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Original articles

E195 **An evaluation of the use of short message service during an avian influenza outbreak on a poultry farm in Young**
Lisa M Stephenson, Janice S Biggs, Vicky Sheppeard, Tracey L Oakman

Short reports

E202 **Q fever and contact with kangaroos in New South Wales**
James Flint, Craig B Dalton, Tony D Merritt, Stephen Graves, John K Ferguson, Maggi Osbourn, Keith Eastwood, David N Durrheim

E204 **Estimates of influenza vaccine coverage from Victorian surveillance systems based in the community, primary care and hospitals**
Benjamin Coghlan, Heath A Kelly, Sandra J Carlson, Kristina A Grant, Karin Leder, Craig B Dalton, Allen C Cheng

Annual reports

E207 **Creutzfeldt-Jakob disease surveillance in Australia: update to December 2014**
Genevieve M Klug, Alison Boyd, Shannon Sarros, Christiane Stehmann, Marion Simpson, Catriona McLean, Colin L Masters, Steven J Collins

E216 **Australian Paediatric Surveillance Unit annual report, 2014**
Marie Deverell, Yvonne Zurynski, Elizabeth Elliott, and all chief investigators of APSU surveillance studies

E221 **Australian Meningococcal Surveillance Programme annual report, 2014**
Monica M Lahra, Rodney P Enriquez

E229 **Australian Group on Antimicrobial Resistance Australian Enterobacteriaceae Sepsis Outcome Programme annual report, 2014**
Jan M Bell, John D Turnidge, Geoffrey W Coombs, Denise A Daley, Thomas Gottlieb, Jenny Robson, Narelle George

E236 **Australian Group on Antimicrobial Resistance Australian Enterococcal Sepsis Outcome Programme annual report, 2014**
Geoffrey W Coombs, Denise A Daley, Yung Thin Lee, Stanley Pang, Julie C Pearson, J Owen Robinson, Paul DR Johnson, Despina Kotsanas, Jan M Bell, John D Turnidge for the Australian Group on Antimicrobial Resistance

E244 **Australian Group on Antimicrobial Resistance Australian *Staphylococcus aureus* Sepsis Outcome Programme annual report, 2014**
Geoffrey W Coombs, Denise A Daley, Yung Thin Lee, Julie C Pearson, J Owen Robinson, Graeme R Nimmo, Peter Collignon, Benjamin P Howden, Jan M Bell, John D Turnidge for the Australian Group on Antimicrobial Resistance

E255 **Australian trachoma surveillance annual report, 2013**
Carleigh S Cowling, Bette C Liu, Thomas L Snelling, James S Ward, John M Kaldor, David P Wilson

E267 **Invasive pneumococcal disease in Australia, 2011 and 2012**
Cindy Toms, Rachel de Kluyver and the Enhanced Invasive Pneumococcal Disease Surveillance Working Group for the Communicable Diseases Network Australia

Policy and guidelines

E285 **Revised surveillance case definitions**
E285 Brucellosis
E286 Flavivirus infection (unspecified) including Zika virus case definition

E289 **New surveillance case definition**
E289 Middle East Respiratory Syndrome Coronavirus (MERS-CoV)

Continued on back page

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Original article

An evaluation of the use of short message service during an avian influenza outbreak on a poultry farm in Young

Lisa M Stephenson, Janice S Biggs, Vicky Sheppeard, Tracey L Oakman

Abstract

In 2013 an avian influenza outbreak occurred in a large poultry farm in Young (approximately 2 hours north-west of Canberra.) The responsible strain was H7N2, which is highly pathogenic and can affect humans. Daily surveillance was required for those individuals who were possibly exposed. This was conducted through the use of daily message through the short message service (SMS). A total of 55 people were identified as having had high risk exposure and requiring monitoring during the surveillance period from 16 to 25 October 2013. A SMS message was sent daily to each contact within 2 groups. (Group 1 were contacts who agreed to take Tamiflu prophylaxis, and Group 2 were contacts who were under surveillance but declined Tamiflu prophylaxis). The average daily response rate for SMS was 66% (median 75%) over a 9 day period. Of those who nominated to receive the daily SMS 98% confirmed they'd received the SMS and it reminded them to take their Tamiflu medication. The public health unit (PHU) team found the use of SMS to be less time consuming than conducting telephone follow-up interviews. The PHU team believed that the use of the technology decreased the likelihood of additional staff being required to assist in the outbreak. Utilising SMS was a new initiative for the PHU and staff found it overall easy to use. These findings confirm there can be significant benefits to using SMS during a large surveillance activity. The application of SMS during this outbreak was estimated at 2.5 times more cost effective than telephone follow-ups and would substantially reduce staffing costs further in the event of a very large outbreak. *Commun Dis Intell* 2016;40(2):E195–E201.

Keywords: avian influenza, H7N2, surveillance, SMS

Introduction

In October 2013 an outbreak of avian influenza (AI) at a large poultry farm in Young, New South Wales was reported to the NSW Department of Primary Industries. The strain was H7N2, which was identified as highly pathogenic. Although most AI viruses do not cause disease in humans, strains H5 and H7 have adapted and spread through human populations.[1] On 15 October, Health Protection NSW requested the local public health unit (PHU) to identify close human contacts of the poultry, offer prophylactic treatment, and place these contacts under surveillance to identify any humans who may have contracted AI. Evidence suggests that some antiviral drugs, notably oseltamivir (Tamiflu), can reduce the duration of viral replication and improve prospects of survival.[2] Since 2010, there have been 376 reported cases of H5N1 in humans including 167 deaths worldwide.[3] The primary risk factor for human infection appears to be direct or indirect exposure to infected live or dead poultry or contaminated environments.

Outbreak setting

The poultry farm in Young is a well-established large poultry farm that houses over 400,000 birds across 8 free range and 6 caged sheds on site. The poultry are only for egg production. The free range sheds are about 700 metres from the cage sheds. In addition to the poultry, the farm has an onsite feed trucking business. The business employs over 103 workers and has a number of truck drivers and contractors visiting the farm. All persons who had close contact with the poultry 7 days prior to onset of illness in the birds were identified as at-risk for infection with AI.

Use of mobile phone technology

In Australia, there are 30.2 million telephones[4] for a population of 22.7 million[5] giving 133 connections per 100 citizens. Mobile phone short message service (SMS) is a communication tool with the potential to support health behaviours. It has been used in a variety of public health and medical monitoring programs such as in Western Australia were it was used to facilitate active monitoring of persons potentially exposed to Ebola virus returning from affected countries.[6] There has been some evidence that the use of SMS has made an effective contribution to advance behaviour change in

prevention programs, such as obesity prevention,[7] smoking cessation and physical activity programs.[8] SMS has also been used as an emergency warning system to provide timely information to disaster affected communities while also being used to rapidly collect information from these communities to improve aid delivery.[9] The Rural Fire Service in Australia uses SMS as an emergency warning system in fire affected areas. Unlike in health programs, a universal SMS is sent to the whole population within the vicinity of an affected area, rather than targeting specific individuals. Reasons for the success of SMS are; the cost is relatively low, its use is widespread, and it is applicable to every model of mobile phone.[9,10]

Surveillance of contacts of avian influenza

The Communicable Diseases Network Australia National Guidelines for Public Health Units for avian influenza recommends daily follow-up of close contacts for up to 10 days after the last exposure to infected birds or environments.[11] Contact management procedures involve offering antiviral prophylaxis, and PHU surveillance officers contacting cases and exposed persons through a daily telephone follow-up. The purpose of this call is to monitor for newly developed symptoms. In the event of a large outbreak this process can be time consuming and a challenge when contacts are likely to continue with their normal daily routines, such as working on the property, and are unavailable to speak on the telephone.

To assist with this large monitoring activity a web-based SMS was used to send messages to mobile telephones of all contacts identified as having high-risk exposure to AI. The purpose of the SMS was to remind identified contacts to self-monitor for influenza-like symptoms until the AI surveillance period expired, report to PHU if any symptoms developed and prompt contacts to take prophylactic Tamiflu medication, if prescribed.

Methods

The PHU identified 80 people at risk of exposure to AI on the farm, 25 were excluded due to either not having close contact with the poultry (within 1 metre of infected poultry without appropriate personal protective equipment) or not being on the property in the days prior to the birds becoming unwell, leaving 55 people identified as high risk of exposure and requiring monitoring during the incubation period from 16 to 25 October 2013. The PHU conducted an initial interview by telephone, arranged oseltamivir prophylaxis for contacts willing to take it, and a NSW Health fact sheet on AI was provided to all contacts and local clinicians. A list of contacts was compiled and sent to the NSW Office of Chief Health Officer (OCHO) Emergency Response Team who manage NSW Health access to a web-based SMS sending service provided by Prodocom® which would send their response in a excel format to the designated email address.

The team sending SMS checked the status of each sent item with a daily delivery report. If a message fails to be delivered, it can be resent. All messages have a maximum of 160 characteristics.

An SMS message was sent daily to each contact within 2 groups. Group 1 were contacts who agreed to take Tamiflu prophylaxis, and Group 2 were contacts who were under surveillance but declined Tamiflu prophylaxis). Originally, the SMS was sent at 10 am (17–19 October 2013) and then was moved to 8 am (20–25 October 2013) with the aim of it being received by the contacts before they started work. On 18 October 3 contacts requested daily telephone contact rather than SMS so were taken off the list leaving a total of 52 contacts in the SMS groups from 19 October. The 2 groups of contacts received a message as described in Box 1.

Box 1: Short message service messages sent to contacts

Group 1: Health reminder: take your flu tablet. Sick today? Cough, fever, sore throat, runny nose, red eyes or gastro? Pls reply Yes or No ASAP. Thanks, Lisa NSW Health

Group 2: Health check: sick today? Cough, fever, sore throat, runny nose, red eyes or gastro? Pls reply Yes or No ASAP. Thanks, Lisa NSW Health

Contacts were asked to respond to the message for the PHU surveillance officers to monitor symptoms. This request to respond to the SMS was discussed with the contacts during the initial telephone interview on the 16 October 2013.

Contacts from whom the PHU had not received a response by 4 pm on any day were telephoned to make sure that they had not developed any symptoms and asked why they had not responded to the SMS. Reasons for non-response were then analysed to determine if there was any way the PHU could improve the response rates by making response easier for those contacts.

Any contacts who replied 'yes' to having symptoms were telephoned immediately by the surveillance officer to discuss the symptoms and to arrange testing if symptoms were consistent with AI.

Data to evaluate the use of SMS monitoring for this outbreak were collected using 3 approaches:

1. A review of all surveillance records was conducted: SMS response data were extracted from the New South Wales Notifiable Conditions Information Management System for the surveillance period 17 to 25 October 2013. Data from the 16 October 2013 were excluded as telephone calls were conducted in the first instance to collect all relevant personal data.
2. Data were collected from contacts who were telephoned if they did not respond to the SMS (same day or > 2 days) as part of AI contact management processes by the PHU team. During their telephone follow-up 3 questions were asked:
    1. Did you receive a text message this morning? (YES/NO)
    2. Did it prompt you to take your flu tablets? (YES/NO)
    3. To help improve our services would you mind telling me why you didn't respond to the SMS?
3. Telephone interviews were conducted in November 2013 with contacts who had responded to the SMS on 8 or more days and were asked:
    1. Did you find receiving a SMS useful? (YES/NO)
    2. Did it prompt you to take your flu tablets? (YES/NO)
    3. Did it make you look out for signs (and symptoms) of flu? (YES/NO)
    4. Are there better ways to contact you? (YES/NO)
    5. If yes, what?
    6. Was responding to the SMS difficult?

Data were entered into Excel v2010 for analysis and basic descriptive statistical analysis was used. Open ended responses were coded and collapsed into themes using a simple content analysis.

Results

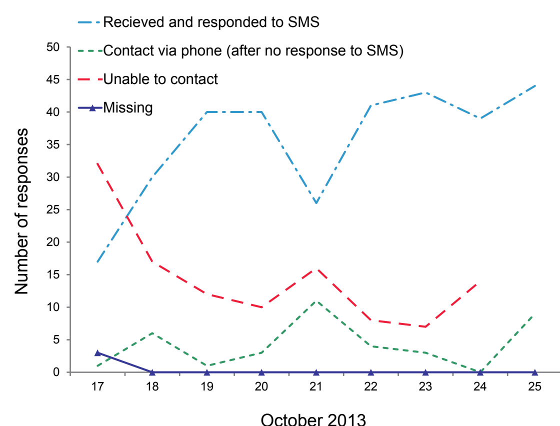
The average daily response rate for SMS was 66% (median 75%) over a 9 day period (Figure 1). The lowest response rate was on 18 October (2nd day using SMS, 32%). The second lowest response rate was 21 October 2013 (48%) mid-way through the surveillance period. On Monday 21 October 2013, 11 contacts were followed up by telephone, which was a 30% increase in telephone calls compared with an average day during the surveillance period. Excluding 21 October the use of the SMS increased over the 9 day period. The response rate of 66% reduced the PHU workload considerably. It was estimated that the use of SMS as a part of the contact management process was over 4 times more cost effective than telephone follow-ups (Box 2). These calculations were based upon a surveillance officer spending 1.5 hours a day administering the SMS process and conducting telephone follow-ups as needed. Conducting the process by telephone was estimated as taking 2 people 4 hours per day over a 9 day period. Overhead costs were excluded from the analysis.

Box 2: Cost analysis of short message service

SMS contact management: Middle grade HSM (1 surveillance officer) x 1.5 hours x 9 days = $590.62 + $152 for sending SMS messages = $742.62

Telephone contact management: Middle grade HSM per hour x 2 persons /4 hours = $3500 x 9 days = $31,500

Figure 1: Short message service responses from contacts over the 9 day surveillance period 17 to 25 October 2013



Non-respondents to short message service

Of the 52 contacts (from 18 October 2013) 7 were consistently un-contactable. Seventeen contacts were followed up by telephone (as no SMS response was received for more than 2 days). Of those who

opted to receive SMS, 98% confirmed they had received the SMS and it reminded them to take their Tamiflu medication.

The reasons for non-response are listed in the Table. Contacts not realising that a reply was required (n = 5) and finding it challenging to respond when working on site (n = 4) were the most common reasons for non-responses.

Responders to short message service

Identified contacts (n = 22, 79%) who frequently responded (≥ 8 days) to SMS over the 9 day period were interviewed over the phone (Figure 2). Over 80% of respondents found the SMS useful. Three respondents felt the fact sheet provided enough information but felt they should respond. Fourteen (64%) said it reminded them to take their Tamiflu tablets. The main reason why the remaining 8 respondents felt that the SMS did not remind them to take their medication was because the time the SMS was received didn't coincide with the time to take their tablets. Nonetheless, over 80% agreed that once they had received their SMS it did prompt them to look out for symptoms described in the SMS. A total of 7 contacts were tested after reporting through SMS that they had symptoms. All results were negative to H7N2. Two positive results were recorded for rhinovirus and respiratory syncytial virus. Two respondents praised the work of the PHU team as they felt 'looked after' during a difficult time.

In response to the question "Were there any associated challenges to participating in the SMS process?" 12 (55%) respondents confirmed they found the SMS process easy (Figure 3). However, the remaining 10 raised challenges to responding and these included; being unable to respond during work time (22%, n = 6) and having to remember to respond in the evening. This finding was consistent with the non-responders. Thirteen per cent (n = 3) said they were not clear if they had to keep responding over the 9 day period.

Figure 2: Responses to follow-up questions to contacts on the use of short message services during the outbreak on the poultry farm in Young

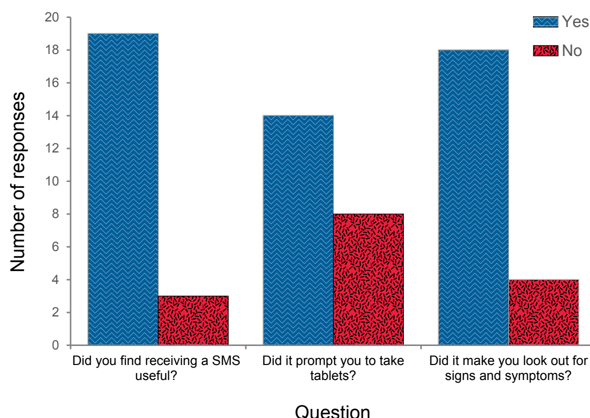
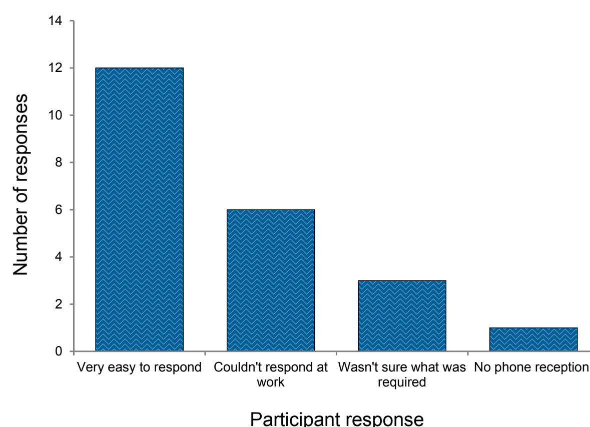


Figure 3: Interview responses identifying the challenges to responding to short message services



Process management

The process of sending out the SMS was reported by the OCHO team to be straightforward and the PHU team found it to be less time consuming than conducting telephone follow up interviews. The process used during this outbreak is described

Table: Contacts reason for not responding to short message service

	Frequency n=16	Proportion of total respondents %
Didn't realise /forgot to respond	5	30
Can't reply when at work	4	24
No service	2	23
No phone credit	3	23
Locked out of house	1	0.5
No receipt of text message	1	0.5

in Figure 4. The PHU team believed that the use of the technology decreased the need for additional staff being required to assist in the outbreak. However, management of the SMS involved liaising across 3 departments (OCHO, PHU and the Communicable Disease Branch) and still required manual administration from the OCHO to set the system up and the PHU team to manage the responses to SMSs, which impacted on workloads. The additional steps in the process for the PHU surveillance officer is described in Figure 5 and involved the SMS responses being sent to a surveillance officer's email address. This presented a problem when surveillance officers changed shifts and subsequently the OCHO had to redirect the emails. Further, the process used required manually matching the mobile phone numbers from the emails to the mobile numbers in a database to find the contact's details.

Impact on public health unit resources

It was estimated that the use of SMS for contact management was 2.5 times more cost effective than telephone follow-ups. These calculations were based upon a surveillance officer spending 1.5 hours a day administering the SMS process and conducting telephone follow ups as and when needed. Conducting the same number of telephone interviews was estimated as taking 2 people 4 hours per day over a 9 day period. Overhead costs were excluded from the analysis.

Figure 4: Process of using short message service as a surveillance tool during the avian influenza outbreak on a chicken farm in Young, October 2013

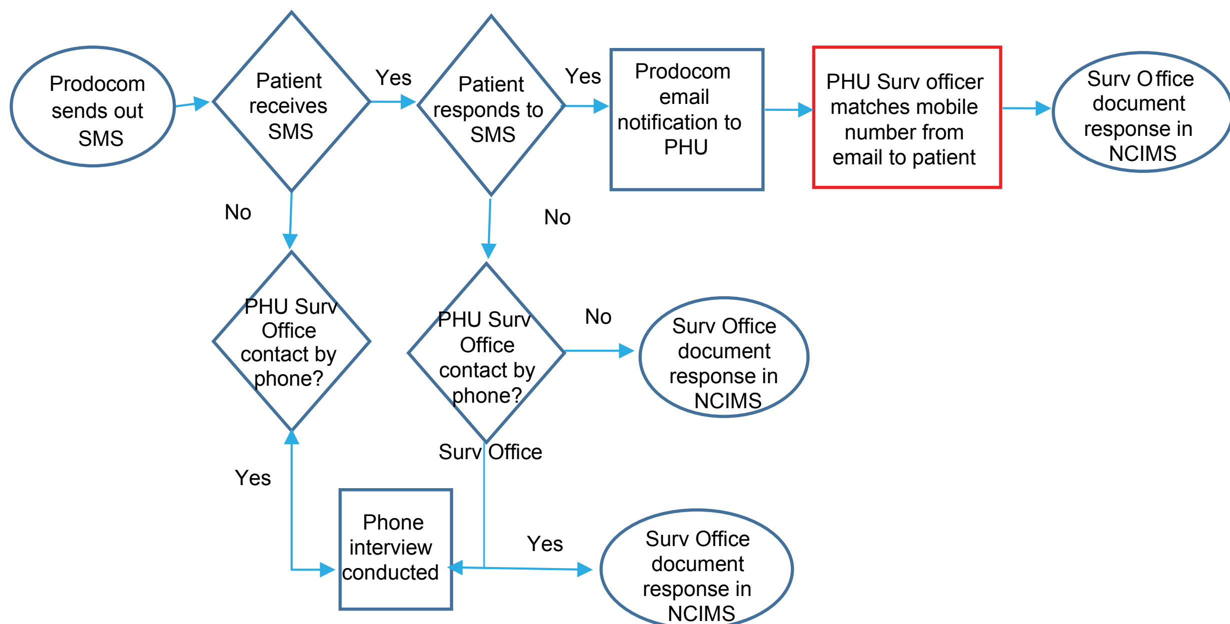
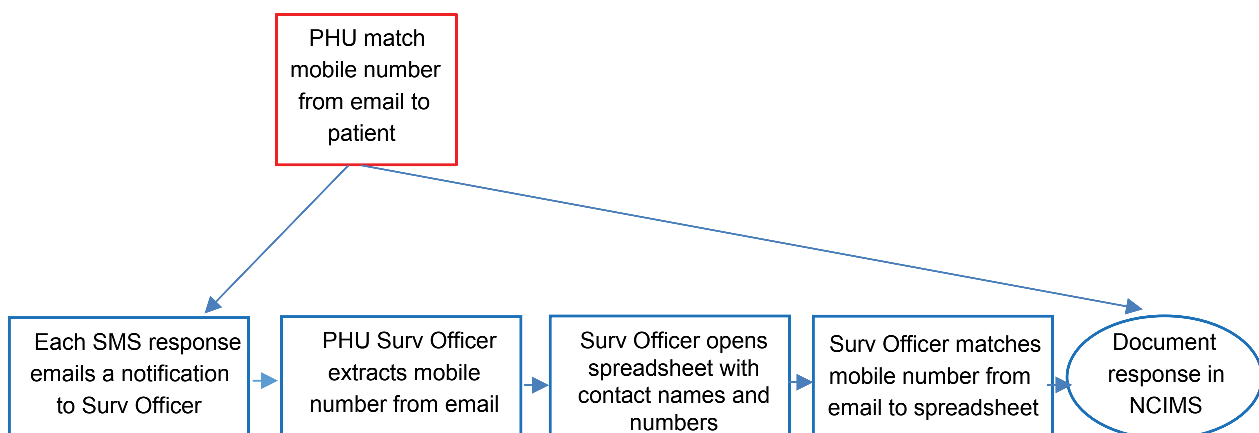


Figure 5: Breakdown of activities for surveillance officer to administer the short message service process



Discussion

Utilising SMS was a new follow-up initiative for the PHU and staff found it overall easy to use. The findings confirm there are significant gains in using SMS during a large surveillance activity, with only minor technical errors (only 1 contact not receiving the SMS). The application of SMS during this outbreak was estimated at 2.5 times more cost effective than telephone follow-ups and would substantially reduce staffing costs further in the event of a very large outbreak. In addition, it was noted by a number of contacts that the SMS increased their confidence in the response to the situation during a difficult time. In larger outbreaks, SMS could be the only way to resource the required follow-up.

Just over 60% of contacts responded to the SMS daily. The findings from the interviews with contacts reported that there were 2 main reasons for the remaining 40% not responding. Firstly, not all contacts were clear about what was required from them, particularly towards the end of the 9 day period when there were no signs of illness. This was reflected in the sudden drop in SMS response rates half way through the surveillance period. And secondly, the timing of the SMS did not fit in with contact's work schedule. While the time was brought forward from 10 am to 8 am, we found that the 8 am SMS may still be within a workday bandwidth and therefore may need to be earlier or during the evening to increase the likelihood of a response. The timing of the SMS needs to be flexible to be able to change with work schedules and convenience of those who are required to respond.

Other contributing factors to non-responses include mobile phone numbers given to public health by those under surveillance where phones were shared by other family members and not always carried by the targeted person. Reduced mobile reception coverage also impacted some responses and alternative contact arrangements could have been arranged at the commencement of surveillance to circumvent the above situations.

The process of sending out a SMS was described as straightforward. Mapping the process highlighted a number of steps involved in tracking contacts responses, which required manual input and was therefore vulnerable to errors because of the amount of paperwork required, particularly with a large number of contacts. It is recommended that these steps be streamlined through exploring if short messages can be sent and received through 1 central database.

This evaluation has a number of limitations. The first is that we were not able to conduct a comprehensive comparison between early detection of illness between telephone follow-ups and the use of SMS. Further studies would be valuable to determine which would be a quicker way to identify sick patients and to determine if people were less or more likely to disclose illness via SMS. Secondly, this study involved adult participants. If contact management was conducted with children and young persons, who may be on pre-paid telephone plans or access parents' telephones, the response rates may be different. This limitation hasn't been highlighted in other studies but requires further consideration.

Using the SMS for daily follow up was a new strategy the PHU had not used before and with digital technology becoming more popular could see SMS surveillance used routinely by PHUs in the future.

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Short reports

Q fever and contact with kangaroos in New South Wales

James Flint, Craig B Dalton, Tony D Merritt, Stephen Graves, John K Ferguson, Maggi Osbourn, Keith Eastwood, David N Durrheim

Q fever is a re-emerging pathogen of increasing public health importance.[1] This zoonosis, caused by *Coxiella burnetii,* can lead to acute and chronic illnesses in humans. While many infections are mild or asymptomatic, severe illness requiring hospitalisation and death can occur.

Domestic cattle, sheep and goats are important reservoirs for *C. burnetii* and exposure to aerosols and dust generated from infected animals is a well-established risk factor.[2] Of particular importance in transmission are the birth products of ruminants, which contain extremely high concentrations of *C. burnetii*. Typical high risk occupations associated with Q fever infection include farming, abattoir work and veterinary practice. Domestic pets, wild animals and ticks are also important reservoirs, although the role they play in the transmission of human disease is not well understood. In Australia, bandicoots, kangaroos, possums, dingoes, cats, foxes and wild pigs have all shown serological evidence of *C. burnetii* infection.[3]

In 2014, two cases of Q fever with non-typical risk exposures were investigated in northern New South Wales. The cases spent a significant amount of time (7–8 hours per day) working outdoors maintaining adjoining parkland properties (a hospital precinct and a golf club) inhabited by a large number of kangaroos. They were confirmed on the basis of clinically compatible symptoms and immunofluorescence serology performed by the Australian Rickettsial Reference Laboratory. The cases were aged 37 and 52 years, both were male and illness onsets were 19 April 2014 and 20 May 2014. Both cases were hospitalised, neither were vaccinated, neither had visited a recognised high risk setting (including farms or abattoirs) nor had contact with cows, goats, sheep, dogs or cats during their incubation period. Both spent considerable time each day (5–7 hours) mowing lawns contaminated with kangaroo faeces. All gardening activity, including lawn mowing, was performed without respiratory protection. The cases both directly handled joeys (juvenile kangaroos still using the mother's pouch) during their incubation period. One of the cases also handled dead kangaroos. As part of the investigation, other outdoor workers at the 2 sites were tested (CFT antibody and intradermal hypersensitivity testing) to determine prior or current Q fever infection. Testing results from one of the sites was made available to public health investigators. A total of 19 outdoor workers were tested, including 10 permanent grounds staff. All were negative for both tests. Tick specimens (n = 49) and kangaroo tissue collected during an unrelated babesiosis outbreak at one of the locations earlier in 2014 were also tested for *C. burnetii* by polymerase chain reaction and all were negative.

Direct contact with infected joeys and/or kangaroos and exposure to infected kangaroo ticks are plausible risk factors for Q fever transmission. Although the kangaroo tissue and ticks did not test positive during this investigation, both kangaroos and the kangaroo tick *Amblyomma triguttatum* have been shown to carry *C. burnetii.*[3,4] Mowing lawns or blowing grass contaminated with faeces or birth products of kangaroos or other native animals are also possible risk factors. Q fever outbreaks have been associated with dust originating from infected ruminant farms[2] and lawn mowing and brush cutting activities have been associated with outbreaks of tularaemia and psittacosis.[5–7] The very high kangaroo population and the extensive contamination of lawns with kangaroo faeces were notable features. However, the absence of *C. burnetii* infection in any of the other grounds staff, including those who spent considerable time mowing the same grounds as one of the confirmed cases, suggests that the direct handling of joeys was the more likely risk factor in this instance. At both sites, the cases were the only outdoor workers who had direct contact with the joeys (including touching the bare skin of the joeys both in and out of the pouch as well as handling dead joeys).

While definitive conclusions cannot be drawn, this investigation adds to a very limited literature on Q fever transmission risks not associated with recognised high risk exposures. The importance of non-typical exposures for *C. burnetii* infection was recently highlighted in Queensland, where it was determined that 60% of acute cases treated at the Townsville hospital had no clear animal or occupational exposures.[8] These cases were more likely to live on the outskirts of the city in areas with denser wildlife populations, including marsupials. It was

hypothesised that the seasonal incidence of Q fever in Townsville was the result of increased wildlife numbers and the drier conditions that follow the wet season.

This investigation points to a possible role for kangaroos in the transmission of *C. burnetii* and the acquisition of Q fever in Australia. Both cases reported large numbers of kangaroos at their workplaces, both had direct contact with joeys and kangaroos during their incubation period, both spent considerable time mowing lawns contaminated with kangaroo faeces, with no other high risk exposures reported. Current occupational guidelines in New South Wales recommend wearing a mask while slashing or cutting grass in areas with livestock or native animals.[9] Additional investigations focusing on non-typical exposures will improve our understanding of the transmission risks and exposure routes from native Australian animals to humans.

Ethics approval

Data collected during this outbreak investigation were covered by New South Wales public health legislation and as such ethics approval was not required.

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Estimates of influenza vaccine coverage from Victorian surveillance systems based in the community, primary care and hospitals

Benjamin Coghlan, Heath A Kelly, Sandra J Carlson, Kristina A Grant, Karin Leder, Craig B Dalton, Allen C Cheng

Introduction

Victoria has a number of complimentary surveillance systems for influenza. Flutracking,[1] an online national community influenza-like illness (ILI) surveillance system, includes Victorian participants; the Victorian Sentinel Practice Influenza Network (VicSPIN)[2] records patients attending general practice clinics throughout the state; and 4 large Victorian hospitals contribute to an Australia-wide adult hospital-based sentinel surveillance system, the Influenza Complications Alert Network (FluCAN).[3] Although these systems primarily collect data on influenza cases to inform public health action, they also collect influenza vaccine status in non-cases, which allows estimation of vaccine coverage and effectiveness.

Comparing data between systems, however, is not straightforward given differences in patient recruitment, processes to determine vaccine status, and means of distinguishing cases and non-cases (controls). Use of a valid control population is one element central for valid estimates of vaccination coverage since controls should ideally represent the broader population at risk. We recently found that vaccine coverage among elderly test-negative controls in primary care and hospitalised patients was similar over 2010 to 2014. However, this does not preclude the possibility of a selection bias in both systems due to the propensity to seek medical care (unpublished data). In this current study, we examined estimates of vaccine coverage in elderly Victorian control patients across all 3 of these influenza surveillance systems, community, primary care and hospitals, to assess whether together they might offer a simple means of estimating vaccine coverage in the broader elderly population at risk. This could be a useful ancillary function of these surveillance systems given that there is presently no routine means of measuring influenza vaccination coverage among groups that are eligible for publicly funded vaccine in Victoria or Australia.

Methods

We compared the proportion of non-cases/control participants aged 65 years or over who had received the influenza vaccine during the influenza season. In Flutracking, vaccine coverage was estimated as the proportion of all Victorian participants aged 65 years or over who reported being vaccinated but did not report an ILI by the end of the influenza season defined as a period of 24 to 26 weeks, typically between April and October in each year. Participants needed to have responded to at least 1 weekly online survey. In VicSPIN, vaccination coverage was estimated as the proportion of all elderly patients with an ILI recruited from a Victorian sentinel general practitioner (GP) site during the influenza season, who were vaccinated according to GP records and who were test-negative for influenza on nucleic acid testing. In FluCAN, vaccination coverage was estimated as the proportion of all elderly patients admitted with an acute respiratory infection to a Victorian sentinel hospital site who reported being vaccinated and were test-negative for influenza on nucleic acid testing.

The Hunter New England Human Research Ethics Committee approved Flutracking surveillance. The FluCAN study was approved by Human Research Ethics Committees of all participating hospitals and from the Australian National University. The *Victorian Public Health and Wellbeing Act 2008* and Public Health and Wellbeing Regulations 2009 provide the legislative authority to collect VicSPIN data.

Results

In Victoria over the 2010 to 2014 seasons, vaccination coverage was similar across all 3 surveillance systems for people aged 65 years or over although the number of participants was small in some years (Table). Flutracking had more participating controls aged 65 years or over than attended VicSPIN GP clinics in all years and more than FluCAN in 3 of the 5 years. Vaccine ascertainment was complete for Flutracking as compared with FluCAN where around 30% of control patients admitted to hospitals did not report vaccination status (unpublished data).

The consistency of these estimates provides reassurance that all systems are likely to be making

Table: Influenza vaccine coverage among Victorian participants aged ≥65 years without influenza from three surveillance systems, 2010 to 2014

Influenza season	Proportion of controls vaccinated by surveillance system					
	Flutracking – online community* (95% CI)	n	VicSPIN – primary care† (95% CI)	n	FluCAN – hospitals† (95% CI)	n
2010	90.2 (76.9–97.3)	41	76.9 (46.2–95.0)	13	78.8 (67.0–87.9)	66
2011	77.9 (66.2–87.1)	68	68.4 (43.4–87.4)	19	81.7 (69.6–90.5)	60
2012	80.5 (72.0–87.4)	113	84.4 (67.2–94.7)	32	76.2 (69.0–82.4)	168
2013	83.7 (76.7–89.3)	147	80.8 (60.6–93.4)	26	81.5 (68.6–90.7)	54
2014	79.8 (73.1–85.4)	178	82.8 (64.2–94.2)	29	80.7 (72.3–87.5)	114
2010–14	81.5 (78.0–84.7)	547	79.8 (71.5–86.6)	119	79.0 (75.0–82.6)	462

* No report of ILI (cough + fever) by end of influenza season.

† Tested negative for influenza by nucleic acid detection using polymerase chain reaction.

unbiased estimates of vaccine coverage in this key target group in whom vaccine is publicly funded. Flutracking participants, however, are known to have different education levels from the general population[4] and information on confounders, other than age and sex, is not collected. Unlike VicSPIN and FluCAN then, estimates of vaccine coverage (and vaccine effectiveness) from Flutracking cannot be adjusted for important factors, including medical illnesses, pregnancy and Indigenous status that also define eligibility for free vaccination in Australia (and the likelihood of severe influenza). Flutracking does ask about Indigenous status, but there are currently few Indigenous participants. In addition, the low specificity of the syndromic case definition of ILI used by Flutracking as opposed to the high specificity of nucleic acid tests[5] has been shown to bias estimates of vaccine effectiveness downwards, particularly if the attack rate of influenza ILI decreases relative to that of non-ILI.[6–9]

Documenting vaccine coverage is important to determine if the program is reaching those targeted for influenza vaccination. Although a whole-of-life vaccine registry has recently been announced, implementation is likely to take several years and the reliability of such a registry for an annually administered vaccine is uncertain.[10] The Australian Institute of Health and Welfare periodically conducts large nationally representative computer assisted telephone surveys of influenza vaccination coverage among adults.[11] However, the last study was in 2009 and findings are typically unavailable until years after data collection, reducing the utility of these data for public health action.

These 3 surveillance systems could together provide more timely estimates of vaccine coverage for high-risk adult groups. The continual expansion of Flutracking, however, could alter the makeup of participants, diverging vaccine coverage estimates from these systems would raise questions about their accuracy (although inaccurate estimates might still be useful to detect changes in vaccine coverage if a surveillance system is consistently under or overestimating vaccine coverage). Estimates for some groups eligible for free vaccine, such as pregnant women and Indigenous Australians, are likely to remain limited as only small numbers are currently recorded by these 3 systems. Specific efforts to recruit particular sub-populations may be necessary.

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Contribution of authors

BC designed the study and, with AC, performed the analysis and drafted the manuscript. KG provided VicSPIN data and SC provided Flutracking data. All authors provided input into the analysis and interpretation of results.

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Annual reports

Creutzfeldt-Jakob disease surveillance in Australia: update to December 2014

Genevieve M Klug, Alison Boyd, Shannon Sarros, Christiane Stehmann, Marion Simpson, Catriona McLean, Colin L Masters, Steven J Collins

Abstract

Nation-wide surveillance of human transmissible spongiform encephalopathies (also known as prion diseases), the most common being Creutzfeldt-Jakob disease, is performed by the Australian National Creutzfeldt-Jakob Disease Registry, based at the University of Melbourne. Prospective surveillance has been undertaken since 1993 and over this dynamic period in transmissible spongiform encephalopathy research and understanding, the unit has evolved and adapted to changes in surveillance practices and requirements concomitant with the emergence of new disease subtypes, improvements in diagnostic capabilities and the overall heightened awareness of prion diseases in the health care setting. In 2014, routine national surveillance continued and this brief report provides an update of the cumulative surveillance data collected by the Australian National Creutzfeldt-Jakob Disease Registry prospectively from 1993 to December 2014, and retrospectively to 1970. *Commun Dis Intell* 2016;40(2):E207–E215.

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

Introduction

In 1993, the Allars' inquiry[1] into the use of cadaver-derived pituitary hormones under The Australian Human Pituitary Hormone Program and the association with 4 medically acquired (iatrogenic) Creutzfeldt-Jakob disease (CJD) deaths recommended broadening of the responsibilities of the nascent Australian surveillance unit while monitoring for further cases of iatrogenic CJD in Australia. The Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR) was established in October 1993 at the University of Melbourne. The monitoring of further Australian iatrogenic CJD cases related to pituitary hormone treatment for infertility or short stature and contaminated dura mater grafts remains one of the core objectives of the ANCJDR. However, the ANCJDR's activities have changed to encompass the surveillance of all types of CJD, including sporadic, genetic and variant CJD and other transmissible spongiform encephalopathies such as Gerstmann-Sträussler-Sheinker syndrome and fatal familial insomnia.

As described previously,[2] human prion disease can arise sporadically or from genetic or iatrogenic aetiologies. Detailed evaluation of each suspected case added to the register is undertaken to determine whether a case can be excluded from suspicion or classified as a definite, probable or possible prion disease case according to World Health Organization (WHO) diagnostic criteria.[3] CJD was made a notifiable disease in all states and territories of Australia as of June 2006. Most initial notifications to the ANCJDR arise through diagnostic testing available through the registry and this occurs prior to Health department notification.

The global incidence of CJD is commonly reported to be 1 case per million per year. However, in most countries with long-standing surveillance systems in place such as France and Switzerland, annual incidence has been reported above this quoted figure.[4] Incidence rates as high as 1.2 to 2.4 cases per million per year have been reported.[4] Temporally, human prion disease incidence increased in most countries, including Australia, as surveillance mechanisms have evolved and diagnostic testing capabilities improved. This was associated with a generally greater awareness of this rare disease in the health care setting.

In 2014 changes occurred that have or will have an influence on the incidence rates of CJD in Australia. These include: difficulties in achieving suspected prion disease autopsies in Queensland between January 2013 and September 2014; and the closure of the New South Wales neuropathology laboratories between December 2012 and March 2015 due to laboratory facility upgrading. In this report, updated surveillance figures to 31 December 2014 are provided for all retrospective (to 1970) and prospective (from 1993) cases ascertained, including discussion of the potential impact in relation to these changes on case notifications, classifications and overall incidence.

Methods

Patients with a suspected human prion disease are prospectively notified to the ANCJDR predominantly through referral for diagnostic cerebrospinal fluid (CSF) 14-3-3 protein detection. Other mechanisms include or have included personal communication with clinicians, families, hospitals and CJD-related groups, health record searches through hospitals or health departments. Once notified to the ANCJDR, referrals are assessed and if the suspicion of prion disease is supported, the case will be added to the register as a formally notified suspected case for continued investigation with the aim of exclusion or classification according to the WHO diagnostic criteria. Investigation of register cases can be prolonged as the ANCJDR requires next-of-kin consent to access and compile the appropriate clinical information from various health information sources for comprehensive evaluation. Response times can vary as the information can be extensive or sources numerous. Medico-demographic questionnaires are offered and forwarded to families if they are willing to contribute, providing valuable information for analysis and evaluation.

The classification of register cases remains as 'incomplete' until all known available information is gathered and reviewed or a definitive result from neuropathological assessment is obtained. Cases may be excluded from the register on the basis of neuropathological examination or after thorough clinical evaluation. A 'definite' classification requires brain tissue examination, including immunohistochemically and 'probable' and 'possible' cases are reliant on specific clinical profile and diagnostic test outcomes being met as previously described.[3] In this report, the total number of confirmed prion disease cases includes those who have been classified as definite or probable cases during 2014.

In conjunction with the ANCJDR's surveillance responsibilities, the registry provides a diagnostic platform for ante– and post-mortem diagnostic testing for human prion diseases. The testing of CSF for the presence of a family of low molecular weight proteins called '14-3-3' is performed weekly by the ANCJDR. This test, first introduced in 1997, has been readily utilised by the health community and referrals have increased substantially since its introduction to around 400 referrals each year. As described previously, the test provides an increasingly larger proportion of initial notifications of suspected human prion disease to the ANCJDR each year. The ANCJDR also facilitates prion protein gene testing as appropriate, and Western blot analysis of tonsil and brain tissue from biopsies or autopsies to supplement immunohistochemical assessment. The ANCJDR actively promotes these diagnostic tests so that these options are available to clinicians and families to achieve the most accurate diagnosis and classification of persons suspected to have prion disease.

Annual human prion disease incidence rates were calculated using direct age-standardisation, based on the Australian Bureau of Statistics 2000 estimated resident population for Australia and for each state and territory.[5] Population based rates of post-mortem examination in suspected human prion disease were calculated using the Australian Bureau of Statistics 1993–2014 Australia demographic statistics for specific states and territories.[6–12] Health information is collected through a combination of public health and surveillance responsibilities, based on the national notification of communicable diseases. ANCJDR surveillance activities for the period reported has been under ethical approval by The University of Melbourne Human Research Ethics Committee.

Statistical analysis (Log-Rank test) was performed using Stata (Intercooled Stata 7, Stata Corporation, College Station, TX).

Results

Seventy-six persons with suspected human prion disease were added to the CJD surveillance register in 2014. Cases were initially notified via a request for the 14-3-3 CSF test (55 cases), personal communication from clinicians (11 cases), hospitals (3 cases), direct health department notifications (2 cases), the CJD Support group network (1 case), a Coronial referral (1 case), family communication (1 case), the Victorian Brain Bank Network (1 case) and the CJD counselling service (1 case). Two of these cases had previously been added to the register and excluded after evaluation. In 2014, these two cases were re-added to the register after further information was provided by the CJD Support group network and after a request for autopsy. One of these cases has again been excluded from the register after clinical advice, while the other remains under investigation. The proportions of the initial notification sources of the 76 cases are consistent with those in previous years and the overall trends for all register cases (Table 1).

Of the 76 cases that were added to register in 2014, five cases were known to the ANCJDR prior to 2014 via the CSF 14-3-3 protein test (3 cases), the CJD support group network (1 case) and the CJD counselling service (1 case). At the time of initial notification, these 5 cases were not added to the register due to a low level of suspicion of prion disease after assessment. In 2014, the provision of

Table 1: Source of initial notification of suspected prion disease cases ascertained between 1993 and 2014

Method	Register cases* (%)	Cases removed from the register† (%)	Overall
CSF 14-3-3 protein test request (Since September 1997)	53.0	49.9	51.8
Personal communications			
Neurologists	13.4	12.2	13.0
Neurologists (mail-out reply cards)	2.6	1.8	2.3
Neuropathologists	7.9	8.6	8.2
Neuropathologists (mail-out reply cards)	0.6		0.3
Pituitary Hormones Task Force	1.7	3.1	2.3
Family	3.0	2.5	2.8
Molecular biologist	0.1		0.05
Hospital	0.6	1.5	0.9
Death certificates	9.4	5.5	7.8
Hospital and health department searches			
Hospital medical records	3.2	7.7	4.9
Health department search/state morbidity data	1.3	3.5	2.2
Direct health department notification	1.5	0.3	1.0
CJD Support Group	0.4	0.1	0.3
CJD counselling service	0.3	0.4	0.3
Combined CSF/genetic test request	0.3	0.9	0.6
Genetic test request	0.3	1.6	0.8
Victorian Brain Bank Network	0.1	0.1	0.2
Coroner's post-mortem request	0.1	0.3	0.1
Press	0.1		0.05
UK surveillance unit	0.1		0.05
Total	100	100	100

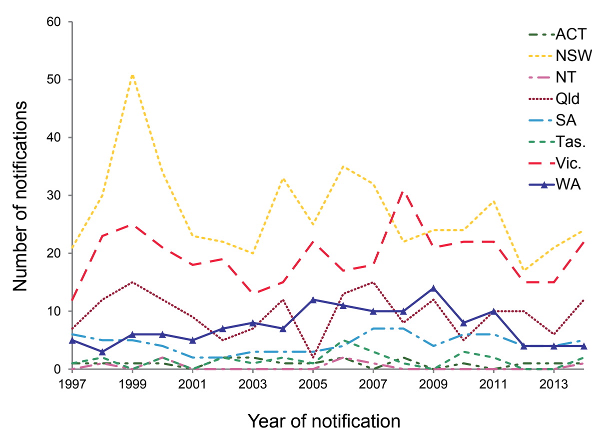
* Registry cases; includes all cases currently on the register as classified cases or cases still under investigation

† Cases removed by the registry; includes all suspected cases excluded from the register after detailed investigation.

further information increased the likelihood of prion disease resulting in formal notification and addition of the cases to the register. The number of case notifications in 2014 was greater than the previous 2 calendar years where lower than expected numbers were observed (53 cases in 2012 and 52 in 2013). In 2013, it was noted that these lower numbers were most likely a reflection of a more clinically discerning approach adopted by the ANCJDR when adding cases to the register.[13]

Excluding the 5 cases added to the register in 2014 but initially notified prior to this time, the number of new suspected cases notified to the ANCJDR in 2014 was 9% lower (71 cases) than the long-term average annual number based on data from 1993 to 2014 (78 cases). This is in sharp contrast to the lower numbers of notifications observed in 2012 and 2013 where 33% to 34% reductions were observed (Figure 1).

Figure 1: Prospective notifications of suspected prion disease cases notified to the ANCJDR, 1997 to 2014, by state or territory and year



By state and territory, there was an increase in the number of suspected case notifications in New South Wales, Queensland, Tasmania and Victoria compared with 2012 and 2013, returning to previously observed levels. The Australian Capital Territory, South Australia and the Northern Territory were unchanged from the previous year. Western Australia continued to have less than half the number of suspected case notifications in 2014 compared with the 1993–2014 long-term average (8 cases per year). This observation was similar to 2012 and 2013. Sizeable relative fluctuations are not surprising with annual CJD notifications given the small absolute case numbers involved. However, it should be noted that since 2009, notifications have been consistently declining in Western Australia. Since 2012, CSF 14-3-3 protein testing referrals from Western Australia have declined and there have been fewer cases within this referral group where the suspicion for prion disease warrants formal addition to the register and therefore case notifications for addition to the register are lower. This is in contrast with previous evidence that elevated CSF referrals correspond with elevated suspected prion disease notifications.[4]

As of 31 December 2014, the majority of the 76 suspected cases added to the register in 2014 were classified as incomplete (59 cases). Nine cases were excluded after detailed clinical follow-up (3 cases) or neuropathological examination (6 cases); 7 cases were classified as definite and 1 as probable prion disease.

Excluding the prion disease-related post-mortem rate in 2014, wherein figures are still provisional, the average proportion of suspected prion disease cases on the register who died between 1993 and 2013 and underwent post-mortem examination is 62%. Over this period, this proportion has steadily increased from an average of 45% during 1993 to 1995 to 71% between 2005 and 2012. This contrasts with the findings of an Australian healthcare setting survey where the national hospital post-mortem rate was 12% in 2002 to 2003.[15] The high prion disease-related post-mortem proportion underpins the high and consistent number of confirmed Australian human prion disease cases recorded over the more recent time period and provides confident understanding of the cause of death in suspected cases ultimately determined as non-prion disease. In contrast to the 2005 to 2012 period, the proportion of suspected prion disease case deaths in 2013 where a post-mortem examination was performed declined by 14% and this was a reflection of a reduced number of autopsies being performed and completed in 2013 in New South Wales and Queensland.

Based on the Australian population, the average crude rate of prion disease-related post-mortems between 1993 and 2014 was 1.4 post-mortems per million per year (range, 0.6 to 2.0), which is considerable given prion disease is particularly rare. By state and territory and for the same period, the lowest rates of suspected prion disease post-mortems performed annually are in the Australian Capital Territory, Tasmania and the Northern Territory (0.8, 1.0 and 0.9 per million per year, respectively), while the highest rates are in Victoria and New South Wales (1.5 per million per year). Despite the smaller populations in Tasmania, the Northern Territory and the Australian Capital Territory, the post-mortem rates are not substantially lower than the rates of more populous states and provide a level of confidence that suspected case deaths in these states and territories have a similar likelihood of undergoing post-mortem examination. In Queensland, where the suspected prion disease autopsy service was interrupted between January 2013 and September 2014, the post-mortem rate in 2013–2014 was substantially diminished (0.2 and 0.0 post-mortems per million per year respectively) compared with the long-term average for Queensland of 1.2 post-mortems per million per year between 1993 and 2012.

In the more populous states and territories, there has been an overall temporal increase in rates between 1993 and 2014 with the exception of South Australia where rates have been variable over this period (Figure 2a, 2b). In the smaller population regions, this positive trend was also present but less robust due to the considerable variation in the annual rates due to small population sizes and case numbers.

Since 2011, a sustained decrease in the post-mortem examination rate in South Australia and Western Australia was observed. In both states, there were a number of suspected prion disease deaths in 2014 where neuropathological examination is pending. Once finalised, the 2014 post-mortem rate is predicted to return to an expected level but clearly will not change the lower rates in 2012 and 2013. In Queensland, the influence of the interrupted autopsy service is demonstrated in Figure 2a with the sharp decline in the annual rate since 2012.

In New South Wales and Victoria, the respective 2013 post-mortem rates returned closer to levels observed prior to the reduced rates of 2012. While this stabilisation has continued in 2014 in Victoria, the rate in New South Wales has again decreased sharply. This is most likely due to a decrease in the completion of neuropathological analysis of post-mortem tissue as a result of the New South Wales neuropathology laboratory service being temporarily closed, rather than a decrease in

post-mortem procedures being performed in New South Wales in 2014. The closure commenced in December 2012 due to the requirement to upgrade the existing neuropathology laboratory facilities. The New South Wales neuropathology service resumed operations in March 2015.

Figure 2a: Rate of post-mortem examination in suspected prion disease case deaths per million population, by state and territory and year

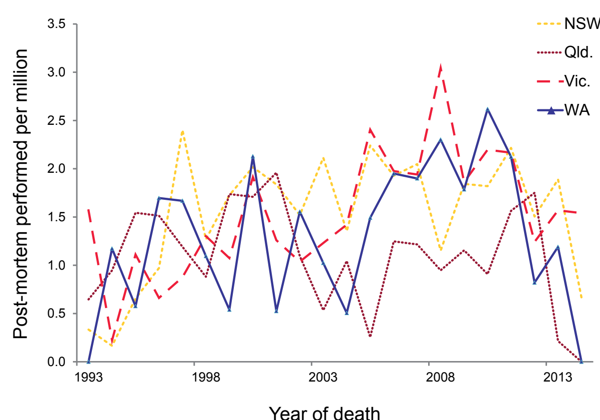
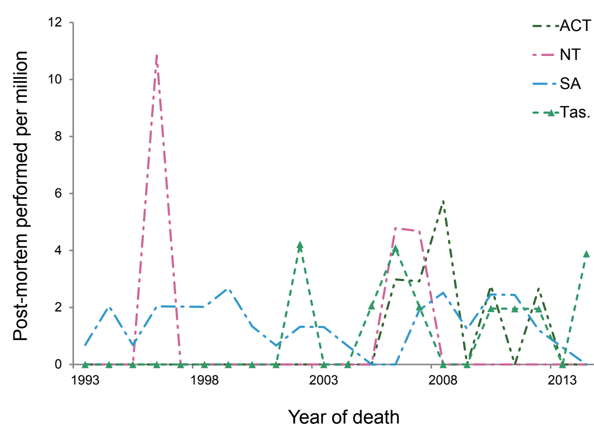


Figure 2b



In total, 11 suspected cases died in New South Wales in 2014 and subsequently underwent a post-mortem procedure to remove tissue for analysis. The immunohistochemical analysis of these 11 tissues is currently pending. Once these examinations have been completed, the post-mortem rate in suspected case deaths in New South Wales should return to expected levels in line with previous years.

As of 31 December 2014, there were 1,048 cases on the register with 781 of these being classified as probable or definite prion disease cases. An additional definite iatrogenic case who was treated in Australia, and died in the United Kingdom is included in Table 2. However this case is not classified as an Australian case due to the location at death and is thereby excluded from the overall statistical analysis of Australian prion disease cases. Since the start of surveillance, 678 suspected prion disease cases have been excluded from the register after detailed follow-up, with 17 of these being excluded in 2014 (13 after neuropathological examination).

In 2014, 16 cases were re-classified from incomplete to definite prion disease and 8 cases to probable prion disease. There were no further cases of possible prion disease classified in 2014 and the total number of possible cases remains at 15 of which 14 cases were sporadic and 1 was iatrogenic CJD (Table 2). Of the 251 incomplete cases, 145 are presently alive. In 2014, the total number of incomplete cases under evaluation has increased from the number observed in 2013 (216 cases). Although the higher number of incomplete cases is not unprecedented, it does highlight the imbalance of new suspected cases with fully evaluated cases with an outcome. Compared with the long-term average (2004–2013) figures, there have been 17% more cases added to the register but 14% fewer cases confirmed as definite or probable and 26% fewer cases excluded in 2014. In particular, the number of definite case classifications has

Table 2: Classification of Australian National Creutzfeldt-Jakob Disease Register cases, Australia, 1970 to 2014

Classification	Sporadic	Familial	Iatrogenic	Variant CJD	Unclassified	Total
Definite	464	49	5*	0	0	518
Probable	249	11	4	0	0	264
Possible	14	0	1	0	0	15
Incomplete					251†	251
Total	727	60	10	0	251	1,048

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

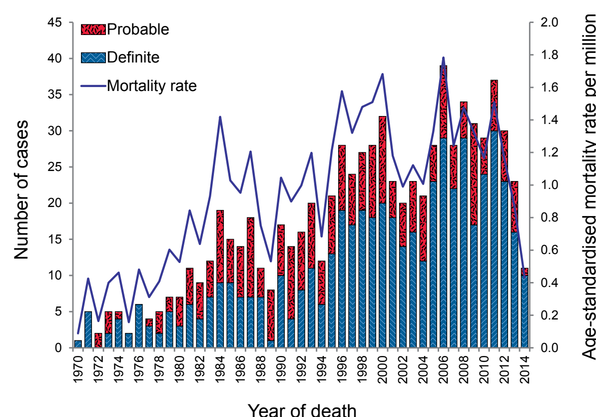
† Includes 145 living cases.

continued to decline (30% decrease compared with the 2004–2013 average), whereas the number of probable cases confirmed has been equivalent with the long-term average (2004–2013). Lower levels of classifications and exclusion of suspected cases is in part due to the inflation of the number of cases classified or excluded in 2012 due to concerted efforts by the ANCJDR to classify outstanding cases but also reflects the impact of the suspension and deferral of autopsy services in Queensland and New South Wales respectively during 2013 and 2014.

Age-standardised mortality rates show that the rate of human prion disease mortality in Australia during the period of 1970 to 2014 is generally increasing, with the exception of 2014, where case evaluation is pending for the majority of deaths (Figure 3) and incidence is therefore provisional. In 2014, the age-adjusted mortality rate was 0.4 deaths per million per year and this would be expected to increase after further investigation and classification of incomplete cases. The mean annual age-adjusted mortality rate during the period from 1970 to 2013 was 1.0 death per million (range, 0.1 to 1.8). For the prospective surveillance period of 1993 to 2013, the mean annual rate was 1.3 deaths per million (range, 0.7 to 1.8). By state and territory, the majority of regions in Australia had a mean age-adjusted mortality rate above 1 case per million per year between 1993 and 2013 (range, 1.0 to 1.5). The exceptions were Tasmania and the Northern Territory with 0.6 and 0.7 deaths per million respectively. Restriction of the surveillance data to the period between 2003 and 2013 allows comparisons between states and territories during a time-frame of relatively consistent surveillance practices, diagnostic capabilities and utility with the exception of MRI diagnostics (Table 3). During this period, Tasmania, the Northern Territory and Queensland had lower than expected mean mortality rates, while Western Australia and Victoria had the highest prion disease mortality in Australia.

The proportions of human prion disease aetiologies represented on the register have remained similar to previous years (Figure 4). Previously we

Figure 3: Number of definite and probable prion disease cases and age-standardised mortality rate*, Australia, 1970 to 2014, by classification and year



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

Table 3: Definite and probable human prion disease deaths and age-adjusted mortality rates, 2003 to 2013, by year and state or territory

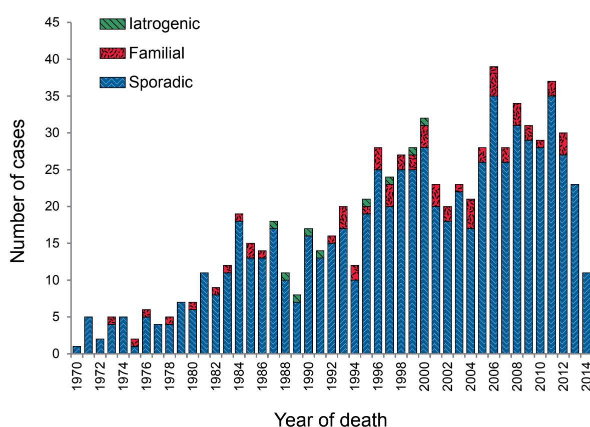
Year	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014*	Total	Mean age-adjusted mortality rate† (deaths/million/year)
ACT		1		1		2		1			1		6	1.4
NSW	7	11	10	12	10	6	11	5	14	6	10	3	105	1.2
NT				2	1								3	0.9
Qld	3			7	2	4	4	2	5	6	3		36	0.7
SA	1	2	1	1	3	5	2	4	4	2	1		26	1.4
Tas.			1	2						1		2	66	0.6
Vic.	9	5	11	10	6	13	9	13	9	12	6	6	109	1.7
WA	3	2	5	4	6	4	5	4	5	3	2		43	1.7
Aus.	23	21	28	39	28	34	31	29	37	30	23	11	334	1.3

* Provisional figures.

† Age-standardised mortality rates (2003–2013) were calculated using the Australian Bureau of Statistics 2000 estimated resident population for Australian states and territories.

reported that the annual number of genetic prion disease cases had declined in recent years[2] although this changed with the classification of 6 confirmed genetic prion disease cases during 2013. In 2014, there were no further cases classified as definite or probable genetic prion disease. Overall, the vast majority of human prion disease cases were sporadic (91%) while genetic and iatrogenic cases represented 8% and 1% respectively of all definite and probable cases.

Figure 4: Definite and probable human prion disease cases, 1970 to 2014, by aetiology and year



Based on 781 definite and probable human prion disease cases, 53% per cent were female. Similar proportions for gender exist for all human prion disease aetiologies. Median ages at death for the overall case group or by specific aetiology are largely unchanged from the previous reporting period. Sixty-six years is the median age at death for all cases overall and only a single year difference between males (66 years) and females (67 years). For sporadic cases, 67 years is the median age at death both overall and for both males and females. For genetic prion disease, there is a 4 year age difference between males (58 years) and females (62 years) and overall the median age of death from genetic prion disease is 60 years. As there have been no further iatrogenic cases identified since the last reporting period at 31 December 2013, there has been no change to the previously reported median age at death for iatrogenic cases.[2]

Duration of illness is typically short for human prion disease, especially sporadic CJD, with the median length of illness duration for all cases combined being 3.9 months. By aetiology, median duration was found to be 3.5 months for sporadic cases (range, 0.9 to 60 months), 6.3 months for iatrogenic cases (range, 2 to 25 months) and 5.8 months for genetic cases (range, 1.5 to 192 months). Within 6 months of disease onset, 70% of all prion disease cases were deceased. By aetiology, 72% of sporadic, 53% of genetic and 56% of iatrogenic human prion disease were deceased 6 months after the onset of symptoms. Survival is significantly shorter in sporadic CJD than the genetic form ($P < 0.0001$ by Log Rank Test).

Between 1 January and 31 December 2014, no variant CJD or further iatrogenic prion disease cases were identified in Australia. The most recent human-derived pituitary gonadotrophin-related CJD death occurred in 1991, while the most recent Lyodura-related CJD death occurred in 2000.

Discussion

In 2014, the number of suspected prion disease notifications returned to levels similar to the long-term average for the 1993 to 2014 surveillance years. This was in contrast to the previous 2 years, when reduced numbers of notifications were attributed to several possible factors including the temporary interruption of the Queensland suspected prion disease autopsy service and changes to the approach to adding cases to the register for investigation by the ANCJDR. However, although these factors were largely unchanged for the majority of 2014, notifications of suspected cases for most jurisdictions (including Queensland) were increased compared with 2012 and 2013, and closer to previous years. This suggests that natural fluctuation strongly contributed to the reduced notifications during 2012 and 2013.

Notifications of suspected cases in Western Australia continued to be lower than expected in 2014, as was observed in 2012 and 2013. As previously discussed, Western Australian health services are relied upon to manage case investigations following notifications and manage autopsy referrals. Changes to the functional role of the ANCJDR in Western Australia during these years may limit the ANCJDR's capacity to ascertain the true level of clinical suspicion for CJD, and may have contributed to a reduced number of formal notifications and subsequently, confirmed cases reported by the ANCJDR. A further contributing factor may be the decrease in CSF testing referrals from Western Australia since 2012, particularly in 2014. The ANCJDR will continue working towards improving ascertainment in Western Australia with the Western Australian Department of Health.

The temporary interruption of the Queensland suspected prion disease autopsy service in 2013 and the majority of 2014 has undoubtedly and, as expected, reduced the number of confirmed prion disease cases in that State during this period. A lower population-based post-mortem rate has con-

tributed to the marked decline in the overall crude numbers and corresponding incidence within these years. Furthermore, this has contributed to an overall lower incidence in Australia in 2013. It would be expected that the finalised 2014 incidence will be similarly influenced. Interestingly, formal case notifications in Queensland returned to expected levels in 2014 after a decline in 2013 and CSF referrals were consistent with previous years, suggesting that the lower Queensland notification number in 2013 was an isolated occurrence rather than a direct consequence of the service interruption.

In New South Wales, the closure of the neuropathology laboratories for refurbishment has extended the time required for reporting, although this appears to have had little effect on formal suspected case notifications and CSF referrals for 14-3-3 testing in 2013 and 2014. Furthermore, incidence has remained consistent with levels prior to the laboratory closure. Post-mortem rates slowed for 2014 as would be expected due to reporting delays. However, it is expected that these figures should improve now that the laboratory is fully operational.

The number of cases classified as definite and probable prion disease in 2014 (24 cases) was equivalent to the number classified in 2013 although lower than the long-term average number classified annually (29 cases) between 2004 and 2013. In comparison with the previous reporting period, fewer definite cases were classified in 2014 as expected due to the reduced autopsy or neuropathology services although this was compensated for by a 2-fold increase in probable case classifications. The concerted effort by the ANCJDR to garner as much clinical information on suspected CJD cases that have not had post-mortem examination to try to achieve an accurate clinical diagnosis will be maintained in the future.

With this continued focus and the resumption of routine autopsy and diagnostic neuropathology services in two of the most populous Australian states, it is expected that the surveillance parameters of CSF referrals, formal suspected case notifications with addition to the register, post-mortem examinations and prion disease incidence, should re-align with previous findings.

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Australian Paediatric Surveillance Unit annual report, 2014

Marie Deverell, Yvonne Zurynski, Elizabeth Elliott, and all chief investigators of APSU surveillance studies

Introduction

The Australian Paediatric Surveillance Unit (APSU) was established in 1993 to facilitate national active surveillance of uncommon diseases of childhood including selected communicable diseases. This report includes data on the following conditions: acute flaccid paralysis (AFP), a surrogate condition for poliovirus infection; congenital cytomegalovirus (cCMV); congenital rubella; perinatal exposure to HIV and paediatric HIV infection; neonatal herpes simplex virus (HSV); congenital varicella; neonatal varicella; and juvenile onset recurrent respiratory papillomatosis (JoRRP). Surveillance of severe complications of influenza was undertaken during the influenza season (July to September 2014).

Methods

Australian Paediatric Surveillance Unit

APSU study protocols and case definitions are developed with collaborating study investigators who provide specialised clinical expertise for each condition studied listed in Table 1. Each month approximately 1,500 paediatricians and other child health clinicians nationally are sent the APSU report card. Over 90% of clinicians report via email; they respond each month whether or not they have a case to report for any of the conditions listed on the report card. The APSU collects de-identified clinical and/or laboratory data via a case report form completed by the doctor looking after the child. Completed case report forms are then forwarded on to study investigators. All study protocols including case definitions and case report forms are available for download on the APSU web site (www.apsu.org.au). The response rate to the monthly report card was 92% in 2014.

Surveillance of AFP is conducted by both the APSU and the Paediatric Active Enhanced Disease Surveillance (PAEDS). PAEDS was initiated in 2007 and is a hospital based surveillance system reliant on active case ascertainment by specialist surveillance nurses in each of the 5 participating hospitals.[1] All cases of AFP from both systems are reported to the Polio Expert Panel (PEP) and are classified according to World Health Organization criteria.

Results

All reported rates are based on child population estimates published by the Australian Bureau of Statistics.[2]

Acute flaccid paralysis

All cases of AFP reported to the APSU are submitted for review to the PEP. In 2014, there were 26 confirmed cases of AFP notified to the APSU. Of these, 14 were reported from Victoria, 6 from Queensland, 2 from New South Wales, 2 from Western Australia, 1 from South Australia and 1 case was reported from the Northern Territory. Cases of AFP were ascertained by the APSU from a variety of hospitals across Australia (Table 2).

All cases were reviewed by the PEP, and classified as non-polio AFP. The main diagnoses associated with reported cases of AFP were Guillain-Barrè syndrome (33%) and transverse myelitis (13%). The National Polio Reference Laboratory (NPRL) combines cases ascertained by the APSU with those ascertained by PAEDS, and a final AFP report produced by NPRL is published in *Communicable Diseases Intelligence*. The NPRL ensures that duplicate notifications that are ascertained by both APSU and PAEDS are counted only once. These data contribute towards Australia's polio monitoring efforts and maintenance of polio-free certification as recommended by the World Health Organization as part of the Global Polio Eradication Initiative.

Congenital cytomegalovirus

In 2014, 24 confirmed cases and 2 probable cases were reported to the APSU. A total of 273 confirmed cases were reported during the entire study period, 1999–2014. Ten cases were reported from New South Wales, 8 from Queensland, 2 from Victoria, 2 from Western Australia, 2 the Northern Territory, 1 from South Australia and 1 from the Australian Capital Territory. Of the 14 cases, 11 were not of Aboriginal or Torres Strait Islander descent, and 3 were unknown.

The data collected since 1999 informed further research including current studies of hyperimmunoglobulin treatment during pregnancy and there is good evidence for the benefits of hygiene

Table 1: Confirmed cases identified in 2014 and for the total study period, and reported rates per 100,000 of the relevant child population

Condition	Date study commenced	Questionnaire returned (%)	Number of confirmed cases 2014	Reported rate for 2014 (per 100,000)	Number of confirmed cases for total study period	Reported rate for total study period (per 100,000 per annum)
Acute flaccid paralysis*	Mar 1995	100	26*	0.59†	839	1.03†
Congenital cytomegalovirus	Jan 1999	56	26	8.44‡	273	6.62‡
Congenital rubella (with defects)	May 1993	No notifications	Nil	Nil	53	0.06§
Perinatal exposure to HIV	May 1993	88	39	12.66‡	626	11.07‡
HIV Infection	May 1993		3	0.06§	87	0.10§
Neonatal – herpes simplex virus infection	Jan 1997	95	10	3.25‡	164	3.54‡
Infant – herpes simplex virus infection	Jan 2012		8	2.66‖	11	1.20‖
Congenital varicella	May 2006	No notifications	Nil	Nil	2	0.08‡
Neonatal varicella	May 2006	No notifications	Nil	Nil	22	0.93‡
Juvenile onset recurrent respiratory papillomatosis (JoRRP)¶	Oct 2011	100	2	0.05†	12	0.09†
Severe complications of influenza**	Influenza season each year since 2008	95	83	1.88†	380	1.27†

* Includes all cases of acute flaccid paralysis (AFP) reported via the Australian Paediatric Surveillance Unit (APSU). All cases have been classified by the Polio Expert Panel as 'non-polio AFP' according to World Health Organization criteria. Number of confirmed cases for the total study period includes both the APSU and the Paediatric Active Enhanced Disease Surveillance data.

† Based on population of children aged less than 15 years.

‡ Based on number of births.

§ Based on population of children aged less than 16 years.

|| Based on population of children aged less than 12 months.

¶ Confirmed cases and probable cases are reported; a probable case is defined as a papilloma visualised by endoscopy but the histology results are pending.

** Influenza surveillance was conducted each year since 2008 during the influenza season, July to September except in the pandemic year (2009) when surveillance occurred from June to October.

All reported rates based on child population estimates published by the Australian Bureau of Statistics.[2]

Table 2: Acute flaccid paralysis cases, by state and hospital

State or territory	Hospital(s)	Number of cases
NSW	Sydney Children's Hospital Randwick; The Children's Hospital at Westmead	2
NT	Alice Springs Hospital	1
SA	Women's and Children's Hospital Adelaide	1
Qld	Mater Hospital, Gold Coast University Hospital; Mackay Base Hospital	6
Vic.	Geelong Hospital; Monash Children's Hospital Clayton; Royal Children's Hospital Melbourne; Bendigo Hospital	14
WA	Princess Margaret Hospital	2

measures in the prevention of cCMV.[3] A study of postnatal valganciclovir therapy for infants with symptomatic congenital CMV disease, has shown moderate benefit for improved long term audiologic and neurodevelopmental outcomes, and no excess risk for adverse events.[4] Concerns regarding efficacy, and potential long term side effects of ganciclovir on gonadal function remain and the continued APSU surveillance for cCMV is extremely important. Whether vaccines under development[5] or trialled in pregnant women[6] can be used in the near future remains an open question in Australia. However, cCMV vaccination is likely to become available to Australians in the future, to prevent cCMV infection.

Congenital rubella

There were no notifications of congenital rubella to the APSU during 2014. The last reported cases of congenital rubella were in 2008 (1 confirmed case) and 2013 (3 confirmed cases). In total, there have been 57 cases of congenital rubella (53 confirmed and 4 probable cases) reported to the APSU during the study period (1993–2014).[7] It is mainly due to the National Immunisation Program that Australia has seen a reduction in congenital rubella infection. However, reports of imported and locally acquired cases among immigrant unvaccinated women during previous years reinforces the need for continued surveillance and a vigilant vaccination program.[7]

Perinatal exposure to HIV and HIV infection

There were 39 confirmed cases of perinatal exposure to HIV reported to the APSU in 2014. In addition, there were 3 cases of HIV infection in children born overseas (2 in Zimbabwe, 1 in Uzbekistan). Of the 39 confirmed cases of perinatal exposure to HIV, 17 were from Victoria, 12 were from New South Wales, 6 from Western Australia, 3 from the Australian Capital Territory and 1 from South Australia. None of the children with perinatal exposure to HIV identified as being of Aboriginal or Torres Strait Islander descent.

The majority of mothers of these cases were receiving antiretroviral therapy (n = 26; 74%). The most common mode of delivery for the perinatally exposed cases was vaginal delivery (n = 11; 31%), followed by elective caesarean (n = 7; 20%) and emergency caesarean (n = 7; 20%). Most mothers (n = 26) avoided breastfeeding their children. Over the total study period (1993–2014) 627 cases of perinatal exposure to HIV and 87 cases of HIV infection have been reported.

Neonatal or infant herpes simplex virus

There were 22 notifications of neonatal or infant HSV in 2014. Eighteen met the case definition criteria. Of these, 10 were neonatal cases (aged < 1 month) and 8 were infant onset cases (aged between 1 month and 1 year). Eight cases were reported from New South Wales, 5 from Queensland, 2 from Western Australia, 2 from Victoria and 1 from Tasmania. Of the 18 confirmed cases, 4 mothers identified as Aboriginal, 5 as Australian, 1 as African, and for 8 ethnicity was not provided.

Eleven had HSV-1; 5 had HSV-2 and in 2 cases the HSV strain was unknown. Eight children presented with central nervous system signs and 4 of these died.

HSV infection leads to significant mortality and morbidity. In the absence of screening in pregnancy, early detection and treatment is needed.[8]

Congenital and neonatal varicella

There were no cases of congenital varicella or neonatal varicella reported to the APSU during 2014. The last case of congenital varicella reported to the APSU was in 2007, while the last case of neonatal varicella was reported in 2013. This supports the success of the varicella vaccination program under the NIP in preventing the most severe manifestations of varicella infection.

Juvenile onset recurrent respiratory papillomatosis

There were 2 notifications of JoRRP to the APSU in 2014: 1 confirmed case (confirmed by visualisation via endoscopy and histology report), and 1 probable case (visualisation by endoscopy, but there was no histology report provided). A total of 12 cases (8 confirmed, 4 probable) were ascertained from 2012 to 2014. Of these 12 cases, 6 were from Western Australia, 5 from Queensland and 1 from New South Wales. The majority of children were Caucasian (n = 10), 1 child was of Aboriginal descent and ethnicity was not provided for 1 case.

JoRRP is a very rare condition characterised by the recurrent growths (papillomas) in the upper airways caused by persistent infection with human papillomavirus (HPV) genotypes HPV 6 or HPV 11. Acquisition of infection occurs via vertical transmission before or during birth, with the susceptible child unable to mount an adequate immune response to permanently clear or suppress the virus. HPV6 and HPV11 are targeted by the

prophylactic quadrivalent HPV vaccine, meaning that JoRRP is now potentially a vaccine preventable disease, because women of child-bearing age become immune to infection with HPV6 and 11, and antibodies generated by immunisation are also known to cross the placenta and provide antibodies detectable in the cord blood of neonates.[9]

Following the commencement of Australia's National HPV Vaccination Program in 2007, genital warts (also caused by HPV types 6 and 11) have almost disappeared in young women and have also markedly declined in young men due to herd protection.[10]

The number of reported cases of JoRRP has also declined from 6 confirmed and 1 probable case in 2012, to 1 confirmed and 2 probable cases in 2013, and 1 confirmed and 1 probable case in 2014.

Severe complications of influenza

A total of 83 children admitted to hospital with serious complications of laboratory confirmed influenza were reported to the APSU from July to September 2014. Of the 83 children, 37 were from Queensland, 28 from New South Wales, 9 from Western Australia, 8 from Victoria and 1 from South Australia. Six cases (7%) identified as being of Aboriginal or Torres Strait Islander descent.

This was a large increase in notifications compared with 2013 when only 13 cases were reported. Seventy-six (92%) had influenza A. Four children had influenza B and 1 child had both influenza A and influenza B. The most common serious complications were pneumonia (57%), seizures (19%) encephalitis (7%), and rhabdomyolysis (4%).

In 2014, 29 children required an intensive care unit admission and 1 child died. Of the 83 children, 62 were previously healthy, while 19 had chronic pre-disposing conditions (neuromuscular disorders, cerebral palsy, asthma, chronic lung disease, Rett syndrome).

Only 5 (6%) of the 83 children were vaccinated for influenza within the last 12 months. Of the 19 children with chronic predisposing conditions, only 3 (16%) were vaccinated. Children with chronic predisposing conditions are recommended and funded for annual influenza vaccination under the National Immunisation Program.

Conclusions and future directions

APSU surveillance provides valuable clinical, treatment and outcome data on infectious and vaccine preventable conditions in Australian children. The data from the APSU contribute significantly to the national surveillance effort, providing valuable information for clinicians, policymakers and the community.

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Australian Meningococcal Surveillance Programme annual report, 2014

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Abstract

In 2014 there were 165 laboratory-confirmed cases of invasive meningococcal disease analysed by the Australian National Neisseria Network. This number was higher than the number reported in 2013, but was the second lowest reported since inception of the Australian Meningococcal Surveillance Programme in 1994. Probable and laboratory confirmed invasive meningococcal disease (IMD) are notifiable in Australia, and there were 170 IMD cases notified to the National Notifiable Diseases Surveillance System (NNDSS) in 2014. This was also higher than in 2013, but was the second lowest number of IMD cases reported to the NNDSS. The meningococcal serogroup was determined for 161/165 (98%) of laboratory confirmed IMD cases. Of these, 80.1% (129 cases) were serogroup B infections; 1.9% (3 cases) were serogroup C infections; 9.9% (16 cases) were serogroup W135; and 8.1% (13 cases) were serogroup Y. Primary and secondary disease peaks were observed in those aged 4 years or less, and in adolescents (15–19 years) respectively. Serogroup B cases predominated in all jurisdictions and age groups, except for those aged 65 years or over, where serogroups Y and W135 combined predominated. The overall proportion and number of IMD caused by serogroup B was higher than in 2013, but has decreased from previous years. The number of cases of IMD caused by serogroup C was the lowest reported to date. The number of IMD cases caused by serogroup Y was similar to previous years, but the number of IMD cases caused serogroup W135 was higher than in 2013. The proportion of IMD cases caused by serogroups Y and W135 has increased in recent years, whilst the overall number of cases of IMD has decreased. Molecular typing was able to be performed on 106 of the 165 IMD cases. In 2014, the most common *porA* genotypes circulating in Australia were P1.7-2,4 and P1.22,14. All IMD isolates tested were susceptible to ceftriaxone and ciprofloxacin. There were 2 isolates that were resistant to rifampicin. Decreased susceptibility to penicillin was observed in 88% of isolates. *Commun Dis Intell* 2016;40(2):E221–E228.

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

Introduction

The Australian National Neisseria Network (NNN) is a 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. Participating NNN laboratories supply phenotypic and genotypic data on 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. These data also 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.

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.[3] 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. When *N. meningitidis* is grown from blood only, the IMD case is classified as septicaemia; cerebrospinal fluid

(CSF) only cultures are classified as meningitis. When *N. meningitidis* is grown from both blood and CSF cultures 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*.

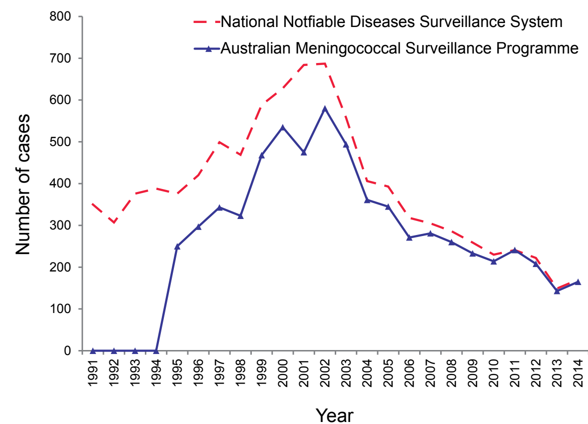
Antibiotic susceptibility testing

Isolates were tested to determine their minimum inhibitory concentration (MIC) values to antibiotics used for therapeutic and prophylactic purposes: ceftriaxone, ciprofloxacin; 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.[4] These are: sensitive (MIC ≤ 0.03 mg/L); less sensitive (MIC 0.06–0.5 mg/L) and resistant (MIC ≥ 1 mg/L).

Results

In 2014, there were 165 laboratory-confirmed cases of IMD analysed by the NNN, and 170 cases notified to the NNDSS. Thus, laboratory data were available for 97% of notified cases of IMD in Australia in 2014 (Figure 1). This was the second lowest annual number of IMD cases recorded by the NNDSS, and by the AMSP. In 2013, there were 149 IMD cases recorded by NNDSS and 143 laboratory confirmed IMD cases reported by the AMSP.) As in previous years, the peak incidence for IMD was in late winter and early spring (1 July to 30 September) (Table 1).

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, 2014



In 2014, the highest number of laboratory confirmed cases was from Queensland (39 cases), which was higher than that reported in this State in 2013 (32 cases). Other states that recorded a rise in IMD cases in 2014 compared with 2013 were: Victoria (33 cases in 2014, compared with 23 in 2013), and South Australia (31 cases in 2014, compared with 21 in 2013). By contrast, New South Wales recorded a fall in the number of IMD cases in 2014 (36 cases) compared with 2013 (43 cases). Numbers for the other states were similar to 2013 (Table 2).

Age distribution

Nationally, the peak number of IMD cases was in children less than 5 years of age, similar to previous years. Between 2007 and 2013, 28% to 36% of cases were in this age group. In 2014, 46/165 (28%) IMD cases occurred in this age group (Table 3). A secondary disease peak has also been observed in previous years among adolescents aged 15 to

Table 1: Laboratory confirmed cases of invasive meningococcal disease, Australia, 2014, by quarter

Serogroup	1 January–31 March	1 April– 30 June	1 July–30 September	1 October–31 December	2014 total
B	20	29	45	35	129
C	2	0	1	0	3
Y	0	7	3	3	13
W135	4	3	4	5	16
NG	0	0	0	0	0
ND	0	2	0	2	4
Total	26	41	53	45	165

NG: Non-groupable; ND: Not determined.

Table 2: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2014, by state or territory and serogroup

	Serogroup						
State or territory	B	C	Y	W135	NG	ND	Total
ACT	1	0	1	0	0	0	2
NSW	21	0	8	6	0	1	36
NT	4	0	0	0	0	0	4
Qld	31	1	2	3	0	2	39
SA	31	0	0	0	0	0	31
Tas.	1	0	0	1	0	0	2
Vic.	27	0	1	4	0	1	33
WA	13	2	1	2	0	0	18
Australia	129	3	13	16	0	4	165
%	78.2	1.8	7.9	9.7	0	2.4	

NG: Non-groupable; ND: Not determined

Table 3: Laboratory confirmed cases of invasive meningococcal disease, Australia, 2014, by age and serogroup

	Age group										
Serogroup	<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	Total
B	24	16	8	5	22	20	13	12	7	2	129
C	1	1	0	0	0	1	0	0	0	0	3
Y	1	0	0	0	2	1	1	1	7	0	13
W135	2	0	0	1	5	1	0	2	5	0	16
NG	0	0	0	0	0	0	0	0	0	0	0
ND	1	0	0	0	1	1	1	0	0	0	4
Total	29	17	8	6	30	24	15	15	19	2	165
% B of within age group	82.8	94.1	100.0	83.3	73.3	83.3	86.7	80.0	36.8		

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

19 years. Of the total cases of IMD, 30/165 (18%) were in those aged 15 to 19 years in 2014, which was the same as the proportion reported for 2013; and similar to the proportion reported in this age group during the period 2007 to 2011 (17% to 20%). The proportion of IMD cases in those aged 25 to 44 (14.5%, 24 cases) was almost double than that in 2013 (7.7%, 11 cases).

Anatomical site of samples and method of confirmation

In 2014, diagnosis was made by a positive culture in 95/165 (58%) cases and, 70/165 (42%) cases were confirmed by NAAT testing (Table 4).

There were 58 diagnoses of meningitis based on cultures or NAAT examination of CSF either

Table 4: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2014, by anatomical source and method of confirmation

Specimen type	Bacterial culture	NAAT	Total
Blood	69	34	103
CSF +/– blood	24	34	58
Other[‡]	2	2	4
Total	95	70	165

NAAT: Nucleic acid amplification testing;
CSF: Cerebrospinal fluid.

alone or with a positive blood sample. There were 103 diagnoses of septicaemia based on cultures or NAAT examination from blood samples alone (Table 4). There were 4 IMD diagnoses by positive joint fluid culture (n = 2) and NAAT (n = 2).

Serogroup data

Number of cases of invasive meningococcal disease by serogroup B, C, Y, W135

The serogroup was determined for 161 of 165 laboratory confirmed cases of IMD in 2014 (Tables 2 and 3). There has been an overall decrease in the number of cases of IMD in Australia in recent years, which was initially predominantly due to a reduction in the number of cases of IMD caused by serogroup C from 2003 to 2007. This was followed by a decline in the numbers IMD cases caused by serogroup B from 194 cases in 2009, to 104 cases in 2013. In 2014, there was an increase in the numbers of IMD cases caused by serogroup B (n = 129). The number of cases of IMD caused by serogroup Y has remained stable since 2011, whereas, the number of cases of serogroup W135 IMD has increased in recent years (7 to 16 cases in 2011 to 2014, compared with 4 to 9 cases in 2007 to 2010). In 2014 there were 16 cases of W135 IMD, the highest number ever reported by the AMSP.

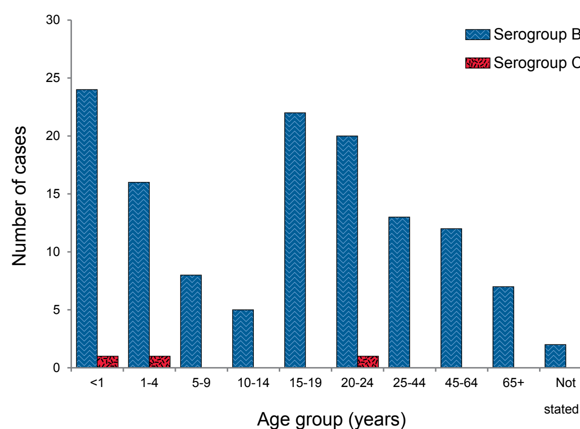
Proportions of serogroup B, C, Y, W135 invasive meningococcal disease

Of the 161 IMD strains for which the serogroup was determined, 80.1% were serogroup B, which was higher than in 2013 (74.8%), but lower than that reported in the years 2006 to 2012 (84% to 88%). The proportion of cases of IMD caused by serogroup B in children less than 5 years in 2014 was lower than in previous years (2008 to 2013) (Table 3, Figure 2). However, in young adults 20–24 years, the number of cases of serogroup B IMD in 2014 was higher than in 2007 to 2011 and 2013 (61% to 67%), and similar to 2012. The proportion of cases of IMD caused by serogroup B in those aged 15–19 has remained relatively stable since 2008, but was lower in 2014. Serogroup B IMD was prominent in IMD in all age groups excepting 65 years or more where, serogroup Y was equally prevalent, and serogroup W135 slightly less so.

The number and proportion of IMD caused by serogroup C in 2014 was lowest since the inception of the Australian Meningococcal Surveillance Programme (1.9%, 3 cases). Two of the 3 cases of IMD caused by serogroup C in 2014 were in those aged less than 20 years in 2014, compared with 1 case in 2013, 2 cases in 2012 and no cases in 2011 in this age group.

Of note, coincident with the decline in serogroup C IMD, the proportion of IMD caused by serogroups Y and W135 has been increasing in recent years. 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%. Similarly, the proportion by serogroup W135 IMD was 8.6% to 9.9% of IMD in 2013 to 2014, higher than the 1.8% to 4.5% reported in the period 2007 to 2011. The number and proportion of IMD cases caused by serogroup Y was highest in people aged 65 years or over in 2014. The number and proportion of IMD cases caused by serogroup W135 was highest in people aged 65 years or over, and also in people aged 15–19 years.

Figure 2: Number of serogroup B and C cases of confirmed invasive meningococcal disease, Australia, 2014, by age group



Genotyping

In 2014, genotyping was performed for 106/165 (64%) IMD cases (Tables 5 and 6). The predominant *porA* genotypes for serogroup B IMD cases were again P1.7-2,4 (14 cases) and P1.22,14 (14 cases). Other *porA* genotypes for serogroup B IMD cases more frequently seen in 2014 were P1.7,16-26 (7 cases); and P1.18-1,34 and P1.22,9 (6 cases each) (Figure 3). The AMSP was not aware of any epidemiological link between any of the cases reported where genotyping was available.

Antibiotic susceptibility testing

Testing for antimicrobial susceptibility was performed for 95/165 (58%) of the IMD cases in 2014. All isolates tested were susceptible to ceftriaxone and ciprofloxacin. There were 2 isolates that were resistant to rifampicin. Using the defined criteria, 11/95 (11.6%) isolates were fully sensitive to penicillin (MIC 0.03 mg/L or less), and 84 (88%) iso-

Table 5: Laboratory confirmed cases of invasive meningococcal disease, Australia, 2014, by *porA* genotype

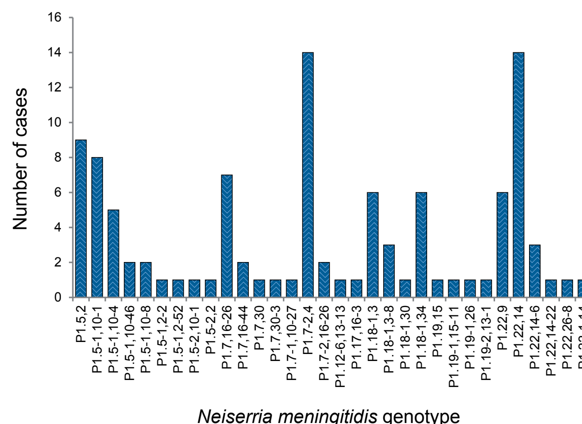
2014 AMSP	Number per serogroup				
Genotype *porA*	B	C	W135	Y	Total
P1.5,2	0	0	9	0	9
P1.5-1,10-1	1	0	1	6	8
P1.5-1,10-4	1	0	1	3	5
P1.5-1,10-46	1	0	0	1	2
P1.5-1,10-8	0	2	0	0	2
P1.5-1,2-2	0	0	0	1	1
P1.5-1,2-52	1	0	0	0	1
P1.5-2,10-1	0	0	0	1	1
P1.5-2,2	0	1	0	0	1
P1.7,16-26	7	0	0	0	7
P1.7,16-44	2	0	0	0	2
P1.7,30	1	0	0	0	1
P1.7,30-3	1	0	0	0	1
P1.7-1,10-27	1	0	0	0	1
P1.7-2,4	14	0	0	0	14
P1.7-2,16-26	2	0	0	0	2
P1.12-6,13-13	1	0	0	0	1
P1.17,16-3	1	0	0	0	1
P1.18-1,3	3	0	3	0	6
P1.18-1,3-8	3	0	0	0	3
P1.18-1,30	1	0	0	0	1
P1.18-1,34	6	0	0	0	6
P1.19,15	1	0	0	0	1
P1.19-1,15-11	1	0	0	0	1
P1.19-1,26	1	0	0	0	1
P1.19-2,13-1	1	0	0	0	1
P1.22,9	6	0	0	0	6
P1.22,14	14	0	0	0	14
P1.22,14-6	3	0	0	0	3
P1.22,14-22	1	0	0	0	1
P1.22,26-8	1	0	0	0	1
P1.22-1,14	1	0	0	0	1
Total	77	3	14	12	106

lates were less sensitive to penicillin (MIC = 0.06–0.5 mg/L). No isolates were resistant to penicillin. The proportion of strains less sensitive to penicillin was the highest recorded by the AMSP.

Discussion

In 2014, there were 165 cases of laboratory confirmed IMD, representing 97% of the number of notifications to the NNDSS.[2] This is both the second lowest number of cases reported since laboratory based surveillance for confirmed IMD cases

Figure 3: Number of *porA* genotypes (where data available) for serogroup B in cases of invasive meningococcal disease, Australia, 2014



(AMSP) began in 1994, and since notification data collection commenced in 1991. This represents less than one-third of the number reported in Australia in 2002 (n = 580), when IMD rates peaked in Australia. The introduction of the serogroup C vaccine to the national immunisation schedule in 2003 has led to a steady decline in the total number of both serogroup C, and the overall number of cases of IMD. The primary peak in IMD infection continues to be in children aged less than 5 years, as reported in previous years, with a secondary peak in adolescents.

The majority of IMD cases in Australia are caused by serogroup B. The proportion and number of IMD cases caused by serogroup C was the lowest reported by the AMSP since the beginning of the program. The number of IMD cases caused by serogroup Y was similar to previous years. The number and proportion of cases caused by serogroup W135 was the highest reported by the AMSP. The proportion of IMD cases caused by serogroups Y and W135 has increased in recent years, coincident with the overall reduction in numbers of IMD cases, and are the predominant serogroups causing IMD in those aged 65 years or over.

As in previous years, genotypic data found no evidence of a substantial number of cases of IMD caused by *N. meningitidis* that have undergone genetic recombination. There have been concerns that the emergence of new and invasive subtypes following extensive vaccine use would occur given the capacity for genetic recombination within meningococci.[5] Therefore the monitoring of meningococcal genotypes is an important part of the NNN program.

Table 6: Distribution of *porA* genotype laboratory confirmed cases of invasive meningococcal disease, Australia, 2014, by state or territory

2014 AMSP	Number per serogroup per state							
Genotype *porA*	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA
P1.5,2		2 W135		2 W135		2 W135	2 W135	2 W135
P1.5-1,10-1		6 Y,1 W135					1 B	
P1.5-1,10-4		1 W135	1 B	1 Y			1 Y	1 Y
P1.5-1,10-46	1 Y						1 B	
P1.5-1,10-8								2 C
P1.5-1,2-2				1 Y				
P1.5-1,2-52		1 B						
P1.5-2,10-1		1 Y						
P1.5-2,2				1 C				
P1.7,16-26		3 B		3 B			1 B	
P1.7,16-44		1 B		1 B				
P1.7,30								1 B
P1.7,30-3					1 B			
P1.7-1,10-27				1 B				
P1.7-2,4		1 B	1 B	7 B			4 B	1 B
P1.7-2,16-26				1 B			1 B	
P1.12-6,13-13								1 B
P1.17,16-3		1 B						
P1.18-1,3		1 W135					3 B, 2 W135	
P1.18-1,3-8				3 B				
P1.18-1,30				1 B				
P1.18-1,34				4 B			1 B	1 B
P1.19,15							1 B	
P1.19-1,15-11		1 B						
P1.19-1,26				1 B				
P1.19-2,13-1								1 B
P1.22,9		2 B					4 B	
P1.22,14		2 B		5 B			3 B	4 B
P1.22,14-6		2 B		1 B				
P1.22,14-22		1 B						
P1.22,26-8					1 B			
P1.22-1,14				1 B				

All isolates were susceptible to ceftriaxone and ciprofloxacin; whilst there were 2 IMD isolates that were resistant to rifampicin. The proportion of IMD isolates with penicillin MIC values in the less sensitive category in 2014 was 88%, and was the highest proportion recorded by the AMSP. In previous years the range was 62% to 75% in 1996 to 2006; 67% to 79% in 2007 to 2009; and 78% to 85% in 2010 to 2013. Thus indicating a right shift in penicillin MIC values of IMD isolates, however, in Australia, the incidence of penicillin resistance in *N. meningitidis* is very low.

In early 2014, a recombinant multi-component meningococcal B vaccine became available in Australia.(6) 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. The AMSP continues to monitor the phenotypic and genotypic features of *N. meningitidis* causing IMD to inform treatment protocols and monitor prevention strategies.

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Australian Group on Antimicrobial Resistance Australian Enterobacteriaceae Sepsis Outcome Programme annual report, 2014

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Abstract

The Australian Group on Antimicrobial Resistance performs regular period-prevalence studies to monitor changes in antimicrobial resistance in selected enteric Gram-negative pathogens. The 2014 survey was the second year to focus on blood stream infections. During 2014, 5,798 Enterobacteriaceae species isolates were tested using commercial automated methods (Vitek 2, BioMérieux; Phoenix, BD) and results were analysed using the Clinical and Laboratory Standards Institute (CLSI) and European Committee on Antimicrobial Susceptibility Testing (EUCAST) breakpoints (January 2015). Of the key resistances, non-susceptibility to the third-generation cephalosporin, ceftriaxone, was found in 9.0%/9.0% of *Escherichia coli* (CLSI/EUCAST criteria) and 7.8%/7.8% of *Klebsiella pneumoniae,* and 8.0%/8.0% *K. oxytoca.* Non-susceptibility rates to ciprofloxacin were 10.4%/11.6% for *E. coli,* 5.0%/7.7% for *K. pneumoniae,* 0.4%/0.4% for *K. oxytoca,* and 3.5%/6.5% in *Enterobacter cloacae.* Resistance rates to piperacillin-tazobactam were 3.2%/6.8%, 4.8%/7.2%, 11.1%/11.5%, and 19.0%/24.7% for the same 4 species respectively. Fourteen isolates were shown to harbour a carbapenemase gene, 7 bla_{IMP-4}, 3 bla_{KPC-2}, 3 bla_{VIM-1}, 1 bla_{NDM-4}, and 1 $bla_{OXA-181-lke}$.*Commun Dis Intell* 2016;40(2):E229–E235.

Keywords: antibiotic resistance; bacteraemia; gram-negative; *Escherichia coli; Enterobacter; Klebsiella*

Introduction

Emerging resistance in common pathogenic members of the Enterobacteriaceae is a world-wide phenomenon, and presents therapeutic problems for practitioners in both the community and in hospital practice. The Australian Group on Antimicrobial Resistance (AGAR) commenced surveillance of the key Gram-negative pathogens, *Escherichia coli* and *Klebsiella* species in 1992. Surveys have been conducted biennially until 2008 when annual surveys commenced, alternating between community– and hospital-onset infections.[1] In 2004, another genus of Gram-negative pathogens in which resistance can be of clinical importance, *Enterobacter* species, was added. *E. coli* is the most common cause of community-onset urinary tract infection, while *Klebsiella* species are less common but are known to harbour important resistances. *Enterobacter* species are less common in the community, but of high importance due to intrinsic resistance to first-line antimicrobials in the community. Taken together, the three groups of species surveyed are considered to be valuable sentinels for multi-resistance and emerging resistance in enteric Gram-negative bacilli. In 2013 AGAR commenced the Enterobacteriaceae Sepsis Outcome Programme (EnSOP), which focused on the collection of resistance and some demographic data on all isolates prospectively from patients with bacteraemia. The 2014 survey was the second EnSOP survey.

Resistances of particular interest include resistance to ß-lactams due to ß-lactamases, especially extended-spectrum ß-lactamases, which inactivate the third-generation cephalosporins that are normally considered reserve antimicrobials. Other resistances of interest are to agents important for treatment of these serious infections, such as gentamicin; and resistance to reserve agents such as ciprofloxacin and meropenem.

The objectives of the 2014 surveillance program were to:

1. monitor resistance in Enterobacteriaceae isolated from blood;
2. examine the extent of co-resistance and multi-resistance; and
3. detect emerging resistance to newer last-line agents such as carbapenems.

Methods

Study design

From 1 January to 31 December 2014, 26 institutions across Australia collected either all or up to 200 isolates from different patient episodes of bacteraemia.

Species identification

Isolates were identified using the routine method for each institution; Vitek®, Phoenix™ Automated Microbiology System, or where available, mass spectrometry (MALDI-TOF).

Susceptibility testing

Testing was performed by 2 commercial semi-automated methods, Vitek 2 (BioMérieux) or Phoenix (BD), which are calibrated to the ISO reference standard method of broth microdilution. Commercially available Vitek AST-N246, or Phoenix NMIC-203 cards were utilised by all participants throughout the survey period. The Clinical and Laboratory Standards Institute (CLSI) M100[2] and European Committee on Antimicrobial Susceptibility Testing (EUCAST) v5.0[3] breakpoints from January 2015 have been employed in the analysis. For analysis of cefazolin, breakpoints of ≤ 4 for susceptible, and ≥ 8 for resistant were applied due to the restricted minimum inhibitory concentration (MIC) range available on the commercial cards, recognising that the January 2015 breakpoint is actually susceptible ≤ 2 mg/L.

Molecular confirmation of resistances

E. coli and *Klebsiella* isolates with ceftazidime or ceftriaxone MIC > 1 mg/L, or cefoxitin MIC > 8 mg/L; *Enterobacter* spp. with cefepime MIC > 1 mg/L; all isolates with ciprofloxacin MIC > 0.25 mg/L; all isolates with meropenem MIC > 0.25 mg/L; and all isolates with amikacin MIC > 32 mg/L were referred to a central laboratory (SA Pathology) for molecular confirmation of resistance.

All referred isolates were screened for the presence of the bla_{TEM}, and bla_{SHV} genes using a real-time polymerase chain reaction (PCR) platform (LC-480) and published primers.[4,5] A multiplex real-time TaqMan PCR was used to detect CTX-M-type genes.[6] Strains were probed for plasmid-borne AmpC enzymes using the method described by Pérez-Pérez and Hanson,[7] and subjected to molecular tests for MBL (bla_{VIM}, bla_{IMP}, and bla_{NDM}), bla_{KPC}, and $bla_{OXA\text{-}48\text{-}like}$ genes using real-time PCR.[8,9] Known plasmid mediated quinolone resistance mechanisms (Qnr, efflux (*qepA, oqxAB*), and aac(6')-Ib-cr) were examined by PCR on all referred isolates with ciprofloxacin MIC > 0.25 mg/L using published methods.[10,11] All *E. coli* were examined for presence of the O25b-ST131 clone and its H30- and H30-Rx subclones.[12–14]

Results

The species isolated, and the numbers of each are listed in Table 1. Three genera, *Escherichia* spp., *Klebsiella* spp. and *Enterobacter* spp. contributed 87.6% of all isolates. Major resistances and non-susceptibilities for the top 6 ranked species are listed in Table 2. Non-susceptibility, (which includes both intermediately resistant and resistant strains), has been included for some agents because these figures provide information about important emerging acquired resistances. Multiple acquired resistances by species are shown in Table 3. Multi-resistance was detected in 13.4% of *E. coli* isolates, 9.7% of *K. pneumoniae*, and 12.1% of *Ent. cloacae*. A more detailed breakdown of resistances and non-susceptibilities by state and territory is provided in the online report from the group (http://www.agargroup.org/surveys).

Table 1: Species tested

Species	Total	%
Escherichia coli	3,493	60.2
Klebsiella pneumoniae	877	15.1
Enterobacter cloacae	343	5.9
Klebsiella oxytoca	226	3.9
Proteus mirabilis	187	3.2
Serratia marcescens	136	2.3
Enterobacter aerogenes	105	1.8
Salmonella species (non Typhi)	94	1.6
Morganella morganii	57	1.0
Citrobacter freundii	53	0.9
Citrobacter koseri	50	0.9
Salmonella Typhi/Paratyphi	26	0.4
Enterobacter asburiae	16	0.3
Raoultella ornithinolytica	15	0.3
Pantoea species	12	0.2
Pantoea agglomerans	12	0.2
Enterobacter species	11	0.2
Providencia stuartii	10	0.2
Other species (n=27)	75	1.3
Total	5,798	

Escherichia coli

Moderately high levels of resistance to ampicillin (and therefore amoxycillin) were maintained (50.1%/51.9%, CLSI/EUCAST criteria), with lower rates for amoxycillin-clavulanate (12.7%/-intermediate, 8.2%/20.9% resistant). Non-susceptibility to third-generation cephalosporins was low (ceftriaxone 9.0%/9.0%, ceftazidime

Table 2: Non-susceptibility and resistance rates for the top 6 ranked species tested

Antimicrobial	Category*	*Escherichia coli* (%)		*Klebsiella pneumoniae* (%)		*Klebsiella oxytoca* (%)		*Enterobacter cloacae* (%)		*Proteus mirabilis* (%)		*Serratia marcescens* (%)	
		CLSI	EUCAST	CLSI	EUCAST	CLSI	EUCAST	CLSI	EUCAST	CLSI	EUCAST	CLSI	EUCAST
Ampicillin	I	1.8	–	†	†	†	†	†	†	0.5	–	†	†
	R	50.1	51.9	†	†	†	†	†	†	16.8	17.3	†	†
Amoxycillinclavulanate‡	I	12.7	–	5.1	–	4.4	–	†	†	8.7	–	†	†
	R	8.2	–	5.3	–	8.8	–	†	†	1.6	–	†	†
Ticarcillin-clavulanate	R	9.4	19.3	7.2	11.4	10.4	12.2	24.4	29.7	1.1	1.7	0.0	2.2
Piperacillintazobactam	R	3.2	6.8	4.8	7.2	11.1	11.5	19.0	24.7	1.1	1.6	0.0	0.0
Cefazolin	R	20.5	/	13.0	/	66.0	/	†	†	26.5	/	†	†
Cefoxitin	R	3.8	/	6.2	/	0.9	/	†	†	0.0	/	†	†
Ceftriaxone	NS	9.0	9.0	7.8	7.8	8.0	8.0	27.6	27.6	0.5	0.5	2.9	2.9
Ceftazidime	NS	4.4	8.0	6.1	8.0	0.4	0.4	24.6	27.0	0.0	0.0	2.2	2.2
Cefepime	NS	3.3	6.4	3.7	6.1	0.0	0.0	4.1	14.4	1.1	1.1	1.5	2.2
Meropenem	NS	0.1	0.1	1.1	1.0	0.0	0.0	2.9	2.3	0.5	0.5	0.7	0.7
Ciprofloxacin	NS	10.4	11.6	5.0	7.6	0.4	0.4	3.5	6.5	2.7	3.2	1.5	3.7
Norfloxacin	NS	10.4	18.2	4.5	13.7	0.0	1.8	3.2	13.5	3.2	5.4	0.7	3.7
Gentamicin	NS	7.5	8.0	5.5	6.1	1.3	1.8	6.7	7.6	1.6	2.2	1.5	1.5
Trimethoprim	R	29.2	29.4	15.5	16.6	4.0	4.4	19.1	19.1	21.7	22.3	1.5	2.2
Nitrofurantoin	NS	6.6	1.6	88.8	/	41.6	/	72.6	/	†	†	†	†

* R = resistant, I = intermediate, NS = non-susceptible (intermediate + resistant), using criteria as published by the Clinical and Laboratory Standards Institute (CLSI) [2014] and European Committee on Antimicrobial Susceptibility Testing (EUCAST) [2014].

† Considered largely intrinsically resistant due to natural β-lactamases; – no intermediate category; / no breakpoints defined

‡ For EUCAST interpretation, the clavulanate is fixed at 2 mg/L, rather than a 2:1 ratio used in CLSI guidelines. As all cards used have a 2:1 ratio of clavulanate no EUCAST category has been applied.

Table 3: Multiple acquired resistances, by species

Species	Total	Number of acquired resistances (CLSI breakpoints)															
		Non-multi-resistant					Multi-resistant										
		0	1	2	3	Cumulative %	4	5	6	7	8	9	10	11	12	13	Cumulative %
Escherichia coli	2,958	1,324	506	505	226		133	109	71	44	22	11	6	0	0	0	
	%	44.8	17.1	17.1	7.6	86.6	4.5	3.7	2.4	1.5	0.7	0.4	0.2	0.0	0.0	0.0	13.4
*Klebsiella pneumoniae**	746	417	192	51	14		17	15	11	6	6	5	4	2	6		
	%	55.9	25.7	6.8	1.9	90.3	2.3	2.0	1.5	0.8	0.8	0.7	0.5	0.3	0.8		9.7
Enterococcus cloacae†	330	163	66	24	37		20	12	5	1	2						
	%	49.4	20.0	7.3	11.2	87.9	6.1	3.6	1.5	0.3	0.6						12.1
Proteus mirabilis	148	3	87	35	13		7	2	0	0	0	0	1				
	%	2.0	58.8	23.6	8.8	93.2	4.7	1.4	0.0	0.0	0.0	0.0	0.7				6.8
Serratia marcescens†	98	1	94	0	1		2										
	%	1.0	95.9	0.0	1.0	98.0	2.0										2.0
*Klebsiella oxytoca**	197	66	98	14	10		7	2									
	%	33.5	49.7	7.1	5.1	95.4	3.6	1.0									4.6
Enterococcus aerogenes†	104	36	31	12	18		5	1	1								
	%	34.6	29.8	11.5	17.3	93.3	4.8	1.0	1.0								6.7
Salmonella spp. (non-Typhi)	71	57	8	4	2												
	%	80.3	11.3	5.6	2.8	100.0											0.0

* Antibiotics included: amoxycillin-clavulanate, piperacillin-tazobactam, cefazolin, cefoxitin, ceftriaxone, ceftazidime, cefepime, gentamicin, amikacin, ciprofloxacin, nitrofurantoin, trimethoprim, meropenem;
Antibiotics excluded: ampicillin (intrinsic resistance), ticarcillin-clavulanate, tobramycin, norfloxacin, nalidixic acid, sulfamethoxazole-trimethoprim (high correlation with antibiotics in the included list)

† Antibiotics included: piperacillin-tazobactam, ceftriaxone, ceftazidime, cefepime, gentamicin, amikacin, ciprofloxacin, nitrofurantoin, trimethoprim, meropenem
Antibiotics excluded: ampicillin, amoxycillin-clavulanate, cefazolin, and cefoxitin, (all four due to intrinsic resistance); also excluded were ticarcillin-clavulanate, tobramycin, norfloxacin, nalidixic acid, sulfamethoxazole-trimethoprim (high correlation with antibiotics in the included list).

4.4%/8.0%). Moderate levels of resistance were detected to cefazolin (20.5%/-) and trimethoprim (29.2%/29.4%). Ciprofloxacin non-susceptibility was found in 10.4%/11.6% of *E. coli* isolates. Resistance to ticarcillin-clavulanate (9.4%/19.3%), gentamicin (7.3%/7.5%), piperacillin-tazobactam (3.2%/6.8%), and cefepime (1.6%/2.9%) were low. Nine isolates had elevated meropenem MICs (≥ 0.5 mg/L). For the extended-spectrum ß-lactamase (ESBL)-producing strains, ciprofloxacin and gentamicin resistance was found in 51.6%/51.6% and 33.8%/34.1% respectively.

In line with international trends among community strains of *E. coli*, most of the strains with ESBL genes harboured genes of the CTX-M type (222/272 = 82%). Over 60% of *E. coli* with CTX-M group 1 types were found to belong to sequence type 131 (O25b-ST131). ST131 accounted for 68% of *E. coli* ESBL phenotypes that were ciprofloxacin resistant (MIC > 1 mg/L), and only 6% of ciprofloxacin susceptible ESBL phenotypes. Ninety-one per cent and 41% of O25b-ST131 were associated with the H30 and *H30-Rx* subclones, respectively, with their reported association with more antibiotic resistances and greater virulence potential.[13]

Klebsiella pneumoniae

K. pneumoniae showed slightly higher levels of resistance to piperacillin-tazobactam and ceftazidime compared with *E. coli*, but lower rates of resistance to amoxycillin-clavulanate, ticarcillin-clavulanate, cefazolin, ceftriaxone ciprofloxacin, gentamicin, and trimethoprim. Thirteen *K. pneumoniae* isolates had elevated meropenem MICs. ESBLs were present in 61 of 69 (88%) presumptively ESBL-positive isolates of *K. pneumoniae,* 47 (77%) of which proved to be of the CTX-M type.

Enterobacter species

Acquired resistance was common to ticarcillin-clavulanate (24.4%/29.7% and 26.7%/40.6%), piperacillin-tazobactam (19.0%/24.7% and 23.1%/31.7%), ceftriaxone (27.3%/27.3% and 37.5%/37.5%), ceftazidime (24.3%/24.6% and 29.8%/33.7%) and trimethoprim (19.1%/19.1% and 1.9%/1.9%) for *Ent. cloacae* and *Ent. aerogenes,* respectively. Cefepime, ciprofloxacin, and gentamicin resistance were all less than 10%. Seventeen of 45 *Ent. cloacae* tested for ESBL based on a suspicious phenotype, harboured ESBL-encoding genes. Eighteen *Ent. cloacae* strains had elevated meropenem MICs.

Carbapenemase resistance

Overall, 14 isolates (14 patients) in 9 institutions from 5 states or territories were found to harbour a carbapenemase gene. Bla_{IMP-4} was detected in *E. cloacae* (5) and *K. pneumoniae* (2); bla_{KPC-2} was detected in 3 *K. pneumoniae* isolates from 1 institution; bla_{VIM-1} was detected in 2 *K. pneumoniae;* bla_{NDM-4} in 1 *E. coli,* and $bla_{OXA-181-like}$ in 1 *K. pneumoniae.*

Discussion

AGAR has been tracking resistance in sentinel enteric Gram-negative bacteria since 1992. From 2008, surveillance was segregated into hospital-versus community-onset infections. The last year of hospital-onset only surveillance was 2011.[15] In 2013, the first survey of antimicrobial resistance among Enterobacteriaceae isolates from bacteraemic patients throughout Australia was conducted using an approach similar to that conducted by the European EARS-Net program. The 2014 survey was the second survey conducted of antimicrobial resistance among Enterobacteriaceae isolates from bacteraemic patients throughout Australia.

CTX-M-producing *E. coli* and *Klebsiella* species and gentamicin- and ciprofloxacin-resistant *E. coli* continued to be a problem in patients with bacteraemia. Of concern is the high proportion of *E. coli* that belong to the ST131 *H30-Rx* subclone, and its reported association with more antibiotic resistance and greater virulence potential.[13] Carbapenem resistance attributable to acquired carbapenemases are still uncommon in patients with bacteraemia in Australia, although 5 different types (IMP, KPC, VIM, NDM and OXA-181-like) were detected from 9 of the participating institutions. Compared with many other countries in our region, resistance rates in Australian Gram-negative bacteria are still relatively low,[16] but similar to those observed in 2014 in many Western European countries.[17]

Multi-resistance is being increasingly observed, especially in *E. coli* and *E. cloacae,* both of which have multi-resistance rates (as defined by AGAR) above 10%. This is likely to drive more broad-spectrum antibiotic use, and increase the resistance selection pressure for important reserve classes, especially the carbapenemases.

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Australian Group on Antimicrobial Resistance Australian Enterococcal Sepsis Outcome Programme annual report, 2014

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Abstract

From 1 January to 31 December 2014, 27 institutions around Australia participated in the Australian Enterococcal Sepsis Outcome Programme (AESOP). The aim of AESOP 2014 was to determine the proportion of enterococcal bacteraemia isolates in Australia that were antimicrobial resistant, and to characterise the molecular epidemiology of the *Enterococcus faecium* isolates. Of the 952 unique episodes of bacteraemia investigated, 94.4% were caused by either *E. faecalis* (54.9%) or *E. faecium* (39.9%). Ampicillin resistance was detected in 0.6% of *E. faecalis* and in 89.4% of *E. faecium*. Vancomycin non-susceptibility was reported in 0.2% and 46.1% of *E. faecalis* and *E. faecium* respectively. Overall 51.1% of *E. faecium* harboured *vanA* or *vanB* genes. For the *vanA/B* positive *E. faecium* isolates, 81.5% harboured *vanB* genes and 18.5% *vanA* genes. The percentage of *E. faecium* bacteraemia isolates resistant to vancomycin in Australia is significantly higher than that seen in most European countries. *E. faecium* consisted of 113 pulsed-field gel electrophoresis pulsotypes of which 68.9% of isolates were classified into 14 major pulsotypes containing 5 or more isolates. Multilocus sequence typing grouped the 14 major pulsotypes into clonal cluster 17, a major hospital-adapted polyclonal *E. faecium* cluster. The geographical distribution of the 4 predominant sequence types (ST203, ST796, ST555 and ST17) varied with only ST203 identified across most regions of Australia. Overall 74.7% of isolates belonging to the four predominant STs harboured *vanA* or *vanB* genes. In conclusion, the AESOP 2014 has shown enterococcal bacteraemias in Australia are frequently caused by polyclonal ampicillin-resistant high-level gentamicin resistant *vanA* or *vanB E. faecium*, which have limited treatment options. *Commun Dis Intell* 2016;40(2):E236–E243.

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

Introduction

Globally enterococci are thought to account for approximately 10% of all bacteraemias, and in North America and Europe are the 4th and 5th leading causes of sepsis respectively.[1,2] Although in the 1970s healthcare-associated enterococcal infections were primarily due to *Enterococcus faecalis,* there has been a steadily increasing prevalence of *E. faecium* nosocomial infections.[3–5] Worldwide, the increase in nosocomial *E. faecium* infections has primarily been due to the expansion of polyclonal hospital-adapted clonal complex (CC) 17 strains. While innately resistant to many classes of antibiotics, *E. faecium* has demonstrated a remarkable capacity to evolve new antimicrobial resistances. In 2009, the Infectious Diseases Society of America highlighted *E. faecium* as one of the key problem bacteria or ESKAPE (*Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa,* and *Enterobacter species*) pathogens requiring new therapies.[6]

The Australian Group on Antimicrobial Resistance (AGAR) is a network of laboratories located across Australia that commenced surveillance of antimicrobial resistance in *Enterococcus* species in 1995.[7] In 2011, AGAR commenced the Australian Enterococcal Sepsis Outcome Programme (AESOP).[8] The objective of AESOP 2014 was to determine the proportion of *E. faecalis* and *E. faecium* bacteraemia isolates demonstrating antimicrobial resistance with particular emphasis on:

1. assessing susceptibility to ampicillin;
2. assessing susceptibility to glycopeptides; and
3. molecular epidemiology of *E. faecium*.

Methods

Twenty-seven laboratories from all 8 Australian states and territories participated in 2014.

Collection period

From 1 January to 31 December 2014, the 27 laboratories collected all enterococcal species isolated from blood cultures. Enterococci with the same species and antimicrobial susceptibility profiles isolated from a patient's blood culture within 14 days

of the first positive culture were excluded. A new enterococcal sepsis episode in the same patient was recorded if it was confirmed by a further culture of blood taken more than 14 days after the initial positive culture. Data were collected on age, sex, date of admission and discharge (if admitted), and mortality at 30 days from date of blood culture collection. To avoid interpretive bias, no attempt was made to assign attributable mortality. Each episode of bacteraemia was designated as 'hospital onset' if the first positive blood culture(s) in an episode was collected more than 48 hours after admission.

Laboratory testing

Enterococcal isolates were identified to the species level by the participating laboratories using one of the following methods: API 20S (bioMérieux), API ID32Strep (bioMérieux), Vitek2® (bioMérieux), Phoenix™ (BD), matrix-assisted laser desorption ionization (MALDI) Biotyper (Bruker Daltonics), Vitek-MS (bioMérieux), polymerase chain reaction (PCR), or conventional biochemical tests. Antimicrobial susceptibility testing was performed by using the Vitek2® (bioMérieux, France) or the Phoenix™ (BD, USA) automated microbiology systems according to the manufacturer's instructions. Minimum inhibitory concentration (MIC) data and isolates were referred to the Australian Collaborating Centre for *Enterococcus* and *Staphylococcus* Species (*ACCESS*) Typing and Research. Clinical and Laboratory Standards Institute (CLSI) and European Committee on Antimicrobial Susceptibility Testing (EUCAST) breakpoints were utilised for interpretation.[9,10] Isolates with either a resistant or an intermediate category were classified as non-susceptible. Linezolid and daptomycin non-susceptible isolates and selected vancomycin susceptible isolates were retested by Etest® (bioMérieux, France) using the Mueller-Hinton agar recommended by the manufacturer. *E. faecalis* ATCC® 29212 was used as the control strain. Molecular testing including *vanA/B* PCR, pulsed-field gel electrophoresis (PFGE) and multilocus sequence typing (MLST) was performed as previously described.[11–13]

A chi-square test for comparison of 2 proportions was performed and 95% confidence intervals (95% CI) were determined using MedCalc for Windows, version 12.7 (Medcalc Software, Ostend Belgium).

Approval to conduct the prospective data collection was given by the research ethics committee associated with each participating laboratory.

Results

From 1 January to 31 December 2014, 952 unique episodes of enterococcal bacteraemia were identified. Although 10 *Enterococcus* species were identified, 54.9% (523 isolates) were *E. faecalis* and 39.9% (380 isolates) were *E. faecium*. Forty-nine enterococci were identified either as *E. casseliflavus* (19 isolates), *E. gallinarum* (13), *E. avium* (9), *E. hirae* (2) *E. raffinosus* (3), *E. durans* (1), *E. cecorum* (1), *and E. mundtii* (1).

A significant difference was seen in patient sex ($P < 0.0001$) with 613 (64.4%) being male (95% CI, 61.4–67.5). The average age of patients was 63 years ranging from 0 to 100 years with a median age of 67 years. Of the 952 episodes, 474 (49.8%) were hospital onset (95% CI, 46.5–52.9). However, a significant difference was seen between *E. faecium* and *E. faecalis,* with 71.8% (95% CI, 67.0–76.3) of *E. faecium* episodes being hospital onset compared with 36.5% (95% CI, 32.4–40.8) for *E. faecalis* ($P < 0.0001$). All-cause mortality at 30 days was 18.5% (95% CI, 15.9–21.3). There was a significant difference in mortality between *E. faecalis* and *E. faecium* episodes (13.2% [95% CI, 10.3–16.6] vs 27.6% [95% CI, 22.9–32.7] respectively, $P < 0.0001$) and between vancomycin susceptible and vancomycin non-susceptible *E. faecium* episodes (22.8% [95% CI, 16.9–29.6] vs 32.9% [95% CI, 25.7–40.7] respectively, $P = 0.05$).

Enterococcus faecalis phenotypic susceptibility

Apart from erythromycin, tetracycline, ciprofloxacin and high-level gentamicin, acquired resistance was rare among *E. faecalis* (Table 1). Ampicillin resistance was detected in 3 isolates and only 1 isolate was vancomycin non-susceptible. Thirty-six (6.9%) *E. faecalis,* were initially reported as linezolid non-susceptible (CLSI breakpoint > 2 mg/L). However by Etest®, 22 of the 35 isolates available for MIC testing by Etest® had a linezolid MIC of ≤ 2 mg/L and were therefore considered linezolid susceptible. Thirteen isolates with an MIC of 4 mg/L, although non-susceptible by CLSI guidelines, were considered susceptible by EUCAST guidelines. Eight (1.6%) isolates were initially reported non-susceptible to daptomycin (CLSI and EUCAST breakpoint > 4 mg/L). However by Etest®, 7 of the 8 isolates had an MIC of < 4 mg/L and were therefore considered susceptible. One isolate had an MIC of 8 mg/L, which was considered non-susceptible. All isolates were susceptible to teicoplanin.

Enterococcus faecium phenotypic susceptibility

The majority of *E. faecium* were non-susceptible to multiple antimicrobials (Table 2). Most isolates were non-susceptible to ampicillin, erythromycin, tetracycline, ciprofloxacin, nitrofurantoin and high-level gentamicin. Overall, 175 (46.1%) were phenotypically vancomycin non-susceptible (MIC > 4 mg/L). Thirty-one (8.2%) and 33 (8.8%) isolates were

Table 1: The number and proportion of Enterococcus faecalis non-susceptible to ampicillin and the non-β-lactam antimicrobials, Australia, 2014

Antimicrobial	Tested	Breakpoint (mg/L)	Non-susceptible n	Non-susceptible %
Ampicillin	522	>8*	3	0.6
		>4†	3	0.6
Vancomycin	523	>4‡	1	0.2
Erythromycin	509	>0.5*	446	87.4
Tetracycline	501	>4*	363	72.5
Ciprofloxacin	477	>1*	122	25.6
Daptomycin	490	>4*	1	0.2
Teicoplanin	521	>8*	0	0
		>2†	0	0
Linezolid	522	>2*	13	2.5
		>4†	0	0
Nitrofurantoin	521	>32*	11	2.1
		>64†	2	0.4
High level gentamicin	519	>128*	198	38.2

* Clinical and Laboratory Standards Institute (CLSI) non-susceptible breakpoint.
† European Committee on Antimicrobial Susceptibility Testing (EUCAST) non-susceptible breakpoint.
‡ CLSI and EUCAST non-susceptible breakpoint.

Table 2: The number and proportion of Enterococcus faecium non-susceptible to ampicillin and the non-β-lactam antimicrobials, Australia, 2014

Antimicrobial	Tested	Breakpoint (mg/L)	Non-susceptible n	Non-susceptible %
Ampicillin	379	>8*	339	89.5
		>4†	343	90.5
Vancomycin	380	>4‡	175	46.1
Erythromycin	371	>0.5*	351	94.6
Tetracycline	369	>4*	194	52.6
Ciprofloxacin	351	>1*	321	91.5
Teicoplanin	377	>8*	31	8.2
		>2†	33	8.8
Linezolid	378	>2*	2	0.5
		>4†	1	0.3
Nitrofurantoin	377	>32*	289	76.5
		>64†	137	36.2
High level gentamicin	377	>128*	233	61.5

* Clinical and Laboratory Standards Institute (CLSI) non-susceptible breakpoint.
† European Committee on Antimicrobial Susceptibility Testing (EUCAST) non-susceptible breakpoint.
‡ CLSI and EUCAST non-susceptible breakpoint.

teicoplanin non-susceptible by CLSI and EUCAST guidelines respectively. Nine (2.4%) isolates were initially reported as linezolid non-susceptible (CLSI breakpoint > 2 mg/L). However by Etest®, 7 of the 9 isolates had a linezolid MIC of ≤ 2 mg/L. One isolate had an MIC of 4 mg/L, which was considered susceptible by EUCAST guidelines but non-susceptible by CLSI guidelines. One isolate had an MIC of 8 mg/L, which was considered non-susceptible by CLSI and EUCAST guidelines.

Genotypic vancomycin susceptibility

VanA/vanB PCR was performed on 512 of the 523 *E. faecalis* isolates. Overall, 7 (1.4%) of the 512 isolates harboured a *vanA* or *vanB* gene. The vancomycin non-susceptible *E. faecalis* isolate (Vitek® vancomycin MIC ≥ 32 mg/L) harboured a *vanB* gene. One phenotypically vancomycin/teicoplanin susceptible isolate (Vitek® vancomycin MIC = 1 mg/L, teicoplanin MIC = ≤ 0.5 mg/L) harboured *vanA*. A further 5 phenotypically vancomycin susceptible *E. faecalis* isolates (Vitek® vancomycin MIC = 1) harboured *vanB* genes.

VanA/B PCR was performed on 370 of the 380 *E. faecium* isolates, including 171 of the 175 vancomycin non-susceptible isolates and 199 of the 205 vancomycin susceptible isolates. Overall, 189 (51.1%) of the 370 isolates harboured a *vanA* or *vanB* gene.

Thirty-one of the vancomycin non-susceptible *E. faecium* isolates harboured *vanA* (Vitek® vancomycin MIC = 8 mg/L [1 isolate] and > 16 mg/L [30 isolates]). A further 140 *E. faecium* vancomycin non-susceptible isolates harboured *vanB* (Vitek® vancomycin MIC = 8 mg/L [2 isolates] and > 16 mg/L [135 isolates]).

VanA or *vanB* genes were detected in 18 vancomycin susceptible *E. faecium* isolates. Four isolates harboured *vanA* (Vitek® vancomycin MIC ≤ 0.5 mg/L [2 isolates], MIC = 1 mg/L [1 isolate] and MIC = 2 mg/L [1 isolate], teicoplanin ≤ 1 mg/L [4 isolates]). Fourteen isolates harboured *vanB* (Vitek® vancomycin MIC ≤ 0.5 mg/L [7 isolates], MIC = 1 mg/L [6 isolates] and MIC = 2 mg/L [1 isolate]).

Of the 154 *vanB E. faecium* isolates, 3 were teicoplanin resistant (MIC > 32 mg/L).

Enterococcus faecium molecular epidemiology

Of the 380 episodes, 369 *E. faecium* isolates were available for typing. By PFGE, 367 isolates were classified into 113 pulsotypes, including 14 major pulsotypes with 5 or more isolates (Table 3). Two isolates were not typable by PFGE. Of the 99 pulsotypes with less than 5 isolates, 90 had only 1 isolate. Overall 253 (68.9%) of the 367 isolates were grouped into the 14 major pulsotypes from which 9 multilocus sequence types (STs) were identified. Using eBURST, the 9 STs were grouped into CC 17.

Geographical distribution of the 9 STs varied (Table 3). For the 4 most prominent STs, ST203 (69 isolates) was identified across most of Australia, ST796 (65 isolates) primarily in Victoria, ST555 (45 isolates) primarily in South Australia and Western Australia and ST17 (34 isolates) primarily in New South Wales. For the remaining 5 STs, ST117 (16 isolates) was found in New South Wales, Queensland and the Australian Capital Territory, ST 761 (7 isolates) in New South Wales and Queensland, ST192 (6 isolates) in Victoria and Tasmania, ST80 (7 isolates) in New South Wales and Western Australia and ST341 (5 isolates) in New South Wales and the Australian Capital Territory.

VanA was detected in two major pulsotypes (29 isolates, Efm18 and Efm85), and *vanB* in 8 major pulsotypes (137 isolates, Efm1, Efm2, Efm3, Efm18, Efm74, Efm75, Efm76, Efm77) (Table 4). Efm18 (ST17) harboured *vanA* and *vanB* genes. Twelve minor pulsotypes (14 isolates) also harboured *vanB* genes. In addition, *vanA* genes were detected in 6 minor pulsotypes (6 isolates).

Discussion

Enterococci are intrinsically resistant to a broad range of antimicrobials including the cephalosporins and sulphonamides. Due to their ability to acquire additional resistance through the transfer of plasmids and transposons and to disseminate easily in the hospital environment, enterococci have become difficult to treat and provide major infection control challenges.

As the AGAR programs are similar to those conducted in Europe[14] comparison of Australia antimicrobial resistance data with other countries is possible.

In the 2013 European Centre for Disease Prevention and Control and Prevention (ECDC) Enterococci surveillance program the European Union/European Economic Area (EU/EEA) population-weighted mean percentage of *E. faecium* resistant to vancomycin was 8.9% (95% CI, 7–12), ranging from 0.0% (95% CI, 0–9) in Estonia, Lithuania, Malta and Sweden to 42.7% (95% CI, 38–48) in Ireland. Cyprus (23.3%), United Kingdom (23.3%), Portugal (22.0%) and Greece (21.2%) were the only other EU/EEA countries to report above 20%.[15]

Table 3: The number and proportion of major Enterococcus faecium (Efm) pulsed-field gel electrophoresis pulsotypes, Australia, 2014, by region

		ACT		NSW		NT		Qld		SA		Tas.		Vic.		WA		Aus.	
Type	ST	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Efm1	ST203	0		6	4.5	0		0		0		0		2	2.1	2	4.0	10	2.6
Efm2		1	9.1	3	2.2	0		14	37.8	16	34.8	0		2	2.1	0		36	9.5
Efm75		2	18.2	5	3.7	0		1	2.7	0		0		1	1.1	0		9	2.4
Efm76		1	9.1	13	9.7	0		0		0		0		0		0		14	3.7
Efm74	ST796	0		0		0		0		2	4.3	1	14.3	62	66.0	0		65	17.1
Efm4	ST555	0		0		0		0		6	13.0	0		1	1.1	21	42.0	28	7.4
Efm77		0		0		0		1	2.7	9	19.6	0		0		7	14.0	17	4.5
Efm5	ST17	0		4	3.0	0		3	8.1	0		0		1	1.1	0		8	2.1
Efm18		0		26	19.4	0		0		0		0		0		0		26	6.8
Efm80	ST117	4	36.4	11	8.2	0		1	2.7	0		0		0		0		16	4.2
Efm78	ST761	0		6	4.5	0		1	2.7	0		0		0		0		7	1.8
Efm24	ST192	0		0		0		0		0		2	28.6	4	4.3	0		6	1.6
Efm85	ST80	0		5	3.7	0		0		0		0		0		1	2.0	6	1.6
Efm3	ST341	1	9.1	4	3.0	0		0		0		0		0		0		5	1.3
Other		2	18.2	43	32.1	1	100	15	40.5	12	26.1	2	28.6	20	21.3	19	38.0	114	30.0
NT		0		0		0		1	2.7	0		1	14.3	0		0		2	0.5
ND		0		7	5.2	0		0		1	2.2	1	14.3	1	1.1	0		11	2.9
Total		11	100	134	100	1	100	37	100	46	100	7	100	94	100	50	100	380	100

NT Non-typeable.

ND Not done.

Table 4: The number and proportion of major Enterococcus faecium (Efm) pulsed-field gel electrophoresis pulsotypes harbouring *vanA* or *vanB* genes, Australia, 2014

Pulsotypes	ST	n	vanA		vanB		Not detected	
			n	%	n	%	n	%
Efm1	ST203	10	0	0.0	3	30.0	7	70.0
Efm2		36	0	0.0	34	94.4	2	5.6
Efm75		9	0	0.0	3	33.3	6	66.7
Efm76		14	0	0.0	13	92.9	1	7.1
Efm74	ST796	65	0	0.0	65	100.0	0	0.0
Efm4	ST555	28	0	0.0	0	0.0	28	100.0
Efm77		17	0	0.0	16	94.1	1	5.9
Efm5	ST17	8	0	0.0	0	0.0	8	100.0
Efm18		26	24	92.3	1	3.8	1	3.8
Efm80	ST117	16	0	0.0	0	0.0	16	100.0
Efm78	ST761	7	0	0.0	0	0.0	7	100.0
Efm24	ST192	6	0	0.0	0	0.0	6	100.0
Efm85	ST80	6	5	83.3	0	0.0	1	16.7
Efm3	ST341	5	0	0.0	5	100.0	0	0.0
Total		253	29	11.5	140	53.3	84	33.2

In AESOP 2014 approximately 40% of enterococcal bacteraemia were due to *E. faecium,* of which 46.1% (95% CI, 41.0–51.2) were phenotypically vancomycin non-susceptible by Vitek2® or Phoenix™. However, 51.1% of *E. faecium* isolates tested (189/370) harboured *vanA/vanB* genes, of which 81.5% were *vanB*. Overall, 9.5% (35/370) of *E. faecium* isolates harboured a *vanA* gene, which is a significant increase from the 2.6% (8/310) of isolates reported in AESOP 2013 ($P = 0.0005$).[16] The majority of *E. faecium* isolates were also non-susceptible to multiple antimicrobials, including ampicillin, erythromycin, tetracycline, ciprofloxacin and high level gentamicin. In AESOP 2011[17] and 2013,16 37.0% and 48.6% of *E. faecium* harboured *vanA/vanB* respectively confirming the incidence of vancomycin resistant *E. faecium* bacteraemia in Australia is increasing.

Fourteen (9.1%) of the 154 *vanB E. faecium* isolates had a vancomycin MIC at or below the CLSI and the EUCAST susceptible breakpoint (≤ 4 mg/L) and would not have been identified using routine phenotypic antimicrobial susceptibility methods. Furthermore, 6 *vanA/B E. faecalis* were also phenotypically vancomycin susceptible (MIC 1 mg/L).

By PFGE, *E. faecium* was shown to be very polyclonal, consistent with the known plasticity of the enterococcal genome. The 14 major *E. faecium* pulsotypes formed part of CC17, a global hospital-derived lineage that has successfully adapted to hospital environments. CC17 is characteristically ampicillin and quinolone resistant and subsequent acquisition of *vanA*– or *vanB*-containing transposons by horizontal transfer in CC17 clones has resulted in vancomycin resistant enterococci with pandemic potential. In AESOP 2014, 4 *E. faecium* STs predominated: ST203 (of which 77% of isolates harboured *vanB* genes); ST796 (100% harboured *vanB*); ST555 (37% harboured *vanB*); and ST17 (71% harboured *vanA* and 3% harboured *vanB*). Two minor PFGE pulsotypes identified in AESOP 2013 have become major pulsotypes in AESOP 2014: Efm80–ST117 (16 isolates) found in New South Wales (11 isolates), the Australian Capital Territory (4 isolates) and Queensland (1 isolate) and Efm85–ST80 (6 isolates), in New South Wales (5 isolates) and Western Australia (1 isolate). The majority of Efm85–ST80 isolates harboured *vanA* genes.

Conclusions

The AESOP 2014 study has shown that although predominately caused by *E. faecalis,* enterococcal bacteraemia in Australia is frequently caused by ampicillin-resistant high-level gentamicin-resistant *vanB E. faecium*. Furthermore, the percentage of *E. faecium* bacteraemia isolates resistant to vancomycin in Australia is significantly higher than that seen in almost all European countries. Although the *vanB* operon continues to be the predominant genotype, the number of *vanA E. faecium* identified in AESOP 2014 has significantly increased when compared with AESOP 2013. In addition to

being a significant cause of healthcare-associated sepsis, the emergence of multiple multi-resistant hospital-adapted *E. faecium* strains has become a major infection control issue in Australian hospitals. Further studies on the enterococcal genome will contribute to our understanding of the rapid and ongoing evolution of enterococci in the hospital environment and assist in preventing their nosocomial transmission.

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Australian Group on Antimicrobial Resistance Australian *Staphylococcus aureus* Sepsis Outcome Programme annual report, 2014

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Abstract

From 1 January to 31 December 2014, 27 institutions around Australia participated in the Australian Staphylococcal Sepsis Outcome Programme (ASSOP). The aim of ASSOP 2014 was to determine the proportion of *Staphylococcus aureus* bacteraemia (SAB) isolates in Australia that are antimicrobial resistant, with particular emphasis on susceptibility to methicillin and to characterise the molecular epidemiology of the isolates. Overall, 18.8% of the 2,206 SAB episodes were methicillin resistant, which was significantly higher than that reported in most European countries. The 30-day all-cause mortality associated with methicillin-resistant SAB was 23.4%, which was significantly higher than the 14.4% mortality associated with methicillin-sensitive SAB ($P<0.0001$). With the exception of the ß-lactams and erythromycin, antimicrobial resistance in methicillin-sensitive *S. aureus* remains rare. However in addition to the ß-lactams, approximately 50% of methicillin-resistant *S. aureus* (MRSA) were resistant to erythromycin and ciprofloxacin and approximately 15% were resistant to co-trimoxazole, tetracycline and gentamicin. When applying the European Committee on Antimicrobial Susceptibility Testing breakpoints, teicoplanin resistance was detected in 2 *S. aureus* isolates. Resistance was not detected for vancomycin or linezolid. Resistance to non-beta-lactam antimicrobials was largely attributable to 2 healthcare-associated MRSA clones; ST22-IV [2B] (EMRSA-15) and ST239-III [3A] (Aus-2/3 EMRSA). ST22-IV [2B] (EMRSA-15) has become the predominant healthcare associated clone in Australia. Sixty per cent of methicillin-resistant SAB were due to community-associated (CA) clones. Although polyclonal, almost 44% of community-associated clones were characterised as ST93-IV [2B] (Queensland CA-MRSA) and ST1-IV [2B] (WA1). CA-MRSA, in particular the ST45-V [5C2&5] (WA84) clone, has acquired multiple antimicrobial resistance determinants including ciprofloxacin, erythromycin, clindamycin, gentamicin and tetracycline. As CA-MRSA is well established in the Australian community it is important that antimicrobial resistance patterns in community and healthcare-associated SAB is monitored as this information will guide therapeutic practices in treating *S. aureus* sepsis. *Commun Dis Intell* 2016;40(2):E244–E254.

Keywords: Australian Group on Antimicrobial Resistance, antimicrobial resistance surveillance; *Staphylococcus aureus*, methicillin sensitive, methicillin resistant, bacteraemia

Introduction

Globally, *Staphylococcus aureus* is one of the most frequent causes of hospital-acquired and community-acquired blood stream infections.[1] Although there are a wide variety of manifestations of serious invasive infection caused by *S. aureus,* in the great majority of these cases the organism can be detected in blood cultures. Therefore, SAB is considered a very useful marker for serious invasive infection.[2]

Although prolonged antimicrobial therapy and prompt source control are used to treat SAB,[3] mortality ranges from as low as 2.5% to as high as 40%.[4–6] Mortality rates however, are known to vary significantly with patient age, clinical manifestation, co-morbidities and methicillin resistance.[7,8] A prospective study of SAB conducted in 27 laboratories in Australia and New Zealand found a 30-day all-cause mortality of 20.6%.[9] On univariate analysis, increased mortality was significantly associated with older age, European ethnicity, methicillin resistance, infections not originating from a medical device, sepsis syndrome, pneumonia/empyema and treatment with a glycopeptide or other non-ß-lactam antibiotic.

The Australian Group on Antimicrobial Resistance (AGAR), a network of laboratories located across Australia, commenced surveillance of antimicrobial resistance in *S. aureus* in 1986.[10] In 2013 AGAR commenced the Australian Staphylococcal Sepsis Outcome Programme (ASSOP).[11] The primary objective of ASSOP 2014 was to determine the proportion of SAB isolates demonstrating antimicrobial resistance with particular emphasis on:

1. assessing susceptibility to methicillin
2. molecular epidemiology of methicillin resistant *S. aureus* (MRSA).

Methods

Twenty-seven laboratories from all 8 Australian states and territories participated in the program in 2014.

From 1 January to 31 December 2014, the 27 laboratories collected all *S. aureus* isolated from blood cultures. *S. aureus* with the same antimicrobial susceptibility profiles isolated from a patient's blood culture within 14 days of the first positive culture were excluded. A new *S. aureus* sepsis episode in the same patient was recorded if it was identified by a culture of blood collected more than 14 days after the last positive culture. Data were collected on age, sex, date of admission and discharge (if admitted), and mortality at 30 days from date of first positive blood culture. To avoid interpretive bias, no attempt was made to assign attributable mortality. Each episode of bacteraemia was designated healthcare onset if the first positive blood culture(s) in an episode were collected > 48 hours after admission.

Laboratory testing

Participating laboratories performed antimicrobial susceptibility testing using the Vitek2® (bioMérieux, France) or the Phoenix™ (BD, USA) automated microbiology systems according to the manufacturer's instructions. *S. aureus* was identified by morphology and positive results of at least one of the following tests: Vitek MS® (bioMérieux, France), matrix-assisted laser desorption ionization biotyper (Bruker Daltonics, Germany), slide coagulase, tube coagulase, appropriate growth on chromogenic agar and demonstration of deoxyribonuclease production. Additional tests such as fermentation of mannitol, growth on mannitol-salt agar or polymerase chain reaction (PCR) for the presence of the *nuc* gene may have been performed for confirmation.

Minimum inhibitory concentration (MIC) data and isolates were referred to the Australian Collaborating Centre for *Enterococcus* and *Staphylococcus* Species (*ACCESS*) Typing and Research. Clinical and Laboratory Standards Institute (CLSI)[12] and European Committee on Antimicrobial Susceptibility Testing (EUCAST)[13] breakpoints were utilised for interpretation. Isolates with a resistant or an intermediate category were classified as non-susceptible. Linezolid and daptomycin non-susceptible isolates were retested by Etest® (bioMérieux) using the Mueller-Hinton agar recommended by the manufacturer. *S. aureus* ATCC 29213 was used as the control strain. High level mupirocin resistance was determined using a mupirocin 200 μg disk according to CLSI guidelines on all isolates with a mupirocin MIC > 8 mg/L by Vitek2® or > 256 mg/L by Phoenix™.[12] Multi-resistance was defined as resistance to 3 or more of the following non-ß-lactam antimicrobials: vancomycin, teicoplanin, erythromycin/clindamycin, tetracycline, ciprofloxacin, gentamicin, co-trimoxazole, fusidic acid, rifampicin, high level mupirocin, or linezolid.

Electrophoresis of chromosomal DNA was performed as previously described on all MRSA using contour-clamped homogeneous electric field DR III system (Bio-Rad Laboratories Pty Ltd, USA).[14] Chromosomal patterns were examined visually, scanned with a Quantity One software (Bio-Rad Laboratories Pty Ltd, USA), and digitally analysed using FPQuest (Applied Maths NV, Belgium). Multilocus sequence typing (MLST) was performed on all unique pulsed-field types as previously described.[15] The sequences were submitted to the Multi Locus Sequence Typing on-line database (http://www.mlst.net) where an allelic profile was generated and an ST assigned.

SCC*mec* typing was performed on all MRSA with a unique pulsed-field pattern using the Clondiag *S. aureus* Genotyping Array Hybridisation Kit (Alere, USA) as previously described.[16]

Detection of Panton-Valentine leucocidin (PVL) determinants and *mecA* was performed by PCR on all MRSA as previously described.[17,18]

Chi-square tests for comparison of 2 proportions and calculation of 95% confidence intervals (95%CI) were performed using MedCalc for Windows, version 12.7 (Medcalc Software, Ostend Belgium).

Approval to conduct the prospective data collection was given by the research ethics committee associated with each participating laboratory.

Results

From 1 January to 31 December 2014, 2,206 unique episodes of *S. aureus* bacteraemia were identified. A significant difference ($P < 0.0001$) was seen in patient sex with 63.2% (1,395) being male (95% CI 60.6–65.7). The average age of patients was 59 years ranging from 0 to 101 years with a median age of 62 years. Overall, 73.2% (1,615) of the 2,206 episodes were community onset (95% CI 71.0%–75.3%). All-cause mortality at 30-days was 16.1% (95% CI 14.5–17.8). Methicillin-resistant

SAB mortality was 23.4% (95% CI 19.1 to 28.1), which was significantly higher than methicillin-susceptible SAB mortality (14.4%, 95% CI 12.7 to 16.3, $P < 0.0001$).

Methicillin-sensitive *Staphylococcus aureus* antimicrobial susceptibility

Overall, 81.2% (1,792) of the 2,206 isolates were methicillin sensitive of which 77.0% (1,380) were penicillin resistant (MIC > 0.12 mg/L). However as ß-lactamase was detected in 87 phenotypically penicillin susceptible isolates, 81.9% of Methicillin-sensitive *Staphylococcus aureus* (MSSA) were considered penicillin resistant. Apart from erythromycin non-susceptibility, resistance to the non-ß-lactam antimicrobials among MSSA was rare, ranging from < 0.1% to 4.1% (Table 1). Four isolates were reported as non-susceptible to daptomycin by Vitek2®. By Etest® all isolates had MICs ≤ 1 mg/L and were therefore considered susceptible. Two isolates were reported as linezolid resistant (MIC > 8 mg/L) by Vitek2®. However by Etest® both isolates had a MIC ≤ 4 mg/L (1.0 and 2.0 mg/L) and were therefore considered linezolid susceptible. When using the EUCAST resistant breakpoint of > 2 mg/L 1 isolate was teicoplanin resistant (MIC = 4 mg/L). However using the CLSI resistant breakpoint of > 8 mg/L the isolate was classified susceptible. All MSSA were vancomycin susceptible. Twenty-eight (1.6%) of the 1,792 isolates had high level mupirocin resistance, of which 19 isolates were referred from Queensland. Inducible resistance to clindamycin was determined by the Vitek2® susceptibility system. Of the 1,622 isolates tested, 9.1% (147) were erythromycin non-susceptible/clindamycin intermediate/susceptible (CLSI and EUCAST breakpoints) of which 75.5% (111) were classified as having inducible clindamycin resistance. Multi-resistance was uncommon in MSSA (1.7%, 30/1,792).

There were no significant differences in interpretation for any drug when CLSI or EUCAST non-susceptibility breakpoints were utilised ($P > 0.05$).

Table 1: The number and proportion of methicillin sensitive Staphylococcus aureus isolates non-susceptible to penicillin and the non-β-lactam antimicrobials, Australia, 2014

Antimicrobial	Tested	Breakpoint (mg/L)	Non-susceptible	
			n	%
Penicillin	1,792	>0.12*	1,467	81.8
Vancomycin	1,792	>2*	0	0.0
Teicoplanin	1,792	>8†	0	0.0
		>2‡	1	0.1
Rifampicin	1,741	>1†	4	0.2
Fusidic acid	1,791	>1‡	74	4.1
Gentamicin	1,792	>4†	14	0.8
		>1‡	18	1.0
Erythromycin	1,790	>2†	177	9.9
		>1‡	181	10.1
Clindamycin	1,790	>0.5†	30	1.7
		>0.25	31	1.7
Tetracycline	1,790	>4†	55	3.1
		>1‡	61	3.4
Co-trimoxazole	1,791	>2/38*	40	2.2
Ciprofloxacin	1,782	>1*	54	3.0
Nitrofurantoin	1,702	>32†	20	1.2
Daptomycin	1,791	>1*	0	0.0
Linezolid	1,792	>4*	0	0.0

* Clinical and Laboratory Standards Institute (CLSI) and European Committee on Antimicrobial Susceptibility Testing (EUCAST) non-susceptible breakpoint.

† CLSI non-susceptible breakpoint.

‡ EUCAST non-susceptible breakpoint.

Methicillin-resistant *Staphylococcus aureus* antimicrobial susceptibility

The proportion of *S. aureus* that were MRSA was 18.8% (95%CI 17.2–20.5). The 414 MRSA identified were either cefoxitin screen positive by Vitek2® (401) or had a cefoxitin MIC > 8 by Phoenix™ (13). All 414 MRSA isolates were phenotypically penicillin resistant. Among the MRSA isolates, non-susceptibility to non-ß-lactam antimicrobials was common except for rifampicin, fusidic acid and nitrofurantion where resistance was below 4.1% (Table 2). There were 6 isolates reported by Vitek2® as non-susceptible to daptomycin. By Etest® 3 isolates had MICs ≤ 1 mg/L and were therefore considered susceptible. Three isolates had MICs > 1 mg/L (1.5, 3 and 4 mg/L) and were considered non-susceptible. By Vitek2®, 2 isolates were linezolid resistant (MIC > 8 mg/L). However by Etest® both isolates had an MIC ≤ 4 mg/L (1 and 1.5 mg/L) and were therefore considered linezolid susceptible. When using the EUCAST resistant breakpoint of > 2 mg/L, 1 isolate was teicoplanin resistant (MIC = 4 mg/L). However, using the CLSI resistant breakpoint of > 8 mg/L the isolate was classified susceptible. All MRSA were vancomycin susceptible. Eight (1.9%) of the 414 MRSA isolates had high level mupirocin resistance of which five isolates were referred from Queensland. Inducible resistance to clindamycin was determined by the Vitek2® susceptibility system. Of the 352 isolates tested by Vitek2®, 31.2% (110) were erythromycin non-susceptible/clindamycin intermediate/susceptible (CLSI and EUCAST breakpoints) of which 88.2% (97) were classified as having inducible clindamycin resistance. Multi-resistance was common in MRSA (24.4%, 101/414).

There were no significant differences in interpretation for any drug when CLSI or EUCAST non-susceptibility breakpoints were utilised ($P > 0.05$).

Methicillin-resistant *Staphylococcus aureus* molecular epidemiology

Of the 414 MRSA identified, 403 were referred to *ACCESS* Typing and Research for strain characterisation. Based on molecular typing, 40.4% (163) and 59.6% (240) of isolates were classified

Table 2: The number and proportion of methicillin-resistant Staphylococcus aureus isolates non-susceptible to penicillin and the non-β-lactam antimicrobials, Australia, 2014

Antimicrobial	Tested	Breakpoint (mg/L)	Non-susceptible (%) n	%
Penicillin	414	>0.12*	414	100.0
Vancomycin	414	>2*	0	0.0
Teicoplanin	414	>8†	0	0.0
		>2‡	1	0.2
Rifampicin	412	>1†	4	1.0
Fusidic acid	414	>1‡	17	4.1
Gentamicin	414	>4†	67	16.2
		>1‡	74	17.9
Erythromycin	414	>2†	204	49.3
		>1‡	204	49.3
Clindamycin	414	>0.5†	68	16.4
		>0.25‡	70	16.9
Tetracycline	414	>4†	65	15.7
		>1‡	81	19.6
Co-trimoxazole	413	>2/38*	61	14.8
Ciprofloxacin	414	>1*	212	51.2
Nitrofurantoin	407	>32†	13	3.2
Daptomycin	412	>1*	3	0.7
Linezolid	414	>4*	0	0.0

* Clinical and Laboratory Standards Institute (CLSI) and European Committee on Antimicrobial Susceptibility Testing (EUCAST) non-susceptible breakpoint.

† CLSI non-susceptible breakpoint.

‡ EUCAST non-susceptible breakpoint.

as healthcare-associated MRSA (HA-MRSA) and community-associated MRSA (CA-MRSA) clones respectively (Table 3).

Healthcare-associated methicillin-resistant *Staphylococcus aureus*

For the 163 HA-MRSA strains, 46.6% (76) were epidemiologically classified as hospital onset and 53.4% (87) were classified as community onset.

Table 3: Proportion of healthcare-associated and community-associated methicillin-resistant *Staphylococcus aureus*, Australia, 2014, by clone, healthcare and community onset, and Panton-Valentine leucocidin carriage

Strain	Total		Onset				PVL positive	
			Healthcare		Community			
	n	%*	n	%	n	%†	n	%
Healthcare-associated MRSA								
ST22-IV [2B] (EMRSA-15)	119	29.5	52	43.7	67	56.3	1	0.8
ST239-III [3A] (Aus-2/3)	43	10.7	23	53.5	20	46.5	0	0.0
ST5-II [2A] (USA100)	1	0.2	1	100.0	0	0.0	0	0.0
Total	163	40.4	76	46.6	87	53.4	1	0.6
Community-associated MRSA								
ST93-IV [2B] (Queensland)	60	14.9	13	21.7	47	78.3	56	93.3
ST1-IV [2B] (WA1)	45	11.2	11	24.4	34	75.6	4	8.9
ST45-V [5C2&5] (WA84)	30	7.4	12	40.0	18	60.0	0	0.0
ST30-IV [2B] (SWP)	20	5.0	4	20.0	16	80.0	18	90.0
ST5-IV [2B] (WA3)	20	5.0	6	30.0	14	70.0	0	0.0
ST78-IV [2B] (WA2)	11	2.7	5	45.5	6	54.5	1	9.1
ST188-IV [2B] (WA38)	5	1.2	2	40.0	3	60.0	0	0.0
ST1-V [5C2]	5	1.2	1	20.0	4	80.0	0	0.0
ST8-IV [2B] (USA300)	5	1.2	0	0.0	5	100.0	5	100.0
ST5-IV [2B] (WA71)	4	1.0	0	0.0	4	100.0	0	0.0
ST72-IV [2B] (Korean)	4	1.0	3	75.0	1	25.0	0	0.0
ST5-IV [2B] (WA121)	4	1.0	0	0.0	4	100.0	4	100.0
ST835-IV [2B] (WA48)	4	1.0	0	0.0	4	100.0	0	0.0
ST953-IV [2B] (WA54)	3	0.7	0	0.0	3	100.0	0	0.0
ST5-V [5C2] (WA81)	3	0.7	0	0.0	3	100.0	0	0.0
ST45-IV [2B] (WA75)	3	0.7	0	0.0	3	100.0	0	0.0
ST5-V [5C2] (WA123)	3	0.7	0	0.0	3	100.0	0	0.0
ST59-IV [2B] (WA15)	2	0.5	1	50.0	1	50.0	0	0.0
ST1420-IV [2B] (WA126)	2	0.5	0	0.0	2	100.0	1	50.0
ST6-IV [2B] (WA51)	1	0.2	1	100.0	0	0.0	0	0.0
ST5-IV [2B]	1	0.2	0	0.0	1	100.0	0	0.0
ST5-IV [2B] (WA105)	1	0.2	0	0.0	1	100.0	0	0.0
ST75-IV (2B] (WA8)	1	0.2	1	100.0	0	0.0	0	0.0
ST5-V [5C2] WA14	1	0.2	0	0.0	1	100.0	0	0.0
ST5-V [5C2] WA86	1	0.2	1	100.0	0	0.0	0	0.0
ST2947-V [5C2] (WA129)	1	0.2	0	0.0	1	100.0	1	100.0
Total	240	59.6	61	25.4	179	74.6	90	37.5
Grand total	403	100.0	137	34.0	266	66.0	91	22.6

PVL Panton-Valentine leucocidin.

* Percentage of all methicillin-resistant *Staphylococcus aureus* (MRSA).

† Percentage of the strain.

Three HA-MRSA clones were identified: 119 isolates of ST22-IV [2B] (EMRSA-15) (29.5% of MRSA and 5.4% of *S. aureus*); 43 isolates of ST239-III [3A] (Aus -2/3 EMRSA) (10.7% and 1.9%) and a single isolate of ST5-II [2A] (USA100/New York Japan MRSA).

ST22-IV [2B] (EMRSA-15) was the dominant HA-MRSA clone in Australia accounting for 73% of HA-MRSA ranging from 0% in the Northern Territory to 100% in Tasmania and Western Australia (Table 4). ST22-IV [2B] (EMRSA-15) was typically PVL negative and using CLSI breakpoints 98.3% and 61.3% were ciprofloxacin and erythromycin resistant respectively.

ST239-III [3A] (Aus-2/3 EMRSA) accounted for 26.4% of HA-MRSA ranging from 0% in Tasmania and Western Australia to 100% in the Northern Territory (Table 4). PVL negative ST239-III [3A] (Aus-2/3 EMRSA) were typically resistant to erythromycin (97.7%), co-trimoxazole (100%), ciprofloxacin (97.7%), gentamicin (97.7%), tetracycline (79%) and clindamycin (74.4%).

Community-associated methicillin-resistant *Staphylococcus aureus*

For the 240 CA-MRSA strains, 25.4% (61) of episodes were epidemiologically classified as hospital onset and 74.6% (179) classified as community onset. Twenty-six different CA-MRSA clones were identified by pulsed-field gel electrophoresis (PFGE) corresponding to 19 MLST/SCC*mec* clones (Table 3). Overall, 77.5% of CA-MRSA were classified into 6 clones each having more than 10 isolates: ST93-IV [2B] (Queensland CA-MRSA) (14.9% of MRSA and 2.7% of *S. aureus*); ST1-IV [2B] (WA1) (11.2% and 2%); ST45-V [5C2&5] (WA84) (7.4% and 1.4%); ST30-IV [2B] (South West Pacific [SWP] CA-MRSA) (5.0% and 0.9%); ST5-IV [2B] (WA3) (4.2% and 0.8%); and ST78-IV [2B] (WA2) (2.7% and 0.5%).

ST93-IV [2B] (Queensland CA-MRSA) accounted for 25% of CA-MRSA ranging from 0% in Tasmania and the Australian Capital Territory to 59.1% in the Northern Territory (Table 5). Typically PVL positive, 83.3% of ST93-IV [2B] (Queensland CA-MRSA) were resistant to the ß-lactams only (50/60) or additionally resistant to erythromycin (10%, 6/60), erythromycin and clindamycin (5%, 3/60), or erythromycin, clindamycin and ciprofloxacin (1.7%, 1/60).

ST1-IV [2B] (WA1) accounted for 18.8% of CA-MRSA ranging from 0% in Tasmania to 100% in the Australian Capital Territory (Table 5). Typically PVL negative, 62.2% of isolates were resistant to the ß-lactams only (28/45) or additionally resistant to erythromycin (8.9%, 4/45), erythromycin and fusidic acid (8.9%, 4/45), high level mupirocin (6.7%, 3/45), ciprofloxacin, erythromycin and fusidic acid (4.4%, 2/45), ciprofloxacin, erythromycin and gentamicin (2.2%, 1/45), clindamycin (2.2%, 1/45), erythromycin, fusidic acid and nitrofurantoin (2.2%, 1/45) or nitrofurantoin (2.2%, 1/45).

ST45-V [5C2&5] (WA84) accounted for 12.5% of CA-MRSA and was isolated primarily in the eastern regions of Australia (Table 5). All isolates were PVL negative and were resistant to the ß-lactams and ciprofloxacin. Isolates were additionally non-susceptible to erythromycin and tetracycline (20%, 6/30), erythromycin, gentamicin and tetracycline (16.7%, 5/30), erythromycin and gentamicin (13.3%, 4/30), erythromycin and clindamycin (10%, 3/30) and one (3.3%) each of erythromycin or erythromycin, clindamycin and tetracycline or erythromycin, clindamycin, gentamicin and tetracycline or clindamycin, erythromycin and nitrofurantoin or erythromycin, nitrofurantoin, gentamicin and tetracycline or erythromycin, fusidic acid, gentamicin and tetracycline.

ST30-IV [2B] (SWP CA-MRSA), accounted for 8.3% of CA-MRSA and was primarily isolated in the eastern regions of Australia (Table 5). Typically PVL positive, 70% of isolates were resistant to the ß-lactams only (14/20). Six isolates were non-susceptible to nitrofurantoin (30%).

ST5-IV [2B] (WA3) accounted for 8.3% of CA-MRSA and was primarily isolated in the eastern regions of Australia (Table 5). PVL negative ST5-IV [2B] (WA3) was typically resistant to the ß-lactams only (50%, 10/20) or additionally resistant to erythromycin (20%, 4/20), erythromycin and high level mupirocin (11.5%, 3/20), erythromycin and fusidic acid (5% 1/20), erythromycin and rifampicin (5% 1/20) or high level mupirocin (5%, 1/20).

ST78-IV [2B] (WA2), accounted for 4.6% of CA-MRSA and was isolated in most regions of the Australian mainland (Table 5). Isolates were resistant to the ß-lactams only (27%, 3/11) or additionally resistant to erythromycin (63.6%, 7/11). One isolate was resistant to tetracycline.

Overall, 85.8% of CA-MRSA were non-multiresistant and 50.4% were resistant to the ß-lactams only. However, 34 (14.2%) CA-MRSA isolates were multiresistant.

Table 4: The number and proportion of healthcare associated methicillin resistant *Staphylococcus aureus* multilocus sequence types, Australia, 2014, by state or territory

Type	ACT		NSW		NT		Qld		SA		Tas.		Vic.		WA		Aus.	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
ST22-IV [2B] (EMRSA-15)	6	66.7	52	76.5	0	0.0	21	70.0	12	80.0	1	100.0	19	70.4	8	100.0	119	73.0
ST239-III (3A] (Aus-2/3 EMRSA)	3	33.3	15	22.1	5	100.0	9	30.0	3	20.0	0	0.0	8	29.6	0	0.0	43	26.4
ST5-II [2A] (USA100)	0	0.0	1	1.5	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	1	0.6
Total	9	100.0	68	100.0	5	100.0	30	100.0	15	100.0	1	100.0	27	100.0	8	100.0	163	100.0

Table 5: The number and proportion of the major community associated methicillin resistant *Staphylococcus aureus* multilocus sequence types, Australia (>10 isolates), 2014, by state or territory

Type	ACT		NSW		NT		Qld		SA		Tas.		Vic.		WA		Aus.	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
ST93-IV [2B] (Qld)	0	0.0	7	12.3	13	59.1	20	30.3	6	27.3	0	0.0	6	17.6	8	21.6	60	25.0
ST1-IV [2B] (WA1)	1	100.0	7	12.3	4	18.2	10	15.2	5	22.7	0	0.0	6	17.6	12	32.4	45	18.8
ST45-V [5C2&5] (WA84)	0	0.0	16	28.1	1	4.5	0	0.0	4	18.2	0	0.0	8	23.5	1	2.7	30	12.5
ST30-IV [2B] (SWP)	0	0.0	3	5.3	0	0.0	14	21.2	0	0.0	0	0.0	2	5.9	1	2.7	20	8.3
ST5-IV [2B] (WA3)	0	0.0	9	15.8	0	0.0	8	12.1	0	0.0	0	0.0	2	5.9	1	2.7	20	8.3
ST78-IV [2B] (WA2)	0	0.0	2	3.5	0	0.0	2	3.2	2	9.1	0	0.0	2	5.9	3	8.1	11	4.6
Other	0	0.0	13	22.8	4	18.1	12	18.2	5	22.7	1	100.0	8	23.5	11	29.7	54	22.5
Total	1	100.0	57	100.0	22	100.0	66	100.0	22	100.0	1	100.0	34	100.0	37	100.0	240	100.0

Panton-Valentine leucocidin

Overall 91 (22.6%) MRSA were PVL positive, of which 98.9% were CA-MRSA (Table 3). PVL positive CA-MRSA clones included the international CA-MRSA clone ST8-IV [2B] USA300.

Discussion

The AGAR surveillance programs collect data on antimicrobial resistance, focussing on bloodstream infections caused by *S. aureus*, Enterococcus and Enterobacteriaceae. All data being collected in the AGAR programs are generated as part of routine patient care in Australia, with most being available through laboratory and hospital bed management information systems. Isolates are referred to a central laboratory where strain and antimicrobial resistance determinant characterisation is performed. As the programs are similar to those conducted in Europe comparison of Australia antimicrobial resistance data with other countries is possible.[19]

In the 2013 European Centre for Disease Prevention and Control and Prevention SAB surveillance program, the European Union/European Economic Area (EU/EEA) population-weighted mean percentage of *S. aureus* resistant to methicillin was 18.0% (95% CI 17–20), ranging from 0.0% (95% CI 0–5) in Iceland to 64.5% (95% CI 59–69) in Romania.[20] In ASSOP 2014, 18.8% (95% CI 17.2–20.5) of the 2,206 SAB episodes were methicillin resistant. This compares with 19.1% (95% CI 17.5–21.0) in ASSOP 2013. Two European countries reported a similar percentage to Australia: Bulgaria (19.2%, 95% CI 14–25), and Ireland (19.9%, 95% CI 18–2). However for 18 of the 30 European countries (primarily the northern Europe countries, Germany, France and the United Kingdom) the percentage of SAB isolates resistant to methicillin was less than that reported in ASSOP 2014. Similar to Europe, which has seen the EU/EEA population-weighted mean percentage decrease significantly from 23.2% in 2009 to 18.0% in 2013, the percentage of methicillin-resistant SAB in Australia has decreased from 23.8% (95% CI 21.4–26.4) in 2007 to 18.8% (95%CI 17.2–20.5) in 2014 ($P < 0.0001$).[21] The decrease in methicillin-resistant SAB is consistent with what has been reported elsewhere[22,23] and is believed to be attributed to the implementation of antimicrobial stewardship and a package of improved infection control procedures including hand hygiene, MRSA screening and decolonisation, patient isolation and infection prevention care bundles.[24–28] However, unlike Europe, Australia has a high prevalence of CA-MRSA and so further reduction in the proportion of SAB due to MRSA may prove problematic.

In ASSOP 2014, the all-cause mortality at 30-days was 16.1% (95% CI 14.5–17.8). In comparison, the 2008 Australian New Zealand Cooperative on Outcomes in Staphylococcal Sepsis reported a significantly higher figure of 20.6% (95% CI 18.8–22.5, $P < 0.0001$), and when adjusted for Australian institutions only was 25.9% (personal communication). MRSA-associated SAB mortality remains high (23.4%, 95% CI 19.1–28.1) and was significantly higher than MSSA-associated SAB mortality (14.4%, 95% CI 12.7–16.3, $P < 0.0001$). Although it has recently been shown that invasive MRSA infection may be more life-threatening partially because of the inferior efficacy of the standard treatment, vancomycin,[9] the emergence of hyper-virulent CA-MRSA clones such as ST93-IV [2B] (Queensland CA-MRSA), causing healthcare-associated SAB is of concern.[29]

With the exception of the ß-lactams and erythromycin, antimicrobial resistance in MSSA remains rare. However, in addition to the ß-lactams approximately 50% of MRSA were resistant to erythromycin and ciprofloxacin and approximately 15% resistant to co-trimoxazole, tetracycline and gentamicin. Resistance was largely attributable to 2 healthcare-associated MRSA clones, ST22-IV [2B] (EMRSA-15), which is typically ciprofloxacin and erythromycin resistant, and ST239-III [3A] (Aus-2/3 EMRSA), which is typically erythromycin, clindamycin, ciprofloxacin, co-trimoxazole, tetracycline and gentamicin resistant. From the early 1980s until recently, the multi-resistant ST239-III [3A] (Aus-2/3 EMRSA) was the dominant HA-MRSA clone in Australian hospitals. However, ST22-IV [2B] (EMRSA-15) has replaced it as the most prevalent HA-MRSA isolated from clinical specimens and this change has occurred throughout most of the country.[30] In the current survey, ST239-III [3A] (Aus-2/3 EMRSA) was the only HA-MRSA clone in the Northern Territory. In ASSOP 2014, approximately 30% of MRSA were characterised as ST22-IV [2B] (EMRSA-15), compared with 24% in ASSOP 2013. CA-MRSA, in particular the ST45-V [5C2&5] (WA84) clone, has acquired multiple antimicrobial resistance determinants including ciprofloxacin, erythromycin, clindamycin, gentamicin and tetracycline.

Resistance was not detected for vancomycin, linezolid or teicoplanin when CLSI interpretive criteria were applied. However two isolates were teicoplanin non-susceptible when EUCAST criteria were applied.

Approximately 25% of SAB caused by CA-MRSA were of healthcare onset. Although in several parts of the United States of Australia the CA-MRSA clone USA300 has replaced the HA-MRSA clone ST5-II [2A] (USA100) as a cause of healthcare-associated

MRSA infection,[31] transmission of CA-MRSA in Australian hospitals is thought to be rare.[32,33] Consequently it is likely that many of the health-care onset CA-MRSA SAB infections reported in ASSOP 2014 were caused by the patient's own colonising strains acquired prior to admission. In Australia CA-MRSA clones such as PVL-positive ST93-IV [2B] (Queensland CA-MRSA) and PVL-negative ST1-IV [2B] (WA1) are well established in the community and therefore it is important to monitor antimicrobial resistance patterns in both community– and healthcare-associated SAB as this information will guide therapeutic practices in treating *S. aureus* sepsis.

In conclusion, ASSOP 2014 has demonstrated antimicrobial resistance in SAB in Australia is a significant problem and continues to be associated with a high mortality. This may be due, in part, to the high prevalence of methicillin-resistant SAB in Australia, which is significantly higher than most EU/EEA countries. Consequently, MRSA must remain a public health priority and continuous surveillance of SAB and its outcomes and the implementation of comprehensive MRSA strategies targeting hospitals and long-term care facilities are essential.

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Australian trachoma surveillance annual report, 2013

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Abstract

Australia remains the only developed country to have endemic levels of trachoma (a prevalence of 5% or greater among children) in some regions. Endemic trachoma in Australia is found predominantly in remote and very remote Aboriginal communities. The Australian Government funds the National Trachoma Surveillance and Reporting Unit to collate, analyse and report trachoma prevalence data and document trachoma control strategies in Australia through an annual surveillance report. This report presents data collected in 2013. Data are collected from Aboriginal and Torres Strait Island communities designated at-risk for endemic trachoma within New South Wales, the Northern Territory, South Australia and Western Australia. The World Health Organization grading criteria were used to diagnose cases of trachoma in Aboriginal children, with jurisdictions focusing screening activities on the 5–9 years age group; but some children in the 1–4 and 10–14 years age groups were also screened. The prevalence of trachoma within a community was used to guide treatment strategies as a public health response. Aboriginal adults aged 40 years or over were screened for trichiasis. Screening coverage for the estimated population of children aged 5–9 years and adults aged 40 years or over in at-risk communities required to be screened in 2013 was 84% and 30%, respectively. There was a 4% prevalence of trachoma among children aged 5–9 years who were screened. Of communities screened, 50% were found to have no cases of active trachoma and 33% were found to have endemic levels of trachoma. Treatment was required in 75 at-risk communities screened. Treatment coverage for active cases and their contacts varied between jurisdictions from 79% to 100%. Trichiasis prevalence was 1% within the screened communities. *Commun Dis Intell* 2016;40(1):E255–E266.

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

Introduction

This is the 8th national trachoma surveillance annual report.[1–7] Trachoma screening and management data for 2013 were provided to the National Trachoma Surveillance and Reporting Unit (NTSRU) by the Northern Territory, South Australia, Western Australia and New South Wales.

Trachoma is one of the major causes of preventable blindness globally.[8] It is an eye infection caused by the bacteria *Chlamydia trachomatis* serotypes A, B, Ba and C. Infection with the relevant *C. trachomatis* serotype causes inflammation of the conjunctiva. Diagnosis of trachoma is by visual inspection, and the detection of follicles (white spots) and papillae (red spots) on the inner upper eyelid. Repeated infections with *C. trachomatis*, especially during childhood, may lead to scarring with contraction and distortion of the eyelid, which may in turn cause the eyelashes to rub against the cornea. This condition is known as trichiasis, which leads to gradual vision loss and blindness.[9–11] Scarring of the cornea due to trichiasis is irreversible. However, if early signs of in-turned eyelashes are found, then surgery is usually effective in preventing further damage to the cornea. The infection can be transmitted through close facial contact, hand-to-eye contact, via fomites (towels, clothing and bedding) or by flies. Trachoma generally occurs in dry, dusty environments and is linked to poor living conditions. Overcrowding of households, limited water supply for bathing and general hygiene, poor waste disposal systems and high numbers of flies have all been associated with trachoma. Children generally have the highest prevalence of trachoma and are believed to be the main reservoirs of infection because the infection in children has a longer duration than in adults.[12]

The Alliance for the Global Elimination of Blinding Trachoma by 2020 (GET 2020) initiative, supported by the World Health Organization (WHO), advocates the implementation of the SAFE strategy, with its key components of surgery (to correct trichiasis), antibiotic treatment, facial cleanliness and environmental improvements. This strategy is ideally implemented through a primary care model within a community framework, ensuring consistency and continuity in screening, control measures, data collection and reporting, as well as the building of community capacity. The target set by both WHO and the Communicable Diseases Network Australia (CDNA) for the elimination of blinding trachoma is a prevalence in children aged 1–9 years of less than 5% over a period of 5 years.[13–15]

Trachoma is usually treated by a single dose of the antibiotic azithromycin repeated on an annual basis, with treatment strategies varying according to trachoma prevalence. Best public health practice involves treatment of all members of the household in which a case resides, whether or not they have evidence of trachoma. In hyperendemic communities, it is recommended that treatment is also extended to all children over 3 kg in weight up to 14 years of age, or to all members of the community over 3 kg in weight.[9,12,16]

Trachoma control in Australia

Australia is the only high-income country where trachoma is endemic. It occurs primarily in remote and very remote Aboriginal communities in the Northern Territory, South Australia and Western Australia. In 2008, cases were also found in New South Wales and Queensland, States where trachoma was believed to have been eliminated. However, cases of trichiasis are believed to be present in all jurisdictions and sub-jurisdictional regions of Australia.[12,17] In 2013, the Australian Government committed $16.5 million to continue, improve and expand trachoma control initiatives in jurisdictions with areas of known endemic levels of trachoma and to jurisdictions with a previous history of trachoma screening activities to ascertain if control programs were also required. Funding was also committed to establishing a strong framework for monitoring and evaluation of trachoma control activities.[18]

The surveillance and management of trachoma in 2013 was guided by the CDNA guidelines for management of trachoma. The 2006 *Guidelines for the Public Health Management of Trachoma in Australia*.[19] were reviewed in 2013 and revised guidelines were formally implemented from January 2014.[9] One of the main changes to the guidelines was to include the option of not screening all endemic communities every year. The Northern Territory trachoma control program in 2013 was guided by the revised *National Guidelines for the Public Health Management of Trachoma in Australia*.[9] The guidelines were developed in the context of the WHO SAFE strategy and make recommendations for improving data collection, collation and reporting systems in relation to trachoma control in Australia.

The National Trachoma Surveillance and Reporting Unit

The NTSRU is responsible for data collation, analysis and reporting related to the ongoing evaluation of trachoma control strategies in Australia. It operates under contract with the Australian Government Department of Health.

The NTSRU has been managed by The Kirby Institute, the University of NSW since the end of 2010.[5–7] For previous reports from 2006 to 2008, the NTSRU was managed by The Centre for Eye Research Australia.[1–3] and the 2009 report was managed by the Centre for Molecular, Environmental, Genetic and Analytic Epidemiology, at the University of Melbourne.[4]

Methods

Each jurisdiction undertook screening and treatment for trachoma according to its respective protocols, and in the context of the national 2006 CDNA *Guidelines for the Public Health Management of Trachoma in Australia,* or the 2014 CDNA *National guidelines for the Public Health Management of Trachoma in Australia,* which recommend specific treatment strategies depending on the prevalence of trachoma detected through screening.[9,19]

In 2006, when the National Trachoma Management Program was initiated, each jurisdiction identified at-risk communities from historical prevalence data and other knowledge, including known transiency into endemic communities. Over time, additional communities have been reclassified as being at risk. Screening for trachoma focuses on the at-risk communities, but a small number of other communities designated as not-at-risk have also been screened, generally if there is anecdotal information suggesting the presence of active trachoma.

The WHO trachoma grading criteria were used to diagnose and classify individual cases of trachoma in all jurisdictions.[20] Data collection forms for data collection at the community level were developed by the NTSRU, based on the CDNA guidelines. Completed forms were forwarded from the jurisdictional coordinators to the NTSRU for checking and analysis. Information provided to the NTSRU at the community level for each calendar year included:

- number of Aboriginal children aged 1–14 years screened for clean faces and the number with clean faces, by age group;
- number of Aboriginal children aged 1–14 years screened for trachoma and the number with trachoma, by age group;
- number of episodes of treatment for active trachoma, household contacts and other community members, by age group;
- number of Aboriginal adults screened for trichiasis, number with trichiasis, and the number who had surgery for trichiasis;
- community-level implementation of WHO SAFE strategies.

While data may be collected for Aboriginal children aged 0–14 years, the focus age group in all regions is the 5–9 years age group.

Community-wide treatment differs between regions. In the Northern Territory, whole-of-community treatment according to the 2014 guidelines indicates the treatment of all people in the community over 3 kg in weight who are living in houses where there are any children less than 15 years of age. In Western Australia and South Australia whole-of-community treatment using the 2006 guidelines refers to active cases, household contacts and all children in the community aged 6 months to 14 years.

Northern Territory

In 2013, the Northern Territory delivered trachoma control activities using the draft revised guidelines, which allowed resources to be directed towards community-wide treatment in high-prevalence communities, and ensured that resources were not consumed by annual screening in areas where the prevalence was already well established. Trachoma screening and management in the Northern Territory was undertaken through collaboration between the Northern Territory Department of Health, Centre for Disease Control (CDC) and Health Development; and the Aboriginal Community Controlled Health Services (ACCHS). Trachoma screening was incorporated into the Healthy School-Age Kids program annual check and conducted by either local primary health-care services or community-controlled services, with support from the CDC trachoma team. The Northern Territory uses school enrolment lists, electronic health records and local knowledge to best determine children aged 5–9 years present in the community at the time of screening. Following screening, treatment was generally undertaken by primary health-care services with support from the CDC trachoma team, particularly where community-wide treatments were required.

In 2013, community screening for trichiasis was undertaken primarily by primary health clinic professionals in the community, ACCHS, or by optometrists or ophthalmologists from the Regional Eye Health Service based in Alice Springs.

South Australia

In 2013, Country Health South Australia was responsible for managing the South Australia trachoma screening and treatment program. Country Health South Australia contracted with ACCHS, the Aboriginal Health Council of South Australia, Nganampa Health Service and local health service providers to ensure coverage of screening services in all at-risk rural and remote areas. South Australia uses the Australian Bureau of Statistics census population estimates[21] as the screening denominator for screened communities. Additional trichiasis screening activities were undertaken by the Eye Health and Chronic Disease Specialist Support Program (EH&CDSSP), coordinated by the Aboriginal Health Council of South Australia and supported by the Medical Specialist Outreach Assistance Program. This program provides regular visits to South Australia remote Aboriginal communities by optometrists and ophthalmologists. Trichiasis screening was undertaken opportunistically for adults by contracted trachoma screening service providers, the EH&CDSSP team and also routinely as part of the Adult Annual Health Checks.

Western Australia

Trachoma screening and management in Western Australia is the responsibility of population health units in the Kimberley, Goldfields, Pilbara and Midwest health regions. In collaboration with the local primary health-care providers, the population health units screened communities in each region within a 2-week period, in August and September. People identified with active trachoma were treated at the time of screening. Each region determines the screening denominator in a different manner: in the Goldfields the denominator is based on the school register, without adjusting for absent children, plus other children present in the community at the time of screening; in the Pilbara the denominator number is based on children present in the community at the time of screening; in the Midwest the denominator is based on the school register with removal of children from the school list who were known to be absent on the day of the screening, plus any other children present in the community at the time of screening; and in the Kimberley the denominator is based on the school register, updated at the time of screening.

Trichiasis screening was undertaken in conjunction with the administration of adult influenza vaccinations. Screening of the target population also occurs with the Visiting Optometrist Scheme in the Kimberley region. In addition, the Goldfields region undertook additional trichiasis screening during the trachoma screening period.

In 2011, Western Australia Health amalgamated several previously distinct communities into one single community for the purpose of trachoma surveillance due to the small populations of each community and the kinship links that result in frequent mobility between these communities. This definition alters trends presented in reports from 2010 to 2013.

New South Wales

In 2013, New South Wales Health piloted a school-based trachoma screening project in 10 potentially at-risk communities in north-western New South Wales. The project aimed to determine if there was any evidence of trachoma in Aboriginal children living in rural and remote communities in New South Wales. Screening and treatment were conducted by the population health unit in Bathurst with support from the NSW Ministry of Health. No trichiasis screening was undertaken in New South Wales. In New South Wales, the denominator used to calculate screening coverage is based on the number of Aboriginal children aged 5–9 years enrolled in the school being screened. The denominator was not adjusted if children were absent on the day of screening.

Data analysis

For the purpose of this report, a community is defined as a specific location where people reside and where there is at least 1 school. At-risk communities are classified by the participating jurisdictions as being at higher risk of trachoma (generally based on prevalence above 5% among the 5–9 years age group). Communities are defined as being not-at-risk by having a baseline prevalence of below 5%; if previously at risk, 5 years with a prevalence below 5%; or having no historical evidence of trachoma prevalence. Community coverage is defined as the number of at-risk communities screened for trachoma as a proportion of those that were identified to possibly have trachoma. Individual screening coverage is the proportion of children in the respective target age groups in a region that was actually screened. Active trachoma is defined as the presence of chronic inflammation of the conjunctiva caused by infection with *C. trachomatis* and includes WHO grades trachomatous inflammation – follicular and trachomatous inflammation – intense.[15] Under the WHO criteria, Clean face is defined as the absence of dirt, dust and crusting on cheeks and forehead. The Clean face target is at least 80% of children within the community having a clean face at the time of screening.[17] Trachomatous trichiasis is defined as the evidence of the recent removal of in-turned eyelashes or at least 1 eyelash rubbing on the eyeball.

Trachoma data were analysed in the age groups 0–4, 5–9 and 10–14 years. Comparisons over time were limited to the 5–9 years age group, which is the target age group for the trachoma screening programs in all regions. Data from 2006 were excluded from assessment of time trends as collection methods in this first year differed from those subsequently adopted.

Projected data for trachoma prevalence

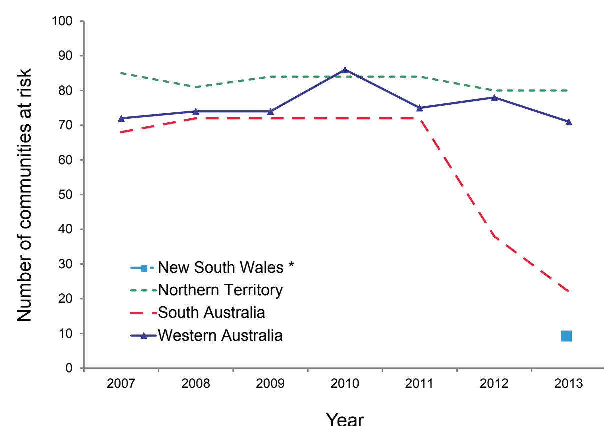
In 2013 the Northern Territory delivered trachoma control activities according to the newly revised guidelines.[9] Under these guidelines, a community would be excluded from screening activities for up to 3 years if high screening coverage had been achieved in the recent past and either prevalence of trachoma was less than 5%, or it was 5% or more without obvious clustering. Exclusion of these communities from screening activities leads to less reliable trachoma surveillance data during the interim period, for the total level, and trend in trachoma, in the region in which community is located. For reporting purposes, the NTSRU carried the most recent prevalence data forward in those communities that did not screen in the 2013 calendar year as a direct program decision, providing what is believed to be a conservative upper bound on average levels of trachoma. This principle will apply to all tables and figures relating to trachoma prevalence data. This method of projecting data was approved by the Trachoma Surveillance and Control Reference Group on 26 November 2013.

Results

Trachoma program coverage

Jurisdictions identified 183 communities as being at risk of trachoma in 2013, including those classified as potentially at-risk for the purposes of a mapping exercise in New South Wales (Table 1). The number of communities designated as being at-risk has plateaued in the Northern Territory, marginally decreased in Western Australia and substantially decreased in South Australia since 2012 (Figure 1). Of 183 at-risk or potentially at-risk communities, 145 communities were determined

Figure 1: Number of communities at risk, 2007 to 2013, selected states, by year and state or territory



* Screened in 2013 only.

to require screening for trachoma, a further 18 were identified to require treatment without screening, equating to 163 communities that were determined to require screening, treatment, or both screening and treatment. Of these, 144 (88%) received the screening, treatment or both screening and treatment that was required. The remaining 20 at-risk communities did not require screening or treatment as their previous year's prevalence was under 5%. A total of 15 communities, 2 each in the Northern Territory and Western Australia and 11 in South Australia, deemed not at-risk were also screened for trachoma in 2013 (Table 1).

Screening coverage

Jurisdictions identified 145 communities in the 4 jurisdictions requiring screening for trachoma in 2013 and of these 127 (88%) were screened for trachoma in 2013 (Table 1, Table 2). Within these communities 4,213 (84%) of an estimated 5,017 resident children aged 5–9 years were screened (Table 2). Screening coverage in children aged 5–9 years in at-risk communities was 81% for the Northern Territory, 90% for South Australia and Western Australia, and 72% for New South Wales (Table 2, Figure 2).

Table 1: Delivery of trachoma control in Australia, 2013, by state or territory

Community risk status and treatment delivered to communities	Number of communities					
	New South Wales	Northern Territory	South Australia	Western Australia	Total	Not at-risk
At risk* (A)	10	80	22	71	183	N/A
Requiring screening for trachoma (B)	10	42	22	71	145	N/A
Screened for trachoma (C)	10	30	16	71	127	15‡
Requiring treatment only (D)	N/A	18	N/A	N/A	18	N/A
Treated† (E)	N/A	17	N/A	N/A	17	N/A
Screened and/or treated for trachoma (F = C+E)	10	47	16	71	144	15
Requiring neither screening or treatment for trachoma (G=A-B-D)	N/A	20	N/A	N/A	20	N/A

* In 2013, New South Wales communities have been designated as 'potentially at risk' for the purposes of a mapping exercise.

† Communities treated without screening in 2013 as per revised guideline instructions.

‡ Not at risk communities were screened in the Northern Territory (2), Western Australia (2) and South Australia (11).

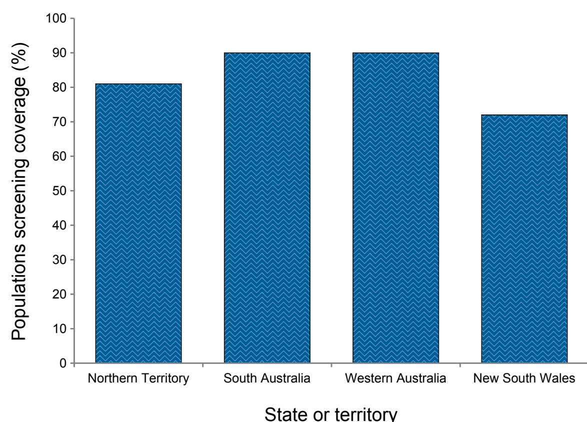
Table 2: Trachoma screening coverage, trachoma prevalence and clean face prevalence in Australia, 2013, by state or territory

	New South Wales	Northern Territory	South Australia	Western Australia	Total	Not at risk
Number of communities screened	10	30	16	71	127	15
Age group (years)	5–9	5–9	5–9	5–9	5–9	5–9
Children examined for clean face	608	1,358	768	1,510	4,244	266
Children with clean face	581	1,057	671	1,112	3,421	241
Clean face prevalence (%)	96	78	87	74	81	91
Estimated number* of Aboriginal children in communities†	795	1,681	857	1,684	5,017	380
Children screened for trachoma	575	1,362	768	1,508	4,213	265
Trachoma screening coverage (%)	72	81	90	90	84	70
Children with active trachoma	3	33	27	57	120	3
Active trachoma prevalence (%)	0.5	2.4	3.5	3.8	2.8	1.1
Active trachoma prevalence using projected data (%)	0.5	5	3.5	3.8	4.0	1.1

* Jurisdictional estimate.

† In communities that were screened for trachoma.

Figure 2: Population screening coverage in children aged 5–9 years in communities that were screened for trachoma, selected states, 2013, by state or territory



Clean face prevalence

A total of 4,244 children aged 5–9 years in 127 at-risk communities were assessed for clean faces during 2013. The overall prevalence of clean faces in children aged 5–9 years was 81%, with 78% in the Northern Territory, 87% in South Australia, 74% in Western Australia and 96% in New South Wales (Table 2).

Trachoma prevalence

The overall prevalence of active trachoma among children aged 5–9 years in screened communities (using projected data, see methodology) was 4%, with 5% in the Northern Territory, 3.5% in Western Australia, 3.8% in South Australia, and 0.5% in New South Wales. The observed trachoma prevalence in communities that were screened in 2013 in the Northern Territory was 2% (Figure 3, Table 2). Since 2009, the prevalence of trachoma in children aged 5–9 years has decreased significantly in all studied jurisdictions, with the projected national trachoma prevalence dropping from 14% in 2009 to 4% in 2013. From 2012 to 2013 the prevalence of trachoma in children aged 5–9 years has remained steady in Western Australia, and increased in the Northern Territory and South Australia (Figure 4). No trachoma was reported or detected in children aged 5–9 years in 91 (50%) communities in 2013, including communities that screened for trachoma in children 5–9 years of age and communities in the Northern Territory that did not screen in accordance with guidelines. Endemic levels of trachoma (> 5%) were reported in 55 communities (33%) in 2013, including communities that screened for trachoma in children aged 5–9 years and communities in the Northern Territory that did not screen in accordance with guidelines (Figure 5).

Figure 3: Trachoma prevalence in children aged 5–9 years in at-risk communities, selected jurisdictions, 2013

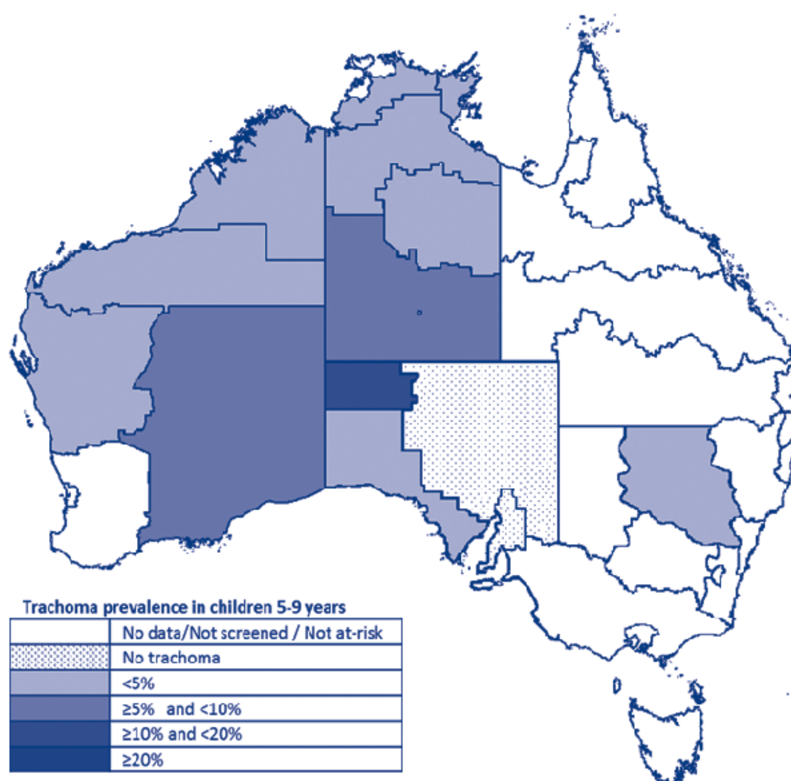
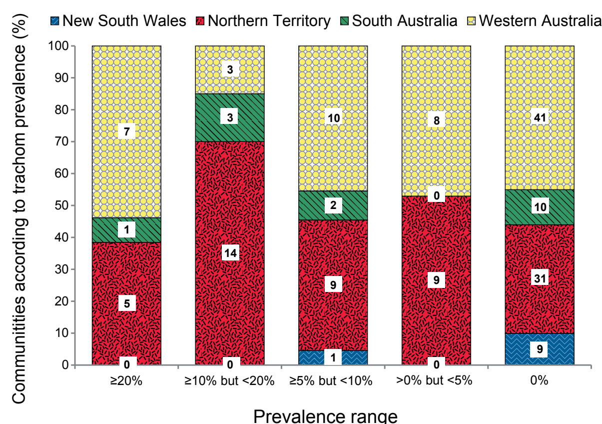


Figure 4: Trachoma prevalence among screened children* aged 5–9 years, 2007 to 2013, selected states, by year and state or territory

* Including communities in the Northern Territory that screened in 2013 and those that did not screen in 2013 in accordance with revised guideline instructions.

Figure 5: Number of screened at-risk communities* according to level of trachoma prevalence in children aged 5–9 years, selected states, 2013, by state or territory



* Including communities in the Northern Territory that screened in 2013 and those that did not screen in 2013 in accordance with revised guideline instructions.

Treatment delivery and coverage

Trachoma treatment strategies were applied in 74 communities, comprising 99% of those requiring treatment (Table 3). Treatment was delivered to active cases and households in 45 communities, and to the whole of community in 29 communities according to the guidelines. Three communities, all in the Northern Territory did not treat according to CDNA guidelines (Table 3). Of all cases detected that required treatment, 99% received treatment (Table 4). Treatment coverage in all jurisdictions was 81%, with 79% in the Northern Territory, 99% in South Australia, 94% in Western Australia and 100% in New South Wales (Table 4). A total of 10,219 doses of azithromycin was delivered (Table 4). An increasing trend of azithromycin distribution is observed in the Northern Territory since 2009, with Western Australia and South Australia trends relatively stable (Figure 6).

Figure 6: Number of doses of azithromycin administered for the treatment of trachoma, Australia, 2007 to 2013, by state or territory

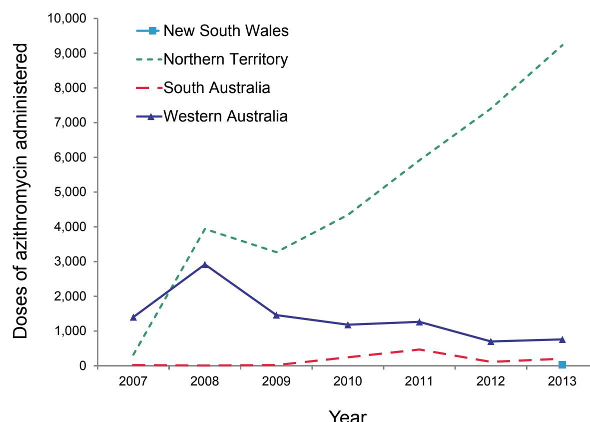


Table 3: Treatment strategies in Australia, 2013, by state or territory

Number of communities	New South Wales	Northern Territory	South Australia	Western Australia	Total
Required treatment for trachoma	1	34	6	34	75
Treated for trachoma	1	33	6	34	74
Screened and treated	1	16	6	34	57
Received treatment only	N/A	17	N/A	N/A	17
Received 6-monthly treatment	N/A	5	N/A	N/A	5
Did not require treatment	9	34	10	37	90
Treated active cases and households	1	12	5	27	45
Treated the whole of community*	0	21	1	7	29
Not treated according to CDNA guidelines	0	3	0	0	3

* In the Northern Territory, whole-of-community treatment was guided by the 2014 guidelines. This is defined as the treatment of all people in the community weighing more than 3 kg and living in households with children less than 15 years of age. In Western Australia and South Australia whole-of-community treatment was guided by the 2006 guidelines. This is defined as active cases, household contacts and all children in the community aged 6 months to 14 years.

Table 4: Trachoma treatment coverage in Australia, 2013

Age group (years)	Northern Territory					South Australia					Western Australia					New South Wales					Total				
	0–4	5–9	10–14	15+	All	0–4	5–9	10–14	15+	All	0–4	5–9	10–14	15+	All	0–4	5–9	10–14	15+	All	0–4	5–9	10–14	15+	All
Active cases requiring treatment	3	33	10		46	1	27	5		33	15	57	22		94	0	3	0		3	19	120	37		176
Active cases who received treatment	3	33	10		46	1	27	5		33	15	56	22		93		3			3	19	119	37		175
Active cases who received treatment (%)	100	100	100		100	100	100	100		100	100	98	100		99		100			100	100	99	100		99
Estimated contacts requiring treatment	1,184	1,497	1,306	7,597	1,1584	25	41	29	78	173	102	158	140	317	717	2	3	1	16	22	1,313	1,699	1,476	8,008	12,496
Number of contacts who received treatment	1,033	1,297	1,057	5,799	9,186	25	41	28	76	170	81	142	128	315	666	2	3	1	16	22	1,141	1,483	1,214	6,206	10,044
Estimated contacts who received treatment (%)	87	87	81	76	79	100	100	100	97	98	79	90	91	99	93	100	100	100	100	100	87	87	82	77	80
Total number of doses of azithromycin delivered	1,036	1,330	1,067	5,799	9,232	26	68	33	76	203	96	198	150	315	759	2	6	1	16	25	1,160	1,602	1,251	6,206	10,219
Estimated overall treatment coverage (%)	87	87	81	76	79	100	100	97	97	99	82	92	93	99	94	100	100	100	100	100	87	88	83	77	81
Doses administered in communities that were treated without screening*	423	498	399	2,264	3,584																423	498	399	2,264	3,584
Doses administered six-monthly*	552	659	574	3,114	4,899																552	659	574	3,114	4,899

* As per revised guidelines 2014.

Trichiasis

Screening for trichiasis was undertaken in 143 communities with 5,635 adults aged over 15 years screened in 2013. The prevalence of trichiasis in adults aged 15 years or over was 1% (55/5,635), and 1% (49/3,856) in adults aged 40 years or over. Surgery for trichiasis was reported to have been undertaken for 31 adults in 2013 (Table 5).

Health promotion activities

Health promotion activities were reported to have been undertaken in 128 communities, including at-risk and not at-risk communities. These included school and community based activities promoting facial cleanliness.

Discussion

Trachoma screening

The number of at-risk communities screened plateaued in the Northern Territory, decreased slightly in Western Australia and decreased substantially in South Australia. It is expected that this decreasing trend will continue in future years. A number of communities screened for the first time in 2013 did not have trachoma, and therefore do not qualify as being at risk for future years.

The revised *National Guidelines for the Public Health Management of Trachoma in Australia* direct communities to focus resources on treatment without annual screening where trachoma prevalence is already well established. Communities with non-endemic levels of trachoma will not be required to screen annually. These guidelines were implemented in the Northern Territory in 2013, and implemented nationwide in 2014. This strategy affected the number of communities screened in the Northern Territory and will have a similar effect in other jurisdictions in future years. Community and child population screening coverage have been used as an indication of the level of program delivery in previous annual trachoma reports. In response to the revised guidelines, the annual report has shifted focus from screening coverage to the extent of implementation of the guidelines with respect to screening, treatment and health promotion activities.

Trachoma prevalence

In past years, the NTSRU had been able to estimate the prevalence using population weights. Due to the poor screening coverage of the 0–4 years age group, it was considered that the results reported were not representative of that age group. In Australia, the prevalence in the 5–9 years age group is accepted as a sufficient measure of the prevalence of trachoma within at-risk communities.

Across all 4 jurisdictions in 2013, the prevalence of trachoma in children 5–9 years was 4%, a figure that includes data projected forward in communities that did not screen due to implementation of the revised guidelines. This rate is consistent with the 2012 national prevalence of 4% for trachoma in children aged 5–9 years. The observed trachoma prevalence in communities that were screened in 2013 was 3%.

Table 5: Trichiasis screening coverage, prevalence and treatment among Aboriginal adults in Australia, 2013

	Northern Territory		South Australia		Western Australia		Total		
Number of communities screened for trichiasis	56		23		64		143		
Age groups	15–39	40+	15–39	40+	15–39	40+	15–39	40+	15+
Estimated population in region*	14,087	7,146	3,385	2,121	5,898	3,450	23,370	12,717	36,087
Adults examined†	1,106	878	512	1,322	161	1,656	1,779	3,856	5,635
With trichiasis (% of adults examined)	6 (0.05%)	33 (4%)	0	8 (0.6%)	0	8 (0.5%)	6 (0.3%)	49 (1%)	55 (1%)
Offered ophthalmic consultation	0	13		8	0	7	0	28	28
Declined ophthalmic consultation	0	2		0	0	1	0	3	3
Surgery in past 12 months	0	23		2	0	6	0	31	31

* Population estimate limited to trachoma endemic regions and does not take into account changing endemic regions over time and transiency between regions.

† Number of adults examined limited to numbers reported. This number does not account for adults who may be examined in routine adult health checks, and may also include multiple screening.

New South Wales detected trachoma in 1 of the 10 communities screened. This community is now considered at-risk and will continue to be monitored. A further 9 communities were screened in 2013.

Trachoma prevalence in 2013 increased slightly in South Australia and the Northern Territory but plateaued in Western Australia, after a decreasing trend from 2009 to 2012 in all jurisdictions. The observed trends in South Australia and Western Australia were most likely due to the decrease in at-risk communities in South Australia and Western Australia. This trend may continue in future years due to implementation of the revised guidelines where communities not at risk cease undergoing screening and the at-risk population becomes more concentrated.

Trachoma treatment

Nationally, 99% of active cases that were identified in 2013 were treated for trachoma. Contact and community-treatment coverage was 81%. The total number of doses of azithromycin administered in 74 communities was 10,219. The majority of these were in the Northern Territory.

Facial cleanliness

Promoting facial cleanliness is a major component of the SAFE strategy, recognising that the presence of nasal and ocular discharge is significantly associated with the risk of both acquiring and transmitting trachoma. New South Wales had the highest prevalence of facial cleanliness at 96% of all children screened. The Northern Territory did not report levels of facial cleanliness in communities that did not screen for trachoma. However it is recommended that jurisdictions implementing the new guidelines continue to screen for facial cleanliness in communities where treatment and health promotion activities are undertaken.

Program delivery and monitoring

Improvements in program delivery have been reported in 2013 with increased coverage of screening and treatment delivery and health promotion activities in Western Australia. However, although treatment coverage in the Northern Territory and South Australia was high, these jurisdictions did not reach their community screening goals due to funding issues with service providers. Data quality also improved in all jurisdictions. However, as many regions chose to focus on the 5–9 years age group, data pertaining to the 0–4 and 10–14 years age groups were not comprehensive.

The newly endorsed CDNA guidelines provide the basis for strengthening trachoma control programs in all jurisdictions by reducing ambiguity in previous guidelines and providing clear guidance on screening and treatment methods. The impact of the new strategies, in particular treatment and screening schedules, may not be evident for several years.

Progress towards Australia's elimination target

The Australian government's commitment to the WHO Alliance of the Global Elimination of Blinding Trachoma by the year 2020 (GET 2020), to which Australia is a signatory, continues with further funding committed to ensuring that trachoma programs are increased and strengthened.

Discussions and plans are required for the next phase of monitoring communities no longer considered at-risk, and planning for the ongoing monitoring of trichiasis once blinding trachoma has been eliminated from Australia.

With the implementation of new guidelines in 2014 and strengthened efforts in health promotion, environmental condition improvements and treatment coverage, as reported in 2013, and decreasing numbers of at-risk communities leading to a more focused trachoma control program in endemic areas, Australia will stay on track to eliminate trachoma by 2020.

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Jurisdictional contributors to trachoma data collection

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Healthy School Age Kids Program: Top End and Central Australia

South Australia

Aboriginal Community Controlled Health Services

Aboriginal Health Council of South Australia

Country Health South Australia

Western Australia

Aboriginal Community Controlled Health Services

Communicable Diseases Control Directorate, Health Department of Western Australia

Goldfields Population Health Unit

Kimberley Population Health Unit

Midwest Population Health Unit

Pilbara Population Health Unit

New South Wales

Population Health unit, Western New South Wales Local Health District

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Invasive pneumococcal disease in Australia, 2011 and 2012

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Abstract

In Australia, there were 1,883 cases (8.3 per 100,000 population) of invasive pneumococcal disease (IPD) notified to the National Notifiable Diseases Surveillance System (NNDSS) in 2011 and 1,823 cases (8.0 per 100,000) in 2012. The overall rate of IPD in Indigenous Australians was 9 times the rate of IPD in non-Indigenous Australians in 2011 and 7 times in 2012. Following the July 2011 introduction of the 13-valent pneumococcal conjugate vaccine (13vPCV) to the National Immunisation Program, rates of IPD in children aged less than 5 years decreased from 19.5 per 100,000 in 2011 to 12.6 per 100,000 in 2012. In Indigenous adults aged 50 years or over the rates of IPD caused by serotypes included in the 23-valent pneumococcal polysaccharide vaccine (23vPPV) continued to increase in both 2011 (47.2 per 100,000) and 2012 (51.2 per 100,000). The rates of IPD in non-Indigenous adults aged 65 years or over caused by serotypes included in the 23vPPV also increased in 2011 (10.1 per 100,000) and 2012 (11.2 per 100,000). There were 134 deaths attributable to IPD in 2011 and 126 in 2012, although it should be noted that deaths may be under-reported. The number of invasive pneumococcal isolates with reduced penicillin susceptibility remained low and reduced susceptibility to ceftriaxone/cefotaxime continued to be rare. *Commun Dis Intell* 2016;40(2):E267–E284.

Keywords: Australia, invasive pneumococcal disease, communicable disease surveillance, epidemiology, annual report

Introduction

Pneumococcal disease, caused by *Streptococcus pneumoniae,* is a major cause of morbidity and mortality worldwide.[1] Pneumococcal disease is generally classed as either noninvasive or invasive pneumococcal disease (IPD). Noninvasive forms of the disease include otitis media, sinusitis and bronchitis. Noninvasive forms of the disease are not nationally notifiable and are not discussed in this report. IPD tends to be more severe and occur when the pathogen enters the blood stream or other sterile sites resulting in clinical manifestations such as pneumonia, bacteraemia, and meningitis.[1,2] This report describes the epidemiology of IPD in Australia for the years 2011 and 2012.

The burden of pneumococcal disease is greatest in infants and the elderly and it is these groups that are mostly targeted by the National Immunisation Program (NIP). In 1999, the 23-valent pneumococcal polysaccharide vaccine (23vPPV) was first funded by the NIP for Aboriginal and Torres Strait Islander adults aged greater than 50 years. NIP-funded 23vPPV has since been extended to include all adults aged 65 years or over and medically at-risk children. The 7-valent pneumococcal conjugate vaccine (7vPCV) was first registered for use in Australia in late 2000 and in mid-2001 it was funded by the NIP for Aboriginal and Torres Strait Islander infants and other at risk children. In January 2005, NIP-funded 7vPCV was extended to all infants nationally, together with a catch-up program for all children aged less than 2 years. In 2009, 10-valent pneumococcal conjugate vaccine (10vPCV) was funded by the NIP for children residing in the Northern Territory, replacing the 7vPCV and the 23vPPV in this group. In 2011, the 13-valent pneumococcal conjugate vaccine (13vPCV) replaced both the 7vPCV and the 10vPCV on the NIP and a supplementary dose of 13vPCV was made available to eligible infants who had completed a primary course of 7vPCV or 10vPCV.[3,4]

Annual and quarterly IPD surveillance reports are published regularly in *Communicable Diseases Intelligence*. In addition, a subset of IPD notification data, including serotype, age, sex Indigenous status, clinical categories and vaccination history are publicly available from the Australian National Notifiable Diseases Surveillance System (NNDSS) IPD Public Data Set.[5]

Methods

Data collection

IPD has been a nationally notifiable disease in Australia since 2001. To varying degrees across jurisdictions, medical practitioners, laboratories and other health professionals are required under state and territory public health legislation to report cases of laboratory confirmed IPD to state and territory health authorities. The *National Health Security Act 2007* provides the legislative basis for the national notification of communicable diseases and authorises the exchange of health information between the state and territory governments and

the Commonwealth. State and territory health departments transfer these notifications regularly to the NNDSS.

Core data, including serotype, sex, age, Indigenous status, pneumococcal vaccination history and outcome (alive or dead), are collected for all notifiable cases of IPD. In addition to the core data, enhanced surveillance data on notified cases of IPD are collected and these include information relating to risk factors, clinical category, antibiotic susceptibilities and, when relevant, date died. In 2011 and 2012, core data were available for all notified cases of IPD, whereas the availability of enhanced data varied across states and territories (Table 1). The data reported in this report on mortality include deaths within the first 1 to 2 weeks of diagnosis and that overall deaths may be under-reported.

The Enhanced Invasive Pneumococcal Disease Surveillance Working Group (EIPDSWG), a working group of the Communicable Diseases Network Australia, ensures routine and standardised reporting of trends and emerging issues relating to IPD.

The data presented in this report represent a point in time analysis of notified cases of IPD in Australia. Cases having a date of diagnosis from 1 January 2011 to 31 December 2012 inclusive were extracted from the NNDSS in July 2014 and analysed with a focus on the age groups targeted by the NIP. Date of diagnosis is a derived field within the NNDSS and represents the onset date of illness, or where the onset date was not known, the earliest of the specimen collection date, the notification date, or the notification receive date. Due to the dynamic nature of the NNDSS, data in this report may vary from data reported in other NNDSS reports and reports of IPD notifications at the state or territory level.

Australian Bureau of Statistics mid-year estimated resident populations were used to calculate notification rates (per 100,000 population).[6]

Statistical significance of the change of rates of IPD notifications overall and in some selected age groups in 2011 and 2012 compared with the previous year was ascertained using incidence rate ratios (IRRs) and 95% confidence interval assuming a Poisson distribution. Statistical analyses were performed using Stata version 10 Texas, USA: Stata Corp.

The evaluation of vaccination status in this report is described in Table 2. These definitions are applied to the vaccination fields reported to the NNDSS and are agreed to by the EIPDSWG.

Table 1: Enhanced invasive pneumococcal disease surveillance data collection performed by states and territories in 2011 and 2012

Age group	State or territory
All ages	Australian Capital Territory, Northern Territory, Queensland (except Metro South and Gold Coast Public Health Units), Tasmania, South Australia, Victoria,[1] Western Australia
Under 5 years	New South Wales, Queensland (Metro South and Gold Coast Public Health Units), Victoria*
Over 50 years	New South Wales and Victoria*

* Prior to 30 June 2012, Victoria followed up the collection of enhanced data on all ages. Between 1 July and 31 December 2012, Victoria only followed up the collection of enhanced data in the under 5 years and the 50 years or over age groups.

Table 2: Definitions of vaccination status and vaccine failure used in this report

Category	Definition
Fully vaccinated	Those 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.
Vaccination validation	Written confirmation of vaccination through the Australian Childhood Immunisation Register, state or territory immunisation register or health record.
Vaccine failure	Where a fully vaccinated child (as defined above) is diagnosed with IPD due to a serotype covered by the administered vaccine.

Case definition

According to the national IPD case definition, only laboratory confirmed cases of IPD are notifiable and therefore reported to the NNDSS. A laboratory confirmed case IPD is defined as the detection of *S. pneumoniae* from a normally sterile site, such as blood or cerebrospinal fluid either by culture or by nucleic acid amplification testing (NAAT).[7,8] Cases that meet this case definition are referred to a pneumococcal reference laboratory for serotype identification.

Serotype identification

The serotype information in this report is obtained through 2 methods. For culture positive specimens isolated from the vast majority of cases, serotypes were determined using the Quellung reaction, the gold standard method for serotyping using antisera produced by Statens Serum Institut, Denmark. Where pneumococcus has been detected by nucleic acid amplification test only, molecular serotyping was used to identify serotypes. Molecular serotyping may also be used to confirm indeterminate or equivocal results produced by the standard serotyping method. The Australian Government, through the National IPD Laboratory Surveillance Project, funds the serotyping of all *S. pneumoniae* isolates causing invasive disease.

Indigenous status

Cases of IPD were reported indicating the Indigenous status of the individual. The definition of an Aboriginal or Torres Strait Islander person within the NNDSS aligns with the Commonwealth definition, that is, an Aboriginal or Torres Strait Islander is determined by descent, self-identification and community acceptance. Completeness of Indigenous status reporting is described in the results section of this report.

Vaccination schedule

There were several amendments to the NIP schedule in 2011 with the most notable being the replacement of the 7vPCV and the 10vPCV for all infants with the 13vPCV and the subsequent catch-up program (Table 3).[3,4] There are now 4 pneumococcal vaccines available in Australia, each targeting multiple serotypes (Table 4).[3] Note that in this report serotype analysis is generally grouped according to vaccine composition. A detailed analysis of sero-

Table 3: Amendments to the National Immunisation Program pneumococcal vaccination schedule for 2011 and 2012

Vaccine type	NIP pneumococcal vaccination schedule
7-valent pneumococcal conjugate vaccine (7vPCV)	From 2005 to July 2011, 7vPCV was funded nationally for all infants as a 3-dose primary vaccination schedule consisting of doses at 2, 4 and 6 months of age without a booster in the 2nd year of life.
10-valent pneumococcal conjugate vaccine (10vPCV)	From October 2009 to September 2011, 10vPCV replaced the use of the 7vPCV in all children aged <2 years in the Northern Territory.
13-valent pneumococcal conjugate vaccine (13vPCV)	From July 2011, the 13vPCV replaced the 7vPCV for all infants. From October 2011, the 13vPCV replaced the 10vPCV for infants in the Northern Territory. From October 2011 to September 2012, a single supplementary dose of 13vPCV for children aged 12–35 months who completed primary vaccination with either 7vPCV or 10vPCV was made available for 12 months. From October 2012, a booster dose of 13vPCV was made available for Aboriginal and Torres Strait Islander children at 12-18 months of age living in the Northern Territory, South Australia, Queensland and Western Australia.
23-valent pneumococcal polysaccharide vaccine (23vPPV)	From October 2011, the 23vPPV booster dose for Aboriginal and Torres Strait Islander children aged 18–24 months living in the Northern Territory, South Australia, Queensland and Western Australia ceased.

Table 4: *Streptococcus pneumoniae* serotypes targeted by pneumococcal vaccines

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

types grouped by 10vPCV has not been included in this report as NIP-funded 10vPCV was restricted to infants residing in the Northern Territory and it was only available on the NIP for a 2 year period.

More information on the scheduling of the pneumococcal vaccination can be found in *The Australian Immunisation Handbook*.[3] The history of pneumococcal vaccination recommendations and practices is available through the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases.[4]

Results

Invasive pneumococcal disease notifications and rates

In 2011, 1,883 cases of IPD were reported to the NNDSS, representing a rate of 8.3 per 100,000 population. This was a 12% increase in the rate of IPD compared with that in 2010 (n = 1,640; 7.4 per 100,000) (IRR 1.12, 95% CI 1.05–1.20, $P < 0.01$). In 2012, 1,823 cases of IPD were reported, representing a rate of 8.0 per 100,000 and a 3.5% decrease in the rate of IPD in 2011 (IRR 0.96, 95% CI 0.90–1.03) (Table 5).

The total number of IPD cases notified to the NNDSS in 2011 was the highest number reported in any year since 2005 when the 7vPCV for all infants and the 23vPPV for all adults aged 65 years or over was introduced to the NIP (Figure 1).

Similar to previous years, the largest number of IPD cases was notified by New South Wales (2011: n = 529; 2012: n = 581) while the Northern Territory recorded the highest jurisdiction specific rate of IPD (2011: 55.8 per 100,000; 2012: 30.5 per 100,000) in both 2011 and 2012. The Australian Capital Territory recorded the lowest rate in 2011 (n = 26, 7.1 per 100,000), while in Victoria recorded the lowest rate in 2012 (6.8 per 100,000) (Table 5).

Similar to previous years, the number of cases of IPD was greatest in the winter months with the peak number of notifications occurring in August in both 2011 (n = 257) and 2012 (n = 271) (Figure 2). The peak number of notifications for those aged less than 5 years occurred slightly earlier, in June in 2011 (n = 44) and May in 2012 (n = 26).

Figure 1: Notified cases and rates of invasive pneumococcal disease, Australia, 2002 to 2012

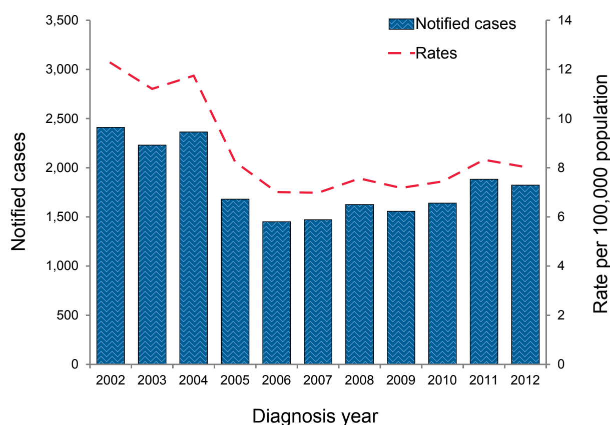
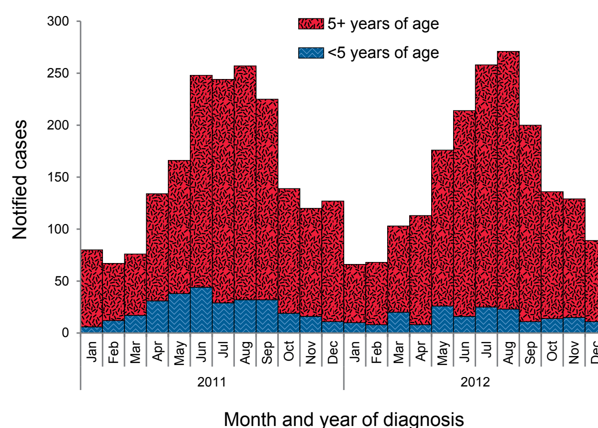


Figure 2: Notified cases of invasive pneumococcal disease, Australia, 2011 and 2012, by month, year of diagnosis and age group



Age and sex distribution

In both 2011 and 2012, in almost all age groups the notification rate of IPD was higher in males than in females. Overall, the male to female ratio was 1.2:1 in 2011 and 1.1:1 in 2012. As with previous years, the highest notification rate in 2011 was among the elderly aged 85 years or over (35.4 per 100,000) and in children aged 1 year (32.6 per 100,000). In 2012, the highest notification rates were again in the elderly aged 75 years or over while the rate in children aged 1 year reduced by 45% to 18.1 per 100,000 (IRR 0.55, 95% CI 0.39–0.78, $P < 0.01$) (Table 6). The lowest rates of IPD occurred in those aged between 10 and 29 years.

In 2011, 166 cases of IPD were notified in children aged under 2 years, representing a rate of 28.3 per 100,000. This was a 7% decrease on the rate of IPD reported in this age group in 2010 (n = 180; 30.5 per 100,000). In 2012, the rate of IPD in children aged under 2 years was 15.9 per 100,000 (n = 94) and a 44% decrease compared with the rate of IPD in 2011 (IRR 0.56, 95% CI 0.43–0.73, $P < 0.01$) (Figure 3).

Table 5: Notified cases and rates of invasive pneumococcal disease, Australia, 2011 and 2012 by state or territory, age group and Indigenous status

Age and Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
2011									
Notified cases aged <5 years									
Indigenous	0	6	13	9	1	0	0	23	52
Non-Indigenous	2	67	4	43	23	5	45	31	220
Unknown	0	0	0	6	0	0	9	0	15
Total	2	73	17	58	24	5	54	54	287
Notified cases aged 5–64 years									
Indigenous	2	11	85	33	12	1	3	93	240
Non-Indigenous	17	150	18	137	50	21	173	53	619
Unknown	0	101	0	37	1	0	40	0	179
Total	19	262	103	207	63	22	216	146	1,038
Notified cases ≥65 years									
Indigenous	0	1	6	5	0	0	0	1	13
Non-Indigenous	5	189	3	53	54	20	124	42	490
Unknown	0	4	0	17	2	0	32	0	55
Total	5	194	9	75	56	20	156	43	558
Total									
Indigenous	2	18	104	47	13	1	3	117	305
Non-Indigenous	24	406	25	233	127	46	342	126	1329
Unknown	0	105	0	60	3	0	81	0	249
Total	26	529	129	340	143	47	426	243	1,883
Rate (per 100,000 population)	7.1	7.3	55.8	7.6	8.7	9.2	7.7	10.3	8.3*
Indigenous status completeness (%)	100	80	100	82	98	100	81	100	87
2012									
Notified cases aged <5 years									
Indigenous	0	4	8	8	2	1	0	4	27
Non-Indigenous	3	62	3	18	9	4	34	16	149
Unknown	0	0	0	8	0	0	3	0	11
Total	3	66	11	34	11	5	37	20	187
Notified cases aged 5–64 years									
Indigenous	0	9	46	54	15	2	4	69	199
Non-Indigenous	16	153	7	135	58	22	133	85	609
Unknown	0	116	0	31	0	0	59	1	207
Total	16	278	53	220	73	24	196	155	1,015
Notified cases ≥65 years									
Indigenous	0	4	4	5	0	1	2	5	21
Non-Indigenous	8	233	4	74	47	15	141	55	577
Unknown	0	0	0	15	0	0	8	0	23
Total	8	237	8	94	47	16	151	60	621
Total									
Indigenous	0	17	58	67	17	4	6	78	247
Non-Indigenous	27	448	14	227	114	41	308	156	1,335
Unknown	0	116	0	54	0	0	70	1	241
Total	27	581	72	348	131	45	384	235	1,823
Rate (per 100,000 population)	7.2	8.0	30.5	7.6	7.9	8.8	6.8	9.6	8.0
Indigenous status completeness (%)	100	80	100	84	100	100	82	100	87

* Statistically significant increase compared with previous year (P<0.01).

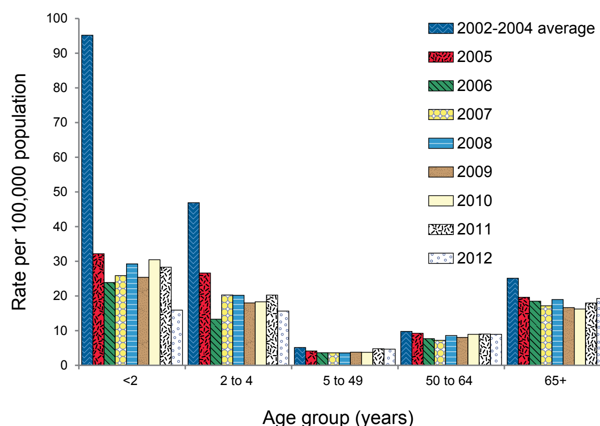
In 2011, 558 cases of IPD were notified in adults aged 65 years or over, representing a rate of 18.0 per 100,000. This was an 11% increase in the rate of IPD reported in this age group in 2010 (n = 485; 16.2 per 100,000). In 2012, 621 cases were reported in adults aged 65 years or over, representing a rate of 19.3 per 100,000 and a 7% increase on the rate of IPD reported in this age group in 2011 (Figure 3).

Table 6: Notification rates per 100,000 of invasive pneumococcal disease, Australia, 2011 and 2012, by age group and sex

Age group	Male		Female		Total	
	2011	2012	2011	2012	2011	2012
0	25.0	11.8	22.9	15.9	24.0	14.1
1	41.5	22.6	23.3	13.3	32.6	18.1*
2	16.9	14.4	15.1	13.8	16.0	14.1
3	14.5	7.9	13.1	7.6	13.8	7.8
4	11.5	8.6	10.7	10.4	11.1	9.5
0–4	21.8	13.0	17.0	12.2	19.5	12.6
5–9	7.9	4.7	6.7	4.3	7.3	4.5
10–14	2.8	3.2	3.9	2.1	3.3	2.7
15–19	3.7	3.7	2.1	1.8	2.9	2.8
20–24	2.8	1.6	2.9	3.0	2.8	2.3
25–29	3.5	3.5	3.4	3.8	3.4	3.7
30–34	4.7	6.1	5.4	4.8	5.0	5.5
35–39	6.2	6.5	4.9	8.1	5.5	7.3
40–44	8.3	8.0	5.0	5.5	6.7	6.7
45–49	8.5	6.6	4.2	6.0	6.3	6.3
50–54	7.3	8.0	5.7	7.8	6.5	7.9
55–59	8.5	9.0	7.8	7.0	8.1	8.0
60–64	12.4	12.5	13.5	10.2	13.1	11.4
65+	20.0	19.9	16.3	18.8	18.0	19.3
65–69	15.5	14.3	12.3	11.2	13.9	12.8
70–74	14.1	16.7	12.5	11.2	13.3	13.9
75–79	18.8	18.4	11.4	21.3	14.8	19.9
80–84	23.9	25.0	21.0	23.4	22.3	24.1
85+	45.8	42.9	29.7	36.4	35.4	38.7
Total	9.0	8.3	7.6	7.8	8.3	8.0

* Statistically significant increase compared with previous year (P<0.01).

Figure 3: Notification rates of invasive pneumococcal disease, Australia, 2002 to 2012, by age group



Invasive pneumococcal disease in the Indigenous population

Indigenous status was reported in 87% of notifications in both 2011 and 2012. In 2011, 305 cases of IPD were notified in the Indigenous population, representing a rate of 53.0 per 100,000 and 16% of all cases. This was a 52% increase compared with the rate of IPD in this group in 2010 (34.8 per 100,000, n = 196) (IRR 1.52, 95% CI 1.27–1.83, $P < 0.01$). In 2012, 247 cases were reported in the Indigenous population, representing a rate of 42.0 per 100,000, 14% of all cases, and a 21% decrease on the rate of IPD in this group in 2011. The increased number of notifications during 2011 and 2012 compared with previous years was mostly due to an outbreak of serotype 1 among the Indigenous populations in the Northern Territory, Western Australia and to a lesser extent Queensland (Table 7). The rate of IPD in the Indigenous population was 9 times higher than the rate of IPD in the non-Indigenous population in 2011 (6.0 per 100,000) and 7 times

Table 7: Notified cases of invasive pneumococcal disease due to serotype 1 in Indigenous Australians, Australia, 2008 to 2012, by state or territory

Year	State or territory								Total
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
2008	0	0	0	2	0	0	0	0	2
2009	0	1	0	1	0	0	0	0	2
2010	0	2	6	0	4	0	0	15	27
2011	0	1	51	12	2	0	0	44	110
2012	0	0	14	20	1	0	0	15	50

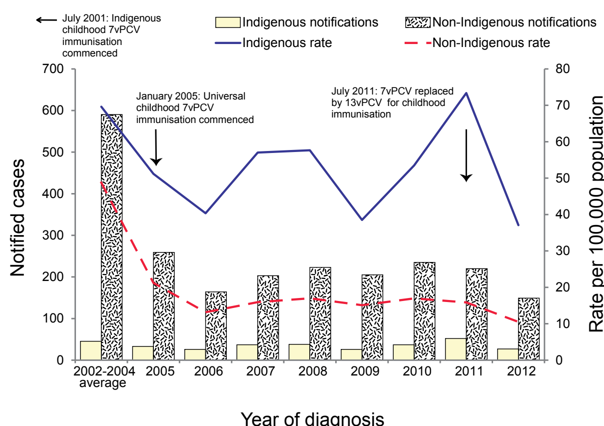
higher in 2012 (6.0 per 100,000). Further analyses of the Indigenous population group are provided throughout the report.

Invasive pneumococcal disease in children by Indigenous status

The rate of IPD in Indigenous children aged less than 5 years was 73.4 per 100,000 in 2011 (n = 52) and 37.1 per 100,000 in 2012 (n = 27). The rate of IPD in Indigenous children aged less than 5 years in 2011 had increased by 37% compared with that in 2010 (n = 37, 53.5 per 100,000); however this change was not statistically significant (IRR 1.37, 95% CI 0.89–2.15). The rate in non-Indigenous children aged less than 5 years was 15.7 per 100,000 in 2011 (n = 220) and 10.6 per 100,000 in 2012 (n = 149) (Figure 4). In 2011 and 2012, the rate of IPD among Indigenous children aged less than 5 years was 4.5 to 5 times higher than the rate of IPD in non-Indigenous children.

Table 8, Figure 5 and Figure 6 show the notified cases and rates of IPD in children aged less than 5 years by Indigenous status and smaller age groups over the last decade.

Figure 4: Notified cases and rates of invasive pneumococcal disease in children aged less than 5 years, Australia, 2002 to 2012, by Indigenous status



The rate of IPD in Indigenous children has shown large fluctuations over the last decade due to the small number of notifications. However, the higher number of notifications among Indigenous children in 2011 is mostly due to an

Figure 5: Notified cases and rates of invasive pneumococcal disease in Indigenous children aged less than 5 years, Australia, 2002 to 2012, by age group

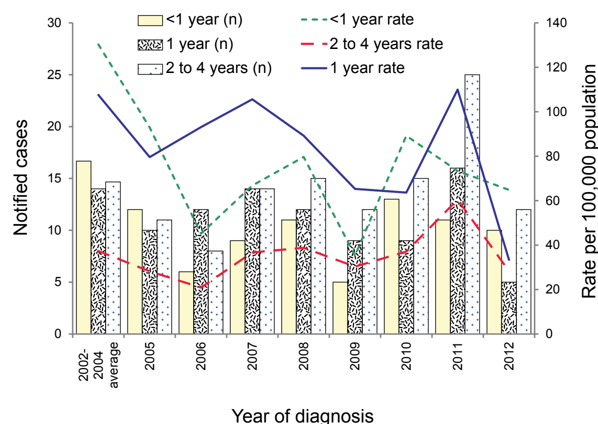


Figure 6: Notified cases and rates of invasive pneumococcal disease in non-Indigenous children aged less than 5 years, Australia, 2002 and 2012, by age group

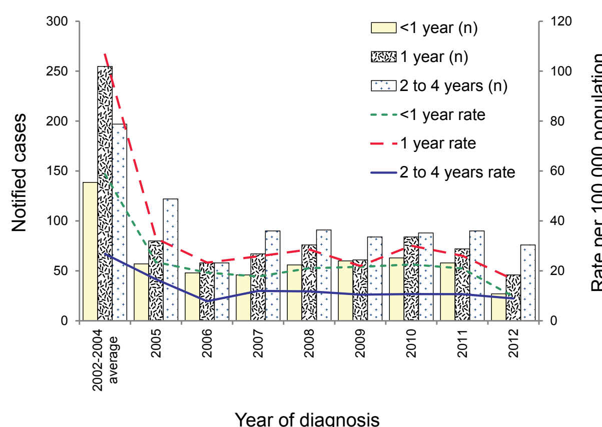


Table 8: Notified cases and rates of invasive pneumococcal disease in children aged less than 5 years, Australia, 2011 and 2012, by age group and Indigenous status

Age group		Indigenous		Non-Indigenous	
		2011	2012	2011	2012
<1 year	Number of cases	11	10	58	27
	Rate per 100,000	73.4	64.8	21.0	9.7
1 year	Number of cases	16	5	72	46
	Rate per 100,000	110.0	33.4	26.0	16.6
2 to 4 years	Number of cases	25	12	90	76
	Rate per 100,000	60.5	28.3	10.7	9.0

outbreak of serotype 1 observed in the Northern Territory, Western Australia and Queensland in that year. In 2011, 38% (20/52) of all notifications in Indigenous children were due to serotype 1 and 65% of those notifications were in Indigenous children aged 2 to 4 years (13/20).

Mortality

In 2011 and 2012, there were 134 and 126 deaths respectively, attributed to IPD and notified to NNDSS. Of those cases reported to have died, approximately 10% (n = 14) in 2011 and 7% (n = 9) in 2012 were reported as Indigenous (Table 9).

In those aged less than 5 years, there were 6 deaths associated with IPD in 2011 and 1 death in 2012 giving a case fatality rate (CFR) of 2.1% and 0.5% respectively. Of those 7 deaths, none were potentially preventable by the 7vPCV. Three of the deaths were caused by serotype 19A, which is included in the 13vPCV. However,

Table 9: Deaths reported to the National Notifiable Diseases Surveillance System and case fatality rates for invasive pneumococcal disease, Australia, 2011 and 2012, by age group, Indigenous status and state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.†
2011									
Notified cases aged <5 years									
Deaths	0	1	0	3	0	0	0	2	6
CFR* %	0.0	N/A	0.0	N/A	0.0	0.0	0.0	3.7	2.1
Notified cases aged 5–64 years									
Deaths	1	15	4	10	5	2	9	6	52
CFR* %	5.3	N/A	3.9	N/A	7.9	9.1	N/A	4.1	5.0
Notified cases ≥ 65 years									
Deaths	0	37	1	5	4	5	18	6	76
CFR* %	0.0	N/A	11.1	N/A	7.1	25.0	11.5	14.0	13.6
Total									
Total deaths	1	53	5	18	9	7	27	14	134
Completeness %	-	-	-	-	-	-	-	-	61
Indigenous status									
Indigenous deaths	0	1	4	3	1	0	0	5	14
Non-Indigenous deaths	1	49	1	12	7	7	21	9	107
Unknown status deaths	0	3	0	3	1	0	6	0	13
2012									
Notified cases aged <5 years									
Deaths	0	0	0	0	0	0	1	0	1
CFR* %	0.0	N/A	0.0	0.0	0.0	0.0	2.7	0.0	0.5
Notified cases aged 5–64 years									
Deaths	1	11	3	7	4	3	6	2	37
CFR* %	6.3	N/A	5.7	N/A	5.5	12.5	N/A	1.3	3.6
Notified cases ≥65 years									
Deaths	2	36	2	9	2	3	24	10	88
CFR* %	25.0	N/A	25.0	9.6	4.3	18.8	15.9	16.7	14.2
Total									
Total deaths	3	47	5	16	6	6	31	12	126
Completeness %	-	-	-	-	-	-	-	-	61
Indigenous status									
Indigenous deaths	0	1	2	2	2	1	0	1	9
Non-Indigenous deaths	3	46	3	12	4	5	29	11	113
Unknown status deaths	0	0	0	2	0	0	2	0	4

* Case fatality rates (CFR) are not presented for those jurisdictions reporting less than 50% completeness of death data in that age group or if that jurisdiction does not actively follow up all cases in that age group as per Table 1.

† Total for Australia includes all jurisdictional data irrespective of individual jurisdictional data completeness.

all 3 cases received their vaccinations prior to the July 2011 introduction of the 13vPCV to the NIP. One death was caused by serotype 7F, which is also included in the 13vPCV. This case died after the introduction of the 13vPCV to the NIP but was too young for vaccination. Further details, including Indigenous status, serotype and vaccination history, of these 7 deaths are shown in Table 10.

In the 65 years or over age group, there were 76 deaths (Indigenous: n = 2; non-Indigenous: n = 74) associated with IPD in 2011 and 88 deaths (Indigenous: n = 4; non-Indigenous: n = 84) in 2012, giving CFRs of 13.6% and 14.2% respectively. Of those deaths, 76% (58/76) in 2011 and 59% (52/88) in 2012 were attributable to a serotype included in the 23vPPV. The most frequently reported 23vPPV serotypes associated with death were 19A (2011: 33%, 19/58; 2012: 27%, 14/52) and 3 (2011: 20%, 12/58; 2012: 25%, 13/52).

Risk factors

Risk factor data were provided for 62% (2,307/3,706) of cases reported in 2011 and 2012 combined. Of the cases with risk factor data reported, 80% (1,850/2,307) of cases reported at least 1 risk factor. Table 11 shows data on the risk factors for IPD in specified population subgroups for 2011 and 2012 combined.

In children aged less than 5 years, the most frequently reported known risk factor in the Indigenous population was 'Other' (65%; 26/40), e.g. asthma, previous pneumonia ,or exposure to

Table 10: Characteristics of deaths attributable to invasive pneumococcal disease in children aged less than 5 years, Australia, 2011 and 2012

Case	Year of diagnosis	Sex	Age (months)	Indigenous status	Serotype	Vaccine type and number of doses	Risk factors
1	2011	Female	22	Non-Indigenous	22F	7vPCV: 3 doses	Childcare attendee
2	2011	Male	5	Non-Indigenous	19A	7vPCV: 1 doses	No risk factor identified
3	2011	Male	12	Indigenous	19A	7vPCV: 1 doses	No risk factor identified
4	2011	Male	12	Non-Indigenous	19A	7vPCV: 3 doses	No risk factor identified
5	2011	Male	7	Non-Indigenous	11A	7vPCV: 3 doses	Unknown
6	2011	Female	1	Indigenous	23B	0	No risk factor identified
7	2012	Female	1	Non-Indigenous	7F	0	Information not supplied

Table 11: Number of risk factors reported for invasive pneumococcal disease notifications, Australia, 2011 and 2012, by risk factor and vaccine targeted population sub-group

Risk factor*	Children aged less than 5 years		Indigenous aged 50 years or over	Non-Indigenous aged 65 years or over
	Indigenous	Non-Indigenous		
Premature (<37 weeks gestation)	10	21	N/A	N/A
Congenital or chromosomal abnormality	4	15	0	0
Anatomic or functional asplenia	0	1	0	14
Immunocompromised	2	22	21	189
Chronic illness	12	20	91	498
Childcare attendee	6	57	N/A	N/A
Previous episode of IPD	4	4	8	14
Other†	26	20	71	371
No risk factor identified	26	105	2	49
Unknown or not reported	13	145	14	302
Total known risk factors	40	119	111	716
Total	79	369	127	1,067

* Case may be reported with more than 1 risk factor.

† Other risk factors include but are not limited to, asthma, previous pneumonia and exposure to smoke.

smoke, followed by chronic illness (30%; 12/40). In the non-Indigenous population, the most frequently reported known risk factor was childcare attendee (48%; 57/119) followed by immunocompromised (18%; 22/119).

In both the adult population groups described in Table 11, the most frequently reported known risk factor was chronic illness (Indigenous aged 50 years or over: 82%, 91/111; non-Indigenous aged 65 years or over: 70%, 498/716) followed by 'Other' (Indigenous aged 50 years or over: 64%, 71/111; non-Indigenous aged 65 years or over: 52%, 371/716).

Pneumococcal serotypes causing invasive disease

Pneumococcal serotypes were identified for 94% (1,775/1,883) of cases in 2011 and 95% (1,729/1,823) of cases in 2012. Of those cases with a serotype identified:

- 8% (139/1,775) of cases in 2011 and 7% (124/1,729) of cases in 2012 were due to a serotype included in the 7vPCV;
- 19% (340/1,775) of cases in 2011 and 18% (312/1,1729) of cases in 2012 were due to one of the additional 3 serotypes (1, 5 and 7F) included in the 10vPCV [10vPCV (non-7vPCV)];
- 32% (561/1,775) of cases in 2011 and 27% (462/1,1729) of cases in 2012 were due to one of the additional 3 serotypes (3, 6A and 19A) included in the 13vPCV [13vPCV (non-10vPCV)]; and
- 69% (1,227/1,775) of cases in 2011 and 67% (1,153/1,729) of cases in 2012 were due to one of the additional 16 serotypes included in the 23vPPV that are not included in the 7vPCV [23vPPV (non-7vPCV)].

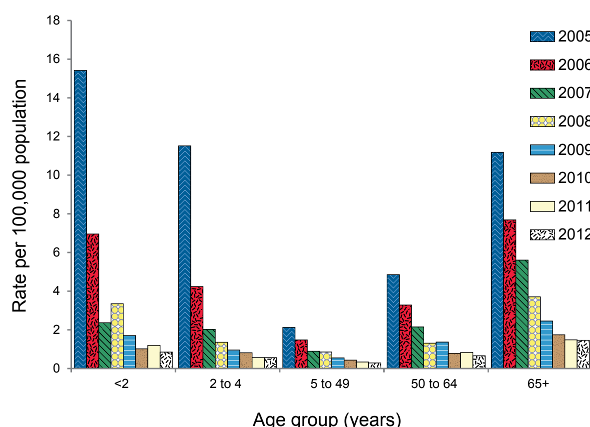
Table 12 and Table 13 shows data on serotypes, grouped by pneumococcal vaccine, age group and Indigenous status for 2011 and 2012. Note that these tables do not include cases with an unknown Indigenous status.

In 2011 and 2012, the most frequently reported serotypes causing IPD were 19A (2011: n = 430; 2012: n = 293), 7F (2011: n = 184; 2012: n = 213), 1 (2011: n = 155; 2012: n = 97), 3 (2011: n = 120; 2012: n = 156), 22F (2011: n = 114; 2012: n = 137) and 6C (2011: n = 108; 2012: n = 96). These 6 serotypes accounted for 59% (1,111/1,883) of all notifications in 2011 and 54% (992/1,823) in 2012.

7-valent pneumococcal conjugate vaccine serotypes

In 2011, 139 cases of IPD due to serotypes included in the 7vPCV were notified, representing a rate of 0.6 per 100,000. This was a 10% decrease on the number in 2010 (n = 155) and an 85% decrease on the number in 2005 (n = 909). In 2012, 124 cases of IPD due to 7vPCV serotypes were notified (0.5 per 100,000), which was an 11% decrease on the number in 2011 and an 86% decrease on the number recorded in 2005. Since the 2005 introduction of the 7vPCV, there has been an overall decrease in the notification rate of IPD due to serotypes included in the 7vPCV across all age groups. However, in recent years this decline appears to have plateaued across all age groups (Figure 7).

Figure 7: Notification rates for invasive pneumococcal disease caused by 7vPCV serotypes, Australia, 2005 to 2012, by age group



In children aged less than 5 years, there were 12 cases of IPD due to 7vPCV serotypes reported in 2011 (0.8 per 100,000) and 10 cases in 2012 (0.7 per 100,000). In both 2011 and 2012, the rate of IPD due to 7vPCV serotypes in Indigenous children aged less than 5 years remained unchanged from the 2010 rate of 1.4 per 100,000 (2011: n = 1; 2012: n = 1). In non-Indigenous children, there were 10 cases of IPD due to 7vPCV serotypes in 2011 (0.7 per 100,000) and 8 cases in 2012 (0.6 per 100,000) (Figure 8). One case of IPD due to a 7vPCV serotype in 2011 and 1 case in 2012 was reported with an unknown Indigenous status.

In the period (January 2005 to July 2011) that the 7vPCV was available on the NIP to children aged less than 5 years, there was an overall increase in the rate of IPD due to non-7vPCV serotypes in this age group, with 257 cases (17.5 per 100,000)

Table 12: Notified cases of invasive pneumococcal disease, Australia, 2011, by pneumococcal vaccine serotypes, age and Indigenous status*

Vaccine type	Indigenous			Non-Indigenous		
	n	%	Cumulative (%)	n	%	Cumulative (%)
<5 years						
7vPCV	1	2	2	10	5	5
10vPCV (non-7vPCV)	20	40	42	22	11	16
13vPCV (non-10vPCV)	10	20	62	123	60	76
Non-conjugate serotypes	19	38	100	49	24	100
Total	50	100		204	100	
23vPPV (non-7vPCV)	41	82		164	80	
5-49 years						
7vPCV	14	7	7	21	7	7
10vPCV (non-7vPCV)	91	48	56	94	30	36
13vPCV (non-10vPCV)	14	7	63	92	29	65
Non-conjugate serotypes	70	37	100	110	35	100
Total	189	100		317	100	
23vPPV (non-7vPCV)	141	75		236	74	
50-64 years						
7vPCV	2	4	4	31	12	12
10vPCV (non-7vPCV)	10	22	26	35	13	25
13vPCV (non-10vPCV)	8	17	43	86	32	57
Non-conjugate serotypes	26	57	100	116	43	100
Total	46	100		268	100	
23vPPV (non-7vPCV)	29	63		184	69	
65+ years						
7vPCV	1	8	8	43	9	9
10vPCV (non-7vPCV)	1	8	17	27	6	15
13vPCV (non-10vPCV)	2	17	33	153	33	48
Non-conjugate serotypes	8	67	100	243	52	100
Total	12	100		466	100	
23vPPV (non-7vPCV)	4	33		269	58	
Total						
7vPCV	18	6	6	105	8	8
10vPCV (non-7vPCV)	122	41	47	178	14	23
13vPCV (non-10vPCV)	34	11	59	454	36	59
Non-conjugate serotypes	123	41	100	518	41	100
Total	297	100		1,255	100	
23vPPV (non-7vPCV)	215	72		853	68	

* Does not include cases with an unknown Indigenous status

reported in 2011. In 2012, following the NIP schedule change from 7vPCV to 13vPCV, there was a 35% reduction in cases due to non-7vPCV serotypes (n = 167; 11.3 per 100,000).

In 2011, 49 cases of IPD due to non-7vPCV serotypes in Indigenous children aged less than 5 years were notified, representing a rate of 69.1 per 100,000. In 2012, 26 cases of IPD due to non-7vPCV serotypes were notified (35.7 per 100,000), which was a 47% decrease on the number of notifications in 2011. In non-Indigenous children aged less than 5 years, the number of notifications due to non-7vPCV serotypes in 2012 (n = 133; 9.4 per 100,000) was a 31% decrease on the number of notifications in this group in 2011 (n = 194; 13.8 per 100,000) (Figure 8).

Table 13: Notified cases of invasive pneumococcal disease, Australia, 2012, by pneumococcal vaccine serotypes, age and Indigenous status

Vaccine type	Indigenous			Non-Indigenous		
	n	%	Cumulative (%)	n	%	Cumulative (%)
<5 years						
7vPCV	1	4	4	8	6	6
10vPCV (non-7vPCV)	9	33	37	12	9	14
13vPCV (non-10vPCV)	2	7	44	55	39	53
Non-conjugate serotypes	15	56	100	66	47	100
Total	27	100		141	100	
23vPPV (non-7vPCV)	17	63		93	66	
5-49 years						
7vPCV	11	7	7	16	6	6
10vPCV (non-7vPCV)	50	33	40	89	31	36
13vPCV (non-10vPCV)	10	7	46	78	27	63
Non-conjugate serotypes	82	54	100	106	37	100
Total	153	100		289	100	
23vPPV (non-7vPCV)	100	65		223	77	
50-64 years						
7vPCV	3	7	7	22	8	8
10vPCV (non-7vPCV)	8	18	25	42	14	22
13vPCV (non-10vPCV)	5	11	36	87	30	52
Non-conjugate serotypes	28	64	100	140	48	100
Total	44	100		291	100	
23vPPV (non-7vPCV)	22	50		208	71	
65+ years						
7vPCV	1	5	5	46	8	8
10vPCV (non-7vPCV)	5	25	30	34	6	15
13vPCV (non-10vPCV)	6	30	60	163	30	44
Non-conjugate serotypes	8	40	100	304	56	100
Total	20	100		547	100	
23vPPV (non-7vPCV)	15	75		314	57	
Total						
7vPCV	16	7	7	92	7	7
10vPCV (non-7vPCV)	72	30	36	177	14	21
13vPCV (non-10vPCV)	23	9	45	383	30	51
Non-conjugate serotypes	133	55	100	616	49	100
Total	244	100		1,268	100	
23vPPV (non-7vPCV)	154	63		838	66	

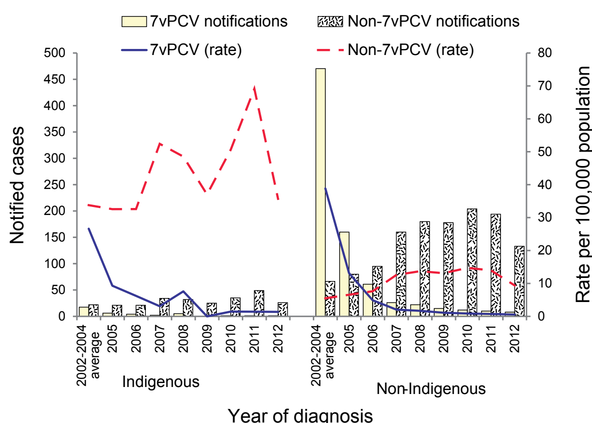
* Does not include cases with an unknown Indigenous status.

13-valent pneumococcal conjugate vaccine serotypes

In 2011, there were 182 cases of IPD in children aged less than 5 years due to the 6 additional serotypes included in the 13vPCV (1, 5, 7F, 3, 6A and 19A) over the 7vPCV [13vPCV (non-7vPCV)], representing a rate of 12.4 per 100,000. In 2012, following the July 2011 NIP schedule change from 7vPCV to 13PCV, there were 82 cases of IPD in children aged less than 5 years due to those 13vPCV (non-7vPCV) serotypes (5.5 per 100,000). This was a 55% decrease on the number of cases reported in 2011.

In Indigenous children aged less than 5 years, there was an overall reduction in the number of IPD cases caused by 13vPCV (non-7vPCV) serotypes,

Figure 8: Notified cases and rates of invasive pneumococcal disease caused by 7vPCV and non-7vPCV in children aged less than 5 years, 2002 to 2012, by Indigenous status

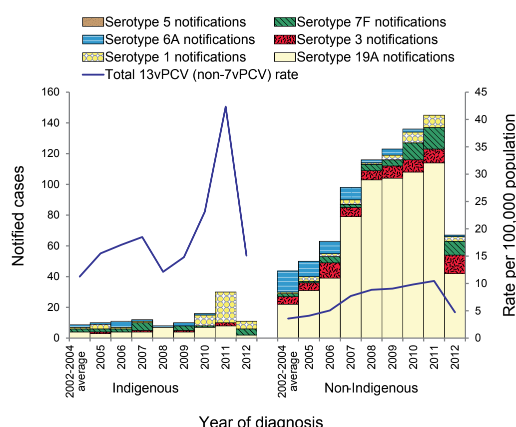


in particular serotypes 1 and 19A, following the introduction of the 13vPCV to the NIP. In 2011, there were 8 cases of IPD in Indigenous children due to serotype 19A and 20 cases due to serotype 1. In 2012, cases of IPD in Indigenous children due to 19A reduced by 75% (n = 2) while cases due to serotype 1 also reduced by 75% (n = 5). Overall, the rate of IPD in Indigenous children due to 13vPCV (non-7vPCV) serotypes reduced from 42.3 per 100,000 in 2011 to 15.1 per 100,000 in 2012 (Figure 9).

Similarly, in non-Indigenous children aged less than 5 years, there was an overall reduction in IPD cases caused by 13vPCV (non-7vPCV) serotypes following the introduction of the 13vPCV to the NIP and this was mostly due to a reduction in IPD caused by 19A. There were 114 cases of IPD in non-Indigenous children due to serotype 19A in 2011 and 42 in 2012. IPD cases in non-Indigenous children due to serotype 7F decreased from 14 cases in 2011 to 9 cases in 2012, while cases due to serotype 3 increased from 9 cases in 2011 to 12 cases in 2012. Overall, the rate of IPD in non-Indigenous children due to 13vPCV (non-7vPCV) serotypes reduced from 10.3 per 100,000 in 2011 to 4.8 per 100,000 in 2012 (Figure 9).

Note that in the last decade there have been no cases of IPD due to serotype 5 notified in children aged less than 5 years.

Figure 9: Notified cases and rates of invasive pneumococcal disease caused by 13vPCV (non-7vPCV) serotypes in children aged less than 5 years, 2002 to 2012, by Indigenous status



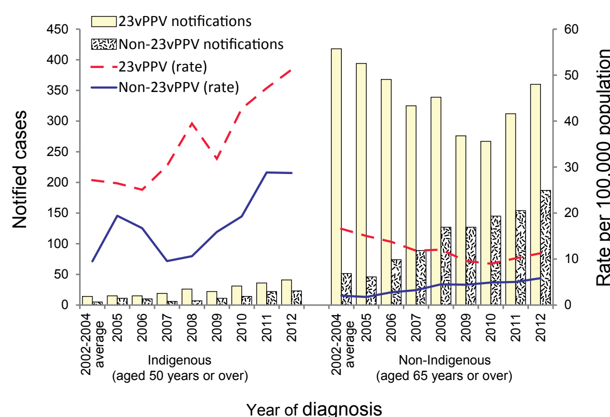
23-valent pneumococcal polysaccharide vaccine serotypes

In Indigenous adults aged 50 years or over, the number of notifications caused by serotypes included in the 23vPPV has continued to show an overall increase with 36 cases (47.2 per 100,000) reported in 2011 and 41 cases (51.2 per 100,000) in 2012. Unlike previous years, the number of notifications due to 23vPPV serotypes in non-Indigenous adults aged 65 years or over increased in both 2011 (n = 312, 10.1 per 100,000) and 2012 (n = 360, 11.2 per 100,000) (Figure 10).

In Indigenous adults aged 50 years or over, the number of notifications due to non-23vPPV serotypes remained steady with 22 cases in 2011 (28.8 per 100,000) and 23 cases in 2012 (28.7 per 100,000). The number of notifications caused by non-23vPPV serotypes in non-Indigenous adults aged 65 years or over continued to show an overall increase with 154 cases in 2011 (5.0 per 100,000) and 187 cases in 2012 (5.8 per 100,000) (Figure 10).

In both 2011 and 2012, the most frequent serotype causing disease in Indigenous adults aged 50 years

Figure 10: Notified cases and rates of 23vPPV and non-23vPPV serotypes causing invasive pneumococcal disease in Indigenous adults aged 50 years or over and non-Indigenous adults aged 65 years or over, 2002 to 2012



or over was serotype 1 (2011: n = 8; 2012: n = 9) and a result of the serotype 1 outbreak observed over that period. The 5-year mean of serotype 1 notifications during the 5 years prior to 2011 was 1.8 notifications. The next most frequent serotype was 19A (n = 6) and 6C (n = 6) in 2011 and 3 in 2012 (n = 5). In both 2011 and 2012, the number of notifications due to serotype 6A in Indigenous adults aged 50 years or over remained similar to previous years (2011: n = 0; 2012: n = 2).

In both 2011 and 2012, the most frequent serotype causing disease in non-Indigenous adults aged 65 years or over was serotype 19A (2011: n = 109; 2012: n = 90). The next most frequent serotype was 6C (n = 56) in 2011 and 3 in 2012 (n = 67). The number of notifications due to serotype 6A in non-Indigenous adults aged 65 years or over has continued to decline, with the number of notifications in 2011 (n = 5) and 2012 (n = 6) being half that of the number notified in 2010 (n = 12).

Vaccine failures

In children aged less than 5 years who were fully vaccinated, there were 6 cases in 2011 and 10 cases in 2012 that were considered to be a vaccine failure according to the definition described in Table 2. In 2011 and 2012 combined, 13 cases were characterised as a 7vPCV failure, 2 cases were a 13vPCV failure and the remaining case was a 10vPCV failure. Serotype 19F was reported as the cause of disease in 63% (n = 10) of these cases (Table 14).

Antibiotic resistance

Penicillin and ceftriaxone/cefotaxime susceptibility data were analysed only for jurisdictions that reported susceptibility data for more than 50% of cases. In 2011, penicillin and ceftriaxone/cefotaxime susceptibility completeness was suitable for reporting for all jurisdictions except Victoria. In 2012, penicillin and ceftriaxone/cefotaxime susceptibility completeness was suitable for reporting for all jurisdictions except Western Australia.

Penicillin

Penicillin susceptibility data were reported for 68% (1,275/1,883) of cases in 2011 and 76% (1,389/1,823) of cases in 2012; and of those, 12% (151/1,275) of cases in 2011 and 10% (132/1,389) of cases in 2012 were reported with reduced susceptibility to penicillin (Table 15). Of those cases with reduced susceptibility to penicillin in 2011 (excluding Victoria), 149 cases were serotyped. Of those serotyped cases, 17% (26/149) were due to a serotype in the 7vPCV, 85% (126/149) were due to a serotype in the 23vPPV and serotypes 9V (n = 4), 19A (n = 93) and 19F (n = 14) accounted for 75% (111/149) of serotyped cases.

Table 14: Characteristics of vaccine failures in children aged less than 5 years, Australia, 2011 and 2012

Case	Year of diagnosis	Age	Indigenous status	Serotype	Vaccine type and number of doses	Clinical category	Risk factors
1	2011	3 years	Non-Indigenous	14	7vPCV: 3 doses	Bacteraemia	Yes
2	2011	4 years	Indigenous	18C	7vPCV: 3 doses	Bacteraemia	Yes
3	2011	10 months	Non-Indigenous	19F	7vPCV: 3 doses	Pneumonia	Unknown
4	2011	2 years	Not reported	19F	7vPCV: 3 doses	Bacteraemia	No
5	2011	2 years	Non-Indigenous	19F	7vPCV: 3 doses	Other	Unknown
6	2011	3 years	Non-Indigenous	19F	7vPCV: 3 doses	Pneumonia	Yes
7	2012	3 years	Indigenous	9V	7vPCV: 1 dose & 10vPCV: 3 doses	Pneumonia	Yes
8	2012	12 months	Non-Indigenous	19A	13vPCV: 3 doses	Pneumonia	Unknown
9	2012	16 months	Non-Indigenous	19F	13vPCV: 3 doses	Septic arthritis	Unknown
10	2012	20 months	Non-Indigenous	19F	7vPCV: 3 doses	Bacteraemia	Unknown
11	2012	22 months	Non-Indigenous	4	7vPCV: 3 doses	Pneumonia	No
12	2012	2 years	Non-Indigenous	19F	7vPCV: 3 doses	Pneumonia	Yes
13	2012	20 months	Non-Indigenous	19F	7vPCV: 3 doses	Bacteraemia	Yes
14	2012	3 years	Non-Indigenous	19F	7vPCV: 3 doses	Bacteraemia	Yes
15	2012	4 years	Non-Indigenous	19F	7vPCV: 3 doses	Pneumonia plus Other sterile site not specified	Yes
16	2012	2 years	Non-Indigenous	6B	7vPCV: 3 doses	Meningitis	Yes

Of those cases with reduced susceptibility to penicillin in 2012 (excluding Western Australia), 126 cases were serotyped. Of those serotyped cases, 17% (22/126) were due to a serotype in the 7vPCV, 60% (76/126) were due to a serotype in the 23vPPV and serotypes 9V (n = 9), 19A (n = 59) and 19F (n = 7) accounted for 60% (75/126) of serotyped cases.

Ceftriaxone/cefotaxime

Ceftriaxone/cefotaxime susceptibility was reported in 59% (1,118/1,883) of cases in 2011 and 68% (1,242/1,823) of cases in 2012; of those, 2% (22/1,118) of cases in 2011 and 2% (20/1,242) of cases in 2012 were reported with reduced susceptibility to ceftriaxone/cefotaxime (Table 15).

Of those cases with reduced susceptibility to ceftriaxone/cefotaxime in 2011 (excluding Victoria), 22 cases were serotyped. Of those serotyped cases, 50% (11/22) were due to a serotype in the 7vPCV, 59% (13/22) were due to a serotype in the 23vPPV and serotypes 9V (n = 2), 19A (n = 10) and 19F (n = 7) accounted for 86% (19/22) of serotyped cases.

Of those cases with reduced susceptibility to ceftriaxone/cefotaxime in 2012 (excluding Western Australia), 18 cases were serotyped. Of those serotyped cases, 39% (7/18) were due to a serotype in the 7vPCV, 61% (11/18) were due to a serotype in the 23vPPV and serotypes 9V (n = 1), 19A (n = 10) and 19F (n = 6) accounted for 94% (17/18) of serotyped cases with reduced susceptibility to the third generation cephalosporins.

Table 15: *Streptococcus pneumoniae* susceptibility to penicillin and ceftriaxone/cefotaxime for selected states and territories,* 2011 and 2012

	9V	19F	All 7vPCV serotypes	19A	All 23vPPV	Not specified	All isolates
2011							
Penicillin							
Resistant	1	6	11	16	27	1	30
Intermediate	3	8	15	77	99	1	121
Sensitive	10	20	75	200	853	30	1,124
Total tested	14	34	101	293	979	32	1,275
Total isolates with reduced susceptibility (%)	4 (29%)	14 (41%)	26 (26%)	93 (32%)	126 (13%)	2 (6%)	151 (12%)
Ceftriaxone/cefotaxime							
Resistant	1	2	3	2	3	0	6
Intermediate	1	5	8	8	10	0	16
Sensitive	10	20	73	264	815	16	1,096
Total tested	12	27	84	274	828	16	1,118
Total isolates with reduced susceptibility (%)	2 (17%)	7 (26%)	11 (13%)	10 (4%)	13 (2%)	0 (0%)	22 (2%)
2012							
Penicillin							
Resistant	6	6	13	20	24	2	47
Intermediate	3	1	9	39	52	4	85
Sensitive	10	25	72	183	876	31	1,257
Total tested	19	32	94	242	952	37	1,389
Total isolates with reduced susceptibility (%)	9 (47%)	7 (22%)	22 (23%)	59 (24%)	76 (8%)	6 (16%)	132 (10%)
Ceftriaxone/cefotaxime							
Resistant	1	2	3	3	4	2	9
Intermediate	0	4	4	7	7	0	11
Sensitive	13	23	76	213	845	23	1,222
Total tested	14	29	83	223	856	25	1,242
Total isolates with reduced susceptibility (%)	1 (7%)	6 (21%)	7 (8%)	10 (4%)	11 (1%)	2 (8%)	20 (2%)

* Susceptibility data are restricted to jurisdictions with completeness suitable for reporting, that is, greater than 50% completeness

Discussion

Following the 2005 introduction of the 7vPCV for all infants on the NIP, Australia achieved a significant reduction in the overall rate of IPD in the community. Whilst Australia has maintained lower rates of IPD since the introduction of 7vPCV, there has also been a small but gradual rate increase in the IPD rate, largely due to non-7vPCV serotypes. In 2011, Australia recorded its highest overall rate of IPD since 2005 which was largely driven by the increased number of cases caused by serotype 19A and a serotype 1 outbreak that occurred amongst the Indigenous populations of the Northern Territory, Western Australia and Queensland.[9,10] Other countries that have implemented a national 7vPCV program, such as the United Kingdom, the United States of America and Norway, have experienced a similar non-7vPCV serotype replacement pattern, with serotype 19A emerging as a dominant serotype, after the introduction of 7vPCV.[11–13]

In Australia from 2005 to 2011, the majority of all cases caused by non-7vPCV serotypes were due to serotypes 19A, 3 and 22F; and more recently serotypes 7F, 1 and 6C. A reduction in the number of notifications due to 19A, 1 and 6C was observed following the mid-2011 NIP schedule change from 7vPCV and 10vPCV to 13vPCV. However, notifications due to serotypes 7F, 3 and 22F continued to increase. Serotype 6C is not included in any of the registered vaccines in Australia but immunogenicity data suggests that immune responses to 6A, which is included in the 13vPCV, could provide cross protection against IPD due to serotype 6C.[14] Infections due to serotypes 3 and 7F continued to rise in 2012, despite their inclusion in the 13vPCV.

The impact of the 2011 NIP schedule change from 7vPCV and 10vPCV to 13vPCV for infants was most evident in children aged less than 5 years. In 2012, and following the introduction of the new vaccine, the rate of IPD due to 13vPCV (non-7vPCV) serotypes halved in this cohort, while the rate of IPD caused by 7vPCV serotypes remained stable. In 2011 and 2012, there were no deaths in children aged less than 5 years that were preventable by the 7vPCV. Four deaths were due to serotypes included in the 13vPCV but each of these cases occurred prior to the introduction of the 13vPCV or in a child too young for vaccination.

The reduction of the IPD rate in non-Indigenous children post the introduction of 13vPCV is largely due to the decline in disease caused by serotype 19A but despite this, serotype 19A remains the most frequently isolated cause of IPD in this cohort. Notifications in non-Indigenous children due to serotype 3 recorded a small increase despite its inclusion in the 13vPCV. Canada has observed a similar rise in serotype 3 notifications following the introduction of the 13vPCV and other studies have suggested that 13vPCV may be less effective in protecting against IPD due to serotype 3.[15–18]

The reduction of the IPD rate in Indigenous children post the introduction of 13vPCV is largely due to the tapering of the serotype 1 outbreak and to a lesser extent the decline in serotype 19A. The serotype 1 outbreak observed during this reporting period contributed to the large increase in IPD notifications recorded in Indigenous children in 2011, in particular, those in children aged between 2 and 5 years.[9,10] It is important to note that the Northern Territory was using a 4 dose 10vPCV vaccine schedule for infants for the period October 2009 to October 2011 and did not experience any cases of serotype 1 disease in 10vPCV vaccinated infants during the outbreak.

In both Indigenous and non-Indigenous adults eligible for the 23vPPV on the NIP, rates of IPD due to 23vPPV serotypes have increased in 2011 and 2012. The rate of IPD due to 23vPPV serotypes in Indigenous adults has shown a marked overall increase since 2006. In recent years, the serotype 1 outbreak has contributed to the rise in notifications. However, excluding the serotype 1 notifications, the Indigenous adult population is still experiencing an overall increase in IPD due to 23vPPV serotypes.

In 2008, a Cochrane review found that there is strong evidence to support the effectiveness of the 23vPPV against IPD. However, several studies included in this review as well as more recent publications, suggest that the protective effect could range from 40% to 80% in different populations.[19–21] The data described in this report with regards to the Indigenous population suggest that either the vaccine has only a moderate effect in this cohort and/or vaccine uptake in this cohort is less than optimal. The most recent data published on the uptake of NIP recommended vaccines in the Aboriginal and Torres Strait Islander population was collected and analysed a decade ago and reported pneumococcal vaccine coverage of only 34% in Indigenous adults aged 50 years or over and that coverage varied between jurisdictions.[22]

Other countries that have introduced the 13vPCV into a childhood vaccination schedule have also observed a reduction in IPD in the adult population within 3 years of the vaccine's introduction.[12,18,23] The observation period of this report constituted only 18 months of 13vPCV use in children and thus far the herd immunity effect on the adult population in Australia is not yet evident.

In 2011 and 2012, the proportion of cases with reduced susceptibility to penicillin returned to

levels seen prior to the slight increase reported in 2010. This was likely due to an overall reduction in cases caused by serotypes 19A following the introduction of the 13vPCV. In both 2011 and 2012, reduced susceptibility to ceftriaxone/cefotaxime remained uncommon in Australia. The proportion of isolates with reduced susceptibility to ceftriaxone/cefotaxime is similar to proportions described in both the United States of America and Asia.[24–26]

Post-immunisation surveillance of IPD in Australia is essential to monitor disease trends, to inform future control strategies, including the targeting of existing and new vaccines and the best options for antibiotic treatment.

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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 January 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, 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 CDNA.

The implementation date for the brucellosis case definition is 1 July 2016, while the implementation date for the flavivirus infection (unspecified) including Zika virus case definition is 1 January 2016. Both supersede any previous versions.

Brucellosis

Reporting

Both **confirmed** and **probable cases** should be notified.

Confirmed case

A confirmed case requires **laboratory definitive evidence** only.

Laboratory definitive evidence

1. Isolation of *Brucella* species

OR

2. Detection of *Brucella* species by nucleic acid testing from a blood sample

OR

3. IgG seroconversion or a significant increase in IgG antibody level (e.g. fourfold or greater rise) to *Brucella*.

Probable case

A probable case requires **laboratory suggestive** AND **clinical evidence.**

Laboratory suggestive evidence

1. A single high agglutination titre to *Brucella*

OR

2. Detection of *Brucella* species by nucleic acid testing from a normally sterile site other than blood.

Clinical evidence

A clinically compatible illness.

Summary of changes to brucellosis surveillance case definition	**Laboratory definitive evidence** Addition of 'detection of *Brucella* species by nucleic acid testing in a blood sample'. IgG seroconversion description re-worded. Removal of 'agglutination and complement fixation titres'. **Laboratory suggestive evidence** Addition of 'detection of *Brucella* species by nucleic acid testing from a sterile site other than blood'.

Flavivirus infection (unspecified) including Zika virus

Note:

It is recognised that some cases of human infection cannot be attributed to a single flavivirus. This may either be because the serology shows specific antibody to more than one virus, specific antibody cannot be assigned based on the tests available in Australian reference laboratories, or a flavivirus is detected that cannot be identified.

Confirmation by a second arbovirus reference laboratory is required if the case cannot be attributed to known flaviviruses.

Occasional human infections occur due to other known flaviviruses, such as Kokobera, Alfuy, Edge Hill and Stratford viruses.

Reporting

Only **confirmed cases** should be notified.

Confirmed case

A confirmed case requires laboratory **definitive evidence** AND **clinical evidence**.

Laboratory suggestive evidence

1. Isolation of a flavivirus that cannot be identified in Australian reference laboratories or which is identified as one of the flaviviruses not otherwise classified

OR

2. Detection of a flavivirus, by nucleic acid testing, that cannot be identified in Australian reference laboratories or which is identified as one of the flaviviruses not otherwise classified

OR

3. IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre of flavivirus specific IgG that cannot be identified or which is identified as being specific for one of the flaviviruses not otherwise classified. There must be no history of recent Japanese encephalitis or yellow fever vaccination

OR

4. Detection of flavivirus IgM in cerebrospinal fluid, with reactivity to more than one flavivirus antigen (Murray Valley encephalitis, Kunjin, Japanese encephalitis and/or dengue) or with reactivity only to one or more of the flaviviruses not otherwise classified

OR

5. Detection of flavivirus IgM in the serum, with reactivity to more than one flavivirus antigen (Murray Valley encephalitis, Kunjin, Japanese encephalitis and/or dengue) or with reactivity only to one or more of the flaviviruses not otherwise classified. This is only accepted as laboratory evidence for encephalitic illnesses. There must be no history of recent Japanese encephalitis or yellow fever vaccination.

Clinical evidence

1. Non-encephalitic disease: acute febrile illness with headache, myalgia and/or rash

OR

2. Encephalitic disease: acute febrile meningoencephalitis characterised by one or more of the following:
    - focal neurological disease or clearly impaired level of consciousness
    - an abnormal computerised tomograph or magnetic resonance image or electrocardiograph
    - presence of pleocytosis in cerebrospinal fluid.

Zika virus case definition

Confirmed and probable cases are nationally notifiable under the disease *Flavivirus infection (unspecified)* using the Organism Name field to specify infection with Zika virus (ZIKV).

Reporting

Both **confirmed** and **probable** cases are nationally notifiable. Both confirmed and probable cases should be further sub-classified into clinical and non-clinical cases.

Confirmed case

A confirmed case requires **laboratory definitive evidence** only. Clinical evidence should be used to sub-classify cases as clinical or non-clinical.

Laboratory definitive evidence

- Detection of ZIKV by nucleic acid testing or virus isolation

OR

- IgG seroconversion or a significant increase in antibody level or a fourfold or greater rise in titre of ZIKV-specific IgG, and recent infection by dengue or other epidemiologically possible flaviviruses has been excluded

OR

- Detection of ZIKV-specific IgM in cerebrospinal fluid, in the absence of IgM to other possible infecting flaviviruses.

Probable case

A probable case requires **laboratory suggestive evidence** AND **epidemiological evidence**. Clinical evidence should be used to sub-classify cases as clinical or non-clinical.

Laboratory suggestive evidence

Detection of ZIKV-specific IgM in the absence of IgM to other epidemiologically possible flaviruses or flavivirus vaccination in the 3 weeks prior to testing.

Notes:

1. If the date of most recent exposure was greater than 4 weeks before the specimen date, then ZIKV-specific IgG must also be positive.
2. If ZIKV-specific IgG was initially negative and subsequent testing greater than 4 weeks after exposure fails to demonstrate seroconversion the case should be rejected.

Epidemiological evidence

Clinical case

- Travel to or residence in a ZIKV receptive country[1] or area in Australia within two weeks prior to symptom onset;

OR

- Sexual exposure to a confirmed or probable case of ZIKV infection within two weeks prior to symptom onset.

Non-clinical case

- Travel to or residence in a ZIKV receptive country[1] or area in Australia within two months prior to specimen date;

OR

- Sexual exposure to a confirmed or probable case of ZIKV infection within two months prior to specimen date.

1. ZIKV receptive countries and areas are outlined on the Global Consensus Map at http://www.healthmap.org/dengue/en/. Areas are considered receptive to ZIKV where the likelihood of local acquisition is placed on the map as 'uncertain' or more.

Clinical case

Both confirmed and probable cases should be further sub-classified into **clinical** or **non-clinical** cases.

Clinical evidence

An acute illness within 2 weeks of exposure with 2 or more of the following symptoms:

- Fever
- Headache
- Myalgia
- Arthralgia
- Rash
- Non-purulent conjunctivitis.

In the absence of clinical evidence, the case will be classified as non-clinical.

Congenital Zika virus case definition

Confirmed and probable cases are nationally notifiable under the disease *Flavivirus infection (unspecified)* using the Organism Name field to specify congenital ZIKV infection.

Reporting

Both **confirmed** and **probable** cases are nationally notifiable.

Confirmed case

A confirmed case requires **laboratory definitive evidence** only.

Laboratory definitive evidence

Fetal (at 20 weeks gestation or more)

Isolation or detection of ZIKV from appropriate clinical samples (i.e. fetal blood, amniotic fluid, chorionic villus sample or post-mortem cerebrospinal fluid or tissue) by viral culture or nucleic acid testing.

Infant (within 28 days following birth)

Isolation or detection of ZIKV from appropriate clinical samples by viral culture or nucleic acid testing, with no history of travel since birth to, or residence in, a ZIKV receptive country[1] or area in Australia.

Probable case

A probable case requires **clinical evidence** AND **epidemiological evidence.**

Clinical evidence

Microcephaly[2–6] or other CNS abnormalities[7] in the infant or fetus (in the absence of any other known cause).

Epidemiological evidence

Confirmed or probable ZIKV infection in the mother during pregnancy.

2. Head circumference <-2SD below mean for gestation.
3. WHO Assessment of infants with microcephaly in the context of ZIKV. Interim guidance. 4 March 2016, WHO/ZIKV/MOC/16.3 Rev.1.
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5. WHO Pregnancy management in the context of ZIKV. Interim guidance. 2 March 2016. WHO/ZIKV/MOC/16.2
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7. These include: ventriculomegaly, calcifications, abnormal sulcation and gyration, brain atrophy, callosal dysgenesis, microophthalmia, eye calcifications.

Summary of changes to Flavivirus infection (unspecified) surveillance case definition	Addition of the Zika virus case definition Addition of the congenital Zika virus case definition

New surveillance case definition

The following new case definition has been developed by the Case Definitions Working Group and endorsed by the Communicable Diseases Network Australia. The implementation date is 1 July 2016.

Middle East Respiratory Syndrome Coronavirus (MERS-CoV)

Reporting

Confirmed and **probable cases** should be notified

Confirmed case

A confirmed case requires **laboratory definitive evidence**

Laboratory definitive evidence

Detection of MERS-CoV coronavirus by polymerase chain reaction (PCR) in a public health reference laboratory using the testing algorithm described in the national guideline (SoNG) and summarised below.[1]

Probable case

A probable case requires **clinical evidence** AND **epidemiological evidence**

Clinical evidence

An acute respiratory infection with clinical, radiological, or histopathological evidence of pulmonary parenchymal disease (e.g. pneumonia or pneumonitis or acute respiratory distress syndrome).

AND

No possibility of laboratory confirmation for MERS-CoV because the patient or samples are not available for testing.

Epidemiological evidence

Close contact with a laboratory-confirmed case

1. To consider a case as laboratory-confirmed, one of the following conditions must be met:
   - A positive PCR result for at least two different specific targets on the MERS-CoV genome.
   - One positive PCR result for a specific target on the MERS-CoV genome and an additional different PCR product sequenced, confirming identity to known sequences of MERS-CoV.

Quarterly report

OzFoodNet quarterly report, 1 April to 30 June 2014

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. In each Australian state and territory, OzFoodNet epidemiologists investigate outbreaks of enteric infection. 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 outbreaks of gastrointestinal illness and clusters of disease potentially related to food, which commenced in Australia between 1 April to 30 June 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 2nd quarter of 2014, OzFoodNet sites reported 419 outbreaks of enteric illness, including those transmitted by contaminated food or water. Outbreaks of gastroenteritis are often not reported to health agencies or the reports may be delayed, meaning that these figures under-represent the true burden of enteric disease outbreaks. In total, these outbreaks affected 7,052 people, 250 of whom were hospitalised, and 35 deaths were reported. This represents a decrease in the number affected and an increase in the number of deaths compared with the 5-year average for the 2nd quarter (7,600 affected, 234 hospitalised and 22 deaths). The majority of outbreaks (n=305) were due to person-to-person transmission (Table 1), with 55% (169 outbreaks) of these occurring in aged care facilities and 32% (97 outbreaks) occurring in child care facilities or schools.

Foodborne and suspected foodborne disease outbreaks

There were 39 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission (Appendix). These outbreaks affected 406 people and resulted in 53 hospitalisations. Two deaths were reported during these outbreaks, 1 associated with a *Listeria monocytogenes* outbreak among chemotherapy patients in a New South Wales hospital and the other with a *Salmonella* Typhimurium 135a outbreak in a Victorian hospital.

This was a decrease on the number of foodborne outbreaks that were reported in the 1st quarter of 2014 (n=49) and an increase on the 5-year mean for the 2nd quarter between 2009 and 2013 (n=32). A limitation of the outbreak data provided by OzFoodNet sites for this report was the potential for variation in the categorisation of the features of outbreaks depending on circumstances and investigator interpretation. Changes in the number of foodborne outbreaks should be interpreted with caution due to the small number each quarter.

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

Transmission mode	Number of outbreaks and clusters	Per cent of total*	Number ill
Foodborne and suspected foodborne	39	9	406
Suspected waterborne	0	0	0
Person-to-person	305	73	5,973
Unknown (*Salmonella* cluster)	22	5	247
Unknown (other pathogen cluster)	4	1	18
Unknown	49	12	408
Total	419	100	7,052

* Percentages do not add to 100 due to rounding.

S. Typhimurium was identified as or suspected to be the aetiological agent in 62% (24/39) of foodborne or suspected foodborne outbreaks during this quarter, a higher proportion than the number from the same quarter in 2013 (30%, 9/30). The aetiological agents for the remaining outbreaks included: norovirus and *S.* Infantis in 2 outbreaks each, *Amanita phalloides* (death cap mushrooms), ciguatoxin, hepatitis E virus (HEV), histamine poisoning, *Campylobacter,* and *L. monocytogenes* for 1 outbreak each. For 5 outbreaks the aetiological agent was unknown.

Sixteen outbreaks (41% of all the foodborne or suspected foodborne outbreaks) reported in this quarter were associated with food prepared in restaurants (Table 2), which is higher than the average number associated with foodborne or suspected foodborne outbreaks in the 2nd quarter from 2009 to 2013 (38%).

To investigate these outbreaks, sites conducted 3 cohort studies, 2 case control studies and collected descriptive case series data for 29 investigations, while for 5 outbreaks no individual patient data were collected. The evidence used to implicate food vehicles included analytical evidence in 4 outbreaks, microbiological evidence in 6 outbreaks, and descriptive evidence in 29 outbreaks.

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

Australian Capital Territory

There were 2 outbreaks of foodborne or suspected foodborne illness reported in the Australian Capital Territory during this quarter. The aetiological agents identified were *A. phalloides* and *S.* Typhimurium.

Description of key outbreak

An outbreak was investigated in April after 3 people with gastroenteritis reported eating a home-cooked meal containing mushrooms. The mushrooms were subsequently identified as *A. phalloides,* and 1 case was transferred to a New South Wales health care facility for ongoing clinical care. It was reported to authorities that the mushrooms had been purchased from a national supermarket chain. However, the investigation showed no evidence to support this. A 4th but unrelated New South Wales case was seen at an Australian Capital Territory hospital before transfer to an New South Wales health care facility for further care.

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 *S.* Typhimurium for 7 outbreaks and *L. monocytogenes,* norovirus, HEV and histamine poisoning for 1 outbreak each.

Description of key outbreaks

An outbreak was investigated in April after a single notification of HEV infection led to the identification of 5 cases among a group of 9 work colleagues. An interview of the 1st notified case found that a work colleague from Victoria also had HEV infection, and that the only common exposure for both cases was a restaurant dinner with 7 other work colleagues on 11 March. Further investigations included interviewing and serological testing of co-dining work colleagues, which identified a further 3 cases. Case interviews revealed that pork pâté was the only food consumed by all the cases. An additional

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

Food preparation setting	Outbreaks	Per cent of foodborne outbreaks	Number ill
Restaurant	16	41	132
Private residence	11	28	65
Bakery	4	10	77
Takeaway	3	8	39
Commercial caterer	1	3	6
Hospital	1	3	22
Institution	1	3	57
Primary produce	1	3	3
Unknown	1	3	5
Other	0	0	0
Total	39	100	406

10 infected individuals, unrelated to the work group, were also linked to this cluster. All 10 cases reported consuming pork pâté at the same restaurant on different dates to the work group (13 March, 15 March, 3 May and 15 May). The New South Wales Food Authority (NSWFA) inspected the restaurant on 2 occasions on 15 and 21 May 2014 and found it to be very well-run with no issues identified in food handling, cooking or cleaning. Pork samples from the restaurant were tested but all were negative for HEV. The pork pâté was made with pork livers and included only one short cooking step. It is suspected that on one or more occasions the pork livers had been inadvertently undercooked leading to the survival of HEV in the pâté. Trace back of the pork livers revealed that a single pig farm supplied the livers that were served as pork pâté on the days the cases reported eating at the restaurant.

In addition to the HEV cases above, 3 notifications of locally acquired HEV from 2013 with no known source of infection were re-investigated. Interviews revealed that 2 cases had also eaten pork pâté at the same restaurant during their incubation period (the 3rd case was thought to be person-to-person transmission). An additional case from October 2013, identified on retrospective testing of stored sera was also linked to the cluster. The viruses from 11 of the 18 cases linked to the restaurant (3 from 2013 and 8 from 2014) were genetically sequenced and were found to be closely related, suggesting a common source. Undercooked pork has been associated with cases of foodborne HEV overseas. An HEV expert panel was convened and it was concluded there was no ongoing public health risk associated with the restaurant.

An outbreak was investigated in April after 3 cases of *L. monocytogenes* were notified within a month. All 3 cases reported attending the same chemotherapy treatment facility in a 2-week period. *Listeria* isolates from the 3 cases had the same binary type (158), multi-locus variable number tandem repeat analysis (MLVA) profile (04-17-16-05-03-11-14-00-16), serotype (1/2b, 3b, 7) and pulsed-field gel electrophoresis (PFGE) type (4:4:5A). Two of the cases reported eating sandwiches on multiple visits to the facility but the 3rd case denied eating anything. The facility sourced its food from a café next door, which was inspected by the NSWFA, and while considered generally well run, a sample of cucumber tested positive for the outbreak strain. As the food provider was a public café, rather than a food provider for vulnerable populations, it was not required to be *Listeria* free. Although not all cases reported eating food provided from the café, the identical typing of the cases and the food isolate (a novel type), indicates that it is likely the case had eaten from the café but could not recall on interview. The café was advised of ways to help reduce the possibility of having *Listeria* on foods, and the chemotherapy facility was advised on the importance of food safety for vulnerable populations, with particular reference to *Listeria*.

Northern Territory

There were 5 outbreaks of suspected foodborne illness reported in the Northern Territory during this quarter. *S.* Typhimurium phage type (PT) 9 was identified as the aetiological agent for 1 outbreak and the remaining 4 outbreaks had unknown aetiology.

Description of key outbreak

An outbreak was investigated in April in response to reports of gastroenteritis among 9 people after a restaurant meal. Two cases tested positive for *S.* Typhimurium PT 9 infection. A cohort study was conducted and high risk foods identified included a ‘Surf and Turf’ dish (eaten by 8 of 9 cases) and the raw-egg Hollandaise sauce, which accompanied this, as well as another dish. The Hollandaise sauce was made on site and an environmental health inspection of the restaurant identified that improper storage of this sauce after preparation was a likely contributing factor to the outbreak. Food samples were taken from the kitchen but none tested positive for *Salmonella*.

Queensland

There were 7 outbreaks of foodborne or suspected foodborne illness reported in Queensland during this quarter. The aetiological agents were identified as *S.* Typhimurium for 4 outbreaks, and ciguatoxin and *Campylobacter* for 1 outbreak each. The remaining outbreak had an unknown aetiology.

Description of key outbreaks

An outbreak was investigated in April after reports of gastroenteritis among 57 of the at least 247 residents of a tertiary residential college. A total of 20 cases were laboratory confirmed with *S.* Typhimurium MLVA 03-09-07-12-524. All students residing at the college at the time of the outbreak were emailed a detailed food history questionnaire in an attempt to identify a common food vehicle. Seventy-seven questionnaires (31%) were returned for analysis. Results were inconclusive. Using a case-control methodology, 2 food items consumed had elevated odds ratios (OR) but were not statistically significant: chilli con carne (OR 2.6, 95% confidence interval (CI) 0.7 to 8.9) and chicken schnitzel (OR 3.3, 95% CI 0.7 to 16.0).

Multiple food samples including left-over ready-to-eat produce, eggs, egg products and poultry were collected from the college cafeteria for microbiological testing. However, all tested negative for *Salmonella*. The microbiological quality of some samples were likely to have been compromised as the left-over food was scheduled to be discarded and had been steamed to remove food residue from the chafing dish. The detection of *Bacillus cereus* and *Staphylococcus aureus* from environmental swabs taken during the investigation indicated inadequate cleaning and sanitising of contact surfaces within the kitchen environment. No vehicle or source of infection was identified during this investigation.

An outbreak of gastroenteritis was investigated in May among attendees of a luncheon that was held in the Wide Bay area of Queensland. Seven of 63 people who attended the event developed gastrointestinal symptoms within 4 days of attendance. Five cases were hospitalised and 4 cases were diagnosed with *Campylobacter* infection. The luncheon was catered by the various attendees and included a fixed menu consisting of roast beef, vegetables, gravy, apple crumble with custard, and jugs of tank water were also available for consumption. No remaining food samples were available for microbiological analysis, but samples collected from the rainwater tank were positive for *Campylobacter.* No genotyping was able to be performed to compare human and non-human isolates. The rainwater tanks were the suspected source of infection for these cases and have since been decommissioned by the local council.

South Australia

There were 2 outbreaks of foodborne or suspected foodborne illness reported in South Australia during this quarter. The aetiological agent was identified as *Salmonella* Typhimurium in both outbreaks.

Tasmania

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

Victoria

There were 8 outbreaks of foodborne or suspected foodborne illness reported in Victoria during this quarter. The aetiological agents were identified as *S.* Typhimurium for 7 outbreaks and norovirus for the remaining outbreak.

Description of key outbreak

An outbreak was investigated in May after 3 notifications of *S.* Typhimurium PT 170/108 MLVA 03-10-07-12-523 in the same family. All 5 family members had consumed a chicken meal and a chocolate mousse cake during their incubation period. Two weeks later a 2nd group of 5 people all reported being ill and 2 were confirmed with the outbreak strain after eating a chocolate mousse cake from the same bakery. Along with the members of this group, all notified cases of *S.* Typhimurium PT 170/108 in the suburbs surrounding the bakery were then interviewed. A total of 24 cases of illness, with 15 being confirmed as *S.* Typhimurium PT 170/108, were found to have consumed chocolate mousse cake from this bakery over a 4-week period. Council found that the premises made large batches of chocolate mousse and cakes and froze them for use over at least 4 weeks. Two samples of the cakes were positive for *S.* Typhimurium PT 170/108. The eggs used in the chocolate mousse were traced back to a distributor who purchased eggs from multiple farms.

Western Australia

There were 4 outbreaks of foodborne or suspected foodborne illness reported in Western Australia during this quarter. The aetiological agents were identified as *S.* Typhimurium in 2 outbreaks and *S.* Infantis for the remaining 2 outbreaks.

Description of key outbreak

An outbreak was investigated in May after 5 cases reported gastrointestinal symptoms from 3 different groups who ate at the same restaurant. Two cases, in 2 separate groups, were confirmed with *S.* Typhimurium PT 9, PFGE 0001 infection. A structured questionnaire was used to obtain specific information from the 21 people in 3 different groups. The data were analysed as a case control study and there was a statistical association between eating the lamb shanks meal (P value = 0.0003) and raw salad (P value = 0.0251) and becoming ill. There were no remaining food samples available for testing and surface swabs from the kitchen were negative for routine pathogens. No staff reported illness. Lamb shanks were cooked prior to the meal and stored in the cool room. Cases also reported that the lamb shanks were not hot when served. An inspection of the kitchen revealed that cross contamination between meats may have occurred in the cool room as uncovered cooked chicken was stored below raw meat that was defrosting.

Cluster investigations

During the quarter, OzFoodNet sites conducted investigations into 75 clusters of infection for which no common food vehicle or source of infection could be identified. Aetiological agents identified during the investigations included 14 *S.* Typhimurium clusters, 2 norovirus clusters, 2 *S.* Saintpaul clusters, 2 *S.* Wangata clusters and 1 cluster each of: *Escherichia coli* O157, *E. coli* O26, *S.* Mbandaka, *S.* Virchow, *C. jejuni,* and 1 mixed cluster of *Campylobacter, S.* Infantis and cryptosporidium.

Comments

A. phalloides poisoning is rare in Australia but causes severe gastrointestinal symptoms that can lead to death. This species resembles the edible straw mushroom (*Volvariella volvacea*), which commonly grows in Asia and may be a contributing factor in their consumption.[1] In 2012, 3 of 4 workers in a restaurant in the Australian Capital Territory became ill after a post-service staff meal containing *A. phalloides,* leading to 2 deaths.[2]

The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission, and in this quarter 73% of outbreaks (n=305) were transmitted via this route, which was slightly lower than for the same quarter in 2013 (n=315) and lower than the 5-year mean (2nd quarter 2009 to 2013) of 330 outbreaks.

S. Typhimurium was identified as the aetiological agent in 62% (24/39) of foodborne or suspected foodborne outbreaks during this quarter (Appendix). Of the 10 confirmed foodborne outbreaks for which an analytical and/or microbiological link to a food vehicle was established, 5 were due to *S.* Typhimurium and 3 of these were associated with the consumption of raw or minimally cooked egg dishes.

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Appendix: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites,* Australia, 1 April to 30 June 2014 (n=39)

State or territory	Month†	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
ACT	Apr	Private residence	*Amanita phalloides* (death cap mushrooms)	3	3	D	Curry containing mushrooms
ACT	Apr	Private residence	*Salmonella* Typhimurium PT 9 MLVA 03-10-14-12-498	2	2	D	Milkshake containing raw egg
NSW	Apr	Restaurant	Hepatitis E virus	7	2	A	Pork liver pate
NSW	Apr	Restaurant	*S.* Typhimurium MLVA 03-13-10-11-523	4	0	D	Unknown
NSW	Apr	Bakery	*S.* Typhimurium MLVA 03-17-10-11-523	33	7	M	Raw egg mayonnaise
NSW	Apr	Takeaway	*S.* Typhimurium MLVA 03-26-07-20-496	11	2	D	Raw egg salad dressing
NSW	Apr	Restaurant	*S.* Typhimurium MLVA 03-09-07-12-523	7	1	M	Multiple foods
NSW	May	Restaurant	*Listeria monocytogenes* Binary type 158 MLVA 04-17-16-05-03-11-14-00-16, serotype 1/2b, 3b, 7 and PFGE 4:4:5A	3	3	D	Unknown
NSW	May	Restaurant	Norovirus	6	0	D	Garden salad
NSW	May	Takeaway	*S.* Typhimurium MLVA 03-10-07-12-523	11	1	D	Vietnamese rolls raw egg
NSW	Jun	Private residence	*S.* Typhimurium MLVA 03-24-12-10-523 (9) and 03-24-13-10-523 (1)	13	0	D	Tiramisu with raw egg
NSW	Jun	Restaurant	*S.* Typhimurium MLVA 03-12-12-09-523	9	1	D	Unknown
NSW	Jun	Private residence	Histamine poisoning	2	2	D	Tuna steaks
NT	Apr	Restaurant	*S.* Typhimurium PT 9	9	3	A	Hollandaise sauce containing raw eggs
NT	Apr	Restaurant	Unknown	5	0	D	Unknown
NT	Apr	Restaurant	Unknown	2	0	D	Unknown
NT	May	Private residence	Unknown	3	0	D	Unknown
NT	Jun	Restaurant	Unknown	2	0	D	Unknown
Qld	Apr	Primary produce	Ciguatoxin	3	0	M	Spanish mackerel
Qld	Apr	Restaurant	*S.* Typhimurium MLVA 03-12-13-09-524	3	2	D	Suspected egg and lettuce sandwiches
Qld	Apr	Bakery	*S.* Typhimurium MLVA 03-09-07-11-524	8	0	D	Various bakery products (custard buns)
Qld	Apr	Institution	*S.* Typhimurium MLVA 03-09-07-12-524	57	4	D	Unknown
Qld	Apr	Unknown	*S.* Typhimurium MLVA 03-09-07-12-524	5	0	D	Unknown
Qld	Apr	Restaurant	Unknown	2	0	D	Suspected raw oysters
Qld	May	Private residence	*Campylobacter*	7	5	M	Tank water
SA	Apr	Bakery	*S.* Typhimuirum PT 170/108	12	3	D	Raw egg butter on Vietnamese roll

Appendix *continued*: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites,* Australia, 1 April to 30 June 2014 (n=39)

State or territory	Month†	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
SA	Apr	Takeaway	*S.* Typhimurium PT 170/108	17	2	D	Raw egg mayonnaise on Vietnamese roll
Vic.	Apr	Private residence	*S.* Typhimurium PT 135a	13	1	D	Tiramisu
Vic.	Apr	Private residence	*S.* Typhimurium PT 135a	4	1	D	Uncooked brownie batter
Vic.	May	Restaurant	Norovirus genogroup II (GII)	45	Unknown	A	Probable food handler
Vic.	May	Private residence	*S.* Typhimurium PT 135a	6	5	D	Raw egg chocolate mousse
Vic.	May	Bakery	*S.* Typhimurium PT 170/108	24	1	M	Raw egg chocolate mousse
Vic.	May	Restaurant	*S.* Typhimurium PT 9	21	Unknown	D	Raw egg aioli/mayonnaise
Vic.	Jun	Hospital	*S.* Typhimurium PT 135a	22	1	D	Unknown
Vic.	Jun	Private residence	*S.* Typhimurium PT 99	2	0	D	Raw egg chocolate mousse
WA	Apr	Commercial caterer	*S.* Infantis	6	0	D	Nasi-Lemak
WA	Apr	Restaurant	*S.* Infantis	2	0	D	Unknown
WA	Apr	Private residence	*S.* Typhimurium PT 135 PFGE 0003	10	1	D	Unknown
WA	Apr	Restaurant	*S.* Typhimurium PT 9 PFGE 0001	5	0	A	Lamb shanks or salad
Total				406	53		

* No foodborne outbreaks were reported in Tasmania during the quarter

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

The number of people affected and hospitalised relate to the findings of the outbreak investigation at the time of writing and not necessarily in the month specified or in this quarter

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 profile Multi-locus variable number tandem repeat analysis profile

PFGE type Pulsed-field gel electrophoresis type

National Notifiable Diseases Surveillance System, 1 January to 31 March 2016

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 67,031 notifications to the National Notifiable Diseases Surveillance System (NNDSS) between 1 January to 31 March 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 except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
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	
Chlamydia	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis - congenital	All jurisdictions
Syphilis <2 years duration	All jurisdictions

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

Disease	Data received from:
Syphilis >2 years or unspecified duration	All jurisdictions
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
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 January to 31 March 2016, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2016	Total 4th quarter 2015	Total 1st quarter 2015	Last 5 years mean 1st 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
Hepatitis B (newly acquired)†	1	5	0	14	2	1	14	6	43	28	40	49.2	0.9	43	49.2
Hepatitis B (unspecified)‡	20	610	20	241	81	5	490	124	1,591	1,512	1,583	1,588.4	1	1,591	1,588.4
Hepatitis C (newly acquired)†	4	7	0	0	11	8	25	30	85	100	108	114.4	0.7	85	114.4
Hepatitis C (unspecified)‡	50	954	63	757	134	51	690	248	2,947	2,741	2,569	2,485.2	1.2	2,947	2,485.2
Hepatitis D	0	4	0	4	3	0	4	0	15	3	11	12.4	1.2	15	12.4
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	1	1	0.6	0	0	0.6
Campylobacteriosis	154	NN	123	1,957	521	361	2,071	730	5,917	6,411	5,540	4,702.2	1.3	5,917	4,702.2
Cryptosporidiosis	11	399	126	1,195	237	7	241	89	2,305	1,092	1,504	1,235.4	1.9	2,305	1,235.4
Haemolytic uraemic syndrome	0	2	0	1	0	0	1	0	4	4	4	5.6	0.7	4	5.6
Hepatitis A	1	16	0	16	2	0	14	6	55	33	88	66.2	0.8	55	66.2
Hepatitis E	1	6	0	2	2	0	4	0	15	16	11	12.8	1.2	15	12.8
Listeriosis	0	12	0	3	1	0	10	1	27	22	16	22.4	1.2	27	22.4
Paratyphoid	0	8	1	4	2	1	10	8	34	22	26	28.0	1.2	34	28.0
STEC§	0	11	0	13	24	0	6	6	60	51	30	33.4	1.8	60	33.4
Salmonellosis	83	1,643	153	1,959	478	109	1,469	638	6,532	3,833	6,413	4,866.4	1.3	6,532	4,866.4
Shigellosis	2	76	76	42	35	1	127	29	388	231	333	227.2	1.7	388	227.2
Typhoid fever	0	17	1	5	2	1	9	2	37	24	43	54.0	0.7	37	54.0
Quarantinable diseases															
Avian influenza in humans	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	1	0	0.4	0	0	0.4
Middle East respiratory syndrome coronavirus	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
Rabies	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
Smallpox	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
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2016, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2016	Total 4th quarter 2015	Total 1st quarter 2015	Last 5 years mean 1st quarter	Ratio	Year to date 2016	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Sexually transmissible infections															
Chlamydia[‖,¶]	390	6,444	716	5,897	1,451	482	138	3,088	18,606	16,230	22,574	22,065.6	0.8	18,606	22,065.6
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0	0.2
Gonococcal infection[¶]	49	1,592	510	854	300	15	1,520	752	5,592	4,639	4,708	3,874.4	1.4	5,592	3,874.4
Syphilis – congenital	0	0	0	0	0	0	0	0	0	0	0	0.8	0	0	0.8
Syphilis < 2 years duration[¶]	4	177	43	188	17	0	231	84	744	680	619	446.2	1.7	744	446.2
Syphilis > 2 years or unspecified duration[‡,¶]	9	143	16	77	14	1	208	13	481	467	500	404.4	1.2	481	404.4
Vaccine preventable diseases															
Diphtheria	0	0	0	1	0	0	0	0	1	0	1	0.4	2.5	1	0.4
Haemophilus influenzae type b	0	1	1	1	0	0	0	1	4	2	2	2.8	1.4	4	2.8
Influenza (laboratory confirmed)	60	1,648	76	2,280	430	48	676	517	5,735	5,945	4,561	2,929.4	2	5,735	2,929.4
Measles	0	8	0	4	0	0	25	0	37	9	27	60.0	0.6	37	60.0
Mumps	0	5	9	11	2	2	15	284	328	280	60	54.6	6	328	54.6
Pertussis	77	3,425	12	790	274	7	827	419	5,831	8,413	4,097	5,596.8	1	5,831	5,596.8
Pneumococcal disease – invasive	2	65	7	28	12	6	45	20	185	301	194	216.8	0.9	185	216.8
Poliovirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Rubella	0	1	0	1	0	0	2	0	4	5	5	9.4	0.4	4	9.4
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Tetanus	0	0	0	1	0	0	1	1	3	2	0	1.2	2.5	3	1.2
Varicella zoster (chickenpox)	23	NN	63	62	98	20	215	108	589	706	536	438.4	1.3	589	438.4
Varicella zoster (shingles)	50	NN	88	14	515	70	593	423	1,753	1,633	1,636	1,273.0	1.4	1,753	1,273.0
Varicella zoster (unspecified)	40	NN	2	1,823	61	40	1,443	410	3,819	3,543	3,070	2,582.0	1.5	3,819	2,582.0
Vectorborne diseases															
Barmah Forest virus infection	0	9	7	118	2	0	0	4	140	86	269	663.0	0.2	140	663.0
Chikungunya virus infection	0	5	0	2	1	1	7	1	17	10	55	25.4	0.7	17	25.4
Dengue virus infection	13	147	31	149	42	3	136	195	716	282	732	588.8	1.2	716	588.8
Flavivirus infection (unspecified)	0	13	0	17	2	0	6	7	45	3	4	4.2	10.7	45	4.2
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	1	2	0.6	0	0	0.6
Kunjin virus infection	0	0	0	0	0	0	0	0	0	1	0	0.0	0	0	0.0
Malaria	5	8	5	22	4	1	9	17	71	59	65	97.6	0.7	71	97.6
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	1	2.0	0	0	2.0
Ross River virus infection	4	181	94	1,163	34	3	40	117	1,636	1,030	5,682	2,796.4	0.6	1,636	2,796.4

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2016, by date of diagnosis

Disease	State or territory								Total 1st quarter 2016	Total 4th quarter 2015	Total 1st quarter 2015	Last 5 years mean 1st 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
Australian bat lyssavirus infection	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0	0.2
Brucellosis	0	1	0	1	0	0	0	0	2	2	4	6.6	0.3	2	6.6
Leptospirosis	0	3	0	43	0	0	3	1	50	17	23	46.4	1.1	50	46.4
Lyssavirus infection (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Ornithosis	0	2	0	0	0	0	1	0	3	9	2	11.0	0.3	3	11.0
Q fever	0	60	0	64	7	0	5	4	140	131	161	118.2	1.2	140	118.2
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0	0.2
Other bacterial infections															
Legionellosis	0	33	0	14	5	2	18	8	80	84	92	91.0	0.9	80	91.0
Leprosy	0	1	1	0	0	1	0	1	4	4	1	1.4	2.9	4	1.4
Meningococcal infection – invasive**	0	13	0	11	7	2	10	2	45	39	27	36.6	1.2	45	36.6
Tuberculosis	8	123	3	45	23	3	76	34	315	341	285	313.0	1	315	313.0
Total	1,061	17,878	2,247	19,894	4,836	1,252	11,435	8,428	67,031	61,079	68,315			67,031	60,263

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

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

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

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

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

¶ 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 January to 31 March 2016, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)‡	1.0	0.3	0.0	1.1	0.5	0.8	0.9	0.9	0.7
Hepatitis B (unspecified)§	19.7	31.7	31.6	19.4	18.8	3.8	32.5	18.0	26.1
Hepatitis C (newly acquired)‡	3.9	0.4	0.0	0.0	2.5	6.1	1.7	4.4	1.4
Hepatitis C (unspecified)§	49.3	49.6	99.6	61.1	31.0	39.1	45.7	36.0	48.4
Hepatitis D	0.0	0.2	0.0	0.3	0.7	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	151.8	NN	194.4	157.8	120.7	276.6	137.2	106.0	142.0
Cryptosporidiosis	10.8	20.7	199.2	96.4	54.9	5.4	16.0	12.9	37.8
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Hepatitis A	1.0	0.8	0.0	1.3	0.5	0.0	0.9	0.9	0.9
Hepatitis E	1.0	0.3	0.0	0.2	0.5	0.0	0.3	0.0	0.2
Listeriosis	0.0	0.6	0.0	0.2	0.2	0.0	0.7	0.1	0.4
Paratyphoid	0.0	0.4	1.6	0.3	0.5	0.8	0.7	1.2	0.6
STEC‖	0.0	0.6	0.0	1.0	5.6	0.0	0.4	0.9	1.0
Salmonellosis	81.8	85.4	241.8	158.0	110.7	83.5	97.3	92.6	107.3
Shigellosis	2.0	4.0	120.1	3.4	8.1	0.8	8.4	4.2	6.4
Typhoid fever	0.0	0.9	1.6	0.4	0.5	0.8	0.6	0.3	0.6
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									
Chlamydia¶,**	384.4	335.0	1,131.7	475.6	336.1	369.3	9.1	448.2	305.5
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	48.3	82.8	806.1	68.9	69.5	11.5	100.7	109.1	91.8
Syphilis – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years duration**	3.9	9.2	68.0	15.2	3.9	0.0	15.3	12.2	12.2
Syphilis > 2 years or unspecified duration§,**	8.9	7.4	25.3	6.2	3.2	0.8	13.8	1.9	7.9
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	1.6	0.1	0.0	0.0	0.0	0.1	0.1
Influenza (laboratory confirmed)	59.1	85.7	120.1	183.9	99.6	36.8	44.8	75.0	94.2
Measles	0.0	0.4	0.0	0.3	0.0	0.0	1.7	0.0	0.6
Mumps	0.0	0.3	14.2	0.9	0.5	1.5	1.0	41.2	5.4
Pertussis	75.9	178.1	19.0	63.7	63.5	5.4	54.8	60.8	95.7
Pneumococcal disease – invasive	2.0	3.4	11.1	2.3	2.8	4.6	3.0	2.9	3.0
Poliovirus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 *continued*: Notification rates of diseases, 1 January to 31 March 2016, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Vaccine preventable diseases, *cont'd*									
Rubella	0.0	0.1	0.0	0.1	0.0	0.0	0.1	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	0.1	0.0	0.0	0.1	0.1	0.0
Varicella zoster (chickenpox)	22.7	NN	99.6	5.0	22.7	15.3	14.2	15.7	14.1
Varicella zoster (shingles)	49.3	NN	139.1	1.1	119.3	53.6	39.3	61.4	42.1
Varicella zoster (unspecified)	39.4	NN	3.2	147.0	14.1	30.6	95.6	59.5	91.7
Vectorborne diseases									
Barmah Forest virus infection	0.0	0.5	11.1	9.5	0.5	0.0	0.0	0.6	2.3
Chikungunya virus infection	0.0	0.3	0.0	0.2	0.2	0.8	0.5	0.1	0.3
Dengue virus infection	12.8	7.6	49.0	12.0	9.7	2.3	9.0	28.3	11.8
Flavivirus infection (unspecified)	0.0	0.7	0.0	1.4	0.5	0.0	0.4	1.0	0.7
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	4.9	0.4	7.9	1.8	0.9	0.8	0.6	2.5	1.2
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	3.9	9.4	148.6	93.8	7.9	2.3	2.6	17.0	26.9
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	0.1	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Leptospirosis	0.0	0.2	0.0	3.5	0.0	0.0	0.2	0.1	0.8
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	0.1	0.0	0.0
Q fever	0.0	3.1	0.0	5.2	1.6	0.0	0.3	0.6	2.3
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	0.0	1.7	0.0	1.1	1.2	1.5	1.2	1.2	1.3
Leprosy	0.0	0.1	1.6	0.0	0.0	0.8	0.0	0.1	0.1
Meningococcal infection – invasive††	0.0	0.7	0.0	0.9	1.6	1.5	0.7	0.3	0.7
Tuberculosis	7.9	6.4	4.7	3.6	5.3	2.3	5.0	4.9	5.2

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

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

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

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

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

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

** 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 October to 30 September cohort, assessed as at 31 December 2015

Alexandra 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, for four 3-month birth cohorts of children assessed at the stated ages between 1 October 2014 and 30 September 2015 using ACIR data up to 31 December 2015. '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 3 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 *Commun Dis Intell* 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 0.6 of a percentage point to 92.3% (Table 1). All jurisdictions experienced small increases in the percentage of children 'fully immunised' by 12 months of age. For individual vaccines due by 12 months of age all jurisdictions achieved coverage greater than 92%.

The rolling annualised percentage of all children 'fully immunised' by 24 months of age for Australia increased for the first time in 4 consecutive reports by 0.8 percentage points to 89.3% (Table 2). All jurisdictions experienced increases in fully immunised coverage for this age group.

Table 1. Percentage of children immunised at 12 months of age for the birth cohort 1 October 2013 to 30 September 2014, preliminary results, by disease and state or territory; assessment date 31 December 2015

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	5,751	99,099	3,705	62,810	20,192	5,934	76,660	34,392	308,543
Diphtheria, tetanus, pertussis (%)	94.8	93.0	93.1	93.0	93.1	92.8	93.1	92.9	93.1
Poliomyelitis (%)	94.8	93.0	93.1	93.0	93.0	92.7	93.1	92.9	93.0
Haemophilus influenzae type b (%)	94.4	92.8	93.0	92.9	92.9	92.8	92.8	92.7	92.9
Hepatitis B (%)	94.4	92.8	93.4	92.8	92.9	92.7	92.8	92.6	92.8
Pneumococcal	94.5	92.7	93.1	92.7	92.8	92.8	92.7	92.5	92.7
Fully immunised (%)	93.8	92.2	92.6	92.4	92.4	92.4	92.2	92.1	92.3

Coverage for individual vaccines due by 24 months was above 90% in all jurisdictions. Coverage for varicella increased by 0.3 of a percentage point to 91.8% and for MMR by 0.6 of a percentage point to 91.2%. This is the second time that a full 12 month wide birth cohort (4 quarters) has been used to assess coverage at 24 months of age for the MMRV vaccine due at 18 months of age (the 1st dose of varicella and the 2nd dose of MMR).

The rolling annualised percentage of all children 'fully immunised' by 60 months of age for Australia increased marginally from the previous report by 0.3 of a percentage point to 92.6% (Table 3). This marginal increase in fully immunised coverage at 60 months of age occurred in all jurisdictions. Coverage for individual vaccines due by 60 months was greater than 91% 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. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007). From September 2014, coverage at 24 months is lower than coverage at 12 and 60 months of age. This is most likely due to the change in the 24 month coverage assessment algorithm as described above. Coverage estimates of fully vaccinated children by 12 months and by 24 months have remained stable since June 2015. There has been a marginal increase in coverage of fully vaccinated children by 60 months from 92.6% to 93.2%.

Figure: Trends in vaccination coverage, Australia, 1997 to 30 September 2015, by age cohorts

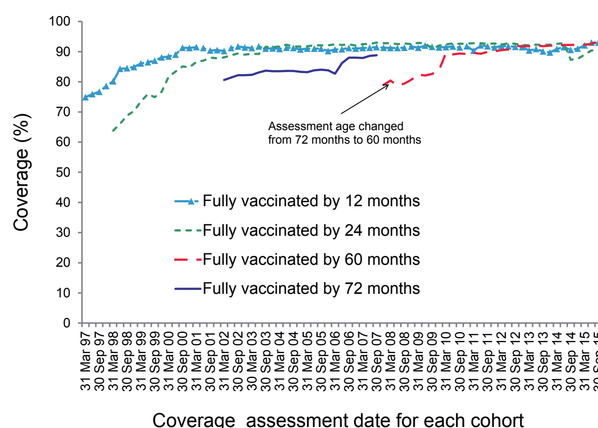


Table 2. Percentage of children immunised at 24 months of age for the birth cohort 1 October 2012 to 30 September 2013, preliminary results, by disease and state or territory; assessment date 31 December 2015

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	5,602	99,789	3,566	62,777	19,953	5,957	77,115	34,200	308,959
Diphtheria, tetanus, pertussis (%)	96.6	95.3	94.9	95.3	95.3	95.6	95.9	95.3	95.5
Poliomyelitis (%)	96.5	95.3	94.9	95.3	95.3	95.5	95.8	95.2	95.4
Haemophilus influenzae type b (%)	95.6	94.2	93.9	94.5	94.3	94.2	94.8	94.2	94.5
Measles, mumps, rubella (%)	92.6	91.0	90.4	91.6	90.8	90.8	91.5	90.2	91.2
Hepatitis B (%)	96.2	95.0	95.1	95.1	94.9	95.3	95.6	94.8	95.2
Meningococcal C (%)	95.0	94.1	94.0	94.5	93.4	94.3	94.4	93.3	94.2
Varicella (%)	93.8	91.7	89.7	91.8	91.4	90.8	92.3	91.0	91.8
Fully immunised (%)	91.0	89.1	87.7	90.2	88.3	88.3	89.7	88.1	89.3

Table 3. Percentage of children immunised at 60 months of age for the birth cohort 1 October 2009 to 30 September 2010, preliminary results, by disease and state or territory; assessment date 31 December 2015

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	5,573	101,773	3,467	65,450	20,244	6,196	76,782	34,460	313,945
Diphtheria, tetanus, pertussis (%)	94.1	93.6	93.5	93.0	91.9	93.9	93.6	91.7	93.2
Poliomyelitis (%)	94.1	93.7	93.5	93.0	91.9	93.9	93.5	91.7	93.2
Measles, mumps, rubella (%)	94.0	93.5	93.7	92.9	91.9	93.8	93.6	91.7	93.1
Fully immunised (%)	93.5	93.0	92.8	92.4	91.2	93.3	93.0	91.1	92.6

Australian Sentinel Practices Research Network, 1 January to 31 March 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, ASPREN practitioners are instructed to swab 20% of all patients presenting with an ILI.

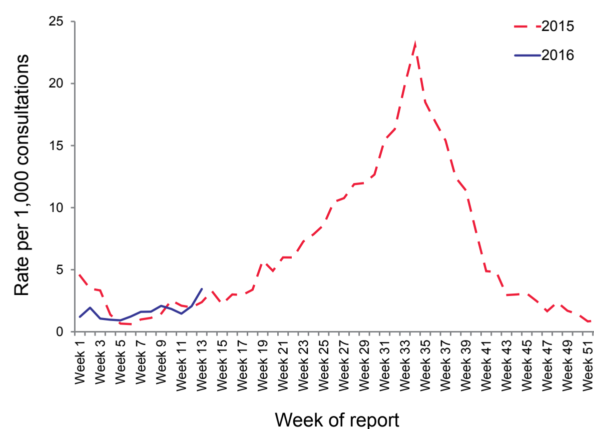
The list of conditions reported is reviewed annually by the ASPREN management committee. In 2016, 4 conditions are being monitored. They are ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in surveillance systems reported in CDI, published in *Commun Dis Intell* 2016;40(1):11.

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 1st quarter of 2016. Each week an average of 222 general practitioners provided information to ASPREN at an average of 15,970 (range 12,767 to 17,970) consultations per week and an average of 110 (range 82 to 134) notifications per week (all conditions).

ILI rates reported from 1 January to 31 March 2016 averaged 1.7 cases per 1,000 consultations (range 0.9 to 3.5 cases per 1,000 consultations). This was lower than the rates in the same reporting period in 2015, which averaged 2.0 cases per 1,000 consultations (range 0.6 to 4.5 cases per 1,000 consultations, Figure 1). ILI rates peaked in week 13 at a rate of 3.5 ILI cases per 1,000 consultations.

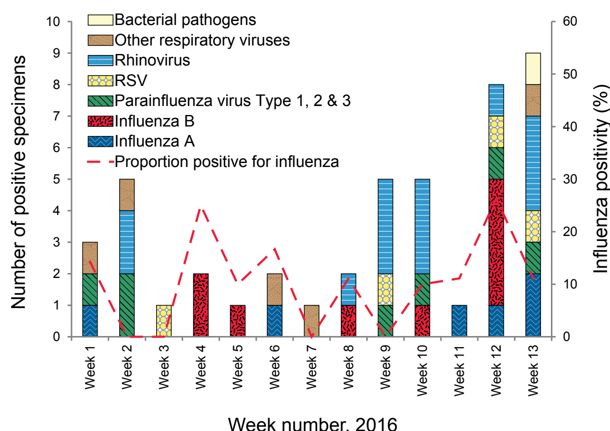
Figure 1: Consultation rates for influenza-like illness, ASPREN, 2015 and 1 January to 31 March 2016, by week of report



The ASPREN ILI swab testing program continued in 2016 with 127 tests being undertaken from 1 January to 31 March. The most commonly reported virus during this reporting period was rhinovirus (10.2% of all swabs performed, Figure 2), with the second most common virus being influenza B (7.1% of all swabs performed).

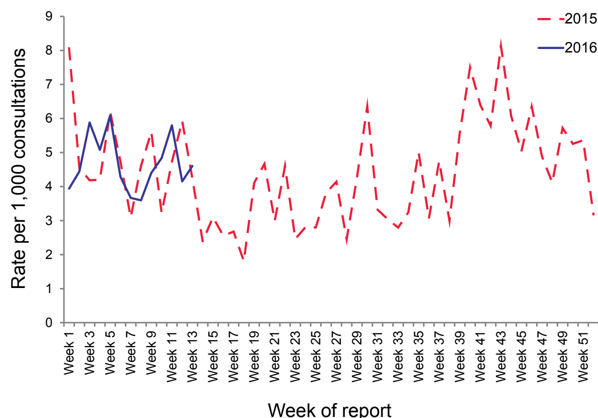
From the beginning of 2016 to the end of week 13, 15 cases of influenza were detected with 9 of these typed as influenza B (7.1% of all swabs performed) and the remaining 6 being influenza A (4.7% of all swabs performed) (Figure 2).

Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January to 31 March 2016, by week of report



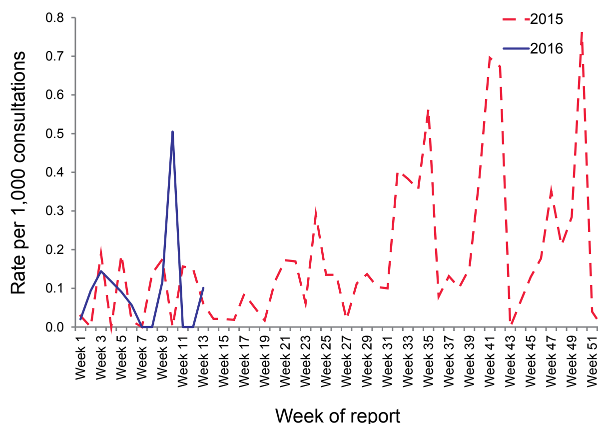
During this reporting period, consultation rates for gastroenteritis averaged 4.7 cases per 1,000 consultations (range 3.9 to 6.1 cases per 1,000, Figure 3). This was similar to the rates in the same reporting period in 2015 where the average was 4.9 cases per 1,000 consultations (range 3.1 to 8.1 cases per 1,000).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 2015 and 1 January to 31 March 2016, by week of report



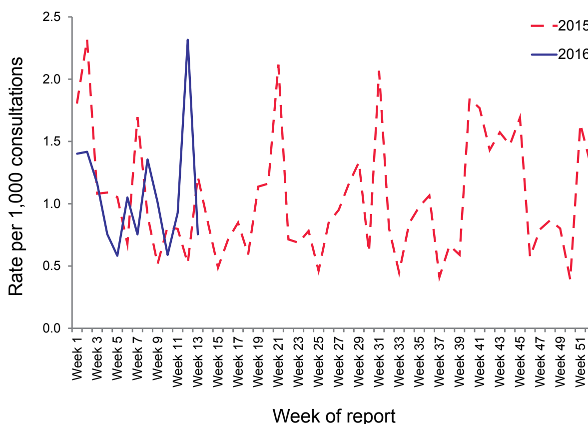
Varicella infections were reported at a higher rate for the 1st quarter of 2016 compared with the same period in 2015. From 1 January to 31 March 2016, recorded rates for chickenpox averaged 0.1 cases per 1,000 consultations (range 0.0 to 0.5 cases per 1,000 consultations, Figure 4).

Figure 4: Consultation rates for chickenpox, ASPREN, 2015 and 1 January to 31 March 2016, by week of report



In the 1st quarter of 2016, reported rates for shingles averaged 1.1 cases per 1,000 consultations (range 0.6 to 2.3 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 1.1 cases per 1,000 consultations (range 0.5 to 2.3 cases per 1,000 consultations).

Figure 5: Consultation rates for shingles, ASPREN, 2015 and 1 January to 31 March 2016, by week of report



Invasive pneumococcal disease surveillance, 1 January to 31 March 2016

Kate Pennington, Anna Glynn-Robinson, 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 1st quarter of 2016 was substantially fewer than the previous quarter and marginally less than the number of notified cases in the 1st 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 1st quarter of 2016, there were 184 cases of IPD reported to the NNDSS, an almost 6% decrease compared with the same period in 2015 (n = 195) (Table 1). In 2015 the most common pneumococcal serotypes causing IPD were 3 (8.8%), 19A (8.5%) and 22F (7.1%). The trend for this quarter was similar; however serotype 19F (6.0%; n = 11) was also common and has been gradually increasing (Table 2).

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. In Indigenous Australians, cases were highest in the under 5 years age group and the 55–59 years age group (Table 3). The proportion of cases reported as Indigenous in this quarter (14%) was less than the proportion observed in the 1st quarter of 2015 (17%; 33/195), but was similar to the proportion overall in 2015 (14%; 208/1,500).

There were 34 cases of IPD reported in children aged less than 5 years, representing 18% of all cases reported this quarter. The number of cases notified in this age group was 9.7% more in this reporting period compared with the 1st quarter of 2015 (n = 31). Of those cases with known serotype, 32% (n = 8) were due to a serotype included in the 13vPCV compared with 42% (n = 12) of cases in the 1st quarter of 2015 (Figure 1). Serotypes 19F, 16F and 23B were the most common serotypes affecting this age group this quarter, noting that serotype 19F is included in the 13vPCV (Table 2).

In the 1st quarter of 2016, there were 6 cases reported in fully vaccinated children aged less than 5 years who were considered to be 13vPCV failures. Serotype 19F was reported as the cause of disease in 4 of these cases (Table 4).

Table 1: Notified cases of invasive pneumococcal disease, Australia, 1 January to 31 March 2016, by Indigenous status, serotype completeness and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total 1st qtr 2016	Total 4th qtr 2015	Total 1st qtr 2015
Indigenous	0	1	5	6	2	0	1	11	26	52	33
Non-Indigenous	2	53	2	19	10	6	36	9	137	233	141
Not stated / Unknown	0	11	0	3	0	0	7	0	21	29	21
Total	2	65	7	28	12	6	44	20	184	314	195
Indigenous status completeness* (%)	100	83	100	89	100	100	84	100	91	91	89
Serotype completeness† (%)	100	77	100	93	75	100	95	100	96	96	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-typable. Serotype incompleteness may include where 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: Frequently notified serotypes of invasive pneumococcal disease, Australia, 1 January to 31 March 2016, by age group

Serotype	Age group			Serotype total
	Under 5 years	5–64 years	Over 65 years	
3	1	7	8	16
19A	1	6	5	12
19F	4	4	3	11
22F	1	5	5	11
8	0	7	3	10
16F	3	1	5	9
23B	3	4	2	9
15A	1	3	4	8
9N	0	5	3	8
10A	0	4	1	5
23A	0	3	2	5
38	1	3	1	5
6C	0	3	2	5
Other*	10	27	11	48
Serotype unknown†	9	11	2	22
Total	34	93	57	184

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

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

Table 3: Notified cases of invasive pneumococcal disease, Australia, 1 January to 31 March 2016, by Indigenous status and age group

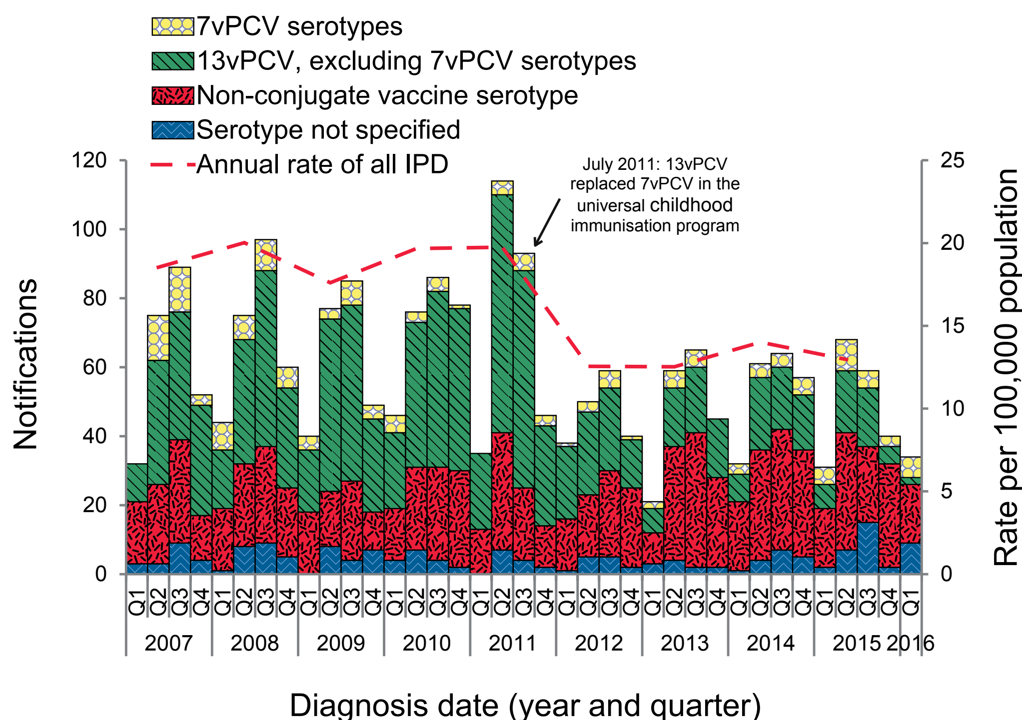
Age group	Indigenous	Non-Indigenous	Not reported*	Total
0-4	4	28	2	34
5-9	0	1	3	4
10-14	0	5	0	5
15-19	0	0	0	0
20-24	2	2	0	4
25-29	2	5	3	10
30-34	1	2	1	4
35-39	2	3	3	8
40-44	2	3	2	7
45-49	1	3	3	7
50-54	4	5	1	10
55-59	5	9	0	14
60-64	1	17	2	20
65-69	1	12	0	13
70-74	0	9	1	10
75-79	0	7	0	7
80-84	0	13	0	13
85+	1	13	0	14
Total	26 (14%)	137 (74%)	21 (11%)	184

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

There were 12 cases of IPD reported among Indigenous Australians aged 50 years or over in the 1st quarter of 2016. Of those cases with a reported serotype, half (n = 6) were due to a serotype included in the 23vPPV (Figure 2). The number of notified cases of IPD among Indigenous Australians aged 50 years or over was just over half (55%) the number reported in the previous quarter (n = 22) and similar to the 1st quarter of 2015 (n = 10). Compared with the previous quarter, the proportion of cases due to serotypes included in the 23vPPV decreased from 75% to 50% among cases with a known serotype. Additionally, there was no apparent predominance of any serotype reported among this population group this quarter.

There were 54 cases of IPD among non-Indigenous Australians aged 65 years or over reported in the 1st quarter of 2016. The number of notified cases of IPD among non-Indigenous Australians aged 65 years or over was around half the number reported in the previous quarter (n = 111) and 11% lower than the 1st quarter of 2015 (n = 61). Of those cases with a reported serotype, 62% (n = 32) were due to a serotype included in the 23vPPV (Figure 3). Compared with the previous quarter, the proportion of IPD due to 23vPPV serotypes among cases with a reported serotype were relatively similar.

Figure 1: Notifications and annual rates* of invasive pneumococcal disease in children aged less than 5 years, Australia, 2007 to 31 March 2016, by vaccine serotype group



* Annual rates are shown on Q2, excluding 2016.

Table 4: Characteristics of 13vPCV failures in children aged less than 5 years, Australia, 1 January to 31 March 2016

Age	Indigenous status	Serotype	Clinical category	Risk factor/s
11 months	Non-Indigenous	19F	Pneumonia	No data available
1 year	Non-Indigenous	19A	Pneumonia and other (pleural empyema)	Childcare attendee and other
2 years	Non-Indigenous	19F	Bacteraemia	Chronic illness
2 years	Non-Indigenous	19F	Pneumonia	Congenital or chromosomal abnormality
4 years	Indigenous	23F	Pneumonia	Previous episode of IPD
4 years	Non-Indigenous	19F	Pneumonia	Childcare attendee

In the 1st quarter of 2016 there were 9 deaths attributed to a variety of IPD serotypes. All of these deaths occurred in non-Indigenous Australians with a median age of 61 years (range 48–83).

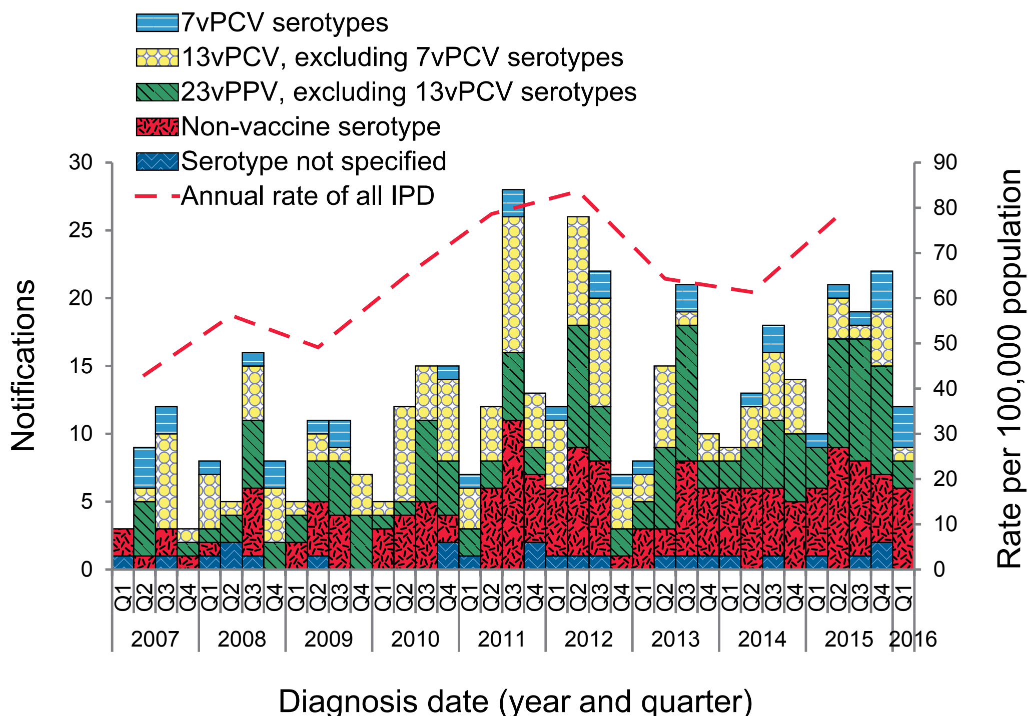
Notes

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).

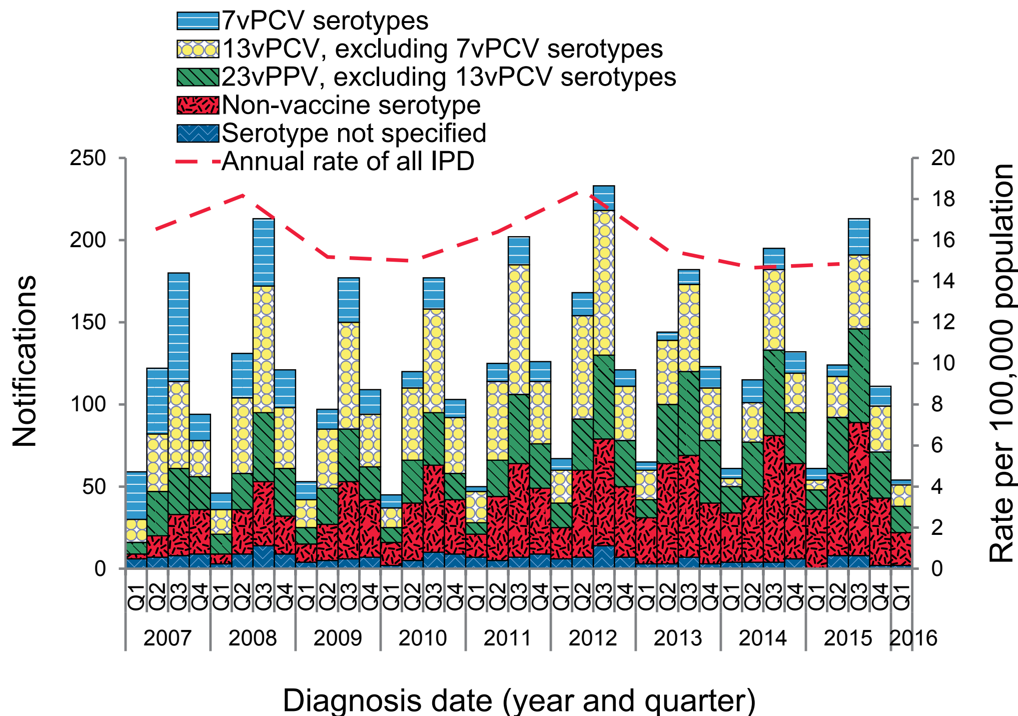
In this report, a 'vaccine failure' is where a fully vaccinated child is diagnosed with IPD due to a serotype covered by the administered vaccine. 'Fully vaccinated' describes cases that have completed the primary course of the relevant vaccine(s) required for their age according to the most recent edition of *The Australian Immunisation Handbook*,

Figure 2: Notifications and annual rates* of all invasive pneumococcal disease in Indigenous Australians aged 50 years or over, Australia, 2007 to 31 March 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 31 March 2016, by vaccine serotype group



* Annual rates are shown on Q2, excluding 2016.

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 4 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.

Acknowledgements

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

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

Communicable Diseases Intelligence

Volume 40 Number 2 Quarterly report June 2016

Contents continued

Quarterly reports

E290 OzFoodNet quarterly report, 1 April to 30 June 2014
The OzFoodNet Working Group

E297 National Notifiable Diseases Surveillance System, 1 January to 31 March 2016

E304 Australian childhood immunisation coverage, October to September cohort, assessed as at 31 December 2015
Alexandra Hendry for the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases

E306 Australian Sentinel Practices Research Network, 1 January to 31 March 2016
Monique B-N Chilver, Daniel Blakeley, Nigel P Stocks for the Australian Sentinel Practices Research Network

E308 Invasive pneumococcal disease surveillance, 1 January to 31 March 2016
Kate Pennington, Anna Glynn-Robinson, Cindy Toms and the Enhanced Invasive Pneumococcal Disease Surveillance Working Group, for the Communicable Diseases Network Australia