Australian Government
Department of Health

Communicable Diseases Intelligence

Volume 40 Number 4 Quarterly report December 2016

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ISSN 1445-4866 Online

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

Website

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

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This journal is indexed by *Index Medicus* and Medline

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Editorial

Rise in invasive serogroup W meningococcal disease in Australia 2013–2015

Mark GK Veitch, Rhonda L Owen

This *Communicable Diseases Intelligence* issue contains an analysis prompted by the recent increase in incidence of invasive meningococcal disease (IMD) due to meningococcal serogroup W (MenW) in Australia.

IMD due to MenW is emerging rapidly in Australia. This editorial provides readers with epidemiological data on MenW current to 2 December 2016 (hereafter YTD i.e. year-to-date); information on the coordinated national public health response; and some of the clinical and public health challenges posed by the emergence of MenW.

Epidemiological situation

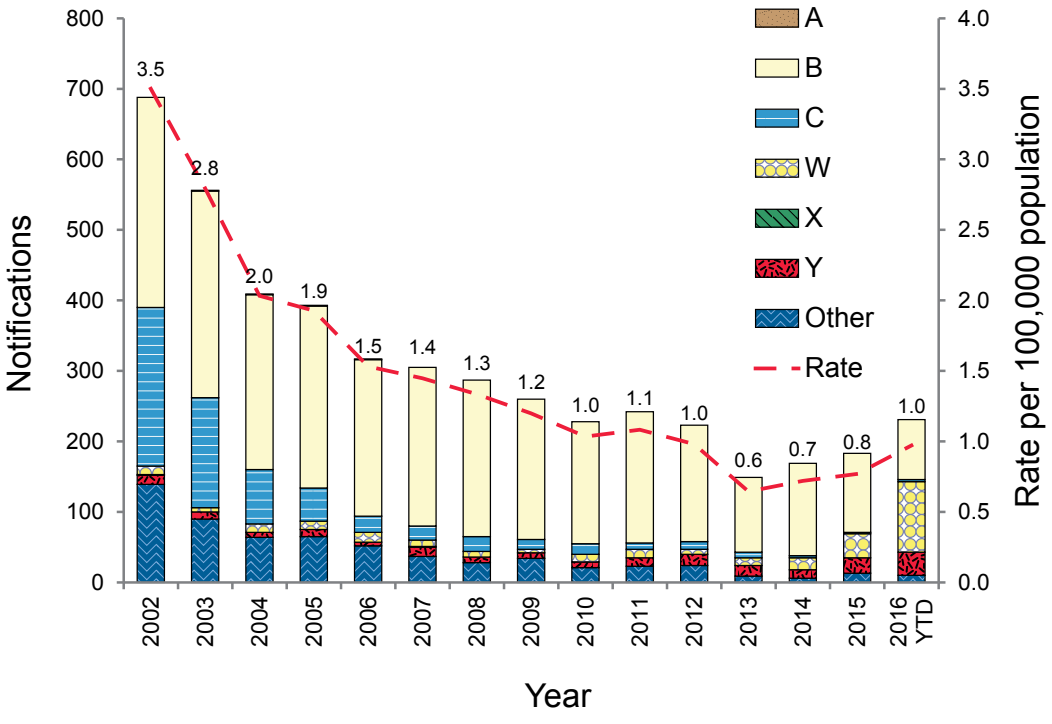
Incidence

The most recent peak in the incidence of IMD in Australia was in 2001 to 2002, due to serogroup C. The rate of notifications of IMD declined by 82% from 2002 (3.5 per 100,000; 688 cases) to 2013 (0.6 per 100,000; 149 cases) (Figure). IMD subsequently increased, with 169 cases in 2014, 183 cases in 2015 and 231 cases YTD in 2016.

From 2002 to 2015, serogroup B meningococcal infection was the most common cause of IMD in Australia. However, in 2016, meningococcal serogroup W (MenW) became the predominant meningococcal serogroup in Australia, accounting for 43% of notifications of IMD (100 of 231 IMD cases in 2016 YTD) (Figure). Since April 2016 one-third or more of IMD notifications each month have been caused by MenW.

Seven of the 11 deaths from IMD notified to the Australian National Notifiable Diseases Surveillance System in 2016 YTD were due to MenW.

Figure: Notifications and rates of invasive meningococcal disease, 2002 to December 2016,*Australia, by serogroup



* As of 2 December 2016.

Temporal pattern

IMD tends to follow a seasonal pattern in Australia, with disease most common between June and September each year. In 2016 notifications peaked later, with 38 cases in both August and October. Notifications substantially decreased in November 2016 (18 cases).

Geographical distribution

MenW is contributing to an increasing burden of IMD in all jurisdictions except the Northern Territory. Compared with 2015, the number of MenW notifications has more than doubled in 2016 YTD, in New South Wales (8 to 26 cases), Queensland (4 to 12 cases), Western Australia (4 to 12 cases), Victoria (17 to 41 cases), South Australia (0 to 4 cases) and Tasmania (1 to 4 cases). In 2016, the Australian Capital Territory reported its 1st case of MenW since 2008.

Age distribution

In 2016 YTD, MenW has been reported in all age groups, except in children aged 10–14 years, and accounts for almost half of IMD in adults aged 65 years or over.

Notifications of MenW have remained low in children aged less than 5 years from 2002 until 2016, with no more than 2 cases reported annually in children aged less than 1 year and no more than 4 cases reported annually in children aged 1–5 years. However in 2016 YTD, there have been 8 cases of MenW reported in children aged less than 1 year and 13 cases in children aged 1–4 years.

International situation

Significant outbreaks of MenW have occurred in countries including the United Kingdom,[1] Brazil, Portugal, Argentina, Chile, Sweden and Taiwan.[2] In Chile, the incidence of MenW increased from <0.1 per 100,000 population in 2010 to 0.7 per 100,000 population in 2012.[3] The epidemiology of MenW in Australia currently has a similar appearance to increases seen internationally, but there remains uncertainty whether the disease in Australia will continue to evolve as experienced elsewhere, such as the United Kingdom and parts of South America.

What is being done nationally about the increase in meningococcal serogroup W?

The Australian Government Department of Health, in collaboration with the states and territories, is closely monitoring the epidemiology of IMD due to MenW in Australia in accordance with the Emergency Response Plan for Communicable Disease Incidents of National Significance (CDPlan).

Following a rapid assessment, the Chief Medical Officer of Australia established an incident management team, supported by advisory groups, with a focus on examining the current epidemiology of MenW and co-ordinating national responses to MenW. A further assessment will be undertaken in early 2017.

Clinical and public health challenges posed by the emergence of meningococcal serogroup W

Clinicians are familiar with the importance of early diagnosis and early treatment of IMD, and the challenge of identifying severe sepsis from early, non-specific signs of infection. While cases of MenW may have features of bacteraemia or, less commonly, meningitis, less typical presentations have occurred, including septic arthritis and epiglottitis in older age groups. In the United Kingdom gastrointestinal symptoms were noted among adolescents, and pneumonia among young children.[4] Our surveillance must therefore track changes in the age distribution, clinical manifestations, outcomes, and phenotypic and genotypic microbiology of IMD, and communicate findings to clinicians and public health practitioners in Australia.

Quadrivalent meningococcal vaccines that provide protection against IMD due to serogroups A, C, W and Y are available on private prescription in Australia. These are currently recommended for some occupational exposures (e.g. laboratory work), overseas travel to particular destinations, and medical conditions that increase the risk from IMD.[5] They may also be prescribed for, and purchased electively by, persons who wish to be personally protected against these serogroups of IMD. The consideration of a population-based vaccination program entails various uncertainties and complexities.

The emergence of a new strain of meningococcal bacteria reflects evolving host-microbial ecology.[6] While vaccination of particular population sectors can be expected to confer direct protection on those vaccinated, the trajectory of the recently emerged MenW strain in the wider Australian population is uncertain, with or without targeted vaccination programs.

Establishing a population-based vaccination program would entail determining the target population or populations so as to prevent disease, avert transmission, and contribute significantly to herd immunity in the wider population. Such a program

would also need to be feasible for jurisdictions and providers to deliver, monitor and thoroughly evaluate, aiming to achieve early high coverage of the target populations.

There remains the need for clinical vigilance by healthcare professionals to suspect and diagnose rare but serious meningococcal disease, and to ensure incident cases are promptly notified to the relevant public health agency.

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Original article

Rise in invasive serogroup W meningococcal disease in Australia, 2013–2015

Nicolee V Martin, Katherine S Ong, Benjamin P Howden, Monica M Lahra, Stephen B Lambert, Frank H Beard, Gary K Dowse, Nathan Saul on behalf of the Communicable Diseases Network Australia MenW Working Group

Abstract

Since 2013, there has been an increase in the number of notified cases of invasive meningococcal disease (IMD) due to serogroup W (MenW) in Australia. In response to this observed increase, the Communicable Diseases Network Australia convened a working group in 2015 to collate and analyse the epidemiology of MenW disease nationally. Enhanced surveillance data collected by jurisdictions were collated and analysed, and whole genome sequencing (WGS) of MenW isolates assessed the genomic relatedness of strains between 2012 and 2015. This report describes that epidemiology. Since 2013, the incidence and proportion of MenW has increased in Australia, rising from an average of 2% of all IMD cases annually (range 0% to 5%) between 1991 and 2012; to 8% (12/149) of cases in 2013, 10% (17/169) in 2014, and 19% (34/182) in 2015. Victoria has been the main affected state, with 50% (17/34) of national cases in 2015. MenW has affected older populations, with a median age between 2003 and 2015 being 44 years. During this period, case fatality was 10.7% (17/159), 2.3 times higher than for all IMD serogroups combined (4.7%, 173/3720). There were 7 deaths due to MenW in 2015 (CFR 21%). WGS has found the majority of Australian isolates cluster within a group of W:P1.5,2:F1-1:ST11 isolates from the United Kingdom and South America, regions where rapid spread and endemic transmission has occurred since 2009. The recent increase in incidence of MenW in Australia is evolving and is being closely monitored. Lessons learned from the international experience will be important in informing the public health response. *Commun Dis Intell* 2016;40(4):E454–E459.

Keywords: meningococcal disease; Australia, *Neisseria meningitidis*

Introduction

Invasive meningococcal disease (IMD) is caused by the bacterium *Neisseria meningitidis*. Meningitis and septicaemia are the most common clinical presentations of IMD, and infection can lead to serious outcomes including death.[1] Historically, most IMD in Australia has been caused by serogroups B (MenB) and C (MenC). The national rate of IMD notification has declined since the introduction of the MenC vaccine on the National Immunisation Program in 2003 but total numbers of IMD have been increasing since 2013.[2,3] The incidence of IMD due to serogroup W (MenW) in Australia has been low. However, since 2013 there has been an increase in both the incidence and proportion of MenW relative to all IMD cases, notably in Victoria.[4,5] There are international reports of a hypervirulent strain of MenW that began circulating in the United Kingdom and Chile in 2009 and has since become endemic in these countries. In this context the epidemiology of IMD in Australia was investigated, with a particular focus on MenW.

Methods

Notifications of IMD in Australia have been routinely collected in the National Notifiable Diseases Surveillance System (NNDSS), a collation of surveillance data collected locally in the 8 states and territories, since 1991. The NNDSS was used to compare the epidemiology of IMD due to non-MenW serogroups with that of MenW during the period 1991 and 2015. A sub-set of these data from 2003 were used to calculate case fatality ratios by serogroup and Indigenous status. Prior to this time, although these fields were available, they were not consistently complete.

Laboratory surveillance of IMD, including phenotypic and genotypic characteristics of invasive strains, has been conducted in Australia by reference laboratories in each state and territory (the Australian National Neisseria Network) since 1984, and these data supplement the NNDSS data.

Additional enhanced data (clinical presentation, hospitalisation status, co-morbidities, complications, risk factors, travel history) provided by states and territories were collated and analysed retrospectively, for MenW cases notified between 2012 and 2015. This period was selected to represent the time-frame in which MenW cases began to increase and for which enhanced clinical and whole genome sequencing (WGS) data were available.

WGS was performed by the Microbiological Diagnostic Unit (MDU) at the University of Melbourne or at the University of Western Australia (Western Australian isolates only), to determine the genomic relatedness of 34 Australian isolates of MenW between 2006 and 2015. Genomic DNA extraction and phylogenomic analysis was performed according to previously established methods within the MDU public health laboratory[6] including the Nullarbor bioinformatic pipeline (https://github.com/tseemann/nullarbor). These 3 data sources were merged in a single Microsoft Excel dataset for this descriptive analysis.

Results

Epidemiology

Australian IMD notifications reached an annual peak of 688 cases in 2002 (rate of 3.5 per 100,000 population), with a decreasing trend evident since then (Figure 1).

Between 1991 and 2015, MenB and MenC were the most common serogroups causing IMD in Australia comprising 49% (4520/9269) and 16% (n=1503) of all cases, respectively. The remaining serogroups have accounted for a very small proportion of cases overall, including MenW, 3% (n=234); serogroup Y, 2% (n=218); and serogroups A, X, and Z each less than 0.1%. Thirty per cent of cases during this time were not grouped (n=2,714) or non-groupable (n=68), with this proportion decreasing from 90% of cases in 1991 to 3% of cases in 2014 (Figure 1).

In 2015, MenB remained the predominant serogroup, comprising 61% (111/182) of all notified IMD cases. MenC decreased from a high of 33% (225/688) of cases in 2002 to approximately 1% (2/182) of cases in 2015 (Figure 1). In contrast, MenW represented an average of 2% of cases annually (range 0% to 5%) between 1991 and 2012, but has been increasing since then: 8% (12/149) of all cases in 2013, 10% (17/169) in 2014, and 19% (34/182) in 2015 (Figure 1).

The largest increase in MenW notifications has occurred in Victoria, accounting for 50% (17/34) of all MenW cases reported nationally in 2015, and nearly 6 times the annual Victorian average of 2.9 cases between 1991 and 2011 (Figure 2).

Figure 2: Notifications of invasive meningococcal disease serogroup W, Australia, 2012 to 2015, by state and territory and month

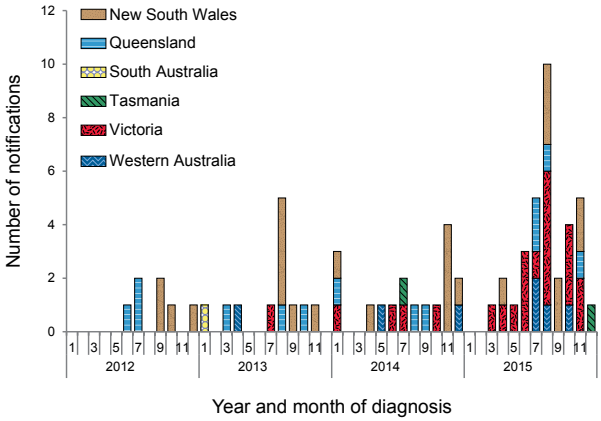
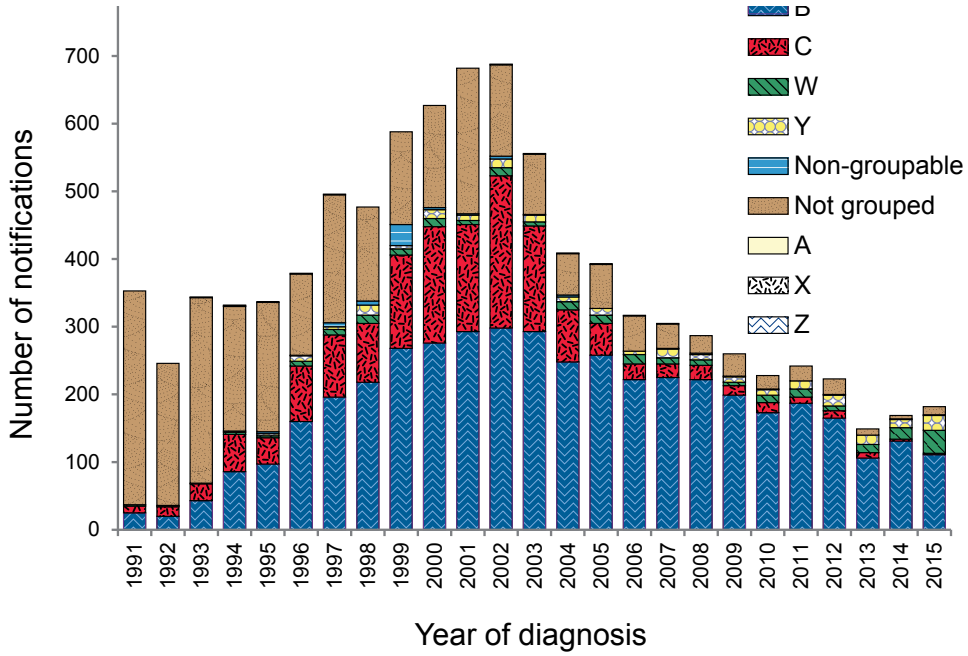


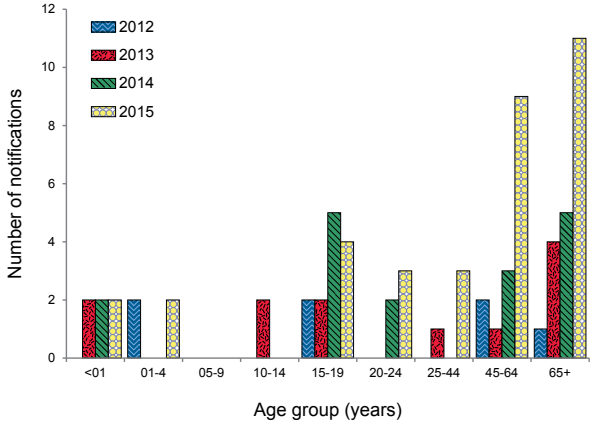
Figure 1: Notifications of invasive meningococcal disease, Australia, 1991 to 2015, by serogroup and year



The age distribution of IMD notifications has a bimodal pattern, strongly influenced by the predominance of MenB notifications, with peaks in children less than 5 years of age, and in adolescents and young adults aged between 15 and 24 years. Between 1991 and 2015, males represented 53% of all IMD cases and predominated in all age groups less than 45 years of age, while females predominated in age groups 45 years or over.

While MenW had similar peaks in the 0–4 and 15–24 age groups, there were also a relatively larger proportion of cases among adults aged 45 years or over, as reflected in the older median age of MenW cases compared with IMD generally (Table 1). Increases in MenW notifications since 2013 were confined to those 15 years of age or over with children less than 10 years of age relatively unaffected to date (Figure 3). Males represented 53% (123/234) of all MenW cases between 1991 and 2015, and represented 60% of cases (75/126) in those less than 35 years of age. Females represented 67% (72/108) of MenW cases aged 35 years or over.

Figure 3: Notifications of invasive meningococcal disease serogroup W, Australia, 2012 to 2015, by age group



Of the 3,720 cases of IMD notified between 2003 and 2015, 4.7% (n=173) were reported as having died with an annual case fatality ratio (CFR) ranging from 3% to 7% (Table 2). MenB has accounted for the majority of deaths in all years except 2003 when MenC had the highest proportion (73%, 19/26) and 2015 when MenW had the highest proportion (58%, 7/12).

Between 2003 and 2011, 11% of all MenW cases were fatal compared with the lower CFR for other serogroups (Table 2). There were 7 deaths due to MenW (CFR 21%) in 2015, accounting for 58% of all IMD deaths for that year. The median age of death for MenW cases between 2012 and 2015 was 26 years, nearly half the median age of death for this serogroup between 2003 and 2011, but older than for all non-MenW cases during this period (Table 1).

Enhanced surveillance data

Enhanced data was collected for all 70 MenW cases notified between 2012 and 2015: New South Wales (n=25), Victoria (n=22), Queensland (n=13), Western Australia (n=7), Tasmania (n=2) and South Australia (n=1).

Eighty per cent (56/70) of the MenW cases had a typical clinical presentation of septicaemia, meningitis, or both while 20% (14/70) had an atypical presentation, including: septic arthritis (n=6), pneumonia (n=4), epiglottitis (n=3) and other (n=1). All 69 cases that had information available on their hospitalisation status were hospitalised. Twenty-three per cent (16/69) of hospitalised cases were admitted to an intensive care unit.

Information on complications was available for 29% (20/70) of cases: 15 had no complications reported, 3 had minor complications, and 2 had major complications (excluding death).

There was no medical or behavioural risk factor information available for 22 cases. Of the remaining cases, 48% (23/48) had at least 1 risk factor identified: 25% (n=12) had a chronic disease; 31% (n=15) reported smoking either current (n=8) or previous (n=7); and 13% (n=6) had an immunological condition. Eight cases (17%) had 2 or more risk factors.

Vaccination against MenW is currently not routinely recommended in Australia excepting for those with medical conditions associated with an increased risk of meningococcal disease, for persons travelling overseas to areas in which MenW is endemic, or for those at occupational risk. Information on vaccination status was available for 35 (50%) of MenW cases, none of whom were vaccinated.

Travel history in the preceding 2 weeks was known for 84% (59/70) of cases for overseas travel and 54% (38/70) for interstate travel. Only 2 cases (3%) reported overseas travel (to England and Papua New Guinea, respectively), and 2 (5%) reported interstate travel (to New South Wales and Queensland respectively).

Genomic relatedness of Australian strains of invasive meningococcal disease serogroup W

Of the 70 MenW cases reported between 2012 and 2015, 51 isolates had typing available at the time of this report, and of these 65% (33/51) were strain type

Table 1: Summary table of invasive meningococcal disease cases, deaths and median age at onset

	Number of cases	Median age at onset	Age at onset range in years	Number of deaths	Median age of death	Age of death range in years	Case fatality rate (%)
All IMD 2003–2015	3,720	17	0–102	173	20	0–95	4.7
MenW 2003–2015	159	44	0–93	17	40	0–89	10.7
MenW 2003–2011	89	40	0–93	10	49	0–89	11.2
MenW 2012–2015	70	48	0–89	7	26	18–78	10
IMD (excluding W) 2012–2015	653	18	0–98	30	17	0–94	4.6
MenW 2015	34	51	0–85	7	26	18–78	20.6
All IMD Indigenous 2003–2015	353	3	0–72	15	3	0–72	4.2
MenW Indigenous 2003–2015	12	1	0–48	0	–		–

Table 2: Notifications, rates, deaths and case fatality ratio of invasive meningococcal disease, Australia, 2003 to 2015, by serogroup

	Serogroup B				Serogroup C				Serogroup W				Serogroup Y				All serogroups			
	Cases	Rate	Deaths	CFR (%)	Cases	Rate	Deaths	CFR (%)	Cases	Rate	Deaths	CFR (%)	Cases	Rate	Deaths	CFR (%)	Cases	Rate	Deaths	CFR (%)
2003	293	1.49	6	2.0	156	0.79	19	12.2	6	0.03	0	0	10	0.05	0	0.0	556	2.82	26	4.7
2004	248	1.24	12	4.8	77	0.39	4	5.2	12	0.06	2	16.7	7	0.04	0	0.0	409	2.05	19	4.6
2005	258	1.28	13	5.0	47	0.23	4	8.5	12	0.06	2	16.7	10	0.05	1	10.0	393	1.95	21	5.3
2006	222	1.09	8	3.6	23	0.11	1	4.3	14	0.07	3	21.4	5	0.02	0	0.0	317	1.55	12	3.8
2007	225	1.08	5	2.2	20	0.10	3	15.0	9	0.04	0	0.0	13	0.06	0	0.0	305	1.46	9	3.0
2008	222	1.04	9	4.1	21	0.10	1	4.7	8	0.04	0	0.0	8	0.04	0	0.0	287	1.35	10	3.5
2009	199	0.92	8	4.0	14	0.06	1	7.1	5	0.02	1	20.0	8	0.04	0	0.0	260	1.20	10	3.8
2010	173	0.79	10	5.8	15	0.07	1	6.7	11	0.05	1	9.1	8	0.04	0	0.0	228	1.03	14	6.1
2011	187	0.84	12	6.4	9	0.04	0	0.0	12	0.05	1	8.3	12	0.05	2	16.7	242	1.08	15	6.2
2012	165	0.73	10	6.1	11	0.05	2	18.2	7	0.03	0	0.0	16	0.07	0	0.0	223	0.98	12	5.4
2013	106	0.46	2	1.9	8	0.03	1	12.5	12	0.05	0	0.0	14	0.06	1	7.1	149	0.64	5	3.4
2014	131	0.56	7	5.3	3	0.01	0	0.0	17	0.07	0	0.0	12	0.05	1	8.3	169	0.72	8	4.7
2015	111	0.47	4	3.6	2	0.01	0	0.0	34	0.14	7	20.6	22	0.09	1	5.0	182	0.77	12	6.6
Total	2,540		106	4.2	406		37	9.1	159		17	10.7	145		6	4.1	3,720		173	4.7

CFR Case fatality rate.

Rate = rate per 100,000 population.

W:P1.5,2:F1-1:ST11 (or close variants) 35% (18/51) were not ST11 based on *in silico* typing (MDU, unpublished data), and results were pending for the remaining 19 cases. All but 1 of the 33 ST11 and close variant isolates clustered with a group that has been identified in the United Kingdom (UK) and South America in recent years.[7–9] The ST11 variant was the dominant strain of MenW identified in 2015 representing 86% (25/29) of MenW strains with results so far available in that year, a 46 percentage point increase from 2014 when 4 of the 10 MenW isolates with results available were ST11.

WGS of the 33 ST11 (and close variants) identified 2 main phylogenetic clusters of MenW, cluster 1 comprised 14 cases (12 from Victoria and 2 from New South Wales), which were all genetically closely related. Meanwhile, Cluster 2 comprised 18 cases (including 5 from Queensland, 6 from Western Australia, 5 from New South Wales, 2 from Victoria) and were not closely related.

Discussion

This analysis was prompted by the recent increase in incidence of MenW in Australia, with this serogroup comprising 19% of all IMD notifications in 2015. As at 5 October 2016, 39% (67/174) of all IMD notifications and 6 of the 8 deaths (CFR of 9%) due to IMD in 2016 were due to MenW.

MenW appears on average to cause more severe disease than other circulating IMD serogroups, with the CFR over the period 2003 to 2015 being 10.7% (20.6% in 2015), compared with 4.7% for all IMD over the same period. Between 2003 and 2015 the median age of onset for MenW (44 years) was higher than that of IMD generally (17 years) and the median age of death for MenW (40 years) was twice that of all IMD. The older age of MenW cases generally may at least partially explain the increased CFR for this serogroup. However, in the more recent period between 2012 and 2015, while the median age of MenW remained older (48 years) the median age of death was younger at 26 years. Victoria has had the highest number of cases of MenW (50% of all cases in 2015), and the most marked increase in incidence, and WGS suggests most isolates from Victoria, and some from New South Wales, may be genetically related, suggesting the possibility of transmission within this region.

There are no contemporary data on nasopharyngeal carriage of meningococci in Australia. Carriage studies might help to inform understanding of the underlying prevalence and transmission dynamics of MenW strains in Victoria and other states and territories, although sample size considerations make such studies difficult to undertake.

WGS has also identified that MenW in Australia is the same hypervirulent strain that has been circulating in the UK and South America since 2009.[7,8] This is consistent with a previous review of Australian meningococcal epidemiology, which identified that intercontinental spread of clonal strains was responsible for the pattern of hypersporadic invasive disease.[10]

Cases due to this strain have rapidly increased to now comprise 25% of IMD cases in the UK in 2014 to 2015, and 59% of all cases in Chile in 2012. MenW is now considered to be endemic in these countries. The initial increase was seen in older adults, but rapidly spread across all age groups within 2 years, particularly in adolescents and infants.[11] Disease severity has been high, and in response to this hypervirulent strain of MenW, targeted vaccination programs have been initiated in the UK and Chile.[7,12]

Conjugate MenC vaccine is part of the funded National Immunisation Program for all children at the age of 12 months. A multi-component MenB vaccine is registered for use in Australia, but is not part of the funded vaccination program. However, the Australian Technical Advisory Group on Immunisation has recommended it for use in children (particularly those <24 months of age) and adolescents aged 15–19 years.[13] A number of safe and effective quadrivalent conjugate meningococcal vaccines(covering serogroups A, C, W and Y) are also registered for use in Australia, and *The Australian Immunisation Handbook* recommends their use in individuals of any age who have predisposing medical conditions associated with increased risk of IMD or who are planning travel to parts of the world where epidemics of group A, C, W or Y meningococcal disease occur.[13] It is notable that based on WGS of the Australian MenW isolates to date, there has been no evidence of Hajj-related outbreak strains, suggesting that vaccination strategies for Hajj attenders may have been successful in preventing importation of these strains.[4] Australian guidelines also recommend post-exposure vaccination of unimmunised household and other higher risk contacts of cases of IMD caused by serogroups C, A, W or Y.[1] Individuals or parents who wish to protect themselves or their children against MenW could discuss receipt of a quadrivalent conjugate vaccine with their immunisation provider, via a private prescription.

The situation in Australia with regards to IMD due to MenW is evolving and continues to be closely monitored. A national working group has been formed under the auspices of the Communicable Diseases Network of Australia to further assess the situation and ensure consistent collection of enhanced data. Lessons learned from the international experience will be important in informing the public health response.

Acknowledgements

Authors would like to gratefully acknowledge the contribution of many colleagues in jurisdictional health authorities and laboratories who assisted with collection and analysis of case data and processing of specimens specifically including:

Members of the Communicable Diseases Network Australia MenW Working Group: Frank Beard, John Bates, Gary Dowse, Monica Lahra, Stephen Lambert, Nicolee Martin, Katherine Ong, Finn Romanes, Nathan Saul, Kerrie Stevens, Deborah Williamson, Ben Howden, Tomasz Kiedrzynski

Members of the Microbiological Diagnostic Unit at the University of Melbourne for their work on WGS and Associate Prof Charlene Kahler, Shakeel Mowlaboccus and Alfred Tay for providing WGS data for Western Australian MenW cases.

Members of the National Neisseria Network

The Department of Health and Human Services Victoria: Kylie Carville, Lucinda Franklin, Asma Sohail, Aicha Brahmi, Rachel Coutts, Carol El-Hayek and Stephen Pellissier

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Associate Professor Stephen B Lambert, Senior Medical Officer, Communicable Diseases Branch, Prevention Division, Queensland Health and UQ Child Health Research Centre, Faculty of Medicine and Biomedical Sciences, The University of Queensland, Queensland

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Epidemiology of bacterial toxin-mediated foodborne gastroenteritis outbreaks in Australia, 2001 to 2013

Fiona J May, Benjamin G Polkinghorne, Emily J Fearnley

Abstract

Bacterial toxin-mediated foodborne outbreaks, such as those caused by *Clostridium perfringens, Staphylococcus aureus* and *Bacillus cereus,* are an important and preventable cause of morbidity and mortality. Due to the short incubation period and duration of illness, these outbreaks are often under-reported. This is the first study to describe the epidemiology of bacterial toxin-mediated outbreaks in Australia. Using data collected between 2001 and 2013, we identify high risk groups and risk factors to inform prevention measures. Descriptive analyses of confirmed bacterial toxin-mediated outbreaks between 2001 and 2013 were undertaken using data extracted from the OzFoodNet Outbreak Register, a database of all outbreaks of gastrointestinal disease investigated by public health authorities in Australia. A total of 107 laboratory confirmed bacterial toxin-mediated outbreaks were reported between 2001 and 2013, affecting 2,219 people, including 47 hospitalisations and 13 deaths. Twelve deaths occurred in residents of aged care facilities. *Clostridium perfringens* was the most commonly reported aetiological agent (81 outbreaks, 76%). The most commonly reported food preparation settings were commercial food preparation services (51 outbreaks, 48%) and aged care facilities (42 outbreaks, 39%). Bacterial toxin outbreaks were rarely associated with food preparation in the home (2 outbreaks, 2%). In all outbreaks, the primary factor contributing to the outbreak was inadequate temperature control of the food. Public health efforts aimed at improving storage and handling practices for pre-cooked and re-heated foods, especially in commercial food preparation services and aged care facilities, could help to reduce the magnitude of bacterial toxin outbreaks. *Commun Dis Intell* 2016;40(4):E460–E469.

Keywords: foodborne illness; bacteria-mediated; gastroenteritis; *Staphylococcus aureus; Bacillus cereus, Clostridium perfringens*

Introduction

Two different types of bacterial toxins can cause gastroenteritis. Preformed toxins are produced by *Staphylococcus aureus* and *Bacillus cereus* (emetic toxin).[1,2] These toxins are formed in the food and are resistant to heat, so the risk of illness is not removed by cooking.[3,4] Onset of illness is rapid, between 30 minutes and 6 hours, and vomiting is the most commonly reported symptom.[3] *In vivo* toxins are produced by *Clostridium perfringens* and *B. cereus* (diarrhoeal toxin), and are formed in the digestive tract after food containing the bacteria is consumed. While adequately cooking food can kill the bacterial vegetative cells, both *C. perfringens* and *B. cereus* produce heat-resistant spores that can survive cooking and subsequently regerminate after cooking. Onset of illness is between 6 and 16 hours. Diarrhoea is commonly reported and vomiting is not common.[5,6] All 3 toxin-producing bacteria are ubiquitous in the environment, and *S. aureus* is a normal component of human flora.[7]

Individual cases of *S. aureus, B. cereus* and *C. perfringens* gastroenteritis are not notifiable diseases in Australia, so gastroenteritis caused by these pathogens are only reported if they are part of an outbreak, defined as two or more cases of the same illness with a common source. Gastrointestinal outbreaks are collated in the national OzFoodNet Outbreak Register.

OzFoodNet was established in 2000 by the Australian Government as a network of epidemiologists with representatives in every state and territory. OzFoodNet focuses on enhanced surveillance for foodborne illnesses.[8] The OzFoodNet Outbreak Register is a Microsoft Access database maintained by OzFoodNet Central (at the Australian Government Department of Health), and has been in use since 2001. State and territory-based OzFoodNet epidemiologists collect and provide summary data quarterly to OzFoodNet Central on all gastrointestinal outbreaks investigated in their jurisdiction. Summaries of outbreaks are published in OzFoodNet quarterly and annual reports.[9,10]

The aim of this study was to describe the epidemiology of bacterial toxin-mediated foodborne outbreaks in Australia between 2001 and 2013, and to identify high risk groups and risk factors to inform prevention measures.

Methods

Data collection

Outbreak Register data were extracted on 2 February 2015. Variables analysed included aetiology, laboratory confirmation of aetiology, food vehicle, state or territory of outbreak, year of outbreak, number of cases, number hospitalised, number of deaths, median age of cases, per cent of cases for each gender, median incubation period and duration, number of cases reporting each symptom, factors contributing to the outbreak (microbial growth and microbial survival), the setting in which food was prepared, the consumption setting and the free text remarks variable. Application of inclusion and exclusion criteria was performed in Microsoft Excel, and data cleaning and analysis was performed in StataSE 13 (Stata Corp, College Station, TX, USA). Missing data, nonsensical data and answers of 'unknown' were treated as unknown responses. Completeness for all variables was defined as useable data, i.e. values other than missing or unknown.

Case definitions

All confirmed bacterial toxin outbreaks included in this analysis were laboratory confirmed according to simplified Centers for Disease Control and Prevention (CDC) guidelines for bacterial toxin outbreaks.[11] Outbreaks were classified as laboratory confirmed if the aetiological agent was isolated or enterotoxin was detected in clinical specimens from 2 or more cases, or at least 10^5 organisms were isolated per gram of epidemiologically implicated food.[11] Confirmed bacterial toxin-mediated foodborne or suspected foodborne outbreaks with onset between 2001 and 2013 were included. Five aetiology categories were created for analysis:

1. '*Bacillus cereus*' – *B. cereus* was listed as the sole aetiology.
2. '*Clostridium perfringens*' – *C. perfringens* was listed as the sole aetiology.
3. '*Staphylococcus aureus*' – *S. aureus* was listed as the sole aetiology.
4. 'Preformed toxin' – both *S. aureus* and *B. cereus* were listed as the confirmed aetiology.
5. '*In vivo* toxin' – both *C. perfringens* and *B. cereus* were listed as the confirmed aetiology.

For further attribution of outbreaks caused by *B. cereus*, the following criteria based on the known characteristics of *B. cereus* illness were used as probable case definitions:[2]

a. Emetic *B. cereus*
   i. incubation period $\leq$ 6 hours and
   ii. $\geq$ 50% of cases reporting vomiting

b. Diarrhoeal *B. cereus*
   i. incubation period $\geq$ 6 hours and
   ii. $<$ 50% of cases reporting vomiting.

Vehicle attribution

Confirmed or suspected food vehicles with a reasonable level of suspicion (for example the implicated food was the only food that was eaten by most or all cases) that were detailed in the Outbreak Register in either the food vehicle variable or in the free-text remarks variable were retained for analysis. To simplify analysis, the food vehicles were categorised according to the method of food preparation as proposed by Weingold et al.[12] Only the information provided in the Outbreak Register was used to apply categories and no assumptions were made about foods commonly served together. An additional food variable was created to record if a high starch food, such as rice or pasta, was reported. The number and percentage of outbreaks reporting each food category are reported.

Data analysis

Median values and ranges were calculated for numerical variables including number of cases, number of hospitalisations, number of deaths, median age, percentage of each gender, median incubation period and duration and percentage for each symptom. Histograms were constructed in Microsoft Excel using outbreak data aggregated by year based on onset date of the outbreak. Overall rates for each state and territory were calculated using population data from the Australian Bureau of Statistics,[13] and compared using Poisson regression using StataSE 13.

The per cent incidence of symptoms was calculated using the number of cases reporting the illness as the numerator, and the number of cases interviewed about the symptom as the denominator, where possible. For outbreaks where the number of cases reporting the symptoms was higher than the number interviewed, or the number interviewed was missing, the total number ill was used as the denominator. If an outbreak had no information for any symptom, the percentage of each symptom was reported as missing.

Results

A total of 107 confirmed bacterial toxin-mediated outbreaks were reported during the period 2001 to 2013, affecting 2,219 people across all states and territories with the exception of Tasmania, where no outbreaks occurred during this period. Of these people, 47 were hospitalised and 13 died; 12 deaths (92%) were residents of aged care facilities. The

number of outbreaks per year by jurisdiction is shown in Figure 1. Victoria had more outbreaks than any other state (46 outbreaks, 43%), followed by Queensland (29 outbreaks, 27%) and New South Wales (22 outbreaks 21%). The rate of bacterial toxin-mediated outbreaks reported per 10 million people for each state or territory is shown in Table 1. Comparing the rates for the states with 20 or more outbreaks, Victoria (6.8 outbreaks per 10 million) reported 2.8 times as many bacterial toxin-mediated outbreaks than New South Wales (2.5 outbreaks per 10 million), and 1.3 times as many outbreaks as Queensland (5.4 outbreaks per 10 million).

Figure 2 shows the number of outbreaks reported each year, by aetiology. Outbreaks caused by *C. perfringens* were the most frequently reported cause of bacterial toxin-mediated outbreaks (81 outbreaks, 76%; Figure 2, Table 2). All but one of the deaths were during outbreaks caused by *C. perfringens* (12 deaths, 92%).

Symptomology

Outbreaks caused by *S. aureus* had the shortest median incubation period (3 hours) of the 3 types of bacterial toxin-mediated infection (Table 3), while outbreaks caused by *C. perfringens* had the longest median incubation period (12 hours). Duration of illness was comparable between outbreaks caused by the different pathogens. Diarrhoea was the most commonly reported symptom in all outbreaks. Vomiting and nausea were most common in outbreaks caused by *S. aureus.*

Food vehicle attribution

The most frequently reported category of food associated with bacterial toxin-mediated foodborne outbreaks was the category of 'solid masses of potentially hazardous foods', such as lasagne, which was reported in 31 outbreaks (29%) (Table 4). An additional 17 outbreaks (16%) were associated with 'liquid or semi-solid mixtures of potentially hazardous foods', such as gravy. Half of the outbreaks (13 outbreaks) caused by *S. aureus,*

Figure 1: Laboratory confirmed bacterial toxin-mediated outbreaks, Australia, 2001 to 2013, by year and state or territory

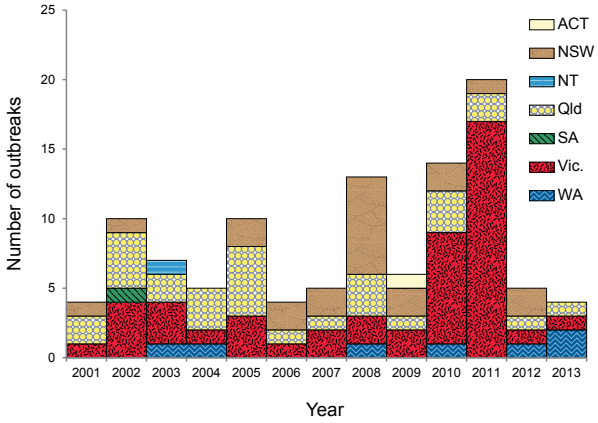

Figure 2: Laboratory confirmed bacterial toxin-mediated foodborne outbreaks, Australia, 2001 to 2013, by aetiology and year

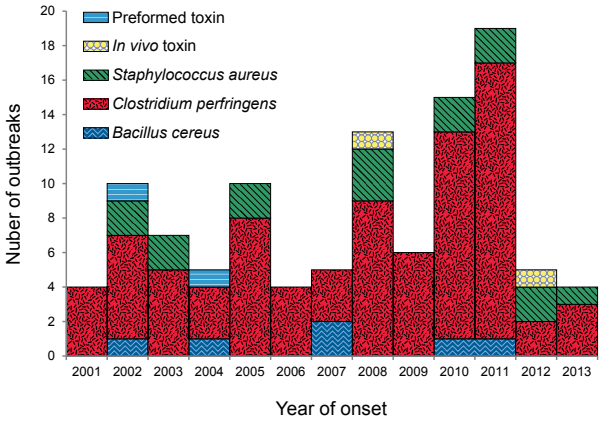

Table 1: Rate of laboratory confirmed bacterial toxin-mediated outbreaks reported per 10 million people, Australia, 2001 to 2013, by state or territory

State or territory*	Number of outbreaks	Rate per 10 million people	Incidence rate ratio	95% confidence interval
Vic.	46	6.8	Reference	Reference
Qld	29	5.4	0.78	0.50 – 1.27
NT	1	3.5	0.52	0.07 – 3.77
NSW	22	2.5	0.36	0.22 – 0.60
WA	7	2.5	0.15	0.17 – 0.82
ACT	1	2.2	0.33	0.05 – 2.37
SA	1	0.5	0.07	0.01 – 0.52
Tas.	0	0.0	0.00	0

Table 2: Epidemiology of bacterial toxin-mediated outbreaks, Australia, 2001 to 2013

		Staphylococcus aureus	*Bacillus cereus*	*Clostridium perfringens*	Preformed toxin	*In vivo* toxin
Number of outbreaks		16	6	81	2	2
Total number of cases		200	114	1,533	288	84
Median number of cases per outbreak (range)		8 (2–38)	18 (3–37)	13 (2–100)	144 (16–272)	42 (9–75)
Hospitalisations		18*	0†	14*	15†	0†
Deaths‡		1†	0†	12†	0†	0†
Per cent of outbreaks with one or more deaths		6.3†	0†	6.2†	0†	0†
Median per cent ill (range)		48 (13.3–88.9)§	26 (14.8–32.0)§	19 (0.7–100)*	8 (8.4)§	52 (4.1–100)†
Median age		31*	36§	81*	39†	20§
Median per cent sex	Male	39*	41§	34*	0‡	50§
	Female	62*	59§	66*	0‡	50§

* 75% to 89% complete.
† ≥90% complete.
‡ Deaths were temporally associated with gastroenteritis but the contribution of gastroenteritis to death is unknown.
§ 50% to 74% complete.

Table 3: Incubation period, duration of illness and median per cent of commonly reported symptoms in bacterial toxin-mediated foodborne outbreaks, Australia, 2001 to 2013, by aetiology

	Staphylococcus aureus		*Bacillus cereus*		*Clostridium perfringens*		Preformed toxin		*In vivo* toxin	
	Median	Range	Median	Range	Median	Range	Median	Range	Median	Range
Incubation period (hours)	3*	2–7	8.5*	2–12	12†	6–17	4*	2–6	12‡	12–12
Duration of illness (hours)	18‡	3–72	23.5‡	0–48	24§	3–204	24‡	24–24	36 *	24–48
Diarrhoea (%)	82§	0–100	97*	0–100	100*	53–100	86*	72–100	100*	100–100
Abdominal pain (%)	67§	0–100	35*	0–88	0*	0–100	88*	76–100	33*	0–67
Vomiting (%)	100§	43–100	14*	0–100	0*	0–74	66*	50–83	0*	0–0
Nausea (%)	83§	0–100	29*	0–100	0*	0–100	87*	74–100	0*	0–0

* ≥90% complete
† <50% complete
‡ 75% to 89% complete.
§ 50% to 74% complete.

B. cereus, preformed toxin and *in vivo* toxin had a starch-based food such as rice, pasta or noodles listed as part of the implicated food vehicle, whereas only 5 outbreaks (6%) caused by *C. perfringens* had starch as part of the food vehicle.

Contributing factors

All bacterial toxin-mediated outbreaks that had a contributing factor for microbial growth recorded (63 outbreaks) had at least one contributing factor for microbial growth that can be categorised as temperature abuse, including 'slow cooling', 'inadequate refrigeration', 'delay between preparation and consumption', 'insufficient cooking', 'inadequate thawing' or 'inadequate hot holding temperature' (Table 5). The temperature abuse growth factor was confirmed for 32 (51%) of these outbreaks. Confirmation was via observation during inspection for 15 outbreaks, verbally during inspection for 15 outbreaks and was only confirmed with measured evidence for 2 outbreaks.

Table 4: Food categories implicated in toxin-mediated outbreaks, Australia 2001 to 2013

	Staphylococcus aureus		*Bacillus cereus*		*Clostridium perfringens*		Preformed toxin		*In vivo* toxin	
	%	n	%	n	%	n	%	n	%	n
Solid masses of potentially hazardous foods	43	7	50	3	24	19	50	1	50	1
Liquid or semi-solid mixtures of potentially hazardous foods	6	1	33	2	16	13	0	0	50	1
Roasted meat/poultry/fish	19	3	0	0	7	6	0	0	0	0
Cook/serve foods	13	2	0	0	4	3	0	0	0	0
Salads prepared with one or more cooked ingredients	6	1	0	0	0	0	0	0	0	0
Salads with raw ingredients	0	0	0	0	0	0	0	0	0	0
Multiple foods	0	0	17	1	0	0	50	1	0	0
Baked goods	6	1	0	0	3	2	0	0	0	0
Sandwiches	0	0	0	0	0	0	0	0	0	0
Beverages	6	1	0	0	0	0	0	0	0	0
Unknown food vehicle	0	0	0	0	47	38	0	0	0	0

Table 5: Contributing factors for microbial growth after contamination of food vehicle, Australia, 2001 to 2013

	Staphylococcus aureus		*Bacillus cereus*		*Clostridium perfringens*		Preformed toxin		*In vivo* toxin	
	%	n	%	n	%	n	%	n	%	n
Foods left at room or warm temperature	56	9	33	2	16	13	100	2	100	2
Inadequate refrigeration	38	6	17	1	11	9	50	1	0	0
Slow cooling	6	1	0	0	30	24	0	0	0	0
Delay between preparation and consumption	6	1	17	1	12	10	50	1	50	1
Inadequate hot holding temperature	13	2	33	2	2	2	0	0	50	1
Insufficient cooking	0	0	33	2	1	1	0	0	0	0
Inadequate thawing	0	0	0	0	0	0	0	0	0	0
Anaerobic packaging/modified atmosphere	0	0	0	0	0	0	0	0	0	0
Other source of contamination	0	0	0	0	0	0	0	0	0	0
Unknown	19	3	17	1	49	40	0	0	0	0

The temperature abuse growth factor was assumed or suspected, or no level of evidence was provided for the remaining 31 outbreaks.

Similarly, 85% of outbreaks that had a contributing factor for microbial survival (40 of 47 outbreaks) had a contributing factor for microbial survival that can be categorised as temperature abuse, including 'insufficient time/temperature during cooking', 'insufficient time/temperature during reheating' or 'inadequate thawing and cooking' (Table 6). The temperature abuse survival factor was confirmed for 9 (23%) of these outbreaks. Confirmation was via observation for 2 outbreaks, verbally during inspection for 6 outbreaks and confirmed with measured evidence for 1 outbreak. The temperature abuse survival factor was assumed or suspected, or no level of evidence was provided for the remaining 31 outbreaks.

Food preparation and consumption settings

The food implicated in bacterial toxin-mediated foodborne outbreaks was prepared and eaten in the same location for 78 outbreaks (73%). The implicated food in the remaining 29 outbreaks (27%) was prepared in a commercial location before being eaten in the home, for example eating a takeaway meal from a restaurant at home. The most commonly reported food preparation locations for all aetiologies were res-

Table 6: Contributing factors for microbial survival after contamination of food vehicle, Australia, 2001 to 2013

	Staphylococcus aureus		*Bacillus cereus*		*Clostridium perfringens*		Preformed toxin		*In vivo* toxin	
	%	n	%	n	%	n	%	n	%	n
Insufficient time/temperature during reheating	0	0	33	2	41	33	0	0	0	0
Insufficient time/temperature during cooking	6	1	33	2	5	4	0	0	100	2
Other source of contamination	13	2	17	1	2	2	100	2	0	0
Inadequate or failed disinfection	6	1	0	0	0	0	0	0	50	1
Inadequate acidification	0	0	0	0	0	0	0	0	0	0
Inadequate thawing and cooking	0	0	0	0	0	0	0	0	0	0
Unknown	75	12	33	2	56	45	50	1	0	0

Table 7: Setting where the food was prepared, Australia 2001 to 2013

	Staphylococcus aureus		*Bacillus cereus*		*Clostridium perfringens*		Preformed toxin		*In vivo* toxin	
	%	n	%	n	%	n	%	n	%	n
Aged care	0	0	0	0	47	38	0	0	50	1
Restaurant	19	3	33	2	19	15	50	1	0	0
Commercial caterer	25	4	33	2	11	9	0	0	50	1
Takeaway	13	2	17	1	2	2	0	0	0	0
Hospital	0	0	0	0	6	5	0	0	0	0
Institution	0	0	0	0	6	5	0	0	0	0
National franchised fast food restaurant	13	2	17	1	1	1	0	0	0	0
Fair/festival/mobile service	6	1	0	0	0	0	50	1	0	0
Private residence	0	0	0	0	2	2	0	0	0	0
Other	6	1	0	0	1	1	0	0	0	0
Camp	6	1	0	0	0	0	0	0	0	0
Child care	6	1	0	0	0	0	0	0	0	0
Grocery store/delicatessen	6	1	0	0	0	0	0	0	0	0
Military	0	0	0	0	1	1	0	0	0	0
Private caterer	0	0	0	0	1	1	0	0	0	0
Unknown	0	0	0	0	1	1	0	0	0	0

taurants (21 outbreaks, 20%) and commercial caterers (16 outbreaks, 15%; Table 7). Only 2 outbreaks (2%) were due to food prepared in a private home. Food preparation businesses (including restaurants, commercial caterers, takeaway locations, grocery stores, delicatessens, fairs, festivals and mobile food services) were the most commonly reported food preparation setting associated with bacterial toxin-mediated foodborne outbreaks, (51 outbreaks, 48%). A total of 42 outbreaks (39%), all caused by *C. perfringens* (alone or with *B. cereus* in the *in vivo* toxin category), were associated with meals prepared and/or consumed in aged care facilities (Table 8). The incidence of aged care associated outbreaks varies from year to year, ranging from no aged care outbreaks in 2001 to 75% of outbreaks in 2013 and a median of 25% of outbreaks per year. Restaurants were the second most frequently reported location for consumption of food (21 outbreaks, 20%; Table 8).

Further attribution of *Bacillus cereus* outbreaks

Using the probable case definition, in particular the percentage of cases that reported vomiting, 1 outbreak caused by *B. cereus* (17%) was likely to have been caused by the emetic toxin (incubation period 2 hours, 100% of cases reported vomiting), while the remaining 5 outbreaks were likely to have been caused by the diarrhoeal toxin (Table 9).

Table 8: Setting where the food was consumed, Australia, 2001 to 2013

	Staphylococcus aureus		*Bacillus cereus*		*Clostridium perfringens*		Preformed toxin		*In vivo* toxin	
	%	n	%	n	%	n	%	n	%	n
Aged care	0	0	0	0	51	41	0	0	50	1
Restaurant	13	2	33	2	17	14	0	0	0	0
Private residence	19	3	50	3	5	4	0	0	0	0
Commercial caterer	13	2	17	1	4	3	50	1	50	1
Other	13	2	0	0	5	4	0	0	0	0
Institution	0	0	0	0	6	5	0	0	0	0
Community	13	2	0	0	4	3	0	0	0	0
Hospital	0	0	0	0	4	3	0	0	0	0
Fair/festival/mobile service	6	1	0	0	0	0	50	1	0	0
Military	0	0	0	0	1	1	0	0	0	0
National franchised fast food restaurant	6	1	0	0	0	0	0	0	0	0
Child care	6	1	0	0	0	0	0	0	0	0
Function	6	1	0	0	0	0	0	0	0	0
Camp	6	1	0	0	0	0	0	0	0	0
Cruise/airline	0	0	0	0	1	1	0	0	0	0
School	0	0	0	0	1	1	0	0	0	0
Unknown	0	0	0	0	1	1	0	0	0	0

Table 9: Informative variables and assessment of probable toxin type for confirmed and suspected *Bacillus cereus* outbreaks (emetic or diarrhoeal), Australia, 2001 to 2013

Probable *B. cereus* toxin type	Incubation (hours)	Symptoms		Food vehicle
		Vomiting (%)	Diarrhoea (%)	
Emetic	2	100	0	Fried rice and honey chicken
Diarrhoeal	6	0	100	Multiple foods
Diarrhoeal	8	16	100	Boiled gefilte fish (fish balls)
Diarrhoeal	9	67	67	Mashed potato and gravy
Diarrhoeal	10	3	97	Rice
Diarrhoeal	12	13	96	Rice (and/or beef curry)

Discussion

This study was the first to examine the epidemiology of bacterial toxin-mediated foodborne outbreaks in Australia. The incidence of bacterial toxin-mediated foodborne outbreaks fluctuated over the 13 year analysis period (2001 to 2013), but there was no overall trend in the change in incidence. During the analysis period, 107 outbreaks were confirmed to be caused by a bacterial toxin in Australia. In comparison, during the same period 571 outbreaks of salmonellosis, 80 outbreaks of ciguatera fish poisoning and 68 outbreaks of campylobacteriosis were investigated in Australia[9,10,14–30] Victoria reported more outbreaks per 100,000 people caused by bacterial toxins than any other jurisdiction. From the data available, it is unclear whether this was due to a higher incidence of bacterial toxin-mediated outbreaks in Victoria, or if other factors were involved.

The median incubation periods and symptomology of *S. aureus,*[31] *B. cereus*[6] and *C. perfringens*[5] in Australian outbreaks were similar to that observed elsewhere. Only 1 outbreak caused by *B. cereus* was likely to have been caused by the emetic toxin (17%). The food vehicle for this outbreak included rice (fried rice and honey chicken), consistent with findings that the *B. cereus* emetic toxin is associated with rice.[2,3,32] This distribution of a greater number of diarrhoeal outbreaks than emetic is consistent with the epidemiology of *B. cereus* in North America and Northern Europe, and different to that seen in countries with high rates of rice consumption such as Japan.[2]

The 2 most commonly reported food vehicle categories were 'solid masses of potentially hazardous foods' and 'liquids or semi-solid mixtures of potentially hazardous foods'. Without careful temperature control, both of these categories of foods can spend a long period of time at temperatures that promote microbial growth due to the density of the food. This finding is in contrast with a study examining bacterial toxin-mediated foodborne outbreaks in the United States of America (USA), which found that 'roasted meat and poultry' was the most commonly reported food category.[33]

All outbreaks that reported a contributing factor for microbial growth and 85% of outbreaks that reported a contributing factor for microbial survival reported at least one factor that was associated with temperature abuse of the food, although this was not always confirmed. Temperature abuse refers to inappropriate holding of food products between 4°C and 60°C, which is the optimal temperature for growth of most pathogenic microorganisms.[34] This is a particular problem with toxin-producing bacteria as even reheating or cooking the food does not remove the preformed toxin (*S. aureus* and emetic *B. cereus*) or the bacterial spores (*C. perfringens* and diarrhoeal *B. cereus*).[2,4,35]

The most commonly reported location for preparation of the food vehicle that was implicated in bacterial toxin-mediated foodborne outbreaks was at food preparation businesses such as restaurants and commercial caterers (48%), while the implicated food was prepared in private homes in only 2% of the outbreaks. This is in contrast to a study in the USA, which found that 16% of bacterial toxin-mediated outbreaks were associated with food prepared in the home.[33] Similarly, a study in the European Union found that homes were the most commonly reported setting for outbreaks of *S. aureus* and the third most commonly reported setting for outbreaks of *C. perfringens*.[36] However, the European Union study did not distinguish between preparation and consumption settings. Education of all food preparation services on safe food practices, with a focus on increased awareness of temperature abuse of foods that are difficult to cool or warm rapidly, including high risk dishes, would reduce the incidence of bacterial toxin-mediated foodborne outbreaks in food preparation businesses.

The most commonly reported location for preparation and consumption of the food implicated in *C. perfringens* outbreaks was aged care facilities (39% of all outbreaks, 51% of *C. perfringens* outbreaks). Foods in aged care facilities are often prepared in bulk and stored for a period of time before serving, increasing the risk of bacterial toxin outbreaks.[37] Food prepared in aged care facilities was not reported as a major risk factor for bacterial toxin-mediated foodborne outbreaks in the USA.[33,38] The short duration and mild symptoms associated with bacterial toxin-mediated illness means that cases and outbreaks in the general community are less likely to be detected and investigated than cases and outbreaks in aged care facilities. However, residents of aged care facilities are a vulnerable population, and the outcome of bacterial toxin-mediated illnesses may be more severe in the aged care population and as such, staff are trained to be particularly observant of symptoms of gastroenteritis.[39] Indeed, almost all deaths were associated with bacterial toxin-mediated outbreaks occurred in aged care facilities (92%), consistent with studies showing higher mortality during foodborne outbreaks in aged care facilities.[37,40–42] Prevention of bacterial toxin-mediated foodborne outbreaks in aged care facilities through education and awareness of ways to avoid temperature abuse of food served in aged care facilities is important in protecting this vulnerable population. Food safety in aged care facilities is regulated by Food Standards Australia New Zealand Standard 3.3.1 *Food Safety Programs for Food Service to Vulnerable Persons* (https://www.comlaw.gov.au/Series/F2012L00290). This standard was introduced in 2008 and requires implementation of a food safety program by food businesses that prepare food for vulnerable people, including the elderly. However, despite the introduction of this Standard during the period of this study, there has been no decrease in the frequency of bacterial toxin-mediated outbreaks in aged care facilities.

Only outbreaks that were laboratory confirmed to be caused by a bacterial toxin were included in this study. As it can be difficult to confirm the causative agent in bacterial toxin-mediated outbreaks, and few laboratories in Australia are able to test for these pathogens, many outbreaks that were possibly caused by bacterial toxins but not laboratory confirmed have not been included in this study. This may have biased the analysis towards outbreaks that were more likely to be confirmed, such as larger outbreaks, outbreaks in vulnerable populations such as aged care or commercial enterprises complying with regulations; the results of this study should be considered in this context. However, a larger study that incorporated suspected outbreaks showed no differences in the epidemiology of confirmed bacterial toxin-mediated outbreaks and suspected bacterial toxin-mediated outbreaks.[43] Similarly, the outbreaks reported in the OzFoodNet Outbreak Register are likely to be only a proportion of the total number of outbreaks caused by bacterial toxins, as these outbreaks are often not reported or investigated due to the short duration and relatively mild symptoms in healthy adults compared with other infectious causes of foodborne gastroenteritis such as *Salmonella*.[44]

In conclusion, bacterial toxin-mediated foodborne outbreaks are most frequently reported to be associated with dense large volume foods prepared by food preparation businesses such as restaurants, and in aged care facilities. As bacterial toxin-mediated outbreaks disproportionately affect the vulnerable residents of aged care facilities, education and training of food handlers in these facilities should be a priority.

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Adverse events following immunisation with bacille Calmette-Guérin vaccination: baseline data to inform monitoring in Australia following introduction of new unregistered BCG vaccine

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Abstract

In recent years there has been a global shortage of bacille Calmette-Guérin (BCG) vaccine and, from September 2012, unregistered vaccines have needed to be used in Australia (a Danish product initially until the end of 2015, and a Polish product used in some jurisdictions from early 2016). We examined rates and types of adverse events following immunisation (AEFI) with BCG vaccine reported to the Therapeutic Goods Administration between 2009 and 2014 in children aged less than 7 years. Reporting rates of AEFI with BCG vaccine increased from 87 per 100,000 doses (registered Sanofi Pasteur product) in 2009 to 201 per 100,000 doses (unregistered Danish Statens Serum Institute product) in 2014, with Victoria having the highest rate each year. Substantial variation between jurisdictions exists, suggesting differential reporting of BCG vaccine doses administered and/or BCG vaccine-related AEFI. The most commonly reported reactions were abscess (31%), injection site reaction (27%) and lymphadenopathy/lymphadenitis (17%). This study provides baseline data on BCG vaccine safety to inform surveillance. Given the current use of unregistered vaccines in the context of vaccine supply issues, improved recording of both administered BCG vaccine doses and the reporting of BCG vaccine-related AEFI are required to facilitate close monitoring of vaccine safety. *Commun Dis Intell* 2016;40(4):E470–E474.

Keywords: adverse events; bacille Calmette-Guérin; vaccination

Introduction

In Australia, bacille Calmette-Guérin (BCG) vaccine programs to protect against tuberculosis (TB) are funded by state and territory governments, rather than under the National Immunisation Program (NIP). Given the low incidence of TB in Australia, current Australian guidelines recommend BCG vaccination for groups of people who are at an increased risk of TB, in particular children aged less than 5 years who will be travelling to or living for an extended period of time in countries with a high prevalence of TB (annual TB incidence of 40 per 100,000 or more), and Aboriginal and Torres Strait Islander neonates in high-incidence communities (currently implemented in Queensland, the Northern Territory and northern South Australia only).[1,2]

BCG vaccine has been in use since 1921.[3] As a result of repeated passage under different conditions in different laboratories, BCG vaccine strains have diverged genetically.[4] The Sanofi Pasteur BCG vaccine is the only product registered for use in Australia but has been unavailable since a recall was issued in June 2012 due to a possible breach in the sterility of the product following a flood at the manufacturing plant.[5] As of September 2012, an alternative unregistered vaccine (BCG Denmark-Serum Statins Institute (SSI)) was supplied under provisions of Section 19A of the *Therapeutic Goods Act 1989,* which allows for importation and supply of products that are registered in the specified countries under Section 19A(3) of the *Therapeutic Goods Act 1989*.[6] However, this product has also been unavailable in Australia since 1 January 2016 and a critical shortage of BCG vaccine has been reported globally since 2013.[7,8] From early 2016, another alternative unregistered Polish BCG vaccine (BCG-10) has been supplied in some jurisdictions in Australia. Whilst BCG-10 has been manufactured and registered for use in Poland since 1955[9] and is also available (but not registered) in some other European countries,[7] Poland is not one of the Section 19A(3) specified countries. As such, it is only able to be supplied via the 'Authorised Prescriber Scheme' or Special Access Scheme.[6]

BCG vaccine is considered safe, however, it is one of the more reactogenic vaccines currently available and reactogenicity may differ between BCG vaccine strains.[10] Currently there are no available longitudinal national data on adverse events following immunisation (AEFI) with BCG vaccine in Australia. To provide baseline data and inform monitoring following introduction of BCG-10 vaccine, or other unregistered vaccines, we examined

rates and reaction types of BCG vaccine-related AEFI between 2009 and 2014 in children aged less than 7 years, by jurisdiction and vaccine type.

Methods

De-identified information on BCG vaccine-related AEFI that were reported to the Therapeutic Goods Administration and entered into the Adverse Drug Reactions System (ADRS) database were extracted from a dataset released to the National Centre for Immunisation Research and Surveillance in March 2015. AEFI data where BCG vaccine was recorded as suspected of involvement were included in the analysis for children vaccinated at less than 7 years of age between 1 January 2009 and 31 December 2014.

The number of BCG vaccine doses administered was obtained from the Australian Childhood Immunisation Register (ACIR). AEFI reporting rates per 100,000 doses and 95% confidence intervals were calculated and analysed by jurisdiction and age group. Adverse reaction types were also examined. Using the date of vaccination, we determined the number of BCG vaccine doses that had been administered for each vaccine product (Sanofi Pasteur or Denmark-SSI) and the number and reporting rate of AEFI related to each of these products.

All data analyses were performed using SAS software version 9.4 (SAS Institute Inc. Cary, NC, USE) and Excel 2010 (Microsoft, Redmond, PA, USA).

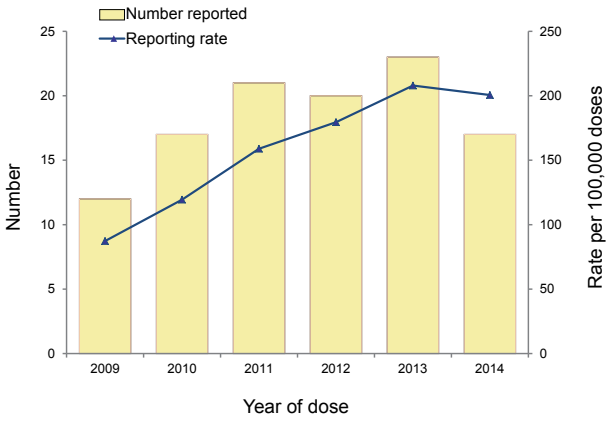
Results

Table 1 shows the number of BCG vaccine doses recorded on the ACIR between 2009 and 2014, by jurisdiction. Queensland is recorded as administering the highest number of doses each year. The ADRS database for the period 1 January 2009 to 31 December 2014 included a total of 110 AEFI reports related to BCG vaccination in children aged less than 7 years. Fifty-eight per cent of BCG AEFI reports were for males, and 68% were for children aged less than 1 year. The rate of reported BCG-related AEFI in children aged less than 7 years varied substantially between Australian states and territories, with Victoria having the highest rate each year (Table 1).

The rate of reported BCG vaccine-related AEFI increased nationally from 87 per 100,000 doses in 2009 to 201 per 100,000 doses in 2014 (Figure 1).

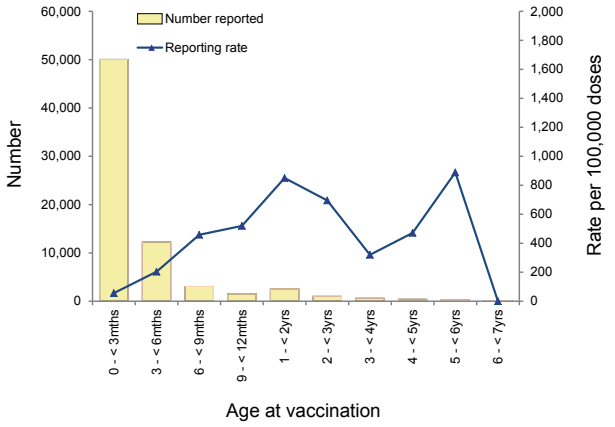
The large majority of BCG doses were recorded as administered to children aged less than 3 months, with the rate of reported BCG vaccine-related AEFI in this age group relatively low at 56 per 100,000 doses (Figure 2).

Figure 1: Adverse events following immunisation with bacille Calmette-Guérin vaccine in children aged less than 7 years, Australia, 2009 to 2014, by year of vaccination



Source: Australian Childhood Immunisation Register and the Australian Adverse Drug Reactions System database.

Figure 2: Number of bacille Calmette-Guérin vaccine doses recorded as administered and adverse events following immunisation reporting rate per 100,000 doses in children aged less than 7 years, Australia, 2009 to 2014, by age at vaccination



Source: Australian Childhood Immunisation Register and the Australian Adverse Drug Reactions System database.

The 110 BCG vaccine-related AEFI reports involved 150 reaction types recorded in the ADRS database. The most commonly reported reactions were abscess (31%), injection site reaction (27%) and lymphadenopathy/ lymphadenitis (17%).

Table 1: Number of bacille Calmette-Guérin vaccine doses recorded as administered and adverse events following immunisation reporting rate per 100,000 doses in children aged less than 7 years, Australia, 2009 to 2014, by state or territory and year

State or territory	2009			2010			2011			2012			2013			2014		
	Doses	AEFI rate / 100,000 doses	95% CI	Doses	AEFI rate / 100,000 doses	95% CI	Doses	AEFI rate / 100,000 doses	95% CI	Doses	AEFI rate / 100,000 doses	95% CI	Doses	AEFI rate / 100,000 doses	95% CI	Doses	AEFI rate / 100,000 doses	95% CI
ACT	202	0	0–1826	181	0	0–2038	166	0	0–2222	145	0	0–2544	86	1,163	29–6479	55	0	0–6707
NSW	2,033	0	0–181	2,111	0	0–175	1,866	0	0–198	1,674	0	0–220	1,180	85	2–472	716	0	0–515
NT	1,125	267	55–779	1,018	98	3–547	1,098	182	22–658	969	103	3–575	780	128	3–714	615	0	0–600
Qld	5,782	0	0–64	6,516	15	1–86	6,025	0	0–61	5,206	19	1–107	6,007	67	18–170	4,264	0	0–87
SA	466	0	0–792	423	0	0–872	358	0	0–1030	276	0	0–1337	183	0	0–2016	87	0	0–4240
Tas.	51	0	0–7233	52	0	0–7094	46	0	0–8019	40	0	0–9222	28	0	0–13175	10	0	0–36889
Vic.	2,693	334	153–634	2,845	457	243–781	2,804	678	408–1058	2,228	763	445–1222	2,359	678	388–1101	2,496	681	397–1090
WA	1,389	0	0–266	1,094	183	22–660	853	0	0–432	607	165	4–918	440	0	0–838	233	0	0–1583
Aus.	13,741	87	45–153	14,240	119	70–191	13,216	159	98–243	11,145	180	110–277	11,063	208	132–312	8,476	201	117–321

Source: Australian Childhood Immunisation Register and Australian Adverse Drug Reactions System database.

Table 2: Bacille Calmette-Guérin vaccine-related adverse event following immunisation number and reporting rate per 100,000 doses in children aged less than 7 years, Australia, 2009 to 2014, by vaccine product and year

Year of BCG vaccination	Sanofi Pasteur product		Denmark-Serum Statins Institute product	
	Number of AEFI	AEFI rate per 100,000 doses (95% CI)	Number of AEFI	AEFI rate per 100,000 doses (95% CI)
2009	12	87 (45 –153)	–	–
2010	17	119 (70–191)	–	–
2011	21	159 (98–243)	–	–
2012	13	191 (102–327)	7	161 (65–332)
2013	–	–	23	208 (132–312)
2014	–	–	17	201 (117–321)

– Denotes bacille Calmette-Guérin (BCG) vaccine product not in use.

AEFI Adverse event following immunisation.

Source: Australian Childhood Immunisation Register and Australian Adverse Drug Reactions System database.

The number of BCG vaccine-related AEFI reports from the Sanofi Pasteur product and the Denmark-SSI product were 63 (57%) and 47 (43%) respectively. Table 2 displays the number of BCG vaccine-related AEFI reports and the reporting rate per 100,000 doses by vaccine product.

Discussion

The rate of reported BCG vaccine-related AEFI in children aged less than 7 years appears to have been increasing in Australia since 2009. Consistent with previously published Australian and international data, we found that 58% of BCG AEFI reports were for male children,[11,12] and that the rate of reported AEFI generally increased with increasing age at time of BCG vaccination.[11,13] The most frequently reported BCG-related AEFI in children under the age of 7 years were localised abscesses, injection site reactions and lymphadenopathy, consistent with other published data from Australia and overseas.[9,11–14]

Our data shows substantial variation between jurisdictions in the reporting of BCG doses administered and BCG-related AEFI. However, this could be influenced by differential reporting of vaccination and/or AEFI. As BCG vaccination is not funded under the NIP and there are no incentives for immunisation providers to report BCG vaccination to the ACIR, it is likely that there is under-reporting of BCG vaccine dose administration. Under-reporting of AEFI is also acknowledged to be a major limitation of all passive AEFI surveillance systems including those used in Australia, due to the reliance on voluntary reporting by immunisation providers, other health professionals and consumers.[15] As such the calculation of AEFI rates in our study is limited by potential inaccuracies in both numerator and denominator data. There has also been a general increase in reporting of AEFI related to other vaccines in children over our study period.[16] Our finding that Victoria consistently had the highest BCG vaccine-related AEFI reporting rate could be in part due to the establishment of an enhanced passive surveillance system in 2007, which was shown to significantly improve AEFI reporting rates in its first 3 years of operation.[17]

Whilst our study cannot draw any conclusions about the different reactogenicity of the Sanofi product and the Denmark SSI product, due to the limitations discussed above, reactogenicity is reported to differ between BCG vaccine strains.[10] The Polish BCG-10 vaccine that has been used in some jurisdictions in Australia in 2016 and is not included in this study, is derived from yet another BCG strain, BCG-Moreau. Although a low (0.2%–0.6%) frequency of BCG-AEFI has been reported from passive surveillance in Poland, understanding of its vaccine safety profile is based on this single study, and should be prospectively monitored using baseline data on other strains from Australia.[9]

Conclusion

As the use of unregistered BCG vaccines is likely to be needed for the foreseeable future in Australia, close monitoring of vaccine safety will be important. Our study provides baseline AEFI data against which to monitor the introduction of new, and particularly unregistered BCG vaccines, in Australia. However, improving data quality in relation to both the recording of administered BCG vaccine doses and the reporting of BCG vaccine-related AEFI will be essential to facilitate this monitoring.

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Public health action following an outbreak of toxigenic cutaneous diphtheria in an Auckland refugee resettlement centre

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Abstract

Global forced displacement has climbed to unprecedented levels due largely to regional conflict. Degraded public health services leave displaced people vulnerable to multiple environmental and infectious hazards including vaccine preventable disease. While diphtheria is rarely notified in New Zealand, a 2 person outbreak of cutaneous diphtheria occurred in refugees from Afghanistan in February 2015 at the refugee resettlement centre in Auckland. Both cases had uncertain immunisation status. The index case presented with a scalp lesion during routine health screen and toxigenic *Corynebacterium diphtheriae* was isolated. A secondary case of cutaneous diphtheria and an asymptomatic carrier were identified from skin and throat swabs. The 2 cases and 1 carrier were placed in consented restriction until antibiotic treatment and 2 clearance swabs were available. A total of 164 contacts were identified from within the same hostel accommodation as well as staff working in the refugee centre. All high risk contacts (n=101) were swabbed (throat, nasopharynx and open skin lesions) to assess C. *diphtheriae* carriage status. Chemoprophylaxis was administered (1 dose of intramuscular benzathine penicillin or 10 days of oral erythromycin) and diphtheria toxoid-containing vaccine offered regardless of immunisation status. Suspected cases were restricted on daily monitoring until swab clearance. A group of 49 low risk contacts were also offered vaccination. Results suggest a significant public health effort was required for a disease rarely seen in New Zealand. In light of increased worldwide forced displacement, similar outbreaks could occur and require a rigorous public health framework for management. *Commun Dis Intell* 2016;40(4):E475–E481.

Keywords: diphtheria, cutaneous, outbreak, refugees, vaccination

Introduction

The global displaced population has climbed to unprecedented levels with a worldwide total of 59.5 million individuals forcibly moved by 2014 and no simple resolution in sight.[1,2] Public health measures are critical for these humanitarian emergencies including management of vaccine preventable diseases (VPD) since displaced people are more likely to be inadequately immunised.[3] Recent studies by the World Health Organization (WHO) reveal 'varied and non-standardised criteria' used by various government agencies to control vaccine preventable outbreaks.[4,5]

Diphtheria is a rare disease in New Zealand primarily due to high immunisation coverage.[6] Diphtheria is caused by a polypeptide exotoxin of *Corynebacterium diphtheria*.[7] Severe clinical illness results from absorption of the toxin to the pharynx, nasal lining or skin, producing low grade fevers, and a pharyngeal pseudomembrane can classically develop over 2 to 3 days.[8] Cutaneous disease can also occur with indolent non-healing skin lesions that ulcerate. Diphtheria antitoxin (DAT) developed in the 1890s in horses hyperimmunised with diphtheria toxoid has dramatically reduced mortality.[9] DAT is used prophylactically in the treatment of diphtheria while the advent and widespread use of diphtheria toxoid containing vaccines makes the disease vanishingly rare in the developed world.[10] Cases are largely observed among unimmunised individuals or their contacts with recent travel history to countries where diphtheria remains endemic.[11] These countries include Afghanistan, Bangladesh, Cambodia, China, India, Indonesia, Malaysia, Nepal, Pakistan, Papua New Guinea, the Philippines, Thailand, Vietnam and the Pacific Islands.

Guidance for the management of diphtheria cases and contacts had been previously developed by the New Zealand Ministry of Health (MoH) following the Centers for Disease Control and Prevention (CDC) recommendations.[12] The disease is notifiable with a confirmed case definition of clinically compatible respiratory and/or cutaneous illness that is laboratory confirmed with a toxigenic isolate of *C. diphtheriae* or epidemiologically linked to a laboratory confirmed case. A probable case is a clinically compatible illness that is not laboratory confirmed.

A recent review of national surveillance data in New Zealand showed 1 case of diphtheria was reported in each of the years 1987, 1998, 2002 and

2008[6] and 2 unrelated cases in 2014.[13] The last recorded outbreak occurred in 2009 consisting of 2 cases in Wellington following the incomplete treatment of a person returning with disease from Samoa and resulting in contact tracing of 27 people.[6] This paper reports the public health response to an outbreak of 2 cases of cutaneous diphtheria in a group of Afghani refugees who had recently arrived in New Zealand from Pakistan, resulting in the follow up and contact tracing of 164 people at the Mangere Refugee Resettlement Centre (MRRC).

Case presentations

Three Afghani children were identified with culture-proven toxigenic *C. diphtheriae;* 2 with cutaneous lesions and 1 with asymptomatic pharyngeal carriage. All had spent the proceeding 4 years in Pakistan prior to arrival in New Zealand in January 2015.

Index case

A 7-year-old Afghani girl was identified at the MRRC when she had her routine health screening, which is mandatory for all new arrivals. She was noted to have an impetigous scalp lesion, which was cultured. Initial microbiology results identified *Staphylococcus aureus*, *Streptococcus pyogenes* and *C. diphtheriae*. This was reported 6 days later to be a *C. diphtheriae* toxin-producing strain by the National Reference Laboratory, Institute of Environmental Science and Research (ESR). This index case had no written record of immunisation. She had been started on flucloxacillin initially with significant resolution of the lesion, and on advice from the paediatric infectious diseases physician she was restricted with her family under voluntary consent and changed to amoxicillin/clavulanate for a further 14 days. She was assessed for consideration of prophylactic DAT but this was not available within New Zealand. The scalp infection improved and she did not develop any clinical signs of respiratory diphtheria. Pharyngeal carriage of *C. diphtheriae* was also noted so she remained in consented restriction until 2 sets of negative swabs from the nasopharynx, throat and scalp lesion were obtained 24 hours apart after completion of the antibiotic course.

Secondary case

A 6-year-old refugee Afghani girl from Pakistan had a small bullious lesion on the plantar surface of her right foot which developed after arrival in New Zealand and while resident at the MRRC. Toxigenic *C. diphtheriae* was isolated from it. She was from the same contact group, hostel block and attended the same school classes as the index case. Throat and nasopharyngeal swabs were both negative. She had been given intramuscular benzathine penicillin the day before the lesion swab result was known and vaccinated with diphtheria toxin containing vaccine (tetanus–diphtheria–acellular pertussis (Tdap) Boostrix© GSK). She remained in consented restriction on 14 days of erythromycin for eradication until 2 sets of clearance negative swabs from the nasopharynx, throat and foot lesion were obtained 24 hours apart on completion of the antibiotic course.

Asymptomatic carrier

The 11-year-old sister of the index case was identified during the contact tracing with asymptomatic pharyngeal carriage of toxigenic *C. diphtheriae*. She had been given intramuscular benzathine penicillin and Tdap vaccine 24 hours before the swab result was known as part of the screening. She remained in consented restriction for 7 days until 2 sets of clearance swabs were obtained from the nasopharynx and throat 24 hours apart.

Public health action

A public health response was initiated on the date of notification (2 February 2015) and managed as per MoH protocols.[12] A total of 164 contacts were identified, of which 1 became the secondary case. There were 3 rounds of contact tracing completed according to priority (Figure).

Group 1 – High risk close contacts including family, people with respiratory symptoms or skin lesions, other contacts in the same hostel, children in the MRRC school class and their families, as well as contact staff (n=101).

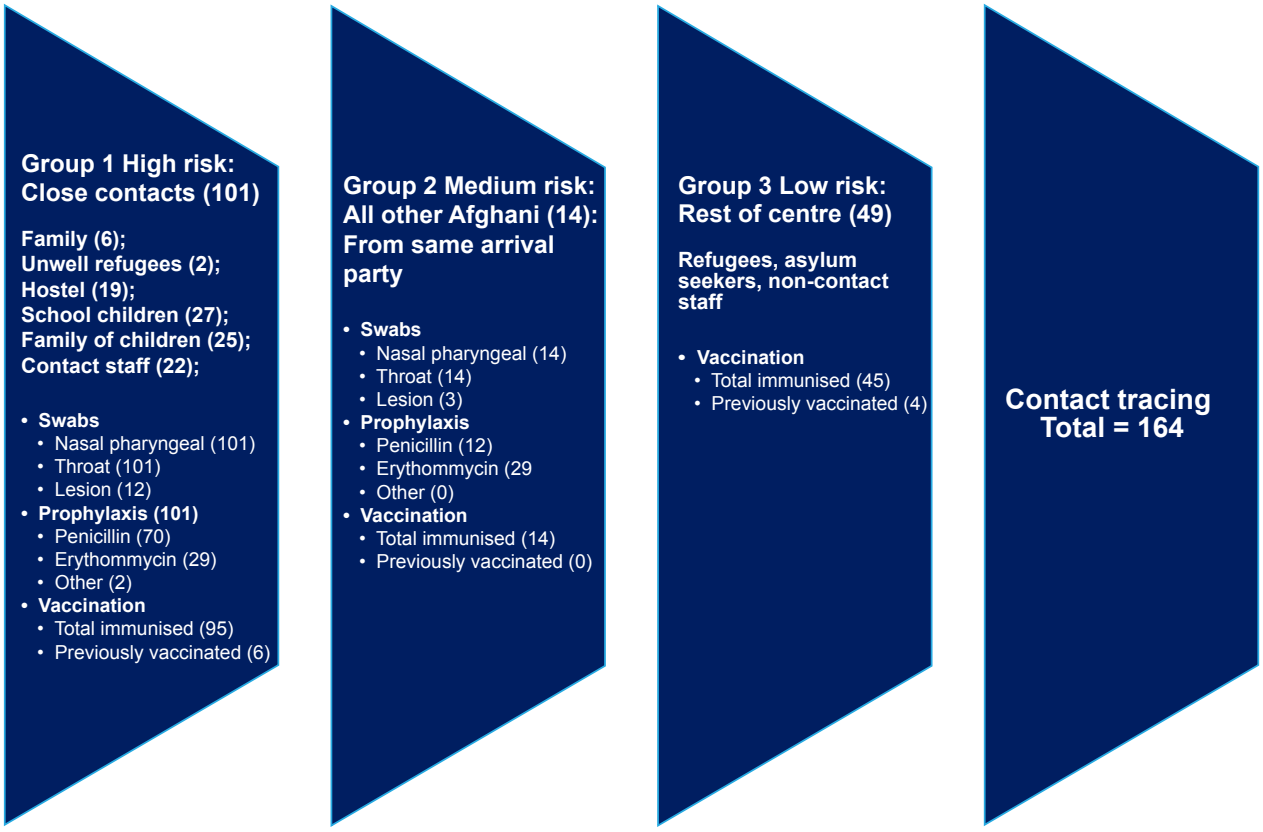
Group 2 – Medium risk contacts included all other Afghani people at the centre who arrived in the same party from Pakistan though not in direct close contact (n=14).

Group 3 – Deemed low risk, were the rest of the MRRC refugees and asylum seekers and non-contact staff (n=49).

Groups 1 and 2 had nasopharyngeal and throat swabs collected for screening for *C. diphtheriae* and open lesion skin wounds were swabbed regardless of history. Groups 1 and 2 were given antibiotic prophylaxis and immunised with Tdap after swabbing. The low risk contact group 3 were offered Tdap vaccine unless they had previously documented vaccine in the past 5 years.

The MRRC is an area that is fenced and gated allowing only staff or pre-approved visitors to have access to the site. Refugees who did not require vol-

Figure: Risk groups for diphtheria contact tracing, Mangere Refugee Resettlement Centre, Auckland, 2015



A total of 164 contacts were followed up at the MRRC in 3 contact tracing rounds according to risk. Groups were classified as high, medium and low risk of toxigenic diphtheria including; high risk close contacts of the Afghani case (group 1), medium risk all other Afghani refugees in the same arrival party (group 2) and low risk, all other refugee, asylum seekers and non-contact staff at the MRRC (group 3).

untary consented restriction, were free to come and go from the centre and so had access to the local community. During the outbreak routine medical assessment, including screening and health care continued as normal.

Outbreak information and the process of informed consent required the use of appropriate interpreters in 4 language streams. All refugees are routinely invited to consent to the collection of health information to provide the appropriate care; plan for and fund health services; carry out teaching and research; and monitor quality. Where their information was used for research or monitoring quality, it would not be published in a manner that identified that person. Approval from a human research ethics committee was not required but extra informed consent was invited for the outbreak management. Full ethical permission is not required for a public health action.

Skin, nasopharyngeal and throat swabs from the 164 contacts were cultured on sheep blood agar plates incubated at 35º C for 24–48 hours in 5% CO_2-enriched atmosphere. Suspicious Gram positive bacilli resembling *Corynebacterium* species morphologically were identified by MALDI-TOF (bioMerieux MS, Durham, NC, US). Isolates confirmed as *C. diphtheriae* were then sent to the ESR for detection of toxigenic strain using polymerase chain reaction amplification of the toxin gene (*tox*).

While the *C. diphtheriae* can persist for several days after antibiotics,[14] the preferred chemoprophylaxis was a single dose of intramuscular benzathine penicillin or 10 days of oral erythromycin where intramuscular doses were impractical or there was penicillin hypersensitivity. Many of the refugee children received oral erythromycin due to practical constraints related to correct intramuscular benzathine penicillin dosing.

Refugees with no documented vaccination history are generally considered non-immune. Tdap was offered rather than adult diphtheria-tetanus (ADT) for vaccination because they would normally be offered Tdap as a catch-up within the

first 4 weeks of arrival. A total of 154 contacts were immunised (137 refugees and 17 staff); while a further 10 contacts who had documented evidence of Tdap or ADT and were not offered a booster. Staff involved in the response were required to know their own vaccine history, but vaccination history was checked via occupational health records where possible. Tdap was also administered to public health outbreak response staff who had not been vaccinated in the previous 5 years.

The public health nurse team donned full personal protective equipment (PPE consisting of gowns, gloves and surgical masks) to swab, administer chemoprophylaxis and immunise the high risk group. All high risk contacts received follow-up daily symptom checks by the public health nurse for 7 days from their last contact with the case. Skin lesions were covered with dressings and the public health nurse assessed them during their daily symptom checks

Probable cases (n=6) who displayed potential clinically compatible diphtheria pharyngeal or cutaneous symptoms were identified, reviewed and treated by medical staff. They were then restricted until negative swab cultures were obtained (usually 2–3 days) in accordance with MoH and CDC manual guidelines.[12]

All contacts were educated on transmission prevention methods and effective hand hygiene measures. A health protection officer from the public health unit assessed the MRRC and provided environmental cleaning recommendations to staff. Extensive commercial cleaning of high risk areas was undertaken, including the hostel accommodation the cases used as a restriction facility, the medical centre and school classroom.

Discussion and public health significance

This outbreak involved 2 cases of confirmed toxigenic cutaneous diphtheria and an asymptomatic throat carrier of toxigenic *C. diphtheriae,* which did not satisfy the case definition though a reservoir for disease. The public health response to this outbreak was the largest recorded group contact trace for diphtheria in New Zealand history since the notification process began in 1956 and involved intensive resource allocation. The emergency action in this refugee resettlement centre for such a rarely notified disease highlights 4 internationally pertinent issues for any future VPD outbreaks occurring in these vulnerable refugee populations. These concerns are detailed in the sections below.

1. *A diphtheria outbreak could readily happen again with international pressure to increase refugee intakes and could involve other VPD, including polio and measles.*

Arriving refugees are a heterogeneous group with varied medical needs based on the country of origin and country of transit, the length of time as a refugee and quality of healthcare prior to arrival.[15] All displaced people are at increased risk of VPD due to poor vaccine availability and failing public health systems in their country of origin.[3] The rapidly increasing annual rate of forced global displacement, more markedly over the last 3 years,[16] in regions of conflict and political instability makes a VPD outbreak more likely at borders and refugee intake centres.

Unlike other immigrants, refugees tend to arrive with minimal documentation of immunisation as well as very limited history of previous clinical illness. Re-vaccination as soon after arrival as practical is recommended within New Zealand where there is no valid documentation.[17] Previous studies have shown it is cost effective to re-start catch-up immunisation soon after resettlement rather than using screening measures such as serological testing.[18] While the MRRC protocol allowed for fully funded catch-up immunisation within the first 4 weeks of arrival at the centre, full immunisation prior to arrival in New Zealand would be preferable. There are a number of logistical barriers to preclude this option including funding, consistent vaccine supply, cold chain and vaccine delivery into areas of political instability.

An extensive public health response was more feasible and manageable because the refugees all reside in 1 site for the first 6 weeks after arrival in New Zealand and would provide further challenges if it had occurred in the community. A purpose built resettlement centre for all refugees and asylum seekers entering the country allowed consistent and strategic health screening soon after arrival. It has the added advantage of quick diagnosis and management of any VPD outbreaks.

2. *There is a need for a consistent and rigorously tested framework for planning and managing of rare VPD emergencies including diphtheria.*

Since diphtheria is rare, testing the disease management framework for best practice has been difficult. For this reason the protocol was risk averse[12] and included a rigorous public health intervention of consented restriction of the cases and infection prevention and control (Standard and Contact Precautions), swabbing, chemoprophylaxis and a complete course of vaccine or a booster as appropriate for all close contacts. A more tested public

health response might be less risk averse and result in more efficient resource allocation. Testing the framework would require an international effort and rely on good communication and formal debrief after such outbreaks, to identify any issues. Internationally sharing information would help to optimise management in future situations.

A recent review of literature by WHO[4] confirms a range of criteria used by organisations and government agencies to plan and implement urgent public health responses such as vaccination in outbreak situations. Very little data are available to evaluate the process of how decisions are made to assess rare disease and VPD management frameworks. There is a clear need for a framework to ensure a standardised and consistently applied methodology for decision making both nationally and internationally. The use of such a framework would minimize mortality, maximize resources, reduce wastage, ensure equity, and ultimately improve accountability to the population at risk and other stakeholders.[4]

3. *Readiness and preparedness of public health services for VPD emergencies including rarely accessed medical treatments such as DAT.*

The readiness and preparedness of public health services to manage and plan for VPD outbreaks needs regular review. This requires the training and availability of staff to diagnose, then manage the acute outbreak with the resultant temporary reduction in other services. The sharing of information and strong communication networks are vital when such a rare disease is involved. Health care workers working with refugees need to have a thorough knowledge of the causes of communicable diseases in developing countries to facilitate the appropriate collection of clinical specimens, avoiding diagnostic delay and result in timely, appropriate management. Mechanisms also need to be in place to deliver this information on a national basis to all primary care health workers who will have long-term care of the refugee populations.

Maintaining high levels of immunity for all New Zealanders, particularly those travelling to high risk areas is recommended. Despite high levels of community and refugee health worker immunity to diphtheria, only a small group of the total number of health workers at the centre had documented immunisation status reflecting the lack of application of the health and safety policies and procedures. There were also special occupational health requirements identified for interpreters, teachers and administrators not usually at high risk of contact.

A national shortage of anti-toxin in New Zealand and neighbouring Australia was identified by this outbreak and global access to DAT continues to be restricted. Despite ongoing efforts to secure supplies, rapid access to DAT would currently not be possible. While a case of clinical pharyngeal or cutaneous diphtheria with systemic manifestations would be unlikely, the MoH's CDC manual 2012 recommends DAT "before laboratory confirmation when there is strong clinical suspicion of diphtheria". While the literature regarding anti-toxin (DAT) in cutaneous diphtheria is unclear,[19] antitoxin is not usually given due to lack of pseudomembranes or cardiac involvement.[20] These 2 children clinically had no evidence of systemic disease and so anti-toxin was not indicated.

Neither case of cutaneous diphtheria identified in this outbreak had typical features. *C. diphtheriae* is a well-recognised cause of skin infections in children both in New Zealand and in Pacific Island countries though the isolates are largely non-toxigenic. While invasive disease has also been reported,[21] cutaneous diphtheria usually presents as indolent, non-healing lesions often with no characteristic features. Children with cutaneous diphtheria rarely develop the pharyngeal form of the disease or systemic manifestations, probably due to a brisk antibody response, but the ulcer acts as a reservoir to infect susceptible hosts. This makes timely diagnosis very important, particularly in situations where there is a closed community with a large number of vulnerable refugee families with suboptimal vaccination and often significant health issues.

4. *There are implications from this diphtheria outbreak for the wider public health and emergency response policy framework including non-VPD outbreaks.*

This outbreak of rarely notified and potentially life threatening diphtheria has implications for wider public health emergency response and planning. Policy needs to include consideration of:

1. the varied needs of this vulnerable group of people about to resettle in a different country;
2. the temporary arrival into a single screening facility is a good model and highlights the need for the appropriately funded purpose built infrastructure for refugees;
3. appropriate information technology infrastructure for the sharing of knowledge and experience after rare VPD and non-VPD outbreaks to the national and international scientific and medical community can direct best practice protocols for management;

4. a well trained group of health professionals that recognise the specific needs of refugees will also be aware of the array of communicable diseases from other parts of the world and their treatment;
5. adequately funded and trained public health units that are equipped to respond to VPD and non-VPD emergencies; and
6. agencies caring for newly arrived refugees need to have a robust health and safety policy to keep their staff and other refugees safe against VPD and non-VPD hazards.

Summary

We describe a 2 person outbreak of diphtheria in a New Zealand refugee centre resulting in the follow-up of 164 contacts and this is timely with the rapid increase in global displacement. It highlights the need for rigorous policy for countries who are receiving refugee populations as these events could readily happen again. The outbreak shows that a rare VPD can be imported from areas of humanitarian need and requires clear systems and protocols for management. Finally it emphasises the need for readiness and preparedness and the importance of integration of VPD and non-VPD into emergency planning.

Acknowledgements

The authors wish to thank the refugees who consented to this study as part of the public health response. Thank you to the public health medical, nursing and administrative staff at Auckland Regional Public Health for their dedicated efforts to control this diphtheria outbreak.

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Australian vaccine preventable disease epidemiological review series: Influenza 2006 to 2015

Jean Li-Kim-Moy, Jiehui Kevin Yin, Cyra Patel, Frank H Beard, Clayton Chiu, Kristine K Macartney, Peter B McIntyre

Abstract

Introduction: Influenza is a major contributor to the preventable health burden of Australians each year. The National Immunisation Program provides influenza vaccine for those at highest risk of severe disease. This review of influenza epidemiology examines current data on influenza disease burden in Australia, in the context of several comparable countries having programs with much broader eligibility for influenza vaccine in children.

Methods: Influenza notifications (2006–2015), hospitalisations, and deaths (2006–2013) were sourced and age-specific rates calculated. Comparisons were made across age groups in the pre-pandemic, pandemic, and post-pandemic periods and by Indigenous and non-Indigenous status.

Results: The 2009 pandemic year and the 2012 non-pandemic season resulted in the highest rates of notification, hospitalisation and death. Influenza notification rates were 4.0 times higher and hospitalisation rates 2.1 times higher during 2011–2013 compared with 2006–2008. Death rates varied widely, but peaks corresponded to high-activity seasons. Influenza hospitalisation rates were highest among those aged <5 and ≥65 years, but influenza-attributable deaths were identified primarily in those aged ≥75 years. Significantly higher notification and hospitalisation rates were seen for all Indigenous people, but higher death rates were largely restricted to the 2009 pandemic year.

Conclusions: Based on notifications, hospitalisations and deaths, burden of disease from influenza is highest at the extremes of life and is significantly higher among Indigenous people of all ages. This pattern of disease burden warrants consideration of widened eligibility for influenza vaccine under the National Immunisation Program to all Indigenous people and all children less than 5 years of age. *Commun Dis Intell* 2016;40(4):E482–E495.

Keywords: influenza, epidemiology, review, Australia, hospitalisation, notifications, deaths, Indigenous

Introduction

Influenza is an acute respiratory viral infection caused by influenza viruses, A, B, or C and which causes a substantial global burden of disease. The World Health Organization estimates that influenza causes 3 to 5 million cases of severe illness, and about 250,000 to 500,000 deaths globally each year.[1] In developed countries such as Australia, influenza is one of the leading vaccine preventable causes of morbidity and mortality each year. Previous epidemiological reviews have highlighted this burden particularly in the young and the elderly.[2,3]

There is a large focus on vaccination each season to prevent influenza disease. Australia has an established National Immunisation Program (NIP) with funded influenza vaccination primarily aimed at individuals at highest risk of severe disease or complications from influenza. Beginning in 1999, funded vaccination was available for all adults aged ≥65 years and Indigenous people aged ≥50 years or 15–49 years with chronic medical conditions predisposing them to severe complications of influenza. Expanded funding to cover all individuals aged ≥6 months with chronic medical conditions, all Indigenous people aged 15–49 years, and pregnant women commenced in January 2010. In March 2015, funded vaccination was extended to Indigenous children aged 6 months to <5 years. In 2016, quadrivalent influenza vaccine replaced trivalent influenza vaccine as the funded influenza vaccine on the NIP.[4]

This report provides information on the age-specific disease burden of influenza during the 2006–2015 period and the population prevalence of medical conditions associated with increased risk of severe influenza, with the aim of informing consideration of extension of current influenza vaccination programs in Australia. We used narrow age ranges to provide further detail of disease burden in children, in light of recent overseas programs such as that undertaken in the United Kingdom, to address overall community influenza burden by wider vaccination of children.[5] Influenza trends were also examined by comparing the pre-pandemic period

(2006–2008), the pandemic year (2009) and the post-pandemic period (2010–2015) during which there was broadened eligibility of funded influenza vaccination under the NIP.

Methods

Data sources

Notifications

Notifications for influenza are reported across Australia to the National Notifiable Diseases Surveillance System (NNDSS) and consist of laboratory-confirmed influenza, either by isolation of influenza virus by culture, nucleic acid testing, or virus antigen testing from an appropriate respiratory tract specimen; or IgG seroconversion (significant increase in antibody level or a 4-fold or greater rise in titre to influenza virus), or a single high titre by complement fixation test or haemagglutination inhibition to influenza virus.[6]

Influenza has been a notifiable disease in all jurisdictions since 2001 except for Tasmania (2002), the Australian Capital Territory (2006) and South Australia (May 2008). Notification data were obtained from NNDSS for the period 2006–2015. For 2006–07 where South Australian notification data may not have been complete, these were not included in rate calculations for those years. Notifications were examined by Indigenous status only in Western Australia and the Northern Territory, where the completeness of Indigenous status was greater than 90%; other jurisdictions had suboptimal completeness of recording of Indigenous status ranging from 6% to 82%.

Hospitalisations

De-identified data on influenza hospitalisations was limited by availability to calendar years 2006–2013 and were obtained from the Australian Institute of Health and Welfare (AIHW) National Hospital Morbidity Database. Influenza hospitalisations were defined as those that were coded as J09 (influenza due to identified avian influenza virus (2006–2012), or avian influenza and the influenza A/H1N1 pandemic strain (2013)), J10 (influenza due to identified virus) or J11 (influenza, virus not identified) for either principal or associated other diagnosis, according to the International Statistical Classification of Diseases and Related Health Problems, 10th Revision, Australian Modification (ICD-10-AM). We interpreted and classified the J09 and J10 codes as virologically-confirmed and J11 as non-virologically confirmed influenza. This dataset was subject to the limitations of being unable to identify duplicated admissions due to inter-hospital transfers or hospital readmissions for an individual during the same illness. We restricted analyses of disparity in influenza hospitalisation between Indigenous and non-Indigenous Australians to data from 2010 onwards, which are considered by AIHW to have acceptable completeness of Indigenous status coding across all jurisdictions.[7]

Deaths

Data on deaths recorded during calendar years 2006–2013 were obtained from Australian Bureau of Statistics (ABS) Causes of Deaths data according to the International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10) coding (J09, J10, or J11) and were analysed by year of death. ICD-10 coding incorporated A/H1N1 pandemic influenza within J09 coding from 2010. We included all cases where influenza was coded as a principal 'underlying' cause of death or any of other contributing causes of death. Data for 2012–2013 were preliminary.

Population estimates

Population estimates were obtained from age-specific mid-year estimated resident population data available from the ABS (Catalogue 3101.0, Table 59, Series B, published 17/12/2015) to calculate population rates.[8,9] National data on total population estimates were complete; however Indigenous population estimates were only available up to 2011 with population projections used for 2012–2013 (Catalogue 3238.0, Table 9, Series B, published 30/04/2014). Additionally, Indigenous population estimates for those ≥ 65 years of age were available as a whole and not further age-stratified.[9] Therefore, in order to obtain population estimates for age groups 65–74 and > 75 years, we used proportions from previously published population projections[10] that were provided by single year of age for all ages and applied them to the most current available census population numbers. Non-Indigenous population estimates were obtained by subtracting Indigenous population estimates from the total estimates for the corresponding age groups.

Population prevalence of medical conditions associated with increased risk of severe influenza

Data on the proportion of the Indigenous, non-Indigenous and total Australian population who report having at least 1 of the medical conditions associated with increased risk of severe influenza by age group, together with the corresponding relative standard error (RSE), were obtained from the relevant survey data provided by the ABS. The estimates for Indigenous Australians were obtained from the 2012–13 National Aboriginal and Torres Strait Islander Health Survey, while

estimates for non-Indigenous ('Other') Australians and the total Australian population were obtained from the 2011–13 ABS National Health Survey (National Health Survey component, 2011–12). All survey data were self-reported. As the medical conditions captured and coded in the surveys do not exactly correspond to those listed in *The Australian Immunisation Handbook,* we selected the conditions that best match the list (Appendix). For the Indigenous survey, only persons living in non-remote areas were asked to self-report severe asthma. As prevalence estimates were captured from different surveys, direct comparisons between Indigenous and non-Indigenous populations cannot be drawn.

Analyses

All collected data were analysed according to the following age groups: 0–5 months, 6–23 months, 2–4 years, 5–11 years, 12–17 years, 18–24 years, 25–49 years, 50–64 years, 65–74 years and ≥75 years. Rates per 100,000 population were calculated using STATA/MP 13.1 (StataCorp LP, USA), both inclusive and exclusive of the 2009 pandemic year. Due to variability in A/H1N1 coding within J09/J10 during the reporting period, data were not sub-analysed by J09/J10. The 95% confidence intervals (CI) of rates were calculated assuming a Poisson distribution. Where relevant, we used non-overlapping confidence intervals to assess the statistical significance of differences in rates. Indigenous versus non-Indigenous rates were examined by rate ratios to assess for disparity of influenza burden. Comparisons of rates were made between pre-pandemic (2006–2008), pandemic (2009) and post-pandemic periods (2010–2013 for hospitalisations and deaths, 2010–2015 for notifications). The 95% confidence intervals for estimates of the prevalence of medical conditions were calculated manually using the following formula: p +/– 1.96(p x RSE); where p is the population prevalence of medical conditions, and RSE is the relative standard error of the prevalence estimate.

Results

Notifications

Notifications for influenza numbered 363,934 during the reporting period 2006–2015. There was a substantial increasing trend in notification rates from 18 per 100,000 (2006) to 423 per 100,000 (2015) (Figure 1). This was further demonstrated by average annual notifications increasing from 7,693 (2006–2008) to 65,528 (2013–2015). The 2009 A/H1N1 pandemic year was associated with a large peak of notifications (272 per 100,000) relative to surrounding seasons. However, this peak rate has subsequently been exceeded in the 2014 and 2015 seasons.

In all non-pandemic years (2006–2015), influenza notification rates were highest in children aged 6–23 months, with high rates for all children aged 0–11 years (244–327 per 100,000) (Table 1). There were relatively lower notification rates for those aged 12–74 years (112–153 per 100,000), with higher rates in the elderly aged ≥75 years (202 per 100,000).

During the 2009 A/H1N1 pandemic, peak notifications occurred in the 12–17 year age group, followed by children aged 5–11 years (Figure 2,

Table 1: Influenza notification rates per 100,000 population, Australia, 2006 to 2015, by year and age group, and total

Age group	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	All years excl. 2009	All years
Age group-specific notification rate per 100,000												
0–5 m	130	322	179	358	130	333	432	199	444	502	300	306
6–23 m	87	259	132	419	134	294	454	228	596	683	327	337
2–4 y	30	127	69	407	96	228	452	210	546	764	296	307
5–11 y	18	67	53	496	94	210	314	190	331	823	244	269
12–17 y	18	46	54	543	70	129	176	100	235	522	153	192
18–24 y	17	48	43	399	65	110	134	67	186	305	112	141
25–49 y	11	41	34	237	60	112	164	116	270	336	132	143
50–64 y	14	37	33	143	45	81	132	114	254	300	119	121
65–74 y	17	37	34	72	33	78	163	103	249	335	130	125
≥75 y	27	73	45	70	34	93	293	130	426	577	202	190
All ages	18	55	43	272	61	122	196	122	288	423	155	167
Total number of notifications												
All ages	3,320	10,586	9,173	59,026	13,469	27,213	44,564	28,308	67,704	100,571	304,908	363,934

Table 1). This contrasts with the pre-pandemic seasons (2006–2008) where the highest notifications occurred in children aged 0–5 months with decreases in the notification rate with increasing age. In the post-pandemic period (2010–2015), the highest notifications were in slightly older children aged 6–23 months and 2–4 years.

Figure 1: Notification rate for influenza, Australia, 2006 to 2015, by year and age group

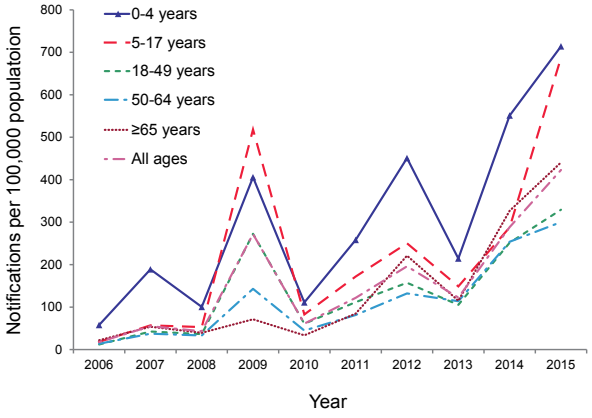
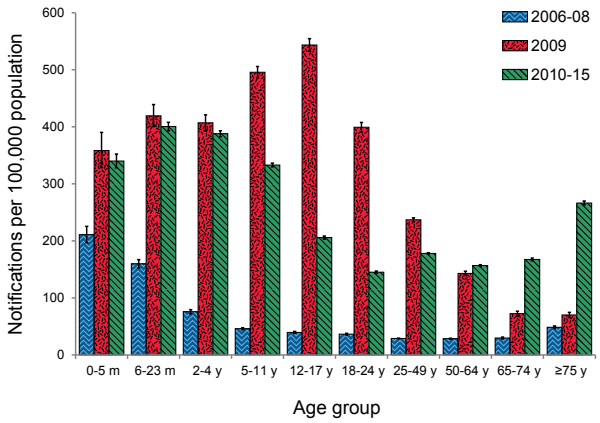
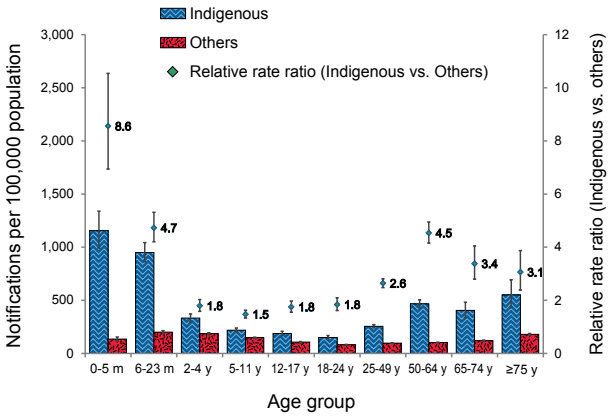


Figure 2: Notification rate for influenza, with 95% confidence intervals comparing pre-pandemic (2006–2008), pandemic (2009) and post-pandemic (2010–1015) time periods, Australia, by age group



Notification rates in Indigenous Australians compared with others, during 2006–2015 (excluding 2009), using Western Australian and Northern Territory notification data only, were statistically significantly higher for all ages, particularly for children aged 0–23 months and adults ≥25 years of age. Rate ratios ranged from 1.5 to 8.6 depending on age (Figure 3).

Figure 3: Notification rate for influenza with 95% confidence intervals, Western Australia and the Northern Territory*, 2006 to 2015 (excluding 2009), by age group and Indigenous status



* Western Australia and the Northern Territory had greater than 90% completeness of Indigenous status recorded.

All rate ratios significant with 95% confidence intervals excluding one.

Hospitalisation

During the period 2006 to 2013 there were 41,140 influenza hospitalisations (average 5,143 per year). The 2 highest years of hospitalisation occurred during the 2009 A/H1N1 pandemic (34 per 100,000) and in 2012 (rate 44 per 100,000) (Table 2). Combining all non-pandemic years, hospitalisation rates were highest in children aged 0–5 months (192 per 100,000), followed by those aged 6–23 months (109 per 100,000) (Table 2), more than 4 and 2 times respectively the rate in those aged ≥75 years. Among children, hospitalisation rates were lower with increasing age. Children ≥5 years of age through to adults <65 years of age had similar and relatively low hospitalisation rates (10–18 per 100,000), with thereafter progressive increases in hospitalisation with increasing age in the elderly (30 per 100,000 for 65–74 years, and 46 per 100,000 for ≥75 years).

In non-pandemic years, large peaks in hospitalisation rates for children aged 0–4 years were seen in 2007 and 2012 and for the elderly aged ≥65 years in 2012 (Figure 4). There was a slight increase in all-age hospitalisation rate during 2006-2013 which was less marked than the increase in notification rate over the same time period (Figures 4 and 5). Overall, the 2009 A/H1N1 pandemic year hospitalisation rate was statistically significantly higher than in other years in individuals aged 5–64 years, but comparable or lower than averaged rates for the post-pandemic period for children aged 0–4 years and those aged ≥65 years (Figure 6).

Table 2: Influenza hospitalisation rates per 100,000 population, Australia, by year and age group and total

Age group	2006	2007	2008	2009	2010	2011	2012	2013	All years excl. 2009	All years
Age group-specific hospitalisation rate per 100,000										
0–5 m	113	236	152	158	113	242	326	152	192	187
6–23 m	64	153	86	102	70	119	170	98	109	108
2–4 y	18	51	27	41	20	39	76	37	39	39
5–11 y	7	17	9	22	7	20	31	17	16	16
12–17 y	6	10	11	21	6	12	15	13	10	12
18–24 y	8	13	9	29	11	16	23	10	13	15
25–49 y	6	14	8	30	12	18	27	19	15	17
50–64 y	6	13	10	33	13	20	35	29	18	20
65–74 y	10	19	15	36	12	31	71	42	30	31
≥75 y	16	38	25	57	19	49	122	46	46	48
All ages	9	21	14	34	14	25	44	26	22	24
Total number of hospitalisations										
All ages	1,879	4,384	2,955	7,335	3,018	5,602	9,930	6,037	33,805	41,140

Figure 4: Rate of ICD-coded hospitalisations for influenza (principal or other diagnosis), Australia, 2006 to 2013, by year and age group

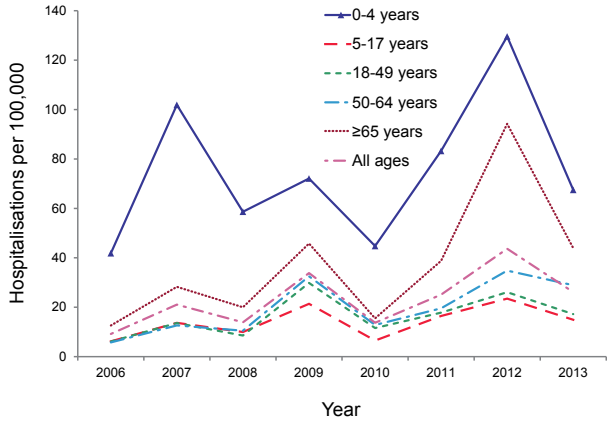


Figure 5: Rate for influenza notifications (2006–2015) and hospitalisations (2006–2013), Australia, by year

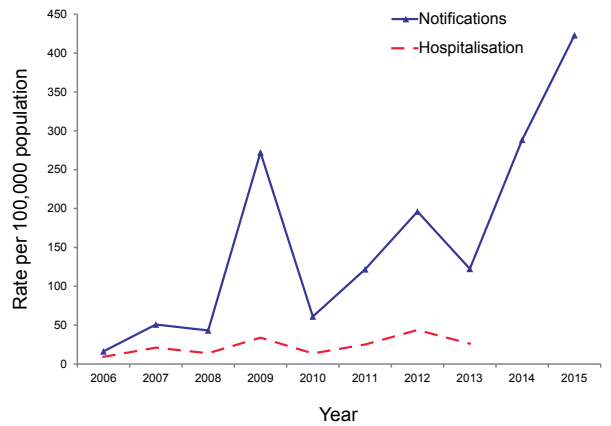
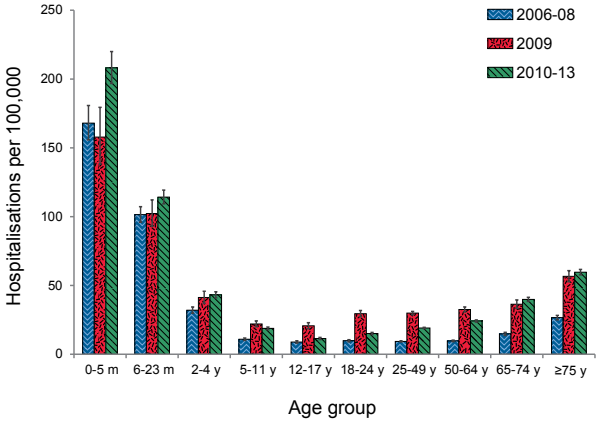


Figure 6: Rate of ICD-coded hospitalisation for influenza (any diagnosis) with 95% confidence intervals, Australia, 2006 to 2013, by age group and time period

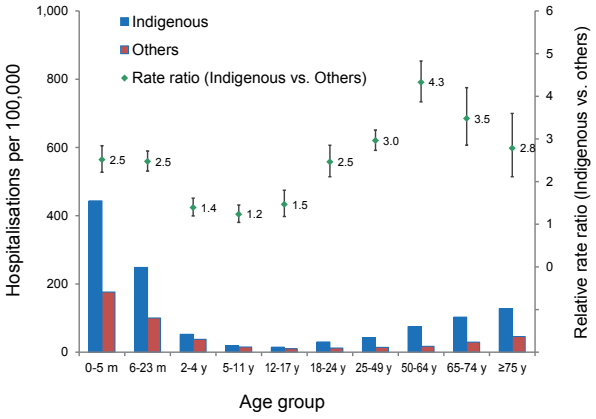


Small but significant increases were noted in hospitalisation rates for all age groups when comparing post-pandemic (2010–2013) with pre-pandemic (2006–2008) time periods (Figure 6). These increased rate ratios were more pronounced in those aged ≥25 years (incidence rate ratio 2.0–2.7). While aggregate hospitalisation rates (2010–2013) for adults aged 65–74 years and ≥75 years during the post-pandemic period were not dissimilar to those in the 2009 pandemic year. These consisted of a combination of lower hospitalisation rates in 2010 and considerably higher rates in 2012 (Figure 4).

The overall completeness of Indigenous status recording among the data used in the Indigenous hospitalisation data analysis was 98%. During 2010

to 2013, rates of influenza-related hospitalisation in Indigenous people were significantly higher than that in non-Indigenous people across all age bands with rate ratios ranging from 1.2 to 4.3 (Figure 7). Compared with the non-Indigenous population, increases in the hospitalisation rate were more pronounced in children aged 0–23 months and adults aged ≥18 years, with the highest rate ratio seen in the subgroup of adults aged 50–64 years (rate ratio 4.3).

Figure 7: Rate of ICD-coded hospitalisation for influenza (any diagnosis) with 95% confidence intervals, Australia, 2010 to 2013, by age group and Indigenous status



All rate ratios significant with 95% confidence intervals excluding one.

The proportion of influenza hospitalisations with diagnostic codes suggesting virological confirmation (J09, J10) remained high throughout the 2006 to 2013 period for children aged 0–4 years (80% to 99%) (Figure 8). In all other age groups there was a progressive increase in the proportion of hospitalisations recorded as having virological confirmation, with the exception of the 2009 pandemic year when a decline in the proportion of virologically confirmed hospitalisations occurred for most age groups.

Deaths

During 2006 to 2013, a total of 807 deaths were coded with influenza as a principal or contributing cause. The all-age death rate was 0.46 per 100,000 (Table 3), peaking both during the 2009 A/H1N1 pandemic (0.89 per 100,000) and in 2012 (0.91 per 100,000) with a smaller peak during 2007 (0.48 per 100,000) (Figure 9). There was a higher proportion of deaths classified as virologically confirmed by ICD coding in the 2009 pandemic (74%) than during other years (19%–54%).

Excluding the pandemic year, the highest rate of death due to influenza during 2006 to 2013 was observed in persons aged ≥75 years (3.66 per 100,000) and 65–74 years (0.65 per 100,000) (Table 3). Death rates in young children aged 0–4 years ranged from 0.20–0.39 per 100,000 with wide confidence intervals due to low absolute numbers of deaths. Mortality was lowest in the age range 5–49 years (0.04–0.11 per 100,000).

Figure 8: Proportion of influenza hospitalisation recorded as being virologically confirmed (J9-10) of total (any diagnosis: J09-J11), Australia, 2006 to 2013, by year and age group

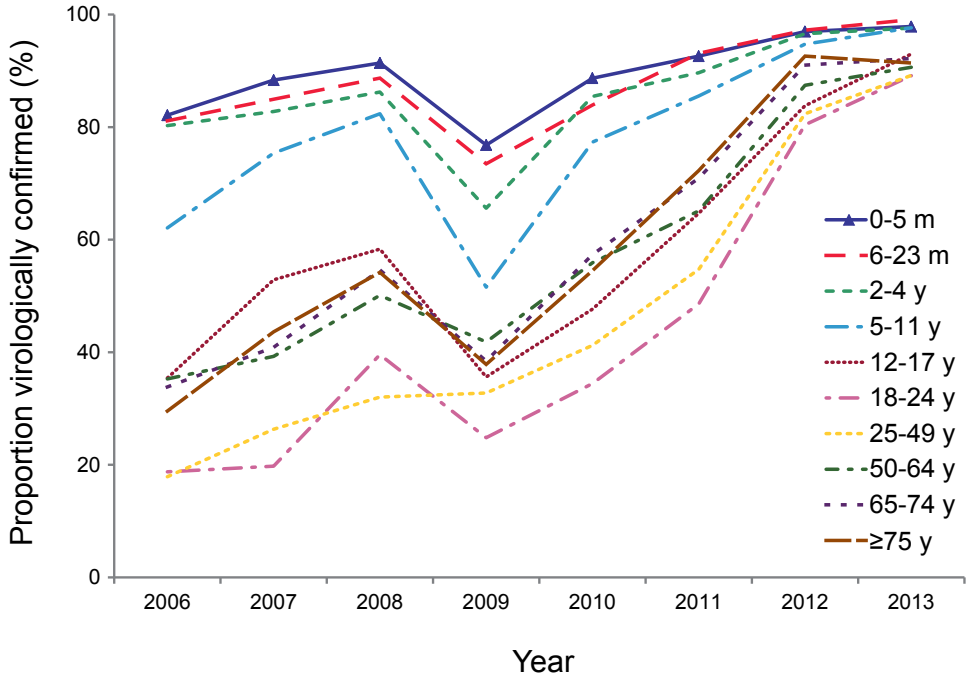


Table 3: Influenza death rates per 100,000 population, Australia, 2006 to 2013 by year and age band and absolute numbers of deaths, by year

Age group	2006	2007	2008	2009	2010	2011	2012	2013	All years excl. 2009	All years
Age group-specific death rate per 100,000										
0–5 m	0.00	0.70	0.00	0.00	0.67	0.69	0.66	0.00	0.39	0.34
6–23 m	0.77	0.00	0.23	0.68	0.23	0.23	0.22	0.00	0.23	0.29
2–4 y	0.00	0.77	0.00	0.24	0.12	0.11	0.45	0.00	0.20	0.21
5–11 y	0.05	0.05	0.00	0.11	0.05	0.10	0.05	0.00	0.04	0.05
12–17 y	0.00	0.06	0.00	0.24	0.06	0.00	0.12	0.00	0.03	0.06
18–24 y	0.05	0.05	0.00	0.32	0.14	0.18	0.05	0.04	0.07	0.10
25–49 y	0.00	0.12	0.05	0.76	0.14	0.16	0.16	0.15	0.11	0.20
50–64 y	0.06	0.24	0.32	1.21	0.50	0.44	0.27	0.29	0.31	0.42
65–74 y	0.21	0.83	0.27	1.55	0.43	0.71	1.57	0.38	0.65	0.76
≥75 y	1.18	4.63	2.73	3.42	1.24	2.92	10.08	2.44	3.66	3.63
All ages	0.12	0.48	0.27	0.89	0.29	0.42	0.91	0.29	0.40	0.46
Total numbers of deaths										
All ages	25	100	57	194	63	93	207	68	613	807

Figure 9: Rate for all influenza deaths virologically confirmed cases, Australia, 2006 to 2013, by year

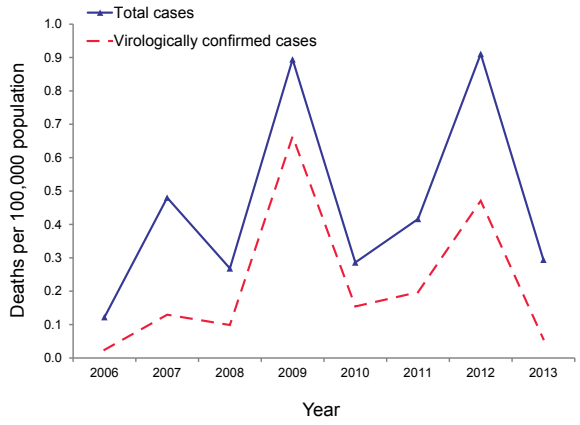
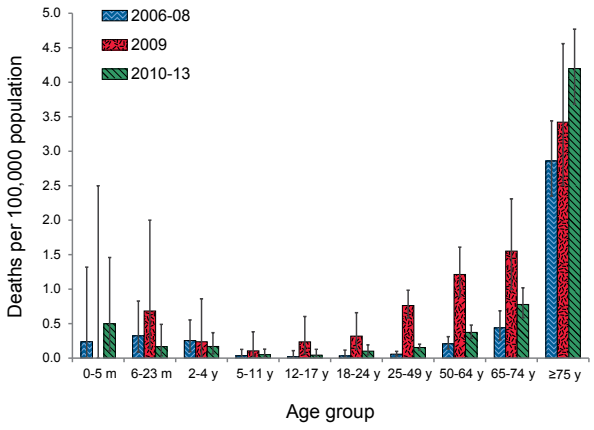


Figure 10: Rate for influenza deaths with 95% confidence intervals, Australia, 2006 to 2013, by age group time period



During the 2009 A/H1N1 pandemic, death rates for those aged 25–64 years were significantly elevated compared with pre- and post-pandemic periods (Figure 10). Smaller, non-significant increases in deaths were seen during the pandemic in the 5–17 year and 65–74 year age bands. In those aged ≥75 years, the death rate was not significantly higher in the pandemic year, but was higher in the post-pandemic period (4.20 per 100,000) compared with the pre-pandemic period (2.86 per 100,000), largely due to a high number of deaths during 2012 in this age group (rate 10.08 per 100,000).

Completeness of recording of Indigenous status within death data was high at 99%. Analysis of deaths by Indigenous status (Figure 11 and 12) demonstrates significantly elevated death rates for Indigenous Australians during the 2009 pandemic with a rate ratio of 3.35 (95%CI 1.9–5.5). During non-pandemic years there was no demonstrated disparity in mortality between Indigenous versus non-Indigenous individuals. During the 2009 pandemic, the increase in deaths was predominantly in Indigenous individuals aged 25–74 years, with non-significant increases in deaths in Indigenous persons aged 6–23 months and ≥75 years difficult to interpret due to low numbers.

Population prevalence of medical conditions associated with increased risk of severe influenza

Data from national surveys conducted between 2011 and 2013 showed that approximately 12.2%

Figure 11: Influenza death rate with Indigenous/ Non-Indigenous rate ratios, Australia, 2006 to 2013, by year and Indigenous status

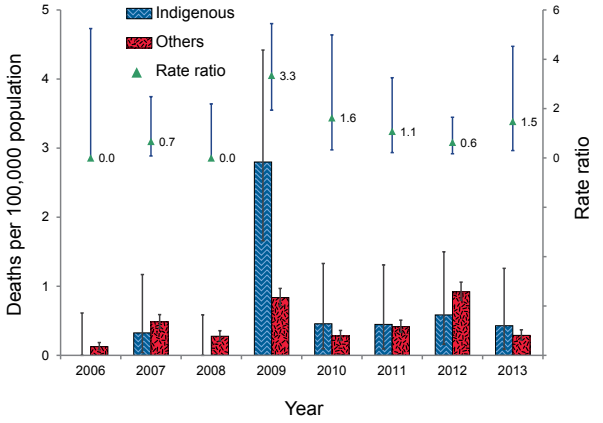
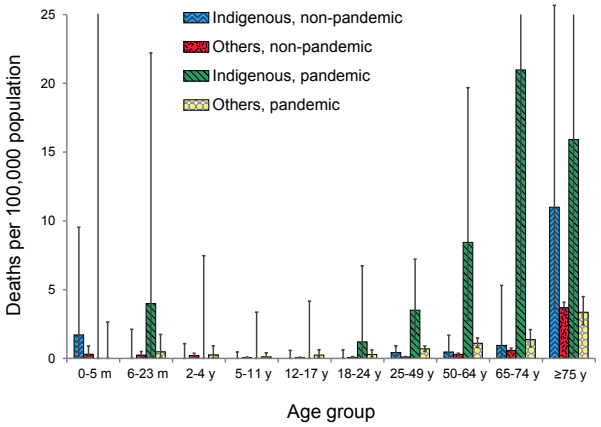


Figure 12: Influenza death rates, Australia, 2006 to 2013, by pandemic versus non-pandemic years and Indigenous status



(95%CI 11.6–12.8%) of the Australian population had at least 1 medical condition that is associated with increased risk of severe influenza (Table 4). The prevalence of these conditions was generally low among children and adolescents (<18 years: 2.7% (95%CI 2.0–3.4%)), and increased with age (e.g. ≥65 years: 38.2% (95% CI 35.9–40.5%)). Among non-Indigenous older adults aged ≥65 years, more than a third reported at least 1 condition. A large proportion of Indigenous adults, even younger adults, reported having at least 1 condition. Importantly, while direct comparisons cannot be made, the prevalence estimates among Indigenous individuals of all age groups are considerably higher than those of the non-Indigenous population (Table 4).

Discussion

Across non-pandemic seasonal influenza years, there was an increasing rate of influenza notifications in Australia during the period 2006–2015, particularly in the post-pandemic period. Comparing data from similar pre– and post-pandemic years, average notification rates were 4.0 times higher than during the pre-pandemic period (2006–2008) than post-pandemic period (2011–2013), while average hospitalisation rates showed a less marked upward trend (2.1 times higher in 2011–2013 than 2006–2008). Notification rates in 2014 and 2015 show even more marked increases (9.7 times) compared with the pre-pandemic period, but comparative data on hospitalisations was not available for these years. Peaks in hospitalisation during 2009 and 2012 matched peak notification rate years, consistent with these being more clinically severe influenza seasons. Death rates peaked in 2007 and 2012 among the non-pandemic years, as well as during the 2009 pandemic.

The 2012 season was associated with the highest hospitalisation and death rates during the study period. That year had predominantly A/H3N2 circulation with a strain considered antigenically drifted from the strain in the 2012 seasonal vaccine but for which cross-protection was expected.[11] There was little A/H1N1pdm09 circulation but substantial co-circulation of both lineages of influenza B, only one of which was included in the trivalent influenza vaccine. Adults aged ≥65 years and children 0–4 years of age contributed most to increases in hospitalisation in 2012. Increased infection rates, with associated morbidity and mortality, are often seen in the elderly when A/H3N2 circulates prominently during a season,[12–14] and this, as well as the aforementioned strain factors may have contributed to a more severe season.

The trend of marked increases in notifications and to a lesser extent hospitalisations, require careful interpretation. As Australia has compulsory laboratory notification, and notification data are influenced heavily by influenza testing, interpretation of trends in true disease burden is difficult if increased testing occurs. Differences in local health seeking behaviour with influenza may also affect rates of testing. Nucleic acid testing using reverse-transcription polymerase chain reaction (PCR) offers a rapid and sensitive method of confirmation of influenza and has become more readily used for confirmatory influenza testing in recent years, in part due to its funding as a Medicare Benefits Schedule testing item since approximately 2005.[15,16] PCR-based notifications to the NNDSS have increased from 29% (2001–2006) to 81% (2007–2013) and increased testing is likely to account for a substantial amount of the increase in notifications.[16]

Increased laboratory testing also influences hospital coding as suggested by the increasing proportion of influenza hospitalisations coded as virologically confirmed (J09 or J10) in individuals ≥5 years of age, especially adults and the elderly, over the study

Table 4: Proportion of Indigenous, Other and all Australians with at least 1 self-reported medical condition associated with an increased risk of severe influenza, by age group

Age group (years)	Age group-specific population prevalence (as proportion of the population)					
	Indigenous*		Others†		Total population†	
	Prevalence	95% C I	Prevalence	95% CI	Prevalence	95% CI
Paediatric						
All 0–17‡	5.7	4.4–7.0	2.6	1.9–3.3	2.7	2.0–3.4
0–4	5.0	3.3–6.7	2.5	1.4–3.6	2.5	1.4–3.6
5–14	6.0	4.1–7.9	2.8	1.6–4.0	2.9	1.8–4.0
Adult						
All 18–64	23.5	21.9–25.1	10.0	9.3–10.7	10.2	9.5–10.9
18–34	11.5	9.6–13.4	4.1	3.4–4.8	4.2	3.5–4.9
35–49	27.1	24.3–29.9	8.5	7.4–9.6	8.8	7.8–9.9
50–64	50.4	45.9–54.9	19.5	17.6–21.4	19.8	18.08–21.6
Elderly						
All ≥65	65.0	58.8–71.2	38.1	35.8–40.4	38.2	35.9–40.5
65–74§	–	–	35.0	32.0–38.0	35.2	32.2–38.2
≥75‡	–	–	41.8	38.2–45.4	42.0	38.4–45.6
All ages (Crude)	17.3	16.2–18.4	12.1	11.5–12.7	12.2	11.6–12.8
All ages (AS)	26.2	24.7–27.7	11.2	10.6–11.8	11.4	10.8–12.0

AS Age-standardised

* Data source for Indigenous population: Australian Bureau of Statistics, 2012–13 National Aboriginal and Torres Strait Islander Health Survey 2012–13.

† Data source for Others and Total population: Australian Bureau of Statistics, National Health Survey 2011–12.

‡ Estimates for the age group 15–17 years have not been provided as they were unreliable (relative standard error exceeded 25%).

§ Estimates for these age groups were not available for Indigenous Australians due to concerns about maintaining confidentiality.

period. Proportions in children aged <5 years have remained high and largely unchanged, probably because in this age group, collection of respiratory samples such as nasopharyngeal aspirates or throat swabs for multiplex respiratory virus PCR testing has been standard clinical practice for some time. The isolated decline in the proportion of virologically confirmed hospitalisations seen during the 2009 pandemic year was likely due to a larger number of clinically diagnosed cases in the latter stages of the pandemic.

The 2009 season, which was dominated by the novel A/H1N1pdm09 strain, was associated with a large peak in notifications, hospitalisations and deaths. However, all-age hospitalisation and death rates were lower than the severe 2012 seasonal influenza year. Age-specific rates of notifications (5–49 years), hospitalisations (5–64 years) and deaths (25–64 years) were significantly higher during the 2009 A/H1N1 pandemic, compared with pre-pandemic and post-pandemic periods. This matches international and Australian findings soon after the onset of the pandemic, which suggested that younger adults, rather than the elderly, had a higher risk of influenza related complications and death with A/H1N1pdm09 than seasonal influenza.[17–19] The elderly, particularly those born prior to 1950, appeared to have reduced susceptibility to infection by the A/H1N1 pandemic strain, possibly due to cross-protective antibodies from exposure to previous similar influenza strains.[20,21]

The burden of hospitalised influenza remains highest in young children aged 0–4 years and in the elderly aged ≥65 years. Correspondingly, children aged <5 years have become a focus for vaccination by the World Health Organization.[22] In particular, the youngest children aged 0–5 months have the highest hospitalisation rate, which matches findings from retrospective[23] and prospective[24] reviews of Australian paediatric influenza hospitalisation through the Influenza Complications Alert Network sentinel network[25] and United States of America data.[26] This supports focussing on maternal vaccination during pregnancy as the only means to provide protection to this age group, as influenza vaccine is not recommended under 6 months of age. Influenza-related deaths were predominantly in the elderly aged ≥75 years, in

whom a large proportion were noted to report having at least 1 medical condition known to increase the risk of severe influenza. A low number of ICD-coded deaths was observed in other age groups. In particular, the 2012 A/H3N2-predominant season had a disproportionately large number of deaths compared with all other seasons including the 2009 pandemic, and was consistent with the increased burden of disease of H3N2 in the elderly.[14]

Indigenous persons are at significantly increased risk from influenza infection, with a substantial proportion of the Indigenous population reporting a current medical condition that increases their risk of severe influenza. Notifications and hospitalisations show similar patterns, with significantly increased rates in all ages compared with non-Indigenous individuals, particularly in young children 0–23 months and adults ≥18 years of age, who have between 2 and 9 times higher notification rates and 2 to 4 times higher hospitalisation rates than the rest of the population. A previous analysis of Indigenous hospitalisations due to influenza during 1999 to 2009 found rates 4.6 times higher than for the rest of the population.[27] In contrast, deaths data during 2006 to 2013 showed that increased Indigenous death rates were found primarily during the 2009 pandemic, in adults aged 25–74 years. During non-pandemic years, the Indigenous influenza-related death rate was not significantly different. Numerous studies have shown that Indigenous populations in Australia and internationally were at significantly increased risk of morbidity and mortality during the 2009 pandemic,[19,28,29] although in Australia, this may be related to elevated rates of background chronic disease rather than Indigenous status per se.[30] Currently all Indigenous persons aged 6 months–<5 years and ≥15 years are eligible for influenza vaccines funded under the NIP.[4] The high proportion of Indigenous persons with at least 1 medical condition that increases risk of severe influenza infection justifies their inclusion in the NIP-eligible population. Our hospitalisation data suggest that although Indigenous children 5–14 years of age have lower rates of hospitalisation for influenza, these rates are significantly higher than in non-Indigenous children. Implementation of the influenza vaccination program would be simplified if influenza vaccine was funded for Indigenous people of all ages, meriting review of current provisions under the NIP.

The estimates of population prevalence of medical conditions associated with an increased risk of severe influenza would inform vaccination program planning, allowing for estimation of the vaccine doses needed to vaccinate the at-risk population who are eligible under the NIP. Despite NIP funding of influenza in those aged ≥65 years and at-risk groups, it is evident that there remains a considerable ongoing burden of influenza. Modelling in the United Kingdom demonstrated cost-effectiveness of universal influenza vaccination for children, based on their importance in the transmission of influenza within the community, leading to reduction in the population-wide influenza burden.[31] This led to a childhood influenza vaccination program using live attenuated influenza vaccine aimed at eventually including all healthy children aged 2–16 years of age.[32,33] Currently, vaccination has been rolled out for children aged 2–4 years and in the first 2 years of school, with initial evaluation showing promising direct as well as indirect herd immunity benefits.[5] Similar modelling using Australian data would inform whether a similar approach, in comparison with strengthening of direct immunisation of at-risk age groups, would be beneficial to influenza control in the Australian setting.

Limitations of our study include under-ascertainment, which is inevitable if administrative data (notifications and ICD-coded hospitalisations) are used. Notification data do not include the large number of possible influenza infections that are not tested. Hospitalisation and death data are reliant on diagnosis by clinicians and accurate coding. An Australian study has estimated that true hospitalisation rates may be up to 11 times higher than that calculated from hospital discharge coding in children.[34] It is likely that notification data underestimate the true incidence of influenza by an even larger factor. Similarly, with regard to under-ascertainment of influenza-related deaths, a New South Wales study found that, of persons with virologically confirmed influenza, only 25% of those who died, and 49% of those hospitalised had influenza coded as a cause of illness.[35] Under-ascertainment is likely to vary according to age group, with a higher level in the elderly in whom influenza testing may be less frequent. Community measures of influenza burden such as influenza-like illness presentations to general practitioners and emergency departments may provide a measure of burden of influenza not captured through testing but were outside the scope of this report.[36] Incompleteness of Indigenous status recording in hospitalisation data is also likely to lead to underestimation of the disparity of influenza disease by Indigenous status. The exact effect of increased influenza testing practices is difficult to estimate. However, significant increases in notification data with only modest increases in hospitalisation rates suggest there is considerable contribution from increased testing, and assessments of trends of overall burden of influenza need to be made with caution. Estimates of the population prevalence of medical conditions associated with increased risk of severe influenza may be underestimates as they are based on self-report, with the respondents asked to nominate only conditions that were current and

long-term (lasting 6 months or more), except for a few National Health Priority Area conditions for which more details were sought. Also, our method only captured the major and the more common conditions that were captured in the relevant surveys.

In summary, this review identifies age groups most affected by an ongoing burden of influenza in Australia, with implications for expansion of NIP funding for influenza vaccines. Whilst children with specified medical risk factors receive free vaccine, healthy children, particularly those aged <5 years, also experience a considerable burden of influenza hospitalisation and although recommended for influenza vaccination in *The Australian Immunisation Handbook*,[37] are currently not eligible for NIP funding unless they also are of Indigenous background. Universally funded influenza vaccine programs similar to those implemented in the United States of America, United Kingdom and parts of Canada[33,38,39] warrant investigation for their suitability in an Australian setting. With increasing evidence of equivalent influenza vaccine effectiveness in children and adults,[24,40,41] ongoing disease surveillance data incorporated into Australian-specific disease modelling, will be critical to evaluating the cost-effectiveness of strategies to improve influenza control in Australia.

Appendix: Medical conditions included in the analysis of the proportion of the population reporting a medical condition associated with an increased risk of severe influenza

These tables list the medical conditions associated with an increased risk of severe influenza that were included in deriving estimates of the cumulative population prevalence of at-risk medical conditions. Respondents that self-reported at least 1 medical condition within the condition groups (A), (B) or (C) below were included. Condition and condition status codes used by the Australian Bureau of Statistics (ABS) for capturing and classifying the conditions selected for inclusion are also given.*

Definitions for the condition status codes used by the ABS are:

1. ever told has condition, still current and long-term
2. ever told has condition, still current but not long-term
3. ever told has condition, not current
4. not known if ever told or not ever told, but condition current and long-term.

Condition group (A)

Respondents were asked about National Health Priority Area (NHPA) conditions, specifically regarding whether the specific condition was medically diagnosed ('ever told') and the duration of the condition.

* Australian Bureau of Statistics, 4363.0.55.001 – Australian Health Survey: Users' Guide, 2011-13, accessed 4 August 2016, http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/4363.0.55.001Chapter3002011-13

Condition group (A)

Condition category	Specific conditions included	ABS condition code	ABS condition status code
Cancer	All malignant neoplasms including those of the digestive organs, respiratory and intrathoracic organs, mesothelial and soft tissue, breast, female genital organs, male genital organs, leukaemia, lymphoma, 'other' and 'site unknown'. Excluded skin cancers with the exception of melanoma	All condition codes listed for malignant neoplasms* except 42, 43, 627, 920	1, 2, 4
Diabetes/high sugar levels	Diabetes/high sugar levels	688, 947, 689, 948, 90, 91	1, 2, 4
Heart and circulatory systems	Rheumatic heart disease	377, 935	1, 2, 3, 4
	Angina	117, 938, 382	1, 2, 4
	Heart attack	383, 936	1, 2, 3, 4
	Heart failure	385	1, 2, 4
	Fluid problems/fluid retention/oedema	135, 941	1, 2, 3, 4

* List of health condition codes available at: Australia Health Survey: Users' Guide, 2011-13, Appendix 3: Classification of health conditions http://www.abs.gov.au/AUSSTATS/abs@.nsf/Latestproducts/4363.0.55.001Appendix202011-13?opendocument&tabname=Notes&prodno=4363.0.55.001&issue=2011-13&num=&view=

Condition group (B)

In addition to specific questions on the NHPA condition, respondents were additionally asked to report having other conditions that were current and long-term (i.e. lasted or were expected to last for 6 months or more). Respondents were not specifically asked if they were medically diagnosed or the duration of the condition. For these conditions, the comparable ABS condition status code would be similar to condition codes 1 and 4.

Condition group (C) – 'Severe asthma that worsened or was out of control over the last 12 months'

Respondents were separately asked about having asthma. This condition group included all persons who reported having asthma that required them to visit a hospital or emergency department 2 or more times in the past 12 months.

Condition group (B)

Condition category	Specific conditions included	ABS condition code
Chronic liver disease	Viral hepatitis	271
	Liver disease not otherwise specified	133
Heart and circulatory systems	Heart valve disease not otherwise specified	136
	Congenital anomaly cardiovascular	381
	Infection of circulatory system	376
	Ischemic heart disease without angina	384
Immunocompromising disorders	Hereditary haemolytic anaemia	76
	HIV-infection/AIDS	83
Neurological conditions that compromise respiratory functions	Convulsions/seizures	465
	Paralysis/weakness	468
	Multiple Sclerosis	503
	Parkinsonism	504
	Epilepsy	505, 982
	Limited function or disability due to a neurological condition	490
Renal disease	Kidney disease and dialysis	702
	Pyelonephritis/pyelitis	203
	Glomerulonephritis/nephrosis	204
	Urinary disability/limited function	705
Respiratory disease	Pulmonary heart disease	390
	Chronic bronchitis	583, 979
	Chronic obstructive pulmonary disease	596, 981
	Respiratory disability/limited function	575
	Other respiratory infection	587
	Other respiratory disease	599

Acknowledgements

We thank Christina Bareja and Kate Pennington, Australian Government Department of Health, for their review of the manuscript.

We thank the Australian Coordinating Registry, state and territory registries of births, deaths and marriages, state and territory coroners, and the National Coronial Information System, for providing access to cause of death data.

We thank the National Centre for Aboriginal and Torres Strait Islander Statistics of the Australian Bureau of Statistics for providing data from the National Health Survey 2011–12 and the National Aboriginal & Torres Strait Islander Health Survey 2012–13 for estimating the population prevalence of certain medical conditions associated with increased risk of influenza.

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases is supported by the Australian Government Department of Health, the NSW Ministry of Health and The Children's Hospital at Westmead. The opinions expressed in this paper are those of the authors, and do not necessarily represent the views of these agencies

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Short report

Haycocknema perplexum: an emerging cause of parasitic myositis in Australia

Luke J Vos, Thomas Robertson, Enzo Binotto

Abstract

Haycocknema perplexum is a rare cause of parasitic myositis, with all cases of human infection reported from Australia. This case involved an 80-year-old Queensland wildlife carer, who presented with muscle weakness, mild eosinophilia and creatine kinase elevation. This case supports an association with native animal contact and highlights the debilitating nature of this infection. *Commun Dis Intell* 2016;40(4):E496–E499.

Keywords: *Haycocknema perplexum*, parasitic myositis, nematode, native animals, Australia

In 1998, Dennett et al. described a new nematode that causes myositis, *Haycocknema perplexum*.[1] Male and female *H. perplexum* live inside individual muscle fibres and are 25 to 45 um in diameter and 300–500 um long.[1] The published literature contains 8 reports of *H. perplexum*, 4 from Tasmania and 4 from northern Queensland.[1-4] This report describes the 9th known case, an 80–year-old female from northern Queensland (Table).

Case report

In October 2012, an 80-year-old wildlife carer was referred to the Cairns Hospital Infectious Diseases clinic with possible chronic Q fever and concerns regarding Lyme disease. She was prescribed rosuvastatin 10 mg at the time of review. She reported a progressive illness involving weakness of her arms and legs over the past 18 months, with functional limitation, such that she was unable to dress or shower independently. In addition, she reported a weight loss of approximately 5 kilograms over the previous 12 months. She reported no dysphagia, dysphonia, shortness of breath or myalgia.

The patient reported extensive native wildlife exposure, such as handfeeding puggles (juvenile echidnas) and caring for various marsupials including koalas, wombats, possums, wallabies and kangaroos. She owned a pet galah. She had sustained multiple tick bites in the past, but reported no direct cattle exposure. Her travel history was extensive, with past travel to South American, African and Asian countries. She reported previous, limited travel to Tasmania.

On examination, she appeared thin, with a weight of 59 kg. Diffuse, non-tender muscle wasting was evident in her upper and lower limbs. Cranial nerve examination was normal. Neurological examination of her limbs revealed symmetrically reduced power of 3 on 5, more marked proximally than distally. Knee jerks were present, with all other limb tendon reflexes absent.

Initial pathology investigations showed an eosinophilia (0.7 x 10^9/L; reference range (RR), < 0.4 x 10^9/L) and elevated serum creatine kinase (CK) level (270; RR, < 160). Levels of inflammatory markers were normal (erythrocyte sedimentation rate, 15 mm/h; RR 0–20; C-reactive protein, < 2; RR, < 5 mg/L). Q fever serology results were consistent with past exposure to Q fever.

Electromyography showed florid myopathic changes, with frequent spontaneous activity and occasional myotonic discharge from the proximal limb muscles. A muscle biopsy from the right anterolateral thigh showed an active inflammatory myopathy, with infiltration of the muscle by mononuclear cells and eosinophilic granulocytes and evidence of muscle injury. Three minute intracellular nematodes were identified in the limited biopsy provided (approximately 15 square mm in cross-sectional area in the cryostat sections). The nematodes were 16 microns in diameter in the plane of section and were characterised by a thin eosinophilic cuticle with surrounding sarcoplasmic retraction halo (Figures 1 and 2). Unfortunately, the tissue retained for electron microscopic examination did not contain any parasites. Although there were only limited nematodes for assessment, given the minute size, intracellular location, and association with eosinophilic myositis, a histopathologic diagnosis of *H. perplexum* parasitic myositis was made.

Initial management of this patient included cessation of rosuvastatin, with no steroids administered at any stage of her illness. She was treated with albendazole 400 mg twice daily for 12 weeks. She tolerated this treatment well, with normalisation of her eosinophil count and CK. Unfortunately, she experienced no functional improvement. Her limb muscle power continued to slowly decline, with increased assistance required for activities of daily living.

Table: Clinical and laboratory features of patients with myositis due to *Haycocknema perplexum*[2,3,4]

Patient, age, sex	Place and year of diagnosis	Prior travel	Animal exposure	Duration of symptoms (years)	Dysphagia	Weakness	Weight loss (kg)	Peak CK level (U/L)*	Corticosteroids	Eosinophil count (x 10^9/L)†	Outcome
1. 33, F	Tasmania, 1994	Extensive, including northern Australia	Botanist, native animals and specimens	5	NA	‡	NA	3,294	Prednisolone prior to diagnosis – worsened	0.8	Good recovery, normal CK
2. 48, M	Tasmania, 1996	Extensive, including North Qld	Limited, field trip to Kakadu	1.5	§	§	7	1,586	Prednisolone prior to diagnosis –worsened	2.0	Good recovery
3. 61, M	North Qld, 2004	None in 20 years, born in Tasmania	Nil noted	3	§	§	NA	1,263	Yes	High	Died
4. 23, F	North Qld, 2005	Extensive travel in Australia	Nil noted	2	§	§	18	1,370	None	1.1	Weak, CK elevated at 6 years
5. 61, F	North Qld, 2006	Nil	Nil noted	2	‡	‡	NA	1,230	None	1.36	Weak, elevated CK at 6 years
6. 50, M	Tasmania, 2011	Extensive, no travel to North Qld	Native animals as pets, eaten bush meat	2	§	§	10	5,700	Prednisolone prior to diagnosis – marked deterioration	Normal	Good recovery, CK 470 U/L
7. 80, F	North Qld, 2012	Extensive, including Tasmania	Native animal carer	1.5	Nil	§	5	270	None	0.7	Weak, normal CK
8. 30, M	North Qld, 2014	Travel limited to North Qld	Nil	2	§	§	20	3,400	Prednisolone prior to diagnosis – new symptoms	1.24	Good recovery
9. 72, M	Tasmania, 2014	WA and Qld	Recreational hunter, eaten bush meat	Years	NA	‡	NA	2,082	Methyl-prednisolone prior to diagnosis	2.4	Good recovery

* Reference range (RR), <160 U/L

† RR, <0.4x 10^9/L

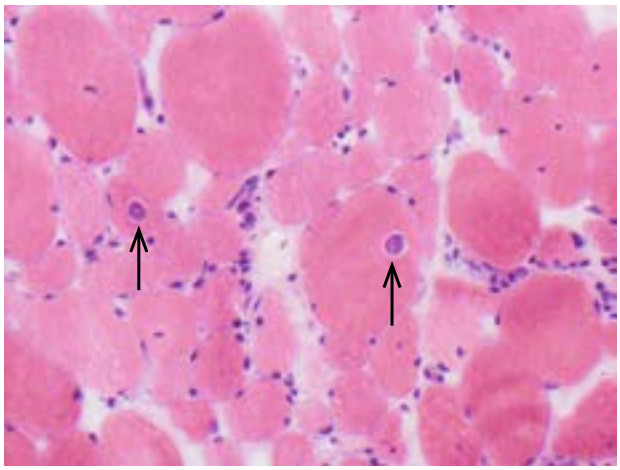
‡ Mild-moderate

§ Moderate-severe

CK Creatine kinase

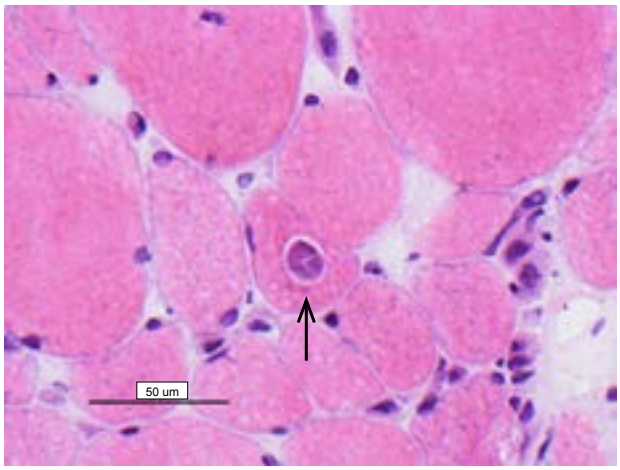
NA Not available

Figure 1: Myositis with two nematodes within myofibres



Cryostat section, haematoxylin-eosin stain, x 200 magnification

Figure 2: Transverse section of a nematode demonstrating a thin eosinophilic cuticle and sarcoplasmic retraction halo



Cryostat section, haematoxylin-eosin stain, x 400 magnification

Discussion

This report of *H. perplexum* infection in an 80-year-old female wildlife carer from northern Queensland represents the 9th known human case. This patient developed severe, functionally limiting limb weakness and a moderate weight loss of 5 kilograms. Laboratory investigations demonstrated a peripheral eosinophilia and mildly elevated CK. Electromyography showed florid myopathic changes, with the diagnosis confirmed on muscle biopsy. Unfortunately she did not improve with albendazole treatment, with continued decline in muscle power.

This case of *H. perplexum* shares several similarities with the previously reported accounts. This patient lived in the Tablelands Region of Far North Queensland, with a history of limited prior travel to Tasmania. Northern Queensland and Tasmania remain the only known locations of *H. perplexum* human infection.[1-4] She described sustained, close contact with native animals. Three of the previous 8 cases detail extensive animal contact.

Treatment recommendations for *H. perplexum* based on limited case reports include Albendazole 400 mg twice daily for 8 to 9 weeks, with a strong caution against steroid use.[5] The patient in this case received 12 weeks of treatment, in view of good medication tolerance and uncertainty regarding optimal treatment duration. Given the limited information on the life cycle and transmission of *H. perplexum*, no infection prevention or control recommendations exist at present.

While *H. perplexum* is a suspected human zoonosis, the natural host remains unknown. *H. perplexum* had previously been assigned to the superfamily Muspiceoidea.[6] Muspiceoids have been isolated from Australian animals that include mice, bats, kangaroos, tree kangaroos, wallabies, pademelons, koalas and biting midge.[6] However, new molecular findings have cast doubt on the earlier classification of *H. perplexum*, with further molecular evidence needed to elucidate phylogenetic relationships for other muspiceoid nematodes.[4] A *Haycocknema*-like nematode has been identified in muscle fibres from a horse imported to Switzerland from Ireland.[7]

The difficulties and delays experienced in the diagnosis of *H. perplexum* raise the possibility of undiagnosed cases in the Australian community. The disease spectrum may include subclinical infection, in addition to severe cases that receive clinical attention. The report of an infection similar to *H. perplexum* in an Irish horse implies a greater potential distribution of human infection outside of Australia. Several of the reported cases suggest the importance of prolonged, close animal contact for acquisition of the parasite. At risk groups may include wildlife carers and veterinarians.

Potential areas for future research into *H. perplexum* include investigation into the natural host, mechanism of transmission and the life cycle. Recent definition of nuclear and mitochondrial gene markers for *H. perplexum* provide a molecular diagnostic tool for parasitic myositis in humans.[4] In addition, polymerase chain reaction-based sequencing provides a useful research tool to survey potential reservoir animals or vectors, including horses, for *H. perplexum* and related genotypes.[4,7]

This, the 9th reported case of human *H. perplexum* infection, adds to the limited clinical information on the presentation and course of this disease. It

supports the potential importance of close wildlife contact as a risk factor in those who develop clinical infection. The case highlights the serious nature of *H. perplexum* infection and the need for further biological and clinical research into this condition.

Acknowledgements

We wish to acknowledge the valuable assistance of Dr Andrew McNabb, Neurologist, Cairns Hospital.

There are no conflicts of interest to declare.

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Policy and guidelines

REVISED SURVEILLANCE CASE DEFINITIONS

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

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

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

The listeriosis case definition will be implemented on 1 January 2017 and supersedes any previous versions.

Listeriosis

Reporting

Only confirmed cases should be notified. Where a mother and fetus (≥20 weeks gestation)/neonate are both confirmed, both cases should be notified.

Confirmed case

A confirmed case requires either:

1. laboratory definitive evidence.

OR

2. Clinical AND epidemiological evidence.

Laboratory definitive evidence

Isolation or detection of *Listeria monocytogenes* from a site that is normally sterile, including fetal gastrointestinal contents.

Clinical evidence

1. A fetus/neonate where the gestational outcome is one of the following:
   a. Stillbirth
   b. Premature birth (<37 weeks gestation)
   c. Diagnosis (within the first month of life) with at least one of the following:
      – Granulomatosis infantiseptica
      – Meningitis or meningoencephalitis
      – Septicaemia
      – Congenital pneumonia
      – Lesions on skin, mucosal membranes or conjunctivae
      – Respiratory distress and fever at birth

   AND

   In the absence of another plausible diagnosis

OR

2. A mother has experienced at least one of the following conditions during pregnancy:
   a. Fever of unknown origin
   b. Influenza like illness
   c. Meningitis or meningoencephalitis
   d. Septicaemia
   e. Localised infections such as arthritis, endocarditis and abscesses
   f. preterm labour/abruption

   AND

   In the absence of another plausible diagnosis

Epidemiological evidence

A maternal/fetal pair where one of either the mother or fetus/neonate is a confirmed case by **laboratory definitive evidence** (up to 2 weeks postpartum).

Notes

1. The clinical AND epidemiological evidence criteria for a confirmed case means that if the mother is a confirmed case by laboratory definitive evidence, then the fetus/neonate is also a confirmed case if they have the defined (fetus/neonate) clinical evidence, and vice versa.
2. Laboratory definitive evidence in a fetus < 20 weeks gestation means the mother only is a confirmed case.

Summary of changes to listeriosis surveillance case definition	Addition of clinical evidence and epidemiological evidence as alternative criteria for a confirmed case. Definition of fetus as ≥20 weeks gestation.

Dengue virus infection

Reporting

Both confirmed cases and probable cases should be notified.

Confirmed case

A confirmed case requires:

Laboratory definitive evidence AND clinical evidence

Laboratory definitive evidence*

Isolation of dengue virus

OR

Detection of dengue virus by nucleic acid testing

OR

Detection of non-structural protein 1 (NS1) antigen in blood by EIA

OR

IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre to dengue virus, proven by neutralisation or another specific test

OR

Detection of dengue virus-specific IgM in cerebrospinal fluid, in the absence of IgM to Murray Valley encephalitis, West Nile virus /Kunjin or Japanese encephalitis viruses

Clinical evidence

A clinically compatible illness (e.g. fever, headache, arthralgia, myalgia, rash, nausea/vomiting)

Probable case

A probable case requires:

Laboratory suggestive evidence AND clinical evidence AND epidemiological evidence

OR

Clinical evidence AND household epidemiological evidence

Laboratory suggestive evidence

Detection of NS1 antigen in blood by a rapid antigen test†

OR

Detection of dengue virus-specific IgM in blood

Clinical evidence

As for confirmed case

Epidemiological evidence

Exposure, between 3 and 14 days prior to onset, in

EITHER

a country with known dengue activity

OR

a dengue-receptive area‡ in Australia WHERE a locally-acquired or imported case has been documented with onset within a month

* Confirmation of the laboratory result by an arbovirus reference laboratory is required if the infection was acquired in Australia but outside a dengue-receptive area as defined in the Dengue National Guideline for Public Health Units.

† Unless dengue NS1 antigen by EIA is negative

‡ As defined in the Dengue CDNA National Guideline for Public Health Units.

Household epidemiological evidence

Living in the same house[§] as a locally-acquired case in a dengue-receptive area[3] of Australia within a month of the onset in the case.

AND

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

§ The case must have spent all the exposure period (from 14 days prior to onset to 3 days prior to onset) living in the same house as the epi-linked confirmed case.

Summary of changes to dengue surveillance case definition	Under laboratory definitive evidence, addition of "by EIA" after the NS1 antigen clause. Clinical evidence simplified. Addition of clinical evidence and household epidemiological evidence as alternative criteria for a probable case. Addition of "NS1 antigen in blood by a rapid antigen test" as laboratory suggestive evidence. Epidemiological evidence updated to specify exposure time and change the description of Australian exposure.

Annual reports

Australian Meningococcal Surveillance Programme annual report, 2015

Monica M Lahra, Rodney P Enriquez for the National Neisseria Network

Abstract

In 2015, there were 174 laboratory-confirmed cases of invasive meningococcal disease analysed by the Australian National Neisseria Network. This number was higher than that reported in 2013 and 2014, which were the lowest and second-lowest totals reported, respectively, since inception of the Australian Meningococcal Surveillance Programme in 1994. Probable and laboratory confirmed invasive meningococcal disease (IMD) is notifiable in Australia. There were 182 IMD cases notified to the National Notifiable Diseases Surveillance System in 2015, again, higher than in 2013 and 2014, which were the lowest and second-lowest totals of IMD cases recorded, respectively, by this system. Meningococcal serogrouping was able to be determined for 168/174 (97%) laboratory confirmed IMD cases. Of these, 64.2% (108 cases) were serogroup B infections, the lowest reported since 2003. Further, the number and proportion of cases of serogroup C IMD, (1.2%, 2 cases), was the lowest yet reported. By contrast, in 2015 in Australia, there was a marked increase in the number and proportion of serogroup W IMD (21.4%, 36 cases), and an increase in serogroup Y IMD (13.1%, 22 cases). The number and proportion of IMD cases caused by serogroups W and Y was the highest reported since the inception of the Australian Meningococcal Surveillance Programme in 1994. Molecular typing results were available for 140 of the 174 IMD cases. Of the 31 serogroup W IMD strains that were able to be genotyped, 25/31 (81%) were sequence type (ST)-11, and have the *porA* antigen encoding gene type P1.5,2, the same genotype as the hypervirulent serogroup W strain of that has been circulating in the United Kingdom and South America since 2009. In 2015, the most common serogroup B *porA* genotype circulating in Australia was P1.7-2,4. The primary IMD age peak was observed in adults aged 45 years or more, which was the first time that this was noted by the AMSP, whilst secondary disease peaks were observed in those aged 4 years or less, and in adolescents (15–19 years). Serogroup B cases predominated in all jurisdictions and age groups, except for those aged 45 years or over where serogroups W and Y predominated. All IMD isolates tested were susceptible to ceftriaxone and ciprofloxacin. One isolate was resistant to rifampicin. Four isolates were resistant to penicillin. Decreased susceptibility to penicillin was observed in 86% of isolates. *Commun Dis Intell* 2014;40(4):E503–E511.

Keywords: antibiotic resistance; disease surveillance; meningococcal disease; *Neisseria meningitidis*

Introduction

The National Neisseria Network (NNN) in Australia is an established, collaborative network of reference laboratories in each state and territory that contribute to the laboratory surveillance system of the pathogenic *Neisseria* species *(N. meningitidis* and *N. gonorrhoeae)*. Since 1994 the NNN has coordinated laboratory data from the examination of *N. meningitidis* cases of invasive meningococcal disease (IMD) for the Australian Meningococcal Surveillance Programme (AMSP).[1] The AMSP is funded by the Australian Government Department of Health. The NNN laboratories supply phenotypic and genotypic data of invasive meningococci for the AMSP. These data supplement the notification data from the National Notifiable Diseases Surveillance System (NNDSS), which includes cases of probable IMD as well as laboratory confirmed IMD. The characteristics of meningococci responsible for IMD, and the associated demographic information, are important considerations for management of individual patients and their contacts; and to inform public health responses for outbreaks or case clusters, locally and nationally. The introduction of the publicly funded conjugate serogroup C meningococcal vaccine onto the National Immunisation Program in 2003 has seen a significant and sustained reduction in the number of cases of serogroup C IMD after 2003.[2] However, IMD remains an issue of public health concern in Australia. In this report please note a nomenclature change for serogroup W135, which will be hereafter referred to as serogroup W in line with the accepted international nomenclature system for *N. meningitidis*.[3]

Methods

Case confirmation of invasive meningococcal disease

Case confirmation is based on isolation of *N. meningitidis*, or a positive nucleic acid amplification testing (NAAT) from a normally sterile site, defined as laboratory definitive evidence of IMD

by the Communicable Diseases Network Australia criteria.[4] Information regarding the site of infection, age and sex of the patients is collated by the NNN for the AMSP.

IMD cases are categorised on the basis of the site from which *N. meningitidis* was isolated, or from which meningococcal DNA was detected (blood, joint fluid, vitreous fluid). When *N. meningitidis* is detected from both blood and cerebrospinal fluid (CSF) from the same patient, the case is classified as one of meningitis.

Phenotyping and genotyping of *Neisseria meningitidis*

Phenotyping is limited to the determination of the serogroup by detection of soluble polysaccharide antigens. Genotyping of both isolates and DNA extracts is performed by sequencing of products derived from amplification of the porin genes *porA, porB* and *FetA*.

Antibiotic susceptibility testing

Isolates were tested to determine their minimum inhibitory concentration (MIC) values to antibiotics used for therapeutic and prophylactic purposes: ceftriaxone, ciprofloxacin; and rifampicin. This program uses the following parameters to define the various levels of penicillin susceptibility or resistance when determined by a standardised agar plate dilution technique: sensitive (MIC $\leq$ 0.03 mg/L); less sensitive (MIC 0.06–0.5 mg/L) and resistant (MIC $\geq$ 1 mg/L).

Results

In 2015, there were 174 laboratory-confirmed cases of IMD analysed by the NNN, and 182 cases notified to the NNDSS. Thus, laboratory data were available for 96% of notified cases of IMD in Australia in 2014 (Figure 1). This number was higher than that reported in 2013 and 2014, which were the lowest and second-lowest totals reported, respectively, by both the NNDSS, and the AMSP. With regard to the number of cases of IMD in Australia, there has been an overall decrease from the peak reported in 2002. As in previous years, the peak incidence for IMD was in late winter and early spring (1 July to 30 September) (Table 1).

The highest number of laboratory confirmed cases was from Victoria (54 cases), which was higher than 2014 (33 cases), and the highest number of cases reported in this state since 2008 (61 cases).

New South Wales also recorded a rise in IMD cases in 2015 compared with 2014 (41 cases in 2015, compared with 36 cases in 2014). In contrast, Queensland recorded a fall in the number of IMD cases in 2015 (30 cases) compared with 2014 (39 cases), and this was the lowest number of cases

Figure 1: Number of invasive meningococcal disease cases reported to the National Notifiable Diseases Surveillance System compared with laboratory confirmed data from the Australian Meningococcal Surveillance Programme, Australia, 2015

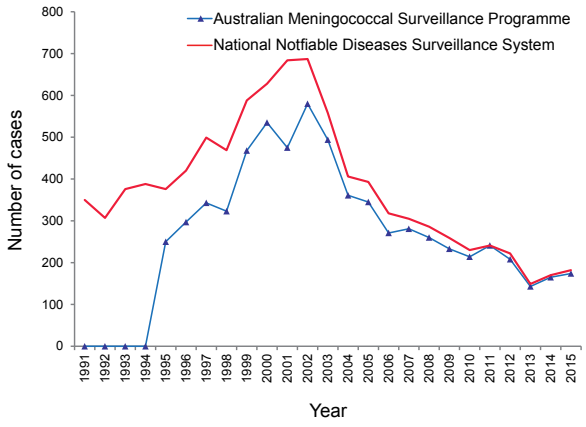


Table 1: Laboratory confirmed cases of invasive meningococcal disease, Australia, 2015, by quarter

Serogroup	1 January to 31 March	1 April to 30 June	1 July to 30 September	1 October to 31 December	2015
B	20	32	35	21	108
C	1	0	0	1	2
Y	1	5	12	4	22
W	2	6	16	12	36
NG	0	2	0	2	4
ND	0	0	0	2	2
Total	24	45	63	42	174

NG: Non-groupable.

ND: Not determined.

reported in this State since the inception of the AMSP in 1994. Numbers for the other states were similar to 2014 (Table 2).

Age distribution

Nationally, for the first time in AMSP reporting, the peak incidence of IMD occurred in adults aged 45 years or more. This age group represented 61/174 (35%) IMD cases in 2015 (Table 3). The number of IMD cases and the proportion of cases within this age group was almost double that in 2014 (21%, 34 cases). Previously, the primary peak incidence of IMD was in children less than 5 years of age; however in 2015 they represented 23% of IMD cases, the lowest percentage of cases noted by the AMSP in this age group in any year. Between 2003 and 2014, the proportion of IMD that occurred in those less than 5 years of age in Australia ranged from 28% to 36% of cases. A secondary disease peak has also been observed in previous years among adolescents aged 15–19 years. Of the total cases of IMD, 33 (19%) were in those aged 15–19 years in 2015, which was similar to the proportion reported in the period 2006 to 2011, and 2013 to 2014 (17%, 20% respectively).

Anatomical site of samples for laboratory confirmed cases

In 2015, diagnosis was made by a positive culture in 117/174 (67%) cases and 57/174 (33%) cases were confirmed by NAAT testing alone (Table 4).

There were 53 diagnoses of meningitis based on cultures or NAAT examination of CSF either alone or with a positive blood sample. There were

Table 2: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2015, by state or territory and serogroup

State or territory	Serogroup						Total
	B	C	Y	W	NG	ND	
ACT	1	0	1	0	0	0	2
NSW	20	2	7	9	3	0	41
NT	1	0	0	0	0	0	1
Qld	19	0	3	5	1	2	30
SA	29	0	1	0	0	0	30
Tas.	0	0	0	1	0	0	1
Vic.	29	0	8	17	0	0	54
WA	9	0	2	4	0	0	15
Australia	108	2	22	36	4	2	174
%	62.1	1.1	12.6	20.7	2.3	1.1	

NG: Non-groupable
ND: Not determined

Table 4: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2015, by anatomical source and method of confirmation

Specimen type	Isolate of MC	PCR positive	Total
Blood	92	23	115
CSF +/- blood	20	33	53
Other‡	5	1	6
Total	117	57	174

PCR Polymerase chain reaction.

Table 3: Laboratory confirmed cases of invasive meningococcal disease, Australia, 2015, by age and serogroup

Serogroup	Age group										Total
	<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
B	9	20	3	3	27	11	9	12	12	2	108
C	1	0	0	1	0	0	0	0	0	0	2
Y	0	1	1	0	1	2	1	4	12	0	22
W	2	2	1	0	4	4	2	10	11	0	36
NG	2	1	0	0	1	0	0	0	0	0	4
ND	2	0	0	0	0	0	0	0	0	0	2
Total	16	24	5	4	33	17	12	26	35	2	174
% B of within age group	56.3	83.3	60.0	75.0	81.8	64.7	75.0	46.2	34.3		

NS: Age not stated.
NG: Non-groupable.
ND: Not determined.

115 diagnoses of septicaemia based on cultures or NAAT examination from blood samples alone (Table 4). There were 3 IMD diagnoses by positive joint fluid culture, 1 IMD diagnosis by positive eye vitreous fluid culture, 1 IMD diagnosis by positive cyst fluid culture, and 1 IMD diagnosis by NAAT where the site was not stated.

Serogroup data

Number of cases of serogroup B, C, Y, W invasive meningococcal disease

The serogroup was determined for 168 of 174 laboratory confirmed cases of IMD (97%) in 2015 (Tables 2 and 3). The overall decrease since 2002 was initially predominantly due to a reduction in the number of cases of IMD caused by serogroup C from 2003 to 2007 following the introduction of the serogroup C vaccine. After 2009 a decline in the number of IMD cases caused by serogroup B was reported, from 194 cases in 2009, to 104 cases in 2013. In 2014, there was an increase in the number of IMD cases caused by serogroup B (n=129), but in 2015 the number of IMD cases caused by serogroup B was similar to 2013. The number of IMD cases caused by serogroup C (n=2) in 2015 was the lowest total reported by the AMSP. The number of cases of IMD caused by serogroup Y (n=22) in 2015 was the highest reported (13 cases in 2014). In contrast, the number of cases of serogroup W IMD has increased in recent years (7-16 cases in 2011-2014, compared with 4-9 cases in 2007-2010), and in 2015 there were 36 cases, the highest number reported by the AMSP, and more than double the number reported in 2014 (n=16).

Proportions of serogroup B, C, Y, W invasive meningococcal disease

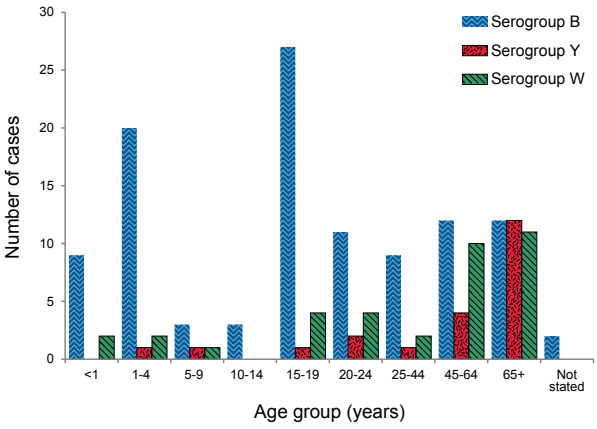
Of the 168 IMD strains for which the serogroup was determined in 2015, 64.3% were serogroup B, which is the lowest overall proportion since 2003. In the years 2006 to 2012 it was 84% to 88%, and in 2013 to 2014 it was 75% to 80%. The proportion and number of IMD caused by serogroup C in 2015 was lowest since the inception of the Australian Meningococcal Surveillance Programme (1.2%).

Whilst the number and proportion of IMD attributable to serogroup B and C declined in 2015, the number and proportion of cases of serogroup Y and W have increased. In 2012 to 2014 serogroup Y accounted for 7.7% to 10.8% of IMD, higher than the proportion reported in the period 2007 to 2011: 3.5% to 5.0%. In 2015 the proportion of IMD cases caused by serogroup Y (13.1%) was the highest yet reported and the predominance (16/22, (73%) was in those aged 45 years or over.

The proportion of cases of IMD caused by serogroup W in Australia ranged from 1.8% to 4.5% in the period 2007 to 2011, and was 8.6% to 9.9% in 2013 to 2014. In 2015, this rose to 21.4% of the total cases of IMD in Australia.

In 2015 the proportion of cases of IMD caused by serogroup B in children less than 5 years, was the lowest since 2000 (Table 3, Figure 2). In young adults 20–24 years, serogroup B IMD was lower than in 2014 (83%), and lower than 2007 to 2010 and 2012 (72% to 88%) but similar to 2011 and 2013 (61% to 67%). The proportion of cases of IMD caused by serogroup B in those aged 15–19 has remained relatively stable since 2008. Serogroup B IMD was prominent in IMD in all age groups excepting 65 years or over where, serogroup Y was equally prevalent, and serogroup W slightly less so. The age group with the highest prevalence of serogroup Y IMD was those aged 65 years or over, and accounted for 12/22 total serogroup Y IMD cases (55%) in 2015. Serogroup W cases were more evenly distributed over the age groups than serogroup Y; however the age group with the highest prevalence of serogroup W IMD was those aged 65 years or over (11/37 cases, 30%), followed by those aged between 45 and 64 years (8/37 cases, 22%).

Figure 2: Number of serogroups B, Y and W cases of confirmed invasive meningococcal disease, Australia, 2015, by age



Genotyping

In 2015, genotyping results were available for 140/174 (81%) IMD cases (Tables 5 and 6). The predominant *porA* genotypes for serogroup B IMD cases were again P1.7-2,4 (29 cases, 35% of serogroup B IMD cases that were typeable) and P1.7,16-26 (14 cases, 17% of serogroup B IMD cases that were typeable) (Figure 3). In 2014, the genotype P1.7-2,4 accounted for 18% (14 cases) of serogroup B IMD cases that were typeable, and

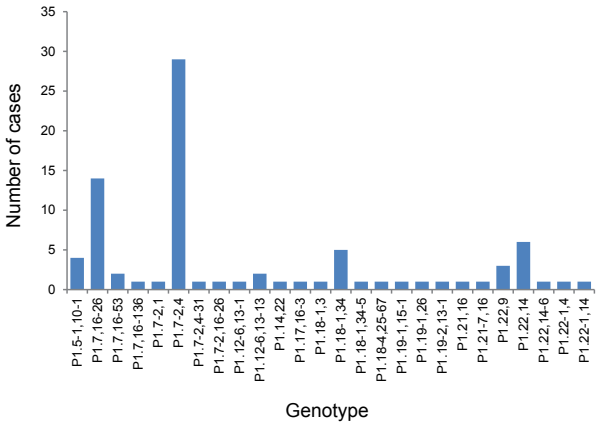
between 2011 and 2014 was the most common genotype detected for serogroup B IMD cases that were typeable (18% to 30%).The predominant *porA* genotype for serogroup Y IMD cases was P1.5-1,10-1 (14 cases, 70% of serogroup Y IMD cases that were typeable). This was higher than in 2014 (6 cases, 50% of serogroup Y IMD cases that were typeable) but the overall numbers were low.

Table 5: Laboratory confirmed cases of invasive meningococcal disease, Australia, 2015, by *porA* genotype

Genotype	B	C	Y	W	NG	Total
P1.5,2	0	0	0	31	0	31
P1.5,2-1	0	1	0	0	0	1
P1.5-1,2-2	0	0	0	1	0	1
P1.5-1,10-1	4	0	14	0	0	18
P1.5-1,10-4	0	0	1	1	0	2
P1.5-1,10-8	0	0	1	0	0	1
P1.5-2,10-1	0	0	3	0	0	3
P1.5-2,10-29	0	0	1	0	0	1
P1.7,16-26	14	0	0	0	0	14
P1.7,16-53	2	0	0	0	0	2
P1.7,16-136	1	0	0	0	0	1
P1.7-2,1	1	0	0	0	0	1
P1.7-2,4	29	0	0	0	0	29
P1.7-2,4-31	1	0	0	0	0	1
P1.7-2,16-26	1	0	0	0	0	1
P1.12-6,13-1	1	0	0	0	0	1
P1.12-6,13-13	2	0	0	0	0	2
P1.14,22	1	0	0	0	0	1
P1.17,16-3	1	0	0	0	0	1
P1.18-1,3	1	0	0	1	0	2
P1.18-1,34	5	0	0	0	1	6
P1.18-1,34-5	1	0	0	0	0	1
P1.18-4,25-67	1	0	0	0	0	1
P1.19,15-1	1	0	0	0	0	1
P1.19-1,26	1	0	0	0	0	1
P1.19-2,13-1	1	0	0	0	0	1
P1.21,16	1	0	0	1	0	2
P1.21-7,16	1	0	0	0	0	1
P1.22,9	3	0	0	0	0	3
P1.22,14	6	0	0	0	0	6
P1.22,14-6	1	0	0	0	0	1
P1.22-1,4	1	0	0	0	0	1
P1.22-1,14	1	0	0	0	0	1
Total	83	1	20	35	1	140

Of the 31 serogroup W IMD strains that were able to be genotyped, 25/31 (81%) were sequence type (ST)-11, and had the *porA* antigen encoding gene type P1.5,2; the same genotype as the hypervirulent serogroup W strain reported in the United Kingdom and South America since 2009[6,7] (Table 7).

Figure 3: Number of *porA* genotypes* for serogroup B in cases of invasive meningococcal disease, Australia, 2015



* Where data available.

Antibiotic susceptibility testing

Testing for antimicrobial susceptibility was performed for 117/174 (67%) IMD cases in 2015. All isolates tested were susceptible to ceftriaxone and ciprofloxacin. There was 1 isolate that was resistant to rifampicin (MIC=1 mg/L). Using the defined criteria, 12/117 (10.3%) isolates were fully sensitive to penicillin (MIC 0.03 mg/L or less), and 101 (86%) isolates were less sensitive to penicillin (MIC=0.06–0.5 mg/L). Four isolates were resistant to penicillin (MIC ≥ 1 mg/L).

Discussion

In 2015, there were 174 cases of laboratory confirmed IMD, representing 96% of the number of notifications to the NNDSS.[2] Whilst this was higher than the number of IMD cases reported in 2014, it represents the second lowest number of IMD cases reported in Australia since laboratory based surveillance (AMSP) began in 1994, and since notification data collection commenced in 1991, and represents about one-third of the peak number of IMD cases reported in Australia in 2002 (n=580). The introduction of the serogroup C vaccine to the national immunisation schedule in 2003 has resulted in a very large and sustained reduction in the number and proportion of sero-

Table 6: Distribution of *porA* genotype laboratory confirmed cases of invasive meningococcal disease, Australia, 2015, by state or territory

Genotype	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA
P1.5,2		9 W		3 W		1 W	15 W	3 W
P1.5,2-1		1 C						
P1.5-1,2-2							1W	
P1.5-1,10-1		3 B,6 Y		1 B,1 Y			6 Y	1 Y
P1.5-1,10-4		1Y		1 W				
P1.5-1,10-8								1 Y
P1.5-2,10-1				1 Y			2 Y	
P1.5-2,10-29	1 Y							
P1.7,16-26		3 B		4 B	1 B		5 B	1 B
P1.7,16-53				2 B				
P1.7,16-136				1 B				
P1.7-2,1				1 B				
P1.7-2,4		4 B		5 B	13 B		5 B	2 B
P1.7-2,4-31							1 B	
P1.7-2,16-26		1 B						
P1.12-6,13-1					1 B			
P1.12-6,13-13					2 B			
P1.14,22				1 B				
P1.17,16-3							1 B	
P1.18-1,3					1 W		1 B	
P1.18-1,34				1 NG	2 B		3 B	
P1.18-1,34-5				1 B				
P1.18-4,25-67		1 B						
P1.19,15-1					1 B			
P1.19-1,26				1 B				
P1.19-2,13-1							1 B	
P1.21,16							1 B	1 W
P1.21-7,16		1 B						
P1.22,9		1 B					2 B	
P1.22,14		1 B		2 B			1 B	2 B
P1.22,14-6							1 B	
P1.22-1,4								
P1.22-1,14				1 B				

Table 7: Laboratory confirmed cases of serogroup W invasive meningococcal disease, Australia, 2015, by whole genome sequence type

	Whole genome sequence type							
W genotype	ST11	ST22	ST23	ST184	ST1287	Not typeable	Not tested	Total
P1.5,2	25	0	0	0	2	1	3	31
P1.5-1,2-2	0	1	0	0	0	0	0	1
P1.5-1,10-4	0	0	1	0	0	0	0	1
P1.18-1,3	0	0	0	1	0	0	0	1
P1.21,16	0	0	0	1	0	0	0	1
Not typeable	0	0	0	0	0	1	0	1
Total	25	1	1	2	2	2	3	36

group C IMD cases in this country. In 2015, the number and proportion of serogroup C IMD cases was the lowest ever reported by the AMSP. In 2015 the majority of IMD cases continue to be caused by serogroup B strains; however the overall proportion IMD caused by serogroup B was the lowest since 2003. In early 2014, a recombinant multicomponent meningococcal B vaccine became available in Australia.[8] This vaccine is not on the immunisation register but is available for purchase privately. Therefore uptake is elective and the impact of its introduction is yet to be determined in this country.

All IMD isolates were susceptible to ceftriaxone and ciprofloxacin; and there was 1 isolate that was resistant to rifampicin, and 4 isolates that were resistant to penicillin. The proportion of IMD isolates with penicillin MIC values in the less sensitive category in 2015 was 86%. In previous years the range was 62% to 75% in 1996–2006; 67% to 79% in 2007–2009; and 78% to 88% in 2010–2014, thus indicating an ongoing increasing trend in penicillin MIC values of IMD isolates. The incidence of penicillin resistance in *N. meningitidis* in Australia however, remains low.

In 2015, a number of changes in IMD epidemiology were observed in Australia. There was a notable increase in the number of IMD cases caused by serogroups W and Y, the highest yet reported by the AMSP, and these were the predominant serogroups causing IMD in those aged 65 years or over. In addition, in 2015, the primary peak of IMD was observed for the first time in adults aged 45 years or more, because of the increased number of serogroup W and serogroup Y IMD cases in this age group. Secondary disease peaks were observed in those aged 4 years or less, and in adolescents (15–19 years).

In 2015, 21% of all IMD notifications were serogroup W, with the highest proportion of cases in New South Wales (22%), Victoria (17%), and Queensland (17%). Whole genome sequencing found that the predominant circulating strain of serogroup W in Australia in 2015, (25/31, 81%), was sequence type (ST)-11, and had the *porA* antigen encoding gene type P1.5,2. This is the same genotype as the hypervirulent serogroup W strain that emerged in the United Kingdom and South America in 2009[6,7] and has spread to now account for 25% of IMD in the United Kingdom in 2014–2015, and 59% of all cases in Chile in 2012. This serogroup W strain is now considered endemic in these regions, is associated with atypical presentations, more severe clinical disease and a higher case fatality rate.[7] In these regions, the initial increase in IMD was seen in older adults, but was subsequently reported in all age groups, particularly in adolescents and infants.[9] In response, vaccination programs have been implemented in both the United Kingdom and in Chile.[6,10] The increase in cases of serogroup W IMD is of significant concern and the NNN is working on further investigations with the Australian Government Department of Health and the Communicable Diseases Network Australia and is closely monitoring the phenotypic and genotypic features of *N. meningitidis* causing IMD in Australia. Additional investigations including whole genome sequencing of all Serogroup W are in place to enhance surveillance practices. The AMSP data are used for informing treatment protocols and to inform the need for and monitor the effect of disease prevention strategies.

Acknowledgements

Meningococcal isolates were received in the reference centres from many laboratories throughout Australia. The considerable time and effort involved in forwarding these isolates is recognised and these efforts are greatly appreciated. These data could not have been provided without this assistance and the help of clinical colleagues and public health personnel. The Australian Government Department of Health provided funding for the National Neisseria Network.

Members of the AMSP in 2015 were: John Bates, Helen Smith and Vicki Hicks, Public Health Microbiology, Forensic and Scientific Services, Coopers Plains, Queensland; Monica Lahra, Rodney Enriquez; Tiffany Hogan; Ratan Kundu and Athena Limnios, Department of Microbiology, SEALS, The Prince of Wales Hospital, Randwick, New South Wales; Dr Michael Maley, Robert Porritt and Joanne Mercer, Department of Microbiology and Infectious Diseases, SSWPS, Liverpool, New South Wales; Kerrie Stevens, The Microbiological Diagnostic Unit Public Health Laboratory, Department of Microbiology and Immunology, University of Melbourne, Parkville, Victoria; Andrew Lawrence, Microbiology and Infectious Diseases Department, SA Pathology, Adelaide, South Australia; Jane Bew, Leanne Sammels and Tony Keil, Department of Microbiology, Princess Margaret Hospital for Children, Subiaco, Western Australia; Belinda McEwan Belinda Chamley and Dr McGregor Department of Microbiology and Infectious Diseases, Royal Hobart Hospital, Hobart, Tasmania; Rob Baird, Kevin Freeman and microbiology staff, Microbiology Laboratory, Territory Pathology, Royal Darwin Hospital, Tiwi, Northern Territory; Angelique Clyde-Smith and Peter Collignon, Microbiology Department, Canberra Hospital, Garran, Australian Capital Territory.

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Flutracking weekly online community survey of influenza-like illness annual report, 2015

Craig B Dalton, Sandra J Carlson, David N Durrheim, Michelle T Butler, Allen C Cheng, Heath A Kelly

Abstract

Flutracking is a national online community influenza-like illness (ILI) surveillance system that monitors weekly ILI activity and impact in the Australian community. This article reports on the 2015 findings from Flutracking. From 2014 to 2015 there was a 38.5% increase in participants to 27,824 completing at least 1 survey with a peak weekly response of 25,071 participants. The 2015 Flutracking national ILI weekly fever and cough percentages peaked in late August at 5.0% in the unvaccinated group, in the same week as the national counts of laboratory confirmed influenza peaked. A similar percentage of Flutracking participants took two or more days off from work or normal duties in 2015 (peak level 2.3%) compared with 2014 (peak level 2.5%) and the peak weekly percentage of participants seeking health advice was 1.6% in both 2014 and 2015. Flutracking fever and cough peaked in the same week as Influenza Complications Alert Network surveillance system influenza hospital admissions. The percentage of Flutracking participants aged 5 to 19 years with cough and fever in 2015 was the highest since 2011. The 2015 season was marked by a transition to predominantly influenza B strain circulation, which particularly affected younger age groups. However, for those aged 20 years and over, the 2015 national Flutracking influenza season was similar to 2014 in community ILI levels and impact. *Commun Dis Intell* 2016;40(4):E512–E520.

Keywords: influenza, surveillance, syndromic surveillance, influenza-like illness, survey, Flutracking.

Introduction

Flutracking provides weekly community level influenza-like illness (ILI) surveillance that is not biased by health seeking behaviour, clinician testing practices or differences in jurisdictional surveillance methods.[1–4] Flutracking provides an indication of differential ILI rates in influenza-vaccinated and unvaccinated participants and impact of illness at a community level.[5] The Flutracking surveillance system has been incorporated into the weekly Australian Influenza Surveillance Report since 2009.[6]

The main aims of Flutracking are to:

1. compare ILI syndrome proportions between vaccinated and unvaccinated participants to detect inter-pandemic and pandemic influenza;
2. provide consistent surveillance of influenza activity across all jurisdictions and over time; and
3. provide year-to-year comparison of the timing, incidence, and impact of influenza on the community.

In this report, we aim to describe

- the epidemiology of ILI and influenza vaccination in the community;
- the performance characteristics of the Flutracking system; and
- the comparison between these estimates with other sources of surveillance data in the 2015 season.

Methods

The Flutracking surveillance system was in operation for 26 weeks in 2015, from the week ending Sunday 26 April to the week ending Sunday 18 October 2015. The cohort of participants is maintained year to year and is boosted by an annual recruitment drive, which usually runs from March to May. In 2015, the recruitment drive was from 1 April to 6 May. Participants may opt out of participating at any time. The recruitment methods in 2015 were similar to those used in 2007–2014.[1]

The weekly survey questions evolved from 2007–2012.[1,7] but no changes have been made to the questions from 2013 onwards.

Descriptive statistics

Descriptive statistics were tabulated and summarised for each state and territory, by age group, gender, education level, Aboriginal and Torres Strait Islander status, and vaccination status.

A participant is defined as anyone who has a survey submitted by them self or on their behalf. A respondent is anyone who submits a survey either for them self or on behalf of a household member.

The participation rate was calculated using the Australian Bureau of Statistics June 2015 Estimated Resident Population[8] for state and territory, age group, gender, education level, and Aboriginal and Torres Strait Islander status.

We analysed the proportion of vaccinated participants aged less than 10 years of age by whether there was at least 1 participant in their household who was a healthcare worker with patient contact.

The mean proportion of participants who responded by 24 hours after the survey was distributed was calculated across all 26 weeks of surveillance. This calculation was also stratified by age groups. Time taken to respond to the survey was calculated for primary participants only (that is, those responding for themselves and possibly other household members). For participants in Western Australia, 2 hours were subtracted from their time to respond, to account for differences in time zones. No adjustment was made for the 30 minute time zone difference in South Australia and the Northern Territory.

Unless otherwise stated, a participant with ILI was defined as having both self-reported fever and cough. For ILI percentage calculations, the numerator was all persons who completed a survey for the current week and reported ILI symptoms, and the denominator was all persons who completed a survey for the current week. Weekly ILI percentages were compared by self-reported vaccination status for participants. The unstratified (by vaccination status) ILI percentages were also compared with national laboratory confirmed influenza notifications for 2009 to 2015.

Weekly percentages of community ILI were calculated. ILI percentages, not stratified by vaccination status, were compared with FluCAN admissions to hospital for influenza from 2012 to 2015.

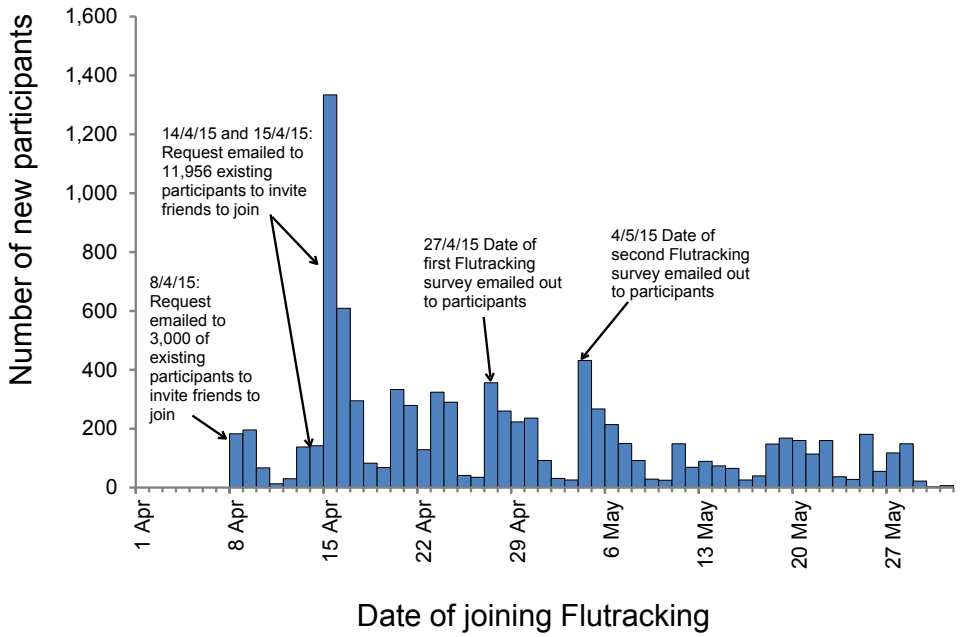
The weekly percentage of participants from 2011 to 2015 who had 1) fever and cough and 2 or more days off work or normal duties; and 2) who visited a general practitioner, emergency department or were admitted to hospital due to fever and cough was compared.

Results

Recruitment in 2015

Participants were encouraged to join at any time during the year. Similar to previous years, the most successful recruitment strategy in 2015 was recruitment through previous participants. There were 17,687 participants who previously participated in 2014 and also completed at least 1 survey in 2015. On 8, 14, and 15 April 2015, a *Welcome Back to Flutracking* email was sent to all active participants (15,082 participants who respond for themselves and other household members) with a suggestion that participants invite friends to join the survey. From 1 January to 7 April 2015, 50 people had enrolled. On 8 and 9 April, respectively 183 and 196 participants enrolled. On 14, 15 and 16 April, respectively 142, 1,334 and 609 participants enrolled. There were an additional 356 participants recruited on 27 April and an additional 432 participants recruited on 4 May: these spikes correspond to the dates the first and second Flutracking survey emails were sent to participants (Figure 1).

Figure 1: Significant Flutracking recruitment events and impact, 2015



The Western Australia Department of Health recruited an additional 1,873 participants into Flutracking to support a research project in 2015.

The 30 organisations with the highest number of participants in Flutracking for 2014 were emailed a 'Congratulations' certificate and asked to invite any new employees to participate in Flutracking in 2015.

Facebook requests to participants to 'like' the Flutracking page resulted in an increase in page likes from 2,497 'likes' (6 April 2015) to 3,138 'likes' (20 April 2015).

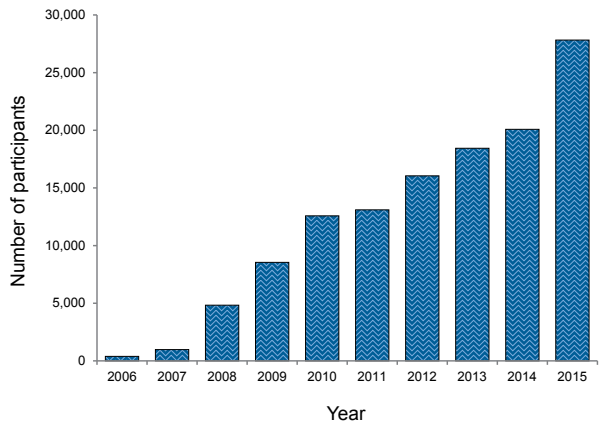
As a result of the above recruitment strategies and media coverage a total of 9,987 participants joined the survey in 2015, compared with the 5,099 that joined in 2014.

Participation in 2015

There were 16,707 respondents for 27,828 participants who completed at least 1 survey in 2015. Of the 26,224 participants who completed a survey during the first 4 survey weeks of 2015, 59.1% completed all available surveys, and 78.0% completed more than 90% of available surveys. From 2014 onwards, once they have finished their current survey, participants have been able to click on any prior surveys from the past 5 weeks to complete. This has increased the percentage of surveys completed for both 2014 and 2015.

There were 27,824 participants who completed at least 1 survey in 2015, compared with 20,087 in 2014 (a 38.5% increase) (Figure 2). At a state and territory level, increases in peak weekly participation were most marked in Western Australia, the Australian Capital Territory, Queensland, and Victoria. In 2015, Tasmania had the highest rate of Flutracking participation per 100,000 persons, followed by the Northern Territory and the Australian Capital Territory (Table 1). New South Wales, Victoria, and Queensland all had lower rates of participation than the general Australian population (Table 1).

Figure 2: Number of participants who completed at least one survey, Australia, 2006 to 2015, by year



Socio-demographic characteristics

Of the participants who completed at least 1 survey and responded to each of the demographic questions in 2015, 59.0% were aged 35–64 years, 61.6% were female, 61.7% had completed a bachelor degree, graduate diploma or certificate or post-graduate degree, and 1.3% identified as Aboriginal and/or Torres Strait Islander (Table 2). Compared with the national profile, the following demographic groups were underrepresented among Flutracking participants: 0–34 years and 65 years or over age groups; males; Aboriginal and Torres Strait Islander participants; and participants with lower levels of educational attainment.

Time to respond to survey each week

Most participants responded within 24 hours of the survey being sent, with a mean 24 hour response of 72.9% over the 26 weeks. The 65 years or over age group had the highest percentage of all age groups responding within 24 hours, with a mean 24 hour response of 81.4% over the 26 weeks.

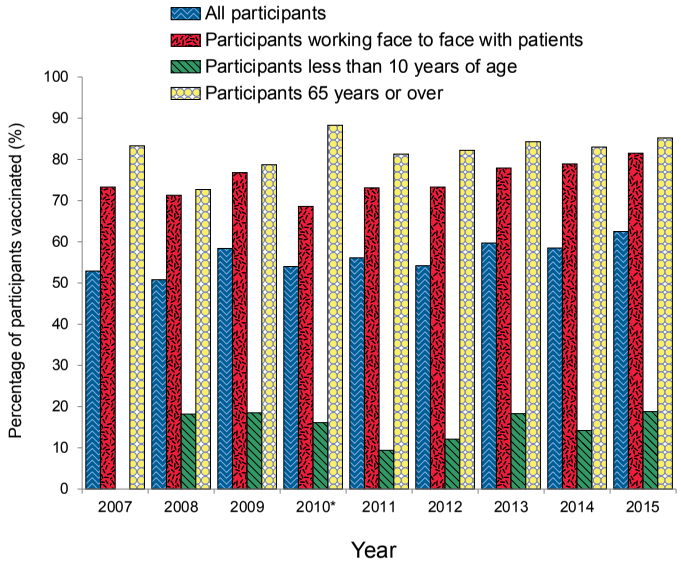
Percentage of participants vaccinated

Seasonal vaccination levels among participants were higher in 2015 than prior years. By the end of the 2015 season (week ending 18 October 2015), 62.5% (13,313/21,293) of participants had received the 2015 seasonal vaccine, compared with 58.5% (9,742/16,642) of participants by the end of 2014. Of the 4,447 participants who identified as working face-to-face with patients in 2015, 3,622 (81.4%) received the vaccine compared with 78.9% by the end of 2014. In 2015, 18.8% (269/1,429) of participants less than 10 years of age were vaccinated with the seasonal influenza vaccine by the end of the season, compared with 14.0% in 2014. In 2015, among households with at least 1 member working face-to-face with patients, 17.5% of participants aged less than 10 years were vaccinated, compared with 16.9% of participants aged less than 10 years in households without a member working face-to-face with patients. In 2015, 85.2% (2,411/2,054) participants aged 65 years or over were vaccinated with the seasonal influenza vaccine by the end of the season, compared with 83.0% in 2014 (Figure 3).

Table 1: Recruitment to Flutracking, 2014 and 2015, by state or territory

State or territory	2014 Number of participants (peak week)	2014 Flutracking participation per 100,000 population	2014 Per cent of participants (peak week)	2015 Number of participants (peak week)	2015 Flutracking participation per 100,000 population	2015 Per cent of participants (peak week)	% Distribution of the underlying Aust. population	Percentage positive change
ACT	662	169.4	3.6	1,062	271.8	4.2	1.6	60.4
NSW	6,357	83.4	34.8	7,525	98.8	30.0	32.0	18.4
NT	801	327.5	4.4	954	390.0	3.8	1.0	19.1
Qld	1,726	36.1	9.4	2,283	47.8	9.1	20.1	32.3
SA	2,840	167.2	15.5	3,355	197.5	13.4	7.1	18.1
Tas.	2,012	389.5	11.0	2,368	458.4	9.4	2.2	17.7
Vic.	2,844	47.9	15.6	3,482	58.6	13.9	25.0	22.4
WA	1,045	40.3	5.7	4,041	155.9	16.1	10.9	286.7
Total	18,287	76.9	100.0	25,070	105.4	100.0	100.0	37.1

Figure 3: Percentage of participants vaccinated with the seasonal influenza vaccine at the final survey of each year, by participant characteristics, Australia, 2007 to 2015, by year



* This percentage calculation is for those participants who received either the monovalent H1N109 influenza vaccine in 2009 or 2010, or received the 2010 seasonal influenza vaccine.

Table 2: Socio-demographic characteristics of Flutracking participants who completed at least one survey during 2014 and 2015

	2014			2015			% Distribution of the Australian population
	Frequency	Per cent	Rate per 100,000*	Frequency	Per cent	Rate per 100,000*	
Age (years)							
0–15	2,638	12.6	55.4	3,446	12.4	72.4	20.0
16–34	3,754	17.9	59.0	5,160	18.5	81.1	26.7
35–49	5,405	25.7	112.7	7,040	25.3	146.8	20.2
50–64	7,311	34.8	170.2	9,384	33.7	218.5	18.1
65 and over	1,909	9.1	53.5	2,793	10.0	78.2	15.0
Total participants	21,017	100.0	88.4	27,823	100.0	117.0	100.0
Gender							
Male	7,461	38.6	63.1	10,132	38.4	85.6	49.7
Female	11,867	61.4	99.3	16,224	61.6	135.8	50.3
Total reported	19,328	100.0	81.3	26,356	100.0	110.8	100.0
Education†							
Year 11 or below (or equiv) or Certificate I/II/III/IV	3,417	21.0	44.7	4,615	20.9	60.3	44.1
Year 12 (or equivalent)	1,299	8.0	45.0	1,849	8.4	64.1	16.6
Advanced Diploma/ Diploma	1,538	9.5	110.4	2,028	9.2	145.6	8.0
Completed Bachelor Degree	3,801	23.4	162.4	5,304	24.0	226.6	13.5
Grad Diploma/ Grad Certificate	2,265	13.9	761.9	3,002	13.6	1009.8	1.7
Postgraduate Degree	3,921	24.1	621.3	5,331	24.1	844.7	3.6
Total who nominated an ABS equivalent education level (15 years and over only)	16,241	100.0		22,129	100.0		
Aboriginal and/or Torres Strait Islander							
Yes	235	1.4	35.1	304	1.3	45.4	3.0
No	16,309	98.6	75.3	23,540	98.7	108.6	97.0
Total reported	16,544	100.0	74.1	23,844	100.0	106.7	100.0

* Calculated using Australian Bureau of Statistics (ABS) June 2015 quarter Estimated Resident Population.

† Note that the rate per 100,000 was calculated using participants aged 15–74 years for all categories except Enrolled Bachelor Degree. The rate per 100,000 for Enrolled Bachelor Degree was calculated using participants aged 15–64 years of age. These age adjustments were necessary to match the age groups in the ABS data.

Percentage of participants with influenza-like illness symptoms

Of participants who completed a survey in the national peak week of ILI for 2015, 4.3% reported fever and cough compared with 4.7% in 2014 and 3.6% in 2013. Of participants who completed at least 1 survey in the national peak 4 weeks of ILI for 2015, 11.7% reported fever and cough, compared with 12.4% in 2014 and 9.6% in 2013 (Table 3).

Detection of influenza-like illness

Figure 4 shows the 2009 to 2015 weekly ILI percentages by vaccination status. Peak ILI activity for 2015 was during the week ending 23 August (5.0% in the unvaccinated group and 3.8% in the vaccinated group). Divergence between the vaccinated and unvaccinated participants' ILI percentages was highest during the following week of week ending 30 August (4.6% in the unvaccinated group and 3.2% in the vaccinated group). The levels of ILI seen in 2015 were similar to the 2014 and 2012 seasons.

Comparison with national laboratory influenza notifications

There was an increase in the number of laboratory confirmed cases of influenza from 7,170 notifications in the peak week of laboratory notifications for 2014 to 10,678 notifications in the peak week of laboratory notifications for 2015. However, the peak weekly 2015 Flutracking ILI percentage unstratified by vaccination status was lower (4.3%) than the peak weekly 2014 percentage (4.7%) (Figure 5). In 2015, the timing of the peak week

Figure 4: Fever and cough percentage stratified by vaccination status, Australia, 2009 to 2015, by week

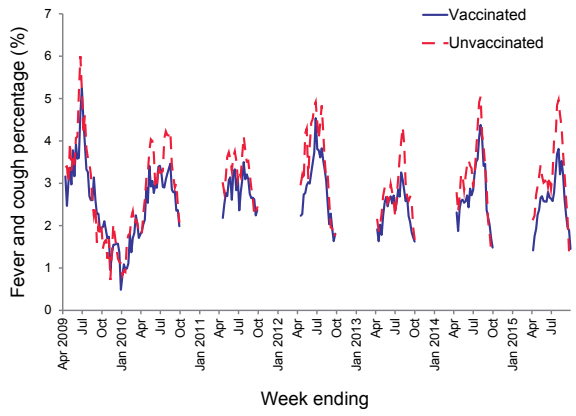
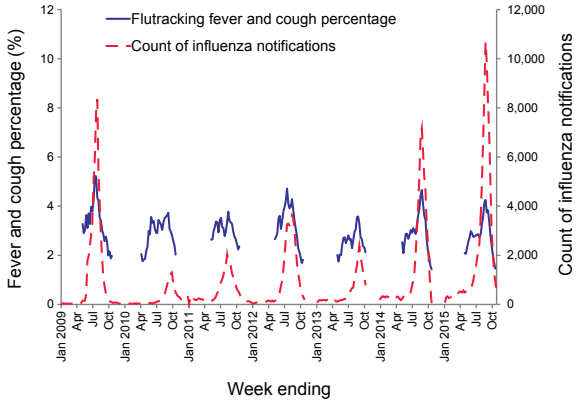


Figure 5: Fever and cough percentage, 1 April to 31 October* compared with national influenza laboratory notifications, Australia, 2009 to 2015, by week



* Not stratified by vaccination status

Table 3: Percentage of participants with influenza-like illness symptoms who completed a survey either in the national peak influenza-like illness week, or completed at least one survey in the national peak 4 weeks of influenza-like illness, Australia, 2013 to 2015

	Participants who completed a survey in the national peak ILI week						Participants who completed at least one survey during the national peak 4 weeks ILI					
	2013*		2014†		2015‡		2013§		2014¶		2015**	
Symptoms	n	%	n	%	n	%	n	%	n	%	n	%
Fever	742	4.8	1,067	5.8	1,289	5.4	2,130	12.5	2,920	15.2	3,568	14.2
Cough	2,208	14.2	2,957	16.2	3,751	15.6	4,816	28.3	6,212	32.4	7,727	30.7
Fever and cough	558	3.6	852	4.7	1,021	4.3	1,634	9.6	2,385	12.4	2,942	11.7
Fever, cough and sore throat	430	2.8	618	3.4	745	3.1	1,263	7.4	1,775	9.3	2,288	9.1

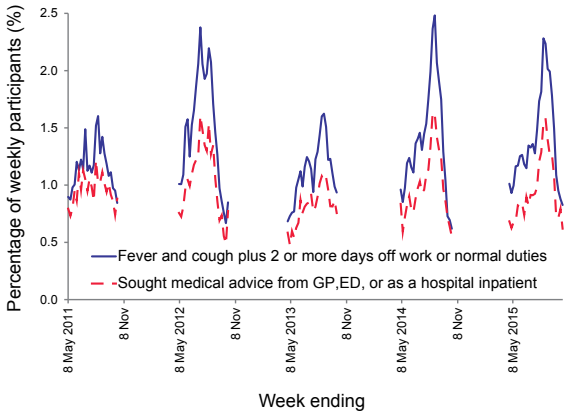
* ending 25 August 2013, N=1 Week 5579.
† Week ending 24 August 2014, N=18,287.
‡ Week ending 23 August 2015, N=23,985.
§ Weeks ending 11 August to 1 September 2013, N=16,988.
¶ Weeks ending 10 August to 31 August 2014, N=19,188.
** Weeks ending 16 August to 6 September 2015, N= 25,196.

of Flutracking ILI levels was the same as the timing of the peak week of laboratory notifications of influenza (week ending 23 August 2015).

Time off work or normal duties and health seeking behaviour

The peak weekly percentage of participants taking time off work or normal duties in 2015 was 2.3% in 2015 and 2.5% in 2014, while the peak weekly percentage of participants seeking health advice was 1.6% in both 2014 and 2015 (Figure 6).

Figure 6: Figure 6: Weekly influenza-like illness severity,*Australia, 2011 to 2015, by week



* The denominator is the number of weekly participants.

GP General practitioner

ED Emergency department

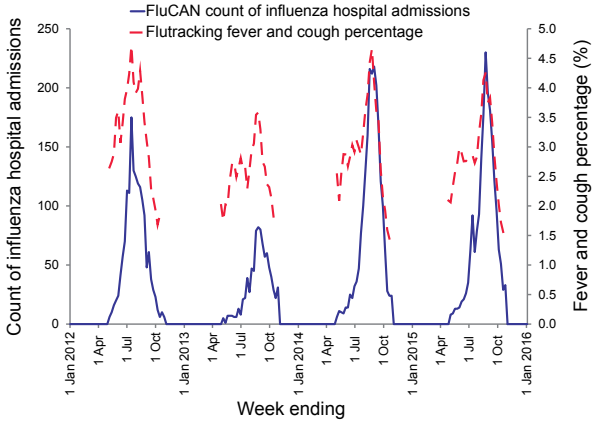
Comparison of Flutracking influenza-like illness with the hospital-based FluCAN surveillance system

There was an increase in the number of influenza admissions with confirmed influenza recorded in the peak week of FluCAN surveillance data (218 in the peak week of 2014 compared with 230 in the peak week of 2015). In 2015 the timing of the peak week of Flutracking ILI levels (fever and cough) was the same as the timing of the peak week of FluCAN hospital admissions for influenza (week ending 23 August 2015) (Figure 7).

Percentage of participants with influenza-like illness by age group

The percentage of Flutracking participants aged 5–19 years with cough and fever in 2015 was the highest since 2011 (Figure 8).

Figure 7: Percentage of Flutracking participants with fever and cough compared with FluCAN influenza hospital admissions, Australia, 2012 to 2015, by week



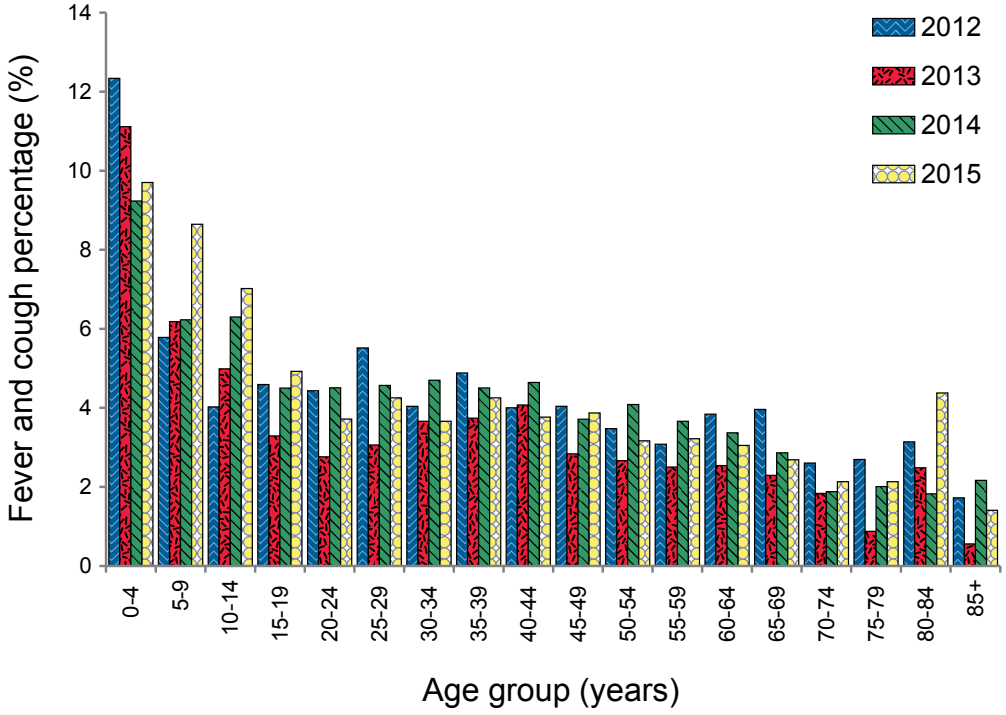
Discussion

The number of participants enrolled to participate in Flutracking increased markedly from 2014 to 2015. Although there were increases in participation in every state and territory in 2015, almost a quarter (24%) of this national increase in participation could be attributed to Western Australia Department of Health encouraging employees to sign up to Flutracking to participate in a vaccine effectiveness study. Dissemination of invitations to potential participants by existing Flutracking participants also continued to be an effective method of recruitment.

As the recruitment strategies were similar in 2015 to 2014, the distribution of participants among each age group, gender, education level, and Aboriginal and Torres Strait Islander status group has remained similar to 2014. There is still under-representation of participants in the 0–34 years and 65 years or over age groups, males, participants with non-tertiary education and Aboriginal and Torres Strait Islander participants. This highlights the need for more targeted recruitment strategies reaching out to under-represented groups through consultation and targeted recruitment campaigns.

Influenza vaccine coverage of Flutracking participants increased in 2015 to its highest recorded level since 2007 for all participants, and more specifically, for participants working face-to-face with patients and participants aged less than 10 years of age. Among children less than 10 years of age, the percentage who received influenza vaccination has gradually increased since 2011. These results do not appear to be biased by children with healthcare

Figure 8: Percentage of participants with fever and cough episodes, by year and age group, Australia, 2012 to 2015*



* Only the 4 peak weeks of fever and cough in Australia for each year were included.

workers in the family. The lower levels of vaccination in this age group in 2011 may be attributed to the increased reports of fever and febrile convulsions in children less than 10 years of age after vaccination with Fluvax and Fluvax Junior in 2010.[9] We acknowledge that Flutracking participants may be more likely to be vaccinated than the general population based on motives to participate in the survey, and because unvaccinated participants are asked about their vaccination status each week.

Based on Flutracking fever and cough weekly percentages, the peak community ILI levels in the 2015 season were slightly lower than the peak community ILI levels in the 2012 and 2014 seasons. However, national influenza laboratory notifications showed an increase in influenza cases from 2014 to 2015. This discrepancy between the relative magnitude of the influenza season as described by laboratory notifications and Flutracking most likely reflected an increase in laboratory testing. Fielding et al noted that public funding for the purchase of new polymerase chain reaction (PCR) testing equipment following the 2009 H1N1 influenza pandemic and Medicare reimbursement for PCR testing, may have influenced provider practice and driven increases in influenza testing.[10]

Flutracking analyses suggest similar levels of ILI in the community in 2015 and 2014, with slightly lower time off normal duties in 2015 and similar health care seeking behaviour. FluCAN's influenza peak number of weekly hospital admissions also suggested that the 2014 and 2015 seasons were not meaningfully different. The total number of FluCAN influenza hospital admissions for 2015 were similar to 2014 (2,060 in 2015 as compared with 2,092 in 2014). Compared with 2014, when there was a mix of influenza A H1N1 and H3N2 with lower levels of influenza B, 2015 was marked by a transition to predominantly influenza B strain circulation with the B/Victoria lineage dominating the B/Yamagata lineage by the end of the influenza season.[6] Influenza B has been regarded as less severe than influenza A infection but no difference was identified in a recent comparison of severity of influenza A and B infection based on outcomes among hospitalised patients.[11] The similar levels of severity overall in 2014 and 2015 seen in Flutracking and FluCAN supports this.

The predominance of influenza B activity in 2015 particularly affected younger age groups. Flutracking ILI percentages were at their highest since 2011 in the 5–19 years age groups and this was reflected in laboratory surveillance.[6] In summary, in 2015 Flutracking demonstrated that the severity of the 2015 season at the community level was similar to 2014, except in participants under 20 years of age who were likely impacted by circulating influenza B strains.

Competing interests

All authors declare that they have no competing interests.

Acknowledgements

The authors would like to acknowledge the University of Newcastle for their continued support, and the Australian Government Department of Health and the Hunter Medical Research Institute for their funding and support. We would also like to acknowledge Stephen Clarke for software and database development, and John Fejsa for his contribution to the design of the project, and the thousands of Flutracking participants who give their time freely each week to contribute to influenza surveillance.

Author contributions

Craig Dalton conceived and designed the project, oversaw the statistical analysis, and contributed to writing of the manuscript. Sandra Carlson contributed to the writing of the manuscript and the statistical analysis. David Durrheim contributed to the design of the project and writing of the manuscript. Michelle Butler also contributed to the statistical analysis. Allen Cheng provided FluCAN data for comparison and contributed to the interpretation of data and writing of the manuscript. Heath Kelly contributed to data interpretation and writing of the manuscript.

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Influenza epidemiology in patients admitted to sentinel Australian hospitals in 2015: the Influenza Complications Alert Network

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Abstract

The Influenza Complications Alert Network (FluCAN) is a sentinel hospital-based surveillance program that operates at sites in all states and territories in Australia. This report summarises the epidemiology of hospitalisations with laboratory-confirmed influenza during the 2015 influenza season. In this observational study, cases were defined as patients admitted to one of the sentinel hospitals with an acute respiratory illness with influenza confirmed by nucleic acid detection. During the period 1 April to 30 October 2015 (the 2015 influenza season), 2,070 patients were admitted with confirmed influenza to one of 17 FluCAN sentinel hospitals. Of these, 46% were elderly (≥ 65 years), 15% were children (< 16 years), 5% were Indigenous Australians, 2.1% were pregnant and 75% had chronic co-morbidities. A high proportion were due to influenza B (51%). There were a large number of hospital admissions detected with confirmed influenza in this national observational surveillance system in 2015 with case numbers similar to that reported in 2014. The national immunisation program is estimated to avert 46% of admissions from confirmed influenza across all at-risk groups, but more complete vaccination coverage in target groups could further reduce influenza admissions by as much as 14%. *Commun Dis Intell* 2016;40(4):E521–E526.

Keywords: influenza; hospitalisation; morbidity; FluCAN

Introduction

Influenza is a common respiratory viral infection that affects up to 5% to 10% of the population each year.[1] Although the proportion of cases requiring hospitalisation is low, because infection with influenza virus is relatively widespread, the incidence of hospitalisation from influenza is of public health significance.[2] In this report we describe the epidemiology of hospitalisation with laboratory-confirmed influenza in the 2015 season in Australia.

Methods

The Influenza Complications Alert Network (FluCAN) is a national hospital-based sentinel surveillance system in Australia.[3] Since 2011, the participating sites have been Canberra Hospital (ACT), Calvary Hospital (ACT), Westmead Hospital (NSW), John Hunter Hospital (NSW), Children's Hospital at Westmead (NSW), Alice Springs Hospital (NT), Royal Adelaide Hospital (SA), Mater Hospital (Qld), Princess Alexandra Hospital (Qld), Cairns Base Hospital (Qld), Royal Hobart Hospital (Tas.), The Alfred Hospital (Vic.), Royal Melbourne Hospital (Vic.), Monash Medical Centre (Vic.), University Hospital Geelong (Vic.), Royal Perth Hospital (WA), and Princess Margaret Hospital (WA).

Ethical approval has been obtained at all participating sites and at Monash University. Hospital bed capacity statistics were obtained from each participating hospital, and national bed capacity was obtained from the last published Australian Institute for Health and Welfare report.[4]

A case was defined as a patient admitted to hospital with influenza confirmed by nucleic acid testing (NAT). For each case, a control was the next tested patient admitted to hospital for an acute respiratory illness where the influenza NAT was negative. Surveillance is conducted from early April to end October (with follow up continuing to the end of November) each year. Admission or transfer to an intensive care unit (ICU) included patients managed in a high dependency unit. The onset date was defined as the date of admission except for patients where the date of the test was more than 7 days after admission, where the onset date was the date of the test. The presence of risk factors and comorbidities was ascertained from the patient's medical record. Restricted functional capacity was defined as those who were not fully active and not able to carry out all activities without restriction prior to the acute illness.[5]

We examined factors associated with ICU admission using multivariable regression. Factors associated with ICU admission were determined using a logistic regression model, with factors retained in the multivariable model if $P < 0.2$.

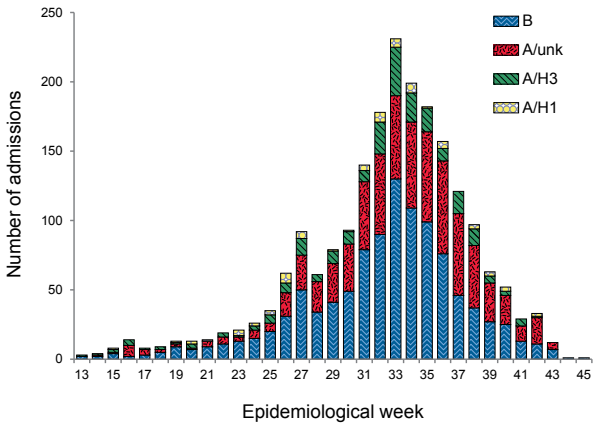
Vaccine coverage was estimated from the proportion of vaccinated test negative control patients in each age group, stratified by the presence of chronic comorbidities. Vaccine effectiveness was estimated from the odds ratio of vaccination in cases vs controls using the formula , with the odds ratio calculated from a conditional logistic regression, stratified by site and adjusted for age group, the presence of chronic comorbidities, pregnancy and Indigenous ethnicity. The proportion of avoidable admissions was calculated using the formula where is the number of unvaccinated individuals and is the number of vaccinated individuals. The proportion of averted admission was calculated using the formula.

Results

During the period 1 April to 30 October 2015 (the 2015 influenza season), 2,070 patients were admitted with laboratory-confirmed influenza to one of 17 FluCAN sentinel hospitals. The peak weekly number of admission was in mid-August (week 33) (Figure). The majority of cases were due to influenza B (51%). The proportion due to influenza B varied by site from 49 of 136 cases (36%) at the Royal Hobart Hospital to 60 of 88 cases (68%) at the Princess Alexandra Hospital, Brisbane.

Of these 2,070 patients, 954 (46%) were more than 65 years of age, 320 (15%) were children (<16 years), 93 (4.5%) were Indigenous Australians, and 1,543 (75%) had chronic co-morbidities (Table 1; Table 2). There were 43 pregnant women, which represented 15% of the 277 female patients aged 16–49 years, or 2.1% of the total. Of the 1,686 patients (81%) where influenza vaccination status was ascertained, 805 (48%) had been vaccinated.

Figure: Date of admission in patients hospitalised with confirmed influenza



By week beginning on listed date; representing date of admission (or date of influenza diagnosis if acquired after more than 7 days in hospital).

Table 1: Demographic characteristics of hospitalised adult patients with confirmed influenza

	Influenza type/subtype									
	A/H1		A/H3		A/unknown		B		Total	
Number of cases	69		225		718		1,058		2,070	
	n	%	n	%	n	%	n	%	n	%
Age group										
<16 years	17	24.6	0	0.0	122	17.0	181	17.1	320	15.5
16-49 years	24	34.8	47	20.9	117	16.3	287	27.1	475	22.9
50-64 years	14	20.3	24	10.7	100	13.9	181	17.1	319	15.4
65-79 years	11	15.9	71	31.6	169	23.5	238	22.5	489	23.6
80+ years	3	4.3	83	36.9	210	29.2	171	16.2	467	22.6
Female*	32	46.4	129	57.3	364	50.8	520	49.1	1,045	50.5
Pregnant	3	4.3	8	3.6	8	1.1	24	2.3	43	2.1
Indigenous	1	1.4	5	2.2	34	4.7	53	5.0	93	4.5
State										
ACT	16	23.2	10	4.4	44	6.1	91	8.6	161	7.8
NSW	25	36.2	80	35.6	65	9.1	198	18.7	368	17.8
NT	0	0.0	0	0.0	21	2.9	22	2.1	43	2.1
Qld	5	7.2	20	8.9	74	10.3	153	14.5	252	12.2
SA	0	0.0	6	2.7	182	25.3	117	11.1	305	14.7
Tas.	5	7.2	67	29.8	15	2.1	49	4.6	136	6.6
Vic.	5	7.2	9	4.0	262	36.5	329	31.1	605	29.2
WA	13	18.8	33	14.7	55	7.7	99	9.4	200	9.7

* Sex missing for 1 patient.

Table 2: Risk factors, severity and outcomes in hospitalised adult patients with confirmed influenza

	Not admitted to ICU		Admitted to ICU		Total	
	n	%	n	%	n	%
Number of cases	1,916		154		2,070	
Pregnancy	41	2.1	2	1.3	43	2.1
Medical comorbidities	1,421	74.2	122	79.2	1,543	74.5
Chronic respiratory illness	625	32.6	58	37.7	683	33.0
Chronic cardiac disease	399	20.8	43	27.9	442	21.4
Diabetes	70	3.7	8	5.2	78	3.8
Chronic liver disease	323	16.9	28	18.2	351	17.0
Chronic neurological illness	233	12.2	22	14.3	255	12.3
Chronic renal disease	513	26.8	49	31.8	562	27.1
Immunocompromised	190	9.9	24	15.6	214	10.3
Malignancy	304	15.9	28	18.2	332	16.0
Obesity	255	13.3	22	14.3	277	13.4
Nursing home resident	149	7.8	3	1.9	152	7.3
Received influenza vaccine	760/1,564	48.6	46/123	37.4	806/1,687	47.8
Influenza type/subtype						
A/H1	60	3.1	9	5.8	69	3.3
A/H3	215	11.2	10	6.5	225	10.9
A/unknown	666	34.8	52	33.8	718	34.7
B	975	50.9	83	53.9	1,058	51.1
In hospital mortality	22	1.1	21	13.6	43	2.1

Presentation and management

For 1,546 patients with laboratory-confirmed influenza where the duration of symptoms was known, the median duration of symptoms prior to admission was 3 days (interquartile range (IQR): 2, 5 days). Of all cases, 102 cases (4.9%) were diagnosed more than 7 days after admission and therefore were likely to be hospital-acquired. Radiological evidence of pneumonia was present in 377 patients (18%).

Of all cases, 107 patients (5.2%) were initially admitted to ICU and a further 47 (2.3%) were subsequently transferred to ICU after initial admission to a general ward. There were no statistically significant differences in the risk of admission by influenza type. Factors associated with ICU admission are detailed in Table 3.

Outcome

The mean length of hospital stay for all patients was 5.0 days. Admission to ICU was associated with a mean hospital length of stay of 12.7 days compared with those not admitted to ICU (4.4 days). Of the 2,053 patients where hospital mortality status was documented, 43 patients died (2.1%), which included 21 patients in ICU. Case fatality was higher in the elderly (31/950; 3.3%) than in non-elderly adults (11/786; 1.4%). Of the 43 deaths, 40 (93%) occurred in patients with comorbidities. The case fatality of influenza-associated pneumonia was 5.9%.

Vaccine coverage and effectiveness

Vaccination status was ascertained in 1,687 of 2,070 cases (81%) and 1,293 of 1,636 test negative control patients (79%). Estimated vaccine coverage was 80.2% (478/596) in the elderly (≥ 65 years), 57.9% (219/378) in non-elderly adults with medical comorbidities and 26.9% (21/78) in children (< 16 years) with medical comorbidities. In the target population, the crude odds ratio of vaccination in cases vs controls was 0.57 (95% CI: 0.48, 0.69) and the adjusted odds ratio of vaccination was 0.54 (95% CI: 0.45, 0.66). The estimated vaccine effectiveness in the target population was therefore 45.3% (95% CI: 34.2%, 54.5%).

Avoidable and averted hospitalisations

Of the 2,070 admissions, 1,645 involved patients at risk of severe influenza. This included 956 elderly patients (of which 19.8% were estimated to be unvaccinated); 551 non-elderly adults with comor-

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

Variable	Crude odds ratio	*P*	Adjusted odds ratio*	*P*
Age				
<16 years	0.89 (0.56, 1.42)	0.615	0.75 (0.43, 1.33)	0.326
16–64 years	1 (referent)		1 (referent)	
65+ years	0.62 (0.43, 0.90)	0.011	0.59 (0.40, 0.88)	0.011
Medical comorbidities	1.33 (0.89, 1.99)	0.167	1.61 (1.05, 2.48)	0.03
Indigenous	1.18 (0.56, 2.48)	0.662	0.99 (0.46, 2.10)	0.975
Pregnancy	0.60 (0.14, 2.51)	0.486	0.48 (0.11, 2.05)	0.324
Restricted functional status	0.98 (0.70, 1.36)	0.894	0.76 (0.51, 1.12)	0.168
Nursing home resident	0.24 (0.07, 0.75)	0.014	0.23 (0.07, 0.76)	0.016
Influenza type/subtype				
A/H1	1.76 (0.84, 3.68)	0.131	1.68 (0.80, 3.54)	0.169
A/H3	0.55 (0.28, 1.07)	0.078	0.62 (0.31, 1.23)	0.169
B	1 (referent)		1 (referent)	
A/unknown	0.92 (0.64, 1.32)	0.638	0.95 (0.66, 1.37)	0.794

* All variables included in multivariate model.

bidities (42.1% unvaccinated) and; 138 children with comorbidities (72.1% unvaccinated). Based on the estimated vaccine effectiveness in this study of 45.3%, complete vaccination would result in 85 (8.9%) fewer admissions of elderly patients, 82 (18.9%) fewer admissions of non-elderly patients with comorbidities and 41 (32%) fewer paediatric admissions with confirmed influenza in participating sentinel hospitals. Conversely, the current vaccination program was estimated to have averted 49.5% of admissions in the elderly, 41.4% of admissions in non-elderly adults with comorbidities and 24.7% of admissions in children with comorbidities with confirmed influenza.

Discussion

In the 2015 season, we documented more than 2,000 cases of influenza, which represents a similar number of admissions to those in 2014 (n=2,097). This was the largest number of admissions documented since hospital-based surveillance commenced in 2009. Based on the bed capacity of sentinel hospitals, this is likely to represent around 17,000 admissions with confirmed influenza nationally. However, as influenza testing is not performed on all patients with acute respiratory presentations, and influenza may also trigger non-respiratory complications such as acute myocardial infarction, this should be regarded as a probable underestimate.

While the peak and duration of case counts, demographics and medical comorbidities were similar to cases in 2014, a striking difference in 2015 was the high proportion of admissions associated with influenza B. Influenza B is often thought to be associated with a milder illness than influenza A. Studies examining excess mortality associated with the influenza season suggest that mortality was lower in influenza B seasons compared with seasons where A/H3N2 predominated.[6–8] We found that the proportion of admissions associated with influenza B was similar to that described in primary care surveillance systems, suggesting that the risk of hospitalisation following infection is similar.

Previous studies have also noted a lower clinical severity of illness associated with influenza B, with pneumonitis or pneumonia being uncommonly reported.[9,10] In contrast, we found that the proportion of patients requiring intensive care admission was similar for those with influenza B compared with influenza A. A lower proportion of elderly patients were admitted to ICU than non-elderly adults, and this difference was not accounted for by the higher prevalence of medical comorbidities in the elderly. Of all female patients of childbearing age, 16% were pregnant. This was a similar proportion to that observed in previous seasons (20.9% of female patients aged 16–49 years who were pregnant during the 2010–14 seasons), and is likely to reflect the susceptibility of pregnant women to severe influenza.

No data were available on the influenza lineages associated with hospitalisation in this surveillance

system, but other surveillance systems have noted co-circulation of both Victoria and Yamagata lineages of influenza B, in all jurisdictions except the Northern Territory (dominated by B/Victoria lineages) and Western Australia (dominated by B/Yamagata lineages). As the trivalent influenza vaccine only contains one influenza B lineage, this may be associated with an attenuated vaccine effectiveness. The Australian Government has recently announced that influenza vaccines funded under the National Immunisation Program in 2016 will be quadrivalent. It remains to be seen if this change will be associated with a change in the proportion of admissions associated with influenza B infection.

We found that around half of the influenza cases were unvaccinated. Our estimates of vaccine coverage is similar to that of previous years, where around 70% to 80% of the elderly, around 60% of non-elderly adults with comorbidities and 30% of children with comorbidities were vaccinated.[11,12] We estimated that up to 209 admissions, or 14% of the admissions with confirmed influenza in the at-risk population at these 17 hospitals are potentially avoidable by improving influenza vaccination coverage in the target group. Conversely, this suggests that there would have more than 2,800 admissions involving patients at risk of influenza in the absence of vaccination and the proportion of admissions in the target group averted by vaccination was 46%. These figures are higher than estimated for the United States of America population, where influenza vaccination is actively promoted for the whole population, rather than specific risk groups.[13]

There are several limitations to this surveillance system. There may be under-ascertainment of influenza due to poor quality sample collection or the lack of use of influenza laboratory tests, despite the diagnosis of influenza having implications for infection control and antiviral use in hospitals. Delayed presentations or secondary bacterial pneumonia may be associated with false negative influenza tests as the influenza infection may be cleared by the time of presentation. Ascertainment in tropical regions is limited by sampling in the winter/dry season only. We have previously found that around 5% of influenza cases in hospital were acquired after admission,[14] and the incremental benefit of immunising close contacts (healthcare workers and household contacts) has not been considered.

In summary, we detected a large number of hospital admissions with laboratory-confirmed influenza in a national observational study in 2015 comparable with 2014 but much higher than in prior years. A high proportion of patients with severe influenza, and almost all deaths, occurred in patients with chronic comorbidities.

Acknowledgements

We thank Neela Joshi Rai, Ristilla Ram, Jo-Anne Thompson, Janette Taylor (Westmead Hospital), Cazz Finucane (Princess Margaret Hospital; Telethon Institute), Janine Roney, Jill Garlick, Virginia Cable, Nigel Pratt (The Alfred Hospital), Kristof Boot, Megan Martin (Mater Hospital), Michelle Thompson, Casey McLeod, Adam Kadmon (Royal Melbourne Hospital), Jocelynne McRae, Laura Rost, Natalie McLaren, Sharon Tan (Children's Hospital at Westmead), Kathryn Ellis, Sammi Xui, Wendy Beckingham, Sandra Root (Canberra and Calvary Hospitals), Stella Green, Sue Richmond (Cairns Base Hospital), Irene O'Meara, Ingrid Potgeiter (Alice Springs Hospital; Menzies School of Health Research), Tina Collins, Michelle Towers (Princess Alexandria Hospital), Susan Wagg (Royal Hobart Hospital), Kate Ellis (University Hospital Geelong, Barwon Health), Doug Dorahy, Lorissa Hopkins (John Hunter Hospital), Jenny McGrath, Louise Milazzo, Sarah Richards, Cathy Short, Cate Green, Mary McAlister, Eve Boxhall, Belinda Hua, Ashleigh Richardson, Catriona Doran (Royal Adelaide Hospital), Ellen MacDonald, Sophie Damianopoulos, Fiona Seroney (Royal Perth Hospital). We acknowledge the support of the Australian Government Department of Health for funding this system.

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Australian Rotavirus Surveillance Program annual report, 2015

Susie Roczo-Farkas, Carl D Kirkwood, Julie E Bines and the Australian Rotavirus Surveillance Group

Abstract

The Australian Rotavirus Surveillance Program, together with collaborating laboratories Australia-wide, reports the rotavirus genotypes responsible for the hospitalisation of children with acute gastroenteritis during the period 1 January to 31 December 2015. During the survey period, 1,383 faecal samples were referred for rotavirus G and P genotype analysis, and of these, 1,031 were confirmed as rotavirus positive. A total of 634 specimens had been collected from children under 5 years of age, while 397 were from older children and adults. Genotype analysis of samples from both children and adults revealed that G12P[8] was the dominant genotype in this reporting period, identified in 48.2% of strains nationally. Genotype G3P[8] was the second most common strain nationally, representing 22.8% of samples, followed by G2P[4] and G1P[8] (9% and 8% respectively). G3P[8] was further divided as equine-like G3P[8] (13.2% of all strains) and other wild-type G3P[8] (9.6%). This report highlights the continued predominance of G12P[8] strains as the major cause of disease in this population. Genotype distribution was distinct between jurisdictions using RotaTeq and Rotarix vaccines. Genotype G12P[8] was more common in states using RotaTeq, while equine-like G3P[8] and G2P[4] were more common in the states and territories using Rotarix. This survey highlights the dynamic change in rotavirus genotypes observed since vaccine introduction, including the emergence of a novel equine-like G3P[8] as a major strain. The prolonged dominance of G12P[8] for a 4th consecutive year further illustrates the unexpected trends in the wild type rotaviruses circulating in the Australian population since vaccine introduction. *Commun Dis Intell* 2016;40(4):E527–E538.

Keywords: rotavirus, gastroenteritis, genotypes, disease surveillance

Introduction

Rotaviruses belong to the Reoviridae family and are triple layered dsRNA viruses that contain a segmented genome, consisting of 11 gene segments that encode 6 structural proteins and 6 non-structural proteins.[1] Rotaviruses are the most common cause of severe diarrhoea in young children worldwide, and are estimated to cause up to 453,000 deaths annually.[2] The significant morbidity and mortality associated with rotavirus infection has led to the development of vaccines, such as Rotarix® (GlaxoSmithKline) and RotaTeq® (Merck). These 2 oral live attenuated rotavirus vaccines have been shown to be safe and highly effective in the prevention of severe diarrhoea due to rotavirus infection,[3,4] leading to both vaccines being licensed in over 125 countries and included in the national vaccination schedules of 63 predominantly high and middle-income countries worldwide.[5] In Australia, rotavirus vaccines have been included in the National Immunisation Program since 1 July 2007, with excellent uptake in subsequent years. RotaTeq is administered in Queensland, South Australia, Victoria and Western Australia, while Rotarix is administered in the Australian Capital Territory, New South Wales, the Northern Territory and Tasmania.[6]

In Australia prior to vaccination being introduced, rotavirus infection had accounted for up to 10,000 childhood hospitalisations for diarrhoea each year.[7] A significant impact on the disease burden has been observed since vaccine introduction, with studies showing a substantial decline in both rotavirus coded and non-rotavirus coded hospitalisation and emergency room visits.[6,8–12]

The Australian Rotavirus Surveillance Program has characterised the G- and P- genotypes of rotavirus strains causing severe disease in Australian children since 1997. Data from this surveillance has shown that strain diversity as well as temporal and geographic changes occur each year; providing critical baseline data.[13] Ongoing characterisation of circulating rotavirus genotypes in the vaccine era will provide insight into whether vaccine introduction has impacted on virus epidemiology, and altered circulating strains, which could have ongoing consequences for the success of current and future vaccination programs.

This report describes the genotype distribution of rotavirus strains causing severe gastroenteritis in Australia for the period 1 January to 31 December 2015.

Methods

Rotavirus positive specimens detected by enzyme immunoassay (EIA) or latex agglutination in collaborating laboratories across Australia were

collected, stored frozen and forwarded to the Australian Rotavirus Reference Centre Melbourne, together with relevant age and sex details. The laboratories contributing samples were:

- ACT Pathology, Canberra, Australian Capital Territory
- The Virology Division, South Eastern Area Laboratory Services, Prince of Wales Hospital, New South Wales
- Virology Department, The Children's Hospital at Westmead, New South Wales
- Centre for Infectious Diseases and Microbiology, Westmead, New South Wales
- The Microbiology Department, John Hunter Hospital, Newcastle, New South Wales
- The Microbiology Department, Royal Darwin Hospital, Casuarina, Northern Territory
- The Microbiology Department, Alice Springs Hospital, Alice Springs, Northern Territory
- Forensic and Scientific Services, Queensland Health, Herston, Queensland
- Microbiology division, Pathology Queensland, Herston, Queensland
- The Queensland Paediatric Infectious Diseases laboratory, Royal Children's Hospital, Brisbane, Queensland
- Queensland Health laboratory in Townsville, Queensland
- Microbiology and Infectious Diseases Laboratory, SA Pathology, Adelaide, South Australia
- The Serology Department, Royal Children's Hospital, Parkville, Victoria
- Department of Microbiology, Monash Medical Centre, Clayton, Victoria
- Division of Microbiology, PathWest LM, The Queen Elizabeth Medical Centre, Nedlands, Western Australia.

Viral RNA was extracted from 10% to 20% faecal extracts using the QIAamp Viral RNA mini extraction kit (Qiagen) according to the manufacturer's instructions. Rotavirus G and P genotypes were determined using an in-house hemi-nested multiplex reverse transcription polymerase chain reaction (RT-PCR) assay. The first round RT-PCR reactions were performed using the Qiagen one step RT-PCR kit, using VP7 conserved primers VP7F and VP7R, or VP4 conserved primers VP4F and VP4R. The second round genotyping PCR reactions were conducted using specific oligonucleotide primers for G types 1, 2, 3, 4, 8, 9 and 12 or P types [4], [6], [8], [9], [10] and [11].[14–19] The G and P genotype of each sample was assigned using agarose gel analysis of second round PCR products.

First round amplicons for VP7 were also purified for sequencing by using a Wizard SV Gel for PCR Clean-Up System (Promega), according to the manufacturer's protocol. Purified DNA together with oligonucleotide primers (VP7F/R) were sent to the Australian Genome Research Facility, Melbourne, and sequenced using an ABI PRISM BigDye Terminator Cycle Sequencing Reaction Kit (Applied Biosystems, Foster City, CA, USA) in an Applied Biosystems 3730xl DNA Analyzer (Applied Biosystems, Foster City, CA, USA). Sequences were edited with Sequencher v.4.10.1. The genotype assignment was accomplished using BLAST (http://blast.ncbi.nlm.nih.gov/Blast.cgi) and RotaC v2.0 (http://rotac.regatools.be).[20]

Any samples that provided a discordant result between the initial antigen detection and genotype assay were further tested using the commercial rotavirus ELISA assay ProSpecT (Thermo Fisher, Aus.), as per manufacturer's instructions to confirm the presence of rotavirus antigen.

Results

Number of isolates

A total of 1,383 faecal specimens were collected during the period 1 January to 31 December 2015 for analysis from 15 collaborating centres across Australia. For this reporting period, no samples were received from Tasmania.

A total of 1,031 samples were confirmed as rotavirus positive. Of these, 634 had been collected from children under 5 years of age, and 397 were from older children and adults. An additional 352 specimens contained either insufficient specimen for genotyping (n= 7), were duplicates of samples already analysed (n=77) or the specimen was not confirmed to be positive for rotavirus (n= 268) and were thus not analysed further.

Age distribution

During the 2015 reporting period, 61.5% of samples were obtained from children under 5 years of age (Table 1). Of these, one-third of all samples (33.3%) were identified in children aged 13–24 months, while the next most common age group was 25–36 months (19.7%).

Genotype distribution

All of the 1,031 confirmed rotavirus samples collected from children and adults underwent

Table 1: Age distribution of gastroenteritis cases

Age range (months)	Age range (years)	Number	% of total	% under 5 years
0–6		120	11.6	18.9
7–12	≤1	82	8.0	12.9
13–24	1–2	211	20.5	33.3
25–36	2–3	125	12.1	19.7
37–48	3–4	53	5.1	8.4
49–60	4–5	43	4.2	6.8
Sub-total		634	61.5	–
61–120	5–10	139	13.5	
121–240	10–20	48	4.7	
241–960	20–80	165	16.0	
961+	>80	45	4.4	
Total		1,031	–	

genotype analysis (Table 2). G12P[8] strains were the most common genotype identified nationally, representing 48.2% of all specimens analysed. This genotype was identified as the dominant type in 4 states, Queensland, Victoria, Western Australia and South Australia, representing 53.8%, 80.2%, 65.4% and 50.8% of strains respectively.

G3P[8] strains were the second most common genotype identified nationally, representing 22.8% of all specimens. These were further divided as equine-like G3P[8] (13.2%) and other wild-type G3P[8] (9.6%). Equine-like G3P[8] were identified in all 7 states or territories that submitted samples, and was the dominant type in the Australian Capital Territory and New South Wales, where it represented 96% and 67.2% respectively. In the Northern Territory, equine-like G3P[8] was the second most common type after G2P[4], representing 36.4% and 48.6% of strains respectively. No other wild-type G3P[8] strains were detected in Rotarix states.

G2P[4] and G1P[8] strains were the third and fourth most common genotypes nationally, representing 8.6% and 8.1% of all specimens respectively. G1P[8] was identified in all locations except the Australian Capital Territory, and was the third most common strain in Queensland and South Australia, representing 11.3% and 13.2% of strains respectively.

Thirty samples (3% of all strains) were categorised into 9 uncommon G- and P- genotype combinations (Table 2). Six G3P[3], 5 G8P[14], 4 G9P[4], and 4 G12P[6] strains were identified. Three G1P[4] strains were also noted, as were 3 G8P[8] strains. A further 2 G8P[14], 2 G12P[4] and a single G10P[14] strain were identified.

A G- or P- genotype could not be assigned to 35 samples, of which 23 (65.7%) were observed in Western Australia. Of the 35 samples, 31 were G- non-typeable, 2 were P- non-typeable and 2 had no G- or P- genotype assigned, although they were positive by EIA. The partially non-typeable samples could be strains that contain unusual or uncommon G- or P- genotypes and would not be typeable with the primers used. While this can also be suggested for the EIA positive/G- and P- non-typeable samples, another possible explanation is that the extracted RNA for these samples contained inhibitors that could have prevented the function of the enzymes used in the RT and/or PCR steps.

Thirty-eight samples were identified that contained a strain that was a component of the RotaTeq or Rotarix vaccine. Such Rotarix strains were found in New South Wales (n=1), and the Northern Territory (n=2), while RotaTeq strains were identified in South Australia (n=5), Victoria (n=1) and Western Australia (n=29). In each instance, a vaccine component was determined by RT-PCR and confirmed by sequence analysis of the VP6 and VP7 gene.

Genotypes identified in samples from children less than 5 years of age

In total, 634 rotavirus samples were collected from children under 5 years of age (Table 3). In this cohort, genotype G12P[8] strains were the most commonly identified, found in 41.6% of samples. G3P[8] was the second most common genotype (23.5%), comprising of 14.8% equine-like G3P[8] and 8.7% other wild-type G3P[8] (Figure 1). G2P[4] strains were the third most common genotype (10.1%). G1P[8] and G9P[8] strains represented minor genotypes in this cohort, and were identified in 8.8% and 2.2% of samples respectively (Table 3).

Figure 1: Wild-type G3P[8] occurrences in infants and children under 5 years of age

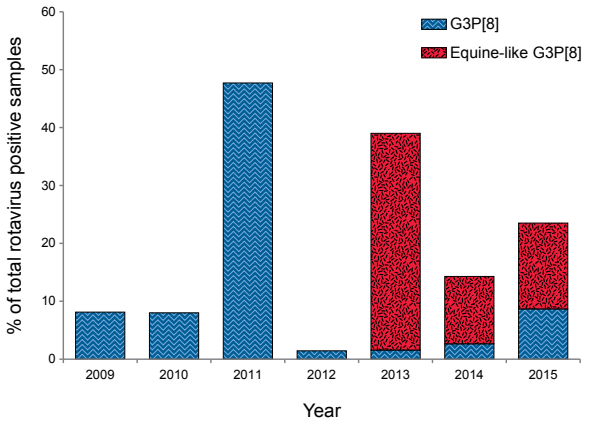


Table 2: Rotavirus G and P genotype distribution in infants, children and adults, 1 January to 31 December 2015

Centre	Type total	G1P[8]		G2P[4]		G3P[8]		G3P[8]*		G4P[8]		G9P[8]		G12P[8]		Non-type†		Vaccine‡		Other		Neg	Insuff
		%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	n	n
Australian Capital Territory																							
ACT	25	–	0	–	0	–	0	96	24	–	0	–	0	–	0	–	0	–	0	4	1	1	0
New South Wales																							
POW	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	0	0
Westmead	35	3	1	9	3	–	0	71	25	–	0	3	1	6	2	3	1	–	0	6	2	2	0
John Hunter	20	10	2	–	0	–	0	60	12	–	0	15	3	5	1	–	0	5	1	5	1	6	0
NSW subtotal:	55	5	3	5	3	–	0	67	37	–	0	7	4	5	3	2	1	2	1	5	3	8	0
Northern Territory																							
Alice Springs	15	–	0	93	14	–	0	–	0	–	0	–	0	–	0	–	0	–	0	7	1	7	0
Darwin	48	–	0	35	17	–	0	54	26	–	0	10	5	–	0	–	0	–	0	–	0	3	0
Western Diagnostic	12	–	0	–	0	–	0	100	12	–	0	–	0	–	0	–	0	–	0	–	0	0	0
Other§	31	3	1	68	21	3	1	–	0	–	0	–	0	–	0	6	2	6	2	13	4	10	2
Northern Territory subtotal:	106	1	1	49	52	1	1	36	38	–	0	5	5	–	0	2	2	2	2	5	5	20	2
Queensland																							
Pathology Brisbane	61	10	6	11	7	7	4	15	9	–	0	5	3	46	28	3	2	–	0	3	2	9	0
Qld Regional	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	3	0
Pathology Townsville	19	16	3	–	0	–	0	–	0	–	0	5	1	79	15	–	0	–	0	–	0	3	0
Queensland subtotal:	80	11	9	9	7	5	4	11	9	–	0	5	4	54	43	3	2	–	0	3	2	15	0
South Australia																							
Adelaide	418	13	55	5	22	21	89	3	12	<1	1	<1	1	51	212	2	7	1	5	3	14	100	5
Victoria																							
RCH	80	6	5	–	0	–	0	9	7	–	0	1	1	80	64	–	0	1	1	3	2	30	0
Monash	1	–	0	–	0	–	0	–	0	–	0	–	0	100	1	–	0	–	0	–	0	0	0
Victoria subtotal:	81	6	5	–	0	–	0	9	7	–	0	1	1	80	65	–	0	1	1	2	2	30	0

Table 2 *continued*: Rotavirus G and P genotype distribution in infants, children and adults, 1 January to 31 December 2015

Centre	Type total	G1P[8] %	G1P[8] n	G2P[4] %	G2P[4] n	G3P[8] %	G3P[8] n	G3P[8]* %	G3P[8]* n	G4P[8] %	G4P[8] n	G9P[8] %	G9P[8] n	G12P[8] %	G12P[8] n	Non-type† %	Non-type† n	Vaccine‡ %	Vaccine‡ n	Other %	Other n	Neg n	Insuff n
Western Australia																							
PathWest	266	4	10	2	5	2	5	3	9	–	0	3	8	65	174	9	23	11	29	1	3	92	0
Total	1031	8	83	9	89	10	99	13	136	<1	1	2	23	48	497	3	35	4	38	3	30	266	7

* Equine-like G3P[8]

† A specimen where G and/or P genotype was not determined.

‡ A specimen where a vaccine component from RotaTeq or Rotarix was found.

§ Faecal specimens from the Northern Territory which were processed in Adelaide.

Neg = Negative for rotavirus

Insuff = Insufficient sample for testing

POW Prince of Wales Hospital

RCH Royal Children's Hospital

Non-typeable† samples:

Northern Territory: 1x G-nt P[8]; 1x G-nt P[4]

New South Wales: 1x G-nt P[8]

Queensland: 1x G-nt P[8]; 1x G12P[nt]

South Australia: 4x G-nt P[8]; 1x G-nt P[6]; 1x G4P[nt] and 1x nt (EIA positive)

Western Australia: 22x G-nt P[8]; 1x nt (EIA positive)

Other samples:

Australian Capital Territory 1x G12P[6]

New South Wales 1x G8P[8], 1x G8P[14], 1x G9P[14]

Northern Territory 1x G1P[4], 4x G3P[3]

Queensland 1x G8P[8], 1x G8P[14]

South Australia 2x G1P[4], 1x G3P[3], 2x G8P[4], 2x G8P[14], 2x G9P[4], 1x G10P[14], 2x G12P[14], 2x G12P[6]

Victoria 1x G9P[4], 1x G12P[6]

Western Australia 1x G3P[3], 1x G8P[8], 1x G8P[14]

Table 3: Rotavirus G and P genotype distribution in infants and children under 5 years of age, 1 January to 31 December 2015

Centre	Type total	G1P[8] %	G1P[8] n	G2P[4] %	G2P[4] n	G3P[8] %	G3P[8] n	G3P[8]* %	G3P[8]* n	G4P[8] %	G4P[8] n	G9P[8] %	G9P[8] n	G12P[8] %	G12P[8] n	Non-type† %	Non-type† n	Vaccine‡ %	Vaccine‡ n	Other %	Other n	Neg n	Insuff n
Australian Capital Territory																							
ACT	14	–	0	–	0	–	0	93	13	–	0	–	0	–	0	–	0	–	0	7	1	1	0
New South Wales																							
POW	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	0	0
Westmead	23	4	1	4	1	–	0	78	18	–	0	4	1	–	0	4	1	–	0	4	1	0	0
John Hunter	10	–	0	–	0	–	0	60	6	–	0	20	2	–	0	–	0	10	1	10	1	1	0
NSW subtotal:	33	3	1	3	1	–	0	73	24	–	0	9	3	–	0	3	1	3	1	6	2	1	0
Northern Territory																							
Alice Springs	14	–	0	93	13	–	0	–	0	–	0	–	0	–	0	–	0	–	0	7	1	7	0
Darwin	44	–	0	34	15	–	0	55	24	–	0	11	5	–	0	–	0	–	0	–	0	3	0
Western Diagnostic	12	–	0	–	0	–	0	100	12	–	0	–	0	–	0	–	0	–	0	–	0	0	0
Other§	27	4	1	64	18	–	0	–	0	–	0	–	0	–	0	7	2	7	2	15	4	7	2
Northern Territory subtotal:	97	1	1	47	46	–	0	37	36	–	0	5	5	–	0	2	2	2	2	5	5	17	2
Queensland																							
Pathology Brisbane	24	17	4	4	1	4	1	17	4	–	0	–	0	54	13	4	1	–	0	–	0	7	0
Qld Regional	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	3	0
Pathology Townsville	8	13	1	–	0	–	0	–	0	–	0	13	1	75	6	–	0	–	0	–	0	2	0
Queensland subtotal:	32	16	5	3	1	3	1	13	4	0	0	3	1	59	19	3	1	–	0	–	0	12	0
South Australia																							
Adelaide	238	15	36	6	15	22	52	3	8	<1	1	<1	1	45	107	2	4	2	5	4	9	39	3
Victoria																							
RCH	55	7	4	–	0	–	0	7	4	–	0	2	1	78	43	–	0	2	1	4	2	17	0
Monash	1	–	0	–	0	–	0	–	0	–	0	–	0	100	1	–	0	–	0	–	0	0	0
Victoria subtotal:	56	7	4	–	0	–	0	7	4	–	0	2	1	79	44	0	0	2	1	4	2	17	0
Western Australia																							
PathWest	164	5	9	1	1	1	2	3	5	–	0	2	3	57	94	12	19	18	29	1	2	55	0
Total	634	9	56	10	64	9	55	15	94	<1	1	2	14	42	264	4	27	6	38	3	21	142	5

* Equine-like G3P[8].

† A specimen where G and/or P genotype was not determined.

‡ A specimen where a vaccine component from RotaTeq or Rotarix was found.

§ Faecal specimens from the Northern Territory which were processed in Adelaide.

Neg Negative for rotavirus

Insuff Insufficient sample for testing

POW Prince of Wales Hospital

RCH Royal Children's Hospital

Other samples:

Australian Capital Territory	1x G12P[6]
New South Wales	1x G8P[14], 1x G9P[4]
Northern Territory	1x G1P[4], 4x G3P[3]
South Australia	1x G1P[4], 1x G3P[3], 2x G8P[4], 2x G9P[4], 1x G10P[14], 2x G12P[4]
Victoria	1x G9P[4], 1x G12P[6]
Western Australia	1x G3P[3], 1x G8P[14]

Genotypes identified in samples from individuals greater than 5 years of age

A total of 397 rotavirus samples were collected from children over the age of 5 years, and adults (Table 4). In this cohort, genotype G12P[8] strains were the most common, found in 58.4% of samples. G3P[8] strains (including equine-like G3P[8]) were the second most common genotype (21.7%), and G1P[8] and G2P[4] strains were equal third most common genotypes, identified in 6.8% and 6.3% respectively. Genotype G9P[8] strains were identified in 2.3% of samples.

Distribution of genotypes according to vaccine type

G and P genotypes from the rotavirus samples collected from infants and children under 5 years of age by vaccine usage were compared (Figure 2). In states where RotaTeq is in use, G12P[8] strains were the dominant genotype in children less than 5 years of age, identified in 54% of samples, while G3P[8] were the second most common, identified in 15.5% of strains. These G3 were further divided as equine-like G3P[8] (4.3%) or other wild type G3P[8] (11.2%). G1P[8] strains were the third most common genotype representing 11% of samples. In locations using Rotarix, equine-like G3P[8] strains were dominant, identified in 50.3% of strains, followed by G2P[4], identified in 32.4% of samples.

Consistency in genotype distribution among states using RotaTeq vaccine was observed, with all 4 RotaTeq states (Queensland, Victoria, South Australia and Western Australia) having G12P[8] as the dominant genotype. However, in states and territories using Rotarix (New South Wales, the Australian Capital Territory and the Northern Territory), the dominant genotype differed, with G2P[4] dominant in Northern Territory and equine-like G3P[8] dominant in New South Wales and the Australian Capital Territory.

Discussion

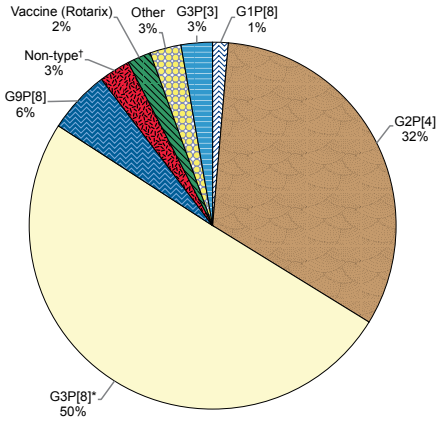
This 2015 Australian Rotavirus Surveillance report describes the annual distribution of rotavirus genotypes and geographic differences in genotypes causing disease in Australia for the period 1 January to 31 December 2015. Genotype G12P[8] remained the dominant genotype nationally, representing 48.2% of all strains from all age groups. Genotype G3P[8] was the second most common genotype nationally, comprising 23% of all strains, but was the dominant genotype in New South Wales and the Australian Capital Territory only. Genotype G2P[4] and G1P[8] were the third and fourth most common, representing 8.6% and 8.1% of all strains respectively.

In the samples collected from infants and children under 5 years of age, genotype G12P[8] continued to remain as the dominant genotype nationally, identified in 41.6% of samples. The second most common genotype was G3P[8], identified in 23.5% of samples, while the third most common was G2P[4], identified in 10% of samples.

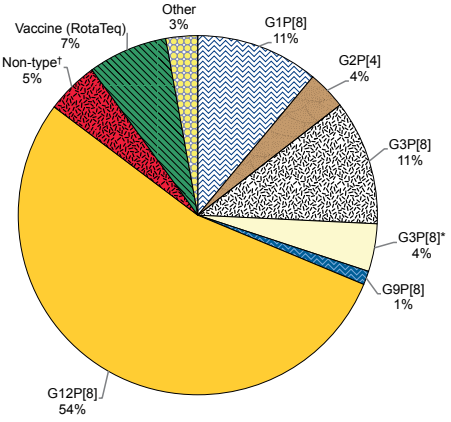
In Australia, G12P[8] emerged in 2012 and subsequently became the dominant strain in 2013 and 2014, representing 33% and 29.6% of all strains respectively.[19,22,23] This figure increased in 2015 to encompass 48.2% of all strains, and has been identified as the dominant strain in all 4 states that use the RotaTeq vaccine (South Australia, Queensland, Victoria and Western Australia). Similar observations have been observed in

Figure 2: Overall distribution of rotavirus G and P genotypes identified in Australian children based on vaccine usage, Australia, 1 January to 31 December 2015

Rotarix states†



RotaTeq states‡



* Equine-like G3P[8][21]

† The Australian Capital Territory, New South Wales, and the Northern Territory

‡ Queensland, South Australia, Victoria, and Western Australia

Table 4: Rotavirus G and P genotype distribution in children over 5 years of age and adults, 1 January to 31 December 2015

Centre	Type total	G1P[4] %	G1P[4] n	G1P[8] %	G1P[8] n	G2P[4] %	G2P[4] n	G3P[8] %	G3P[8] n	G3P[8]* %	G3P[8]* n	G8P[8] %	G8P[8] n	G8P[14] %	G8P[14] n	G9P[8] %	G9P[8] n	G12P[6] %	G12P[6] n	G12P[8] %	G12P[8] n	Non-type† %	Non-type† n	Neg n	Insuff n
Australian Capital Territory																									
ACT	11	–	0	–	0	–	0	–	0	100	11	–	0	–	0	–	0	–	0	–	0	–	0	0	0
New South Wales																									
POW	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	0	0
Westmead	12	–	0	–	0	17	2	–	0	58	7	8	1	–	0	–	0	–	0	17	2	–	0	2	0
John Hunter	10	–	0	20	2	–	0	–	0	60	6	–	0	–	0	10	1	–	0	10	1	–	0	5	0
NSW subtotal	22	–	0	9	2	9	2	–	0	59	13	5	1	–	0	4	1	–	0	14	3	–	0	–	0
Northern Territory																									
Alice Springs	1	–	0	–	0	100	1	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	0	0
Darwin	4	–	0	–	0	50	2	–	0	50	2	–	0	–	0	–	0	–	0	–	0	–	0	0	0
Western Diagnostic	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	0	0
Other‡	4	–	0	–	0	75	3	25	1	–	0	–	0	–	0	–	0	–	0	–	0	–	0	3	0
Northern Territory subtotal	9	–	0	–	0	67	6	11	1	22	2	–	0	–	0	–	0	–	0	–	0	–	0	3	0
Queensland																									
Pathology Brisbane	37	–	0	5	2	16	6	8	3	14	5	3	1	3	1	8	3	–	0	41	15	3	1	2	0
Qld regional	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	0	0
Pathology Townsville	11	–	0	18	2	–	0	–	0	–	0	–	0	–	0	–	0	–	0	73	8	9	1	1	0
Queensland subtotal:	48	–	0	8	4	13	6	6	3	10	5	2	1	2	1	6	3	–	0	48	23	4	2	3	0
South Australia																									
Adelaide	180	1	1	11	19	4	7	21	37	2	4	–	0	1	2	–	0	1	2	58	105	2	3	61	2
Victoria																									
RCH	25	–	0	4	1	–	0	–	0	12	3	–	0	–	0	–	0	–	0	84	21	–	0	13	0
Monash	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	–	0	0	0
Victoria subtotal:	25	–	0	4	1	–	0	–	0	12	3	–	0	–	0	–	0	–	0	84	21	–	0	13	0
Western Australia																									
PathWest	102	–	0	1	1	4	4	3	3	4	4	1	1	–	0	5	5	–	0	78	80	4	4	37	0
Total	397	<1	1	7	27	6	25	11	44	11	42	1	3	1	3	2	9	1	2	58	232	2	9	124	2

* Equine-like G3P[8]

† A specimen where G and/or P genotype was not determined.

‡ Faecal specimens from the Northern Territory which were processed in Adelaide.

Neg Negative for rotavirus

Insuff Insufficient sample for testing

POW Prince of Wales Hospital

RCH Royal Children's Hospital.

Nicaragua, where RotaTeq was added to the infant schedule in 2006; more than a 2-fold increase in rotavirus cases in children under 5 years of age was attributed to a G12 outbreak in the 2012/2013 season.[24] Since 2000, over 30 countries have reported an emergence or re-emergence of G12 rotavirus strains.[25–27] Furthermore, G12P[8] have been identified as the dominant strain in multiple countries since 2010.[24,28–31] During the 2010-2011 season, 65% of all strains in Basque Country, Spain were identified as G12P[8].[31] At this time, vaccine coverage was estimated to be below 30%, even though both vaccines were available in Basque Country since 2006–2007.[31] In 2013, G12P[8] represented 82% of all strains in Nicaragua; 86% in St Louis, Missouri and 89% in Atlanta, Georgia.[24,29,30] In addition, G12P[8] emerged as a dominant strain in Brazil, from 22.7%–27.3% in 2012 to 86.6% in 2014, although G12 had decreased to represent only 2.7% of all strains in 2013.[28,32,33]

Whilst not yet dominant, G12 has been identified as an important genotype in countries such as Italy, Saudi Arabia, Cameroon, and North and South America.[34–38] In Delhi, India, a study investigating strain diversity of samples collected from 2007–2012, established that G12 represented 14.8% of all genotypes identified. The majority of G12 strains found in the study had VP4 genotype P[6] (67.4%), while P[8] represented only 7% of all G12 cases. Another combination, G12P[4], was identified and represented 14% of all G12 strains identified.[39] G12P[4] and G12P[6] have also been identified in Australia in 2013 and 2015, however they were uncommon and represented less than 1% for each surveillance period. Taken together, these data demonstrate that G12 strains are an important cause of rotavirus disease both in Australian and global settings.

This survey reports the persistence of G3P[8] strains as the second most common genotype across Australia. Previously, G3P[8] strains generally represented 4% to 11% of strains over a 1-year reporting period. However, in 2013, G3P[8] emerged as the second most common genotype, representing 31% of all strains.[19] Full genome analysis of these G3P[8] samples revealed that in 2013, a novel inter-genogroup reassortant strain (denoted as equine-like G3P[8], or G3P[8]* in Figure 2) had emerged, which had a distinct VP7 antigenic profile compared to other wild-type Australian G3P[8] strains.[21] The genome constellation of equine-like G3P[8] contained an equine-like G3 VP7, a P[8] VP4 and a genogroup 2 backbone I2-R2-C2-M2-A2-N2-T2-E2-H2.[21] This strain continued to persist, representing 37% of all strains in 2013, 12% in 2014 and 15% of 2015 samples from infants and children under 5 years of age (Figure 1).

The state and territory based tender process has resulted in the use of either RotaTeq or Rotarix vaccines in states and territories in Australia and provides a unique opportunity to observe and compare rotavirus strain diversity. Since vaccine introduction, significant differences in genotype distribution in infants and children under 5 years of age have been observed between jurisdictions using each vaccine.[40] In the current survey, equine-like G3P[8] was dominant in locations using Rotarix (50.3%), although no other wild-type G3P[8] strains were identified; whereas in RotaTeq states, other wild-type G3P[8] were more common (11.25%) compared with equine-like G3P[8] (4.3%).

The emergence of G12P[8] and novel genotypes globally have raised concerns regarding vaccine efficacy; however a review of vaccine trials and surveillance studies up to 2012 demonstrated that in high income countries such as Belgium and the United States of America, the monovalent Rotarix (G1P[8]) and pentavalent RotaTeq (G1-4, P[8]) vaccines continue to be highly efficacious against commonly circulating rotavirus strains.[41] The pooled effectiveness of Rotarix was 94% against homotypic strains, to 87% against fully heterotypic strains.[41] RotaTeq exhibited similar effectiveness; from 83% against homotypic strains to 75% against single-antigen non-vaccine type strains.[41] Nevertheless, a recent study from Belgium reported that multiple deduced amino acid differences existed between the VP7 and VP4 antigenic epitopes of vaccine and currently circulating strains, such as G12 and G9.[42] The VP7 proteins of G12P[8] strains alone contained 9 amino acid differences compared with the strains of RotaTeq, and 16 amino acid differences to Rotarix strain.[42] These differences, together with the high evolutionary rate of the G12 VP7 gene (1.66×10^{-3} substitutions/site/year), raise the concern that this strain could ultimately escape the rotavirus neutralising antibody response induced by vaccines.[27,42] Vaccine effectiveness studies are required to determine if current vaccines are able to protect successfully against novel strains, such as the equine-like G3P[8] and G12P[8].

The continued dominance of G12P[8] over a 4-year period in states using RotaTeq support the concept hypothesis that vaccine-related selective pressure may be arising. Vaccine-related selective pressure have also been implicated in the persistence of G2P[4] in countries such as Belgium, where Rotarix provides high vaccine coverage.[43,44] This observation is of particular interest, as the 2015 Australian data described in this report found that in infants and children under 5 years of age, there was a marked increase in G2P[4] cases to 32% in jurisdictions using Rotarix, reflecting a trend also observed in 2012.[45]

In this reporting period, over 1,000 rotavirus positive samples were collected from across Australia. Caution in interpreting the data is recommended, as the increase in the number of samples received may not necessarily reflect the number of cases being greater, since not all samples are forwarded by collaborating hospitals and diagnostic laboratories. Regardless, a robust increase in the number of samples sent from South Australia, Western Australia and Victoria have been noted, particularly in 2013 and 2015 in which the majority of samples from these states were genotyped as G12P[8]. The emergence of G12 and novel strains such as the equine-like G3P[8], G3P[3], G10P[14] and G8 strains demonstrate a dynamic virus population in the current post vaccine era. Therefore, the continued variations in the wild type strain population will remain a challenge to vaccine effectiveness and will require continued monitoring.

Acknowledgements

The Rotavirus Surveillance Program is supported by grants from the Australian Government Department of Health, GlaxoSmithKline and CSL. Dr CD Kirkwood is supported by a NHMRC senior research fellowship. The Murdoch Childrens Research Institute (MCRI) is supported by the Victorian Government's Operational Infrastructure Support program.

We thank H Tran and S Thomas for providing technical assistance.

Rotavirus positive specimens were collected from numerous centres throughout Australia. The significant time and effort involved in the collection, storage, packaging, compiling data and forwarding of specimens was much appreciated.

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Quarterly report

OzFoodNet quarterly report, 1 July to 30 September 2014

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health established the OzFoodNet network in 2000 to nationally collaborate the investigation of foodborne disease. In each Australian state and territory, OzFoodNet epidemiologists investigate outbreaks of enteric infection. In addition, OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of foodborne or suspected foodborne disease outbreaks, as well as clusters of disease potentially related to food. These investigations commenced in Australia between 1 July and 30 September 2014.

Data were received from OzFoodNet epidemiologists in all Australian states and territories. The data in this report are provisional and subject to change.

During the 3rd quarter of 2014, OzFoodNet sites reported 343 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health authorities or reporting may be delayed, which results in current figures under-representing the true burden of enteric disease outbreaks within Australia. In total, these outbreaks affected 6,204 people, of whom 123 were hospitalised, and 28 deaths were reported during this quarter. This represents a decrease in both the number affected and the number of deaths, compared with the 5-year average for the 3rd quarter (11,581 affected, 295 hospitalised and 41 deaths) between 2009 and 2013. The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission and in this quarter 71% (n=242) were transmitted via this route (Table 1). This was lower by number but the same proportion as the same quarter in 2013 (71%, n=305) and was lower than the 5-year mean for the 3rd quarter for 2009 to 2013 (80%, n=481). Furthermore, of the reported person-to-person outbreaks this quarter, 57% (194 outbreaks) occurred in aged care facilities and 21% (72 outbreaks) occurred in child care facilities.

Foodborne and suspected foodborne disease outbreaks

There were 25 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission (Appendix). These outbreaks affected 253 people and resulted in 23 hospitalisations. No deaths were reported as a result of these outbreaks. This was a decrease from the number of foodborne outbreaks that were reported in the 2nd quarter of 2014 (n=39) and lower than the 5-year mean for the 3rd quarter between 2009 and 2013 (n=31).

Table 1: Outbreaks and clusters of gastrointestinal illness and number ill reported by OzFoodNet, Australia, 1 July to 30 September 2014, by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total outbreaks and clusters*	Number ill
Foodborne and suspected foodborne	25	7	253
Suspected waterborne	0	0	0
Person-to-person	242	71	5,292
Unknown (Salmonella cluster)	11	3	59
Unknown (Other pathogen cluster)	4	<1	7
Unknown	61	19	593
Total	343	100	6,204

* Percentages may not add to 100 due to rounding.

The data within this report, provided by OzFoodNet sites, has associated limitations, including the potential variation in categorisation of features of outbreaks, depending on varied circumstances and investigator interpretation. Changes in the number of foodborne disease outbreaks should be interpreted with caution due to the small number each quarter.

Salmonella Typhimurium was identified as the aetiological agent in 40% (10/25) of foodborne or suspected foodborne disease outbreaks during this quarter (Appendix). This equates to a slightly lower proportion than the same quarter in 2013 (41%, 11/27). The aetiological agents responsible for the remaining outbreaks during this quarter included: ciguatoxin (3 outbreaks); *Clostridium perfringens* (2 outbreaks); and histamine, *Salmonella* Saintpaul, monophasic *Salmonella* Typhimurium, and *Shigella sonnei* biotype (BT) G (1 outbreak each). The remaining 6 outbreaks were of unknown aetiology.

Ten (40%) foodborne or suspected foodborne disease outbreaks reported this quarter were associated with food prepared in restaurants (Table 2), which was lower than the 5-year mean during the 3rd quarter between 2009 and 2013 (46%).

To investigate these outbreaks, sites conducted 2 cohort studies and collected descriptive case series data for 13 investigations; while for 10 outbreaks no individual patient data were collected. The evidence used to implicate food vehicles included analytical evidence in 2 outbreaks, microbiological evidence in 1 outbreak, and descriptive evidence in 22 outbreaks.

The following jurisdictional summaries describe key outbreaks and public health actions that occurred during the quarter.

Australian Capital Territory

There were no outbreaks of foodborne or suspected foodborne illness reported in the Australian Capital Territory during this quarter.

New South Wales

There were 11 outbreaks of foodborne or suspected foodborne illness reported in New South Wales during this quarter. The aetiological agents were identified as *Salmonella* Typhimurium for 4 outbreaks and histamine for 1 outbreak. No aetiological agent was identified for the remaining 6 outbreaks.

Description of key outbreak

An outbreak of acute gastrointestinal illness was investigated at a resort in September. Twenty cases were identified and 14 of these were confirmed as *S.* Typhimurium, with 13 having multi-locus variable number tandem repeat analysis (MLVA) pattern 03-12-11-14-523 and 1 having MLVA 03-12-11-15-523. Onset dates ranged from 20–22 September 2014 and 5 of the 20 cases were hospitalised. The median age of cases was 18 years (range 2–64 years) and 75% of cases were identified as male. A cohort study was conducted involving several groups who ate at the resort. This involved interviewing 16 cases and 44 well individuals. The study found that chocolate milk served with the breakfast buffet on 20 and 21 September was significantly associated with illness (consumed by 15/16 cases and 1/30 well individuals; relative risk (RR) 28.6, 95% confidence interval (CI) 4.1–198.0, $P<0.005$). Other foods found to be potentially associated with illness were various cereals (consumed by 6/8 cases and 12/38 well individuals; RR 4.7, 95% CI 1.1–20.7, $P = 0.04$) and beef sausages (consumed by 5/9 cases and 1/23 well individuals; RR 5.6, 95% CI 2.1–14.9, $P=<0.005$).

Table 2: Outbreaks of foodborne or suspected foodborne disease and number ill reported by OzFoodNet, Australia, 1 July to 30 September 2014, by food preparation setting

Food preparation setting	Outbreaks	Per cent of foodborne outbreaks	Number ill
Restaurant	10	40	75
Aged care facility	4	16	42
Bakery	3	12	50
Primary produce	3	12	13
Camp	1	4	30
Private residence	1	4	18
Takeaway	1	4	13
Fair, festival, other temporary/mobile service	1	4	7
School	1	4	5
Total	25	100	253

There were no egg related dishes significantly associated with illness. The outbreak investigation found that a commercial stick blender used to prepare the chocolate milk was also used for blending raw eggs and raw chicken products. The blender was swabbed on 2 occasions; however laboratory testing did not detect *Salmonella* spp. Although the source of the outbreak was not determined, there was strong epidemiological evidence implicating cross-contaminated chocolate milk as the source of this outbreak.

Northern Territory

There were 2 outbreaks of foodborne or suspected foodborne illness reported in the Northern Territory during this quarter. *Shigella sonnei* biotype (BT) G was identified as the aetiological agent for 1 outbreak and *Salmonella* Saintpaul was the aetiological agent responsible for the 2nd outbreak.

Description of key outbreak

An outbreak of salmonellosis was investigated in the Northern Territory in August affecting 30 of 67 people who attended a school camp in a remote area. Four children had laboratory confirmed *S.* Saintpaul, but no cases were reported to have been hospitalised. Earlier in the year this same camp was associated with a cluster of 7 cases of *S.* Saintpaul, which implicated different schools. Following a cohort study, a batch of cordial was identified as being likely contaminated (either ice, water or the container) and was subsequently defined as the source of the outbreak (RR 3.8, 95% CI 1.3-11.0, $P=<0.005$). The camp remained closed until major renovations were completed.

Queensland

There were 5 outbreaks of foodborne or suspected foodborne illness reported in Queensland during this quarter. The aetiological agents were identified as *S.* Typhimurium for 2 outbreaks and ciguatoxin for 3 outbreaks.

Description of key outbreak

An outbreak of S. Typhimurium (MLVA 03-12-12-09-524) was reported in September affecting 12 individuals (10 laboratory-confirmed and 2 epidemiologically-linked) in a metropolitan area. Specimen collection dates were between 19 and 25 September and interviews concluded that all cases had reported eating egg-based meals from the same café prior to onset of their symptoms. Eggs Benedict (containing hollandaise sauce) was consumed by 9 of the 12 cases, potato rosti was consumed by 7 of the 12, and bacon and eggs were consumed by 2 of the 12 cases. The potato rosti consisted of a ball of potato that was bound with egg and then deep fried prior to consumption. Investigations identified that this particular food item was prepared earlier, stored frozen and cooked to order, raising concerns as to whether an adequate temperature was being achieved in the centre of the rosti during the cooking step. Multiple food samples and environmental swabs of food preparation surfaces were collected during the investigation; however all samples were negative for *Salmonella*. A number of hygiene issues with potential for cross-contamination were identified during environmental health investigations and were subsequently rectified by the café.

South Australia

There were 3 outbreaks of foodborne or suspected foodborne illness reported in South Australia during this quarter. The aetiological agents were identified as *S.* Typhimurium in 2 of the outbreaks and monophasic *S.* Typhimurium in 1 outbreak.

Description of key outbreak

An outbreak of gastroenteritis was investigated in September in metropolitan Adelaide linked to a restaurant. Twelve people were affected and all cases were subsequently confirmed as *S.* Typhimurium phage type (PT) 44 (MLVA 03-10-08-09-523). Resulting interviews concluded that 11 of the cases had consumed different types of eggs (scrambled, poached, or fried) for breakfast at the same restaurant, all during the same weekend in late August 2014. The 12th case was identified as a chef at the restaurant who had prepared the eggs that weekend, however the information he provided regarding his onset of illness was deemed inconsistent and therefore his role in the outbreak was unclear. An inspection of the restaurant was carried out and the restaurant was shut down until improvements were made. Food samples, including eggs, were collected from the restaurant and 1 sample returned a positive result for *S.* Typhimurium PT 44 (MLVA 03-10-08-09-523). The positive result was for a sample of breadcrumbs used to coat meat and zucchini. The breadcrumbs sampled were not used for breakfast foods. Furthermore, it was not confirmed whether eggs were used during the coating process.

Tasmania

There were no outbreaks of foodborne or suspected foodborne illness reported in Tasmania during this quarter.

Victoria

There were 3 outbreaks of foodborne or suspected foodborne illness reported in Victoria during this quarter. The aetiological agents were identified as *S.* Typhimurium for 2 outbreaks and *Clostridium perfringens* for the remaining outbreak.

Description of key outbreak

Following the notification of 3 cases of *Salmonella* during July, a routine investigation identified that all cases had eaten at the same Vietnamese bakery within a 6 day period. A further 7 cases of gastroenteritis were reported during the investigation and 5 of these were subsequently confirmed with *Salmonella*. Eight isolates were typed as *S.* Typhimurium PT 135. In total, 10 individuals were affected during the outbreak and 5 of these were hospitalised. Seven food samples were collected, including chicken liver pâté, roast pork, mayonnaise and vegetables. One pâté sample was positive for *S.* Typhimurium PT 135 and 3 subsequent environmental samples were negative for *Salmonella*.

Western Australia

There was 1 outbreak of foodborne illness reported in Western Australia during this quarter. The aetiological agent was identified as *Clostridium perfringens*.

Description of key outbreak

An outbreak of gastroenteritis at a residential care facility involving 19 residents was investigated in September. Onset dates were from 21–24 September, with 14 of 19 residents becoming ill on the morning of 24 September, predominantly with diarrhoea. Microbiological examination found 7 specimens positive for *C. perfringens* toxin. Six of 7 were also culture positive for *C. perfringens* and 5 of these were typed by pulsed-field gel electrophoresis, with 4 having indistinguishable patterns. The majority of ill residents were from the dementia ward and had a soft, minced or pureed diet and all of these diets were processed in a vitamiser. An environmental investigation found that remains of food were left in the vitamiser after it had undergone its normal cleaning process and therefore it was suspected that an unclean vitamiser may have led to the *C. perfringens* contamination.

Cluster investigations

During the quarter, OzFoodNet sites conducted investigations into 76 clusters of infection for which no common food vehicle or source of infection could be identified. Aetiological agents that were able to be identified during the investigations included 1 *Campylobacter* cluster, 6 *S.* Typhimurium clusters, 1 *Yersinia enterocolitica* cluster, 1 adenovirus cluster, 1 *S.* Weltevreden cluster, 1 *S.* Onderstepoort cluster, 1 *S.* Hessarek cluster, 1 *S.* Mbandaka cluster and 1 *S.* Kottbus cluster.

Comments

Three outbreaks in this quarter affecting 50 people were associated with Vietnamese style bakeries selling bánh mì thit, which are sandwiches consisting of a crusty roll, with mayonnaise, pâté, roast meat (traditionally pork, but sometimes chicken in Australia) and salad. This dish can become risky if the pâté is made with lightly cooked livers or the mayonnaise is made by blending raw egg through butter.

Acknowledgements

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

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Appendix: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites,* 1 July to 30 September 2014 (n=25)

State or territory	Month†	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
NSW	Jul	Restaurant	Histamine	8	0	D	Unknown
NSW	Jul	Restaurant	Unknown	5	0	D	Unknown
NSW	Jul	Restaurant	*Salmonella* Typhimurium MLVA 03-24-12-10-523	4	0	D	Unknown
NSW	Aug	Restaurant	Unknown	3	0	D	Suspected oysters
NSW	Sept	Restaurant	Unknown	8	0	D	Unknown
NSW	Sept	Restaurant	Unknown	4	0	D	Unknown
NSW	Sept	Bakery	*S.* Typhimurium MLVA 03-26-13-08-523	13	0	D	Raw egg mayonnaise
NSW	Sept	Restaurant	Unknown	3	0	D	Unknown
NSW	Sept	Restaurant	*S.* Typhimurium MLVA 03-12-11-14/15-523	20	5	A	Cross-contaminated chocolate milk
NSW	Sept	Aged care facility	Unknown	8	0	D	Roast beef
NSW	Sept	Aged care facility	*S.* Typhimurium MLVA 03-25-13-10-523	6	2	D	Unknown
NT	Jul	Fair, festival, other temporary/mobile service	*Shigella sonnei* BT G	7	0	D	Unknown
NT	Aug	Camp	*S.* Saintpaul	30	0	A	Cordial
Qld	Aug	Primary produce	Ciguatoxin	2	0	D	Unknown
Qld	Aug	Restaurant	*S.* Typhimurium MLVA 03-12-13-09-524	9	2	D	Unknown
Qld	Sept	Primary produce	Ciguatoxin	9	0	D	Spanish mackerel
Qld	Sept	Primary produce	Ciguatoxin	2	0	M	Spanish mackerel
Qld	Sept	Restaurant	*S.* Typhimurium MLVA 03-12-12-09-524	12	1	D	Eggs Benedict with potato rosti
SA	Sept	Private residence	Monophasic *S.* Typhimurium MLVA 04-15-12-00-490	18	1	D	Pork spit roast
SA	Sept	Restaurant	*S.* Typhimurium PT 44 MLVA 03-10-08-09-523	12	3	D	Suspected eggs
SA	Sept	School	*S.* Typhimurium PT 9	5	1	D	Chicken burger

Appendix *continued*: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites,* 1 July to 30 September 2014 (n=25)

State or territory	Month†	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
Vic.	Jul	Aged care facility	*Clostridium perfringens*	9	0	D	Unknown
Vic.	Jul	Bakery	*S.* Typhimurium PT 135	10	5	D	Chicken liver pâté
Vic.	Sept	Bakery	*S.* Typhimurium PT 170	27	3	D	Unknown
WA	Sept	Aged care facility	*Clostridium perfringens*	19	0	D	Soft/minced/vitamised food

* No foodborne outbreaks were reported in Tasmania or the Australian Capital Territory during the quarter.

† Month of outbreak is the month of onset of the first case or month of notification of the first case or month the investigation of the outbreak commenced.

The number of people affected and hospitalised relates to the findings of the outbreak investigation at the time of writing and not necessarily in the month specified or in this quarter. The number of people affected does not necessarily equal the number of laboratory-confirmed cases.

A Analytical epidemiological association between illness and 1 or more foods.

D Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission.

M Microbiological confirmation of aetiological agent in the suspected vehicle and cases.

PT Phage type

MLVA Multi-locus variable number tandem repeat analysis profile

BT Biotype

National Notifiable Diseases Surveillance System, 1 July to 30 September 2016

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 63,863 notifications to the National Notifiable Diseases Surveillance System (NNDSS) between 1 July to 30 September 2016 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions
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
Paratyphoid	All jurisdictions
Shiga toxin/verotoxin-producing *Escherichia coli*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid fever	All jurisdictions
Quarantinable diseases	
Avian influenza in humans	All jurisdictions
Cholera	All jurisdictions
Middle East respiratory syndrome coronavirus	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions
Syphilis - congenital	All jurisdictions

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

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

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2016, by date of diagnosis*

Disease	State or territory								Total 3rd quarter 2016	Total 2nd quarter 2016	Total 3rd quarter 2015	Last 5 years mean 3rd quarter	Ratio	Year to date 2016	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Hepatitis B (newly acquired)†	0	0	1	10	0	0	12	5	28	42	36	42.0	0.7	111	132.2
Hepatitis B (unspecified)‡	29	582	24	267	55	12	443	150	1,562	1,878	1,751	1,730.8	0.9	5,016	4,896.2
Hepatitis C (newly acquired)†	4	5	0	28	6	4	23	31	101	129	109	105.8	1.0	371	328.0
Hepatitis C (unspecified)‡	45	931	40	594	90	64	576	279	2,619	2,821	2,304	2,350.4	1.1	8,203	6,815.0
Hepatitis D	0	2	0	2	2	0	5	0	11	13	12	11.6	0.9	39	37.4
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	1.8
Campylobacteriosis	134	NN	125	1,673	880	267	1,462	971	5,512	5,191	5,448	4,372.4	1.3	16,631	13,102.8
Cryptosporidiosis	14	130	24	189	52	12	184	51	656	1,501	482	389.4	1.7	4,470	2,454.8
Haemolytic uraemic syndrome	0	0	0	3	0	0	1	1	5	1	6	3.8	1.3	10	13.0
Hepatitis A	1	3	0	2	0	0	10	5	21	24	28	38.2	0.5	100	140.6
Hepatitis E	0	3	1	0	0	0	3	1	8	5	7	6.0	1.3	28	29.2
Listeriosis	0	4	0	1	1	0	3	4	13	23	14	15.0	0.9	63	56.4
Paratyphoid	0	4	0	1	1	0	3	4	13	12	14	8.8	1.5	58	53.2
STEC§	0	15	0	4	55	0	8	9	91	57	21	33.2	2.7	191	93.6
Salmonellosis	55	813	110	682	332	41	780	359	3,172	4,519	2,645	2,330.4	1.4	14,234	10,447.6
Shigellosis	0	65	32	48	8	5	145	23	326	349	264	164.8	2.0	1,038	544.6
Typhoid fever	1	6	0	2	0	0	6	5	20	20	18	18.8	1.1	77	97.2
Quarantinable diseases															
Avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Cholera	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	3.0
Middle East respiratory syndrome coronavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.4

Table 2 *continued:* Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2016, by date of diagnosis*

Disease	State or territory								Total 3rd quarter 2016	Total 2nd quarter 2016	Total 3rd quarter 2015	Last 5 years mean 3rd quarter	Ratio	Year to date 2016	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Sexually transmissible infections															
Chlamydial infection[‖,¶]	313	6,435	607	5,728	1,310	254	236	2,888	17,771	19,628	16,483	19,761.6	0.9	56,054	61,987.2
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.4
Gonococcal infection[¶]	57	1,721	396	1,069	249	18	478	862	4,850	6,018	4,597	3,594.4	1.3	16,489	11,264.0
Syphilis <2 years duration[¶]	2	166	61	168	25	0	184	100	706	795	756	500.0	1.4	2,295	1,396.8
Syphilis >2 years or unspecified duration[‡,¶]	3	94	12	74	39	2	321	9	554	457	478	419.4	1.3	1,487	1,238.4
Syphilis – congenital	0	0	0	2	0	0	0	0	2	0	2	2.0	1.0	2	3.4
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	3	1	0.6	0.0	4	1.8
Haemophilus influenzae type b	0	2	0	1	0	0	2	0	5	5	8	6.2	0.8	14	13.8
Influenza (laboratory confirmed)	1,348	27,510	357	14,786	3,807	739	8,294	5,551	62,392	7,305	79,584	40,008.6	1.6	75,501	48,598.8
Measles	0	0	0	1	7	0	2	4	14	19	19	56.6	0.2	70	150.4
Mumps	1	20	67	21	5	1	8	37	160	189	200	75.8	2.1	687	191.2
Pertussis	140	2,480	45	418	537	11	626	455	4,712	3,857	5,647	5,380.6	0.9	14,439	15,431.8
Pneumococcal disease – invasive	14	203	14	105	55	12	142	93	638	439	589	637.4	1.0	1,259	1,314.0
Poliovirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	2	0	0	0	1	0	0	3	9	3	8.0	0.4	15	24.8
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	1	0.4	0.0	0	0.6
Tetanus	0	0	0	0	0	0	0	0	0	1	0	0.4	0.0	4	2.4
Varicella zoster (chickenpox)	25	NN	19	148	78	14	44	167	495	592	679	618.4	0.8	1,677	1,527.4
Varicella zoster (shingles)	70	NN	96	16	486	74	70	410	1,222	1,713	1,484	1,210.8	1.0	4,719	3,729.0
Varicella zoster (unspecified)	40	NN	0	1,883	85	32	328	408	2,776	3,741	3,350	2,763.0	1.0	10,306	7,917.4
Vectorborne diseases															
Barmah Forest virus infection	0	4	1	33	0	0	1	2	41	83	83	307.8	0.1	249	1,509.8
Chikungunya virus infection	0	12	0	2	1	1	12	2	30	15	15	16.0	1.9	63	63.8
Dengue virus infection	12	80	14	60	24	10	101	112	413	701	267	275.0	1.5	1,858	1,261.2
Flavivirus infection (unspecified)	0	5	0	13	0	0	2	6	26	18	1	2.4	10.8	98	11.0
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.6	0.0	0	1.6
Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.4
Malaria	1	20	6	13	3	0	20	18	81	74	61	89.6	0.9	226	263.6
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	3.6
Ross River virus infection	1	37	37	127	4	0	16	41	263	1,010	809	644.6	0.4	2,499	4,800.6

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 July to 30 September 2016, by date of diagnosis

Disease	State or territory								Total 3rd quarter 2016	Total 2nd quarter 2016	Total 3rd quarter 2015	Last 5 years mean 3rd quarter	Ratio	Year to date 2016	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Australian bat lyssavirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Brucellosis	0	4	0	2	0	0	0	0	6	4	6	6.4	0.9	12	18.2
Leptospirosis	0	1	0	12	1	0	5	1	20	39	17	17.4	1.1	112	99.2
Lyssavirus infection (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	3	0	5	1	3	12.0	0.4	8	34.4
Q fever	0	48	2	51	7	0	17	0	125	115	162	115.0	1.1	386	347.2
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Other bacterial infections															
Legionellosis	1	26	0	12	9	0	19	22	89	101	87	106.4	0.8	272	300.8
Leprosy	0	1	0	1	0	0	1	4	7	4	5	3.0	2.3	15	7.8
Meningococcal infection – invasive**	2	34	0	11	7	2	20	8	84	46	67	66.6	1.3	175	151.0
Tuberculosis	6	139	6	49	24	1	95	44	364	296	312	346.0	1.1	978	949.6
Total	2,319	41,609	2,097	28,312	8,245	1,577	14,711	13,142	112,012	63,863	128,935			242,613	

* 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 began reporting hepatitis C newly acquired from 1 September 2016. Previous notifications are reported under hepatitis unspecified.

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

§ Infection with Shiga toxin/verotoxin-producing *Escherichia coli.*

|| Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens.

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

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

NN Not notifiable

NEC Not elsewhere classified

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

Table 3: Notification rates of diseases, 1 July to 30 September 2016, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)‡	0.0	0.0	1.6	0.8	0.0	0.0	0.8	0.8	0.5
Hepatitis B (unspecified)§	29.7	30.6	39.3	22.3	12.9	9.3	29.8	23.2	26.3
Hepatitis C (newly acquired)‡	4.1	0.3	0.0	2.3	1.4	3.1	1.5	4.8	1.7
Hepatitis C (unspecified)§	46.0	48.9	65.4	49.7	21.2	49.5	38.8	43.1	44.0
Hepatitis D	0.0	0.1	0.0	0.2	0.5	0.0	0.3	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	137.1	NN	204.5	140.0	207.2	206.7	98.4	149.9	136.4
Cryptosporidiosis	14.3	6.8	39.3	15.8	12.2	9.3	12.4	7.9	11.0
Haemolytic uraemic syndrome	0.0	0.0	0.0	0.3	0.0	0.0	0.1	0.2	0.1
Hepatitis A	1.0	0.2	0.0	0.2	0.0	0.0	0.7	0.8	0.4
Hepatitis E	0.0	0.2	1.6	0.0	0.0	0.0	0.2	0.2	0.1
Listeriosis	0.0	0.2	0.0	0.1	0.2	0.0	0.2	0.6	0.2
Paratyphoid	0.0	0.2	0.0	0.1	0.2	0.0	0.2	0.6	0.2
STEC‖	0.0	0.8	0.0	0.3	12.9	0.0	0.5	1.4	1.5
Salmonellosis	56.3	42.7	180.0	57.1	78.2	31.7	52.5	55.4	53.3
Shigellosis	0.0	3.4	52.4	4.0	1.9	3.9	9.8	3.6	5.5
Typhoid fever	1.0	0.3	0.0	0.2	0.0	0.0	0.4	0.8	0.3
Quarantinable diseases									
Avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Middle East respiratory syndrome coronavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmitted infections									
Chlamydial infection¶,**	320.2	337.8	993.0	479.3	308.4	196.6	15.9	445.9	298.8
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	58.3	90.3	647.8	89.4	58.6	13.9	32.2	133.1	81.6
Syphilis < 2 years duration**	2.0	8.7	99.8	14.1	5.9	0.0	12.4	15.4	11.9
Syphilis > 2 years or unspecified duration§,**	3.1	4.9	19.6	6.2	9.2	1.5	21.6	1.4	9.3
Syphilis – congenital	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Influenza (laboratory confirmed)	1,378.9	1,444.2	584.0	1,237.1	896.3	572.1	558.4	857.1	1,049.2
Measles	0.0	0.0	0.0	0.1	1.6	0.0	0.1	0.6	0.2
Mumps	1.0	1.0	109.6	1.8	1.2	0.8	0.5	5.7	2.7
Pertussis	143.2	130.2	73.6	35.0	126.4	8.5	42.1	70.3	79.2
Pneumococcal disease – invasive	14.3	10.7	22.9	8.8	12.9	9.3	9.6	14.4	10.7

Table 3 *continued*: Notification rates of diseases, 1 July to 30 September 2016, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Vaccine preventable diseases, cont'd									
Poliovirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.1	0.0	0.0	0.0	0.8	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
Varicella zoster (chickenpox)	25.6	NN	31.1	12.4	18.4	10.8	3.0	25.8	12.2
Varicella zoster (shingles)	71.6	NN	157.1	1.3	114.4	57.3	4.7	63.3	30.2
Varicella zoster (unspecified)	40.9	NN	0.0	157.6	20.0	24.8	22.1	63.0	68.7
Vectorborne diseases									
Barmah Forest virus infection	0.0	0.2	1.6	2.8	0.0	0.0	0.1	0.3	0.7
Chikungunya virus infection	0.0	0.6	0.0	0.2	0.2	0.8	0.8	0.3	0.5
Dengue virus infection	12.3	4.2	22.9	5.0	5.7	7.7	6.8	17.3	6.9
Flavivirus infection (unspecified)	0.0	0.3	0.0	1.1	0.0	0.0	0.1	0.9	0.4
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	1.0	1.0	9.8	1.1	0.7	0.0	1.3	2.8	1.4
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	1.0	1.9	60.5	10.6	0.9	0.0	1.1	6.3	4.4
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.2	0.0	0.2	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.1	0.0	1.0	0.2	0.0	0.3	0.2	0.3
Lyssavirus infection (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.1	0.0	0.0	0.0	0.0	0.2	0.0	0.1
Q fever	0.0	2.5	3.3	4.3	1.6	0.0	1.1	0.0	2.1
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	1.0	1.4	0.0	1.0	2.1	0.0	1.3	3.4	1.5
Leprosy	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.6	0.1
Meningococcal infection – invasive††	2.0	1.8	0.0	0.9	1.6	1.5	1.3	1.2	1.4
Tuberculosis	6.1	7.3	9.8	4.1	5.7	0.8	6.4	6.8	6.1

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

† Rate per 100,000 of population. Annualisation Factor was 4.0

‡ Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis. Queensland began reporting hepatitis C newly acquired from 1 September 2016. Previous notifications are reported under hepatitis unspecified.

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

|| Infection with Shiga toxin/verotoxin-producing *Escherichia coli*.

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral and throat samples, except for South Australia, which reports only cervical, urine and urethral specimens.

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

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

NEC Not elsewhere classified.

NN Not notifiable.

Australian childhood immunisation coverage, 1 April 2015 to 31 March 2016 cohort, assessed as at 30 June 2016

Alexandra J Hendry for the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

Introduction

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS) provides commentary on the trends in ACIR data. For further information please contact NCIRS at: telephone +61 2 9845 1423, email: alexandra.hendry@health.nsw.gov.au

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

The data show the percentage of all children 'fully immunised' at 12 months, 24 months and 60 months of age, for four 3-month birth cohorts of children assessed at the stated ages between 1 April 2015 and 31 March 2016 using ACIR data up to 30 June 2016. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, and is outlined in more detail below.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of three doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of *Haemophilus* B conjugate (PRP-OMP) containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, and 3 doses of 13-valent pneumococcal conjugate vaccine. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 doses of a DTP-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP Hib, Infanrix Hexa or Hiberix vaccine (3 doses only of Infanrix Hexa or Hiberix if given after 11.5 months of age), or 4 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, 2 doses of a measles, mumps and rubella-containing (MMR) vaccine, 1 dose of meningococcal C vaccine, and 1 dose of varicella vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

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

Results

The rolling annualised percentage of all children 'fully immunised' by 12 months of age for Australia increased marginally from the previous report by 0.3 of a percentage point to 93.0% (Table 1). Small increases in the percentage of children 'fully immunised' by 12 months of age were experienced in all jurisdictions and coverage was greater than 92% in all jurisdictions. For individual vaccines due by 12 months of age all jurisdictions achieved coverage greater than 93%.

The rolling annualised percentage of all children 'fully immunised' by 24 months of age for Australia has continued to rise with coverage increasing by 0.6 percentage points to reach 90.7% (Table 2). Coverage for individual vaccines due by 24 months of age reached above 94% in all jurisdictions, except for the measles, mumps and rubella vaccine and varicella. Coverage for these antigens at 24 months

Table 1: Percentage of children immunised at 12 months of age for the birth cohort 1 April 2014 to 31 March 2015, preliminary results, by disease and state or territory; assessment date 30 June 2016

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,660	98,635	3,779	62,133	19,834	5,790	77,117	34,551	307,499
Diphtheria, tetanus, pertussis (%)	95.3	93.8	93.6	93.8	93.7	93.9	94.0	93.4	93.8
Poliomyelitis (%)	95.2	93.8	93.6	93.8	93.7	93.9	94.0	93.4	93.8
Haemophilus influenzae type b (%)	94.9	93.5	93.5	93.6	93.4	93.8	93.6	93.2	93.5
Hepatitis B (%)	95.1	93.7	94.0	93.8	93.6	94.0	93.8	93.1	93.7
Pneumococcal	94.9	93.4	93.5	93.5	93.4	93.8	93.6	93.1	93.5
Fully immunised (%)	94.5	92.9	93.0	93.2	93.0	93.5	93.0	92.6	93.0

of age have however, continued to improve with measles, mumps and rubella coverage increasing from the previous report by 0.5 of a percentage point to 92.3% and varicella coverage increasing by 0.3 of a percentage point to 92.4%.

The rolling annualised percentage of all children 'fully immunised' by 60 months of age for Australia increased from the previous report by 0.2 of a percentage point to 92.9% (Table 3). Coverage for individual vaccines due by 60 months of age was 92% or greater in all jurisdictions.

The Figure shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. Overall there is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007). Coverage by 24 months did fall below the 12 and 60 month coverage estimates following the change in the 24 month coverage assessment algorithm to include MMR dose 2 instead of MMR dose 1, varicella dose 1, and meningococcal C dose 1. However, 'fully immunised' coverage by 24 months has been steadily increasing since this change and as at 31 March 2016 reached 91.3%, which is an increase of 0.5 of a percentage point from the previous quarterly report. In the 1st quarter of 2016 there have also been marginal increases in the 'fully immunised' coverage estimates by 12 and 60 months to 93.3% and 93.0% respectively.

The National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases is supported by the Australian Government Department of Health, the NSW Ministry of Health and The Children's Hospital at Westmead. The opinions expressed in this paper are those of the authors, and do not necessarily represent the views of these agencies.

Figure: Trends in vaccination coverage, Australia, 1997 to 31 March 2016, by age cohorts

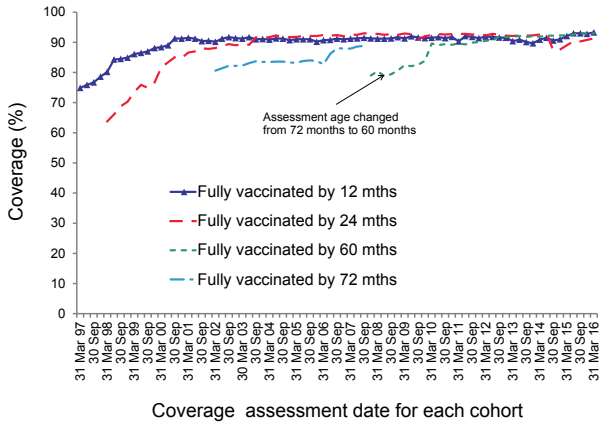

Table 2: Percentage of children immunised at 24 months of age for the birth cohort 1 April 2013 to 31 March 2014, preliminary results, by disease and state or territory; assessment date 30 June 2016

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	5,681	99,173	3,549	62,337	19,969	5,953	76,890	33,960	307,512
Diphtheria, tetanus, pertussis (%)	97.0	95.7	95.4	95.7	95.8	96.1	96.2	95.7	95.9
Poliomyelitis (%)	96.9	95.7	95.4	95.7	95.7	96.1	96.1	95.7	95.8
Haemophilus influenzae type b (%)	95.9	94.7	94.4	95.0	94.8	95.1	95.3	94.7	94.9
Measles, mumps, rubella (%)	93.4	92.2	91.5	92.6	92.4	93.0	92.6	91.2	92.3
Hepatitis B (%)	96.7	95.5	95.8	95.6	95.5	96.0	95.9	95.4	95.7
Meningococcal C (%)	95.5	94.6	94.3	94.9	94.5	95.0	94.9	94.1	94.7
Varicella (%)	93.6	92.3	90.7	92.5	92.5	92.7	92.8	91.2	92.4
Fully immunised (%)	91.8	90.4	88.9	91.4	90.5	90.7	91.0	89.6	90.7

Table 3: Percentage of children immunised at 60 months of age for the birth cohort 1 April 2010 to 31 March 2011, preliminary results, by disease and state or territory; assessment date 30 June 2016

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	5,550	101,092	3,497	64,952	20,112	6,229	76,211	34,455	312,098
Diphtheria, tetanus, pertussis (%)	94.1	93.9	92.7	93.3	93.0	94.5	93.9	92.0	93.5
Poliomyelitis (%)	94.2	94.0	92.8	93.3	93.0	94.5	94.0	92.0	93.5
Measles, mumps, rubella (%)	94.3	94.0	93.1	93.2	93.0	94.5	93.9	92.2	93.6
Fully immunised (%)	93.5	93.4	91.9	92.7	92.3	93.8	93.3	91.3	92.9

Australian Gonococcal Surveillance Programme, 1 January to 31 March 2016

Monica M Lahra, Rodney P Enriquez, The World Health Organisation Collaborating Centre for STD and Neisseria Reference Laboratory, South Eastern Area Laboratory Services, Prince of Wales Hospital for The National Neisseria Network

Introduction

The Australian National Neisseria Network (NNN) comprises reference laboratories in each State and Territory that report data on sensitivity to an agreed group of antimicrobial agents for the Australian Gonococcal Surveillance Programme (AGSP). The antibiotics are penicillin, ceftriaxone, azithromycin and ciprofloxacin, which are current or potential agents used for the treatment of gonorrhoea. Azithromycin combined with ceftriaxone is the recommended treatment regimen for gonorrhoea in the majority of Australia. However, there are substantial geographic differences in susceptibility patterns in Australia and in certain remote regions of the Northern Territory and Western Australia gonococcal antimicrobial resistance rates are low, and an oral treatment regimen comprising amoxycillin, probenecid and azithromycin is recommended for the treatment of gonorrhoea. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatments.[1] Additional data on other antibiotics are reported in the AGSP annual report. The AGSP has a program-specific quality assurance process.

Results

A summary of the proportion of isolates with decreased susceptibility to ceftriaxone, and the proportion resistant to azithromycin, penicillin, and ciprofloxacin for the 1st quarter of 2016 are shown in Table 1.

Ceftriaxone

Ceftriaxone minimum inhibitory concentration (MIC) values in the range 0.06–0.125 mg/L have been reported in the category decreased susceptibility since 2005.

In the 1st quarter of 2016 the states that reported isolates with decreased susceptibility to ceftriaxone were New South Wales, Victoria, Queensland, urban/rural Western Australia and the Australian Capital Territory. No decreased susceptibility to ceftriaxone was reported in South Australia, the remote regions of Western Australia, the Northern Territory or Tasmania. New South Wales and Victoria reported a decrease in the proportion of *Neisseria gonorrhoeae* isolates with decreased susceptibility to ceftriaxone when compared with the same quarter in 2015. Compared with the annual data for 2015, New South Wales, Victoria, and

Table 1: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to azithromycin, penicillin, and ciprofloxacin, Australia, 1 January to 31 March 2016, by state or territory

State or territory	Number of isolates tested	Decreased susceptibility		Resistance					
		Ceftriaxone		Azithromycin		Penicillin		Ciprofloxacin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	22	1	4.5	2	9.1	2	9.1	6	27.3
New South Wales	590	14	2.4	5	0.8	205	34.7	206	34.9
Queensland	166	2	1.2	4	2.4	43	25.9	37	22.3
South Australia	88	0	0.0	26	29.5	37	42.0	24	27.3
Tasmania	7	0	0.0	0	0.0	3	42.9	2	28.6
Victoria	445	3	0.7	7	1.6	111	24.9	157	9.4
Northern Territory Urban & Rural	14	0	0.0	0	0.0	4	28.6	3	21.4
Northern Territory Remote	63	0	0.0	0	0.0	2	3.2	4	6.3
Western Australia Urban & Rural	152	3	2.0	6	3.9	29	19.1	35	23.0
Western Australia Remote	25	0	0.0	0	0.0	1	4.0	0	0.0
Australia	1,572	23	1.5	50	3.2	437	27.8	474	30.2

South Australia reported a decrease in the proportion of *N. gonorrhoeae* isolates with decreased susceptibility to ceftriaxone. Queensland and urban/rural Western Australia reported an increase in the proportion of *N. gonorrhoeae* isolates with decreased susceptibility to ceftriaxone, while the other states reported similar results.[2]

From New South Wales there were 14 of 590 strains with decreased susceptibility to ceftriaxone. Of those, all were multidrug-resistant (MDR); 12 (86%) were from males; and 6 (43%) were isolated from extragenital sites (rectal and pharyngeal). From Victoria, 3 of 445 strains had decreased susceptibility to ceftriaxone. All were MDR and from males; and 2 (67%) were isolated from extragenital sites. From Queensland, 2 of 166 strains had decreased susceptibility to ceftriaxone and of those, 1 (50%) was MDR, 1 (50%) was from a male, and 1 (50%) was from an extragenital site. From urban/rural Western Australia there were 3 of 152 strains with decreased susceptibility to ceftriaxone; all of which were MDR, from males, and from extragenital sites. From the Australian Capital Territory there was 1 of 22 strains with decreased susceptibility to ceftriaxone, it was MDR, from a male, but not from an extragenital site.

In recent years the proportion of strains with decreased susceptibility to ceftriaxone has been of increasing concern in Australia and overseas, as this is phenotypic of the genotype with the key mutations that are the precursor to ceftriaxone resistance.[3] There are recent reports of ceftriaxone 500 mg treatment failures in patients from Victoria and New South Wales in patients with pharyngeal gonococcal infections. In these patients the infecting gonococcal strains had ceftriaxone MIC values in the range 0.03–0.06 mg/L.[4,5] Until 2013 there had not been an isolate reported in Australia with a ceftriaxone MIC value >0.125 mg/L.[2] In late December 2013 there was a new multidrug-resistant gonococcal strain (A8806) with a ceftriaxone MIC of 0.5 mg/L, the highest ever reported in Australia that was isolated from a female traveller from Central Europe. This infection was acquired in Sydney from another traveller, also from Europe. The patient was tested in the Northern Territory, but had travelled to north-eastern Queensland before the results were available, and was treated there. To date there has been no evidence of spread of this strain.[6]

The category of ceftriaxone decreased susceptibility as reported by the AGSP includes the MIC values 0.06 and 0.125 mg/L (Table 2).

Azithromycin

Azithromycin resistance is defined as a MIC to azithromycin equal to or greater than 1.0 mg/L.

In the 1st quarter of 2016, all states reported isolates with resistance to azithromycin, with the exception of Tasmania, the Northern Territory and remote Western Australia. Notably the reported proportion of *N. gonorrhoeae* isolates with resistance to azithromycin in South Australia was 26/88 (29.5%) in the 1st quarter of 2016, compared with none in the same quarter in 2015. None of these strains had high level resistance, all were resistant to penicillin (beta-lactamase producing); and sensitive to ceftriaxone and ciprofloxacin. Enhanced surveillance, case reviews, and genotypic investigations are in process in South Australia with further results to follow.

In the Australian Capital Territory there were 2/22 (9.1%) isolates from the 1st quarter of 2016 that were resistant to ceftriaxone compared with none in the same quarter in 2015. The states that reported a decrease in the proportion of *N. gonorrhoeae* isolates with resistance to azithromycin when compared with the same quarter in 2015 were New South Wales, Queensland, Tasmania, and urban/rural Western Australia. The other states reported similar results from the same quarter of 2015.

Penicillin

Penicillin resistant *N. gonorrhoeae* are defined as those isolates with a MIC to penicillin equal to or greater than 1.0 mg/L. Penicillin resistance includes penicillinase producing *N. gonorrhoeae* (PPNG), and *N. gonorrhoeae* that have chromosomally mediated resistance to penicillin (CMRP). In certain areas of the Northern Territory and Western Australia, which are classified as remote, a treatment regimen based on oral amoxicillin, probenecid and azithromycin is used. Due to the distance specimens must travel in these remote regions to a laboratory, low numbers of cultures are collected, and thus, by necessity, nucleic acid amplification testing (NAAT) is used. In remote

Table 2: Percentage of gonococcal isolates with decreased susceptibility to ceftriaxone MIC 0.06–0.125 mg/L, Australia, 2011 to 2015, and 1 January to 31 March 2016

Ceftriaxone MIC mg/L	2011	2012	2013	2014	2015	2016 Q1
0.06 (%)	3.2	4.1	8.2	4.8	1.7	1.5
0.125 (%)	0.1	0.3	0.6	0.6	0.1	0.0

Western Australia the introduction of a targeted NAAT, developed by the NNN to detect PPNG, is in use to enhance surveillance.[7,8]

Ciprofloxacin

Ciprofloxacin resistance includes isolates with an MIC to ciprofloxacin equal to or greater than 1.0 mg/L.

Dual therapy of ceftriaxone plus azithromycin is the recommended treatment for gonorrhoea as a strategy to temper development of more widespread resistance.[6] Patients with infections in extragenital sites, where the isolate has decreased susceptibility to ceftriaxone, are recommended to have test of cure cultures collected. Continued surveillance to monitor *N. gonorrhoeae* with elevated MIC values, coupled with sentinel site surveillance in high risk populations remains important to inform therapeutic strategies, to identify incursion of resistant strains, and to detect instances of treatment failure.

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Australian Gonococcal Surveillance Programme, 1 April to 30 June 2016

Monica M Lahra, Rodney P Enriquez, The World Health Organisation Collaborating Centre for STD and Neisseria Reference Laboratory, South Eastern Area Laboratory Services, Prince of Wales Hospital, for The National Neisseria Network

Introduction

The Australian National Neisseria Network (NNN) comprises reference laboratories in each state and territory that report data on sensitivity to an agreed group of antimicrobial agents for the Australian Gonococcal Surveillance Programme (AGSP). The antibiotics are penicillin, ceftriaxone, azithromycin and ciprofloxacin, which are current or potential agents used for the treatment of gonorrhoea. Azithromycin combined with ceftriaxone is the recommended treatment regimen for gonorrhoea in the majority of Australia. However, there are substantial geographic differences in susceptibility patterns in Australia and in certain remote regions of the Northern Territory and Western Australia gonococcal antimicrobial resistance rates are low, and an oral treatment regimen comprising amoxycillin, probenecid and azithromycin is recommended for the treatment of gonorrhoea. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatments.[1] Additional data on other antibiotics are reported in the AGSP annual report. The AGSP has a program-specific quality assurance process.

Results

A summary of the proportion of isolates with decreased susceptibility to ceftriaxone, and the proportion resistant to azithromycin, penicillin, and ciprofloxacin for the 2nd quarter of 2016 are shown in Table 1.

The category of ceftriaxone decreased susceptibility as reported by the AGSP includes the minimum inhibitory concentration (MIC) values 0.06 and 0.125 mg/L. A summary of the proportion of isolates with decreased susceptibility to ceftriaxone, for 2011 to 2015, and the first 2 quarters of 2016 is shown in Table 2.

Ceftriaxone

Ceftriaxone MIC values in the range 0.06–0.125 mg/L have been reported in the category decreased susceptibility since 2005.

The proportion of *Neisseria gonorrhoeae* isolates with decreased susceptibility to ceftriaxone in Australia for the 2nd quarter of 2016 was higher than the previous quarter, and was higher when compared with the same quarter in 2015 and with the annual data for 2015.

Table 1: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to azithromycin, penicillin, and ciprofloxacin, Australia, 1 April to 30 June 2016, by state or territory

State or territory	Number of isolates tested Q2, 2016	Decreased susceptibility		Resistance					
		Ceftriaxone		Azithromycin		Penicillin		Ciprofloxacin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	35	0	0.0	7	20.0	1	2.9	10	28.6
New South Wales	586	38	6.5	21	3.6	275	46.9	196	33.4
Queensland	210	12	5.7	1	0.5	75	35.7	57	27.1
South Australia	110	1	0.9	25	22.7	45	40.9	38	34.5
Tasmania	8	1	12.5	0	0.0	4	50.0	2	25.0
Victoria	418	4	1.0	16	3.8	116	27.8	144	34.4
Northern Territory Urban & Rural	10	0	0.0	0	0.0	0	0.0	3	30.0
Northern Territory Remote	35	0	0.0	0	0.0	2	5.7	1	2.9
Western Australia Urban & Rural	186	1	0.5	4	2.2	33	17.7	43	23.1
Western Australia Remote	42	0	0.0	1	2.4	1	2.4	1	2.4
Australia	1,640	57	3.5	75	4.6	552	33.7	495	30.2

Table 2: Percentage of gonococcal isolates with decreased susceptibility to ceftriaxone MIC 0.06–0.125 mg/L, Australia, 2011 to 2015, 1 January to 31 March 2016, and 1 April to 30 June 2016

Ceftriaxone MIC mg/L	2011	2012	2013	2014	2015	2016 Q1	2016 Q2
0.06 (%)	3.2	4.1	8.2	4.8	1.7	1.5	3.4
0.125 (%)	0.1	0.3	0.6	0.6	0.1	0.0	0.1

In the 2nd quarter of 2016 the states that reported isolates with decreased susceptibility to ceftriaxone were New South Wales, Queensland, Victoria, South Australia, urban/rural Western Australia and Tasmania. There were no isolates with decreased susceptibility to ceftriaxone reported in the Northern Territory, the remote regions of Western Australia, or the Australian Capital Territory. New South Wales, Queensland, and Tasmania reported an increase in the proportion of *N. gonorrhoeae* isolates with decreased susceptibility to ceftriaxone when compared with the same quarter in 2015, and with the annual data for 2015. Victoria, urban and rural Western Australia, and South Australia reported a decrease in the proportion of *N. gonorrhoeae* isolates with decreased susceptibility to ceftriaxone when compared with the same quarter in 2015, and with the annual data for 2015. Other states reported similar results.[2]

From New South Wales, there were 38 of 586 strains with decreased susceptibility to ceftriaxone. Of those, 21 (55%) were multidrug-resistant (MDR); 33 (87%) were from males; and 13 (34%) were isolated from extragenital sites (rectal and pharyngeal). From Queensland, there were 12 of 210 strains with decreased susceptibility to ceftriaxone and of those, 8 (67%) were MDR, 8 (67%) were from males, and 42 (33%) were from extragenital sites. From Victoria, 4 of 418 strains had decreased susceptibility to ceftriaxone. All were MDR and from males; and 1 (25%) was isolated from an extragenital site. From South Australia there was 1 of 110 strains with decreased susceptibility to ceftriaxone, the strain was MDR, from a male, and from an extragenital site. From urban/rural Western Australia, 1 of 186 strains had decreased susceptibility to ceftriaxone, the strain was MDR, from a male, but not from an extragenital site. From Tasmania there was 1 of 8 strains with decreased susceptibility to ceftriaxone, the strain was MDR, from a male, and from an extragenital site.

In recent years the proportion of strains with decreased susceptibility to ceftriaxone has been of increasing concern in Australia and overseas, as this is phenotypic of the genotype with the key mutations that are the precursor to ceftriaxone resistance.[3] There are recent reports of ceftriaxone 500 mg treatment failures in patients from Victoria and New South Wales in patients with pharyngeal gonococcal infections. In these patients the infecting gonococcal strains had ceftriaxone MIC values in the range 0.03–0.06 mg/L.[4,5] Until 2013 there had not been an isolate reported in Australia with a ceftriaxone MIC value >0.125 mg/L.[2] In late December 2013, there was a new multidrug-resistant gonococcal strain (A8806) with a ceftriaxone MIC of 0.5 mg/L, the highest ever reported in Australia that was isolated from a female traveller from Central Europe. This infection was acquired in Sydney from another traveller, also from Europe. The patient was tested in the Northern Territory, but had travelled to north eastern Queensland before the results were available, and was treated there. To date there has been no evidence of spread of this strain.[6]

Azithromycin

Azithromycin resistance is defined as a MIC to azithromycin equal to, or greater than 1.0 mg/L.

In the 2nd quarter of 2016, all states, with the exception of Tasmania and the Northern Territory, reported isolates with resistance to azithromycin. Notably the reported proportion of *N. gonorrhoeae* isolates with resistance to azithromycin in South Australia for the 2nd quarter 2016 was 25/110 (22.7%). This proportion of resistance follows on from the 1st quarter of 2016, which was previously reported as 26/88 (29.5%); and compares with none (0/54) in the same quarter of 2015, and 7/251 (2.8%) for all of 2015. None of these strains had high level resistance, 24/25 (96%) were resistant to penicillin (beta-lactamase producing); and all were sensitive to ceftriaxone and ciprofloxacin. Enhanced surveillance, case reviews, and genotypic investigations are in process in South Australia with further results to follow.

In the Australian Capital Territory there were 7 of 35 (20%) isolates that were resistant to azithromycin compared with 2 of 22 (9.1%) in the previous quarter, and none (0/69) in 2015. The other states that reported an increase in the proportion of *N. gonorrhoeae* isolates with resistance to azithromycin when compared with the previous quarter, and when compared with 2015, were New South Wales (5/590, 0.8% in 1st quarter of 2016, and

43/1,905, 2.3% in 2015) and Victoria (7/445, 1.6% in the 1st quarter of 2016, and 30/1,695, 1.8% in 2015). Queensland and urban/rural Western Australia reported a decrease when compared with the previous quarter, and with 2015. The other states reported similar results when compared with the previous quarter, and with 2015.

Penicillin

Penicillin resistant *N. gonorrhoeae* are defined as those isolates with a MIC to penicillin equal to or greater than 1.0 mg/L. Penicillin resistance includes penicillinase producing *N. gonorrhoeae* (PPNG), and *N. gonorrhoeae* that have chromosomally mediated resistance to penicillin (CMRP). In certain areas of the Northern Territory and Western Australia, which are classified as remote, a treatment regimen based on oral amoxicillin, probenecid and azithromycin is used. Due to the distance specimens must travel in these remote regions to a laboratory, low numbers of cultures are collected, and thus, by necessity, nucleic acid amplification testing (NAAT) is used. In remote Western Australia the introduction of a targeted NAAT, developed by the NNN to detect PPNG, is in use to enhance surveillance.[7,8]

Ciprofloxacin

Ciprofloxacin resistance includes isolates with an MIC to ciprofloxacin equal to or greater than 1.0 mg/L.

Dual therapy of ceftriaxone plus azithromycin is the recommended treatment for gonorrhoea as a strategy to temper development of more widespread resistance.[8] Patients with infections in extragenital sites, where the isolate has decreased susceptibility to ceftriaxone, are recommended to have test of cure cultures collected. Continued surveillance to monitor *N. gonorrhoeae* with elevated MIC values, coupled with sentinel site surveillance in high risk populations remains important to inform therapeutic strategies, to identify incursion of resistant strains, and to detect instances of treatment failure.

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Australian Meningococcal Surveillance Programme, 1 July to 30 September 2016

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

Introduction

The reference laboratories of the National Neisseria Network, Australia report laboratory data on invasive meningococcal disease (IMD) cases confirmed by laboratory testing using culture and non-culture based techniques for the Australian Meningococcal Surveillance Programme (AMSP). Culture positive cases, where Neisseria meningitidis is grown from a normally sterile site or skin lesions, and non-culture based diagnoses, derived from results of nucleic acid amplification testing (NAAT) and serological techniques, are defined as IMD according to Public Health Laboratory Network definitions. Data contained in quarterly reports are usually restricted to a description of the numbers of cases by jurisdiction and serogroup, where known. Some minor corrections to data in the Table may be made in subsequent reports if additional data are received. A full analysis of laboratory confirmed cases of IMD in each calendar year is contained in the AMSP annual reports published in *Communicable Diseases Intelligence*. For more information see *Commun Dis Intell* 2016;40(1):E179.

Results

Of note in this quarter of 2016 is the number and proportion of IMD caused by serogroup W. This is particularly evident in New South Wales and Victoria, which have both shown substantial increases in both the number and proportion of serogroup W cases compared with previous years. In the years 2007–2011 the proportion of IMD caused by serogroup W in Australia ranged from 1.8% to 4.5%, and increased to 8.6% to 9.9% in 2013–2014. In 2015 this increased markedly to 31/81 (21.4%) IMD cases in Australia. In 2015 25 serogroup W IMD strains were genotyped, and 81% were sequence type (ST)-11, and had the *porA* antigen encoding gene type P1.5,2, the same genotype as the hypervirulent strain reported in the United Kingdom and South America since 2009. Nationally, enhanced surveillance strategies including whole genome sequencing and phylogenetic inference has been applied to the recent emergence of *N. meningitidis* serogroup W in Australia.

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Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 July to 30 September 2016, by serogroup and state or territory

State or territory	Year	Serogroup A Q3	A YTD	B Q3	B YTD	C Q3	C YTD	Y Q3	Y YTD	W135 Q3	W135 YTD	ND Q3	ND YTD	All Q3	All YTD
Australian Capital Territory	2016	0	0	1	1	0	0	0	0	1	1	0	0	2	2
	2015	0	0	0	1	0	0	1	1	0	0	0	0	1	2
New South Wales	2016	0	0	8	18	0	1	6	9	13	22	1	3	28	53
	2015	0	0	4	16	0	1	5	7	4	6	0	1	13	31
Northern Territory	2016	0	0	0	2	0	0	0	0	0	0	0	0	0	2
	2015	0	0	0	1	0	0	0	0	0	0	0	0	0	1
Queensland	2016	0	0	6	11	0	0	2	8	5	10	1	3	14	32
	2015	0	0	7	16	0	0	1	2	3	3	0	1	11	22
South Australia	2016	0	0	6	17	0	0	0	0	2	2	0	0	8	19
	2015	0	0	15	24	0	0	0	0	0	0	0	0	15	24
Tasmania	2016	0	0	0	0	0	0	0	0	2	4	0	0	2	4
	2015	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Victoria	2016	0	0	2	9	0	1	2	4	8	23	1	1	13	38
	2015	0	0	7	23	0	0	4	6	6	12	0	0	17	41
Western Australia	2016	0	0	1	3	0	0	0	0	6	9	0	0	7	12
	2015	0	0	3	7	0	0	1	2	3	3	0	0	7	12
Total	2016	0	0	24	61	0	2	10	21	37	71	3	7	74	162
	2015	0	0	36	88	0	1	12	18	16	24	0	2	64	133

Australian Sentinel Practices Research Network, 1 July to 30 September 2016

Monique B-N Chilver, Daniel Blakeley, Nigel P Stocks for the Australian Sentinel Practices Research Network

Introduction

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

The network consists of general practitioners and nurse practitioners, Australia wide, who report syndromic presentations on a number of defined medical conditions each week. ASPREN was established in 1991 to provide a rapid monitoring scheme for infectious diseases that can inform public health officials of the epidemiology of pandemic threats in the early stages of a pandemic, as well as play a role in the evaluation of public health campaigns and research of conditions commonly seen in general practice. Reporters currently submit data via automated data extraction from patient records, web-based data collection or paper form.

In 2010, virological surveillance was established allowing ASPREN practitioners to collect nasal swab samples for laboratory viral testing of a proportion of influenza-like illness (ILI) patients for a range of respiratory viruses including influenza A and influenza B. In 2016, practitioners are instructed to swab 20% of all patients presenting with an ILI.

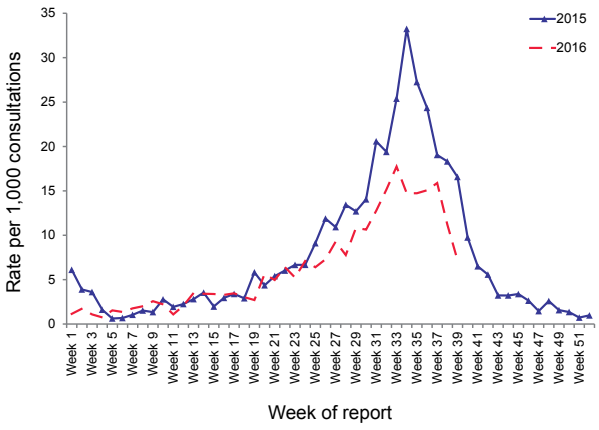
The list of conditions reported is reviewed annually by the ASPREN management committee. In 2016, 4 conditions are being monitored. They include ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, published in *Commun Dis Intell* 2016;40(1):E180.

Results

Sentinel practices contributing to ASPREN were located in all 8 states and territories in Australia. A total of 240 general practitioners regularly contributed data to ASPREN in the 3rd quarter of 2016. Each week an average of 208 general practitioners provided information to ASPREN at an average of 15,265 (range 11,414 to 16,914) consultations per week and an average of 259 (range 165 to 367) notifications per week (all conditions).

ILI rates reported from 1 July to 30 September 2016 averaged 12.5 cases per 1,000 consultations (range 7.2 to 17.7 cases per 1,000 consultations) weighted / 12.6 cases per 1,000 consultations (range 7.7 to 18.2 cases per 1,000 consultations) unweighted. This was lower than the rates in the same reporting period in 2015, which averaged 19.6 cases per 1,000 consultations (range 10.9 to 33.2 cases per 1,000 consultations, Figure 1) weighted / 15.1 cases per 1,000 consultations (range 10.8 to 23.1 cases per 1,000 consultations, Figure 1) unweighted.

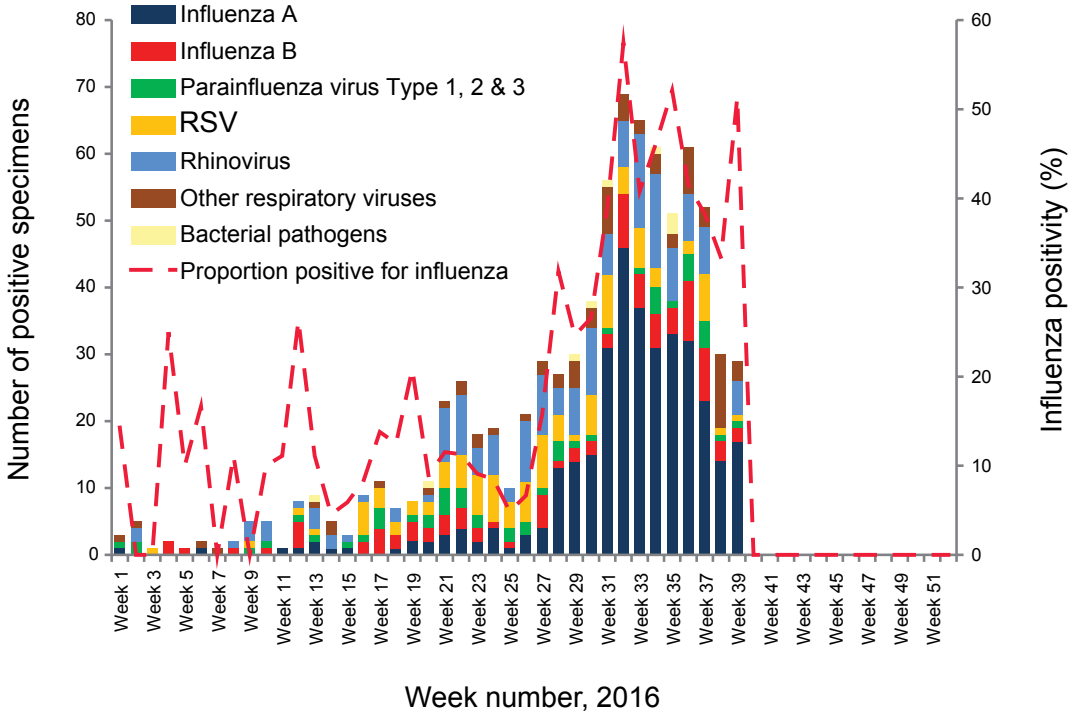
Figure 1: Consultation rates for influenza-like illness, ASPREN, 2015 and 1 January to 30 September 2016, by week of report



The ASPREN ILI swab testing program continued in 2016 with 929 tests being undertaken from 1 July to 30 September. The most commonly reported virus during this period was influenza A (33.4% of all swabs performed, Figure 2), with the 2nd most common virus being rhinovirus (10.5%).

From the beginning of 2016 to the end of week 39, 428 cases of influenza were detected with 340 of these typed as influenza A (22%) with the remaining 88 being influenza B (5.7%) (Figure 2).

Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January to 30 September 2016, by week of report



During this reporting period, consultation rates for gastroenteritis averaged 3.9 cases per 1,000 consultations (range 2.7 to 5 cases per 1,000, Figure 3). This was similar to the rates in the same reporting period in 2015 where the average was 3.9 cases per 1,000 consultations (range 2.5 to 6.4 cases per 1,000).

Varicella infections were reported at a similar rate for the 3rd quarter of 2016 compared with the same period in 2015. From 1 July to 30 September 2016, recorded rates for chickenpox averaged 0.2 cases per 1,000 consultations (range 0.0 to 0.5 cases per 1,000 consultations, Figure 4).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 2015 and 1 January to 30 September 2016, by week of report

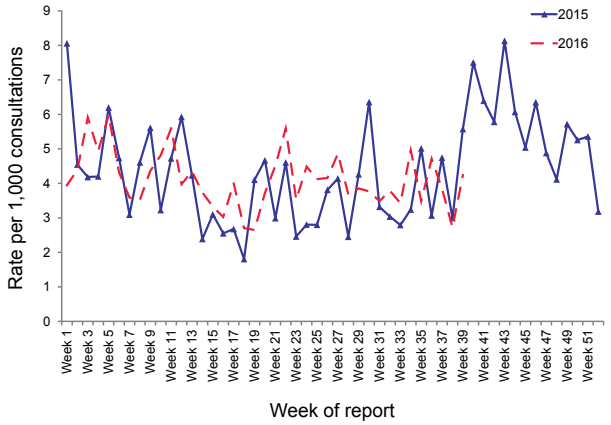
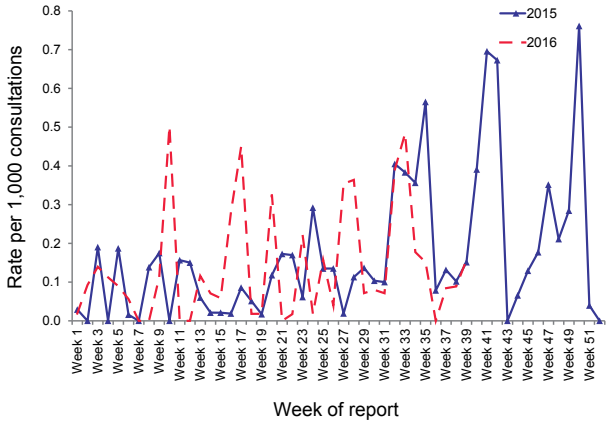
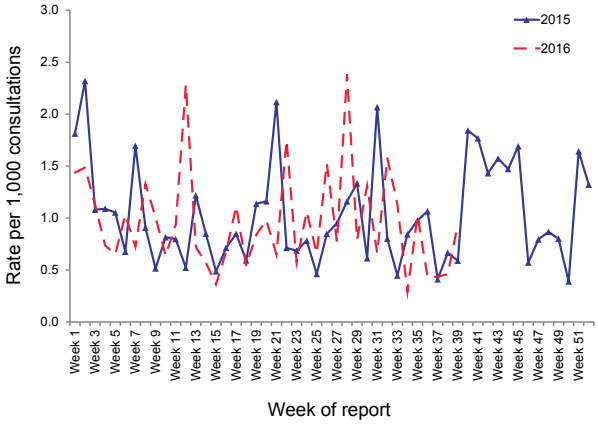


Figure 4: Consultation rates for chickenpox, ASPREN, 2015 and 1 January to 30 September 2016, by week of report



In the 3rd quarter of 2016, reported rates for shingles averaged 0.9 cases per 1,000 consultations (range 0.3 to 2.4 cases per 1,000 consultations, Figure 5). This was similar to the rates in the same reporting period in 2015 where the average shingles rate was 0.9 cases per 1,000 consultations (range 0.4 to 2.1 cases per 1,000 consultations).

Figure 5: Consultation rates for shingles, ASPREN, 2015 and 1 January to 30 September 2016, by week of report



Invasive pneumococcal disease surveillance, 1 April to 30 June 2016

Anna Glynn-Robinson, Kate Pennington, Cindy Toms and the Enhanced Invasive Pneumococcal Disease Surveillance Working Group, for the Communicable Diseases Network Australia

Summary

The number of notified cases of invasive pneumococcal disease (IPD) in the 2nd quarter of 2016 was substantially more than the previous quarter and marginally more than the number of notified cases in the 2nd quarter of 2015. Overall, the decline in disease due to the serotypes targeted by the 13-valent pneumococcal conjugate vaccine (13vPCV) has been maintained across all age groups, since the 13vPCV replaced the 7-valent pneumococcal conjugate vaccine (7vPCV) in the childhood immunisation program from July 2011.

Key points

In the 2nd quarter of 2016, there were 442 cases of IPD reported to the National Notifiable Disease Surveillance System, a 10% increase when compared with the same period in 2015 (n=401) (Table 1). The observed increase in notifications in the 2nd quarter of 2016 was attributable to the 65 years or over age group, in which case numbers increased by 30% (n=167) when compared with the same reporting period of 2015 (n=127). In the 2nd quarter of 2016 the most common pneumococcal serotypes causing IPD were 3 (9.3%), 19A (8.4%), 22F (6.5%) and 23B (5.4%) (Table 2). Compared with the 1st quarter of 2016, these proportions were similar for serotypes 3 (9.3%) and 22F (6.0%), but higher for serotype 19A (6.0%).

In non-Indigenous Australians, the number of notified cases was highest in children aged less than 5 years and older adult age groups, especially those aged 65 years or over (Table 3). In Indigenous Australians, case numbers were highest in children aged less than 5 years and the 50–54 years age group. The proportion of cases reported as Indigenous this quarter (8%; 34/442) was less than the proportion observed in the 2nd quarter of 2015 (15%; 60/401), and the proportion reported in the 1st quarter of 2016 (14%; 26/182).

In children aged less than 5 years, there were 71 cases of IPD reported, representing 16% of all cases reported in this quarter. The number of cases notified in this age group was similar in this reporting period when compared with the 2nd quarter of 2015 (68/401). Of those cases with known serotype, 37% (20/54) were due to a serotype included in the 13vPCV compared with 45% (28/62) of cases in the 2nd quarter of 2015 (Figure 1). Serotypes 3, 19A and 23B were the most common serotypes affecting this age group in this quarter, noting that serotype 3 and 19A are included in the 13vPCV (Table 2).

Table 1: Notified cases of invasive pneumococcal disease, Australia, 1 April to 30 June 2016, by Indigenous status, serotype completeness and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total 2nd qtr 2016	Total 1st qtr 2016	Total 2nd qtr 2015
Indigenous	0	5	7	6	2	3	3	8	34	26	60
Non-Indigenous	7	112	4	70	31	14	85	30	353	141	299
Not stated / Unknown	0	30	0	1	0	0	23	1	55	15	42
Total	7	147	11	77	33	17	111	39	442	182	401
Indigenous status completeness* (%)	100	80	100	99	100	100	79	97	88	92	90
Serotype completeness† (%)	100	80	100	92	3	94	92	90	81	92	95

* 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-typeable. 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.

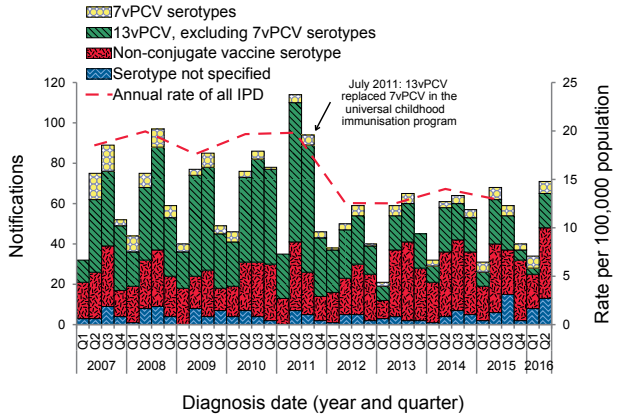
Table 2: Distribution of serotypes causing invasive pneumococcal disease in notified cases, Australia, 1 April to 30 June 2016, by age group

Serotype	Age group			Serotype total
	Under 5 years	5–64 years	Over 65 years	
3	7	20	14	41
19A	8	20	9	37
22F	5	11	13	29
23B	7	9	8	24
9N	3	14	6	23
15A	2	8	7	17
19F	4	5	7	16
16F	2	7	6	15
11A	3	6	5	14
15C	4	6	3	13
23A	1	6	6	13
33F	1	6	5	12
6C	–	6	6	12
7F	–	7	5	12
10A	2	4	4	10
8	–	6	3	9
15B	2	2	4	8
38	–	2	4	6
35B	–	1	4	5
Other*	3	27	14	44
Unknown†	17	32	33	82
Total	71	205	166	442

* Serotypes that only occur in less than 5 cases per quarter are grouped as 'Other' and include 'non-typeable' isolates this quarter.

† 'Serotype unknown' includes those serotypes reported as 'no isolate', 'not referred', 'not viable', 'typing pending' and 'untyped'.

Figure 1: Notifications and annual rates* of invasive pneumococcal disease in children aged less than 5 years, Australia, 2007 to 30 June 2016, by vaccine serotype group



* Annual rates are shown on Q2, excluding 2016.

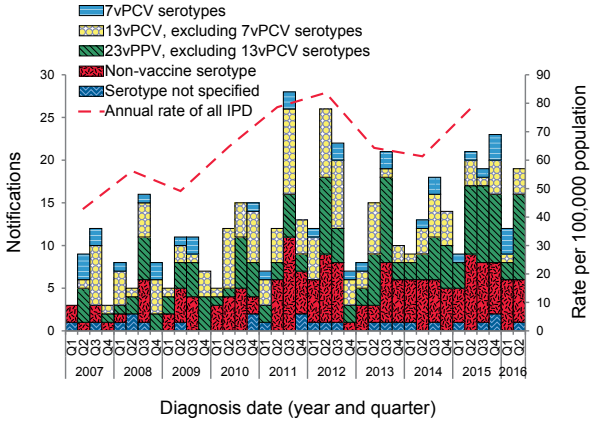
In the 2nd quarter of 2016, there were 14 cases reported in fully vaccinated children aged less than 5 years who were considered to be 13vPCV failures. Serotype 19A was reported as the cause of disease in 6 of these cases, followed by serotype 3 with 5 cases (Table 4).

Among Indigenous Australians aged 50 years or over, there were 20 cases of IPD reported this quarter. Of those cases with a reported serotype, 67% (12/18) were due to a serotype included in the 23-valent polysaccharide pneumococcal vaccine (23vPPV) (Figure 2). The number of notified cases of IPD in this age group increased by 66% compared with the number reported in the previous quarter (n=12), but was similar to the 2nd quarter of 2015 (n=21). Compared with the previous quarter, the proportion of cases due to serotypes included in the 23vPPV increased from 50% to 67% among cases with a known serotype.

Additionally, there was no apparent predominance of any serotype reported among this population group this quarter.

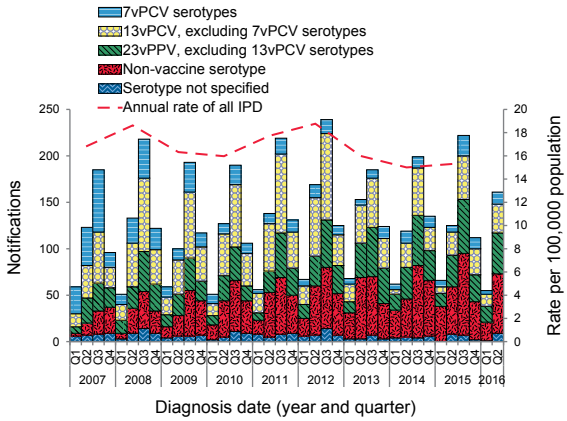
Among non-Indigenous Australians aged 65 years and over there were 161 cases of IPD reported this quarter. The number of notified cases of IPD in this age group almost tripled compared with the number reported in the previous quarter (n=55) and was 28% higher than the 2nd quarter of 2015 (n=125); however, the overall proportion of cases remained similar. Of those cases with a reported serotype, 60% (76/127) were due to a serotype included in the 23vPPV (Figure 3), which was a small reduction when compared with the previous quarter (63%). For this quarter, serotypes 3 (n=33) and 19A (n=31) were the predominant serotypes for this population group, noting that serotype 3 and 19A are included in the 23vPPV.

Figure 2: Notifications and annual rates* of all invasive pneumococcal disease in Indigenous Australians aged 50 years or over, Australia, 2007 to 30 June 2016 , by vaccine serotype group



* Annual rates are shown on Q2, excluding 2016.

Figure 3: Notifications and annual rates* of all invasive pneumococcal disease in non-Indigenous Australians† aged 65 years or over, Australia, 2007 to 30 June 2016, by vaccine serotype group



* Annual rates are shown on Q2, excluding 2016.

† Non-Indigenous Australians includes cases reported with as non-Indigenous, not stated, blank or unknown.

Table 3: Notified cases of invasive pneumococcal disease, Australia 1 April to 30 June 2016, by Indigenous status and age group

Age group	Indigenous status			Total
	Indigenous	Non-Indigenous	Not reported*	
00–04	6	63	2	71
05–09	1	6	4	11
10–14	1	7	3	11
20–24	1	1	2	4
25–29	1	4	3	8
30–34	–	7	6	13
35–39	1	7	2	10
40–44	2	6	5	13
45–49	1	10	13	24
50–54	10	22	1	33
55–59	5	35	2	42
60–64	–	36	–	36
65+	5	149	12	166
Total	34	353	55	442

* Not reported is defined as not stated or unknown Indigenous status.

During this quarter there were 24 deaths attributed to a variety of IPD serotypes. Of these deaths, 22 occurred in non-Indigenous Australians, with a median age of 64 years (range 0 to 94 years) and 2 occurred in Indigenous Australians. There were 2 deaths reported in children less than 5 years of age, which were associated with serotype 3 and serotype 11A. Both of these cases were under the age of 2 months and therefore not eligible for vaccination.

Notes

The data in this report are provisional and subject to change as laboratory results and additional case information become available. More detailed data analysis of IPD in Australia and surveillance methodology are described in the IPD annual report series published in *Communicable Diseases Intelligence*.

Table 4: Characteristics of 13vPCV failures in children aged less than 5 years, Australia, 1 April to 30 June 2016

Age	Indigenous status	Serotype	Clinical category	Risk factor/s
11 months	Non-Indigenous	3	Pneumonia and other (pleural empyema)	No risk factor identified
1 year	Non-Indigenous	3	Pneumonia	Other
1 year	Non-Indigenous	19A	Pneumonia	No data available
2 years	Non-Indigenous	19A	Pneumonia	Other
2 years	Non-Indigenous	19A	Pneumonia	Childcare attendee
2 years	Non-Indigenous	19A	Bacteraemia	Childcare attendee
2 years	Indigenous	19F	Bacteraemia	Congenital or chromosomal abnormality
2 years	Unknown	19F	Bacteraemia	Immunocompromised
2 years	Non-Indigenous	3	Pneumonia and other (pleural effusion)	No risk factor identified
3 years	Non-Indigenous	3	Pneumonia	No data available
3 years	Non-Indigenous	19A	Pneumonia and other (other sterile site)	Childcare attendee
3 years	Non-Indigenous	19A	Bacteraemia	Childcare attendee
3 years	Non-Indigenous	3	Pneumonia	Childcare attendee
4 years	Non-Indigenous	19F	Other(other sterile site)	No data available

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, individuals with specific underlying conditions associated with increased risk of IPD and older Australians. More information on the scheduling of the pneumococcal vaccination can be found on the Immunise Australia Program website (www.immunise.health.gov.au).

In this report, a 'vaccine failure' is where a fully vaccinated child is diagnosed with IPD due to a serotype covered by the administered vaccine. 'Fully vaccinated' describes cases that have completed the primary course of the relevant vaccine(s) required for their age according to the most recent edition of *The Australian Immunisation Handbook,* at least 2 weeks prior to disease onset with at least 28 days between doses of vaccine. NB: A young child who has had all the required doses for their age but is not old enough to have completed the primary course would not be classified as fully vaccinated.

There are 3 pneumococcal vaccines available in Australia, each targeting multiple serotypes (Table 5). Note that in this report serotype analysis is generally grouped according to vaccine composition.

Follow-up of all notified cases of IPD is undertaken in all states and territories except New South Wales and Victoria who conduct targeted follow-up of notified cases aged under 5 years, and 50 years or over for enhanced data. Follow-up in notified cases of IPD is undertaken in Queensland in all areas except Metro South and Gold Coast Public Health Units who conduct targeted follow-up of notified cases for those aged under 5 years only.

Acknowledgements

Report prepared with the assistance of Rachael Corvisy on behalf of the Enhanced Invasive Pneumococcal Disease Surveillance Working Group.

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Table 5: *Streptococcus pneumoniae* serotypes targeted by pneumococcal vaccines

Serotypes	7- valent pneumococcal conjugate vaccine (7vPCV)	10-valent pneumococcal conjugate vaccine (10vPCV)	13-valent pneumococcal conjugate vaccine (13vPCV)	23-valent pneumococcal polysaccharide vaccine (23vPPV)
1		✓	✓	✓
2				✓
3			✓	✓
4	✓	✓	✓	✓
5		✓	✓	✓
6A			✓	
6B	✓	✓	✓	✓
7F		✓	✓	✓
8				✓
9N				✓
9V	✓	✓	✓	✓
10A				✓
11A				✓
12F				✓
14	✓	✓	✓	✓
15B				✓
17F				✓
18C	✓	✓	✓	✓
19A			✓	✓
19F	✓	✓	✓	✓
20				✓
22F				✓
23F	✓	✓	✓	✓
33F				✓

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Invasive pneumococcal disease surveillance, 1 July to 30 September 2016

Rachael Corvisy, Jennie Hood and the Enhanced Invasive Pneumococcal Disease Surveillance Working Group, for the Communicable Diseases Network Australia

Summary

The number of notified cases of invasive pneumococcal disease (IPD) in the 3rd quarter of 2016 was substantially more than the previous quarter and marginally more than the number of notified cases in the 3rd quarter of 2015. Overall, the decline in disease due to the serotypes targeted by the 13-valent pneumococcal conjugate vaccine (13vPCV) has been maintained across all age groups since the 13vPCV replaced the 7-valent pneumococcal conjugate vaccine (7vPCV) in the childhood immunisation program from July 2011.

Key points

In the 3rd quarter of 2016, there were 637 cases of IPD reported to the National Notifiable Disease Surveillance System, an 8% increase when compared with the same period in 2015 (n=589) (Table 1). In the 3rd quarter of 2016 the most common pneumococcal serotypes causing IPD were 3 (8.9%), 9N (7.5%), 19A (6.1%) and 23A (6.0%) (Table 2). Compared with the 2nd quarter of 2016, there was a slight decrease in serotypes 3 (9.3%) and 19A (8.4%) and an increase in serotypes 9N (5.2%) and 23A (2.9%).

In non-Indigenous Australians, the number of notified cases was highest in children aged less than 5 years and older adult age groups, especially those aged 60 years or over (Table 3). In Indigenous Australians, cases were highest in children aged less than 5 years and the 40–44 years age group. The proportion of cases reported as Indigenous this quarter (11%; 72/637) was the same as that observed in the 3rd quarter of 2015 (11%; 63/589), and higher than the proportion reported in the 2nd quarter of 2016 (7%; 32/439).

In children aged less than 5 years, there were 73 cases of IPD reported, representing 11% of all cases reported in this quarter. The number of cases notified in this age group was slightly higher in this reporting period when compared with the 3rd quarter of 2015 (10%; 58/589). Of those cases with known serotype, 35% (18/52) were due to a serotype included in the 13vPCV compared with 50% (22/44) of cases in the 3rd quarter of 2015 (Figure 1). Serotypes 3, 19A, 19F and 23B were the most common serotypes affecting this age group in this quarter, noting that serotype 3, 19A and 19F are included in the 13vPCV (Table 2).

In the 3rd quarter of 2016, there were 10 cases reported in fully vaccinated children aged less than 5 years who were considered to be 13vPCV failures.

Table 1: Notified cases of invasive pneumococcal disease, Australia, 1 July to 30 September 2016, by Indigenous status, serotype completeness and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total 3rd qtr 2016	Total 2nd qtr 2016	Total 3rd qtr 2015
Indigenous	0	11	11	19	5	0	0	26	72	32	63
Non-Indigenous	14	155	2	86	50	12	115	67	501	368	455
Not stated / Unknown	0	38	0	0	0	0	26	0	64	39	71
Total	14	204	13	105	55	12	141	93	637	439	589
Indigenous status completeness* (%)	100	81	100	100	100	100	82	100	90	91	88
Serotype completeness† (%)	100	91	92	97	64	92	89	94	90	93	93

* 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-typeable. 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.

Table 2: Distribution of serotypes causing invasive pneumococcal disease in notified cases, Australia, 1 July to 30 September 2016, by age group

Serotype	Age groups			Serotype total
	Under 5 years	5–64 years	Over 65 years	
3	9	26	22	57
9N	3	32	13	48
19A	4	20	15	39
23A	2	14	22	38
22F	–	20	17	37
19F	4	15	16	35
6C	1	7	22	30
23B	4	12	10	26
8	–	22	4	26
15A	2	9	11	22
11A	2	10	8	20
33F	2	13	4	19
16F	–	9	9	18
35B	3	5	9	17
7F	–	15	2	17
15B	1	4	6	11
17F	1	8	2	11
10A	1	5	4	10
24F	2	2	4	8
31	–	4	4	8
15C	2	1	4	7
38	1	2	4	7
9V	–	6	1	7
10F	1	5	–	6
24	2	1	2	5
35F	1	1	3	5
4	–	4	1	5
7C	1	1	3	5
Unknown	21	34	10	65
Other	3	15	10	28
Total	73	322	242	637

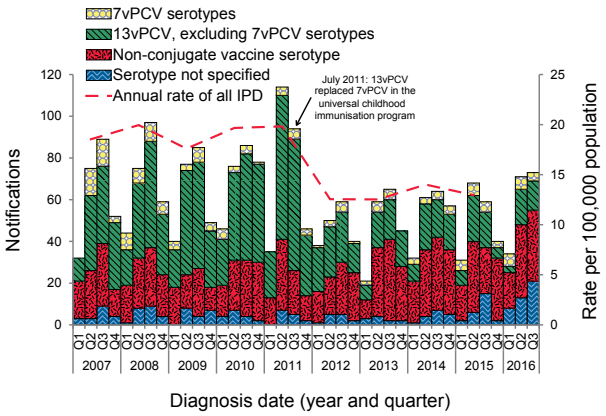
* Serotypes that only occur in less than 5 cases per quarter are grouped as 'Other' and include 'non-typeable' isolates this quarter.

† 'Serotype unknown' includes those serotypes reported as 'no isolate', 'not referred', 'not viable', 'typing pending' and 'untyped'.

Serotype 19A was the most common serotype associated with 13vPCV failure reported this quarter (n=4), followed by serotypes 3 and 19F (n=3 each; Table 4).

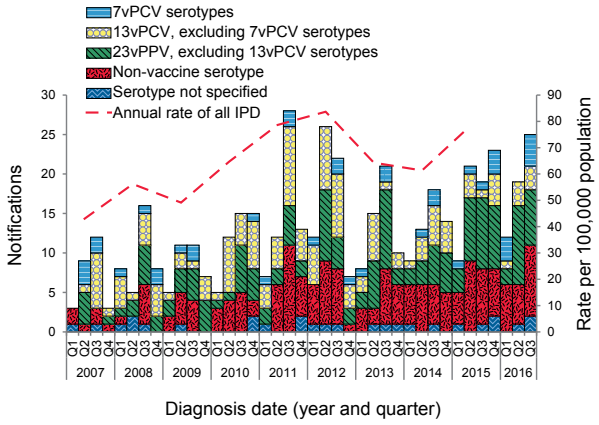
Among Indigenous Australians aged 50 years or over, there were 25 cases of IPD reported this quarter. Of those cases with a reported serotype, 61% (14/23) were due to a serotype included in the 23-valent polysaccharide pneumococcal vaccine (23vPPV) (Figure 2). The number of notified cases of IPD in this age group increased by 32% compared with the number reported in the previous quarter (n=19) and the 3rd quarter of 2015 (n=19). Compared with the previous quarter, the proportion of cases due to serotypes included in the 23vPPV decreased from 72% to 61% among cases with a known serotype. The most common serotypes causing disease in this group this quarter were serotypes 3 and 8 (n=3 each); these serotypes are included in the 23vPPV.

Figure 1: Notifications and annual rates* of invasive pneumococcal disease in children aged less than 5 years, Australia, 2007 to 30 September 2016, by vaccine serotype group



* Annual rates are shown on Q2, excluding 2016.

Figure 2: Notifications and annual rates* of all invasive pneumococcal disease in Indigenous Australians aged 50 years or over, Australia, 2007 to 30 September 2016 by vaccine serotype group



* Annual rates are shown on Q2, excluding 2016.

Table 3: Notified cases of invasive pneumococcal disease, Australia 1 July to 30 September, by Indigenous status and age group

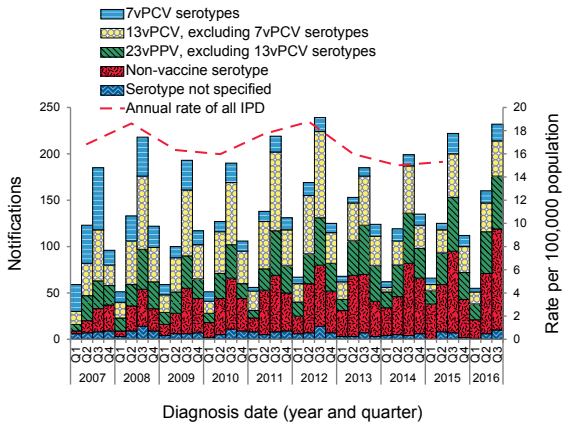
Age group	Indigenous status			Total
	Indigenous	Non-Indigenous	Not reported*	
00–04	12	60	1	73
05–09	2	16	3	21
10–14	–	8	1	9
15–19	4	5	2	11
20–24	3	7	2	12
25–29	1	6	8	15
30–34	7	13	7	27
35–39	3	15	7	25
40–44	10	9	11	30
45–49	5	11	7	23
50–54	3	33	2	38
55–59	7	32	1	40
60–64	6	60	5	71
65+	9	226	7	242
Total	72	501	64	637

* Not reported is defined as not stated or unknown Indigenous status.

Among non-Indigenous Australians aged 65 years or over there were 233 cases of IPD reported this quarter. The number of notified cases of IPD in this age group increased by 46% when compared with the previous quarter (n=160) but was similar to the number reported in the 3rd quarter of 2015 (n=222). Of those cases with a reported serotype, 51% (113/222) were due to a serotype included in the 23vPPV (Figure 3), which was a small reduction when compared with the previous quarter (58%). For this quarter, serotypes 6C (n=22), 3 (n=21) and 23A (n=21) were the predominant serotypes for this population group.

During this quarter there were 49 deaths attributed to a variety of IPD serotypes. Of these deaths, 44 occurred in non-Indigenous Australians, with a median age of 69 years (range 0–95 years). A total of 5 deaths occurred in Indigenous Australians.

Figure 3: Notifications and annual rates* of all invasive pneumococcal disease in non-indigenous Australians† aged 65 years or over, Australia, 2007 to 30 September 2016 , by vaccine serotype group



* Annual rates are shown on Q2, excluding 2016.

† Non-Indigenous Australians includes cases reported with as non-Indigenous, not stated, blank or unknown.

Table 4: Characteristics of 13vPCV failures in children aged less than 5 years, Australia, 1 July to 30 September 2016

Age	Indigenous status	Serotype	Clinical category	Risk factor/s
7 months	Indigenous	19A	Other, septic arthritis	No data available
1 year	Indigenous	19A	Bacteraemia	No data available
1 year	Non-Indigenous	19F	Unknown	No risk factor identified
1 year	Non-Indigenous	19F	Bacteraemia	No data available
2 years	Non-Indigenous	3	Pneumonia	No data available
2 years	Non-Indigenous	3	Pneumonia	Other
2 years	Non-Indigenous	19A	Pneumonia	No risk factor identified
2 years	Non-Indigenous	19F	Pneumonia	Premature (<37 weeks gestation), Chronic Illness, Childcare attendee
3 years	Indigenous	19A	Bacteraemia	Immunocompromised, Chronic illness, Other
4 years	Non-Indigenous	3	Pneumonia	No risk factor identified

There were 2 deaths reported in children less than 5 years of age, which were associated with serotype 6C and serotype 11A. One of these cases was under the age of 2 months and therefore not eligible for vaccination.

Notes

The data in this report are provisional and subject to change as laboratory results and additional case information become available. More detailed data analysis of IPD in Australia and surveillance methodology are described in the IPD annual report series published in *Communicable Diseases Intelligence*.

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, individuals with specific underlying conditions associated with increased risk of IPD and older Australians. More information on the scheduling of the pneumococcal vaccination can be found on the Immunise Australia Program website (www.immunise.health.gov.au).

In this report, a 'vaccine failure' is where a fully vaccinated child is diagnosed with IPD due to a serotype covered by the administered vaccine. 'Fully vaccinated' describes cases that have completed the primary course of the relevant vaccine(s) required for their age according to the most recent edition of *The Australian Immunisation Handbook*, at least 2 weeks prior to disease onset with at least 28 days between doses of vaccine. NB: A young child who has had all the required doses for their age but is not old enough to have completed the primary course would not be classified as fully vaccinated.

There are 3 pneumococcal vaccines available in Australia, each targeting multiple serotypes (Table 5). Note that in this report serotype analysis is generally grouped according to vaccine composition.

Follow-up of all notified cases of IPD is undertaken in all states and territories except New South Wales and Victoria who conduct targeted follow-up of notified cases aged under 5 years, and 50 years or over for enhanced data. Follow-up in notified cases of IPD is undertaken in Queensland in all areas except Metro South and Gold Coast Public Health Units who conduct targeted follow-up of notified cases for those aged under 5 years only.

Acknowledgements

Report prepared with the assistance of Mark Trungove on behalf of the Enhanced Invasive Pneumococcal Disease Surveillance Working Group.

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Table 5: *Streptococcus pneumoniae* serotypes targeted by pneumococcal vaccines

Serotypes	7- valent pneumococcal conjugate vaccine (7vPCV)	10-valent pneumococcal conjugate vaccine (10vPCV)	13-valent pneumococcal conjugate vaccine (13vPCV)	23-valent pneumococcal polysaccharide vaccine (23vPPV)
1		✓	✓	✓
2				✓
3			✓	✓
4	✓	✓	✓	✓
5		✓	✓	✓
6A			✓	
6B	✓	✓	✓	✓
7F		✓	✓	✓
8				✓
9N				✓
9V	✓	✓	✓	✓
10A				✓
11A				✓
12F				✓
14	✓	✓	✓	✓
15B				✓
17F				✓
18C	✓	✓	✓	✓
19A			✓	✓
19F	✓	✓	✓	✓
20				✓
22F				✓
23F	✓	✓	✓	✓
33F				✓

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The CDI staff wish to thank the following reviewers for their valued assistance throughout the year.
Ross Andrews, Frances Birrell, Sandra Carlson, Anna Colwell, Paul Corben, Chris Coulter, Craig Dalton, Stephanie Davis, Justin Denholm, David Durrheim, Mark Ferson, Marianne Gale, Katherine Gibney, Anna Glynn-Robinson, Alan Hampson, Sonia Harmen, Rebecca Hundy, Vicki Krause, Stephen Lambert, Gary Lum, Helen Marshall, Peter Massey, Bridget O'Connor, Rhonda Owen, Mahomed Patel, Nevada Pingault, Gayle Pollard, Razlyn Rashim, Alice Richardson, Katrina Roper, Dave Spratt, Liza Szabo, Ee Laine Tay, Kathryn Taylor, Stephanie Williams

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