#### Department of Health

# Communicable Diseases Intelligence

Volume 38 Number 3

Quarterly report

September 2014

#### Epidemiology of pertussis in Australia

#### **Editorial**

## E177 Pertussis control in Australia – the current state of play

Dr Helen Quinn, Senior Research Fellow, National Centre for Immunisation and Research of Vaccine Preventable Diseases

#### **Original articles**

# E179 Australian vaccine preventable disease epidemiological review series: pertussis, 2006–2012

Alexis Pillsbury, Helen E Quinn, Peter B McIntyre

E195 Finding the 'who' in whooping cough: vaccinated siblings are important pertussis sources in infants 6 months of age and under

Christina Bertilone, Tania Wallace, Linda A Selvey

E201 A state-wide information campaign during a pertussis epidemic in New South Wales, 2010

> Paula J Spokes, Alexander E Rosewell, Alex S Stephens, Jeremy M McAnulty

#### **Annual reports**

#### E208 Immunisation coverage, 2012

Brynley P Hull, Aditi Dey, Rob I Menzies, Julia M Brotherton, Peter B McIntyre

### E232 Surveillance of adverse events following immunisation in Australia, 2012

Deepika Mahajan, Aditi Dey, Jane Cook, Bronwen Harvey, Rob I Menzies, Kristine K Macartney

# E247 Australian Enterococcal Sepsis Outcome Progamme, 2011

Geoffrey W Coombs, Julie C Pearson, Denise A Daley, Tam Le, James O Robinson, Thomas Gottlieb, Benjamin P Howden, Paul DR Johnson, Catherine M Bennett, Timothy P Stinear, John D Turnidge for the Australian Group on Antimicrobial Resistance

#### **Quarterly reports**

#### E253 National Notifiable Diseases Surveillance System, 1 April to 30 June 2014

#### E260 Australian childhood immunisation coverage, 1 October to 31 December cohort, assessed as at 31 March 2013

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

#### E262 Australian Meningococcal Surveillance Programme quarterly report, 1 April to 30 June 2014

Monica M Lahra for the Australian Meningococcal Surveillance Programme

#### E263 Australian Sentinel Practices Research Network, 1 April to 30 June 2013

Monique Chilver, Daniel Blakeley for the Australian Sentinel Practices Research Network

# E266 Invasive pneumococcal disease surveillance Australia, 1 April to 30 June 2014

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

#### Policy and guidelines

E271 Recommended composition of the Australian influenza vaccine for the 2015 season ISSN 1445-4866 Online

This work is copyright. You may download, display, print and reproduce the whole or part of this work in unaltered form for your own personal use or, if you are part of an organisation, for internal use within your organisation, but only if you or your organisation do not use the reproduction for any commercial purpose and retain this copyright notice and all disclaimer notices as part of that reproduction. Apart from rights to use as permitted by the Copyright Act 1968 or allowed by this copyright notice, all other rights are reserved and you are not allowed to reproduce the whole or any part of this work in any way (electronic or otherwise) without first being given the specific written permission from the Commonwealth to do so. Requests and inquiries concerning reproduction and rights are to be sent to the Online, Services and External Relations Branch, Department of Health, GPO Box 9848, Canberra ACT 2601, or by email to copyright@health.gov.au

Communicable Diseases Intelligence aims to diseminate information on the epidemiology and control of communicable diseases in Australia. Communicable Diseases Intelligence invites contributions dealing with any aspect of communicable disease epidemiology, surveillance or prevention and control in Australia. Submissions can be in the form of original articles, short reports, surveillance summaries, reviews or correspondence. Instructions for authors can be found in Commun Dis Intell 2014;38(1):E99–E104.

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

#### **Editor**

Margaret Curran

#### **Deputy Editor**

Katrina Knope

#### **Associate Editor**

Timothy Sloan-Gardner

#### **Editorial and Production Staff**

Alison Milton, Leroy Trapani

#### **Editorial Advisory Board**

Peter McIntyre (Chair), David Durrheim, Cheryl Jones, John Kaldor, Martyn Kirk, Brett Sutton

#### Website

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

#### Contacts

Communicable Diseases Intelligence is produced every quarter by: Health Emergency Management Branch Office of Health Protection
Australian Government Department of Health GPO Box 9848, (MDP 6)
CANBERRA ACT 2601;

Telephone: +61 2 6289 2717 Facsimile: +61 2 6289 2700 Email: cdi.editor@health.gov.au

This journal is indexed by Index Medicus and Medline

#### **Disclaimer**

Opinions expressed in *Communicable Diseases Intelligence* are those of the authors and not necessarily those of the Australian Government Department of Health or the Communicable Diseases Network Australia. Data may be subject to revision.

### Editorial

# PERTUSSIS CONTROL IN AUSTRALIA — THE CURRENT STATE OF PLAY

Dr Helen E Quinn, Senior Research Fellow, National Centre for Immunisation and Research of Vaccine Preventable Diseases

Pertussis is highly contagious. Neither natural infection nor vaccination provides lifelong immunity. As a consequence, epidemic peaks of pertussis occur every 3–4 years against a background of endemic transmission. There is little doubt that the use of pertussis vaccines has significantly impacted the global burden of this disease and prevented millions of deaths. However, pertussis continues to be a public health concern with growing evidence of a pertussis resurgence in a number of developed countries and deaths still occurring in vulnerable young infants, demonstrating the inadequacies of current pertussis control.<sup>1</sup>

Australia has long held the position of having one of the highest reported rates of pertussis in the world due to comprehensive laboratory surveillance for pertussis. Australia has been an early adopter of processes such as mandatory reporting by laboratories of positive test results to notification systems, serological testing for pertussis diagnosis in adults and widespread community use of polymerase chain reaction (PCR) testing for pertussis diagnosis. This issue of *Communicable Diseases Intelligence* (CDI) contains several articles on various aspects of pertussis in Australia.

The article by Pillsbury et al. reports changes in diagnostic testing of notified cases over time and among different age groups. As the authors conclude, there is little doubt that all Australian states and territories have recently experienced another pertussis epidemic. However the magnitude of notified cases has clearly been augmented by increased PCR testing. The ongoing use of this highly sensitive, rapid, relatively non-invasive and affordable diagnostic tool means that a new baseline for notified endemic pertussis rates in Australia has been established and notified cases numbers will never return to levels seen prior to 2008.

Pillsbury et al. also demonstrate the shift in notified disease, from older adolescents and adults to children, in the most recent epidemic. Whilst the largest burden and most severe disease has consistently occurred in the youngest infants, children aged 6 months to 9 years had elevated notification rates in the recent epidemic, which had not been seen since the introduction of acellular pertussis

vaccines. Vaccination coverage is not an issue; for the last decade coverage for the primary series of diphtheria-tetanus-pertussis acellular (DTPa)containing vaccine and the pre-school booster have remained steady and high, at 92% for 3 doses at 12 months of age, increasing to 95% at 24 months, and 90% for 4 doses at 5 years of age. Instead, waning of vaccine induced immunity appears to be a factor. The increase in cases aged 6 months to 4 years may have resulted from the removal of the 18 month dose from the National Immunisation Program in 2003, thereby increasing the interval between the last dose of the primary series and the first booster dose. An Australian study showed that the vaccine effectiveness (VE) of 3 doses of DTPa against notified pertussis was 84% in infants aged 6–11 months, declining progressively from 2 years of age (71%) to less than 50% by 4 years of age.2 Likewise for children aged 5 years or over, studies from both Queensland and the United States of America have demonstrated progressive waning of immunity with increasing age and time since last dose.<sup>3,4</sup> It is a clear that a third generation of pertussis vaccines, providing long lasting protection, are required. Options being considered include less reactogenic whole cell vaccines, acellular vaccines with new adjuvants or live attenuated vaccines; however these are all some years away.

Whilst the majority of these vaccinated children experience mild pertussis and do not require hospitalisation, they do pose a risk as a source of infection for vulnerable young infants, who suffer the greatest morbidity and mortality from pertussis. Although the most common source for infant pertussis is usually the mother, in settings where disease activity is high among young children, siblings can pose a significant risk.<sup>5</sup> The article in this issue of CDI by Bertilone et al. has again confirmed this in the Australian setting, with siblings identified as the source for 35% of cases in a 4 year period in Perth, Western Australia. Among these sibling sources, the majority were aged 2–3 years and fully vaccinated for pertussis according to the National Immunisation Program.

There are a number of immunisation strategies aimed at preventing morbidity and mortality in young infants. Neonatal vaccination has been trialled; however study results are mixed as to whether a birth dose negatively impacts on the efficacy of future immunisations. The cocooning strategy (vaccinating close contacts of infants to reduce the likelihood of exposure) has been recommended in Australia since 2003. Although never funded at a national level, some states and territories introduced funded cocoon programs in response to the recent epidemic, which varied by target group, length and delivery method. A study of the New South Wales cocoon program showed that when both parents were immunised at least 4 weeks prior to onset of disease in cases, the risk of pertussis before 4 months of age was reduced by 51%.6 However, the effectiveness of a cocoon strategy is limited by timely vaccine uptake among adult household contacts. The article by Spokes et al. in this issue describes a survey to assess the effectiveness of an information campaign conducted in New South Wales at the time of the epidemic, promoting adult vaccination and pertussis awareness. Receipt of a NSW Health letter about the pertussis epidemic and vaccination was significantly associated with the uptake of an adult pertussis booster by respondents and other adults in the household. As in previous studies, advice from a general practitioner was one of the key reasons for receiving an adult pertussis booster in those who had been vaccinated. Unfortunately, cocooning will only provide indirect protection to the infant, which may not be enough in settings with waning acellular pertussis vaccine immunity among children, who may also be the sibling of a young infant. Maternal vaccination provides another alternative strategy, with the passive transfer of antibodies from the mother giving the infant direct protection against pertussis. Maternal vaccination is one of the recommended strategies in the 10th edition of *The Australian Immunisation* Handbook. It is being used at a population level in countries such as the United States of America, England and New Zealand, and a funded program has recently commenced in Queensland. Recent

evidence from England and the United Kingdom suggests that maternal vaccination against pertussis is both highly effective and safe.<sup>7,8</sup>

The control of pertussis in Australia will remain a challenge due to the continual fluctuations in pertussis immunity at a population level. As we await the development of better pertussis vaccines, ongoing monitoring of disease morbidity and vaccine effectiveness is necessary. This will inform considerations around the most appropriate policy options for pertussis control as the evidence arises.

#### References

- World Health Organization. Meeting of the Strategic Advisory Group of Experts on immunization, April 2014—conclusions and recommendations. Wkly Epidemiol Rec 2014;89(21):221–236.
- Quinn HE, Snelling TL, Macartney KK, McIntyre PB. Duration of protection after first dose of acellular pertussis vaccine in infants. *Pediatrics* 2014;133(3):e513–e19.
- Misegades LK, Winter K, Harriman K, Talarico J, Messonnier NE, Clark TA, et al. Association of childhood pertussis with receipt of 5 doses of pertussis vaccine by time since last vaccine dose, California, 2010. JAMA 2012;308(20):2126–2132.
- Sheridan SL, McCall BJ, Davis CA, Robson JM, Hull BP, Selvey CE, et al. Acellular pertussis vaccine effectiveness for children during the 2009–2010 pertussis epidemic in Queensland. Med J Aust 2014;200(6):334–338.
- Wiley KE, Zuo Y, Macartney KK, McIntyre PB. Sources of pertussis infection in young infants: a review of key evidence informing targeting of the cocoon strategy. Vaccine 2013;31(4):618–625.
- Quinn HE, Snelling TL, Habig A, Chiu C, Spokes PJ, McIntyre PB. Parental Tdap boosters and infant pertussis: A Case-Control Study. Pediatrics 2014;134(4):713–720.
- Amirthalingam G, Andrews N, Campbell H, Ribeiro S, Kara E, Donegan K, et al. Effectiveness of maternal pertussis vaccination in England: an observational study. Lancet 2014; pii: S0140-6736(14)60686-3.
- Donegan K, King B, Bryan P. Safety of pertussis vaccination in pregnant women in UK: observational study. BMJ 2014;349:g4219.

E178

# Original article

# AUSTRALIAN VACCINE PREVENTABLE DISEASE EPIDEMIOLOGICAL REVIEW SERIES: PERTUSSIS, 2006–2012

Alexis Pillsbury, Helen E Quinn, Peter B McIntyre

#### **Abstract**

Despite pertussis vaccine being available since the 1940s and immunisation programs using combined diphtheria-tetanus-pertussis vaccine since the mid-1950s, pertussis has been the most commonly notified vaccine preventable disease in Australia over the past 20 years. Pertussis notification and hospitalisation data have been available nationally since 1993, and provide different perspectives for understanding epidemiological trends. This report follows on from a previous review of Australian pertussis epidemiology from 1995–2005 and summarises routinely collected notification, hospitalisation and mortality data for 2006-2012. During the latter 7-year period, which incorporated epidemics in all jurisdictions, and in which acellular vaccines (as opposed to whole cell vaccines) were used exclusively, the average annual notification rate was more than 2.8 times that of the previous decade. In contrast, hospitalisation and mortality rates remained similar. The pattern of age-specific notification rates changed substantially, with cases aged 15 years or over representing 93% of total cases in 2006, but only 58% by 2012; the steepest increases were seen in children 2-4 and 6-9 years of age. In South Australia, where acellular vaccines were introduced into the primary schedule 2 years earlier than in other jurisdictions except the Northern Territory, a peak in notifications among those aged 5–9 and 10–12 years was observed earlier. Likely contributors to both the overall increase in notifications and changes in age distribution include increased diagnostic testing and more rapid waning of effectiveness following vaccination with acellular compared with whole cell vaccines, exacerbated by cessation of the 18-month dose in the National Immunisation Program from 2003. Commun Dis Intell 2014;38(3):E179-E194.

Keywords: pertussis, disease surveillance, immunisation

#### Introduction

In Australia, universal childhood immunisation with combined diphtheria-tetanus-pertussis (DTP) vaccine began in 1953 and was continued in the national

schedule when it commenced in 1975. Since 1982, the primary schedule has recommended infant doses at 2, 4 and 6 months, but both the number and timing of booster doses and vaccines in use have changed substantially since then. Recent modifications have included the switch for all scheduled doses from the diphtheria-tetanus-whole cell pertussis vaccine (DTPw) to the diphtheria-tetanus-acellular pertussis vaccine (DTPa) in 1999; the change in recommendation for the 5th dose to be administered at 4 years rather than 4 to 5 years of age in 2000; the removal of the 18-month booster in 2003; and the addition in 2004 of the adolescent booster reduced antigen content diphtheria-tetanus-acellular pertussis vaccine dose (dTpa) recommended with varying ages of administration by jurisdiction.<sup>1</sup>

The pertussis immunisation program is well established in Australia and vaccination coverage is high, at 92.2% for 3 DTPa doses in those aged 12 months and 91.7% for 4 DTPa doses in those aged 5 years.<sup>2</sup> Nevertheless, pertussis continues to be the most commonly notified vaccine preventable disease in Australia,<sup>3</sup> with increases in national notification rates over the past 20 years, in different age groups and epidemic cycles. Similar trends have occurred in other developed countries, though typically later than in Australia.<sup>4–7</sup>

Several changes in diagnostic testing are likely to have contributed to the observed increase in pertussis notifications. Firstly, the availability and use of serologic testing in adolescents and adults increased from the early 1990s. Secondly, from 2000, polymerase chain reaction (PCR) became available as a diagnostic test, initially in hospitals and then, with changes in reimbursement arrangements, also in primary care from 2007.8 Thirdly, the use of PCR in primary care was facilitated by laboratories accepting specimens collected by throat swab, as well as nasopharyngeal aspirate, which particularly facilitated testing of young children. Laboratories are legally mandated to report positive tests for pertussis under state and territory public health legislation. In the case of notifications based on PCR, which are accepted as confirmed cases without supplementary clinical criteria being required, diagnostic testing changes directly contributed to the rise in notifications.

Beyond the influence of changes in diagnostic practice, recent evidence has shown that protection from the acellular vaccine, universally adopted in Australia in 1999 and in South Australia and the Northern Territory from 1997, is not as long-lasting as that from the whole cell vaccine. <sup>9–11</sup> In turn, the shorter duration of immunity from the acellular vaccines has the potential to magnify the impact of changes to the vaccination schedule with subsequent epidemic cycles. This is likely to have occurred among children aged 1–3 years following the removal of the 18-month booster. <sup>12</sup>

This analysis provides a detailed overview of Australian pertussis trends nationally, regionally and by age group from 2006–2012, following a similar review for the period 1995–2005. Trends are considered both in historical context and in the context of recent changes to the National Immunisation Program (NIP).

#### **Methods**

#### **Data sources**

#### **Notifications**

In Australia, pertussis is notifiable in each state and territory; both confirmed and probable pertussis cases are required to be notified. For the period under review, under the Communicable Diseases Network Australia national case definition, a confirmed case required either laboratory confirmation or a combination of laboratory suggestive and clinical evidence. A confirmed case could also consist of clinical and epidemiological evidence. A probable case required clinical evidence only. Laboratory confirmation included isolation of *Bordetella pertussis* or detection by PCR. Laboratory suggestive evidence included serology (single point high titre or seroconversion) or an immunofluorescence assay.<sup>13</sup>

For this report, notification data were obtained from the NNDSS. All state and territory pertussis notifications with a diagnosis date between 1 January 2006 and 31 December 2012 were included. The diagnosis date field is derived from the date of onset, or where not supplied, the earliest recorded date entered for either date of specimen, date of notification, or date when the notification was received. Laboratory diagnostic data were available for all states and territories except Tasmania; limited data were available for South Australia. For all other jurisdictions, data completeness ranged from 86.3% (Victoria) to 99.3% (Australian Capital

Territory). For the purpose of this review, where multiple diagnostic methods were recorded in the dataset, the case was classified as having been diagnosed by the most sensitive method.<sup>14</sup> Typically, this was PCR.

As part of this review, an ecological analysis of vaccine cohorts based on individual jurisdiction of birth was conducted. This analysis involved South Australia and New South Wales as representing the 2 differing time periods when DTPa was adopted by states and territories. South Australia and the Northern Territory introduced the acellular vaccine in 1997; the other states and territories did so in 1999. For each of these 2 jurisdictions, further sub-grouping was performed based on birth cohort and subsequent eligibility for different vaccine types: whole cell vaccine for all doses; whole cell vaccine for the primary series; or acellular vaccine for all doses. Rates over time for children aged 5–9 and 10–12 years were then calculated for these groups.

This report forms an extension of a previous analysis that reviewed pertussis trends from 1995–2005. Data from the previous analysis have been referred to and incorporated into several graphs in order to provide broader context.<sup>3</sup>

#### Hospitalisation and mortality data

Hospitalisation data were obtained from the Australian Institute of Health and Welfare National Hospital Morbidity Database, which compiles administrative, demographic and clinical information about patients admitted to public and private hospitals. For this report, all hospital admissions between 1 January 2006 and 31 December 2010 were included. Eligible hospital admissions were extracted based on the International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification (ICD-10-AM), code A37 (whooping cough), or a subcode, listed as the principal or other diagnosis.

Mortality data were obtained from the NNDSS data field, which recorded whether the notified case had died from pertussis. This data field has been reliable since 2000.<sup>15</sup>

#### Population estimates

National, jurisdictional and age-specific mid-year estimated resident population data were obtained from the Australian Bureau of Statistics (ABS).<sup>16</sup>

#### Data analysis

Annual notification numbers and diagnostic test data were reviewed nationally, regionally, and by age group for the time period 2006–2012. National, regional and age group-specific rates were calculated using ABS population data. Similarly, hospitalisations were reviewed for 2006–2010 with national, regional and age group-specific rates calculated. Medians were used to summarise length of stay data for hospitalised cases. Mortality data were reviewed nationally by age group for the period of 2006–2012.

Analysis was conducted using Stata 12 and Excel 2010.

Ethical approval was not required for this review as de-identified population based data were collected and summarised for routine public health surveillance only.

#### Results

#### Secular trends

From 2006 to 2012, 156,200 notifications of pertussis were recorded by the NNDSS (Table 1). The average annual national rate for this 7-year period was 103.1 per 100,000 population, varying from a low of 23.1 in 2007 to a high of 173.3 per 100,000 in 2011. Though national notification rates initially fell during the period 2006–2007 from those in 2005, rates steadily increased from 2008 through 2011. This pattern of decreasing notifications fol-

lowed by a consistently upward trend with varying peak years was largely repeated across all jurisdictions but in different time frames. In 2006 and 2007, notification rates for those less than 15 years of age were lower than those aged 15 years or over, but this pattern reversed in 2008–2012 such that notification rates in those less than 15 years of age became more than double those in persons aged 15 years or over (Figure 1).

Figure 1: Incidence rates and incidence rate ratios for pertussis comparing children aged less than 15 years with the remainder of the population, Australia, 1995–2012, by year of onset

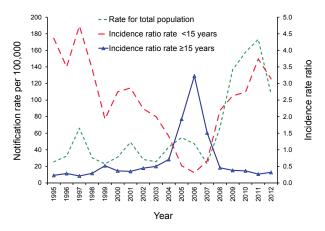


Table 1: Notification and hospitalisation rates per 100,000 population for pertussis, Australia, 2006 to 2012,\* by state or territory

					State or	territory				
Year	Data source	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
2006	Notifications	77.2	54.0	46.1	53.1	139.0	8.4	20.8	12.9	47.2
	Hospitalisations	0.3	3.2	4.3	2.6	4.9	0.4	1.0	0.8	2.3
2007	Notifications	28.4	23.5	12.6	36.7	23.9	5.1	20.2	6.3	23.1
	Hospitalisations	0.6	1.5	1.4	2.1	1.4	0.2	1.2	0.3	1.3
2008	Notifications	41.7	108.0	216.4 <sup>†</sup>	53.0	92.5	39.9	32.7	21.3	66.8
	Hospitalisations	2.0	5.6	14.0	2.7	3.9	2.2	2.0	1.9	3.6
2009	Notifications	99.2	176.1	94.8	142.4	332.6	122.8	70.3	34.7	136.8
	Hospitalisations	4.0	9.4	7.9	6.9	14.2	5.8	3.8	2.4	7.0
2010	Notifications	197.6	130.4	142.8	185.8	453.6 <sup>†</sup>	55.3	29.5	63.2	157.7
	Hospitalisations	3.0	4.6	10.4	6.8	17.4	2.8	6.0	3.3	6.2
2011	Notifications	225.4 <sup>†</sup>	182.3 <sup>†</sup>	163.4	200.9 <sup>†</sup>	143.4	68.9	156.3 <sup>†</sup>	169.6 <sup>†</sup>	173.3 <sup>†</sup>
2012	Notifications	117.7	80.7	128.0	168.3	54.4	246.5 <sup>†</sup>	79.8	142.8	107.6
Total number not	ifications	2,826	53,573	1,820	36,926	20,039	2,777	27,795	10,444	156,200
Total number hos	Total number hospitalisations		1,700	85	911	676	57	750	194	4,408
Average rate not	ifications	114.3	108.6	116.2	122.0	177.8	79.0	74.0	67.0	103.1
Average rate hos	pitalisations	2.0	4.9	7.7	4.3	8.5	2.3	2.8	1.8	4.1

Hospitalisation data available through 2010.

<sup>†</sup> Peak incidence rates for each state or territory for the period 2006–2012.

For the period 2006–2010, 4,408 hospitalisations were coded as pertussis equating to 4.7% of the number of notifications over this period and skewed significantly towards the lowest end of the age spectrum. Nevertheless, secular trends in hospitalisation rates generally reflected those of notification rates (Table 1). Over this period, 73.8% (n = 3,255) of total ICD-10-AM coded pertussis hospitalisations had a whooping cough code as the principal diagnosis, decreasing with age from 89.2% for those aged less than 6 months, to 50.0% for those aged 65 years or over. During 2006–2010, there was little difference between the average percentage of all age primary diagnoses coded as B. pertussis (49.0%) compared with those coded as whooping cough unspecified. However, for those aged less than 6 months, the percentage of diagnoses coded as B. pertussis (57.6%) increased, in 2010 becoming higher than the proportion coded as whooping cough unspecified for the first time in the 5-year period since 2006.

#### State and territory variations

Together, New South Wales (n = 53,573) and Queensland (n = 36,926) contributed 57.9% of all national notifications during the 7-year period 2006–2012. South Australia had the highest annual notification rate at 453.6 per 100,000 (2010) as well as the highest average jurisdictional rate for the 7-year period at 177.8 per 100,000 (Table 1). All states and territories, however, reported peak average annual notification rates, which ranged from 1.8 to 4.6 times higher than those for the same jurisdictions during the previous decade.<sup>3</sup> With respect to timing, the Northern Territory and Tasmania had earlier epidemic peaks occurring in 2008–2009; most other states and territories peaked later between 2010–2011, with Western Australia the last to reach epidemic levels in 2011–2012 (Table 1, Figure 2, Figure 3). By 2012, notification rates had decreased in most jurisdictions, with the notable exception of Tasmania where rates had climbed. Jurisdictional hospitalisation rates from 2006–2010 demonstrated that hospitalisation patterns closely followed notification patterns, but on a reduced scale (Figure 3).

#### Age distribution

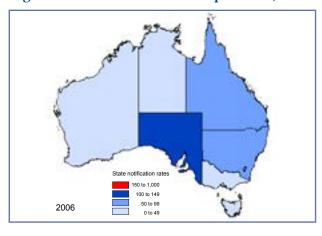
During the 7-year period, the average notification rate for those aged less than 15 years was 205.6 per 100,000 (range: 16.4–434.3) compared with 79.0 per 100,000 (range: 24.8–118.5) for those aged 15 years or over (Figure 1). For infants and children aged less than 15 years, notification rates increased steeply from 2007, while rates for those aged 15 years or over were lower, with a less

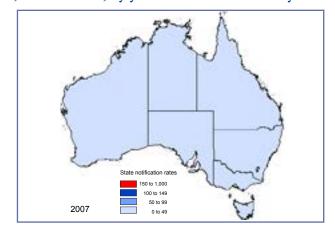
dramatic increase through 2010. By 2012, rates for both of these broad age groups were decreasing. The proportion of total notified cases aged 15 years or over was 93.0% in 2006, decreasing to 57.8% by 2012.

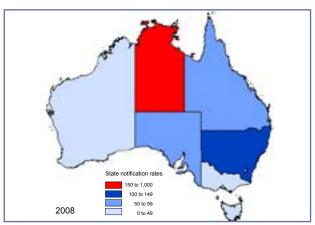
During the 2008–2011 epidemic period, the highest notification rates were seen in infants aged 0-5 months of age. Among children aged 6 months to 4 years, the highest notification rates were seen in the 3rd year, peaking at 411.0 per 100,000 in 2011 (Figure 4). However, the highest rates among all age groups were in children aged 5-9 years which reached 426.5 per 100,000 in 2010 and 556.2 in 2011. From 2008, rates among children in the 5–9 year age group increased progressively for each single year age group, peaking at 627.9 and 651.0 per 100,000 for those aged 7 and 8 years respectively. Children aged 10–14 years also had relatively high average notification rates, peaking in 2011 at 397.0 per 100,000. Within this age group, during 2006–2012, rates were more than 1.8 times higher for those aged 10–12 years compared with those aged 13–14 years, peaking at 659.5 per 100,000 in 2011 for those aged 10 years. Among older age groups, rates were considerably lower, averaging 62.1 and 47.2 per 100,000 for those aged 15–19 and 20–29 years respectively. For those aged 30–64 years, rates were highest for those aged 40-44 years, averaging 99.8 per 100,000. For those aged 65 years or over, the average rate was 86.0 per 100,000.

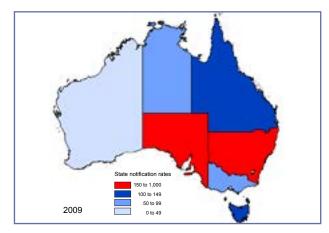
Age trends varied across states and territories, with the lowest notification rates among those 15 years of age or over (Appendix). In the Australian Capital Territory and New South Wales, the highest notification rates were in children aged 5–9 years during 2010–2011. In the Northern Territory, Queensland, South Australia, Tasmania, Victoria and Western Australia the highest notification rates were seen among infants less than 6 months of age in varying individual years between 2008 and 2012. The highest agespecific jurisdictional notification rates were in South Australia, where rates of 1,119.3 per 100,000 were recorded for infants aged less than 6 months and 1,117.4 per 100,000 among children aged 5–9 years in 2010. Tasmania experienced similarly high rates for infants less than 6 months of age in 2012, at 1,328.3 per 100,000. In South Australia, high rates were also experienced by those aged 10–12 years, with a peak of 1,158.9 per 100,000 in 2010. Figure 5 displays rates for those aged 5–9 and 10-12 years for South Australia compared with the same age groups in New South Wales, by birth cohort. Rates peaked for those aged 5–9 and 10–12 years in South Australia a year earlier than in New South Wales.

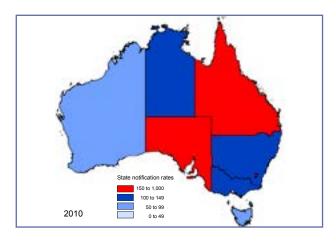
Figure 2: Notification rates for pertussis, Australia, 2006 to 2012, by year and state or territory

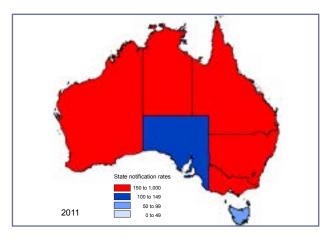


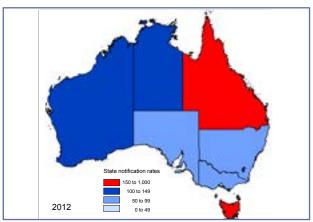












CDI Vol 38 No 3 2014 E183

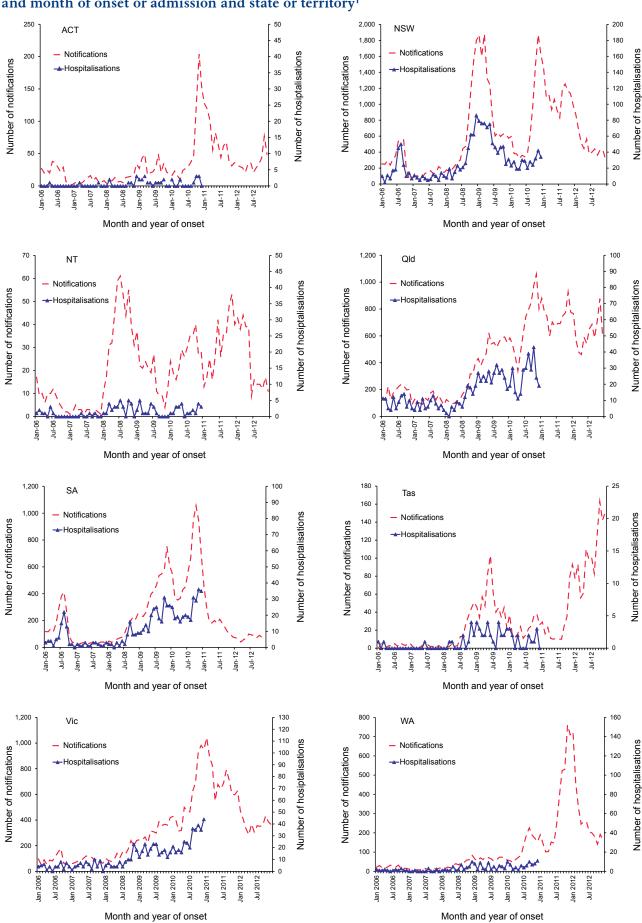


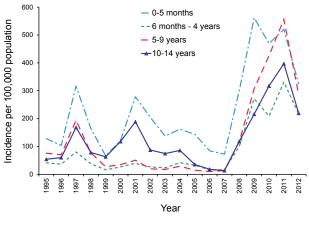
Figure 3: Notification and hospitalisation patterns of pertussis, Australia, 2006 to 2012,\* by year and month of onset or admission and state or territory<sup>†</sup>

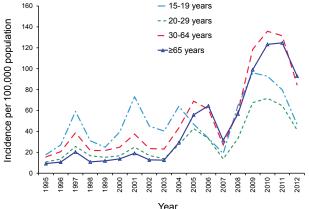
E184 CDI Vol 38 No 3 2014

Hospitalisation data available through 2010.

<sup>†</sup> Scales vary between states and territories.

Figure 4: Age-specific incidence of pertussis for groups aged < 15 years and ≥15 years,\* Australia, 1995 to 2012, by age group and year





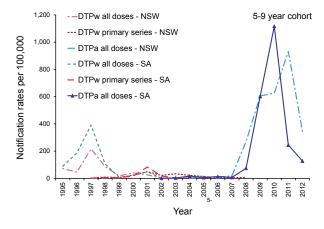
\* Scales differ between figures.

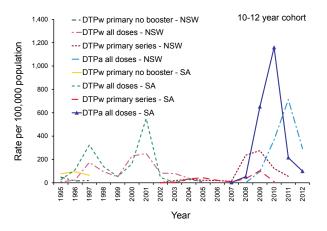
Historically, among infants less than 6 months of age, rates of ICD coded hospitalisations have been higher than notification rates. From 2007, however, notification rates for this age group have been consistently higher than hospitalisation rates (Figure 6). The highest hospitalisation rates occurred among those aged 3 months or less, increasing steeply between 2007 and 2009. Though rates were comparatively lower for infants and children aged 6 months to 4 years, this age group also experienced a sharp increase in hospitalisations from 2007–2009. Of all persons aged over 4 years, adults aged 65 years or over had the highest hospitalisation rates (Figure 7).

#### Diagnostic method

Compared with the previous decade, the completeness of NNDSS diagnostic testing data has improved. For the period of 1995–2005, the method of diagnosis was recorded for 50.1% of notifications, increasing to 85.8% of notifications for the period 2006–2012. Over the 7-year period,

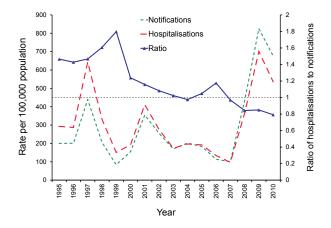
Figure 5: Incidence of pertussis for children aged 5–9 and 10–12 years,\* New South Wales and South Australia, 1995 to 2012, by birth cohort and year of onset





Scales differ between figures.

Figure 6: Ratio of national pertussis hospitalisation to notification rates for infants less than 6 months of age, Australia, 1995 to 2010\*

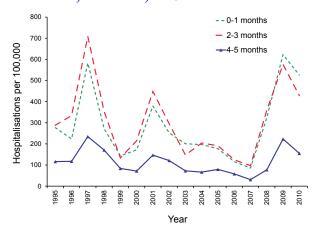


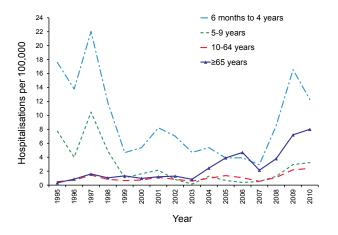
Hospitalisation data available through 2010.

CDI Vol 38 No 3 2014

an increasing proportion of notifications had PCR recorded as the method of diagnosis, increasing from 6.9% in 2006 to 58.7% in 2012. The increase in the percentage of diagnoses by PCR testing

Figure 7: Age-specific hospitalisation rates of pertussis for groups aged < 6 months and ≥6 months, Australia, 1995 to 2010\*,†





- \* Scales differ between figures.
- † Hospitalisation data available through 2010.

was most rapid in young children, with a more gradual increase among adults. The proportion of PCR confirmed diagnoses decreased and the proportion of serologic diagnoses increased with age (Figure 8). During the 7-year period, for those aged less than 1 year, culture alone was recorded as the diagnostic method for 67 (1.5%) notifications (range: 0%–6.0%). For the same age group, both culture and PCR together were recorded for 1.8% of notifications (range: 0%–4.3%).

#### Severe morbidity and mortality

During the period of investigation, 11 notified cases were reported to have died from pertussis. Of these, 10 deaths were in unvaccinated infants less than 2 months of age and one was an adult aged 70 years. There were twice as many infants less than 6 months of age hospitalised for pertussis than adults aged 65 years or over, but the median length of stay per hospital admission was longer for the older adult age group (median: 7 days) than for the infant group (median: 4 days; Table 2).

#### **Discussion**

For the period 2006–2012, the average annual pertussis notification rate was 2.8 times that for 1995–2005.<sup>3</sup> Unlike the previous review period, the expected 3–4 year epidemic cycles of approximately 12 months duration<sup>17</sup> were replaced by sustained epidemics, which peaked in 2009 or 2011 in different jurisdictions. This national picture is similar to previously published jurisdictional reviews of notifications for the same time period.<sup>18,19</sup>

Although cases are still believed to be underreported,<sup>20,21</sup> improved surveillance and laboratory diagnostics as well as heightened awareness among clinicians have led to increased testing and notification of disease.<sup>22–24</sup> In particular, the general availability of commercial PCR kits<sup>25</sup> as well as the

Table 2: Number of pertussis coded hospitalisations, median length of hospital stay and number of pertussis deaths, Australia, 2006 to 2012,\*,† by age group

	Hospita	l admissions	Length of	stay (days)†	Deaths*
Age group	n	(Rate per 100,000 population)	Median	Range	n
<6 months	1,832	257.9	4	(1–292)	10
6 months-4 years	557	9.0	2	(1–313)	0
5-9 years	113	1.7	2	(1–477)	0
10-64 years	1,166	1.4	3	(1–139)	0
≥65 years	740	4.9	7	(1–364)	1
All ages	4,408	4.1	3	(1–477)	11

Deaths are for the period 2006 to 2011.

<sup>†</sup> Length of stay is for the period 2006 to 2010

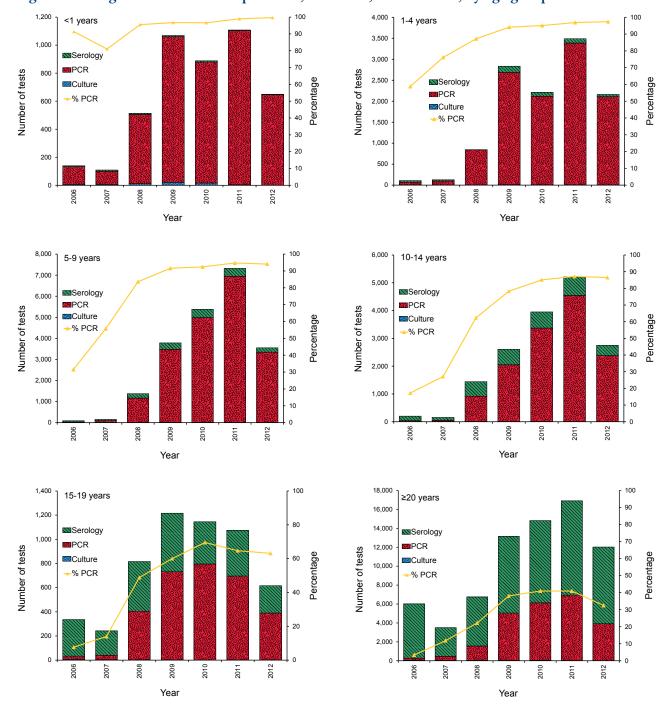


Figure 8: Diagnostic methods for pertussis, Australia, 2006 to 2012, by age group\*

Scales differ between age groups. Polymerase chain reaction.

**PCR** 

high sensitivity of PCR testing, as has been noted elsewhere, <sup>26</sup> have encouraged clinicians to test more, thus contributing to the rise in notifications.

It is unlikely, however, that the increase in pertussis notifications documented here is solely attributable to increased testing and diagnosis.8,12,26 The hospitalisation rates presented in this report, which rose similarly to notification rates but not to the same absolute magnitude, support this claim, as hospitalisation data are likely to be less influenced by diagnostic practices. Because PCR testing has been widely employed by hospitals since about 2000, any changes in hospital coding are likely to have been less dramatic than the more recent adoption of PCR testing in primary care.8 It is notable that hospitalisation rates in the 2009 epidemic were similar to those recorded in 1997, despite the increased availability of PCR testing from 2000.27 If anything, this would suggest that hospitalisation for pertussis was less common in the

most recent acellular vaccine era. Similarly, the number of deaths for this 7-year review period remained comparable to the number of deaths notified in the previous decade.

During the period 1995–2005, notification rates for Australian adults were much higher than in comparable countries internationally.<sup>28–30</sup> In contrast, in 2006–2012 the highest rates occurred among younger age groups, specifically in infants aged less than 6 months and children aged 5–9 years. Recent outbreaks in the United States of America (US), the Unitied Kingdom (UK), Canada and New Zealand have also been characterised by high rates in infants too young to be vaccinated. 4,31-34 High rates in infants have been common for several decades. When notifications were largely reliant upon clinical diagnoses, higher rates were often detected in hospitalisation data because infant cases admitted to hospital were not notified. Since the increased use of PCR testing in Australia, however, this differential between notification and hospitalisation data for infants has diminished.35

Recent trials have investigated the delivery of pertussis vaccine at birth. <sup>36–39</sup> The cocooning strategy (vaccinating close contacts of infants to reduce the likelihood of exposure) and maternal vaccination have been recommended for preventing infection in very young infants and have been given equal preference in the most recent *Australian Immunisation Handbook*. <sup>40</sup> Maternal vaccination has also been recommended by the US, UK, Canada and New Zealand. <sup>31,32,41,42</sup> Systematic evaluation of the various strategies to protect newborn infants is vital to prevent deaths and severe disease from pertussis in this vulnerable group.

The efficacy of DTPa vaccines that contain three or more antigens has been estimated at 71%–78% for the prevention of milder pertussis and 84% for typical disease.43,44 While data demonstrate that receipt of the 1st dose of the primary DTPa series can reduce incidence of severe illness in infants, 45,46 DTPa vaccine does not appear to confer immunity for as long as the DTPw vaccine.<sup>10</sup>, 47-52 Consequently, high rates of pertussis in children may have resulted in part from the NIP switch to the DTPa vaccine. Specific estimates of the duration of immunity afforded by the wholecell vaccine range from 4–14 years, though studies suggest that immunity conferred by the acellular vaccine may only last 5 years or less.<sup>9,53</sup> Average notification rates from 2006-2012 for children aged 7 and 8 years were more than four times as high as those experienced by the same age group from 1995–2005. This suggests that immunity waned in the period following receipt of the 4 year old dose, for which coverage was estimated

to be high,<sup>54–56</sup> before the adolescent booster could be administered.<sup>57</sup> Similarly, high rates among US children aged 8–12 years were documented from 2005 and correspond to the first cohort of children to have received a schedule containing all acellular vaccines.<sup>53,58</sup> In Australia, at the state and territory level, the DTPa vaccine was adopted for the primary series in South Australia and the Northern Territory in 1997 before being adopted Australia-wide for all childhood doses in 1999. This is likely to have contributed to notification rates for those aged 5–9 and 10–12 years in South Australia peaking earlier than in New South Wales, despite these states sharing similar overall epidemic patterns.

NIP schedule changes, specifically the 2003 removal of the 18-month booster dose, also likely influenced notification rates among younger age groups by exacerbating the impact of the decreased efficacy and longevity of the acellular vaccine. The removal of the 18-month booster expanded the time interval between doses from 6 months to 4 years of age leaving those aged 1–3 years vulnerable to waning immunity and resulting in record high notification rates for those aged 1–4 years from 2008. Australian serosurvey results from 2007 support this claim, reporting that among children 1–4 years of age, prevalence of undetectable immunoglobulin G (IgG) levels had increased from 25% in 1997–1998 to 62% in 2007.

Based on evidence of waning immunity, as well as evidence that toddlers serve as an important source of infection for infants too young to be vaccinated, <sup>59–61</sup> the current immunisation handbook advises that an additional dose of DTPa in the 2nd year of life will minimise the likelihood of a child developing pertussis prior to their scheduled booster dose at 3.5 to 4 years of age. <sup>40</sup> This is in line with the World Health Organization's 2010 recommendation that a booster be given in the 2nd year of life unless country-specific epidemiological evidence supports delaying this until preschool. <sup>62</sup>

The decreased notification rates among older adolescents and young adults demonstrated in this analysis were likely partially influenced by the 2003 NIP addition of the adolescent booster recommended for those aged 15–17 years. Despite variation in coverage and timing of programs across state and territories, 63,64 the dTpa employed for the adolescent dose has been demonstrated as moderately effective, both in a clinical trial and a field setting. 64,66,67 Though duration of immunity associated with a single booster dose is thought to be limited, 68 both the US and Canada have reported temporally similar decreases in disease among adolescents following the addition of adolescent boosters. 69,70 In response to concern

about waning immunity, the Australian Technical Advisory Group on Immunisation has recommended shifting the 5th dose to 11–13 years to decrease the time between the primary childhood series and adolescent booster.<sup>71</sup>

Though notification rates appeared to decrease somewhat in 2012, average rates for the period of investigation were dramatically higher than those experienced in the previous decade. Because of the increase in community PCR testing, baseline pertussis rates may well remain higher than they were prior to this change in diagnostic practice. Nevertheless, in light of the fact that this review demonstrates that pertussis notification rates are once again highest among the young, strategies targeted at reducing disease among infants must continue to be pursued. Due to the dynamic nature of pertussis immunity, it is imperative to continue exploring a broad range of both scientific and policy solutions.

#### **Acknowledgements**

The National Centre for Immunisation for Research and Surveillance of Vaccine Preventable Diseases is supported by the Australian Government Department of Health, the NSW Ministry of Health and The Children's Hospital at Westmead. We wish to acknowledge the Vaccine Preventable Diseases Surveillance Section, Health Emergency Management Branch, Office of Health Protection, Australian Government Department of Health for data from the National Notifiable Diseases Surveillance System, and the Hospitals Unit, Australian Institute of Health and Welfare for data from the National Hospital Morbidity Database.

This report is part of an ongoing series produced by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases documenting vaccine preventable disease epidemiological trends.

#### **Author details**

Ms Alexis Pillsbury,<sup>1,2</sup> Master of Philosophy (Applied Epidemiology) Scholar Dr Helen E Quinn,<sup>1,3</sup> Research Fellow Professor Peter B McIntyre,<sup>1,3</sup> Director

- National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, The Children's Hospital at Westmead and the University of Sydney, Westmead, New South Wales
- 2. National Centre for Epidemiology and Population Health, Australian National University, Acton, Australian Capital Territory
- Discipline of Paediatrics and Child Health, University of Sydney, The Children's Hospital at Westmead, Westmead, New South Wales

Corresponding author: Ms Alexis Pillsbury, National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, Locked Bag 4001, WESTMEAD NSW 2145. Telephone: +61 2 9845 1232. Facsimile: +61 2 9845 1418. Email: alexis.pillsbury@health.nsw.gov.au

#### References

- National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases. Significant events in diphtheria, tetanus and pertussis vaccination practice in Australia. 2013 [online] Accessed on 8 July 2014. Available from: http://ncirs.edu.au/immunisation/history/Diphtheria-tetanus-pertussis-history-December-2013.pdf
- Hull BP, Dey A, Menzies RI, Brotherton J, McIntyre PB. Annual immunisation coverage report, 2012. Commun Dis Intell In press 2014.
- 3. Quinn HE, McIntyre, P. Pertussis epidemiology in Australia over the decade 1995–2005 trends by region and age group. Commun Dis Intell 2007;31(2):205–215.
- Cherry JD. Epidemic pertussis in 2012—the resurgence of a vaccine-preventable disease. N Engl J Med 2012;367(9):785–787.
- 5. Wirsing von König CH, Riffelman M. Pertussis: an old disease in new clothes. Euro Surveill 2007;12(9):E1–E2.
- Winter K, Harriman K, Zipprich J, Schechter R, Talarico J, Watt J, et al. California pertussis epidemic, 2010. J Pediatr 2012161(6):1–6.
- Centers for Disease Control and Prevention. Pertussis epidemic—Washington, 2012. MMWR Morb Mortal Wkly Rep 2012;61(28):517–522.
- Kaczmarek MC, Valenti L, Kelly HA, Ware RS, Britt HC, Lambert SB. Sevenfold rise in likelihood of pertussis test requests in a stable set of Australian general practice encounters, 2000–2011. Med J Aust 2013;198(11):624–628.
- Wendelboe AM, Van Rie A, Salmaso S, Englund JA. Duration of immunity against pertussis after natural infection or vaccination. *Pediatr Infect Dis J* 2005;24(5 Suppl):S58–S61.
- Witt MA, Arias L, Katz PH, Truong ET, Witt DJ. Reduced risk of pertussis among persons ever vaccinated with whole cell pertussis vaccine compared to recipients of acellular pertussis vaccines in a large US cohort. Clin Infect Dis 2013;56(9):1248–1254.
- 11. Witt MA, Katz PH, Witt DJ. Unexpectedly limited durability of immunity following acellular pertussis vaccination in pre-adolescents in a North American outbreak. *Clin Infect Dis* 2012;54(12):1730–1735.
- Campbell P, McIntyre P, Quinn H, Hueston L, Gilbert G, McVernon J. Increased population prevalence of low pertussis toxin antibody levels in young children preceding a record pertussis epidemic in Australia. PLoS One 2012;7(4):1–7.
- 13. Australian Government Department of Health and Ageing. Australian national notifiable diseases case definitions: pertussis case definition. 2004 [online] Accessed on 20 October 2012. Available from: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-surveil-nndss-casedefs-cd\_pertus.htm
- 14. Australian Government Department of Health and Ageing. Australian national notifiable diseases case definitions. 2011 [online] Accessed on 24 October 2012. Available from: http://www.health.gov.au/internet/ main/publishing.nsf/Content/cdna-casedefinitions.htm

- Georgousakis M. Pertussis deaths in Australia—what has changed? Public Health Association of Australia 13th National Immunisation Conference; 19–21 June; Darwin 2012.
- 16. Australian Bureau of Statistics. Australian Demographic Statistics, Dec 2012. Catalogue Number 3101.0. Updated on 25 May 2010 [online]. Available from: http://www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/3101.0Main+Features1Dec%20 2012?OpenDocument
- Wood N, Quinn HE, McIntyre P, Elliott E. Pertussis in infants: preventing deaths and hospitalisations in the very young. J Paediatr Child Health 2008;44(4):161–165.
- Spokes PJ, Quinn HE, McAnulty JM. Review of the 2008–2009 pertussis epidemic in NSW: notifications and hospitalisations. N S W Public Health Bull 2010;21(7–8):167–173.
- Clarke MF, Rasiah K, Copland J, Watson M, Koehler AP, Dowling K, et al. The pertussis epidemic: informing strategies for prevention of severe disease. *Epidemiol Infect* 2013;141(3)463–471.
- Allen CJ, Ferson MJ. Notification of infectious diseases by general practitioners: a quantitative and qualitative study. Med J Aust 2000;172(7):325–328.
- 21. Cherry JD. The epidemiology of pertussis: a comparison of the epidemiology of the disease pertussis with the epidemiology of Bordetella pertussis infection. *Pediatrics* 2005;115(5):1422–1427.
- 22. McIntyre P, Wood N. Pertussis in early infancy: disease burden and preventive strategies. Curr Opin Infect Dis 2009;22(3):215–223.
- 23. Bamberger ES, Srugo I. What is new in pertussis? Eur J Pediatr 2008;167(2):133–139.
- 24. Cherry JD. The present and future control of pertussis. Clin Infect Dis 2010;51(6):663–667.
- 25. McIntyre PB, Sintchenko V. The "how" of polymerase chain reaction testing for *Bordetella pertussis* depends on the "why". Clin Infect Dis 2013;56(3):332–334.
- Fisman DN, Tang P, Hauck T, Richardson S, Drews SJ, Low D, et al. Pertussis resurgence in Toronto, Canada: a population-based study including test-incidence feed-back modeling. BMC Public Health 2011;11:694.
- 27. Chiu C, Dey A, Wang H, Menzies R, Deeks S, Majahan D, et al. Vaccine preventable diseases in Australia, 2005 to 2007. Commun Dis Intell 2010;34(Suppl):S1–S167.
- Centers for Disease Control and Prevention. Pertussis (whooping cough) surveillance and reporting. [online].
   Accessed on 3 December 2013. Available from: http://www.cdc.gov/pertussis/surv-reporting.html
- McIntyre P. Is Australia the world capital of pertussis? National pertussis workshop: strategies to prevent severe pertussis in the next decade; 25–26 August 2011; Sydney, Australia.
- Kanitz E. Pertussis in Europe from 1980–2010 incidence and vaccination coverage. European Scientific Conference on Applied Infectious Disease Epidemiology (ESCAIDE); 6–8 November 2011; Stockholm, Sweden.
- Public Health England. Pertussis (whooping cough) immunisation for pregnant women. [online]. 2013 Accessed on 23 November 2013. Available from: http://www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/WhoopingCough/ImmunisationForPregnantWomen/http://www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/WhoopingCough/ImmunisationForPregnantWomen/

- 32. Auckland Regional Public Health Service. Information about whooping cough (pertussis) immunisation during pregnancy. [online]. 2013 Accessed on 23 November 2013. Available from: http://www.arphs.govt.nz/Portals/0/Health%20Information/Communicable%20 Disease/Pertussis/Pertussis%20Fact%20sheet%20 Jan%202013/Whooping%20cough%20immunisation%20in%20pregrnancy%20January%202013.pdf
- 33. Chiappini E, Stival A, Galli L, de Martino M. Pertussis re-emergence in the post-vaccination era. *BMC Infect Dis* 2013;13:151.
- Alphonso C. Whooping cough makes deadly return across Canada. The Globe and Mail. 23 July 2012.
- 35. Torvaldsen S, McIntyre P. Do variations in pertussis notifications reflect incidence or surveillance practices? A comparison of infant notification rates and hospitalisation data in NSW. N S W Public Health Bull 2003;14(4–5):81–84.
- Belloni C, De Silvestri A, Tinelli C, Avanzini MA, Marconi M, Strano F, et al. Immunogenicity of a threecomponent acellular pertussis vaccine administered at birth. Pediatrics 2003;111(5 Pt 1):1042–1045.
- Halasa NB, O'Shea A, Shi JR, Lafleur BJ, Edwards KM. Poor immune responses to a birth dose of diptheria, tetanus, and acellular pertussis vaccine. J Pediatr 2008;153(3):327–332.
- 38. Knuf M, Schmitt HJ, Wolter J, Schuerman L, Jacquet JM, Kieninger D, et al. Neonatal vaccination with an acellular pertussis vaccine accelerates the acquisition of pertussis antibodies in infants. *J Pediatr* 2008;152(5):655–660.
- Wood N, McIntyre P, Marshall H, Roberton D. Acellular pertussis vaccine at birth and one month induces antibody responses by two months of age. *Pediatr Infect Dis* J 2010;29(3):209–215.
- Australian Technical Advisory Group on Immunisation. The Australian Immunisation Handbook. 10th edn. Canberra: Australian Government Department of Health and Ageing; 2013.
- 41. Matlow JN, Pupco A, Bozzo P, Koren G. Tdap vaccination during pregnancy to reduce pertussis infection in young infants. Can Fam Physician 2013;59(5):497–498.
- 42. Centers for Disease Control and Prevention. Updated recommendations for use of tetanus toxoid, reduced diphtheria toxoid and acellular pertussis vaccine (Tdap) in pregnant women and persons who have or anticipate having close contact with an infant aged < 12 months—Advisory Committee on Immunization Practices (ACIP), 2011. MMWR Morb Mortal Wkly Rep 2011;60(41):1424–1426.</p>
- Zhang L Prietsch SO, Axelsson I, Halperin SA. Acellular vaccines for preventing whooping cough in children. Cochrane Database Syst Rev 2012;14(3):CD001478.
- Jefferson T, Rudin M, DiPietrantonj C. Systematic review of the effects of pertussis vaccines in children. Vaccine 2003;21(17–18):2003–2014.
- 45. Foxwell AR, McIntyre P, Quinn H, Roper K, Clements MS. Severe pertussis in infants: estimated impact of first vaccine dose at 6 versus 8 weeks in Australia. *Pediatr Infect Dis J* 2011;30(2):161–163.
- Cortese MM, Baughman AL, Zhang R, Srivastava PU, Wallace GS. Pertussis hospitalizations among infants in the United States, 1993–2004. *Pediatrics* 2008;121(3):484–492.

- 47. Stehr K, Cherry JD, Heininger U, Schmitt-Grohé S, Uberall M, Laussucq S, et al. A comparative efficacy trial in Germany in infants who received either the Lederle/Takeda acellular pertussis component DTP (DTaP) vaccine, the Lederle whole-cell component DTP vaccine, or DT vaccine. Pediatrics 1998;101(1 Pt1):1–11.
- 48. Schmitt HJ, von König CH, Neiss A, Bogaerts H, Bock HL, Schulte-Wissermann H, et al. Efficacy of acellular pertussis vaccine in early childhood after household exposure. *JAMA* 1996;275(1):37–41.
- 49. Liese JG, Meschievitz CK, Harzer E, Froeschle J, Hosbach P, Hoppe JE, et al. Efficacy of a two-component acellular pertussis vaccine in infants. *Pediatr Infect Dis J* 1997;16(1):1038–1044.
- Simondon F, Preziosi MP, Yam A, Kane CT, Chabirand L, Iteman I, et al. A randomized double-blind trial comparing a two-component acellular to a whole-cell pertussis vaccine in Senegal. Vaccine 1997;15(15):1606–1612.
- Olin P, Rasmussen F, Gustafsson L, Hallander HO, Heijbel H. Randomised controlled trial of two-component, three-component, and five-component acellular pertussis vaccines compared with whole-cell pertussis vaccine. Ad Hoc Group for the Study of Pertussis Vaccines. Lancet 1997;350(9091):1569–1577.
- Sheridan SL, Ware RS, Grimwood K, Lambert SB. Number and order of whole cell pertussis vaccines in infancy and disease protection. JAMA 2012;308(5):454–456.
- Klein NP, Bartlett J, Rowhani-Rahbar A, Fireman B, Baxter R. Waning protection after fifth dose of acellular pertussis vaccine in children. N Engl J Med 2012;367(11):1012–1019.
- Hull B, Deeks S, Menzies R, McIntyre P. Immunisation coverage annual report, 2007. Commun Dis Intell 2009;33(2):170–187.
- Hull BP, Mahajan D, Dey A, Menzies RI, McIntyre PB. Immunisation coverage annual report, 2008. Commun Dis Intell 2010;34(3):241–258.
- Hull B, Dey A, Mahajan D, Menzies R, McIntyre P. Immunisation coverage annual report, 2009. Commun Dis Intell 2011;35(2):132–148.
- 57. Sheridan SL, McCall BJ, Davis CA, Robson JM, Hull BP, Selvey CE, et al. Acellular pertussis vaccine effectiveness for children during the 2009–2010 pertussis epidemic in Queensland. *Med J Aust* 2014;200(6):334–338.
- Misegades LK, Winter K, Harriman K, Talarico J, Messonnier NE, Clark TA, et al. Association of childhood pertussis with receipt of 5 doses of pertussis vaccine by time since last vaccine dose, California, 2010. JAMA 2012;308(20):2126–2132.
- de Greeff SC, Mooi FR, Westerhof A, Verbakel JM, Peeters MF, Heuvelman CJ, et al. Pertussis disease burden in the household: how to protect young infants. Clin Infect Dis 2010;50(10):1339–1345.

- 60. Wiley KE, Zuo Y, Macartney KK, McIntyre PB. Sources of pertussis infection in young infants: a review of key evidence informing targeting of the cocoon strategy. *Vaccine* 2013;31(4):618–625.
- Jardine A, Conaty SJ, Lowbridge C, Staff M, Vally H. Who gives pertussis to infants? Source of infection for laboratory confirmed cases less than 12 months of age during an epidemic, Sydney, 2009. Commun Dis Intell 2010;34(2):116–121.
- World Health Organization. Meeting of the Strategic Advisory Group of Experts on Immunization, April 2010

   conclusions and recommendations. Wkly Epidemiol Rec 2010;85(22):197–212.
- 63. Quinn HE, McIntyre PB. The impact of adolescent pertussis immunization, 2004–2009: lessons from Australia. Bull World Health Organ 2011;89(9):666–674.
- Rank C, Quinn HE, McIntyre PB. Pertussis vaccine effectiveness after mass immunization of high school students in Australia. Pediatr Infect Dis J 2009;28(2):152–155.
- Ward JI, Cherry JD, Chang SJ, Partridge S, Lee H, Treanor J, et al. Efficacy of an acellular pertussis vaccine among adolescents and adults. N Engl J Med 2005;353(15):1555–1563.
- Baxter R, Bartlett J, Rowhani-Rahbar A, Fireman B, Klein NP. Effectiveness of pertussis vaccines for adolescents and adults: case-control study. BMJ 2013;347:f4249.
- Barlow RS, Reynolds LE, Cieslak PR, Sullivan AD. Vaccinated children and adolescents with pertussis infections experience reduced illness severity and duration, Oregon, 2010–2012. Clin Infect Dis 2014;58(11):1523–1529.
- 68. Booy R, Van Der Meeren O, Ng SP, Celzo F, Ramakrishnan G, Jacquet JM. A decennial booster dose of reduced antigen content diphtheria, tetanus, acellular pertussis vaccine (Boostrix™) is immunogenic and well tolerated in adults. Vaccine 2010;29(1):45–50.
- 69. Skoff TH, Cohn AC, Clark TA, Messonier NE, Martin SW. Early impact of the US Tdap vaccination program on pertussis trends. *Arch Pediatr Adolesc Med* 2012;166(4):344–349.
- Skowronski DM, Janjua NZ, Sonfack Tsafack EP, Ouakki M, Hoang L, De Serres G. The number needed to vaccinate to prevent infant pertussis hospitalization and death through parent cocoon immunization. Clin Infect Dis 2012;54(3):318–327.
- 71. Australian Government Department of Health and Ageing. Australian Technical Advisory Group on Immunisation bulletin 44th meeting: 24–25 February 2011. [online]. Canberra: Australian Government Department of Health and Ageing; 2011. Accessed on 20 September 2013. Available from: http://www.health.gov.au/internet/immunise/publishing.nsf/Content/atagi-meet44bulletin

Appendix: Age-specific notification and hospitalisation rates per 100,000 population for pertussis, Australia, 2006 to 2012,\* by state or territory

					Age group			
State or territory	Data source	<6 months	6 months- 4 years	5-9 years	10-14 years	15–19 years	20-64 years	>65 years
ACT								
2006	Notifications	0.0	32.4	29.2	9.3	36.7	94.4	100.4
	Hospitalisations	0.0	0.0	0.0	0.0	0.0	0.5	0.0
2007	Notifications	43.9	15.5	4.9	0.0	27.9	32.8	39.2
	Hospitalisations	86.8	0.0	0.0	0.0	0.0	0.0	0.0
2008	Notifications	87.1	25.1	58.6	47.1	23.5	41.6	46.7
	Hospitalisations	86.9	0.0	0.0	0.0	0.0	1.8	2.9
2009	Notifications	247.8	78.3	82.4	51.9	46.9	104.4	143.5
	Hospitalisations	371.7	4.9	0.0	0.0	0.0	0.9	5.6
2010	Notifications	159.1	99.3	503.5	377.1	74.4	172.7	221.5
	Hospitalisations	156.0	9.3	0.0	0.0	0.0	1.3	5.4
2011	Notifications	320.6	171.4	363.7	343.2	104.0	201.6	333.5
2012	Notifications	78.0	62.4	130.7	247.9	66.3	106.5	153.2
Average ra	ate notifications	137.0	71.2	169.2	152.4	54.2	109.1	153.4
Average ra	ate hospitalisations	144.0	3.0	0.0	0.0	0.0	0.9	2.9
NSW								
2006	Notifications	161.4	27.4	13.4	18.7	35.2	64.1	60.5
	Hospitalisations	176.9	7.4	0.7	1.1	0.2	1.2	5.0
2007	Notifications	137.7	28.1	24.2	18.3	17.9	23.1	22.3
	Hospitalisations	109.1	5.1	0.5	0.0	0.0	0.3	2.0
2008	Notifications	571.7	233.3	278.9	270.1	135.7	65.9	50.4
	Hospitalisations	456.8	16.3	3.0	0.7	1.3	1.4	3.6
2009	Notifications	911.2	579.4	605.0	342.0	141.1	97.2	63.1
	Hospitalisations	728.3	28.0	3.4	3.8	1.7	2.0	8.3
2010	Notifications	418.2	284.1	626.2	365.0	88.3	62.9	44.9
	Hospitalisations	285.8	12.1	3.2	0.9	0.6	1.9	4.1
2011	Notifications	648.9	492.4	931.1	526.2	77.2	77.0	55.3
2012	Notifications	337.2	239.2	343.6	205.2	33.3	38.3	32.4
Average ra	ate notifications	459.8	273.2	403.3	248.1	75.8	61.3	46.8
Average ra	ate hospitalisations	355.0	13.9	2.1	1.3	0.8	1.4	4.6
NT								
2006	Notifications	109.2	6.3	0.0	0.0	64.4	59.0	51.3
	Hospitalisations	163.8	6.3	0.0	0.0	0.0	3.0	10.3
2007	Notifications	53.9	12.5	5.8	0.0	0.0	13.1	48.2
	Hospitalisations	54.0	6.2	0.0	0.0	0.0	0.7	0.0
2008	Notifications	880.6	277.5	368.1	317.9	121.9	176.0	275.7
	Hospitalisations	827.1	49.6	5.7	0.0	12.1	2.1	9.2
2009	Notifications	465.8	126.3	137.5	119.7	42.0	87.7	61.0
	Hospitalisations	619.5	12.1	5.7	0.0	5.9	1.4	0.0
2010	Notifications	317.5	172.4	315.0	194.0	90.4	116.2	164.8
	Hospitalisations	690.8	6.0	5.7	0.0	5.9	4.7	8.2
2011	Notifications	597.8	197.9	535.0	448.5	61.9	86.7	210.7
2012	Notifications	452.0	196.4	377.2	300.8	36.9	80.4	71.7
Average ra	ate notifications	414.5	142.6	249.5	196.7	59.9	89.1	127.8
Average ra	ate hospitalisations	476.9	16.0	3.4	0.0	4.9	2.4	5.5

E192 CDI Vol 38 No 3 2014

Appendix continued: Age-specific notification and hospitalisation rates per 100,000 population for pertussis, Australia, 2006 to 2012,\* by state or territory

					Age group			
State or territory	Data source	<6 months	6 months- 4 years	5-9 years	10-14 years	15–19 years	20-64 years	>65 years
Qld								
2006	Notifications	61.6	13.7	8.3	36.9	47.6	55.8	95.7
	Hospitalisations	50.8	2.9	0.4	0.3	0.0	1.9	7.1
2007	Notifications	52.9	7.3	6.8	11.7	31.7	37.7	79.0
	Hospitalisations	52.7	1.2	0.7	1.0	0.3	1.5	4.5
2008	Notifications	147.6	28.0	26.4	39.3	38.3	51.7	96.7
	Hospitalisations	120.0	2.7	0.4	0.3	0.7	1.2	7.5
2009	Notifications	404.7	136.8	181.2	162.0	101.3	132.4	170.7
	Hospitalisations	353.8	14.0	2.8	2.4	1.0	3.1	9.1
2010	Notifications	471.7	179.6	334.4	262.1	102.9	163.1	210.7
	Hospitalisations	353.1	12.2	2.8	2.0	1.0	2.9	11.3
2011	Notifications	488.2	263.2	471.8	404.6	105.4	152.9	191.6
2012	Notifications	344.6	233.6	397.6	339.6	74.6	119.4	176.9
Average ra	ate notifications	287.6	127.9	207.9	180.0	72.3	103.2	148.6
Average ra	ate hospitalisations	193.0	6.9	1.4	1.2	0.6	2.1	8.0
SA								
2006	Notifications	11.1	7.4	19.9	26.5	83.0	174.4	169.9
	Hospitalisations	44.4	4.9	1.0	1.0	2.9	4.0	11.0
2007	Notifications	0.0	14.7	7.4	9.9	16.0	28.5	26.2
	Hospitalisations	10.6	2.4	2.1	0.0	0.0	1.2	2.5
2008	Notifications	163.3	51.4	74.3	74.7	57.0	97.6	113.8
	Hospitalisations	192.3	3.6	1.1	1.0	1.0	2.4	6.1
2009	Notifications	847.8	338.6	602.2	582.7	181.8	313.7	247.9
	Hospitalisations	673.1	26.7	12.7	6.0	2.0	7.6	18.5
2010	Notifications	1,119.3	493.1	1117.4	872.4	238.1	392.6	334.6
	Hospitalisations	794.4	39.5	9.6	14.0	6.0	7.9	25.1
2011	Notifications	277.2	212.2	244.9	191.1	58.5	132.2	137.8
2012	Notifications	108.1	50.5	127.9	93.6	21.9	46.4	52.1
Average ra	ate notifications	366.0	169.9	310.7	263.5	94.1	169.8	154.4
Average ra	ate hospitalisations	352.5	15.9	5.3	4.4	2.4	4.7	12.8
Tas.								
2006	Notifications	62.8	3.7	0.0	11.7	0.0	8.7	12.6
	Hospitalisations	31.4	0.0	0.0	0.0	0.0	0.0	1.4
2007	Notifications	30.2	7.3	3.2	2.9	2.9	4.5	8.2
	Hospitalisations	0.0	3.6	0.0	0.0	0.0	0.0	0.0
2008	Notifications	303.9	70.8	60.7	83.2	58.4	28.0	26.7
	Hospitalisations	180.1	10.4	0.0	0.0	0.0	0.3	1.3
2009	Notifications	722.1	224.3	162.1	211.3	134.5	107.4	57.0
	Hospitalisations	295.7	23.5	0.0	0.0	2.9	2.7	3.9
2010	Notifications	258.5	82.5	62.0	54.2	67.3	51.9	39.0
	Hospitalisations	320.4	6.6	0.0	0.0	2.9	0.0	1.3
2011	Notifications	256.2	114.9	156.1	197.8	26.5	51.7	41.4
2012	Notifications	1,328.3	677.7	786.9	691.2	130.0	141.7	111.0
Average ra	ate notifications	422.4	171.1	173.9	175.3	60.0	56.7	43.9
Average ra	ate hospitalisations	164.7	9.1	0.0	0.0	1.2	0.6	1.6

CDI Vol 38 No 3 2014 E193

Appendix continued: Age-specific notification and hospitalisation rates per 100,000 population for pertussis, Australia, 2006 to 2012,\* by state or territory

					Age group			
State or territory	Data source	<6 months	6 months- 4 years	5–9 years	10-14 years	15–19 years	20-64 years	>65 years
Vic.								
2006	Notifications	30.7	2.8	3.7	6.2	15.5	23.7	32.8
	Hospitalisations	67.6	0.0	0.0	0.0	0.0	0.3	2.5
2007	Notifications	58.8	14.5	13.1	17.6	17.8	21.3	21.2
	Hospitalisations	81.6	2.1	0.3	0.0	0.0	0.5	1.3
2008	Notifications	141.3	25.5	25.9	41.6	20.7	34.0	28.6
	Hospitalisations	134.6	4.7	0.3	0.3	0.6	0.8	1.8
2009	Notifications	302.3	74.8	72.4	81.1	43.5	68.7	72.0
	Hospitalisations	328.8	4.8	0.9	1.2	0.3	1.2	3.5
2010	Notifications	470.3	133.7	207.6	256.3	75.6	113.1	119.5
	Hospitalisations	437.5	8.2	3.0	0.9	1.1	2.3	7.7
2011	Notifications	434.8	189.7	282.3	263.3	75.4	134.0	164.1
2012	Notifications	232.9	98.9	106.6	95.6	31.2	70.0	101.7
Average ra	ate notifications	241.8	79.5	102.5	108.2	40.1	67.5	79.3
Average ra	ate hospitalisations	212.4	4.1	0.9	0.5	0.4	1.1	3.4
WA								
2006	Notifications	66.9	17.1	10.3	5.6	12.3	13.1	13.2
	Hospitalisations	74.3	4.3	0.0	0.0	0.0	0.1	0.0
2007	Notifications	7.0	4.9	2.2	2.8	5.3	7.2	7.2
	Hospitalisations	13.8	1.6	0.0	0.0	0.0	0.0	0.8
2008	Notifications	198.2	30.6	20.9	10.3	10.5	20.2	24.9
	Hospitalisations	184.1	3.9	0.0	0.0	0.0	0.4	0.8
2009	Notifications	240.7	53.1	58.4	31.2	17.4	31.2	30.8
	Hospitalisations	201.1	8.2	0.7	0.7	0.0	0.7	0.4
2010	Notifications	313.0	89.3	122.4	118.5	40.1	49.7	59.3
	Hospitalisations	239.2	7.2	1.4	1.3	0.0	1.2	2.9
2011	Notifications	610.9	285.9	520.3	474.1	71.7	108.4	116.8
2012	Notifications	375.7	284.0	247.9	263.1	63.7	104.5	144.5
Average ra	ate notifications	267.2	116.4	145.1	131.2	31.9	49.9	60.0
Average ra	ate hospitalisations	145.9	5.2	0.4	0.4	0.0	0.5	1.0

<sup>\*</sup> Hospitalisation data available through 2010.

E194 CDI Vol 38 No 3 2014

# FINDING THE 'WHO' IN WHOOPING COUGH: VACCINATED SIBLINGS ARE IMPORTANT PERTUSSIS SOURCES IN INFANTS 6 MONTHS OF AGE AND UNDER

Christina Bertilone, Tania Wallace, Linda A Selvey

#### Abstract

Objectives: To describe the epidemiology of pertussis, and to identify changes in the source of pertussis in infants 6 months of age and under, during the 2008–2012 epidemic in south metropolitan Perth.

Design and setting: Analysis of all pertussis cases notified to the South Metropolitan Population Health Unit and recorded on the Western Australian Notifiable Infectious Disease Database over the study period. Information on the source of pertussis was obtained from enhanced surveillance data.

Results: Notification rates were highest in the 5-9 years age group, followed by the 0-4 years and 10-14 years age groups. There was a significant increase in the proportion of known sources who were siblings from the early epidemic period of 2008-2010, compared with the peak epidemic period of 2011-2012 (14.3% versus 51.4%, p=0.002). The majority of sibling sources were fully vaccinated children aged 2 and 3 years.

Conclusions: The incidence of pertussis was highest in children aged 12 years and under in this epidemic. At its peak, siblings were the most important sources of pertussis in infants 6 months and younger, particularly fully vaccinated children aged 2 and 3 years. Waning immunity before the booster at 4 years may leave this age group susceptible to infection. Even if cocooning programs could achieve full vaccination coverage of parents and ensure all siblings were fully vaccinated according to national schedules, waning immunity in siblings could provide a means for ongoing transmission to infants. Recent evidence suggests that maternal antenatal vaccination would significantly reduce the risk of pertussis in infants 3 months of age and under. Commun Dis Intell 2014;38(3):E195-E200.

Keywords: pertussis, whooping cough, infants, source, vaccination, immunisation

#### Introduction

The incidence of pertussis (whooping cough) has risen both in Australia and internationally over

recent years, and large epidemics have occurred.<sup>1,2</sup> Increased clinician awareness and laboratory testing are likely to be partially responsible for the apparent increase in disease incidence.<sup>3</sup> However, the epidemiology of pertussis in Australia and the United States of America has also changed in recent times, with an increasing proportion of disease occurring in children.4-7 Possible reasons for this include the increasing use of less effective acellular vaccines<sup>8-10</sup> and increasing circulation of Bordetella pertussis strains deficient of vaccine antigen.11,12 Within vaccinated populations, the fewer whole cell vaccines received, the greater the risk of pertussis.<sup>8,10</sup> Additionally, immunity from acellular pertussis vaccination wanes more rapidly than that from whole cell vaccination. 13-15 Pertussis morbidity and mortality are greatest in infants under the age of 6 months, who are too young to have completed a primary vaccination course. The implications of these changes for the source of infant pertussis remain unclear.

Household contacts are the most likely sources of infant pertussis, but there is variation in the proportion of sources reported to be parents as opposed to siblings. A recently published Australian review on infant pertussis sources reported the source as a parent in 55% (range 39%–57%) and a sibling in 16%–43%. The proportion of sources that were siblings varied widely between studies, in comparison to the proportion that were parents, which were more consistent. The conclusion was that siblings may be more important sources of infant pertussis than previously realised. 16

A prolonged outbreak of pertussis occurred in Australia, including south metropolitan Perth, between 2008 and 2012. A cocooning strategy involving the vaccination of caregivers of newborns was implemented in Western Australia and ran for 2011 and 2012 in attempts to protect newborns during the outbreak. This strategy can only be effective if caregivers are the main source of pertussis in infants.

Over the study period, the South Metropolitan Population Health Unit (SMPHU) collected enhanced surveillance data for pertussis cases in children under 5 years of age. These data are not collected or reported at the national level so provide valuable additional information, particularly regarding source of infection, to that routinely collected for the National Notifiable Diseases Surveillance System. This study aimed to describe the epidemiology of the epidemic in south metropolitan Perth in relation to the source of infant pertussis, as well as any changes in the epidemiology and the source that occurred over the 5-year period.

#### **Methods**

The SMPHU is responsible for the follow up of notifiable diseases for the area covered by the South Metropolitan Health Service, which spans all of metropolitan Perth south of the Swan River and services approximately 37% of the Western Australian population.<sup>17</sup> Over the study period, the SMPHU collected enhanced surveillance data for pertussis cases in children under 5 years of age. The process involves a trained public health nurse interviewing the treating doctor and caregiver of the notified case, in order to obtain further information such as the likely source of infection and any high risk contacts. Enhanced surveillance defines a source of pertussis as a contact of the notified case who had either prolonged coughing illness or known pertussis infection, who was in contact with the notified case during the latter's incubation period (from 6 to 21 days prior to symptom onset). In the case of multiple possible sources, the source was assumed to be the individual who first became symptomatic, provided that the source's infectious period coincided with the notified case's incubation period.

Enhanced surveillance data for notified cases in infants 6 months of age and under were examined retrospectively, as well as pertussis notification data recorded on the Western Australian Notifiable Infectious Disease Database (WANIDD) for all age groups. All confirmed and probable cases meeting the case definition for pertussis were included if the optimal date of onset of pertussis occurred any time from 1 January 2008 to 31 December 2012, and residential postcode was within the SMPHU catchment area. The optimal date of onset refers to the earliest date recorded on WANIDD reflecting disease onset. In some situations, such as those where the caregiver of the notified case could not be contacted by telephone, enhanced surveillance data were not available. Notified cases and sources were defined as being fully vaccinated for age if on the optimal date of onset of illness they had received all pertussis vaccinations recommended by the Western Australian immunisation schedule for their age. This would potentially include vaccinations given within the 14 days preceding disease onset. The dates of vaccination for the

source were not available so any such cases would be misclassified as being fully vaccinated for age at disease onset. Notified cases from the 2008–2010 and 2011–2012 periods were compared because this distinction allowed comparison of the precocooning period with the cocooning period, and the early epidemic period with the peak epidemic period. Differences in age specific risk of infection as well as source of infant pertussis in the 2 periods were assessed.

Denominator data for notification rates were obtained from the Epidemiology Branch of the WA Department of Health. All analyses were performed in SPSS version 21. All comparisons were performed using chi-squared analyses or Fisher's exact test for categorical variables, and Mann-Whitney U testing for continuous variables. The study was approved by the Curtin University Human Research Ethics Committee (protocol approval SPH-16-2013). Ethics approval was not sought elsewhere, as this study formed part of the core business of the SMPHU.

#### Results

There were 3,611 cases of pertussis notified to the SMPHU from 2008 to 2012, with this period demonstrating a dramatic increase in notifications in comparison with previous years (Figure 1). Of these cases, 37.3% (n = 1348) occurred in children 12 years of age or under. At the peak of the epidemic in the December 2011 quarter, notification rates were markedly higher in children in age categories 14 years of age and under in comparison with the remainder of the population (Figure 1, Figure 2). The notification rate for the 5–9 years age group in the December 2011 quarter was 341.4 per 100,000, and 243.0 per 100,000 for the 10–14 years age group. Notification rates peaked in adults in this quarter also, but the amplitude of the peak was much less marked (56.0 per 100,000). Notification rates in children 4 years of age and under did not peak until the following quarter, at 206.8 per 100,000.

Of the 115 cases of pertussis in infants 6 months of age and under, enhanced surveillance data were available for 106 (92.2%). The optimal date of onset was the date of symptom onset for 111 of 115 cases, and the laboratory specimen date for the remaining four. There were no significant differences between those who had undergone enhanced surveillance and those who had not, comparing gender (p = 0.74), age (p = 0.56), ethnicity (p = 1.00) and hospitalisation status (p = 0.48).

The source was identified in 65 of 106 cases (61.3%). Two potential sources were identified for two of these cases, and one for the remaining 104 cases.

Figure 1: Notification rates of pertussis, south metropolitan Perth, 2008 to 2012, by quarter and age group

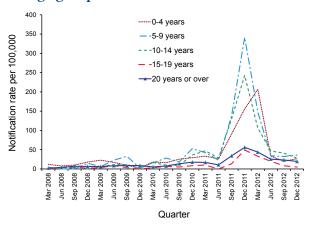
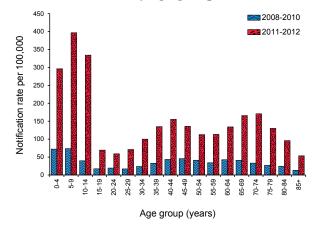


Figure 2: Notification rate of pertussis, south metropolitan Perth, 2008 to 2010 compared with 2011 to 2012, by age group



The proportion of sources whose diagnosis was confirmed with laboratory testing was unknown. Over the 5-year period, the source was a parent in 38.5% (n = 25) of cases and a sibling in 35.4% (n = 23) of cases. The most likely source of pertussis differed in the 2008–2010 period compared with the 2011–2012 period (Table). The proportion of parents as a source was lower in the 2011–2012 period (32.4%, n = 12 versus 46.4%, n = 13). However this difference was not statistically significant (p = 0.25). In contrast, the proportion of sources that were siblings was significantly higher in the 2011–2012 period (51.4%, n = 19 versus 14.3%, n = 4; p = 0.002).

During the 2011–2012 peak epidemic period, the ages of 14 of 19 sibling sources were known. Eight of these sources were aged from 2 to 4 years with five being fully vaccinated, one partially vaccinated, one unvaccinated, and one of unknown vaccination status. The true number of children in the 2–4 years age group may have been higher as the ages of 5 children were not recorded. Three sources were aged 6–11 years, and three were aged 12–19 years. Of all children in south metropolitan Perth diagnosed with pertussis in 2008–2012 and aged from 7 months to 4 years, 78.1% (n = 267) were fully vaccinated for age.

#### **Discussion**

Recent studies have shown an increasing incidence of pertussis in children but the implications of this for the source of infant pertussis have not been fully described. Identifying the source of pertussis in infants 6 months of age and under is crucial for the development of effective preventive strategies in this age group. However, the most likely source of infection will reflect local epidemiology, and if

Table: Source of pertussis in infants 6 months of age and under, south metropolitan Perth, 2008 to 2010 compared with 2011 to 2012

		2008-20 <sup>-</sup>	10		2011-20 <sup>-</sup>	12		Total	
	n	Known source %	Notified cases %	n	Known source %	Notified cases %	n	Known source %	Notified cases %
Parent	13	46.4	24.5	12	32.4	19.4	25	38.5	21.7
Sibling	4	14.3	7.5	19	51.4	30.6	23	35.4	20
Other household contact	3	10.7	5.7	2	5.4	3.2	5	7.6	4.3
Grand parent	3	10.7	5.7	3	8.1	4.8	6	9.2	5.2
Cousin	3	10.7	5.7	0	0	0	3	4.6	2.6
Other household contact	2	7.1	3.8	1	2.7	1.6	3	4.6	2.6
Total known source	28			37			65		
Notified cases with available enhanced surveillance data	45			61			106		
Notified cases 6 months of age and under	53			62			115		

the age specific risk of infection changes during epidemics, the source of pertussis in infants could vary at different points in the epidemic cycle. This study demonstrates changes in the source of infant pertussis corresponding with changing age specific risk of infection during an epidemic period.

Notification rates were highest in children in this epidemic, particularly at its peak in the 2011–2012 period. This correlated with a dramatic rise in the proportion of sibling sources. There are several possible explanations for the high notification rates in children. Recent studies suggest that acellular pertussis vaccine immunity wanes more rapidly than that of the whole cell pertussis vaccine.<sup>8,Î0,13–Ĭ5</sup> The vaccine effectiveness of the whole cell pertussis vaccine previously administered in Australia was estimated at 91% (95% CI 85.5%–94.4%) in infants aged 8-23 months, and 84.5% (95%) CI 78.3%–88.9%) in the 2–4 years age group.<sup>19</sup> In contrast, a recent Australian study reported the vaccine effectiveness of acellular vaccine to be 83.5% (95% CI 79.1%-87.8%) in infants aged 6–11 months, falling to 70.7% (95% CI 64.5%– 75.8%) in children aged 2 years, and 59.2% (95%) CI 51.0%–66.0%) in children aged 3 years.<sup>20</sup> In the whole cell pertussis vaccine effectiveness study, children had received 5 doses of pertussis vaccine by age 5 (2, 4, 6, 18 months and 4 years). In contrast, the acellular pertussis vaccine effectiveness for the children aged 2 and 3 years was calculated for children receiving 3 doses of vaccine, reflecting the current pertussis vaccination schedule of 2, 4, 6 months and 4 years.<sup>20</sup>

The high notification rates in children and the higher percentage of sibling sources could also be epidemic specific features, given the timing of this study. This is feasible as studies of contact patterns have shown high levels of assortative mixing in children.<sup>21</sup> Age specific infection risk and infant pertussis source types may be different in the inter-epidemic period. This would be congruent with the findings of this study, given that proportions of sources that were parents and siblings in the 2008–2010 period were comparable with those reported in previous literature. 16 Even if high incidence of pertussis in children and high proportions of siblings as sources are purely epidemic specific features, there are still implications for infant pertussis control measures during epidemics.

Cocooning programs are challenging to implement and there is no definitive evidence that they are successful in reducing the incidence of infant pertussis.<sup>22,23</sup> Parents remain susceptible to pertussis for 14 days following immunisation, due to the time taken to mount an immune response.<sup>24</sup> The earlier parental immunisation is performed post-natally, the better protected infants will be,

making hospital-based vaccination ideal. Barriers to this have been identified, including legal issues related to vaccinating fathers (who are not hospital patients), and the need to provide after-hours services.<sup>25</sup> In Western Australia in 2011, an estimated 60% of mothers and 41% of fathers of newborns had been administered government funded pertussis vaccine, although the timing of this vaccination post-natally is unknown (2012 data not available at the time of publication).<sup>26</sup> These rates were similar to coverage rates reported in Victoria for the duration of their state wide cocooning program, where it was found that of those eligible, 68% of mothers and 49% of fathers were vaccinated.<sup>22</sup> In metropolitan areas of Victoria, 6% of mothers and 10% of fathers were vaccinated in the maternity hospital, compared with 70% of mothers and 42% of fathers in rural areas, suggesting that (particularly in metropolitan areas) vaccination may not have been given early enough in the neonatal period.<sup>22</sup> In this study, although the proportion of sources that were parents was lower in the cocooning period (2011–2012) compared with the pre-cocooning period (2008–2010), this observation did not reach statistical significance. While this may be a real finding, there were insufficient numbers in this study to determine that. If the difference in the proportion of source cases that were parents in the 2 periods were real, cocooning may explain this reduction, but it is likely to be insufficient to explain the observed increase in the proportion of sibling sources.

The increasing proportion of sibling sources over time reflected the increasing proportion of pertussis notifications in children 12 years of age and under over the 2008–2012 epidemic. In the peak epidemic period, sibling sources of infection were most likely to be aged 2 or 3 years. This suggests that the impact of high notification rates was greatest in the youngest siblings, despite the greatest numbers of cases occurring in children aged 7–11 years. Possible reasons for this include that siblings tend to be close in age, and that younger children are generally less able to control respiratory secretions. The only other recent Australian study of infant pertussis sources had similar findings, demonstrating that siblings aged 3 and 4 years were particularly important sources of infant pertussis during the 2009 epidemic in New South Wales.<sup>27</sup> Dutch research published in 2010 speculated that the high proportion of infant pertussis sources that were siblings (41%) in their study may have been related to the introduction of acellular pertussis vaccine in the Netherlands, as well as prior use of a less effective whole cell vaccine.<sup>24</sup> In that study, the source was a sibling aged 1–4 years in 18% of cases (95% CI 12%–25%), a sibling aged 5-8 years in 15% of cases (95%) CI 9%–21%), and a sibling aged 9–13 years in

8% of cases (95% CI 4%–13%). The vaccination schedule for that population involved vaccination at 2, 3, 4 and 11 months, with a booster at 4 years introduced 5 years prior to the commencement of the study. There is a possibility that with the introduction of acellular pertussis vaccine, the interval between primary vaccination and booster doses in both the Dutch and Australian populations is now too long, resulting in waning immunity before the booster at 4 years. Even if all household contacts of newborns (including siblings) could be routinely fully vaccinated, the issue of breakthrough disease prior to the booster at 4 years would leave a certain proportion of siblings as possible infant pertussis sources, limiting the effectiveness of cocooning.

Vaccination in the 3rd trimester of pregnancy is an alternative measure for prevention of infant pertussis, with the benefit of placental transfer of maternal IgG to the infant. The vaccine effectiveness of the maternal antenatal vaccination program in the United Kingdom was estimated at 91% (84%–95%) CI) for infants aged 3 months or less.<sup>28</sup> Following the introduction of the program, significant reductions in infant pertussis mortality, numbers of confirmed cases and numbers of hospitalisations were reported.<sup>28</sup> Adverse event surveillance has not detected any significant complications of maternal vaccination to date,<sup>29</sup> but further investigation is required into the possibility of infant immune response blunting.<sup>28</sup> Neonatal vaccination is an alternative possible means of infant pertussis control but similar concerns exist regarding immune blunting, requiring further study.<sup>30</sup> More research is also required to determine whether these observed antibody responses translate into lower incidence of pertussis in infants.

This study is a retrospective review of the data collected as part of the routine surveillance of pertussis, meaning there are several limitations. The source of pertussis was unable to be identified in 38.7% (n = 41) of cases who underwent enhanced surveillance. Previously published Australian studies on the source of infant pertussis have been unable to identify a source in 31%<sup>27</sup> and 49%<sup>31</sup> respectively. This could be due to the source being an asymptomatic or mildly unwell household contact, or a contact from outside the household unknown to the notified case or caregiver undergoing interview. If previously vaccinated adults are more likely to experience mild or asymptomatic illness, the proportion of infant pertussis sources that were parents could be underestimated in studies relying on the recall of the notified case and epidemiologic linkage rather than laboratory testing. However, siblings were the most common source of infant pertussis in a recently published study, which performed laboratory testing on all household contacts in order to identify the source.<sup>24</sup>

Another reason for the higher proportion of siblings noted in the 2011–2012 period could be that as the epidemic progressed, clinician awareness of pertussis in younger children increased, with a concurrent increase in laboratory testing. If this were the case, previous reports of sibling sources of infant pertussis may have underestimated the true proportion of sources attributable to siblings. Regardless, there are still implications for infant pertussis prevention and control measures.

This study has shown that a rapid increase in notification rates in children at the peak of the 2008–2012 epidemic in south metropolitan Perth was accompanied by a significant increase in siblings as sources of pertussis in young infants. In the face of widespread vaccination with a less effective acellular pertussis vaccine, it seems likely that notification rates will remain high in children. Fully vaccinated siblings aged 2 and 3 years were the most important infant pertussis sources in the peak epidemic period of this study, suggesting that immunity may wane in this age group before the vaccine booster at 4 years. Even if it were possible to fully cocoon infants through a combination of parental vaccination and ensuring siblings were fully vaccinated, the possibility of transmission via breakthrough disease in siblings would persist. The risk of sibling transmission to infants would be significantly reduced through the addition of a pertussis vaccine booster at 18 months and maternal antenatal vaccination, for which evidence of effectiveness at preventing pertussis in infants 3 months of age or less is mounting.

#### **Acknowledgements**

The authors would like to acknowledge the work of the public health nurses at the South Metropolitan Population Health Unit during the study period, who collected the data analysed in this research.

#### **Author details**

Dr Christina Bertilone<sup>1</sup> Dr Tania Wallace<sup>2</sup> A/Prof Linda A Selvey<sup>3</sup>

- Public Health Registrar, South Metropolitan Population Health Unit, WA Department of Health, Fremantle, Western Australia
- Public Health Physician, South Metropolitan Population Health Unit, WA Department of Health, Fremantle, Western Australia
- 3. Director of Epidemiology and Biostatistics, School of Public Health, Curtin University, Perth, Western Australia

Corresponding author: Dr Christina Bertilone, South Metropolitan Population Health Unit, Western Australia Department of Health, PO Box 546, FREMANTLE WA 6160. Telephone: +61 8 9431 0200. Email: christina.bertilone@health.wa.gov.au

#### References

- NNDSS Annual Report Writing Group. Australia's notifiable disease status, 2011: Annual report of the National Notifiable Diseases Surveillance System. Commun Dis Intell 2013;37(4):E313–E393.
- Campbell P, McIntyre P, Quinn H, Hueston L, Gilbert GL, McVernon J. Increased population prevalence of low pertussis toxin antibody levels in young children preceding a record pertussis epidemic in Australia. PLoS One 2012;7(4):e35874.
- Kaczmarek MC, Valenti L, Kelly HA, Ware RS, Britt HC, Lambert SB. Sevenfold rise in likelihood of pertussis test requests in a stable set of Australian general practice encounters, 2000–2011. Med J Aust 2013;198(11):624– 628.
- Spokes PJ, Quinn HE, McAnulty JM. Review of the 2008–2009 pertussis epidemic in NSW: notifications and hospitalisations. N S W Public Health Bull 2010;21(7–8):167–173.
- Clarke MF, Rasiah K, Copland J, Watson M, Koehler AP, Dowling K, et al. The pertussis epidemic: informing strategies for prevention of severe disease. *Epidemiol Infect* 2013; 141(3):463–471.
- Gabutti G, Rota MC. Pertussis: a review of disease epidemiology worldwide and in Italy. Int J Environ Res Public Health 2012;9(12):4626–4638.
- Skoff TH, Cohn AC, Clark TA, Messonnier NE, Martin SW. Early impact of the US Tdap vaccination program on pertussis trends. Arch Pediatr Adolesc Med 2012;166(4):344.
- Sheridan SL, Ware RS, Grimwood K, Lambert SB. Number and order of whole cell pertussis vaccines in infancy and disease protection. [Erratum in JAMA 2012;308(14):1432.] JAMA 2012;308(5):454–456.
- Witt MA, Katz PH, Witt DJ. Unexpectedly limited durability of immunity following acellular pertussis vaccination in preadolescents in a North American outbreak. Clin Infect Dis 2012;54(12):1730–1735.
- Witt MA, Arias L, Katz PH, Truong ET, Witt DJ. Reduced risk of pertussis among persons ever vaccinated with whole cell pertussis vaccine compared to recipients of acellular pertussis vaccines in a large US cohort. Clin Infect Dis 2013;56(9):1248–1254.
- Lam C, Octavia S, Ricafort L, Sintchenko V, Gilbert GL, Wood N, et al. Rapid increase in pertactin-deficient Bordetella pertussis isolates, Australia. Emerg Infect Dis 2014;20(4):626–633.
- Octavia S, Sintchenko V, Gilbert GL, Lawrence A, Keil AD, Hogg G, et al. Newly emerging clones of Bordetella pertussis carrying prn2 and ptxP3 alleles implicated in Australian pertussis epidemic in 2008–2010. J Infect Dis 2012;205(8):1220–1224.
- Klein NP, Bartlett J, Rowhani-Rahbar A, Fireman B, Baxter R. Waning protection after fifth dose of acellular pertussis vaccine in children. N Engl J Med 2012;367(11):1012–1019.
- Misegades LK, Winter K, Harriman K, Talarico J, Messonnier NE, Clark TA et al. Association of childhood pertussis with receipt of 5 doses of pertussis vaccine by time since last vaccine dose, California, 2010. JAMA 2012;308(20):2126–2132.
- Sheridan SL, McCall BJ, Davis CA, Robson JM, Hull BP, Selvey CE, et al. Acellular pertussis vaccine effectiveness for children during the 2009–2010 pertussis epidemic in Queensland. Med J Aust 2014;200(6):334–338.

E200

- Wiley KE, Zuo Y, Macartney KK, McIntyre PB. Sources of pertussis infection in young infants: a review of key evidence informing targeting of the cocoon strategy. Vaccine 2013;31(4):618–625.
- 17. Government of Western Australia Department of Health. Population health statistics. [online] Accessed on 20 August 2013. Available from: http://www.public.health.wa.gov.au/3/1489/1/population\_health\_statistics.pm
- Baxter R, Bartlett J, Rowhani-Rahbar A, Fireman B, Klein NP. Effectiveness of pertussis vaccines for adolescents and adults: case-control study. BMJ 2013;347:f4249.
- 19. Torvaldsen S, Simpson JM, McIntyre PB. Effectiveness of pertussis vaccination in New South Wales, Australia, 1996–1998. Eur J Epidemiol 2003;18(1):63–69.
- Quinn HE, Snelling TL, Macartney KK, McIntyre PB. Duration of protection after first dose of acellular pertussis vaccine in infants. *Pediatrics* 2014;133(3):e513–E519.
- 21. Rohani P, Zhong X, King AA. Contact network structure explains the changing epidemiology of pertussis. *Science* 2010;330(6006):982–985.
- 22. Donnan EJ, Fielding JE, Rowe SL, Franklin LJ, Vally H. A cross sectional survey of attitudes, awareness and uptake of the parental pertussis booster vaccine as part of a cocooning strategy, Victoria, Australia. BMC Public Health 2013;13(1):676.
- Rivero-Santana A, Cuéllar-Pompa L, Sánchez-Gómez LM, Perestelo-Pérez L, Serrano-Aguilar P. Effectiveness and cost-effectiveness of different immunization strategies against whooping cough to reduce child morbidity and mortality. Health Policy 2014;115(1):82–91.
- 24. de Greeff SC, Mooi FR, Westerhof A, Verbakel JM, Peeters MF, Heuvelman CJ, et al. Pertussis disease burden in the household: how to protect young infants. *Clin Infect Dis* 2010;50(10):1339–1345.
- 25. Healy CM, Rench MA, Baker CJ. Implementation of cocooning against pertussis in a high-risk population. *Clinical Infect Dis* 2011;52(2):157–162.
- Western Australia Department of Health, South Metropolitan Public Health Unit. Review of notifiable diseases in the South Metropolitan Area Health Service – 2011. Perth: Department of Health Western Australia; 2012
- Jardine A, Conaty SJ, Lowbridge C, Staff M, Vally H. Who gives pertussis to infants? Source of infection for laboratory confirmed cases less than 12 months of age during an epidemic, Sydney, 2009. Commun Dis Intell 2010;34(2):116–121.
- Amirthalingam G, Andrews N, Campbell H, Ribeiro S, Kara E, Donegan K, et al. Effectiveness of maternal pertussis vaccination in England: an observational study. *Lancet* 2014;doi:10.1016/S0140–6736(14)60686– 60683.
- Donegan K, King B, Bryan P. Safety of pertussis vaccination in pregnant women in UK: observational study. BMJ 2014;349:g4219.
- 30. Edwards KM, Berbers GA. Immune responses to pertussis vaccines and disease. *J Infect Dis* 2014;209(suppl 1):S10–S15.
- 31. Elliott E, McIntyre P, Ridley G, Morris A, Massie J, McEniery J et al. National study of infants hospitalized with pertussis in the acellular vaccine era. *Pediatr Infect Dis* J 2004;23(3):246–252.

# A STATE-WIDE INFORMATION CAMPAIGN DURING A PERTUSSIS EPIDEMIC IN NEW SOUTH WALES, 2010

Paula J Spokes, Alexander E Rosewell, Alex S Stephens, Jeremy M McAnulty

#### Abstract

Pertussis notifications increased dramatically in New South Wales in 2008, exceeding the rates in previous epidemic years. A state-wide, multifaceted campaign was launched in March 2009 to provide information about pertussis prevention. A population-based survey was conducted using a Computer Assisted Telephone Interviewing facility to assess the effectiveness of sending letters to households with young infants. A representative sample of 1,200 adults across all 8 area health services was interviewed between July 2009 and September 2010, with responses weighted against the state population. Many respondents (39.7%) reported receiving the letter, while fewer (29.6%) reported receiving an adult pertussis booster in the last year, mostly in response to General Practitioner advice (40.4%). Letter receipt was associated with the uptake of an adult pertussis booster in the past 12 months by respondents (OR 5.8; 95%CI 4.1, 8.2) and other adults in the household (OR 5.1; 95%CI 3.5, 7.5), as well as knowledge about pertussis prevention. Health providers remain crucial for vaccination decision making; however letters may have contributed to an increased uptake of pertussis booster vaccination and knowledge. Health authorities may consider mailing households in future pertussis epidemics as a component of a wider communication strategy. Commun Dis Intell 2014;38(3):E201-E207.

Keywords: pertussis, communication, letter, cocoon, evaluation, vaccination, knowledge, behaviour

#### Introduction

Pertussis is a significant cause of morbidity and hospitalisation in Australia, with cyclical transmission driving outbreaks every few years. The true burden of pertussis is underestimated. Rates of infection remain highest in infants, particularly those aged less than 6 months. Findings from recent population-based serosurveys are consistent with pertussis infection in the previous 12 months in a significant proportion of adolescents. Pertussis among the elderly is becoming increasingly recognised in settings with high coverage diphtheria-tetanus-pertussis acellular vaccine (DTPa) programs. While pertussis affects people of all ages, infants less than 2 months of age are at the greatest risk of severe disease, hospitalisation

and death.<sup>5</sup> Mothers, fathers, siblings and adult carers have all been shown to be important sources of pertussis infection for infants in the Australian setting.<sup>6–8</sup>

Pertussis notification rates increased dramatically in New South Wales in 2008, exceeding those of previous years, with infants less than 1 year of age most affected.<sup>2</sup> At this time, the national immunisation schedule provided DTPa for infants at 2, 4 and 6 months of age, with a booster dose at 4 years of age. Since 2003 a single booster dose of dTpa for adults has been recommended nationally for adults planning a pregnancy, working with children and health care workers.9 From March 2009, two time-limited outbreak control measures were implemented in New South Wales: 1) pertussis vaccination became funded for new parents and adult carers of infants aged less than 12 months, and 2) the 1st infant dose of DTPa was recommended to be brought forward to 6 weeks of age. Subsequently, it was estimated the latter of these measures would reduce the average notifications, hospitalisations, and hospital bed-days by 8%, 9%, and 12%, respectively, with larger reductions in an epidemic year.<sup>10</sup>

To provide timely information about the latest pertussis prevention and control recommendations to adult carers of young infants, a state-wide information campaign was launched in March 2009.<sup>11</sup> The campaign included direct communications to clinicians about pertussis and recommendations for immunisation of infants and their carers as well as the production of posters and leaflets for health care facilities and the public. Another key component of the campaign was a letter to households through the Australian Childhood Immunisation Register (ACIR) (on behalf of NSW Health). ACIR contains contact details for nearly all children registered with Medicare.12 Letters were mailed to households with children born in the previous 12 months in March 2009, and households with children born after this date were prospectively mailed each month following registration with Medicare at the time of birth. The campaign cost an average of about \$1 per letter sent. The letter recommended that parents and adult carers ensure their infant received its vaccinations on time and provided information that the 1st dose could be given as early as 6 weeks of age; that parents and other adults with regular close contact with their

CDI Vol 38 No 3 2014

child receive a free booster vaccination; and that parents keep the child away from people with a coughing illness.

#### **Aim**

To assess whether targeted letters to adult carers in households with young infants were associated with the increased uptake of adult pertussis vaccination or recall of pertussis prevention messages.

#### **Methods**

#### Study design

A pertussis questionnaire was included in the New South Wales population based household survey, continuously conducted by the Health Survey Program to monitor the health of the New South Wales population using computer assisted telephone interviewing (CATI).<sup>13</sup> The questionnaire collected self-reported information on parents' knowledge of pertussis and related behaviours. Telephone interviews were conducted by the NSW Health CATI survey facility from July 2009 to the end of September 2010. An amendment to the existing New South Wales Population Health Survey for the inclusion of the pertussis questionnaire was granted by the New South Wales Population and Health Services Research Ethics Committee in June 2009.

#### Survey sample

The study population was all New South Wales residents with a private telephone, living in households with at least 1 child less than 3 years of age. The CATI program uses addresses from the electronic telephone white pages assigned to each of the 8 NSW Health Services Areas by geo-coding. Households were contacted using list assisted random digit dialling. One person from the household was randomly selected to participate in the survey, using age order, having firstly identified the position of the household reporter. Respondents aged less than 16 years were interviewed via a parent or carer selected as a proxy respondent.<sup>13</sup> Up to 7 calls were made to establish initial contact with a household, and 5 calls were made in order to contact a selected respondent.

#### Sample size calculation

We estimated the baseline prevalence of adults receiving a pertussis booster vaccination in the previous 12 months to be 11%, increasing to 20% among the 50% who reported receiving the intervention. The sample size required (power = 80%, alpha = 0.05, design effect=2) to detect this difference in the uptake of pertussis booster vaccination

between those who reported receiving a letter and those who did not was at least 548 in each group. The sample size requirement, including a 10% buffer, was estimated at 1,200.

#### Questionnaire development

The pertussis questionnaire module gathered information to assess the impact of the letter on the self-reported recall of knowledge and behaviours relating to pertussis prevention, including vaccination uptake, as well as barriers to adoption of the recommendations. Participants were asked about the recent pertussis vaccination history of adult responders as well as other adults in the household. Adults who reported receiving pertussis vaccination within the previous 12 months were asked about the main prompt for this. The vaccination status of children in the household as well as the recall of individual pertussis prevention measures was also collected. Information on demographics and household characteristics were taken from other modules within the NSW Health Survey. Responses for households who reported receiving a letter (intervention group) were compared to those who did not report receiving a letter. Response categories were divided into indicators of interest, don't knows and those who refused were removed.

#### **Participation rates**

Response and cooperation rates for the NSW Health survey were calculated in line with established guidelines. <sup>14</sup> In brief, the minimum response rate was calculated as the number of complete interviews divided by the number of interviews (complete plus partial) plus the number of non-interviews (refusal and break-off plus non-contacts plus others) plus all cases of unknown eligibility (unknown if housing unit, plus unknown, other). The minimum cooperation rate was the number of complete interviews divided by the number of interviews (complete plus partial) plus the number of non-interviews that involve the identification of and contact with an eligible respondent (refusal and break-off plus other).

#### Statistical analysis

The survey data were weighted to adjust for probability of selection and for differing non-response rates among males and females and different age groups, the number of household members, number of residential telephone connections and the sampling fraction in each health area. Further information about the weighting process is provided elsewhere.<sup>13</sup> Design based analysis was undertaken to account for features of the sample design and provide approximately unbiased estimates and standard errors. Data were manipulated

and analysed using SAS version 9.3. The association between the reported receipt of letter status and knowledge and behavioural outcomes were examined using SAS procedures SURVEYFREQ and SURVEYLOGISTIC to analyse the data and calculate prevalence and odds ratio estimates and 95 per cent confidence intervals.

#### Results

The NSW Health survey response rate during the period the pertussis survey was conducted was 42.2%, and the cooperation rate was 62.6%. Refusal rates and contact rates for the overall survey during the study period were 25.3% and 79.0% respectively. There were 1,200 participants across all 8 areas health services: 424 households in 2009 and 776 in 2010. The sex, remoteness, Aboriginality, English as a second language and number of children less than 3 years of age in the household of respondents did not differ by reported receipt of letter status. There was a significant dif-

ference by reported receipt of letter status in the proportion of households that had private health insurance (Table 1), but not for household income or socioeconomic disadvantage quintile.

Overall, less than half (39.7%) the respondents reported receiving a letter, while 29.6% of all respondents reported receiving a pertussis booster in the last year, mostly in response to general practitioner (GP) advice (40.4%) but also in response to the letter (10.0%) as the primary prompts. Of the adults that did not receive a booster, about half (48.3%) were not aware of the recommendation, while less (11.1%) decided not to be vaccinated. The majority of respondents (82.9%) recalled one or more key messages about how to protect babies from pertussis. The strength of association between reported receipt of letters and pertussis adult boosters was modestly higher in males. There was an increasing trend of reported letter receipt over time, and with an increasing number of children in the household.

Table 1: NSW Health Survey participants: socio-demographic characteristics by reported letter receipt, 2009 to 2010

	Variable		Letter %	No letter %	OR %	(95%CI)
	Sex	Female	55.3	54.6	0.97	(0.71, 1.22)
		Male	44.7	45.4	0.97	(0.71, 1.32)
	Remoteness	Major cities	63.4	63.9	1.29	(0.46, 3.63)
		Inner regional	27.3	23.8	1.11	(0.39, 3.20)
		Outer regional	8.4	11.7	1.77	(0.59, 5.25)
		Remote & very remote	0.0	0.0	Reference	category
	Language other than	Yes	18.3	22.0	0.79	(0.53, 1.10)
	English spoken at home	No	81.7	78.0	0.79	(0.53, 1.19)
	Socio economic	1st least disadvantaged	19.1	16.8	0.84	(0.51, 1.39)
	disadvantage quintile	2nd	22.2	23.2	1.00	(0.62, 1.61)
		3rd	21.9	20.4	0.89	(0.55, 1.43)
Socio		4th	16.9	18.7	1.06	(0.63, 1.78)
demographic characteristics		5th most disadvantaged	20.0	20.9	Reference	category
	Not Aboriginal or Torres	Yes	2.3	4.2	0.56	(0.24, 1.20)
	Strait Island Origin	No	97.7	95.8	0.56	(0.24, 1.29)
	Private Health Insurance*	Yes	60.6	50.3	1.52	(1,11, 2.06)
		No	39.4	49.7	1.52	(1,11, 2.06)
	Household income	More than \$80,000	47.0	42.9	1.30	(0.57, 2.98)
		\$60,000-\$80,000	17.0	13.0	0.73	(0.34, 1.56)
		\$40,000-\$60,000	13.6	13.0	0.59	(0.28, 1.25)
		\$20,000-\$40,000	9.1	15.4	0.70	(0.35, 1.40)
		\$10,000-\$20,000	4.7	6.1	Reference	category
	Number of children under	1	82.0	82.5	0.96	(0 GE 1 42)
	3 in household	2 & 3	18.0	17.5	0.96	(0.65, 1.43)

 <sup>\*</sup> Significant difference between groups, p<0.05</li>

Adult participants who reported receipt of a letter had higher odds of reporting uptake of pertussis vaccination in the last year (OR 5.8; 95%CI 4.1, 8.2), and reporting other adults in the house were vaccinated for pertussis in the last year (OR 5.1; 95%CI 3.5, 7.5) (Table 2). Participants who reported receipt of a letter also had higher odds of recalling knowledge of various personal prevention measures, including: get the baby vaccinated (OR 1.6; 95%CI 1.1, 2.2), check that siblings are vaccinated (OR 2.4; 95%CI 1.5, 3.8), other adults should be vaccinated (OR 1.9; 95%CI 1.3, 2.7) and to keep the baby away from coughing people (OR 1.6; 95%CI 1.1, 2.1) (Table 3). There was no difference in reported household pertussis incidence and reports of receiving a letter.

#### **Discussion**

State-wide population level communication can positively impact on knowledge and attitudes to the use of personal protective measures among the general public and among health professionals during multi-faceted campaigns.<sup>15,16</sup> In our study, reported receipt of the information letter was positively associated with knowledge of the recommendations to ensure: 1) adults in the household are vaccinated; 2) siblings and others are vaccinated; 3) the baby is vaccinated; and 4) the baby is kept away from coughing people.

During the 2009 influenza pandemic, adoption of non-pharmaceutical protective measures was associated with perceptions of disease severity, risk of acquisition, outbreak duration, public trust

Table 2: NSW Health Survey participants: personal protective behaviours by reported letter receipt, 2009 to 2010

	Variable	Letter %	No letter %	OR %	(95%CI)		
	Reported had adult whooping cough	Yes	52.9	16.2	5.83	(4.14, 8.21)	
	booster in past 12 months*	No	47.1	83.8	5.65		
	Reported other children up to date with	Yes	94.1	95.5	0.76	(0.20, 4.07)	
Personal protective	vaccinations	No	5.9	4.5	0.76	(0.29, 1.97)	
behaviours	Reported other adults had pertussis	Yes	42.4	12.6	5.09	(2.47.7.45)	
	booster vaccine in last 12 months*	No	57.6	87.4	5.09	(3.47, 7.45)	
	Reported all children <3 years up to	Yes	94.6	93.7	1.18	(0.61.2.20)	
	date with vaccinations	No	5.4	6.3	1.10	(0.61, 2.29)	

<sup>\*</sup> Significant difference between groups, p<0.05

Table 3: NSW Health Survey participants: pertussis knowledge of personal protective measures by reported letter receipt, 2009 to 2010

	Variable	Letter %	No letter %	OR %	(95%CI)		
	Know to get the baby vaccinated*	Yes	71.6	61.3	1.60	(1.14, 2.23)	
		No	28.4	38.7	1.00	(1.14, 2.23)	
	Know to get the baby's first vaccine	Yes	7.2	5.0	1.46	(0.80, 2.69	
	at 6 weeks	No	92.8	95.0	1.40	(0.00, 2.09	
	Know to get all scheduled vaccines	Yes	8.0	7.1	1.13	(0.65, 1.96)	
Knowledge of personal protective	on time	No	92.0	92.9	1.10		
measures	Know to check that siblings and other	Yes	15.1	6.9	2.38	(1 40 2 02)	
	people in the house are vaccinated*	No	84.9	93.1	2.36	(1.48, 3.83)	
	Known that adults in the household	Yes	25.6	15.5	1.87	(1 20, 2 60)	
	should get vaccinated*	No	74.4	84.5	1.07	(1.30, 2.69)	
	Know to keep the baby away from	Yes	40.7	30.6	1.56	(1 14 2 14)	
	coughing people*	No	59.3	69.4	1.30	(1.14, 2.14)	

<sup>\*</sup> Significant difference between groups, p<0.05.

E204 CDI Vol 38 No 3 2014

in the health authorities, soundness of information, the public's ability to control their risk, and whether specific behaviours were effective in risk reduction.<sup>17</sup> Decision making regarding the use of pharmaceutical interventions such as vaccination is similarly complex and driven by multiple factors, including the emotional and experiential.<sup>18</sup> However, the use of vaccination reminder messages has demonstrated their effectiveness in influencing decision making to improve immunisation coverage across a variety of settings, modes (text, letter, postcard and phone), age groups and vaccines, 19 but not in some rural settings.<sup>20</sup> Reminders may be particularly effective at prompting vaccination when parents are not familiar with changes in vaccine recommendations<sup>21</sup> such as occurred during the New South Wales pertussis outbreak. In our study, receipt of a letter in households with infants was associated with a 6-fold increase in the uptake of the pertussis booster by adult respondents (OR 5.8; 95%CI 4.1, 8.2) and other adults in the household (OR 5.1; 95%CI 3.5, 7.5). This stands to reason as letters: 1) were sent during an outbreak when risk perceptions may have been high; 2) were sent directly from trusted health authorities; and 3) provided information about the severity of disease in infants, and how to obtain effective, freely available interventions to enable recipients to control the risk within their household.

There were several limitations to the study. We could not establish causality with this study methodology and did not evaluate the GP or mass media component of the campaign, which may have been a source of confounding. We relied on self-reported vaccination status, creating the potential for misclassification bias. While previous CATI surveys measuring vaccination status have identified significant bias and the need for validation,<sup>22</sup> the NSW Health Survey program methodology has been validated for the collection of health information at the population level.<sup>23</sup> Households without a residential telephone were excluded from the survey, which may have decreased the proportion of younger people and females in the sample.<sup>24</sup> Respondents were asked about how many children aged less than 3 years were in the household. As such, we were not able to analyse the results by age. In addition, we did not measure the extent to which the letters contributed to decision making as the non-primary prompt. However, given that the repetition of messaging is crucial for behaviour change,<sup>25</sup> it is conceivable the letters may have contributed to vaccination decision making more broadly. The increasing trend of letter recall over time, and with an increasing number of children in the household may have been due to an increased likelihood of recall associated with being sent a

letter with each newborn infant during the mail out period and an increasing proportion of those eligible to receive a letter sampled over time.

Importantly, letter recall was not associated with parental knowledge that babies could get their first vaccination at 6 weeks of age. It is conceivable this message may have held less relevance for the proportion of mothers who would have received the letter retrospectively, after their baby would have already received the 1st dose in their primary course of pertussis vaccinations, but this would not be an explanation for the parents who were sent a letter prospectively. In comparison, getting other household members vaccinated, including adults who were more likely to be unvaccinated, may have held greater relevance. Recent data from other sources indicates that the continued provision of information to parents to bring forward the first vaccination for pertussis has increased the proportion of infants vaccinated at 6 weeks.

Outbreak communication is most effective when standardised messages are disseminated through multiple channels. While some media-only communication interventions designed to alter public behaviour directly have had little impact on behaviour, when media communication is used in combination with a community component, significant changes in behaviour have been reported.<sup>27</sup> While we did not evaluate the impact of NSW Health's complementary communication strategy through mass media and with general practitioners, previous studies have demonstrated that communication disseminated directly to GPs, who are known to influence parental vaccination decision making,<sup>28</sup> prepared them to effectively respond to disease outbreaks<sup>29</sup> and increased their awareness and knowledge of health issues.<sup>30</sup> Previous studies have found GPs to be the key health information provider and decision influencer for families. In our study, 10.0% of adults reported receiving a pertussis booster in response to the letters, while 40.4% reported it was following GP advice (as the primary prompt). Direct communication to GPs during the outbreak is likely to have contributed to parental vaccination decision making, complementing the direct communication to parents of young infants and newborns.

While targeted reminder letters to parents have been previously used to prompt vaccination of children, their use at a population level in an outbreak scenario to provide prevention and control messages to new parents was a novel approach. This targeted messaging, in combination with broader public and clinician communications, was associated with a 6-fold increase in pertussis boosters by adult respondents in the study. Given that recent evidence has shown that vaccinating mothers prior

to giving birth (pre-conception or third trimester) is the most effective indirect way to protect infants from pertussis,<sup>31</sup> it will be important to consider similar targeted mechanisms to provide pertussis prevention information to women who are planning, or approaching the third trimester of pregnancy. The potential for broader social media strategies to complement such targeted approaches should be further explored.

#### Conclusion

Health providers remain crucial for vaccination decision making. However, direct correspondence with households may have contributed to increased uptake of pertussis booster vaccination and knowledge, as a component of a wider communication strategy. Many respondents reported receiving a letter and some reported it as the main prompt to get vaccinated. Health authorities may consider mailing households in future pertussis epidemics as a component of the wider communication strategy.

#### **Acknowledgements**

We wish to thank the NSW in-house computer assisted telephone interview facility for the data collection support they provided during this evaluation, as well as the technical support provided by Margo Barr.

#### **Author details**

Paula J Spokes,<sup>1</sup> Manager, Surveillance Alexander E Rosewell,<sup>1</sup> Epidemiologist, vaccine-preventable diseases Alex S Stephens,<sup>2</sup> Trainee biostatistician Jeremy M McAnulty,<sup>1</sup> Director

- Communicable Diseases Branch, Health Protection, NSW Ministry of Health, North Sydney, New South Wales
- NSW Biostatistical Officers Training Program, NSW Ministry of Health, Sydney, New South Wales

Corresponding author: MrAlexander Rosewell, Epidemiologist, vaccine-preventable diseases, Communicable Diseases Branch, Health Protection, NSW Ministry of Health, Locked Bag 961, NORTH SYDNEY NSW 2059. Telephone: +61 2 9391 9675. Facsimile: +61 2 9391 9189. Email: arosw@doh.health.nsw.gov.au

#### References

- Chiu C, Dey A, Wang H, Menzies R, Deeks S, Mahajan D, et al. Vaccine preventable diseases in Australia, 2005 to 2007. Commun Dis Intell 2010;34 (Supp):S1–S167.
- Spokes PJ, Quinn HE, McAnulty JM. Review of the 2008–2009 pertussis epidemic in NSW: notifications and hospitalisations. N S W Public Health Bull 2010;21(7–8):167–173.
- Wood N, McIntyre P. Pertussis: review of epidemiology, diagnosis, management and prevention. Paediatr Respir Rev 2008;9(3):201–211; quiz 211–212.

- Liu BC, McIntyre P, Kaldor JM, Quinn HE, Ridda I, Banks E. Pertussis in older adults: prospective study of risk factors and morbidity. Clin Infect Dis 2012;55(11):1450–1456.
- Georgousakis M, Quinn H, Wang H, Snelling T, Macartney K, McIntyre PB. Pertussis deaths in Australia—what has changed? Proceedings of the 13th National Immunisation Conference; 19–21 June 2012; Darwin, Australia.
- Chuk L-MR, Lambert SB, May ML, Beard FH, Sloots TP, Selvey CE, et al. Pertussis in infants: how to protect the vulnerable? Commun Dis Intell 2008;32(4):449–456.
- Elliott E, McIntyre P, Ridley G, Morris A, Massie J, McEniery J, et al. National study of infants hospitalized with pertussis in the acellular vaccine era. Pediatr Infect Dis J 2004;23(3):246–252.
- Jardine A, Conaty SJ, Lowbridge C, Staff M, Vally H. Who gives pertussis to infants? Source of infection for laboratory confirmed cases less than 12 months of age during an epidemic, Sydney, 2009. Commun Dis Intell 2010;34(2):116–121.
- Australian Government Department of Health and Ageing, National Health and Medical Research Council. The Australian Immunisation Handbook 9th Edition. Canberra: Australian Government Department of Health and Ageing. 2008. Accessed on 29 May 2012. Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/handbook-home
- Foxwell AR, McIntyre P, Quinn H, Roper K, Clements MS. Severe pertussis in infants: estimated impact of first vaccine dose at 6 versus 8 weeks in Australia. Pediatr Infect Dis J 2011;30(2):161–163.
- Della Bosca J. Media release: Whooping Cough vaccine not just for kids. North Sydney: NSW Ministry of Health. 2009.
- 12. Hull BP, McIntyre PB, Heath TC, Sayer GP. Measuring immunisation coverage in Australia. A review of the Australian Childhood Immunisation Register. *Aust Fam Physician* 1999;28(1):55–60.
- Barr M, Baker D, Gorringe M, Fritsche L. NSW Population Health Survey: Description of Methods. North Sydney: NSW Ministry of Health. Accessed on 2 July 2012. Available from: http://www.health.nsw.gov.au/surveys/ other/Documents/health survey methods.pdf
- 14. The American Association for Public Opinion Research. Standard Definitions: Final Dispositions of Case Codes and Outcome Rates for Surveys 7th edn. Deerfield, IL: The American Association for Public Opinion Research. 2011. Accessed 2 July 2012. Available from: http://www.aapor.org/AM/Template.cfm?Section=Standard\_Definitions2&Template=/CM/ContentDisplay. cfm&ContentID=3156
- 15. Buchbinder R. Population based intervention to change back pain beliefs and disability: three part evaluation. *BMJ* 2001;322(7301):1516–1520.
- 16. Sly DF, Heald GR, Ray S. The Florida "truth" antitobacco media evaluation: design, first year results, and implications for planning future state media evaluations. *Tob Control* 2001;3;10(1):9–15.
- 17. Rubin GJ, Amlot R, Page L, Wessely S. Public perceptions, anxiety, and behaviour change in relation to the swine flu outbreak: cross sectional telephone survey. BMJ 2009;339:b2651–b2651.
- Helms C, Leask J, Robbins SC, Chow MYK, McIntyre P. Implementation of mandatory immunisation of healthcare workers: observations from New South Wales, Australia. Vaccine 2011;29(16):2895–2901.

- Jacobson Vann JC, Szilagyi P. Patient reminder and patient recall systems to improve immunization rates. Cochrane Database Syst Rev 2005;(3):CD003941.
- Evaluation of Vaccination Recall Letter System for Medicaid-Enrolled Children Aged 19–23 Months — Montana, 2011. MMWR Morb Mortal Wkly Rep 2012;61(40);811–815.
- 21. Stockwell MS, Kharbanda EO, Martinez RA, Lara M, Vawdrey D, Natarajan K, et al. Text4Health: impact of text message reminder-recalls for pediatric and adolescent immunizations. *Am J Public Health* 2012;102(2):e15–e21.
- Andrews RM. Assessment of vaccine coverage following the introduction of a publicly funded pneumococcal vaccine program for the elderly in Victoria, Australia. Vaccine 2005;23(21):2756–2761.
- Shenson D, Dimartino D, Bolen J, Campbell M, Lu PJ, Singleton JA. Validation of self-reported pneumococcal vaccination in behavioral risk factor surveillance surveys: experience from the sickness prevention achieved through regional collaboration (SPARC) program. Vaccine 2005;23(8):1015–1020.
- 24. Pennay DW, Bishop N. Profiling the "mobile phone only" population: a study of Australians with a mobile phone and no landline telephone. Melbourne: The Social Research Centre Pty Ltd. 2009. Accessed on 2 July 2012. Available from: http://www.docstoc.com/docs/49351161/PROFILING-THE-MOBILE-PHONE-ONLY-POPULATION-A-study-of

- Ross-Degnan D. Changing behavior to maintain a healthy home. Pediatr Infect Dis J 2000;19(10 Suppl):S117–S119.
- World Health Organization. WHO outbreak communication guidelines WHO/CDS/2005.28. Geneva, Switzerland: World Health Organization. 2005. Accessed on 3 July 2012. Available from: http://www.who.int/csr/resources/publications/WHO\_CDS\_2005\_28/en/index.html
- 27. Redman S, Spencer EA, Sanson-Fisher RW. The role of mass media in changing health-related behaviour: a critical appraisal of two models. *Health Promot Int* 1990;5(1):85–101.
- Bartlett MJ, Burgess MA, McIntyre PB, Heath TC. Parent and general practitioner preferences for infant immunisation. Reactogenicity or multiple injections? Aust Fam Physician 1999;28(Suppl 1):S22–S27.
- Rosewell A, Patel M, Viney K, Marich A, Lawrence GL. Impact of faxed health alerts on the preparedness of general practitioners during communicable disease outbreaks. Commun Dis Intell 2010;34(1):23–28.
- Mathew M, Goldstein AO, Kramer KD, Ripley-Moffitt C, Mage C. Evaluation of a Direct Mailing Campaign to Increase Physician Awareness and Utilization of a Quitline Fax Referral Service. J Health Commun 2010;15(8):840–845.
- 31. Quinn H, Habig A, Snelling T, Chiu C, Spokes P, McIntyre P. Parental tdap boosters and infant pertussis: a case-control study. *Pediatrics* 2014;134(4):713–720.

# Annual report

# IMMUNISATION COVERAGE, 2012

Brynley P Hull, Aditi Dey, Rob I Menzies, Julia M Brotherton, Peter B McIntyre

#### Abstract

This, the 6th annual immunisation coverage report, documents trends during 2012 for a range of standard measures derived from Australian Childhood Immunisation Register (ACIR) data, and National Human Papillomavirus (HPV) Vaccination Program Register data. These include coverage at standard age milestones and for individual vaccines included on the National Immunisation Program (NIP) and coverage in adolescents and adults. The proportion of Australian children 'fully vaccinated' at 12, 24 and 60 months of age was 91.7%, 92.5% and 91.2%, respectively. For vaccines available on the NIP but not assessed during 2012 for 'fully vaccinated' status or for eligibility for incentive payments (rotavirus and pneumococcal at 12 months and meningococcal C and varicella at 24 months) coverage varied. Although pneumococcal vaccine had similar coverage at 12 months to other vaccines, coverage was lower for rotavirus at 12 months (83.6%) and varicella at 24 months (84.4%). Although 'fully vaccinated' coverage at 12 months of age was lower among Indigenous children than non-Indigenous children in all jurisdictions, the extent of the difference varied, reaching a 15 percentage point differential in South Australia but only a 0.4 percentage point differential in the Northern Territory. Overall, Indigenous coverage at 24 months of age exceeded that at 12 months of age nationally and for all jurisdictions, but as receipt of varicella vaccine at 18 months is excluded from calculations, this represents delayed immunisation, with some contribution from immunisation incentives. The 'fully vaccinated' coverage estimates for vaccinations due by 60 months of age for Indigenous children exceeded 90% at 91% in 2012. Unlike in 2011, at 60 months of age, there was no dramatic variation in coverage between Indigenous and non-Indigenous children for individual jurisdictions. As previously documented, vaccines recommended for Indigenous children only, hepatitis A and pneumococcal vaccine, had suboptimal coverage at 60.1% and 73.1%, respectively, although there was a considerable improvement in coverage from 2011, 57.7% and 68.2% respectively. On-time receipt (before 49 months of age) of vaccines by Indigenous children at the 60-month milestone age improved substantially between 2011 (19%) and 2012 (38%) but the disparity in on-time vaccination between Indigenous and non-Indigenous children worsened at the 60-month age milestone from 2011 (from 1.8 to 5.4 percentage points) and remained the same

for the 12 and 24-month age milestones. By late 2012, the percentage of children who received the 1st dose of DTPa vaccine dose at less than 8 weeks of age was greater than 50% in all but 1 jurisdiction and greater than 70% for New South Wales, the Australian Capital Territory and Tasmania. Further, by late 2012, the percentage of children who received the 4th dose of DTPa vaccine dose at less than 4 years of age was greater than 30% in 3 jurisdictions. The percentage of children whose parents officially objected to vaccination in Australia was 1.7% and this figure varied by jurisdiction. However, there is a further 2.1% of children whose parents don't officially object but whose children have no vaccines recorded on the ACIR. Coverage data for the 3rd dose of HPV from the national HPV register in the school catch up program was similar to 2011 at 71% but was substantially lower for the catch up program for females outside school (44%-69%), although this was an improvement from 2011. Commun Dis Intell 2014;38(3):E208-E231.

Keywords: immunisation coverage, immunisation delay, vaccine objection, human papilloma virus vaccine coverage

#### Introduction

This is the 6th annual immunisation coverage report, with the first 4 reports beginning in 2007.<sup>1-4</sup> It consolidates regular reports produced by the National Centre for Immunisation Research and Surveillance<sup>5-19</sup> using Australian Childhood Immunisation Register (ACIR) data and highlights important trends and significant issues over the preceding 12 months. It follows the format of the previous reports, providing a detailed summary for 2012 that includes vaccination coverage at standard milestone ages, coverage for vaccines not included in standard coverage assessments, timeliness of vaccination, coverage for Indigenous children and data for small geographic areas on the prevalence of vaccine objectors. This report also includes data from previously published sources on adolescents who are not on the ACIR. Readers are referred to the first report for a more detailed explanation of the background to this series of annual reports and the range of analyses presented.<sup>1</sup> This report uses the longstanding international practice of reporting at key milestone ages, to measure coverage against national targets and to track trends over time. Table 1 shows the Australian National Immunisation Program Schedule for 2012.

High levels of reporting to the Australian Childhood Immunisation Register are maintained by a system of incentive payments for immunisation providers and carers. These have been discussed in detail elsewhere<sup>18</sup> and are highlighted in Table 2. However, changes to immunisation policy, the incentive payment system and changes to the 'fully immunised' coverage algorithms may have an impact on reported vaccination coverage; some recent changes are highlighted in the Box and are also referred to in this report.

#### **Methods**

# The Australian Childhood Immunisation Register

The ACIR was established on 1 January 1996, by incorporating demographic data from Medicare on all enrolled children under the age of 7 years.<sup>5</sup> Participation in the ACIR is opt-out so it constitutes a nearly complete population register, as approximately 99% of children are registered with Medicare by 12 months of age.<sup>5</sup> Children not enrolled in Medicare can also be added to the ACIR

via a supplementary number. Since 2001, immunisations given overseas may be recorded if a provider endorses their validity. Data are transferred to the ACIR when a recognised immunisation provider supplies details of an eligible immunisation either automatically from medical practice software or through the Internet using the Medicare Australia website or by submitting paper encounter forms. The existence of medical contraindications and conscientious objection to immunisation is also recorded on the ACIR. All vaccination records for a child remain on the register indefinitely, but no new immunisation encounter records are added after the 7th birthday.

Immunisations recorded on the Register must be rendered in accordance with the guidelines issued by the National Health and Medical Research Council as stated in *The Australian Immunisation Handbook*.<sup>20</sup> Notifications falling outside these guidelines or duplicate notifications prompt an enquiry with the provider and, if their validity cannot be established, they are rejected.

Table 1: Australian National Immunisation Program Schedule for children, adolescents and adults in 2012

Age							Vaccine	*			
Childhood	vaccines	3									
Birth	НерВ										
2 months	НерВ	DTPa	Hib	IPV				13vPCV		Rotavirus	
4 months	НерВ	DTPa	Hib	IPV				13vPCV		Rotavirus	
6 months	НерВ	DTPa	Hib	IPV				13vPCV		Rotavirus <sup>†</sup>	
12 months			Hib		MMR		HepA <sup>‡</sup>		MenCCV		
18 months						VV	HepA <sup>‡§</sup>	13vPCV <sup>‡</sup>			
24 months							HepA <sup>§</sup>	13vPCV§			
48 months		DTPa		IPV	MMR						
Adolescent	vaccine	s									
12 years	НерВ∥	dTpa				VVII					HPV¶
15 years		dTpa								Influenza**,††,	23vPPV <sup>‡‡</sup>
Adult vacci	Adult vaccines										
≥ 50 years										Influenza**,††	23vPPV**
65 years										Influenza <sup>††</sup>	23vPPV

- \* A glossary of vaccine abbreviations is provided at the end of the report.
- † 3rd dose of rotavirus vaccine at 6 months is dependent on vaccine brand used in each state or territory.
- ‡ Aboriginal and Torres Strait Islander children in Western Australia and the Northern Territory.
- § Aboriginal and Torres Strait Islander children in Queensland and South Australia.
- || Catch up only.
- ¶ Females only.
- \*\* For Indigenous people only.
- †† Annual vaccination, all aged ≥6 months with medical risk factors, non-Aboriginal adults ≥65 years.
- ‡‡ Aboriginal adults with medical risk factors.

Table 2: History of payments provided by the Australian Government for immunisation in Australia, 1996 to 2013

Year introduced	Parent/carer	General practice
1996		ACIR notification payment
		\$6* per notification of completion of all vaccines at each age-based National Immunisation Program (NIP) schedule point
1998	Childcare Assistance Rebate and/or the Childcare	GPII Service Incentive Payment
	Cash Rebate <sup>†</sup> Amount varied depending on income, number and age of child(ren) as well as type and duration of care used.	\$18.50 per notification of completion of all vaccines at each of the aged-based NIP schedule points
	Maternity Immunisation Allowance (MIA)†	GPII Outcomes Bonus payment
	\$200 <sup>‡</sup> per 'fully vaccinated' child at 19 months of age if child had received all vaccines due up to 18 months of age	\$3.50 per 'fully immunised' whole-patient equivalent (WPE)§ if practice coverage rate for children <7 years of age is over 90%
2000	Childcare Rebate replaced previous Childcare Assistance and Cash rebates <sup>‡</sup>	
	Amount varied depending on income, number and age of child(ren) as well as type and duration of care used	
July 2004	Means testing removed from Maternity Immunisation Allowance <sup>¶</sup>	
July 2008		National HPV Register notification payment introduced
		\$6 per dose notified
October 2008		GPII Service Incentive Payment ceased
		–\$111 per child ≤7 years of age <sup>¶</sup>
January 2009	MIA split into 2 payments	
	\$129** paid in 2 instalments paid for 'fully immunised' children aged between 18–24 months and between 4–5 years	
June 2010		National HPV Register notification payment ceased <sup>1</sup>
		–\$6 per dose
July 2012	MIA ceased¶	GPII Outcomes Bonus payment ceased
	-\$258 per 'fully immunised' child	–\$3.50 per WPE aged ≤7 years <sup>¶</sup>
	Immunisation status of children aged 1, 2 and 5 years linked to the existing Family Tax Benefit Part A supplement <sup>†</sup>	
	Maximum of \$726 per age milestone, total \$2,178 per 'fully immunised' child	

In Queensland the ACIR notification payment is \$3 per notification, totalling \$18 if all vaccines at each of the 6 aged-based schedule points on the NIP are notified to the ACIR.

E210 CDI Vol 38 No 3 2014

<sup>†</sup> Means tested.

<sup>‡</sup> Amount in 1998. This had increased annually to \$233 by 2008 prior to being split into 2 payments totalling \$258 per child.

<sup>§</sup> The whole patient equivalent is a numerical representation of the proportion of care provided to a patient (aged ≤ 7 years) at a general practice during a 12 month reference period, compared with the overall care provided to that patient by all other general practices that child may have visited during the same period. The WPE is calculated from the Medical Benefits Schedule fee value of non-referred services.

<sup>||</sup> GPII outcomes bonus payment applied to general practices with 70%, 80% and 90% coverage levels in 1st year, then 80% and 90% coverage levels in 2nd year and 90% coverage levels from 3rd year onwards. In addition, general practices must have been registered with the GPII scheme and had ≥10 WPEs to qualify for the payment.

<sup>¶</sup> Denotes removal of a payment or payment condition.

<sup>\*\*</sup> Amount from 2009 to end June 2012.

## Box: Recent significant changes in immunisation policy, immunisation incentives and coverage calculation algorithms

July 2013 – The combined *Haemophilus influenzae* type b (Hib) and meningococcal serogroup C (MenC) vaccine, Menitorix® at 12 months of age, was added to the National Immunisation Program (NIP) schedule. This combination vaccine replaces the single dose of monovalent meningococcal C conjugate vaccine (MenCCV) and booster dose of monovalent Hib vaccine previously scheduled at 12 months of age.

A combination measles, mumps, rubella, varicella (MMRV) vaccine for children aged 18 months was added to the NIP. The MMRV vaccine replaces the separate measles, mumps, rubella (MMR) vaccine currently given at 4 years, and the varicella vaccine (for chickenpox) currently given at 18 months.

Pneumococcal conjugate vaccine, meningococcal C vaccine and varicella vaccine (as MMRV) included in the algorithms used to calculate 'fully vaccinated' coverage at 12 and 24 months.

The hepatitis A vaccination schedule for Indigenous children changed so that dose 1 is administered to Indigenous children at 12 months of age and dose 2 at 18 months of age in all 4 jurisdictions (the Northern Territory, Western Australian, Queensland and South Australia).

February 2013 – Males and females aged 12–13 years received the human papillomavirus (HPV) vaccine at school. Males aged 14–15 years also received the vaccine as part of a catch-up program until the end of the 2014 school year.

October 2012 – Prevenar 13® (13-valent pneumococcal conjugate vaccine, 13vPCV) replaced 23-valent pneumococcal polysaccharide vaccine (23vPPV) as a booster dose in Indigenous children.

July 2012 – Eligibility for the Family Tax Benefit Part A supplement required that children are assessed as

fully immunised, replacing the Maternity Immunisation Allowance.

July 2011 – Prevenar 13® (13vPCV) replaced Prevenar® (7-valent pneumococcal conjugate vaccine, 7vPCV) on the NIP for children at 2, 4 and 6 months of age in all states and territories except the Northern Territory (adopted 13vPCV from 1 October 2011). Also, a single supplementary dose of 13vPCV was funded for children aged 12–35 months who had not received a dose of 13vPCV or 10vPCV in their primary course. This 'supplementary dose' was available from October 2011 through to the end of September 2012. It was also available through the NIP to medically at-risk and Indigenous children on the 3+1 schedule, to be given at least 2 months after the booster dose.

December 2009 – Changes in the coverage calculation algorithms that tightened the rules regarding the receipt of *Haemophilus influenzae* type b and hepatitis B vaccines for children aged 12 and 24 months to lead to more accurate measures of *Haemophilus influenzae* type b and hepatitis B vaccine coverage in Australia.

October 2009 – The recommendation by the Australian Technical Advisory Group on Immunisation (ATAGI) that the 4th dose of diphtheria-tetanus-pertussis (acellular) (DTPa) vaccine can be given from 3½ years of age instead of the previously recommended 4 years of age.

March 2009 – The recommendation by NSW Health and ATAGI to parents and immunisation providers to consider bringing the 1st dose of DTPa forward to 6 weeks of age to provide earlier protection.

January 2009 – Changes to the overdue rules so that children were classified as overdue for pre-school boosters at 4 years and 1 month instead of the previous 5 years of age. This applied to parental and provider incentive payments.

### Measuring immunisation coverage using the Australian Childhood Immunisation Register

The cohort method has been used for calculating coverage at the population level (national and state or territory)<sup>21</sup> since the ACIR's inception. Cohort immunisation status is assessed at 12 months of age (for vaccines due at 6 months), 24 months of age (for vaccines due at 12 and 18 months), and 60 months of age (for vaccines due at 48 months). A minimum 3-month lag period is allowed for late notification of immunisations to the register, but only immunisations given on or before a child's 1st, 2nd or 5th respective birthdays are considered.<sup>21</sup> If a child's records indicate receipt of the last dose of a vaccine that requires more than 1 dose to complete the series, it is assumed that earlier vaccinations in the sequence have been given. This assumption has been shown to be valid.6,9

Three-month birth cohorts are used for time trend analyses, while 12-month wide cohorts are used for other analyses in this report such as for small area analysis. The 12-month wide cohorts used in this report are children born between 1 January and 31 December 2011 for the 12-month milestone age; children born between 1 January and 31 December 2010 for the 24-month milestone age; and children born between 1 January and 31 December 2007 for the 60-month milestone age.

The proportion of children designated as 'fully immunised' is calculated using the number of children completely immunised with the vaccines of interest by the designated age as the numerator, and the total number of Medicare-registered children in the age cohort as the denominator. 'Fully immunised' at 12 months of age was defined as a child having a record on the ACIR of a 3rd dose of the combined DTPa-hepB-IPV-Hib vac-

cine. 'Fully immunised' at 24 months of age was defined as a child having a record on the ACIR of a 3rd dose of the combined DTPa-hepB-IPV-Hib vaccine, a 4th dose of *Haemophilus influenzae* type b polysaccharide conjugated to tetanus toxoid vaccine, and a 1st dose of an measles-mumps-rubella (MMR)-containing vaccine. 'Fully immunised' at 60 months of age was defined as a child having a record on the ACIR of a 4th dose of combined DTPa-IPV vaccine, and a 2nd dose of an MMR-containing vaccine.

Immunisation coverage estimates were also calculated for individual National Immunisation Program (NIP) vaccines, including the 6 NIP vaccines not routinely reported in *Communicable Diseases Intelligence* and not part of 'fully immunised' calculations at 12 and 24 months of age. They were: a 3rd dose of pneumococcal polysaccharide vaccine and 2nd or 3rd dose of rotavirus vaccine by 12 months of age; a 1st dose of a varicella vaccine and a 1st dose of meningococcal C vaccine by 24 months of age; a 2nd dose of hepatitis A vaccine in Indigenous children by 30 or 36 months of age; and a dose of pneumococcal vaccine in Indigenous children by 30 months of age.

Changes to immunisation policy and changes to the 'fully immunised' coverage algorithms have had an impact on vaccination coverage presented in this report. In December 2007, the coverage algorithm for immunisations due at 48 months of age was changed to assess children at 60 months, not 72 months of age. In January 2009, changes were made to the overdue rules so that children were classified as overdue for pre-school boosters at 49 months instead of the previous 60 months of age. This applied to parental and provider incentive payments (Table 2). In March 2009, a recommendation was made by ATAGI to parents and immunisation providers to consider bringing the 1st dose of DTPa forward to 6 weeks of age to provide earlier protection against pertussis infection. From the September 2009 coverage assessment date onwards, changes were made in the coverage calculation algorithms that tightened the rules regarding receipt of Hib and hepatitis B vaccines for children aged 12 and 24 months of age. Prior to September 2009, if a child aged 12 months of age had a record on the ACIR of a 2nd or 3rd dose of any child Hib vaccine, he or she was considered 'fully vaccinated'. From September 2009, a child needed a record on the ACIR of a 3rd dose of any Hib vaccine or a 2nd dose of either PedvaxHIB or Comvax to be assessed as 'fully vaccinated'. Prior to September 2009, if a child aged 12 months of age had a record on the ACIR of a 2nd or 3rd dose of any hepatitis B vaccine, he or she was considered 'fully vaccinated'. From

September 2009, a child needed a record on the ACIR of a 3rd dose of any hepatitis B vaccine or a 2nd dose of Engerix B (paediatric), Comvax, or hepatitis B (paediatric) vaccine, to be assessed as 'fully vaccinated'. In October 2009, a recommendation was made by ATAGI that the 4th dose of DTPa containing vaccine can be given from 42 months of age instead of the previously recommended 48 months of age.

#### **Timeliness**

Age-appropriate immunisation was defined as receipt of a scheduled vaccine dose within 30 days of the recommended age. For example, a child who received the 1st dose of DTPa (due at 60 days of age) when he or she was more than 90 days of age was classified as late for that dose. For descriptive purposes, we categorised the outcome measure for each dose as either vaccine dose 'no delay', 'delay of between 1 and 6 months', or 'delay greater than 6 months'. Doses received 'too early' (greater than 30 days prior to when it was due), and doses never administered or recorded were excluded. Timeliness is measured in 12-month birth cohorts. Children included in the timeliness analysis were assessed at 1-3 years after doses were due, to allow time for late vaccinations to be recorded. Therefore, cohorts assessed for timeliness are not the same as those assessed for coverage milestones. The interval between doses was not evaluated. Timeliness of different vaccines and doses was also compared by plotting the cumulative percentage receiving each vaccine dose by age, with the proportion ever immunised set as 100%.

### Remoteness status

The area of residence of children was defined as 'Major Cities', 'Inner Regional', 'Outer Regional', 'Remote', and 'Very Remote' using the Accessibility/ Remoteness Index of Australia (ARIA),<sup>22</sup> which was developed by GISCA (now the Australian Population and Migration Research Centre) as a joint project with the Australian Department of Health in 1997/98. ARIA is an unambiguously geographical approach to defining remoteness. The most widely used ARIA product is ARIA+. ARIA+ is a continuous varying index with values ranging from 0 (high accessibility) to 15 (high remoteness), and is based on road distance measurements from over 12,000 populated localities to the nearest Service Centres in 5 size categories based on population size. For the timeliness analysis, we combined the 2 'Regional' categories into 1 category ('Inner and Outer Regional') and the 2 'Remote' categories into 1 category ('Remote and Very Remote').

### Indigenous status

Indigenous status on the ACIR is recorded as 'Indigenous', 'non-Indigenous' or 'unknown', as reported by the child's carer to Medicare, or by the immunisation provider to the ACIR. For this report we considered 2 categories of children: 'Indigenous' and 'non-Indigenous'. Children with unknown Indigenous status were presumed to be 'non-Indigenous'. Coverage estimate time trends are presented from 2004 only, due to poor rates of reporting Indigenous status prior to then.<sup>23</sup>

### Small area analysis

Analysis for small areas was calculated for the Australian Bureau of Statistics (ABS)-defined Statistical Area 3 (SA3) areas,<sup>24</sup> chosen because each is small enough to show differences within jurisdictions but not too small to render maps unreadable. Maps were created using version 12 of the MapInfo mapping software<sup>25</sup> and the ABS Census Boundary Information. As postcode is the only geographical indicator available from the ACIR, the ABS Postal Area to SA3 Concordance 2006 was used to match ACIR postcodes to SA3s, in order to create a SA3 field for each child in the relevant study cohort.<sup>26</sup>

### Vaccine objection

A child must be registered with Medicare before its parent(s) can lodge an official objection to immunisation. Parents can also object to immunisation but also refuse to lodge any official objection to the ACIR. We used the percentage of children with no vaccines recorded on the ACIR as a proxy measure of the number of these children. <sup>15</sup> Some children with no vaccines recorded on the ACIR will be officially registered as 'vaccine objectors' and some will not be registered as such. Registered vaccine objectors are eligible for parent incentive payments even if their children are unvaccinated. Proportions of children with: official vaccine objector status and no vaccines recorded on the ACIR; official vaccine objector status and at least 1 vaccine recorded on the ACIR and; no official vaccine objector status and no vaccines recorded on the ACIR were calculated from the cohort of children registered with Medicare, and born between 1 January 2010 and 31 December 2010. We chose this cohort when calculating proportions so that children under the age of 12 months were not included, to allow sufficient time for registration of objection and exclude infants late for vaccination.

### Human papillomavirus vaccine coverage

The National Human papillomavirus Vaccination Program is listed on the NIP

schedule, funded under the Immunise Australia Program, and delivered to females through an ongoing school-based program usually in the first year of secondary school. From 2013, males are also being offered Human papillomavirus (HPV) vaccination. From 2007 to 2009, there was a time-limited catch up program delivered through schools, general practices and community immunisation services for females up to age 26 years. Immunisation against HPV is achieved with a course of 3 doses of vaccine, over a 6-month period. Data on the National HPV Vaccination Program are provided by the National HPV Vaccination Program Register, which is operated by the VCS Inc. The purpose of this legislated register is to support the implementation of the vaccination program and to provide data for monitoring and evaluation.<sup>27</sup> States and territories provide data to the HPV register from their school based programs. Doses administered in general practice or by community providers outside of the school program are notified on a voluntary basis, with a notification payment provided only to general practitioners (GPs) during the 2007–2009 catch up program. The World Health Organization proposes using 15 years as the reference age for HPV vaccination coverage for the purposes of international comparison. Data on notified HPV coverage was obtained from National HPV Vaccination Program Register.<sup>28</sup>

### Coverage in the elderly

As there has not been an Adult Vaccination Survey<sup>29</sup> undertaken in Australia since 2009, no data is presented in this 2012 report on influenza and pneumococcal (23vPPV) vaccination coverage in the elderly. The next Adult Vaccination Survey is planned for 2015.

### Results

### Coverage estimates

### Overall

Coverage estimates in 2012 for full-year birth cohorts at the 3 milestone ages of 12 months, 24 months and 60 months are provided in Tables 3, 4 and 5. Nationally and for almost all jurisdictions, 'fully immunised' coverage and coverage for all individual vaccines (except rotavirus and varicella vaccines) for the 12-month, 24-month and 60-month age groups exceed the 1993 Immunise Australia Program's target of 90%. However, coverage at 60 months of age in Western Australia was below this target for all vaccines and 'fully immunised'.

Table 3: Percentage of children immunised by 12 months of age, 2012, by vaccine and state or territory\*

Vaccine <sup>†</sup>	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,377	97,849	3,792	62,005	19,776	6,221	72,624	32,468	300,112
Diphtheria, tetanus, pertussis (%)	93.4	91.8	92.4	92.5	92.3	93.1	92.9	91.0	92.2
Poliomyelitis (%)	93.4	91.7	92.4	92.4	92.3	93.0	92.8	90.9	92.2
Haemophilus influenzae type b (%)	93.3	91.6	92.4	92.4	92.2	92.8	92.7	90.8	92.0
Hepatitis B (%)	92.9	91.5	92.3	92.1	92.0	92.8	92.4	90.4	91.8
Fully immunised <sup>‡</sup> (%)	92.8	91.4	92.2	92.1	91.9	92.7	92.3	90.2	91.7
Rotavirus (%)	86.5	86.3	86.6	81.5	84.4	87.8	83.5	77.7	83.6
13vPCV (%)	92.7	90.5	91.7	91.6	91.6	90.6	91.3	89.2	90.9

For the birth cohort born in 2011.

Source: Australian Childhood Immunisation Register.

Table 4: Percentage of children immunised by 24 months of age, 2012, by vaccine and state or territory\*

Vaccine <sup>†</sup>	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,238	97,741	3,626	62,470	19,631	6,040	72,729	32,643	299,987
Diphtheria, tetanus, pertussis (%)	95.7	94.6	95.9	94.5	94.5	95.5	95.5	93.3	94.7
Poliomyelitis (%)	95.7	94.6	95.9	94.5	94.5	95.5	95.4	93.3	94.7
Haemophilus influenzae type b (%)	95.8	95.0	96.1	94.6	94.6	95.8	95.5	93.5	94.9
Hepatitis B (%)	95.0	94.2	95.5	94.0	94.2	95.4	95.0	92.5	94.2
Measles, mumps, rubella (%)	94.5	93.8	95.1	94.0	93.7	95.1	94.5	92.5	93.9
Fully immunised <sup>‡</sup> (%)	93.1	92.3	94.0	92.6	92.4	94.1	93.2	90.5	92.5
Varicella (%)	88.9	83.2	86.1	87.1	83.8	85.9	84.5	81.8	84.4
MenC (%)	94.3	93.4	94.5	93.4	93.4	94.9	94.0	91.5	93.4

<sup>\*</sup> For the birth cohort born in 2010.

Source: Australian Childhood Immunisation Register

Table 5: Percentage of children immunised by 60 months of age, 2012, by vaccine and state or territory\*

Vaccine	ACT	NSW	NT	Qld.	SA	Tas.	Vic.	WA	Australia
Total number of children	5,009	99,730	3,611	64,466	20,207	6,543	74,163	33,136	306,865
Diphtheria, tetanus, pertussis (%)	93.0	91.7	90.8	91.9	90.8	92.5	92.9	89.5	91.7
Poliomyelitis (%)	92.9	91.6	90.9	91.8	90.7	92.4	92.8	89.5	91.7
Measles, mumps, rubella(%)	92.6	91.5	90.8	91.8	90.5	92.7	92.7	89.4	91.6
Fully immunised (%)	92.3	91.2	90.5	91.4	90.2	92.2	92.4	88.9	91.2

<sup>\*</sup> For the birth cohort born in 2007.

Source: Australian Childhood Immunisation Register

E214 CDI Vol 38 No 3 2014

<sup>†</sup> A glossary of vaccine abbreviations is provided at the end of the report.

<sup>‡ &#</sup>x27;Fully immunised' – 3 doses of a diphtheria, tetanus and pertussis-containing vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP-containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of any other hepatitis B vaccines.

<sup>†</sup> A glossary of vaccine abbreviations is provided at the end of the report.

<sup>‡ &#</sup>x27;Fully immunised' – 3 or 4 doses of a DTPa-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP-containing Hib vaccine or 4 doses of any other Hib vaccine, 3 or 4 doses of Comvax hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of a measles, mumps and rubella-containing vaccine.

<sup>† &#</sup>x27;Fully immunised' – 4 or 5 doses of a diphtheria-tetanus-DTPa-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

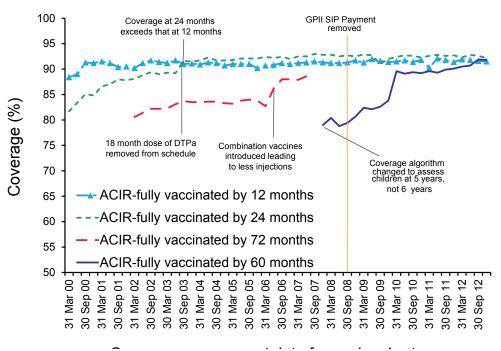
Figure 1 shows time trends in 'fully immunised' childhood vaccination coverage in Australia, assessed at 12 months, 24 months, and at 60 months of age, for 3-month cohorts born from 1 January 1999 to 31 December 2011. There is a clear trend of increasing vaccination coverage over time for all age groups assessed, with the 2 youngest age cohorts having the highest coverage for most of the period. The proportion 'fully immunised' at 12 months of age increased steadily from 75% for the 1st cohort in 1997 to 91.5% by 31 December 2012. At the 24-month milestone, 'fully immunised' coverage estimates also increased steadily from 64% for the 1st cohort to 92.2% by December 2012. 'Fully immunised' coverage estimates assessed at 72 months of age, for vaccines due at 48 months, were first reported in Communicable Diseases Intelligence in 2002, and increased steadily from 80.6% in early 2002 to 87.3% in late 2007, including a noticeable increase in June 2006, corresponding with the introduction of combination vaccines. From the beginning of 2008, when the assessment age was changed from 72 months to 60 months, 'fully immunised' coverage was substantially lower at 80.7% in December 2008, which was related to delayed immunisation. However, during 2009–2011, coverage for the 60-month age group rose substantially and kept increasing throughout 2012 to be comparable with coverage for the 2 younger age groups at 91.8%.

### Individual vaccines

DTPa and polio coverage at 12 months of age remained relatively stable from the latter part of 2001 to 2012 (Figure 2). Coverage for the Hib and hepatitis B vaccines at 12 months of age (prior to the change in algorithm to measure coverage that occurred in the latter half of 2009) are becoming similar to those for DTPa and polio in the last 2 cohorts of 2009 and on to 2012 and to more accurately reflect the situation (Figure 2). Coverage for 13-valent pneumococcal conjugate vaccine (13vPCV) rose steadily from below 90% in mid-2007 to be just below that for all other vaccines due at this age at around 91%, except for rotavirus vaccine. Rotavirus vaccine coverage rose steeply from late 2008 from below 70% to almost 84% in late 2011 and kept increasing throughout 2012 to be at 83.6%.

For most of the study period, hepatitis B coverage was higher than for all other vaccines at 24 months of age, at just under 95%, due to the different coverage algorithm described above (Figure 3). Coverage was lowest for MMR and Hib, the only vaccines that have a 12-month dose used in calculations, but in 2012, coverage is similar for all vaccines at just under 95%, except for varicella vaccine.

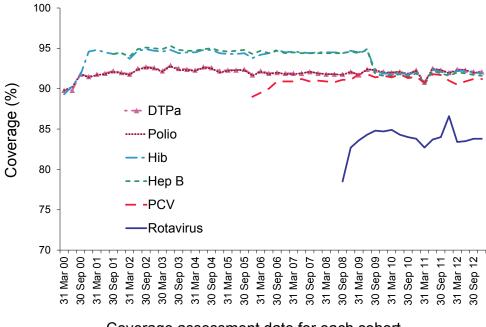




By 3-month birth cohorts born between 1 January 1999 and 31 December 2011. Coverage assessment date was 12 months after the last birth date of each cohort.

Source: Australian Childhood Immunisation Register.

Figure 2: Trends in vaccination coverage estimates for individual vaccines at 12 months of age (DTPa, IPV, Hib, HepB, 13vPCV, and rotavirus),\*,† Australia, 2000 to 2012

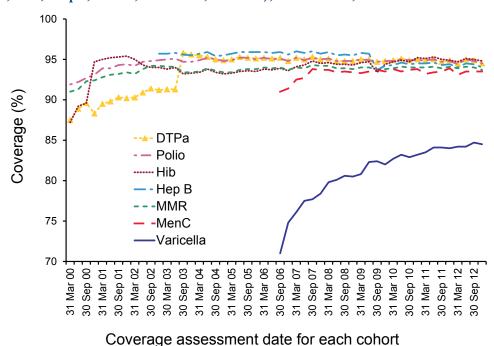


- Coverage assessment date for each cohort
- \* 3rd dose of DTPa, IPV, and 13vPCV, 2nd or 3rd dose of Hib, HepB, and rotavirus.
- † A glossary of vaccine abbreviations is provided at the end of the report.

By 3-month birth cohorts born between 1 January 1999 and 31 December 2011. Coverage assessment date was 12 months after the last birth date of each cohort.

Source: Australian Childhood Immunisation Register.

Figure 3: Trends in vaccination coverage estimates for individual vaccines at 24 months of age (DTPa, IPV, Hib, HepB, MMR, MenCCV, and VV),\*,† Australia, 2000 to 2012



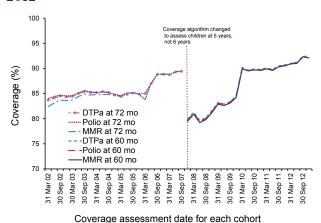
- \* 3rd or 4th dose of DTPa, 3rd dose of IPV, 3rd or 4th dose of Hib, 2nd or 3rd dose of HepB, 1 dose of MMR, MenCCV, and VV.
- † A glossary of vaccine abbreviations is provided at the end of the report.

By 3-month birth cohorts born between 1 January 1998 and 31 December 2010. Coverage assessment date was 24 months after the last birth date of each cohort.

Source: Australian Childhood Immunisation Register.

There was a marked increase in coverage for individual vaccines at 60 months of age following the change in the due/overdue rules in January 2009, with coverage increasing to levels similar to when coverage was assessed at 72 months of age (Figure 4). Coverage for all individual vaccines is at almost 92% in 2012, probably related, in part, to completed immunisation by 48 months of age being introduced in 2009 as a requirement for GP incentive payments.

Figure 4: Trends in vaccination coverage estimates for individual vaccines (DTPa, IPV, and MMR)\*.† at 60 months of age (72 months prior to December 2007), Australia, 2002 to 2012



- \* 4th dose of DTP and polio, 2nd dose of MMR.
- † A glossary of vaccine abbreviations is provided at the end of the report.

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

Source: Australian Childhood Immunisation Register.

### Coverage estimates for Indigenous children

Unlike in previous years, immunisation coverage is now only lower for Indigenous children than non-Indigenous for the 12 months age group, with little or no difference at 24 and 60 months of age (Table 6). The coverage differential between Indigenous and non-Indigenous children for individual vaccines varies, with coverage at 24 months of age for most vaccines being almost identical for both groups and greater among Indigenous children for Hib, MMR and meningococcal C vaccines.

The proportion of Indigenous children 'fully immunised' by 24 months of age remained consistently higher than at 12 months and 60 months of age until 2012 where coverage for the 60-month age group rose to levels comparable with that for the

24-month group (Figure 5). As for non-Indigenous children, coverage at 60 months of age increased following the change in due/overdue rules.

Although coverage at 12 months was lower among Indigenous children in all jurisdictions, the extent of the difference varied, reaching 15 and 11 percentage point differentials in South Australia and Western Australia, respectively, but only a 0.4 percentage point differential in the Northern Territory (Table 7). By age 24 months, the coverage disparity between Indigenous and non-Indigenous children ranged from 5.5 percentage points higher in the Northern Territory and 6 percentage points lower in South Australia (Table 7).

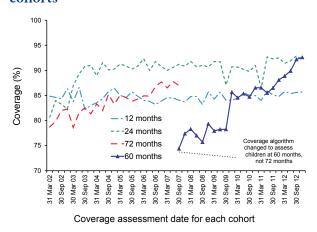
Table 6: Vaccination coverage estimates, 2012, by age, vaccine and Indigenous status

Vaccine*	Milestone age	Indigenous	Non- Indigenous				
DTPa	12 months†	85.7	92.5				
	24 months <sup>‡</sup>	94.2	94.7				
	60 months§	91.3	92.0				
IPV	12 months†	85.7	92.5				
	24 months‡	94.2	94.7				
	60 months§	91.3	91.9				
Hib	12 months†	85.7	92.4				
	24 months‡	94.8	94.9				
	60 months§	N/I	N/I				
НерВ	12 months†	85.6	92.1				
	24 months <sup>‡</sup>	94.2	94.2				
	60 months§	N/I	N/I				
MMR	12 months <sup>†</sup>	N/I	N/I				
	24 months <sup>‡</sup>	94.3	93.9				
	60 months§	91.7	91.8				
VV	12 months†	N/I	N/I				
	24 months‡	82.2	84.5				
	60 months§	N/I	N/I				
MenCCV	12 months <sup>†</sup>	N/I	N/I				
	24 months <sup>‡</sup>	93.8	93.4				
	60 months§	N/I	N/I				
13vPCV	12 months <sup>†</sup>	85.5	91.2				
	24 months‡	N/I	N/I				
	60 months§	N/I	N/I				
Rotavirus	12 months†	70.3	84.2				
	24 months <sup>‡</sup>	N/I	N/I				
	60 months§	N/I	N/I				

- A glossary of vaccine abbreviations is provided at the end of the report.
- † Birth cohort born 1 January 2011 31 December 2011
- ‡ Birth cohort born 1 January 2010– 31 December 2010
- § Birth cohort born 1 January 2007 31 December 2007
- N/I Not included in coverage estimates for that group.

At 60 months of age, there was variation between individual jurisdictions, ranging from coverage 5 percentage points lower for Indigenous children

Figure 5: Trends in 'fully immunised' vaccination coverage estimates for Indigenous children in Australia, 2002 to 2012, by age cohorts



in South Australia to 7 percentage points higher in the Northern Territory, compared with non-Indigenous children (Table 7).

Coverage for National Immunisation Program vaccines not routinely reported elsewhere

Pneumococcal conjugate vaccine and rotavirus vaccine

The 7-valent pneumococcal conjugate vaccine was first added to the NIP in January 2005 and was replaced in July 2011 by 13vPCV for all Australian children at 2, 4 and 6 months of age. Since coverage was first calculated for this vaccine in early 2006, it has remained high, with a slight increase from 89% to 91.2% (Figure 2). Coverage is greater than the 1993 Immunise Australia Program target of 90% in all jurisdictions except for Western Australia where it was 89.2% (Table 3).

Rotavirus vaccine was added to the NIP in July 2007, so coverage for 2 or 3 doses (depending on vaccine) at 12 months of age could be calculated only from the December 2008 quarter onwards. Rotavirus coverage was lower nationally

Table 7: Percentage of children fully immunised by 12 months, 24 months and 60 months of age, 2012, by Indigenous status and state or territory

	State or territory										
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.		
12 months – fully	immunise	d (%)*									
Indigenous	81.5	85.9	91.9	87.2	77.1	90.1	86.3	78.9	85.6		
Non-Indigenous	93.0	91.6	92.3	92.5	92.5	92.9	92.4	90.9	92.0		
12 months – fully immunised (incl rotavirus & pneumococcal polysaccharide vaccine) (%)†											
Indigenous	73.0	74.2	78.6	67.2	59.9	81.0	71.4	54.5	68.9		
Non-Indigenous	84.4	82.8	86.1	83.7	85.6	83.1	83.4	79.4	83.0		
24 months - fully	immunise	d (%)‡									
Indigenous	91.6	91.8	96.9	93.0	86.7	93.9	90.2	89.4	92.1		
Non-Indigenous	93.1	92.3	92.2	92.6	92.6	94.1	93.2	90.5	92.5		
24 months - fully	immunise	d (incl vari	cella & Men	C) (%) <sup>†</sup>							
Indigenous	84.6	77.6	89.3	83.1	72.0	80.5	77.1	75.2	80.1		
Non-Indigenous	86.7	81.2	82.0	85.8	82.7	84.7	82.7	79.8	82.6		
60 months - fully	immunise	d (%)§									
Indigenous	91.0	91.4	93.9	92.0	85.7	93.1	92.4	86.9	91.0		
Non-Indigenous	92.8	91.3	89.4	91.5	90.9	89.7	92.0	89.3	91.4		

<sup>\* &#</sup>x27;Fully immunised' – 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP-containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of any other hepatitis B vaccines.

E218 CDI Vol 38 No 3 2014

<sup>†</sup> A glossary of vaccine abbreviations is provided at the end of the report.

<sup>‡ &#</sup>x27;Fully immunised' – 3 or 4 doses of a DTPa-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP-containing Hib vaccine or 4 doses of any other Hib vaccine, 3 or 4 doses of Comvax hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine.

<sup>§ &#</sup>x27;Fully immunised' – 4 or 5 doses of a DTPa-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

(Figure 2), and had greater variation between jurisdictions compared with other vaccines given at 2, 4 and 6 months, which may be due to the strict upper age limits for this vaccine. Reported coverage in 2012 for 2 doses of Rotarix® or 3 doses of Rotateq® vaccine at 12 months of age varied from 87.8% and 86.6% in Tasmania and the Northern Territory respectively (both Rotarix®) to 77.7% in Western Australia (Rotateq®) (Table 3).

### Meningococcal C vaccine and varicella vaccine

Meningococcal C vaccine was added to the NIP in January 2003. Since coverage was first calculated for this vaccine in early 2006, it has remained at high levels with an increase over 2 years from 88% to almost 93.5% (Figure 3). There was little variation in 2012 by jurisdiction with all jurisdictions experiencing coverage levels greater than 91% and some, the Northern Territory and Tasmania, approaching 95% (Table 4).

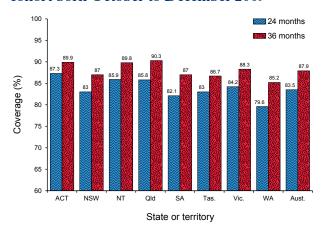
Varicella vaccine was added to the NIP in November 2005. Reported coverage for this vaccine has consistently been 10–15 percentage points lower than that for all the other vaccines assessed at the 24-month milestone, being 84.4% for the latest assessment in 2012 (Figure 3). This is probably partly due to the shorter time varicella has been on the NIP and the age of administration (18 months). The 18-month schedule point was historically associated with lower coverage when there was an 18-month pertussis booster prior to 2003. Between 2003 and 2005, there was a gap of over 2 years when no vaccine was administered at 18 months. Reported varicella vaccine coverage in 2012 also shows considerable variation by jurisdiction from 81.8% in Western Australia to 88.9% in the Australian Capital Territory (Table 4).

Due to the 18-month schedule point being historically associated with lower coverage; there being only a 6-month time period to catch up for varicella vaccination, and that there is the possibility that parents may delay the varicella vaccination, we compared varicella coverage estimates assessed at 24 months to estimates assessed at 36 months by jurisdiction (Figure 6). The differences in the expected increases in coverage varied by jurisdiction from a 5.6 percentage points increase in Western Australia to a 2.6 percentage points increase in the Australian Capital Territory. Three jurisdictions reach 90% varicella coverage or very close to 90% when the vaccine is assessed at 36 months.

Data are also available from the ACIR on the number of reports from GPs stating that children born since May 2004 have natural immunity to varicella and do not require varicella vaccination. Reports of natural immunity to varicella total greater than

27,810 since May 2004 (not shown), corresponding to approximately 1.1% of the cohort. It is likely that there is underreporting of presumed natural immunity by GPs but this is unlikely to fully account for lower varicella coverage.

Figure 6: A comparison of coverage of varicella vaccine assessed in 2012 at 24 months of age versus 36 months of age, by state or territory, cohort born October to December 2009



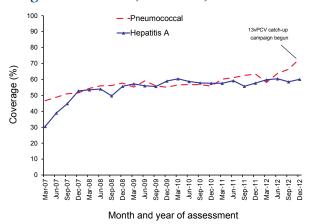
Hepatitis A vaccine and 23-valent pneumococcal polysaccharide vaccine

Hepatitis A vaccine was available in Australia prior to the development of the ACIR in 1996 and has been included on the NIP for Indigenous children in the Northern Territory, South Australia, Western Australia, and in Queensland since November 2005, but was used earlier than this in North Queensland. Since March 2007, coverage of 2 doses of hepatitis A vaccine for Indigenous children by 30 months of age in Western Australia and the Northern Territory and 36 months of age in Queensland and South Australia has increased from 58% to 60% in December 2012 (Figure 7). An additional 10% of children had received 1 dose of hepatitis A vaccine by 18 or 24 months of age, putting national coverage for at least 1 dose of hepatitis A vaccine for 2012 at 70.5% in Indigenous children compared to 60% for 2 doses (Table 8). There is a variation in reported hepatitis A vaccine coverage by jurisdiction, from a low of 32% in South Australia to a high of 85.4% in the Northern Territory (Table 8).

The pneumococcal vaccine has been recommended and funded as a booster at 18–24 months of age since 2001for Indigenous children in 4 jurisdictions (the Northern Territory, South Australia, Western Australia, and Queensland). Coverage has gradually increased from 47% in March 2007 to 73% in December 2012 (Figure 7). From 2011 to

2012, coverage increased by almost 10 percentage points. There is a large variation in pneumococcal vaccine coverage by jurisdiction from a low of 56.5% in South Australia to a high of 90.2% in the Northern Territory (Table 8).

Figure 7: Trends in coverage estimates for hepatitis A\* and pneumococcal<sup>†</sup> vaccines for Indigenous children, Australia, <sup>‡</sup> 2007 to 2012



- Two doses assessed at 30 months for Western Australia and the Northern Territory, 2 doses assessed at 36 months for Queensland and South Australia.
- † 18-month dose assessed at 30 months of age.
- Northern Territory, Queensland, South Australia and Western Australia only.

Table 8: Vaccination coverage\* for hepatitis A (Indigenous only) and pneumococcal (Indigenous only), 2012, by state or territory<sup>†</sup>

	Vaccine type <sup>‡</sup>								
State or territory	Hepatitis A§	Pneumococcal <sup>∥</sup>	13vPCV <sup>¶</sup>						
NT	85.4 (92.7)	90.2	87.3						
Qld	58.2 (64.2)	73.0	70.5						
SA	32.2 (50.8)	56.5	41.3						
WA	56.8 (75.5)	66.3	55.3						
Aust.	60.1 (70.5)	73.1	67.6						

- \* For the last 3-month cohorts assessable in 2012.
- † Northern Territory, Queensland, South Australia and Western Australia only.
- ‡ A glossary of vaccine abbreviations is provided at the end of the report.
- § Indigenous only: 2 doses by 30 months of age for Western Australia and the Northern Territory (1 dose by 18 months of age), 2 doses by 36 months of age for Queensland and South Australia (1 dose by 24 months of age).
- Indigenous only: 1 dose of 13vPCV (4th dose) or 23vPPV (1st dose) or 10vPCV (4th dose in the Northern Territory) by 36 months of age for Australia.
- ¶ Indigenous only: 1 dose of 13vPCV (4th dose) by 36 months of age for Australia.

### Hepatitis B birth dose

Since 1999, coverage for the birth dose of hepatitis B vaccine has slowly decreased from 14.9% to 3.3% in 2012 (data not shown). It's highly likely that these estimates are considerable underestimations of the true coverage figures with significant underreporting to the ACIR of the administration of the dose being the likely reason. A 2006 unpublished report found a minimum estimate for coverage of the birth dose of hepatitis B vaccine of 85%, based on provider-completed parent-held written records.<sup>30</sup>

# Recommendation to give 1st dose of DTPa from 6 weeks of age and the 4th dose of DTPa from 3.5 years of age

In response to a pertussis epidemic and to provide early protection to young infants, it was recommended by the ATAGI in March 2009, and promoted in that year during epidemics in New South Wales and Tasmania (later in other jurisdictions), that immunisation providers give the 1st dose of DTPa vaccine at 6 weeks of age instead of 8 weeks of age. Prior to this, very few children received the vaccine dose at less than 8 weeks of age, but for New South Wales, Tasmania and the Australian Capital Territory the percentage rose over the 3 years with more than 70% of children receiving the dose prior to 8 weeks of age in December 2012 (Figure 8). By late 2012, this percentage was greater than 50% in all but 1 jurisdiction. Another change in DTPa vaccine scheduling occurred in October 2009 when the pre-school booster dose of DTPa-IPV was scheduled at 3.5-4 years instead of 4 years of age. This change in schedule took longer to take effect with no jurisdiction giving the vaccine in great numbers at 3.5–4 years of age until November 2010 (Figure 9). As at December 2012, more than 30% of children in 3 jurisdictions were receiving the dose at 3.5–4 years of age, in South Australia and the 2 territories (Figure 9).

### Timeliness of immunisation

Timeliness has been examined for vaccines requiring both multiple doses (DTPa, 13vPCV and MMR) and a single dose (MenCCV) at 12 and 24 months of age.

As demonstrated in previous studies, the proportion with vaccination delay increased with older age (Figure 10). The greatest proportion with any delay was seen with the 2nd dose of MMR vaccine with 57% of doses given late and 9% given more than 6 months late. This analysis is for doses due in 2010 allowing up to 3 years for capturing delayed doses, as explained in the methods. These figures are very similar to those in the 2011 report.

Figure 8: The percentage of children who received their 1st dose of diphtheria-tetanus-pertussis acellular vaccine at age 6-< 8 weeks, January 2009 to December 2012 birth cohort, by jurisdiction and month of receipt

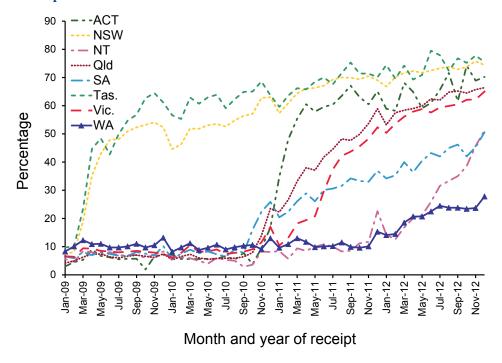


Figure 9: The percentage of children who received their 4th dose of diphtheria-tetanus-pertussis acellular vaccine at age 3 years and 6 months – < 4 years, September 2009 – December 2012 birth cohort, by jurisdiction and month of receipt

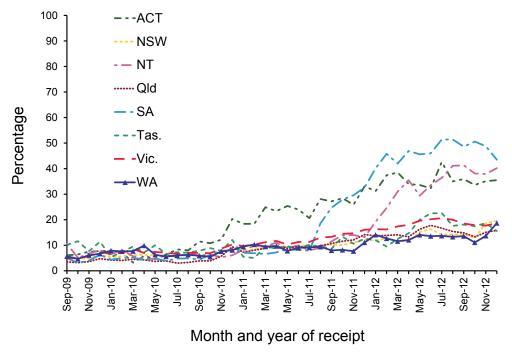
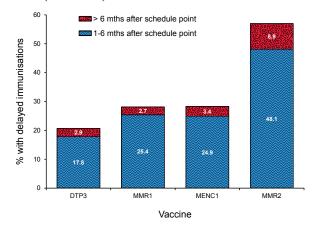


Figure 10: Vaccination delay for cohorts born in 2010 (DTPa3, MMR1, MenCCV1) and 2006 (MMR2\*)



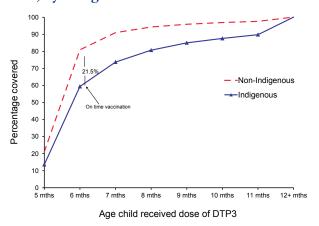
A glossary of vaccine abbreviations is provided at the end of the report.

For the 3rd dose of DTPa, there was greater delay for Indigenous children than non-Indigenous children, with a 21.5% differential in on-time vaccination at less than 7 months of age (Figure 11). The same pattern was found for timeliness of the 1st dose of MMR, but with a smaller differential of 13% (Figure 12). Although Indigenous children had only slightly lower coverage than non-Indigenous children by 24 months of age, they were more likely to have delayed vaccination and this differential in on-time vaccination between Indigenous and non-Indigenous continues to be high (the corresponding differentials for the 3rd dose of DTPa and 1st dose of MMR from the 2011 report were 22% and 13%, respectively).

Vaccination with the 3rd dose of DTPa and the 1st dose of MMR was delayed by more than 1 month for 26%–38% of Indigenous children and 17%–26% of non-Indigenous children (Table 9). The proportion with long delays (i.e. greater than 6 months) was 2-4 times higher in Indigenous children than in non-Indigenous children, with greater differences for the 3rd dose of DTPa and for Indigenous children living in Major Cities and in Inner and Outer Regional areas. Delays of 1-6 months were also more frequent for Indigenous children, although less marked, especially for the 1st dose of MMR. The proportion with short delays was greater among Indigenous children residing in Remote and Very Remote areas than in Major Cities for the 3rd dose of diphtheria-tetanus-pertussis vaccine (38% versus 26%), but not for the 1st dose of MMR.

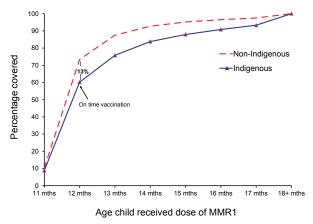
Vaccination delay for Indigenous children by jurisdiction was measured for the 3rd dose of

Figure 11: Timeliness\* of the 3rd dose of DTPa<sup>†</sup> vaccine (DTP3) for the cohort born in 2010, by Indigenous status



- \* Percentage covered = number of children who received vaccine dose at particular ages/the total number of children who received the vaccine dose.
- † A glossary of vaccine abbreviations is provided at the end of the report.

Figure 12: Timeliness\* of the 1st dose of MMR<sup>†</sup> vaccine (MMR1) for the cohort born in 2010, by Indigenous status



- Percentage covered = number of children who received vaccine dose at particular ages/the total number of children who received the vaccine dose
- † A glossary of vaccine abbreviations is provided at the end of the report.

13vPCV, with greater delays of 1–6 months in Western Australia (35.7%) and the Northern Territory (36.7%) (Figure 13). This is an improvement from 2011 where the corresponding figures were 40% and 37.1%, respectively. The proportion of Indigenous children with long delays increased substantially from 2011 in all jurisdictions with South Australia increasing from 6.2% to 9.8% and Western Australia increasing from 5.8% to 11.1%.

In contrast to the 3rd dose of DTPa and the 1st dose of MMR, analysis of timeliness of immunisation for a vaccine due at 48 months of age, the 2nd dose of MMR, showed a much smaller differential in delay in receiving this vaccine between non-Indigenous children and Indigenous children, with a 5.3% differential at less than 49 months of age (Figure 14). However, timeliness for both groups was considerably improved from the previous report in 2011.

Trends in timeliness of the 3rd dose of pneumococcal vaccine and the 1st dose of MMR vaccine by Indigenous status are provided in Figures 15 and 16. Timeliness for the 3rd dose of pneumococcal vaccine improved marginally over time for non-Indigenous children; however no improvements were seen for Indigenous children. In contrast, there were no noteworthy changes over time in timeliness of the 1st dose of MMR for either Indigenous or non-Indigenous children.

Figure 13: Vaccination delay for Indigenous children for the 3rd dose of pneumococcal conjugate vaccine for the cohort born in 2010, by jurisdiction

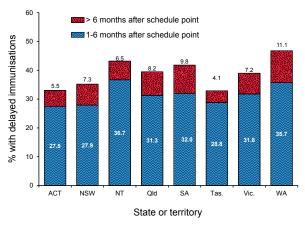
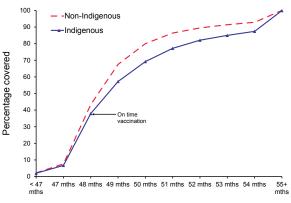


Figure 14: Timeliness\* of the 2nd dose of measles-mumps-rubella vaccine (MMR2) for the cohort born in 2006, by Indigenous status



Age child received dose of MMR2

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

Table 9: Vaccination delay, by Indigenous and remoteness status for the cohort of children born in 2010 and assessed in 2012, Australia

Vaccine dose	Indigenous status	Remoteness	1–6 months after schedule point %	> 6 months after schedule point %
Diphtheria-tetanus-	Indigenous	Major Cities	26.4	10.4
pertussis acellular3		Inner and Outer Regional	30.6	10.9
		Remote and Very Remote	38.1	8.5
	Non-Indigenous	Major Cities	17.0	2.4
		Inner and Outer Regional	17.9	2.9
		Remote and Very Remote	17.0	2.4
Measles-mumps-rubella1	Indigenous	Major Cities	33.7	7.8
		Inner and Outer Regional	34.7	6.3
		Remote and Very Remote	32.0	6.0
	Non-Indigenous	Major Cities	25.1	2.4
		Inner and Outer Regional	24.8	2.6
		Remote and Very Remote	26.3	2.2

<sup>\*</sup> A glossary of vaccine abbreviations is provided at the end of the report.

### Objection to vaccination

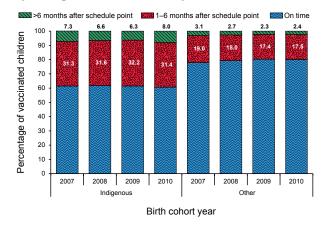
The percentage of vaccine objectors and children with no vaccines recorded on the ACIR for all jurisdictions and Australia is shown in Table 10. Of the 3 groups, the largest is those parents who don't officially object and also don't have any vaccines recorded on the ACIR. The total rate of objection for Australia was 3.7% and this varied by jurisdiction with a high of 4.8% in Western Australia and a low of 3.1% in the Australian Capital Territory.

The total proportions of children whose parents object to vaccination are presented by SA3 in Figure 17. The map shows pockets of high levels of objection within jurisdictions in 2012, particularly in coastal areas of northern and south-east Queensland, northern New South Wales, the Adelaide Hills in South Australia, and south-west Western Australia with rates of objection reaching over 10% in many areas. These areas have had consistently high levels of objection over many years.

# Figure 15: Timeliness of the 3rd dose of

pneumococcal conjugate vaccine, 2007 to 2010,

by Indigenous status and year of birth



### Human papillomavirus vaccine coverage

Vaccination coverage, as notified to the HPV register, for dose 3 of the HPV vaccine for females aged 15 years in 2012 is shown in Table 11. For Australia, 70.8% of females completed a full course of the vaccine. This is a slight drop from the 71.2% in 2011. Coverage varied by jurisdiction from a low of 62.6% in Tasmania to a high of 84.1% in the Northern Territory. Coverage in all age groups was higher for earlier doses, being as high as 82% for the 1st dose in females aged 14–15 years (Figure 18). Coverage was higher in the younger age groups than the older age groups, with only 44% of females aged 20-26 years fully vaccinated according to data notified to the Register. HPV coverage by Indigenous status is not available due to limitations in Indigenous status reporting on the HPV register.

Figure 16: Timeliness of the 1st dose of measles-mumps-rubella vaccine, 2007 to 2010, by Indigenous status and year of birth

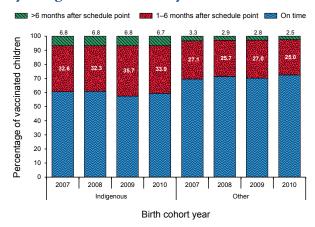


Table 10: The percentage of vaccine objectors and children with no vaccines recorded on the Australian Childhood Immunisation Register for the cohort born 1 January to 31 December 2010 and assessed in 2012, by state or territory

	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Number	5,241	97,981	3,626	62,779	19,675	6,038	73,059	32,756	301,155
Object* and no vaccines recorded	0.71	0.78	0.47	1.45	1.30	0.98	1.00	1.09	1.03
Object* and at least 1 vaccine recorded	0.67	0.50	0.66	0.79	0.82	0.53	0.60	0.85	0.65
No objection and no vaccines recorded	1.72	2.09	2.15	2.02	2.05	1.79	1.74	2.82	2.05
Total	3.10	3.37	3.28	4.26	4.17	3.30	3.34	4.76	3.73

<sup>\*</sup> Recorded on the Australian Childhood Immunisation Register as an objector through lodging an "Immunisation exemption conscientious objection" form to the Australian Government Department of Human Services.

Figure 17: Proportion of total objectors to immunisation for the cohort born January 2010 to December 2010, Australia, 2012

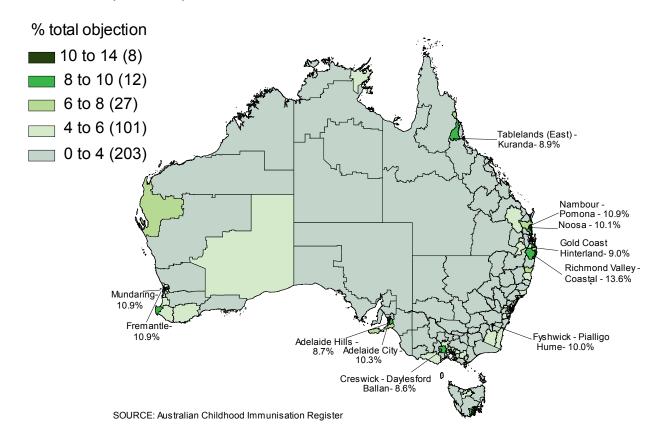


Table 11: Percentage of girls turning 15 years in 2011 and 2012 immunised for dose 3 of human papillomavirus vaccine, by state or territory

Ye	ar	ACT	NSW	NT	Qld.	SA	Tas.	Vic.	WA	Australia
			72.7	79.5	70.2	66.0	64	74.5	64.8	71.2
2012	2	74.0	70.7	84.1	68.7	70.1	62.6	73.6	69.9	70.8

Source: National Human Papillomavirus Vaccination Program Register, July 2013. Includes valid doses and too close doses for Clinically Complete Consumers.

### **Provider type**

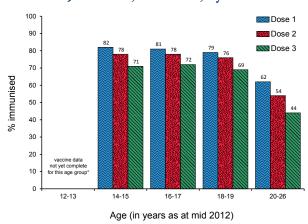
GPs administer the large majority of immunisations in Australia (Figure 19) with the proportion given by GPs having increased by almost 5% over the past 11 years (data not shown). Local government clinics also administer a substantial proportion of immunisations, especially in some jurisdictions. The only other category of provider administering major numbers of immunisations nationally is community health centres. Regional differences are marked, with immunisations almost entirely administered by GPs in some jurisdictions (New South Wales, Queensland, South Australia, Tasmania and Western

Australia), while in others a majority are given by local government (Victoria) and community health clinics (the Northern Territory).

## Mechanisms of reporting to the Australian Childhood Immunisation Register

The proportion of vaccinations on the ACIR lodged by the various reporting mechanisms available by jurisdiction is shown in Figure 20. The most common method of reporting for all but 1 jurisdiction was through Online claiming. Almost 80% of vaccinations given in Tasmania were reported through this method. In contrast, 85% of vaccinations given to children in the Northern Territory were reported using electronic data interchange via the Internet.

Figure 18: Human papillomavirus vaccination coverage for females vaccinated between April 2007 and June 2013, Australia, by dose number



In some states those aged 12–13 years in 2012 are not eligible for vaccination until 2013. Notification of 2013 doses to the register is in progress.

Source: National Human Papillomavirus Vaccination Program Register, July 2013.

Technical notes:

Includes valid doses and too close doses for Clinically Complete Consumers.

Population is Estimated Resident Population provided by the Australian Bureau of Statistics – Cat 3101.0 Australia Demographic Statistics, Tables 51 to 58: Estimated Resident Population by Single year of Age by State and Territory, published 2012.

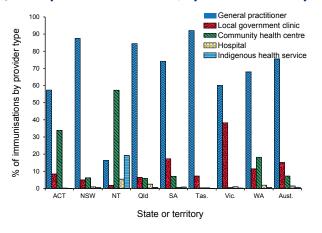
Age is age as at date of Estimated Resident Population

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

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

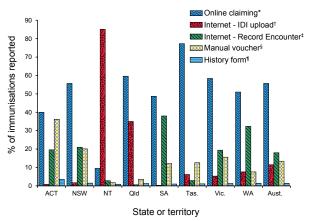
Published with permission from the Australian Government Department of Health.

Figure 19: Proportion of immunisations on the Australian Childhood Immunisation Register given by various provider types, January to December 2012, by state or territory



The only jurisdiction still reporting vaccinations using manual forms in considerable numbers was the Australian Capital Territory (36%).

Figure 20: Proportion of immunisations on the Australian Childhood Immunisation Register lodged by various reporting mechanisms from January to December 2012, by state or territory



- \* Online claiming Medicare Australia online claiming a software application that allows the transmission of Australian Childhood Immunisation Register data via the immunisation provider's desktop software.
- † IDI = approved immunisation providers can send immunisation details using the IDI upload facility through the Australian Childhood Immunisation Register secure area within Medicare Australia's website.
- Record encounter = approved immunisation providers can send immunisation details using the record encounter facility through the Australian Childhood Immunisation Register secure area within Medicare Australia's website.
- § Manual voucher by completing an Immunisation encounter form and sending it to Medicare Australia.
- ¶ History form to record a child's vaccination details that may be missing from the Immunisation Register. This form must be completed by a doctor or immunisation provider and sent to the Immunisation Register.

### **Discussion**

These data show that 1993 Immunise Australia Program 'fully vaccinated' coverage targets (90%) have been reached nationally for children 12, 24 and 60 months of age. This was also the case for most jurisdictions.

'Fully immunised' coverage at 24 months of age exceeds that at 12 months of age, and this is likely to be related to the longer time available for late vaccinations to be assessed, due to the exclusion of varicella vaccine at 18 months from the calculation of 'fully vaccinated', and also the absence of any other vaccines administered between those ages. There may also be an impact of immunisation incentives. National coverage for vaccines

due at 48 months of age continued to improve during 2012 with coverage greater than 91% for all 4 cohorts. This increase is due to improved timeliness of vaccination, and is probably related to the change to the overdue rules in January 2009, where children became overdue for their pre-school boosters at 49 months of age instead of the previous 60 months. This change had an impact on eligibility for child care benefits for parents and outcome payments for providers. It was accompanied by a letter from Medicare Australia advising parents of the change, and the follow-up of overdue children by local health authorities.

There is earlier evidence that immunisation incentives to providers positively impacted on coverage estimates.<sup>13</sup> However, the initial analyses in this report provide no evidence of a reduction in coverage associated with the removal of Service Incentive Payments in October 2008, while coverage at 60 months has increased following the due and overdue rules changes. More analysis is required however, to examine the impact of these changes in more detail.

A number of vaccines that are included in the NIP are not included when calculating 'fully immunised' status or in eligibility for incentive payments. Coverage estimates for 13vPCV and meningococcal C vaccines are comparable with estimates for vaccines that are included in 'fully vaccinated' calculations, but estimates for varicella and rotavirus are still substantially lower. During 2012, there was only a small change in national coverage for varicella from 2011 (from 83.9% to 84.4%) and national coverage for rotavirus vaccine decreased marginally by 0.2 of a percentage point from 2011 (from 83.8% to 83.6%). However, when we assessed varicella coverage at 36 months of age instead of 24 months we observed higher estimates across all jurisdictions ranging from 2.6 to 5.6 percentage points higher, suggesting many parents delay the administration of the varicella vaccine by more than 6 months. For rotavirus vaccines, strict upper age limits for administration may explain lower coverage when compared with other vaccines assessed at 12 months of age, whilst varicella is the only vaccine due at 18 months, and this milestone was historically problematic and lapsed for a 2 year period (2003–2005) when there were no vaccines scheduled. The implications also vary. In the case of rotavirus vaccine, coverage of 80% or greater has been associated with substantial herd immunity and decreases in rotavirus hospitalisations in Australia and elsewhere.31,32 In contrast, modelling studies suggest that low coverage with varicella vaccine may result in a shift of disease to older age groups with higher disease severity.<sup>33</sup> This will change from July 2013 with the inclusion

of 13vPCV, MenCCV, and varicella (as MMRV) in the algorithms used to calculate fully immunised coverage at 12 and 24 months of age.

Coverage for vaccines recommended for Indigenous children only (i.e. hepatitis A and a booster dose of pneumococcal vaccine) remained sub-optimal during 2012 but increased substantially from 2011 for the 23-valent pneumococcal polysaccharide vaccine (nationally, from 63% to 73%). This is likely due to the change in the pneumococcal vaccine used as a booster dose in Indigenous children from 23vPPV to 13vPCV in October 2012 and the 13vPCV catch up program in 2011 that continued until September 2012. The extent of underreporting to the ACIR for these vaccines is unknown but may be more than for 'universal' vaccines, given the lack of incentive payments for notification to the ACIR. However, lower coverage for vaccines targeted at Indigenous people has been a relatively consistent finding using a range of different methods for both children<sup>14</sup> and adults.<sup>34</sup> Both a lack of provider knowledge about the recommendations for high risk groups, and poor identification of Indigenous children by immunisation providers are likely to be important contributing factors. Differences in schedules between jurisdictions may also contribute. During 2012, coverage for both vaccines was much higher in the Northern Territory, which gives the vaccines at 6 months younger (Hepatitis A, 12 and 18 months, 23vPPV 18 months), than in South Australia and Queensland (18 and 24, and 24 months). However, coverage for both vaccines in 2012 for Queensland was similar to that in Western Australia even though the vaccines are given 6 months later in Queensland. The presence of other vaccines on the schedule at the same age may assist achieving higher coverage at 12 months and 18 months of age. Failure to receive a 2nd dose by 10% of children also contributed to the low coverage for hepatitis A vaccine. However, a protective antibody response after 1 dose is expected from a majority of children.<sup>35</sup>

Although coverage data reveal that most children eventually complete the scheduled vaccination series by the 24-month milestone, many still do not do so in a timely manner. On-time vaccination in 2012 as measured in this report for vaccines assessed at 12 and 24 months of age has improved only for the 3rd dose of pneumococcal vaccine assessed at 12 months of age. However, timeliness cannot be measured in the most recent cohort, as time must be allowed for late vaccination to be received. Poorer timeliness in Indigenous children has been noted previously in infants. Timeliness has continued to improve at 60 months of age for both Indigenous and non-Indigenous children. However, as coverage and timeliness of vaccines assessed at 60 months of age has improved,

the disparity in timeliness between Indigenous and non-Indigenous children has increased, as improvements in non-Indigenous children were not fully duplicated in Indigenous children. Delayed vaccination is a concern, especially for diseases where multiple vaccine doses are required for protection and the disease risk among young infants is significant (e.g. pertussis). Immunisation at the earliest appropriate age should be a public health goal for countries such as Australia where high levels of vaccine coverage at milestone ages have been achieved.

The ACIR has shown the rapid uptake of new vaccines and consistently high coverage for all vaccines, unlike some other developed countries.<sup>36,</sup> <sup>37</sup> In comparison with similar countries, reported coverage at 12 months of age is higher,<sup>37</sup> and, with almost 4% of children not vaccinated due to parental refusal, targeting of on-time vaccination is required to significantly improve the current levels of greater than 91% 'fully immunised' at 12 months of age. Areas of low coverage have been identified in many remote areas and areas containing higher proportions of vaccine objectors. Further vaccination coverage estimates in small areas has been provided by the National Health Performance Authority for children in 2011–12.<sup>38</sup>

Coverage data for HPV from the national HPV register reflect a successful school-based program with lower coverage for the catch-up program.<sup>39, 40</sup> Under-notification to the HPV register of doses administered in general practice and the community contributes to the apparently lower coverage in women currently aged over 20 years, with independent coverage estimates from population surveys in this age group suggesting undernotification of around 5%–15%.<sup>40, 41</sup> The 10%–18% drop in coverage between dose 1 and 3 may also reflect under-notification of doses missed in school and caught up in general practice but not notified to the register, as well as demonstrating the challenges in delivering a 3 dose vaccination course to adolescents.

Australia's HPV vaccination program remains the most broadly targeted program in the world, with no other country having provided a free catch up program up to the age of 26 years. The coverage achieved in the program has been sufficient to result in demonstrable decreases in HPV prevalence in young women, <sup>42</sup> genital warts <sup>43</sup> and cervical abnormalities. <sup>44</sup>

Unfortunately, coverage data are not available for Indigenous adolescents. For adults, data are only available from the Aboriginal and Torres Strait Islander Health Survey, last conducted in 2004–05.45

Data provided in this report reflect continuing successful delivery of the NIP in Australia, while identifying some areas for improvement. Coverage for varicella and rotavirus vaccines is below that for other vaccines. Timeliness of vaccination could be improved, particularly for Indigenous infants, and coverage for vaccines recommended only for Indigenous infants is lower than for other vaccines. From July 2013, varicella and other NIP vaccines (meningococcal C and pneumococcal conjugate vaccines) have been included in coverage assessments for 'fully immunised', and thereby in eligibility for provider and parent incentives.<sup>46</sup> It will be important to evaluate the impact of this change in coming years and given the encouraging improvements in timely coverage seen with the changes to reimbursement introduced in 2009 for the 48-month milestone, this promises to have a favourable impact. especially for varicella vaccine where high coverage is crucial to long-term outcomes of the program.

### **Author details**

Brynley Hull<sup>1</sup> Aditi Dey<sup>1</sup> Rob Menzies<sup>1</sup> Julia Brotherton<sup>2</sup> Peter McIntyre<sup>1</sup>

- National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, The Children's Hospital at Westmead and University of Sydney, Westmead, New South Wales
- 2. Victorian Cytology Service Registries: (National HPV Vaccination Program Register and Victorian Cervical Cytology Registry), Carlton South, Victoria

Corresponding author: Mr Brynley Hull, National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, The Children's Hospital at Westmead and University of Sydney, Locked Bag 4001, WESTMEAD NSW 2145. Email: brynley.hull@health.nsw.gov.au

### **Abbreviations**

7vPCV
 7-valent pneumococcal conjugate vaccine
 10vPCV
 10-valent pneumococcal conjugate vaccine
 13vPCV
 13-valent pneumococcal conjugate vaccine

23vPPV 23-valent pneumococcal polysaccharide vaccine

ABS Australian Bureau of Statistics

ACIR Australian Childhood Immunisation Register ARIA Accessibility/Remoteness Index of Australia

ATAGI Australian Technical Advisory Group on Immunisation

DTP diphtheria-tetanus-pertussis

DTPa Diphtheria-tetanus-pertussis (acellular) vaccine

dTpa Adolescent/adult diphtheria-tetanus-pertussis (acellular) vaccine

GP general practitioner

GPII General Practice Immunisation Incentives Scheme

HepA hepatitis A virus HepB hepatitis B virus

Hib Haemophilus influenzae type b HPV human papillomavirus vaccine IPV inactivated poliovirus vaccine MenC meningococcal serogroup C

MenCCV meningococcal C conjugate vaccine
MIA Maternity Immunisation Allowance

MMR measles-mumps-rubella

MMRV measles-mumps-rubella-varicella NIP National Immunisation Program

PRP-OMP Haemophilus influenzae type b polysaccharide conjugated to the outer membrane protein of

Neisseria meningitidis vaccine

SA3 Australian Bureau of Statistics Statistical Area 3

VV Varicella vaccine

WPV whole-patient equivalent

### References

- Hull B, Deeks S, Menzies R, McIntyre P. Immunisation coverage annual report, 2007. Commun Dis Intell 2009;33(2):170–187.
- Hull B, Mahajan D, Dey A, Menzies RI, McIntyre PB. Immunisation coverage annual report, 2008. Commun Dis Intell 2010;34(3):241–258.
- 3. Hull B, Dey A, Mahajan D, Menzies RI, McIntyre PB. Immunisation coverage annual report, 2009. Commun Dis Intell 2011;35(2):132–148.
- 4. Hull B, Dey A, Menzies RI, McIntyre PB. Immunisation coverage annual report, 2011. Commun Dis Intell 2013;37(4):22.
- Hull BP, McIntyre PB, Heath TC, Sayer GP. Measuring immunisation coverage in Australia. A review of the Australian Childhood Immunisation Register. Aust Fam Physician1999;28(1):55–60.
- Hull BP, McIntyre PB. Immunisation coverage reporting through the Australian Childhood Immunisation Register

  – an evaluation of the third-dose assumption. Aust N Z J Public Health 2000;24(1):17–21.
- Hull BP, McIntyre PB, Sayer GP. Factors associated with low uptake of measles and pertussis vaccines – an ecologic study based on the Australian Childhood Immunisation Register. Aust N Z J Public Health 2001;25(5):405–410.
- Hull B, Lawrence G, MacIntyre CR, McIntyre P. Immunisation coverage: Australia 2001. Canberra: Commonwealth Department of Health and Ageing; 2002
- Hull BP, Lawrence GL, MacIntyre CR, McIntyre PB. Estimating immunisation coverage: is the 'third dose assumption' still valid? Commun Dis Intell 2003;27(3):357–361.
- Hull BP, Lawrence GL, MacIntyre CR, McIntyre PB. Is low immunisation coverage in inner urban areas of Australia due to low uptake or poor notification? Aust Fam Physician2003;32(12):1041–1043.
- 11. Hull BP, Lawrence GL, MacIntyre CR, McIntyre PB. Immunisation coverage in Australia corrected for underreporting to the Australian Childhood Immunisation Register. Aust N Z J Public Health 2003;27(5):533–538.
- 12. Hull BP, McIntyre PB, Couzos S. Evaluation of immunisation coverage for Aboriginal and Torres Strait Islander children using the Australian Childhood Immunisation Register. Aust N Z J Public Health 2004;28(1):47–52.
- Lawrence GL, MacIntyre CR, Hull BP, McIntyre PB. Effectiveness of the linkage of child care and maternity payments to childhood immunisation. Vaccine 2004;22(17–18):2345–2350.
- Hull BP, McIntyre PB. What do we know about 7vPCV coverage in Aboriginal and Torres Strait Islander children? Commun Dis Intell 2004;28(2):238–243.
- Lawrence GL, Hull BP, MacIntyre CR, McIntyre PB. Reasons for incomplete immunisation among Australian children. A national survey of parents. Aust Fam Physician 2004;33(7):568–571.
- 16. Hull BP, McIntyre PB. Timeliness of childhood immunisation in Australia. *Vaccine* 2006;24(20):4403–4408.
- Hull BP, Deeks S, Menzies R, McIntyre PB. What do we know about 7vPCV coverage in Aboriginal and Torres Strait islander children? A 2007 update. Commun Dis Intell 2008;32(2):257–260.

- Hull BP, Deeks SL, McIntyre PB. The Australian Childhood Immunisation Register—A model for universal immunisation registers? Vaccine 2009;27(37):5054–5060.
- Hull B, Dey A, Menzies RI, McIntyre PB. Immunisation coverage annual report, 2010. Commun Dis Intell 2013;37(1):19.
- Australilan Government Department of Health and Ageing. The Australian Immunisation Handbook. 10th edn. Canberra: Australian Government Department of Health and Ageing; 2013.
- O'Brien ED, Sam GA, Mead C. Methodology for measuring Australia's childhood immunisation coverage. Commun Dis Intell 1998;22(3):36–37.
- Australian Population and Migration Research Centre. Accessibility/Remoteness Index of Australia (ARIA+). [online] 2011. Accessed on 20 November 2013. Available from: http://www.adelaide.edu.au/apmrc/research/projects/category/aria.html
- 23. Rank C, Menzies RI. How reliable are Australian Childhood Immunisation Register coverage estimates for indigenous children? An assessment of data quality and coverage. Commun Dis Intell 2007;31(3):283–287.
- 24. Australian Bureau of Statistics. Australian Statistical Geography Standard (ASGS). 2011. Accessed on. Available from: http://www.abs.gov.au/websitedbs/d3310114.nsf/home/australian+statistical+geography+standard+%28asgs%29
- 25. MapInfo. MapInfo version 12.5 [computer program]. Connecticut: Pitney Bowes Software Inc.; 2014.
- Australian Bureau of Statistics. Australian Statistical Geography Standard (ASGS): Correspondences, July 2011. [online]. Accessed on 21 November 2013. Available from: http://www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/1270.0.55.006Main+Features1July%20 2011?OpenDocument
- Gertig DM, Brotherton JM, Saville M. Measuring human papillomavirus (HPV) vaccination coverage and the role of the National HPV Vaccination Program Register, Australia. Sex Health 2011;8(2):171–178.
- 28. Australian Government Department of Health. Human Papillomavirus (HPV). 2013. Accessed on 21 November 2013. Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/immunise-hpv
- 29. Australian Institute of Health and Welfare. 2009 Adult Vaccination Survey: summary results. 2011. Accessed on 21 November 2013. Available from: http://www.aihw.gov.au/publication-detail/?id=10737418409
- 30. Lawrence G HB, McIntyre P. Monitoring immunisation coverage: Australia 2005. National Centre For Immunisation Research and Surveillance; 2006. p. 84.
- Buttery JP, Lambert SB, Grimwood K, Nissen MD, Field EJ, Macartney KK, et al. Reduction in rotavirus-associated acute gastroenteritis following introduction of rotavirus vaccine into Australia's National Childhood vaccine schedule. Pediatr Infect Dis J 2011;30(1 Suppl):S25–S29.
- Dey A, Wang H, Menzies R, Macartney K. Changes in hospitalisations for acute gastroenteritis in Australia after the national rotavirus vaccination program. Med J Aust 2012;197(8):453–457.
- 33. Brisson M, Edmunds W, Gay N, Law B, De Serres G. Modelling the impact of immunization on the epidemiology of varicella zoster virus. *Epidemiol Infect* 2000;125(3):651–669.

- 34. Menzies R, Turnour C, Chiu C, McIntyre P. Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia 2003 to 2006. Commun Dis Intell 2008;32(Suppl):S2–S67.
- 35. Plotkin S, Orenstein WA, Offit PA. Vaccines 5th edn. Elsevier; 2008.
- National, state, and local area vaccination coverage among children aged 19–35 months—United States, 2012. MMWR Morb Mortal Wkly Rep 2013;62(36):733–740.
- Health and Social Care Information Centre. NHS Immunisation Statistics England 2012–13. England; 2013.
- 38. National Health Performance Authority. Healthy Communities: Immunisation rates for children on 2011–12. Sydney, Australia; 2013.
- 39. Ward KF, Menzies RI, Quinn HE, Campbell-Lloyd S. School-based vaccination in NSW. N S W Public Health Bull 2010;21(9–10):237–242.
- Brotherton J, Gertig D, Chappell G, Rowlands L, Saville M. Catching up with the catch-up: HPV vaccination coverage data for Australian women aged 18–26 years from the National HPV Vaccination Program Register. Commun Dis Intell 2011;35(2):197–201.

- Brotherton JM, Mullins RM. Will vaccinated women attend cervical screening? A population based survey of human papillomavirus vaccination and cervical screening among young women in Victoria, Australia. Cancer Epidemiol 2012;36(3):298–302.
- Tabrizi SN, Brotherton JM, Kaldor JM, Skinner SR, Cummins E, Liu B, et al. Fall in human papillomavirus prevalence following a national vaccination program. J Infect Dis 2012;206(11):1645–1651.
- Ali H, Donovan B, Wand H, Read TR, Regan DG, Grulich AE, et al. Genital warts in young Australians five years into national human papillomavirus vaccination programme: national surveillance data. BMJ 2013;346:f2032.
- Brotherton JM, Fridman M, May CL, Chappell G, Saville AM, Gertig DM. Early effect of the HPV vaccination programme on cervical abnormalities in Victoria, Australia: an ecological study. *Lancet* 2011;377(9783):2085–2092.
- 45. Australian Bureau of Statistics. National Aboriginal and Torres Strait Islander Health Survey, 2004–05. Canberra; 2006.
- Department of Health. Immunise Australia Program. 2013. Accessed on 5 December 2013. Available from: http://www.health.gov.au/internet/immunise/publishing.nsf/Content/home

# Surveillance of adverse events following immunisation in Australia, 2012

Deepika Mahajan, Aditi Dey, Jane Cook, Bronwen Harvey, Rob I Menzies, Kristine K Macartney

### Abstract

report summarises Australian surveillance data for adverse events following immunisation (AEFI) reported to the Therapeutic Goods Administration (TGA) for 2012. It also describes reporting trends over the 13-year period 1 January 2000 to 31 December 2012. There were 1,897 AEFI records for vaccines administered in 2012, a decrease of 22% from 2,417 in 2011. The decrease in 2012 compared with 2011 was mainly attributable to a drop in the reports following receipt of the 23-valent pneumococcal polysaccharide vaccine (405 reduced to 133). However, reporting rates for some other vaccines such as rotavirus and varicella vaccines were higher in 2012 than 2011. Although an increase was observed in estimated reporting rates for rotavirus and varicella in children aged < 7 years in 2012 compared with 2011, it was not statistically significant. There were 370 AEFI records (37.2 per 100,000 doses) for the pneumococcal conjugate vaccine in 2012, which was fewer than in 2011 (43.4 per 100,000 doses). The most commonly reported reactions were injection site reactions (40%), fever (22%), allergic reactions (19%) and rash (10%). Only 7% of all the reported adverse events were categorised as serious. There were 2 reports of death, which were investigated by the TGA and no clear causal relationship with vaccination was found. Commun Dis Intell 2014;38(3):E232-E246.

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

### Introduction

This report summarises national passive surveillance data for adverse events following immunisation (AEFI) reported to the Therapeutic Goods Administration (TGA) to 28 February 2013. The report focuses on AEFI reported for vaccines administered during 2012 and trends in AEFI reporting over the 13-year period 1 January 2000 to 31 December 2012.

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

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

Reports summarising national AEFI surveillance data have been published regularly since 2003.<sup>2–11</sup> Trends in reported adverse events following immunisation are heavily influenced by changes to vaccine funding and availability provided through the National Immunisation Program (NIP). These changes impact on the interpretation of trend data and have been described in detail elsewhere.<sup>2–11</sup> Recent changes that impact on AEFI surveillance data presented in this report are:

- i. Implementation of the 4th dose of Prevenar 13®, (13vPCV, a 13-valent pneumococcal conjugate vaccine) for Indigenous children from October 2012 replacing the booster dose of Pneumovax23®12 (Table 1).
- ii. Replacement of Prevenar<sup>®</sup> (7-valent pneumococcal conjugate vaccine, 7vPCV) by Prevenar 13<sup>®</sup> from July 2011. The Northern Territory Government provided a free dose of Prevenar 13<sup>®</sup> at 18 months for children who previously received a primary course of Synflorix<sup>®</sup> (10vPCV) or a mixed primary pneumococcal course with Synflorix<sup>®</sup> and Prevenar<sup>®13</sup> (Table 1).

To assist readers, a glossary of the abbreviations of the vaccines referred to in this report is provided at the end of this report.

### **Methods**

AEFI are notified to the TGA by state and territory health departments, health professionals, vaccine manufacturers and members of the public. 14,15 All reports are assessed using internationally consistent criteria 16 and entered into the Australian Adverse Drug Reactions System (ADRS) database. All serious reports for drugs and vaccines are reviewed by medical officers in the TGA. Other reports are used in data mining and signal detection activities. Where there is insufficient information in a report to determine causality for a serious adverse event the TGA will contact the reporter on up to 3 occasions to elicit further information.

Table 1: Changes to the Australian Standard Vaccination Schedule (2003–2012)<sup>2-11</sup>

Date	Intervention
2003	Commencement of the meningococcal C conjugate vaccine (MenCCV) immunisation program.
	18-month dose of DTPa vaccine removed from the National Immunisation Program.
2004	dTpa funded at 15–17 years of age replacing the diphtheria-tetanus dose.
2005	From January 2005, universal funded infant 7-valent pneumococcal conjugate vaccine (7vPCV) program replaced the previous targeted childhood program, with a catch-up program for children aged <2 years.
	Universal 23-valent pneumococcal polysaccharide vaccine (23vPPV) for adults aged ≥65 years replaced previous subsidy through the Pharmaceutical Benefits Scheme.
	From November 2005, universal funded immunisation against varicella at 18 months of age with a school-based catch-up program for children at 10–13 years of age not previously vaccinated and without a history of varicella infection (no funded catch-up for children 2–10 years of age).
	IPV funded to replace OPV, in combination vaccines.
2007	From April 2007, funded immunisation against human papillomavirus for all Australian girls aged 12–13 years delivered through a school-based program, with a temporary catch-up program through schools or primary care providers for females aged 13–26 years until December 2009.
	From July 2007, universal funded immunisation against rotavirus at 2 and 4 months of age (Rotarix®) or at 2, 4 and 6 months of age (Rotateq®).
2008	Western Australia commenced a seasonal influenza vaccination program for all children aged 6 months to <5 years (born after 1 April 2003).
	In March 2008, Queensland, South Australia and Victoria changed from using two combination vaccines (quadrivalent DTPa-IPV and Hib-HepB) to the single hexavalent DTPa-IPV-HepB-Hib vaccine.
2009	By late 2009, all states and territories were using the single hexavalent DTPa-IPV-Hib-HepB (Infanrix hexa®) vaccine for all children at 2, 4 and 6 months of age, due to an international shortage of <i>Haemophilus influenzae</i> type b (Hib) (PedvaxHib® [monovalent] and Comvax® [Hib-HepB]) vaccines.
	Influenza A(H1N1)pdm09 vaccine (Panvax®) was rolled out across Australia from 30 September 2009 for people aged ≥10 years. From December 2009, the pandemic vaccine was made available to children aged 6 months to 10 years.
2010	Annual vaccination with seasonal trivalent influenza vaccine (TIV, containing 3 influenza strains: A/H1N1, A/H3N2 and B) was funded under the National Immunisation Program for people aged ≥6 months with medical risk factors (previously subsidised through the Pharmaceutical Benefits Scheme) and all Indigenous people aged ≥15 years (previously all Indigenous adults ≥50 years and 15–49 years with medical risk factors).
	On 23 April 2010, the use of the 2010 seasonal TIV in children <5 years of age was suspended by Australia's Chief Medical Officer due to an increased number of reports of fever and febrile convulsions post vaccination. A subsequent investigation identified that Fluvax® and Fluvax junior® (CSL Biotherapies), but neither of the other two available brands registered for use in young children, were associated with an unacceptably high risk of febrile convulsions. The recommendation to resume the use of seasonal influenza vaccine in children aged 6 months to 5 years, using brands other than Fluvax® and Fluvax junior®, occurred in August 2010.
2011	From 1 July 2011, Prevenar 13® replaced Prevenar® on the National Immunisation Program for children at 2, 4 and 6 months of age in all states and territories except the Northern Territory, which adopted 13vPCV from 1 October 2011.
	1 October 2011 to 30 September 2012 – all children aged between 12–35 months who had completed a primary pneumococcal vaccination course with 7vPCV were eligible to receive a free supplementary dose of Prevenar 13®
	On 25 March 2011, the Therapeutic Goods Administration issued a recall of Batch N3336 of the 23 valent pneumococcal polysaccharide vaccine 23vPPV, Pneumovax® 23. April 2011 – health professionals were advised not to administer a second or subsequent dose of Pneumovax 23 vaccine. December 2011 – Revised recommendations regarding which patients should be re-vaccinated under the National Immunisation Programwere provided.
2012	From 1 October 2012, a 4th dose of Prevenar 13®, (13vPCV, a 13-valent pneumococcal conjugate vaccine) was listed on the National Immunisation Program for Indigenous children, aged 12–18 months, residing in Queensland, South Australia, Western Australia and the Northern Territory. This replaced the booster dose of Pneumovax23®, (23vPPV, a 23-valent pneumococcal polysaccharide vaccine) administered between 18 and 24 months of age for Indigenous children from these jurisdictions.

CDI Vol 38 No 3 2014 E233

### Adverse event following immunisation data

De-identified information on all AEFI reported to the TGA from 1 January 2000 to 28 February 2013 and stored in the ADRS database were released to the National Centre for Immunisation Research and Surveillance (NCIRS) in March 2013. Readers are referred to previous AEFI surveillance reports for a description of the surveillance system.<sup>2,3</sup>

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

- a. the vaccination occurred between 1 January 2000 and 31 December 2012, or
- b. for records where the vaccination date was not recorded, the date of onset of symptoms or signs occurred between 1 January 2000 and 31 December 2012.

### Study definitions of adverse event following immunisation outcomes and reactions

AEFI were defined as 'serious' or 'non-serious' based on information in the report sent to the TGA and criteria similar to those used by the World Health Organization<sup>16</sup> and the US Vaccine Adverse Events Reporting System (VAERS).<sup>17</sup> In this report, an AEFI is defined as 'serious' if it meets one or more of the following criteria: (1) results in death (2) is life-threatening (3) requires inpatient hospitalisation or prolongation of existing hospitalisation (4) results in persistent or significant disability/incapacity (5) is a congenital anomaly/birth defect, or (6) is a medically important event or reaction.

The causality ratings of 'certain', 'probable' and 'possible' are assigned to individual records by the TGA. They describe the likelihood that a suspected vaccine or vaccines was/were associated with the reported reaction at the level of the individual vaccine recipient. Factors that are considered in assigning causality ratings include the timing (minutes, hours), the spatial relationship between symptoms and signs and the vaccination (for injection site reactions), and whether one or more vaccines were administered. These factors are outlined in more detail elsewhere.<sup>3</sup> However,

\* The term 'AEFI record' is used throughout this report because a single AEFI notification or report to the Office of Product review can generate more than 1 record in the ADRS database. This may occur if there is a time sequence of separate adverse reactions in a single patient, such as systemic and local reactions. in many instances a causal association between vaccines administered to an individual and events that subsequently occurred cannot be clearly ruled in or out. In addition, children in particular often receive several vaccines at the same time. Therefore, all administered vaccines are usually listed as 'suspected' of involvement in a systemic adverse event as it is usually not possible to attribute the event to a single vaccine.

Typically, each record lists several reaction terms, that is symptoms, signs and/or diagnoses that have been coded by TGA staff from the reporter's description into standardised terms using the Medical Dictionary for Regulatory Activities (MedDRA®).<sup>18</sup>

To analyse reported AEFI, MedDRA® coding terms were grouped to create a set of reaction categories. Firstly, reaction categories were created that were analogous to the reactions listed and defined in the *Australian Immunisation Handbook*, 9th edition.¹⁵ Where MedDRA® coding terms could not be categorised into categories as per the Immunisation Handbook, additional categories were created for those that were listed in more than 1% of records (e.g. headache, dizziness, change in heart or respiratory rate or rhythm). Reaction terms listed in less than 1% of records were grouped into broader categories based on the organ system where the reaction was manifested (e.g. gastrointestinal, neurological).

### Data analysis

All data analyses were performed using SAS software version 9.3.<sup>19</sup> Average annual population-based reporting rates were calculated for each state and territory and by age group using population estimates obtained from the Australian Bureau of Statistics.<sup>20</sup>

Reporting rates per 100,000 administered doses were estimated where information was available on the number of doses administered. This was done for vaccines funded through the NIP for children aged < 7 years.

Denominator data to estimate reporting rates for influenza and 23vPPV for people aged ≥ 18 years were obtained from a national adult coverage survey conducted in 2009.<sup>21</sup> For 23vPPV, the number of people vaccinated in 2012 was derived from the number of people who reported receipt of the vaccine within the previous 5 years, divided by five. The number of administered doses of each of the 10 childhood vaccines was obtained from the Australian Childhood Immunisation Register, a national population-based register of approximately 99% of children aged < 7 years.<sup>22</sup>

Records are classified as 'suspected' if the report contains sufficient information to be valid and the relationship between reported reactions and drugs are deemed to be biologically plausible.

### Notes on interpretation

Caution is required when interpreting the data presented in this report. Due to reporting delays and the late onset of some AEFI, the data are considered preliminary, particularly for the 4th quarter of 2012. Data published in previous reports for 2000–2011<sup>2–11</sup> may differ from that presented in this report for the same period because this report has been updated to include delayed notifications to the TGA that were not included in prior publications and data may be updated and recoded when follow-up information is received.

The information collated in the ADRS database is intended primarily for signal detection and hypothesis generation. While reporting rates can be estimated using appropriate denominators, they cannot be interpreted as incidence rates due to under-reporting and biased reporting of suspected events, and the variable quality and completeness of information provided in individual notifications.<sup>2–11,23</sup>

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

### Comparison with online Database of Adverse Events Notifications

In August 2012, the TGA made a searchable database, the Database of Adverse Event Notifications (DAEN) available on its web site. DAEN contains reports of all adverse event reports for medicines (including vaccines).<sup>24</sup> The data in this report are more detailed than that available on DAEN, and were provided to NCIRS by the TGA from the ADRS database. The numbers published in this report may be different to the numbers in the DAEN database, due to different dates of data extraction and amendment to reports where further information has become available. In addition, this report provides several features that are not available from the DAEN database, including long-term trends and population and dose-based reporting rates, put in the context of changes in vaccine policy and use, and reporting practices.

### **Results**

The ADRS database included a total of 1,897 records where the date of vaccination (or onset of adverse event, if vaccination date was not reported) was between 1 January and 31 December 2012.

In 2012, 84% of AEFI (n = 1,599) were reported to the TGA via states and territories (except Tasmania where all AEFIs are directly reported to the TGA), while the rest were reported directly to the TGA; 10% (n = 193) by doctors or health care providers, 3% (n = 50) by members of the public, 1% (n = 23) by hospitals, and 2% (n = 32) by drug companies.

### Reporting trends

The overall reporting rate for 2012 was 8.4 per 100,000 population, compared with 10.8 in 2011. The rate was lower compared with the previous years following a peak in 2010 (17.4) predominantly due to reports in children following vaccination with the 2010 seasonal trivalent influenza vaccine; in 2009 (11.0) associated with the influenza A(H1N1)pdm09 vaccine program; and in 2011 (10.8) following an increase in reports following 23vPPV in adults.<sup>9,10,11</sup>

There was a decline in the reported events and reporting rate per 100,000 population during 2012 and the vast majority of reported events from all reporter types, were of a non-serious nature similar to the previous years (Figure 1). There were marked variations in reporting levels in association with previous changes to the NIP from 2000 onwards (Figures 2a, 2b, 2c). The drop in reports in 2012 was predominantly associated with a drop in reports following 23vPPV vaccines in adults (Figure 2a). However, an increase was observed in estimated reporting rates for rotavirus and varicella in children aged < 7 years in 2012 compared with 2011, but it was not statistically significant (Table 2, Figure 2c).

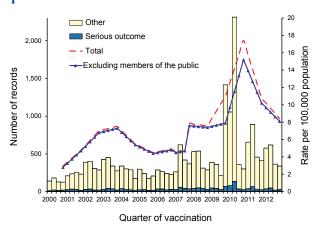
A seasonal pattern of AEFI reporting was apparent in 2012 and previous years, with the highest number of AEFI notifications for vaccinations administered in the first half of the year (Figure 1). The usual seasonal pattern of AEFI reporting in adults, with peaks in the first half of the year, was also apparent in 2012 (Figure 2a), corresponding to the months when older Australians receive the 23vPPV and influenza vaccines (March to June). However, more AEFI reports following influenza vaccine were received in 2011 and 2012 than in the years prior to 2009 (pre-pandemic era) (Figure 2a).

### Age distribution

In 2012, the highest population-based AEFI reporting rate occurred in infants < 1 year of age and 1 to < 2 years of age, the age groups that received the highest number of vaccines (Figure 3). Compared with 2011, AEFI reporting rates per 100,000 population increased among the < 1 year age group (5% increase from 134.9 to 142.3 per 100,000 population in 2011 and 2012 respectively),

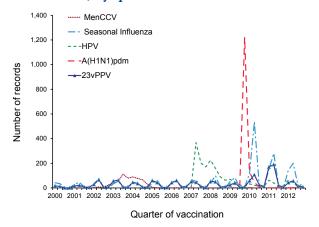
and the 1 to <2 year age group (5% increase, 53.8 to 56.7 per 100,000). Rates declined for all other age groups especially in the  $\ge$ 65 years age group (60% decline, 12.1 to 4.9 per 100,000).

Figure 1: Adverse events following immunisation, Australian Adverse Drug Reactions System database, 2000 to 2012, by quarter of vaccination



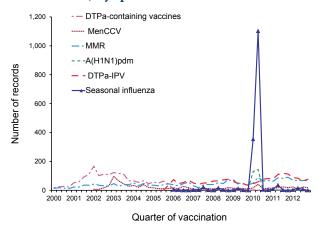
For reports where the date of vaccination was not recorded, the date of onset or the date the event was reported to the Therapeutic Goods Administration was used as a proxy for vaccination date.

Figure 2a: Adverse events following immunisation for people aged ≥7 years for frequently reported vaccines, Australian Adverse Drug Reactions System database, 2000 to 2012, by quarter of vaccination



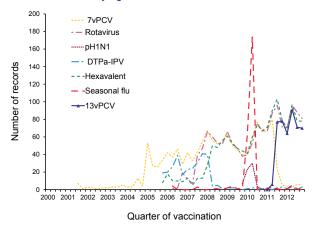
For reports where the date of vaccination was not recorded, the date of onset or the date the event was reported to the Therapeutic Goods Administration was used as a proxy for vaccination date.

Figure 2b: Adverse events following immunisation for children aged 1 to < 7 years for frequently reported vaccines, Australian Adverse Drug Reactions System database, 2000 to 2012, by quarter of vaccination



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

Figure 2c: Adverse events following immunisation for children aged < 1 year for frequently suspected vaccines,\* Australian Adverse Drug Reactions System database, 2000 to 2012, by quarter of vaccination



Meningococcal C conjugate vaccine was introduced into the National Immunisation Program schedule on 1 January 2003; 7-valent pneumococcal conjugate vaccine on 1 January 2005; DTPa-IPV and DTPa-IPV-HepB-Hib (hexavalent) vaccines in November 2005; rotavirus (RotaTeq® and Rotarix®) vaccines on 1 July 2007; pH1N1 influenza vaccine for children 6 months to 10 years on December 2009; seasonal trivalent influenza vaccine in 2010; and the 13-valent pneumococcal conjugate vaccine (13vPCV) on 1 July 2011 (Table 1).

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

E236 CDI Vol 38 No 3 2014

Table 2: Vaccine types listed as 'suspected' of involvement in records of adverse events following immunisation, Australian Adverse Drug Reactions System database, 2012,\* by age group

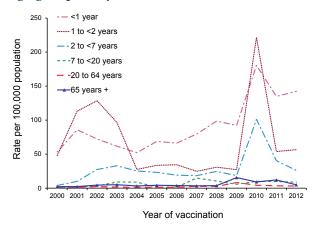
	AEFI records‡	Vaccine doses§		ing rate oses <sup>∥</sup> (95% CI)	
Vaccines <sup>†</sup>	n	n	2012	2011	
<7 years					
DTPa-containing vaccines	664	1,153,117	57.6 (53.3–62.1)	66.7 (62.0-71.6)	
DTPa-IPV	320	298,769 107.1 (95.7–119.5)		138.9 (125.9–152.9)	
Pentavalent (DTPa-IPV-HepB)	1	274	364.9 (10.9–2032.8)	396.8 (11.9–2222.2)	
Hexavalent (DTPa-IPV-HepB-Hib)	343	854,074	40.2 (36.0-44.6)	40.4 (36.2–45.0)	
Pneumococcal conjugate - PCV	370	993,880	37.2 (33.5–41.2)	48.3 (42.3–54.8)	
Rotavirus vaccine	338	537,171	62.9 (56.4–70.0)	56.3 (50.0-63.1)	
Measles-mumps-rubella	283	591,536	47.8 (42.4–53.8)	54.8 (49-61.1)	
Meningococcal C conjugate	79	297,603	26.6 (21.0–33.1)	26.3 (20.8–32.8)	
Varicella	67	280,568	23.9 (18.5–30.3)	21.7 (16.6–27.9)	
Haemophilus influenzae type b	66	284,223	23.2 (18.0–29.5)	25.5 (19.9–32.1)	
Seasonal influenza	32	na	na	na	
Total (<7 years)¶	973	4,138,379	23.5 (22.1–25.0)	27.6 (26.0–29.3)	
7–17 years					
HPV	151	na	na	na	
Hepatitis B	124	na	na	na	
dTpa	98	na	na	na	
Varicella	39	na	na	na	
Seasonal influenza	17	na	na	na	
Total (7-17 years)	317	na	na	na	
18-64 years					
Seasonal influenza**	271	3,170,300	8.6 (7.9–9.6)	7.1 (6.2–8.1)	
dTpa	67	na	na	na	
23vPPV**	40	132,520	30.2 (21.6–41.1)	63.4 (50.6–78.4)	
Total (18–64 years) <sup>††</sup>	311	3,302,820	9.4 (8.4–10.5)	9.4 (8.4–10.5)	
≥65 years					
Seasonal influenza**	78	2,176,000	3.6 (2.8–4.5)	5.9 (4.9-7.0)	
23vPPV**	77	317,400	24.3 (19.2–30.3)	90.7 (80.6–101.8)	
dTpa	17	na	na	na	
Total (≥65 years) <sup>††</sup>	155	2,493,400	6.2 (5.3–7.3)††	16.7 (15.2–18.4)††	

AEFI Adverse events following immunisation.

- \* Influenza A(H1N1)pdm09 was reported in the table in previous reports but is not shown in this table as it is no longer in use and no reports were received.
- † Records where at least one of the vaccines shown in the table was suspected of involvement in the reported adverse event
- 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 31 December 2012. More than one vaccine may be coded as 'suspected' if several were administered at the same time.
- § Number of vaccine doses recorded on the Australian Childhood Immunisation Register and administered between 1 January and 31 December 2012.
- || The estimated reporting rate per 100,000 vaccine doses recorded.
- ¶ Number of AEFI records excluding influenza vaccines.
- \*\* Number of administered doses of seasonal influenza vaccine estimated from the 2009 Australian Institute of Health and Welfare national adult vaccination survey.<sup>21</sup>
- †† Seasonal influenza and 23vPPV only.
- na Not applicable

CDI Vol 38 No 3 2014 E237

Figure 3: Reporting rates of adverse events following immunisation per 100,000 population, Australian Adverse Drug Reactions System database, 2000 to 2012, by age group and year of vaccination



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

Reporting rates per 100,000 doses (excluding influenza due to the absence of reliable dose data) decreased overall and for most individual vaccines in children aged < 7 years in 2012 compared with 2011 except for rotavirus and varicella vaccines (Table 2).

Reporting rates per 100,000 vaccine doses were lower overall in 2012 compared with 2011 for adults aged  $\geq 65$  years (down to 6.2 in 2012 from 16.7 in 2011) and especially for 23vPPV (down to 24.3 in 2012 from 90.7 in 2011) (Table 2).

### Geographical distribution

Population-based reporting patterns varied between states and territories during 2012 (Table 3) as in previous years.<sup>2–11</sup> The highest reporting rates were from the Northern Territory, the Australian Capital Territory, Western Australia and Victoria (20.0, 13.1, 11.7, 11.5, respectively) while New South Wales had the lowest rate (4.4). Reporting rates dropped in most jurisdictions in 2012 compared with 2011 except in Western Australia, which experienced an increase that was not statistically significant (Table 3).

### **Vaccines**

Thirty-two different vaccines were included in the 1,897 records received in 2012 (Table 4). The percentage of records where only 1 vaccine was reported as being the suspected vaccine differed by the vaccine administered, typically varying according to whether multiple vaccines were routinely co-administered for the patient's age. The percentage of records assigned causality ratings of 'certain' or 'probable' also varied. Adverse events such as

Table 3: Adverse events following immunisation, Australian Adverse Drug Reactions System database, 2012, by state or territory

	AEFI r	ecords	Annua	l reporting rate per	100,000 popula	tion*
State or territory	n %		Overall	'Certain'/ 'probable' causality rating <sup>†‡</sup>	'Serious' outcome	Aged <7 years
Australian Capital Territory	49	3	13.1 (9.7–17.3)	2.7	0.3	5.3
New South Wales	321	17	4.4 (3.9–4.9)	0.8	0.6	1.7
Northern Territory	47	2	20.0 (14.7–26.6)	6.0	1.3	10.2
Queensland	328	17	7.2 (6.4–8.0)	1.9	0.5	3.5
South Australia	142	7	8.6 (7.2–10.1)	1.1	0.4	3.9
Tasmania	42	2	8.2 (5.9–11.1)	1.6	0.4	1.8
Victoria	650	34	11.5 (10.7–12.5)	2.0	0.6	7.7
Western Australia	285	15	11.7 (10.4–13.2)	2.5	0.7	5.4
Other§	33	2	na	na	na	na
Total		100	8.4 (8.0-8.7)	1.6	0.5	4.3

AEFI Adverse events following immunisation.

- \* Average annual rates per 100,000 population calculated using the Australian Bureau of Statistics mid-2011 population estimates <sup>20</sup>
- † Causality ratings were assigned to AEFI records using criteria described previously.<sup>2,3</sup>
- ‡ AEFI records defined as 'serious' (i.e. recovery with sequelae, hospitalisation, life-threatening or death).
- § Records where the jurisdiction in which the AEFI occurred was not reported or was unclear. AEFI records in this category were notified mainly by pharmaceutical companies (n=32), and General Practioners (n=1).

E238 CDI Vol 38 No 3 2014

Table 4: Vaccine types listed as 'suspected' in records of adverse events following immunisation, Australian Adverse Drug Reactions System database, 2012

	AEFI <sub>.</sub>	susp vacci	ne ected ne or	ʻprok caus	tain'/ pable' sality		ous'	_		roup <sup>  </sup>	
Suspected vaccine	records		only <sup>†</sup>		ing‡ %¶		ome <sup>§</sup> %¶		ears %¶	1	ears %¶
type*	n 406	n 242	%¶	n		n 27		n		n 266	
Influenza	406	342	84	56	14	37	9	32	8	366	90
PCV	370	35	10	19	5	34	10	370	100	0	0
DTPa-IPV-HepB-Hib	344	21	6	19	6	33	10	343	99	1	1
Rotavirus	338	52	15	11	3	44	13	338	100	0	0
DTPa-IPV	324	133	41	113	35	10	3	320	99	2	1
MMR	298	31	10	13	4	14	5	283	95	14	5
dTpa	186	132	71	47	25	10	5	2	1	182	98
Hepatitis B	160	53	33	13	8	7	4	10	6	150	94
HPV	155	62	40	12	8	9	6	0	0	155	100
23vPPV	133	80	60	43	32	9	7	10	8	122	92
Varicella	111	59	53	6	5	9	8	67	60	41	37
MenCCV	87	6	7	5	6	6	7	79	91	8	9
Hib	67	0	0	0	0	6	9	66	99	1	1
Typhoid	20	5	25	1	5	3	15	2	10	18	90
dT	16	10	63	5	31	0	0	0	0	16	100
BCG	15	15	100	10	67	0	0	15	100	0	0
Hepatitis A	14	3	21	2	14	3	21	4	29	10	71
Hepatitis A-Typhoid	13	6	46	1	8	0	0	0	0	13	100
Yellow fever	12	4	33	0	0	2	17	1	8	11	92
DTPa	11	8	73	5	45	1	9	2	18	9	82
Hepatitis A + B	11	8	73	1	9	0	0	1	9	10	91
Rabies	8	4	50	3	38	0	0	1	12	7	88
IPV	6	3	50	0	0	0	0	1	17	5	83
dTpa-IPV	5	3	60	2	40	0	0	0	0	5	100
10vPCV	3	0	0	0	0	0	0	2	67	1	33
Q fever	3	3	100	0	0	1	33	0	0	3	100
Japanese encephalitis	2	1	50	1	50	0	0	0	0	2	100
Men4PV	2	0	0	0	0	0	0	0	0	2	100
Cholera	2	1	50	1	50	0	0	0	0	2	100
Tetanus	2	2	100	0	0	0	0	0	0	2	100
DTPa-IPV-HepB	1	1	100	0	0	0	0	1	100	0	0
Total**	1,897	1,078	57	374	20	137	7	973	51	908	48

AEFI Adverse events following immunisation.

- \* The abbreviations of vaccine names are included at the end of this report.
- † AEFI records where only one vaccine was suspected of involvement in a reported adverse event.
- ‡ Causality ratings were assigned to AEFI records using criteria described previously.<sup>2,3</sup>
- § 'Serious' outcomes are defined in the Methods section.
- AEFI records are not shown if both age and date of birth were not reported.
- ¶ Percentages are calculated for the number of AEFI records where the vaccine was suspected of involvement in the AEFI, e.g. HPV was 'suspected' in 155 AEFI records. This was the only suspected vaccine in 40% of the 155 AEFI records. Eight per cent had 'certain' or 'probable' causality ratings, 6% were defined as 'serious' and 100% were for those aged ≥7 years.
- \*\* Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and an AEFI record may list more than 1 vaccine.

CDI Vol 38 No 3 2014 E239

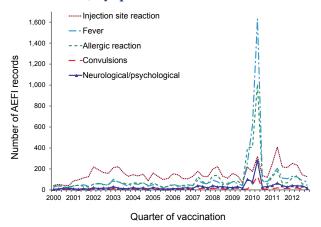
injection site reactions, for which the attribution of causality is more straightforward, were more often reported as certain or probable. There were also slight variations in the number with outcomes defined as 'serious', which have remained low as in previous years.

The individual vaccines most frequently suspected to have been involved in AEFI events were seasonal influenza vaccine (n = 406,21%), 13vPCV (n = 350, 18%) and hexavalent DTPa-IPV-HepB-Hib (n = 344, 18%) (Table 4).

### Reactions

The most frequently reported adverse events in 2012 were injection site reactions (ISRs) (n = 761, 40%), fever (n = 420, 22%), allergic reaction (n = 355, 19%), and rash (n = 195, 10%) (Tables 5a and 5b, Figure 4). Some of the severe reactions included convulsions (n = 54, 3%) including 25 cases of febrile convulsion, hypotonic-hyporesponsive episode (HHE) (n = 33, 2%), anaphylactic reaction (n = 16, 1%), intussusception (n = 16, 1%), thrombocytopenia (n = 2, 0.1%), and Guillain-Barrè syndrome (GBS) (n = 1, 0.05%) (Table 5a).

Figure 4: Selected frequently reported adverse events following immunisation, Australian Adverse Drug Reactions System database, 2000 to 2012, by quarter of vaccination



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

Of the 761 cases of ISR, 385 (51%) were in children aged < 7 years. The vaccines most commonly suspected of involvement in AEFI for children aged < 7 years were: DTPa-IPV (n = 257); MMR (n = 149); 13vPCV (n = 55); and hexava-

lent vaccine (n = 52). For those aged  $\geq$ 7 years (n = 370), these were seasonal influenza vaccine (n = 149), 23vPPV (n = 104) and dTpa (n = 84) either given alone or co-administered with other vaccines. As expected, reports for 23vPPV were predominantly in those aged  $\geq$ 65 years (63%), while AEFI with seasonal influenza vaccine was commonly reported in the 18–64 years age group (70%). The dTpa vaccine was most commonly recorded as causing ISR in AEFI records in the 12–17 years age group (40%) and the 18–64 years age group (40%).

The number of reports in each reaction category has changed over time (Figure 4). Much of the variation in reporting of ISR related to specific changes in the immunisation schedules for vaccines that are known to have higher rates of ISR, including DTPa-containing vaccines, MenCCV, 23vPPV and HPV vaccines. 2-11,25,26

The increase in ISR during 2012 was predominantly associated with non-influenza vaccines, particularly 23vPPV and DTPa-containing vaccines. In children aged 2 to <7 years, ISR was mainly associated with DTPa-IPV vaccine (86%, n = 251). In the 18–64 years age group, it is associated with seasonal influenza vaccine (60%, n = 109), while in the 12–17 years age group, the increase in ISR was associated with adult dTpa vaccine (44%, n = 186), HepB (34%, n = 160), and HPV (30%, n = 155).

Increases in reports of fever were largely associated with time periods when new vaccines were added to the NIP in the reporting period, such as rotavirus and HPV in 2007 and the extension of seasonal influenza vaccine from the elderly to others at high risk in 2010.

### Severity of outcomes

Fifty-three per cent (n = 999) of reported events in 2012 were defined as 'non-serious', 7% (n = 137) were defined as 'serious' (i.e. requiring hospitalisation, resulting in persistent or significant disability or incapacity; medically important reaction or event; experiencing a life-threatening event or death); 26% (n = 495) were recorded as not fully recovered, while information on severity could not be determined due to insufficient data for 14% (n = 266) of events (Table 6). This was similar to the proportions of serious AEFI in previous years. 9,10,11 Of the reported events recorded as not fully recovered at the time of reporting, the largest proportion were from Victoria (39%, n = 191), followed by Western Australia (21%, n = 106) and Queensland (16%, n = 77). Ninety-three per cent of cases recorded as 'not fully recovered' had missing information in various fields including

Table 5a: Reaction categories of interest\* mentioned in records of adverse events following immunisation, Australian Adverse Drug Reactions System database, 2012

	AEFI	Only reaction reported <sup>†</sup>		'Certain'	/'probable'	Age group <sup>§</sup>				
	records			causali	ty rating <sup>‡</sup>	<7 years		≥7 years		
Reaction category*	n	n	% <sup>  </sup>	n	% <sup>∥</sup>	n	% <sup>  </sup>	n	% <sup>  </sup>	
Injection site reaction	761	273	36	321	42	385	51	370	49	
Fever	420	8	2	9	2	239	57	175	42	
Allergic reaction¶	355	60	17	12	3	163	46	191	54	
Rash**	195	72	37	8	4	142	73	52	27	
Syncope	125	84	67	13	10	29	23	96	77	
Abnormal crying	74	10	14	0	0	72	97	1	1	
Arthralgia	64	4	6	2	3	5	8	59	92	
Convulsions	54	28	52	0	0	40	74	14	26	
Lymphadenopathy/itis††	46	12	26	14	30	22	22	34	74	
Hypotonic-hyporesponsive episodes	33	26	79	1	3	33	100	0	0	
Anaphylactic reaction	16	12	75	3	19	5	31	11	69	
Intussusception	16	14	88	2	13	1	100	0	0	
Abscess	10	3	30	5	50	8	80	2	20	
Arthritis	5	0	0	1	20	0	0	5	100	
Brachial neuritis	2	1	50	0	0	0	0	2	100	
Death	2	1	50	0	0	1	50	1	50	
Thrombocytopenia	2	0	0	1	50	1	50	1	50	
Encephalitis	1	0	0	0	0	1	100	0	0	
Encephalopathy	1	0	0	0	0	1	100	0	0	
Guillain-Barré syndrome	1	0	0	0	0	0	0	1	100	
Parotitis	1	0	0	0	0	1	100	0	0	
Orchitis	1	0	0	0	0	1	100	0	0	
Osteitis	0	0	0	0	0	0	0	0	0	
Sepsis	0	0	0	0	0	0	0	0	0	
Total <sup>‡‡</sup>	1,897	734	39	374	20	973	51	908	48	

AEFI Adverse events following immunisation.

- \* Reaction categories were created for the AEFI of interest listed and defined in *The Australian Immunisation Handbook* 9th edition (p 58–65 and 360–363)<sup>15</sup> as described in the Methods section.
- † AEFI records where only one reaction was reported.
- ‡ Causality ratings were assigned to AEFI records using criteria described previously.<sup>2,3</sup>
- § Not shown if neither age nor date of birth were recorded.
- Percentages relate to the number of AEFI records in which the specific reaction term was listed, e.g. of 761 AEFI records listing injection site reaction, 36% listed only one type of reaction while 42% had a causality rating of 'certain' or 'probable' and 51% were for children aged <7 years.
- ¶ Allergic reaction includes skin reactions including pruritus, urticaria, periorbital oedema, facial oedema, erythema multiforme etc. (excludes skin reactions presented elsewhere in this table); and/or gastrointestinal (e.g. diarrhoea, vomiting)
  symptoms and signs but does not include other abdominal symptoms such as abdominal pain, nausea, flatulence,
  abnormal faeces, hematochezia etc. and does not include anaphylaxis.
- \*\* Includes general terms of rash but does not include pruritic rash.
- †† Includes lymphadenitis following Bacille Calmette-Guérin BCG vaccination and the more general term of 'lymphadenopathy'.
- the Total number of AEFI records analysed, not the total in each column as categories are not mutually exclusive and an AEFI record may list more than 1 reaction term.

CDI Vol 38 No 3 2014 E241

Table 5b: 'Other'\* reaction terms listed in records of adverse events following immunisation, Australian Adverse Drug Reactions System database, 2012

	AEFI	Only r	eaction	'Certain'/'probable'		Age group <sup>ş</sup>				
	records				ty rating	<7 years		≥7 years		
Reaction term*	n	n	%∥	n	%∥	n	% <sup>  </sup>	n	%∥	
Malaise	152	3	2	5	3	47	31	102	67	
Headache	139	2	1	6	4	8	6	130	94	
Neurological/psychological	138	3	2	3	2	87	63	48	35	
Gastrointestinal – RVV¶	122	15	12	3	2	122	100	0	0	
Nausea	111	1	1	6	5	5	5	105	95	
Dizziness	98	4	4	4	4	2	2	94	96	
Myalgia	72	2	3	5	7	9	13	61	85	
Pain	69	0	0	3	4	6	9	61	88	
Abdominal pain	60	2	3	4	7	31	52	29	48	
Reduced sensation	60	5	8	2	3	0	0	58	97	
Respiratory	47	5	11	1	2	15	32	31	66	
Erythema	38	5	13	1	3	23	61	14	37	
Pallor	37	1	3	1	3	26	70	10	27	
ENT	26	2	8	2	8	5	19	20	77	
Oedema	27	0	0	0	0	14	52	12	44	
Somnolence	25	1	4	0	0	16	64	9	36	
Circulatory	24	2	8	4	13	12	50	12	50	
Increased sweating	21	0	0	2	10	6	29	13	62	
Tremor	18	1	6	1	6	2	11	16	89	
Vision impaired	16	1	6	1	6	2	13	14	88	
Weakness	11	0	0	0	0	0	0	11	100	
Flushing	9	0	0	0	0	1	11	8	89	
Other	225	32	14	20	9	111	49	109	48	
general non-specific	38	7	18	5	13	22	58	16	42	
infection	19	7	37	0	0	8	42	10	53	
neurological	19	9	47	2	11	9	47	10	53	
eye or ear	17	3	8	1	6	8	47	9	53	
metabolic/endocrine	16	0	0	1	6	14	88	2	12	
cardiovascular	15	1	7	3	20	9	0	6	40	
musculoskeletal	13	0	0	0	0	2	15	11	85	
gastrointestinal <sup>††</sup>	12	0	0	0	0	5	42	7	58	
respiratory	12	1	8	0	0	5	42	7	58	
skin**	11	2	18	0	0	7	64	3	27	
psychological	9	0	0	1	11	4	44	5	56	
renal/urogenital	7	0	0	0	0	3	43	4	57	
haematological	7	2	29	1	14	2	29	5	71	
miscellaneous	4	0	0	0	0	1	25	2	50	
pregnancy/congenital	2	0	0	0	0	2	100	0	0	

AEFI Adverse events following immunisation.

E242 CDI Vol 38 No 3 2014

<sup>\*</sup> Reaction terms not listed in *The Australian Immunisation Handbook 9th edition*<sup>15</sup> but included in AEFI records in the ADRS database. The top part of the table shows reaction terms included in 1% or more of AEFI records, the bottom part of the table shows reaction terms, grouped by organ system, that were included in less than 1% of AEFI records.

<sup>§</sup> Not shown if neither age nor date of birth were recorded.

Percentages relate to the number of AEFI records in which the specific reaction term was listed, e.g. of 152 AEFI records listing malaise, 2% listed only 1 type of reaction while 3% had a causality rating of 'certain' or 'probable' and 31% were for children aged <7 years.

<sup>¶</sup> Gastrointestinal – RVV includes gastrointestinal reactions following rotavirus vaccination only.

<sup>\*\*</sup> Other, skin includes purpura, petechie, blister, burning, dermatitis, dry skin etc. but does not include skin reactions.

<sup>††</sup> Other, gastrointestinal does not include reaction categories coded as gastrointestinal reactions or Gastrointestinal – RVV signs and symptoms.

hospitalisation. For reports where not 'fully recovered' was recorded, 83% (n = 409) were reported by states and territories, 11% (n = 56) by health care providers, 4% (n = 19) by members of the public, 1% each by pharmacist (n = 4) and drug companies (n = 5), and 0.4% (n = 2) by hospitals. Of those without information describing severity, the most commonly reported adverse reactions were ISR (41%, n = 110), fever (22%, n = 58) and allergic reactions (20%, n = 52).

Only 20% of records (n = 374) were assigned causality ratings of either 'certain' (n = 347, 18%) or 'probable' (n = 27, 2%), and the rest (80%) were rated as 'possible'.

The 'serious' reactions (n = 137) were ISR (n = 21, 15%); fever (n = 25, 18%); allergic reactions (n = 17, 12%); convulsions (n = 21, 15%), including 12 febrile convulsions; diarrhoea/vomiting (n = 16, 12%); HHE (n = 7, 5%); anaphylaxis(n = 6, 4%); GBS (n = 1, 1%); intussusception (n = 9, 7%); 11 cases of syncope (8%); and 2 reports of death (2%), one of which was a case of idiopathic thrombocytopenic purpura. Other relatively severe reactions that were not classified as 'serious', either because they did not satisfy the criteria, or due to a lack of information about the outcome and/ or hospitalisation status, included: convulsion (n = 33, 61%), including 13 febrile convulsions; HHE (n = 26, 79%); anaphylaxis (n = 10, 63%); and intussusception (n = 9, 56%).

All the reported cases of HHE (33) were from children aged < 7 years. Twenty-eight reports (85%) followed co-administration of hexavalent, PCV and rotavirus vaccines. Another 2 cases followed vaccination with Hib, MenC and MMR while a further 3 cases followed the vaccines administered simultaneously in combinations of: DTPa, PCV and rotavirus; DTPa-IPV, PCV and rotavirus; and PCV, Hep A and varicella vaccine.

The single reported case of GBS was in a person aged > 60 years following co-administered seasonal influenza (Fluvax®) and 23vPPV vaccines. The person experienced corrhyzal symptoms for 4 days after receiving the vaccines and developed symptoms of GBS approximately 11 days post vaccination.

All of the 16 reports of intussusception were from infants (< 1 year of age). Fifteen reports (94%) were following hexavalent, 13vPCV and rotavirus vaccines administered together, while 1 report was following rotavirus vaccine administered alone.

Five of the 16 reports of anaphylaxis were in children aged < 7 years. Three cases were infants and two were following hexavalent and 13vPCV vaccine administered together, while one reported anaphylaxis following hexavalent alone. Another 2 cases in children aged < 7 years were following vaccination with DTPa-IPV and MMR administered together. Eight of the 11 cases of anaphylaxis in those aged > 7 years followed receipt of one of the influenza

Table 6: Outcomes of adverse events following immunisation, Australian Adverse Drug Reactions System database, 2012

	AEFI records		'Certain'/ 'probable' causality rating*		<7 y	Age g	roup <sup>†</sup> ≥7 years	
Outcome	n	% <sup>‡</sup>	n	% <sup>§</sup>	n	% <sup>§</sup>	n	%§
Non-serious	999	53	171	17	498	50	494	49
Not recovered at time of report	495	26	140	28	247	50	245	49
Not known (missing data) – total	266	14	52	20	153	58	109	41
Serious:	137	7	11	8	75	55	60	44
recovered with sequelae	1		_		_		1	
hospital treatment – admission	125		11		72		51	
life-threatening event	9		_		2		7	
Death	2		_		1		1	
Total	1,897	100	374	20	973	51	908	48

AEFI Adverse events following immunisation.

- \* Causality ratings were assigned to AEFI records using criteria described previously.<sup>2,3</sup>
- † AEFI records where both age and date of birth were not recorded are not shown (16 missing).
- ‡ Percentages relate to the total number of AEFI records (n = 1,897).
- § Percentages relate to the number of AEFI records with the specific outcome, e.g. of 999 AEFI records with a 'non-serious' outcome, 17% had causality ratings of 'certain' or 'probable' and 50% were for children aged <7 years.</p>

vaccines administered alone or in combination with other vaccines, while one each of the remaining three followed adult dTpa vaccine, HepB vaccine and 23vPPV vaccine administered alone.

Two deaths were recorded as being temporally associated with receipt of vaccines:

- A 19-month-old child who had received varicella vaccine 7 weeks prior to death. Seven weeks after vaccination the child presented with vomiting, fever and drowsiness. The cause of death was recorded as encephalitis.
- A 28-year-old person, who became unwell 2–3 days post vaccination with an unspecified influenza vaccine. The person developed thrombotic thrombocytopenic purpura (TTP) and died 2 days after onset of symptoms. The cause of the death was documented as TTP.

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

### Pneumococcal conjugate vaccine

In 2012, there were 370 reports of adverse events following pneumococcal conjugate vaccine in people aged < 7 years, with 34 of these cases being coded as serious.

The reporting rates were 37.2 per 100,000 doses for PCV (Table 2). Seven per cent of reports involving PCV (n = 26) were for PCV administered alone. Of the 26 cases, 92% (n = 24) were children between 12 months and < 36 months, who received vaccine under the catch-up program offered to children between 12 months and 35 months of age. Seventy-three per cent (n = 269) of the reports involving PCV vaccines were for PCV co-administered with rotavirus vaccine.

The spectrum of reactions for PCV included 91 (25%) reports each of vomiting or diarrhoea and fever; 73 (20%) of rash; 61 of allergic reactions (17%); 55 ISR (16%); 56 screaming (15%); 29 cases of HHE (8%); 18 cases of convulsions (including 6 febrile convulsions) (5%); 6 case of syncope (2%); and 2 cases of anaphylaxis (0.5%). The total number of AEFIs don't add up as some reports had multiple reactions.

### **Discussion**

There was an overall drop in the total number of AEFI reports and population-based reporting rates in 2012 compared with 2011, predominantly due to a large decline in reports following vaccination with the 23vPPV vaccine.

Reporting rates per 100,000 doses for children aged < 1 years and 1 to < 2 years were similar to the corresponding period in 2011, but significantly lower for children aged 2 to < 7 years [54 (95% CI: 48.5–59.4) vs 80 (73.7–87.0)]. The decrease in overall reporting of AEFI in children aged 2 to < 7 years in 2012 is primarily due to a drop in the reporting of ISR following vaccination with DTPa-IPV in that age group in 2012 compared with 2011. Although reporting rates for DTPa-IPV vaccines were lower in 2012 compared with 2011, reporting rates remained higher than for other years (92 in 2008; 72 in 2009; 94 in 2010). 9,10

The increase in the reports following rotavirus vaccine may be because in the majority of the records (77%), rotavirus vaccine was administered with PCV. The likelihood of developing at least 1 AEFI with the administration of multiple vaccines is greater than with just 1 vaccine. From 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®. The increased AEFI reports following PCV administration in toddlers 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 2nd year of life) compared with earlier doses in infancy. A similar trend was also observed for the other systemic reactions.<sup>27</sup> Some may also be attributed to the 'Weber effect',28 which describes increased reporting frequently observed following the introduction of new vaccines.

### Conclusion

The total number of reported AEFI in 2012 was reduced by 22% compared with 2011.

Reports of ISR following DTPa-IPV at 4 years decreased in 2012 compared with 2011 but remained higher than in previous years. Reporting rates for most of the vaccines were similar to 2011 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.

### **Acknowledgements**

We thank Brynley Hull and Donna Armstrong, National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, for assisting in the preparation of this report.

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases is supported by the Australian Government Department of Health, the New South Wales Ministry of Health and The Children's Hospital at Westmead, New South Wales.

### **Author details**

Deepika Mahajan<sup>1</sup> Aditi Dey<sup>1</sup> Jane Cook<sup>2</sup> Bronwen Harvey<sup>2</sup> Rob Menzies<sup>1</sup> Kristine Macartney<sup>1</sup>

- National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, University of Sydney and The Children's Hospital at Westmead, Sydney, New South Wales
- Office of Product Review, Therapeutic Goods Administration, Canberra, Australian Capital Territory

Corresponding author: Dr Deepika Mahajan, National Centre for Immunisation Research and Surveillance, Locked Bag 4001, WESTMEAD NSW 2145. Telephone: +61 2 9845 1433. Facsimile: +61 2 9845 1418. Email: Deepika. mahajan@health.nsw.gov.au

### References

- Council for International Organizations of Medical Sciences (CIOMS) c/o World Health Organization. Definition and Application of Terms for Vaccine Pharmacovigilance: Report of CIOMS/WHO Working Group on Vaccine Pharmacovigilance. 2012. Accessed on 16 August 2012. Available from: http://www.cioms. ch/frame\_vaccine\_pharmacovigilance.htm
- Lawrence G, Menzies R, Burgess M, McIntyre P, Wood N, Boyd I, et al. Surveillance of adverse events following immunisation: Australia, 2000–2002. Commun Dis Intell 2003;27(3):307–323.
- Lawrence G, Boyd I, McIntyre P, Isaacs D. Surveillance of adverse events following immunisation: Australia 2002 to 2003. Commun Dis Intell 2004;28(3):324–338.
- Lawrence GL, Boyd I, McIntyre PB, Isaacs D. Annual report: surveillance of adverse events following immunisation in Australia, 2004. Commun Dis Intell 2005;29(3):248–262.
- Lawrence G, Boyd I, McIntyre P, Isaacs D. Annual report: surveillance of adverse events following immunisation in Australia, 2005. Commun Dis Intell 2006;30(3):319–333.
- Lawrence GL, Aratchige PE, Boyd I, McIntyre PB, Gold MS. Annual report on surveillance of adverse events following immunisation in Australia, 2006. Commun Dis Intell 2007;31(3):269–282.
- Lawrence G, Gold MS, Hill R, Deeks S, Glasswell A, McIntyre PB. Annual report: surveillance of adverse events following immunisation in Australia, 2007. Commun Dis Intell 2008;32(4):371–387.

- Menzies R, Mahajan D, Gold MS, Roomiani I, McIntyre P, Lawrence G. Annual report: surveillance of adverse events following immunisation in Australia, 2008. Commun Dis Intell 2009;33(4):365–381.
- Mahajan D, Roomiani I, Gold M, Lawrence G, McIntyre P, Menzies R. Annual report: surveillance of adverse events following immunisation in Australia, 2009. Commun Dis Intell 2010;34(3):259–276.
- Mahajan D, Cook J, McIntyre P, Macartney K, Menzies R. Annual report: surveillance of adverse events following immunisation in Australia, 2010. Commun Dis Intell 2011;35(4):263–280.
- 11. Mahajan D, Cook J, Dey A, Macartney K, Menzies R. Annual report: surveillance of adverse events following immunisation in Australia, 2011. Commun Dis Intell 2012;36(4):E315–E332.
- 12. Australian Government Department of Health and Ageing. Immunise Australia program. Pneumococcal Disease: Recent changes to pneumococcal vaccine for children Program providing a supplementary dose of Prevenar 13®. Accessed on 7 August 2013. Available from: http://immunise.health.gov.au/internet/immunise/publishing.nsf/Content/immunise-pneumococcal
- Centre for Disease Control. Change to Northern Territory Childhood Immunisation Schedule to introduce 13 valent pneumococcal vaccine 1 October 2011. Northern Territory Disease Control Bulletin 2011;18:37.
- Australian Technical Advisory Group on Immunisation. The Australian Immunisation Handbook. 8th edn. Canberra: Australian Government Department of Health and Ageing, 2003.
- National Health and Medical Council. The Australian Immunisation Handbook. 9th edn. Canberra: Australian Government Department of Health and Ageing, 2008.
- Uppsala Monitoring Centre. WHO Collaborating Centre for International Drug Monitoring. Accessed on 30 August 2010. Available from: http://www.who-umc.org/
- Zhou W, Pool V, Iskander JK, English-Bullard R, Ball R, Wise RP, et al. Surveillance for safety after immunization: Vaccine Adverse Event Reporting System (VAERS)—United States, 1991–2001. [Erratum appears in MMWR Morb Mortal Wkly Rep 2003(06):113]. MMWR Morb Mortal Wkly Rep 2003;52(SS-1):1–24.
- Brown EG, Wood L, Wood S. The medical dictionary for regulatory activities (MedDRA). Drug Saf 1999;20(2):109–117.
- 19. The SAS system for Windows [computer program]. Version 9.3. Cary, N.C.: SAS Institute Inc, 2012.
- Australian Bureau of Statistics. Population by Age and Sex, Australian States and Territories, June 2011. Australian Bureau of Statistics: Cat no. 31010DO002\_201106; 2011.
- Australian Institute of Health and Welfare. 2009 Adult Vaccination Survey: summary results. Cat. No. PHE 135 Canberra: Australian Institute of Health and Welfare; 2011.
- 22. Additional reports Childhood immunisation coverage. Commun Dis Intell 2008;32(2):288–289.
- 23. Varricchio F, Iskander J, DeStefano F, Ball R, Pless R, Braun MM, et al. Understanding vaccine safety information from the Vaccine Adverse Event Reporting System. *Pediatr Infect Dis J* 2004;23(4):287–294.
- 24. Therapeutic Goods Administration. Database of Adverse Event Notifications. 1 August 2012. Accessed on 28 August 2012. Available from: http://www.tga.gov.au/safety/daen.htm

- Joura EA, Leodolter S, Hernandez-Avila M, Wheeler CM, Perez G, Koutsky LA, et al. Efficacy of a quadrivalent prophylactic human papillomavirus (types 6, 11, 16, and 18) L1 virus-like-particle vaccine against high-grade vulval and vaginal lesions: a combined analysis of three randomised clinical trials. Lancet 2007;369(9574):1693–1702.
- 26. Reisinger KS, Block SL, Lazcano-Ponce E, Samakoses R, Esser MT, Erick J, et al. Safety and persistent immunogenicity of a quadrivalent human papillomavirus types 6, 11, 16, 18 L1 virus-like particle vaccine in preadolescents and adolescents: a randomized controlled trial. *Pediatr Infect Dis J* 2007;26(3):201–209.
- 27. Therapeutic Goods Administration. Australian Public Assessment Report for Pneumococcal Polysaccharide Conjugate Vaccine (Prevenar 13®). Accessed on 7 August 2013. Available from: http://www.tga.gov.au/pdf/auspar/auspar-prevenar13.pdf
- 28. Simon LS. Pharmacovigilance: towards a better understanding of the benefit to risk ratio. *Ann Rheum Dis* 2002;61(Suppl II):ii88–ii89.

### **Abbreviations of vaccine types**

7vPCV 7-valent pneumococcal conjugate vaccine

10vPCV 10-valent pneumococcal conjugate vaccine

13vPCV 13-valent pneumococcal conjugate vaccine

23vPPV 23-valent pneumococcal polysaccharide vaccineBCG Bacille Calmette-Guérin

(i.e. tuberculosis)

dT diphtheria-tetanus – adolescent and adult formulation

DTPa diphtheria-tetanus-pertussis (acellular) – paediatric formulation

dTpa diphtheria-tetanus-pertussis (acellular) – adolescent and adult formulation

dTpa-IPV combined dTpa and inactivated poliovirus

DTPa-HepB combined diphtheria-tetanus-pertussis (acellular) and hepatitis B

DTPa-IPV combined diphtheria-tetanus-pertussis (acellular) and inactivated poliovirus

(quadrivalent)

DTPa-IPV-HepB combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus and

hepatitis B (pentavalent)

DTPa-IPV-HepB-Hib combined diphtheria-tetanus-pertussis (acellular), inactivated poliovirus,

hepatitis B and *Haemophilus influenzae* type b vaccine (hexavalent)

HepB hepatitis B

Hib *Haemophilus influenzae* type b

Hib-HepB combined *Haemophilus influenzae* type b and hepatitis B

HPV human papillomavirus

IPV inactivated poliovirus vaccine

Men4PV meningococcal polysaccharide tetravalent vaccine

MenCCV meningococcal C conjugate vaccine

MMR measles-mumps-rubella

pH1N1 pandemic H1N1 influenza 2009

# Australian Enterococcal Sepsis Outcome Progamme, 2011

Geoffrey W Coombs, Julie C Pearson, Denise A Daley, Tam Le, James O Robinson, Thomas Gottlieb, Benjamin P Howden, Paul DR Johnson, Catherine M Bennett, Timothy P Stinear, John D Turnidge for the Australian Group on Antimicrobial Resistance

### Abstract

From 1 January to 31 December 2011, 29 institutions around Australia participated in the Australian Enterococcal Sepsis Outcome Programme (AESOP). The aim of AESOP 2011 was to determine the proportion of enterococcal bacteraemia isolates in Australia that are antimicrobial resistant, with particular emphasis on susceptibility to ampicillin and the glycopeptides, and to characterise the molecular epidemiology of the Enterococcus faecalis and E. faecium isolates. Of the 1,079 unique episodes of bacteraemia investigated, 95.8% were caused by either E. faecalis (61.0%) or E. faecium (34.8%). Ampicillin resistance was detected in 90.4% of E. faecium but not detected in E. faecalis. Using Clinical and Laboratory Standards Institute breakpoints (CLSI), vancomycin non-susceptibility was reported in 0.6% and 31.4% of E. faecalis and E. faecium respectively and was predominately due to the acquisition of the vanB operon. Approximately 1 in 6 vanB E. faecium isolates however, had an minimum inhibitory concentration at or below the CLSI vancomycin susceptible breakpoint of  $\leq 4$  mg/L. Overall, 37% of E. faecium harboured vanA or vanB genes. Although molecular typing identified 126 E. faecalis pulsedfield gel electrophoresis (PFGE) pulsotypes, more than 50% belonged to 2 pulsotypes that were isolated across Australia. E. faecium consisted of 73 PFGE pulsotypes from which 43 multilocus sequence types were identified. Almost 90% of the E. faecium were identified as clonal complex 17 clones, of which approximately half were characterised as sequence type 203, which was isolated Australia-wide. In conclusion, the AESOP 2011 has shown that although polyclonal, enterococcal bacteraemias in Australia are frequently caused by ampicillin-resistant vanB E. faecium. Commun Dis Intell 2014;38(3):E247-E252.

Keywords: antimicrobial resistance surveillance; Enterococcus faecium, Enterococcus faecalis, vancomycin resistant, bacteraemia

### Introduction

Globally, enterococci are thought to account for approximately 10% of all bacteraemias, and in North America and Europe are the 4th and 5th leading cause of sepsis, respectively. Although

in the 1970s, healthcare-associated enterococcal infections were primarily due to Enterococcus faecalis,<sup>3</sup> there has been a steadily increasing prevalence of *E. faecium* nosocomial infections.<sup>4,5</sup> While innately resistant to many classes of antibiotics, E. faecium has demonstrated a remarkable capacity to evolve new antimicrobial resistances. By the early 1990s vancomycin resistant E. faecium had become the 2nd most common nosocomial pathogen in the United States of America (USA),6 and was endemic in many North American hospitals.<sup>7</sup> Vancomycin resistance in *E. faecium* bacteraemia isolates ranges from 5%-35% in Europe to 60% in North America.<sup>8,9</sup> In 2009, the Infectious Diseases Society of America highlighted E. faecium as one of the key problem bacteria or ESKAPE (Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumanii, Pseudomonas aeruginosa, and Enterobacter species) pathogens requiring new therapies.<sup>10</sup>

The Australian Group on Antimicrobial Resistance (AGAR) is a network of laboratories located across Australia that commenced surveillance of antimicrobial resistance in *Enterococcus* species in 1995. In 2011, AGAR commenced the Australian Enterococcal Sepsis Outcome Programme (AESOP). The objective of the AESOP 2011 was to determine the proportion of *E. faecalis* and *E. faecium* bacteraemia isolates demonstrating antimicrobial resistance with particular emphasis on:

- assessing susceptibility to ampicillin;
- assessing susceptibility to glycopeptides; and
- molecular epidemiology of *E. faecalis* and *E. faecium*.

### Methodology

### **Participants**

Twenty-nine laboratories including 26 public and 3 private laboratories from all 8 Australian states and territories participated in the AESOP 2011 study.

### Collection period

From 1 January to 31 December 2011, 29 laboratories collected all enterococcal species isolated from blood cultures. Enterococci with the same species and antimicrobial susceptibility profiles isolated

from a patient's blood culture within 14 days of the 1st positive culture were excluded. A new enterococcal sepsis episode in the same patient was recorded if it was confirmed by a further culture of blood taken more than 14 days after the initial positive culture.

### Laboratory testing

Ampicillin susceptibility testing was performed according to each laboratory's routine standardised methodology. Clinical and Laboratory Standards Institute (CLSI) breakpoints were utilised for interpretation.<sup>12</sup> Of the 1,033 E. faecalis and E. faecium sepsis isolates, 963 (93.2%) were referred to the Australian Collaborating Centre for Enterococcus and Staphylococcus Species (ACCESS) Typing and Research for vancomycin and teicoplanin minimum inhibitory concentration (MIC) estimation by Etest (bioMérieux) according to the manufacturer's guidelines. Isolates with a CLSI intermediate or resistant category were classified as non-susceptible. Molecular testing including *vanA/B* polymerase chain reaction, pulsed-field gel electrophoresis (PFGE) and multilocus sequence typing (MLST) was performed as previously described. [3-15]

### **Results**

From 1 January to 31 December 2011, 1,079 unique episodes of enterococcal bacteraemia were identified. Although 8 *Enterococcus* species were identified, 61.0% (658 isolates) were *E. faecalis* and 34.8% (375) were *E. faecium*. Forty-five enterococci were identified either as *E. casseliflavus* (15 isolates), *E. gallinarum* (14), *E. avium* (8), *E. raffinosus* (4), *E. durans* (2) or *E. hirae* (2). One isolate could not be identified to the species level.

### Phenotypic susceptibility results

Overall 90.4% (339) of the 375 isolates of *E. fae*cium were ampicillin resistant. Ampicillin resistance was not detected in the 658 E. faecalis isolates. Vancomycin and teicoplanin MICs were performed on the 622 E. faecalis and 341 E. faecium referred to ACCESS Typing and Research. The vancomycin MICs for the *E. faecalis* isolates ranged from 0.25->256 mg/L with a mode of 2 mg/L. The 3 vancomycin non-susceptible isolates (MIC >4 mg/L) had MICs of 16, 32 and >256 mg/L, respectively. The E. faecalis teicoplanin MICs ranged from 0.064-2 mg/L with a mode of 0.25 mg/L. None of the E. faecalis isolates had a teicoplanin MIC above the CLSI susceptible breakpoint of  $\leq 8$  mg/L. The vancomycin MICs for the *E. faecium* isolates ranged from 0.25–>256 mg/L with a mode of 1.0 mg/L. Overall, 31.4% (107 isolates) of E. faecium had a

vancomycin MIC >4 mg/L. The *E. faecium* teicoplanin MICs ranged from 0.047–>256 mg/L, with a mode of 0.5 mg/L. Five isolates had a teicoplanin MIC >8 mg/L.

### Genotypic vancomycin susceptibility results

The 3 vancomycin non-susceptible E. faecalis isolates harboured a vanB gene. Two and 104 of the 107 E. faecium vancomycin non-susceptible isolates harboured vanA and vanB genes respectively. The *vanA/vanB* negative vancomycin non-susceptible E. faecium isolate had a MIC of 6 mg/L. Twenty of the 234 vancomycin susceptible E. faecium isolates (MIC  $\leq 4$  mg/L) also harboured vanB genes. The 2 vanA E. faecium isolates had a vancomycin MIC >256 mg/L. Thirty-three vancomycin non-susceptible E. faecium isolates had a vancomycin MIC within the CLSI intermediate category of 8–16 mg/L. Only 71 (57.2%) of the 124 vanB E. faecium isolates had a MIC above the CLSI vancomycin resistant breakpoint ( $\geq$ 32 mg/L). The 2 vanA E. faecium isolates had teicoplanin MICs of 32 and 64 mg/L. Of the 124 vanB E. faecium isolates one was teicoplanin intermediate (MIC 16 mg/L) and three were resistant (MIC >32 mg/L) by CLSI criteria.

### Molecular epidemiology

By PFGE, 618 of the 622 *E. faecalis* were classified into 126 pulsotypes of which 9 pulsotypes (Efs1 to Efs9) had 10 or more isolates. Four isolates could not be typed by PFGE. Of the 117 pulsotypes that have less than 10 isolates, 66 pulsotypes were represented by only 1 isolate. Geographically, the 9 major pulsotypes were widely distributed, with the 2 predominant pulsotypes EFs1 (191 isolates) and Efs2 (103 isolates), isolated across Australia (Table 1). The 3 *vanB E. faecalis* were detected in pulsotype Efs2.

By PFGE, the 341 E. faecium isolates were classified into 73 pulsotypes from which 43 multilocus sequence types (STs) were identified. Five STs had more than 10 isolates (Table 2). The 2 major STs, ST203 (159 isolates) and ST17 (47 isolates) were isolated across Australia. ST341 (38 isolates) and ST252 (11 isolates) were isolated only in the eastern regions of Australia, and ST555 (34 isolates) was isolated in the western and central regions. Using eBURST, 89.1% (304/341 isolates) of *E. faecium* isolates, including the 5 major STs, were grouped into clonal complex (CC) 17. Van genes were identified in the 5 major STs and in ST80 and ST414. Although only 8.5% (4/47) of ST17 isolates harboured vanB, vanB genes were identified in 50.6% (79/153) of ST203 isolates. Other STs harbouring the vanB genes included ST341 (33/38), ST555 (2/34), ST252 (1/11), ST80 (2/6), ST414 (2/2), and a single non-CC17 isolate, ST863. Two CC17 STs harboured the vanA gene; ST341 and ST80.

Table 1: Number and proportion of major Enterococcus faecalis pulsed-field gel electrophoresis pulsotypes, Australia, 2011, by state or territory

	Ā	ACT	ž	NSW	Z	۲	o	QId	o)	SA	Ta	Tas.	>	Vic.	3	WA	Aus.	IS.
Type	٦	%	_	%	_	<b>%</b>	_	%	_	%	_	<b>%</b>	_	%	_	%	٦	%
Efs1	10	30.3	22	33.3	2	28.6	22	32.2	18	25.4	9	40.0	31	33.7	12	19.4	191	30.7
Efs2	6	27.3	21	12.3	2	28.6	29	17.0	17	23.9	က	20.0	13	14.1	6	14.5	103	16.6
Efs3	0	0.0	9	3.5	0	0.0	~	9.0	7	2.8	_	6.7	7	2.2	_	1.6	13	2.1
Efs4	0	0.0	က	1.8	0	0.0	œ	4.7	0	0.0	0	0.0	~	1.1	_	1.6	13	2.1
Efs5	0	0.0	7	6.4	0	0.0	~	9.0	0	0.0	0	0.0	0	0.0	0	0.0	12	1.9
Efs6	0	0.0	2	1.2	0	0.0	~	9.0	2	2.8	_	6.7	က	3.3	က	4.8	12	1.9
Efs7	_	3.0	က	1.8	0	0.0	2	2.9	2	2.8	0	0.0	~	1.1	0	0.0	12	1.9
Efs8	0	0.0	ဇ	1.8	0	0.0	7	1.2	က	4.2	0	0.0	~	1.7	7	3.2	7	1.8
Efs9	0	0.0	4	2.3	0	0.0	က	1.8	7	2.8	0	0.0	0	0.0	~	1.6	10	1.6
Other	13	39.4	09	35.1	ဇ	42.9	92	38.0	25	35.2	4	26.7	39	42.4	32	51.6	241	38.7
LN	0	0.0	~	9.0	0	0.0	-	9.0	0	0.0	0	0.0	_	1:1	~	1.6	4	9.0
Total	33	100.0	171	100.0	7	100.0	171	100.0	71	100.0	15	100.0	92	100.0	62	100.0	622	100.0

NT Not typed.

The category type 'other' includes the 117 pulsed-field gel electrophoresis pulsotypes that had less than 10 isolates per pulsotype.

Table 2: Number and proportion of major Enterococcus faecium multilocus sequence types, Australia, 2011, by state or territory

	4	ACT	SN.	NSM	Ł	F	QIQ	р	SA	<b>⋖</b> .	Ta	Tas.	Vic.	ناء	3	WA	Aus.	ÿ.
Type	٦	%	_	%	۵	%	_	%	_	%	_	%	_	%	_	%	_	%
Efm ST203	6	40.9	37	39.8	2	33.3	34	60.7	4	25.9	2	50.0	36	57.1	25	58.1	159	46.6
Efm ST17	က	13.6	19	20.4	0	0.0	7	12.5	7	3.7	7	20.0	7	11.1	7	16.3	47	13.8
Efm ST341	7	31.8	26	28.0	0	0.0	က	5.4	0	0.0	0	0.0	7	3.2	0	0.0	38	11.1
Efm ST555	0	0.0	0	0.0	ဗ	20.0	0	0.0	27	20.0	0	0.0	0	0.0	4	9.3	34	10.0
Efm ST252	0	0.0	7	2.1	0	0	က	5.4	0	0.0	0	0.0	9	9.5	0	0.0	7	3.2
Other	က	13.6	6	9.7	_	16.7	6	16.0	11	20.4	0	0.0	12	19.1	7	16.3	52	15.2
Total	22	100.0	93	100.0	9	100.0	26	100.0	54	100.0	4	100.0	63	100.0	43	100.0	341	100.0

The category type 'other' includes the 38 multilocus sequence types that had less than 10 isolates per sequence type.

### **Discussion**

AESOP 2011 was the 1st ongoing sepsis program performed by AGAR, and was conducted primarily to determine the proportion of *E. faecalis* and *E. faecium* bacteraemia isolates demonstrating antimicrobial resistance, with particular emphasis on assessing susceptibility to penicillin and the glycopeptides and the distribution of different enterococcal clones. As this is the 1st year of an ongoing program, it is difficult to determine the public health significance of these results. We suggest these results should be used as a marker for future AESOPs.

Similar to the situation in the USA and in Europe, 16,17 enterococcal bacteraemia in Australia, and notably bacteraemia caused by multidrugresistant E. faecium, has become a significant problem. In the AESOP 2011 study, approximately 1 in 3 cases of enterococcal bacteraemia was due to *E. faecium*, of which 90.4% (339 of 375 episodes) were ampicillin resistant and 36.9% (126 of 341 episodes) harboured either vanA or vanB genes. However, unlike Europe and the USA, where vancomycin resistance in E. faecium has predominately been due to the acquisition of the vanA operon, almost all of the AESOP 2011 E. faecium blood culture isolates harbouring van genes carried the *vanB* operon (98.4%). Recent studies however, have demonstrated a significant presence of vanB E. faecium in both North America and Europe.<sup>18–20</sup>

vanB vancomycin resistant enterococci (VRE) is now more prevalent than vanA VRE in several European centres including Sweden, Spain and Germany, 21-23 whilst recent Canadian national surveillance demonstrates vanB comprises 10% of their VRE.<sup>24</sup> It is thought this increased occurrence of vanB-positive E. faecium in the Northern Hemisphere may be due to the increased use of antibiotics selecting enterococci and VRE as well as to methodological reasons (e.g. reduced European Committee on Antimicrobial Susceptibility Testing (EUCAST) MIC breakpoints for glycopeptides; increased use and sensitive performance of chromogenic VRE agars; increased use of molecular diagnostic assays).<sup>25</sup> Twenty (16.1%) of the 124 vanB E. faecium isolates had a vancomycin MIC at or below the CLSI and the EUCAST susceptible breakpoint (≤4 mg/L) and would not have been identified using routine phenotypic antimicrobial susceptibility methods.

With the use of PFGE, both enterococcal species were shown to be very polyclonal, confirming the enormous plasticity of the enterococcal genome. The majority of *E. faecium* isolates formed part of CC17, a global hospital-derived lineage that

has successfully adapted to hospital environments. CC17 is characteristically ampicillin and quinolone resistant and subsequent acquisition of *vanA*— or *vanB*-containing transposons by horizontal transfer in CC17 clones has resulted in VRE with pandemic potential.

The study has a number of limitations. Although achieving national coverage, the participating laboratories service only a minority of the Australian hospitalised population. Further, MIC assays for vancomycin and teicoplanin were performed by a commercial gradient diffusion method and not the standard reference broth microdilution method.

### **Conclusions**

The AESOP 2011 study has shown though predominately caused by *E. faecalis*, enterococcal bacteraemia in Australia is frequently caused by ampicillin-resistant *vanB E. faecium*. Molecular typing characterised over 50% of *E. faecalis* isolates as 2 PFGE pulsotypes and almost 90% of *E. faecium* isolates as CC17 clones of which approximately half were ST203. Further studies of the enterococcal genome will contribute to our understanding of the evolution of enterococci in the hospital environment and assist in preventing their nosocomial transmission.

### **Acknowledgements**

We gratefully acknowledge Hui-leen Tan, Yung Ching Lee and Lynne Wilson from the Department of Microbiology and Infectious Diseases, PathWest Laboratory Medicine WA, Royal Perth Hospital; Frances O'Brien and Ka Yan Wong from the Australian Collaborating Centre for *Enterococcus* and *Staphylococcus* Species (*ACCESS*) Typing and Research, School of Biomedical Sciences, Curtin University; and the Western Australia Genome Resource Centre, Department of Clinical Immunology and Biochemical Genetics, Royal Perth Hospital for the molecular typing of enterococci.

Members of the AGAR in 2011 were:

### **Australian Capital Territory**

Peter Collignon and Susan Bradbury, The Canberra Hospital

### **New South Wales**

Tom Gottlieb and Graham Robertson, Concord Hospital

Miriam Paul and Richard Jones, Douglass Hanly Moir Pathology James Branley and Donna Barbaro, Nepean Hospital

George Kotsiou and Peter Huntington, Royal North Shore Hospital

Colin MacLeod and Bradley Watson, Royal Prince Alfred Hospital

Iain Gosbell and Annabelle LeCordier, Liverpool Hospital

David Mitchell and Lee Thomas, Westmead Hospital

### **Northern Territory**

Jann Hennessy and Rob Baird, Royal Darwin Hospital

### Queensland

Enzo Binotto and Bronwyn Thomsett, Pathology Queensland Cairns Base Hospital

Graeme Nimmo and Narelle George, Pathology Queensland Central Laboratory

Petra Derrington and Sharon Dal-Cin, Pathology Queensland Gold Coast Hospital

Chris Coulter and Sonali Coulter, Pathology Queensland Prince Charles Hospital

Joan Faoagali and Joel Douglas, Pathology Queensland Princess Alexandra Hospital

Jenny Robson and Georgia Peachey, Sullivan Nicolaides Pathology

### South Australia

Kelly Papanoum and Nicholas Wells, SA Pathology, Flinders Medical Centre

Morgyn Warner and Fleur Manno, SA Pathology, Royal Adelaide Hospital

John Turnidge and Jan Bell, SA Pathology, Women's and Children's Hospital

#### **Tasmania**

Mhisti Rele and Kathy Wilcox, Launceston General Hospital

Louise Cooley and Rob Peterson, Royal Hobart Hospital

### **Victoria**

Denis Spelman and Michael Huysmans, The Alfred Hospital

Benjamin Howden and Peter Ward, Austin Hospital

Tony Korman and Despina Kotsanas, Monash Medical Centre

Suzanne Garland and Gena Gonis, Royal Women's Hospital

Mary Jo Waters and Linda Joyce, St Vincent's Hospital

#### Western Australia

David McGechie and Rebecca Wake, PathWest Laboratory Medicine, Fremantle Hospital

Barbara Henderson and Ronan Murray, PathWest Laboratory Medicine, Queen Elizabeth II Hospital

Keryn Christiansen, Denise Daley and Geoffrey Coombs, PathWest Laboratory Medicine, Royal Perth Hospital

Victoria D'Abrera and Sindy Budalich, St John of God Pathology

### **Author details**

Dr Geoffrey W Coombs<sup>1,2</sup>
Ms Julie C Pearson<sup>2</sup>
Ms Denise A Daley<sup>3</sup>
Ms Tam Le<sup>1</sup>
Dr James O Robinson<sup>1,2</sup>
Dr Thomas Gottlieb<sup>4</sup>
Dr Benjamin P Howden<sup>5,6</sup>
Dr Paul DR Johnson<sup>5</sup>
Dr Catherine M Bennett<sup>7</sup>
Dr Timothy P Stinear<sup>6</sup>
Prof John D Turnidge<sup>8,9</sup>

- Australian Collaborating Centre for Enterococcus and Staphylococcus Species (ACCESS) Typing and Research, School of Biomedical Sciences, Curtin University, Perth, Western Australia
- Department of Microbiology and Infectious Diseases, PathWest Laboratory Medicine, WA, Royal Perth Hospital, Perth, Western Australia
- 3. Australian Group on Antimicrobial Resistance, Royal Perth Hospital, Perth, Western Australia
- Department of Microbiology and Infectious Diseases, Concord Hospital, Concord, New South Wales
- Microbiology Department, Austin Health, Heidelberg, Victoria
- 6. Department of Microbiology and Immunology, University of Melbourne, Melbourne, Victoria
- 7. Population Health, Deakin University, Melbourne
- SA Pathology, Department of Microbiology and Infectious Diseases, Women's and Children's Hospital, North Adelaide, South Australia

 Departments of Pathology, Paediatrics and Molecular and Biomedical Sciences, University of Adelaide, Adelaide, South Australia

Corresponding author: Dr Geoffrey Coombs, Australian Collaborating Centre for Enterococcus and Staphylococcus Species (ACCESS) Typing and Research, School of Biomedical Sciences, Curtin University, Perth, Western Australia. Telephone: +61 8 9224 2446. Facsimile: +61 8 9224 1989. Email: Geoff.Coombs@curtin.edu.au

### References

- Pinholt M, Ostergaard C, Arpi M, Bruun NE, Schønheyder HC, Gradel KO, et al. Incidence, clinical characteristics and 30-day mortality of enterococcal bacteraemia in Denmark 2006–2009: a population-based cohort study. Clin Microbiol Infect 2014;20(2):145–515.
- Deshpande LM, Fritsche TR, Moet GJ, Biedenbach DJ, Jones RN. Antimicrobial resistance and molecular epidemiology of vancomycin-resistant enterococci from North America and Europe: a report from the SENTRY antimicrobial surveillance program. Diagn Microbiol Infect Dis 2007;58(2):163–170.
- 3. Murray BE. The life and times of the *Enterococcus*. Clin Microbiol Rev 1990;3(1):46–65.
- Simonsen GS, Småbrekke L, Monnet DL, Sørensen TL, Moller JK, Kristinsson KG, et al. Prevalence of resistance to ampicillin, gentamicin and vancomycin in Enterococcus faecalis and Enterococcus faecium isolates from clinical specimens and use of antimicrobials in five Nordic hospitals. J Antimicrob Chemother 2003;51(2):323–331.
- Treitman AN, Yarnold PR, Warren J, Noskin GA. Emerging incidence of Enterococcus faecium among hospital isolates (1993 to 2002). J Clin Microbiol 2005;43(1):462–463.
- Frieden TR, Munsiff SS, Low DE, Willey BM, Williams G, Faur Y, et al. Emergence of vancomycin-resistant enterococci in New York City. Lancet 1993;342 (8863):76–79.
- Murray BE. Vancomycin-resistant enterococcal infections. N Engl J Med 2000;342(10):710–721.
- 8. European Centre for Disease Prevention and Control. European Antimicrobial Resistance Surveillance Network. [online]. Accessed on April 2014. Available from: http://www.ecdc.europa.eu/en/activities/surveillance/EARS-Net/Pages/index.aspx
- Bearman GM, Wenzel RP. Bacteremias: A leading cause of death. Arch Med Res 2005 36(6):646–659.
- Boucher HW, Talbot GH, Bradley JS, Edwards JE, Gilbert D, Rice LB, et al. Bad bugs, no drugs: no ESKAPE! An update from the Infectious Diseases Society of America. Clin Infect Dis 2009;48(1):1–12.
- Christiansen KJ, Turnidge JD, Bell JM, George NM, Pearson JC, Australian Group on Antimicrobial Resistance. Prevalence of antimicrobial resistance in Enterococcus isolates in Australia, 2005: Report from the Australian Group on Antimicrobial Resistance. Commun Dis Intell 2007;31(4):392–397.
- Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. Twentysecond informational supplement M100-S22. Villanova, PA, USA; 2012.

- Kulski JK, Wilson RD, Bending R, Grubb W. Antibiotic resistance and genomic analysis of enterococci in an intensive care unit and general wards. *Pathology* 1998;30(1):68–72.
- Tenover FC, Arbeit RD, Goering RV, Mickelsen PA, Murray BE, Persing DH, et al. Interpreting chromosomal DNA restriction patterns produced by pulsed-field gel electrophoresis: Criteria for bacterial strain typing. J Clin Microbiol 1995;33(9):2233–2239.
- Homan WL, Tribe D, Poznanski S, Li M, Hogg G, Spalburg E, et al. Multilocus sequence typing scheme for Enterococcus faecium. J Clin Microbiol 2002;40(6):1963–1971.
- Wisplinghoff H, Bischoff T, Tallent SM, Seifert H, Wenzel RP, Edmond MB. Nosocomial bloodstream infections in US hospitals: Analysis of 24,179 cases from a prospective nationwide surveillance study. Clin Infect Dis 2004;39(3):309–317.
- de Kraker ME, Jarlier V, Monen JC, Heuer OE, van de Sande N, Grundmann H. The changing epidemiology of bacteraemias in Europe: Trends from the European Antimicrobial Resistance Surveillance System. Clin Microbiol Infect 2013;19(9):860–868.
- Klare I, Fleige C, Geringer U, Witte W, Werner G. Performance of three chromogenic VRE screening agars, two Etest® vancomycin protocols, and different microdilution methods in detecting vanB genotype Enterococcus faecium with varying vancomycin MICs. Diagn Microbiol Infect Dis 2012;74(2):171–176.
- Mendes RE, Farrell DJ, Sader HS, Jones RN. Oritavancin microbiologic features and activity results from the surveillance program in the United States. Clin Infect Dis 2012;54(Suppl 3):S203–S213.
- Jones RN, Sader HS, Flamm RK. Update of dalbavancin spectrum and potency in the USA: report from the SENTRY Antimicrobial Surveillance Program (2011). Diagn Microbiol Infect Dis 2013;75(3):304–307.
- 21. Soderblom T, Aspevall O, Erntell M, Hedin G, Heimer D, Hokeberg I, et al. Alarming spread of vancomycin resistant enterococci in Sweden since 2007. Euro Surveill 2010;15(29):pii: 19620.
- López M, Cercenado E, Tenorio C, Ruiz-Larrea F, Torres C. Diversity of clones and genotypes among vancomycin-resistant clinical *Enterococcus* isolates recovered in a Spanish hospital. *Microb Drug Resist* 2012;18(5):484–491.
- 23. Werner G, Klare I, Fleige C, Geringer U, Witte W, Just HM, et al. Vancomycin-resistant vanB-type Enterococcus faecium isolates expressing varying levels of vancomycin resistance and being highly prevalent among neonatal patients in a single ICU. Antimicrob Res Infect Control 2012;1(1):21.
- McCracken M, Wong A, Mitchell R, Gravel D, Conly J, Embil J, et al. Molecular epidemiology of vancomycinresistant enterococcal bacteraemia: results from the Canadian Nosocomial Infection Surveillance Program, 1999–2009. J Antimicrob Chemother 2013;68(7):1505– 1509.
- Klarel, Witte W, Wendt C, Werner G. [Vancomycin-resistant enterococci (VRE). Recent results and trends in development of antibiotic resistance]. Bundesgesundheitsblatt, Gesundheitsforschung, Gesundheitsschutz 2012;55(11– 12):1387–400. German

## Quarterly reports

# National Notifiable Diseases Surveillance System, 1 April to 30 June 2014

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 54,644 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 April to 30 June 2014 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
STEC, VTEC*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis - congenital	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis > 2 years or unspecified duration	All jurisdictions

CDI Vol 38 No 3 2014 E253

Table 1 continued: Reporting of notifiable diseases by jurisdiction

Vaccine preventable diseases   Diphtheria	Disease	Data received from:
Haemophilus influenzae type b         All jurisdictions           Influenza (laboratory confirmed)         All jurisdictions           Measles         All jurisdictions           Mumps         All jurisdictions           Pertussis         All jurisdictions           Pneumococcal disease (invasive)         All jurisdictions           Poliomyelitis         All jurisdictions           Rubella         All jurisdictions           Rubella - congenital         All jurisdictions           Varicella zoster (chickenpox)         All jurisdictions except New South Wales           Varicella zoster (thickenpox)         All jurisdictions except New South Wales           Varicella zoster (unspecified)         All jurisdictions except New South Wales           Varicella zoster (unspecified)         All jurisdictions except New South Wales           Varicella zoster (inspecified)         All jurisdictions except New South Wales           Vectorborne diseases         Arbovirus infection (NEC)         All jurisdictions           Barmah Forest virus infection         All jurisdictions           Japanese encephalitis virus infection         All jurisdictions           Kunjin virus infection         All jurisdictions           Ruser virus infection         All jurisdictions           Ross River virus infection         All jurisdictions	Vaccine preventable diseases	
Influenza (laboratory confirmed) Measles Mumps All jurisdictions Mumps Pertussis All jurisdictions Pneumococcal disease (invasive) All jurisdictions Rubella All jurisdictions Rubella - congenital All jurisdictions Rubella - congenital 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 Varicella zoster (unspecified) All jurisdictions except New South Wales Varicella zoster (unspecified) All jurisdictions except New South Wales Varicella zoster (unspecified) All jurisdictions	Diphtheria	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           Yaricella 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           Varicella zoster (unspecified)         All jurisdictions except New South Wales           Varicella zoster (unspecified)         All jurisdictions except New South Wales           Varicella zoster (unspecified)         All jurisdictions           Lapinase encephalitis virus infection         All jurisdictions           Murray Valley encephalitis virus infection         All jurisdictions	Haemophilus influenzae type b	All jurisdictions
Mumps Pertussis All jurisdictions Pneumococcal disease (invasive) Poliomyelitis All jurisdictions Poliomyelitis All jurisdictions Rubella All jurisdictions Rubella All jurisdictions Rubella - congenital All jurisdictions Rubella - congenital All jurisdictions Rubella - congenital 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 Varicella zoster (unspecified) All jurisdictions except New South Wales Vectorborne diseases Vectorborne diseases Arbovirus infection (NEC) All jurisdictions Barmah Forest virus infection All jurisdictions Dengue virus infection All jurisdictions All jurisdictions All jurisdictions All jurisdictions Murary Valley encephalitis virus infection All jurisdictions Murray Valley encephalitis virus infection All jurisdictions All jurisdictions All jurisdictions Australian bat lyssavirus All jurisdictions Prucellosis All jurisdictions All jurisdictions All jurisdictions All jurisdictions All jurisdictions Cornithosis All jurisdictions	Influenza (laboratory confirmed)	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 (unspecified)         All jurisdictions except New South Wales           Varicella zoster (unspecified)         All jurisdictions except New South Wales           Varicella zoster (unspecified)         All jurisdictions except New South Wales           Vectorborne diseases         Vectorborne diseases           Arbovirus infection (NEC)         All jurisdictions           Barmah Forest virus infection         All jurisdictions           Dengue virus infection         All jurisdictions           Kunjin virus infection         All jurisdictions           Murray Valley encephalitis virus infection         All jurisdictions           Murray Valley encephalitis virus infection         All jurisdictions           Rose River virus infection         All jurisdictions           Australian bat lyssavirus         All jurisdictions           Bruccellosis         All jurisdictions           Leptospirosis <td>Measles</td> <td>All jurisdictions</td>	Measles	All jurisdictions
Pneumococcal disease (invasive) Poliomyelitis Rubella Rubella - congenital Rubella - congenital All jurisdictions Rubella - congenital All jurisdictions All jurisdictions Tetanus Varicella zoster (chickenpox) All jurisdictions except New South Wales Varicella zoster (shingles) Varicella zoster (shingles) All jurisdictions except New South Wales Varicella zoster (unspecified) All jurisdictions except New South Wales Varicella zoster (unspecified) All jurisdictions except New South Wales Vectorborne diseases All jurisdictions All jurisdictions All jurisdictions All jurisdictions All jurisdictions Dengue virus infection All jurisdictions All jurisdictions All jurisdictions All jurisdictions Malaria All jurisdictions Murray Valley encephalitis virus infection All jurisdictions All jurisdictions All jurisdictions All jurisdictions All jurisdictions All jurisdictions Australian bat lyssavirus All jurisdictions	Mumps	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         All jurisdictions           Arbovirus infection (NEC)         All jurisdictions           Barmah Forest virus infection         All jurisdictions           Dengue virus infection         All jurisdictions           Muriani (Aurian)         All jurisdictions           Mularia         All jurisdictions           Murray Valley encephalitis virus infection         All jurisdictions           Murray Valley encephalitis virus infection         All jurisdictions           Ross River virus infection         All jurisdictions           Australian bat lyssavirus         All jurisdictions           Brucellosis         All jurisdictions           Leptospirosis         All jurisdictions           Lyssavirus (NEC)         All jurisdictions           Ornithosis         All jurisdictions           Q fev	Pertussis	All jurisdictions
Rubella - congenital 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 Dengue virus infection All jurisdictions Japanese encephalitis virus infection All jurisdictions Kunjin virus infection All jurisdictions Murray Valley encephalitis virus infection All jurisdictions Murray Valley encephalitis virus infection All jurisdictions Ross River virus infection All jurisdictions All jurisdictions  Australian bat lyssavirus All jurisdictions Brucellosis Leptospirosis Leptospirosis Leptospirosis Leptospirosis All jurisdictions All jurisdictions Ornithosis All jurisdictions All jurisdictions All jurisdictions  All jurisdictions  All jurisdictions  All jurisdictions  All jurisdictions  Leptospirosis All jurisdictions All jurisdictions  All jurisdictions	Pneumococcal disease (invasive)	All jurisdictions
Rubella - congenital 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 Varicella zoster (unspecified) All jurisdictions except New South Wales Vectorborne diseases  Vectorborne diseases Arbovirus infection (NEC) Barmah Forest virus infection All jurisdictions Barmah Forest virus infection All jurisdictions Dengue virus infection All jurisdictions All jurisdictions All jurisdictions All jurisdictions Murray Valley encephalitis virus infection All jurisdictions Murray Valley encephalitis virus infection All jurisdictions All jurisdictions All jurisdictions  Poonoses  Anthrax All jurisdictions All jurisdictions Australian bat lyssavirus All jurisdictions  Ceptospirosis All jurisdictions	Poliomyelitis	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  Murray Valley encephalitis virus infection All jurisdictions  Murray Valley encephalitis virus infection All jurisdictions  Ross River virus infection All jurisdictions  Authrax All jurisdictions  Australian bat lyssavirus All jurisdictions  Brucellosis All jurisdictions  Leptospirosis All jurisdictions  Leptospirosis All jurisdictions  Q fever All jurisdictions  Ortitosis All jurisdictions  Orter bacterial infections  Legionellosis All jurisdictions  Leprosy All jurisdictions	Rubella	All jurisdictions
Varicella zoster (chickenpox) Varicella zoster (shingles) All jurisdictions except New South Wales Varicella zoster (unspecified) All jurisdictions except New South Wales  Vectorborne diseases  Arbovirus infection (NEC) Barmah Forest virus infection Dengue virus infection All jurisdictions Dengue virus infection All jurisdictions	Rubella - congenital	All jurisdictions
Varicella zoster (shingles) Varicella zoster (unspecified) All jurisdictions except New South Wales  Vectorborne diseases  Arbovirus infection (NEC) Barmah Forest virus infection Dengue virus infection All jurisdictions Japanese encephalitis virus infection All jurisdictions All jurisdictions All jurisdictions Munipi virus infection All jurisdictions Mulrary Valley encephalitis virus infection All jurisdictions Murray Valley encephalitis virus infection All jurisdictions Ross River virus infection All jurisdictions  Authrax Authrax All jurisdictions  All jurisdictions  Brucellosis All jurisdictions  Leptospirosis Leptospirosis All jurisdictions All jurisdictions  All jurisdictions	Tetanus	All jurisdictions
Varicella zoster (unspecified)  Vectorborne diseases  Arbovirus infection (NEC)  Barmah Forest virus infection  Dengue virus infection  All jurisdictions  All jurisdictions  Dengue virus infection  All jurisdictions  All jurisdictions  All jurisdictions  All jurisdictions  Kunjin virus infection  All jurisdictions  Mularia  All jurisdictions  Murray Valley encephalitis virus infection  All jurisdictions  Murray Valley encephalitis virus infection  All jurisdictions  All jurisdictions  All jurisdictions  All jurisdictions  All jurisdictions  All jurisdictions  Australian bat lyssavirus  All jurisdictions  Brucellosis  Leptospirosis  Leptospirosis  All jurisdictions  All jurisdictions  All jurisdictions  Q fever  All jurisdictions  All jurisdictions  Q fever  All jurisdictions  All jurisdictions  All jurisdictions  Cornithosis  All jurisdictions	Varicella zoster (chickenpox)	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         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	Varicella zoster (shingles)	All jurisdictions except New South Wales
Arbovirus infection (NEC) Barmah Forest virus infection Dengue virus infection All jurisdictions All jurisdictions Japanese encephalitis virus infection All jurisdictions Kunjin 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 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 Q fever All jurisdictions All jurisdictions Q fever All jurisdictions	Varicella zoster (unspecified)	All jurisdictions except New South Wales
Barmah Forest virus infection Dengue virus infection All jurisdictions Japanese encephalitis virus infection Kunjin 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 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 Q fever All jurisdictions Q fever All jurisdictions Q fever All jurisdictions  Other bacterial infections  Legionellosis Leprosy All jurisdictions	Vectorborne diseases	
Dengue virus infection Japanese encephalitis virus infection Kunjin 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 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 Q fever All jurisdictions  Other bacterial infections  Legionellosis Leprosy All jurisdictions	Arbovirus infection (NEC)	All jurisdictions
Japanese encephalitis virus infection Kunjin virus infection Malaria All jurisdictions Murray Valley encephalitis virus infection All jurisdictions Murray Valley encephalitis virus infection Ross River virus infection All jurisdictions  Zoonoses  Anthrax All jurisdictions Australian bat lyssavirus Brucellosis Leptospirosis Leptospirosis Lyssavirus (NEC) Ornithosis Q fever All jurisdictions All jurisdictions Q fever All jurisdictions All jurisdictions Unitrose  Cother bacterial infections Legionellosis Leprosy All jurisdictions	Barmah Forest virus infection	All jurisdictions
Kunjin virus infection Malaria All jurisdictions Murray Valley encephalitis virus infection Ross River virus infection All jurisdictions Ross River virus infection All jurisdictions  Zoonoses  Anthrax Australian bat lyssavirus All jurisdictions Brucellosis All jurisdictions Leptospirosis All jurisdictions Lyssavirus (NEC) All jurisdictions Q fever All jurisdictions Q fever All jurisdictions Tularaemia All jurisdictions  Other bacterial infections Legionellosis All jurisdictions All jurisdictions All jurisdictions Other bacterial infections All jurisdictions	Dengue 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	Japanese encephalitis virus infection	All jurisdictions
Murray Valley encephalitis virus infection Ross River virus infection  All jurisdictions  All jurisdictions  Anthrax All jurisdictions  Australian bat lyssavirus Brucellosis All jurisdictions  All jurisdictions  Leptospirosis All jurisdictions  Lyssavirus (NEC) All jurisdictions  All jurisdictions  Q fever All jurisdictions  All jurisdictions  All jurisdictions  Q fever All jurisdictions	Kunjin virus infection	All jurisdictions
Ross River virus infection  Zoonoses  Anthrax All jurisdictions Australian bat lyssavirus Brucellosis All jurisdictions Leptospirosis Leptospirosis Lyssavirus (NEC) All jurisdictions Ornithosis Q fever All jurisdictions Tularaemia All jurisdictions  Other bacterial infections  Leptospirosis All jurisdictions All jurisdictions All jurisdictions All jurisdictions All jurisdictions All jurisdictions  Other bacterial infections  Leptosy All jurisdictions	Malaria	All jurisdictions
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	Murray Valley encephalitis virus infection	All jurisdictions
Anthrax Australian bat lyssavirus Brucellosis All jurisdictions All jurisdictions Leptospirosis All jurisdictions Lyssavirus (NEC) All jurisdictions Ornithosis All jurisdictions All jurisdictions Q fever All jurisdictions Tularaemia All jurisdictions Other bacterial infections Legionellosis Leprosy Meningococcal infection All jurisdictions	Ross River virus infection	All jurisdictions
Australian bat lyssavirus Brucellosis Leptospirosis Leptospirosis All jurisdictions Lyssavirus (NEC) All jurisdictions Ornithosis Q fever All jurisdictions Tularaemia All jurisdictions Other bacterial infections Legionellosis Leprosy Meningococcal infection  All jurisdictions	Zoonoses	
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 All jurisdictions	Anthrax	All jurisdictions
Leptospirosis All jurisdictions Lyssavirus (NEC) All jurisdictions Ornithosis All jurisdictions Q fever All jurisdictions Tularaemia All jurisdictions  Other bacterial infections  Legionellosis Leprosy Meningococcal infection All jurisdictions All jurisdictions All jurisdictions All jurisdictions All jurisdictions All jurisdictions	Australian bat lyssavirus	All jurisdictions
Lyssavirus (NEC)  Ornithosis  All jurisdictions  Q fever  All jurisdictions  Tularaemia  Other bacterial infections  Legionellosis  Leprosy  Meningococcal infection  All jurisdictions  All jurisdictions  All jurisdictions  All jurisdictions  All jurisdictions  All jurisdictions	Brucellosis	All jurisdictions
Ornithosis Q fever All jurisdictions Tularaemia All jurisdictions  Other bacterial infections  Legionellosis Leprosy Meningococcal infection  All jurisdictions All jurisdictions All jurisdictions All jurisdictions All jurisdictions	Leptospirosis	All jurisdictions
Q fever All jurisdictions Tularaemia All jurisdictions  Other bacterial infections  Legionellosis All jurisdictions  Leprosy All jurisdictions  Meningococcal infection All jurisdictions	Lyssavirus (NEC)	All jurisdictions
Tularaemia All jurisdictions  Other bacterial infections  Legionellosis All jurisdictions  Leprosy All jurisdictions  Meningococcal infection All jurisdictions	Ornithosis	-
Tularaemia All jurisdictions  Other bacterial infections  Legionellosis All jurisdictions  Leprosy All jurisdictions  Meningococcal infection All jurisdictions	Q fever	All jurisdictions
Other bacterial infections       Legionellosis     All jurisdictions       Leprosy     All jurisdictions       Meningococcal infection     All jurisdictions	Tularaemia	
Leprosy All jurisdictions  Meningococcal infection All jurisdictions	Other bacterial infections	·
Meningococcal infection All jurisdictions	Legionellosis	All jurisdictions
Meningococcal infection All jurisdictions	Leprosy	All jurisdictions
	Meningococcal infection	All jurisdictions
		-

<sup>\*</sup> Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (STEC/VTEC). NEC Not elsewhere classified.

E254 CDI Vol 38 No 3 2014

Table 2: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2014, by date of diagnosis\*

			•	State or t	erritory				Total 2nd	Total 1st	Total 2nd	Last 5 years		Year	Last 5 years
Disease	ACT	NSM	۲	Qid	SA	Tas	Vic	WA	quarter 2014	quarter 2013	quarter 2013	mean znd quarter	Ratio	to date 2014	Y I D mean
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) <sup>†</sup>	_	1	7	6	0	0	41	7	44	28	43	9.03	6.0	102	106.2
Hepatitis B (unspecified) <sup>‡</sup>	26	631	46	239	26	20	458	137	1,654	1,644	1,765	1,669.4	1.0	3,283	3,374.6
Hepatitis C (newly acquired)⁺	9	2	~	0	7	4	30	44	101	86	78	105.6	1.0	199	216.8
Hepatitis C (unspecified) <sup>‡</sup>	44	890	47	663	66	61	518	256	2,578	2,515	2,494	2,553.0	1.0	5,066	5,168.0
Hepatitis D	0	2	0	4	0	0	4	0	13	10	17	11.4	1.1	23	21.2
Gastrointestinal diseases															
Botulism	0	0	0	_	0	0	0	0	_	0	_	0.4	2.5	~	1.0
Campylobacteriosis	134	Z	70	1,492	413	194	1,624	722	4,649	4,816	3,202	3,546.6	1.3	9,415	7,849.8
Cryptosporidiosis	4	91	22	210	89	10	173	87	999	869	1,124	813.2	0.8	1,523	2,253.2
Haemolytic uraemic syndrome	0	~	_	_	0	0	_	0	4	6	က	3.4	1.2	13	7.8
Hepatitis A	~	13	0	∞	0	0	16	7	40	86	42	65.2	9.0	125	130.6
Hepatitis E	_	17	0	_	0	0	~	0	20	10	2	8.6	2.3	30	22.4
Listeriosis	0	80	0	7	7	0	80	0	20	22	17	18.0	<u>+-</u>	42	45.2
STEC, VTEC§	0	6	0	6	7	0	က	0	32	4	29	20.2	1.6	73	26.0
Salmonellosis	71	1,063	145	1,311	327	45	890	353	4,202	5,260	3,016	2,633.6	1.6	9,403	6,685.2
Shigellosis	_	31	24	35	ဂ	0	110	15	219	324	114	124.8	1.8	538	299.2
Typhoid	0	10	_	_	~	0	4	4	21	45	29	24.4	6.0	92	74.0
Quarantinable diseases															
Cholera	0	0	0	0	0	0	_	0	_	_	_	2.0	0.5	7	2.6
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.7	0.0	0	0.7

CDI Vol 38 No 3 2014 E255

Table 2 continued: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2014, by date of diagnosis\*

Disease Sexually transmissible infections				State or territory	erritory				Total 2nd	Total 1st	Total 2nd	Last 5 years		Year	Last 5 years
Sexually transmissible infections	ACT	NSM	Ł	Øld	SA	Tas	Vic	WA	quarter 2014	quarter 2013	quarter 2013	méan 2nd quarter	Ratio	to date 2014	YTD mean
Chlamydial infection⊪ı	305	5,661	792	5,089	1,410	483	4,827	2,832	21,399	22,686	20,771	19,414.2	1.1	43,946	39,218.2
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Gonococcal infection <sup>¶</sup>	28	1,219	494	743	220	7	869	222	3,965	4,293	3,868	3,069.2	1.3	8,206	6,094.4
Syphilis – congenital	0	0	0	0	0	0	0	0	0	0	2	0.8	0.0	0	2.2
Syphilis <2 years duration⁴	2	146	13	96	2	က	145	32	445	446	411	353.6	1.3	885	715.6
Syphilis > 2 years or unspecified duration*.¶	<u>ი</u>	92	10	09	30	ო	226	12	445	480	459	374.0	1.2	919	713.0
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	0	0	0.8	0.0	0	1.0
Haemophilus influenzae type b	0	7	0	2	0	0	_	_	9	က	2	5.8	1.0	6	9.6
Influenza (laboratory confirmed)	68	1,023	48	1,480	202	81	880	480	4,567	3,867	2,505	5,980.4	0.8	8,391	7,421.6
Measles	0	7	_	32	2	0	17	10	72	175	17	20.8	3.5	247	58.8
Mumps	_	16	0	15	_	_	က	7	39	64	62	48.0	0.8	102	93.0
Pertussis	45	418	18	327	104	15	692	372	2,068	2,357	2,545	5,829.0	0.4	4,396	12,998.4
Pneumococcal disease (invasive)	4	129	7	22	29	12	107	25	397	217	439	463.2	6.0	611	679.4
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	7	0	~	0	0	0	0	က	9	7	9.8	0.3	6	22.0
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	~	0.2	0.0	0	0.2
Tetanus	0	0	0	0	0	0	0	0	0	_	_	9.0	0.0	_	2.4
Varicella zoster (chickenpox)	7	Z	23	35	77	9	194	119	461	484	434	430.6	1.1	940	803.4
Varicella zoster (shingles)	15	Z	28	7	470	29	377	330	1,328	1,414	1,277	928.8	1.4	2,726	1,926.6
Varicella zoster (unspecified)	64	Z	_	1,280	61	28	845	289	2,568	2,783	2,224	1,882.6	1.4	5,326	3,795.8
Vectorborne diseases															
Arbovirus infection (NEC)	0	7	0	7	0	0	_	0	10	4	2	3.0	3.3	24	6.4
Barmah Forest virus infection	0	29	12	126	0	0	ო	4	214	335	1,561	605.4	4.0	545	1,351.8
Dengue virus infection	∞	119	18	103	21	9	ည	135	415	929	503	302.4	4.1	1,069	847.4
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	2	0.4	0.0	0	9.0
Kunjin virus infection**	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	1.0
Malaria	က	59	_	71	7	<b>~</b>	က	15	75	91	86	0.96	0.8	166	206.8
Murray Valley encephalitis virus infection**	0	0	0	0	0	0	0	0	0	0	0	1.8	0.0	0	4.0
Ross River virus infection	7	211	26	691	15	4	25	254	1,299	1,639	1,314	1,378.6	0.9	2,925	3,366.0

E256 CDI Vol 38 No 3 2014

Table 2 continued: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2014, by date of diagnosis

				State or territory	erritory				Total 2nd	Total 1st	Total 2nd	Last 5 vears		Year	Last 5 vears
Disease	ACT	NSM	Ä	Qid	SA	Tas	Vic	WA	quarter 2014	quarter 2013	quarter 2013	mean 2nd quarter	Ratio	to date 2014	YTD mean
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Brucellosis	0	2	0	0	0	0	_	0	က	9	က	5.8	0.5	6	12.8
Leptospirosis	0	_	0	28	0	0	0	_	30	27	33	43.6	0.7	22	97.8
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	2	0	0	0	0	က	0	2	7	16	15.4	0.3	16	29.4
Q fever	0	34	0	99	2	0	က	7	110	129	138	0.96	<del>1.</del>	237	191.2
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.3
Other bacterial infections															
Legionellosis	0	19	_	19	7	7	34	22	108	06	111	97.4	1.1	195	178.8
Leprosy	0	0	0	~	0	0	_	7	4	က	4	2.8	4.1	7	4.0
Meningococcal infection <sup>+†</sup>	_	13	0	7	4	0	9	9	4	26	27	53.8	0.8	29	98.2
Tuberculosis	2	101	9	34	10	0	117	30	303	321	289	281.2	1.1	619	598.6
Total	829	12,095	1,959	14,321	4,019	1,054	13,146	7,191	54,644	58,432	51,100			112,553	

The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (>2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.

Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland reports hepatitis C newly acquired under hepatitis unspecified.

Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.

infection with Shiga toxin/verotoxin-producing Escherichia coli.

ncludes Chlamydia trachomatis identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens. From 1 July 2013 case definition changed to exclude all ocular infections.  $\omega =$ 

The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

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

Only invasive meningococcal disease is nationally notifiable. However, New South Wales and the Australian Capital Territory also report conjunctival cases. #

Not notifiable  $\frac{1}{2}$  Not elsewhere classified

Totals comprise data from all states and territories. Cumulative figures are subject to retrospective revision so there may be discrepancies between the number of new notifications and the increment n the cumulative figure from the previous period

CDI 2014 E257 Vol 38 No 3

Table 3: Notification rates of diseases, 1 April to 30 June 2014, by state or territory. (Annualised rate per 100,000 population)\*\*,

			S	tate or t	erritory				
Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired) <sup>‡</sup>	1.0	0.6	3.3	0.8	0.0	0.0	1.0	1.1	0.8
Hepatitis B (unspecified) <sup>§</sup>	27.3	34.1	76.3	20.5	23.2	15.6	31.9	21.7	28.6
Hepatitis C (newly acquired)‡	6.3	0.3	1.7	0.0	2.6	3.1	2.1	7.0	1.7
Hepatitis C (unspecified)§	47.2	48.0	77.9	57.0	23.7	47.6	36.1	40.6	44.6
Hepatitis D	0.0	0.3	0.0	0.3	0.0	0.0	0.3	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	140.5	NN	116.1	128.2	98.9	151.2	113.2	114.6	118.3
Cryptosporidiosis	4.2	4.9	36.5	18.0	16.3	7.8	12.1	13.8	11.5
Haemolytic uraemic syndrome	0.0	0.1	1.7	0.1	0.0	0.0	0.1	0.0	0.1
Hepatitis A	1.0	0.7	0.0	0.7	0.0	0.0	1.1	0.3	0.7
Hepatitis E	1.0	0.9	0.0	0.1	0.0	0.0	0.1	0.0	0.3
Listeriosis	0.0	0.4	0.0	0.2	0.5	0.0	0.6	0.0	0.3
STEC,VTEC <sup>  </sup>	0.0	0.5	0.0	8.0	2.6	0.0	0.2	0.0	0.6
Salmonellosis	74.4	57.4	240.5	112.7	78.3	32.7	62.0	56.0	72.7
Shigellosis	1.0	1.7	39.8	3.0	0.7	0.0	7.7	2.4	3.8
Typhoid fever	0.0	0.5	1.7	0.1	0.2	0.0	0.3	0.6	0.4
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Human pathogenic avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmitted infections									
Chlamydial infection <sup>¶,**</sup>	319.8	305.6	1,313.5	437.3	337.6	376.5	336.4	449.4	370.0
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	29.4	65.8	819.3	63.8	52.7	8.6	48.7	87.6	68.6
Syphilis – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis <2 years duration**	5.2	7.9	21.6	8.2	1.2	2.3	10.1	5.1	7.7
Syphilis > 2 years or unspecified duration <sup>§,**</sup>	9.4	5.1	16.6	5.2	7.2	2.3	15.8	1.9	7.7
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.2	0.0	0.0	0.1	0.2	0.1
Influenza (laboratory confirmed)	71.3	55.2	79.6	127.2	121.4	63.1	61.3	76.2	79.0
Measles	0.0	0.4	1.7	2.7	1.2	0.0	1.2	1.6	1.2
Mumps	1.0	0.9	0.0	1.3	0.2	0.8	0.2	0.3	0.7
Pertussis	47.2	22.6	29.9	28.1	24.9	11.7	53.6	59.0	35.8
Pneumococcal disease (invasive)	4.2	7.0	11.6	4.9	6.9	9.4	7.5	8.3	6.9
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	0.1	0.0	0.0	0.0	0.0	0.1
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

E258 CDI Vol 38 No 3 2014

Table 3 continued: Notification rates of diseases, 1 April to 30 June 2014, by state or territory. (Annualised rate per 100,000 population)\*,†

			S	tate or t	erritory				
Disease	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases, cont'd									
Varicella zoster (chickenpox)	7.3	NN	38.1	3.0	18.4	4.7	13.5	18.9	11.7
Varicella zoster (shingles)	15.7	NN	96.2	0.9	112.5	52.2	26.3	52.4	33.8
Varicella zoster (unspecified)	67.1	NN	1.7	110.0	14.6	21.8	58.9	45.9	65.3
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.1	0.0	0.6	0.0	0.0	0.1	0.0	0.2
Barmah Forest virus infection	0.0	3.2	19.9	10.8	0.0	0.0	0.2	2.2	3.7
Dengue virus infection	8.4	6.4	29.9	8.9	5.0	4.7	0.3	21.4	7.2
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 <sup>††</sup>	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	3.1	1.6	1.7	1.8	0.5	8.0	0.2	2.4	1.3
Murray Valley encephalitis virus infection††	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	2.1	11.4	160.9	59.4	3.6	3.1	1.7	40.3	22.5
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Leptospirosis	0.0	0.1	0.0	2.4	0.0	0.0	0.0	0.2	0.5
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.1	0.0	0.0	0.0	0.0	0.2	0.0	0.1
Q fever	0.0	1.8	0.0	5.7	1.2	0.0	0.2	0.3	1.9
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	0.0	1.0	1.7	1.6	2.6	1.6	2.4	3.5	1.9
Leprosy	0.0	0.0	0.0	0.1	0.0	0.0	0.1	0.3	0.1
Meningococcal infection <sup>‡‡</sup>	1.0	0.7	0.0	0.9	1.0	0.0	0.4	1.0	0.7
Tuberculosis	5.2	5.5	10.0	2.9	2.4	0.0	8.2	4.8	5.2

<sup>\*</sup> The date of diagnosis is the onset date or where the date of onset was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. For hepatitis B (unspecified), hepatitis C (unspecified), leprosy, syphilis (> 2 years or unspecified duration) and tuberculosis, the public health unit notification receive date was used.

NEC Not elsewhere classified.

NN Not notifiable.

CDI Vol 38 No 3 2014 E259

<sup>†</sup> Rate per 100,000 of population. Annualisation Factor was 4.0

<sup>‡</sup> Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland reports hepatitis C newly acquired under hepatitis C unspecified.

<sup>§</sup> Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined or is greater than 24 months.

<sup>|</sup> Infection with Shiga toxin/verotoxin-producing Escherichia coli.

<sup>¶</sup> Includes Chlamydia trachomatis identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens. From 1 July 2013 case definition changed to exclude all ocular infections.

<sup>\*\*</sup> The national case definitions for chlamydial, gonococcal and syphilis diagnoses include infections that may be acquired through a non-sexual mode (especially in children – e.g. perinatal infections, epidemic gonococcal conjunctivitis).

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

<sup>4.</sup> Only invasive meningococcal disease is nationally notifiable. However, New South Wales and the Australian Capital Territory also report conjunctival cases.

# Australian Childhood Immunisation Coverage, 1 October to 31 December Cohort, assessed as at 31 March 2014

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

### Introduction

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

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months, for 3-month birth cohorts of children assessed at the stated ages between October and December 2013 using ACIR data as at 31 March 2014. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, varicella, and meningococcal C conjugate vaccines, and is outlined in more detail below.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of three doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing *Haemophilus* influenzae type b (Hib) vaccine or 3 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine and 3 doses of 13-valent pneumococcal conjugate vaccine. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP Hib vaccine or 4 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 60 months of age is defined

as a child having a record on the ACIR of 4 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

A full description of the basic methodology used can be found in *Commun Dis Intell* 1998;22(3):36–37.

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) provides commentary on the trends in ACIR data. For further information please contact NCIRS at: telephone +61 2 9845 1435, email: brynley.hull@health.nsw.gov.au

### Results

The percentage of children 'fully immunised' by 12 months of age for Australia decreased from the previous quarter by 0.4 of a percentage point to 89.7% (Table 1). This decrease, following on from a decrease in the previous quarter, is likely due to the recent inclusion of the 13-valent pneumococcal conjugate vaccine in the coverage calculation algorithm for 'fully immunised' at 12 months of age. Except for the Northern Territory, almost all jurisdictions experienced decreases in coverage for all individual vaccines due at 12 months of age, ranging from 0.1 of a percentage point to 1.5 percentage points.

The percentage of children 'fully immunised' by 24 months of age for Australia increased marginally from the previous quarter by 0.1 of a percentage

Table 1. Percentage of children immunised at 12 months of age for the birth cohort 1 October to 31 December 2012, preliminary results, by disease and state or territory; assessment date 31 March 2014

				State or	territory				
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Number of children	1,393	25,202	915	15,551	5,112	1,501	19,535	8,493	77,702
Diphtheria, tetanus, pertussis (%)	93.5	90.1	91.4	91.4	90.3	88.7	91.1	90.8	90.7
Poliomyelitis (%)	93.5	89.9	91.5	91.4	90.2	88.5	91.0	90.6	90.6
Haemophilus influenzae type b (%)	93.1	89.9	91.3	91.3	90.1	88.5	90.8	90.4	90.5
Hepatitis B (%)	93.1	89.7	91.1	91.1	90.0	88.5	90.5	90.1	90.3
Pneumococcal	93.1	89.8	91.1	91.1	90.0	88.3	90.4	90.2	90.3
Fully immunised (%)	92.7	89.1	90.7	90.7	89.5	87.6	89.8	89.5	89.7
Change in fully immunised since last quarter (%)	-0.6	-0.4	+0.7	-0.4	-0.2	-1.6	-0.7	0.0	-0.4

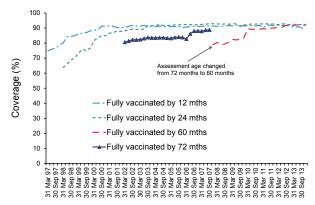
E260 CDI Vol 38 No 3 2014

point to 92.3% (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

The percentage of children 'fully immunised' by 60 months of age for Australia increased marginally from the previous quarter by 0.2 of a percentage point to 92.0% (Table 3). This maintains the improvement in coverage for this age milestone. There were no important changes in coverage for any individual vaccines due at 60 months of age or by jurisdiction.

The Figure shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007). Coverage at 12 months is still lower than coverage at 24 and 60 months of age.

Figure: Trends in vaccination coverage, Australia, 1997 to 31 December 2013, by age cohorts



Coverage assessment date for each cohort

Table 2. Percentage of children immunised at 24 months of age for the birth cohort 1 October to 31 December 2011, preliminary results, by disease and state or territory; assessment date 31 March 2014\*

				State or	territory				
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,344	24,127	837	14,916	4,973	1,573	18,566	7,938	74,274
Diphtheria, tetanus, pertussis (%)	95.4	94.8	94.5	95.5	94.7	95.5	95.3	94.3	95.0
Poliomyelitis (%)	95.4	94.7	94.5	95.5	94.7	95.5	95.2	94.2	95.0
Haemophilus influenzae type b (%)	93.8	93.4	94.1	94.5	93.2	93.6	93.9	92.9	93.7
Measles, mumps, rubella (%)	94.0	93.8	94.9	95.0	94.1	93.7	94.2	93.3	94.1
Hepatitis B (%)	95.1	94.2	94.5	95.0	94.2	95.0	94.9	93.4	94.5
Fully immunised (%)	92.9	91.8	92.7	93.5	92.0	92.6	92.4	91.1	92.3
Change in fully immunised since last quarter (%)	-0.7	0.0	+0.2	+0.7	-0.3	-1.0	-0.5	+1.0	+0.1

<sup>\*</sup> The 12 months age data for this cohort were published in Commun Dis Intell 2013;37(3):E275.

Table 3. Percentage of children immunised at 60 months of age for the birth cohort 1 October to 31 December 2008, preliminary results, by disease and state or territory; assessment date 31 March 2014

				State or	territory				
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,353	25,434	812	15,907	5,037	1,705	18,839	8,170	77,257
Diphtheria, tetanus, pertussis (%)	93.0	92.7	91.4	93.1	91.5	93.4	93.0	89.5	92.5
Poliomyelitis (%)	92.9	92.6	91.1	93.1	91.4	93.0	93.0	89.5	92.4
Measles, mumps, rubella (%)	92.4	92.6	91.5	93.2	91.6	93.4	93.0	89.4	92.4
Fully immunised (%)	92.2	92.2	90.9	92.8	91.0	92.6	92.6	88.9	92.0
Change in fully immunised since last quarter (%)	+1.3	+0.1	-1.3	+0.9	+0.2	-0.2	+0.2	-0.6	+0.2

# AUSTRALIAN MENINGOCOCCAL SURVEILLANCE PROGRAMME QUARTERLY REPORT, 1 APRIL TO 30 June 2014

Monica M Lahra for the Australian Meningococcal Surveillance Programme

### Introduction

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

tion and serogroup, where known. Some minor corrections to data in the Table may be made in subsequent reports if additional data are received. A full analysis of laboratory confirmed cases of IMD in each calendar year is contained in the annual reports of the programme published in *Communicable Diseases Intelligence*. For more information see *Commun Dis Intell* 2014;38(1):E97.

### Results

Laboratory confirmed cases of invasive meningococcal disease for the period 1 April to 30 June 2014 are shown in the Table.

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2014, by serogroup and state or territory

		Serogroup													
State or		Α		В		С		Y		W135		ND		All	
territory	Year	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD
Australian Capital Territory	2014	0	0	1	1	0	0	0	0	0	0	0	0	1	1
	2013	0	0	0	1	0	0	1	1	0	0	0	0	1	2
New South Wales	2014	0	0	6	9	0	0	5	5	1	2	1	1	13	17
	2013	0	0	5	9	0	2	1	1	0	0	1	1	7	13
Northern Territory	2014	0	0	0	2	0	0	0	0	0	0	0	0	0	2
	2013	0	0	1	1	0	0	0	0	0	0	0	0	1	1
Queensland	2014	0	0	9	16	0	0	0	0	0	1	2	2	11	19
	2013	0	0	6	13	1	1	0	1	1	1	0	0	8	16
South Australia	2014	0	0	4	9	0	0	0	0	0	0	0	0	4	9
	2013	0	0	5	9	0	0	1	1	0	1	0	0	6	11
Tasmania	2014	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2013	0	0	0	1	0	0	0	0	0	0	0	0	0	1
Victoria	2014	0	0	6	9	0	0	1	1	1	2	0	0	8	12
	2013	0	0	2	8	0	1	0	0	0	0	1	1	3	10
Western Australia	2014	0	0	5	6	0	2	0	0	1	1	0	0	6	9
	2013	0	0	1	8	1	1	0	0	1	1	0	0	3	10
Total	2014	0	0	31	52	0	2	6	6	3	6	3	3	43	69
	2013	0	0	20	50	2	5	3	4	2	3	2	2	29	64

# Australian Sentinel Practices Research Network, 1 April to 30 June 2013

Monique Chilver, Daniel Blakeley for the Australian Sentinel Practices Research Network

### Introduction

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is funded by the Australian Government Department of Health, owned and operated by the Royal Australian College of General Practitioners and directed through the Discipline of General Practice at the University of Adelaide.

The network consists of general practitioners who report presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can alert public health officials of epidemics in their early stages as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Electronic, web-based data collection was established in 2006.

In June 2010, ASPREN's laboratory influenza-like illness (ILI) testing was implemented, allowing for viral testing of 25% of ILI patients for a range of respiratory viruses including influenza A, influenza B and influenza A H1N1(2009).

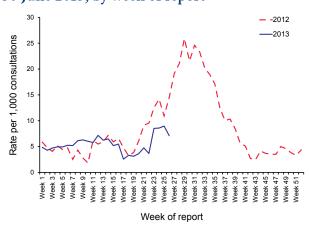
The list of conditions is reviewed annually by the ASPREN management committee. In 2014, 4 conditions are being monitored. They include ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, published in *Commun Dis Intell* 2014;38(1):E96.

### **Results**

Sentinel practices contributing to ASPREN were located in all 8 states and territories in Australia. A total of 275 general practitioners contributed data to ASPREN in the 2nd quarter of 2013. Each week an average of 231 general practitioners provided information to ASPREN at an average of 22,309 (range 15,235–27,263) consultations per week and an average of 248 (range 134–356) notifications per week.

ILI rates reported from 1 April to 30 June 2013 averaged 6 cases per 1,000 consultations (range 3–9 cases per 1,000 consultations). This was lower than rates in the same reporting period in 2012, which averaged 8 cases per 1,000 consultations (range 3–14 cases per 1,000 consultations, Figure 1).

Figure 1: Consultation rates for influenzalike illness, ASPREN, 2012 and 1 January to 30 June 2013, by week of report



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

ILI swab testing continued in 2013. The most commonly reported virus during this reporting period was rhinovirus (20% of all swabs performed, Figure 2), with the 2nd most common virus being respiratory syncytial virus (8% of all swabs performed).

From the beginning of 2013 to the end of week 26, 60 cases of influenza were detected and comprised of influenza A (untyped) (5% of all swabs performed) and influenza B (3% of all swabs performed) (Figure 2).

During this reporting period, consultation rates for gastroenteritis averaged 4 cases per 1,000 consultations (range 3–7 cases per 1,000, Figure 3). This was similar to rates in the same reporting period in 2012 where the average was 4 cases per 1,000 consultations (range 3–6 cases per 1,000).

Varicella infections were reported at a higher rate for the 2nd quarter of 2013 compared with the same period in 2012. From 1 April to 30 June 2013, recorded rates for chickenpox averaged 0.22 cases per 1,000 consultations (range 0.00–0.74 cases per 1,000 consultations, Figure 4).

Proportion positive for influenza 9 50 4 30 20 10 0 Figure 2: Influenza-like illness swab testing results, ASPREN, 2012 and 1 January to 30 June 2013, by week of report Respiratory syncytial virus Parainfluenza virus type 3 Week 51 Meek 49 Metapneumovirus Week 47 Meek 45 Week 43 Week 41 Week 39 Week 37 Week 35 Week number, 2013 **Meek 33** Week 31 Parainfluenza virus type 2 Week 29 Week 27 Week 25 Week 23 Influenza B Rhinovirus Pertussis Week 21 Week 19 Week 17 Week 15 Week 13 Week 11 Influenza A untyped / other Parainfluenza virus type 1 Mycoplasma pneumoniae **Меек** 9 Week 7 Week 5 Week 3 Adenovirus Week 1 200 180 160 140 120 100 8 8 4 20 Number of positive specimens

Influenza positivity (%)

E264 CDI Vol 38 No 3 2014

Figure 3: Consultation rates for gastroenteritis, ASPREN, 2012 and 1 January to 30 June 2013, by week of report

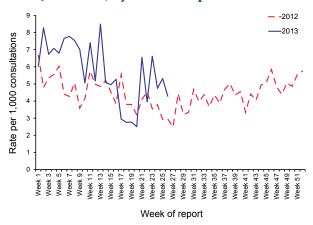
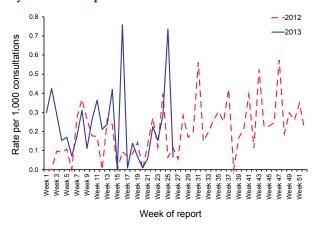


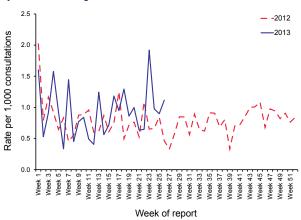
Figure 4: Consultation rates for chickenpox, ASPREN, 2012 and 1 January to 30 June 2013, by week of report



In the 2nd quarter of 2013, reported rates for shingles averaged 0.98 cases per 1,000 consultations (range 0.56–1.92 cases per 1,000 consultations, Figure 5). This was slightly higher than in the same reporting period in 2012 where the average shingles rate was 0.75 case per 1,000 consultations

(range 0.46–1.25 cases per 1,000 consultations).

Figure 5: Consultation rates for shingles, ASPREN, 2012 and 1 January to 30 June 2013, by week of report



## Invasive pneumococcal disease surveillance Australia, 1 April to 30 June 2014

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

### Introduction

Invasive pneumococcal disease (IPD) is caused by the bacterium Streptococcus pneumoniae and results in illnesses such as pneumonia, bacteraemia and meningitis. There are currently more than 90 serotypes recognised worldwide, approximately half of which are found in Australia where IPD has been a nationally notifiable disease since 2001. This quarterly report documents trends in notified cases of IPD occurring in Australia in the 2nd quarter of 2014. In this quarterly report, 3 age groups have been selected for focused analyses. These age groups align with groups that carry the greatest burden of disease and against which the National Immunisation Program is targeted. The data in this report are provisional and subject to change as laboratory results and additional case information become available.

Detailed IPD surveillance methodology is described each year in the 1st quarter report and in the annual reports published in *Communicable Diseases Intelligence*.

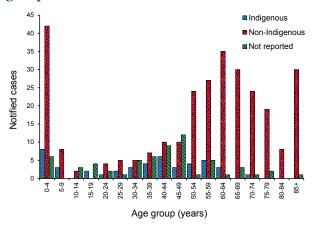
In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk and older Australians.\*

### Results

There were 397 cases of IPD reported to the National Notifiable Diseases Surveillance System in the 2nd quarter of 2014, bringing the year to date total to 611 cases. The number of cases notified in the reporting period was an 86% increase from the 1st quarter (n=214). This trend is consistent with the usual peak in the number of cases of IPD in the winter months. This trend was observed in all analyses included in this report. For the year to 30 June there was a 6.1% reduction on the number of cases reported for the same period in 2013 (n=651) (Table).

Overall, and in cases reported as non-Indigenous Australian, notified cases were highest in the under 5 years age group followed by the 60–64 years age group (Figure 1). In cases reported as Indigenous, the most prevalent age group was the under 5 years (n=8) followed by the 40–44 years age group (n=6).

Figure 1: Notifications of invasive pneumococcal disease, Australia, 1 April to 30 June 2014, by Indigenous status and age group



### **Data completeness**

During the reporting period, Indigenous status was reported for 84% (n=335) of cases and sero-type information was available for 91% (n=360) of all cases reported (Table).

## Invasive pneumococcal disease in children aged less than 5 years

In the 2nd quarter of 2014, 14% (n=56) of notified cases were aged less than 5 years. This was an increase on the number of cases reported in the previous quarter (n=31), but similar to the number reported during the same period of 2013 (n=57) (Figure 2).

The majority (89%, n=50) of cases aged less than 5 years were reported with serotype information. Of these, 42% (n=21) were reported with a serotype included in the 7vPCV or the 13vPCV.

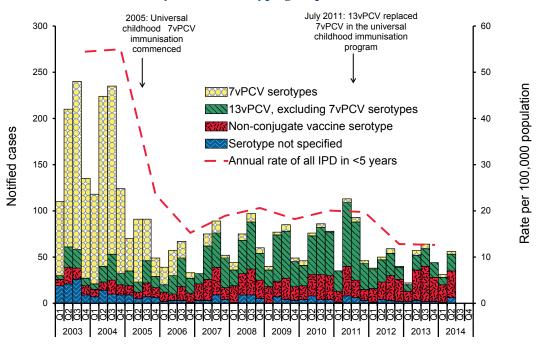
<sup>\*</sup> The 7-valent pneumococcal conjugate vaccine (7vPCV) was added to the National Immunisation Program schedule for Indigenous and medically at-risk children in 2001 and for all children up to 2 years of age in 2005. The 13-valent pneumococcal conjugate vaccine (13vPCV) replaced the 7vPCV in the childhood immunisation program from July 2011. The 23-valent pneumococcal polysaccharide vaccine (23vPPV) was added to the National Immunisation Program schedule for Aboriginal and Torres Strait Islander peoples aged 50 years or over in 1999 and for non-Indigenous Australians aged 65 years or over from January 2005.

Table: Notified cases of invasive	pneumococcal disease,	Australia, 1	April to 30 June 201	14, by
Indigenous status, serotype com				

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

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

Figure 2: Notifications and rates of invasive pneumococcal disease in those aged less than 5 years, Australia, 2003 to 30 June 2014, by vaccine serotype group

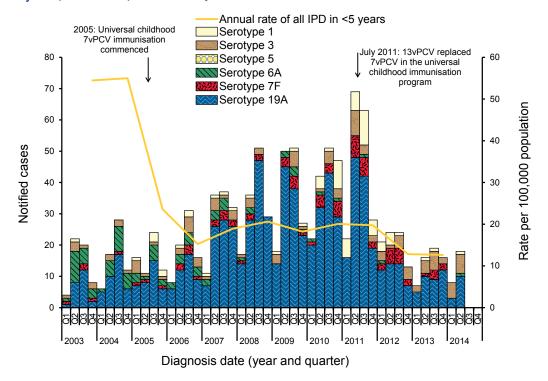


Diagnosis date (year and quarter)

Over the period 2007 to 2011 notified cases aged less than 5 years with disease caused by the 6 additional serotypes (1, 3, 5, 6A, 7F and 19A) that would be covered by the 13vPCV, increased steadily, particularly those caused by serotype 19A (Figure 3). However, cases of serotype 19A have decreased since the 4th quarter of 2011, reflecting the introduction of the 13vPCV into the universal

childhood immunisation program in mid-2011. In the 2nd quarter of 2014, there were 10 cases aged less than 5 years with disease due to serotype 19A, 6 cases due to serotype 3 and 1 case each of serotypes 1 and 6A. In this age group, no cases were reported with disease caused by serotype 5 or 7F; a previously common serotype.

Figure 3: Notifications of invasive pneumococcal disease caused by serotypes targeted by the 13-valent pneumococcal conjugate vaccine\* and rates of all invasive pneumococcal disease, aged less than 5 years, Australia, 2003 to 30 June 2014



\* Excludes those targeted by 7-valent pneumococcal conjugate vaccine

### Invasive pneumococcal disease in Indigenous Australians aged 50 years or over

In the 2nd quarter of 2014, 3% (n=13) of notified cases were reported as Indigenous Australians aged 50 years or older (Figure 4). This was a 58% increase compared with the 1st quarter (n=9) and was slightly lower than the number reported during the same period in 2013 (n=15).

All but one of the cases notified in the 2nd quarter of 2014 were reported with serotype information. Of these, approximately half (n=7) were reported with disease due to serotypes targeted by the 23vPPV. The remaining cases reported disease due to a non-vaccine serotype (n=5).

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

In the 2nd quarter of 2014, 28% (n=111) of notified cases were reported as nonIndigenous and aged 65 years or over. This was an increase in the number of cases reported in the previous quarter (n=58) and a 21% reduction on the number reported during the same period of 2013 (n=141) (Figure 5).

The majority (94%, n=104) of cases reported in this quarter were reported with serotype information. Of these cases, 64% (n=67) were reported with a serotype targeted by the 23vPPV. While the burden of disease in this age group has remained relatively stable, the profile of serotypes causing disease has changed over time. Disease due to serotypes targeted by the 7vPCV has reduced substantially in this age group, which is likely to be due to herd immunity impacts from the childhood immunisation program.

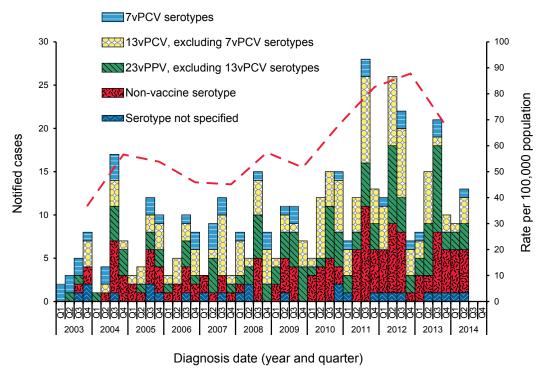
## Mortality due to invasive pneumococcal disease

Nationally, there were 21 deaths attributed to 13 different IPD serotypes during this reporting period. No deaths in the under 5 years age group were due to serotypes included in the 7vPCV or 13vPCV.

### Conclusion

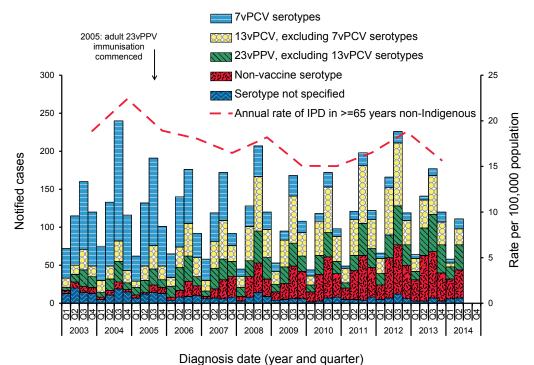
The number of notified cases of IPD in the 2nd quarter of 2014 was an 86% increase on the previous quarter, which was consistent with the seasonal increase of IPD during winter. To 30 June, the total number of cases in 2014 was a 6% reduction on the number of cases reported for

Figure 4: Notifications and rates of invasive pneumococcal disease in Indigenous Australians aged 50 years or over, Australia, 2003 to 30 June 2014, by vaccine serotype group



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

Figure 5: Notifications and rates of invasive pneumococcal disease in non-Indigenous Australians aged 65 years or over, Australia, 2003 to 30 June 2014, by vaccine serotype group



CDI Vol 38 No 3 2014

the same period in 2013. Nationally, the pattern of disease has not changed from the 2nd quarter of 2013. Specifically, the decline in disease due to the serotypes targeted by the 13vPCV has been maintained since the 13vPCV replaced the 7vPCV in the childhood immunisation program from July 2011. Similarly, IPD associated with non-vaccine serotypes has remained unchanged in all groups targeted for IPD vaccination. Disease in non-Indigenous Australians aged 65 years or over has remained relatively stable but the profile of serotypes causing disease has diversified.

### **Acknowledgements**

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

Enhanced Invasive Pneumococcal Disease Surveillance Working Group contributors to this report include (in alphabetical order): David Coleman (Tas.), Heather Cook (NT), Rachel de Kluyver (Health), Lucinda Franklin (Vic.), Carolien Giele (WA), Robin Gilmour (NSW), Michelle Green (Tas.), Vicki Krause (NT), Rob Menzies (NCIRS), Shahin Oftadeh (Centre for Infectious Diseases and Microbiology- Public Health, Westmead Hospital), Sue Reid (ACT), Stacey Rowe (Vic.), Vitali Sintchenko (Centre for Infectious Diseases and Microbiology- Public Health, Westmead Hospital), Helen Smith (Queensland Health Forensic and Scientific Services), Janet Strachan (Microbiological Diagnostic Unit, University of Melbourne), Cindy Toms (Health), Hannah Vogt (SA), Angela Wakefield (Qld).

### **Author details**

Corresponding author: Dr Rachel de Kluyver, Vaccine Preventable Diseases Surveillance Section, Office of Health Protection, Australian Government Department of Health, GPO Box 9484, MDP 14, Canberra, ACT 2601. Telephone: +61 2 6289 1463. Facsimile: +61 2 6289 1070. Email: Rachel.de.kluyver@health.gov.au

E270

## Policy and guidelines

## RECOMMENDED COMPOSITION OF THE AUSTRALIAN INFLUENZA VACCINE FOR THE 2015 SEASON

On 25 September 2014, the World Health Organization recommended that vaccines for the 2015 Southern Hemisphere influenza season contain the following:

A/California/7/2009 (H1N1)pdm09 - like virus

A/Switzerland/9715293/2013 (H3N2) - like virus

B: a B/Phuket/3073/2013 – like virus (Yamagata lineage)

The WHO also recommended that quadrivalent vaccines, which include 2 influenza B viruses, contain the above 3 viruses and a B/Brisbane/60/2008-like virus (Victoria lineage).

The Australian Influenza Vaccine Committee met on 9 October 2014, and recommended that the Therapeutic Goods Administration (TGA) adopt the WHO recommendations.

For further information please see the TGA web site (http://www.tga.gov.au/about/committees-aivc-2015-recommendations.htm) and the WHO web site (http://www.who.int/influenza/vaccines/virus/recommendations/consultation201409/en/).

CDI Vol 38 No 3 2014 E271