4	6	0	0	0	18	1.8
WA	453,747	5	6	0	1	0	0	0	5	1.0
Australia	4,260,157	43	77	8	13	1	1	3	51	1.2

* Australian Bureau of Statistics, estimated population at 30 June 2011. Available at http://www.abs.gov.au/

† Based on a non-polio AFP rate of 1 case per 100,000 children less than 15 years of age

Table 2: Surveillance for AFP cases in children less than 15 years, Australia, 2012, compared with the WHO performance indicators

WHO surveillance performance indicator for AFP cases in children <15 years	Performance of Australia's AFP surveillance	
	Number of cases/specimens 2012	Comparison with WHO indicator 2012
AFP cases		
≥1.0 non-polio AFP case / 100,000 children (43 cases for Australia in 2012).	51 cases classified as non-polio AFP	1.2 (51 / 43) non-polio AFP cases / 100,000 children <15 years
Adequate specimen collection		
≥80% of classified AFP cases with adequate specimens* (41 cases for Australia in 2012).	15 AFP cases with adequate specimens collected	29% (15 / 51) classified non-polio AFP cases with adequate specimens

* Adequate specimen collection is defined as 2 faecal specimens collected at least 24 hours apart and within 14 days of onset of paralysis

ised as being Sabin-like. Seven poliovirus type 1 and two poliovirus type 2 isolates were characterised as Sabin-like from sources other than AFP.

Quality Assurance Programs

In 2012, the NERL passed the annual WHO quality assurance panels for poliovirus isolation by cell culture and poliovirus RT-PCR for intratypic differentation and vaccine derived poliovirus. The WHO distributed the first official poliovirus sequencing proficiency panel and the laboratory scored full marks for sequencing RNA templates consisting of wild, Sabin and Sabin prototype mixtures of poliovirus. The laboratory also participated in the Royal College of Pathologists of Australasia quality assurance panel for enterovirus detection by RT-PCR.

Table 3: Specimens referred to the NERL Australia, 2012

Result	Specimens from AFP cases in patients < 15 years of age	Specimens from AFP cases in patients ≥15 years of age	Specimens from sources other than AFP	TOTAL
Non-polio enterovirus	5	0	150	155
Rhinovirus	1	0	2	3
No enterovirus identified	58	4	35	97
Total	64	4	187	255

Table 4: Enterovirus test results from the NERL Australia, 1995 to 2012

Year	Poliovirus		Non-polio enterovirus	No enterovirus detected	EVID results referred†	Total samples reviewed
	Sabin-like	Non-Sabin-like*				
1995	190	0	200	13	0	403
1996	224	0	198	9	0	431
1997	124	0	76	0	0	200
1998	52	0	15	4	0	71
1999	60	1	9	9	0	79
2000	45	0	44	47	0	136
2001	46	5	33	75	0	159
2002	36	0	21	49	0	106
2003	9	0	15	47	0	71
2004	6	0	26	61	0	93
2005	18	0	10	39	0	67
2006	2	0	6	71	29	108
2007	0	2	32	115	107	256
2008	0	0	20	92	77	189
2009	1	0	63	78	113	255
2010	0	0	170	39	108	317
2011	0	0	174	61	205	440
2012	0	0	155	97	123	375

* Untyped enterovirus or uncharacterised poliovirus isolates were referred for further testing after completion of a laboratory inventory in 1999 and 2001; the six non-Sabin-like isolates were identified as wild type poliovirus prototype strains and destroyed. Wild poliovirus type 1 was imported from Pakistan in 2007. A Sabin-like poliovirus type 1 was identified from an unimmunised infant in 2009.

† Enterovirus Identification (EVID) results include retrospective data made available via the ERNLA.

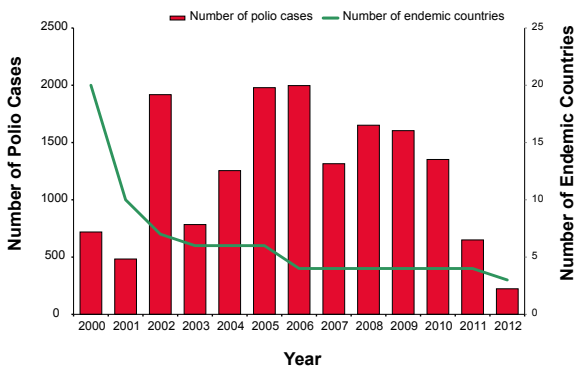
Discussion

In 2012, Australia reached the WHO surveillance target of ≥1 non-polio AFP case per 100,000 children, for the fifth year in a row. The continued participation of clinicians and health care workers in notifying cases of AFP to the APSU and VIDRL along with the involvement of the ward based nurses in the PAEDS is essential in reaching this target, indicative of a sensitive surveillance system. Collection of adequate faecal specimens from AFP cases in Australia has never met the WHO surveillance target and represents a gap in clinical surveillance for imported cases of polio. The establishment of supplementary virological surveillance for poliovirus by the typing of enteroviruses and testing of environmental samples at sentinel sites provides an additional means of monitoring Australia's polio-free status.

Since the initial target for global polio eradication by the year 2000 set by the World Health Assembly (WHA) in 1988, a number of subsequent target years set for achieving polio eradication have not been met, the most recent being the end of 2012.[13] However significant achievements have been attained since 1988, including the last reported case of wild poliovirus type 2 in 1999 and a reduction in the number of polio endemic countries worldwide from 125 to 3 by 2012. However, cases of wild poliovirus have reviewed around 1,200 annually between 2002 and 2010 (Figure 3).[14] After Egypt was certified polio-free in 2006, it has proved difficult to eradicate the virus from the remaining areas of wild poliovirus transmission (Afghanistan, India, Nigeria and Pakistan).

A new strategy was initiated from 2005 with the introduction of monovalent oral polio vaccine for poliovirus type 1 and poliovirus type 3 followed by bivalent oral polio vaccine for poliovirus types 1 and 3 in 2009.[15] The judicious use of trivalent, bivalent and monovalent oral polio vaccines reduced the number of polio cases in the polio endemic countries and those with re-established transmission to 217 and 6, respectively, in 2012.[5] Changes to the WHO laboratory testing protocols were also introduced from 2006 to shorten the time taken to confirm cases of polio.[16] The WHO Global Polio Laboratory Network introduced a new cell culture algorithm that halved the reporting time from 28 days to 14 days and implemented real time RT-PCR protocols that reduced the timeframe for poliovirus intratypic differentiation from 14 days to 7 days.

Figure 3: Cases of wild polio virus infection in endemic countries, 2000 to 2012, by year



Data from: http://www.polioeradication.org/Dataandmonitoring/Poliothisweek/Wildpolioviruslist.aspx, accessed 27 March 2013.

A further response in 2010 was the establishment of an Independent Monitoring Board (IMB) by the WHA to monitor and guide the progress of the Global Polio Eradication Initiative's 2010-2012 Strategic Plan.[17] The IMB was convened quarterly to review developments in the polio program and provided independent advice regarding the requirements for the plan to succeed. The 2010 to 2012 strategic plan aimed to stop wild poliovirus transmission in two of the four endemic countries by the end of 2011, with only one country, India, achieving that goal.[4] Considering that India reported 741 cases of polio as recently as 2009, representing 46% of the cases worldwide, its certification as polio-free in January 2012 was a significant milestone for the eradication program. Despite this, the January 2012 IMB report concluded that global polio eradication would not be achieved if it continued on its current path.[17] One of the board's conclusions was to place a greater emphasis on people management, including rating the importance of having well-trained vaccinators who are valued and inspired as the most important group in the programme. The cessation of polio vaccination in response to the deliberate killing of polio vaccinators in Pakistan in late 2012 reinforced the board's opinion of their key role.[13]

In January 2012, in response to a recommendation by the IMB, the WHO Executive Board called "the completion of poliovirus eradication a programmatic emergency for global public health". This was adopted as a resolution by the WHA the following May.[18] By the end of 2012, 223 cases of polio were reported for the year, the lowest annual total ever (Figure 3). Transmission of wild poliovirus was restricted to four countries, the lowest since the program began; the three endemic countries of Afghanistan, Nigeria and Pakistan, and Chad with re-established transmission.[5] Furthermore, wild poliovirus type 3 was last reported in Afghanistan and Pakistan in April 2012, potentially leaving Nigeria as the last country to be endemic for this serotype.

The Polio Eradication Initiative appears to be at a critical juncture. A concerted international effort is required to support the final stages of wild

poliovirus eradication to avoid a repeat of 2001. This was the last time that relatively few cases were reported, and was followed in subsequent years by a rapid rise in the number of cases in the endemic countries and frequent occurrences of re-established transmission in others. To restrict the international spread of wild poliovirus, the IMB recommended in its seventh report that by May 2013 the International Health Regulations Expert Review Committee issue a standing recommendation that travellers from the remaining endemic countries receive pre-travel vaccination or a check of their vaccination status until polio transmission in the country ceases.[17]

Until certification of global wild poliovirus eradication, Australia remains at risk of an importation as occurred from Pakistan in 2007.[9] The continued performance of the clinical and virological surveillance systems for poliovirus to a high international standard is essential to monitor Australia's polio-free status in order to detect and rapidly respond to any future polio importation event.

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Annual report of the Australian National Enterovirus Reference Laboratory 2010-2011

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Abstract

Australia conducts clinical surveillance for cases of polio-like illness in children in accordance with the World Health Organization (WHO) recommended surveillance criteria for acute flaccid paralysis (AFP). AFP cases are ascertained either by clinicians notifying the Australian Paediatric Surveillance Unit or designated nurses enrolling cases as part of the Paediatric Active Enhanced Disease Surveillance system at four sentinel tertiary paediatric hospitals. The National Enterovirus Reference Laboratory (NERL), formerly the National Poliovirus Reference Laboratory, is accredited by the World Health Organization (WHO) for the testing of faecal specimens from cases of AFP and operates as a Poliovirus Regional Reference Laboratory for the Western Pacific Region. In 2010 and 2011, for the 3rd and 4th consecutive years, Australia met the WHO AFP surveillance performance indicator. This is indicative of a sensitive surveillance system capable of detecting an imported case of polio in children. However, the faecal collection rate for the virological investigation of AFP cases was below the WHO surveillance performance indicator in both years and represented a gap in Australia's polio surveillance. Enterovirus and environmental surveillance were established in Australia as virological surveillance to complement the clinical surveillance schemes. No poliovirus was detected by the clinical or virological surveillance schemes in 2010 or 2011 and Australia maintained its polio-free status. India was declared polio-free in January 2012, a significant step towards global polio eradication, leaving Afghanistan, Nigeria and Pakistan as the remaining countries endemic for wild poliovirus.

Keywords: poliovirus, acute flaccid paralysis, surveillance, enterovirus, poliomyelitis, eradication, vaccination

Introduction

The National Enterovirus Reference Laboratory (NERL), formerly the National Polio Reference Laboratory, is responsible for the virological testing of faecal specimens from cases with a clinical suspicion of poliomyelitis. This includes cases of acute flaccid paralysis (AFP), a major clinical presentation of poliomyelitis, in children less than 15 years of age and cases of suspected poliomyelitis in patients of any age. The World Health Organization (WHO) recommends that two faecal specimens be collected for virological investigation at least 24 hours apart and within 14 days of the onset of paralysis from cases of AFP to exclude poliovirus as the causative agent. It is a requirement of the WHO polio eradication program that the specimens are tested in a WHO accredited laboratory, which for Australia is the NERL at the Victorian Infectious Diseases Reference Laboratory (VIDRL).

Enterovirus and environmental surveillance programs were established to provide virological surveillance for poliovirus to complement the clinical surveillance program focussed on AFP cases in children. Enteroviruses other than poliovirus have been associated with AFP and poliovirus infection may manifest clinically without paralysis. The Enterovirus Reference Laboratory Network of Australia (ERLNA) was established in 2009, bringing together public diagnostic virology laboratories. ERLNA aims to identify the types of enteroviruses detected in clinical specimens to exclude poliovirus and establish the epidemiology of non-polio enteroviruses in Australia. WHO supports environmental surveillance as another aspect of polio surveillance through the testing of sewage samples. Clinical and virological surveillance schemes for poliovirus serve to monitor Australia's polio-free status.

From November 2005, inactivated poliomyelitis vaccine (IPV) replaced oral poliomyelitis vaccine (OPV) in the National Immunisation Program.[1] IPV is administered to children at 2, 4 and 6 months of age, with a booster dose at 4 years of age. With the removal of OPV (containing live attenuated virus) from the immunisation schedule, any poliovirus identified by Australian virology laboratories requires further investigation to determine its origin, as it potentially represents an importation event.

It is important that Australia maintains high levels of polio vaccine coverage to avoid a resurgence of poliomyelitis in the event of a wild poliovirus importation. In 2010, China reported an outbreak of polio due to wild type virus in Xinjiang province, which borders Pakistan and from where the importation originated. A total of 21 cases of polio were reported ranging in age from 6 months to 42 years before the outbreak was controlled through mass immunization programs.[2] The WHO provides a weekly update of the global polio eradication situation including a list of countries reporting cases due to wild poliovirus (http://www.polioeradication.org/Dataandmonitoring/Poliothisweek.aspx).

The Australian Immunisation Handbook recommends that individuals who are at continuing risk of infection, such as health care workers should have a polio vaccine booster every 10 years.[1]

The last wild poliovirus isolated in India was in January 2011. The country was declared polio-free 12 months later reducing the number of countries that have never interrupted wild poliovirus transmission to three; Afghanistan, Nigeria and Pakistan.[2] It was only in 2009 that India reported 741 cases of polio due to wild type poliovirus which accounted for 46% of the cases worldwide. India's polio-free status is a significant public health achievement that supports the feasibility of the global polio eradication strategy.

This report summarises the polio surveillance program in Australia for 2010 and 2011, encompassing AFP surveillance in children and virological surveillance.

Methods

AFP Surveillance

AFP surveillance was initiated by the Australian Government in 1995 in collaboration with the Australian Paediatric Surveillance Unit (APSU) as part of Australia's commitment to the WHO poliomyelitis eradication program. Since 2000, AFP surveillance has been co-ordinated by VIDRL in collaboration with the APSU. In late 2007, the Paediatric Active Enhanced Disease Surveillance (PAEDS) surveillance scheme was established as a collaboration between the APSU and the National Centre of Immunisation Research and Surveillance. PAEDS is a hospital based surveillance system for paediatric conditions of public health interest, including AFP, at four tertiary paediatric hospitals in Adelaide, Melbourne, Perth and Sydney.[3] In April 2011 the Polo Expert Committee which is responsible for reviewing AFP cases to determine if they are compatible with polio was renamed the Polio Expert Panel (PEP) by the Communicable Disease Network Australia (CDNA).

The strategy adopted for AFP surveillance is as follows:

- Paediatricians reviewing a patient less than 15 years of age who presents with AFP, or clinicians reviewing a patient of any age suspected of poliomyelitis, are requested to notify the NERL (telephone 03 9342 2607, email polio@mh.org.au). Notification of the AFP case is also included on the paediatrician's monthly report card to the APSU (http://www.apsu.org.au/). Upon receipt of the notification the AFP National Surveillance Co-ordinator, based at VIDRL, forwards a clinical questionnaire for the clinician to complete.
- Alternatively, AFP cases are ascertained by PAEDS nursing staff from medical records and with parental agreement are enrolled in the surveillance system.
- Two faecal specimens are collected 24 to 48 hours apart and within 14 days of onset of paralysis. The collection of specimens within these time frames enables them to be classified as adequate by WHO.
- The faecal specimens are tested free of charge by the NERL, which is accredited by WHO for this purpose.
- The PEP which is convened by the Department of Health (DoH), reviews the clinical and laboratory data for all notified cases of AFP, irrespective of whether they are an eligible or ineligible case. An eligible case is: an Australian child under 15 years of age with AFP (including Guillain-Barré syndrome) or an Australian of any age with paralytic illness if polio is suspected. Examples of ineligible cases are where the patient is aged 15 years or older, an overseas resident and cases notified in error or later determined to be non-AFP. The PEP classifies cases of AFP as:
    - Poliomyelitis due to wild poliovirus, vaccine-derived poliovirus (VDPV) or vaccine associated paralytic poliomyelitis (VAPP)
    - Non-polio AFP or
    - Non-AFP.

A follow-up questionnaire is sent to notifying clinicians if the PEP requires more information regarding the AFP case before a final classification can be made.

After each PEP meeting the Australian AFP data is forwarded to WHO for inclusion in the global AFP surveillance data published in the Weekly Epidemiological Record (available at http://www.who.int/wer/en/). Ineligible cases are not reported to WHO.

The WHO AFP surveillance performance indicator for a polio non-endemic country is 1 case of non-polio AFP per 100,000 children aged less than 15 years each year. For Australia in 2009 this equated to 41 cases, based on the Australian Bureau of Statistics (ABS) population data released in December 2008. An AFP surveillance scheme that satisfies the surveillance performance indicator is deemed sufficiently sensitive to detect a wild poliovirus importation in children of that country.

The WHO surveillance performance indicator for laboratory testing is that at least 80% of notified AFP cases have adequate faecal specimens collected and tested in a WHO accredited laboratory.

At the end of each calendar year, a number of AFP notifications remain unclassified if insufficient clinical and laboratory data were available to enable the PEP to review the cases. The PEP classifies the remaining AFP notifications as "polio compatible-zero evidence" if a final review reveals no evidence of clustering amongst the unclassified cases.

Virus Culture

Upon receipt at the NERL, faecal specimens are treated with Minimum Essential Medium containing Hank's salts, chloroform (9.1% v/v) and foetal bovine serum (2%). The suspension is clarified and the supernatant inoculated onto a series of mammalian cell lines. Two WHO recommended cell lines are used for the isolation of poliovirus; L20B (a transgenic mouse epithelial cell line expressing the human poliovirus receptor, CD155) and RD-A (human rhabdomyosarcoma).[4,5] Up to September 2011 the NERL utilised two additional cell lines for the isolation of poliovirus and non-polio enteroviruses: Buffalo Green Monkey Kidney (BGMK) and human embryonic lung (HEL).

Diagnostic laboratories in Australia are encouraged to refer enteroviruses of unknown serotype to the NERL for further characterisation as poliovirus infection can lead to clinical presentations without paralysis such as aseptic meningitis.

A series of tests known as intratypic differentiation (ITD) are performed on poliovirus isolates to determine whether the virus is a wild poliovirus strain, OPV strain (Sabin-like) or a VDPV. In 2009 WHO introduced diagnostic poliovirus real time reverse transcriptase PCR (rRT-PCR) developed by the Centers for Disease Control and Prevention USA, as the primary ITD method.[6] The Australian NERL sequences the complete poliovirus VP1 genomic region, which contains a major neutralizing antibody binding site. The VP1 genomic sequence provides valuable biological information, including the number of mutations within a significant region of the OPV virus strain and enables the phylogenetic analysis of wild poliovirus to rapidly determine the likely source of the virus, as utilised in the 2007 importation.[7,8]

Enterovirus Surveillance

The ERLNA was established primarily as a means of detecting imported poliovirus amongst untyped enteroviruses from clinical specimens. The network consists of 10 public sector diagnostic virology laboratories:

Australian Capital Territory

Canberra Hospital: Prof. Peter Collignon, Dr Karina Kennedy, Ms Jennifer Ridgway

New South Wales

Infectious Diseases and Immunology, the University of Sydney: Prof. Peter McMinn

Queensland

Queensland Health and Scientific Services: Dr Russell Simmons, Dr Bruce Harrower, Dr David Warrilow

South Australia

Microbiology and Infectious Diseases, Flinders Medical Centre: Prof. David Gordon

SA Pathology, Institute of Medical and Veterinary Science: Dr Tuck Weng, Ms Kok, Ms Lyn Payne

Victoria

Department of Microbiology, Royal Children's Hospital: Dr Andrew Daley, Ms Poppy Adamopoulos

National Enterovirus Reference Laboratory, VIDRL: Dr Bruce Thorley, Mr Jason Roberts

Viral Identification Laboratory, VIDRL: Dr Chris Birch, Ms Gina Papadakis

Western Australia

Department of Clinical Microbiology, Sir Charles Gairdner Hospital: Dr Avram Levy, Dr Simon Williams, Dr David Williams, Dr David Speers

Department of Microbiology, Princess Margaret Hospital for Children: Dr Leanne Sammels, Ms Katie Lindsay, Prof. Tony Keil

The NERL encourages members of the ERLNA to perform their own virus typing. It has advised members of the ERLNA on enterovirus detection, supplied laboratory and computer analysis protocols and performed tests in parallel with other laboratories for quality assurance purposes. The NERL receives untyped enteroviruses from three laboratories for typing on a regular basis. The other laboratories perform their own enterovirus typing and report the results to the NERL for inclusion in the National Enterovirus Database.

The NERL screens clinical specimens for enterovirus using a semi-nested RT-PCR directed to highly conserved sequence in the five non-translated region (NTR).[9] Enterovirus typing is

primarily performed by amplifying a fragment of the VP1 genomic region according to a published method,[10] but the complete nucleotide sequence of VP1 is required to type some enteroviruses. The enterovirus typing RT-PCR is directed to a region of sequence divergence that allows differentiation between enterovirus genomes. As a consequence, the enterovirus sequence based typing assay is not as sensitive as the pan-enterovirus detection assay. This can result in an enterovirus being detected by pan-enterovirus RT-PCR in a clinical specimen without subsequent identification by the VP1 enterovirus typing assay.

Environmental surveillance

The laboratory cell culture protocol implemented by the NERL is based on a two-phase separation procedure published by WHO. Further advice was obtained from the Enterovirus Laboratory at the National Public Health Institute, Finland, a Global Specialised Laboratory in the WHO Polio Laboratory Network.[11] In brief, 800 mL of sewage is collected prior to any biological or chemical treatment and referred to the NERL within 24 hours. At the laboratory, 500 mL of the sample is centrifuged and the supernatant vigorously shaken at 4°C with dextran, polyethylene glycol and sodium chloride. The mixture is incubated overnight at 4°C in a separating funnel and the lower organic phase is collected the next day and used to re-suspend any pellet stored after the initial centrifugation. The final solution is clarified as for a faecal specimen and inoculated onto the L20B and RD-A cell lines and observed microscopically for cytopathic effect.

Results

Classification of AFP cases

A total of 57 notifications of AFP were received in 2010 (Table 1) and 78 notifications in 2011 (Table 2). The PEP classified 41 cases as non-polio AFP involving children less than 15 years of age with onset of paralysis in 2010, and 60 cases in 2011 (Tables 1 and 2). This equates to a non-polio AFP rate of 1.0 case per 100,000 children less than 15 years of age in 2010 and a rate of 1.4 per 100,000 in 2011. Thus, the WHO AFP surveillance performance criterion for a polio-free country of one case of non-polio AFP per 100,000 children less than 15 years of age was met in both years (Table 3).

In 2009, the PEP resolved to follow a WHO recommendation and report AFP notifications that could not be classified due to a lack of clinical information as "polio compatible – zero evidence". In 2010-11, a total of three AFP notifications, one each from New South Wales, Queensland and Victoria were reported to WHO as polio compatible-zero evidence (Tables 1 and 2).

Four AFP cases were notified by more than one clinician in 2010 and were regarded as duplicate notifications while nine cases were duplicated in 2011 (Tables 1 and 2). Ten AFP notifications in 2010 and eight cases in 2011, did not meet the criteria for an eligible case, either involving patients greater than 14 years of age or the cases were later reported as non-AFP. The cases involving patients greater than 14 years of age were all classified by the PEP as non-polio AFP but were not reported to the WHO as the global polio surveillance program focuses on AFP in children less than 15 years of age as an age group at high risk of poliovirus infection.

Notification of AFP cases by state and territory

In 2010 and 2011, AFP cases were reported from all jurisdictions in Australia except the Australian Capital Territory (Tables 1 and 2). After excluding duplicate notifications and ineligible cases, the non-polio AFP rates per jurisdiction exceeded the WHO AFP surveillance performance indicator of 1.0 case per 100,000 children in New South Wales, South Australia and Victoria in both years. Western Australia did not reach the surveillance indicator in 2010, with a non-polio AFP rate of 0.5 per 100,000, but it was well exceeded in Western Australia in 2011, with a rate of 2.0 per 100,000. The increase in AFP cases notified in Western Australia in 2011 may indicate a surveillance failure in 2010, and/or the year-to-year variation in incidence of a rare childhood condition in a relatively small population. This is further demonstrated in Tasmania, which did not notify any AFP cases in 2010 but reported a non-polio AFP rate of 2.0 per 100,000 in 2011 (Tables 1 and 2).

Faecal collection from AFP cases

WHO defines adequate specimens for poliovirus culture as being two faecal specimens collected at least 24 hours apart and within 14 days of the onset of paralysis. A further surveillance criterion set by WHO is for adequate faecal collection from 80% of the eligible AFP cases.

In 2010, a total of 54 faecal specimens from 29 of the 41 eligible cases were tested at the NERL. Twelve (29%) of the eligible cases had adequate specimens collected while another 12 (29%) cases had only one specimen collected within 14 days of onset.

In 2011, a total of 69 faecal specimens were received from 36 of the 60 eligible cases. Nineteen (32%) of the non-polio AFP cases had adequate specimens collected, and a further 13 (22%) cases had one specimen collected within 14 days of onset.

Table 1: Notification of AFP cases 2010, by state or territory

State/ Territory	Estimated population aged <15 years*	Expected number of AFP cases in 2010†	Total number of notifications	Ineligible notifications	Duplicate notifications	Polio compatible-zero evidence	Eligible cases with final classification by PEP	Non-polio AFP rate per 100,000 children
ACT	64,981	1	0	0	0	0	0	0
NSW	1,343,184	13	23	3	4	1	15	1.2
NT	52,857	1	0	0	0	0	0	0
QLD	886,584	8	6	0	0	0	6	0.8
SA	291,569	3	4	0	0	0	4	1.3
TAS	97,579	1	0	0	0	0	0	0
VIC	1,008,841	10	20	5	0	1	14	1.4
WA	438,532	4	4	2	0	0	2	0.5
Australia	4,184,127	41	57	10	4	2	41	1.0

* Australian Bureau of Statistics, estimated population at 30 June 2009. Available at www.abs.gov.au.

† Based on a non-polio AFP rate of 1 case per 100,000 children less than 15 years of age

PEP Polio Expert Panel

AFP Acute flaccid paralysis

Table 2: Notification of AFP cases 2011, by state or territory

State/ Territory	Estimated population aged <15 years*	Expected number of AFP cases in 2011†	Total number of notifications	Ineligible notifications	Duplicate notifications	Polio compatible-zero evidence	Cases classified by the PEP as non- polio AFP	Non-polio AFP rate per 100,000 children
ACT	66,077	1	0	0	0	0	0	0
NSW	1,355,128	13	24	2	2	0	20	1.5
NT	53,079	1	2	1	0	0	1	1
QLD	901,689	9	5	0	0	1	4	0.4
SA	293,041	3	4	0	0	0	4	1.3
TAS	97,626	1	2	0	0	0	2	2
VIC	1,017,432	10	30	4	5	0	21	2.1
WA	446,058	4	11	1	2	0	8	2
Australia	4,230,130	42	78	8	9	1	60	1.4

* Australian Bureau of Statistics, estimated population at 30 June 2010. Available at www.abs.gov.au.

† Based on a non-polio AFP rate of 1 case per 100,000 children less than 15 years of age

PEP Polio Expert Panel

AFP Acute flaccid paralysis

Australia has never achieved the WHO criterion of collection of adequate specimens from 80% of AFP cases nationally (Figure 2). At the jurisdictional level, Western Australia was the only state to reach the WHO target, with adequate specimens collected from seven of the eight cases (88%) classified in 2011.

Laboratory testing of specimens

AFP cases

In 2010, a total of 54 faecal specimens were referred from 31 cases of AFP involving patients aged less than 15 years, while in 2011, 69 faecal specimens were referred (Table 4). The specimens included faeces, faecal extracts, swabs and cell culture isolates. No poliovirus was isolated from any of these specimens.

Non-polio enteroviruses (NPEV) were reported from three AFP cases in 2010. In the first case, echovirus 19 was isolated from an unimmunised three year old with a diagnosis of anterior horn cell disease confirmed by a MRI consistent with myelitis.[12] The patient had an upper respiratory tract infection three weeks prior to presentation. There was no history of recent travel or contact with an overseas visitor. Initial laboratory investigation was hampered by the CSF being inhibitory by RT-PCR. The local hospital isolated enterovirus by virus culture from a nasopharyngeal aspirate and faeces, which was confirmed by RT-PCR. The original specimens and virus isolates were referred to the NERL and echovirus 19 was identified from the virus isolates by sequencing a fragment of the capsid encoding region of the virus genome.

Echovirus 3 was identified from the first of two faecal specimens referred from the second AFP case involving a 12 year old patient diagnosed with meningitis. The specimen was collected nine days after the onset of symptoms while the second specimen was collected 13 days after onset.

In the third case, enterovirus 68 (EV68) was identified from a single faecal specimen received by the NERL from a patient with spinal cord ischaemia. The virus was detected by RT-PCR from the faecal extract while virus culture was negative.

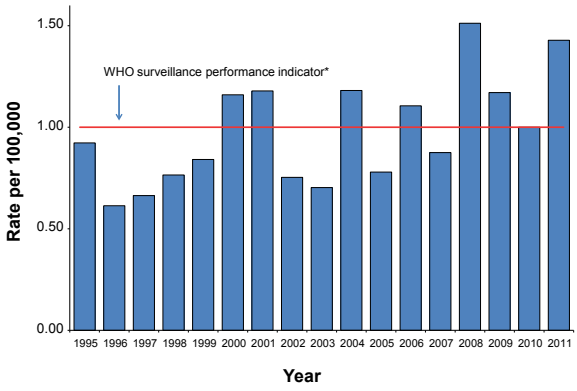
In 2011, a NPEV was detected in two faecal specimens from one AFP case. An atypical enterovirus cytopathic effect was observed in virus culture and was later confirmed by pan-enterovirus RT-PCR. The virus was typed from the stool extract by sequencing a fragment of the VP1 genomic region and identified as coxsackievirus type A24.

In 2010 and 2011, a total of 13 specimens were received from eight cases involving patients greater than 14 years of age, which is outside of the WHO AFP surveillance criterion. No enterovirus was isolated from the specimens.

Enterovirus Surveillance

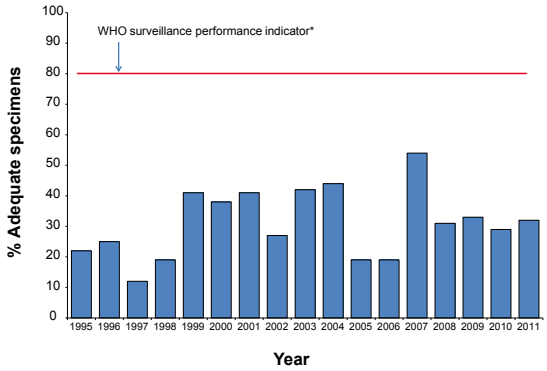
No poliovirus was detected by the ERLNA in 2010 and 2011. The ERLNA typed 234 NPEVs in 2010 and 331 in 2011. Coxsackievirus A6 (CA6) and enterovirus 71 (EV71) were amongst the leading enteroviruses detected in Australia in 2010 and 2011. Both viruses are typically associated with hand, foot and mouth disease in children. B5 was the predominant subgenogroup of EV71 detected in Australia in 2010 and 2011 and was detected in post-mortem specimens from an infant in 2010. Coxsackievirus B1 was detected in both the western and eastern states in 2009 and 2010. Echovirus 25 was the most common enterovirus identified in 2011.

Figure 1: Non-polio AFP rate classified by the PEP 1995 to 2011



* The WHO AFP surveillance performance indicator for a polio non-endemic country is one case per 100,000 children <15 years of age.

Figure 2: Percentage of AFP cases with adequate faecal specimens, 1995 to 2011



* The criterion for the WHO surveillance performance indicator is the collection of two faecal specimens more than 24 hours apart and within 14 days of the onset of symptoms.

Table 3: Surveillance for AFP cases in children less than 15 years, Australia, 2010 to 2011, compared with the WHO performance indicators

WHO surveillance performance indicator for AFP cases in children less than 15 years*	Australia's AFP surveillance performance	
	2010: 41 AFP cases expected	2011: 42 AFP cases expected
Non-polio AFP case rate of 1.0 / 100,000 children	Non-polio AFP rate 1.0 / 100,000 children (41 cases classified)	Non-polio AFP rate 1.43 per 100,000 children (60 cases classified)
More than 80% of classified AFP cases with adequate faecal specimens†	29% (adequate specimens received from 12 / 41 AFP cases)	32% (adequate specimens received from 19 / 60 AFP cases)

* Population data derived from the Australian Bureau of Statistics, estimated population, at 30 June 2009 and 2010. Available at www.abs.gov.au. Based on Australia's population less than 15 years of age.

† Adequate faecal specimens defined as 2 faecal specimens collected at least 24 hours apart and within 14 days of onset of paralysis.

Table 4: Test results for faecal specimens from AFP cases involving children < 15 years of age referred to the NERL, 2010 and 2011

Result	2010	2011
Non-polio enterovirus*	3	2
No enterovirus isolated	51	67
Total	54	69

* In 2010, non-polio enteroviruses identified from three AFP cases were echovirus 19, echovirus 3 and enterovirus 68. In 2011, coxsackievirus A24 was identified from two faecal specimens of one AFP case.

It was mainly associated with fever and most cases originated from Western Australia.

Environmental Surveillance

In 2010, sentinel sites for environmental surveillance for poliovirus were established at Armidale and two sites in Newcastle at Burwood Beach and Shortland. The Burwood Beach site was replaced by Byron Bay in 2011. Fifteen collections (5 from each site) were tested in 2010 and a further nine collections (3 from each site) in 2011. No poliovirus was reported from any of the 500 mL grab samples processed by the NERL.

The sewage extracts were tested in parallel by cell culture and a pan-enterovirus RT-PCR. The pan-enterovirus RT-PCR is a validated in-house test and was utilised to confirm the cell culture results as not all human enteroviruses can infect the RD-A cell line. All enterovirus isolates from cell culture and positive detections by RT-PCR were investigated to determine the virus type by nucleic acid sequencing and the results from the two methods were the same. All samples except one from each of the Newcastle sites and one from Byron Bay were positive for NPEV in virus culture, which serves as an indicator organism for the collection, transportation and laboratory procedures.

Regional reference laboratory activities

The following activities were performed as a Polio Regional Reference Laboratory in 2010 and 2011:

- Brunei Darussalam: Specimens from three AFP cases were received in each year. Sabin poliovirus types 1, 2 and 3 were isolated from one case in 2011, consistent with recent immunisation.
- Pacific Island countries: Specimens from 14 and 9 AFP cases were received in 2010 and 2011, respectively. No poliovirus was isolated from any of these but NPEVs were isolated from 6 of the 20 cases. In 2010, 14 specimens were referred from Fiji to investigate the cause of a hand, foot and mouth disease outbreak. Coxsackievirus A6 was detected in 11 of these. Twenty faecal specimens were referred from a gastroenteritis outbreak in Fiji in 2011. A NPEV was isolated from only one of the specimens, indicating that an enterovirus was unlikely to have caused the outbreak.
- Papua New Guinea: Specimens from 17 and 9 AFP cases were referred in 2010 and 2011, respectively. Sabin-like poliovirus type 3 was isolated from one case in 2010. Fourteen NPEVs were isolated by cell culture or detected by RT-PCR from 14 of the 26 cases.
- Philippines: Four poliovirus type 2 and 3 poliovirus type 3 viruses were referred from AFP cases for ITD in 2010 and all were characterised as Sabin-like. A poliovirus type 3 and a poliovirus type 1 were characterised as Sabin-like from sources other than AFP in 2010 and 2011, respectively.

Table 5: Enterovirus test results at the NERL, Australia, 1995 to 2011

Year	Poliovirus		Non-polio enterovirus	No enterovirus detected	EVID results referred §	Total samples reviewed
	Sabin-like	Non-Sabin-like				
1995	190	0	200	13	0	403
1996	224	0	198	9	0	431
1997	124	0	76	0	0	200
1998	52	0	15	4	0	71
1999*	60	1	9	9	0	79
2000	45	0	44	47	0	136
2001*	46	5	33	75	0	159
2002	36	0	21	49	0	106
2003	9	0	15	47	0	71
2004	6	0	26	61	0	93
2005	18	0	10	39	0	67
2006	2	0	6	71	29	108
2007†	0	2	32	115	107	256
2008	0	0	20	92	77	189
2009‡	1	0	63	78	113	255
2010	0	0	170	39	108	317
2011	0	0	174	61	205	440

* Untyped enterovirus or uncharacterised poliovirus isolates were referred for further testing after completion of a laboratory inventory. The six isolates tested as non-Sabin-like and were subsequently identified as wild type poliovirus prototype strains and were destroyed.

† Wild poliovirus type 1 was imported from Pakistan.

‡ A Sabin-like poliovirus type 1 was identified from an unimmunised infant.

§ Enterovirus Identification results include retrospective data made available via the ERNLA.

Quality Assurance Programs

In 2010 and 2011, the NERL passed the annual quality assurance panels for poliovirus isolation by cell culture and poliovirus RT-PCR for ITD and vaccine derived poliovirus. In 2011, WHO introduced a trial poliovirus sequencing proficiency panel with the intention of making this an annual test from 2012. The NERL maintained accreditation as a Polio Regional Reference Laboratory after WHO conducted a two day on-site review of laboratory procedures and documentation in October 2010. Accreditation is assessed in the interim years by the submission of an annual checklist and subject to passing the annual laboratory proficiency tests.

Discussion

Clinical surveillance for cases of AFP in children is a sensitive means of detecting imported cases of poliomyelitis in a polio-free country by targeting an age group that is at high risk of infection if not immunised. This occurred in China when an outbreak of polio was reported in the far western province of Xinjiang due to an importation of a wild poliovirus type 1 from Pakistan in August 2011.[2] Initially four polio cases were confirmed through AFP surveillance, in children aged between four months and two years, who had had onset of paralysis the preceding month. A further 17 cases of polio were confirmed up to October 2011 and included two fatalities. Polio cases were also reported in adults. The outbreak was stopped by concerted vaccination campaigns targeting people up to 39 years of age.[2] This serves as a salutary reminder that polio can quickly spread in a population with inadequate polio vaccination coverage at any age.

Australia met the WHO AFP surveillance performance indicator of at least 1.0 non-polio AFP case per 100,000 children less than 15 years of age in 2010 and 2011, reporting a rate of 1.0 and 1.4 per 100,000 respectively. This was the fourth year in a row that Australia has met the indicator used by WHO to assess whether a national AFP surveillance system is likely to detect an imported case of polio in a child. At the state and territory level, New South Wales, South Australia and Victoria exceeded the performance indicator rate in both years, while the Northern Territory, Tasmania and

Western Australia only did so in 2011. Queensland did not reach the surveillance indicator in either year despite it being the only jurisdiction where AFP is notifiable. Six of Australia's eight jurisdictions met the WHO performance indicator in 2011, a result that reinforces the overall sensitivity of the national AFP surveillance system.

Another important aspect of AFP surveillance is the testing of stool specimens to exclude poliovirus as the causative agent. Notwithstanding the strong performance of AFP case ascertainment in Australia in recent years, the number of AFP cases with adequate stool specimens has averaged 31% for the last four years. This compares to the WHO surveillance performance indicator of 80%, a target that Australia has never met. Australia's standard of performance against this surveillance indicator is not unusual for developed nations. The reasons for poor rates of faecal specimen collection are manyfold. This could include an unwillingness or inability of the patient to provide a faecal specimen, prioritising laboratory tests of specimens from other sites such as cerebrospinal fluid and relying upon neurological diagnostic procedures such as magnetic resonance imaging and nerve conduction studies.

Virological surveillance for poliovirus was introduced in Australia to complement the clinical surveillance program for AFP cases in children. It has two components, the typing of enteroviruses through the ERLNA and environmental surveillance by testing grab samples of sewage at sentinel sites. No poliovirus was isolated through either of these surveillance systems providing additional data in support of Australia's continued polio-free status. In addition to testing for poliovirus, enterovirus typing facilitates the detection of NPEVs of public health importance, such as EV71, and will enable the epidemiology of enteroviruses circulating in Australia to be better understood. The predominant subgenogroup of EV71 detected in Australia in 2010 and 2011 was B5 and, based on reports from the ERLNA this subgenogroup was mainly associated with fever and hand, foot and mouth disease. EV71 B5 was also detected in faecal material from a post-mortem sample from an infant suffering from a suspected viral illness in 2010 but causality cannot be confirmed due to the detection of the virus from a non-sterile site. A report from New South Wales in 2011 linked EV71 meningoencephalitis with the death of a 63 year old male who had received rituximab as treatment for non-Hodgkin's lymphoma.[13] The EV71 was typed as subgenogroup C2. Anti-CD20 monoclonal antibody therapy can deplete B cells, which are required to clear enterovirus infection.

As a result of the broader focus on enterovirus surveillance in support of poliovirus surveillance, the Polio Reference Laboratory was renamed the National Enterovirus Reference Laboratory from July 2011.

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Surveillance for Creutzfeldt-Jakob disease in Australia: update to December 2012

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Abstract

Nation-wide surveillance for transmissible spongiform encephalopathies including Creutzfeldt-Jakob disease (CJD) is undertaken by the Australian National Creutzfeldt-Jakob disease Registry (ANCJDR), based at the University of Melbourne. Surveillance has been undertaken since 1993. During this period the unit has evolved and adapted to changes in surveillance practices and requirements, the emergence of new disease subtypes, improvements in diagnostic capabilities and the overall heightened awareness and understanding of CJD and other transmissible spongiform encephalopathies in the health care setting. In 2012, routine surveillance continued. This brief report provides an update on the surveillance data collected by the ANCJDR prospectively from 1993 to December 2012, and retrospectively to 1970. It also highlights the recent release of the revised Australian CJD Infection Control Guidelines.

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

Introduction

In 1993 the Allar's inquiry into the use of cadaver-derived pituitary hormones under The Australian Human Pituitary Hormone Program and the association with four medically acquired (iatrogenic) Creutzfeldt-Jakob disease (CJD) deaths recommended the formation of an Australian surveillance unit to monitor further cases of iatrogenic CJD in Australia.[1] The Australian National Creutzfeldt-Jakob disease Registry (ANCJDR) was established in October 1993 at the Department of Pathology at the University of Melbourne. The monitoring of further Australian iatrogenic CJD cases related to pituitary hormone treatment for infertility or short stature and contaminated dura mater grafts remains one of the core objectives of the ANCJDR. However, the ANCJDR's activities have changed to encompass the surveillance of all types of CJD including sporadic, genetic and variant CJD and other transmissible spongiform encephalopathies (TSEs) such as Gerstmann Sträussler-Sheinker Syndrome (GSS) and fatal familial insomnia (FFI).

Sporadic CJD currently accounts for between 85% and 90% of all CJD cases internationally.[2] Cases are defined as sporadic when there is no discernible transmission event and when there is no family history and/or negative prion protein gene (*PRNP*) testing. Familial TSEs include genetic CJD, GSS and FFI. These cases are classified as such if there is a disease-specific mutation in *PRNP* or a TSE is confirmed in a 1st degree relative. *PRNP* mutations include single nucleotide substitutions and poly-nucleotide insertions and deletions. Polymorphisms in *PRNP* such as at codon 129 are thought to influence the disease phenotype (including in relation to particular mutations), as well as susceptibility to sporadic and some forms of iatrogenic CJD. Classification of iatrogenic CJD cases is dependent on a typical clinical profile and recognition of a transmission risk.

Since 1993 there has been considerable change in the understanding of surveillance for TSEs. This is due to the appearance of new disease subtypes, greater clinical awareness, improved and varied diagnostic capabilities, continued scientific research and the world wide focus on CJD through the emergence of variant CJD (vCJD) in 1996. In response to these changes, the ANCJDR has adapted with available resources to meet the increasing demands for diagnostic testing, clinical and expert infection control advice, and the steadily growing number of suspected case notifications directed to the ANCJDR for evaluation.

Methods

Various mechanisms have been established by the ANCJDR in order to ascertain all cases of TSE in Australia since 1970. During 2012 cases were referred through several mechanisms including diagnostic test referral, personal communication by clinicians, families, health departments and hospitals and the CJD counselling service. As of June 2006, CJD was listed as a notifiable disease in all states and territories.

Since September 1997 the ANCJDR has offered national diagnostic testing for the presence of a family of low molecular weight proteins called 14-3-3 proteins in cerebrospinal fluid (CSF). This single test has provided an increasingly larger proportion of the annual suspected case notifications. It is currently the predominant (49.9%) and continually increasing source of notifications. The CSF 14-3-3 protein test is now the most broadly utilized diagnostic tool for CJD. As of 2012, all CSF samples tested by the ANCJDR were followed-up at three months and, if necessary, again at nine months after initial referral to determine the outcome for the patient.

This is performed to assist in the determination of the sensitivity and specificity of the diagnostic test. The follow-up at three months was introduced to enable a more timely prompt for clinicians and has led to more case outcomes being determined than follow up at nine months only. If death has occurred within three months of the original referral and CJD is considered a possible diagnosis, further case evaluation is performed.

After notification of a suspected case and a detailed evaluation, the ANCJDR utilises internationally recognised case definitions for the classification of definite, probable and possible cases.[3-5] Suspected CJD cases retain an incomplete status until evaluation is complete. Definite cases are those that have been neuropathologically confirmed either by brain biopsy or post-mortem examination. Probable cases are classified on the basis of clinical profile and a typical electroencephalogram (EEG) and/or a positive 14-3-3 CSF test and/or a characteristic magnetic resonance imaging (MRI) with high T2 weighted image signal in the caudate/putamen. In addition to dementia, probable cases must display at least two of the following; myoclonus; visual or cerebellar signs; pyramidal or extrapyramidal features; and/or akinetic mutism with an illness duration of less than two years. Possible cases fulfil the same clinical profile in the absence of a typical EEG, characteristic MRI and either no or a negative 14-3-3 CSF test result. The method of classification of possible cases is in accordance with the European Creutzfeldt Jakob Disease (EUROCJD) and World Health Organization (WHO) promulgated diagnostic criteria and has been in use since 1 January 2001.[5]

Annual TSE incidence rates were calculated using direct age-standardisation, based on the Australian Bureau of Statistics 2000 estimated resident population for Australia and each state and territory.[6]

Results

In 2012, 53 new suspected TSE cases were added to the ANCJDR for evaluation. The source of notification for these new cases included requests for a CSF 14-3-3 protein test (62%), personal communication from a neurologist, neuropathologist, clinician or hospital (23%), health department notification (7%), communication from a family (6%) or from the CJD counselling service (2%). The proportion reported from each source is consistent with those in 2011. CSF referral has accounted for 74% of all referrals since 2000, with 21% by direct personal communication (comprising medical practitioners, 16%, families, 4% and hospitals, 1%).

In 2012 notification numbers declined nationally by 37% compared to the previous year (Figure 1). Contributing to this decrease were fewer notifications in several states including Victoria (32%), New South Wales (45%), Western Australia (60%) and Tasmania (100%). The remaining states and territories remained unchanged from the previous year. This decline in notifications is unexplained and these rates will be closely monitored in 2013.

In 2012, 38 of the 53 notified cases were still under investigation. The annual proportion of suspected cases notified between 1993 and 2011 that were subsequently classified as definite or probable TSE cases ranges from 32% to 78%, with a mean of 46%.

Figure 1: Prospective notifications of suspected TSE cases notified to the ANCJDR, 1993 to 2012, by state or territory and year

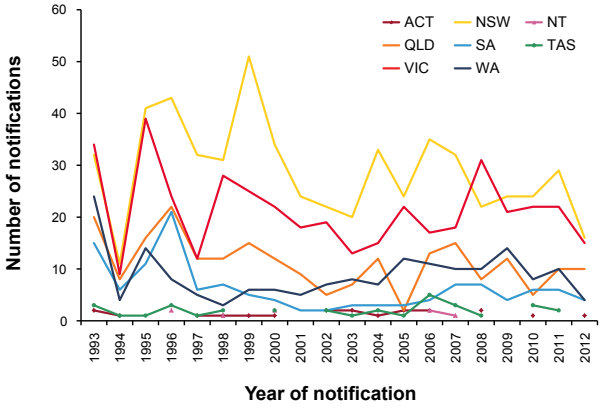
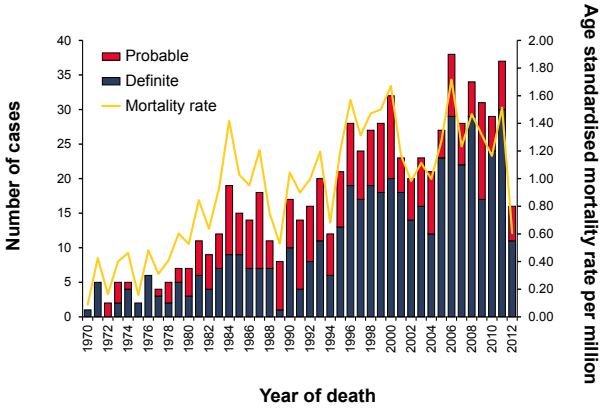


Figure 2: Number of definite and probable TSE cases and age-standardised mortality rate in Australia, 1970 to 2012, by classification and year



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

Figure 3: Definite and probable TSE cases, 1970 to 2012, by aetiology and year

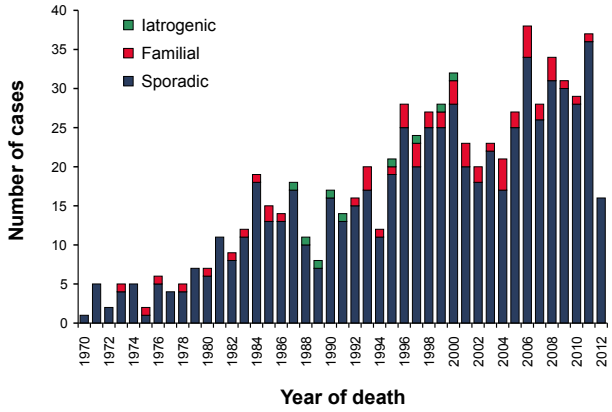
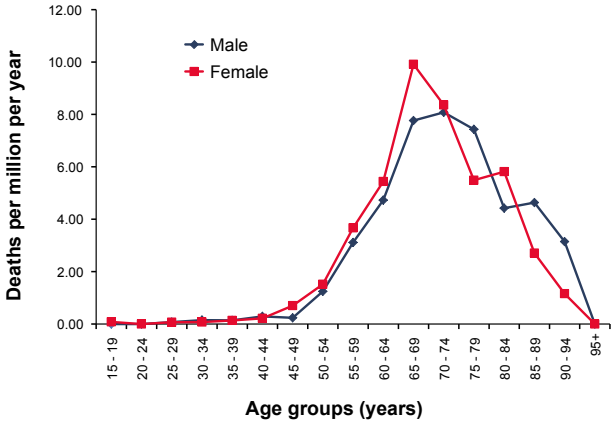


Figure 4: Mortality rates for all TSE cases 1993 to 2012, by sex and age group



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

As of 31 December 2012, 962 suspected TSE cases were on the register with 733 of these being classified as Australian probable or definite TSE cases (Table 1).

An additional 638 cases were excluded after detailed follow-up. Of the 53 new suspected cases added to the register in 2012, 3 were excluded (2 following neuropathological examination), 38 are incomplete, 8 were classified as definite CJD and 4 as probable CJD. During 2012, 45 suspected cases were excluded from the register (10 after neuropathological examination) and 38 cases were classified as sporadic CJD and 1 as familial TSE. There are currently 14 cases of possible CJD of which 13 are sporadic and 1 iatrogenic. Of the 214 incomplete cases, 135 are presently alive. In comparison to the rapid increase in the number of incomplete cases on the register observed between 2003 and 2010 (average 22% increase per year), an overall reduction in the size of this group was recorded in 2012 (12% decrease).

Between 1 January 2012 and 31 December 2012, there were no new cases of iatrogenic CJD. The most recent human-derived pituitary gonadotrophin-related CJD death occurred in 1991, while the most recent Lyodura-related CJD death occurred in 2000. As of 31 December 2012, there had been no known cases of vCJD in Australia.

Between 1970 and 2000, the annual incidence of TSEs in Australia steadily increased (Figure 2).

As for other international CJD surveillance programs, the increase probably reflects case ascertainment bias stemming from improved recognition, investigation, case confirmation and reporting. The incidence of TSE in Australia declined and stabilized at around 1.0 case per million per year during 2001-2004, but increased to 1.72 cases per million per year in 2006. Incidence remained at around 1.3 to 1.4 cases per million per year between 2007 and 2012.

The majority of the confirmed Australian TSE cases have been of sporadic aetiology (92%) and this has been a consistent observation from 1970 to 2012. Familial and iatrogenic cases constitute 7% and 1%

Table 1: Classification of ANCJDR cases 1970 to 2012

Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total
Definite	433	44	5*	0	0	482
Probable	238	10	4	0	0	252
Possible	13	0	1	0	0	14
Incomplete	0	0	0	0	214**	4
Total	684	54	10	0	214	962

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

** includes 135 living cases

Table 2: TSE deaths and age-adjusted mortality rates, 2000 to 2012, by year and state or territory

	TSE cases by year of death														Mean age-adjusted mortality rate (deaths/ million/year)
	00	01	02	03	04	05	06	07	08	09	10	11	12	Alive	Total	
ACT			1		1		1		2		1				6	1.25
NSW	12	9	7	7	11	10	12	10	6	11	5	14	2	1	117	1.24
NT							2	1							3	0.74
QLD	7	3	3	3			7	2	4	4	2	5	5		45	0.78
SA	2			1	2	1	1	3	5	2	4	4			25	1.13
TAS			2			1	2						1		6	0.85
VIC	9	10	5	9	5	11	9	6	13	9	13	9	6	1	115	1.62
WA	2	1	2	3	2	4	4	6	4	5	4	5	2		44	1.49
AUS	32	23	20	23	21	27	38	28	34	31	29	37	16	2	361	1.25

Age-standardised mortality rates were calculated using the Australian Bureau of Statistics 2000 estimated resident population for Australian states and territories

respectively of all definite and probable cases. Between 1993 and 2012 the average number of familial cases classified in Australia was 2 cases per year. Since 2009 only one familial case has been classified per year. The overall proportion of cases classified as familial TSE has declined (Figure 3).

Between 2002 and 2012 the majority of states and territories had age-adjusted mortality rates above or close to 1.0 case per million per year (Table 2). The highest mean mortality rates were observed in Victoria and Western Australia (1.62 and 1.49 deaths per million per year, respectively).

From 1970 to 2012 there were more female TSE cases (54%) than male for all forms of TSE combined. This was also true for sporadic (54%) and genetic (55%) forms. A comparison of age and sex-specific mortality shows the similarity of rates between males and females with some exceptions in the older age groups (Figure 4).

The median age of death from all forms of TSE between 1970 and 2012 was 67 years with little difference between the sexes (men, 66 years, women, 67 years). For familial cases, a difference did exist between the sexes, as the median age at death was 52 years in males and 62 years in females. The range in age at death from TSE was broad for both the sporadic (25-90 years) and familial (18-82 years) group with median age at death being 67 and 59 years respectively. For the eight iatrogenic cases, death occurred between the ages of 26 and 62 years and disease duration from onset to death was between 2 and 25 months (median, 6.25 months). For all TSE cases, 92.9% of deaths occured over the age of 50. This demonstrates that older age groups are at risk of developing TSEs and this is consistent for all TSE aetiologies. Of the 39 cases confirmed with a TSE in 2012, all deaths occurred in those over the age of 50 years.

Between 1970 and 2012, disease duration from onset varied between the three aetiologies. Sporadic TSE cases had much shorter disease duration than both iatrogenic and familial cases with 50% of deaths occurring within 3.5 months of onset (range, 0.9 – 60 months). From 1970 to 2012 familial cases were associated with a significantly greater survival time in comparison to sporadic TSE with the median illness duration of 6 months (range, 1.5 – 192 months). Within 6 months of disease onset, 72% of sporadic cases, 53% of familial cases and 56% of iatrogenic cases had died.

Discussion

There are several possible explanations regarding the range in the annual number of notifications and the proportion of suspected cases that were subsequently confirmed as TSE cases. These include the prospective surveillance approach employed, diagnostic capacity changes such as the CSF 14-3-3 protein test and MRI, enhanced clinician awareness, a greater public health profile for CJD through the focus on variant CJD. In addition, the notifiable status of CJD was established in all states and territories by June 2006. Specifically, Tasmania (May 2003), Victoria (Jan 2004), Western Australia (Jan 2004), New South Wales (April 2004), Northern Territory (Dec 2004), Australian Capital Territory (Sept 2005), Queensland (Dec 2005), South Australia (June 2006).

In 2012 there was a high number of cases confirmed or excluded by neuropathology. There was also a 4-fold increase in the number of probable cases and a two-fold increase in the number of cases excluded

from the ANCJDR after detailed evaluation. These changes led to a 12% decrease in the number of incomplete or unresolved cases on the register. As in 2011, the ANCJDR made a concerted effort during 2012 to focus staff resources on performing case reviews and classifying outstanding, incomplete cases.

Fluctuating peaks in the incidence of TSE might be expected in such a rare disease. The ANCJDR believes that there are a number of factors responsible for the 2000-04 decline. These include a reduction in the number of probable cases classified due to broadened surveillance responsibilities, difficulties experienced following changes to the privacy legislation, and changes to the registration of cases referred for CSF 14-3-3 protein testing. The subsequent peak incidence in 2006 aligns with a increasing trend in notifications in 2005–06. This can be attributed predominantly to increased ante-mortem notifications through the CSF 14-3-3 protein test, which has enabled a greater number of post-mortem examinations to be actively investigated for suspected TSE. Ultimately, with more post-mortem examinations being performed, a greater number of suspected TSE cases have been confirmed. Currently, the proportion of all suspected cases notified to the ANCJDR between 1993 and 2011, (including those cases excluded from the register after evaluation and where death is known to have occurred) who have undergone a post-mortem examination is 62%. The ANCJDR also believes that the peak incidence rates of 1.67 and 1.72 cases per million per year observed in 2000 and 2006 respectively provide a more accurate estimation of the true incidence of TSEs in Australia, underscoring the importance for post-mortem examinations to be actively promoted in all suspect cases. The mean annual age-adjusted TSE mortality rate for the 1993 to 2012 period was 1.26 deaths per million per year. This rate aligns with the reported figures for other countries with similar surveillance mechanisms to those in Australia.[7]

The ANCJDR has maintained a non-systematic approach to the prion protein genotyping of confirmed TSE cases. This may have contributed to our lower percentage of familial cases (7%) compared with European CJD surveillance programmes, which report that between 12% and 14% of cases are familial.[8] In recent years the free *PRNP* genetic testing service provided by the ANCJDR to CJD patients and families has been decentralised due to their preference for an "on-demand" service. Although the ANCJDR still performs routine genetic testing three times annually, testing is now also undertaken by external, independent laboratories and genetic services. The separation of *PRNP* testing from the ANCJDR may have inadvertently contributed to the reduced proportion of genetic TSE cases observed over the last few years (Figure 3).

In 2012 the Australian CJD Infection Control Guidelines were revised by the Communicable Diseases Network Australia and published in January 2013.[9] These guidelines provide updated information for health care and funeral industry professionals and families of CJD patients. They aim to provide greater clarity for infection control and ensure ease of use and the avoidance of unnecessary discrimination or disadvantage for families affected by CJD.[10]

During 2012, the ANCJDR continued nation-wide surveillance for all forms of TSE and has identified a decrease in the number of suspected cases notified for evaluation. Overall disease incidence has not been affected by this decline; however, it remains to be determined how this will influence incidence rates in 2013. Notifications will be closely monitored during 2013.

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Australian Trachoma Surveillance Annual Report, 2011

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Abstract

Australia remains the only developed country to have endemic trachoma in some regions. Endemic levels of trachoma in Australia are found predominantly in remote and very remote Aboriginal communities. Data are collected from Aboriginal communities designated at-risk for endemic trachoma (defined as a prevalence of 5% or greater among children) in the Northern Territory, South Australia and Western Australia. This report presents data collected in 2011. The World Health Organization (WHO) grading criteria were used to diagnose cases of trachoma in Aboriginal children with jurisdictions focusing screening activities on the 5-9 year age group. The prevalence of trachoma within a community was used to guide appropriate treatment strategies as a public health response. Aboriginal adults aged 40 years or older were screened for trichiasis. Population screening coverage for trichiasis in 2011 was 9% with a prevalence of 2% in those adults screened. Trachoma screening coverage of the estimated population of children aged 5-9 years in at-risk communities was 65%. Trachoma prevalence among children aged 5-9 years who were screened was 7%. Of the communities screened, 47% were found to have no cases of active trachoma and 40% were found to have endemic levels. Treatment was required in 80 at-risk communities screened. Treatment coverage of active cases and their contacts varied between jurisdictions, ranging from 53% to 98%. This report provides evidence of increasing coverage of trachoma screening and control activities. In the Northern Territory and Western Australia, there is also evidence of a decline in the prevalence of infection that may be attributable to an improvement in control activities. Despite these apparent advances, trachoma prevalence remains at endemic levels in many communities in remote Australia. Continued efforts are required to ensure that Australia remains on track to reach the goal of elimination by 2020 or sooner.

Keywords: active trachoma, antibiotic resistance, control activities, endemic, facial cleanliness, Northern Territory, SAFE control strategy, South Australia, surveillance, Western Australia

Introduction

This is the sixth national trachoma surveillance annual report. Trachoma screening and management data for 2011 were provided to the National Trachoma Surveillance and Reporting Unit (NTSRU) by the Northern Territory, South Australia and Western Australia.

Trachoma is an eye infection caused by *Chlamydia trachomatis*. The infection can be transmitted through close facial contact, hand-to-eye contact, via fomites (towels, clothing and bedding) or by flies. Repeated infections with *C. trachomatis*, especially during childhood, may lead to scarring, and distortion of the eyelid which may cause the eyelashes to rub against the cornea; this is known as trichiasis and can lead to blindness.[1,2,3]

Australia is the only high-income country in the world where trachoma is endemic. It occurs primarily in remote and very remote Aboriginal communities in the Northern Territory, South Australia and Western Australia. In 2008 cases were also found in New South Wales and Queensland, where trachoma was believed to have been eliminated.[4,5,6] Australia is a signatory to the Global Elimination of Blinding Trachoma (GET) 2020 initiative, supported by the World Health Organization (WHO) Alliance. This aims to eliminate blinding trachoma by 2020 through the Surgery, Antibiotics, Facial cleanliness and Environmental improvements (SAFE) strategy. In accordance with the GET 2020 initiative the Australian Government committed $16 million over a 4-year period from 2009 towards eliminating trachoma in Australia. The funding is for the improvement and expansion of screening and control activities, as well as establishment of a strong framework for monitoring and evaluation, including the NTSRU.

Methods

Each participating jurisdiction undertook screening and treatment activities for trachoma according to its respective protocols, and in the context of the national 2006 Communicable Disease Network Australia (CDNA) *Guidelines for the public health management of trachoma in Australia*.[2] The guidelines recommend specific treatment strategies depending on the prevalence of trachoma detected through screening.

In 2006 when the National Trachoma Management Program was initiated, each participating jurisdiction identified at-risk communities from historical prevalence data and other knowledge such as data about movement of people between communi-

ties. Over time, additional communities have been reclassified as being at risk. Screening for trachoma focuses on the at-risk communities, but a small number of other communities designated as not being at risk were also screened if there was anecdotal information suggesting the presence of active trachoma. In 2011, jurisdictional authorities designated 207 remote Aboriginal communities as being at risk of endemic trachoma. All jurisdictions have adopted a school-based screening approach, with the focus on the 5-9 year age groups which have the highest levels of school attendance.

The WHO trachoma grading criteria were used to diagnose and classify individual cases of trachoma. Data collection forms for use at the community level were developed by the National Trachoma Surveillance Reference Group (TSRG), based on the CDNA Guidelines. Completed forms were forwarded from the jurisdictional coordinators to the NTSRU for checking and analysis. Information provided to the NTSRU at the community-level for each calendar year included the number of Aboriginal children aged 1-14 years screened for clean faces and the proportion with clean faces:

- by age group; the number of Aboriginal children aged 1-14 years screened for trachoma and the proportion with trachoma
- by age group; the number of episodes of treatment for active trachoma including household contacts and other community members, by age group.

Information provided also included:

- the number of Aboriginal adults screened for trichiasis, number with trichiasis
- the number who had surgery for trichiasis, as well as community level implementation of WHO SAFE strategies.

Northern Territory

Trachoma screening and management in the Northern Territory was undertaken through collaboration between the Northern Territory Department of Health (Centre for Disease Control and Health Development) and Aboriginal community controlled health services (ACCHS). Trachoma screening was incorporated into the Healthy School Age Kids (HSAK)[7] annual check and conducted by either local primary health care services or ACCHS with support from the Centre for Disease Control (CDC) Trachoma Team. Following screening, treatment was generally undertaken by primary health care services with support from the CDC Trachoma Team. Community screening for trichiasis was initiated in a small number of communities by the CDC Trachoma Team. Some adult screening took place during community visits by the CDC Trachoma Team staff, ACCHS, optometrists or ophthalmologists from the Regional Eye Health Service based in Alice Springs.

South Australia

In 2011, Country Health South Australia (CHSA) was responsible for managing the South Australian trachoma screening and treatment program. CHSA contracted with local health service providers, Aboriginal community controlled health services, the Aboriginal Health Council of South Australia (AHCSA) and Nganampa Health Service to ensure coverage of screening services in all at-risk rural and remote areas. Additional screening activities were undertaken by the Eye Health and Chronic Disease Specialist Support Program (EH&CDSSP), coordinated by the Aboriginal Health Council of South Australia and supported by the Medical Specialist Outreach Assistance Program (MSOAP) and the Office for Aboriginal and Torres Strait Islander Health, Department of Health. This program provides regular visits to South Australian remote Aboriginal communities by optometrists and ophthalmologists. Trichiasis screening was undertaken opportunistically for adults by both the EH&CDSSP team and the trachoma screening service providers, and is also undertaken routinely as part of the Adult Annual Health Checks.

Western Australia

Trachoma screening and management in Western Australia is the responsibility of Population Health Units (PHUs) in the Kimberley, Goldfields, Pilbara and Midwest Health Regions. In collaboration with the local primary health care providers, the PHUs screened at-risk communities in each region through schools within a two-week period, mostly at the end of August or early September. Treatment is undertaken at the time of screening. Trichiasis screening was undertaken in conjunction with adult influenza vaccinations. Screening the target population also occured with the Visiting Optometrist Scheme (VOS) in the Kimberley. In 2011 Western Australia changed the definition of community, specifically amalgamating several previously distinct communities into one single community. This alters the trends presented in this report compared to previous reports

Data analysis

For the purpose of this report, a community is defined as a specific location where people reside and there is at least one school. Community coverage is defined as the proportion of at-risk communities screened

for trachoma. Individual screening coverage is the proportion of children in the target age group in a community who were actually screened.

Population data were based on the 2006 Australian census as in previous reports.[8] The population for communities in subsequent years were projected forward using Australian Bureau of Statistics standard estimates of population increase (1.6%, 1.8% and 2.1% in the Northern Territory, Western Australia and South Australia respectively). Population estimates based on the 2006 census do not account for policy changes such as the Northern Territory Intervention, which may have resulted in unexpected population movements. Prevalence of active trachoma was calculated using the number of children screened as the denominator.

Trachoma data were analysed in the age groups 1-4, 5-9 and 10-14 years. Comparisons over time were limited to the 5-9 year age group, for which screening coverage has been consistent over time. Data from 2006 were excluded from the assessment of time trends as collection methods in this first year differed from those subsequently adopted. Statistical significance in prevalence trend rates for communities that screened consistently from 2007 was tested with the chi-square test for trend. Adherence to the CDNA treatment guidelines was assessed by calculating the estimated proportion of active cases and contacts requiring treatment that were in fact treated.

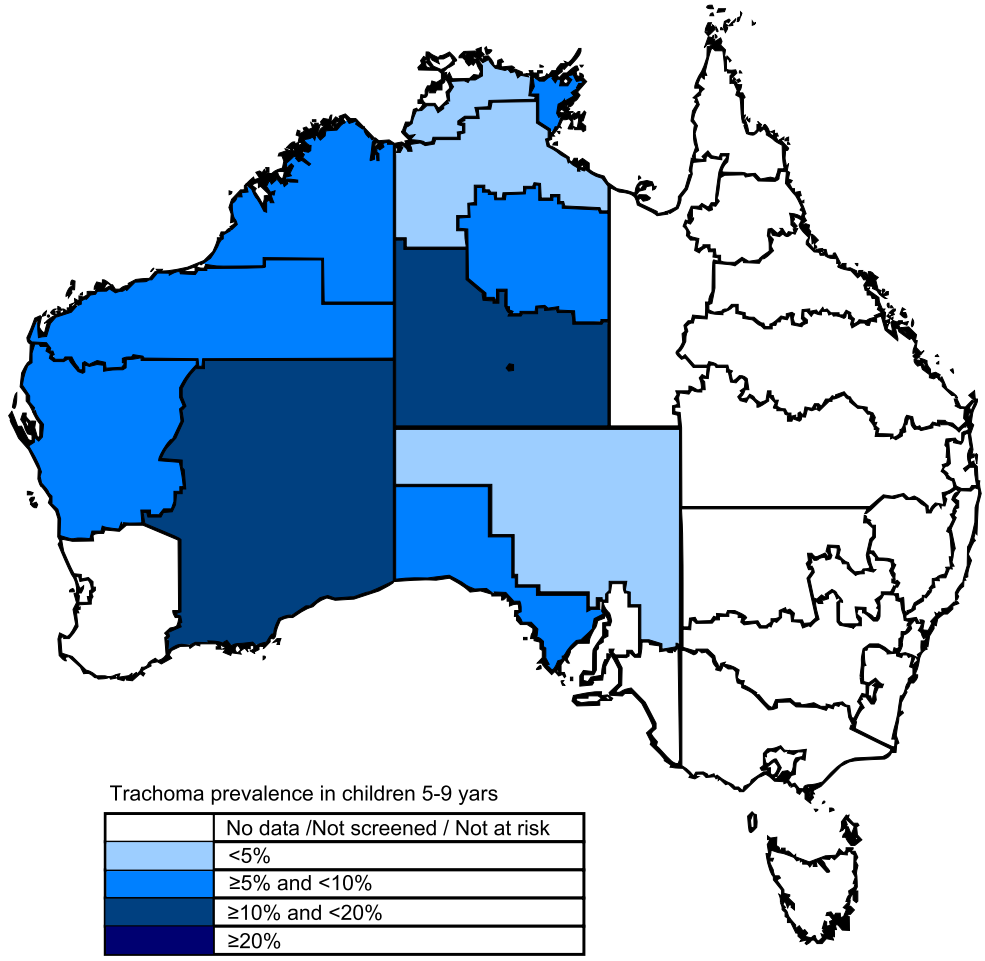
Health promotion resources and programs to promote trachoma and trichiasis awareness, facial cleanliness, and personal and environmental hygiene, and environmental conditions were not reported for the majority of communities and therefore data are not presented here.

Results

Trachoma

In 2011, a total of 152 (73%) of 207 designated at-risk communities were screened for trachoma across 11 endemic regions in the Northern Territory, South Australia and Western Australia (Figure 1). In communities screened for trachoma, 4,746 (65%) of an

Figure 1: Trachoma prevalence in children aged 5-9 years in screened communities, 2011, by region



The numerator and denominator associated with each region refer to the number of communities screened and the number of at-risk communities within each region, respectively.

Table 1: Trachoma screening coverage and prevalence among at-risk communities, 2011, by state or territory

	Northern Territory				South Australia				Western Australia				Total			
Number of communities at risk	86				46				75				207			
Number of communities screened	65				19				68				152			
Age group (years)	**1-4**	**5-9**	**10-14**	**Total**	**1-4**	**5-9**	**10-14**	**Total**	**1-4**	**5-9**	**10-14**	**Total**	**1-4**	**5-9**	**10-14**	**Total**
Estimated Aboriginal population at risk	3,637	3,909	3,653	11,199	938	931	935	2,804	2,265	2,498	2,095	6,858	6,840	7,338	6,683	20,861
Children examined for trachoma	439	2,530	1,785	4,754	151	718	468	1,337	203	1,498	946	2,647	793	4,746	3,199	8,738
Screening coverage	12%	65%	49%	42%	16%	77%	50%	48%	9%	60%	45%	39%	12%	65%	48%	15%
Children with active trachoma	19	175	91	285	2	29	5	36	3	123	38	164	24	327	134	485
Active trachoma prevalence	4%	7%	5%	6%	1%	4%	1%	3%	1%	8%	4%	6%	3%	7%	4%	6%
Trachoma prevalence 1-9 years (weighted by population)*	6%				3%				5%				5%			
Range of trachoma prevalence in 5-9 year olds in regions within jurisdictions	4% - 14%				3% - 7%				5% - 12%				3% - 14%			

* Calculated as the proportions of children with active trachoma in age groups 1-4 and 5-9 years, weighted by the estimated population sizes of each age group. This was done in order to account for uneven coverage with respect to age groups.

Table 2: Number of trachoma cases requiring treatment, and treatment coverage among at-risk communities, 2011, by state or territory

	Northern Territory					South Australia					Western Australia					Total				
Number of communities at risk	86					46					75					207				
Number of communities requiring treatment	43					8					29					80				
Age group (years)	**0-4**	**5-9**	**10-14**	**15+**	**All**	**0-4**	**5-9**	**10-14**	**15+**	**All**	**0-4**	**5-9**	**10-14**	**15+**	**All**	**0-4**	**5-9**	**10-14**	**15+**	**All**
Active cases requiring treatment	19	175	91	N/A	285	2	29	5	N/A	36	3	123	38	N/A	164	24	327	134	N/A	485
Active cases who received treatment	19	150	66	N/A	235	2	27	5	N/A	34	3	121	35	N/A	159	24	298	106	N/A	428
% Active cases received treatment					82%					94%					97%					88%
Estimated contacts requiring treatment (according to jurisdictional interpretation of guidelines)					8,772					466					1,304					9,509
Number of contacts who received treatment	626	841	512	2,636	4,615	36	51	52	316	455	118	254	177	556	1,105	780	1,146	741	3,508	6,175
Estimated overall treatment coverage *					53%					98%					85%					65%

* Estimated using average number of household contacts per child in communities who reported number of contacts requiring treatment and population statistics (see Methodology for deta

Table 3: Trichiasis screening coverage, prevalence and treatment among Aboriginal adults aged over 40 years, 2011, by state or territory

	Northern Territory	South Australia	Western Australia	Total
Adult population size of at-risk communities	7,007	1,921	4,538	13,466
Number of communities at-risk	86	46	75	207
Number of communities screened for trichiasis (% of at risk communities)	8 (9%)	7 (15%)	5 (7%)	20 (10%)
Number of adults examined (% of adults population)	212 (3%)	712 (37%)	255 (6%)	1,179 (9%)
Number of adults with trichiasis (% of adults examined)	9 (4%)	8 (1%)	2 (1%)	19 (2%)
Number of adults offered ophthalmic consultation	1	2	3	6
Number of adults receiving trichiasis surgery in past 12 months	0	0	0	0

estimated 7,338 resident children aged 5-9 years were screened. In 2011, screening coverage for children aged 5-9 years in the Northern Territory, 77% in South Australia and 60% in Western Australia (Table 1) (Figures 2 and 3).

In screened at-risk communities in all jurisdictions, the prevalence of trachoma among children aged 5-9 years, was 7%; comprising 7% in the Northern Territory, 4% in South Australia, and 8% in Western Australia. There was a decreasing trend in trachoma prevalence in the Northern Territory and Western Australia compared to data from the previous three years (Figure 4). Prevalence ranges of children aged 5-9 years within regions were between 4% and 14% Northern Territory, 3% and 7% within regions in South Australia and between 5% and 12% in Western Australia. The overall prevalence of trachoma among 1-9 year old children in screened communities was 5% (weighted by population).

In approximately half (47%; 72/152) of at risk communities screened no active trachoma was detected. The proportion of screened communities with no trachoma detected in 2011 increased in the Northern Territory and Western Australia, compared to 2010 and the proportion with endemic trachoma (>5% prevalence) decreased in these jurisdictions (Figures 5 and 6). In 14% (21/152) of at risk communities screened, hyper-endemic levels (>20% prevalence) of trachoma were found and in all participating jurisdictions (Figure 7). The highest prevalence in a single community was 60%, found in Western Australia, where fewer than 10 children were screened. A decreasing trend has been observed in communities that have been consistently screened and treated accordingly from 2007 to 2011. Low screening coverage in previous years in South Australia prevented the examination of time trends in trachoma in this state.

Treatment coverage

Active trachoma cases requiring treatment were detected in 80 of 152 communities screened, with 88% of active cases found to be receiving appropriate treatment for trachoma. Treatment coverage of contacts of the active cases detected with trachoma was 65% overall (Table 2). Since 2009, the Northern Territory has also undertaken 6-monthly treatment in hyper-endemic communities (>20% prevalence of trachoma). This strategy was undertaken in more communities in 2010, particularly in the Alice Springs remote region and may have contributed to the notable decrease in trachoma in that region, from 33% in 2010 to 14% in 2011.

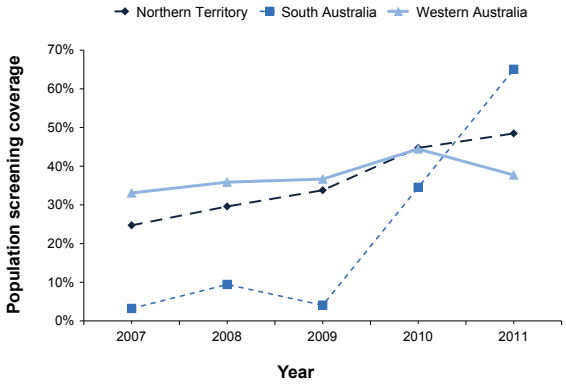
Trichiasis

Trichiasis screening coverage was low in all jurisdictions, with 1,179 adults (less than 10%) of an estimated at-risk population of 13,466 reported to have been screened across the Northern Territory, South Australia and Western Australia (Table 3). Overall trichiasis prevalence among those screened was 2% (n=19). No trichiasis surgery was reported by the jurisdictions. Screening coverage and prevalence of trichiasis has remained stable from 2010 – 2011.

Facial cleanliness

For individuals, the overall prevalence of clean faces in children aged 5-9 years was 76%, with 74% in the Northern Territory, 88% in South Australia and 75% in Western Australia, generally stable since 2010. The proportion of screened at-risk communities

Figure 2: Population screening coverage of children aged 5-9 years, 2007 to 2011, by year and state or territory



The numerator and denominator associated with each region refer to the number of communities screened and the number of at-risk communities within each region, respectively.

Figure 3: Trachoma prevalence among screened children aged 5-9 years, 2007 to 2011, by year and state or territory

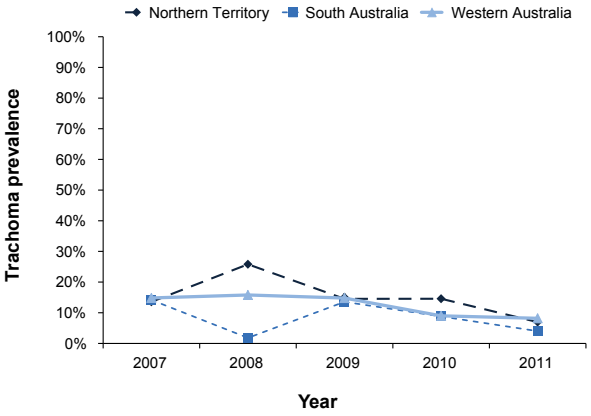
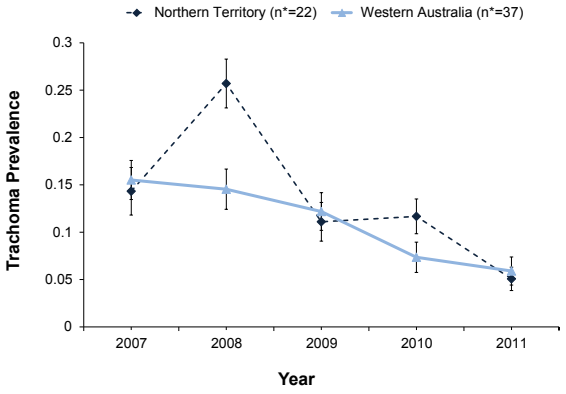


Figure 4: Trachoma prevalence* among children aged 5-9 years, in communities consistently screened* 2007 to 2011, by year and state or territory



* with 95% confidence intervals

† With at least 10 children screen each year between 2007 and 2011

Figure 5: Proportion of screened communities in which no trachoma was reported among children aged 5-9 years, 2007 to 2011, by year and state or territory

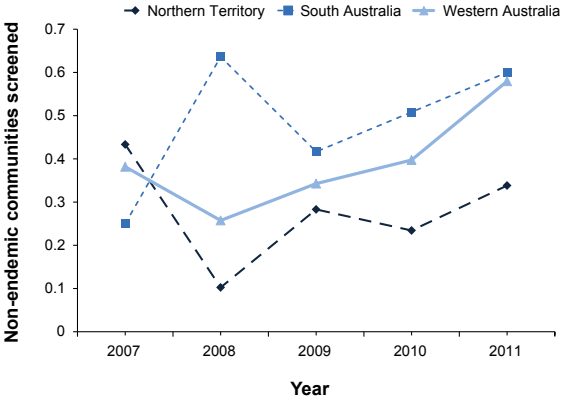
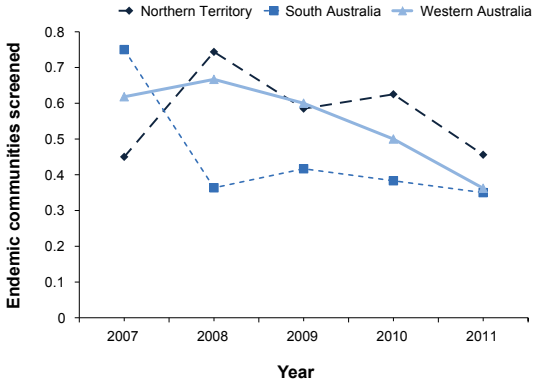
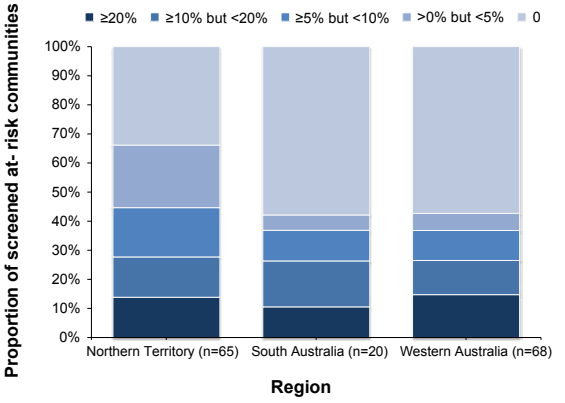


Figure 6: Proportion of screened communities with endemic trachoma* among children aged 5-9 years, 2007 to 2011, by year and state or territory



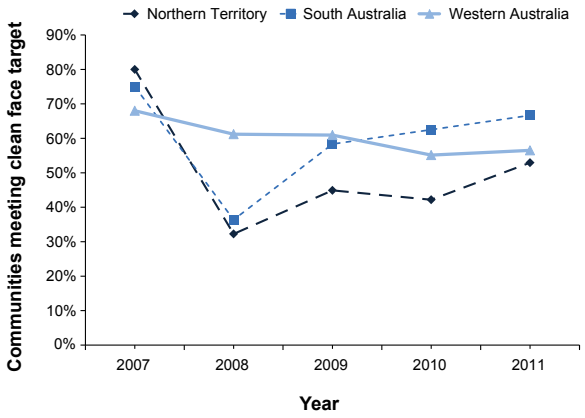
* Prevalence greater than 5%

Figure 7: Proportion of screened at-risk communities according to level of trachoma prevalence among children aged 5-9 years, 2011, by trachoma prevalence and state or territory



n denotes number of communities screened

Figure 8: Percentage of screened communities meeting clean face target in children aged 5-9 years, 2007 to 2011, by year and state or territory



meeting the target of facial cleanliness of 80% among screened children aged 5-9 years in 2011 was 53% in the Northern Territory, 67% in South Australia and 57% in Western Australia (Figure 8).

Discussion

Screening coverage

Screening coverage was measured as both the proportion of at-risk communities screened and the proportion of 5-9 year old children screened in at-risk communities. Screening was predominantly conducted through primary school-based initiatives. Screening of older (10-14 year old) and younger (1-4 year old) children also took place, but less consistently. By both screening measures, the screening coverage substantially improved in South Australia in 2011. Coverage of 5-9 year old children has improved steadily in the Northern Territory and Western Australia over the past four years.

Trachoma prevalence in screened communities

Endemic trachoma is defined as a prevalence of active trachoma of 5% or more in screened communities among children aged 1-9 years. Although the focus of screening was among 5-9 year old children, the prevalence in the larger age band was estimated from available data. Across all three jurisdictions in 2011, the prevalence of trachoma among 1-9 year old children in screened communities was 5% (unweighted by population, ranging from 0% to 60%), representing a decrease from the 2010 combined prevalence of 13% (unweighted by population, ranging from 0% to 75%). At a regional level, the prevalence of trachoma among children aged 5-9 (focus of screening activities in 2011 across all jurisdictions was in the 5-9 year age group) years ranged from 3% to 14%.

There is strong evidence of a decrease in overall trachoma prevalence in screened communities in the Northern Territory and Western Australia. This conclusion was also reached when analyses were restricted to the communities that had been screened, and treated every year since 2007. The number of communities found to have a prevalence of above 5% (endemic trachoma) in screened children aged 5-9 years in the Northern Territory and Western Australia decreased, and there was an increasing trend in the number of communities that reported no trachoma in screened children aged 5-9 years.

The target set by both WHO and CDNA for the elimination of blinding trachoma is a community prevalence in children aged 1-9 years of less than 5%, over a period of five years. Several communities designated as being at-risk have reported prevalence levels of less than 5% over the past three years, and are therefore on track to be designated as not at risk if this status is maintained for two more years. However, overall there is more progress to be made with many communities still exceeding endemic trachoma levels.

Trachoma treatment

The CDNA guidelines recommend the treatment of active cases and their household contacts. When prevalence is greater than 10% and cases are not clustered within a few households, "community wide" treatment is advised. The approach to community-wide treatment differs between jurisdictions. In the Northern Territory, interpretation of the recommendation has been applied to treating the entire community, whereas in South Australia and Western Australia all children aged between 6 months and 14 years and household contacts are treated.

Across all three jurisdictions, 65% of those found through screening to have trachoma, or to be the household contact of an active case, were recorded as having been treated appropriately. Of active cases across all jurisdictions, 88% received treatment. At the jurisdictional level, 53%, 98% and 85% of the population requiring treatment were treated in the Northern Territory, South Australia and Western Australia, respectively. This variance in coverage may reflect the differing treatment strategies undertaken by the jurisdictions. In 2010, 57% of active cases were treated according to CDNA guidelines, and 70% of contacts across all jurisdictions received treatment. At the jurisdictional level in 2010, 64% of contacts in the Northern Territory and 91% in Western Australia were treated. Data regarding treatment for trachoma in South Australia was not made available in 2010. WHO and current CDNA targets are for 100% treatment of active cases and contacts.[2,8]

Trichiasis

Screening rates for trichiasis among Aboriginal adults aged over 40 years across all jurisdictions remained low, and therefore, the reporting systems may not provide an accurate estimate of trichiasis prevalence in Aboriginal communities. Furthermore, prevalence figures only include data collected in communities currently designated as communities at risk of trachoma and do not take into account the possibility that endemic areas have changed over time. The limited number of adults screened for trichiasis in all jurisdictions, the prevalence of trichiasis in each community screened was 1% or greater, which indicates endemic trachoma in those communities. Ophthalmic referral processes were reported to be functioning within the majority of communities, but the effectiveness of the system has not been verified. In 2011, no episodes of trichiasis surgery were reported. However, this may not reflect the true level of ophthalmic consultation and surgical activities.

Facial cleanliness

Facial cleanliness is a major component of the SAFE strategy. The WHO sets a target of 80% facial cleanliness for children screened within communities. This high target illustrates that the presence of nasal and ocular discharge is a significant risk factor for both acquiring and transmitting trachoma. All jurisdictions had levels of facial cleanliness either just under or over this target of 80%. Ongoing health promotion endorsing facial cleanliness and general positive personal and environmental hygiene practices will improve facial cleanliness prevalence and decrease the risk of trachoma transmission.

Environment

Data on environmental conditions were not well reported in 2011, with the majority of communities not providing relevant data. Early in 2012 the Trachoma Surveillance and Control Reference Group (TSCRG) decided that the previously used methods of data collection did not accurately capture the environmental conditions that are recognised to influence trachoma prevalence and transmission. These conditions include adequate housing to decrease overcrowding; functional housing hardware including taps, showers and washing machines; effective maintenance systems for housing hardware; and adequate community waste disposal systems to prevent flies. The TSCRG and NTSRU are currently collaborating with environmental health agencies to develop more effective reporting processes for this component of the SAFE strategy.

Data quality and surveillance systems

Despite considerable improvement in several aspects of program delivery and monitoring in 2011, there are several issues that remain to be adequately addressed.

The analyses in this report have used population denominator estimates based on projections from census figures. These estimates are recognised as having the potential for substantial error in communities that are small or for which there is considerable mobility of community members, however we have no means for determining the extent or direction of any bias that may be present.

There are differences apparent across jurisdictions in the interpretation of the 2006 CDNA *Guidelines for the Public Health Management of Trachoma in Australia*. There is also a need to ensure that the guidelines are up to date. In 2011, the CDNA agreed to undertake a review of the Guidelines to incorporate the latest information on the screening, treatment and management of trachoma. The document is central to supporting trachoma control programs in the Northern Territory, South Australia and Western Australia, and new programs being established in New South Wales and Queensland. The Trachoma Framework Review Working Group, acting as a CDNA subcommittee, will guide the review process, and the NTSRU will manage the review process.

During 2011, the NTSRU developed a web-based interface program to increase the likelihood of consistent reporting across jurisdictions and regions through the use of a standard and simple to use data entry system. The system also allows for more efficient data validation and reporting to stakeholders, including communities. It is anticipated that all components of the web-interface data entry and reporting system will be fully operational in the course of 2012, and this should improve the quality of the data.

Progress towards Australia's elimination target

As a signatory to the WHO Alliance of Global Elimination of Trachoma by the year 2020, Australia is committed to ensuring that trachoma levels continue to decrease to below endemic levels in at-risk communities through increasing and improving effectiveness of all components of the SAFE strategy. This report provides evidence of increasing coverage of trachoma screening and control activities. In the Northern Territory and Western Australia, there is also evidence of a decline in the prevalence of infection that may be attributable to improvement in control activities. Despite these apparent advances, trachoma prevalence remains at endemic levels in many communities of remote Australia. Continued efforts are required to ensure that Australia remains on track to reach the goal of elimination by 2020 or sooner.

Acknowledgements

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Jurisdictional contributors to trachoma data collection

Northern Territory

- Aboriginal Community Controlled Health Services
- Aboriginal Medical Services Alliance of the Northern Territory
- Centre for Disease Control, Northern Territory Department of Health, Northern Territory
- Healthy School Age Kids Program: Top End and Central Australia

South Australia

- Aboriginal Community Controlled Health Services
- Aboriginal Health Council of South Australia
- Country Health South Australia

Western Australia

- Aboriginal Community Controlled Health Services
- Communicable Diseases Control Directorate, Health Department of Western Australia
- Goldfields Population Health Unit
- Kimberley Population Health Unit
- Midwest Population Health Unit
- Pilbara Population Health Unit

The National Trachoma Surveillance and Control Reference Group

The NTSRU is guided by the National Trachoma Surveillance and Control Reference Group, members of which include representatives from the following organisations:

- Office for Aboriginal and Torres Strait Islander Health; Department of Health
- National Aboriginal Community Controlled Health Organisations
- Communicable Disease Network Australia
- Northern Territory Department of Health
- Western Australia Country Health Service
- Country Health South Australia
- Melbourne School of Population Health, University of Melbourne
- Population & Preventive Health, University of Notre Dame
- The National Trachoma Surveillance and Reporting Unit; The Kirby Institute, University of New South Wales

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Supplementary report: surveillance of adverse events following immunisation among children aged less than seven years in Australia, 1 January to 30 June 2012

Deepika Mahajan, Jane Cook, Aditi Dey, Kristine Macartney, Rob Menzies

Key words: AEFI, adverse events, vaccines, surveillance, immunisation, vaccine safety

Introduction

This report summarises national passive surveillance data reported to the Therapeutic Goods Administration (TGA) to 31 August 2012 for adverse events following immunisation (AEFI) for children aged <7 years who received vaccines between 1 January and 30 June 2012. The report includes all vaccines administered to children in this age group with a focus on the vaccines included in the funded National Immunisation Program (NIP) schedule.[1]

Recent changes to the NIP have impacted on AEFI surveillance data presented in this report. On 1 July 2011, Prevenar® (7-valent pneumococcal conjugate vaccine, 7vPCV) was replaced with Prevenar 13® (13-valent pneumococcal conjugate vaccine, 13vPCV) for children at 2, 4 and 6 months and a 4th dose for medically at risk children at 12 months of age in all states and territories except the Northern Territory (which adopted 13vPCV from 1 October 2011).[2] In addition, children aged between 12 and 35 months who had completed a primary course with 7vPCV were eligible to receive a free supplementary dose of Prevenar 13® from 1 October 2011 to 30 September 2012. Also from 1 October 2011, the Northern Territory Government provided a free dose of Prevenar 13® at 18 months for children who had previously received a primary course of Synflorix® (10vPCV) or a mixed primary pneumococcal course with Synflorix® and Prevenar®.[3]

Methods

Case definition and coding

The data reported here are provisional only. It is important to note that an AEFI is defined as a medical event that is temporally but not necessarily causally associated with immunisation. Readers are referred to previous reports for a description of the national AEFI passive surveillance system, methods used to analyse the data and information regarding limitations and interpretation of the data.[4-9] Often several vaccines and reaction codes are listed in an AEFI record so that the number of vaccines and reaction codes will exceed the total number of AEFI records. For the purpose of this report, an AEFI is defined as 'serious' if it is life-threatening, had recovery with sequelae, or if it was associated with admission to hospital, prolongation of hospitalisation, or death.

Denominator calculations

Average annual population-based AEFI reporting rates were calculated using mid-2011 population estimates. Reporting rates per 100,000 doses were calculated for 10 vaccines on the NIP schedule for which reliable dosing data were available from the Australian Childhood Immunisation Register (ACIR), for children from birth to age <7 years.

Results

There was a total of 484 AEFI records (annualised reporting rate of 47.9 per 100,000 population) for NIP and non-NIP vaccines administered to children aged <7 years in the first 6 months of 2012. This was lower than the corresponding period in 2011 (532 records; 52.7 per 100,000 population). Of the 484 records, 37 (8%) were events defined as being 'serious' i.e. recovery with sequelae, requiring hospitalisation, experiencing a life-threatening event or death. All AEFI records were assigned a causality rating. Eighteen percent (n=87) were rated as 'certain', 1% 'probable' (n=5), while the rest were 'possible'. Forty-one percent (n=200) of records were for children aged <1 year, 17% (n=81) for those aged 1 to < 2 years and 42% (n=203) were for the 2 to <7 year age group. The male to female ratio was 1.2:1, similar to previous years.[5,6]

Eighty-eight percent of AEFI (n=424) were reported to the TGA via states and territories. The remainder were reported directly to the TGA, 9% (n=42) by doctors/health care providers, 2% (n=8) by members of the public, 1% (n=7) by hospitals and 0.6% (n=3) by pharmaceutical companies.

Thirty-seven reports listed one or more vaccine(s) for which accurate dose denominator data (number of people who received the vaccine) were not available from the ACIR. These were influenza (n=24), bacille Calmette-Guérin (BCG) (n=7), hepatitis B (n=6), 23-valent pneumococcal polysaccharide

(n=4), and hepatitis A (n=2) vaccines. AEFI reporting rates per 100,000 doses were calculated for the remainder of records (n=447) (Table).

The overall AEFI rate for those reports for which accurate dose denominator data were available was 20.0 per 100,000 doses, with 1.5 per 100,000 classified as being 'serious' which is slightly lower than for the same period in 2011 (25 per 100,000 and serious 2.3 per 100,000 doses respectively). In 2012, reporting rates were similar to or lower than those in 2011 for all age groups and vaccine types (Table). There was a 28% reduction in reports for children aged 2 to <7 years, and no change in other age groups. There were substantial decreases in reported AEFI following receipt of *Haemophilus influenzae* type b vaccine (Hib) (39%); diphtheria tetanus acellular pertussis inactivated poliomyelitis (DTPa-IPV) (19%); measles mumps rubella (MMR) (18%); hexavalent (DTPa-IPV-HepB-Hib) (16%); meningococcal C conjugate (MenC) (15%); varicella (13%) and rotavirus (6%) (Figure). During 2012, pneumococcal conjugate vaccine was suspected of involvement in 180 events (173 for 13vPCV and 7 for 7vPCV) being for children aged <7 years with 15 coded as being serious, all for 13vPCV, consistent with vaccine usage i.e. with 13vPCV replacing 7vPCV in July 2011.

Table: Rates of AEFI per 100,000 vaccine doses, children aged less than 7 years, TGA database, January to June 2012

	Jan-Jun 2011		Reporting rate per 100,000 doses† (95% CI)	
	AEFI records* n	Vaccine doses§ n	Jan–June 2012	Jan–June 2011
Vaccine (NIP vaccines)‡				
DTPa-containing vaccines	333	582,301	57 (51.2-63.7)	68 (61.1-75.4)
DTPa-IPV	177	164,040	108 (92.6-125.0)	133 (114.5-153.1)
Pentavalent (DTPa-IPV-HepB)	1	95	NA*	0
Hexavalent (DTPa-IPV-HepB-Hib)	155	418,166	37 (31.5-43.4)	44 (37.8-51.3)
Haemophilus influenzae type b	24	141,139	17 (10.9-25.3)	28 (20.0-38.8)
Haemophilus influenzae type b-hepatitis B	0	127	0	0
Measles-mumps-rubella	139	309,159	45 (37.8-53.1)	55 (47.0-64.8)
Meningococcal C conjugate	33	148,609	22 (15.3-31.2)	26 (18.5-36.2)
Pneumococcal conjugate (7vPCV)	7	25,423	28 (11.1-56.7)	40 (34.1-47.1)
Pneumococcal conjugate (13vPCV)	173	495,731	35 (29.9-40.5)	NA
Varicella	29	144,068	20 (13.5-28.9)	23 (14.9-31.2)
Rotavirus	153	339,487	45 (38.2-52.8)	48 (40.9-56.5)
Age group				
<1 year	187	1,188,402	16 (13.6-18.2)	18 (15.6-20.9)
1 to <2 years	72	602,677	12 (9.3-15.0)	13 (9.8-16.2)
2 to <7 years	188	394,965	48 (41.0-54.9)	68 (59.5-78.3)
AEFI category‡				
Total	**447**	**2,186,044**	**20 (18.6-22.4)**	**25 (22.8-27.5)**
'Certain' or 'probable' causality rating	84	2,186,044	3.8 (3.1-4.8)	4.0 (3.3-5.3)
'Serious' outcome	33	2,186,044	1.5 (1.0-2.1)	2.3 (1.7-3.1)

* Number of AEFI records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 30 June 2012. More than one vaccine may be coded as 'suspected' if several were administered at the same time.

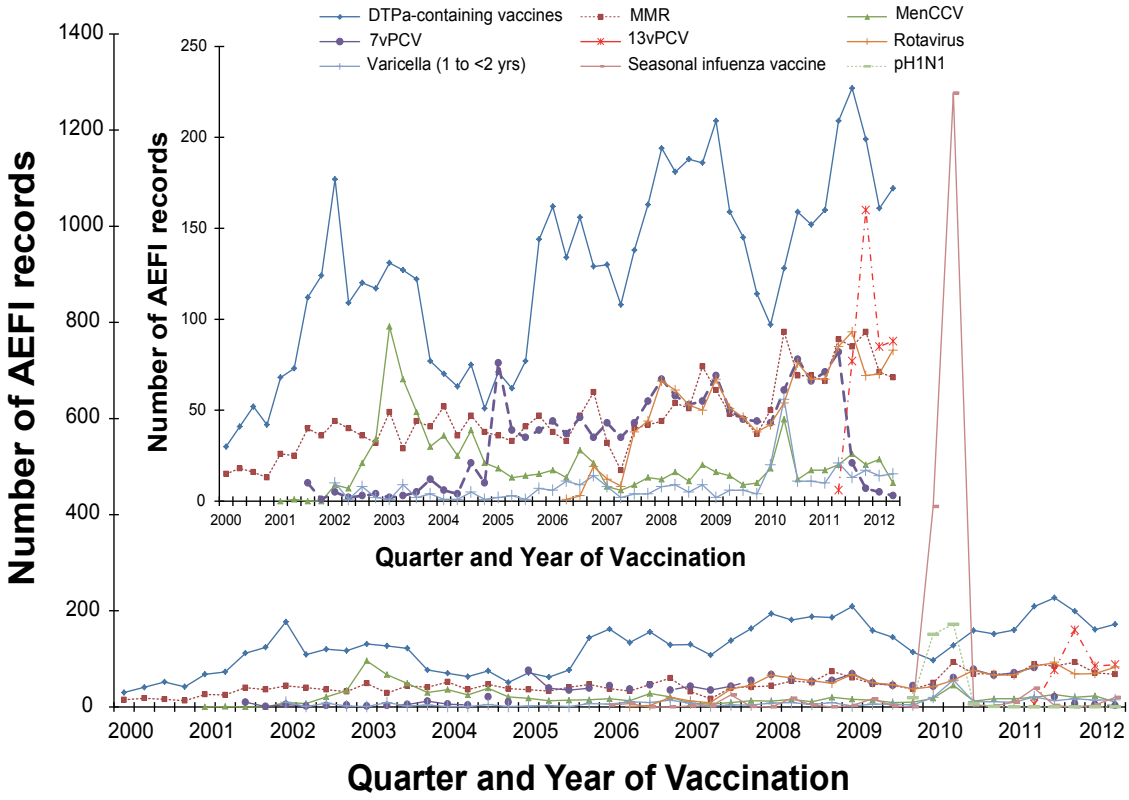
† The estimated AEFI reporting rate per 100,000 vaccine doses recorded on the ACIR.

‡ Records where at least one of the ten vaccines shown in the table was suspected of involvement in the reported adverse event. AEFI category includes all records (i.e. total), those assigned 'certain' or 'probable' causality ratings, and those with outcomes defined as 'serious'. Causality ratings were assigned using the criteria described previously.[8] A 'serious' outcome is defined as recovery with sequelae, hospitalisation, life-threatening event or death.

§ Number of vaccine doses recorded on the Australian Childhood Immunisation Register (ACIR) and administered between 1 January and 30 June 2012.

NA Not applicable

Figure: Reports of AEFI, TGA database, 1 January 2000 to 30 June 2012, for vaccines recently introduced onto the NIP*



Inset excludes pH1N1 and seasonal influenza vaccine.

* Meningococcal C conjugate vaccine (MenCCV) was introduced into the NIP schedule on 1 January 2003; 7-valent pneumococcal conjugate vaccine (7vPCV) on 1 January 2005; DTPa-IPV and DTPa-IPV-HepB-Hib vaccines in November 2005; and Rotavirus (RotaTeq® and Rotarix®) vaccines 1 July 2007. In early 2008, Queensland, South Australia and Victoria changed from DTPa-IPV to DTPa-IPV-HepB-Hib for children at 2, 4 and 6 months of age. pH1N1 influenza vaccine for children 6 months to 10 years on December 2009; seasonal trivalent influenza vaccine in 2010; and the 13-valent pneumococcal conjugate vaccine (13vPCV) on 1 July 2011.

The most commonly reported reaction categories were injection site reaction (ISR) (n=196;40%), fever (n=122;25%), allergic reactions (n=95;20%), rash (n=69;14%), gastroenteritis following rotavirus vaccination (n=55;11%), screaming (n=38;8%) and seizure (n=23;5%). The largest number of reports were from Victoria (41%) followed by Queensland (17%), Western Australia (15%), New South Wales (13%), and South Australia (7%).

Of the 196 reports of ISR, 85% were following DTPa-containing vaccines (73% with DTPa-IPV vaccine and 10% with hexavalent DTPa-IPV-HepB-Hib vaccine given either alone or conjointly with other vaccines). Seventy-six percent (n=149) of the reported ISR were from children aged 2 to <7 years and 95% (n=142) of those were following vaccination with DTPa-containing vaccines (139/142 were following vaccination with DTPa-IPV vaccine administered alone [37] or conjointly [102] with other vaccines).

Eight percent (n=37) of the 484 AEFI records had outcomes defined as being 'serious', a rate of 1.5 per 100,000 doses which was lower than the corresponding period in 2011 (2.3 per 100,000). There was one report of a life-threatening event and all the events in children (n=37) defined as being 'serious' were admitted to hospital, with no reported deaths.

The report of a life-threatening event was a premature infant who developed febrile seizures, encephalopathy and laryngospasm eight hours following vaccination with seasonal influenza vaccine (Fluvax®). The child inadvertently received the Fluvax® brand of influenza vaccine which has not been registered for use in <5 year olds since April 2010.[10]

Forty-one percent (n=15) of the 'serious' reports were following vaccination with hexavalent DTPa-IPV-HepB-Hib, 13vPCV, and rotavirus vaccines co-administered together. Serious and other significant AEFI included convulsions (n=23; 11 were serious of which 10 were hospitalised), hypotonic-hyporesponsive episodes (HHE, n=10; 3 hospitalised), intussusception (n=8; 5 hospitalised) and one case of idiopathic thrombocytopenic purpura (ITP) who was also hospitalised. Of the 10 cases of convulsions requiring hospitalisation, 7 were febrile convulsions. There were 15 reports of febrile convulsions in total. The most common vaccines given on their own cited in reports of convulsions were seasonal influenza vaccine (n=2),

DTPa-IPV (n=1), MMR (n=1), and varicella (n=1). The other reports of convulsions were following co-administration of hexavalent DTPa-IPV-HepB-Hib /13vPCV/rotavirus (n=5), Hib/MenC/MMR (n=3), DTPa-IPV/MMR (n=3), Hib/MenC/13vPCV/MMR (n=2), and one each of hexavalent DTPa-HepB/IPV-Hib-13vPCV-MenC, DTPa-IPV/MMR/varicella, HepB/MenC, HepB/Hib/MMR, and 23vPPV/Hep A/varicella vaccines.

Nine of the 10 reports of HHE were following receipt of DTPa-containing vaccines, with hexavalent DTPa-IPV-HepB-Hib/ 13vPCV/rotavirus given conjointly in 8 reports and hexavalent DTPa-IPV-HepB-Hib/ 7vPCV/rotavirus in one report. The report following non-DTPa vaccines were Hib/MenC/MMR.

There were 8 reports of intussusception in 2012; 6 occurred following receipt of hexavalent DTPa-IPV-HepB-Hib/13vPCV/rotavirus, one report hexavalent DTPa-IPV-HepB-Hib/7vPCV/rotavirus administered together while one report was following rotavirus vaccine administered alone.

The only case of ITP was an infant following administration of Hib/MenC/MMR vaccines 3 days post vaccination and was most likely due to MMR.

Discussion

There was a slight decrease in the total number of AEFI records and population-based reporting rates for the first six months of 2012 compared with the corresponding period in 2011.

Reporting rates per 100,000 doses for <1 year olds and 1 to <2 year olds were similar to the corresponding period in 2011, but significantly lower for children aged 2 to<7 years [48 (95% CI: 41.0 to 54.9) vs 68 (59.5 to 78.3)]. The decrease in reporting of AEFI in children aged 2 to <7 years in 2012 is primarily because of the drop in the reporting of ISR following vaccination with DTPa-IPV in that age group in 2012 compared to 2011. There was an increase in DTPa-IPV related ISR in 2 to <7 year olds in 2011 which might partly be due to general changes in AEFI surveillance nationally, discussed in a previous report.[5] Although reporting rates for DTPa-IPV vaccines were lower in 2012 compared to 2011, reporting was still higher than in previous years (78 in 2008; 82 in 2009; 78 in 2010) and therefore maintaining an upward trend.[6,7]

The increase in the reports following rotavirus vaccine may be because in the majority of the cases (86%), rotavirus vaccine was administered with 13vPCV and hexavalent vaccine. The chance of developing at least one AEFI with the administration of multiple vaccines is greater than with just one vaccine. Since October 2011, children aged between 12 and 35 months who had completed a primary pneumococcal vaccination course with 7vPCV have been eligible to receive a free supplementary dose of Prevenar 13®.[2] The increased AEFI reports following 13vPCV might be in part because it is being given as a 4th dose of PCV vaccine. Data from the clinical studies of Prevenar 13® demonstrated similar rates of injection site reactions when comparing 7vPCV with 13vPCV, with an increase following the 4th dose of either 7vPCV or 13vPCV in the second year of life compared with earlier doses in infancy. A similar trend was also observed for the other systemic reactions.[11] Some may also be attributed to the 'Weber effect',[12] which describes increased reporting frequently observed following the introduction of new vaccines.

Conclusion

The total number of AEFI reported in children aged <7 years in the first half of 2012 was lower than in the same period in 2011. Reports of ISR following DTPa-IPV at 4 years decreased in 2012 compared to 2011 but were still higher than in previous years. Reporting rates for most of the vaccines were similar to or lower in 2012, particularly in the 2 to <7 year age group.

The majority of AEFIs reported to the TGA were mild transient events and the data reported here are consistent with an overall high level of safety for vaccines included in the NIP schedule.

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The Australian Immunisation Handbook

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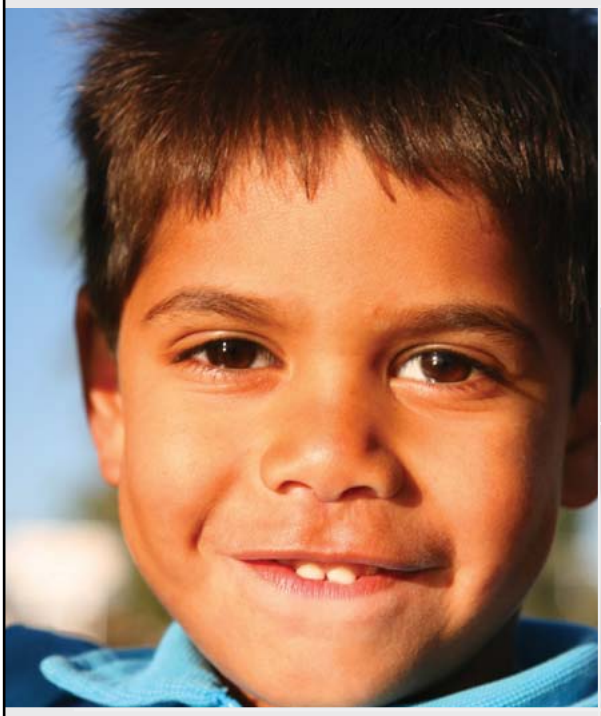


On the 28 March 2013, The Hon Tanya Plibersek MP, Minister for Health, launched the 10th edition of the Australian Immunisation Handbook.

Developed by the Australian Technical Advisory Group on Immunisation, and approved by the National Health Medical Research Council, the latest edition of the *Handbook* introduces new vaccines, contains new and updated recommendations on vaccine use and outlines the importance of vaccination during pregnancy.

New vaccines to the immunisation schedule include extending the Human Papillomavirus (HPV) vaccine to boys, the new combined MMRV vaccine, and a replacement pneumococcal vaccine.

The *Handbook* includes important information about catch-up vaccination schedules, vaccination for special risk groups, vaccination for groups with special requirements, and vaccination for international travel. An easy-to-read summary table is included which provides recommendations for vaccines during pregnancy. There is also information about managing rabies and Australian bat lyssavirus exposures.

In April 2013, copies of the *Handbook* were distributed to Australian immunisation providers including general practitioners, specialist doctors (e.g cardiologists, gerontologists, gynaecologists, obstetricians, oncologists and paediatricians), nurses, midwives, Aboriginal Health workers and travel clinics.

The *Handbook* has also been sent to hospitals, medical colleges, state and territory health units, Medicare Locals, National Aboriginal Community Controlled Health Organisations, Immunisation Committees, non-government organisations, pharmaceutical companies, migrant health services, health insurance companies, universities and libraries.

An electronic version of the *Handbook* is available on the Immunise Australia at www.immunise.health.gov.au. Copies of The *Handbook* can be ordered on the Immunisation Australia website - Publications & Resources.

Enquiries regarding the *Handbook* can be forwarded to handbook@health.gov.au

IMMUNISATION

Original articles

Assessing the threat of chikungunya virus emergence in Australia

Elvina Viennet, Katrina Knope, Helen Faddy, Craig Williams, David Harley

Abstract

Background: Chikungunya virus (CHIKV) is a major threat to Australia given the distribution of competent vectors, and the large number of travellers returning from endemic regions. We describe current knowledge of CHIKV importations into Australia, and quantify reported viraemic cases, with the aim of facilitating the formulation of public health policy and ensuring maintenance of blood safety.

Methods: Cases reported to the National Notifiable Disease Surveillance System (NNDSS) from 2002 to 2012 were analysed by place, month of acquisition, and place of residence. Rates of chikungunya importation were estimated based on reported cases and on the numbers of short-term movements.

Results: Between 2002 and 2012, there were 168 cases of chikungunya virus (CHIKV) imported into Australia. Victoria and New South Wales had the largest number of notifications. The main sources were Indonesia, India and Malaysia. The number of cases increased from 2008 to reach a peak in 2010 (n=64; 40%). Although Indonesia accounted for the majority of CHIKV notifications in Australia, travel from India had the highest CHIKV importation rate (number of imported cases per 100,000 travellers).

Conclusions: The Australian population is increasingly at risk from CHIKV. Arrivals from endemic countries have increased concurrently with vector incursions via imported goods, as well as via local movement from the Torres Strait to North Queensland ports. An outbreak of CHIKV could have a significant impact on health, the safety of the blood supply and on tourism. Case and vector surveillance as well as population health responses are crucial for minimising any potential impact of CHIKV establishment in Australia.

Keywords: Chikungunya, importation, risk, travellers, Australia, vectors, viraemic cases

Introduction

Chikungunya virus (CHIKV) is a mosquito-borne Alphavirus of the family *Togaviridae*. The virus is endemic in Africa, India, South-East Asia and the Western Pacific and is considered to be emerging or re-emerging in many regions of the world.[1-4] The disease was first detected in 1952 in Africa following an outbreak on the Makonde Plateau.[5,6] The name chikungunya is derived from the Makonde* root verb, meaning "to become contorted" or "that which bends up" in reference to the stooped posture developed when arthritic symptoms appear. The virus was originally observed in Central and East Africa, circulating in a sylvatic cycle between forest-dwelling mosquitoes, non-human primates,[4,7] with sporadic human cases. In urban centres of Africa and throughout Asia, CHIKV is transmitted from viraemic humans via mosquitoes to available non-immune human hosts.[8] Although it is not demonstrated that cross-protection after infection with other alphaviruses (Ross River—RRV, O'nyong nyong—ONNV viruses) occurs in humans, it has been shown in animal models.[9-12] Large outbreaks of CHIKV have become more frequent in many endemic regions, including a number of Indian Ocean and Pacific Island nations, including Papua New Guinea, as well as emergent cases in historically non-endemic areas, such as Italy.[13,14,15]

These outbreaks have led to considerable problems for public health authorities, not only in relation to adequate vector control and epidemiological surveillance but also on the sustainability of the blood supply.[16,17] For example, in La Réunion Island in 2005, in which more than 30% of the population was infected during an outbreak, local blood donation was suspended to prevent transfusion-transmitted infection and pathogen inactivation was introduced to avoid critical shortages in platelet components.[16]

Potential Australian CHIKV vectors include; *Aedes vigilax, Aedes procax, Coquillettidia linealis,* and *Aedes notoscriptus* [all competent RRV and Barmah Forest virus (BFV) vectors] as well as *Aedes aegypti* and *Aedes albopictus*.[18,19] However, except for the last two, transmission is unlikely due to limited contact between these vectors and people.[18] Currently, *Ae. aegypti* is distributed widely throughout northern and central Queensland, with Goomeri (235 km north of Brisbane) the southern limit near the coast and

* The Makonde Plateau is a border area between Tanzania and Mozambique. "Chikungunya" is from the Makonde language.

Charleville (740km north west of Brisbane) the south western limit.[20,21] *Ae. albopictus* is currently restricted to the Torres Strait Islands off Cape York.[22,23] However incursions of this species, which is a more cold-tolerant mosquito than *Ae. aegypti*, do occur. In India, during an outbreak in 2005-06, *Ae. aegypti* was the main vector associated with disease transmission.[24] However, in the Indian Ocean CHIKV outbreak in 2005-2006, a mutation in the virus increased the transmission ability of *Ae. albopictus*.[25] Because this mosquito can survive in temperate climates, it has become a worldwide concern as a CHIKV vector.

The genus *Alphavirus* contains seven antigenic complexes. CHIKV belongs in a complex with ONNV, BFV, Semliki Forest (SFV), RRV, Sindbis (SINV), and Mayaro viruses (MAYV); members of the complex cause rheumatic manifestations including arthralgia.[26,27] For CHIKV infections the extrinsic incubation period (EIP) appears to be short in *Ae. albopictus*, as little as two days after the infective blood meal.[28] However, the EIP can be as long as 15 days when taken as time to reach maximum transmission efficiency.[29] The intrinsic incubation period ranges from 1 to 12 days (typically 2–4 days).[13] This period is followed by sudden onset of high fever, severe myalgia and arthralgia, with headaches, a skin rash and photophobia.[15,30,31] The symptoms usually resolve within 1-2 weeks but arthralgia may persist for weeks or months following the acute illness.[27] Infections may rarely be complicated by encephalopathy and hepatic failure.[30] CHIKV can also be transmitted to neonates by vertical transmission.[32,33] During recent epidemics in the Indian Ocean region, maternal-foetal transmission, severe neonatal disease, and adult mortality were reported.[34,35]

CHIKV infection is diagnosed on the basis of clinical and epidemiological criteria and can easily be confused with disease caused by other alphaviruses such as SINV, RRV and BFV, as well as dengue virus infection (Flavivirus) and hence requires laboratory confirmation. The most commonly used methods for laboratory diagnosis are serological tests and the quantitative reverse transcription-polymerase chain reaction (qRT-PCR) that detects the presence of viral RNA in serum.[36,37]

In Australia, the principal CHIKV vectors are present in suitable environments near susceptible populations. In addition, countries endemic for CHIKV are frequently visited by tourists, which eventually may result in chikungunya infectious cases in visitors or residents returning to Australia.[38-41] At present, the risk of CHIKV becoming established in Australia is restricted to areas where the vectors are present in sufficient density (Torres Strait Islands for *Ae. albopictus* and North Queensland for *Ae. aegypti*). CHIKV transmission in Queensland and the Torres Strait islands would have significant population health implications, including a potential impact on the supply of fresh blood components. In this study, we undertook an analysis of imported CHIKV cases in order to understand importation pathways and assess the risk of chikungunya emergence in Australia.

Methods

Chikungunya surveillance system

Chikungunya is notifiable in all Australian States and Territories except the Australian Capital Territory. It is not currently nationally notifiable, but a national case definition was implemented in 2010, and Australia's National Notifiable Diseases Surveillance System (NNDSS) includes a separate disease category for chikungunya. Before 2010, cases of chikungunya were sent to the NNDSS under the disease group "arbovirus Not Elsewhere Classified (NEC)" and the Northern Territory still maintains this practice.

Case definition

Under the Communicable Diseases Network Australia (CDNA) surveillance case definition for CHIKV, a confirmed case requires definitive laboratory evidence before notification.[42] Definitive laboratory evidence is:

- isolation of CHIKV
- detection of the virus by nucleic acid testing
- seroconversion or a significant rise in antibody level to chikungunya virus, in the absence of a corresponding change in antibody levels to RRV and BFV or
- detection of CHIKV-specific IgM, in the absence of IgM to RRV and BFV.

If the suspected case has not travelled to an endemic or epidemic country, then confirmation by a second reference laboratory is required.

Data collection

Notification data

Data on notifications of CHIKV infection were extracted from the NNDSS (8 February 2013). These data were subject to retrospective revision and may vary from that reported in published NNDSS reports and reports of notification data by states and territories. Notifications of chikungunya under "arbovirus NEC" were included. 'Diagnosis date (month and year of diagnosis)' represents the onset date, or where the date of onset was not known, the earliest of specimen collection date, notification

date, or date notification was received. Data span 1 January 2002 to 31 December 2012. 'Place of acquisition' was based on where the infection was believed to have been acquired, and the incubation period and time spent in a location or the place of recent travel. Data on place of acquisition were obtained from the NNDSS, and checked (up to 30 June 2011) and completed by State and Territory data managers for National Arbovirus and Malaria Advisory Committee (NAMAC) annual reports. Where insufficient information was available on the country or region of acquisition, 'Overseas – unknown/inadequately described' was recorded. Place of acquisition is usually obtained through public health follow-up of each case. 'State/Territory' is the state or territory of residence of the case. Cases residing in one jurisdiction but diagnosed in another are notified by the state of residence. Duration of travel was not recorded.

Overseas travel data

We accessed overseas arrivals and departures tables from the Australian Bureau of Statistics (ABS) website for 2008 to 2012.[43] Earlier years were not analysed because there were fewer than five cases of CHIKV per year before 2008. We analysed the number of short-term movements (resident departures and visitor arrivals) from Indonesia, Malaysia and India, the three main sources of importation identified. Monthly data are complete from January 2008 to December 2012 inclusive.

Data analysis

Short-term resident departures and visitor arrivals in Australia have been analysed for the study period.[43] Short-term resident departures (STRD) are defined as Australian residents intending to stay abroad for less than 12 months. Short-term visitor arrivals (STVA) are defined as overseas visitors intending to stay in Australia for less than 12 months. Short-term movement is defined as less than one year in duration.

The total and mean number of short-term movements (STRD and STVA) per year have been calculated for Indonesia, Malaysia and India, and were used to determine the percentage increase in these travel categories from 2008 to 2012. Importation rates based on short-term resident departures alone, and short-term resident departures summed with visitor arrivals over the period 2008 to 2012 have been calculated per 100,000 persons.

We used short-term movement information as these data include the country departed to or arrived from, whereas such information is not included with long-term movement data. We selected short-term visitor arrivals (overseas visitors intending to stay less than one year), and STRD (Australian residents intending to stay abroad less than one year). STRD were used rather than returns, because short-term resident returns are not available by country. We assumed near equivalence of STRD and short-term resident returns. But short-term resident returns may be fewer than STRD if some people decide to stay longer than one year, or die overseas. We assumed that numbers in these categories (longer than intended stay and overseas deaths) would be low.

Results

Number of CHIKV notifications

Since 2002 there have been 168 CHIKV notifications in Australia, 160 of which were during the study period (2008 to 2012). There was no clear seasonality in importation rate, although importations were slightly more common in the period October to April (Figure 1). In 2010, case numbers were highest in October (14 cases, comprising 22% of notifications for the year). The number of cases peaked in 2010 (n= 64) accounting for 40% of cases during the study period. In 2011 and 2012, 38 and 16 cases were reported, respectively.

Source of CHIKV importations

All CHIKV cases in Australia were acquired overseas. The three most common countries of acquisition were Indonesia (28.0%), India (18.5%), and Malaysia (10.0%) (Figure 2). The remaining 43.5% were from other countries, and of these 15.0% were unknown or inadequately described at the time of notification.

State of residence

Of the 160 cases, the majority (57.0%) returned to the two most populous states, Victoria and New South Wales (Table 1). Except for the Australian Capital Terrority, in which CHIKV is not notifiable, all states and territories reported cases. India and Indonesia respectively accounted for 26.0% and 24.0% of cases reported from Victoria and New South Wales.

Predicted number of imported cases based on short-term travel data

Over the study period, there was an increase in SDRD and STVA arrivals for the three main source countries for CHIKV infections (Figures 3 and 4). From 2008 to 2012, the number of STRD to Indonesia increased (+140%) as did, to a lesser extent, departures from India (+57%) and Malaysia (+36%). The number of STVA also increased from 2008 to 2012 (+54% for Indonesia and Malaysia, +35% for India).

We utilised STRD as well as visitor arrival numbers, along with notification source data, to estimate CHIKV importation rates in travellers returning from Indonesia, India and Malaysia. Our analysis

Figure 1: Number of reported cases of CHIKV, 2008 to 2012, by month and year

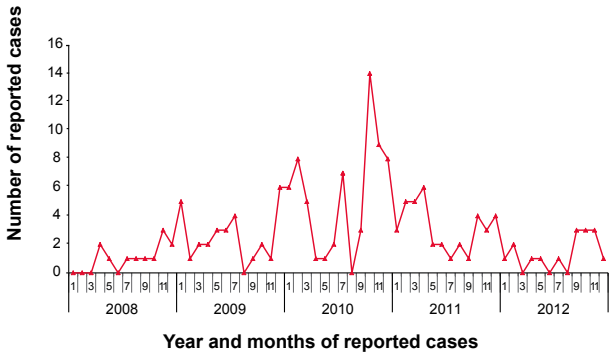


Figure 2: Number of reported cases of CHIKV, 2008 to 2012, by country of origin and year

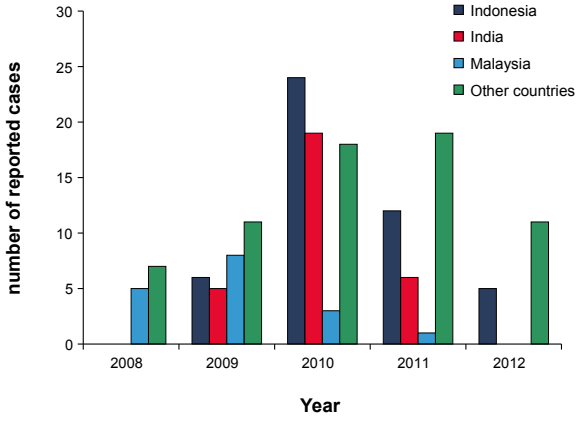
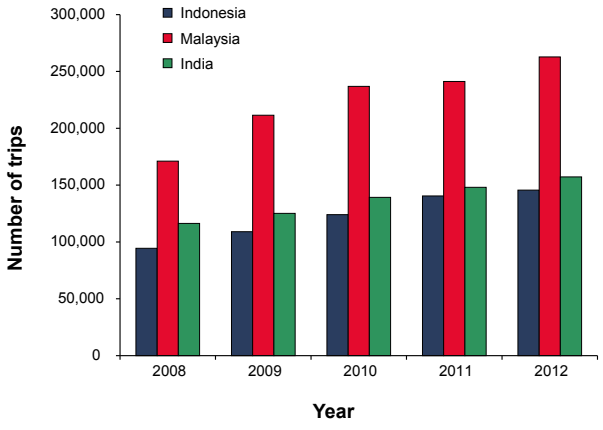


Table 1: Number of reported cases of CHIKV infection by country of acquisition and state or territory, 2008 to 2012

Country of acquisition	State/Territory							
	NSW	NT	QLD	SA	TAS	VIC	WA	Total
Indonesia	11	8	5	0	1	11	11	47
India	9	0	2	0	0	15	4	30
Malaysia	5	0	3	0	1	4	4	17
East Timor	0	4	0	0	0	4	1	9
Sri Lanka	0	0	0	1	0	3	0	4
Thailand	0	0	1	1	0	4	0	6
Vietnam	1	0	0	0	0	1	1	3
Bangladesh	3	0	0	0	0	0	0	3
Other countries*	5	1	2	1	0	6	2	17
Unknown/inadequately described	5	0	2	6	0	4	7	24
Total	39	13	15	9	2	52	30	160

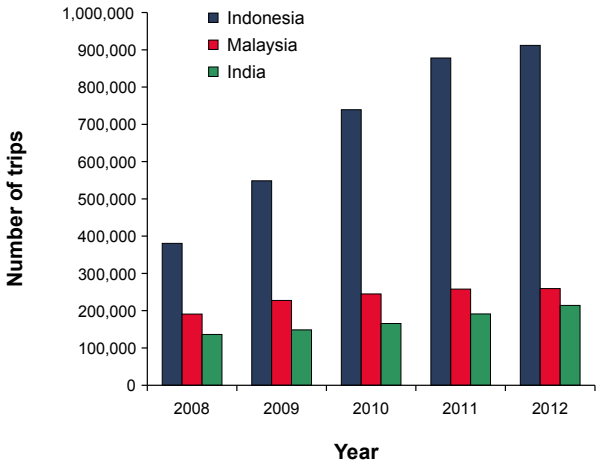
* 12 countries constituting 'Other countries' were known sources of chikungunya infection.

Figure 3: Total number of STRD from Australia, visiting Indonesia, Malaysia and India, 2008 to 2012



Data sourced from ABS

Figure 4: Total number of STVA, visiting Indonesia, Malaysia and India, 2008 to 2012



Data sourced from ABS

Table 2: CHIKV importation rates per 100,000 passengers, 2008 to 2012, by country

	Indonesia	Malaysia	India
n imported cases*	47	17	30
Total Passengers (STRD†)	3,458,100	1,180,600	855,600
CHIKV Import. Rates§	1.35	1.43	3.50
Total Passengers (STRD + STVA‡)	4,071,500	2,304,000	1,541,500
CHIKV Import. Rates§	1.15	0.73	1.94

* n imported cases: number of CHIKV imported cases

† STRD: Short-Term Resident Departures

§ CHIKV Import. Rates: Importation rates of chikungunya per 100,000 persons

‡ STVA: Short-Term Visitor Arrivals

suggests that, although Indonesia was the greatest source of infection acquisition, risk for CHIKV acquisition was highest for India (Table 2).

Discussion

Australia is at risk of local CHIKV transmission. Factors that determine Australia's risk include an immunologically naïve population (unless cross-protection with other alphaviruses occurs), regular introductions of the virus, presence of competent mosquito vectors, and an appropriate climate for exotic vectors. Climate change has the potential to increase vector range, as increased temperature and humidity could increase the areas in Australia receptive to vectors. The movement of workers and human behaviour might have an important role in emergence of CHIKV, as well as the introduction and establishment of *Ae. albopictus*. Indeed in Malaysia, migrant workers coming from endemic neighbouring countries are suspected to be the major source for CHIKV re-emergence.[30] Moreover, a recent study has shown that an increasing number of chikungunya cases have been reported to the Ministry of Health of Malaysia and the country may become endemic for CHIKV.[44] Due to the presence of *Ae. aegypti* and *Ae. albopictus* in the north of Australia, a local outbreak is likely to occur. Knowledge of vector competence of local mosquitoes is a starting point for understanding the risk of autochthonous transmission.[18] It is important to predict population health response requirements on the basis of knowledge of epidemic risk relative to number and timing of viraemic imports and relevant vector biology.

One hundred and sixty imported CHIKV cases were reported between 2008 and 2012. Under-reporting, due either to misdiagnosis or asymptomatic infection, is highly probable. Even for RRV, which is endemic, under-reporting is considerable.[26] These data give an overview of viraemic importations over the last 5 years and enable assessment of the most important source countries. Fifty seven per cent of reported cases come from Indonesia, India and Malaysia, countries where CHIKV has recently re-emerged. There was no strict importation seasonality during the study period. The inclusion of 15% of cases with unknown or inadequately described source represents significant missing data in the NNDSS.

We utilised travel data, along with the sources of CHIKV notifications in Australia to identify the highest risk source countries. Although Indonesia accounted for the majority of CHIKV notifications in Australia, India had the highest CHIKV importation rate. Travel to India has been steadily increasing in recent years, although not as rapidly as travel to other countries and CHIKV cases in Australia may increase in the future if this trend continues. It is also noteworthy that rates of CHIKV infection in returning travellers from East Timor could be higher than rates for India, Malaysia and Indonesia, given the relatively small number of total arrivals from that country. However, the total infections acquired in East Timor accounted for a small percentage of cases (<6% of cases between 2008 and 2012).

Notwithstanding the efforts of state and territory health departments (notably the Northern Territory Health Department and Queensland Health) to successfully manage exotic mosquito-borne diseases and their vectors, [45,46] the introduction of exotic vectors cannot be wholly prevented. It is likely that *Ae. albopictus* will become established on the Australian mainland.[23] Although numerous detections of *Ae. albopictus* have been successfully managed without the establishment of this species as yet, incursions continue to occur. In recent times *Ae. albopictus* was detected near Melbourne (December 2012) and is the subject of an on-going surveillance and control program (S. Lynch, Victorian Department of Primary Industries, *pers. comm.* 20 Dec 2012). The mosquitoes entered via a consignment of lucky bamboo (*Dracaena*). In Australia, other mosquito species such as *Ae. vigilax, Ae. procax, Ae. notoscriptus* and *Cq. linealis*,[47-49] could potentially transmit CHIKV,[18] although this is highly unlikely because of their behavior and ecology.

CHIKV has re-emerged after two to four decades, in some countries e.g. after 39 years in the Democratic Republic of Congo, 32 years in India, and 20 years in Indonesia.[51] In Southeast Asian endemic regions, where the original Asian genotype circulated for several decades, new strains belonging to the Indian Ocean Lineages (IOL) have emerged,[52,53] and caused major outbreaks especially in Malaysia.[53-57] The shift in viral genotypes is a major threat not only for the Asian region but also for the Western Pacific and Australia, where *Ae. aegypti* and *Ae. albopictus* are present in Queensland.

If local CHIKV transmission were to occur in Australia, this virus might cause considerable problems for public health authorities and impact on the nation's blood supply. CHIKV presents a risk to transfusion safety. During an outbreak in La Réunion Island in 2005 in which 30% of the population were infected, local blood donation was suspended and pathogen reduction of platelet components was implemented as an additional safety measure.[16] Dengue is episodic in north Queensland. A recent study has demonstrated that local outbreaks pose a relatively high risk to the safety of Australia's blood supply, with the large 2009 epidemic costing the Australian Red Cross Blood Service in excess of one million Australian dollars.[17] If CHIKV were to become established here with similar seasonal outbreaks in the north, it is likely to cause similar impacts on blood safety.

There are neither specific treatments for CHIKV nor a licensed vaccine. However, some treatments exist to relieve symptoms, for example non-steroidal anti-inflammatory drugs, as well as ribavirin and chloroquine.[13,58,59] A candidate live-attenuated virus vaccine (LAV) based on the wild-type Thai CHIKV strain has shown promising results,[60] provoking a good immune response in humans. In addition, a novel CHIK vaccine candidate, called CHIKV/IRES, has been developed and also shows promising results.[61]

Conclusion

This study highlights the main source of CHIKV viraemic reported cases and assists risk determination which could facilitate the formulation of public health policy and ensure the maintenance of the safety of the blood supply. The re-emergence of chikungunya in Asia and Indian Ocean islands and the emergence in the South Pacific regions, with several chikungunya outbreaks in Papua New Guinea, emphasise the potential of the virus to cause large outbreaks in susceptible populations. Overseas-travel, particularly for holidays, is probably the primary mechanism for CHIKV introduction to Australia. Therefore, epidemiology, human movements, vector biology and ecology are all crucial to population health planning for potential CHIKV importation into Australia.[62]

The Australian population is increasingly at risk for CHIKV establishment as the number of visitors coming from countries endemic for CHIKV and the numbers of residents going to visit these countries have increased in recent years. This risk will continue to increase if these countries remain attractive and affordable visitor destinations, and if in-country control efforts or Australian surveillance and traveller education programs are ineffective. In addition to direct morbidity costs, a CHIKV outbreak could significantly impact blood supply and tourism.

It would be useful to determine how long people stay in the endemic country, for what purpose (work, family visit, travel) and obtain information about the host (age, sex, income, level of education) and virus (strains). Australian authorities must continue to implement vector surveillance and control programs for the major vectors, *Ae. aegypti* and *Ae. albopictus* and ensure that the ongoing biosecurity measures are maintained in order to keep the country free of *Ae. albopictus*. Media and stakeholders should be kept well informed. Greater knowledge of the characteristics of each imported case is needed. Modeling of transmission risk is also important in order to predict future vector distribution and disease risk.

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An outbreak of staphylococcal food poisoning in a commercially catered buffet

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Abstract

Staphylococcal food poisoning is a common cause of foodborne illness. In Australia, since 2000, approximately 30% of foodborne *Staphylococcus aureus* outbreaks reported to OzFoodNet have been associated with foods prepared by commercial caterers. We conducted a retrospective cohort analysis of an outbreak of gastrointestinal illness among participants of an elite sporting event during which 22 individuals became ill after eating a commercially catered buffet dinner in June 2012. All recalled eating fried rice which had been intended for lunch service earlier that day and 20 of the 22 reported eating chicken stir-fry. Though no food samples were available for analysis, laboratory analysis conducted on four faecal specimens resulted in *S. aureus* being cultured from one specimen and *S. aureus* enterotoxin detected in another. The known epidemiology of staphylococcal food poisoning suggests a food contaminated by an infected food handler which was subject to temperature abuse may have caused the outbreak. As *S. aureus* foodborne outbreaks are often underreported, this investigation is a valuable contribution to the evidence-base and understanding of foodborne illness due to *S. aureus* and staphylococcal enterotoxin.

Keywords: Staphylococcus aureus, enterotoxins, outbreak, foodborne, rice, chicken

Introduction

Staphylococcal food poisoning (SFP) is a common cause of foodborne illness worldwide.[1-7] SFP occurs following ingestion of staphylococcal enterotoxins which are heat resistant and are produced in food following contamination by staphylococci, typically *Staphylococcus aureus*. Foods including sliced meat, meat products, salads, pastries, custards, raw milk and cheese products present a particular contamination risk.[2] Such a large population of staphylococci is indicative of unhygienic food handling procedures and temperature abuse over a period of time to allow for bacterial growth.[3]

In Australia, little published information exists describing past SFP outbreaks. OzFoodNet, however, collects information on all reported foodborne illness outbreaks. Between January 2000 and March 2012, OzFoodNet recorded 14 *S. aureus* outbreaks affecting 429 people (25 hospitalised; 1 death). In just under a third of these outbreaks, meals containing chicken were implicated. Twenty-nine per cent of these outbreaks were associated with food prepared by a commercial caterer (OzFoodNet Outbreak Register. June 2012. Unpublished data).

The outbreak

On 2 June 2012, 22 individuals who had participated in an elite sporting event in Sydney experienced gastrointestinal symptoms after eating a buffet dinner served by the commercial catering company servicing the event. The day of the outbreak was the final day of the two week event and reportedly less busy at dinner time than previous meals. The 22 individuals were part of a larger cohort of up to 40 people who queued for dinner service earlier than the other 500 attendees due to the timing of their responsibilities at the event. Within hours of eating, all 22 fell ill with symptoms including vomiting, diarrhoea and abdominal cramping. Six people were transported to hospital. The event organiser reported that only the early dining group was affected.

This report summarises the epidemiological and microbiological investigations into the cause of the outbreak.

Methods

Epidemiological investigation

As this epidemiological investigation was conducted as part of the required public health response to a reported outbreak, it was not necessary to obtain ethical approval.

In order to develop hypotheses regarding the cause of the outbreak, preliminary interviews were conducted by telephone with several of the cases who attended the emergency department (ED) due to the severity of their symptoms. We drafted a food exposure questionnaire based on information from these interviews and information from a copy of the menu provided by the caterers. The questionnaire sought basic demographic details, food exposures (lunch and dinner), symptom description and duration, and illness history. Individuals were also

asked whether they were aware of anyone who had been ill with gastrointestinal symptoms prior to or following the outbreak.

A case was defined as anyone who ate the catered buffet dinner on 2 June 2012 at the early time (16:00 to 17:30) and experienced vomiting and/or diarrhoea and abdominal cramping commencing between 17:45 and 21:15. A confirmed case was someone meeting the case definition with *S. aureus* or *S. aureus* toxin detected in a stool specimen.

The names of the cases as well as others who were thought to have dined early were provided by the event organisers, Ambulance Service NSW, and other interviewed attendees. Based on the knowledge gleaned from these interviews, we conducted a retrospective cohort investigation to identify risk factors for developing illness. Interview data were collated and attack rates and risk ratios were calculated for specific food exposures. Analysis was conducted using SAS© software (version 9.3).

Microbiological and environmental investigations

No food samples were available for testing. Faecal specimens were collected from 5 of the individuals who attended the ED. Initial testing for *Clostridium difficile*, *Salmonella, Shigella* and *Campylobacter* species and norovirus was conducted by the hospital laboratory.

Four specimens were available to be sent to Queensland Health Forensic and Scientific Services laboratory where they were cultured for a full range of enteric pathogens (including *Salmonella*, *Shigella* and *Campylobacter* species) and toxin-mediated foodborne illness causing bacteria (*S. aureus* and *Bacillus cereus*). Samples were cultured on Baird Parker Agar for two days at 37°C for *S. aureus* and Phenol-Red Egg Yolk Polymixin Agar for *B. cereus*. Three faecal samples were tested for staphylococcal enterotoxin using the Tecra enzyme-linked immunosorbent assay (TECRA). A site inspection was conducted by NSW Food Authority and is the subject of a separate internal report.

Results

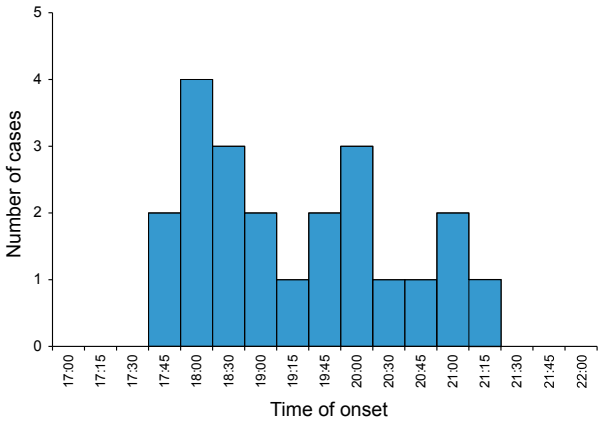
Epidemiological results

A total of 36 persons who ate an early dinner served by the caterer were interviewed, with the majority interviewed 2 to 3 days after the incident. The median age of people interviewed was 40 years (range 12 to 72 years); 78% were female. Among the 36 persons interviewed, 22 (61%) were identified as cases, including two persons with laboratory-confirmed illnesses.

Of the 22 cases, 18 (82%) were female, ranging from 12 to 69 years old (median 34 years). Of those who did not fall ill, 10 (71%) were female, ranging from 21 to 72 years old (median 46 years).

Dinner times reported by cases ranged from 16:00 to 17:30. The epidemic curve illustrates the time distribution of symptom onsets among cases ranging over a 4 hour period on 2 June (Figure 1). Incubation periods ranged from 1 hour to 4.75 hours (average 2.5 hours). Illness typically began with the sudden onset of vomiting, followed by a period of concurrent vomiting and diarrhoea, with a median duration of 4 hours (range 2 to 13 hours). Of the 22 cases, 21 experienced vomiting (96%); 17 had diarrhoea (77%) and 10 reported abdominal cramping (46%). Six people (27%) were transported to a local ED. No interviewees were aware of others with symptom onset of gastrointestinal illness prior to or following the outbreak.

Figure 1: Number of cases of gastrointestinal illness after the catered dinner on 2 June 2012, by time of onset (n=22)



A number of food items were served during lunch and dinner. A selection of bread, cold meats (ham, chicken, turkey and silverside), salad and fried rice were available at lunch. Green salad, coleslaw, meatballs, cannelloni, boiled rice, fried rice, chicken stir-fry, bread rolls, jelly and yoghurt were served for dinner. Fried rice intended for lunch service on the day of the outbreak was reportedly served to the early diners because the boiled rice for dinner service was not ready in time.

All interviewees had eaten dinner early at the catered buffet while only 14 (39%) ate lunch there. Ninety-one per cent of cases ate both chicken stir-fry and fried rice at dinner with attack rates and rate differences of 74% for chicken stir-fry and 71% for fried rice (Table 1). The risk ratios for both dishes were undefined. Similarly, we were unable to conduct further analysis using stratification. Therefore it was not possible to identify an association with either chicken stir-fry or fried rice.

Table 1: Relative risks and attack rates for food items consumed by the cohort

Salad	5	7	71	17	29	59	1.22 (0.70-2.13)	0.68
Coleslaw	2	2	100	20	34	59	1.70 (1.28-2.25)	0.51
Meatballs	15	24	63	7	12	58	1.07 (0.61-1.89)	1.00
Cannelloni	14	25	56	8	11	73	0.77 (0.47-1.27)	0.47
Fried rice	22	31	71	0	5	0	undefined	0.005
Chicken stir-fry	20*	27	74	0	7	0	undefined	0.0006
Yoghurt	5	8	63	17	28	61	1.03 (0.56-1.90)	1.00
Jelly	8†	13	62	13	22	59	1.04 (0.60-1.81)	1.00
Bread roll	11‡	17	65	7	14	50	1.29 (0.69-2.43)	0.48

* 2 missing
† 1 missing
‡ 5 missing

Microbiological and environmental results

Initial screening results for all five specimens were negative for norovirus, *C. difficile, Salmonella, Shigella* and *Campylobacter* species.

Queensland Health Forensic and Scientific Services laboratory cultured *S. aureus* in one specimen. Another specimen tested positive for *S. aureus* enterotoxin.

Though no food samples remained for laboratory testing, the catering company confirmed that food handling policies were in place to prevent contamination as well as time and temperature abuse. No evidence of time and temperature abuse was observed during the site inspection. The catering company also reported that no staff members were known to be suffering from gastrointestinal illness during the sporting event.

Discussion

S. aureus is one of the most common pathogens in humans, estimated to colonise approximately 25% of healthy adults.[2] Multiple pathogenic strains produce enterotoxins which, when ingested, can cause gastroenteritis.[8] In Australia, *S. aureus* intoxication accounted for 1% of all suspected and confirmed foodborne outbreaks reported to OzFoodNet between January 2000 and March 2012. Meals including chicken, beef, seafood, and lamb, as well as pasta salad and rice dishes have all been implicated as source of infection in these *S. aureus* enterotoxin outbreaks (OzFoodNet Outbreak Register. June 2012. Unpublished data).

Our findings suggested that chicken stir fry and/or fried rice were the food vehicles responsible for illness. Although it was not possible to determine risk ratios for fried rice and chicken stir-fry, the attack rates and rate differences calculated support this conclusion. It was not possible to consider these exposures independently as all cases who were able to recollect reported eating both food items.

SFP outbreaks result from contamination of food with *S. aureus* from food handlers either through skin infection on uncovered hands or arms, or via coughing or sneezing over food that is not subjected to further cooking. Current industry guidelines require food handlers to ensure their bodies, and anything from their bodies or clothing, do not contaminate food or food preparation areas.[9] For the bacteria to grow to sufficient numbers, the contaminated food must be left in temperature conditions where the bacteria are able to proliferate. *S. aureus* produces pre-formed toxins that have an emetic and diarrheal effect.[3]

In this investigation, there was no evidence of temperature abuse and we were unable to definitively identify a cause of the outbreak. The environmental investigation revealed no food safety breaches, and the absence of food samples made it impossible to identify the food vehicle responsible for the outbreak. The only apparent difference in foods served to the early diners was the fried rice which had been intended for lunch service.

To prevent toxin-based outbreaks, it is important that commercial food providers adhere to strict temperature protocols and ensure good food handling practices. Management and staff need to be alert to the presence of infected skin lesions or discharges from nasal passages, ears or eyes in food handlers. Appropriate measures should be taken to ensure that no ill individuals can contaminate food or food contact surfaces.[10]

Investigation of toxin-mediated foodborne illness is particularly problematic due to short onset times and duration of symptoms. Furthermore, as *S. aureus* is not a notifiable disease outbreaks often go undetected. This outbreak was only likely to have been reported due to the nature of the sporting event and the large number of individuals affected.

Limitations

This investigation was limited in several ways. Though interviews were conducted as soon as possible following the outbreak, a number of individuals had difficulty remembering all foods consumed. A high proportion of individuals who dined early strongly believed that the fried rice intended for lunch was the infection source. Moreover, participants had extensively discussed the outbreak and theories on its cause, predominantly through social media, potentially introducing bias to the investigation.

The microbiological investigation was also impacted by limitations. Firstly, initial analyses of faecal specimens were restricted to in-house PCR assays and not cultured as per the NSW Health outbreak protocol which specifies that all faecal specimens related to potential outbreaks undergo routine enteric culture. Nevertheless, *S. aureus* is unlikely to be grown using routine culture, and the delay which ensued from the need to transport samples to Queensland for toxin testing would have decreased the yield when appropriately cultured there. Given the time delay between onset and receipt of the samples and the variable storage temperatures of the samples during that time, it is unsurprising that only 1 positive result was returned. This underlines the importance of good communication between public health investigators and laboratories so that specimens are tested according to the clinical and epidemiological picture. Additionally, vomitus specimens would have been preferable for analysis as staphylococcal enterotoxin is cleared from the gut quite quickly. Unfortunately, no samples of vomitus were collected as this is not a routine practice in EDs and vomiting had resolved before the public health investigation commenced.

Conclusion

Information obtained from case interviews and the results of microbiological testing of human specimens support a conclusion that enterotoxigenic *S. aureus* bacteria were responsible for this outbreak. We were unable to definitively identify a food vehicle in this outbreak. *S. aureus* associated outbreak reports are rarely published in Australia despite being such a common cause of foodborne illness worldwide. This investigation improves our understanding of the epidemiology of foodborne *S. aureus* outbreaks in Australia.

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Review of Australia's Polio Surveillance

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Abstract

With eradication almost within reach, the importance of detecting every poliomyelitis case has taken on additional significance. The selected surveillance strategy must be effective and efficient. A review of polio surveillance in Australia was conducted to consider whether current strategies were optimal. Document review and semi-structured key informant interviews were used to conduct the review. Interviews were recorded, transcribed and thematically analysed. The review was an iterative process with feedback on the findings sought from interviewees.

Since Western Pacific Regional polio-elimination status was certified, one imported adult case was detected in 2007 in Australia, with no evidence of further transmission, and no Australian paediatric cases identified. Respondents reported that: it was not possible to prevent importations; paediatric cases were more likely to be identified than adult cases; and there may be a low level of suspicion among clinicians.

Case detection and outbreak mitigation were considered key reasons to undertake polio surveillance. While Australia has not achieved one of the key World Health Organization (WHO) surveillance targets, this did not compromise Australia's polio-free status. Identified issues with polio surveillance were the potential for an importation with high attendant investigation and containment costs, low stool sample collection rates, and the opportunity to improve safeguards around the importation and laboratory storage of biological samples containing poliovirus. The review found strong support for ongoing polio surveillance, particularly to detect imported cases and to demonstrate commitment to maintaining a polio-free region. Existing polio surveillance strategies were considered appropriate for Australia.

Keywords: Polio, surveillance, evaluation, epidemiology, acute flaccid paralysis

Introduction

Global polio occurrence is at its lowest level, with only 223 wild polio cases reported in 2012. However the goal of eradication is elusive with three countries, Pakistan, Afghanistan and Nigeria remaining endemic and cases also reported in Chad and Niger during 2012.[1] Under the International Health Regulations (2005), poliomyelitis caused by wild poliovirus is one of four specific diseases that must be notified to the World Health Organization (WHO) on detection.[2]

In the 1980s, acute flaccid paralysis (AFP) surveillance was implemented globally as the key surveillance strategy for validating the eradication of polio.[3] AFP is a marker syndrome for poliomyelitis and a number of other conditions including Guillain-Barré Syndrome (GBS), the most common cause of AFP.[4] Identification of all AFP cases prevents paralytic polio being missed and adequate investigation, including the timely collection of two stool samples, ensures that polio has been excluded as a diagnosis.[5] As part of the certification process to declare WHO Regions polio-free, WHO recommended implementation of AFP surveillance in all member countries.[6]

Australia is one of a decreasing number of developed countries to maintain AFP surveillance. Over the past five years Australia has consistently achieved the non-polio AFP surveillance target of one case per 100,000 children aged less than 15 years, but the stool collection surveillance targets, of two stool specimens collected from 80% of cases classified as non-polio AFP, has never been met.

Poliomyelitis has been a notifiable condition in Australia since 1922.[7] Queensland is the only state where AFP is notifiable. Australia has high immunisation rates. In 2012, Australia had a 92.3% national average coverage rate at 12 months with three doses of polio containing vaccine,[8] and has experienced no community polio outbreaks since the 1970s. The WHO Western Pacific Region, which includes Australia, was declared polio free in 2000.[9] In 2007, one imported polio case was detected in a Melbourne student returning from a visit to Pakistan, without further known local transmission.[10,11]

A number of Western Pacific countries, including Papua New Guinea, remain classified as 'high risk' for polio outbreaks by the WHO (Personal communication, Dr Sigrun Roesel, WHO). Australia is currently classified as 'low risk', but continues to receive a large number of short term arrivals, students, migrants and refugees from countries classified as endemic or 'high risk' or that continue to use oral poliomyelitis vaccine (OPV).[12]

From 2000 a range of surveillance strategies were implemented in Australia to document the absence of circulating wild poliovirus, and detect AFP cases to confirm ongoing eradication. The surveillance

systems continued to monitor vaccine-associated paralytic poliomyelitis (VAPP) until 2005, when use of OPV was discontinued in the immunisation schedule.[13] A response plan for polio importations and potential outbreaks has also been developed. Australia has two peak polio committees, the National Certification Committee for Poliomyelitis Eradication (NCC) and the Polio Expert Panel (PEP). Australia hosts a WHO accredited National Enterovirus Reference Laboratory (NERL) at the Victorian Infectious Diseases Reference Laboratory (VIDRL).

This review of current Australian polio surveillance activities was undertaken to ensure that the current suite of strategies provide optimal surveillance for a high income country with sophisticated medical and laboratory infrastructure, and a long history of freedom from endemic polio circulation. The review specifically examined whether Australia was able to detect an imported case of poliomyelitis, determine if surveillance helped to mitigate the risk of an outbreak, and whether there was sufficient evidence to demonstrate that Australia was free of circulating wild poliovirus.

Methods

This polio surveillance review was conducted by an independent epidemiologist from the Hunter Medical Research Institute, University of Newcastle, engaged by the NCC, between April and November 2012.

The framework for the review was adapted from the Centers for Disease Control and Prevention (CDC) framework for evaluating surveillance systems,[14] the WHO guide for monitoring and evaluating surveillance and response systems for communicable diseases,[15] and techniques commonly used in public health evaluations.[16,17] Generous timeframes and small numbers of interviewees permitted the use of semi-structured, face-to-face interviews that enabled an in-depth investigation of respondents' views of the surveillance system. Interview guides were prepared and tailored to expert informants' roles. A desktop review of relevant documents was also conducted, which included published articles, unpublished government reports and other grey literature.

Concepts and identified issues were explored and validated in subsequent interviews. Expert informants were chosen in consultation with the NCC, based on their knowledge, roles, or involvement with the surveillance system. Interviewees included personnel of the NERL and Enterovirus Reference Laboratory Network of Australia (ERLNA), paediatricians, policy makers, surveillance system administrators, research nurses, academics and members of the polio peak committees. Twenty seven key informants were interviewed face to face, in Western Australia, Victoria, New South Wales, Queensland and the Australian Capital Territory, and a further nine by phone or email. During interviews, interviewees were encouraged to identify other key informants. Additional key informants were interviewed until information saturation, where no new information is obtained from the interviews, was reached. One hundred percent of the approached informants participated in the interviews. Interviews were recorded and transcribed.[16] Thematic analysis was applied to transcriptions using NVivo software. Situational analyses have been undertaken where appropriate.

The review was iterative, with feedback sought from key informants on identified issues, gaps in understanding and draft recommendations. The reliability of identified themes was tested during subsequent interviews and the document review.

Findings from the interviews and draft recommendations were presented to the NCC, for discussion and comment, prior to preparation of the final report.

Human ethics approval was not required as this was a service evaluation and quality assurance exercise, thus not requiring such clearance.

Results

System description

The stated objective of Australian poliovirus surveillance is to conduct surveillance for poliovirus in Australia to detect imported cases, mitigate the risk of an outbreak and provide additional virological evidence that Australia continues to be free of circulating wild poliovirus (Personal communication, Nicolee Martin, Department of Health). Poliomyelitis surveillance system components include AFP surveillance, and virological, laboratory and environmental surveillance (Table 1). The VIDRL coordinates most polio surveillance activities in Australia, including:

- The NERL
- National AFP surveillance system
- The ERLNA
- Environmental surveillance.

AFP surveillance focuses on children less than 15 years of age, with research nurses actively identifying potential AFP cases for inclusion in the surveillance system or clinicians notifying AFP cases through the APSU. There is no active surveillance system to detect polio specifically. AFP case detection in

Table 1: Australia's polio surveillance system, 2012

Surveillance System	System component	Description	Findings
Acute Flaccid Paralysis (AFP)	Australian Paediatric Surveillance Unit (APSU)	Commenced in 1995. Approximately 90% (~ 1360) of paediatric clinicians submit a monthly report card to the APSU. It includes request for the collection and testing of two stool samples and the completion of a clinical questionnaire.	The system may not be timely. Provides the only method to access regional and non-tertiary hospital AFP cases. Important mechanism for communicating with paediatricians. Low workload for respondents. Clinicians may not report AFP cases through the APSU system at PAEDS hospitals.
	Paediatric Active Enhanced Disease Surveillance (PAEDS)	Commenced in 2007. Four tertiary paediatric hospitals – Perth, Adelaide, Melbourne, Sydney. Brisbane is expected to commence in 2013. Uses hospital-based research nurses to actively identify cases of AFP, seek consent and ensure the collection of two stool samples. AFP is one of four conditions collected through the system.	Becoming the most important system for AFP surveillance. Some frustration that stool collection rates have not improved uniformly since implementation of the system. Some challenges in ensuring clinician engagement.
	Mandatory notification in Queensland	AFP is notifiable under the Queensland *Public Health Act 2005* by a clinician on the basis of clinical or provisional diagnosis.	AFP should not be made nationally notifiable.
Virological and enterovirus	National Enterovirus Reference Laboratory (NERL)	World Health Organization-accredited polio reference laboratory. Receives samples from AFP surveillance.	Effective mechanism for enterovirus (including poliovirus) surveillance. Provides enterovirus testing in the Region.
	Enterovirus Reference Laboratory Network of Australia (ERLNA)	Established in 2008. Coordinated by the NERL. Sends untyped enterovirus samples for testing to the NERL. In 2011, 331 enteroviruses were typed by members of the ERLNA.	Provides epidemiological data on enteroviruses in Australia.
Environmental	Three sentinel sites in Newcastle, Byron Bay and Armidale	Implemented in 2010. Sites were chosen based on local public health support. Population size served and areas with large overseas student populations from endemic areas (Newcastle and Armidale) or relatively low immunisation coverage and regular international visitors (Byron Bay). Sewerage samples from the sentinel environmental sites are tested for poliovirus and other enteroviruses at the NERL	Successful implementation of sentinel sites. Useful to trial a site at a major metropolitan location. Demonstrates that an outbreak is contained rather than used for case detection. Retention of sentinel sites maintains skills and capacity.
National Notifiable Diseases Surveillance System (NNDSS)	Poliomyelitis nationally notifiable	Notifiable since 1922. Poliomyelitis (paralytic infection) and Poliovirus (non-paralytic infection) are currently notifiable. Includes wild poliovirus infection, Vaccine-associated paralytic poliomyelitis (VAPP) and Vaccine derived poliovirus (VDPV) infection.	Passive notification of poliomyelitis and poliovirus. One adult case notified in 2007.

Australia occurs actively through two systems; the APSU monthly reporting system, and Paediatric Active Enhanced Disease Surveillance (PAEDS). Samples from AFP cases are forwarded to the NERL, a WHO-accredited polio reference laboratory. De-identified AFP case information is reviewed by the PEP every two months for classification.

Environmental surveillance for enteroviruses is currently implemented at three sentinel sites in Australia. Testing for poliovirus and other enteroviruses is conducted at the NERL for stool samples from the AFP surveillance system, adults where polio is suspected, and sewerage samples from the sentinel environmental sites.

Australia reports key polio surveillance indicators to the WHO and also provides an annual report to the Regional Certification Committee with evidence to verify that Australia continues to remain polio-free.

System performance

Following the introduction of the PAEDS system in 2007, Australia met the non-polio AFP rate for children <15 years of age every year for the years 2008—2011. Prior to this the surveillance target was only achieved sporadically; in 2000, 2001, 2004 and 2006.[18] Australia however has never achieved stool sample collection rates that meet the WHO surveillance criterion.[8] In 2011, 34% of non-polio AFP cases had adequate stool samples collected.[19] Factors identified by the respondents to improve stool sample collection rates included; active, daily visits with ward staff, monitoring whether stool samples had been submitted, and regular feedback and engagement of clinicians

Surveillance objective 1: Is Australia able to detect an imported case of poliomyelitis?

Respondents unanimously agreed that it was not possible to prevent importations. Most felt that undetected importations were likely to have occurred in Australia. Reasons cited for the possibility of missed importations were that most poliovirus infections are asymptomatic and would not present to a hospital, clinicians may have missed cases because of a low level of suspicion, or the case would have presented in a non-classical manner (without paralysis). One respondent observed, "We're looking for a needle in a haystack really."

"We are actually a well-protected country at threat of importation (of polio)."

- Polio surveillance interviewee

Most respondents thought that a paediatric flaccid paralysis case would be detected because of AFP surveillance but that an adult case might be missed. The risk of outbreaks was mitigated by high vaccination coverage. Systematic environmental sampling for polioviruses was viewed as complementing AFP surveillance, although detection would only be limited to areas under surveillance.[20] No polioviruses were detected through environmental surveillance in 2010, 2011 or 2012, but other enteroviruses were successfully detected.[8]

Surveillance objective 2: Does surveillance help to mitigate the risk of an outbreak?

Respondents commented that the early detection of a poliomyelitis case was one of the main reasons to undertake surveillance. Early detection and a rapid public health response should mitigate the risk of further community transmission. They noted that the NERL had the capacity to rapidly increase virological testing in the event of an outbreak. This was successfully demonstrated during the 2007 polio importation. Virological surveillance amongst contacts and exposed high risk groups would help to determine whether an outbreak had been controlled. In particular, environmental surveillance conducted locally in the outbreak region, could help in assessing whether community transmission had occurred and would serve to demonstrate that an outbreak had been contained.

"It's nice to have (environmental surveillance) as a surveillance strategy in your back pocket if you're going to invest heavily in a community response."

- Polio surveillance interviewee

Respondents noted that there was a response plan that would be activated in the event of detection of a single polio case to limit further transmission of poliovirus.[21] A number of respondents commented on the public health and economic imperative for containing an outbreak as early as possible. The costs associated with the importation of a single polio case were substantial; however, a larger outbreak could have an even more profound economic impact. Effective surveillance (including virological and environmental), early detection and immediate response were considered necessary to mitigate the risk of any future outbreak.

Surveillance objective 3: Is there sufficient evidence to demonstrate that Australia is free of circulating wild poliovirus?

Respondents were unanimous that there was sufficient evidence to demonstrate that Australia continues to be free of circulating wild poliovirus,

as ratified annually since 2000 by the RCC. They indicated that AFP surveillance helped to demonstrate that Australia remains polio free and should continue in its current form.

While Australia did not achieve all the required WHO polio surveillance indicators, respondents considered that there was still sufficient evidence that Australia remained polio-free, with adequate AFP detection and accessibility to high quality laboratory services. The supplemental surveillance systems (environmental and enterovirus) were viewed as providing additional evidence that there was no circulating wild poliovirus. Prior to certification, WHO recognised that countries may have difficulty meeting all the reporting requirements, and that supplemental surveillance could be used to provide assurance that the country remained polio-free.

Identified gaps and issues

The major surveillance gaps identified were in:

- the detection of adult cases
- ensuring that clinicians would recognise a poliomyelitis case
- the risk of importations
- the need to improve stool sample collection rates
- the opportunity to improve safeguards around the importation and laboratory storage of biological samples containing poliovirus.

In general, respondents thought there was a low level of clinical suspicion for polio. It was acknowledged that this is because the disease is rare and it is unlikely that most clinicians have seen a case of poliomyelitis. Detection of cases are generally considered to rely on astute clinicians considering poliomyelitis as a possible diagnosis in AFP cases.

There was some concern that PAEDS had only demonstrated limited success in improving stool sample collection rates. Respondents recommended that active engagement of clinicians by the research nurses, or through the identifiction of a local clinical champion may improve clinician participation in stool sample collection. Respondents thought that Australia should be aiming for the highest possible stool collection rate in patients in which there was no obvious diagnosis. Stool sample collection should be based on the clinical imperative to test for diagnostic purposes.

"I think there is reluctance among clinicians to do unnecessary investigations."

- Polio surveillance interviewee

The possible inclusion of additional surveillance systems was mentioned by respondents. These included AFP being made nationally notifiable and through the Australian and New Zealand Paediatric Intensive Care (ANZPIC) registry. However the former had not demonstrated success in Queensland and the latter lacked the required timeliness.

Respondents considered that individuals infected with poliovirus could have entered Australia without detection. However they did not feel that this was of major concern as there had been no detected poliomyelitis outbreaks and that this was unlikely to occur because of Australia's high vaccination coverage. The majority of respondents believed that there was only a limited risk that a broader community outbreak would have gone undetected by the system. Respondents commented that there are a number of groups that pose a higher risk of poliovirus importation into Australia than others, particularly those from endemic countries. They suggested that it would be useful to explore whether the current policies around vaccination of immigrants, refugees and travellers to and from endemic areas were adequate to address importation risks.

Many respondents noted their concerns about the lack of safeguards regarding the importation of biological samples that might contain poliovirus. They felt that it was of concern that a stool specimen containing poliovirus could be imported into Australia with relative ease. They noted that laboratories importing biological materials need to obtain an import permit for handling of these materials. However, as poliovirus is currently designated as a Risk Level 2 organism (moderate individual risk, low community risk),[6] the controls around importation were limited. Respondents felt that it was critical that Australia should know where all poliovirus specimens were held, that they were secure and that importation of specimens potentially containing poliovirus were strictly controlled.

The future

Most respondents thought that if global polio eradication was achieved, Australia should maintain the current AFP and other surveillance strategies for at least three years post-eradication. Enterovirus surveillance should however, continue indefinitely post eradication to improve the epidemiological understanding of other important enteroviruses in Australia, including EV71.

Respondents commented that surveillance may need to be enhanced if polio eradication was not achieved.

Discussion

The thematic analysis of responses by enterovirus and public health surveillance experts and the document review, found that Australia meets some but not all of its polio surveillance objectives, and that there is room for improvement. Table 2 documents the recommendations arising from the polio surveillance review.

There is strong support for the continuation of polio surveillance, particularly to detect imported cases and to demonstrate solidarity with maintaining a polio-free status in the region. While recognising that the polio surveillance system has developed in a relatively *ad hoc* manner and that there are some remaining gaps, the existing polio strategies were considered appropriate for Australia. Maintenance of the established AFP surveillance system requires a relatively small economic investment and was considered likely to successfully identify symptomatic, paralytic polio in children. PAEDS is becoming the most important surveillance mechanism for detecting AFP cases; however APSU, in addition to detecting AFP cases, serves a supplementary function as an important mechanism for communicating with all Australian paediatricians. Enterovirus and environmental surveillance were considered important supplementary surveillance systems, with complementary strengths, and the NERL was recognised as being a highly credible organisation playing an integral role in national and regional polio surveillance.

Table 2: Recommendations arising from the review of Australia's polio surveillance system, 2012

	Recommendations
1.	Australia should continue to undertake active polio surveillance.
2.	Existing polio surveillance strategies should occur for three years post-eradication and enterovirus surveillance should continue post-eradication. If eradication is not achieved, surveillance will need to be re-evaluated and may need to be enhanced.
3.	The consolidated purpose, objectives and activities of the Australian polio surveillance system, including Australia's commitment to the WHO Global Polio Eradication Initiative, should be documented by the Department of Health (DoH).
4.	Acute flaccid paralysis (AFP) surveillance should continue in its current form through Australian Paediatric Surveillance Unit (APSU) and the Paediatric Active Enhanced Disease Surveillance system (PAEDS) with regular case review by Polio Expert Panel and reporting of classified cases to the WHO.
5.	Stool collection rates should be improved including through enhancing the effectiveness of the PAEDS program.
6.	Polio should remain a nationally notifiable condition but AFP should not be nationally notifiable.
7.	Sentinel environmental surveillance sites to supplement AFP surveillance should be maintained and sentinel environmental surveillance should be trialed in a major metropolitan area.
8.	Enhanced communications to raise awareness of the importance of completing global poliovirus eradication and highlighting the need for clinicians to remain vigilant for cases of poliomyelitis should be developed by DoHA.
9.	The DoHA should review current policies relating to vaccination of immigrants, refugees and travellers to and from endemic countries to determine if these policies are adequate to address risks of importation.
10.	A review of biosecurity arrangements for the laboratory containment of polioviruses should be conducted in collaboration with accountable individuals.

There were ongoing concerns about the potential importation of poliovirus without adequate controls. The potential to apply the new Biosecurity legislation to address risks associated with the importation of biological samples containing poliovirus should be explored (Biosecurity Bill 2012).[22]

Respondents believed that Australia had a responsibility to meet World Health Assembly (WHA) member requirements to maintain surveillance of such quality that Australia would be able to detect cases and respond to them.

Polio eradication is a global public health emergency and every effort should be made to complete this task.[23] Australia should continue to maintain high immunisation coverage, support global eradication efforts financially, and sustain current polio surveillance to ensure that this public health goal is achieved.

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Adolescent school-based vaccination in Australia

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Abstract

Adolescents have become an increasingly prominent target group for vaccination in Australia and other developed countries. Over the past decade, voluntary school-based vaccination programs have evolved to become the primary method of delivering adolescent vaccines funded under Australia's National Immunisation Program (NIP). These programs operate at a state and territory level and offer NIP vaccines to adolescents in specific school grades using local teams of trained vaccine providers. This paper summarises the current operation of voluntary school-based vaccination programs in Australia. Information was obtained through a literature review, semi-structured interviews with those managing and implementing school-based vaccination programs in each jurisdiction and a review of program resources. Available coverage data was obtained from each state or territory. Vaccines are delivered at the school, during school hours, and typically target late primary or early secondary school grades. Written parental consent is required for any vaccine to be administered. Operation of the programs is influenced by various factors at the school and provider level. Despite variability in program implementation, collection and analysis of coverage data, comparable coverage has been achieved across all states and territories. Coverage is higher than that reported by other countries where adolescent vaccines are mandated for school entry or available only through community vaccination providers. Voluntary school-based vaccination programs are an established mechanism for the delivery of adolescent vaccines in Australia and vaccines offered will continue to evolve in light of national recommendations. Current gaps in evidence include a detailed understanding of the influence of procedural factors on uptake, the best ways to maximise consent form return and, standardisation of coverage data reporting.

Key words: immunisation, vaccination, adolescent, school vaccination

Introduction

Vaccination has long been a successful strategy in the control and elimination of communicable diseases predominantly those affecting children.[1] However, morbidity continues to occur in adolescents due to vaccine-preventable diseases such as hepatitis B, varicella and pertussis.[2] In light of this and the availability of new vaccines best delivered in adolescence (i.e. human papillomavirus - HPV), they have become a prominent target group for routine vaccination both in Australia and internationally.[3-5] Vaccination of adolescents aims to maximise protection against future disease risk, to boost existing but waning immunity, or to catch-up those who may not have been adequately vaccinated as children.[6] To contribute to disease reduction, adolescent vaccination programs must be successfully implemented and achieve coverage for each vaccine sufficient to control disease.[7] To do so, efficient and effective ways of delivering vaccines to this often hard to reach group are needed. Current evidenced-based approaches to achieving this include school-linked mandates and voluntary on-site school-based vaccination.[8]

Since the 1970s voluntary, school-based vaccination programs have been implemented in Australia, although their use has been varied due to differences in political support, legislative and public health systems between states and territories.[9] Provision of vaccines through schools has the potential to reach the majority of adolescents in Australia, as school attendance is mandatory until mid to late adolescence and attendance rates are high, particularly in lower school grades.[10] School-based vaccination is popular with parents,[11,12] and has achieved higher levels of coverage compared with vaccinating adolescents in the primary care setting.[13] In addition, school-based vaccination largely overcomes the issue of cost and access to vaccines for adolescents, as they are not required to make a specific appointment with a doctor, there is no consultation fee and the vaccines are free.

This approach to adolescent vaccination is not without its challenges. Some barriers to the vaccination of adolescents are similar to those of vaccinating younger children (e.g. vaccine refusal and low awareness).[14] However there are additional challenges unique to this group, such as obtaining appropriate and valid parental consent in the light of the increasingly autonomous decision making capacity of adolescents.[8,15]

This paper summarises the Australian approach to school-based vaccination, highlighting successes, challenges and future considerations with this approach.

Methods

Review of school-based vaccination implementation in Australia

Information about the history and current operations of school-based vaccination programs in Australia was collected through a review of published literature, semi-structured interviews with those managing and implementing school-based vaccination programs in each state or territory and a review of program resources available in 2010. Descriptive content analysis and thematic analysis were used to objectively identify relevant information from these data and systematically group it into key themes,[16, 17] constructing an overview of school-based vaccination program implementation in Australia.

Coverage

Coverage data by calendar year, vaccine, dose number and school grade were requested from each state and territory at the time of interview. Data were requested for all calendar years in which a vaccine was routinely offered through school-based vaccination programs in the state or territory. Due to known differences in data sources and methods of collection and analysis, an explanation of these was also requested. In addition, coverage data for HPV vaccine for 2007 was sourced from the published literature.[18]

As available coverage data was not uniform in presentation, had different denominators and was limited to aggregated proportions of jurisdiction-wide coverage, manipulation was required to develop standard estimates for comparison. The Australian Bureau of Statistics (ABS) estimates of annual full-time equivalent school enrolments in Australia were used as a standard denominator.[10] Available proportions of state or territory-wide annual coverage was used to estimate the number of students vaccinated by vaccine, dose number, jurisdiction and calendar year (numerator). These were pooled to obtain estimates of average national coverage for each vaccine, dose and calendar year which were then weighted by the enrolled population and averaged across 2004 to 2009.

Results

Implementation of school-based vaccination in Australia

Interview participants

Interviews were conducted with all state and territory immunisation program managers (n=8), along with other health department staff associated with immunisation program management in some jurisdictions (Victoria, Western Australia, South Australia and Tasmania). Seven staff primarily responsible for coordinating the implementation of school-based vaccination programs in their relevant jurisdiction were interviewed in: the Australian Capital Territory (n=1), Western Australia (n=1), Victoria (n=1), Northern Territory (n=2) and Queensland (n=2). Staff were not interviewed in New South Wales, South Australia or Tasmania as sufficient detail on program implementation had been obtained from earlier interviews. Of the 19 interviewees, the majority had been involved in program management and/or delivery for 6 to 10 years (n=7) or more than 10 years (n=7).

Policy

From 2007 all states and territories used routine voluntary school-based vaccination as the primary mechanism to deliver adolescent vaccines on the NIP although some jurisdictions had previously implemented these programs.[9] Based on the nationally recommended age range for administration each state or territory health department decides on the school grade(s) in which vaccines will be offered and develops policies and procedures for these programs in their jurisdiction. Adolescent vaccines offered through school-based vaccination programs are not mandated for secondary school entry or attendance. The education sector allows vaccination programs to operate in schools on a goodwill basis, recognising the important public health benefit of these programs.[19] Vaccines currently offered though school-based vaccination programs in Australia are a mixture of newly introduced vaccines (e.g. HPV), booster doses (e.g. diphtheria-tetanus-pertussis - dTpa), and time limited, catch-up programs for particular age cohorts (e.g. Hepatitis B, varicella; Table 1).[3]

Funding

Until recently the Australian Government funded each state and territory to purchase vaccines on the NIP through a tender process. The responsibility for purchasing NIP vaccines is currently being transferred to the Australian Government, which will directly provide states and territories with vaccines for school-based vaccination programs.[20] Over the years, the Australian Government has also provided time-limited funding to support school-based delivery of some vaccines.

Each state and territory government provides varying levels of funding to support the implementation of their own school-based vaccination program. The immunisation area in each state and territory health department is primarily responsible for the overall management and operation of the program. Most provide funds to regional organisations, such as local or regional government health services to implement the program (Table 2). Funding formulas vary

Table 1: Vaccines offered through school-based vaccination programs, 2012, by state and territory

Vaccine	Nationally recommended age group	School grade	Jurisdiction
Hepatitis B (2 doses, adult formulation)	10–13 years*	Last year primary First year secondary	WA, TAS† NSW, ACT, VIC, QLD, SA, TAS†
Varicella‡	10–13 years*	Last year primary First year secondary Second year secondary	WA, TAS† NSW, ACT, VIC, QLD, SA, TAS† NT
Human papillomavirus (HPV) (3 doses)	12–13 years (females only)	Last year primary First year secondary	WA, TAS† NSW, ACT, VIC, NT, QLD, SA, TAS†
Diphtheria-tetanus-acellular pertussis (dTpa)	11–17 years*§	Last year primary First year secondary Second year secondary Third year secondary Fourth year secondary	WA, TAS† NSW‖ NT, SA ACT, QLD VIC, TAS†, NSW‖
Pneumococcal 23vPPV¶	15–49 years (Indigenous only)	Fourth year secondary	NT

Abbreviations for each state/territory: ACT = Australian Capital Territory, NSW = New South Wales, NT = Northern Territory, QLD = Queensland, SA = South Australia, TAS = Tasmania, VIC = Victoria and WA = Western Australia.

* Recommended for one cohort only within the specified age range. Dose schedules may vary between jurisdictions.

† In Tasmania each local government decides in which school grades vaccines are offered.

‡ Varicella vaccine is recommended for those who have not already received the vaccine or have no clinical history of chicken-pox. A second dose is recommended for students ≥ 14years of age but not funded or provided routinely through school-based vaccination programs, except in Qld where the follow up vaccination is offered by a school-based vaccination provider or general practitioner.

§ From October 2009, Australian Technical Advisory Group on Immunisation (ATAGI) recommended that the adolescent booster dose of pertussis be given in the first year of secondary school, which is from 11 years of age.

‖ From 2010 dTpa was routinely offered in grade 7 (first year secondary school) in NSW. This vaccine will also be offered as a catch–up in grade 10 from start 2009 to end 2012. From 2013 this vaccine will be routinely offered only in grade 7 in NSW.

¶ Pneumococcal polysaccharide vaccine, 23-valent.

between jurisdictions with some offering additional funds to support the operation of these programs in rural/remote areas.

Program reach

The age-based structure of school grades varies between states and territories.[10] Most school-based vaccination programs target lower secondary school grades (ages 11—14 years). The exception is Western Australia and Tasmania, where most vaccines are offered in the final year of primary school when children are equivalent in age to those in the first year of secondary school in other jurisdictions (Table 1).

Nationally recommended vaccines for adolescents are also offered in some 'non-traditional' schools at the discretion of each state and territory. These include schools that cater for students who have special educational needs and those that provide tuition to school-aged youth who have recently arrived in Australia and whose first language is not English.[21] Students attending these schools are vaccinated based on age, as opposed to school grade. Routine school-based vaccination programs may not reach adolescents in some remote communities who do not regularly attend school or those who are home-schooled. Adolescents in these communities are offered nationally recommended vaccines for their age by local public health providers and/or their local doctor. There have been isolated reports of schools refusing to participate in school-based vaccination programs, often due to a philosophical objection to vaccination.[22]

Program delivery

School-based vaccination programs are delivered at a regional level, with local teams of trained vaccine providers offering vaccines in schools within a defined geographical region (Table 2). Vaccines are offered at the school, during school hours, and more than one vaccine may be offered at each vaccination clinic. Dates for school visits are organised by local program providers annually, with each school receiving a minimum of three visits per school year although some have up to five.

Table 2: School-based vaccination program delivery in Australia, by state and territory

	Australian Capital Territory	New South Wales	Northern Territory	Queensland	South Australia	Tasmania	Victoria	Western Australia
Local coordination	Maternal and Child Health Unit of ACT Health Department	PHU*	Community Health and Remote Health units	3 area coordinators based at PHU* (Northern, Central, Southern)	Local government or area health services (metro) Country Health SA (rural)	Local government	Local government	Local government or Child and Adolescent Health (metro), PHUs* (rural)
Vaccines administered by	Maternal and Child Health nurses	Nurses employed via local health districts† (metro), Community Health nurses (rural)	Health Promoting School Nurses‡ and Community/ Health Department nurses	Community Health nurses, council Immunisation Program Nurses§ or general practitioners/ practice nurses	Council and Community Health nurses§ and general practitioners/ practice nurses	Council nurses§	Council nurses§	Community Health & council nurses (metro), PHU* nurses (rural)§
Age of student where parental consent required	<15yrs[62]	all school students regardless of age[24]	all students vaccinated on school grounds‖	<15yrs[25]	<16yrs[63]	<18yrs[64]	<18yrs[65]	<18yrs[66]

* Public health units are located within regional offices of some state/territory health departments . They are responsible for surveillance activities and public health response within their geographical region.

† The NSW Health service is divided into 15 Local Health Districts, which are regional units of the state health department, and which direct and manage publically funded health services within their geographical areas.

‡ Nurse located within public high schools in the Northern Territory providing holistic health care to the school community through management of clinical and health promotion services within the high school and the feeder primary schools.

§ Predominantly registered nurses (Registered Nurses/Division 1) or Immunisation Program Nurses (in Qld who have completed an accredited course). Also includes endorsed enrolled nurses (Endorsed Enrolled Nurses/Division 2) in some areas.

‖ Personal communication: Immunisation Senior Project Officer, Centre for Disease Control, Department of Health and Families, Northern Territory: 2010

Operational issues such as the number of schools visited each day, the number of visits to each school and the order in which vaccines are offered are influenced by numerous factors including school size, geographic location of schools, curriculum activities and numbers of schools in the area served by each local school-based vaccination provider.

Arrangements to catch up students who miss out on scheduled vaccines offered through school-based vaccination programs are varied. Some jurisdictions enable vaccines requiring multiple doses to be administered at subsequent visits to the school but this is often only possible for earlier doses. Most commonly, any age-eligible students can obtain missed doses for free from their local vaccination provider (consultation fees may apply).

State and territory policy and procedure documents detail the planning, set up, operation and reporting procedures for school-based vaccination programs.[23-25] Clinics at the school are set-up to maximise the flow of students from the pre-defined waiting area where they read and/or hear pre-vaccination information. They then move to the nurse's station where they are vaccinated out of sight from other students (where possible) and then to the designated observation area where they wait for a minimum of 15 minutes before returning to the classroom. Clinic operation and set-up is designed to maximise efficiency and optimise the vaccination experience for all involved,[26] whilst reducing the potential for errors and mass psychogenic responses which may occur in such programs.[27]

Each jurisdiction develops their own information materials (e.g. brochures) that are primarily targeted at parents and/or guardians and delivered *en masse* as part of, or along with, consent forms. Written information about the operational aspects of the program and the vaccines offered is provided to school teachers and/or principals in only half of the states and territories. There is no requirement for jurisdictional health(as of 2010) or education departments to provide education about the vaccines offered or the diseases that they protect against.[19] However half of the states and territories provide some education to students or offer materials for teachers to do so (Table 3).[28,29] There is frequently limited opportunity to provide meaningful education to students in their first year of secondary school due to the need for vaccination clinics to commence early in the school year in order to offer all vaccines and complete multi-dose schedules.

State and territory health departments maintain comprehensive records of vaccines delivered through their school-based vaccination programs. However reporting mechanisms, the type of data, and the timing of collection varies. Queensland and the Northern Territory have state and territory-wide vaccination registers that store records of all NIP vaccines administered in these jurisdictions.[30,31] More recently, Western Australia and New South Wales have developed statewide adolescent school-based vaccination registers (Table 3).[32,33] In jurisdictions without registers, individual line listings or aggregate numbers of students vaccinated are reported to state and territory health departments. Recording of adolescent vaccines administered outside school-based vaccination programs is variable. In some jurisdictions, there are established mechanisms for voluntary reporting of these data to either the state and territory health department (e.g. fax-back form) or directly to state and territory immunisation registers. State and territory health departments are required to report individual records of HPV vaccines administered in school-based vaccination programs to the National HPV Vaccination Program Register (NHPVR).[34]

Coverage data are most frequently used by each state and territory health department to allocate funding to those organisations responsible for implementing the program and to monitor program performance. They are often shared on request with those making decisions about vaccination programs at a state and national level.

Consent

Each state and territory has their own legislation governing the age of consent to medical treatment. In some jurisdictions the education department also influences school-based vaccination program consent policy. Thus, the age at which adolescents have the right to provide consent for themselves, and are permitted to do so in a school-based vaccination program, varies between states and territories (Table 2).

In general, written parental consent is required for all vaccines delivered through school-based vaccination programs in Australia, though in the absence of this verbal consent documented and witnessed by the school-based vaccination provider is acceptable in most states and territories. Each jurisdiction develops standard consent forms that are either vaccine-specific (e.g. for HPV vaccine only) or grade-specific (i.e. all vaccines offered in a selected grade on one consent form), or a combination of these. Consent for multiple doses of the same vaccine (e.g. hepatitis B) is provided on the same consent form. With the exception of New South Wales, 'neutral consent' is employed in all jurisdictions whereby consent forms are returned regardless of the consent decision. In New South Wales, 'opt-in' consent is employed whereby consent forms are returned only if the parent/guardian consents to their child receiving the vaccine covered by that form.[24]

Table 3: School-based vaccination program materials and resources 2010, by state and territory

Materials and recourse	ACT	NSW	NT	QLD	SA	TAS	VIC	WA
Policy and procedures document*	✓	✓		✓	✓	✓	✓	✓
Standard consent form across state/territory†	✓	✓	✓	✓	✓	HPV only	✓	✓
Vaccine-specific parent information†	✓	✓	✓	✓	✓	✓	✓	✓
Post-vaccination care information‡	✓	✓	✓	✓	✓	✓	✓	✓
Student record of vaccination	✓	✓	✓	✓	✓	✓	✓	✓
Parent notification card/letter for missed vaccine		✓	✓					✓
Policy for independent nurse immunisers§		✓		✓	✓	✓	✓	✓
Jurisdictional immunisation register incl. adolescent vaccines		✓	✓	✓				✓
School-based vaccination program information on dedicated page of the state/territory health department website	Schedule only	✓	Schedule only‖	✓	✓	On local government websites	Schedule only	✓
Education material/resources for students¶			✓		✓	✓	✓	
Specific system for notification of adolescent vaccines administered outside the school-based vaccination program**			NT immunisation register	VIVAS††	✓			✓
Standard information for principals/schools	✓	✓		✓				✓
Student pre-vaccination checklist/advice card		✓			✓			

Abbreviations for each state/territory: ACT = Australian Capital Territory, NSW = New South Wales, NT = Northern Territory, QLD = Queensland, SA = South Australia, TAS = Tasmania, VIC = Victoria and WA = Western Australia.

* Overarching document outlining jurisdictional policy and procedures for preferred model of delivery. Usually includes standard forms and templates.

† In SA, ACT, Qld and NT the consent form and disease/vaccine information for parents is provided in the same resource, not two separate pieces of paper.

‡ This is either included on the student record of vaccination or provided on a separate piece of paper following vaccination.

§ This document links to specific state/territory poisons legislation and outlines scope of practice for the delivery of vaccines in school-based vaccination program by nurses qualified to administer vaccines independent of a doctor, usually under standing orders at a jurisdictional, local or organisational level.

‖ This website has since been updated to include more detail on vaccines available, school grades in which these are offered and consent forms.

¶ Education materials (i.e. classroom lessons, videos) made available through the state/territory health department for those implementing the program or classroom teachers to educate students about vaccines offered through school-based vaccination programs and the diseases they protect against. Education may be provided in some, but not necessarily all areas of the state/territory. This excludes pre-vaccination information sheets for students provided just prior to vaccination.

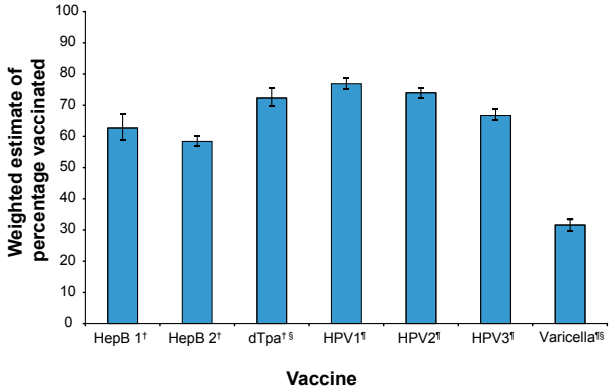
** For community vaccination providers (e.g. general practitioners) to report nationally recommended vaccines administered to adolescents to the relevant state/territory health department.

†† Vaccination Information and Vaccination Administration System.

Most commonly, the jurisdictional health department provides hard copies of standard consent forms to local school-based vaccination provider/s. They disseminate these to schools in their area, with timing and practices largely dependent upon the population and geographic size of the service provider's area. Teachers predominantly assume the responsibility of disseminating consent forms to students in their class. Students are responsible for taking consent forms home and returning them to school once completed.

Several strategies have been trialed to increase consent form return by school-based program providers in Australia, but there is limited published evidence for these. Resending the consent form/information package to non-responders was shown to be significantly associated with increased return rates in Western Australia.[32] Some interviewees reported that distributing consent forms at the beginning of the school year yielded a greater response than posting them with school enrolment forms at the end of the school year. In one jurisdiction, telephoning non-responders to request a completed form or obtaining verbal consent frequently led to a 100% response rate and posting consent forms with a reply paid envelope did not alter return rates and resulted in fewer opportunities for reminders, as school teachers were not directly involved.

Figure 1: Average national coverage for adolescent vaccines routinely delivered through school-based vaccination programs in Australia, 2004 to 2009*



* Error bars are the lowest and highest weighted national coverage estimate recorded from 2004 to 2009.

† Hepatitis B1 and Hepatitis B2 includes data from 2004–2009, excluding ACT (2007), SA (2004–2007), Qld (2004–2006) and NT (All years).

‡ dTpa includes data from 2005–2009, excluding NSW (2006–2008), Tas (2005–2006), SA (2007), Qld (2005–2006).

§ Data for dTpa and Varicella vaccines from the NT includes only those students vaccinated in the school-based vaccination program. Adding eligible adolescents vaccinated in the community to those vaccinated at school considerably increased overall coverage (Table 4).

‖ HPV dose 1–3 (HPV1–HPV3) includes data from 2007–2009 only. Estimates include all school grades offered the vaccine across these calendar years.

¶ Varicella includes data from 2006–2009, excluding ACT (2007), WA (2006), Qld (2006). Coverage estimates exclude students who reported a clinical history of chickenpox or having received the vaccine prior to being offered it through school-based vaccination program.

Coverage

Coverage data for the school-based vaccination program was somewhat complete across jurisdictions for all vaccines offered from 2004 to 2007, with complete coverage data for all vaccines and jurisdictions available for 2008 and 2009 (Table 4). In most jurisdictions, coverage data for vaccines offered through school-based vaccination programs prior to 2004 was insufficient or not available. Data for Tasmania were not available from all local government areas with the number providing data varying by calendar year and vaccine. Whilst the estimates for the Northern Territory are based on doses received in the school setting, a considerable number of age eligible dTpa and varicella vaccine doses were recorded on the Northern Territory immunisation register as being delivered in the community. Coverage estimates for South Australia, Queensland, Western Australia and Victoria included an unknown number of vaccine doses administered to age-eligible adolescents outside the school-based vaccination program, as reported to the relevant state and territory health department, but these were understood to be minimal. As not all doses are routinely reported by community vaccination providers (e.g. general practitioners), reported coverage for subsequent doses, may be higher than for previous doses. Dose assumptions, as have been described in the calculation of Australian childhood immunisation coverage rates,[35] are used when calculating school-based vaccination coverage in some jurisdictions (e.g. Victoria).

There is no published coverage data for vaccines delivered through schools in Australia prior to the National Measles Control Campaign in 1998, which predominantly focussed on primary school children.[36] Data from early adolescent school-based vaccination programs in South Australia shows coverage for the grade 8 (13 to 14 years) schoolgirl rubella program (1990–94) ranged from 71% to 78%, which was similar to that achieved when the measles-mumps-rubella (MMR) vaccine was delivered to all grade 8 students from 1995 to 1997 (78 to 81%). Coverage from time-limited catch-up national adolescent school-based vaccination campaigns for meningococcal C and dTpa show variation in coverage by school grade. When multiple school grades are vaccinated in the same year, higher uptake is evident in younger age groups,

Table 4: Percentage coverage for vaccines delivered through routine school-based vaccination programs, 2004 to 2009, by state or territory and vaccine

Year	ACT	NSW	NT	QLD	SA	TAS*	VIC	WA	National†
Hepatitis B dose 1‡									
2004	77	56	–	n/a	n/a	n/a	82	n/a	67
2005	77	59	–	n/a	n/a	n/a	61	n/a	60
2006	76	57	–	n/a	n/a	76	58	n/a	59
2007	–	58	–	61	n/a	79	60	69	61
2008	82	65	–	65	74	76	56	69	65
2009	66	63	–	65	80	63	56	70	61
Hepatitis B dose 2‡									
2004	69	43	–	n/a	n/a	n/a	76	n/a	57
2005	72	48	–	n/a	n/a	n/a	74	n/a	59
2006	69	46	–	n/a	n/a	69	70	n/a	57
2007	–	49	–	50	n/a	72	74	67	59
2008	69	48	–	58	70	63	72	64	60
2009	59	50	–	78	73	58	75	65	59
dTpa									
2005	83	72	58 (25)§	n/a	86	n/a	78	77	76
2006	81	–	73 (36)§	n/a	84	n/a	78	69	77
2007	80	–	81 (45)§	61	–	70	78	71	71
2008	89	–	82 (55)§	63	75	59	76	76	71
2009	81	68	88 (59)§	62	77	59	74	81	70
Varicella									
2006	15	33	33 (13)§	n/a	37	n/a	13	n/a	30
2007	–	36	28 (13)§	32	33	11	33	29	32
2008	12	34	32 (18)§	32	22	20	41	44	32
2009	19	34	36 (23)§	35	43	23	34	29	34
HPV dose 1‖									
2007	80	83	80	74	70	68	83	71	76
2008	80	77	75	74	80	49	77	69	75
2009	73	80	79	76	83	59	80	82	79
HPV dose 2‖									
2007	75	80	71	79	65	65	78	67	74
2008	78	75	72	69	77	46	74	66	72
2009	71	77	73	72	80	59	77	79	76
HPV dose 3‖									
2007	63	74	64	62	64	56	71	60	65
2008	65	68	62	64	72	40	67	60	66
2009	58	69	67	66	71	55	72	72	69

Abbreviations for each state/territory: ACT = Australian Capital Territory, NSW = New South Wales, NT = Northern Territory, QLD = Queensland, SA = South Australia, TAS = Tasmania, VIC = Victoria and WA = Western Australia.

Other abbreviations: n/a = data not available and – = vaccine not offered.

* The school enrolment population used for Tasmania is the average of grade 6 and 7 enrolments. Data for Tasmania does not include all council areas.

† National estimates are weighted by Australian Bureau of Statistics (ABS) full-time equivalent school enrolment population for the grade/s targeted for each year and vaccine. Estimates exclude states/territories for which data was not available for that year/vaccine

‡ The NT does not offer hepatitis B in routine school-based vaccination programs due to a long standing universal infant vaccination program in which the adolescent cohort has been vaccinated.

§ The first figure is proportion of age-eligible students enrolled in targeted school grade(s) (as per ABS enrolments) vaccinated in either the school or community setting, with the figure in brackets the proportion of these vaccinated in the school-based vaccination program only.

‖ HPV vaccine was offered across multiple school grades in 2007 and 2008 though this varied by state/territory.[9] The denominator is the total ABS enrolments for all grades offered the vaccine in each calendar year. The numerator is the number of enrolled students from all grades offered the vaccine who reported receiving it.

¶ Jurisdictional coverage estimates obtained from Brotherton et al.[18]

with the highest uptake being achieved in the last two years of primary school and first two years of secondary school.[33,37]

The average weighted national coverage estimates for adolescent vaccines routinely delivered through school-based vaccination program varied (6%–10%) within a jurisdiction for each vaccine across calendar years (Figure 1, Table 4). Coverage differed somewhat across jurisdictions for each vaccine, most notably for dTpa, hepatitis B (dose 2) and varicella. For most vaccines, coverage was generally lower in the first year the vaccine was offered, compared to any subsequent years, though this was not the case in all states and territories (Table 4). Across the calendar years, national coverage for the two-dose hepatitis B catch-up program ranged from 57% to 60% nationally, which is slightly lower than dTpa (70% to 77%) and the first dose of HPV (75% to 79%). The uptake of a single catch-up dose of varicella vaccine has been consistently low (30% to 34%) as it is influenced by the number of students reporting a history of chickenpox or previous vaccination. Although most jurisdictions routinely request parents to report a student's previous history of the disease and/or vaccination, this is not validated, complete or routinely included in coverage estimates. In the Northern Territory, a comparison of school versus community delivered varicella and dTpa vaccines from 2005–09 showed a declining trend in community vaccination (e.g. 37% in 2006 to 29% in 2009 for dTpa), however for the observed period there was an increase in the number vaccinated overall (Table 4).

For 2009, the 3 dose coverage estimate for HPV vaccine was slightly lower (1.8%) than that reported by the NHPVR.[38] This may be due to differences in data collection and analysis methods and/or inclusion of more reported doses delivered outside of routine school-based vaccination programs in NHPVR estimates. Thus, data presented here should be considered minimum estimates of coverage for the adolescent population.

Discussion

In Australia, school-based vaccination is now the primary method to deliver nationally recommended vaccines to adolescents. However there is substantial variation between states and territories in how programs are funded, managed and implemented. This is largely due to differences in state and territory health systems, legislation, geography, and population size and characteristics. Despite this, the approach has largely overcome missed opportunities for vaccinating adolescents in traditional healthcare settings and good coverage has been achieved in all states and territories.

Whilst there is currently no national benchmark for the implementation of adolescent school-based vaccination programs, existing arrangements align with international standards for child and adolescent immunisation practices in terms of vaccine availability, communication, patient assessment, vaccine storage, administration and documentation.[39] Integral to the success of these programs are refined policy and procedural guidelines, highly skilled and well-trained vaccine service providers, and comprehensive and efficient consent processes. Maximising consent form return rates is a continuing challenge facing school-based programs in Australia and internationally.[40-42] The need for national consistency regarding the inclusion and wording of some questions on school-based vaccination program consent forms (e.g. Indigenous status) warrants future consideration. Dissemination of the results of trialed implementation approaches and strategies may enhance the current evidence base. However gaps still remain in understanding the association between many procedural factors and vaccine uptake/completion.

Gaining support from parents and teachers, and ensuring that they are adequately informed are ongoing challenges, as is working with the education sector who may not view schools as an appropriate place for the delivery of health care in an already crowded school curriculum.[26,43] There is international evidence to support an increase in parental knowledge following the provision of an information sheet as well as increased rates of parental consent following education.[44-48] However, challenges with ensuring written information about vaccination for adolescents reaches parents and that they read and understand it have been identified.[49,50]

The coverage achieved in Australia's school-based vaccination program is higher than in settings where adolescent vaccines are delivered through the community sector or private practice.[51,52] Australia's coverage for HPV vaccine is one of the highest in the world to date.[53-55] Coverage for dTpa, varicella and hepatitis B were higher than that achieved through routine school-based vaccination in British Columbia, Canada,[53] despite the inclusion in their estimates of those with prior natural infection and those who self-reported receiving vaccines outside the school setting. A nationally agreed standard for the collection of coverage data from school-based vaccination programs in Australia would assist in developing a more accurate understanding of nationwide coverage achieved in these programs, which is imperative for future monitoring and evaluation of adolescent vaccination programs.

As has been demonstrated in previous studies, there is higher uptake of vaccines in lower school grades, particularly the final year of primary school

and first year of high school.[33,37] This may be due to higher attendance rates in lower school grades,[56] or that parental consent is less frequently required for older adolescents and where it is, it may be more challenging to obtain due to reduced rates of school attendance and increasing independence and cognitive maturity at this age. The trend of lower coverage for newly introduced vaccines, especially in the first year of the program has been observed previously in both Australia and other countries.[33,37,57] This may be due to a lack of awareness or misconceptions about the vaccine and/or its availability in the school-based vaccination program that may be overcome with time and use of sound communication strategies.

Obtaining details of adolescent vaccines delivered outside school-based vaccination programs remains a challenge. Various methods have been employed to capture these data, though they rely heavily on provider initiative to report doses administered to their state/territory immunisation register or health department without any financial incentive, as is provided for routine reporting of childhood vaccines.[58] In the absence of a national register for adolescent vaccines, a computer-assisted telephone interview (CATI) survey, as is used to determine population-wide coverage in adults could be an alternative mechanism to determine population-wide coverage.[59] However, there are numerous limitations (e.g. responder bias) with this method as have been experienced in the United States.[60] For some diseases, serosurveillance would be an alternative way of assessing vaccine-derived and population immunity, though it too has limitations.[61]

Future vaccination, disease surveillance and control programs will need to take into consideration adolescent cohorts vaccinated through routine school-based vaccination programs. The continuation of hepatitis B catch-up vaccination for the cohort of adolescents who received the universal birth dose at the commencement of this program will require consideration to ensure an acceptable level of seroprevalence is achieved in this cohort. Future pertussis revaccination strategies for adults will need to incorporate guidelines for those who received dTpa as an adolescent. HPV vaccination status will be important when following up disease and when developing future cervical cancer screening practices. In addition, monitoring acceptance and uptake of school-based vaccination programs among future generations of parents and students as well as addressing identified gaps in evidence will be important to ensure the continued success of voluntary school-based vaccination in Australia.

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A history of adolescent school-based vaccination in Australia

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Abstract

As adolescents have become an increasingly prominent target group for vaccination, school-based vaccination has emerged as an efficient and effective method of delivering nationally recommended vaccines to this often hard to reach group. School-based delivery of vaccines has occurred in Australia for over 80 years and has demonstrated advantages over primary care delivery for this part of the population. In the last decade school-based vaccination programs have become routine practice across all Australian states and territories. Using existing records and the recollection of experts we have compiled a history of school-based vaccination in Australia, primarily focusing on adolescents.

Key words: immunization, vaccination, adolescent, school vaccination

Introduction

Many developed countries, including Australia, now recommend routine vaccination of adolescents. This provides protection against future disease risk, boosts existing but waning immunity, and forms part of catch-up programs for those who may not have been adequately vaccinated as children.[1,2] To attain high vaccine uptake and to contribute to disease control, efficient and effective ways of delivering vaccines to this often hard to reach group are needed.[3] Schools have long served as sites to successfully deliver one-off mass vaccination of children and adolescents, both in Australia and overseas.[4-6] Over the last two decades, school-based vaccination programs have been developed in each Australian state and territory to routinely deliver adolescent vaccines on the National Immunisation Program (NIP). This, approach has demonstrated significant advantages over delivery through general practitioners (GPs) alone.[7,8] However it has not been uniformly introduced nationally due to the differences in political support and the legislative and public health systems across the states and territories.

Managed at a state or territory level, current school-based vaccination programs in Australia offer nationally recommended and Australian Government funded vaccines to adolescents in specific school grades using local teams of trained vaccination providers. State and territory governments primarily fund service delivery, although the Australian Government has provided funding for the delivery of some vaccines. Vaccines are routinely offered to eligible students in targeted school grades, typically late primary school or early secondary school and are not mandated for school entry. Participation is voluntary, and written parental consent is required for each course of vaccine.

Much of the historical literature about vaccination in Australia omits detail on school-based delivery. There is currently no single complete account of the evolution of adolescent school-based vaccination in Australia as there is for childhood vaccination.[9,10] As part of a review of adolescent school-based vaccination in Australia information on the evolution of each state or territory school-based vaccination program was obtained through interview with representatives from eight jurisdictional health departments. Where possible, formal written records were identified to supplement and verify the information provided at the time of interview. This information was used to compile the following historical account of school-based vaccination in Australia, primarily focusing on adolescent programs though including relevant national catch-up campaigns targeting younger school-aged children.

Pre-1970s

One of the earliest recorded national school-based vaccination programs in Australia provided the diphtheria–tetanus toxoid (dT) vaccine from 1932 to 1936.[11] This was followed by the introduction of the bacille Calmette-Guerin (BCG) vaccine, delivered through schools in some jurisdictions from the late 1940s to the mid-1980s. The Northern Territory was the only jurisdiction to continue routinely offering this until 1990 (Table 1).[12,13] Some states and territories also used schools to deliver polio vaccine during the 1950s and 1960s as part of larger mass vaccination programs.

1970s to the late- 1990s

The national schoolgirl rubella vaccination program commenced in 1970–71 to vaccinate females prior to, but as close as possible to potential pregnancy with a view to reducing the incidence of congenital rubella. The vaccine was offered to one cohort of girls aged 10–14 years and delivered in school grades 6, 7 or 8 in all jurisdictions except Queensland.[9,14] From 1993-94 to 1997, the schoolgirl rubella program was replaced by the routine vaccination of both boys and girls with the combined measles-mumps-rubella (MMR) vaccine.[14] This program set the scene for

a more consistent adoption of school-based vaccination in some states and territories. Although some continued with the school-based approach to deliver adult diphtheria–tetanus vaccine (ADT) and oral polio vaccine (OPV) to adolescents, more than 25 years elapsed before the next major national school-based vaccination program was implemented (Table 1).

Following a recommendation of the Australian Technical Advisory Group on Immunisation (ATAGI), hepatitis B catch-up vaccination commenced in 1998-99 for all adolescents aged 10–16 years.[15] Three doses of the vaccine were incorporated into existing school-based vaccination programs in some jurisdictions, although in Western Australia, New South Wales and most areas of Queensland the vaccine was initially provided by GPs.[16,17] The Northern Territory had provided universal infant hepatitis B vaccination from 1990 hence, most school children already received the vaccine. Consequently, a one-off catch-up program for all school students was implemented during 1998-99.[18]

National school-based vaccination campaigns from the late 1990s to early 2000s

School-based vaccination developed further in the late 1990s and early 2000s when the Australian Government funded all jurisdictions to deliver two 'whole of school' vaccination programs. The first of these was the National Measles Control Campaign in 1998; a catch-up campaign for all primary school students (5–12 years of age).[19] One-off funding for this program from the Australian Government saw the establishment of state/territory coordinated school-based vaccination programs in some jurisdictions and the enhancement of existing programs in others. This was followed in 2003 by the National Meningococcal C Vaccination Program, targeting all children aged 1 to 19 years.[20] The catch-up component of the program was delivered in both primary and secondary schools in two phases; students aged 15 to 19 years (grades 9 to 12); and students aged 6 to 14 years (pre-school to grade 8), plus any students aged 15 to 19 years who had missed being vaccinated in phase one. In addition to funding the vaccine, the Australian Government provided time-limited funds to support school-based delivery of the catch-up program in all states and territories.

Routine school-based vaccination from early 2000s

As more evidence of the success of school-based vaccination emerged and the number of vaccines recommended for ongoing delivery to adolescents grew, school-based vaccination programs were established or re-established in more states and territories. In 2000, the nationally recommended cohort for receipt of the adolescent catch-up dose of hepatitis B was revised to include only 10 to 13 year olds, and from 2000–04 jurisdictions shifted to a two dose schedule.[10] Hepatitis B vaccine is now provided routinely in all jurisdictions, either in the last year of primary school or the first year of secondary school. However it is currently scheduled to cease around the time when the first age-cohort eligible for the universal birth dose enters secondary school.

The availability of an adult formulation of diphtheria-tetanus-acellular pertussis vaccine (dTpa),[21,22] and the recognition of increasing pertussis incidence in adolescents prompted the addition of dTpa vaccine to the NIP in November 2003.[23] This replaced the previously recommended ADT booster dose for 15 to 17 year olds. The Australian Government funded all states and territories to provide school-based delivery of the vaccine from 2004.[24] In some jurisdictions the implementation simply involved the replacement of ADT with dTpa in existing school-based programs; in others it required establishment or re-establishment of these programs (Table 1). The vaccine continues to be offered in later secondary school grades, though more recently there has been a recommendation for it to be offered at a younger age (11 to 13 years) in either the last year of primary school (grade 6 or 7) or first year of secondary school (grade 7 or 8).[25]

In 2005, varicella vaccine was included on the funded NIP schedule at 18 months and as a catch-up for non-immune adolescents aged 10 to 13 years.[26] From 2006 the adolescent dose was delivered through existing jurisdiction-wide school-based vaccination programs in all states and territories except Queensland.[27] The catch-up dose of varicella vaccine for adolescents is currently scheduled to cease after 2015, when the first cohort eligible for the infant dose will reach the age at which the adolescent catch-up dose is currently offered.

By the mid-2000s all states and territories except Queensland had established routine jurisdiction-wide school-based vaccination programs. In Queensland, nationally recommended adolescent vaccines were predominantly delivered through general practitioners, although routine school-based vaccination programs operated in some local government areas and health service districts. Time-limited statewide school-based vaccination occurred to deliver Australian Government funded national vaccination campaigns until 2007 when the statewide school-based vaccination program commenced in the state.[8]

The most recent addition to the routine school-based vaccination program in Australia came with the National Human Papillomavirus (HPV) Vaccination Program. From April 2007 the quad-

Table 1: School-based vaccination programs, Australia, from 1970–2013

Year commenced	Vaccine	Year ceased	Jurisdiction(s)*	School grade[†] [‡]
Measles-mumps-rubella (MMR) containing vaccines				
1971	Monovalent rubella (females only)	1993	NSW, NT, Vic,Tas, WA, ACT	Grade 6 or 7
			SA	Grade 8
1993	MMR	1997	WA	Grade 7
1994		1997	NSW, ACT, Vic, NT	Grade 7 NSW
				Grade 6 ACT, Vic & NT
			SA	Grade 8
			Tas	Grade 6 or 7
1998	MMR	1998	All	All primary school[19]
Diphtheria-tetanus-pertussis containing vaccines (ADT, dTpa)				
1980	ADT	2003	ACT	Grade 9
1994		2000	Vic	Grade 10
		2003	SA	1994–2001:Grade 10
				2002–03: Grade 9
			Tas	Grade 10
1996		2003	NT	Grade 10
1998		2003	WA	Grade 10
2004	dTpa	Ongoing	NSW§	2004: All secondary school grades
				2005: Grade 7
				2009–12: Grade 10
				2010 onwards: Grade 7
			SA	2004: Grade 9
				2005–06: Grade 8
				2008 onwards: Grade 9
			Qld§ ‖	2004–06: Grade 10
				2007 onwards: Grade 10
			NT	2004–Nov 2005: Grade 10 only
				Nov 2005–07: Grades 8 & 10
				2008 onwards: Grade 8
			WA	2004: Grade 7 & Grades 8–12
				2005 onwards: Grade 7
			Tas, Vic	Grade 10
			ACT	Grade 9
Tuberculosis				
1986	Bacillus Calmette-Guerin (BCG)	1990	NT	Grade 9
Oral polio vaccine (OPV)				
1994	OPV[§]	2000	Vic	Grade 10
		2002	SA, Tas	Grade 10
		2003	ACT	Grade 9
1996	OPV	2002	NT	Grade 10
Hepatitis B (2 and 3 dose schedules)[¶]				
1998	Hepatitis B	1999	NT**	First year of school to Grade 10
			Vic	Grade 7
			Tas	Grade 6 or 7

Table 1 *continued*: School-based vaccination programs, Australia, from 1970–2013

Year commenced	Vaccine	Year ceased	Jurisdiction(s)*	School grade†‡
1998/9	Hepatitis B	Ongoing at present††	ACT	1999–00:Grade 6 2001–06: Grade 6 2008–12: Grade 7
			SA	Grade 8
Hepatitis B				
2002		Ongoing at present††	WA	Grade 7
2004			NSW§	Grade 7
2007			Qld	Grade 8
Pneumococcal (Aboriginal and Torres Strait Islander only)				
2001	Pneumococcal polysaccharide vaccine, 23 valent (23vPPV)	Ongoing	NT	2001: Grade 10 to 12 2002: Grade 11 or 12 2003 onwards: Grade 10
Meningococcal C				
2003	Meningococcal C	2004	ACT	2003: Grade 6, 9 & 10–12 2004: Pre-school – Grade 5 & Grade 8
		2004	NSW, WA	2003: All secondary school 2004: All primary school
		2004	QLD‖	2003: All secondary school 2004: All primary school & Grade 8
		2005	SA	2003: Grades 9–12 2004: Grades 8–9 2005 All primary school
		2005	Vic, Tas	2003: Grades 9–12 2004–05:Grades 1–9
		2006	NT	2003: Grades 10–11 2004: Pre-school to Grade 9 2005–2006: Any student aged 1–19yrs in 2003–04 not previously vaccinated
Varicella‡‡				
2006	Varicella	2015	NT, SA	Grade 8
		2015	NSW, ACT, Vic, WA	Grade 7
		2015	Tas	Grade 6 or 7
		2015	Qld	2006: Grade 8[e] 2007 onwards: Grade 8
Human papillomavirus (HPV)				
2007	Quadrivalent HPV vaccine (females only)	Ongoing	NSW, ACT	2007: Grades 7 & 10–12 2008: Grades 7 & 9–10 2009 onwards:Grade 7
			NT, WA	2007: Grades 10–12 2008: Grades 7–10 2009 onwards: Grade 7
			SA	2007: Grades 8–12 2008 onwards: Grade 8

Table 1 *continued*: School-based vaccination programs, Australia, from 1970–2013

Year commenced	Vaccine	Year ceased	Jurisdiction(s)*	School grade†‡
			Tas	2007: Grades 7 & 10–12
				2008: Grades 7–10
				2009 onwards: Grade 7
			Vic	2007: Grade 7, 10–12
				2008: Grade 7, 9–10
				2009 onwards: Grade 7
			Qld	2007: Grades 10–12
				2008: Grades 8–10
				2009 onwards: Grade 8
2013	Quadrivalent HPV vaccine (males) §§	Ongoing	NSW, ACT, NT, Vic, Tas	2013 & 2014: Grades 7 & 9
				2015 onwards:Grade 7 only
			SA	2013: Grades 9
				2014: Grades 8 & 9
				2015 onwards: Grade 8
			Qld	2013 & 2014: Grades 8 & 10
				2015 onwards: Grade 8
			WA	2013: Grades 8, 9 & 10
				2014 onwards: Grade 8

* Abbreviations for each state/territory: ACT = Australian Capital Territory, NSW = New South Wales, NT = Northern Territory, Qld = Queensland, SA = South Australia, Tas = Tasmania, Vic = Victoria and WA = Western Australia.

† Ages for each school grade differ between jurisdictions. Approximate ages are as follows: pre-school, 3–6 years; grade 1, 6–7yrs; grade 2, 7–8yrs; grade 3, 8–9yrs; grade 4, 9–10yrs; grade 5, 10–11yrs; grade 6, 11–12yrs; 7, 12–13 years; grade 8, 13–14 years; grade 9, 14–15 years; grade 10, 15–16 years; grade 11, 16–17 years; grade 12, 17–19 years.

‡ In South Australia, Western Australia and Queensland grade 7 is the final year of primary school. In all other jurisdictions grade 7 is the first year of secondary school.

§ Year vaccine first routinely provided through state-wide school-based vaccination program. Prior to this, vaccine may have been provided through local government run school-based programs in some areas of the state.

‖ Prior to 2007 in Queensland, nationally recommended adolescent vaccines were mainly delivered by general practitioners. However, some local governments and Health Service Districts implemented their own school-based vaccination programs but these were not managed by centrally by Queensland Health. Only those vaccines provided through Queensland-wide school-based vaccination programs including those provided in one-off national mass-vaccination campaigns (e.g. MMR, Meningococcal C) are listed in this table.

¶ Change from 3-dose to 2-dose dose schedule occurred in 2001 in Victoria and in The Australian Capital Territory, 2002 in South Australia and 2003 in Tasmania. All other jurisdictions delivered only 2-dose scheduled through school-based vaccination programs.

** A one-off cohort based catch-up program for all those born since 1982 was run from February 1998 to April 1999. Hepatitis B vaccine is not offered in the NT routine school-based vaccination program as universal infant hepatitis B vaccination commenced in 1990 with at- risk infants vaccinated since 1988.

†† Under review at time of publication.

‡‡ Varicella vaccine is recommended for one cohort of those aged 10–13 years unless they have a clinical history of chicken pox or have already received the vaccine.

rivalent HPV vaccine became freely available to females aged 12 to 26 years with those aged 12 to 18 years primarily vaccinated at school.[28] During 2007–08 a catch-up program for all female secondary school students was delivered using an accelerated schedule with targeted school grades varying across jurisdictions.[29] From 2009 HPV vaccine has been provided routinely to girls aged 12 to 13 years of age in either the last year of primary school (grade 7 or 8) or first year of secondary school (grade 7 or 8). From February 2013 school-based vaccination programs commenced offering HPV vaccine to males aged 12-13 years (grade 7 or 8) on an ongoing basis. Catch-up for the HPV vaccine series (3 doses) is also being offered to males aged 14 to 15 years until the end of the 2014 school year.

Impact of school-based vaccine programs in Australia

As part of larger mass vaccination programs, school-based delivery of diphtheria, polio, and tuberculosis vaccines contributed substantially to the rapid decline of these diseases in the mid to late 1900s.[9,11] Although coverage data from early programs is limited, state/territory data from routine school-

based vaccination programs illustrates good uptake (65% to 80%), particularly in late primary school and lower secondary school grades.[24,29,30] Uptake in routine school-based vaccination programs has been consistently higher than that achieved by other strategies to vaccinate adolescents, such as mandates for school entry,[31] or GP delivery.[7,8,16] Evaluations of the impact of several school-based vaccination programs have identified significantly higher levels of immunity in population cohorts vaccinated through these programs compared with those who have not.[32-34] Despite evidence of their success there is still room for improvement in school-based program delivery in Australia. This includes understanding operational factors that optimise uptake and enhancing coverage beyond the current levels to maximise population immunity as disease incidence decreases.

Conclusion

The foundations for school-based vaccination in Australia have been laid, its success demonstrated and challenges highlighted. This strategy will continue to evolve in response to available evidence and increased availability of, and recommendations for, adolescent vaccines.

Acknowledgements

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Quarterly reports

Enhanced Invasive Pneumococcal Disease Surveillance Working Group – Quarterly Surveillance Report

Christina Bareja and the Enhanced Invasive Pneumococcal Disease Surveillance Working Group

Invasive pneumococcal disease (IPD) is caused by the bacterium *Streptococcus pneumoniae* and results in illnesses such as pneumonia, bacteraemia and meningitis. There are currently 92 serotypes recognised worldwide and it has been a nationally notifiable disease in Australia since 2001. The Communicable Diseases Network Australia (CDNA) established the Enhanced Invasive Pneumococcal Disease Surveillance Working Group (EIPDSWG) in 2000 to assist in developing and implementing a nationally standardised approach to the enhanced surveillance of IPD in Australia. This quarterly report documents trends in notifications of IPD occurring in Australia in the first quarter of 2013 (1 January to 31 March 2013).

Notification data are collected by all Australian states and territories under jurisdictional public health legislation and are forwarded to the Commonwealth under the *National Health Security Act 2007*. Notifications are collated nationally in the National Notifiable Diseases Surveillance System (NNDSS). The data in this report are provisional and subject to change as laboratory results and additional case information become available. The data are analysed by diagnosis date and were extracted on 16 May 2013. Consideration of vaccination status of cases is outside the scope of this report. More detailed analyses will be available in national surveillance reports on vaccine preventable diseases published by the National Centre for Immunisation Research and Surveillance (NCIRS).

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk and older Australians. The 7-valent pneumococcal conjugate vaccine (7vPCV) was added to the National Immunisation Program (NIP) schedule for Indigenous and medically at-risk children in 2001 and for all children up to 2 years of age in 2005. The 13-valent pneumococcal conjugate vaccine (13vPCV) replaced the 7vPCV in the childhood immunisation program from July 2011. The 23-valent

Table 1: Notified cases of IPD, Australia, 1 January to 31 March 2013, by Indigenous status, serotype and state or territory

	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total QTR 1 2013	Total QTR 4 2012	Total QTR 1 2012	Year to date 2013
Indigenous status												
Indigenous	0	3	12	5	2	0	2	9	33	-	-	-
Non-Indigenous	2	47	3	31	14	5	34	12	148	-	-	-
Not stated/ Unknown	0	15	0	2	0	1	15	0	33	-	-	-
Total	**2**	**65**	**15**	**38**	**16**	**6**	**51**	**21**	**214**	**360**	**235**	**214**
Indigenous status completeness* (%)	100	77	100	95	100	83	71	100	85	-	-	-
Serotype completeness† (%)	100	77	93	100	69	100	100	100	90	-	-	-

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

† Serotype completeness is defined as the reporting of a valid *S. pneumoniae* serotype or reporting that no isolate was available as diagnosis was by PCR, that the isolate was not referred to the reference laboratory, was non-typable or not viable.

pneumococcal polysaccharide vaccine (23vPPV) was added to the NIP schedule for Aboriginal and Torres Strait Islander peoples aged 50 years or older in 1999 and for non-Indigenous Australians aged 65 years or older from January 2005.

Reporting period 1 January to 31 March 2013

There were 214 cases of IPD reported to the NNDSS in the first quarter of 2013 (Table 1), similar to the number of cases reported in the same period in 2012 (235 cases).

Overall, Aboriginal and Torres Strait Islander status was reported for 85% (n=181) of cases, ranging from 71% of cases reported by Victoria to 100% of cases reported by the Australian Capital Territory, the Northern Territory, South Australia and Western Australia. Of cases with reported Indigenous status, Aboriginal and Torres Strait peoples accounted for 18% (n=33) of all cases notified in the quarter (Table 1).

Serotype information was available for 90% (n=193) of all cases reported in the quarter, ranging from 69% of cases reported by South Australia to 100% of cases reported by the Australian Capital Territory, Queensland, Tasmania, Victoria and Western Australia. The organism from one case of IPD could not be typed as the isolate was deemed not viable by the reference laboratory. This case is categorised together with cases with unknown serotypes in this report.

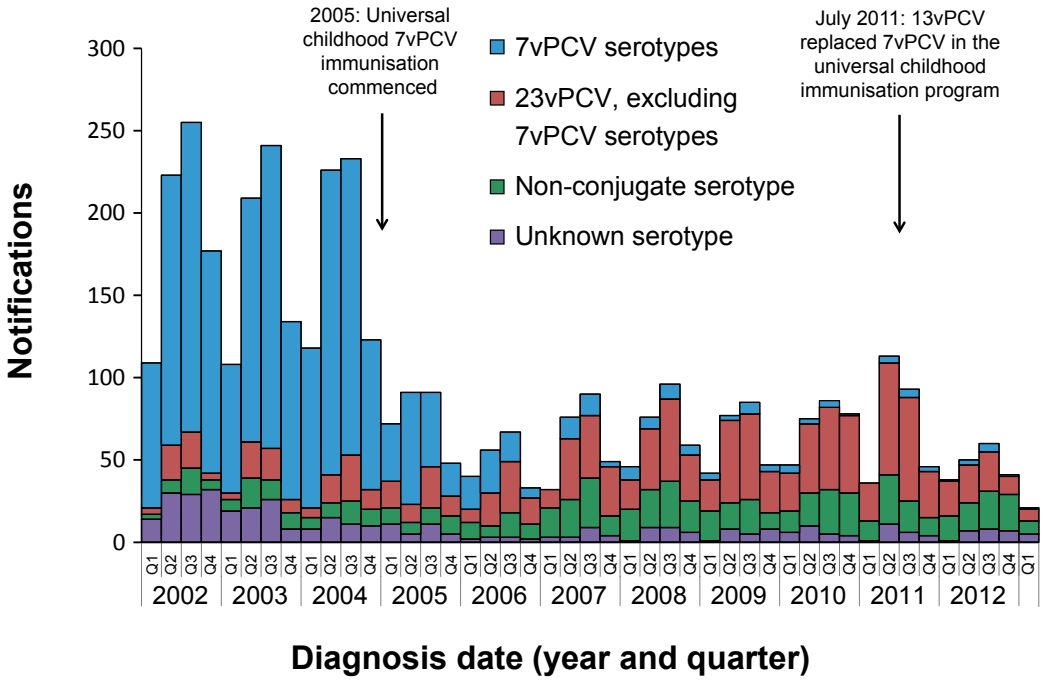
IPD in children aged less than 5 years

In the first quarter of 2013, 10% (n=21) of notified cases were aged less than 5 years. This was almost half the number reported during the same period of 2012 (n=38) and the smallest number of cases reported in any quarter since national reporting began (Figure 1: Notified cases of IPD aged less than 5 years, Australia, 2002 to the first quarter of 2013, by serotypes*).

The majority (76%, 16/21) of cases aged less than 5 years reported in the first quarter of 2013 had serotype information. Of these, half (50%, 8/16) were reported with a serotype included in the 7vPCV or the 13vPCV.

Notification of cases aged less than 5 years with disease caused by the 6 additional serotypes targeted by the 13vPCV increased steadily over the period 2007 to 2011; particularly those caused by serotype 19A (Figure 2: Notified cases of IPD caused by serotypes targeted by the 13-valent pneumococcal conjugate vaccine (excluding those targeted by 7-valent pneumococcal conjugate vaccine), aged less than 5 years, Australia, 2002 to the first quarter of 2013). However, notifications of this type have decreased since the

Figure 1: Notified cases of IPD aged less than 5 years, Australia, 2002 to the first quarter of 2013, by serotypes*



* Serotypes grouped according to targeted vaccines

Figure 2: Notified cases of IPD caused by serotypes targeted by the 13-valent pneumococcal conjugate vaccine (excluding those targeted by 7-valent pneumococcal conjugate vaccine), aged less than 5 years, Australia, 2002 to the first quarter of 2013

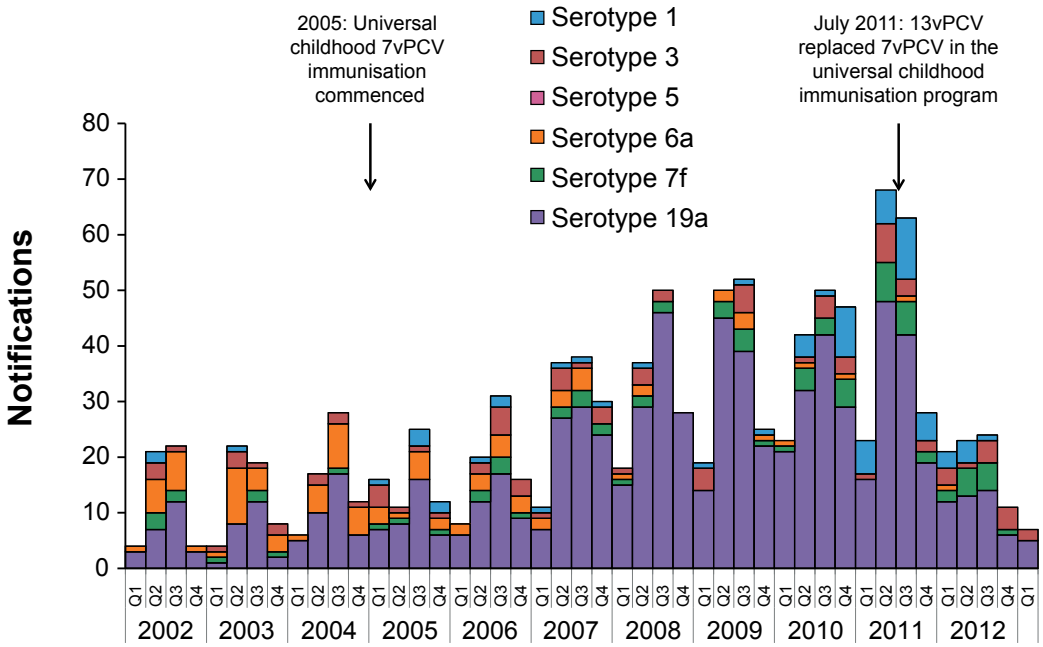


Figure 3: Notified cases of IPD in Indigenous Australians aged 50 years or older, Australia, 2002 to the first quarter of 2013, by serotype*

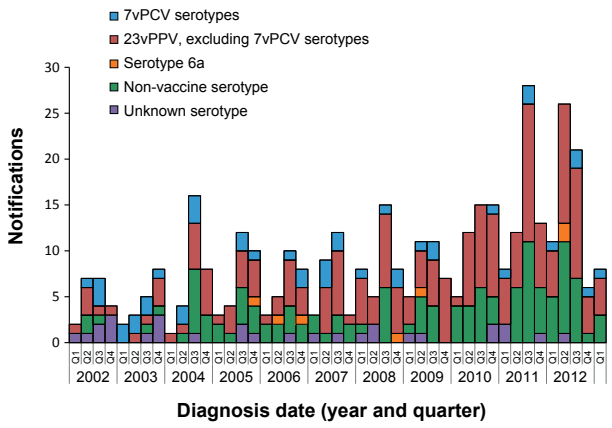
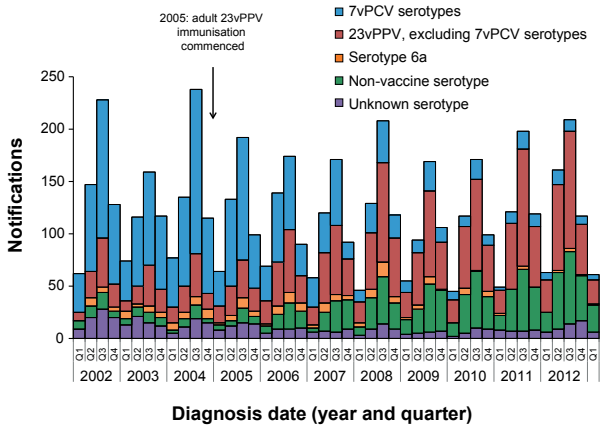


Figure 4: Notified cases of IPD in non-Indigenous Australians aged 65 years or older, Australia, 2002 to the first quarter of 2013, by serotype*



* Serotypes grouped according to targeted vaccines

Note: In 1999 23vPPV immunisation commenced for Indigenous Australians aged 50 years and over

fourth quarter of 2011, reflecting the introduction of the 13vPCV on the universal childhood immunisation program in mid-2011. In the first quarter of 2013, there were five cases aged less than 5 years with disease due to serotype 19A and two cases due to serotype 3. No cases in this age group were reported with disease caused by serotypes 1, 5, 6A or 7F.

* Serotypes grouped according to targeted vaccines

IPD in Indigenous Australians aged 50 years or older

In the first quarter of 2013, 4% (n=8) of notified cases were reported in Indigenous Australians aged 50 years or older. The number of cases notified in this group in the reporting period was similar to the number of cases notified in the previous quarter (n=6) (Figure 3: Notified cases of IPD in Indigenous Australians aged 50 years or older, Australia, 2002 to the first quarter of 2013,

by serotype*). There were fewer notifications in the reporting period compared with the same quarter in the previous year (n=11).

All cases reported in this quarter were reported with serotype information. More than half (n=5) of the cases were reported with disease due to serotypes targeted by the 23vPCV, with the remaining reported with disease due to a non-vaccine serotype.

IPD in non-Indigenous Australians aged 65 years or older

In the first quarter of 2013, 29% (n=61) of notified cases were reported as non-Indigenous Australians aged 65 years or older. The number of cases notified was about one quarter of the number of cases notified at the seasonal peak in the third quarter of 2012 (n=209) (Figure 4: Notified cases of IPD in non-Indigenous Australians aged 65 years or older, Australia, 2002 to the first quarter of 2013by serotype grouped according to targeted vaccines). Slightly fewer notifications were reported in the quarter compared with the same quarter in the previous year (n=63).

The majority (90%, 55/61) of cases reported in this quarter were reported with serotype information. Of these cases, more than one half (51%, 28/55) were reported with a serotype targeted by the 23vPPV. While the burden of disease in this age group has remained relatively stable, the profile of serotypes causing disease has changed over time. Disease due to serotypes targeted by the 7vPCV has reduced substantially in this age group likely due to herd immunity impacts from the childhood immunisation program.

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Meningococcal Surveillance Australia Reporting period 1 January to 31 March 2013

Monica M Lahra, Rodney Enriquez for the Australian Meningococcal Surveillance Programme

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of cases confirmed by laboratory testing using culture and by non-culture based techniques. Culture positive cases, where *Neisseria meningitidis* is grown from a normally sterile site or from a skin lesion, and non-culture based diagnoses, derived from results of nucleic acid amplification assays (NAA) and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in quarterly reports are restricted to a description of the numbers of cases by jurisdiction and serogroup, where known. Some minor corrections to data in the Table may be made in subsequent reports if additional data are received. A full analysis of laboratory confirmed cases of IMD in each calendar year is contained in the annual reports of the programme published in *Communicable Diseases Intelligence* (CDI). For more information see *Comm Dis Intell* 2013;37(1):E61

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, first quarter 2013, and 2012 by jurisdiction and serogroup

Jurisdiction	Yr	Serogroup													
		A		B		C		Y		W135		ND/other		All	
		Q1	*ytd*	Q1	*ytd*	Q1	*ytd*	Q1	*ytd*	Q1	*ytd*	Q1	*ytd*	Q1	*ytd*
Australian Capital Territory	2013	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2012	0	0	1	1	0	0	0	0	0	0	0	0	1	1
New South Wales	2013	0	0	7	7	3	3	0	0	0	0	0	0	10	10
	2012	0	0	7	7	0	0	0	0	0	0	2	2	9	9
Northern Territory	2013	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2012	0	0	0	0	0	0	0	0	0	0	1	1	1	1
Queensland	2013	0	0	7	7	0	0	1	1	1	1	0	0	9	9
	2012	0	0	10	10	1	1	0	0	0	0	0	0	11	11
South Australia	2013	0	0	4	4	0	0	0	0	1	1	0	0	5	5
	2012	0	0	0	0	1	1	0	0	0	0	0	0	1	1
Tasmania	2013	0	0	1	1	0	0	0	0	0	0	0	0	1	1
	2012	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Victoria	2013	0	0	6	6	1	1	0	0	0	0	0	0	7	7
	2012	0	0	7	7	0	0	0	0	0	0	0	0	7	7
Western Australia	2013	0	0	7	7	0	0	0	0	0	0	0	0	7	7
	2012	0	0	1	1	1	1	1	1	0	0	0	0	3	3
Australia	2013	0	0	32	32	4	4	1	1	2	2	0	0	39	39
	2012	0	0	26	26	3	3	1	1	0	0	3	3	33	33

National Notifiable Diseases Surveillance System tables

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 53,347 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 January and 31 March 2013 (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
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions

Table 1 : Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Varicella zoster (chickenpox)	All jurisdictions except New South Wales
Varicella zoster (shingles)	All jurisdictions except New South Wales
Varicella zoster (unspecified)	All jurisdictions except New South Wales
Vectorborne diseases	
Arbovirus infection (NEC)	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

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

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2013, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2013	Total 4th quarter 2012	Total 1st quarter 2012	Last 5 years mean 1st quarter	Ratio	Year to date 2013	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Hepatitis B (newly acquired)†	1	10	0	12	3	2	9	11	48	53	53	57.4	0.8	48	57.4
Hepatitis B (unspecified)‡	29	564	52	209	79	12	492	201	1,638	1,523	1,630	1,683.8	1.0	1,638	1,683.8
Hepatitis C (newly acquired)†	6	13	1	NN	22	5	34	30	111	106	123	101.8	1.1	111	101.8
Hepatitis C (unspecified)‡	33	808	57	612	93	48	512	288	2,451	2,368	2,541	2,656.0	0.9	2,451	2,656.0
Hepatitis D	0	1	1	4	2	0	4	0	12	7	8	9.2	1.3	12	9.2
Gastrointestinal diseases															
Botulism	0	1	0	0	0	0	1	0	2	0	0	0.2	10.0	2	0.2
Campylobacteriosis	105	NN	47	918	386	199	1,399	417	3,471	3,814	4,814	4,540.0	0.8	3,471	4,540.0
Cryptosporidiosis	19	590	26	286	37	42	575	143	1,718	432	1,453	1,246.2	1.4	1,718	1,246.2
Haemolytic uraemic syndrome	0	3	0	1	0	0	0	0	4	4	7	5.2	0.8	4	5.2
Hepatitis A	1	30	0	7	6	0	18	5	67	44	44	68.8	1.0	67	68.8
Hepatitis E	0	7	0	0	0	0	4	3	14	6	15	14.4	1.0	14	14.4
Listeriosis	1	11	1	6	0	1	6	3	29	27	27	27.4	1.1	29	27.4
STEC, VTEC§	1	10	0	7	22	0	4	0	44	32	36	32.4	1.4	44	32.4
Salmonellosis	72	1,131	119	1,092	252	113	1,009	362	4,150	2,761	3,961	3,802.2	1.1	4,150	3,802.2
Shigellosis	4	39	28	23	6	1	24	18	143	129	191	193.4	0.7	143	193.4
Typhoid Fever	1	23	0	12	5	0	24	3	68	31	56	43.2	1.6	68	43.2
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	0	0.8	0.0	0	0.8
Highly Pathogenic Avian Influenza in Humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe Acute Respiratory Syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0

Table 2: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2013, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2013	Total 4th quarter 2012	Total 1st quarter 2012	Last 5 years mean 1st quarter	Ratio	Year to date 2013	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection[‖¶]	324	5,345	647	4,807	1,370	411	4,992	2,981	20,877	19,277	22,536	18,491.8	1.1	20,877	18,491.8
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Gonococcal infection[‖]	39	1,101	435	702	117	12	788	528	3,722	3,381	3,610	2,648.0	1.4	3,722	2,648.0
Syphilis – congenital	0	0	0	1	0	0	0	0	1	0	0	1.2	0.8	1	1.2
Syphilis < 2 years[‖]	4	163	4	76	8	6	99	26	386	344	350	350.0	1.1	386	350.0
Syphilis > 2 years or unspecified duration[‡‖]	2	77	15	83	31	5	183	35	431	296	332	333.8	1.3	431	333.8
Vaccine preventable diseases															
Diphtheria	0	0	0	0	1	0	0	0	1	0	0	0.0	0.0	1	0.0
Haemophilus influenzae type b	0	2	0	1	0	0	0	0	3	3	3	4.4	0.7	3	4.4
Influenza (laboratory confirmed)	44	397	132	795	318	8	326	289	2,309	2,902	1,274	1,062.4	2.2	2,309	1,062.4
Measles	0	1	0	1	2	0	3	3	10	29	10	42.8	0.2	10	42.8
Mumps	0	23	0	8	1	2	9	28	71	40	40	59.2	1.2	71	59.2
Pertussis	48	635	38	1,217	156	323	776	399	3,592	5,800	7,240	6,726.2	0.5	3,592	6,726.2
Pneumococcal disease (invasive)	2	65	15	38	16	6	51	21	214	345	235	216.4	1.0	214	216.4
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	1	0	0	0	0	0	0	1	5	13	12.8	0.1	1	12.8
Rubella Congenital	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Tetanus	0	1	0	0	1	0	0	1	3	4	1	1.8	1.7	3	1.8
Varicella zoster (Chickenpox)	7	NN	39	48	90	2	134	49	369	490	402	351.2	1.1	369	351.2
Varicella zoster (Shingles)	5	NN	44	13	463	70	288	296	1,179	1,131	1,085	836.0	1.4	1,179	836.0
Varicella zoster (Unspecified)	31	NN	1	1,187	36	20	668	313	2,256	2,269	2,078	1,660.0	1.4	2,256	1,660.0
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	1	4	0	0	0	0	5	1	3	3.8	1.3	5	3.8
Barmah Forest virus infection	3	133	130	661	33	0	25	443	1,428	642	454	624.8	2.3	1,428	624.8
Dengue virus infection	3	59	18	165	17	3	96	122	483	218	696	477.4	1.0	483	477.4
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	1	0.2	0.0	0	0.2
Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	0	0.6	0.0	0	0.6

Table 2: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2013, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2013	Total 4th quarter 2012	Total 1st quarter 2012	Last 5 years mean 1st quarter	Ratio	Year to date 2013	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Malaria	8	24	7	44	2	2	21	29	137	95	76	108.0	1.3	137	108.0
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	1	2.4	0.0	0	2.4
Ross River virus infection	1	115	70	495	51	4	57	546	1,339	688	2,298	2,272.8	0.6	1,339	2,272.8
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Australian bat lyssavirus	0	0	0	1	0	0	0	0	1	0	0	0.0	0.0	1	0.0
Brucellosis	0	0	0	5	0	0	0	0	5	6	7	7.8	0.6	5	7.8
Leptospirosis	0	2	0	12	0	0	0	0	14	10	48	60.6	0.2	14	60.6
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	1	0	0	0	0	3	3	7	31	12	17.0	0.4	7	17.0
Q fever	0	28	0	47	7	0	9	2	93	81	108	95.2	1.0	93	95.2
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.5	0.0	0	0.5
Other bacterial infections															
legionellosis	0	20	0	20	14	2	23	16	95	98	95	73.6	1.3	95	73.6
Leprosy	0	1	00	0	0	0	1	0	2	2	0	1.4	1.4	2	1.4
Meningoxoxxal disease (invasive)**	1	8	0	10	6	1	7	6	39	39	34	44.8	0.9	39	44.8
Tuberculosis	5	86	13	53	19	2	93	33	304	382	322	302.0	1.0	304	302.0
Total	**800**	**11,529**	**1,941**	**13,683**	**3,672**	**1,302**	**12,767**	**7,653**	**53,347**	**49,946**	**58,323**			**53,347**	

* 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.

** 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 January to 31 March 2013, by state or territory. (Annualised rate per 100,000 population)[*†]

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)[‡]	1.1	0.6	0.0	1.1	0.7	1.6	0.7	1.9	0.9
Hepatitis B (unspecified)[§]	32.5	31.7	92.5	18.7	19.6	9.6	35.9	35.1	29.7
Hepatitis C (newly acquired)[‡]	6.7	0.7	1.8	NN	5.4	4.0	2.5	5.2	2.5
Hepatitis C (unspecified) [§]	37.0	45.4	101.4	54.8	23.0	38.5	37.3	50.3	44.4
Hepatitis D	0.0	0.1	1.8	0.4	0.5	0.0	0.3	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Campylobacteriosis	117.7	NN	83.6	82.2	95.6	159.8	102.0	72.8	92.9
Cryptosporidiosis	21.3	33.1	46.3	25.6	9.2	33.7	41.9	25.0	31.1
Haemolytic uraemic syndrome	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Hepatitis A	1.1	1.7	0.0	0.6	1.5	0.0	1.3	0.9	1.2
Hepatitis E	0.0	0.4	0.0	0.0	0.0	0.0	0.3	0.5	0.3
Listeriosis	1.1	0.6	1.8	0.5	0.0	0.8	0.4	0.5	0.5
STEC, VTEC[‖]	1.1	0.6	0.0	0.6	5.4	0.0	0.3	0.0	0.8
Salmonellosis	80.7	63.5	211.8	97.7	62.4	90.8	73.6	63.2	75.2
Shigellosis	4.5	2.2	49.8	2.1	1.5	0.8	1.8	3.1	2.6
Typhoid Fever	1.1	1.3	0.0	1.1	1.2	0.0	1.8	0.5	1.2
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
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	0.0
Sexually transmissible infections									
Chlamydial infection[¶**]	363.3	300.1	1,151.5	430.3	339.1	330.1	364.1	520.2	378.4
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection[¶]	43.7	61.8	774.2	62.8	29.0	9.6	57.5	92.1	67.5
Syphilis - congenital	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years[¶]	4.5	9.2	7.1	6.8	2.0	4.8	7.2	4.5	7.0
Syphilis > 2 years or unspecified duration[¶]	2.2	4.3	26.7	7.4	7.7	4.0	13.3	6.1	7.8
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Influenza (laboratory confirmed)	49.3	22.3	234.9	71.2	78.7	6.4	23.8	50.4	41.9
Measles	0.0	0.1	0.0	0.1	0.5	0.0	0.2	0.5	0.2
Mumps	0.0	1.3	0.0	0.7	0.2	1.6	0.7	4.9	1.3
Pertussis	53.8	35.7	67.6	108.9	38.6	259.4	56.6	69.6	65.1
Pneumococcal disease (invasive)	2.2	3.6	26.7	3.4	4.0	4.8	3.7	3.7	3.9
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella Congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.1	0.0	0.0	0.2	0.0	0.0	0.2	0.1

Table 3: Notification rates of diseases, 1 January to 31 March 2013, by state or territory. (Annualised rate per 100,000 population)*†

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases, *cont'd*									
Varicella zoster (Chickenpox)	7.8	NN	69.4	4.3	22.3	1.6	9.8	8.6	9.9
Varicella zoster (Shingles)	5.6	NN	78.3	1.2	114.6	56.2	21.0	51.7	31.6
Varicella zoster (Unspecified)	34.8	NN	1.8	106.3	8.9	16.1	48.7	54.6	60.4
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	1.8	0.4	0.0	0.0	0.0	0.0	0.1
Barmah Forest virus infection	3.4	7.5	231.4	59.2	8.2	0.0	1.8	77.3	25.9
Dengue virus infection	3.4	3.3	32.0	14.8	4.2	2.4	7.0	21.3	8.8
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	9.0	1.3	12.5	3.9	0.5	1.6	1.5	5.1	2.5
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	1.1	6.5	124.6	44.3	12.6	3.2	4.2	95.3	24.3
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	0.1	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.1	0.0	1.1	0.0	0.0	0.0	0.0	0.3
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	0.0	0.0	0.0	0.2	0.5	0.1
Q fever	0.0	1.6	0.0	4.2	1.7	0.0	0.7	0.3	1.7
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	0.0	1.1	0.0	1.8	3.5	1.6	1.7	2.8	1.7
Leprosy	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Meningococcal disease (invasive)††	1.1	0.4	0.0	0.9	1.5	0.8	0.5	1.0	0.7
Tuberculosis	5.6	4.8	23.1	4.7	4.7	1.6	6.8	5.8	5.5

* 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

Policy items

National Tuberculosis Advisory Committee 2012 Committee Report

Emily Gearside and the National Tuberculosis Advisory Committee

The National Tuberculosis Advisory Committee (NTAC) was established in 1999 as a subcommittee of Communicable Diseases Network Australia (CDNA). The terms of reference of NTAC are:

- to provide strategic, expert advice to CDNA on a coordinated national and international approach to Tuberculosis (TB) control; and
- to develop and review nationally agreed strategic and implementation plans for the control of TB in Australia.

NTAC membership is comprised of the following:

- representation from those responsible for the TB programs in their respective jurisdictions, namely nurse managers with TB expertise, public health physicians, clinicians practising in TB clinics, thoracic physicians, and infectious disease physicians
- a representative from the Commonwealth Department of Health (DoH)
- a representative from the Public Health Laboratory Network (PHLN)
- an human immunodeficiency virus-tuberculosis (HIV-TB) specialist nominated by the Australasian Society for Infectious Disease (ASID)
- a paediatric specialist nominated by the Royal Australasian College of Physicians, Paediatrics & Child Health Division; and
- a representative from the Department of Immigration and Citizenship (DIAC).

The Tuberculosis Data Quality Working Group (TBDQWG) is a working group of NTAC, with representation from jurisdictions, DoHA and the Mycobacterium Reference Laboratory Network. The TBDQWG ensures that high quality data informs routine and timely reports on trends and emerging issues in TB.

Secretariat support for NTAC and TBDQWG is provided by DoHA. This report documents activities undertaken by NTAC in 2012. NTAC formally met on four occasions: face-to-face in April and October and via teleconference in February and August. Dr Justin Waring, of Western Australia, was appointed Chair of NTAC in March 2011 and has been endorsed to continue as Chair until March 2015. Ms Amanda Christensen (of New South Wales) was the Deputy Chair of NTAC during 2012. NTAC developed and finalised a comprehensive work plan which sets out the work that the committee aims to complete by the end of 2015. This has helped the committee to focus on its core work and to structure its meeting agendas accordingly.

NTAC expanded its membership in 2012 to include a representative from DIAC. The high burden of TB in the overseas-born population of Australia demonstrates that TB control in Australia is largely dependent on our pre- and post- migration activities in relation to new arrivals.

Guidelines and Publications

NTAC publishes information to assist the TB community. In 2012 NTAC published a number of documents and progressed work towards finalising others.

NTAC published the *Position statement on interferon-γ release assays in the detection of latent tuberculosis infection* (IGRA position statement). This is a general guide to the appropriate practice to be followed in the detection of latent tuberculosis infection (LTBI).[1] The statement recommends that the tuberculin skin test (TST) remains the preferred test for LTBI in most patient groups. NTAC recommends that IGRAs may be used as supplemental tests to improve specificity in screening immunocompetent subjects and in addition to TST in immunocompromised patients considered at high risk of LTBI. The specific recommendations in various patient groups are listed in the document.

The committee also published an annual report, *Tuberculosis notifications in Australia, 2008 and 2009* summarising the incidence of tuberculosis in Australia as reported to the National Notifiable Diseases Surveillance System during 2008 and 2009.[2]

NTAC published *The Strategic Plan for Control of Tuberculosis in Australia: 2011–2015* (the Strategic Plan).[3] This document articulates challenges, priorities and actions for the control of TB in Australia in the coming years. The Strategic Plan acknowledges that the burden of TB in Australia will depend on future immigration policy, the control of TB in new arrivals

and the detection of TB as migrants age. NTAC notes the particular challenge of TB within neighbouring countries posing direct public health threats to Australia as seen in the Treaty Zone between the outer Torres Strait Islands of Queensland and the various villages of the South Fly District of the Western Province of Papua New Guinea. NTAC has identified Australia's priority populations – groups at higher risk of TB than most of the population. These are persons in close contact with active disease, Indigenous Australians, overseas-born persons (including secondary and tertiary students and health care workers) and other groups (elderly and immuno-suppressed persons and those with TB and HIV co-infection). In maintaining TB control in Australia, NTAC calls for a continued high standard of diagnosis and treatment, requiring continuation of current TB control infrastructure, support for global TB control activities and improving existing and developing new diagnostics, treatments and vaccinations.

In 2012 NTAC finalised *The Bacille Calmette Guérin vaccine (BCG): information and recommendations for use in Australia* (the BCG document).[4] The BCG document is an update to the previously published 2006 document and provides an update on the role of BCG vaccination in tuberculosis control and prevention in Australia. No significant changes were made to the previous recommendations.

NTAC members assisted in the preparation of the CDNA National Guidelines for the Public Health Management of Tuberculosis, part of the Series of National Guidelines (SoNGs). The purpose of this document is to provide nationally consistent guidance to public health units in responding to a notifiable TB event, and is expected to be published in 2013.

NTAC members contributed to the development of the Tuberculosis Screening and Management in Immigration Detention section of the *DIAC Detention Services Manual*.[5] The purpose of this section is to provide clear guidelines in screening and investigation for TB at the health induction assessment for irregular arrivals as well as an overview of the principles of the current management of TB in Immigration Detention Facilities.

In Australia TB control is managed through state and territory-based programs, rather than a national program as for most other countries. The jurisdictional boundaries of these programs are well defined, and communication and cooperation between the programs is effective. In 2012, NTAC began work on a paper which defines the essential requirements for TB programs in Australia and will aim to finalise this paper in 2013. In addition members are drafting a proposal to formalise a National TB Program in Australia. This proposal does not intend to change the current operational structure or funding of the existing jurisdictional TB programs or of NTAC, but would recognise the current partnerships between TB services in jurisdictions and the Commonwealth.

This article has provided a brief overview of the work undertaken by NTAC during 2012. Members of NTAC also participate in a variety of other TB related activities at the local, national and international level. Through NTAC they are able to effectively provide a national perspective on issues discussed and to disseminate information from these other sources. NTAC looks forward to continuing and expanding its activities in the future.

For further information on NTAC including NTAC publications contact:

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Author details

Report compiled by Emily Gearside on behalf of NTAC. For a list of current NTAC members, please visit the website at: http://www.health.gov.au/ntac

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2. Christina Barry, Justin Waring, Richard Stapledon, Anastasios Konstantinos 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.
3. National Tuberculosis Advisory Committee. The Strategic Plan for Control of Tuberculosis in Australia: 2011–2015 *Commun Dis Intell* 2012; 36(3):E286-E293.
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Administration

REVISED SURVEILLANCE CASE DEFINITIONS

This report provides the revised Surveillance case definitions approved by the Communicable Diseases Network Australia (CDNA) since 1 January 2013.

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

The following case definitions have been reviewed by CDWG and endorsed by CDNA.

These case definitions were implemented on 1 July 2013 and supersede any previous versions.

Chlamydia

(Excluding eye infections)

(Effective 1 July 2013)

Reporting

Only confirmed cases should be notified.

Confirmed case

A confirmed case requires laboratory definitive evidence.

Laboratory definitive evidence

Isolation of *Chlamydia trachomatis*

OR

Detection of *C. trachomatis* by nucleic acid testing

OR

Detection of *C. trachomatis* antigen.

END

Chlamydia changes

Added to title (Excluding eye infections)

Diphtheria

(Effective 1 July 2013)

Reporting

Both confirmed cases and probable cases should be notified.

Confirmed case

A confirmed case requires laboratory definitive evidence AND clinical evidence.

Laboratory definitive evidence

Isolation of toxigenic *Corynebacterium diphtheriae* or toxigenic *Corynebacterium ulcerans*.

Probable case

A probable case requires:

Laboratory suggestive evidence AND clinical evidence

OR

Clinical evidence AND epidemiological evidence.

Laboratory suggestive evidence

Isolation of *Corynebacterium diphtheriae* or *C. ulcerans* (toxin production unknown).

Clinical evidence

At least one of the following:

Pharyngitis and/or laryngitis (with or without a membrane)

OR

Toxic (cardiac or neurological) symptoms.

Epidemiological evidence

An epidemiological link is established when there is:

Contact between two people involving a plausible mode of transmission at a time when:

a) one of them is likely to be infectious (usually up to 2 weeks and seldom more than 4 weeks after onset of symptoms)

AND

b) the other has an illness which starts within approximately 2-5 days after this contact

AND

At least one case in the chain of epidemiologically linked cases (which may involve many cases) is laboratory confirmed.

END

Diphtheria changes

Confirmed case

At the end of confirmed case, added “AND clinical evidence”

Hepatitis E

(Effective 1 July 2013)

Reporting

Only confirmed cases should be notified.

Confirmed case

A confirmed case requires:

laboratory definitive evidence

OR

Laboratory suggestive evidence AND clinical evidence AND epidemiological evidence.

Laboratory definitive evidence

Detection of hepatitis E virus by nucleic acid testing

OR

Detection of hepatitis E virus in faeces by electron microscopy

OR

IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to hepatitis E virus.

Laboratory suggestive evidence

Detection of IgM or IgG to hepatitis E virus.

Clinical evidence

A clinically compatible illness without other apparent cause.

Epidemiological evidence

Travel to a country with known hepatitis E activity between 15 – 64 days prior to onset OR epidemiological link to a confirmed case

END

Hepatitis E changes

Confirmed case

Added “OR Laboratory suggestive evidence AND clinical evidence AND epidemiological evidence”

Laboratory definitive evidence

Replaced “Detection of IgM or IgG to hepatitis E virus. If the person has not travelled outside Australia in the preceding 3 months, the antibody result must be confirmed by specific immunoblot” with “IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to hepatitis E virus”

Added Laboratory suggestive evidence, Clinical evidence and Epidemiological evidence;

Laboratory suggestive evidence

Added “Detection of IgM or IgG to hepatitis E virus”

Clinical evidence

Added “A clinically compatible illness without other apparent cause”

Epidemiological evidence

Added “Travel to a country with known hepatitis E activity between 15 – 64 days prior to onset OR epidemiological link to a confirmed case”

Pertussis

(Effective 1 July 2013)

Reporting

Both confirmed cases and probable cases should be notified.

Confirmed case

A confirmed case requires either:

Laboratory definitive evidence

OR

Laboratory suggestive evidence AND clinical evidence

Probable case

A probable case requires clinical evidence AND epidemiological evidence

Laboratory definitive evidence

Isolation of *Bordetella pertussis*

OR

Detection of *B. pertussis* by nucleic acid testing

OR

Seroconversion in paired sera for *B.pertussis* using whole cell or specific *B.pertussis* antigen(s) in the absence of recent pertussis vaccination[1]

Laboratory suggestive evidence

In the absence of recent vaccination[1]

Significant change (increase or decrease) in antibody level (IgG, IgA) to *B. pertussis* whole cell or *B. pertussis* specific antigen(s)

OR

Single high IgG and/or IgA titre to Pertussis Toxin (PT)

OR

1 In the absence of recent vaccination

Single high IgA titre to Whole Cell *B.pertussis* antigen.

Clinical evidence

A coughing illness lasting two or more weeks

OR

Paroxysms of coughing OR inspiratory whoop OR post-tussive vomiting.

Epidemiological evidence

An epidemiological link is established when there is:

Contact between two people involving a plausible mode of transmission at a time when:

a) one of them is likely to be infectious (from the catarrhal stage, approximately one week before, to three weeks after onset of cough)

AND

b) the other has an illness which starts within 6 to 20 days after this contact

AND

At least one case in the chain of epidemiologically linked cases (which may involve many cases) is a confirmed case with either laboratory definitive or laboratory suggestive evidence.

Pertussis changes

Confirmed case

Removed “Clinical evidence AND epidemiological evidence”

Probable case

Added “AND epidemiological evidence”

Laboratory definitive evidence

Added “OR Seroconversion in paired sera for *B. pertussis* using whole cell or specific *B. pertussis* antigen(s) in the absence of recent pertussis vaccination.

Added footnote “In the absence of recent vaccination”.

Laboratory suggestive evidence

Changed to “In the absence of recent vaccination Significant change (increase or decrease) in antibody level (IgG, IgA) to *B. pertussis* whole cell or *B. pertussis* specific antigen(s) OR Single high IgG and or IgA titre to Pertussis Toxin (PT) Single high IgA titre to Whole Cell *B.pertussis* antigen.

Added footnote “In the absence of recent vaccination”.

To “A probable case requires clinical evidence only” added “ AND epidemiological evidence”

Removed Clinical evidence for probable cases.

Moved Clinical evidence and Epidemiological evidence to Probable Case.

Errata

Annual report of National Arbovirus and Malaria Advisory Committee 2010-11

Communicable Diseases Intelligence 2013, 37 (1) E1-E20

Change in authorship

The corrected authorship is as follows:

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

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Annual immunisation coverage report, 2010

Communicable Diseases Intelligence 2013, 37 (1) E21-E39

Tables 2, 3 and 4

The corrected tables appear below. In the previous version, the data was under incorrect state and territory headings.

Annual report of the National Notifiable Diseases Surveillance System, 2010

Communicable Diseases Intelligence 2012, 36 (1) E1-E69

Table 6

The corrected part of the table appears below. In the previous version, the numbers in the table for zoonoses were listed against incorrect diseases within the zoonotic disease group.

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

Vaccine	Jurisdiction								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
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/territory*

Vaccine	Jurisdiction								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
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 years of age, by vaccine and state/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

Table 6 *cont'd*: Notifications and notification rate for communicable diseases, Australia, 2005 to 2010, (per 100,000 population)

Disease	Number of notifications						5 year mean	Ratio	Notification rate per 100,000 population					
	2005	2006	2007	2008	2009	2010			2005	2006	2007	2008	2009	2010
Zoonoses														
Anthrax	0	1	1	0	0	1	0.4	2.5	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	0.0	0.0
Brucellosis	41	50	37	45	32	21	41.0	0.5	0.2	0.2	0.2	0.2	0.2	0.1
Leptospirosis	129	145	108	112	146	131	128.0	1.0	0.6	0.7	0.5	0.5	0.7	0.6
Lyssavirus (NEC)	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	164	165	93	102	65	56	117.8	0.5	0.8	0.8	0.4	0.5	0.3	0.3
Q fever	352	411	449	376	310	323	379.6	0.9	1.7	2.0	2.1	1.7	1.4	1.4
Tularaemia	0	0	0	0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0