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Invited commentary

Developing research priorities for Australia's response to infectious disease emergencies

Sharon R Lewin, Ross Andrews, Jodie McVernon, Julie Milland, Miranda Z Smith, Tania C Sorrell on behalf of the investigators of the Australian Partnership for Preparedness Research on Infectious Disease Emergencies

Key points

- The Australian Partnership for Preparedness Research on Infectious Disease Emergencies (APPRISE) is a National Health and Medical Research Council (NHMRC)-funded Centre of Research Excellence (CRE). The Centre is a nationally-distributed, multidisciplinary team of experts that will conduct high-impact research to strengthen Australia's emergency response to infectious diseases.
- Announced in June 2016, this CRE is unique on the basis of the total funding and the scope of work. In the first year, the remit is to complete a targeted consultation of stakeholders across Australia to determine what research activities and protocols, training opportunities and pathways, collaborative partnerships and cross-sectoral linkages should be prioritised to ensure Australia is equipped for a more coordinated, effective and evidence-based response to infectious disease outbreaks.
- The consultation's stakeholder engagement process includes interviews and workshops with government and non-government stakeholders, ranging from government policy makers, clinicians, researchers and infection prevention specialists to consumers.
- Proposed research priorities will be discussed, with analysis and consideration of stakeholder views then incorporated into a final consultation report.
- This report, to be tabled in June 2017, will be evaluated by the NHMRC and the Australian Health Protection Principal Committee (AHPPC) and approval will be required prior to funding allocation.

Introduction

Multidisciplinary and targeted research is essential to support knowledge and practice before, during and after infectious disease outbreaks and pandemics. Countries such as the United Kingdom, the United States of America and Canada have research structures that support emergency responses to infectious disease outbreaks. Australia's research response to past outbreaks has been limited, in part due to delays inherent in grant funding calls made in reaction to such events. At the same time, it has been recognised that research co-ordination and translation must be improved in order to address the most important policy and practical needs associated with a rapidly evolving outbreak.

The APPRISE CRE is a national network of researchers in organisations across Australia funded by the NHMRC. The CRE will establish a framework for rapid and practical research responses to future outbreaks of:

- new pathogens emerging in Australia;
- new pathogens emerging outside Australia; and
- existing pathogens that become of local or regional concern.

The principal goals of APPRISE are to:

- establish a sustainable multidisciplinary research team across Australia to perform high-quality and high-impact infectious disease emergency response research – the team should have strong links to national and international networks
- develop a research strategy for the emergency response to infectious diseases across clinical, laboratory and public health domains – the strategy should be guided by ongoing consultation with stakeholders; and
- generate and execute the best evidence for the emergency response through capacity-building and training and effective communication with frontline health workers, policy makers and consumers.

To fulfil the goals of APPRISE and meet the funding conditions outlined by the NHMRC, a consultation process is underway to address and set research priorities across the CRE's 4 inter-related

'pillars': clinical research; public health research; laboratory research; and key populations including Aboriginal and Torres Strait Islander peoples. Each pillar will be supported and interact with 4 cross-cutting platforms: ethics; data management; education and training; and leadership and integration.

Developing proposed research priorities through consultation

NHMRC funding beyond the first year is dependent upon a successful and targeted consultation of stakeholders across Australia to define research priorities and establish methods and networks for future collaboration. Broad stakeholder engagement is essential to ensure the national research priorities are fully informed and to provide the best possible framework for future collaboration and networking.

The consultation will be a staged process (Table). Stakeholder engagement will include group interviews, one-on-one interviews and workshops. Stakeholders who will be consulted about research priorities include:

- public health organisations (government and non-government);
- clinical and infection prevention organisations (government and non-government);
- laboratory research stakeholders;
- representatives of at-risk populations;
- regional stakeholders;
- emergency and defence stakeholders; and
- consumers.

The APPRISE Expert Reference Panel (Table) will provide feedback and confirm the outcomes of the consultation phase, which will include:

- identifying research priorities;
- building the foundation for ongoing collaboration and engagement;
- recommending methods to translate research outcomes to policy and practice; and
- developing strategies to ensure the long-term sustainability of APPRISE beyond the 5-year timeframe of the CRE.

Evaluating the research priority report

The AHPPC will evaluate the consultation process report and recommend whether the NHMRC should approve the research priorities and ongoing funding of the CRE.

The complex planning and ongoing engagement needed among diverse stakeholders to predict, prepare for and respond to infectious disease outbreaks requires a solid evidence base. The consultation and collaborative priority setting phase that is now underway is a vital part of developing the research needed for an effective emergency response to infectious diseases in Australia.

The development of a cross-sectoral and multidisciplinary team to facilitate cohesive and rapid research responses in infectious disease emergencies will strengthen Australia's capacity to deal with the next infectious outbreak or pandemic, whatever form it takes.

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Table: Six stages of stakeholder engagement

Stage	Description
1	Establish Expert Reference Panel – federal and state or territory health officers and medical advisors, Indigenous communities, veterinary health experts, public health researchers
2	Literature and document review
3	Stakeholder mapping
4	Stakeholder consultations
5	Confirm research priorities
6	Report to the National Health and Medical Research Council for evaluation

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Original article

Influenza testing trends in sentinel surveillance general practices in Victoria 2007 to 2014

Genevieve A Cowie, Benjamin C Cowie, James E Fielding

Abstract

The Victorian Sentinel Practice Influenza Network conducts syndromic surveillance for influenza-like illness (ILI), with testing for laboratory confirmation of a proportion of cases at the discretion of general practitioners. The aim of this study was to evaluate the consistency of sentinel general practitioners' swabbing practice within and between influenza seasons. Aggregated, weekly, non-identified data for May to October each year from 2007 to 2014 were used to calculate the proportion of patients presenting with ILI (defined as cough, fever and fatigue), proportion of ILI patients swabbed and proportion of swabs positive for influenza. Data on the proportion of consultations for ILI and the proportion of ILI patients swabbed were aggregated into time-period quintiles for each year. Analysis of variance was used to compare ILI patients swabbed for each aggregated time-period quintile over all 8 years. Spearman's correlation and Bland-Altman analyses were used to measure association and agreement respectively between ILI proportions of consultations and swabs positive for influenza in time period quintiles within each year. Data were aggregated by year for the rest of the analyses. Between 2007 and 2014 there was a slight decrease in the proportion of positive tests and the proportion of ILI patients was generally a good proxy for influenza test positivity. There was consistency in testing within and between seasons, despite an overall testing increase between 2007 and 2014. There was no evidence for temporal sampling bias in these data despite testing not being performed on a systematic basis. This sampling regimen could also be considered in other similar surveillance systems. *Commun Dis Intell* 2017;41(1):E4–E9.

Keywords: influenza, influenza-like illness, sentinel surveillance, primary health care, Australia

Introduction

A range of influenza surveillance methods and settings are used both internationally and within Australia in order to assess the onset, magnitude and duration of influenza seasons.[1–3] These include syndromic surveillance of influenza-like illness (ILI) that serves as a proxy for influenza activity. This is simple to operate, relatively cheap as no testing is required (there can be reluctance to test children), can be delivered online, is generally consistent over time and is not subject to biases in testing practices. However, the positive predictive value of ILI for influenza can vary due to the use of different case definitions across systems, differing application of definitions, incidence of influenza and other respiratory viruses during the season and the surveillance setting (e.g. primary health care, hospital outpatients, emergency departments or hospital admissions).[4–6] Syndromic surveillance is therefore usually combined with laboratory testing using modern diagnostic techniques such as real-time polymerase chain reaction (PCR)[7] enabling more accurate case ascertainment, a better understanding of circulating types, subtypes and strains, and to inform the formulation of vaccines.[8]

The Victorian Government Department of Health and Human Services funds the Victorian Sentinel Practice Influenza Network (VicSPIN), operated by the Epidemiology Unit at the Victorian Infectious Diseases Reference Laboratory. The Network is comprised of approximately 100 participating general practitioners (GPs), and aims for representation of approximately 1 GP per 200,000 population in metropolitan areas and 1 per 50 to 100,000 in rural areas.[9,10] It operates each year between May and October. This system conducts syndromic and laboratory surveillance of ILI and influenza among general practice patients to assess the onset, magnitude and duration of each influenza season, circulating strains and vaccine effectiveness. Each week, GPs are requested to report their total number of consultations and the number that met the ILI case definition.[2] Nose and/or throat swabbing of ILI patients within 4 days of symptom onset for PCR testing is done at the discretion of the general practitioner rather than systematically,[8] which has the potential to introduce selection bias.[11] Whom GPs select to swab and the frequency at which they do it may vary throughout the season due to many factors such as individual GPs' preferences, the patient's

vaccination status[12] and influenza incidence or perception of it,[13] which may in turn be influenced by media reporting.[14] Swabbing of ILI patients by VicSPIN GPs over the whole season has ranged from 39% in 2005[10] to 69% in 2013.[12]

The aim of this study was to evaluate whether the percentage of patients swabbed by sentinel GPs has changed within and between influenza seasons.

Methods

Aggregated, weekly, non-identified data obtained from VicSPIN practices were obtained for the surveillance periods of May to October each year from 2007 to 2014, to calculate the proportion of patients in GP consultations presenting with ILI (defined as cough, fever and fatigue),[8,15] the proportion of ILI patients swabbed and proportion of swabs positive for influenza. Weekly data on the proportion of consultations that were for ILI and the proportion of ILI patients swabbed were aggregated into time-period quintiles for each year. Analysis of variance (ANOVA) was used to compare the proportion of ILI patients swabbed during the same time period quintile over all 8 years as well as quintiles within each season, with Bonferroni correction for multiple comparisons applied when the initial result was significant. Spearman's rank correlation was used to measure the strength of association and Bland-Altman analyses to measure agreement between the proportions of consultations for ILI and swabs positive for influenza within each year.

Data were aggregated by year for the rest of the analyses. Rates of ILI consultations, ILI patients swabbed and swabs positive for influenza were analysed using Pearson's chi-square for strength of association and the chi-square test for trend.

Weeks where the number of patients swabbed exceeded the number of ILI patients were excluded. Where time-period quintiles were used, weeks at the beginning (weeks 18 to 21) and end (weeks 42 to 43) of the season were excluded as the small numbers during these periods caused large fluctuations in proportions calculated.

Statistical calculations including ANOVA, Pearson correlation and Bland Altman analyses were performed in Microsoft Excel, with *P*-values and chi-square analyses computed using DanielSoper.com Statistics Calculators version 3.0 beta.[16] Other online epidemiological calculators used were Social Science Statistics[17] for Spearman's rank correlation tests and AusVet[18] for chi-square analyses.

Data in this study were collected, used and reported under the legislative authorisation of the Victorian Public Health and Wellbeing Regulations 2009. Monash University granted the study exemption from ethical review (project number CF15/1277 – 2015000606).

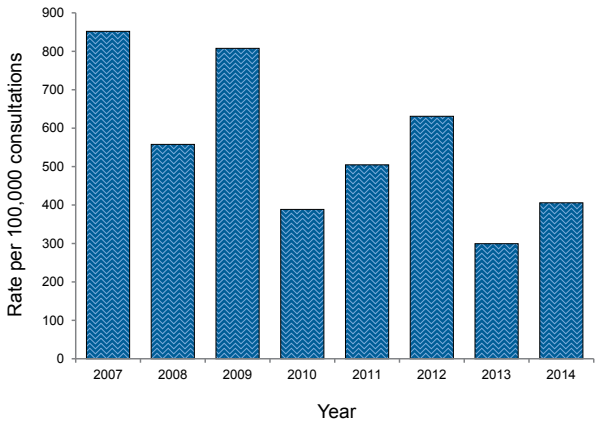
Results

Over the 208 weeks of data collection between 2007 and 2014, sentinel GPs reported 1,384,141 patient consultations. The reported number of patients swabbed exceeded the reported number of ILI consultations in 7 weeks, resulting in the exclusion of 126 (1.7%) swabbed patients and 100 (1.3%) ILI patients. A further 747 ILI patients including 436 swabbed patients were excluded from the beginning and end of seasons for the time period quintile analyses.

Influenza-like illness consultations by year

Between 2007 and 2014, 7,421 patients presented with ILI, with a median of 889 patients per year; the minimum of 481 was in 2013 and the maximum of 1,567 was in the pandemic year of 2009. This corresponded to 299 and 807 ILI patients per 100,000 consultations respectively (Figure 1). The annual variation in ILI consultation rates was statistically significant ($\chi^2 = 800.96$, df = 7, $P < 0.01$).

Figure 1: Rate of influenza-like illness patients per 100,000 consultations, VicSPIN, 2007 to 2014, by year



Influenza-like illness swabbed by year

There were 4,542 ILI patients swabbed between 2007 and 2014, with a median of 479 patients (67%) per year. The minimum and maximum number of patients was 314 in 2013 and 1,071 in 2009 respectively. The percentage of ILI patients swabbed per year increased from a low of 42% in 2007 to a high of 75% in 2014. The difference between the annual

percentage of ILI patients swabbed during this period was statistically significant ($\chi^2 = 384.07$, df = 7, $P < 0.001$). However, the trend was non-linear ($\chi^2 = 198.97$, df = 6, $P < 0.001$). The percentage of ILI patients swabbed from 2007 to 2008 was significantly lower than from 2009 to 2014 ($\chi^2 = 333.38$, df = 1, $P < 0.01$). The percentage of swabbed ILI patients varied between 60% and 75% between 2009 and 2014 (Figure 2).

Percentage of swabs positive by year

There were 1,624 swabs positive for influenza over the 8 years of data collection with a median of 182 positive swabs per year or 37% of swabs taken. The minimum percentage positive was 22% in 2013 and the maximum was 47% in 2007. There was a slight, but statistically significant ($\chi^2 = 10.63$, df = 1, $P < 0.01$), non-linear downward trend in the percentage of ILI patients with influenza-positive swabs during the study (Figure 3).

Figure 2: Percentage and number of influenza-like illness patients swabbed, VicSPIN, 2007 to 2014, by year

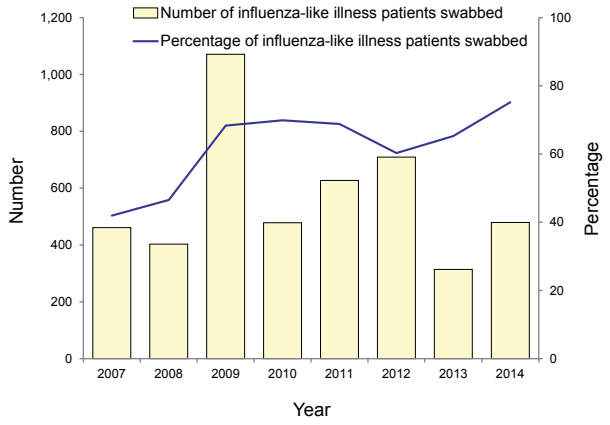


Figure 3: Percentage and number of swabs positive for influenza, VicSPIN, 2007 to 2014, by year

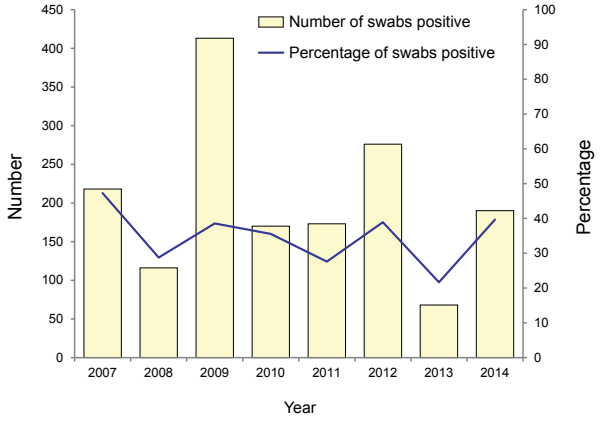
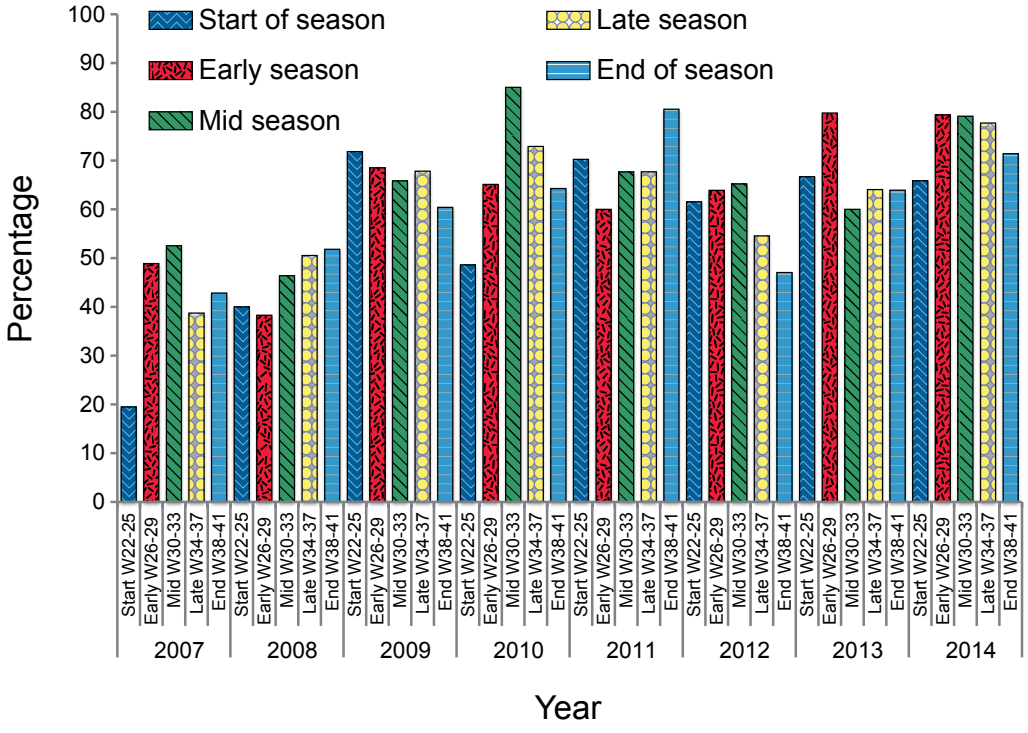


Figure 4: Percentage of influenza-like illness patients swabbed, VicSPIN, 2007 to 2014, by time period quintile



W = Weeks

Influenza-like illness patients swabbed during seasons

The percentage of ILI patients swabbed for every year, divided into seasonal quintiles, is shown in Figure 4.

The percentage of ILI patients swabbed did not significantly differ between time-period quintiles during each annual influenza season (Table 1). Similarly, no significant differences were observed when the same time-period quintiles over all 8 seasons were compared ($F = 0.87$, $F_{critical} = 2.64$, $df = 4$, $P = 0.49$).

Table 1: ANOVA F test values for the proportion of influenza-like illness patients swabbed for influenza, VicSPIN, 2007 to 2014, (degrees of freedom = 4)

Year	n	F	$F_{critical}$	*P* value
2007	440	2.37	3.06	0.10
2008	364	2.33	3.06	0.10
2009	977	0.13	3.06	0.97
2010*	436	4.35	3.06	0.02*
2011	574	0.96	3.11	0.46
2012	639	2.78	3.06	0.07
2013	290	1.43	3.18	0.28
2014	447	0.77	3.11	0.56

* Not significant after Bonferroni correction

Correlation and agreement of influenza-like illness patients with swabs positive for influenza

When compared by time period quintiles, the proportion of patients with ILI correlated positively with the proportion of swabs positive for influenza in every year; and was statistically significant in all but 2008 and 2011 (Table 2). All years when the incidence of influenza was assumed to be high, that is, when the percentages of swabs positive for influenza was greater than the median for 2007 to 2014, were statistically significant.

Bland Altman analyses by time quintiles in each year showed that the fixed differences between the proportion of patients presenting with ILI and the proportion of swabs positive for influenza shown by the intercepts, were not significant. However, in addition to the fixed differences, in all years there were significant increasing differences between the 2 proportional measures as their magnitude increased, as indicated by the slope.

Discussion

The lack of variation in swabbing practice with time-period quintiles indicates that swabbing practice by VicSPIN GPs was generally consistent within and between influenza seasons from 2007 to 2014, despite the overall increase in testing since 2009. Consequently, although VicSPIN GPs do not systematically sample ILI patients for influenza testing, no temporal sampling bias was apparent.[19]

There was an increase in testing from 2009 among sentinel GPs, although laboratory testing, without explicit caps, has always been available to them.

Table 2: Spearman's correlation and Bland Altman analyses for proportions of consultations that are influenza-like illness and proportions of swabs positive for influenza, VicSPIN, 2007 to 2014

		Spearman's rho, df=3		Bland Altman agreement			
Year	n	r_s	*P* value (2 tailed)	Intercept‡	*P* value intercept	Slope	*P* value slope
2007†	440	1.0	<0.001*	-0.0003	0.98	1.9103	*P*<0.001
2008	364	0.7	0.19	-0.008	0.24	1.9651	*P* <0.001
2009†	977	1.0	<0.001*	-0.002	0.67	1.887	*P* <0.001
2010	436	0.9	0.04*	-0.0032	0.37	1.9634	*P* <0.001
2011	574	0.6	0.28	-0.0054	0.16	1.9597	*P* <0.001
2012†	639	0.9	0.04*	0.0137	0.2	1.8572	*P* <0.001
2013	290	0.9	0.04*	-0.0026	0.12	1.9654	*P* <0.001
2014†	447	0.9	0.04*	0.0022	0.63	1.9405	*P* <0.001

* $P < 0.05$

† Greater than median percentage influenza positive for 2007 to 2014.

‡ Intercept = fixed difference between proportions of consultations that are influenza-like illness and proportions of swabs positive for influenza.

The pandemic year of 2009 may have simply prompted greater awareness and concern about influenza among sentinel GPs, however there have also been wider changes in attitudes to testing in primary care over that time. PCR testing with its rapid availability of results has become increasingly widely used after introduction of government reimbursement through Medicare in 2005 and funding for purchase of new PCR machines after the 2009 pandemic.[1,2,20] PCR testing has increased to the point where it is thought to be responsible for the substantial increase in statutory notifications over the 2010 to 2015 period, since syndromic surveillance has not seen similar increases in seasonal incidence.[1,21]

The slight downward trend in the proportion of swabs positive between 2007 and 2014 may be partly related to the observed increase in testing over the same period. However, this relationship is likely to be obscured by several factors, including variability in influenza incidence and other causes of ILI, and the age groups affected in a given year, for instance, GPs may be more reluctant to swab children, and nursing home resident surveillance is covered by a different part of the system in Victoria.[12,22,23]

Theoretically, swabs are more likely to be positive in years with higher incidence of influenza. There was a positive correlation between the percentage of consultations with diagnosis of ILI and percentage of swabs positive in all 8 years of data collection. This was statistically significant in all but 2 years, including those of presumed higher influenza incidence. This confirms that ILI is a reasonable proxy for interpreting the severity of influenza seasons in this dataset.

A limitation of this study is that the small sample size for the time-period quintile analyses may have underpowered the study to detect statistically significant variation in swabbing, as well as correlation and agreement of swabs positive for influenza with consultations for ILI. Furthermore, no conclusions about other sources of sampling bias such as vaccination status, age or other factors can be drawn from this study as they were not examined.

VicSPIN gives timely ILI and laboratory test data from a broadly representative sample of GPs, providing an important part of influenza surveillance in Victoria and Australia as a whole. Between 2007 and 2014 there was a slight decrease in the proportion of positive tests and the proportion of patients with ILI was generally a reasonable proxy for influenza test positivity in these primary care patients, especially in high incidence years. During the study period, GPs in the VicSPIN surveillance program did not vary their ILI testing practices by time period quintiles, despite an overall increase in testing since 2009. This study supports the current VicSPIN practice of testing for influenza at the discretion of the GP and the confidence that can be placed in the surveillance data that are produced. Achieving systematic sampling is likely to require considerable effort and may reduce GP participation with little impact on the value of surveillance. This sampling regime could also be considered in other similar surveillance systems.

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References

1. Fielding JE, Regan AK, Dalton CB, Chilver MB, Sullivan SG. How severe was the 2015 influenza season in Australia? *Med J Aust* 2016.;204(2):60–61.
2. Kelly HA, Grant KA, Tay EL, Franklin L, Hurt AC. The significance of increased influenza notifications during spring and summer of 2010-11 in Australia. *Influenza Other Respir Viruses* 2013;7(6):1136–1141.
3. Clothier HJ, Atkin L, Turner J, Sundararajan V, Kelly HA. A comparison of data sources for the surveillance of seasonal and pandemic influenza in Victoria. *Commun Dis Intell* 2006;30(3):345–349.
4. Navarro-Mari JM, Pérez-Ruiz M, Cantudo-Muñoz P, Petit-Gancedo C, Jiménez-Valera M, et al. Influenza-like illness criteria were poorly related to laboratory-confirmed influenza in a sentinel surveillance study. *J Clin Epidemiol* 2005;58(3):275–279.
5. Moretti ML, Sinkoc V, Cardoso LG, Camargo GJ, Bachur LF, Hofling CC, et al. Lessons from the epidemiological surveillance program, during the influenza A (H1N1) virus epidemic, in a reference university hospital of Southeastern Brazil. *Rev Soc Bras Med Trop* 2011;44(4):405–411.
6. Sreng B, Touch S, Sovann L, Heng S, Rathmony H, Huch C, et al. A description of influenza-like illness (ILI) sentinel surveillance in Cambodia, 2006–2008. *Southeast Asian J Trop Med Public Health* 2010;41(1):97–104.
7. Baumbach J, Mueller M, Smelser C, Albanese B, Sewell CM. Enhancement of influenza surveillance with aggregate rapid influenza test results: New Mexico, 2003–2007. *Am J Public Health* 2009;99 Suppl 2:S372–S377.
8. Fielding JE, Grant KA, Tran T, Kelly HA. Moderate influenza vaccine effectiveness in Victoria, Australia, 2011. *Euro Surveill* 2012;17(11). pii: 20115.

9. Fielding JE, Miller ER, Adams J, Hawking B, Grant K, Kelly HA. Influenza surveillance in Victoria, 2006. *Commun Dis Intell* 2007;31(1):100–106.
10. Turner JL, Fielding JE, Clothier HJ, Kelly HA. Influenza surveillance in Victoria, 2005. *Commun Dis Intell* 2006;30(1):137–143.
11. Thompson DL, Baumbach J, Jungk J, Sewell CM, Smelser C, Landen M. Does outpatient laboratory testing represent influenza burden and distribution in a rural state? *Influenza Other Respir Viruses* 2013;7(5):686–693.
12. Carville KS, Grant KA, Sullivan SG, Fielding JE, Lane CR, Franklin L, et al. Understanding influenza vaccine protection in the community: an assessment of the 2013 influenza season in Victoria, Australia. *Vaccine* 2015;33(2):341–345.
13. Moore K, Black J, Rowe S, Franklin L. Syndromic surveillance for influenza in two hospital emergency departments. Relationships between ICD-10 codes and notified cases, before and during a pandemic. *BMC Public Health* 2011;11:338.
14. Haddock RL, Damian YS, Duguies LA, Paulino YC. Guam's influenza epidemic(s) of 2009. *Hawaii Med J* 2010;69(6 Suppl 3):50–51.
15. Thursky K, Cordova SP, Smith D, Kelly H. Working towards a simple case definition for influenza surveillance. *J Clin Virol* 2003;27(2):170–179.
16. Soper DS. DanielSoper.com Statistics Calculators. 2016. Available from: http://www.danielsoper.com/statcalc3/default.aspx Accessed on 17 February 2016.
17. Stangroom J. Spearman's rho calculator. 2016. Available from: http://www.socscistatistics.com/tests/spearman/Default.aspx Accessed on 5 December 2016.
18. AusVet Animal Health Services. EpiTools epidemiological calculators: Chi-squared test for trend. 2016. Available from: http://epitools.ausvet.com.au/content.php?page=trend Accessed on 18 February 2016.
19. Porta MS, International Epidemiological Association. Surveillance. In: *A dictionary of epidemiology*. 5th edn. Oxford; New York: Oxford University Press: 2008. p. 239.
20. Carman WF, Wallace LA, Walker J, McIntyre S, Noone A, Christie P, et al. Rapid virological surveillance of community influenza infection in general practice. *BMJ* 2000;321(7263):736–737.
21. Kaczmarek MC, Ware RS, Lambert SB. The contribution of PCR testing to influenza and pertussis notifications in Australia. *Epidemiol Infect* 2016;144(2):306–314.
22. Kelly H, Carville K, Grant K, Jacoby P, Tran T, Barr I. Estimation of influenza vaccine effectiveness from routine surveillance data. *PLoS ONE* 2009;4(3):e5079.
23. Turner J, Kelly H. A medical locum service as a site for sentinel influenza surveillance. *Euro Surveill* 2005;10(4):96–98.

An outbreak of *Salmonella* Saintpaul gastroenteritis after attending a school camp in the Northern Territory, Australia

Anthony DK Draper, Claire N Morton, Joshua NI Heath, Justin A Lim, Peter G Markey

Abstract

An outbreak of salmonellosis occurred following attendance at a school camp between 5 and 8 August 2014 in a remote area of the Northern Territory, Australia. We conducted a retrospective cohort study via telephone interviews, using a structured questionnaire that recorded symptoms and exposures to foods and activities during the camp. A case was anyone with laboratory confirmed *Salmonella* Saintpaul infection or a clinically compatible illness after attending the camp. Environmental health officers from the Environmental Health Branch undertook an investigation and collected water and environmental samples. We interviewed 65 (97%) of the 67 people who attended the camp. There were 60 students and 7 adults. Of the 65 people interviewed, 30 became ill (attack rate 46%); all were students; and 4 had laboratory confirmed *S.* Saintpaul infection. The most commonly reported symptoms were diarrhoea (100% 30/30), abdominal pain (93% 28/30), nausea (93% 28/30) and fever (70% 21/30). Thirteen people sought medical attention but none required hospitalisation. Illness was significantly associated with drinking cordial at lunch on 7 August (RR 3.8, 95% CI 1.3–11, $P < 0.01$), as well as drinking cordial at lunch on 8 August (RR 2.1, 95% CI 1.1–4.2, $P=0.01$). *Salmonella* spp. was not detected in water samples or wallaby faeces collected from the camp ground. The epidemiological investigation suggests the outbreak was caused by environmental contamination of food or drink and could have occurred during ice preparation or storage, preparation of the cordial or from inadequate sanitising of the cooler from which the cordial was served. This outbreak highlights the risks of food or drink contamination with environmental *Salmonella*. Those preparing food and drink in campground settings should be vigilant with cleaning, handwashing and disinfection to prevent outbreaks of foodborne disease. *Commun Dis Intell* 2017;41(1):E10–E15.

Keywords: outbreak, *Salmonella* Saintpaul, gastroenteritis, salmonellosis, foodborne disease, cohort study, public health, camp, environmental *Salmonella*

Introduction

Salmonella Saintpaul is a common *Salmonella* serotype in the Northern Territory of Australia. In 2014 it was the 3rd most commonly reported serovar, accounting for 11% of all salmonellosis notifications.[1] About half of all *S.* Saintpaul infections in the Northern Territory occur in children under 4 years of age, with the majority of these thought to be environmentally acquired.[2] Known reservoirs of *S.* Saintpaul are reptiles (including geckos and lizards),[3–5] amphibians[6–10] and wallabies and kangaroos.[11] In 2013, *S.* Saintpaul was detected in ovine, equine, feline, bovine, primate, kangaroo, beef, vegetables, tree nuts and parkland soil samples that were tested and recorded in the National Enteric Pathogen Surveillance System.[12] However, there have been relatively few outbreaks of *S.* Saintpaul recorded in Australia. In 2000 an outbreak occurred at a construction site in regional Queensland with 28 workers reporting gastroenteritis.[13] Tank water contaminated with mice and frog faeces was identified as the cause. A subsequent outbreak, also in Queensland saw 21 people affected with contaminated bore water the likely cause.[14] In 2006, a nationwide outbreak occurred with 36 cases associated with consumption of rockmelon grown in Northern Australia.[15]

On 14 August 2014, the Northern Territory Centre for Disease Control was alerted to a possible outbreak of gastroenteritis among school students who had recently attended a school camp between 5 and 8 August 2014. This was discovered while conducting hypothesis generating interviews with routine salmonellosis notifications, when it was revealed that 2 cases attended the same school and had attended camp in the week preceding their illness. The school camp was at a remote outback location and was attended by 67 people who slept in tents. Food and drink was prepared and served from a kitchen housed in a caravan. The initial cases reported that other attendees were also sick. An outbreak investigation was initiated to identify the cause of illness and implement appropriate public health measures to prevent further cases.

Methods

Epidemiological investigation

We requested details of other school groups that attended the camp grounds in the week prior to and following 5 to 8 August 2014 in order to determine whether other school groups were affected. There were no reports of illness in other groups.

Once the existence of an outbreak was confirmed, a retrospective cohort study was undertaken in order to try to determine which exposures were associated with illness.

We developed and administered a questionnaire that recorded details on symptoms and health seeking behaviour as well as exposures at the camp based on the camp menu, itinerary, activities and observations made at the environmental health site visits on 18 and 19 August. Active case finding was also undertaken through this questionnaire. We obtained verbal consent from a parent or a guardian prior to conducting telephone interviews with cases. All cases were provided with a salmonellosis fact sheet.

A probable case was defined as a person who attended the camp between 5 and 8 August 2014 and subsequently developed a diarrhoeal illness. A confirmed case was defined as any person who had a diarrhoeal illness and had *S*. Saintpaul isolated from a faecal sample. Data were collected and entered into Microsoft Excel 2010 (Microsoft, USA) and statistical analysis was conducted using StataIC® 13 (StataCorp, USA). Univariate analysis of exposures was conducted and we calculated relative risks (RR); 95% confidence intervals (CI) and *P* values were considered significant at the 0.05 level. Fisher's exact test was used when counts were <5. When a RR was infinite, exact logistic regression was used to calculate an odds ratio (OR) and 95% CI. The χ^2 test was used to analyse gender. Age was analysed using the Mann Whitney Wilcoxon Rank Sum test. We conducted multivariate analysis using logistic regression on all variables which had a $P < 0.05$ after univariate analysis.

Ethics approval was not sought for this investigation as it was conducted under the auspices of public health legislation.[16]

Environmental health investigation

Environmental health officers (EHOs) and an epidemiologist from the Northern Territory Department of Health visited the camp facility on 18 August and then again on 19 August to identify potential sources of infection and any contraventions of the *Northern Territory Food Act*[17] *and Northern Territory Public and Environmental Health Act*.[16] During the investigation, food preparation and storage areas were inspected, food preparation methods investigated, drinking water and sewage disposal infrastructure was inspected, and samples of water from the bore head, header tanks, ablution blocks, drinking water taps, and kitchen facilities were taken. Environmental samples were taken of the lake water, which was used for irrigation of the grounds, and wallaby faeces from the ground where tents were pitched. There was no food leftover from the camp to sample. Cleaning and disinfection of the kitchen, ablution facilities and camping equipment was also investigated.

Laboratory investigation

Water samples were collected from various locations at the campsite and tested at the Northern Territory Department of Primary Industry and Fisheries (DPIF) Water Microbiology Laboratory in Darwin for the presence of coliforms, *Escherichia coli* and enterococci. The results were reported against the Australian Drinking Water Guidelines.[18] Water samples from the hand wash basin, water tank and kitchen tap at the camp were tested by ProMicro, Hillarys, Western Australia for the presence of coliforms, *E. coli* and *Salmonella* spp. A heterotrophic colony count was also performed.

Samples of wallaby faeces were tested for the presence of *Salmonella* spp at the Northern Territory DPIF Veterinary Laboratory in Darwin, Northern Territory.

Stools were cultured using standard techniques. Tests for *Cryptosporidium* and *Giardia* were conducted using antigen detection tests. When *Salmonella* was cultured, isolates were sent to SA Pathology or the Microbiological Diagnostic Unit at the University of Melbourne for serotyping.

Results

Epidemiological investigation

We contacted 65 of 67 (response rate 97%) of those who attended the camp (60 students and 7 adults). Of the 65 people we interviewed, 30 became ill (attack rate 46%) (Table 1). All cases were students. Four people submitted stool samples and all had laboratory confirmed *S*. Saintpaul infection. Another 26 people met the case definition as probable cases. Thirteen people sought medical attention but none required hospitalisation.

There was no statistically significant difference between males and females becoming ill nor was there a difference in ages.

In addition to diarrhoea, 27% (8/30) experienced bloody diarrhoea, 93% (28/30) experienced abdominal pain, 70% (21/30) experienced fever, 93% (28/30) experienced nausea and 47% (14/30) experienced vomiting (Table 1). The median incubation period was 45 hours (range 7 to 160 hours). The epidemic curve was typical of a point source salmonellosis outbreak (Figure).

Table 1: Demographic characteristics and symptoms of cases who attended a school camp in the Northern Territory, 5 to 8 August 2014

Characteristic	n	%
Gender		
Male	18	60
Female	12	40
Symptoms		
Diarrhoea	30	100
Nausea	28	93
Abdominal pain	28	93
Lethargy	26	87
Headache	25	83
Fever	21	70
Vomiting	14	47
Bloody diarrhoea	8	27
Health seeking behaviour		
Sought medical attention	13	43
Hospitalised	0	0

Figure: Epidemiological curve of outbreak cases by onset day after attending a school camp in the Northern Territory, 5 to 8 August 2014 (n=30)

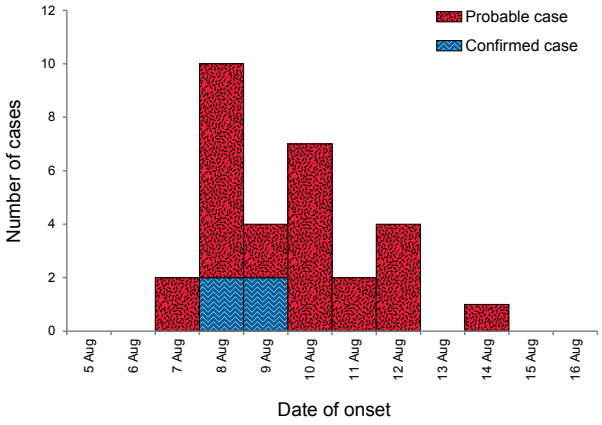

Table 2 shows a selection of risk factors from the univariable analysis which in total measured exposure to 170 variables. Drinking cordial at lunch on 7 August was statistically associated with illness (RR 3.8, 95% CI 1.3–11, $P < 0.01$), as was drinking cordial at lunch on 8 August (RR 2.1, 95% CI 1.1–4.2, $P < 0.01$). Other exposures that had a significant P value but with CI that included 1 were; drinking from a tap near the showers at the camp (RR 1.8, 95% CI 1.0–3.0, $P < 0.04$), using a tent supplied by the camp ground (RR 3.4, 95% CI 0.9–12.4, $P = 0.02$ and eating chicken casserole for dinner on 6 August (OR 8.9, 95% CI 1.2–∞, $P = 0.02$). Drinking cordial at any time between lunch on 7 August and the end of the camp on 8 August had a RR of 2.7 (95% CI 0.9-7.5, $P = 0.03$).

Table 2: Univariable analysis of selected risk factors for salmonellosis among attendees of a school camp in the Northern Territory, 5 to 8 August 2014

Exposure	Exposed			Unexposed			Crude RR	95% CI	*P* value
	Cases	Total	AR%	Cases	Total	AR%			
Drank cordial at lunch on 7 August	26	41	63	3	18	17	3.8	1.3–11.0	0.0015*
Drank cordial at lunch on 8 August	21	55	38	7	31	23	2.1	1.1–4.2	0.0144
Used a supplied tent	28	49	57	2	12	17	3.4	0.9–12.4	0.0217*
Ate chicken casserole at dinner on 6 August	29	54	54	0	6	0	8.9†	1.2–∞†	0.0242
Drank water from tap near showers	16	25	64	12	33	36	1.8	1.0–3.0	0.0370

AR = Attack rate

RR = Relative risk

CI = Confidence interval

* Fisher's exact

† Odds ratio and confidence intervals calculated using exact logistic regression.

Multivariate analysis resulted in a final model that included eating a cheese sandwich at lunch on 7 August (OR 0.2, 95% CI 0.0–0.9, $P = 0.03$), using a supplied tent (OR 6.8, 95% CI 1.1–40.7, $P = 0.04$) and eating chicken casserole for dinner on 6 August, which had an undefined OR as there were no cases who reported not eating it.

Environmental health investigation

The environmental health investigation observed that there were a large amount of animal droppings throughout the camping, activities and play areas. The ablution facilities were not in a clean state and dust, staining and grime was observed on the toilets, showers, hand basins, shower curtains, walls and floors. Drinking water at the camp site was from an untreated bore water supply and stored in header tanks that were gravity fed to the kitchen and ablution facilities. Patrons at the camp also had access to drinking water taps that were unclean and unprotected from environmental contamination. Untreated lake water was used to irrigate the camp ground. The kitchen was in a poor structural state, there was inadequate hand washing facilities and staff had a lack of food safety knowledge which may have resulted in poor practices in the kitchen. No staff illness was reported prior to or after the school camp. A large water container was observed outside the kitchen and dining area which was used to dispense cordial. It was visibly dirty and required cleaning.

Ice was produced onsite using untreated bore water that was poured into open steel containers that were frozen in a chest freezer. This chest freezer was also used to store other items including raw meats. Cordial at the camp contained untreated bore water and the ice prepared onsite.

Children who attended the camp participated in a number of activities which included abseiling, riding a flying-fox, lake walks, orienteering, playing ball games and cane toad hunting. The cane toad hunting activity occurred on the night of 6 August 2014 and involved some students collecting cane toads with gloved hands, placing them in a bag and then euthanasing them by placing the bag in the freezer.

EHOs directed the camp proprietor to treat the drinking water with chlorine prior to its use as a potable drinking water supply. The proprietor was also directed to refurbish or replace the kitchen facilities. Formal legal notices were issued to the proprietor under the *Northern Territory Food Act* and the *Northern Territory Public and Environmental Health Act 2011*. The proprietor voluntarily closed the camp in order to address the issues identified.

Laboratory investigation

Bore water samples collected from the kitchen tap, hand wash basin, shower tap, boys toilet tap, water tanks and from the bore head were all negative for *E. coli*, enterococci and the heterophilic colony count was also negative when assessed against the *Guidelines for Drinking Water in Australia*.[18] Lake water samples collected from a tap used to irrigate a lawn area at the camp ground were tested and recorded a total coliform count of $> 2{,}420$ per 100 mL, 18 *E. coli* per 100 mL and 38 enterococci per 100 mL. No *Salmonella* was detected in any of the samples tested above.

Samples of wallaby faeces tested negative for *Salmonella* spp.

Discussion

The results of this outbreak investigation suggests that food or beverage served at the camp on 7 August and possibly 8 August was responsible for the outbreak, and that a breakdown in cleanliness and food handling practices were the likely contributing factors. *S.* Saintpaul is a common environmental *Salmonella* serovar in the NT and it is likely that this outbreak was caused by the introduction of this organism from the environment into food or drink served at the camp. Unfortunately, no food or drink samples were collected and the specific cause of the outbreak cannot be determined. However, the environmental health investigation identified multiple opportunities for contamination to occur and resulted in formal legal notices being issued to the proprietor under the Northern Territory *Food Act* and the Northern Territory *Public and Environmental Health Act 2011.*

The results of the epidemiological investigation are inconclusive, particularly after multivariable analysis, but univariate analysis revealed an association with consuming cordial at lunch on either 7 or 8 August. The environmental investigation supports this. It revealed that ice used to make cordial was being produced on site in open topped containers that were filled with tap (bore) water. The bore water, along with other water samples at the camp tested negative for pathogens and other indicator organisms and was not considered a cause of the outbreak, unless it became contaminated during preparation of ice or cordial. Containers of ice, which were open containers with no covering, were placed in the chest freezer which was also used for storage of other items. This presented a risk of cross contamination to the ice from other items in the freezer. Apart from food items, it is also plausible that cane toads could have been euthanised in the same freezer. *Salmonella* can survive for extended periods at temperatures below

freezing.[19] When ready for use, the ice was further processed and placed into the containers by hand, which presented another opportunity for contamination. Concerns about the lack of hand washing facilities, availability of hot water for hand washing and actual practices around hand washing presented a further risk of contamination of the ice and add to the number of plausible mechanisms for contamination of the cordial throughout this ice-making process. Additionally, the lack of cleaning and disinfection of equipment, including the cordial container, could have led to contamination of the cordial during service.

There are other possible causes of the outbreak although these are less compelling. Eating the chicken casserole for dinner on the evening of 6 August also yielded a statistically significant association with illness after univariate analysis (OR 8.9, 95% CI 1.2–∞, $P = 0.02$) but there was less confidence in this association compared with that of the cordial. Chicken casserole has an inherent kill step and camp attendees reported that the casserole was served 'piping hot', which makes the chicken casserole an unlikely vehicle for *Salmonella* infection in this instance.

Sleeping in a supplied tent (i.e. supplied by the campground) also had a significantly high risk ratio after univariate analysis (RR 3.4, 95% CI 0.9–12.4, $P = 0.02$) and it is possible that exposure to contaminated tents led to the outbreak. However, exposure to the supplied tents would have led to earlier onset of illness and a pattern of illness consistent with a continuous exposure over the 2 days of the camp. This is in contrast to the pattern seen in the epidemic graph, which strongly suggests a point-source outbreak of illness (or one with a narrow window of exposure) with many cases initially becoming ill at the same time. The majority became ill 6 to 36 hours after lunch on 7 August coinciding with the average incubation period for salmonellosis.[20]

Drinking water from the taps near the shower block was associated with illness after univariate analysis with a relative risk of 1.8 (95% CI 1.0–3.0, $P = 0.03$). This association however, was much weaker than that of drinking cordial at lunch on August 7 and was considered unlikely as a cause as it was also a continuous exposure.

Multivariate analysis was attempted but the final model, which included eating a cheese sandwich at lunch on 7 August using a supplied tent and eating chicken casserole for dinner on 6 August was considered not to be a plausible causal pathway for the outbreak for the same reasons outlined above.

No other foods, activities or exposures in the questionnaire, including hunting cane toads were statistically associated with illness. Nevertheless, it could be that the source of salmonellosis was an unknown environmental exposure on 7 and 8 August. However, regardless of the specific cause, it is likely that the various environmental and infrastructure issues identified by the EHOs contributed to the risk of contamination and therefore illness in the patrons, whether directly from the environment or indirectly through contamination of food or drink prepared at the camp, in this instance, the cordial served at lunch on 7 August.

A major limitation of the investigation was that there was no food, drink or ice available for sampling and thus *S.* Saintpaul was not able to be detected in food or drink samples. Only bore water, lake water and wallaby faeces was collected for sampling and *S.* Saintpaul was not detected in any samples. The exact cause of the outbreak could not be determined but poor food handling practice, sanitation and hygiene could have facilitated contamination.

We were able to contact almost the entire cohort of persons who attended the camp, which minimised selection bias.

The large number of variables tested in this cohort study meant that some exposures could have been identified as being significantly associated with illness by chance alone rather than being a true association.

Conclusion

We conclude that an outbreak of *S.* Saintpaul at a school camp was most likely caused by environmental contamination of food or drink. There were multiple possible mechanisms for contamination to occur due to poor food safety knowledge, poor hygiene and structural deficiencies at the camp.

In order to prevent outbreaks such as this it is essential that those preparing food in campgrounds and outdoor settings have appropriate knowledge of safe food handling procedures and recognise the risks of contaminating food or water with pathogens from the environment, including *Salmonella*. Food handlers, including volunteers need to be adequately trained in safe food preparation procedures including hand washing, cleaning, disinfecting and recognising cross-contamination risks. It is also important to appropriately maintain facilities for food preparation and service. It is important to investigate outbreaks of environmental *Salmonella* in order to identify risks, undertake appropriate public health action and ensure public safety.

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References

1. Draper, A. OzFoodNet – Enhancing Foodborne Disease Surveillance Across Australia – Northern Territory 2014 Annual Report. 2015.
2. Northern Territory Notifiable Disease Surveillance (NTNDS) System. 2015.
3. Oboegbulem SI, Iseghohimhen AU. Wall geckos (Geckonidae) as reservoirs of *Salmonellae* in Nigeria: problems for epidemiology and public health. *Int J Zoonoses* 1985;12(3):228–232.
4. Friedman CR, Torigian C, Shillam PJ, Hoffman RE, Heltzel D, Beebe JL, et al. An outbreak of salmonellosis among children attending a reptile exhibit at a zoo. *J Pediatr* 1998;132(5):802–807.
5. Reptile-associated salmonellosis – selected states, 1994–1995. *MMWR Morb Mortal Wkly Rep* 1995;44(17):347–350.
6. Murray CJ. *Salmonellae* in the environment. *Rev Sci Tech* 1991;10(3):765–785.
7. Parish ME. Coliforms, *Escherichia coli* and *Salmonella* serovars associated with a citrus-processing facility implicated in a salmonellosis outbreak. *J Food Prot* 1998;61(3):280–284.
8. Minette HP. Epidemiologic aspects of salmonellosis in reptiles, amphibians, mollusks and crustaceans—a review. *Int J Zoonoses* 1984;11(1):95–104.
9. Bartlett KH, Trust TJ, Lior H. Small pet aquarium frogs as a source of *Salmonella*. *Appl Environ Microbiol* 1977;3(5)3:1026–1029.
10. Everard CO, Tota B, Bassett D, Ali C. *Salmonella* in wildlife from Trinidad and Granada, W.I. *J Wildl Dis* 1979;15(2):213–219.
11. Thomas AD, Forbes-Faulkner JC, Speare R, Murray C. Salmonelliasis in wildlife from Queensland. *J Wildl Dis* 2001;37(2):229–238.
12. Powling J, Howden B. National Enteric Pathogens Surveillance System Non-Human Annual Report 2013. Melbourne; 2014.
13. Taylor R, Sloan D, Cooper T, Morton B, Hunter I. A waterborne outbreak of *Salmonella* Saintpaul. *Commun Dis Intell* 2000;24(11):336–340.
14. Stafford R, Bell R. Enhancing Foodborne Disease Surveillance Across Australia. Annual report 2005 – Queensland. Queensland Government Department of Health. 2006.
15. Munnoch SA, Ward K, Sheridan S, Fitzsimmons GJ, Shadbolt CT, Piispanen JP, et al. A multi-state outbreak of *Salmonella* Saintpaul in Australia associated with cantaloupe consumption. *Epidemiol Infect* 2009;137(3):367–374.
16. Northern Territory *Public and Environmental Health Act 2011*. Available from: http://www.austlii.edu.au/au/legis/nt/num_act/paeha20117o2011337/ Accessed on 20 January 2016.
17. Northern Territory *Food Act*. Available from: http://www.austlii.edu.au/au/legis/nt/consol_act/fa57/ Accessed on 20 January 2016.
18. Australian Drinking Water Guidelines Paper 6 Water Quality Management Strategy. Canberra: National Health and Medical Research Council, National Resource Management Ministerial Council, Commonwealth of Australia, 2011. Available from: https://www.nhmrc.gov.au/guidelines-publications/eh52
19. *Salmonella*. In: Hocking, AL, ed. *Foodborne Microorganisms of Public Health Significance*. 6th edn. Waterloo DC: Australian Institute of Food Science and Technology Incorporated; 2003.
20. Salmonellosis. In: Heymann DL, ed. *Control of Communicable Diseases Manual*. 20th edn. Washington: American Public Health Association; 2015.

An outbreak of salmonellosis associated with duck prosciutto at a Northern Territory restaurant

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Abstract

In June 2015, an outbreak of salmonellosis occurred among people who had eaten at a restaurant in Darwin, Northern Territory over 2 consecutive nights. We conducted a retrospective cohort study of diners who ate at the restaurant on 19 and 20 June 2015. Diners were telephoned and a questionnaire recorded symptoms and menu items consumed. An outbreak case was defined as anyone with laboratory confirmed *Salmonella* Typhimurium PT9 (STm9) or a clinically compatible illness after eating at the restaurant. Environmental health officers inspected the premises and collected food samples. We contacted 79/83 of the cohort (response rate 95%); 21 were cases (attack rate 27%), and 9 had laboratory confirmed STm9 infection. The most commonly reported symptoms were diarrhoea (100%), abdominal pain (95%), fever (95%) and nausea (95%). Fifteen people sought medical attention and 7 presented to hospital. The outbreak was most likely caused by consumption of duck prosciutto, which was consumed by all cases (OR 18.6, CI 3.0–∞, $P < 0.01$) and was prepared on site. *Salmonella* was not detected in any food samples but a standard plate count of 2×10^7 colony forming units per gram on samples of duck prosciutto demonstrated bacterial contamination. The restaurant used inappropriate methodology for curing the duck prosciutto. Restaurants should consider purchasing pre-made cured meats, or if preparing them on site, ensure that they adhere to safe methods of production. *Commun Dis Intell* 2017;41(1):E16–E20.

Keywords: outbreak, *Salmonella*, gastroenteritis, foodborne disease, cohort study, public health, duck prosciutto

Introduction

In 2011, 33% of the foodborne and suspected foodborne outbreaks investigated by OzFoodNet, Australia's foodborne disease surveillance network, were associated with restaurants and caterers.[1] *Salmonella* Typhimurium was the aetiological agent in 37% of all foodborne and suspected foodborne outbreaks.[1] It is important to investigate all *S.* Typhimurium outbreaks in order to identify implicated foods, undertake appropriate public health action and ensure food safety. This information is vital for food safety authorities to protect the public by formulating policy and practice that minimises risk of foodborne illness.

On 26 June 2015, the Northern Territory Centre for Disease Control was notified of 2 salmonellosis cases via routine laboratory surveillance. Administration of salmonellosis hypothesis generating questionnaires revealed that both cases had eaten at the same restaurant on 19 and 20 June 2015 respectively. Initial enquiries identified further patrons had also become ill on these nights. An outbreak investigation was launched to identify the cause of illness and implement appropriate public health measures to prevent further cases.

Methods

Epidemiological investigation

We performed a retrospective cohort study via telephone using a structured questionnaire developed from the restaurant's menu. Patrons were contacted using the restaurant's electronic booking system, which contained a name and phone number for each table as well as an itemised bill. When further diners were identified, the patron who made the booking was asked to provide names and contact details of the other diners. Kitchen staff who worked on the implicated dates had also eaten food from the kitchen and so were included in the cohort. Ethics approval was not sought as the investigation was conducted under the auspices of public health legislation.[2]

A probable case was defined as anyone who ate dinner at the restaurant on 19 or 20 June 2015 and then developed a clinically compatible illness (diarrhoea) within 92 hours of consuming food at the restaurant. A confirmed case was defined as anyone who ate dinner at the restaurant on 19 or 20 June 2015 with a laboratory confirmed *Salmonella* infection, later refined to *Salmonella* Typhimurium PT9 (STm 9) infection when phage typing became available. The cohort study included confirmed and probable cases as outbreak cases. As the same menu was served on both 19 and 20 June, diners from both nights were analysed as a single cohort. Data were collected and

entered into Microsoft Excel 2010 (Microsoft, USA) and analysed using StataIC® 13 (StataCorp, USA). Binary and categorical outcomes were reported as numbers and proportions, and compared using the chi square test, or Fisher's exact test when cell counts were less than 5. Age was the only non-categorical variable reported. This was non-normally distributed and so medians were compared using the Wilcoxon rank sum test. To determine the risk of becoming unwell in exposed and unexposed groups, we calculated relative risks (RR). When a RR was infinite, exact logistic regression was used to calculate an odds ratio (OR) and 95% confidence intervals (CI). All results were considered significant at the 5% level.

Environmental health investigation

Environmental health officers (EHO) from the Northern Territory Department of Health visited the restaurant on 29 and 30 June 2015. During their investigation, EHOs inspected food preparation and storage areas, collected information about food preparation procedures, staff training and illness, and undertook temperature monitoring of food storage equipment. Samples of frozen duck fillets and frozen duck prosciutto were acquired for microbiological testing.

Laboratory investigation

Food samples were tested by the Food and Environmental Laboratory, SA Pathology, Adelaide South, South Australia. Samples were tested for the presence of coliforms, *Escherichia coli, Salmonella, Listeria* and thermophilic *Campylobacter*. A standard plate count (SPC) was also performed.

Stool samples were submitted and were either tested by polymerase chain reaction or by traditional culture. When *Salmonella* was cultured, isolates were sent to SA Pathology or the Microbiological Diagnostic Unit at the University of Melbourne for subtyping and phage typing.

Results

Epidemiological investigation

We contacted 95% (76/80) of the diners who attended the restaurant over the designated dates as well as 3 staff (response rate 95%, 79/83). Four diners were uncontactable. Of the 79 people we interviewed, 21 met the case definition (9 confirmed and 12 probable), representing an attack rate of 27%. Fifteen people sought medical attention and 7 presented to hospital. Two other diners became ill over a week after eating at the restaurant from presumed secondary transmission after caring for sick family members in the home.

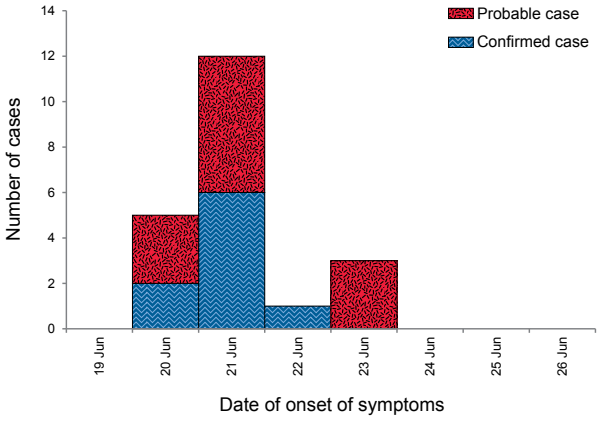
Demographic details for outbreak cases are shown in Table 1. There was no statistically significant difference between males and females becoming ill (RR 1.3, 95% CI 0.4–4.17, $P = 0.6$) but people who became ill were more likely to be younger (median age 30 years, range 16–74) than those that didn't become ill (median age 39 years, range 18–94) ($P < 0.05$).

In addition to diarrhoea, outbreak cases reported abdominal pain (95% 20/21), fever (95% 20/21), nausea (95% (20/21), vomiting (43% (9/21) and bloody diarrhoea (5% 1/21) (Table 1). The median incubation period was 21 hours (range of 4 to 60 hours). The epidemic curve was typical of a point source salmonellosis outbreak (Figure).

Table 1: Demographic characteristics and symptoms of outbreak cases that ate at a Darwin restaurant (n=21)

Characteristic	n	%
Gender		
Male	12	57
Female	9	43
Symptoms		
Diarrhoea	21	100
Abdominal pain	20	95
Fever	20	95
Nausea	20	95
Lethargy	19	90
Sought medical attention	15	71
Headache	13	62
Vomiting	9	43
Hospitalised	7	33
Bloody diarrhoea	1	5

Figure: Epidemiological curve of outbreak cases by onset day after eating at a Darwin restaurant. (n=21)



Attack rates for various exposures are shown in Table 2. On both nights the restaurant served a degustation menu and an *a la carte* menu. All cases ate from the degustation menu and this was significantly associated with illness (OR 11.8, 95% CI 1.8–∞, $P < 0.01$). The degustation menu consisted of 5 courses; duck prosciutto, papardelle, snapper, lamb ratatouille and a dessert of either crème brulee or a chocolate brownie. Duck prosciutto was found to be significantly associated with illness (OR 18.6, 95% CI 3.0–∞, $P < 0.01$). Duck prosciutto was consumed by every person who became ill and as a result, multivariate analysis was unable to be performed.

Environmental health investigation

The initial environmental health inspection of the premises observed that all cool room and refrigerator temperatures in the kitchen were satisfactory and that the structural condition and equipment in food production areas were suitable for the processing of safe food. The kitchen did not produce any foods containing raw or semi-cooked egg products.

A second environmental health inspection focused on the preparation of the degustation menu and in particular the preparation of the duck prosciutto from duck fillets, which was done on site. Duck fillets were cured in salt and brown sugar for a period of 2 days at room temperature in the kitchen and then dried for 24 hours in what was reported by restaurant staff to be a 'cool' dry area, again at room temperature. This area was deemed by EHOs to be unsatisfactory as it was neither cool nor dry and presented a risk of external contamination due to its location in a busy corridor separate from the kitchen. The duck prosciutto was then cryovacced and re-frozen. The environmental health investigation also determined that the cleaning and sanitising of the cryovac machine was not sufficient. The duck prosciutto would then be removed from the freezer to be cut and served as required. Duck prosciutto was removed from the menu as a result of the second inspection on 30 June 2015 and the restaurant was prohibited from preparing any prosciutto on site. Formal legal notices were issued to the restaurant for breaches of the Northern Territory *Food Act,*[3] requiring improved food handling skills and implementation of food safety systems including improved cleaning and sanitising practices.

Laboratory investigation

The samples of duck prosciutto and duck meat collected were from the same lot that was served on the nights implicated in the outbreak. Laboratory testing did not detect the presence of *E. coli*, *Salmonella*, *Listeria* or thermophilic *Campylobacter* in the samples of frozen duck meat or duck prosciutto. The SPC reading for the frozen duck fillets was 1.7×10^3 organisms per gram. The SPC reading for the frozen duck prosciutto was 2×10^7 organisms per gram and the frozen duck prosciutto contained a coliform count of 1.1×10^3 organisms per gram. This increase in bacteria during processing of the duck to prosciutto confirms that poor temperature control and improper drying and storage resulted in an unsatisfactory level of micro-organisms in the food.[4]

All 9 stool samples collected from people who were ill tested positive for STm9.

Table 2: Univariable analysis of selected exposures for salmonellosis at a Darwin restaurant (n=79)

	Exposed			Unexposed					
Food eaten	Cases	Total	AR%	Cases	Total	AR%	RR	95% CI	*P* value
Degustation menu	18	57	32	0	19	0	11.8*	1.8–∞*	0.0039†
A la carte menu	0	19	0	18	57	32	0.0*	0-0.5*	0.0025†
Duck prosciutto	21	56	38	0	23	0	18.6*	3.0–∞*	0.0002†
Parpadelle	19	40	32	2	20	10	3.2	0.8-12.6	0.0438†
Lamb ratatouille	20	43	32	1	16	6	5.1	0.7-35.1	0.0327
Snapper	19	60	32	2	19	11	3.0	0.8-11.8	0.0587†
Crème brulee	8	32	25	12	45	27	0.9	0.4-2.0	0.8694
Chocolate brownie	12	35	34	8	42	19	1.8	0.8-3.9	0.1289

AR = Attack rate

RR = Relative risk

CI = Confidence interval

* Odds ratio and confidence intervals calculated using exact logistic regression.

† Fisher's exact

Discussion

The results of the epidemiological investigation implicated the consumption of duck prosciutto as the cause of this outbreak; it had a highly significant relative risk and was eaten by all cases. Although no *Salmonella* was detected in samples of frozen duck fillet or frozen duck prosciutto, an unsafe SPC reading of 2×10^7 organisms per gram from samples of duck prosciutto indicates that the prosciutto was contaminated.

Non-typhoidal *Salmonella* bacteria are carried in the intestines of wild and domestic animals including ducks and other poultry. Humans become infected through eating contaminated meat or eggs from those animals or from coming in contact with an environment that has been contaminated by those animals.[5] In Australia, *S.* Typhimurium PT9 has been previously detected in samples of duck eggs as well as duck carcasses and offal.[6] An efficient kill-step such as cooking or curing prevents these bacteria infecting humans. Generally, the process of curing meats should involve meat with a pH > 6 undergoing a dry salting process followed by drying at low temperatures (10°C to 15°C) and low relative humidity (70% to 85%). The salt level and low temperature control growth in the early stage of the process and then the drying at low temperature and relative humidity should inactivate some pathogens and inhibit growth of others.[7]

Between 2001 and 2015, there have been few outbreaks of salmonellosis in Australia associated with duck meat or eggs; 1 *S.* Typhimurium PT9 outbreak caused by duck eggs, 1 *S.* Typhimurium (not phage typed) outbreak caused by duck's gizzards, 2 *S.* Typhimurium outbreaks (PT135 and MLVA 03-25-16-11-524) caused by duck paté and 1 *S.* Typhimurium PT11 outbreak caused by roast duck (Unpublished data from the OzFoodNet Outbreak Register; 2001–2015). We have been unable to find any previously reported outbreaks linked to duck prosciutto either in Australia or overseas.

A large number of bacteria were detected in the samples of duck prosciutto but not in the frozen duck fillets from which it was prepared. This is strong evidence that contamination occurred during the drying and curing process. Given that poultry is a known high-risk food for *Salmonella* infection, we believe that it is likely that small amounts of bacteria in the frozen duck fillets that were below detectable limits, could have multiplied during the curing and drying process. However, we cannot discount the possibility that bacteria were introduced to the duck meat during the curing and drying process by improper and unhygienic handling, applying a curing solution that potentially promoted microbial growth, or undertaking the drying and curing process in an area that allowed for potential contamination to occur. The environmental health investigation observed that the curing and drying of the duck fillets occurred at non-air-conditioned room temperature (Darwin median maximum June temperature 30.6°C)[8] and not at the recommended 10°C to 15°C.[7] The site where the curing and drying of the duck prosciutto occurred was not a low humidity environment and occurred in an area with high volumes of human traffic, which could have contributed to external contamination of the meat.

The risk of salmonellosis from poultry, particularly chicken, is recognised at the primary production level.[9] Food Standards Australia New Zealand assume the hazards of concern in all poultry species are largely the same as those for chicken.[10] Poultry has a higher *Salmonella* risk rating than red meat[7] and as a result, food handlers need to possess good food knowledge and follow well documented food safety procedures. Restaurants should evaluate the risk of preparing their high risk raw foods on site as opposed to purchasing from producers who have expertise in the field. This outbreak investigation highlights the risk that occurs when raw high-risk foods are prepared by staff who lack familiarity with the scientific processes that underpin preparation methods, are inadequately trained and operate without food safety systems.

We minimised measurement bias by using a standard questionnaire based on the menu at the restaurant with prompts for all food items, including individual food items on each plate. The restaurant is considered a prestigious venue and more likely to be only frequented by diners on special occasions and this probably improved participant recall. Selection bias was minimised through having access to a full booking list from the restaurant and the study's high response rate. We were able to contact almost the entire cohort of persons who ate at the restaurant over the 2 evenings.

A major limitation of the investigation is that *S.* Typhimurium PT9 was not detected in food samples collected from the restaurant. Only 2 samples of duck prosciutto and frozen duck fillets were collected for sampling. The distribution of contamination throughout a sample is not uniform and this limited amount of sampling reduced the sensitivity of food testing in this outbreak investigation.

Conclusion

This outbreak of *Salmonella* Typhimurium PT9 at a Darwin restaurant was most likely caused by consumption of duck prosciutto. The duck pro-

sciutto was likely contaminated during the drying and curing process, which was conducted under conditions that facilitated the introduction, growth and survival of bacteria. This investigation highlights the importance of restaurants establishing and implementing food safety procedures in order to limit the potential for such outbreaks to occur. Scientific principles underpin safe food handling process and it is important not to deviate from safe methods, particularly when preparing high risk foods which are becoming increasingly popular in Australian restaurants and homes.

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References

1. OzFoodNet Working Group. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet network, 2011. *Commun Dis Intell* 2015;39(2):E236–E364.
2. Northern Territory *Public and Environmental Health Act 2011*. Available from: http://www.austlii.edu.au/au/legis/nt/num_act/paeha20117o2011337/ Accessed on 20 January 2016.
3. Northern Territory *Food Act*. Available from: http://www.austlii.edu.au/au/legis/nt/consol_act/fa57/ Accessed on 20 January 2016.
4. Food Standards Australia New Zealand. Guidelines for microbiological examination of ready-to-eat foods. Canberra: Food Standards Australia New Zealand, 2001.
5. Salmonellosis. In: Heymann DL, ed. *Control of Communicable Diseases Manual*. 20th edn. Washington: American Public Health Association; 2015.
6. Powling J, Howden B. National Enteric Pathogens Surveillance System Non-Human Annual Report 2011. Melbourne; 2012.
7. Meat and Livestock Australia. Guidelines for the safe manufacture of smallgoods. 2nd edn. North Sydney: Meat and Livestock Australia, 2015. Available from: https://www.mla.com.au/Research-and-development/Search-RD-reports/RD-report-details/Product-Integrity/2nd-edition-Guidelines-for-the-Safe-Manufacture-of-Smallgoods/1152
8. Australian Bureau of Meteorology. Climate statistics for Australian Locations – Summary statistics Darwin Airport. Australian Bureau of Meteorology, 2016. Available from: http://www.bom.gov.au/climate/averages/tables/cw_014015.shtml
9. Australian Government. Australia New Zealand Food Standards Code – Standard 4.2.2 – Primary Production and Processing Standard for Poultry meat (Australia Only). Available from: https://www.comlaw.gov.au/Details/F2012L00292
10. Food Standards Australia New Zealand. Initial assessment report – Primary Production and Processing Standard for Poultry Meat. Canberra: Food Standards Australia New Zealand, 2004. Available from: http://www.foodstandards.gov.au/code/proposals/Pages/proposalp282primaryp2442.aspx

The molecular epidemiology of norovirus outbreaks in Victoria, 2014 to 2015

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Abstract

Noroviruses are a leading cause of outbreaks of gastroenteritis. This study examined the incidence and molecular characteristics of norovirus outbreaks in healthcare and non-healthcare settings in Victoria, Australia, over 2 years (2014–2015). Norovirus was detected in 65.7% and 60.4% of gastroenteritis outbreaks investigated for the years 2014 and 2015 respectively. There was a significant decline in the number of norovirus outbreaks in the period 2014 to 2015 although in both years norovirus outbreaks peaked in the latter part of the year. Norovirus Open Reading Frame (ORF) 2 (capsid) genotypes identified included GI.2, GI.3, GI.4, GI.5, GI.6, GI.9, GII.2, GII.3, GII.4, GII.6, GII.7, GII.8, GII.13 and GII.17. GII.4 was the most common genotype detected. In addition, the following ORF 1/ORF 2 recombinant forms were confirmed: GII.P4_NewOrleans_2009/GII.4_Sydney_2012, GII.P12/GII.3, GII.Pb (GII.21)/GII.3, GII.Pe/GII.2 and GII.Pe/GII.4_Sydney_2012. A significant decline was noted in the chief norovirus strain GII.Pe/GII.4_Sydney_2012 between 2014 and 2015 but there was a re-emergence of a GII.P4_NewOrleans _2009 norovirus strain. Outbreaks involving the GII.P17/GII.17 genotype were also detected for the first time in Victoria. GI genotypes circulating in Victoria for the 2 years 2014 and 2015 underwent a dramatic change between the 2 years of the survey. Many genotypes could occur in both healthcare and non-healthcare settings although GI.3, GII.6, and GII.4 were significantly more common in healthcare settings. The study emphasises the complex way in which norovirus circulates throughout the community. *Commun Dis Intell* 2017;41(1):E21–E32.

Keywords: norovirus, outbreaks, genotypes, healthcare, non-healthcare, setting, RT-PCR, nucleotide sequencing

Introduction

Noroviruses are single-stranded positive sense RNA viruses, classified in the genus *Norovirus* within the Family Caliciviridae.[1] Noroviruses are currently classified into 6 genogroups of which genogroups I, II and IV (GI, GII, GIV) occur in human infections.[1] The incidence and clinical significance of GIV noroviruses in human infections are little understood.[2]

Noroviruses are now recognised as a major cause of morbidity and mortality and can cause gastroenteritis in individuals of all ages; it has been estimated that, annually, norovirus infection causes 70,000 to 200,000 deaths around the world.[3] Globally, norovirus is considered to be associated with approximately one-fifth of all diarrhoea cases[4] and in Australia, norovirus appears to be the major known gastroenteritis pathogen.[5] Although vaccine strategies against norovirus are under development,[3] the genetic diversity of the noroviruses has complicated this process.[3,6]

The human norovirus genome comprises 3 open reading frames (ORFs).[1] ORF 1 encodes the non-structural polyprotein, ORF 2 the major capsid protein and ORF 3 the minor capsid protein.[1] Norovirus genotype classification can be based on the ORF 1 region or the ORF 2 region[7] but recombination can occur at the ORF 1–ORF 2 intersect[8] so in some recombinant noroviruses the ORF 1 and ORF 2 genotypes are different.

At least 29 ORF 2 human norovirus genotypes have been identified[1] although the GII.4 genotype appears to be the most common norovirus genotype in human disease.[3,9] Furthermore, GII.4 noroviruses undergo mutation and recombination such that a major new GII.4 variant epidemic strain normally appears every 2 to 4 years.[3] It is therefore of interest that recent studies in China[10,11] and Japan[12] indicate that a new epidemic strain, GII.17, may have emerged. These observations prompted de Graaf et al.[13] to raise the question whether the emergence of a novel GII.17 norovirus is a sign that the GII.4 era was coming to an end.

Overview studies of norovirus molecular epidemiology remain an area of active interest in Australia.[14,15] The current report extends previous work by examining the characteristics of norovirus outbreaks in Victoria in 2014 to 2015 and their associated genotypes. In particular the study examined quantitative and qualitative aspects of 3 areas: what is the relationship between seasonality and norovirus incidence; what norovirus genotypes were detected and how did they change over time and what was the relationship between norovirus genotype and outbreak setting.

Materials and methods

Definition of gastroenteritis outbreak

For the purposes of this study an outbreak was defined as a gastroenteritis cluster that was apparently associated with a common event or location and in which 4 or more individuals had symptoms of gastroenteritis. For an outbreak in a particular setting to be so defined, at least 2 individuals had to develop gastroenteritis within 4 days of each other and for an outbreak linked to a suspect food source, at least 2 individuals had to develop gastroenteritis within 4 days of consuming the suspect food.

Specimens

The faecal specimens included in this study were those sent to the Victorian Infectious Diseases Reference Laboratory (VIDRL) for norovirus testing from outbreaks that occurred during 2014 to 2015. VIDRL, which is National Association of Testing Authorities, Australia accredited, is the main public health laboratory for viral identification in the state of Victoria. As such, it receives faecal material from gastroenteritis outbreaks reported to the Victorian Health Department. Outbreak specimens are also occasionally sent by other institutions such as hospitals. Only outbreaks that occurred in Victoria were included in the study.

Faecal processing, RNA extraction and reverse transcription-polymerase chain reaction testing

Faecal specimens were prepared as a 20% (vol/vol) suspension in Hanks' complete balanced salt solution (Sigma-Aldrich Company, Irvine, UK) and the suspension clarified with a single 10 minute centrifugation as previously described.[16] This clarified extract was then used for RNA extraction followed by reverse transcription-polymerase chain reaction (RT-PCR). RNA extraction was carried out using the Corbett automated extraction procedure (now Qiagen Sciences, Germantown, MD, USA).[17]

Six 2-round RT-PCR procedures (protocols 1 to 6; Table 1) were then used for norovirus detection. For the first round of each of the 6 protocols the Qiagen (GmbH, Hilden, Germany) One step RT-PCR kit that combined the RT step and the first round PCR was utilised. For the second round PCR the Qiagen *Taq* DNA polymerase kit was used. All PCR protocols utilised a GeneAmp PCR System 9700 thermocycler (Applied Biosystems, Foster City, CA, USA).

Nucleotide sequencing and genotype classification

Nucleotide sequencing was carried out for protocols 1, 2, 3 and 5 (Table 2). Protocol 4 was directed to the possibility of detecting a GI-GI recombinant and protocol 6 to the possibility of detecting a GII-GI recombinant but no PCR product was obtained with either protocol. Sequence analysis made use of the software MacVector (Oxford Molecular Limited, Madison, WI) and genotyping made use of the norovirus genotyping tool (http://www.rivm.nl/mpf/norovirus/typingtool).[24]

Experimental plan

All faecal specimens in the study were initially tested by the protocol 1 RT-PCR. Nucleotide sequencing was carried out on 1 positive specimen, chosen at random, from each outbreak. One specimen from every outbreak was also tested by both protocols 2 and 3 (ORF 2 GI and ORF 2 GII RT-PCRs). Nucleotide sequencing was then performed on all positive norovirus specimens from protocols 2 and 3. In addition ORF 1–ORF 2 RT-PCRs (protocols 4–6) were carried out to confirm the recombination status of specimens where the ORF 1 and ORF 2 RT-PCR protocols gave different genotypes.

Statistical analysis

Statistical analysis was carried out using the χ^2 test[25] and the partitioning of χ^2 test.[26] For the partitioning of χ^2 test 3 terms, 'period', 'grouping' and 'class' were defined as follows. A period was one of the 2-month intervals for which the outbreak frequency was used in the analysis. A grouping was a set of genotypes combined so that the total frequency was sufficient for the χ^2 test to be valid. A class was the set of 2-monthly periods or groupings such that the individual periods or groupings did not differ significantly from each other in the characteristic of interest.

To investigate the change in the number of norovirus outbreaks between 2014 and 2015, the null hypothesis was that the number of outbreaks was the same in each year and equal to half of the total number of outbreaks in 2014 to 2015. The χ^2 test (1 degree of freedom) was then applied. If the probability was less than 0.05 the difference was taken to be significant.

To investigate the distribution of norovirus outbreaks among different months of the year separately for 2014 and 2015, the partitioning of χ^2 test was used. First, the months were combined in pairs, to smooth out fluctuations, and the null hypothesis was that the number of outbreaks was

Table 1: Reverse transcription-polymerase chain reaction (RT-PCR). protocols used

Genogroup detected (Protocol number)	ORF	Primers (5' to 3')*	Comments	References	Fragment size for genotype analysis (position relative to reference strain)
GI and GII (protocol 1)	ORF 1	**NV 4562** GAT GCD GAT TAC ACA GCH TGG G **NV 4611** CWG CAG CMC TDG AAA TCA TGG **NV 4692** GTG TGR TKG ATG TGG GTG ACT TC **NV 5296** CCA YCT GAA CAT TGR CTC TTG **NV 5298** ATC CAG CGG AAC ATG GCC TGC C **NV 5366** CAT CAT CAT TTA CRA ATT CGG	Two-round hemi-nested RT-PCR both detects and distinguishes between GI and GII noroviruses.	Yuen et al.[18] Bruggink et al.[19]	440bp (4484–4923†)
GI (protocol 2)	ORF 2	**COG1F** CGY TGG ATG CGN TTY CAT GA **G1SKR** CCA ACC CAR CCA TTR TAC A	Two-round RT-PCR. The second round is a booster step, which uses the same primers as the first round.	Bruggink et al.[19] McIver et al.[20]	198bp (5415–5612‡)
GII (protocol 3)	ORF 2	**G2F3** TTG TGA ATG AAG ATG GCG TCG A **G2SKR** CCR CCN GCA TRH CCR TTR TAC AT	Two-round RT-PCR. The second round is a booster step, which uses the same primers as the first round.	McIver et al.[20] Dunbar et al.[21]	195bp (5133–5327†)
GI-GI (protocol 4)	ORF 1-ORF 2	**NV 4562** GAT GCD GAT TAC ACA GCH TGG G **G1SKR** CCA ACC CAR CCA TTR TAC A	Two-round RT-PCR. The second round is a booster step, which uses the same primers as the first round.	Bruggink et al.[22]	NA
GII-GII (protocol 5)	ORF 1-ORF 2	**NV 4692** GTG TGR TKG ATG TGG GTG ACT TC **G2SKR** CCR CCN GCA TRH CCR TTR TAC AT	Two-round RT-PCR. The second round is a booster step, which uses the same primers as the first round.	Bruggink et al.[22]	345–753bp (in range 4484–5273†)
GII-GI (protocol 6)	ORF 1-ORF 2	**NV 4692** GTG TGR TKG ATG TGG GTG ACT TC **G1SKR** CCA ACC CAR CCA TTR TAC A	Two-round RT-PCR. The second round is a booster step, which uses the same primers as the first round.	Bruggink et al.[23]	NA

* D=AGT, H=ACT, W=AT, M=AC, R=AG, K=GT, Y=CT, N=AGCT

† Reference strain Camberwell (accession number AF145896)

‡ Reference strain Norwalk (accession number M87661)

NA = not applicable

Table 2: Settings of norovirus positive outbreaks 2014 to 2015

Healthcare settings	Number of norovirus positive outbreaks	Percentage of healthcare outbreaks	Percentage of total norovirus positive outbreaks
Aged care facility	199	85.8	69.3
Disabled care facility	10	4.3	3.5
Early parenting centre	2	0.9	0.7
Hospital	16	6.9	5.6
Hospital – Maternity Ward	1	0.4	0.3
Hospital – Psychiatric Ward	3	1.3	1.0
Hospital – Rehabilitation Unit	1	0.4	0.3
Total	232	100.0	80.8
Non-healthcare settings	**Number of norovirus positive outbreaks**	**Percentage of non-healthcare outbreaks**	**Percentage of total norovirus positive outbreaks**
Camp	1	1.8	0.3
Camp – school	2	3.6	0.7
Childcare centre	27	49.1	9.4
Gathering	6	10.9	2.1
Restaurant	12	21.8	4.2
School	1	1.8	0.3
Special accommodation	2	3.6	0.7
Suspect food	4	7.3	1.4
Total	55	100.0	19.2

the same in each 2-monthly period. The χ^2 test (5 degrees of freedom) was then applied. In 2014 there was a difference in norovirus incidence for different 2-monthly periods ($\chi^2 = 30.53$, 5 degrees of freedom, $P = 0.00001$) so that it was valid to partition χ^2. In 2015 there was also a difference in norovirus incidence for different 2-monthly periods ($\chi^2 = 37.74$, 5 degrees of freedom, $P = 0.0000004$) so that it was again valid to partition χ^2. Secondly, to partition χ^2 for each year, 2-monthly periods were combined to give classes, with the null hypothesis that all the 2-monthly periods in each class had the same number of outbreaks. The 2-monthly periods for each class were chosen so that when a χ^2 test was applied with the null hypothesis used, the value of χ^2 was small and it could be taken that there was no significant difference between the number of outbreaks in the 2-monthly periods in the class. Thirdly, a new table was created with each entry being the total number of outbreaks in a class covering the total months corresponding to the class. A χ^2 test was applied to this new table with the null hypothesis being that the number of outbreaks per 2-monthly period was the same for all classes in the table. If the value of χ^2 was sufficiently high to correspond to a probability of less than 0.05 it was taken that there was a difference in the incidence per 2-monthly period of the different time intervals for the table.

To investigate the significance of the change in the proportion of norovirus outbreaks of the 2 major ORF 1/ORF 2 genotypes (GII.Pe/GII.4_Sydney_2012 and GII.Pe/GII.4) between 2014 and 2015 the ORF 1/ORF 2 genotypes were first divided into 3 groupings, GII.Pe/GII.4_Sydney_2012, GII.Pe/GII.4 and the other genotypes ('other genotypes'). The null hypothesis was that the proportion of outbreaks due to the 3 genotype groupings was the same in 2014 and in 2015. The χ^2 test (2 degrees of freedom) was then applied. A significant difference was found ($\chi^2 = 6.162$, 2 degrees of freedom, $P = 0.046$) so that partitioning of χ^2 could be applied. On this basis the genotype groupings could then be grouped into 2 classes, with GII.Pe/GII.4_Sydney_2012 and GII.Pe/GII.4 in the first class and 'other genotypes' in the second class. For the first class genotypes, the null hypothesis was that the proportion of the outbreaks associated with each of the 2 genotypes was the same in 2014 as in 2015. This null hypothesis was confirmed ($\chi^2 = 0.005$, 2 degrees of freedom, $P = 0.997$). Then a new table was set up, with each row giving the numbers of outbreaks in 2014 and 2015 for 1 of the 2 classes. The null hypothesis was that the proportion of outbreaks for each of the 2 classes was the same in 2014 and 2015. Application of the χ^2 test indicated the null hypothesis was not supported ($\chi^2 = 6.157$, 2 degrees of freedom, $P = 0.046$).

To investigate whether the proportion of outbreaks of a particular genotype was higher in a particular type of setting (healthcare vs non-healthcare) than for other genotypes, a table was set up giving the frequencies of outbreaks in the healthcare and non-healthcare settings for each genotype. The frequencies of the minor genotypes were then combined so that frequencies in the table were sufficiently high that the χ^2 test could be validly used. This gave a table with 5 groupings of genotypes, GI.3, GII.4, GII.6, GII.3 and 'other genotypes'. The null hypothesis was that the proportion of outbreaks in healthcare settings (and consequently the proportion of outbreaks in non-healthcare settings) was the same for each of the 5 groupings of genotypes. The χ^2 test was then applied. As there were significant differences in the fraction of outbreaks in healthcare settings among these 5 groupings of genotypes ($\chi^2 = 24.16$, 4 degrees of freedom, $P = 0.00007$), partitioning of χ^2 could be applied. On this basis, the genotype groupings could then be organised into classes, with the null hypothesis that the proportion of outbreaks in healthcare settings for each genotype grouping in the class was the same for all genotype groupings in the class. The first class comprised GI.3, GII.4 and GII.6 and a χ^2 test applied to the genotype groupings in the class gave $\chi^2 = 2.45$, 4 degrees of freedom, $P = 0.65$. The second class comprised GII.3 and 'other genotypes' and a χ^2 test applied to the genotype groupings in the class gave $\chi^2 = 0.83$, 4 degrees of freedom, $P = 0.935$. Thus the null hypothesis was confirmed for each class and the proportion of outbreaks in healthcare settings for each genotype grouping in the class could be taken to be the same.

A new table was set up, with each row giving the numbers of outbreaks in healthcare and non-healthcare settings for 1 of the 2 classes. The null hypothesis was that the proportion of outbreaks in healthcare settings (and in non-healthcare settings) for all the classes, was the same and the χ^2 test was applied to the new table. A high value of χ^2 corresponding to a probability of less than 0.05 was taken to indicate that the classes differed in the proportion of outbreaks of that genotype in healthcare settings (and consequently in non-healthcare settings) and this was found to be the case ($\chi^2 = 21.01$, 4 degrees of freedom, $P = 0.00003$). Each class could then be considered as a whole to determine the relative frequency in healthcare and non-healthcare settings. The first class was then tested to determine whether genotypes were more prevalent in healthcare than in non-healthcare settings, the null hypothesis being that the number of outbreaks in healthcare settings was the same as the number of outbreaks in non-healthcare settings. The null hypothesis was not supported ($\chi^2 = 102.76$, 1 degree of freedom, $P = 5 \times 10^{-24}$).

Ethics

Data collection for the current study is covered by public health legislation and specific ethics approval was not required. No information is given that would allow the identification of any individuals in the study.

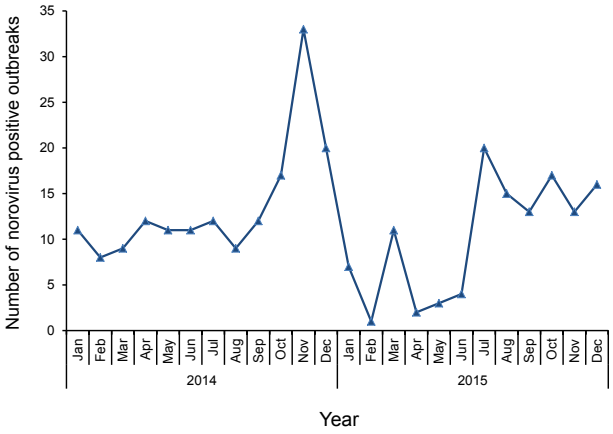
Results

Norovirus outbreak incidence, seasonal periodicity and setting

For the calendar year 2014, specimens from 251 gastroenteritis outbreaks were received for testing and of these 165 (65.7%) were positive for norovirus by the ORF 1 PCR (protocol 1) and/or an ORF 2 PCR (protocols 2 and 3). For the calendar year 2015, specimens from 202 gastroenteritis outbreaks were received for testing and of these 122 (60.4%) were positive for norovirus by the ORF 1 PCR (protocol 1) and/or an ORF 2 PCR (protocols 2 and 3). Thus norovirus was the chief viral agent associated with gastroenteritis outbreaks in Victoria for both 2014 and 2015. However, there was a significant decline in the number of norovirus outbreaks identified in 2015 compared with 2014 ($\chi^2 = 6.44$, 1 degree of freedom, $P = 0.011$).

The seasonal periodicity of all norovirus outbreaks for the period 2014 to 2015 is given in Figure 1. For 2014, partitioning of χ^2 was applied and it was found that outbreak incidence did not change significantly in the period January–October ($\chi^2 = 2.64$, 5 degrees of freedom, $P = 0.75$). However, there was a significant difference in incidence between January–October and November–December

Figure 1: Number of norovirus positive outbreaks (i.e. norovirus positive by ORF 1 and/or ORF 2 RT-PCR) per month for the years 2014 to 2015



($\chi^2 = 28.37$, 5 degrees of freedom, $P = 0.00003$), and the incidence rose significantly in the period November–December.

For 2015, partitioning of χ^2 was also applied. Outbreak incidence did not change significantly in the period January–June ($\chi^2 = 2.22$, 5 degrees of freedom, $P = 0.82$) and did not change significantly in the period July–December ($\chi^2 = 0.66$, 5 degrees of freedom, $P = 0.985$). However, there was a significant difference in incidence between January–June and July–December ($\chi^2 = 35.70$, 5 degrees of freedom, $P = 0.000001$), so that the incidence rose significantly in the period July–December compared with January–June.

Therefore in both years the number of outbreaks rose in the latter part of the year, but the time when the rise occurred was not the same in 2015 as in 2014.

Norovirus outbreak settings could be divided into 2 groups: healthcare and non-healthcare (Table 2). Although most outbreaks came from aged care facilities, a large number of settings were represented in the study. It was notable that the percentage of norovirus outbreaks from the healthcare and non-healthcare categories was similar in 2014 and 2015. In particular, in 2014, 135 (81.3%) of the norovirus outbreaks were from healthcare settings and 31 (18.7%) from non-healthcare settings. In 2015, 98 (80.3%) of the norovirus outbreaks were from healthcare settings and 24 (19.7%) from non-healthcare settings. Thus any alteration in frequency patterns of the various genotypes detected could not have resulted from altered sampling patterns.

Norovirus genotype analysis

A summary of all ORF 1 and ORF 2 norovirus genotypes identified in the study is given in Table 3. It can be seen that a broad range of norovirus genotypes were detected. In terms of ORF 2 (capsid) genotypes, the frequencies, in descending order, were: GII.4 (144/229), GI.3 (18/229), GII.6 (18/229), GII.3 (16/229), GI.2 (7/229), GII.2 (7/229), GII.17 (6/229), GI.4 (3/229), GI.9 (3/229), GII.7 (2/229), GI.5 (1/229), GI.6 (1/229), GII.8 (1/229) GII.13 (1/229) and the mixed outbreak GI.3 plus GII.3 (1/229).

The ORF 1 genotype sometimes differed from the ORF 2 genotype in a given outbreak (Table 3). To test whether these variable genotype combinations represented ORF 1/ORF 2 recombinant forms, nucleotide sequencing in the ORF 1-ORF 2 intersect region was carried out and the following recombinant forms were confirmed: GII.P4_NewOrleans_2009/GII.4_Sydney_2012, GII.P12/GII.3, GII.Pb(GII.P21)/GII.3, GII.Pe/GII.2 and GII.Pe/GII.4_Sydney_2012. A representative sequence of each of these 5 recombinant genotypes has been deposited in GenBank with the accession numbers KX064756 to KX064760 respectively.

There were significant changes in the incidence of some genotypes over the 2-year period 2014 to 2015, notably the decline in the incidence of GII.Pe/GII.4_Sydney_2012 and GII.Pe/GII.4, the re-emergence of GII.P4_NewOrleans_2009, the emergence of GII.17 norovirus and a dramatic alteration in the mix of GI norovirus genotypes (Table 3). These 4 areas are next considered.

Decline in GII.Pe/GII.4_Sydney_2012 and GII.Pe/GII.4 strains over 2014 to 2015

The chief norovirus strains over the period of the study, GII.Pe/GII.4_Sydney_2012 and GII.Pe/GII.4, declined in incidence from 2014 to 2015 (Table 3). In 2014, these strains were found in 60/117 (51.3%) and 27/117 (23.1%) respectively of outbreaks with both ORF 1 and ORF 2 sequence available, whereas in 2015 they were found in 32/80 (40.0%) and 14/80 (17.5%) respectively of outbreaks with both ORF 1 and ORF 2 sequence available. Application of the partitioning of χ^2 test then showed that the 2 chief genotypes did decline significantly from 2014 to 2015 ($\chi^2 = 6.157$, 2 degrees of freedom, $P = 0.046$).

Re-emergence of GII.P4_NewOrleans_2009

During the study period there was a re-emergence of the ORF 1 form GII.P4_NewOrleans_2009 norovirus. GII.P4_NewOrleans_2009 was not detected in 2014 and was first identified in July 2015; thereafter it was detected in a further 5 outbreaks. The 6 outbreaks occurred in a range of settings, 4 in healthcare settings and 2 in non-healthcare settings.

Three of the 6 ORF 1 GII.P4_NewOrleans_2009 norovirus strains were found to be linked to the ORF 2 genotype GII.4_Sydney_2012 (Table 3). Sequence analysis of the ORF 1 fragment showed 98.2% to 98.6% similarity with the reference strain GII.P4_NewOrleans_2009 (GU445325); sequence analysis of the ORF 2 fragment showed 97.4% to 97.9% similarity with the reference strain GII.4_Sydney_2012 (JX459908). Application of the bridging RT-PCR protocol 5 (Table 1) for 1 specimen confirmed GII.P4_NewOrleans_2009/GII.4_Sydney_2012 was a true recombinant form.

Emergence of GII.17 norovirus

Six outbreaks involving the GII.P17/GII.17 genotype were detected (Table 3). In 2014, there were 2 such outbreaks, both in aged care facilities. In

2015, 3 of 4 outbreaks occurred in aged care facilities and 1 in a boarding school. GII.P17/GII.17 could infect individuals over a broad range of ages; of 11 individuals from 6 outbreaks where ages were available and nucleotide sequences for both the ORF 1 and ORF 2 regions were obtained, the spread of ages was 17 to 95 years.

Sequence analysis indicated GII.17 norovirus underwent minor changes in 2014 to 2015. Sequence analysis (protocols 1 and 3; Table 1) indicated that both the GII.17 ORF 1 and ORF 2 regions respectively of the two 2014 GII.P17/GII.17 noroviruses were identical. When the 2014 ORF 1 region was compared with the ORF 1 region of the four 2015 GII.P17/GII.17 noroviruses, they were found to have 97.3% to 98.0% similarity; 1 of the nucleotide differences resulted in an amino acid change. When the 2014 GII.17 ORF 2 region was compared with the GII.17 ORF 2 region of the four 2015 GII.P17/GII.17 noroviruses, they were found to have 97.4% to 98.5% similarity; none of the nucleotide differences resulted in an amino acid change.

Table 3: Genotypes found in norovirus positive outbreaks 2014 to 2015

ORF 1	ORF 2	Number of norovirus positive outbreaks		
		2014	2015	Total
GI.P2	GI.2	7	0	7
GI.P3	GI.3	2	15	17
GI.P3	–	0	1	1
GI.P4	GI.4	3	0	3
GI.P5	GI.5	1	0	1
GI.P6	GI.6	1	0	1
GI.P9	GI.9	3	0	3
GI.Pa	GI.3	1	0	1
GII.P4_NewOrleans_2009	GII.4_Sydney_2012	0	3	3
GII.P4_NewOrleans_2009	GII.4*	0	1	1
GII.P4_NewOrleans_2009	–	0	2	2
GII.P12	GI.3 & GII.3	1	0	1
GII.P12	GII.3	3	6	9
GII.P16	GII.4*	0	1	1
GII.P16	GII.13	1	0	1
GII.P17	GII.17	2	4	6
GII.P17	–	0	1	1
GII.Pb (GII.P21)	GII.3	4	3	7
GII.Pb (GII.P21)	–	2	0	2
GII.Pe	GII.2	1	1	2
GII.Pe	GII.4_Sydney_2012	60	32	92
GII.Pe	GII.4*	27	14	41
GII.Pe	–	27	19	46
–	GII.2	0	5	5
–	GII.4_Sydney_2012	3	0	3
–	GII.4*	0	3	3
–	GII.6	11	7	18
–	GII.7	0	2	2
–	GII.8	1	0	1
–	–	4	2	6
Total		165	122	287

– No sequence available.

* GII.4 variant identity could not be determined by the norovirus genotyping tool.[24]

Nucleotide sequence analysis was also carried out to determine sequence similarity between the GII.P17/GII.17 strains found in this study and GII.P17/GII.17 strains recently identified in the United States of America (USA) and Japan. Analysis of a 753bp fragment corresponding to the ORF 1/ORF 2 intersect region showed a 2014 sequence from the current study was 98.4% similar to the USA strain KR083017[27] and 99.3% similar to the Japanese strain AB983218.[12] A similar analysis of 4 strains from the four 2015 GII.17 outbreaks from the current study showed a 99.3% to 99.7% similarity to KR083017[27] and a 98.1% to 98.5% similarity to AB983218.[12] Thus the Australian strains showed high similarity with the USA and Japanese strains.

One 753bp GII.P17/GII.17 sequence from an individual in the first GII.P17/GII.17 outbreak in 2015 has been lodged in GenBank as KT734635.

Alteration in genogroup I genotypes

Based on ORF 2 sequences it was noted that the proportion of genogroup I (GI) outbreaks in 2014 (14.4%) was similar to the proportion in 2015 (15.5%) (Table 3). However, examination of the GI genotypes circulating in Victoria for the 2 years 2014 and 2015 indicated a dramatic shift in the variety of genotypes detected from 2014 to 2015 (Table 3). In particular, in 2014, 7 GI ORF 1/ORF 2 genotype combinations were detected, whereas in 2015 only 1 GI ORF 1/ORF 2 genotype combination (GI.P3/GI.3) was detected.

An examination of representative GI.3 norovirus nucleotide sequences from both the ORF 1 and ORF 2 regions of the genome (Figure 2) indicates that there were substantial alterations in the genome in the period corresponding to the transition between 2014 and 2015. None of the 'definitive' nucleotide changes in either ORF 1 or ORF 2 resulted in an amino acid change (Figure 2).

It can be seen that for both ORF 1 sequences and ORF 2 sequences there was a major change early in 2015 and this is denoted by a horizontal line; the 3 sequences above the line correspond to the 2 GI.3 outbreaks in 2014 and the first GI.3 outbreak in 2015 and the 14 sequences below the line correspond to GI.3 outbreaks in the remainder of 2015. In Figure 2A (ORF 1) it can be seen that there were 25 (6%) definitive changes (i.e. a change that, once it had occurred, remained fixed for the rest of 2015) in a sequence 440 bp long. In Figure 2B (ORF 2) it can be seen there were 6 (3%) definitive changes in a sequence 198 bp long.

Relationship between ORF 2 genotype and outbreak setting

An examination of Table 4 indicates there was a relationship between some ORF 2 genotypes and outbreak setting and this was then examined by statistical methods. For the statistical analysis, 5 genotype groupings, based on genotype frequency, were chosen. These groupings were GI.3, GII.3, GII.4, GII.6 and all the other genotypes ('other genotypes'). Application of the partitioning of χ^2 test followed by the χ^2 test indicated that GI.3, GII.4 and GII.6 were much more common in healthcare settings than in non-healthcare settings ($\chi^2 = 102.76$, 1 degree of freedom, $P = 5 \times 10^{-24}$).

Discussion

The findings of the current study indicate that norovirus remained a common gastroenteritis virus that infected individuals in a broad range of settings. Norovirus outbreaks occurred throughout the year but a seasonal peak was noted in 2014 and 2015, although the timing was different.

A great diversity in norovirus genotypes was found to be circulating within Victoria in 2014 to 2015. This included 6 ORF 2 GI genotypes and 8 ORF 2 GII genotypes. The genotype diversity of circulating norovirus was further emphasised by the identification of 5 ORF 1/ORF 2 recombinant forms.

A key finding was that there was a progressive decline in norovirus outbreaks in Victoria in the period 2013 to 2015. Using the same sampling and testing protocols, 190 norovirus outbreaks were identified in 2013.[14] In the current study there were 165 norovirus outbreaks in 2014 and 122 in 2015. This decline was linked, to some degree, to changes in the prevalence of the predominant epidemic strain GII.Pe/GII.4_Sydney_2012. These findings indicate that this predominant GII.4 strain is losing its potency to infect, presumably as a result of increasing herd immunity,[28] and some speculation as to what may follow this strain is appropriate. Two potential candidates emerge, GII.P4_NewOrleans_2009 and GII.17.

The first potential candidate that may supplant the diminishing GII.Pe/GII.4_Sydney_2012 is a new strain of GII.P4_NewOrleans_2009 that appeared in 2015. Three of the new GII.P4_NewOrleans_2009 strains were identified by sequencing analysis as GII.4_Sydney_2012 in ORF 2. These observations indicate that a previously dominant ORF 1 form, GII.P4_NewOrleans_2009, had recombined with the currently dominant ORF 2 form GII.4_Sydney_2012 to produce a novel strain. At the end of 2015, GII.P4_NewOrleans_2009/GII.4_Sydney_2012 was still relatively rare.

Figure 2: Nucleotide sequence alignments of 1 specimen from each GI.3 outbreak where both ORF 1 (Figure 2A) and ORF 2 (Figure 2B) sequences were available for a given individual from a given outbreak

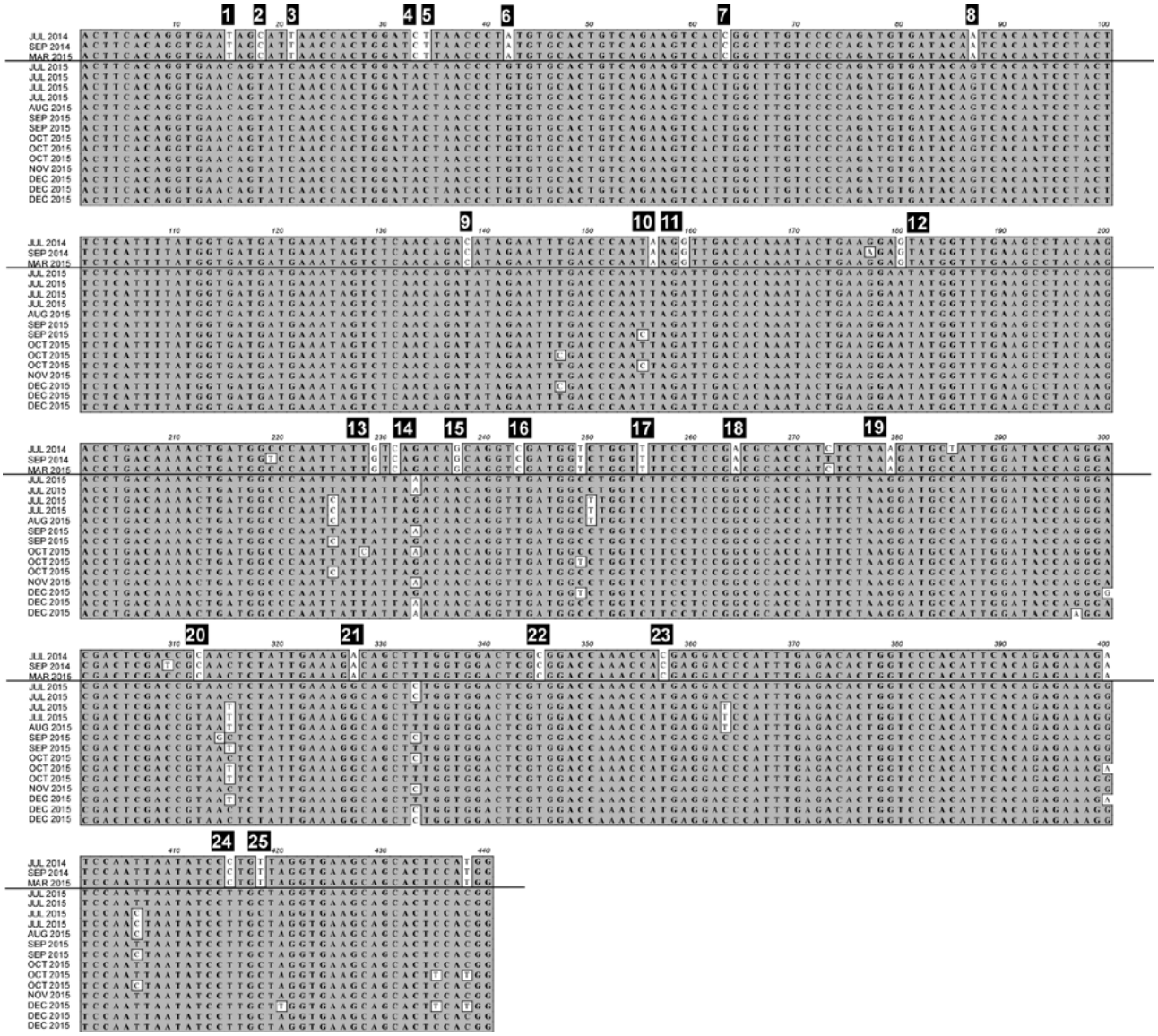


Figure 2B

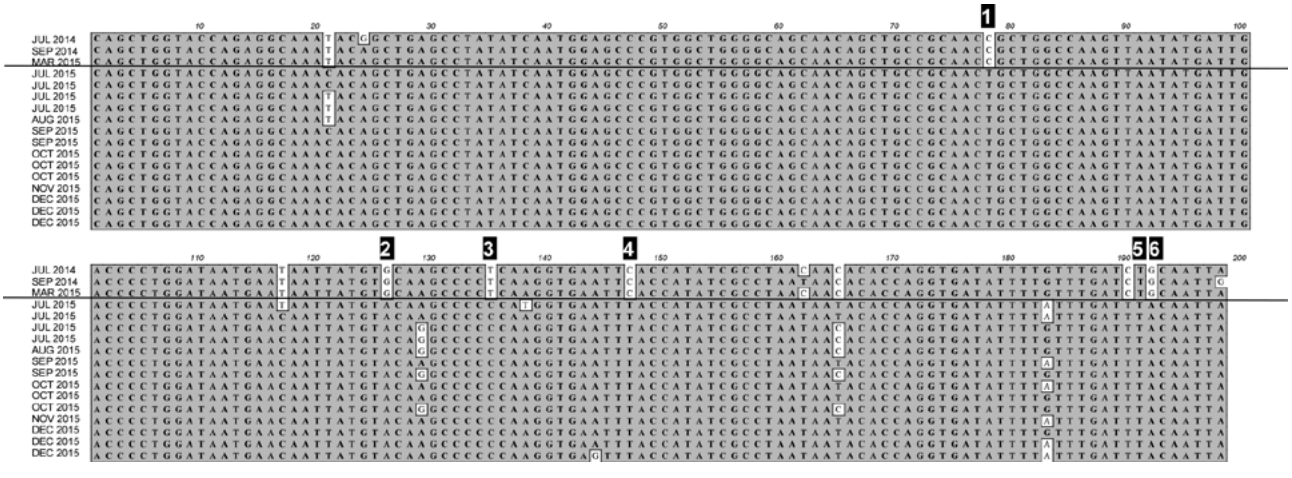


Table 4: Norovirus ORF 2 genotypes detected in healthcare and non-healthcare categories from 2014 and 2015

Norovirus ORF 2 genotypes	Norovirus positive outbreaks in healthcare settings				Norovirus positive outbreaks in non-healthcare settings			
	2014		2015		2014		2015	
	n	%	n	%	n	%	n	%
GI.2	3	2.2	0	0.0	4	12.9	0	0.0
GI.3	3	2.2	12	12.2	0	0.0	3	12.5
GI.3 and GII.3	0	0.0	0	0.0	1	3.2	0	0.0
GI.4	3	2.2	0	0.0	0	0.0	0	0.0
GI.5	1	0.7	0	0.0	0	0.0	0	0.0
GI.6	1	0.7	0	0.0	0	0.0	0	0.0
GI.9	3	2.2	0	0.0	0	0.0	0	0.0
GII.2	1	0.7	1	1.0	0	0.0	5	20.8
GII.3	3	2.2	5	5.1	4	12.9	4	16.7
GII.4	80	59.7	49	50.0	10	32.3	5	20.8
GII.6	9	6.7	5	5.1	2	6.5	2	8.3
GII.7	0	0.0	1	1.0	0	0.0	1	4.2
GII.8	1	0.7	0	0.0	0	0.0	0	0.0
GII.13	1	0.7	0	0.0	0	0.0	0	0.0
GII.17	2	1.5	3	3.1	0	0.0	1	4.2
No sequence available	23	17.2	22	22.4	10	32.3	3	12.5
Total	134	100.0	98	100.0	31	100.0	24	100.0

The second potential candidate that may supplant GII.Pe/GII.4_Sydney_2012, is GII.17 norovirus. Recent studies in China[10,11] and Japan[12] have identified a new GII.17 norovirus as an apparent new norovirus epidemic strain. Studies in the USA[27] and Taiwan[29] have confirmed the presence of GII.17 norovirus strains in these countries but reports from other areas are lacking. A comparison with published sequence data on GII.17 norovirus strains recently reported in the USA[27] and Japan[12] showed the Australian, USA and Japanese strains were similar. The current study indicates that GII.17 does not appear to be a major genotype in gastroenteritis outbreaks in Victoria and recent studies in New South Wales and Western Australia[15] indicate that GII.17 is not a major genotype in those states either.

Previous studies in Victoria (2002 to 2010)[22] have indicated that norovirus gastroenteritis outbreaks associated with GI norovirus are relatively rare compared with outbreaks associated with GII norovirus and the data for the current study confirms this finding. Nevertheless, a dramatic change in the diversity of GI noroviruses occurred in Victoria over the period 2013 to 2015. In 2013, ORF 2 sequence analysis identified 7 ORF 2 GI genotypes associated with gastroenteritis outbreaks in Victoria with GI.4 being the chief genotype detected.[14] In the current study, in 2014, 6 ORF 2 GI norovirus genotypes were detected, with GI.2 being the most common. In 2015, however, a dramatic change occurred with only 1 ORF 2 genotype detected, GI.3.

Genetic analysis of GI.3 noroviruses over 2014 to 2015 indicated there were substantial changes in the genome in the period corresponding to the transition between 2014 and 2015. Although the relative number of GI outbreaks, based on ORF 2 sequencing, was similar in 2014 (14.4%) and 2015 (15.5%), it remains to be seen whether the genetic changes that have occurred in GI norovirus in Victoria in 2015 will result in a greater incidence of GI norovirus in coming years.

A key area in the understanding of how norovirus circulates through the community involves an examination of the relationship between norovirus genotype and outbreak setting. Previous studies in this laboratory have established that such a relationship does exist [22,30,31] and the current study supports and extends these previous observations. In particular, it was shown that the ORF 2 genotypes GI.3, GII.6, and GII.4 were significantly more common in healthcare settings than in non-healthcare settings.

In summary, this study emphasises the complex way in which norovirus circulates throughout the community and the associated genetic changes

the virus undergoes as it does so. The ongoing monitoring of these variables may eventually lead to the development of a clear model of how human norovirus can continually re-invent itself.

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References

1. Green KY. *Caliciviridae*: the noroviruses. In: Knipe DM, Howley PM, eds. *Fields Virology*, 6th edn. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins, 2013. pp 582–608.
2. La Rosa G, Pourshaban M, Iaconelli M, Muscillo M. Detection of genogroup IV noroviruses in environmental and clinical samples and partial sequencing through rapid amplification of cDNA ends. *Arch Virol* 2008;153(11):2077–2083.
3. Aliabadi N, Lopman BA, Parashar UD, Hall AJ. Progress toward norovirus vaccines: considerations for further development and implementation in potential target populations. *Expert Rev Vaccines* 2015;14(9):1241–1253.
4. Lopman BA, Steele D, Kirkwood CD, Parashar UD. The vast and varied global burden of norovirus: prospects for prevention and control. *PLoS Med* 2016;13(4):e1001999.
5. Hall G, Kirk MD, Becker N, Gregory JE, Unicomb L, Millard G, et al. Estimating foodborne gastroenteritis, Australia. *Emerg Infect Dis* 2005;11(8):1257–1264.
6. Bernstein DI, Atmar RL, Lyon GM, Treanor JJ, Chen WH, Jiang X, et al. Norovirus vaccine against experimental human GII.4 virus illness: a challenge study in healthy adults. *J Infect Dis* 2015;211(6):870–878.
7. Kroneman A, Vega E, Vennema H, Vinje J, White PA, Hansman G, et al. Proposal for a unified norovirus nomenclature and genotyping. *Arch Virol* 2013;158(10):2059–2068.
8. Bull RA, Tanaka MM, White PA. Norovirus recombination. *J Gen Virol* 2007;88(12):3347–3359.
9. Bok K, Green KY. Norovirus gastroenteritis in immunocompromised patients. *N Engl J Med* 2012;367(22):2126–2132.
10. Fu J, Ai J, Jin M, Jiang C, Zhang J, Shi C, et al. Emergence of a new GII.17 norovirus variant in patients with acute gastroenteritis in Jiangsu, China, September 2014 to March 2015. *Euro Surveill* 2015;20(24):pii=21157.
11. Lu J, Sun L, Fang L, Yang F, Mo Y, Lao J, et al. Gastroenteritis outbreaks caused by norovirus GII.17, Guangdong Province, China, 2014–2015. *Emerg Infect Dis* 2015;21(7):1240–1242.
12. Matsushima Y, Ishikawa M, Shimizu T, Komane A, Kasuo S, Shinohara M, et al. Genetic analyses of GII.17 norovirus strains in diarrheal disease outbreaks from December 2014 to March 2015 in Japan reveal a novel polymerase sequence and amino acid substitutions in the capsid region. *Euro Surveill* 2015;20(26):pii=21173.
13. de Graaf M, van Beek J, Vennema H, Podkolzin AT, Hewitt J, Bucardo F, et al. Emergence of a novel GII.17 norovirus—End of the GII.4 era? *Euro Surveill* 2015;20(26):pii=21178.
14. Bruggink LD, Dunbar NL, Catton MG, Marshall JA. Norovirus genotype diversity associated with gastroenteritis outbreaks in Victoria in 2013. *Commun Dis Intell* 2015;39(1):E34–E41.
15. Lim KL, Hewitt J, Sitabkhan A, Eden J-S, Lun J, Levy A, et al. A multi-site study of norovirus molecular epidemiology in Australia and New Zealand, 2013–2014. *PLoS One* 2016;11(4):e0145254.
16. Witlox KJ, Karapanagiotidis T, Bruggink LD, Marshall JA. The effect of fecal turbidity on norovirus detection by reverse transcriptase polymerase chain reaction. *Diagn Microbiol Infect Dis* 2010;66(2):230–232.
17. Witlox KJ, Nguyen TN, Bruggink LD, Catton MG, Marshall JA. A comparative evaluation of the sensitivity of two automated and two manual nucleic acid extraction methods for the detection of norovirus by RT-PCR. *J Virol Methods* 2008,150(1–2):70–72.
18. Yuen LKW, Catton MG, Cox BJ, Wright PJ, Marshall JA. Heminested multiplex reverse transcription-PCR for detection and differentiation of Norwalk-like virus genogroups 1 and 2 in fecal samples. *J Clin Microbiol* 2001;39(7):2690–2694.
19. Bruggink LD, Witlox KJ, Sameer R, Catton MG, Marshall JA. Evaluation of the RIDA®QUICK immunochromatographic norovirus detection assay using specimens from Australian gastroenteritis incidents. *J Virol Methods* 2011;173(1):121–126.
20. McIver CJ, Bull RA, Tu ETV, Rawlinson WD, White PA. In: McIver CJ, ed. *A compendium of laboratory diagnostic methods for common and unusual enteric pathogens–an Australian perspective. Norovirus* and *Sapovirus*. Melbourne: The Australian Society for Microbiology Inc: 2005. pp 191–198.
21. Dunbar NL, Bruggink LD, Marshall JA. Evaluation of the RIDAGENE real-time assay for the detection of GI and GII norovirus. *Diagn Microbiol Infect Dis* 2014;79(3):317–321.
22. Bruggink LD, Oluwatoyin O, Sameer R, Witlox KJ, Marshall JA. Molecular and epidemiological features of gastroenteritis outbreaks involving genogroup I norovirus in Victoria, Australia, 2002–2010. *J Med Virol* 2012;84(9):1437–1448.
23. Bruggink LD, Dunbar NL, Marshall JA. Norovirus genotype diversity in community-based sporadic gastroenteritis incidents: a five year study. *J Med Virol* 2015;87(6):961–969.
24. Kroneman A, Vennema H, Deforche K, van der Avoort H, Penaranda S, Oberste MS, et al. An automated genotyping tool for enteroviruses and noroviruses. *J Clin Virol* 2011;51(2):121–125.
25. Remington RD, Schork MA. Statistics with applications to the biological and health sciences. Englewood Cliffs: Prentice-Hall Inc; 1970. pp 235–244.
26. Agresti A. Categorical data analysis. New York: John Wiley & Sons; 1990. pp 50–54.

27. Parra GI, Green KY. Genome of emerging norovirus GII.17, United States, 2014. *Emerg Infect Dis* 2015;21(8):1477–1479.

28. Lindesmith LC, Constantini V, Swanstrom J, Debbink K, Donaldson EF, Vinje J, et al. Emergence of a norovirus GII.4 strain correlates with changes in evolving blockade epitopes. *J Virol* 2013;87(5):2803–2813.

29. Lee C-C, Feng Y, Chen S-Y, Tsai C-N, Lai M-W, Chiu C-H. Emerging norovirus GII.17 in Taiwan. *Clin Infect Dis* 2015;61(11):1762–1764.

30. Bruggink L, Sameer R, Marshall J. Molecular and epidemiological characteristics of norovirus associated with community-based sporadic gastroenteritis incidents and norovirus outbreaks in Victoria, Australia, 2002–2007. *Intervirology* 2010;53(3):167–172.

31. Bruggink L, Marshall J. The relationship between health care and non-healthcare norovirus outbreak settings and norovirus genotype in Victoria, Australia, 2002–2005. *J Microbiol Immunol Infect* 2011;44(4):241–246.

Evaluation of bacille Calmette-Guérin immunisation programs in Australia

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Abstract

Background: bacille Calmette-Guérin (BCG) immunisation programs in Australia are funded and operated by the individual states and territories. In recent years BCG vaccine shortages have required use of unregistered products. We aimed to evaluate BCG immunisation programs in Australia, with particular reference to program implementation and national consistency.

Methods: Between September and November 2015, 12 key stakeholders, representing Australian states and territories, completed surveys. We analysed BCG vaccination coverage data from the Australian Childhood Immunisation Register (ACIR), and data on adverse events following immunisation (AEFI) with BCG vaccine from the Therapeutic Goods Administration's Adverse Drug Reactions System, for 2001 to 2014.

Results: Access to BCG vaccination varies between jurisdictions, with some states providing this only in major city locations. Analysis of ACIR data suggests significant differences in vaccine delivery between jurisdictions, but varying levels of under-reporting to the ACIR were also acknowledged. The rate of BCG AEFI appeared to increase between 2011 and 2014; however, these data need to be interpreted with caution due to small numbers, likely under-reporting of both numerator (AEFI) and denominator (vaccine doses administered), and the general increase in reporting of AEFI related to other vaccines in children over this period.

Conclusions: BCG immunisation programs aim to prevent severe forms of tuberculosis in young children who live in or travel to high burden settings. A range of factors, particularly inconsistent vaccine supply are leading to low, variable and inequitable vaccine delivery across Australian jurisdictions. Improved BCG vaccination uptake and AEFI data quality are required for accurate monitoring of program delivery and vaccine safety – this is particularly important given the current need to use unregistered vaccines. Improved and consistent access to BCG vaccine is suggested to optimise equity for at-risk children Australia-wide. *Commun Dis Intell* 2017;41(1):E33–E48.

Keywords: adverse reaction; bacille Calmette-Guérin; immunisation; tuberculosis

Introduction

Bacille Calmette-Guérin (BCG) vaccine has been in use since 1921. Vaccination of young children is considered an important strategy in almost all national tuberculosis (TB) programs, particularly in countries with a high burden of TB.[1] As of 2013, the incidence of TB in Australia was 5.5 per 100,000 population,[2] one of the lowest rates of TB in the world.[3] However, 28% of Australia's population are overseas-born and many are from TB endemic countries.[4] People born overseas make up the majority of TB cases in Australia, with an incidence of 18.4 per 100,000 in 2013. The incidence of TB among the Australian-born population has remained relatively stable in recent years, although with a marked disparity between Aboriginal and Torres Strait Islander (henceforth referred to as Indigenous) and non-Indigenous populations (incidence 4.6 and 0.8 per 100,000, respectively, in 2013).[2]

There is strong evidence that BCG vaccination in infancy provides a more than 70% protection against severe disseminated forms of TB, including miliary TB and TB meningitis.[1] The efficacy of BCG vaccine against pulmonary TB in adults is less consistent and has ranged from 0% to 80% in controlled trials.[5] Australian national guidelines on the use of BCG vaccine (*The Australian Immunisation Handbook* and the National Tuberculosis Advisory Committee (NTAC) guidelines, *The BCG vaccine: information and recommendations for use in Australia*)[6,7] recommend vaccination principally for young children who will be travelling to or living in regions with a high prevalence of TB for extended periods (preferably 2–3 months prior to departure), and Indigenous neonates in communities with a high incidence of TB (currently implemented in Queensland, the Northern Territory and northern South Australia only).[6,7]

Unlike other childhood vaccines, BCG vaccine coverage in Australia is not routinely reported. In addition, unlike most other vaccines recommended and funded for use in children in Australia, BCG is not delivered under the National Immunisation Program (NIP).[8] Rather BCG immunisation programs are funded and operated by individual states and territories. In recent years recurrent BCG vaccine shortages have required states and territories to prioritise and conserve stocks and/or

use unregistered products. There was a shortage of BCG vaccine for several months in 2012 following a recall of the only BCG vaccine registered in Australia (BCG Vaccine, Sanofi Pasteur).[9] A replacement unregistered vaccine (BCG Vaccine, Danish Strain 1331, Statens Serum Institute (SSI) in Denmark) was sourced under Section 19A(3) of the *Therapeutic Goods Act 1989*,[10] which allows for importation of unregistered products from specified countries with comparable regulatory standards, during shortages of registered products. The SSI product was supplied from September 2012 to the end of 2015, when it also became unavailable. All alternative products currently available can only be sourced and supplied via the Authorised Prescriber Scheme or Special Access Scheme of the *Therapeutic Goods Act*.[10,11] The difficulties in sourcing appropriate alternative products have been exacerbated in recent years by a global BCG vaccine shortage.[12] Of note, subsequent to our study, the complex issues and barriers to the use of potentially available unregistered vaccines led to most Australian jurisdictions suspending their BCG immunisation programs. As of August 2016 only the New South Wales and Victorian programs were active, using an unregistered Polish vaccine.

BCG vaccine causes adverse events in about 5% of the recipients. Common adverse events include infection site abscess in 2.5%, lymphadenitis in 1%, and up to 1% require medical attention.[13] Serious or long-term complications are rare.[13,14] The vaccines used in Australia in recent years are derived from different BCG strains, each of which may have a different reactogenicity profile.[15] Only passive surveillance of adverse events following immunisation (AEFI) with BCG vaccine occurs in Australia.[16]

No national level evaluation of BCG immunisation programs in Australia has previously been conducted. In the context of the issues outlined above, we aimed to evaluate BCG immunisation programs in Australia, with particular reference to program implementation and national consistency, and to promote discussion on improving program delivery (Box).

Methods

Data sources

The data sources for each specific evaluation topic are summarised in Table 1.

Document review

We conducted a detailed search of the Australian Government Department of Health and state and territory government health web sites to identify relevant documents on BCG immunisation policy and programs, including guidelines, fact sheets, media releases, provider and patient resources (e.g. brochures, posters) and reports. Health departments were also asked to provide any additional relevant documents.

Key stakeholder survey

Key stakeholder surveys were conducted between September and November 2015 to gain an in-depth understanding of program implementation as well as strengths and weaknesses/challenges (Appendix). Purposive sampling, using a sampling matrix (Table 2), was used to recruit a representative sample across key stakeholder groups and jurisdictions. Jurisdictional-level TB program managers and coordinators were approached directly while immunisation providers and local program coordinators were referred by other participants (respondent-driven and snowballing sampling).

Box: Evaluation objectives

The specific objectives of the evaluation were:

1. to review policy and practice regarding the use of bacille Calmette-Guérin vaccine at a national and jurisdictional level in Australia; and
2. to describe and assess how bacille Calmette-Guérin vaccine programs are implemented across different states and territories in Australia, with specific regard to:
   a. availability, accessibility and awareness;
   b. vaccine uptake/coverage;
   c. reporting and follow-up of adverse events following immunisation related to bacille Calmette-Guérin vaccine;
   d. consistency between jurisdictional programs and with national guidelines; and
   e. strengths, challenges and recommendations.

Table 1: Summary of data sources used for each specific objective

Objective	Evaluation topics	Data sources
To review policy and practice regarding the use of bacille Calmette-Guérin vaccine at a national and jurisdictional level in Australia		Document review
To describe and assess how bacille Calmette-Guérin vaccine programs are implemented across different states and territories in Australia	Availability, accessibility and awareness	Key stakeholder survey
	Vaccine uptake/coverage	Australian Childhood Immunisation Register
	Reporting and follow-up of adverse events following immunisation related to bacille Calmette-Guérin vaccine	Key stakeholder survey Australian Adverse Drug Reactions System database
	Consistency between jurisdictional programs and with national guidelines	Document review
	Strengths, challenges and recommendations	Key stakeholder survey

Table 2: Matrix of interview participants

Participants	NSW	NT	Qld	Tas.	SA	Vic.	WA
Tuberculosis (TB) medical advisor			X	X	X	X	
Jurisdictional TB program manager/coordinator	X	X	X			X	X
Epidemiologist			X				
TB/chest clinic clinical nurse consultants			X				
Remote area immunisation provider			X				

X = One key informant.

No response from the Australian Capital Territory.

A semi-structured questionnaire was developed by staff from the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases, based on previous national immunisation program evaluations. The questionnaire contained both open and closed questions and sought information on:

a. program implementation including availability and access to BCG vaccine for providers and consumers;

b. communication strategies and resources to engage providers and consumers';

c. reporting of BCG vaccination and AEFI; and

d. strengths and challenges of the program and recommendations for improvements.

The questionnaire was piloted and modified to incorporate feedback. All key stakeholders were sent the questionnaire by email and the completed questionnaires were returned within 2 to 4 weeks.

Australian Childhood Immunisation Register

Vaccination coverage estimates and the number of BCG doses administered were obtained from the Australian Childhood Immunisation Register (ACIR). The ACIR was established in 1996 and is the primary source of vaccination coverage data in Australia. Detailed description of the operation of ACIR has been published previously.[17] At the time of this evaluation the ACIR recorded details of vaccinations given to children aged less than seven years, irrespective of whether NIP funded or not. Analysis of ACIR data was undertaken for vaccinations notified between 2001 and 2014, for data released to NCIRS in April 2015.

Australian Adverse Drug Reactions System database

De-identified data on AEFI with BCG vaccine between 2001 and 2014 reported to the Therapeutic Goods Administration (TGA) and stored in the Australian Adverse Drug Reactions System (ADRS) database were extracted from a dataset released to NCIRS in March 2015. ADRS

is a national passive surveillance system for AEFI data reported to the TGA by state and territory health departments, health professionals, vaccine manufacturers and members of the public. All AEFI reports are assessed using internationally consistent criteria[18] before being entered into the ADRS database.

Data analysis

Both qualitative and quantitative data were analysed. Content analysis was conducted on interview transcripts to identify prominent themes. BCG vaccination data were extracted from the NCIRS ACIR dataset and analysed by year of administration of vaccine and age group. BCG vaccination coverage data were analysed by jurisdiction and Indigenous status using 12-month wide birth cohorts for children born in 2012, 2013 and 2014. The percentage vaccinated for each cohort was calculated using ACIR data as at 30 September 2015. BCG-related AEFI data extracted from the NCIRS ADRS dataset were analysed by year and whether classified as serious, with rates per 100,000 doses calculated. Quantitative data analysis was performed using SAS software version 9.4 (SAS Institute Inc. Cary, NC, USA) and Excel 2010 (Microsoft, Redmond, PA, USA).

Stakeholder engagement/sharing of lessons learnt

The National Tuberculosis Advisory Committee (NTAC) was consulted on the design of the evaluation and provided comment on a draft report. The findings were also shared with the National Immunisation Committee, Communicable Diseases Network Australia, and the Australian Technical Advisory Group for Immunisation.

The study was conducted by NCIRS as part of its national immunisation program evaluation role and using de-identified aggregated data; ethics committee approval was not required.

Results

Document review

Guidance on the use of BCG vaccine in Australia is provided in both *The Australian Immunisation Handbook*[7] and the NTAC guidelines *The BCG vaccine: information and recommendations for use in Australia*.[9] We also identified jurisdiction-specific BCG immunisation policies or guidelines from all states and territories of Australia except for the Australian Capital Territory and Tasmania. Recommendations in current national and jurisdictional guidelines are compared in Table 3. There are minor differences between the 2 national guidelines, and more substantial differences between some of the jurisdictional guidelines.[6,7,19–25]

Key stakeholder interviews and associated data analysis

Twelve key stakeholders from across 5 stakeholder groups (Appendix), including representatives from 7 jurisdictions (New South Wales, the Northern Territory, Queensland, Tasmania, South Australia, Victoria and Western Australia, completed a semi-structured questionnaire covering program implementation issues across the following areas.

Vaccine administration

Availability of bacille Calmette-Guérin vaccine for providers

Participants stated that BCG vaccine is purchased through different routes, either via: their jurisdictional Department of Health Immunisation Branch (South Australia, Victoria and Western Australia); hospitals (New South Wales, Northern Territory); or central pharmacy (Queensland).

Demand for BCG vaccine at the state or territory level was reported to be measured variably. It is estimated from the previous year's usage in New South Wales, the Northern Territory and Queensland. In Western Australia, usage is monitored regularly by the central clinic. In Victoria, measurement was reported to have recently changed from estimation using the previous year's usage to a more detailed picture of actual and projected demand including: the number of children on the waiting list by month; mean or median waiting time from referral to administration of BCG; and the number of children vaccinated by month, age and gender and country of origin of parents.

All stakeholders, except one, stated that the recurrent shortages of BCG vaccine and uncertainty of supply had significantly impacted on BCG vaccine availability in their jurisdiction in the past 3 years.

Consumer access to bacille Calmette-Guérin vaccination

Consumer access to BCG vaccination varied between jurisdictions. New South Wales, Queensland, Western Australia and the Northern Territory provide access at regional and remote locations while South Australia and Victoria provide access only in central major city locations. Moreover, vaccine is not provided by general practitioners and only provided by travel medicine clinics (n=4) in one jurisdiction (Table 4).

Table 3: Recommendations in national and jurisdictional guidance documents on bacille Calmette-Guérin immunisation, with comparison to National Tuberculosis Advisory Committee guidelines*

NTAC guidelines[7]	Immunisation handbook[6]	NSW[19,20]	NT[21]	Qld[22]	SA[23]	Vic.[24]	WA[25]
Bacille Calmette-Guérin vaccination recommended for							
1. Indigenous neonates in communities with a high incidence of TB.	✓	–	✓	✓	✓	✓†	–
2. Neonates and children aged ≤5 years who will be travelling or living in countries or areas with a high prevalence of TB for extended periods	✓ (specifies 'young children, particularly those aged <5 years', country with incidence of >40 per 100,000)	✓ (specifies children aged <5 years and ≥3 months in country with incidence of ≥40 per 100,000)	✓ (specifies aged <5 years who have high probability of travelling to countries of high TB incidence for extended period)	✓ (specifies ≥3 months of age in country with incidence of ≥40 per 100,000)	✓ (specifies ≥3 months of age)	✓ (Depends on age and duration e.g. all children aged <5 years going to country with incidence of > 100 per 100,000 for children aged ≥4 weeks (<4 weeks for children aged <1 year); all aged <1 years going to country with incidence of >40 per 100,000 for ≥4 weeks)	✓ (specifies 'could be vaccinated' if aged >6 months in area with incidence of >50 per 100,000)
3. Neonates born to parents with leprosy or a family history of leprosy	✓	✓	✓	✓ (specifies parent with leprosy only)	✓	✓	✓ (specifies 'could' be vaccinated')
						Children aged <5 years who live in household that includes immigrants or unscreened visitors, recently arrived from countries of high TB incidence (>40 per 100,000)	

Table 3 *continued*: Recommendations in national and jurisdictional guidance documents on bacille Calmette-Guérin immunisation, with comparison to National Tuberculosis Advisory Committee guidelines*

NTAC guidelines[7]	Immunisation handbook[6]	NSW[19,20]	NT[21]	Qld[22]	SA[23]	Vic.[24]	WA[25]
Bacille Calmette-Guérin vaccination should be considered for							
1. Children aged >5 years who will be travelling or living in countries or areas with a high prevalence of TB for extended periods	✓	–	✓	–	–	✓ (specifies >2–3 months, also young adults)	–
2. Health care workers who may be at high risk of exposure to drug resistant TB	✓	–	✓	–	✓	✓	✓
			Children aged <5 years who will be living in Northern Territory Indigenous communities			Children, adolescents and young adults exposed to index active MDR TB case Persons over the age of 5 years through to young adulthood living or travelling for extended periods in countries of high TB prevalence	New-born children of migrants who have arrived from countries with high incidence of TB in last 5 years, or who have household contact with people who have arrived from a high incidence country in last 5 years. Children <6 years who are household contacts of newly diagnosed leprosy case.

* No guidance documents identified for Tasmania or the Australian Capital Territory; stakeholders from those jurisdictions advised that they follow national guidelines.

† No communities meet this criterion (personal communication)

Table 4: Bacille Calmette-Guérin vaccine provision, Australia, by type of provider, state or territory and sub-region location

	Major cities	Regional	Remote
NSW	TB/chest clinic (onsite)	TB/chest clinic (onsite)	TB/chest clinic (onsite)
NT	TB/chest clinic (onsite) Maternity ward	TB/chest clinic (onsite) Maternity ward	TB/chest clinic (onsite) TB/chest clinic (outreach)
Qld	TB/chest clinic (onsite)	TB/chest clinic (onsite) TB/chest clinic (outreach) Maternity ward	TB/chest clinic (onsite) TB/chest clinic (outreach) Maternity ward
SA	TB/chest clinic (onsite)	Not available	Not available
Tas.	TB/chest clinic (onsite)	TB/chest clinic (onsite)	Not available
Vic.	Royal Children's Hospital and Monash Medical Centre. Four private travel health clinics also give bacille Calmette-Guérin vaccine.	Service provided through St John of God Hospital in Geelong	Not available
WA	TB/chest clinic (onsite)	Regional public health units – very occasional usage	Remote public health units – very occasional usage

One stakeholder suggested that BCG vaccination services could be improved if the vaccine could be provided in a single dose vial.

"Provide vaccine in a single dose cost-effective vial so that BCG could be given at any clinic. Currently clients need to fit in with the allocated days for BCG clinics, and then book into the appointment system e.g. one BCG clinic per month with 30 max neonates to be booked."

Access to BCG vaccine in rural and remote areas was a common concern identified in the key stakeholder surveys, and several stakeholders suggested increasing the number of vaccination sites regionally and/or in additional locations in major cities. Moreover, maintaining an adequate number of appropriately trained and accredited staff, particularly in areas with low demand and/or high staff turnover, was identified as a common challenge.

All participating key stakeholders stated that in their state or territory eligible individuals are referred to BCG immunisation providers through both general practitioner (GP) referral and patient self-referral. In Queensland and Western Australia, eligible individuals are also referred by travel medicine clinics, child health clinics and community health nurses. The average waiting time for consumers to access BCG vaccine, as reported by stakeholders, varied by jurisdiction and remoteness of location (Table 5).

Communication strategies and resources to promote awareness

Communication strategies varied between jurisdictions. Most commonly BCG immunisation guidelines and consumer information were disseminated through health department web sites, online learning packages, media releases, brochures and face-to-face education for providers.

Most of the key stakeholders identified GPs, antenatal clinics and child health clinics as potential groups among which greater promotion of BCG vaccination for eligible individuals could occur, with particular focus suggested in areas where parents who are likely to be taking young children to live overseas.

The recurrent shortages of BCG vaccine were reported to have been a considerable barrier in promoting the vaccine and public awareness.

"We have been told that there may not be supplies of BCG after December 2015 so are not promoting the vaccine at present."

Reporting of vaccination coverage and adverse events

When asked how likely it is that BCG vaccination information will be entered into the ACIR, key stakeholder responses varied widely by jurisdiction and in the case of Queensland, where stakeholders from different settings were interviewed, within the jurisdiction. Stakeholders from the Northern Territory and Queensland reported that BCG vaccination information is 'always' entered into the ACIR and one stakeholder from

Table 5: Reported waiting times and/or frequency of bacille Calmette-Guérin clinic services for access to bacille Calmette-Guérin vaccination

Waiting time for BCG vaccination	Major cities	Regional	Remote
NSW	≥2 weeks	Data not provided	Data not provided
NT	<1 week	Regional and remote area are dependent on scheduled visits by accredited vaccine providers	Regional and remote area are dependent on scheduled visits by accredited vaccine providers
Qld	Data not provided	Data not provided Regional clinics are organised every 3–4 weeks.	Remote outreach clinics are organised usually every 6 months.
SA	1–<2 weeks	Need to come to Adelaide	Need to come to Adelaide
Tas.	≥2 weeks	Data not provided	Data not provided
Vic.	≥2 weeks	≥2 weeks	≥2 weeks
WA	1–<2 weeks	Data not provided	Data not provided

Victoria stated that it is done 'most of the times'. For the rest of the stakeholders (Queensland, Victoria, New South Wales, Western Australia, South Australia and Tasmania) responses ranged from 'sometimes' to 'never'.

One key stakeholder identified IT system barriers to the transfer of data from the jurisdictional immunisation register to the ACIR. Another respondent highlighted the need for a *"great deal of encouragement to chest clinic staff to use ACIR"*.

BCG vaccine-related AEFI are reported to the TGA, as with other vaccines. Stakeholders did not spontaneously report any particular issues with these arrangements.

Strengths and challenges

Key stakeholders' opinions about the strengths and challenges of their BCG immunisation programs are summarised in Table 6.

Analysis of Australian Childhood Immunisation Register and adverse events following immunisation data

Australian Childhood Immunisation Register data

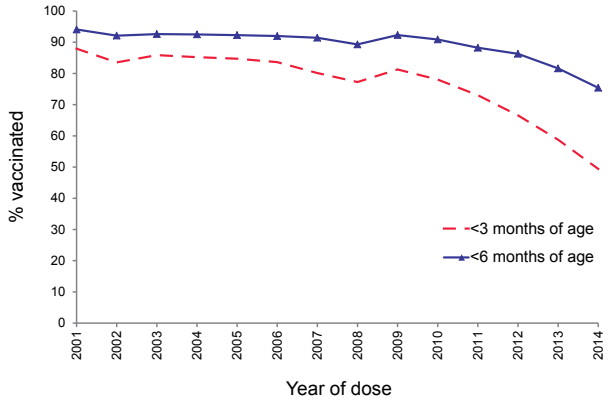
Table 7 shows the number of BCG vaccine doses recorded in the ACIR as administered between 2001 and 2014, by age group. The total number of doses recorded as administered varied by year with the highest number in 2010 and the lowest in 2014. The age distribution of recorded doses changed over time. In 2001, 94.1% of BCG vaccine doses were recorded as administered to infants aged less than 6 months, compared with 75.4% in 2014 (Figure).

Table 8 shows the number of BCG doses recorded on the ACIR and the percentage vaccinated by state or territory and Indigenous status for 12-month wide birth cohorts between 2012 and 2014. Substantial differences in the vaccination rate between jurisdictions and between Indigenous and non-Indigenous children are observed. The

Table 6: Summary of bacille Calmette-Guérin immunisation program strengths and challenges identified by key stakeholders

Strengths	Challenges
TB clinics provide good advisory service to travel vaccine services, GPs and patients	Availability of vaccine
Remote and outreach clinics to Indigenous communities in some jurisdictions	High demand for BCG vaccine in some metropolitan areas
Incorporated within routine childhood immunisation program (NT only)	Increasing access in rural and regional areas
Routine administration of bacille Calmette-Guérin (BCG) vaccine to Aboriginal neonates (NT only)	Maintaining adequate number of appropriately trained and accredited staff, particularly in areas with low demand and/or high staff turnover
Dual vaccination strategy by TB Control Units and maternity units to capture infants at birth in high risk areas (NT only)	Wastage, especially in regional area clinics due to multi-dose vials and product life following reconstitution
	Informing at-risk groups of the availability of the vaccine
	Providing routine clinic times for vaccine administration
	Ability to catch up following periods of shortage and rationing, due to the need for Mantoux tuberculin skin test in children >6 months

Figure: Percentage of bacille Calmette-Guérin vaccinated children vaccinated at less than 3 months of age and less than 6 months of age, Australia, 2001 to 2014



Source: Australian Childhood Immunisation Register

Northern Territory and Queensland had the highest proportion of both Indigenous and non-Indigenous children vaccinated with BCG. However, in 2014 there was a substantial reduction in the proportion of the birth cohort recorded as having BCG vaccine in all states and territories.

Adverse events following immunisation data

According to ADRS data, the overall rate of reported BCG vaccine-related AEFI among children aged less than 7 years was 89.8 per 100,000 doses administered, and the rate of serious AEFI was 11.9 per 100,000 doses (Table 9). Reporting rates appear to be increasing from 2011 onwards. The number and rate of reported serious AEFI did not show any pattern over time.

Discussion

This is the first national level evaluation of BCG immunisation programs in Australia. We identified only minor differences between the two national guidelines (the NTAC guidelines and *The Australian Immunisation Handbook*), but more substantial differences in some of the jurisdictional-specific guidelines. For example, the Victorian guidelines recommend BCG vaccination for children aged less than 5 years living in a household that includes immigrants or visitors recently arrived from countries of high TB incidence, and the Western Australia guidelines state vaccination should be considered for neonates in such households. Further discussion in national forums such as NTAC may be useful to explore the reasons for such discrepancies, and to determine whether greater national consistency can be achieved.

Table 7: Bacille Calmette-Guérin vaccine doses administered to children aged less than 7 years, Australia, 2001 to 2014, by age group

	Bacille Calmette-Guérin vaccine doses administered													
Age (months)	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014
<3	7,687	7589	8,634	9,575	10,054	10,430	9,976	8,902	11,168	11,114	9,652	7418	6,502	4,189
3–5	537	779	679	819	896	1,042	1,407	1,390	1,517	1,828	2,011	2,200	2,526	2,203
6–8	100	191	187	211	222	276	254	302	278	379	463	546	735	652
9–11	69	90	103	118	125	124	160	173	90	196	241	269	356	388
≥ 12	347	435	452	513	569	598	650	758	688	723	849	712	944	1,044
Total	8,740	9,084	10,055	11,236	11,866	12,470	12,447	11,525	13,741	14,240	13,216	11,145	11,063	8,476

Source: Australian Childhood Immunisation Register

Table 8: Number of bacille Calmette-Guérin doses administered and proportion of birth cohort vaccinated, Australia, 2012 to 2014, by state or territory and Indigenous status

Year	Indigenous status	ACT	NSW	NT*	Qld*	SA*	Tas.	Vic.	WA	Aust.
2012	Indigenous	2	8	767	1,548	17	0	12	29	2,383
	birth cohort	129	4,895	1,336	5,130	755	454	1,063	2,036	15,798
	% vaccinated	1.6	0.2	57.4	30.2	2.3	0.0	1.1	1.4	15.1
	Non-Indigenous	126	1,589	216	3,951	245	38	2,496	541	9,202
	birth cohort	5,473	97,920	2,294	59,255	19,663	5,475	77,089	32,678	299,847
	% vaccinated	2.3	1.6	9.4	6.7	1.3	0.7	3.2	1.7	3.1
	Total	128	1,597	983	5,499	262	38	2,508	570	11,585
	birth cohort	5,602	102,815	3,630	64,385	20,418	5,929	78,152	34,714	315,645
	% vaccinated	2.3	1.6	27.1	8.5	1.3	0.6	3.2	1.6	3.7
2013	Indigenous	0	12	530	1,667	8	1	13	21	2,252
	birth cohort	134	4,884	1,234	4,905	804	409	1,159	1,991	15,520
	% vaccinated	0.0	0.3	43.0	34.0	1.0	0.2	1.1	1.1	14.5
	Non-Indigenous	77	1,092	215	3,579	162	24	2,381	394	7,924
	birth cohort	5,428	93,934	2,312	56,967	18,881	5,532	75,132	31,835	290,021
	% vaccinated	1.4	1.2	9.3	6.3	0.9	0.4	3.2	1.2	2.7
	Total	77	1,104	745	5,246	170	25	2,394	415	10,176
	birth cohort	5,562	98,818	3,546	61,872	19,685	5,941	76,291	33,826	305,541
	% vaccinated	1.4	1.1	21.0	8.5	0.9	0.4	3.1	1.2	3.3
2014	Indigenous	0	5	453	1,424	5	0	8	17	1,912
	birth cohort	133	5,032	1,338	5,252	826	432	1,266	2,066	16,345
	% vaccinated	0.0	0.1	33.9	27.1	0.6	0.0	0.6	0.8	11.7
	Non-Indigenous	51	664	127	1,992	75	8	2,068	180	5,165
	birth cohort	5,505	93,649	2,339	57,334	19,360	5,401	75,474	32,316	291,378
	% vaccinated	0.9	0.7	5.4	3.5	0.4	0.2	2.7	0.6	1.8
	Total	51	669	580	3,416	80	8	2,076	197	7,077
	birth cohort	5,638	98,681	3,677	62,586	20,186	5,833	76,740	34,382	307,723
	% vaccinated	0.9	0.7	15.8	5.5	0.4	0.1	2.7	0.6	2.3

* Bacille Calmette-Guérin vaccination is recommended in Indigenous neonates in communities with a high incidence of tuberculosis

Source: Australian Childhood Immunisation Register

Reliable supply of BCG is a major challenge, along with access issues related to the availability of vaccine in multi-dose formulations only, and the additional training required in BCG vaccination and pre-vaccination screening. If cost-effective single dose vials could be sourced (currently not available from any manufacturer globally to our knowledge), this could facilitate wider provision of BCG vaccination, but would also require an increased pool of appropriately trained service providers. BCG is currently provided in general practices and maternity wards in the Northern Territory and Queensland in areas with a high proportion of Indigenous population. This practice may be an appropriate option for consideration in high use areas within other states and territories.

The total number of BCG vaccine doses recorded as administered in the ACIR varied by year. Despite the increase in population and no significant changes in national guideline recommendations over the study period, the number of doses recorded as administered in 2014 was less than that in 2001. The age distribution also changed over time, with 94% of BCG vaccine doses recorded as administered to infants aged less than 6 months in 2001, compared with 75% in 2014. However, it is unclear to what extent the ACIR data on the number of doses and age distribution reflect real (e.g. supply-related) issues or data quality issues. There was considerable variation between jurisdictions in the reported likelihood of entry of BCG vaccination data into the ACIR. Analysis of ACIR data also showed a wide variation between

Table 9: Adverse events following bacille Calmette-Guérin immunisation among children aged less than 7 years, Australia, 2001 to 2014

Year	Number of adverse events		Vaccine doses*	Rate per 100,000 doses			
				All		Serious	
	All	Serious		n	95% CI†	n	95% CI†
2001	2	0	8,740	22.9	0–54.6	0.0	
2002	1	0	9,084	11.0	0–32.6	0.0	
2003	0	0	10,055	0.0		0.0	
2004	2	0	11,236	17.8	0–42.5	0.0	
2005	5	4	11,866	42.1	0–79.1	33.7	0–66.7
2006	12	4	12,470	96.2	41.8–150.7	32.1	0–63.5
2007	5	2	12,447	40.2	0–75.4	16.1	0–38.3
2008	6	0	11,525	52.1	0–93.7	0.0	
2009	12	2	13,741	87.3	37.9–136.7	14.6	0–34.7
2010	17	2	14,240	119.4	62.7–176.1	14.0	0–33.5
2011	21	2	13,216	158.9	91.1–226.8	15.1	0–36.1
2012	20	0	11,145	179.5	100.9–258.0	0.0	
2013	23	2	11,063	207.9	123.0–292.8	18.1	0–43.1
2014	17	1	8,476	200.6	105.3–295.8	11.8	0–34.9
Total	143	19	159,304	89.8	75.1–104.5	11.9	0–17.3

* Bacille Calmette-Guérin vaccine doses recorded in Australian Childhood Immunisation Register.

† Rate of adverse events reported in Australian Adverse Drug Reactions System, per 100,000 administered doses.

jurisdictions in the proportion of non-Indigenous infants reported as receiving BCG, suggesting significant differences in vaccine delivery between states and territories and/or significant under-reporting to the ACIR. The main two jurisdictions with Indigenous programs (Northern Territory and Queensland) have a substantially higher proportion of non-Indigenous children reported as vaccinated compared with other states, suggesting differing implementation of the non-Indigenous program and/or better reporting, in the Northern Territory and Queensland.

The absence of any state or territory or nationally based requirements for transparent reporting on BCG vaccine coverage, as well as any provider and parental incentives to record data, may contribute to under-reporting to the ACIR. In the case of NIP vaccines, for which such requirements and incentives exist, under-reporting has relatively little impact with coverage rates routinely over 90%.[17] BCG vaccine is also not on the NIP and is often administered by specialist providers in chest clinics, rather than GPs who routinely utilise ACIR for NIP vaccines. Data on the number of BCG vaccine doses administered to older children and adults is not readily available, although this may change with the extension of the ACIR to a whole of life register.

Analysis of the TGA ADRS database showed an apparent increase in the rate of BCG vaccine related adverse events notification since 2011. However, BCG vaccine related AEFI data need to be interpreted with caution due to the small numbers involved, likely under-reporting of both numerator (AEFI) and denominator (vaccine doses administered) data, delayed reporting of some AEFI and the general increase in reporting of AEFI related to other vaccines in children over this period.[26] More detailed analysis of BCG related AEFI data reported between 2009 and 2014 has been reported in a separate publication.[27]

In addition to limitations related to data quality issues, this evaluation was of limited scope and did not assess a range of relevant issues including resourcing and cost of jurisdictional programs, whether any costs are charged to consumers, and whether jurisdictions conduct any formal follow-up of BCG-vaccinated individuals in relation to AEFI. The sample size of key stakeholders interviewed for the evaluation was also relatively small, particularly in relation to the number of BCG immunisation providers interviewed. Therefore, the views provided by participants may not necessarily represent those of stakeholders more broadly. No consumers were interviewed. Further study could be undertaken to build on existing evidence regarding poor awareness of BCG vaccine and

programs in parents of children in the target population [28] and to explore some of the information and issues reported by stakeholders in this evaluation, such as waiting times for BCG vaccination.

Concerns have been raised regarding low awareness of current BCG immunisation guidelines among both parents and providers.[28,29] Communication strategies and resources could be developed to target GPs, antenatal clinics and child health clinics, particularly in areas where parents are likely to be taking young children overseas to TB endemic countries. However, the potential effectiveness of such measures is difficult to quantify, as the size of the eligible target population is not known. Available sources suggest the target population is vastly greater than that being currently provided BCG. Further work should be undertaken to define the eligible target population size and distribution, for example using data on departures from Australia for overseas travel to TB endemic countries for young children.[28,29]

It is also difficult to quantify the number of cases potentially preventable by promoting greater awareness of and uptake of BCG vaccination in accordance with current guidelines. The TB notification rates in children are much higher among overseas-born compared with Australia-born children (9.6 (n = 294) vs. 0.6 (n = 230) cases per 100,000 children aged less than 15 years between 2003 and 2012).[30] Limited data are available on the mode of disease acquisition in Australian-born children (e.g. whether via travel to a TB endemic country or via family member contact in Australia), eligibility for vaccination according to current national guidelines, and vaccination status. A review of 2003 to 2012 TB notification data found 42% (226/538) had a history of travel to or through, or residence in, a high-risk country, but did not present any further breakdown of these figures.[30] A hospital audit of all children (< 18 years of age) treated for TB at the Children's Hospital at Westmead between January 2008 and December 2011 found that among 22 TB cases, 21 had a history of immigration or travel to a TB endemic country and 4 of known TB contact within Australia.[31] More comprehensive information on mode of disease acquisition would help inform steps to secure supply of BCG vaccine and/or implement TB screening programs and pre-emptive latent tuberculosis infection treatment in returned travellers.

Conclusions

BCG immunisation programs in Australia are considered important for preventing severe forms of TB in infants and young children who live in or travel to high burden settings. The increasing rate of drug resistant TB globally generates additional importance in terms of the need to provide individual protection. The NTAC plays a key coordinating role in promoting consistency of program delivery, as do recommendations in *The Australian Immunisation Handbook*. However, inconsistent vaccine supply and different state-based procurement processes are major current challenges that are contributing to low, variable and inequitable vaccine delivery. It is important that BCG vaccine-related AEFI data are monitored closely given the adverse event profile of this live attenuated vaccine and particularly in light of the continuing need to use unregistered BCG vaccines. Improved data quality in relation to reporting of BCG vaccination uptake and AEFI is required for more accurate monitoring of both program delivery and vaccine safety. Improvements in access to BCG vaccine and communication strategies are suggested to optimise equity for at-risk children Australia-wide. There could be potential for greater centralisation of some aspects of vaccine procurement and program delivery, for example through inclusion of BCG vaccine on the NIP, to help facilitate such improvements. We hope that publication of this evaluation report promotes further discussion on improving BCG immunisation program delivery across Australia.

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Appendix: Key stakeholder survey questions

1.0 Your role in the BCG vaccination program			
1.1	Job title		
1.2	Department/Section		
1.3	Professional background		
1.4	What is your role in the BCG vaccination program?		
2.0 Program Implementation: Availability (for providers) of BCG vaccine			
2.1	Which area/s of your state/territory government is/are responsible for purchase of BCG vaccine?		
2.2	How is the demand for BCG vaccine in your state/ territory estimated?		
2.3	Please describe any issues which impacted BCG vaccine availability in your state/ territory in the last 3 years (i.e. since the National Tuberculosis Advisory Committee Update in October 2012)		
2.4	Based on your experiences, do you have any recommendations to improve BCG vaccine availability?		
3.0 Program Implementation: Access (for consumers) to BCG vaccine			
3.1	What are the current modes of service for providing BCG vaccine in your state/ territory/area?	Location	Type of Services (please tick ✓)
		Major Cities	☐ TB/Chest Clinic (onsite) ☐ TB/Chest Clinic (outreach) ☐ Other (specify) ……
		Regional	☐ TB/Chest Clinic (onsite) ☐ TB/Chest Clinic (outreach) ☐ Other (specify) ……
		Remote	☐ TB/Chest Clinic (onsite) ☐ TB/Chest Clinic (outreach) ☐ Other (specify) …….
3.2	How eligible individuals are referred to BCG immunisation providers in your state/territory/area?	(✓ tick all that apply) ☐ GP referral ☐ Patient self-referral ☐ Other (Specify) …….	
3.3	How long is the average waiting time for patients to access BCG vaccine?	Location	Average waiting time (please tick ✓)
		Major Cities	☐ <1 week ☐ 1–<2 weeks ☐ >=2week ☐ Other (specify) ……
		Regional	☐ <1 week ☐ 1–<2 weeks ☐ >=2week ☐ Other (specify) ……
		Remote	☐ <1 week ☐ 1–<2 weeks ☐ >=2week ☐ Other (specify) ……
3.4	Does waiting time vary by location?	☐ Yes If Yes, specify ……. ☐ No	
3.5	Please describe any issues which impacted consumer's access to BCG vaccine in your state/ territory/area in the last 3 years?		
3.6	Based on your experiences, do you have any recommendations to improve consumer's access to BCG vaccine?		

4.0 Communication strategies & resources: Awareness			
4.1	Does your jurisdiction have specific state/territory policies/guidelines for BCG vaccination?	☐ Yes If Yes, specify …… ☐ No	
4.2	How are these guidelines promoted? (if answered Yes to 4.1)		
4.3	Since 2012, what provider/community groups/ organisations have been targeted to inform about the BCG vaccination program?		
4.4	Since 2012, were any state/territory/jurisdictional resources (in addition to guidelines) developed for the program?	☐ Yes If Yes, specify …… ☐ No	
4.5	What methods have been used to advise relevant target groups about the BCG vaccination program?	Target group	Method (✓ tick all that apply)
		Providers (e.g. GP, travel medicine clinic)	☐ Media ☐ Brochures ☐ Webpage/online ☐ Letters ☐ Other (specify) …… ☐ None
		Migrants	☐ Media (mainstream) ☐ Media (ethnic) ☐ Brochures ☐ Webpage/online ☐ Other (specify) …… ☐ None
		Indigenous communities	☐ Media (mainstream) ☐ Media (ethnic) ☐ Brochures ☐ Webpage/online ☐ Other (specify) …… ☐ None
		Travellers	☐ Media (mainstream) ☐ Media (ethnic) ☐ Brochures ☐ Webpage/online ☐ Other (specify) …… ☐ None
		Other	☐ Media (mainstream) ☐ Media (ethnic) ☐ Brochures ☐ Webpage/online ☐ Other (specify) …… ☐ None
4.6	Please describe any issues which impacted public/provider awareness of BCG immunisation recommendations in your state/territory/area in the last 3 years?		
4.7	Based on your experiences, do you have any recommendations to improve public/provider awareness of BCG immunisation recommendations?		

5.0 Data					
5.1	How do you collect records of BCG vaccine in your state/territory/area?	(✓ tick all that apply) ☐ Electronic register ☐ Database of BCG vaccinations only ☐ Paper-based records only ☐ Other (Specify) …. ☐ None			
5.2	How many doses of BCG vaccines were administered in your state/territory/area in the last three years? (Please give total number of doses administered in each year)	Jurisdiction/Year	2012	2013	2014
		State/Territory/Area			
		No.			
5.3	How many doses of BCG vaccines were wasted in your state/territory/area in last there year? (Please give total number of doses wasted in each year)	Jurisdiction/Year	2012	2013	2014
		State/Territory/Area			
		No.			
5.4	How likely is BCG vaccination information to be entered into ACIR in your state/territory/area?	(please tick ✓) ☐ Never ☐ Rarely ☐ Sometimes ☐ Most of the times ☐ Always			
5.5	Do you have any recommendations to improve ACIR reporting of BCG vaccines in your state/territory/ area?				
5.6	How are adverse events following BCG immunisation reported in your state/territory/area?	(✓ tick all that apply) ☐ To TGA ☐ To state or territory health department ☐ Other (Specify) ……			
5.7	How likely are adverse events following BCG immunisation to be reported in your state/territory/ area?	(please tick ✓) ☐ Never ☐ Rarely ☐ Sometimes ☐ Most of the times ☐ Always			
5.8	Do you have any recommendations to improve the level of reporting adverse events following BCG immunisation in your state/territory/area?				
5.9	Has your jurisdiction undertaken any internal evaluation/s specific to the BCG vaccination program?				
5.10	Are there any other data collected or available on BCG vaccination from your jurisdiction which has not been previously mentioned?				
6.0 Program strengths and challenges **From your perspective and compared with other vaccination programs**					
6.1	What, if any, are the strengths of the BCG vaccination program in your state/territory and/or area?				
6.2	What, if any, are the challenges facing the BCG vaccination program in your state/territory and/or area?				
6.3	What, if any, are the issues/problems which you have encountered with implementing the BCG vaccination program in your state/territory and/or area?				
6.4	Based on your experiences, do you have any additional recommendations for improving BCG vaccination uptake in your state/territory and/or area?				
6.5	Do you have any further comments?				

References

1. Trunz BB, Fine P, Dye C. Effect of BCG vaccination on childhood tuberculous meningitis and miliary tuberculosis worldwide: a meta-analysis and assessment of cost-effectiveness. *Lancet* 2006;367(9517):1173–1180.
2. Toms C, Stapledon R, Waring J, Douglas P. Tuberculosis notifications in Australia, 2012 and 2013. *Commun Dis Intell* 2015;39(2):E217–E235.
3. World Health Organization. Global Tuberculosis Report 2014. Geneva: World Health Organization; 2014.
4. Australian Bureau of Statistics, Migration, Australia, 2014–15, Media Release. Available from http://www.abs.gov.au/ausstats/abs@.nsf/lookup/3412.0Media%20Release12014-15 Accessed August 2016.
5. Brewer TF. Preventing tuberculosis with bacillus Calmette-Guérin vaccine: a meta-analysis of the literature. *Clin Infect Dis* 2000;31 Suppl 3:S64–S67.
6. Australian Technical Advisory Group on Immunisation. *The Australian Immunisation Handbook*. 10th edn. Canberra: Australian Government Department of Health and Ageing; 2013.
7. National Tuberculosis Advisory Committee. The BCG vaccine: information and recommendations for use in Australia: National Tuberculosis Advisory Committee Update October 2012. *Commun Dis Intell* 2013;37(1):E65–E72.
8. Australian Government Department of Health, National Immunisation Program Schedule (From 20 April 2015). Available from http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/national-immunisation-program-schedule Accessed August 2016.
9. Australian Government Department of Health, Therapeutic Goods Administration. Tuberculosis (BCG) vaccine: urgent medicine recall. 2012. Available from: https://www.tga.gov.au/product-recall/tuberculosis-bcg-vaccine Accessed on 10 April 2016.
10. Commonwealth of Australia. *Therapeutic Goods Act 1989*. Available from: http://www5.austlii.edu.au/au/legis/cth/consol_act/tga1989191/ Accessed November 2015.
11. Australian Government Department of Health, Therapeutic Goods Administration. Access to unapproved therapeutic goods via the Special Access Scheme November 2009. Available from https://www.tga.gov.au/sites/default/files/access-sas-guidelines.pdf Accessed August 2016.
12. Marais B, Seddon JA, Detjen AK, van der Werf MJ, Grzemska M, Hesseling AC, et al. Interrupted BCG vaccination is a major threat to global child health. *Lancet Respir Med* 2016;4(4):251–253.
13. World Health Organization. BCG vaccine. WHO position paper. *Wkly Epidemiol Rec* 2004;79(4):27–38.
14. Turnbull FM, McIntyre PB, Achat HM, Wang H, Stapeldon R, Gold M, et al. National study of adverse reactions after vaccination with bacille Calmette-Guérin. *Clin Infect Dis* 2002;34(4):447–453.
15. Ritz N, Hanekom WA, Robins-Browne R, Britton WJ, Curtis N. Influence of BCG vaccine strain on the immune response and protection against tuberculosis. *FEMS Microbiol Rev* 2008;32(5):821–841.
16. Mahajan D, Cook J, Dey A, Macartney K, Menzies RI. Surveillance of adverse events following immunisation in Australia annual report, 2011. *Commun Dis Intell* 2012;36(4):E315–E332.
17. Hull BP, Dey A, Menzies RI, Brotherton JM, McIntyre PB. Immunisation coverage, 2012. *Commun Dis Intell* 2014;38(3):E208–E231.
18. Uppsala Monitoring Centre. WHO Collaborating Centre Available from for International Drug Monitoring. Available from: http://www.who-umc.org/ Accessed November 2016.
19. NSW Ministry of Health. BCG (Bacille Calmette Guerin) Vaccination. 2013. Available from: http://www0.health.nsw.gov.au/policies/pd/2013/pdf/PD2013_032.pdf Accessed July 2015.
20. NSW Ministry of Health. Request a BCG Vaccination 2016. Available from: http://www.health.nsw.gov.au/Infectious/tuberculosis/Pages/BCG-decision-tree.aspx Accessed June 2016.
21. Northern Territory Government, Centre for Disease Control. Tuberculosis Vaccination (BCG). August 2014. Available from: http://digitallibrary.health.nt.gov.au/prodjspui/handle/10137/993 Accessed July 2015.
22. Queensland Health. BCG Vaccination for Children Aged Less Than Five Years. 2013. Available from: https://www.health.qld.gov.au/chrisp/tuberculosis/documents/factsheets/english/fs-bcg-immu-english.pdf Accessed July 2015.
23. Government of South Australia, Central Northern Adelaide Health Service, Royal Adelaide Hospital. Patient information on BCG vaccination. 2008. Available from: https://www.rah.sa.gov.au/thoracic/health_programs/documents/TBEDUBCGVaccination_000.pdf Accessed July 2015.
24. Department of Health and Human Services, Victoria. Management, control and prevention of tuberculosis: Guidelines for health care providers. 2015. Available from: https://www2.health.vic.gov.au/about/publications/policiesandguidelines/tuberculosis-guidelines-2015 Accessed November 2015.
25. Government of Western Australia, Department of Health. Tuberculosis BCG vaccination. Available from: http://healthywa.wa.gov.au/Articles/S_T/Tuberculosis-BCG-Vaccination Accessed August 2016.
26. Mahajan D, Dey A, Cook J, et al. Surveillance of adverse events following immunisation in Australia annual report, 2013. *Commun Dis Intell* 2015;39:E369–E386.
27. Hendry AJ, Dey A, Beard FH, Khandaker G, Hill R, Macartney KK. Adverse events following immunisation with BCG vaccination: baseline data to inform monitoring in Australia following introduction of new unregistered BCG vaccine, 2016. *Commun Dis Intell* 2017;41(1):E470–E474.
28. Gwee A, Rodrigo R, Casalaz D, Ritz N, Curtis N. Infants born in Australia to mothers from countries with a high prevalence of tuberculosis: to BCG or not to BCG? *Med J Aust* 2013;199(5):324–326.
29. Beard F, Macartney K. Infants born in Australia to mothers from countries with a high prevalence of tuberculosis: to BCG or not to BCG? *Med J Aust* 2014;200(3):149.
30. Teo SS, Tay EL, Douglas P, Krause VL, Graham SM. The epidemiology of tuberculosis in children in Australia, 2003–2012. *Med J Aust* 2015;203(11):440.
31. Britton PN, Yeung V, Lowbridge C, Isaacs D, Marais BJ. Spectrum of disease in children treated for tuberculosis at a tertiary children's hospital in Australia. *J Pediatric Infect Dis Soc* 2013;2(3):224–231.

Community acquired syndromes causing morbidity and mortality in Australia

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Abstract

Introduction: The clinical and economic burden of infectious diseases is a substantial public health problem. The determination of the relative contributions of these diseases to the overall healthcare burden can inform priority setting, planning, and decision-making in healthcare and establish a baseline for future comparisons. Few recent studies have presented definitive data on the incidence of infectious diseases requiring hospitalisation in the Southern Hemisphere. We identified the age-specific number of hospitalisations and severe infections requiring intensive care unit admissions in the Geelong region. This was then extrapolated to calculate incidence data of these selected infectious diseases in Australia.

Methods: This observational study was performed in Geelong, the second largest city in Victoria (population of 194,566 adults ≥ 20 years). University Hospital Geelong is a public hospital with the only emergency department in Geelong during the years 2011 and 2013. Patients were identified using the International Classification of Diseases, 10th Revision Australian Modification discharge codes and diagnoses were confirmed using clinical, radiological and laboratory criteria.

Results: Between 2011 and 2013, there were 1,506 admissions for community-acquired pneumonia (CAP) (245.3 per 100,000 person years), 1,613 admissions for skin and soft tissue infections (SSTIs) (271.2 per 100,000 person years), 479 for pyelonephritis (79.7 per 100,000 person years), 131 for influenza (22.4 per 100,000 person years), and 52 for meningitis (8.9 per 100,000 person years).

Conclusion: SSTI, CAP, and pyelonephritis are common syndromes responsible for admission to hospital in Australia, with an incidence that increases with age. CAP is a major cause of morbidity and mortality in the Australian population. Influenza is associated with the greatest percentage of severe infections requiring intensive care unit admission. *Commun Dis Intell* 2017;41(1):E49–E57.

Introduction

An important input to decision-making and planning in health is an accurate, consistent and comparative description of the burden of diseases and service demand.[1] While most reported data are based on the secondary use of administrative databases, there is concern about the accuracy of coded information.[2] There is a paucity of definitive data on the incidence of common infectious diseases requiring hospitalisation in the Southern Hemisphere. Additionally, while several studies have examined the epidemiology of specific diseases and syndromes, they have not been generally compared within the same population to allow for determination of the relative contributions of different infectious diseases to the overall healthcare burden.[3,4]

The Geelong region provides an ideal opportunity to study the epidemiology of disease as it has a well-defined population and is demographically similar with regards to age distribution, ethnic makeup, and socioeconomic status to the overall Australian population.[5] Epidemiological data from this population has been used in Australian studies of osteoporosis (Geelong Osteoporosis Study), diabetes (Fremantle Diabetes Study), inflammatory bowel disease, and infectious diseases internationally.[6–8]

We aimed to estimate the age-specific incidence, mortality and length of stay of selected common infectious diseases (including the incidence of severe infection) in Australia by extrapolating incidence data from the Geelong region.

Methods

Study setting

Geelong is the second largest city in Victoria with an adult population (≥ 20 years) of approximately 194,566 (Appendix 1).[5] The median age of the population is 40 years, the population is ethnically diverse, and health care is both public and private. University Hospital Geelong is a public hospital, which had the only emergency department in Geelong during the years 2011 and 2013. Discharge data for all hospital admissions are coded according to the International Classification of Diseases, 10th Revision Australian Modification (ICD-10-AM).[9]

Data collection

Patients (≥ 20 years) with community-acquired pneumonia (CAP), skin and soft tissue infection (SSTI), influenza, meningitis, and pyelonephri-

tis were identified from discharge coding at the University Hospital Geelong. Ages above 20 were included as the Australian Bureau of Statistics divides resident population in factions, allowing suitable comparison from ages ≥ 20 onwards. A list of ICD-10-AM codes for these conditions is included in Appendix 2. This study included the above diagnoses that were identified as the presenting problem (P prefix) and ICD-10-AM code.

We excluded the following: those not admitted to hospital overnight, those transferred from other regional hospitals, and those with a postcode of residence outside the Geelong region. Data from those transferred to private hospitals was utilised for estimates of infection incidence but not used to estimate the total length of stay or presence of severe infections. The medical records of patients were examined manually by researchers (SS, ES) to confirm community onset and diagnosis against defined study criteria. Patients were excluded if they were subsequently admitted to any hospital within 14 days of the current admission, or if they developed infection within 14 days of an interventional procedure (e.g. pneumonia post gastroscopy). Individual patients were represented in the data more than once for separate incidents of infection over the study period. Data extracted included; age, gender, length of hospital stay, admission to an intensive care unit (ICU), mortality, and postcode of residence.

Definitions

Severe and prolonged infection

Severe infection was defined as an admission to ICU secondary to CAP, SSTI, influenza, meningitis, or pyelonephritis. Prolonged infection was defined as a hospital stay of more than 14 days.

Community-acquired pneumonia

A diagnosis of CAP met the following criteria:[10]

1. A history of at least 2 of the following: new onset purulent sputum, change in character of sputum or increased respiratory secretions, new onset or worsening cough, dyspnea or tachypnea, rales or bronchial breath sounds, worsening gas exchange or oxygen saturation or increased oxygen requirements.
2. At least 1 of the following 3: fever > 38.0°C, leukopenia (white cell count < 4,000 cells per mm3) or leukocytosis (white cell count ≥ 12,000 cells per mm3). If patient age was greater than 70 years, altered mental status with no other recognisable cause also met this criterion.

Evidence of new infiltrates, consolidation, and/or cavitation on chest x-ray.

Skin and soft tissue infections

Patients with a clinical diagnosis of cellulitis, erysipelas, abscesses, furuncles and carbuncles, necrotising infections, and infections associated with bites (human and animal) according to the Infectious Diseases Society of America criteria were included.[11] Surgical site infections were excluded.

Infected skin ulcers were only included if antibiotic therapy was initiated. Patients with isolated bursitis without overlying cellulitis were excluded. Patients were excluded if the sole site of infection involved bone (osteomyelitis) or muscle. If patients also had additional superficial infection (for example osteomyelitis in the setting of superficial SSTI) they were included. Patients with wounds secondary to bites were excluded if they were admitted for repair and washout prophylactically to prevent infection, however, patients who presented with an infected bite wound without prior intervention were included.

Pyelonephritis

Patients with pyelonephritis were included if they met the following criteria.

1. Temperature > 38.0°C or < 36.0°C and/or bacteraemia with a uro-pathogen.
2. The documented presence of symptoms referable to the upper or lower urinary tract including flank and/or costo-vertebral angle tenderness, and symptoms of cystitis (dysuria, frequency, supra pubic tenderness) and/or confusion in patients over 70 years of age OR a positive urine culture (with no more than 2 species of organism, at least 1 of which was quantified as 10^5 colony forming units per milliliter of urine.

Patients with an indwelling catheter or ureteric stent in situ were excluded.

Influenza

All laboratory-confirmed influenza A or B cases (by nucleic acid testing or culture from an appropriate upper respiratory tract swab specimen) were included. Data on patients with confirmed influenza at the University Hospital Geelong are collected annually for a national sentinel surveillance program.[12]

Meningitis

Patients were diagnosed with meningitis if they had either positive growth of a pathogen in the cerebrospinal fluid; OR clinical findings consistent with meningitis, i.e. fever, nuchal rigidity, and/or change in mental status with cerebrospinal fluid findings consistent with meningitis (i.e., elevated white blood cell count, diminished glucose concentration, and elevated protein concentration).

Statistical analysis

Age-specific incidence was calculated for 2011 to 2013 based on the number of cases and the person-time at risk in 10-year age groups. Denominator information for the Geelong (statistical area level 4) statistical region was obtained for the mid-interval population (2012) from the Australian Bureau of Statistics.[5] Incidence rates for each infection, by age group, were calculated using Microsoft Excel. Age- and sex-stratified incidence was calculated and extrapolated using the Australian Census annual population estimates for the year 2012. Standard errors for age stratified strata was calculated using the Poisson distribution, using the method of Rothman implemented in Stata 14.1 (College Station, Texas).[13] The standard error for directly standardised counts was estimated using the weighted sum of the age-specific variances.

Ethical considerations

This study was approved by the Barwon Health Human Research and Ethics Committee (local reference number 14/89).

Results

Community acquired pneumonia

During the study period, there were 1,506 admissions with CAP occurring among 1,432 patients (4.9% were recurrent admissions) and 1,880 admissions identified using ICD-10-AM criteria that did not meet inclusion criteria. The mean length of hospital stay was 7 days (median 5 days; range 1–54 days) and 11.3% of admissions were prolonged. CAP was associated with the highest inpatient mortality and the highest number of ICU admissions compared with the other infectious diseases in this study, with 118 deaths (8.2% of admissions) and 148 ICU admissions (9.8% of admissions) over the 3 years (Table 1).

Patients were of mean age 69.3 years (median 75; range 20–102 years) (Table 1). The annual incidence of CAP was 245.3 per 100,000 person years. The incidence of CAP increased with age, with an incidence in those over 80 years of age of 1,453.6 per 100,000 person years (Table 2 and Figure).

Skin and soft tissue infection

During the study period, there were 1,613 admissions due to SSTI occurring among 1,583 patients. This included 1,133 diagnoses of cellulitis, 292 cases of abscess, 146 infected ulcers, and 42 necrotising soft tissue infections. A total of 2,598 admissions were excluded, as they did not meet the criteria. The mean length of hospital stay was 5.3 days (median 3 days; range 1–49 days) and 7.0% of patients had a prolonged length of stay. A total of 24 patients (1.5%) were admitted to ICU and 19 patients (1.2%) died.

The annual incidence of SSTI was 271.2 per 100,000 person years and rose from 136.8 per 100,000 in the 20–29-years age group to 716.2 per 100,000 in persons aged over 80 years (Figure). Overall, SSTIs were estimated to result in 45,999 admissions to hospital each year (Table 2).

Pyelonephritis

During the study period, there were 479 admissions among 465 patients with a diagnosis of pyelonephritis (Table 1). A total of 3,962 admissions were excluded based on the defined criteria for pyelonephritis. The average length of stay was 6.6 days (median 4 days; range 1–48 days).

The incidence of pyelonephritis progressively increased after the age of 70 years with a much higher incidence among women than men (98.3 versus 59.9 per 100,000 person years in men) (Figure). The average age of patients presenting with pyelonephritis was 69.8 years, the highest compared with any other infection reviewed (median 73; range 20–93 years) (Table 1).

Figure: Estimated annual incidence of selected community-acquired infectious diseases in Australia, 2011 to 2013. Stratified according to age group

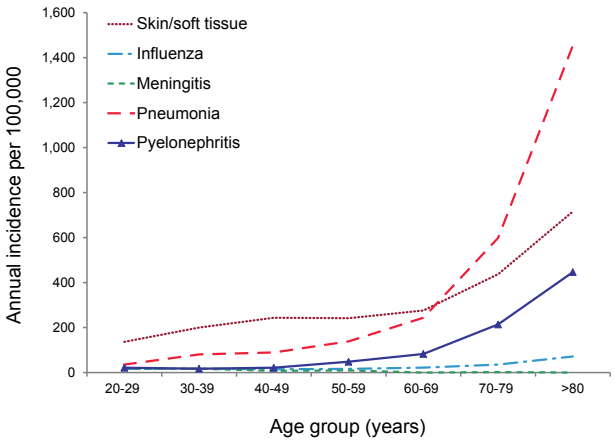

Table 1: Selected community acquired infections in people aged >20 years, Geelong Region, 2011 to 2013

Infection	Number of admissions	Number of patients	Annual incidence per 100,000 person years	Mean patient age (median; range) in years	Mean length of stay (median; range) in days	Overall mortality		Number of ICU admissions		Percentage of prolonged stay
						n	%	n	%	
CAP	1,506	1,432	245.3	69.3 (75; 20–102)	7 (5; 1–65)	118	7.8	148	9.8	10.7
SSTI	1,613	1,583	271.2	56.4 (56; 20–97)	5.3 (3; 1–49)	19	1.2	24	1.5	7.0
Pyelonephritis	479	465	79.7	69.8 (73; 20–93)	6.6 (4; 1–48)	10	2.1	3	0.6	10.2
Influenza	131	131	22.4	57.4 (59; 20–97)	5.8 (4; 1–54)	6	4.6	22	16.8	5.4
Meningitis	52	52	8.9	35.7 (33; 20–78)	4.9 (3; 1–21)	0	0.0	6	11.5	5.8

CAP = community-acquired pneumonia

SSTI = skin and soft tissue infection

ICU = intensive care unit

Table 2: Annual incidence of selected common community-acquired infections in the Geelong Region 2011 to 2013 and estimated number of Australian hospital admissions per year

Infection/age group (years)	Geelong population	Number of admissions	Incidence per 100,000 person years		Australian population	Estimated Australian cases	
			%	95% CI*		n	95% CI
CAP	583,698	1,432	245.3	232.7–258.0	16,961,179	36,624	30,117–43,131
20–29	32,643	35	35.7	24.9–49.7	3,315,513	1,185	825–1,648
30–39	32,638	79	80.7	63.9–100.6	3,143,423	2,536	2,008–3,161
40–49	35,715	96	89.6	72.6–109.4	3,166,479	2,837	2,298–3,464
50–59	34,594	144	138.8	117.0–163.4	2,890,170	4,010	3,382–4,721
60–69	28,605	209	243.5	211.6–278.9	2,248,930	5,477	4,760–6,272
70–79	17,759	319	598.8	534.8–668.2	1,328,080	7,952	7,103–8,874
>80	12,612	550	1,453.6	1,334.7–1,580.4	868,584	12,626	11,593–13,727
SSTI	583,698	1,583	271.2	257.9–284.5	16,961,179	43,773	37,542–50,004
20–29	32,643	134	136.8	114.6–162.1	3,315,513	4,537	3,801–5,373
30–39	32,638	196	200.2	173.1–230.2	3,143,423	6,293	5,442–7,238
40–49	35,715	261	243.6	214.9–275.0	3,166,479	7,714	6,806–8,708
50–59	34,594	251	241.9	212.9–273.7	2,890,170	6,990	6,152–7,910
60–69	28,605	237	276.2	242.1–313.7	2,248,930	6,211	5,445–7,054
70–79	17,759	233	437.3	383.0–497.2	1,328,080	5,808	5,086–6,604
>80	12,612	271	716.2	633.5–806.8	868,584	6,221	5,502–7,008

Infection/age group (years)	Geelong population	Number of admissions	Incidence per 100,000 person years		Australian population	Estimated Australian cases	
			%	95% CI*		n	95% CI
Pyelonephritis	583,698	465	79.7	72.4–86.9	16,961,179	11,911	8,211–15,612
20–29	32,643	21	21.4	13.3–32.8	3,315,513	711	440–1,087
30–39	32,638	17	17.4	10.1–27.8	3,143,423	546	318–874
40–49	35,715	23	21.5	13.6–32.2	3,166,479	680	431–1,020
50–59	34,594	50	48.2	35.8–63.5	2,890,170	1,392	1,034–1,836
60–69	28,605	71	82.7	64.6–104.4	2,248,930	1,861	1,453–2,347
70–79	17,759	114	214.0	176.5–257.1	1,328,080	2,842	2,344–3,414
>80	12,612	169	446.7	381.9–519.3	868,584	3,880	3,317–4,511
Influenza	583,698	131	22.4	18.6–26.3	16,961,179	3,625	1,800–5,450
20–29	32,643	16	16.3	9.3–26.5	3,315,513	542	310–880
30–39	32,638	17	17.4	10.1–27.8	3,143,423	546	318–874
40–49	35,715	16	14.9	8.5–24.3	3,166,479	473	270–768
50–59	34,594	17	16.4	9.5–26.2	2,890,170	473	276–758
60–69	28,605	19	22.1	13.3–34.6	2,248,930	498	300–778
70–79	17,759	19	35.7	21.5–55.7	1,328,080	474	285–740
>80	12,612	27	71.4	47.0–103.8	868,584	620	408–902
Meningitis	583,698	52	8.9	6.5–11.3	16,961,179	1628	592–2,663
20–29	32,643	16	16.3	9.3–26.5	3,315,513	542	310–880
30–39	32,638	17	17.4	10.1–27.8	3,143,423	546	318–874
40–49	35,715	8	7.5	3.2–14.7	3,166,479	237	102–466
50–59	34,594	10	9.6	4.6–17.7	2,890,170	279	134–512
60–69	2,8605	0	0.0	0.0–4.3	2,248,930	0	0–97
70–79	17,759	1	1.9	0.0–10.5	1,328,080	25	1–139
>80	12,612	0	0.0	0.0–9.7	868,584	0	0–85

CI: confidence intervals

CAP = community-acquired pneumonia

Of all admissions with pyelonephritis, 441 (92.0%) had positive urine microbiology results, 153 patients (32%) were bacteraemic and 27 cases were identified based on clinical symptoms and signs consistent with systemic illness. Causative pathogens isolated from blood culture, urine, or both; in order of incidence included *Escherichia coli*, *Klebsiella pneumoniae*, *Enterococcus faecalis*, *Pseudomonas aeruginosa*, *Proteus mirabilis*, *Morganella morganii*, *Enterobacter aerogenes*, and *Citrobacter koseri*.

A total of 3 patients were admitted to ICU (0.6%) and 10 (2.1%) died.

Influenza

During the study period, there were 131 cases of influenza. The mean age of patients was 57.4 years (median 59; range 20–97 years). Although the mean length of stay was 5.8 days, the range was 1–54 days (median 4 days). Influenza contributed to the largest percentage of admissions to ICU (17%) for any infection in this study (Table 1). In addition, 5.4% of patients had a prolonged length of stay and nearly 5% of the patients died.

The annual incidence of influenza was 22.4 per 100,000 person years. The majority of cases (95 of 131, 72.5%) occurred in the winter months of July to September. Overall, influenza is expected to result in more than 15,000 admissions to hospital in Australia annually (Table 2).

Meningitis

During the study period, 52 patients were admitted with meningitis (128 did not meet criteria). Of these, 7 cases (14.3%) had bacterial meningitis, and of the 42 patients with aseptic meningitis, a viral etiology was diagnosed in 16 cases. The mean age of patients with meningitis was the youngest of all infections at 35.7 years (median 33; range 20–78) with only 1 patient above the age of 60 years. The annual incidence of meningitis was 8.9 per 100,000 person years and the average length of stay was 4.9 days (median 3 days; range 1–21 days). A total of 6 (11.5%) required ICU admission and there were no deaths.

Discussion

In contrast with most previous population-based studies using coded data alone, the current study used clinical criteria and the results of investigations to rigorously define cases of common infectious diseases.[4] We found a considerable proportion of ICD10-AM coded infections were excluded based on clinical criteria, and therefore coded data are likely to over-estimate disease incidence. The exception to this is the incidence of influenza, which is likely an under-estimation due to missed diagnosis secondary to under-utilisation of Influenza-swabs.[14] Nevertheless, these common infections requiring hospitalisation represent a significant burden; based on age stratified incidence standardised to the Australian population, we estimate that there were over 36,000 admissions with CAP, over 43,000 admissions with SSTI, over 11,000 admissions with pyelonephritis, over 3,600 admissions with influenza, and over 1,500 admissions with meningitis in Australian adults each year (Table 2).

A study of the global burden of disease in 2001 estimated that lower respiratory tract infection is the 4th leading cause of death in high-income countries, and responsible for 4.4% of total deaths.[15] In this study we estimated a mortality of 7.8% in hospitalised adults. This was almost double that of the other community-acquired infections in this study. Our reported incidence of CAP requiring hospitalisation is similar to that found in other population based studies, such as Marston et al. where CAP incidence was 266.8 per 100,000 overall, and 1,012.3 per 100,000 in the elderly (> 65 years).[16]

SSTI was the most common infectious disease requiring admission in this study, and the incidence was higher in males and in the elderly. Other studies have noted an increasing incidence since the 1960s.[2] The reason for this is unclear but is likely multifactorial, including increased health professional awareness, increase in healthcare associated methicillin resistant *Staphylococcus aureus*, prevalence of diabetes, the growing number of people on dialysis, injecting drug use, and travel.[17]

There is limited epidemiological data of the burden of pyelonephritis.[18] A 2005 study estimated that the annual societal cost of treatment of acute pyelonephritis was estimated to be $2.14 billion in the United States of America.[19] Our findings are similar to that of Foxman et al, who described an incidence of approximately 117 per 100,000 among women and a lower incidence of 24 per 100,000 in men.[20] Our results show an annual incidence of pyelonephritis of 98.3 per 100,000 person years among women compared with 59.9 per 100,000 person years among men. The incidence increases with age (Figure).

Of the infections studied here, patients admitted with influenza were at the highest risk of requiring ICU admission, with around 1 in 6 patients admitted to ICU. This is consistent with Australian national surveillance, in which 17% of patients with influenza admitted to hospital required an ICU stay in 2013.[12] This reinforces the need to improve influenza vaccine coverage.[21,22] While the

incidence of severe influenza in adults requiring hospitalisation varies from season to season, the observed incidence in this study is broadly similar to other studies.[23]

Albeit less common than other community-acquired infections, meningitis is of significant public health interest. Although meningitis was associated with a high percentage of ICU admissions (12%) second only to Influenza (17%), there were no deaths in this young population with a median age of 33 years. Meningitis was uncommonly diagnosed in this study with an incidence of 8.9 per 100,000 person years, and the majority of cases were under the age of 40 years. These findings are in keeping with the declining incidence and mortality of meningitis in the setting of effective immunisation programs.[24,25]

In Australia, the highest rates of meningococcal disease notification are in children under 5 years of age with a second peak in the 15–24 years age group. Death secondary to meningitis in Australia is lowest in the 5–24 year age group and highest in the over 60 years age group.[26]

Incidence data for invasive meningococcal disease in Australia reveals that the introduction of a single dose of meningococcal C conjugate vaccine in the second year of life has resulted in near elimination of serogroup C disease in all age groups in Australia.[23] Interestingly, invasive pneumococcal disease and bacterial meningitis both declined dramatically among children and adults in the United States of America when the 7-valent pneumococcal conjugate vaccine and the *Haemophilus influenzae* type b conjugate vaccine were introduced for infants, although a similar reduction was not seen in Australia with the introduction of the *H. influenzae* PRP-T conjugate vaccine among Indigenous children.[25,27]

There are limitations to this study. Although the city of Geelong is geographically separated from Melbourne, we cannot exclude the possibility that a small number of patients may have been treated at hospitals outside the region. Disease burden may have also been underestimated because we only analysed data on patients aged 20 years or over and excluded patients with these infections admitted directly to private hospitals bypassing the emergency department. We also excluded patients transferred to private hospitals from our length of stay estimates, which may have affected the results. Also of note, this study is not representative of the Indigenous population as the estimated Aboriginal or Torres Strait Islander population in the Geelong region is 0.8% compared with 2.5% Australia-wide.[28] We acknowledge that the small number of patients with meningitis limited the statistical precision of the population incidence of this disease, although declining incidence of meningitis is consistent with other Australian data.[26]

Diagnostic codes were used to identify cases, and while coded data may lack accuracy, we mitigated this limitation by analysing medical records against defined clinical, radiological and laboratory criteria. In addition, although cases were reviewed carefully on an individual basis to assess the cause of hospital admission and the eventual cause of death, given the difficulty in assigning the cause of death and length of stay to any 1 infection in a complex patient presentation, the mortality and length of stay data should be interpreted with caution.

Overall, we found that these selected infectious diseases result in annual hospital admissions for an estimated 0.58% (97,561 admissions) of the total population of Australia aged 20 years or over (16,961,179).[28] More importantly, 54,495, i.e. 55.9% of these admissions are in people over 60 years of age. Of note, this study investigated the incidence of selected infections requiring hospitalisation but did not look at the direct cost burden or incidence in the community. Further studies should examine both the cost-burden of hospitalisation and the incidence of these selected infections treated in the community setting. This would help assess the true burden to the health-care system and measure the effectiveness of preventive strategies.

Conclusion

Knowledge of the burden and preventability of selected infections can inform priority setting and resource deployment in health care. This study assessed admissions to hospital and therefore health care utilisation for selected community acquired infections. Our results show that SSTI, CAP, and pyelonephritis are common infections leading to hospital admissions with an incidence that increases with age. Pneumonia is responsible for significant morbidity and mortality in Australia. Assessment of the epidemiology and incidence of common syndromes is needed to guide healthcare planning.

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Appendix 1: Adult population structure of Geelong statistical region and Australia, 2012

Age group (years)	Geelong			Australia		
	Male	Female	Total	Male	Female	Total
20–29	16,569	16,074	32,643	1,685,119	1,630,394	3,315,513
30–39	16,163	16,475	32,638	1,571,153	1,572,270	3,143,423
40–49	17,568	18,147	35,715	1,569,694	1,596,785	3,166,479
50–59	16,985	17,609	34,594	1,430,255	1,459,915	2,890,170
60–69	13,960	14,645	28,605	1,117,447	1,131,483	2,248,930
70–79	8,416	9,343	17,759	638,160	689,920	1,328,080
>80	4,912	7,700	12,612	341,239	527,345	868,584
Total	94,573	99,993	1,945,66	83,530,67	8,608,112	16,961,179

Appendix 2: Screening ICD-10 codes used to identify hospital admissions for common community-acquired infections

Skin/soft tissue infection	Cellulitis – all sites Erisipelas A26.9, A26.0, A26.7, A26.8 Gangrene, gangrenous R02 Cutaneous, spreading R02 Fournier's N49.8, female N76.8 Limb (lower) (upper) R02 Mouth A69.0 Perineum R02 Scrotum N49.2 Gas gangrene A48.0 Impetigo L40.1, H03.8, H62.4, L01.0 Wound infection with foreign body T89.01 Necrotising fasciitis – M72.4, M72.9, M72.6 Bites (animal and human) W50–W64 Abscess Ankle, foot, heel, thigh, thumb, toe (any), leg (any part), limb (lower) (upper), hand, arm (any part), axilla, wrist, web/palmar space, shoulder (region) – all L02.4 Nail L03.01, L03.02 Head L02.8 Back, groin, mons pubis, navel L02.2 Buttock L02.3 Neck (region) L02.1 Lip K13.0 Orbit, orbital H05.0 Cutaneous L02.9 Face (any part except ear, eye, nose) – L02.0 Pilonidal L05.0
Community-acquired pneumonia	J12 to J18
Meningitis	G00
Influenza	J09 – J11
Pyelonephritis	Pyelonephritis N12 Sepsis A41.9 plus Urinary T83.5 Urinary NEC T83.5 *Escherichia coli* A41.51 Gram-negative (organism) A41.50 Anaerobic A41.50 *Escherichia coli* A41.51 Pseudomonas A41.52 Specified NEC A41.58 Pseudomonas A41.52 Infection urinary (tract) NEC N39.0 Complicating pregnancy O23.4 Newborn P39.3 Puerperal (postpartum) O86.2 Tuberculous A18.1

References

1. Australian Institute of Health and Welfare. Australian Burden of Disease Study: Impact and Causes of Illness and Death in Australia 2011. Australian Burden of Disease Study series no. 3. BOD 4. Canberra: Australian Institute of Health and Welfare; 2016.
2. Goto M, Ohl ME, Schweizer ML, Perencevich EN. Accuracy of administrative code data for the surveillance of healthcare-associated infections: a systematic review and meta-analysis. *Clin infect Dis* 2014;58(5):688–696.
3. Andersen LK, Davis MD. The epidemiology of skin and skin-related diseases: a review of population-based studies performed by using the Rochester Epidemiology Project. *Mayo Clin Proc* 201388(12):1462–1467.
4. Griffin MR, Zhu Y, Moore MR, Whitney CG, Grijalva CG. US hospitalizations for pneumonia after a decade of pneumococcal vaccination. *N Engl J Med* 2013;369(2):155–163.
5. National Regional Profile: Greater Geelong City Part A (Statistical Subdivision). Available from: http://www.abs.gov.au Accessed on 22 October 2015.
6. Pasco JA, Nicholson GC, Kotowicz MA. Cohort profile: Geelong osteoporosis study. *Int J Epidemiol* 2012;41(6):1565–1575.
7. Davis TM, Bruce DG, Davis WA. Cohort profile: the Fremantle diabetes study. *Int J Epidemiol* 2013;42(2):412–421.
8. Wilson J, Hair C, Knight R, Catto-Smith A, Bell S, Kamm M, et al. High incidence of inflammatory bowel disease in Australia: A prospective population-based Australian incidence study. *Inflamm Bowel Dis* 2010;16(9):1550–1556.
9. World Health Organization. International Classification of Diseases: ICD-10 online versions. Available from: http://www.who.int/classifications/icd/icdonlineversions/en/ Accessed on 26 October 2015.
10. Watkins RR, Lemonovich TL. Diagnosis and management of community-acquired pneumonia in adults. *Am Fam Physician* 2011;83(11):1299–1306.
11. Stevens DL, Bisno AL, Chambers HF, Dellinger EP, Goldstein EJ, Gorbach SL, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. *Clin Infect Dis* 2014 59(2):e10–e52.
12. Cheng AC, Dwyer DE, Holmes M, Irving LB, Brown SG, Waterer GW, et al. Influenza epidemiology, vaccine coverage and vaccine effectiveness in sentinel Australian hospitals in 2013: the Influenza Complications Alert Network. *Commun Dis Intell* 2013;38(2):E143–E149.
13. Rothman KJ, Greenland S, Lash TL, eds. *Modern epidemiology.* Lippincott Williams & Wilkins; 2008.
14. Harper SA, Bradley JS, Englund JA, File TM, Gravenstein S, Hayden FG, McGeer AJ, et al. Seasonal influenza in adults and children—diagnosis, treatment, chemoprophylaxis, and institutional outbreak management: clinical practice guidelines of the Infectious Diseases Society of America. *Clin Infect Dis* 2009;48(8):1003–1032.
15. Lopez AD, Mathers CD, Ezzati M, Jamison DT, Murray CJ. Global and regional burden of disease and risk factors, 2001: systematic analysis of population health data. *Lancet* 2006; 367(9524):1747–1757.
16. Marston BJ, Plouffe JF, File TM Jr, Hackamn BA, Salstrom SJ, Lipman HB, et al. Incidence of community-acquired pneumonia requiring hospitalization: results of a population-based active surveillance study in Ohio. *Arch Intern Med* 1997;157(15):1709–1718.
17. Dryden MS. Skin and soft tissue infection: microbiology and epidemiology. *Int J Antimicrob Agents* 2009;34 Suppl 1:S2–S7.
18. Czaja CA, Scholes D, Hooton TM, Stamm WE. Population-based epidemiologic analysis of acute pyelonephritis. *Clin Infect Dis* 2007;45(3):273–280.
19. Brown P, Ki M, Foxman B. Acute pyelonephritis among adults: cost of illness and considerations for the economic evaluation of therapy. *Pharmacoeconomics* 2005;23(11):1123–1142.
20. Foxman B, Klemstine KL, Brown PD. Acute pyelonephritis in US hospitals in 1997: hospitalization and in-hospital mortality. *Ann Epidemiol* 2003;13(2):144–150.
21. Lee KW, Murrell E. Influenza prevention and treatment strategies in the elderly. *Annals of Long Term Care* 2006;14(9):20.
22. Bridges CB, Thompson WW, Meltzer MI, et al. Effectiveness and cost-benefit of influenza vaccination of healthy working adults: a randomized controlled trial. *JAMA* 2000;284(13):1655–1663.
23. Reed C, Chaves SS, Kirley PD, Emerson R, Aragon D, Hancock EB, et al. Estimating influenza disease burden from population-based surveillance data in the United States. *PloS One* 2015;10(3):e0118369.
24. Lawrence GL, Wang H, Lahra M, Booy R, McIntyre PB. Meningococcal disease epidemiology in Australia 10 years after implementation of a national conjugate meningococcal C immunization programme. *Epidemiol Infect* 2016144(11):1–10.
25. Thigpen MC, Whitney CG, Messonnier NE, Zell ER, Lynfield R, Hadler JL, et al. Bacterial meningitis in the United States, 1998–2007. *N Engl J Med* 2011;364(21):2016–2025.
26. Chiu C, Dey A, Wang H, Menzies R, Deeks S, Mahajan D, et al. Vaccine preventable diseases in Australia, 2005 to 2007. *Commun Dis Intell* 2010;34 Suppl:S1–S167.
27. Menzies RI, Bremner KM, Wang H, Beard FH, McIntyre PB. Long-Term Trends in *Invasive Haemophilus* influenzae type b disease among Indigenous Australian children following use of PRP-OMP and PRP-T vaccines. *Pediatr Infect Dis J* 2015;34(6):621–626.
28. Australian Bureau of Statistics. Census of Population and Housing - Counts of Aboriginal and Torres Strait Islander Australians, 2011. ABS cat. No:2075.0 [Online]. Available from: http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/2075.0main+features32011 Accessed on 8 August 2016.

Policies and guidelines

REVISED SURVEILLANCE CASE DEFINITIONS

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

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

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

These case definitions were implemented on 1 January 2017 and supersede any previous versions.

Diphtheria case definition

Reporting

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

Confirmed case

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

Laboratory definitive evidence

Isolation of toxigenic* *Corynebacterium diphtheriae* or toxigenic* *C. ulcerans* from site of clinical evidence.

Clinical evidence – confirmed case

Upper respiratory tract infection

OR

Skin lesion

Probable case

A probable case requires:

Laboratory suggestive evidence AND **clinical evidence**

OR

Clinical evidence AND **epidemiological evidence**.

Laboratory suggestive evidence

Isolation of *C. diphtheriae* or *C. ulcerans* from a respiratory tract specimen (toxin production unknown).

Clinical evidence – probable case

Upper respiratory tract infection with an adherent membrane of the nose, pharynx, tonsils or larynx

Epidemiological evidence

An epidemiological link is established when there is:

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

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

AND

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

AND

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

* as indicated by detection of toxin gene by nucleic acid testing

Summary of changes to diphtheria surveillance case definition	**Confirmed case** **Laboratory definitive evidence** Added 'from site of clinical evidence' **Clinical evidence – confirmed case** Changed to 'Clinical evidence - confirmed case Replaced 'Pharyngitis and/or laryngitis (with or without a membrane) OR Toxic (cardiac or neurological) symptoms' With 'Upper respiratory tract infection OR Skin lesion' **Laboratory suggestive evidence** Added 'from a respiratory tract specimen (toxin production unknown).' Added **Clinical evidence – probable case** Upper respiratory tract infection with an adherent membrane of the nose, pharynx, tonsils or larynx

Varicella-zoster infection (not elsewhere classified) case definition

Reporting

Only confirmed cases should be notified.

Confirmed case

A confirmed case requires laboratory definitive evidence, either in the absence of clinical

Information or where clinical evidence does not meet criteria for varicella-zoster infection (chickenpox) or varicella-zoster infection (shingles).

Laboratory definitive evidence

1. Isolation of varicella-zoster virus.

OR

2. Detection of varicella-zoster virus by nucleic acid testing.

OR

3. Detection of varicella-zoster virus antigen by direct fluorescent antibody testing.

OR

4. Detection of varicella-zoster virus-specific IgM in an unvaccinated person.

Summary of changes to varicella zoster infection (not elsewhere classified) surveillance case definition	Name change: changed unspecified to not elsewhere classified. Removal of wording 'from a skin or lesion swab' from relevant criteria to broaden the types of laboratory specimen that can be tested

Annual reports

Australian Gonococcal Surveillance Programme annual report, 2015

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

Abstract

The Australian Gonococcal Surveillance Programme (AGSP) has continuously monitored antimicrobial resistance in clinical isolates of *Neisseria gonorrhoeae* from all Australian states and territories since 1981. In 2015, there were 5,411 clinical isolates of gonococci from public and private sector sources tested for *in vitro* antimicrobial susceptibility by standardised methods. Current treatment recommendations for the majority of Australian states and territories is a dual therapeutic strategy of ceftriaxone and azithromycin. Decreased susceptibility to ceftriaxone (minimum inhibitory concentration or MIC value 0.06–0.125 mg/L) was found nationally in 1.8% of isolates, which was lower than that reported in the AGSP annual report 2014 (5.4%). The highest proportions were reported from South Australia and New South Wales (3.6% and 2.7% respectively). High level resistance to azithromycin (MIC value ≥256 mg/L) was again reported in 2015, with 1 strain in each of New South Wales and urban Western Australia. There was no reported Azithromycin resistance in the Australian Capital Territory, the Northern Territory, or remote Western Australia. The proportion of strains resistant to penicillin in urban and rural Australia ranged from 8.7% in Tasmania to 33% in the Australian Capital Territory. In rural and remote Northern Territory, penicillin resistance rates remain low (2.2%). In remote Western Australia relatively low numbers of strains are available for testing, however there is now widespread molecular testing for penicillin resistance in Western Australia to monitor resistance and inform guidelines and these data are included in the AGSP annual report. Quinolone resistance ranged from 11% in the urban and rural areas of the Northern Territory, to 41% in South Australia. Quinolone resistance rates remain comparatively low in remote areas of the Northern Territory (3.3%) and remote areas of Western Australia (3.4%). There was no reported quinolone resistance in Tasmania, but the number of isolates tested was relatively low. Azithromycin resistance ranged from 1.8% in Victoria to 5.8% in Queensland. *Commun Dis Intell* 2017;41(1):E60–E67.

Keywords: antimicrobial resistance; disease surveillance; gonococcal infection; *Neisseria gonorrhoeae*

Introduction

Concerns regarding gonococcal antimicrobial resistance (AMR) persist internationally, and continues to be viewed as an urgent public health threat as identified by the United States Centers for Disease Control and Prevention in 2013.[1] The corollary of the emergence and spread of multidrug-resistant gonorrhoea is predicted to pose significant collateral health and financial costs.[1] The reliance on ceftriaxone and azithromycin for treatment in most settings continues with the future direction of gonococcal treatment uncertain, and there are no new or ideal alternative therapeutic strategies identified in the event of the spread of AMR.[2] In recent years in Australia, there has been a significant increase in rates of gonococcal disease observed in both males and females in the eastern states (Victoria, New South Wales and Queensland), and males in the Australian Capital Territory.[3] In contrast, gonococcal disease notification rates in the Indigenous populations from the remote regions of the Western states of Northern Territory and Western Australia, are markedly higher but relatively stable.[3] However, in these remote regions of Northern Territory and Western Australia with much higher rates of gonococcal disease notification rates, the AMR rate remains paradoxically low in locally-acquired infections, and an oral penicillin based therapeutic strategy remains recommended for use.[4]

In 2013, the Australian Gonococcal Surveillance Programme (AGSP) reported that the proportion of strains with decreased susceptibility to ceftriaxone nationally was 8.8%, double that reported in 2012 (4.4%). New South Wales and Victoria reported the highest proportions (11.8%) and these states also had the highest increases in disease notifications.[5] Coincident with this, in 2013, was the reporting of high level resistance to azithromycin (MIC value >256 mg/L), in 2 strains from Victoria and 2 from Queensland.[6] Also in 2013, an imported multidrug-resistant gonococcal strain, known as the A8806 strain, with a ceftriaxone MIC of 0.5 mg/L, the highest ever reported in Australia, was identified in Australia.[7] This A8806 strain showed key genetic similarities to the ceftriaxone-resistant strain H041, reported from a single case in Japan and not subsequently reported.[7]

Enhanced surveillance in the Northern Territory and Queensland has not detected further evidence of the A8806 strain in 2014 or 2015 (unpublished data from the National Neisseria Network).

In the context of the heightened awareness of AMR, and increasing disease notification rates reported in Australia and elsewhere, the widespread move to nucleic acid amplification testing (NAATs), has been identified as a concern as broad based antimicrobial susceptibility testing is not possible with NAATs. However, directed NAATs such as the assay developed to detect *Neisseria gonorrhoeae* penicillinase production[8,9] (the primary cause of penicillin resistance in remote regions in Australia) was the first documented use of molecular testing for gonococcal antimicrobial resistance detection and surveillance to monitor AMR, and inform local treatment guidelines.[9]

Of the World Health Organization (WHO) estimated 106 million new *N. gonorrhoeae* infections reported in those aged 15–49 years annually worldwide, almost two-thirds occur in the Asia–Pacific Region.[10] The WHO data from the Asia–Pacific indicates that, along with a disproportionate burden of gonococcal disease, there are high levels of gonococcal AMR in the region. Compounding these factors is the concern that uncontrolled antimicrobial use in countries in these regions provides ideal conditions for the development of AMR.[11] AMR in *N. gonorrhoeae* has long been influenced by the introduction of multi-resistant strains from overseas.[12] In this context the importation and spread of resistant gonococcal strains and/or resistance developing under selection pressure is an ongoing concern.

Strategies for treating and controlling gonorrhoea are based on regimens effecting cure in a minimum of 95% of cases. Surveillance data, derived from continuous monitoring of resistance to the antibiotics in clinical use, is therefore critical to monitor AMR, detect imported or novel resistance and to inform treatment guidelines.[13] The WHO has called for enhanced surveillance as a fundamental component of the Global Action Plan to control the spread and impact of gonococcal AMR.[14]

The National Neisseria Network (NNN) is a collaboration of *Neisseria* reference laboratories in each state and territory that perform phenotypic and genotypic testing of clinical isolates of pathogenic *Neisseria* species. Clinical isolates are referred to the jurisdictional NNN laboratories from both public and private sector laboratories representing as wide a section of the community as possible, for determination of phenotypic and genotypic characteristics, including antimicrobial resistance, and additional investigations where required. The AGSP is a key activity of the NNN and has continuously monitored the susceptibility of *N. gonorrhoeae* since 1981, making it the longest, continually running, national surveillance system for gonococcal AMR. In this AGSP annual report we will also report the molecular surveillance data from the implementation of the penicillinase-producing *Neisseria gonorrhoeae* (PPNG) assay in remote Western Australia to supplement the AGSP data. This is amid increasing concerns nationally of the status of gonococcal AMR in Australia.

Methods

The NNN AMR data for gonococcal isolates are collated for the AGSP quarterly and annual reports. Gonorrhoea is a notifiable disease in Australia and each confirmed case is notified to the National Notifiable Diseases Surveillance System (NNDSS). The number of isolates tested by the NNN and reported by the AGSP represents a proportion of the number of cases reported to the NNDSS. The NNN tests approximately one-third of the number of notified cases in Australia.

The NNN laboratories test gonococcal isolates for susceptibility to penicillin (representing this group of antibiotics); ceftriaxone (representing later generation cephalosporin antibiotics); ciprofloxacin (representing quinolone antibiotics); azithromycin; spectinomycin; and for high level plasmid mediated resistance to tetracycline using previously described standardised methodology to determine the MIC values.[15–16] The MIC value is the least concentration of an antibiotic that inhibits *in vitro* growth under defined conditions. The AGSP conducts a program-specific quality assurance program.[17]

Antibiotic susceptibility data from each jurisdiction are submitted quarterly to the coordinating laboratory (the Neisseria Reference Laboratory and WHO Collaborating Centre for Sexually Transmitted Diseases, Sydney), which collates the results for reporting. Where available, the AGSP collects data on the gender of the patient, country of acquisition, and site of isolation of gonococcal strains. Data from isolates from all jurisdictions is predominantly from urban centres. Data from the Northern Territory and Western Australia are further divided into urban versus rural and remote as therapeutic recommendations differ.

Statistics

Statistical analysis was performed using Prism % version 5.0d. Results were compared using Fisher's exact test for differences in proportions.

Results

Number of isolates

There were 5,411 gonococcal isolates tested in NNN laboratories in 2015, representing 28% of the 19,092 cases of gonococcal infection notified to the NNDSS in 2015 (Table 1). This was lower than the proportion tested in 2014 (31%) and lower than the range of 33% to 42% referred between 2008 and 2013.

Source of isolates

There were 4,505 isolates from men (83%) and 791 (17%) from women (Table 2). There were 6 isolates from patients of unknown gender. The proportion of gonococcal isolates from males and females tested by the AGSP has remained stable over recent years (2009–2014); ranging between 18% and 20% for women and 80% and 83% for men. The infected site was reported as 'other' or not specified for 35 isolates from males and 14 isolates from females (Table 2). Isolates from urine samples were regarded as genital tract isolates.

Table 1: Number of Australian Gonococcal Surveillance Programme gonococcal isolates tested as a proportion of National Notifiable Diseases Surveillance System gonorrhoea notifications, Australia, 2015, by state or territory

State or territory	Number of isolates tested	Number of cases notified	Number of isolates tested/ number of cases notified %
Australian Capital Territory	69	141	49
New South Wales	1,905	5,460	35
Northern Territory	258	1,851	14
Queensland	728	3,033	24
South Australia	251	798	31
Tasmania	23	56	41
Victoria	1,695	5,497	31
Western Australia	482	2,256	21
Australia	5,411	19,092	28

Table 2: Gonococcal isolates, Australia, 2015, by sex, site and state or territory tested

Site	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Male									
Genital	28	877	150	385	105	9	722	256	2,532
Rectal	23	474	2	110	55	4	446	49	1,163
Pharynx	11	323	2	56	33	6	305	23	759
DGI	0	7	2	4	0	0	3	0	16
Other/NS	1	9	2	8	4	2	7	2	35
Total	63	1,690	158	563	197	21	1,483	330	4,505
Female									
Genital	6	172	94	151	40	2	190	142	797
Rectal	0	3	0	2	6	0	1	3	15
Pharynx	0	32	0	2	6	0	19	5	64
DGI	0	0	4	4	1	0	0	1	10
Other/NS	0	3	1	6	1	0	2	1	14
Total	6	210	99	165	54	2	212	152	900
Unknown									
Total	0	5	1	0	0	0	0	0	6
Total	69	1,905	258	728	251	23	1,695	482	5,411

DGI Disseminated gonococcal infection
NS Not specified

Antibiotic susceptibility patterns

As in past years the patterns of gonococcal antibiotic susceptibility differed between the various states and territories. The data are presented by region as well as aggregated for Australia (Table 3).

Ceftriaxone

From 2001 onwards, gonococcal isolates categorised as having decreased susceptibility to ceftriaxone by the AGSP criteria (MIC values 0.06–0.125 mg/L) have been reported in Australia. The proportion of gonococci with decreased susceptibility to ceftriaxone nationally, increased incrementally from 0.6% in 2006, to 4.4% in 2012, then in 2013 doubled to 8.8%. In 2014, the proportion decreased to 5.4% and again decreased in 2015 to 1.8% (Table 4).

Ceftriaxone decreased susceptibility includes the MIC values 0.06 and 0.125 mg/L. The right shift in the distribution of ceftriaxone MIC values over recent years (2011–2013) (Table 5), is statistically significant with a sustained increase in the proportion of strains with an MIC value of 0.06 mg/L (2011–2012: ($P = 0.02$, 95% CI: 1.04–62), and 2012–2013 ($P < 0.0001$, 95% CI: 1.70–2.38)). The proportion of strains nationally with an MIC value of 0.06 mg/L–0.125 mg/L decreased in 2014 to 5.4% and then in 2015 to 1.7%.

Table 3: Proportion of gonococcal isolates with resistance to azithromycin, penicillin and ciprofloxacin and decreased susceptibility to ceftriaxone reported, Australia, 2015, by state or territory

State or territory	Number of isolates tested	Decreased susceptibility Ceftriaxone		Resistance Azithromycin		Penicillin		Ciprofloxacin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	69	0	0.0	0	0.0	23	33.3	18	26.1
New South Wales	1,905	52	2.7	43	2.3	588	30.9	684	35.9
Queensland	728	7	1.0	42	5.8	201	27.6	186	25.5
South Australia	251	9	3.6	7	2.8	52	20.7	103	41.0
Tasmania	23	0	0.0	1	4.3	2	8.7	0	0.0
Victoria	1,695	25	1.5	30	1.8	257	15.2	383	23.0
Northern Territory/ Urban	76	0	0.0	0	0.0	11	14.5	8	10.5
Northern Territory/ Remote & Rural	182	0	0.0	0	0.0	4	2.2	6	3.3
Western Australia/Urban & Rural	395	5	1.3	15	3.8	77	19.5	82	20.8
Western Australia/Remote	87	0	0.0	0	0.0	2	2.3	3	3.4
Australia	5,411	98	1.8	138	2.6	1,217	22.5	1,473	27.2

Table 4: Number of gonococcal isolates with decreased susceptibility to ceftriaxone (MIC 0.06–0.125 mg/L), Australia, 2011 to 2015, by state or territory

State or territory	Decreased susceptibility to ceftriaxone 2011		2012		2013		2014		2015	
	n	%	n	%	n	%	n	%	n	%
Australian Capital Territory	2	3.1	2	3.6	0	0.0	2	2.7	0	0.0
New South Wales	58	4.4	76	4.5	183	11.8	119	7.1	52	2.7
Northern Territory	2	0.4	0	0.0	4	1.5	4	1.7	0	0.0
Queensland	18	2.3	17	2.4	33	4.9	21	3.2	7	1.0
South Australia	1	0.7	1	0.7	4	1.9	2	1.0	9	3.6
Tasmania	0	0.0	0	0.0	11	24.4	0	0.0	0	0.0
Victoria	50	5.3	105	8.4	181	11.8	95	6.6	25	1.5
Western Australia	3	0.7	6	1.2	13	2.7	15	3.0	5	1.0
Australia	134	3.2	207	4.4	429	8.8	258	5.4	98	1.8

The proportion of strains with a ceftriaxone MIC 0.125 mg/L also increased from 0.1% in 2010 and 2011, to 0.3% in 2012 and to 0.6% in 2013 and 2014. These differences were not significant, which may be attributable to the low number of strains in this MIC category. In 2015, the proportion of strains with an MIC value of 0.125 mg/L decreased to 0.1% (Table 5). No isolates of *N. gonorrhoeae* with an MIC value greater than 0.125 mg/L were reported from Australia in 2015.

Azithromycin

Nationally, the proportion of isolates exhibiting resistance (2.6%) (Table 3) was slightly higher than that reported for 2014 (2.4%) and 2013 (2.1%) and higher than in 2011 to 2012 (1.1% to 1.3%). The proportion of isolates exhibiting resistance was highest in Queensland (5.8% in 2015, compared with 3.5% in 2014), followed by urban Western Australia (3.8% in 2015, compared with 5.3% in 2014). In 2015, there were 2 isolates, 1 from New South Wales and 1 from urban Western Australia that both exhibited high level resistance to azithromycin (MIC value ≥ 256 mg/L).

Penicillin

Resistance to the penicillin group of antibiotics (penicillin, ampicillin and amoxycillin with or without clavulanic acid) in gonococci is a result of the production of a specific beta-lactamase: penicillinase; and/ or by the aggregation of chromosomally-controlled resistance mechanisms. These are denoted respectively, PPNG; and chromosomally mediated resistant to penicillin (CMRP). Chromosomal resistance is defined as an MIC to penicillin of 1 mg/L or more.

In 2015 in Australia, 1,217 (22.5%) isolates were penicillin resistant; a proportional decrease from 2014 (29%) and lower than 2012–2013 (32% to 35%), 2010–2011 (25% to 29%), and 2008–2009 (36% to 44%). In 2015, there were 511 (9.4%) isolates with CMRP; and 706 (13%) with PPNG. In 2014, the proportion of isolates with CMRP was 14%, and 15% were PPNG.

Penicillin resistance in the Northern Territory

In 2015, there were 258 isolates tested from the Northern Territory. There were 76 from Darwin and surrounding urban areas, and 182 from remote areas of the Northern Territory (Alice Springs, Katherine and other areas).

Of the isolates tested from the Northern Territory, 11 (14%) from the city of Darwin and surrounding urban areas were penicillin resistant: (2 CMRP and 9 PPNG) (Table 3: Northern Territory – Urban). Of these, 1 also had decreased susceptibility to ceftriaxone. In contrast, from the remote regions of the Northern Territory, 4 (2.2%) strains tested were penicillin resistant (1 CMRP and 3 PPNG). None of these strains had decreased susceptibility to ceftriaxone.

Penicillin resistance in Western Australia

In 2015 there were 482 isolates tested from Western Australia, 87 from remote regions and 395 from rural and urban regions. Of the isolates tested from rural and urban regions, 19% were reported as resistant, whereas of the 87 from remote regions there were 2 isolates (2.3%) that were penicillin resistant (both PPNG).

In addition to the isolate based surveillance for penicillin, specimens that were *N. gonorrhoeae* positive by NAAT in Western Australia were tested using a PPNG assay now routinely in use at PathWest.[8,9] In 2015, there were 60,790 specimens tested and 1,201 gonococcal detections by NAATs at PathWest confirmed by postcode to be from across Western Australia and of those, 952 (79%) were able to be tested for PPNG. Perth continues to have high rates of PPNG; detected in 58/396 extracts tested (15%). Much lower numbers of specimens were tested from other populated regions: Wheatbelt 0/6; Great Southern 1/8 (12%); and SouthWest 5/14 (36%) and therefore results should be interpreted with caution. Conversely, the remote regions continue to have lower rates of PPNG positive *N. gonorrhoeae*: 2/120 (1.6%) from the Pilbara and 0/341 (0%) from the Kimberley

Table 5: Proportion (%) of gonococcal isolates tested in Australia with MIC values at 0.06 mg/L and 0.125 mg/L 2011 to 2015

Ceftriaxone MIC mg/L	2011	2012	2013	2014	2015
0.06	3.2%	4.1%	8.2%	4.8%	1.7%
0.125	0.1%	0.3%	0.6%	0.6%	0.1%

region. Lower rates of PPNG were also reported from the Midwest and Goldfields (4.7 % and 0 % respectively), but these rates must also be interpreted with caution as lower numbers were tested in these regions (43 and 22 respectively). These data support and enhance the isolate-based surveillance findings of the AGSP, and indicate that PPNG rates remain low in the remote regions of Western Australia. All PPNG positive *N. gonorrhoeae* from remote regions were determined to be in non- Indigenous residents or residents in the major regional centres. There was no PPNG positive *N. gonorrhoeae* detected from the remote Indigenous community (personal communication from Dr David Speers, PathWest).

Quinolone antibiotics

The AGSP uses ciprofloxacin as the representative quinolone. Quinolone resistant *N. gonorrhoeae* (QRNG) are defined as MICs ≥ 1 mg/L. The resistance mechanism in *N. gonorrhoea* has thus far been mediated only by chromosomal mechanisms so that incremental changes in MIC values are observed.

In 2015, 1,473 of the 5,411 gonococci examined (27%) were resistant to ciprofloxacin (Table 3). This was lower than the proportion of isolates resistant in 2014 (36%), and overall there has been a trend of decreasing proportions since 2008, when 54% of isolates were reported as ciprofloxacin resistant.

High-level tetracycline resistance

High-level tetracycline resistant *N. gonorrhoeae* (TRNG) is used as an epidemiological marker, even though tetracyclines are not a recommended treatment for gonorrhoea and are rarely, if ever used for treatment of gonorrhoea in Australia. The proportion of TRNG detected nationally between 2006 and 2014 has ranged from 12% to 21%. In 2015, the proportion of TRNG was 16%.

TRNG were present in all jurisdictions in 2015, with the highest proportions in remote Northern Territory (47%), urban and rural Northern Territory (21%) and urban and rural Western Australia (20%).

Spectinomycin

In 2015, all isolates tested were susceptible to spectinomycin.

Discussion

The WHO recommends that treatment regimens for gonorrhoea are based on epidemiological surveillance of the distribution and extent of AMR, and that a resistance rate of 5% or more is the nominal threshold for change of treatment recommendations.[13] The AGSP has continuously monitored antimicrobial resistance in Australia since 1981, and has established quality assurance and quality control for gonococcal AMR testing with the AGSP External Quality Assurance Program, and WHO *N. gonorrhoeae* reference strains, thus ensuring the quality of the AGSP data.[17,18]

The overall number of gonococcal strains examined by the AGSP in 2015 was higher both in number and proportion when compared with 2014. The clinical isolates were referred from both the public and private health sectors, constituting a comprehensive sample of 33% of all notifications nationally. However, the increasing use of molecular diagnostic assays as an alternative to bacterial culture, in both urban and remote settings, threatens the scope of gonococcal AMR surveillance programs worldwide. This is because resultant decrease in the numbers of strains cultured thus limits the proportion with AMR testing and therefore limits AMR surveillance data. Whilst the advantages of molecular diagnostic assays over culture, in terms of sensitivity, and robustness and reliability for remote settings where cultures may not survive transportation, their primary disadvantage is that they cannot test broadly for AMR. However, molecular AMR testing strategies can give targeted and specific information, which is clinically and epidemiologically important,[2] and can contribute to surveillance programs; and be used to inform treatment guidelines.[9] This report again includes PPNG NAAT data from Western Australia, providing additional situational AMR surveillance data for the AGSP in a region where penicillin based treatment strategies are in place. Introduction of this assay is planned for the Northern Territory where penicillin-based treatment strategies are also in use, to provide enhanced surveillance data for 2016.

The primary focus for gonococcal AMR surveillance for the majority of Australia, and in most countries, is the monitoring of ceftriaxone MIC values. Gonococci with MIC values in the range 0.06–0.125 mg/L are reported to have decreased susceptibility to ceftriaxone and these strains have been found in increasing proportions in Australia, with the rate doubling over the period 2012 to 2013 from 4.4% to 8.8%.[5] In 2014, there was a decrease in the proportion of isolates with decreased susceptibility to ceftriaxone reported nationally 5.4% and this decreased further in 2015 to 1.8%.

However, little reassurance should be taken from this, as fluctuation of clones within a population is to be expected. In recent years increasing proportions of strains with decreased susceptibility to the

cephalosporin antibiotics has been accompanied by an increasing number of reports of treatment failures; and multidrug-resistant strains with high level resistance to ceftriaxone have been reported from Japan, France, Spain and now Australia.[7,15,16,19] All of these strains with high level resistance to ceftriaxone have been shown to have a mosaic penicillin binding protein 2 (PBP2), encoded by a mosaic *PenA* gene, with as few as one additional amino acid substitution required to confer high level resistance.[20] However, molecular studies have shown that strains harbouring the mosaic PBP2 are present in a significant proportion of circulating *N. gonorrhoeae* strains globally and paradoxically these strains, with a mosaic PBP2, may not have an elevated ceftriaxone MIC value, but are potentially only one point mutation from high level ceftriaxone resistance, and are under constant selection pressure. Given these considerations, the level of concern about the development of ceftriaxone resistance has heightened globally.[20]

In 2012, the WHO Global Action Plan nominated the criteria for decreased susceptibility to ceftriaxone as an MIC value ≥ 0.125 mg/L.[14] The proportion of strains tested by the AGSP with a ceftriaxone MIC value of 0.125 mg/L also doubled from 0.3% in 2012 to 0.6% in 2013 and 2014, then decreased in 2015 to 0.1%, the same as reported for 2011.

A dual therapy strategy of ceftriaxone with oral azithromycin for uncomplicated gonococcal infection continues to be recommended in Australia.[4] In 2013, high level resistance to azithromycin in gonococci was reported for the first time in Australia in 4 strains; 2 from Victoria and 2 from Queensland, and of these, 2 were most likely acquired from China.[6] In 2014, there were 2 further strains reported with high level azithromycin resistance in New South Wales. In 2015, there were again 2 sporadic incidents, 1 from New South Wales and 1 from urban Western Australia where isolates exhibited high level resistance to azithromycin (MIC value ≥ 256 mg/L). This continues to be closely observed as evidence of co-evolving cephalosporin and azithromycin resistance is being observed outside Australia and is of significant concern.[20]

The proportion of gonococci with high-level tetracycline resistance in Australia increased from 2006 to 2008 and stabilised at 21% in 2009 to 2010. The proportion of TRNG decreased to 18% in 2011, then to 14% in 2012 and remained unchanged (14%) in 2013. In 2014, there was an increase to 19%. In 2015 the proportion of TRNG was 16%. Outside the remote regions of Western Australia and the Northern Territory penicillin and ciprofloxacin resistance rates remain high. There was no resistance to spectinomycin reported in the jurisdictions testing for this antibiotic.

The recent fluctuations in proportions of *N. gonorrhoeae* with decreased susceptibility to ceftriaxone offer little reassurance in the context of what is known about gonococcal AMR, which continues to be recognised as a global public health threat. Broad-based disease control strategies, including the rational use of antibiotics, have been called for. The WHO Global Action Plan states that disease control strategies and the understanding of the global scope of AMR need to continue to be informed by surveillance programs of AMR, nationally and internationally.[14] The ongoing need for close and enhanced monitoring of gonococcal AMR can be supported to a limited extent by molecular-based assays; however isolate-based surveillance programs, and sentinel site surveillance in high risk populations are critically important to inform therapeutic strategies and to detect instances of treatment failure.

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References

1. Centers for Disease Control and Prevention. Antibiotic Resistance Threats in the United States. Atlanta, Georgia; 2013. Available from: https://www.cdc.gov/drugresistance/threat-report-2013/
2. Goire N, Lahra MM, Chen M, Donovan B, Fairley CK, Guy R, et al. Molecular approaches to enhance surveillance of gonococcal antimicrobial resistance. *Nat Rev Microbiol* 2014;12(3):223–229.
3. Roberts-Witteveen A, Pennington K, Kaldor J, Waddell R, Lahra MM, et al. Epidemiology of gonorrhoea notifications in Australia 2007–12. *Sex Health* 2014;11(4):324–331.
4. Australasian Sexual Health Alliance. Australian STI Management Guidelines. 2014. [online]. Available from: http://www.sti.guidelines.org.au/
5. Lahra MM, Australian Gonococcal Surveillance Programme. Australian Gonococcal Surveillance Programme annual report, 2013. *Commun Dis Intell* 2015;39(1):E137–E145.
6. Stevens K, Zaia A, Tawil S, Bates J, Hicks V, Whiley D, et al. *Neisseria gonorrhoeae* isolates with high-level resistance to azithromycin in Australia. *J Antimicrob Chemother* 2015;70(4):1267–1268.
7. Lahra MM, Ryder N, Whiley DM. A new multidrug-resistant strain of *Neisseria gonorrhoeae* in Australia. *N Engl J Med* 2014;371(19):1850–1851.
8. Goire N, Freeman K, Tapsall JW, Lambert SB, Nissen MD, Sloots TP, et al. Enhancing gonococcal antimicrobial resistance surveillance: a real-time pcr assay for detection of penicillinase-producing *Neisseria gonorrhoeae* by use of noncultured clinical samples. *J Clin Microbiol* 2011;49(2):513–518.
9. Speers DJ, Fisk RE, Goire N, Mak DB. Non-culture *Neisseria gonorrhoeae* molecular penicillinase production surveillance demonstrates the long-term success of empirical dual therapy and informs gonorrhoea management guidelines in a highly endemic setting. *J Antimicrob Chemother* 2014;69(5):1243–1247.
10. WHO, UNICEF, UNAIDS. Progress Report Global HIV/AIDS response: Epidemic update and health sector progress towards universal access 2011. Geneva: World Health Organization; 2011. Available from: http://www.who.int/hiv/pub/progress_report2011/en/
11. Lahra MM, Lo YR, Whiley DM. Gonococcal antimicrobial resistance in the Western Pacific Region. *Sex Transm Infect* 2013;89 Suppl 4:19–23.
12. Tapsall JW, Limnios EA, Murphy DM. An analysis of trends in antimicrobial resistance in *Neisseria gonorrhoeae* isolated in Australia, 1997–2006. *J Antimicrob Chemother* 2008;61(1):150–155.
13. Tapsall JW. Antibiotic Resistance in *Neisseria gonorrhoeae*. Geneva, Switzerland: World Health Organization; 2001.
14. World Health Organization. Global action plan to control the spread and impact of antimicrobial resistance in *Neisseria gonorrhoeae*. WHO Department of Reproductive Health and Research; 2012.
15. Ohnishi M, Golparian D, Shimuta K, Saika T, Hoshina S, Iwasaku K, et al. Is *Neisseria gonorrhoeae* Initiating a future era of untreatable gonorrhea?: Detailed characterization of the first strain with high-level resistance to ceftriaxone. *Antimicrob Agents Chemother* 2011;55(7):3538–3545.
16. Unemo M, Golparian D, Nicholas R, Ohnishi M, Gallay A, Sednaoui P. High-level cefixime- and ceftriaxone-resistant *Neisseria gonorrhoeae* in France: Novel *penA* mosaic allele in a successful international clone causes treatment failure. *Antimicrob Agents Chemother* 2012;56(3):1273–1280.
17. Australian Gonococcal Surveillance Programme. Use of a quality assurance scheme in a long-term multicentric study of antibiotic susceptibility of *Neisseria gonorrhoeae*. *Genitourin Med* 1990;66(1):437–444.
18. Unemo M, Fasth O, Fredlund H, Limnios A, Tapsall J. Phenotypic and genetic characterization of the 2008 WHO *Neisseria gonorrhoeae* reference strain panel intended for global quality assurance and quality control of gonococcal antimicrobial resistance (AMR) surveillance for public health purposes. *J Antimicrob Chemother* 2009;63 (6):1142–1151
19. Cámara J, Serra J, Ayats J, Bastida T, Carnicer-Pont D, Andreu A, et al. Molecular characterization of two high-level ceftriaxone-resistant *Neisseria gonorrhoeae* isolates detected in Catalonia, Spain. *J Antimicrob Chemother* 2012;67(8):1858–1860.
20. Whiley DM, Lahra MM, Unemo M. Prospects of untreatable gonorrhea and ways forward. *Future Microbiol* 2015;10(3):313–316.

Immunisation coverage annual report, 2014

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Abstract

This 8th annual immunisation coverage report shows data for 2014 derived from the Australian Childhood Immunisation Register and the National Human Papillomavirus Vaccination Program Register. This report includes coverage data for 'fully immunised' and by individual vaccines at standard age milestones and timeliness of receipt at earlier ages according to Indigenous status. Overall, 'fully immunised' coverage has been mostly stable at the 12– and 24-month age milestones since late 2003, but at 60 months of age, it has increased by more than 10 percentage points since 2009. As in previous years, coverage for 'fully immunised' at 12 months of age among Indigenous children was 3.7% lower than for non-Indigenous children overall, varying from 6.9 percentage points in Western Australia to 0.3 of a percentage point in the Australian Capital Territory. In 2014, 73.4% of Australian females aged 15 years had 3 documented doses of human papillomavirus vaccine (jurisdictional range 67.7 % to 77.4%), and 82.7% had at least 1 dose, compared with 71.4% and 81.5%, respectively, in 2013. The disparity in on-time vaccination between Indigenous and non-Indigenous children in 2014 diminished progressively from 20.2% for vaccines due by 12 months to 11.5% for those due by 24 months and 3.0% at 60 months of age. *Commun Dis Intell* 2017;41(1):E68–E90.

Keywords: immunisation coverage, delay, Indigenous, vaccine objection, human papillomavirus

Introduction

This is the 8th annual immunisation coverage report, with the first report having focused on 2007 data.[1–6] This report complements other reports providing data on immunisation coverage in Australia[7] and highlights important trends and significant issues. It follows the format of previous reports, providing a detailed summary for 2014 that includes vaccination coverage at standard milestone ages, coverage for vaccines not included in standard coverage assessments, timeliness of vaccination, coverage for Indigenous children, analysis of 'partially immunised' children, and data for small geographic areas on the prevalence of vaccine objection. This report also includes data on adolescents outside the Australian Childhood Immunisation Register (ACIR) age group from previously published sources. Readers are referred to the first report for a more detailed explanation of the background to this series of annual reports and the range of analyses presented.[1] This report uses the long-standing international practice of reporting at key milestone ages to measure coverage against national targets and to track trends over time. Table 1 shows the Australian National Immunisation Program (NIP) Schedule for 2014.

High levels of reporting to the ACIR are maintained by a system of incentive payments for immunisation providers and carers. These have been discussed in detail elsewhere.[6,8] Some recent changes to immunisation policy, the incentive payment system and changes to the 'fully immunised' coverage algorithms are highlighted in the Box and also referred to in this report.

Methods

The Australian Childhood Immunisation Register

The ACIR was established on 1 January 1996 by incorporating demographic data from Medicare on all enrolled children under the age of 7 years.[9] Participation in the ACIR is 'opt-out' so it constitutes a nearly complete population register, as approximately 99% of children are registered with Medicare by 12 months of age.[9] Children not enrolled in Medicare can also be added to the ACIR via a supplementary number. Since 2001, vaccinations given overseas may be recorded if a provider endorses their validity. Data are transferred to the ACIR when a recognised immunisation provider supplies details of an eligible vaccination. This could occur automatically from medical practice software or through the Internet using the Medicare Australia web site or by submitting paper encounter forms. The existence of medical contraindications and conscientious objection to immunisation is also recorded on the ACIR. From 2016, conscientious objection is no longer a valid exemption to immunisation linked to family payments and therefore will no longer be recorded on the ACIR.[10] All vaccination records for a child remain on the register indefinitely, but no new vaccination records are added after the 7th birthday. However, from 2016 this will change to allow the recording of vaccinations given up to 19 years of age.[10]

Vaccinations recorded on the ACIR must be rendered in accordance with the guidelines issued

Table 1: Australian National Immunisation Program Schedule for children, adolescents and adults, 2014

Age	Vaccine									
Childhood vaccines										
Birth	Hep B									
2 months	Hep B	DTPa	Hib	Polio				13vPCV	Rotavirus	
4 months	Hep B	DTPa	Hib	Polio				13vPCV	Rotavirus	
6 months	Hep B	DTPa	Hib	Polio				13vPCV	Rotavirus*	
12 months			Hib-Men C†		MMR		Hep A‡	13vPCV§		
18 months						MMRV¶	Hep A‡	13vPCV§		
24 months							Hep A‡			
48 months		DTPa		Polio	MMR**			23vPPV††		
Adolescent vaccines										
12–15 years		dTpa				VZV				HPV‡‡
15–49 years									Flu§§	23vPPV¶¶
Adult vaccines										
≥50 years									Flu§§	23vPPV¶¶
65 years									Flu§§	23vPPV
Pregnant women (any age)									Flu***	

* 3rd dose of rotavirus vaccine at 6 months of age is dependent on vaccine brand used in each state or territory.

† In July 2013, the combined *Haemophilus influenzae* type b (Hib) and meningococcal serogroup C (Men C) vaccine, Menitorix®, was added to the NIP Schedule at 12 months of age. This combination vaccine replaces the single dose of monovalent meningococcal C conjugate vaccine (Men C) and booster dose of monovalent Hib vaccine previously scheduled at 12 months of age.

‡ Aboriginal and Torres Strait Islander children – doses at 12 months and 18 months of age in the Northern Territory and Western Australia and 18 and 24 months of age in Queensland and South Australia (schedule changed in July 2013 so doses administered at 12 months and 18 months of age in all four jurisdictions).

§ Booster dose for medically at risk children at 12 months of age and Aboriginal and Torres Strait Islander children in the Northern Territory, Western Australia, Queensland and South Australia at 12–18 months of age.

¶ Measles-mumps-rubella-varicella vaccine introduced onto NIP Schedule on 1 July 2013.

** To be given only if MMRV vaccine was not given at 18 months of age. The dose of measles-mumps-rubella vaccine at 4 years of age ceased on 1 January 2016.

†† Medically at-risk children

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

§§ Annual vaccination, all aged ≥6 months with medical risk factors, Aboriginal and Torres Strait Islander people aged ≥15 years, non-Indigenous adults aged ≥65 years.

¶¶ Aboriginal and Torres Strait Islander people: aged ≥15 years with medical risk factors; all aged ≥50 years.

*** At any stage of pregnancy.

by the Australian Technical Advisory Group on Immunisation (ATAGI).[11] Notifications falling outside these guidelines, or duplicate notifications, prompt an enquiry with the provider and, if their validity cannot be established, they are rejected.

Measuring immunisation coverage using the Australian Childhood Immunisation Register

The cohort method has been used for calculating coverage at the population level (national and state and territory)[12] since the ACIR's inception. Cohort immunisation status is assessed at 12 months of age (for vaccines due at 6 months), 24 months of age (for vaccines due at 12 and 18 months), and 60 months of age (for vaccines due at 48 months). A minimum 3-month lag period is allowed for late notification of vaccinations to the ACIR, but only vaccinations given on or before a child's 1st, 2nd or 5th respective birthdays are considered.[12] If a child's records indicate receipt of the last dose of a vaccine that requires more than 1 dose to complete the series, it is assumed that earlier vaccinations in the sequence have been given. This assumption has been shown to be valid.[13,14]

Three-month birth cohorts are used for time trend analyses, while both 3-month wide and 12-month

Box: Significant changes in immunisation policy, immunisation incentives and coverage calculation algorithms, Australia, 2011 to 2014

July 2014 – Immunisation coverage assessment algorithm for the 24-month milestone amended to require a dose of meningococcal vaccine, a dose of varicella vaccine and a 2nd dose of measles-mumps-rubella (MMR) vaccine to be classified as fully immunised.

July 2013 – Immunisation coverage assessment algorithm for the 12-month milestone amended to include a 3rd dose of pneumococcal conjugate vaccine (PCV) in the assessment of fully immunised.

Combined *Haemophilus influenzae* type b (Hib) and meningococcal serogroup C (Men C) conjugate vaccine, Menitorix®, added to the National Immunisation Program (NIP) Schedule at 12 months of age, replacing the single dose of monovalent Men C vaccine and booster dose of monovalent Hib vaccine previously scheduled at 12 months of age.

Combination measles-mumps-rubella-varicella (MMRV) vaccine added to the NIP at 18 months of age, replacing MMR dose previously scheduled at 4 years of age and varicella vaccine dose previously scheduled at 18 months of age. MMR vaccination at 4 years of age continued in parallel until first cohort eligible for MMRV vaccine reached 4 years of age.

Hepatitis A vaccination schedule for Indigenous children changed so that dose 1 administered at 12 months of age and dose 2 at 18 months of age in all four relevant jurisdictions (the Northern Territory, Western Australia, Queensland and South Australia).

February 2013 – Human papillomavirus vaccine funded under the NIP for males aged 12–13 years, delivered in school-based programs.

July 2012 – Eligibility for Family Tax Benefit Part A supplement required that children are assessed as fully immunised during the financial years that they turn 1, 2 and 5 years or have an approved exemption, replacing the Maternity Immunisation Allowance.

October 2011 – 13-valent PCV (13vPCV) replaced 23-valent pneumococcal polysaccharide vaccine as booster dose in Indigenous children living in the Northern Territory, Western Australia, Queensland and South Australia.

July 2011 – 13vPCV replaced 7-valent PCV on the NIP for children at 2, 4 and 6 months of age in all states and territories except the Northern Territory (adopts 13vPCV from 1 October 2011).

wide cohorts are used for all other analyses in this report. The 12-month wide cohorts used in this report are children born between 1 January and 31 December 2013 for the 12-month milestone age; children born between 1 January and 31 December 2012 for the 24-month milestone age; and children born between 1 January and 31 December 2009 for the 5-year (60-month) milestone age.

The proportion of children designated as 'fully immunised' is calculated using the number of children completely immunised with the vaccines of interest by the designated age as the numerator, and the total number of Medicare-registered children in the age cohort as the denominator. 'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, 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-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.

Immunisation coverage estimates were also calculated for individual NIP vaccines, including the 3 NIP vaccines given in early childhood but not routinely reported in the quarterly coverage reports published in *Communicable Diseases Intelligence*[15] and not part of 'fully immunised' calculations at 12, 24 and 60 months of age. These are: a 2nd or 3rd dose of rotavirus vaccine by 12 months of age; a 2nd dose of hepatitis A vaccine in Indigenous children by 30 months of age; and a booster dose of pneumococcal vaccine in Indigenous children by 30 months of age.

Changes to immunisation policy and changes to the 'fully immunised' coverage algorithms have had an impact on vaccination coverage presented in this report. From July 2012, eligibility for the Family Tax Benefit Part A supplement required that children needed to be assessed as fully immunised, replacing the Maternity Immunisation Allowance. To meet the immunisation requirements for the Family Tax Benefit Part A supplement, parents needed to have their children immunised during the financial years that each child turned 1, 2 and 5 years of age. Children needed to be up-to-date with immunisation or have an approved exemption.

From the December 2013 quarterly coverage report, the 3rd dose of pneumococcal conjugate vaccine was included in coverage requirements for 'fully immunised' at the 12-month milestone. From the December 2014 quarterly coverage report, a dose of meningococcal vaccine and a dose of varicella vaccine were included in the coverage requirements for 'fully immunised' at the 24-month milestone, along with the 2nd dose of MMR instead of the 1st dose as previously. The 2nd dose of MMR remained in the coverage assessment algorithm for the 60-month milestone age.

Timeliness

Age-appropriate timely vaccination was defined as receipt of a scheduled vaccine dose within 30 days of the recommended age. For example, a child who received the 1st dose of DTPa (due at 60 days of age) when he or she was more than 90 days of age was classified as late for that dose. For descriptive purposes, we categorised the delay outcome measure for each dose as either delay of 1 to < 6 months or delay ≥ 6 months. Timeliness is measured in 12-month birth cohorts. Children included in the timeliness analysis were assessed at 1 to 3 years after doses were due, to allow time for late vaccinations to be recorded. Therefore, cohorts assessed for timeliness are not the same as those assessed for coverage milestones. The interval between doses was not evaluated. Timeliness of different vaccines and doses was also compared by plotting the cumulative percentage receiving each vaccine dose by age.

Remoteness status

The area of residence of children was defined as Major cities, Inner regional, Outer regional, Remote, and Very remote using the Accessibility/Remoteness Index of Australia (ARIA+).[16] ARIA+ is a continuous varying index with values ranging from 0 (high accessibility) to 15 (high remoteness), and is based on road distance measurements from over 12,000 populated localities to the nearest Service Centres in 5 categories based on population size. For our analysis, we combined the 2 regional categories (Inner regional and Outer regional) into 1 category and the 2 remote categories (Remote and Very remote) into 1 category. ARIA Accessibility/Remoteness categories were assigned for each child using their recorded postcode of residence on the ACIR.

Indigenous status

Indigenous status on the ACIR is recorded as Indigenous, non-Indigenous or unknown, as reported by the child's carer to Medicare or by the immunisation provider to the ACIR. For this report we considered two categories of children: Indigenous and non-Indigenous; children with unknown Indigenous status were presumed to be non-Indigenous. Coverage estimate time trends are presented from 2002 only, due to poor rates of reporting Indigenous status prior to then.[17]

Small area analysis

Analysis for small areas was done by Australian Bureau of Statistics (ABS)-defined Statistical Area 3 (SA3),[18] chosen because each is small enough to show differences within jurisdictions but not too small to render maps unreadable. Maps were created using version 15 of the MapInfo mapping software[19] and the ABS Census Boundary Information. As postcode is the only geographical indicator available from the ACIR, the ABS Postal Area to SA3 Concordance 2011 was used to match ACIR postcodes to SA3s.[20]

Objection to vaccination and incomplete immunisation

Until 2016, parents who registered vaccination objection were eligible for parental incentive payments even if their children were unvaccinated. However some parents who objected to vaccination did not register an objection. We calculated the proportions of children with registered vaccination objection status and no vaccines recorded on the

ACIR, registered vaccination objection status and at least 1 vaccine recorded on the ACIR, no registered vaccination objection status and no vaccines recorded on the ACIR, and no registered vaccination objection status and not 'fully immunised' by 24 months of age, from the cohort of children registered with Medicare and born between 1 October and 31 December 2012. Some of the children in the latter 2 groups may be incompletely immunised due to unregistered vaccination objection. We chose this cohort for calculation of proportions of these groups in 2014 so that children under the age of 12 months were excluded, to allow sufficient time for registration of objection.

Human papillomavirus vaccine coverage

Human papillomavirus (HPV) vaccine is included on the NIP, with the vaccine delivered to females and, since 2013, males through an ongoing school-based program usually in the 1st year of secondary school. From 2007 to 2009, there was a time-limited catch-up program delivered through schools, general practices and community immunisation services for females up to age 26. Males were offered a time-limited catch-up program in 2013–2014, at the age of 14–15 years. A full course of HPV immunisation was defined as 3 doses of quadrivalent HPV vaccine. Data on HPV vaccination are provided by the National HPV Vaccination Program Register, which is operated by the Victorian Cytology Service. Data for males represent the vaccination coverage achieved during the catch-up program for males aged 14–15 years during 2013–2014. The purpose of this legislated register is to support the implementation of the vaccination program and to provide data for monitoring and evaluation. States and territories provide data to the HPV Register from their school-based programs. Doses administered in general practice or by community providers outside of the school program are notified on a voluntary basis, with a notification payment provided only to general practitioners (GPs) during the 2007 to 2009 catch-up program. The World Health Organization recommends using 15 years as the reference age for HPV vaccination coverage for the purposes of international comparison.

Coverage in the elderly

While an Adult Vaccination Survey (AVS)[21] has not been undertaken in Australia since 2009, data from a Newspoll Omnibus Survey in 2014 are presented.[22] From September 2016 the ACIR will expand to become the Australian Immunisation Register. This Register will capture all vaccines administered throughout a person's life (birth to death) from that point forward, given through general practice and community clinics.

Indigenous adolescent and adult coverage

Indigenous adolescent and adult coverage estimates are obtained from the 2012–2013 Aboriginal and Torres Strait Islander Health Survey.

Results

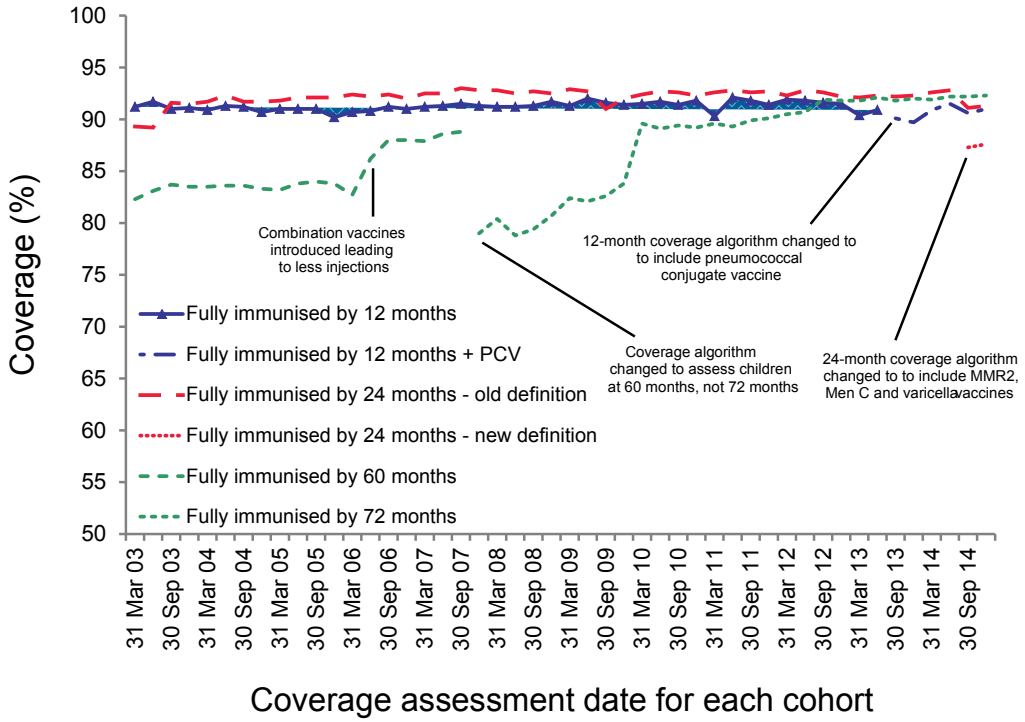
Coverage estimates

Fully immunised

Coverage estimates in 2014 for full-year birth cohorts at the 3 milestone ages of 12 months, 24 months and 60 months are provided in Tables 2, 3 and 4. The proportion of Australian children classified as 'fully immunised' was 91.5% at 12 months, 86.8% at 24 months and 92.0% at 60 months of age, compared with 2013 coverage at these milestones of 90.8%, 92.1% and 91.2%, respectively. Nationally and for almost all jurisdictions, 'fully immunised' coverage (except at the 24-month age milestone) and coverage for all individual vaccines (except rotavirus vaccine, varicella vaccine and dose 2 of MMR vaccine) at all 3 age milestones was above 90%, the target at that time.

Figure 1 shows time trends in 'fully immunised' childhood vaccination coverage in Australia, assessed at 12 months, 24 months and 60 months of age, for 3-month cohorts born from 1 January 1997 to 31 December 2013. Coverage has been largely stable at the 12– and 24-month age milestones since late 2003. However, during 2013, 'fully immunised' coverage at the 12-month age milestone for vaccines due at 6 months of age declined by 1.8 percentage points, partly due to the inclusion of 13-valent pneumococcal conjugate vaccine (PCV) in the coverage assessment algorithm, and then increased by 1.2% in 2014. In the latter half of 2014, 'fully immunised' coverage at the 24-month age milestone declined by 5.5 percentage points. The bulk of this decrease was due to the coverage assessment algorithm being amended in July 2014 to include a dose of meningococcal vaccine, a dose of varicella vaccine and a 2nd dose of MMR vaccine. This is demonstrated in Table 2, which shows 'fully immunised' coverage at the 24-month age milestone to have only dropped by 0.4 of a percentage point when calculated using the old algorithm. For vaccines due at 48 months of age, 'fully immunised' coverage dropped to 80.4% in January 2008, following the change in assessment age from 72 months to 60 months, but then rose substantially in 2009 and 2010 and kept increasing throughout 2011 to 2014 to a level higher than that for the 12-month and 24-month age milestones in 2014.

Figure 1: Trends in 'fully immunised' vaccination coverage estimates, Australia, 2003 to 2014*



* By 3-month birth cohorts born between 1 January 1999 and 31 December 2013. Coverage assessment date was 12 months after the last birth date of each cohort.

PCV = pneumococcal conjugate vaccine

Source: Australian Childhood Immunisation Register.

Table 2: Percentage of children as immunised by 12 months of age, Australia, assessed in 2014, by vaccine and state or territory*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,560	98,101	3,641	61,747	19,593	5,893	75,544	33,710	303,789
Diphtheria, tetanus, pertussis	95.1	92.4	91.3	92.8	92.2	92.0	92.7	92.7	92.6
Polio	95.1	92.3	91.3	92.8	92.1	92.0	92.6	92.7	92.5
Haemophilus influenzae type b	94.8	92.0	91.1	92.5	91.9	91.7	92.3	92.3	92.3
Hepatitis B	94.6	92.0	91.3	92.4	91.8	91.7	92.2	92.1	92.2
Pneumococcal conjugate	94.6	91.8	91.1	92.3	91.6	91.7	92.1	91.8	92.0
Fully immunised†	93.9	91.3	90.6	92.0	91.2	91.2	91.6	91.3	91.5
Rotavirus	90.4	87.4	86.4	82.2	83.5	86.1	83.3	80.0	84.3

* Cohort born 1 January 2013 – 31 December 2013.

† 'Fully immunised' – 3 doses of a diphtheria-tetanus-pertussis-containing vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP-containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, 3 doses of any hepatitis B vaccine or 2 doses of either Engerix-B (paediatric), Comvax or H-B-VAX II (paediatric), and 3 doses of pneumococcal conjugate vaccine.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Table 3: Percentage of children immunised by 24 months of age, Australia, assessed in 2014, by vaccine and state or territory*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,549	101,937	3,638	63,676	20,223	5,902	77,235	34,355	312,515
Diphtheria, tetanus, pertussis	96.3	95.1	95.4	95.1	94.7	95.2	95.5	94.6	95.2
Polio	96.3	95.1	95.4	95.1	94.7	95.1	95.5	94.5	95.1
Haemophilus influenzae type b	94.8	93.7	94.9	94.1	93.2	93.8	94.0	93.1	93.8
Hepatitis B	95.6	94.6	95.4	94.6	94.2	94.8	94.9	93.8	94.6
Measles, mumps, rubella	91.9	88.7	89.2	90.3	88.5	88.0	89.1	86.2	88.9
Varicella	92.3	89.6	90.1	90.4	88.9	88.6	89.8	87.7	89.6
Meningococcal C	94.6	93.5	94.7	94.1	93.2	94.0	93.7	92.7	93.6
Fully immunised – old definition†	93.3	91.5	93.4	92.7	91.5	91.8	92.1	91.1	91.9
Fully immunised‡	90.1	86.4	86.4	88.7	86.3	85.3	87.0	84.3	86.8

* Cohort born 1 January 2012 to 31 December 2012.

† 'Fully immunised – old definition' – 3 doses of a diphtheria-tetanus-pertussis-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP-containing *Haemophilus influenzae* type b (Hib) vaccine or 4 doses of any other Hib vaccine, and 3 doses of hepatitis B vaccine.

‡ 'Fully immunised' – 3 doses of a diphtheria-tetanus-pertussis-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP-containing Hib vaccine or 4 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, 2 doses of a measles-mumps-rubella-containing vaccine, 1 dose of varicella vaccine, and 1 dose of meningococcal C vaccine.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Table 4: Percentage of children immunised by 60 months of age, Australia, assessed in 2014, by vaccine and state or territory*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,347	100,617	3,532	64,869	20,299	6,399	75,454	33,930	310,447
Diphtheria, tetanus, pertussis	93.9	92.9	92.6	92.6	91.4	92.9	92.9	91.1	92.6
Polio	93.9	92.9	92.6	92.6	91.3	92.9	92.9	91.0	92.6
Measles, mumps, rubella	93.7	92.9	93.0	92.6	91.3	92.8	92.9	91.0	92.5
Fully immunised†	93.3	92.4	92.0	92.2	90.7	92.1	92.4	90.4	92.0

* Cohort born 1 January 2009 to 31 December 2009.

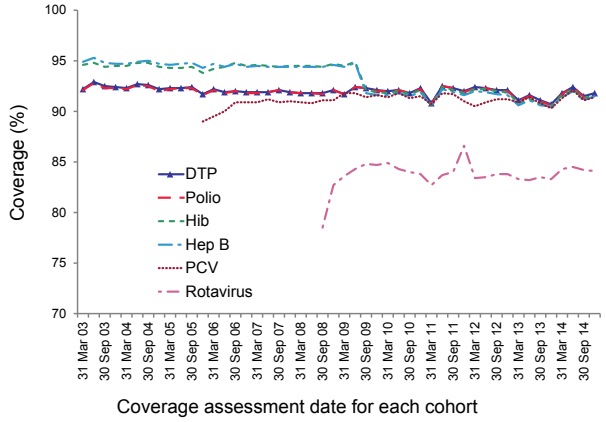
† 'Fully immunised' – 4 or 5 doses of a diphtheria-tetanus-pertussis-containing vaccine, 4 doses of polio vaccine, and 2 doses of an measles-mumps-rubella-containing vaccine.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Individual vaccines

Coverage at 12 months of age for individual antigens in the relevant combination vaccine (DTPa-hepB-polio-Hib) increased in 2014 (Figure 2). Coverage for 3 doses of PCV by 12 months of age rose steadily from below 90% in mid-2007 to 91.4% in late 2014, just below the level for all other vaccines assessed at this age except for rotavirus vaccine. After being added to the NIP in July 2007, rotavirus vaccine coverage for 2 or 3 doses (depending on whether the vaccine in use is Rotarix® or RotaTeq®) at 12 months of age rose steeply from about 75% in late 2008 to almost 84% in late 2011 and has remained largely stable since, with minor fluctuations. Rotavirus vaccine coverage was lower nationally (Figure 2) and had greater variation between jurisdictions than other vaccines given at 2, 4 and 6 months of age. Reported coverage in 2014 at 12 months of age varied from 90.4% in the Australian Capital Territory for 2 doses of Rotarix® vaccine, to 80.0% in Western Australia for 3 doses of RotaTeq® vaccine (Table 2).

Figure 2: Trends in vaccination coverage estimates for individual vaccines* at 12 months of age, Australia, 2003 to 2014



* 3rd dose of DTPa, polio and pneumococcal conjugate vaccines, 2nd or 3rd dose of Hib and rotavirus vaccines, and 3rd dose of hepatitis B vaccine.

By 3-month birth cohorts born between 1 January 1999 and 31 December 2013. Coverage assessment date was 12 months after the last birth date of each cohort.

DTPa = diphtheria-tetanus-acellular pertussis

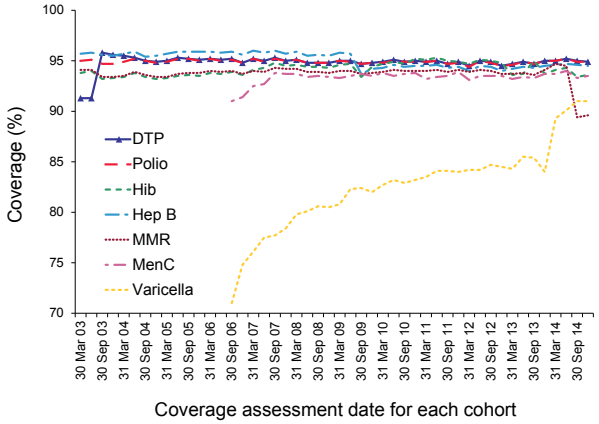
Hib = *Haemophilus influenzae* type b

Hep B = Hepatitis B

PCV = Pneumococcal conjugate vaccine

Source: Australian Childhood Immunisation Register.

Figure 3: Trends in vaccination coverage estimates for individual vaccines* at 24 months of age, Australia, 2003 to 2014



* 3rd dose of DTPa, 3rd dose of polio, 3rd or 4th dose of Hib, 3rd dose of hepatitis B, 2nd dose of MMR (from September 2014), 1st dose of meningococcal C and varicella.

By 3-month birth cohorts born between 1 January 1998 and 31 December 2012. Coverage assessment date was 24 months after the last birth date of each cohort.

DTPa = Diphtheria-tetanus-acellular pertussis

Hib = *Haemophilus influenzae* type b

Hep B = Hepatitis B

MMR = Measles-mumps-rubella

MenC = Meningococcal C

MMRV = Measles-mumps-rubella-varicella

Source: Australian Childhood Immunisation Register.

In 2014, coverage at 24 months of age was around 93% to 95% for all vaccines (except varicella and the 2nd dose of MMR) (Figure 3). In the latter half of 2014, coverage for MMR declined by 5.1 percentage points. This was due to the 2nd dose of MMR, now due at 18 months of age, being assessed at 24 months of age for the first time, instead of the 1st dose as previously. Varicella coverage at 24 months of age increased sharply in mid-2013, following replacement of monovalent varicella vaccine with measles-mumps-rubella-varicella (MMRV) vaccine as the vaccine due at 18 months of age (a comparison between varicella coverage before and after introduction of MMRV vaccine is discussed in a separate section). As the 18-month schedule point has historically been associated with lower coverage when assessed at the 24-month age milestone, given that there is only a 6-month time period for catch-up, we compared varicella coverage assessed at 36 months and 60 months of age to that assessed at 24 months, by jurisdiction (Figure 4). Coverage by jurisdiction was 6.0 to 10.4 percentage points higher at 60 months, with all jurisdictions reaching over 90% varicella coverage when assessed at 60 months.

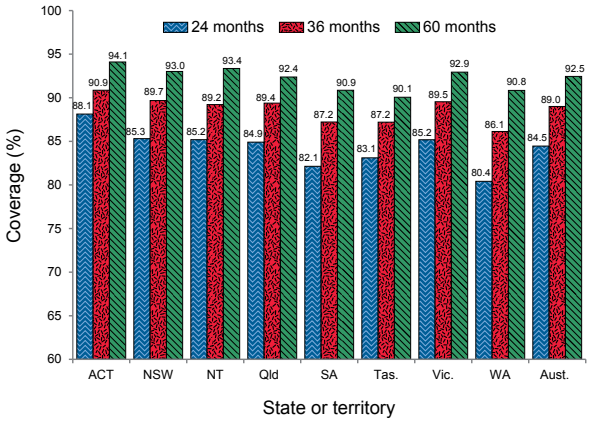
For vaccines due at 48 months of age, trends in individual vaccine coverage were similar to that seen for 'fully immunised' coverage, that is, a marked drop in January 2008 following the change in assessment age from 72 months to 60 months, followed by a marked increase in 2009 and 2010 and ongoing increase to a level higher than when coverage was assessed at 72 months of age (Figure 5). Coverage for both vaccines due at 48 months (DTPa and MMR) was greater than 92% in 2014.

A comparison of varicella coverage before and after introduction of measles-mumps-rubella-varicella vaccine

In July 2013, MMRV was introduced at the 18 months of age schedule point, replacing the single dose of varicella vaccine previously scheduled at this age point and the 2nd dose of MMR previously scheduled at 48 months of age.

Table 5 provides varicella coverage for two 3-month wide birth cohorts 2 years apart, allowing comparison of coverage before and after introduction of MMRV vaccine. For Australia as a whole, varicella coverage increased by 3.7 percentage points from pre– to post-introduction of MMRV. Increases occurred in

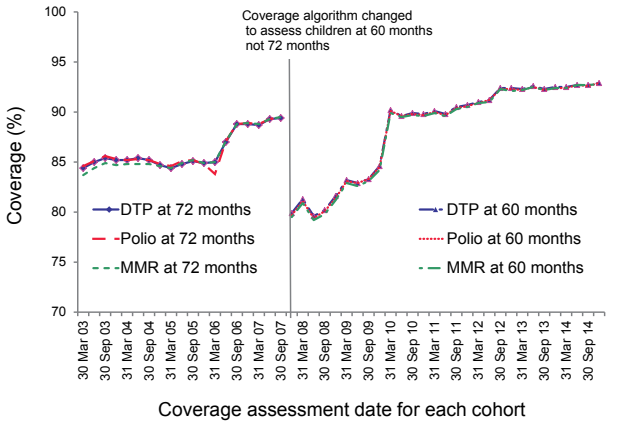
Figure 4: Comparison of 1-dose varicella vaccine coverage at 24 months of age versus 36 months of age and 60 months of age, Australia, assessed in December 2014, by state or territory



Cohort born 1 October to 31 December 2009.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Figure 5: Trends in vaccination coverage estimates for individual vaccines* at 60 months of age (assessed at 72 months prior to December 2007), Australia, 2003 to 2014



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

By 3-month birth cohorts born between 1 January 1996 and 31 December 2009. Coverage assessment date was 72 months after the last birth date of each cohort up to December 2007 and then 60 months after the last birth date of each cohort.

DTPa = Diphtheria-tetanus-acellular pertussis

MMR = Measles-mumps-rubella

Source: Australian Childhood Immunisation Register.

all jurisdictions except the Northern Territory and Tasmania, ranging up to a 5.2 percentage point increase in South Australia and Victoria.

Indigenous coverage estimates

Individual vaccines

Immunisation coverage estimates in 2014 for the 3 age milestones by Indigenous status, vaccine and jurisdiction are provided in Tables 6 and 7. As in previous years, 'fully immunised' coverage at 12 months of age among Indigenous children was lower than for non-Indigenous children in all jurisdictions, with the differential varying from 6.9 percentage points in Western Australia to 0.3 percentage points in the Australian Capital Territory, and 3.7% overall. 'Fully immunised' coverage at 24 months of age among Indigenous children in 2014 was 3.3 percentage points lower, with the differential varying from 6.4 percentage points in Victoria to 0.5 of a percentage point in New South Wales. The lower coverage gap for New South Wales may reflect the NSW Aboriginal Immunisation Health Care Worker Program that began as a 3-year pilot program in 2012. 'Fully immunised' coverage at 60 months of age in 2014 was 1.6 percentage points higher among Indigenous children compared with non-Indigenous children, with coverage in Indigenous children at this age milestone higher in all jurisdictions except Victoria, South Australia and Tasmania.

The coverage differential between Indigenous and non-Indigenous children for individual vaccines varied in 2014, with coverage lower for Indigenous

Table 5: Comparison of varicella coverage (%) before and after introduction of measles-mumps-rubella-varicella vaccine, Australia, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Before MMRV introduction*	87.2	85.2	89.2	87.9	83.7	87.1	84.9	83.9	85.6
After MMRV introduction†	91.8	89.2	84.1	89.5	88.9	87.1	90.1	88.5	89.3

* Cohort born 1 April – 30 June 2011, assessed at 24 months.

† Cohort born 1 April – 30 June 2013, assessed at 24 months.

MMRV = measles-mumps-rubella-varicella

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Table 6: Vaccination coverage estimates (%), Australia, assessed in 2014, by age, vaccine and Indigenous status

Vaccine	Milestone age	Indigenous	Non-Indigenous
Diphtheria, tetanus, acellular pertussis	12 months*	88.3	92.8
	24 months†	95.3	95.1
	60 months‡	94.0	92.5
Polio	12 months*	88.2	92.8
	24 months†	95.3	95.1
	60 months‡	94.0	92.5
Haemophilus influenzae type b	12 months*	88.2	92.5
	24 months†	94.7	93.7
	60 months‡	N/I	N/I
Hepatitis B	12 months*	88.2	92.3
	24 months†	95.2	94.6
	60 months‡	N/I	N/I
Measles-mumps-rubella	12 months*	N/I	N/I
	24 months†	86.1	89.0
	60 months‡	94.3	92.4
Varicella	12 months*	N/I	N/I
	24 months†	86.3	89.7
	60 months‡	N/I	N/I
Meningococcal C conjugate	12 months*	N/I	N/I
	24 months†	94.5	93.6
	60 months‡	N/I	N/I
Pneumococcal conjugate	12 months*	86.4	91.2
	24 months†	N/I	N/I
	60 months‡	N/I	N/I
Rotavirus	12 months*	73.9	84.8
	24 months†	N/I	N/I
	60 months‡	N/I	N/I

* Cohort born 1 January 2013 – 31 December 2013.

† Cohort born 1 January 2012 – 31 December 2012.

‡ Cohort born 1 January 2009 – 31 December 2009.

N/I Not included in coverage estimates for that group.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Table 7: Percentage of children fully immunised by 12 months, 24 months and 60 months of age, Australia, assessed in 2014, by Indigenous status and state or territory

	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
12 months – fully immunised*									
Indigenous	93.6	90.5	88.3	86.8	87.5	87.4	87.5	84.8	88.0
Non-Indigenous	93.9	91.3	92.0	92.4	91.4	91.5	91.6	91.7	91.7
24 months – fully immunised†									
Indigenous	88.2	85.9	85.4	84.2	80.8	80.8	80.7	79.2	83.7
Non-Indigenous	90.2	86.4	87.0	89.1	86.5	85.6	87.1	84.6	87.0
60 months – fully immunised‡									
Indigenous	97.4	95.2	95.4	93.4	89.3	91.4	91.1	92.7	93.6
Non-Indigenous	93.2	92.3	89.9	92.1	90.7	92.2	92.4	90.3	92.0

* 'Fully immunised' – 3 doses of a diphtheria-tetanus-pertussis-containing vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP-containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, and 3 doses of pneumococcal conjugate vaccine. Cohort born 1 January 2013 – 31 December 2013

† 'Fully immunised' – 3 doses of a diphtheria-tetanus-pertussis-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP-containing Hib vaccine or 4 doses of any other Hib vaccine, 3 doses of hepatitis B vaccine, 2 doses of a measles-mumps-rubella-containing vaccine, 1 dose of meningococcal C vaccine, and 1 dose of varicella vaccine. Cohort born 1 January 2012 – 31 December 2012

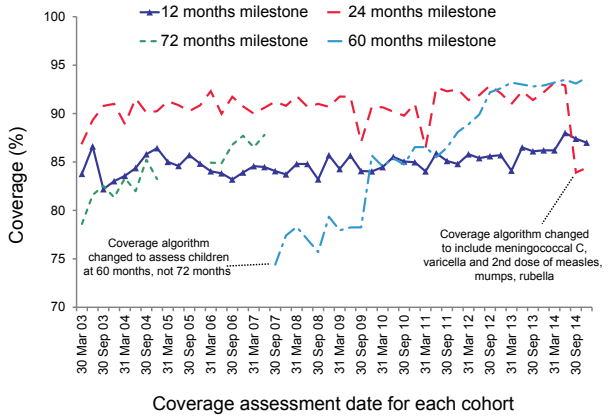
‡ 'Fully immunised' – 4 doses of a DTPa-containing vaccine, 4 doses of polio vaccine, and 2 doses of a measles-mumps-rubella-containing vaccine. Cohort born 1 January 2009 – 31 December 2009

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

children for all vaccines at 12 months of age, but higher at 24 months of age for DTPa, polio, hepatitis B, Hib and meningococcal C vaccines, and at 60 months of age for DTPa, polio and MMR.

The proportion of Indigenous children 'fully immunised' by 24 months of age was consistently higher than at 12 and 60 months of age until 2012, when coverage at 60 months rose to levels comparable to that at 24 months (Figure 6). During the latter half of 2014, the proportion of Indigenous children 'fully immunised' by 24 months of age decreased from 92.9% to 84.4%, due to the changes to the 'fully immunised' coverage algorithm, as noted above.

Figure 6: Trends in 'fully immunised' vaccination coverage estimates for Indigenous children in Australia, 2003 to 2014, by age milestones



MMR = measles, mumps, rubella

Source: Australian Childhood Immunisation Register.

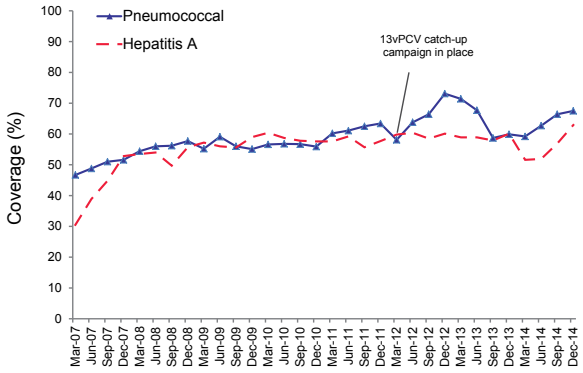
Adolescents and adults

For Indigenous adults and adolescents, according to the 2012/13 Aboriginal and Torres Strait Islander Health Survey, 13% of Indigenous Australians aged 15 years and over had a pneumococcal vaccination in the previous 5 years and 34% of Indigenous Australians aged 15 and over had an influenza vaccination in the previous 12 months.

Pneumococcal booster and hepatitis A vaccine for Indigenous children in some jurisdictions

Hepatitis A vaccine has been included on the NIP since November 2005 for Indigenous children in the Northern Territory, Queensland, South Australia and Western Australia, but was used earlier than this in north Queensland. Since March 2007, coverage of 2 doses of hepatitis A vaccine for Indigenous children by 30 months of age in Western Australia and the Northern Territory and 36 months of age in Queensland and South Australia had increased from 30.5% to 60.1% in December 2013 (Figure 7). By the latter half of 2014, coverage had increased to its highest recorded level of 62.9% with all 4 jurisdictions assessing 2 doses at 30 months of age from July 2013. An additional 17% of children had received 1 dose of hepatitis A vaccine by 18 months of age, putting national coverage in 2014 for Indigenous children (the Northern Territory, Queensland, South Australia and Western Australia only) for at least 1 dose of hepatitis A vaccine at 79.8% (Table 8). There was variation in reported hepatitis A vaccine coverage by jurisdiction, from a low of 37.5% in South Australia to a high of 86.2% in the Northern Territory for 2-dose coverage (Table 8).

Figure 7: Trends in coverage estimates for hepatitis A* and pneumococcal† vaccines for Indigenous children, Australia,‡ 2007 to 2014



* 18-month dose assessed at 30 months of age in all 4 jurisdictions from July 2013.

† 18-month dose assessed at 30 months of age.

‡ Northern Territory, Queensland, South Australia and Western Australia only.

13vPCV = 13-valent pneumococcal conjugate vaccine

Source: Australian Childhood Immunisation Register.

A pneumococcal booster dose at 18–24 months of age has been recommended and funded for Indigenous children in the same 4 jurisdictions (the Northern Territory, Queensland, South Australia and Western Australia) since 2001; firstly as 23-valent pneumococcal polysaccharide vaccine then as 13-valent pneumococcal conjugate vaccine (13vPCV), from July 2013 in Queensland, South Australia and Western Australia, and from October 2013 in the Northern Territory. Coverage gradually increased from 47.0% in March 2007 to 63.4% in December 2011 (Figure 7). In 2012

coverage increased by 15 percentage points following the 13vPCV catch-up campaign that took place that year. Coverage then fell 14 percentage points during 2013 but increased by 9 percentage points in 2014 to 67.5%. There was a large variation between jurisdictions in coverage for the booster dose of pneumococcal conjugate vaccine, from a low of 41.2% in South Australia to a high of 85.4% in the Northern Territory (Table 8).

Table 8: Vaccination coverage* (%) for Indigenous children for hepatitis A and pneumococcal, Australia, 2014, by state or territory

State or territory	Vaccine type	
	Hepatitis A† 2 dose (1 dose)	13vPCV‡
NT	86.2 (92.1)	85.4
Qld	59.2 (77.4)	68.2
SA	37.5 (65.6)	41.2
WA	65.5 (82.1)	60.1
Australia§	63.0 (79.8)	66.9

* Cohort born 1 April 2012 – 30 June 2012.

† Indigenous only: 2 doses by 30 months of age.

‡ Indigenous only: 4th dose of 13-valent pneumococcal conjugate vaccine (13vPCV) by 30 months of age.

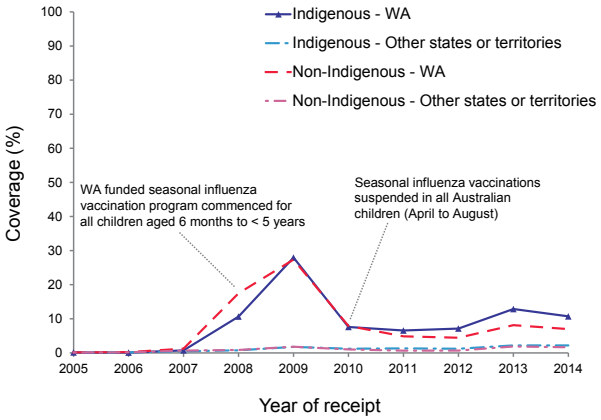
§ Northern Territory, Queensland, South Australia and Western Australia only.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Seasonal influenza vaccine coverage

Seasonal influenza vaccine has been recommended and funded for children with underlying medical conditions for many years. In 2008 a funded seasonal influenza vaccination program for all children aged from 6 months to less than 5 years was introduced in Western Australia. In 2013 seasonal influenza vaccine was recommended (but not funded) for all Australian children aged from 6 months to less than 5 years, and in 2015 it was included on the NIP for all Aboriginal and Torres Strait Islander children aged 6 months to less than 5 years. This report provides baseline coverage data up to 2014 to inform future monitoring of coverage under this program, although these data should be interpreted with caution due to the potential for under-reporting, possibly due to influenza vaccine notifications by immunisation providers not resulting in any notification payments unlike other vaccines on the NIP. Influenza vaccine coverage recorded in the ACIR in this age group was less than 2.5% in both Indigenous and non-Indigenous Australian children (excluding Western Australia) from 2005 through to 2014 (Figure 8). Coverage in Western Australian children rose to around 28% in 2009 following introduction of their universal funded program, but has been substantially lower since, following the much publicised issues of increased incidence of high fevers and febrile convulsions post-vaccination with one particular vaccine brand in 2010. The percentage of influenza vaccinated children aged 6 months to less than 5 years recorded in the ACIR as having received 2 doses in their first year of influenza vaccination, as recommended, ranged from 32% to 61% between 2007 and 2014 for both Indigenous and non-Indigenous children, apart from a sharp decline to less than 10% in 2010 following suspension of influenza vaccine use in this age group in that year (data not shown).

Figure 8: Trends in coverage estimates* for seasonal influenza vaccine in children aged 6 months to less than 5 years, Australia, 2005 to 2014, by Indigenous status and state or territory



* Coverage definition = any influenza vaccination in 2014, so at least 1 influenza vaccination. If a child received 2 vaccinations in 2014 they weren't counted twice.

Source: Australian Childhood Immunisation Register.

Timeliness of immunisation

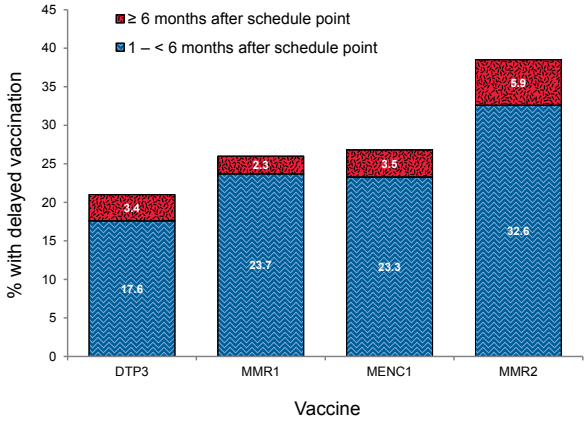
We examined timeliness of immunisation in 2014 for vaccines requiring multiple doses (DTPa, PCV and MMR) or a single dose (meningococcal C) at 12 and 24 months of age.

As demonstrated in previous reports, the proportion with vaccination delay increased with older age (Figure 9). The greatest proportion with any delay was seen with the 2nd dose of MMR vaccine due at 48 months, with 38.5% of doses given late

and 5.9% given very late at ≥6 months (Figure 9). These figures are an improvement from the 2013 report (50.7% and 6.9%, respectively).

For the 3rd dose of DTPa vaccine, there was greater delay for Indigenous children than for non-Indigenous children, with a 20.2% differential in on-time vaccination by 7 months of age (Figure 10). The same pattern was found for timeliness of the 1st dose of MMR vaccine by 13 months of age, but with a smaller differential of 11.5% (Figure 11). This differential in on-time vaccination between Indigenous and non-Indigenous children was slightly improved from the 2013 report, where the corresponding differentials for the 3rd dose of DTPa and 1st dose of MMR were 21.6% and 12.9%, respectively. In contrast to the 3rd dose of DTPa and the 1st dose of MMR, analysis of timeliness of immunisation for a vaccine due at 48 months of age, the 2nd dose of MMR, showed a much smaller differential in delayed receipt between Indigenous and non-Indigenous children, of 3% by 49 months of age (Figure 12a). We also examined the timeliness of immunisation for the 2nd dose of MMR as the coverage algorithm for the 24-month age group changed in July 2014 to include this dose at 18 months of age. Timeliness of the 2nd dose of MMR improved for non-Indigenous children from 58.0% (when due at 48 months) to 68.0% (when due at 18 months) but decreased for Indigenous children (from 55.0% to 52.7%, respectively) (Figure 12b). As a consequence, there was a greater differential in on-time vaccination between Indigenous and non-Indigenous children in 2014 (15.3 percentage points).

Figure 9: Vaccination delay for cohorts born in 2012 (DTPa3, MMR1, MENC1) and 2008 (MMR2), Australia, assessed in 2014



DTPa3 = 3rd dose of a diphtheria-tetanus-acellular pertussis-containing vaccine (due at 6 months of age)

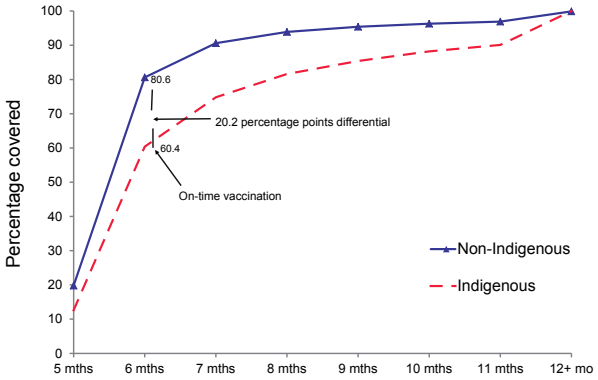
MMR1 = 1st dose of a measles-mumps-rubella vaccine (due at 12 months of age)

MENC1 = 1st dose of a meningococcal C vaccine (due at 12 months of age)

MMR2 = 2nd dose of a measles-mumps-rubella vaccine (due at 48 months of age)

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Figure 10: Timeliness* of the 3rd dose of diphtheria-tetanus-acellular pertussis vaccine (DTPa3), Australia, by Indigenous status

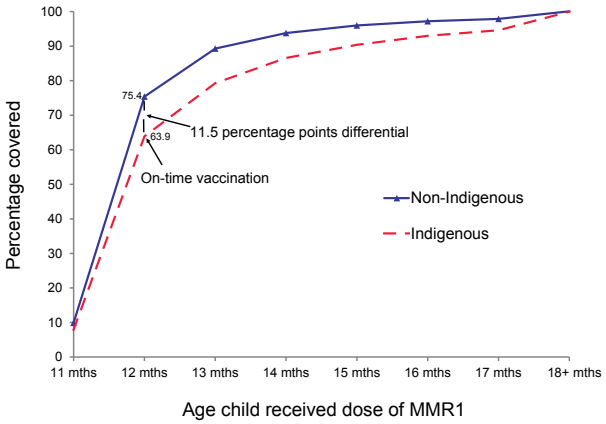


* Percentage covered = number of children who received vaccine dose at particular ages / the total number of children who received the vaccine dose, expressed as a percentage.

Cohort born in 2012.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Figure 11: Timeliness* of the 1st dose of measles-mumps-rubella vaccine (MMR1), Australia, by Indigenous status



* Percentage covered = number of children who received vaccine dose at particular ages / the total number of children who received the vaccine dose, expressed as a percentage.

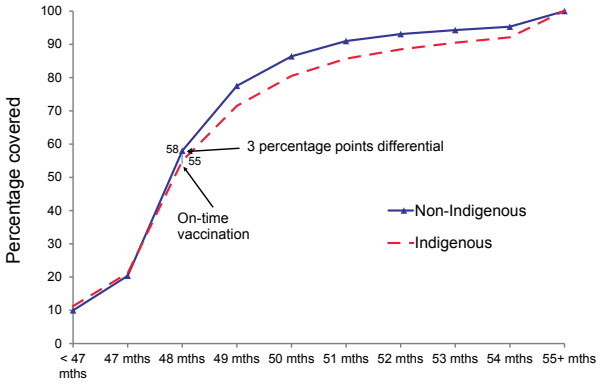
Cohort born in 2012.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Delayed receipt of the 3rd dose of DTPa and the 1st dose of MMR by 1 to <6 months was found in 28% to 38% of Indigenous children and 17% to 24% of non-Indigenous children in 2014, depending on remoteness status (Table 9). Vaccination delay was greater for Indigenous children than for non-Indigenous children for both vaccines across all categories (major cities, inner/outer regional and remote/very remote areas).

Vaccination delay for Indigenous children by jurisdiction was measured for the 3rd dose of PCV, with the highest proportions experiencing delays of 1 to <6 months in Western Australia (34.5%) and the Northern Territory (33.6%), and delays of ≥6 months in South Australia (12.6%) and Western Australia (11.5%) (Figure 13).

Figure 12a: Timeliness* of the 2nd dose of measles-mumps-rubella vaccine (MMR2) due at 48 months, Australia, by Indigenous status

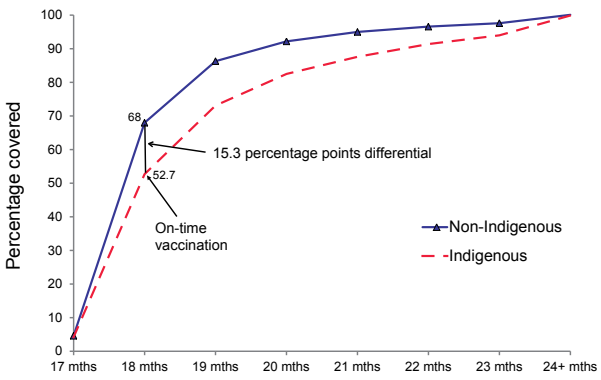


* Percentage covered = number of children who received vaccine dose at particular ages / the total number of children who received the vaccine dose, expressed as a percentage.

Cohort born in 2008.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Figure 12b: Timeliness* of the 2nd dose of measles-mumps-rubella vaccine (MMR2) due at 18 months, Australia, by Indigenous status



* Percentage covered = number of children who received vaccine dose at particular ages / the total number of children who received the vaccine dose, expressed as a percentage.

Cohort born in 2012.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Table 9: Vaccination delay for children 2 year of age,* Australia, 2014, by Indigenous and remoteness status

Vaccine dose	Indigenous status	Remoteness category	1–<6 months after schedule point %	≥6 months after schedule point %
DTPa3	Indigenous	Major cities	27.7	9.6
		Inner and Outer regional	29.4	10.5
		Remote and Very remote	37.5	8.8
	Non-Indigenous	Major cities	16.5	2.9
		Inner and Outer regional	18.1	3.2
		Remote and Very remote	18.3	2.4
MMR1	Indigenous	Major cities	31.5	5.5
		Inner and Outer regional	31.3	5.8
		Remote and Very remote	33.0	3.7
	Non-Indigenous	Major cities	23.2	2.2
		Inner and Outer regional	23.3	2.1
		Remote and Very remote	23.5	2.0

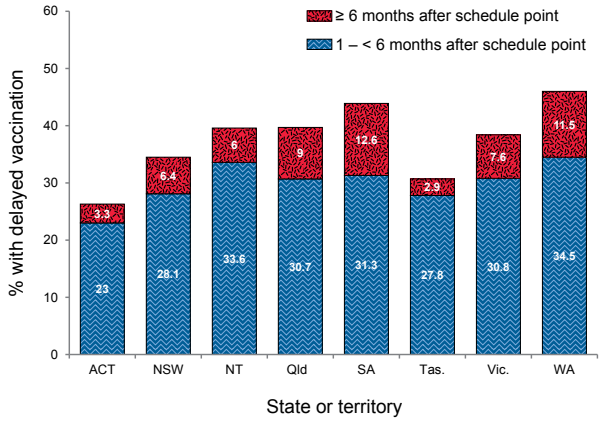
* The cohort of children born in 2012 and assessed in 2014.

DTPa3 = 3rd dose of diphtheria-tetanus-acellular pertussis vaccine

MMR1 = 1st dose of measles-mumps-rubella vaccine

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Figure 13: Vaccination delay for Indigenous children for the 3rd dose of pneumococcal conjugate vaccine, Australia, 2014, by state or territory



Cohort born in 2012.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Trends in timeliness of the 3rd dose of PCV and the 1st dose of MMR vaccine by Indigenous status are provided in Figures 14 and 15. Timeliness for the 3rd dose of PCV improved marginally over time for non-Indigenous children, from 78% in the 2007 birth cohort to 80% in the 2012 birth cohort; however, no improvements were seen for Indigenous children (Figure 14). Timeliness for the 1st dose of MMR vaccine improved over time for non-Indigenous children, from 70.0% in the 2007 birth cohort to 75.0% in the 2012 birth cohort. Timeliness also improved marginally over time for Indigenous children, from 61.0% in the 2007 birth cohort to 63.0% in the 2012 birth cohort (Figure 15).

Figure 14: Timeliness of the 3rd dose of pneumococcal vaccine, Australia, 2007 to 2012, assessed in 2014, by Indigenous status and year of birth

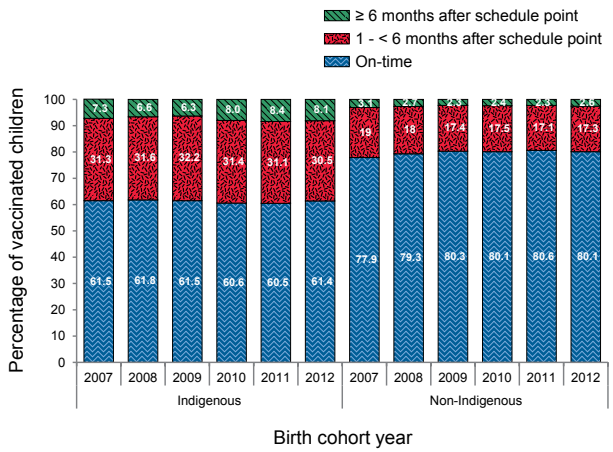
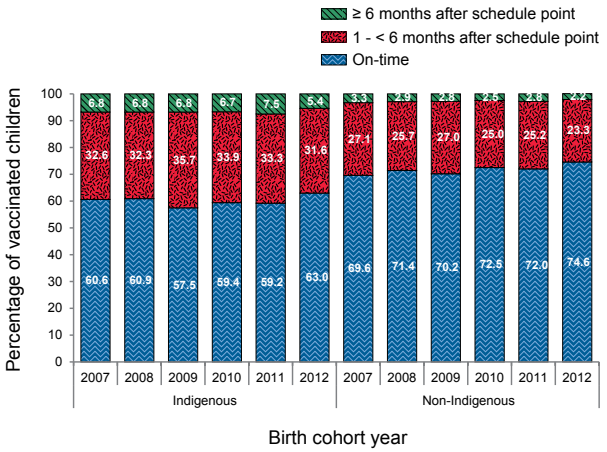


Figure 15: Timeliness of the 1st dose of measles-mumps-rubella vaccine, Australia, 2007 to 2012, assessed in 2014, by Indigenous status and year of birth



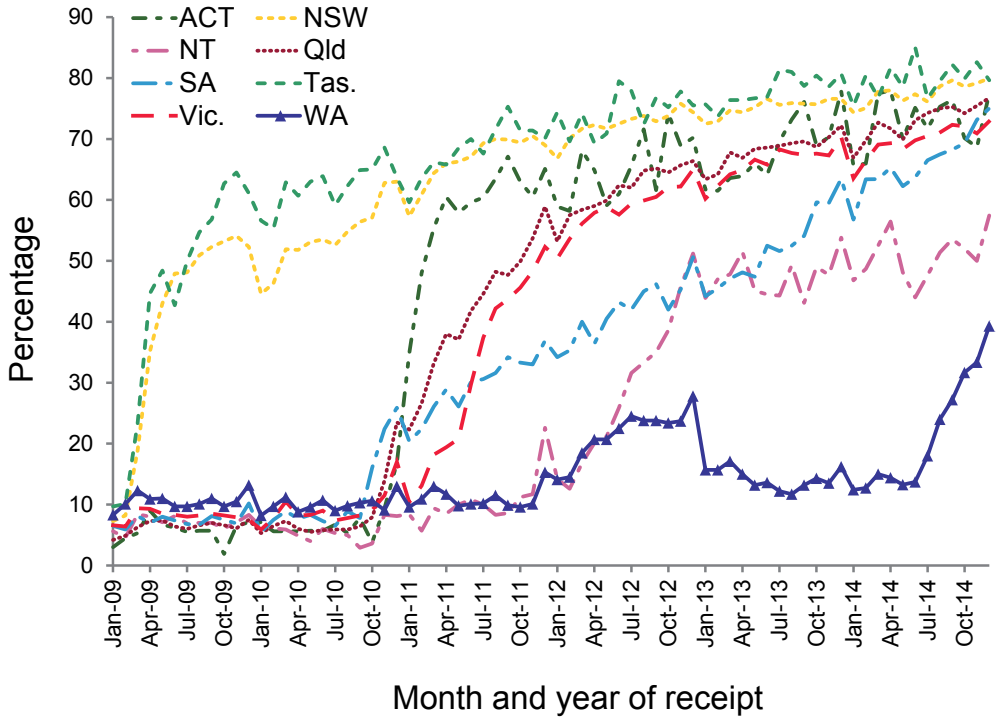
Source: Australian Childhood Immunisation Register.

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

In response to a pertussis epidemic, and to provide early protection to young infants, the Australian Technical Advisory Group on Immunisation (ATAGI) recommended in March 2009 that immunisation providers give the 1st dose of DTPa vaccine at 6 weeks of age instead of 8 weeks (2 months) of age. This was promoted in that year during epidemics in New South Wales and Tasmania and later in other jurisdictions. Prior to this, very few children received the vaccine dose at less than 8 weeks of age. Over the next few years the percentage rose and by late 2014 it was greater than 70% in all jurisdictions except Western Australia and the Northern Territory (Figure 16).

ATAGI also recommended in October 2009 that the pre-school booster dose of DTPa-IPV could be given from 3.5 rather than 4 years of age. Take-up of this recommendation was slower, with no jurisdiction giving the vaccine in any great numbers at 3.5 to 4 years of age until November 2010 (Figure 17). As at December 2014, more than 35% of children in three jurisdictions (the Australian Capital Territory, the Northern Territory and South Australia) were receiving the dose at 3.5 to 4 years of age (Figure 17).

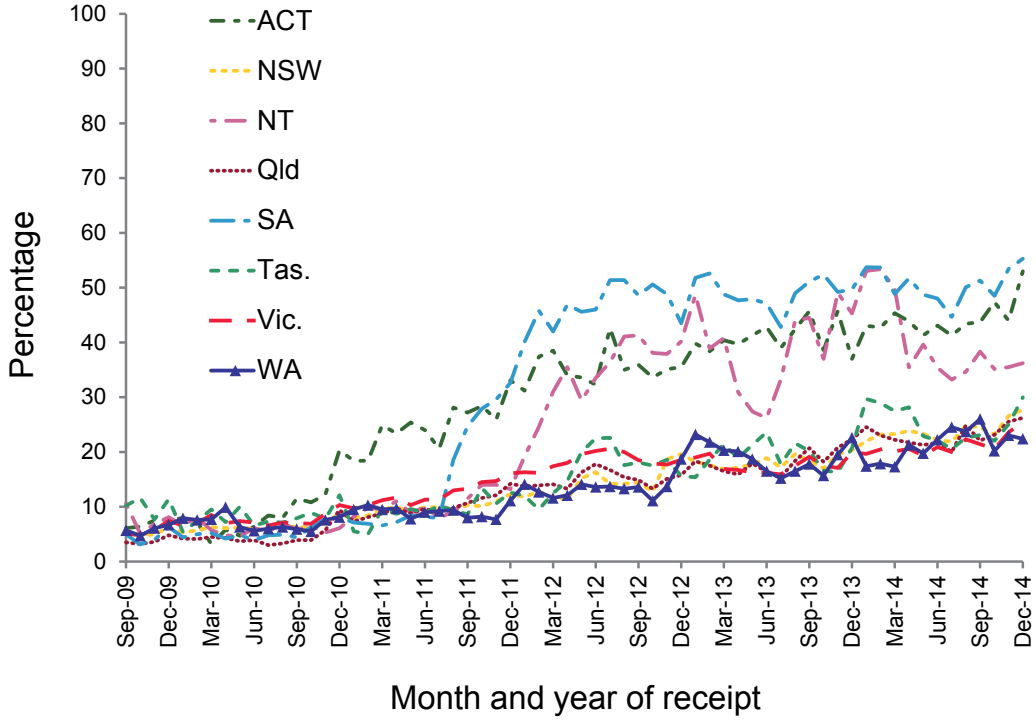
Figure 16: Percentage of children who received their 1st dose of DTPa/Hexa vaccine at age 6 to < 8 weeks, Australia, 2009 to 2014, by state or territory and month of receipt



* DTPa/Hexa = combined diphtheria-tetanus-acellular pertussis (DTPa), polio, *Haemophilus influenzae* type b and hepatitis B vaccine.

Source: Australian Childhood Immunisation Register.

Figure 17: Percentage of children who received their 4th dose of DTPa/Hexa vaccine at age 3.5 years to <4 years, Australia, 2009 to 2014, by state or territory and month of receipt



* DTPa/Hexa = combined DTPa-IPV-Hib-Hep B vaccine

Source: Australian Childhood Immunisation Register.

Objection to vaccination and incomplete immunisation

Registered objection

The proportions of children with registered vaccination objection status and no vaccines recorded on the ACIR, registered vaccination objection status and at least 1 vaccine recorded on the ACIR, no registered vaccination objection status and no vaccines recorded on the ACIR, and no registered vaccination objection status and not 'fully immunised' by 24 months of age (partially immunised), for all jurisdictions and Australia, is shown in Table 10. Some of the children in the latter 2 groups may be incompletely immunised due to unregistered vaccine objection. Of the 4 groups, the largest is those without a registered objection and partially immunised.

The rate of registered objection in 2014 for Australia was 1.8%. This varied by jurisdiction with a high of 2.4% in Queensland and a low of 1.1% in Tasmania.

The proportions of children with a registered objection to vaccination are presented by Statistical Area 3 (SA3) in Figure 18. The map shows pockets of high levels of registered objection in 2014, particularly in coastal areas of northern and south-east Queensland, northern New South Wales, the Adelaide Hills and the Margaret River/Busselton region. These areas have had consistently high levels of registered objection over many years.

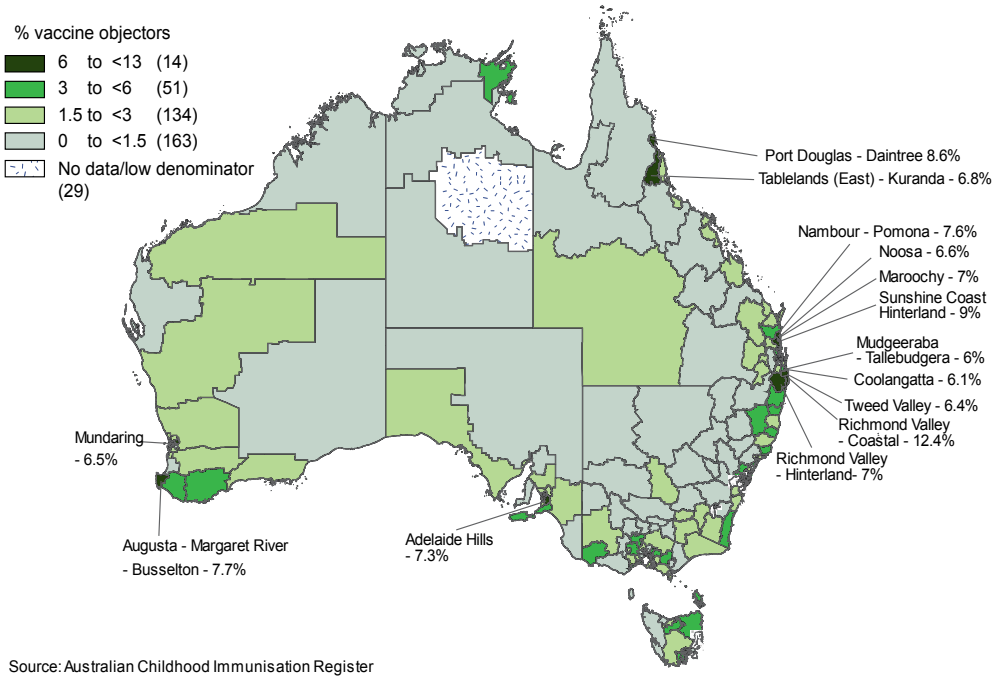
Partially immunised children

The percentage of partially immunised children (excluding those with a registered vaccination objection) who were up-to-date in 2014 for specific vaccines due by 24 months of age is shown in Table 1, by jurisdiction. The vaccines that partially immunised children were most commonly missing by 24 months of age were the 2nd dose of MMR and the dose of varicella (Table 11).

Human papillomavirus vaccine coverage

Vaccination coverage, as notified to the HPV Register, for dose 3 of the HPV vaccine for females and males aged 15 years in 2014 is shown in Table 12. For females in Australia, 73.4% completed a full course of the vaccine, up from 71.4% in 2013. Coverage varied by jurisdiction from a low of 67.7% in Tasmania to a high of 77.4% in Victoria in 2014. Coverage in all age groups was higher for earlier doses, as high as 87.0% for the 1st dose in females aged 12–13 years (Figure 19).

Figure 18: Proportion of children with recorded vaccination objection, Australia, 2014, by Statistical Area 3



Source: Australian Childhood Immunisation Register

Cohort born January 2012 – December 2012, assessed in 2014.

* Number of SA3s in each category in parentheses.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Table 10: Percentage of children aged 2 years* with registered vaccination objection and whether no/some vaccines recorded on the Australian Childhood Immunisation Register, Australia, assessed in 2014, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aus.
Total number of children	1,273	22,002	737	13,716	4,383	1,270	17,280	7,334	67,995
Objection† and no vaccines recorded	1.0	1.0	0.6	1.7	1.4	0.7	1.0	1.3	1.2
Objection† and at least 1 vaccine recorded	0.6	0.4	0.6	0.7	0.7	0.4	0.6	0.8	0.6
No objection and no vaccines recorded	1.5	1.7	2.6	1.7	1.6	0.9	1.6	1.8	1.7
No objection and partially immunised‡	6.9	9.9	9.2	7.4	9.8	12.5	9.2	9.7	9.2

* Cohort born 1 October – 31 December 2012 and assessed in 2014.

† Vaccination objection recorded on the Australian Childhood Immunisation Register.

‡ Record of at least 1 vaccine recorded on the Australian Childhood Immunisation Register, no recorded vaccination objection, and not 'fully immunised' by 24 months of age. 'Fully immunised' – 3 doses of a diphtheria-tetanus-acellular pertussis-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP-containing *Haemophilus influenzae* type b vaccine or 4 doses of any other *Haemophilus influenzae* type b vaccine, 3 doses of hepatitis B vaccine, 2 doses of a measles-mumps-rubella-containing vaccine, 1 dose of varicella vaccine, and 1 dose of meningococcal C vaccine.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Table 11: Percentage of partially immunised* children aged 2 years† who have received individual vaccines, Australia, 2014, by state or territory

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	100	2,597	84	1,202	526	194	1,930	865	7,498
3 doses of diphtheria, tetanus, acellular pertussis vaccine	88.0	77.9	83.3	79.2	80.6	75.3	81.6	79.5	79.6
3 doses of polio vaccine	88.0	77.2	83.3	79.0	80.4	75.3	81.3	78.5	79.1
4 doses of *Haemophilus influenzae* type b vaccine	59.0	58.5	61.9	61.7	60.1	60.3	56.6	59.4	58.8
3 doses of hepatitis B vaccine	82.0	74.0	83.3	76.0	76.6	74.7	78.2	74.3	75.8
1 dose of meningococcal C conjugate vaccine	62.0	63.5	73.8	69.6	68.6	68.0	65.3	66.1	65.8
2 doses of measles, mumps, rubella vaccine	23.0	27.2	34.5	28.0	28.5	29.4	28.7	23.7	27.5
1 dose of varicella vaccine	25.0	27.1	21.4	21.3	25.3	26.8	26.3	23.0	25.3

* Record of at least 1 vaccine recorded on the Australian Childhood Immunisation Register, no recorded vaccination objection, and not 'fully immunised' by 24 months of age. 'Fully immunised' – 3 doses of a diphtheria-tetanus-acellular pertussis-containing vaccine, 3 doses of polio vaccine, 3 or 4 doses of PRP-OMP-containing *Haemophilus influenzae* type b vaccine or 4 doses of any other *Haemophilus influenzae* type b vaccine, 3 doses of hepatitis B vaccine, 2 doses of a measles-mumps-rubella-containing vaccine, 1 dose of varicella vaccine, and 1 dose of meningococcal C vaccine.

† Cohort born 1 October – 31 December 2012 and assessed in 2014.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Coverage was higher in the younger age groups than the older age groups, with only 52.0% of females aged 20–26 years fully vaccinated. For males in Australia, 61.4% completed a full course of the vaccine. Coverage varied by jurisdiction from a low of 55.1% in Tasmania to a high of 67.8% in Victoria in 2014 (Table 12). As with data for females, coverage in all age groups was higher for earlier doses, as high as 75.0% for the 1st dose in males aged 14–15 years (data not shown). HPV coverage by Indigenous status is not available due to limitations in Indigenous status reporting on the HPV Register.

Coverage in the elderly

According to a Newspoll Omnibus Survey, 73% of Australian adults aged 65 years or over had received an influenza vaccination in 2014 as at mid-June.

Provider type

GPs administer the large majority of immunisations in Australia (Figure 20); the proportion given by GPs has increased over the past 12 years by almost 5% (data not shown). Regional differ-

Table 12: Coverage (%) for 3 doses of human papillomavirus vaccine for girls 15 years of age in 2011, 2012, 2013 and 2014, and males age 15 years in 2014, by state or territory, Australia

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
2011	74.2	74.5	87.0	72.4	68.0	66.5	76.5	64.6	72.9
2012	74.4	71.0	84.5	69.4	71.0	64.7	74.2	70.1	71.4
2013	74.0	68.6	81.4	71.0	72.4	64.1	75.2	71.2	71.4
2014	70.0	69.8	77.3	73.5	73.1	67.7	77.4	76.0	73.4
2014 males*	64.2	56.9	55.4	61.1	63.3	55.1	67.8	61.0	61.4

* Reflects male catch-up vaccination program 2013–2014. Routine immunisation ongoing at age 12–13 years.

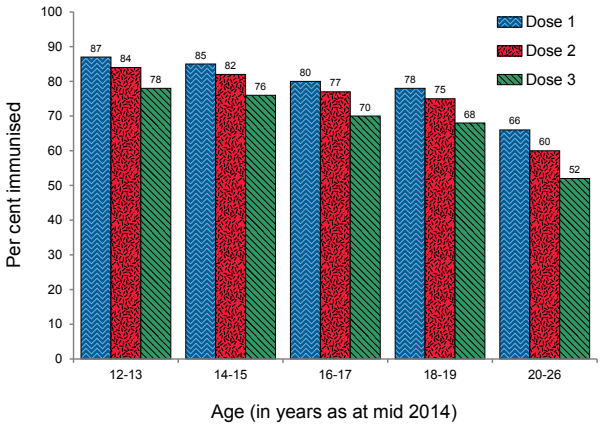
Includes doses that comply with the recommended vaccine dosage and administration as per *The Australian Immunisation Handbook* (up to 3 doses administered at prescribed intervals).

Denominator data used is Australian Bureau of Statistics Estimated Resident Population on 2012 boundaries (final) as at 30 June in the relevant year.

Earlier coverage reports utilised interim Australian Bureau of Statistics Estimated Resident Population data. As a result, small changes in coverage rates may be apparent if compared with earlier reports.

Source: National Human Papillomavirus Vaccination Program Register, January 2016.

Figure 19: Human papillomavirus vaccination coverage for females, Australia, mid-2014, by dose number and age group



Technical notes:

Data extracted from the National Human Papillomavirus Vaccination Program Register (HPV Register) as at 19 January 2016.

Includes doses that comply with the recommended vaccine dosage and administration as per *The Australian Immunisation Handbook* (up to 3 doses administered at prescribed intervals).

Population is Estimated Resident Population 2014 (as at 30/06/2014) from the Australian Bureau of Statistics Cat 3101.0 Australian Demographic Statistics, Tables 51 to 58: Estimated resident population by single year of age by state and territory. Interim data published December 2014.

Age is age as at date of Estimated Resident Population estimate (30 June 2014).

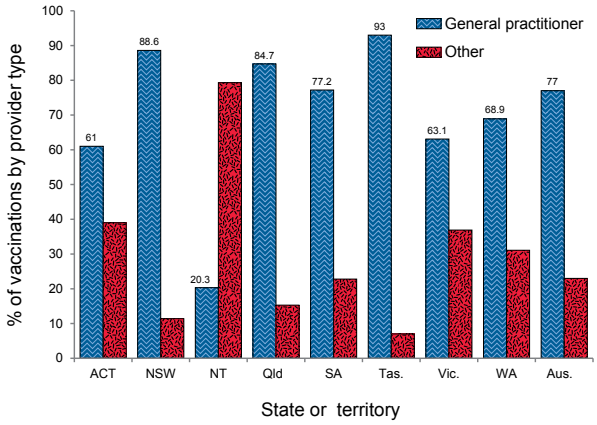
Coverage is calculated as doses administered and reported to the HPV Register / Estimated Resident Population expressed as a percentage.

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

Source: National HPV Vaccination Program Register, June 2016.

ences are marked, with over 80% of immunisations administered by GPs in New South Wales, Queensland and Tasmania, and the majority of immunisations given by GPs in all other jurisdictions except for the Northern Territory.

Figure 20: Proportion of vaccinations on the Australian Childhood Immunisation Register given by provider type, January to December 2014, by state or territory, Australia



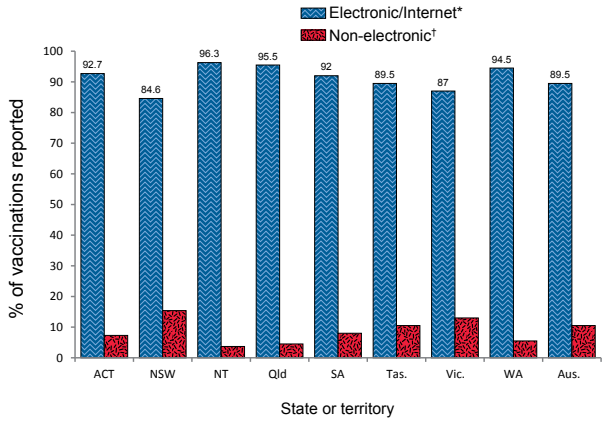
Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Mechanisms of reporting to the Australian Childhood Immunisation Register

The proportions of vaccinations on the ACIR lodged by electronic/online mechanisms versus non-electronic mechanisms by jurisdiction are shown in Figure 21. Most reporting in 2014

occurred through electronic/online mechanisms, for all jurisdictions, with the proportion reported through this method varying from 96.3% in the Northern Territory to 84.6% in New South Wales.

Figure 21: Proportion of immunisations on the Australian Childhood Immunisation Register lodged by type of reporting mechanism, January to December 2014, by state or territory, Australia



* Online claiming: Medicare Australia online claiming, a software application that allows the transmission of Australian Childhood Immunisation Register (ACIR) data via the immunisation provider's desktop software, or Internet Data Interchange where approved immunisation providers can send immunisation details using the Internet Data Interchange upload facility through the ACIR secure area within Medicare Australia's web site, or Health Professionals Online Services, where approved immunisation providers can submit immunisation encounters which are records of immunisations, view immunisation history, request reports and update provider contact details.

† Manual voucher: by completing an immunisation encounter form and sending it to Medicare Australia, or an immunisation history form to record a child's vaccination details that may be missing from the ACIR. This form must be completed by a doctor or immunisation provider and sent to the ACIR.

Source: Australian Childhood Immunisation Register, data as at 31 March 2015.

Discussion

In 2014, 'fully immunised' coverage was higher than in 2013 nationally for children at 12 and 60 months of age (91.5% and 92.0% versus 90.8% and 91.2%, respectively) but lower for children at 24 months of age (86.8% versus 92.1%). However, this decline in coverage at the 24-month age milestone was predominantly a measurement artefact due to the immunisation coverage assessment algorithm for 'fully immunised' at the 24-month milestone being amended in July 2014 to include a dose of meningococcal vaccine, a dose of varicella vaccine and a 2nd dose of MMR vaccine.

Aside from minor fluctuations and the impact of changes to assessment algorithms, 'fully immunised' coverage has been largely stable at the 12– and 24-month age milestones since late 2003. The more than 10 percentage points increase in coverage at 60 months of age since 2009 is likely due to a focus on improved timeliness of vaccination, facilitated by a change to the ACIR overdue rules in January 2009, where children became overdue for their pre-school boosters at 49 months of age instead of the previous 60 months. This change had an impact on eligibility for parent incentive payments and outcome payments for providers.

Coverage estimates for varicella vaccine and the 2nd dose of MMR were substantially lower than for other vaccines included in the algorithm for 'fully immunised' at the 24-month milestone. Varicella vaccine coverage is probably lower due to both the shorter time it has been on the NIP and the age of administration (18 months). The 18-month schedule point was historically associated with lower coverage levels prior to 2003, when there was an 18-month pertussis booster, and there was a gap of over 2 years from 2003 to 2005 when no vaccine was administered at 18 months. When we assessed varicella vaccine coverage at 60 months of age instead of 24 months, we observed much higher estimates across all jurisdictions, ranging from 6.0 to 10.4 percentage points higher. We also found that national varicella vaccine coverage increased by 3.7 percentage points after the introduction of MMRV vaccine in mid-2013, so further increases in coverage may occur as a result of this schedule change. For rotavirus vaccines, strict upper age limits for administration, which reduce the ability to receive late doses, are likely to explain lower coverage when compared to other vaccines assessed at 12 months of age. The implications of lower coverage for rotavirus and varicella vaccines also differ. In the case of rotavirus vaccine, coverage of 80% or greater has resulted in substantial herd immunity and decreases in rotavirus hospitalisations in Australia and elsewhere.[23,24] In contrast, modelling studies suggest that low coverage (70%–90%) with varicella vaccine may result in a shift of disease to older age groups with higher disease severity.[25]

Coverage for vaccines recommended for Indigenous children only (i.e. hepatitis A and a booster dose of pneumococcal vaccine) remained suboptimal in 2014. The extent of under-reporting to the ACIR for these vaccines is unknown but may be more than for universal vaccines, given the lack of incentive payments for notification to the ACIR. However, lower coverage for vaccines targeted at Indigenous people has been a relatively consistent finding using a range of different methods for both children[26] and adults.[27] Both a lack of provider knowledge about the recommendations for high-

risk groups, and poor identification of Indigenous children by immunisation providers, are likely to be important contributing factors. While coverage for 2 doses of hepatitis A vaccine was only 63%, an additional 17% of Indigenous children received a single dose, which provides a protective antibody response in most children.[28]

Although most children eventually complete the scheduled vaccination series by the 24-month milestone, many still do not do so in a timely manner. On-time vaccination for vaccines assessed at 12 and 24 months of age in 2014 increased for Indigenous children but decreased for non-Indigenous children. However, while the differential in on-time vaccination between Indigenous and non-Indigenous children in 2014 did improve marginally from 2013, timeliness is still a significant problem for Indigenous children in Australia. Poorer timeliness in Indigenous children aged 2 years of age has been noted previously.[29] Timeliness continued to improve for vaccines due at 48 months of age and assessed at 60 months of age, for both Indigenous and non-Indigenous children. In 2014 more than 70% of children in all jurisdictions except Western Australia and the Northern Territory received the 1st dose of DTPa vaccine prior to 8 weeks of age, in line with recommendations encouraging early protection of young infants from pertussis infection.

Immunisation at the earliest appropriate age should be a public health goal for countries such as Australia where high levels of vaccine coverage at milestone ages have been achieved. This is especially so for the 2nd dose of the measles vaccine where vaccination delay has consistently been an issue. The change in scheduling of this dose to 18 months of age that occurred in mid-2013 has led to an improvement in the timeliness of this dose but in non-Indigenous children only.

Analysis of ACIR data has demonstrated the rapid uptake by the population of new vaccines in the Australian setting, unlike some other developed countries.[30,31] Only 1.8% of children are registered as having parental vaccination objection and some others are likely unvaccinated due to unregistered objection. However, incomplete immunisation is also often due to access and logistic issues. Further in-depth analysis and interpretation of the data about incompletely immunised children will be the subject of an upcoming National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases report. Further vaccination coverage estimates in small areas have been provided by the National Health Performance Authority for children in 2013 to 2015.[32]

Coverage data for the HPV vaccine from the National HPV Vaccination Program Register reflect a successful school-based program with lower but still substantial coverage for the catch-up program in older females.[33,34] The coverage achieved in the program has resulted in demonstrable decreases in HPV prevalence in young women,[36] and in genital warts[37] and cervical abnormalities.[38] Coverage achieved during the catch-up program for males aged 14–15 years in 2013 to 2014 indicates that the vaccine is acceptable to most parents of boys but that further work may be needed to normalise HPV vaccination for males and raise coverage to the same level as achieved in females. Ongoing routine HPV vaccination of both sexes, and a possible change to a 2-dose schedule for adolescents aged under 15 years at 1st dose in future, as endorsed by the World Health Organization in 2014,[39] will hopefully facilitate further increases in HPV vaccine coverage.

Data provided in this report reflect continuing successful delivery of the NIP in Australia, while identifying some areas for improvement. Coverage for rotavirus vaccine, varicella vaccine and the 2nd dose of MMR vaccine is below that for other vaccines. Timeliness of vaccination could be improved, particularly for Indigenous children, and coverage for vaccines recommended only for Indigenous children is lower than for other vaccines.

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List of vaccine abbreviations

13vPCV	13-valent pneumococcal conjugate vaccine
23vPPV	23-valent pneumococcal polysaccharide vaccine
Comvax	*Haemophilus influenzae* type b conjugate (meningococcal protein conjugate) and hepatitis B (recombinant) vaccine
dTpa	diphtheria-tetanus-acellular pertussis (adults, adolescents and children aged ≥ 10 years formulation)
DTPa	diphtheria-tetanus-acellular pertussis (children aged < 10 years formulation)
DTPa-IPV	diphtheria-tetanus-acellular pertussis-inactivated poliovirus
Engerix-B	recombinant DNA hepatitis B vaccine (paediatric formulation)
Flu	influenza
H-B-VAX II	hepatitis B (paediatric formulation)
Hep A	hepatitis A
Hep B	hepatitis B
Hib	*Haemophilus influenzae* type b
HPV	human papillomavirus
MMR	measles-mumps-rubella
MMRV	measles-mumps-rubella-varicella
PCV	pneumococcal conjugate vaccine
PRP-OMP	*Haemophilus influenzae* type b conjugate vaccine
VZV	varicella-zoster virus

References

1. Hull B, Deeks S, Menzies R, McIntyre P. Immunisation coverage annual report, 2007. *Commun Dis Intell* 2009;33(2):170–187.
2. Hull BP, Mahajan D, Dey A, Menzies RI, McIntyre PB. Immunisation coverage annual report, 2008. *Commun Dis Intell* 2010;34(3):241–258.
3. Hull B, Dey A, Mahajan D, Menzies R, McIntyre PB. Immunisation coverage annual report, 2009. *Commun Dis Intell* 2011;35(2):132–148.
4. Hull B, Dey A, Menzies R, McIntyre PB. Annual immunisation coverage report, 2010. *Commun Dis Intell* 2013;37(1):E21–E39.
5. Hull BP, Dey A, Menzies RI, Brotherton JM, McIntyre PB. Immunisation coverage annual report, 2011. *Commun Dis Intell* 2013;37(4):E291–E312.
6. Hull BP, Dey A, Menzies RI, Brotherton JM, McIntyre PB. Immunisation coverage annual report, 2012. *Commun Dis Intell* 2014;38(3):E208–E231.
7. National Health Performance Authority. Healthy communities: Immunisation rates for children in 2012–13. 2014. Available from: http://www.nhpa.gov.au/internet/nhpa/publishing.nsf/Content/Our-reports Accessed on 2 December 2014.
8. Hull BP, Deeks SL, McIntyre PB. The Australian Childhood Immunisation Register – a model for universal immunisation registers? *Vaccine* 2009;27(37):5054–5060.
9. Hull BP, McIntyre PB, Heath TC, Sayer GP. Measuring immunisation coverage in Australia: a review of the Australian Childhood Immunisation Register. *Aust Fam Physician* 1999;28(1):55–60.
10. Australian Government Department of Health. Update: No Jab No Pay – Immunisation catch-up arrangements. 2015. Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/clinical-updates-and-news/$File/Update-No-Jab-No-Pay-Immunisation-Catch-Up-Arrangements(D15-1126865).pdf Accessed on 30 November 2015.
11. Australian Technical Advisory Group on Immunisation. *The Australian Immunisation Handbook*. 10th edn. Canberra: Australian Government Department of Health; 2013.
12. O'Brien ED, Sam GA, Mead C. Methodology for measuring Australia's childhood immunisation coverage. *Commun Dis Intell* 1998;22(3):36–37.
13. Hull BP, McIntyre PB. Immunisation coverage reporting through the Australian Childhood Immunisation Register – an evaluation of the third-dose assumption. *Aust N Z J Public Health* 2000;24(1):17–21.
14. Hull BP, Lawrence GL, MacIntyre CR, McIntyre PB. Estimating immunisation coverage: is the 'third dose assumption' still valid? *Commun Dis Intell* 2003;27(3):357–361.
15. Hull B. Australian childhood immunisation coverage, 1 October to 31 December cohort, assessed as at 31 March 2014. *Commun Dis Intell* 2014;38(3):E260–E261.

16. Australian Population and Migration Research Centre. ARIA and accessibility. Accessibility/Remoteness Index of Australia – ARIA+ (2011). 2011. Available from: http://www.adelaide.edu.au/apmrc/research/projects/category/aria.html Accessed on 17 November 2014.
17. Rank C, Menzies RI. How reliable are Australian Childhood Immunisation Register coverage estimates for Indigenous children? An assessment of data quality and coverage. *Commun Dis Intell* 2007;31(3):283–287.
18. Australian Bureau of Statistics. Australian Statistical Geography Standard (ASGS). 2011. Available from: http://www.abs.gov.au/websitedbs/d3310114.nsf/home/australian+statistical+geography+standard+%28asgs%29 Accessed on 17 November 2014.
19. MapInfo Pro version 15.0. Stamford, Connecticut, USA. 2015.
20. Australian Bureau of Statistics. Australian Statistical Geography Standard (ASGS): Correspondences, July 2011. 2012. Available from: http://www.abs.gov.au/AUSSTATS/abs@.nsf/Lookup/1270.0.55.006Main+Features1July%202011?OpenDocument Accessed on 17 November 2014.
21. Australian Institute of Health and Welfare. 2009 Adult Vaccination Survey: summary results. 2011. Available from: http://www.aihw.gov.au/publication-detail/?id=10737418409 Accessed on 2 December 2014.
22. Newspoll. Newspoll Omnibus Survey June 2014 – Summary Report Flu Vaccinations. 2014. Available from: http://www.immunise.health.gov.au/internet/immunise/publishing.nsf/Content/762A8FB9101D1759CA257D49002227B6/$File/summ-report-flu-vaccinations-survey2014.pdf Accessed on 30 November 2015.
23. Buttery JP, Lambert SB, Grimwood K, Nissen MD, Field EJ, Macartney KK, et al. Reduction in rotavirus-associated acute gastroenteritis following introduction of rotavirus vaccine into Australia's national childhood vaccine schedule. *Pediatr Infect Dis J* 2011;30(1 Suppl):S25–S29.
24. Dey A, Wang H, Menzies R, Macartney K. Changes in hospitalisations for acute gastroenteritis in Australia after the national rotavirus vaccination program. *Med J Aust* 2012;197(8):453–457.
25. Brisson M, Edmunds W, Gay N, Law B, De Serres G. Modelling the impact of immunization on the epidemiology of varicella zoster virus. *Epidemiol Infect* 2000;125(3):651–669.
26. Hull BP, McIntyre PB. What do we know about 7vPCV coverage in Aboriginal and Torres Strait Islander children? *Commun Dis Intell* 2004;28(2):238–243.
27. Menzies R, Turnour C, Chiu C, McIntyre P. Vaccine preventable diseases and vaccination coverage in Aboriginal and Torres Strait Islander people, Australia, 2003 to 2006. *Commun Dis Intell* 2008;32 Suppl:S2–S67.
28. Plotkin S, Orenstein WA, Offit PA. Vaccines. 5th edn. Philadelphia, PA: Saunders Elsevier; 2008.
29. Hull BP, McIntyre PB. Timeliness of childhood immunisation in Australia. *Vaccine* 2006;24(20):4403–4408.
30. Centers for Disease Control and Prevention. National, state, and local area vaccination coverage among children aged 19–35 months – United States, 2012. *MMWR Morb Mortal Wkly Rep* 2013;62(36):733–740.
31. Health and Social Care Information Centre. NHS immunisation statistics, England, 2012–13. 2013. Available from: http://www.hscic.gov.uk/catalogue/PUB11665 Accessed on 2 December 2014.
32. National Health Performance Authority. Healthy communities: Immunisation rates for children in 2013–15. 2016. Accessed on 20 February 2016. Available from: http://www.nhpa.gov.au/internet/nhpa/publishing.nsf/Content/Our-reports
33. Brotherton JM, Murray SL, Hall MA, Andrewartha LK, Banks CA, Meijer D, et al. Human papillomavirus vaccine coverage among female Australian adolescents: success of the school-based approach. *Med J Aust* 2013;199(9):614–617.
34. Brotherton J, Gertig D, Chappell G, Rowlands L, Saville M. Catching up with the catch-up: HPV vaccination coverage data for Australian women aged 18–26 years from the National HPV Vaccination Program Register. *Commun Dis Intell* 2011;35(2):197–201.
35. Brotherton JM, Liu B, Donovan B, Kaldor JM, Saville M. Human papillomavirus (HPV) vaccination coverage in young Australian women is higher than previously estimated: independent estimates from a nationally representative mobile phone survey. *Vaccine* 2014;32(5):592–597.
36. Tabrizi SN, Brotherton JM, Kaldor JM, Skinner SR, Liu B, Bateson D, et al. Assessment of herd immunity and cross-protection after a human papillomavirus vaccination programme in Australia: a repeat cross-sectional study. *Lancet Infect Dis* 2014;14(10):958–966.
37. Ali H, Donovan B, Wand H, Read TR, Regan DG, Grulich AE, et al. Genital warts in young Australians five years into national human papillomavirus vaccination programme: national surveillance data. *BMJ* 2013;346:f2032.
38. Gertig DM, Brotherton JM, Budd AC, Drennan K, Chappell G, Saville AM. Impact of a population-based HPV vaccination program on cervical abnormalities: a data linkage study. *BMC Med* 2013;11:227.
39. World Health Organization. Meeting of the Strategic Advisory Group of Experts on immunization, April 2014 – conclusions and recommendations. *Wkly Epidemiol Rec* 2014 May 23;89(21):221–236.

Quarterly reports

OzFoodNet quarterly report, 1 October to 31 December 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 that commenced in Australia between 1 October and 31 December 2014.

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

Summary

During the 4th quarter of 2014 (1 October to 31 December), OzFoodNet sites reported 601 outbreaks of enteric illness, including those transmitted by contaminated food or water. Outbreaks of gastroenteritis are often not reported to health authorities, which results in current figures under-representing the true burden of enteric disease outbreaks within Australia. There were 10,072 people affected in these outbreaks with 310 hospitalisations and 35 deaths. This was similar to the number of people affected, hospitalised and who died compared with the 5-year average for the 4th quarter from 2009 to 2013 (9,665 affected; 267 hospitalised; 31 deaths). The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission. In this quarter, 78% of outbreaks (n=469) were due to transmission via this route (Table 1). This was lower than the same quarter in 2013 (n=543) but slightly higher than the 5-year mean (4th quarter, 2009–2013) of 423 outbreaks due to person-to-person transmission. Of the reported person-to-person outbreaks this quarter, 53% (247 outbreaks) occurred in aged care facilities and 32% (150 outbreaks) occurred in child care facilities.

Foodborne and suspected foodborne disease outbreaks

There were 54 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission (Table 1, Appendix). There were 683 people affected by these outbreaks, with 59 hospitalisations and 1 death reported. This was the highest number of foodborne and suspected foodborne disease outbreaks ever reported for a quarter by OzFoodNet (2001–2014). The second highest number was the 1st quarter in 2012 (n=51). The second highest number for the 4th quarter

Table 1: Outbreaks and clusters of gastrointestinal illness and number ill reported by OzFoodNet, 1 October to 31 December 2014, by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total outbreaks and clusters*	Number ill
Foodborne and suspected foodborne	54	9	683
Suspected waterborne	1	<1	6
Person-to-person	469	78	8,703
Unknown (*Salmonella* cluster)	9	1	82
Unknown (other or multiple pathogens) cluster	2	<1	31
Unknown	66	11	567
Total	601	100	10,072

* Percentages do not add to 100 due to rounding.

was in 2009 (n=49). These figures are more than double the number of foodborne outbreaks that were reported in the 3rd quarter of 2014 (n=25) and a 46% increase on the 5-year mean for the 4th quarter between 2009 and 2013 (n=37). Despite the increased total number of outbreaks, the total number of people affected (n=682) was only 65% of that for the same quarter in 2013 (n=1,044). 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. Hence, changes in the number of foodborne outbreaks should be interpreted with caution.

Salmonella Typhimurium was identified as or suspected to be the aetiological agent in 41% (22/54) of foodborne or suspected foodborne outbreaks during this quarter, a higher proportion than the number from the same quarter in 2013 (27%; 10/37). The aetiological agents for the remaining outbreaks during this quarter included: norovirus (8 outbreaks); ciguatoxin (6 outbreaks); *Salmonella* Chester (2 outbreaks); *Staphylococcus aureus* (2 outbreaks); and *Salmonella* Singapore, *Clostridium perfringens*, *C. bifermentans*, *Cryptosporidium* and *Campylobacter* (1 outbreak each). For 9 outbreaks, the aetiological agent was unknown.

Approximately 54% (29/54) of all the foodborne or suspected foodborne outbreaks reported in this quarter were associated with food prepared in restaurants (Table 2), which was higher than the average percentage of foodborne or suspected foodborne outbreaks associated with restaurants in the 4th quarter from 2009 to 2013 (39%).

To investigate these outbreaks, OzFoodNet sites conducted 9 cohort studies, 3 case control studies and collected descriptive case series data for 36 investigations. No individual patient data were collected for 6 outbreaks. The evidence used to implicate food vehicles included the following: analytical evidence in 10 outbreaks; microbiological evidence in 14 outbreaks; descriptive evidence in 29 outbreaks; and both analytical and microbiological evidence in 1 outbreak. Of the 25 confirmed foodborne outbreaks for which an analytical and/or a microbiological link to a food vehicle was established, *S.* Typhimurium was the aetiological agent for 36% (9/25).

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

Australian Capital Territory

During this quarter, the Australian Capital Territory reported 1 outbreak of foodborne or suspected foodborne illness. The aetiological agent identified was *S.* Typhimurium phage type (PT) 135, with multi-locus variable number tandem repeat analysis (MLVA) profile 03-17-09-11-523.

Description of key outbreak

Three people became ill after drinking eggnog made with raw egg at a Christmas luncheon in a private residence. Two of the 3 cases were con-

Table 2: Outbreaks of foodborne or suspected foodborne disease and number ill reported by OzFoodNet, 1 October to 31 December 2014, by food preparation setting

Food preparation setting	Outbreaks	Per cent of foodborne outbreaks*	Number ill
Restaurant	29	54	405
Primary produce	7	13	23
Private residence	5	9	33
Commercial caterer	4	7	105
Aged care	2	4	13
Camp	1	2	30
Takeaway	1	2	26
Other	1	2	16
Bakery	1	2	13
Community	1	2	5
Fair/festival/mobile service	1	2	4
Total	54	100	683

* Percentages may not add to 100 due to rounding.

firmed positive with *S.* Typhimurium PT 135, MLVA 03-17-09-11-523; both of whom presented to an emergency department, resulting in 1 hospital admission. The median incubation period was 19 hours. The eggs used in the drink were commercially produced free range eggs. Four leftover eggs from the implicated carton tested negative for *Salmonella*.

New South Wales

There were 11 outbreaks of foodborne or suspected foodborne illness reported in New South Wales during this quarter. *S.* Typhimurium was the aetiological agent identified for 9 outbreaks. *Sta. aureus* was the aetiological agent identified in 1 outbreak, and for 1 outbreak the aetiological agent was unknown.

Description of key outbreaks

In October 2014, an outbreak of acute gastrointestinal illness was investigated in 27 members of a tour group: 11 showed symptoms of diarrhoea and vomiting (all without fever); and 4 were hospitalised. The group had flown to New South Wales from the Gold Coast in Queensland that morning and had consumed restaurant-prepared sushi rolls prior to their departure. Onset of symptoms began 4 hours after consumption and all cases developed vomiting within 10 to 20 minutes of each other. All symptoms ceased within 8.5 hours. Three stool samples tested positive for *Sta. aureus* enterotoxin. Environmental health officers from Queensland conducted an inspection of the restaurant and detected hygiene deficits. Hand washing was only performed with hand sanitiser and not with soap and water. Surfaces and utensils also showed inadequate cleaning and sanitising. Swabs of kitchen surfaces were positive for *Sta. aureus* and *Bacillus cereus* but boiled rice tested negative for the pathogens. Contaminated sushi from the restaurant was the likely source of illness for this outbreak.

In November, 35 of 75 people developed gastroenteritis 24 hours after consuming a meal at a sports club. The food was prepared by a catering company. Ten stool samples were positive for *S.* Typhimurium MLVA 03-12-11-14-523. All cases reported consuming lamb ragout. Health authorities inspected the caterers and found that leftovers at the venue, a sample of cooked pork, and a floor swab of the cool room were all positive for *S.* Typhimurium MLVA 03-12-11-14-523. It remains unclear how the food became contaminated as the cooking step of the dishes served should have been sufficient to kill any *Salmonella* present at that stage. The contamination is suspected to be due to post cooking cross-contamination of the batch served to this sports club. The same product served to other groups did not cause any illness.

Northern Territory

There were 4 outbreaks of suspected foodborne illness reported in the Northern Territory during this quarter. No aetiological agent was identified for any of these outbreaks.

Queensland

There were 15 outbreaks of foodborne or suspected foodborne illness reported in Queensland during this quarter. The aetiological agents identified were: ciguatoxin (6 outbreaks); *S.* Typhimurium (4 outbreaks); *S.* Chester (2 outbreaks); and *C. bifermentans* and *Sta aureus* (1 outbreak each). The remaining outbreak had an unknown aetiology.

Description of key outbreaks

Three separate outbreaks of *S.* Typhimurium PT U307 MLVA 03-12-11-12-524 were reported in December 2014 affecting at least 28 people (23 laboratory-confirmed). All 3 outbreaks occurred in restaurant settings (Brisbane and the Gold Coast). Traceback investigations identified that eggs supplied to the restaurants were obtained from the same egg producer. Following an investigation of the implicated egg farm, the same *Salmonella* strain (MLVA 03-12-11-12-524) found in the cases, was also identified in samples of used chicken feed and drag swabs. This strain was also detected in the implicated eggs sampled from a retail store. A voluntary trade level recall was subsequently undertaken by the egg producer.

Two outbreaks of *S.* Chester were investigated during the quarter that affected at least 9 people. In both outbreaks, the cases were from an African community and the consumption of lamb offal was reported. The first outbreak involved 3 cases who developed illness following a church function. One case was hospitalised as a result of their infection. The suspect meal was a stew made from lamb intestine, tripe, kidney and liver that was shared among attendees. In the 2nd outbreak, 6 cases (4 laboratory-confirmed) reportedly consumed a lamb intestine dish that had been prepared at a private residence for a function. Three cases were hospitalised. Poor food handling and temperature abuse were suspected to have contributed to both outbreaks. Following these outbreaks, health authorities contacted community leaders to provide food safety advice to the African community. OzFoodNet has only previously investigated 2 other *S.* Chester outbreaks, 1 in 2009 associated

with fresh chillies used to make chilli sauce, and no food vehicle was determined for the other outbreak in 2004.

South Australia

There were 7 outbreaks of foodborne or suspected foodborne illness reported in South Australia during this quarter. The aetiological agents identified were *S.* Typhimurium in 5 outbreaks and *Campylobacter* in 1 outbreak. The remaining outbreak had an unknown aetiology.

Description of key outbreaks

Four *Salmonella* outbreaks were linked to restaurants in Adelaide affecting a total of 36 people in October and December 2014. In 1 café outbreak reported in December, 11 cases were positive for *S.* Typhimurium PT 9 MLVA 03-14-06-12-550, including 4 hospitalisations. Environmental investigations of the food premises identified a number of food handling and preparation practices that could have contributed to the outbreak. A raw egg aioli made at the café tested positive for the same *Salmonella* strain identified in the cases. The investigation also reported that the egg brand used in this café was the same as that found in another café with a concurrent outbreak under investigation. In this outbreak, 7 cases of *S.* Typhimurium PT 9 MLVA 03-15-06-12-550 were identified in individuals who had eaten at the second café over a 4-day period. No common food item was consumed by all cases at the café. The MLVA profile of cases from both outbreaks was closely related with only one repeat difference at the third locus. Food samples taken from the second café were negative for *Salmonella*. An inspection at the egg processing plant demonstrated appropriate food safety practices. Advice was provided to the staff and owner of the second café on the production, storage and handling of raw egg products.

An outbreak of campylobacteriosis was investigated after 3 children who ate at a hotel on the same evening developed gastroenteritis. A case control study was conducted by contacting individuals on the booking list from the hotel. Twenty-two people reported having diarrhoea after the event and 5 tested positive for *Campylobacter*. A multivariate analysis indicated consumption of freshly cooked prawns was significantly associated with diarrhoeal illness (adjusted odds ratio (aOR) 8.5, 95% confidence interval (CI) 2.6–27.9, $P < 0.005$). An additional case of campylobacteriosis was reported from an individual who ate at the hotel the following night. An environmental inspection was conducted and no issues were identified at the premises. A trace-back indicated that the batch of prawns used at the hotel was distributed to other food businesses, but there were no reports of cases associated with any other premises. No sporadic cases of campylobacteriosis interviewed concurrently with the outbreak reported consuming prawns.

Tasmania

One outbreak of suspected foodborne illness was reported in Tasmania during this quarter. Norovirus was identified as the aetiological agent.

Description of key outbreak

An outbreak was investigated in November following reports of illnesses among a group of interstate visitors. Nine cases of gastroenteritis were identified among the 18 persons who could be contacted. Six people could not be contacted. The majority of the cases were female (78%) with a median age of 73 years. Diarrhoea and vomiting were experienced by 89% of the cases. Other symptoms reported included: lethargy (85%); fever (75%); nausea (63%); abdominal pain (57%); and headaches (33%). Onsets occurred between 8 and 10 November 2014. The median duration of illness was 36 hours. Three specimens were collected, with 2 being positive for norovirus. Four cases were admitted to hospital. A cohort study was conducted as part of the investigation. Fruit salad eaten at a dinner held on 7 November was the only statistically significant food item in the analysis (risk ratio (RR) 3.3, CI 1.2–9.5, $P < 0.005$). However, 3 cases did not report eating this food item. This finding appeared unlikely to explain all illnesses, and may have been a chance finding among many exposures in a relatively small cohort. No other exposure had a statistically significant positive association with illness. Inconspicuous environmental contamination at one or more sites to which the group was exposed early in the tour remains a possible source of the outbreak. The environmental investigation identified that the rain water supply at the business where guests stayed for the majority of their tour was insufficiently managed and operating in contravention of the Private Water Supply provisions of the *Public Health Act* 1997. Immediate guidance was provided to the business on how to manage and treat the tank water supply to ensure potability. This matter was managed separately to the outbreak investigation by the local council environmental health officer. The result of the epidemiological study showed no association between the consumption of this water and the development of illness. No definitive source of illness was identified for the outbreak.

Victoria

There were 11 outbreaks of foodborne or suspected foodborne illness reported in Victoria during this

quarter. The aetiological agents were identified as: norovirus (5 outbreaks); *S.* Typhimurium (2 outbreaks); and *S.* Singapore, *C. perfringens*, and *Cryptosporidium* (1 outbreak each). The aetiology for the remaining outbreak was unknown.

Description of key outbreaks

Two cases of haemolytic uraemic syndrome (HUS) were reported by 2 metropolitan hospitals on the same day in late October. The cases were both young children who lived in neighbouring suburbs. Investigation of both cases revealed that they had consumed the same brand of 'bath milk', which is unpasteurised cow's milk, not intended for human consumption. A 3rd case of HUS, notified earlier in the year had also reported consuming the same brand of unpasteurised milk. One of the HUS cases was also confirmed with cryptosporidiosis, so an investigation of recently notified cases of cryptosporidiosis living in the same geographical location as the HUS cases commenced. Twelve cases of cryptosporidiosis were identified for follow up and 11 were interviewed. Two of these cases reported consuming the same brand of unpasteurised milk as the HUS cases in their incubation period. Onset dates for these 2 cases of cryptosporidiosis and the 2 recent HUS cases were within 10 days of each other. This outbreak investigation led to the introduction of tighter controls around the sale of unpasteurised milk sold for cosmetic purposes in Victoria, as detailed in the comments section below.

Five people from 2 separate groups who had eaten food from the same café in October were reported to be ill. The groups had eaten 1 day apart and both groups reported consumption of fried chicken and beef wraps. An investigation commenced in conjunction with the local government health department. The initial cases were subsequently confirmed with *S.* Singapore. No booking list was kept at the café so case finding was limited to self-reported illness to the council or the department and detection of cases of *S.* Singapore in the notifiable diseases surveillance system. A total of 15 cases of illness were identified in people who consumed food from the café on 1 of 5 consecutive days in October. Eight of the 15 cases were confirmed with *S.* Singapore. Fourteen cases consumed a beef wrap and for 3 of the cases, it was the only food item consumed from the café during the outbreak period. Despite a thorough investigation of the preparation method and source of ingredients for the beef wraps, it was not possible to identify the exact means by which they became contaminated. However, the council identified several cleanliness issues during their investigation and a possible explanation was that a raw ingredient used in the wraps, such as a fresh herb, was cross contaminated during preparation.

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 norovirus in 2 outbreaks and *Salmonella* Typhimurium in 1 outbreak. The aetiology for the remaining outbreak was unknown.

Description of key outbreaks

In December 2014, gastroenteritis was reported among attendees of a graduation dinner at a restaurant. Interviews were conducted with 40 of 48 attendees and 21/40 reported diarrhoea and/or vomiting after the event. The median incubation period was 36 hours (range 12 to 53 hours) and the median duration of illness was 2 days (range 1 to 5 days). One faecal specimen was positive for norovirus. Food was served as a buffet, which included hot pasta dishes, salads, roast potatoes and roast beef, and several desserts. Consumption of salads was statistically associated with illness (odds ratio (OR) 7.35, CI 1.3–42.4, $P=0.03$). There were no reports of staff illness prior to the meal. The evidence suggests the outbreak was due to salad(s) contaminated with norovirus, but the source of the norovirus was unknown.

Cases of gastroenteritis were reported following a function at a bowling club in December 2014. Nineteen of the 60 attendees were interviewed, with 13 reporting illness. Symptoms reported included diarrhoea (100%), abdominal pain (92%) and vomiting (8%) with a median incubation period of 12 hours and median duration of diarrhoea of 11 hours. No faecal specimens were tested, but the characteristics were suggestive of *C. perfringens* food poisoning. Food had been prepared by a caterer and included roast meats salads and several desserts. The result of the analytical study found a statistical association between eating roast meats and becoming ill (OR not defined, CI 1.65–not defined, $P = 0.015$. The OR and upper CI were undefined because all the cases ate the roast meats. The local government conducted the environmental health investigation of the food business and at the time of the investigation, the food business could not provide evidence of safe food handling, processing and hygiene.

Comments

Whilst the sale of unpasteurised (raw) cow's milk for human consumption is illegal in all states and territories of Australia, a niche market existed in 2014 selling raw milk for cosmetic use, often labelled as 'bath milk'. Raw milk can contain a number of disease-causing pathogens, including *Campylobacter* and *Salmonella* species, *Listeria monocytogenes,* Shiga toxin-producing *Escherichia*

coli, *Cryptosporidium*, and *Sta. aureus*.[1] Following the outbreak reported above, in February 2015, Dairy Food Safety Victoria (DFSV) introduced a new licence condition for the Victorian dairy industry, stating that 'any licence holders who intend to sell, deliver or supply milk or milk products not intended for human consumption must advise DFSV and obtain approval of the proposed treatment of these products to ensure they are clearly differentiated from dairy food.[2]

Cluster investigations

OzFoodNet sites conducted investigations into 11 clusters of infection during this quarter. No common food vehicle or source of infection could be identified. Aetiological agents identified during the investigations were: *S.* Typhimurium (7 clusters); *S.* Bovismorbificans, *S.* Saintpaul, *Clostridium perfringens* (1 cluster each); and a cluster with multiple aetiological agents detected (norovirus, astrovirus, sapovirus, and *Campylobacter*).

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References

1. Langer A, Ayers T, Grass J, Lynch M, Angulo F, Mahon B. Nonpasteurized Dairy Products, Disease Outbreaks, and State Laws—United States, 1993–2006. *Emerg Infect Dis* 2012;18(3):385–391.
2. Dairy Food Safety Victoria (DFSV). (2015). *Annual Report 2014–2015*. Available from https://www.dairysafe.vic.gov.au/publications-media/annual-reports

Appendix: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites, 1 October to 31 December 2014 (n=54)

State or territory	Month*	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
ACT	Dec	Private residence	*Salmonella* Typhimurium PT 135, MLVA 03-17-09-11-523	3	1	D	Eggnog
NSW	Oct	Restaurant	*S.* Typhimurium MLVA 03-12-12-09-523	13	0	D	Unknown
NSW	Oct	Restaurant	*Staphylococcus aureus*	11	4	M	Sushi
NSW	Oct	Fair/festival/mobile service	*S.* Typhimurium MLVA 03-12-12-09-523	4	1	D	Unknown
NSW	Oct	Restaurant	*S.* Typhimurium MLVA 03-09-07-12-523	4	0	D	Burger
NSW	Oct	Takeaway	*S.* Typhimurium MLVA 03-10-07-11-523	26	0	D	Chocolate mousse cake made with lightly cooked eggs
NSW	Oct	Aged care facility	Unknown	8	0	D	Unknown
NSW	Oct	Restaurant	*S.* Typhimurium MLVA 03-12-11-14/15-523	38	6	AM	Unknown
NSW	Nov	Restaurant	*S.* Typhimurium MLVA 03-12-11-14-523	35	0	M	Pre-prepared meals
NSW	Nov	Other	*S.* Typhimurium MLVA 03-09/10-08-12-523	16	4	M	Unknown
NSW	Dec	Restaurant	*S.* Typhimurium MLVA 03-17-09-11-523	19	4	M	Unknown
NSW	Dec	Restaurant	*S.* Typhimurium MLVA 03-09-08-11-523	4	1	D	Unknown
NT	Oct	Primary produce	Suspected bacterial toxin	2	0	D	Unknown
NT	Nov	Restaurant	Unknown	4	0	D	Unknown
NT	Nov	Restaurant	Suspected viral gastroenteritis	3	0	D	Unknown
NT	Dec	Restaurant	Suspected bacterial toxin	2	0	D	Unknown
Qld	Oct	Primary produce	Ciguatoxin	3	0	M	Coronation trout
Qld	Oct	Primary produce	Ciguatoxin	3	0	M	Coral trout
Qld	Oct	Primary produce	Ciguatoxin	4	0	M	Coral trout
Qld	Oct	Primary produce	Ciguatoxin	4	0	D	Coral trout
Qld	Oct	Private residence	*S.* Chester	3	1	D	Offal (lamb intestine)
Qld	Oct	Restaurant	*S.* Typhimurium MLVA 03-12-12-09-524	11	2	D	Unknown
Qld	Oct	Bakery	Unknown	13	0	D	Birthday cake
Qld	Nov	Private residence	*Staphylococcus aureus*	7	0	M	Taro cake
Qld	Nov	Primary produce	Ciguatoxin	2	0	M	Coral trout
Qld	Nov	Restaurant	*Clostridium bifermentans*	28	0	M	Butter chicken
Qld	Nov	Private residence	*S.* Chester	6	3	D	Offal (lamb intestine)
Qld	Nov	Restaurant	*S.* Typhimurium PT U307, MLVA 03-12-11-12-524	12	Unknown	D	Chocolate mousse
Qld	Dec	Restaurant	*S.* Typhimurium PT U307, MLVA 03-12-11-12-524	10	Unknown	M	Deep fried ice cream
Qld	Dec	Primary produce	Ciguatoxin	5	0	D	Coral trout
Qld	Dec	Restaurant	*S.* Typhimurium PT U307, MLVA 03-12-11-12-524	6	0	D	Chocolate mousse
SA	Oct	Restaurant	*Campylobacter*	22	2	A	Prawns
SA	Oct	Restaurant	Unknown	25	0	D	Unknown
SA	Oct	Restaurant	*S.* Typhimurium PT 9	10	2	D	Unknown
SA	Dec	Restaurant	*S.* Typhimurium PT 9, MLVA 03-14-06-12-550	11	4	M	Raw egg aioli
SA	Dec	Private residence	*S.* Typhimurium PT 9, MLVA 03-24-13-10-523	14	3	A	Raw egg tiramisu
SA	Dec	Restaurant	*S.* Typhimurium PT 9, MLVA 03-15-06-12-550	7	0	D	Unknown
SA	Dec	Restaurant	*S.* Typhimurium PT 9, MLVA 03-24-13-10-523	8	1	D	Unknown

Appendix *continued*: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites, 1 October to 31 December 2014 (n=54)

State or territory	Month*	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
Tas.	Nov	Restaurant	Norovirus	9	4	A	Suspected fruit salad
Vic.	Oct	Community	Cryptosporidium	5	3	M	Suspected unpasteurised milk
Vic.	Oct	Restaurant	*S.* Singapore	15	3	D	Beef wraps
Vic.	Oct	Restaurant	Suspected bacterial toxin	13	0	D	Suspected rice and/or beans
Vic.	Nov	Aged care	*Clostridium perfringens*	5	0	D	Unknown
Vic.	Nov	Restaurant	*S.* Typhimurium PT 170/108, MLVA 03-09-10-15-524/525	19	3	M	Mixed foods including rice paper rolls
Vic.	Nov	Commercial caterer	Norovirus	20	2	A	Brownies and /or fruit salad
Vic.	Nov	Commercial caterer	Norovirus	53	1	A	Thai beef salad
Vic.	Dec	School/private residence	*S.* Typhimurium PT 44, MLVA 03-10-09-09-524	10	0	A	Beef appetiser or frittata
Vic.	Dec	Commercial caterer	Norovirus	19	1	A	Lamb, lettuce and tomato
Vic.	Dec	Restaurant	Norovirus	14	0	A	Potato salad/food handler contamination
Vic.	Dec	Restaurant	Norovirus	26	0	D	Unknown
WA	Oct	Restaurant	*S.* Typhimurium PT 9, PFGE0001	4	1	D	Slow cooked pork hock
WA	Dec	Commercial caterer	Suspected bacterial toxin	13	0	A	Roast meats (turkey, pork, beef)
WA	Dec	Restaurant	Norovirus	21	0	A	Leafy salad (green salad or prawn salad)
WA	Dec	Camp	Norovirus	30	2	D	Unknown, possibly multiple foods
Total				682	59		

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. The number of people affected does not necessarily equal the number of laboratory-confirmed cases.

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

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

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

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

PT Phage type

MLVA Multi-locus variable number tandem repeat analysis profile

PFGE Pulsed-field gel electrophoresis type

National Notifiable Diseases Surveillance System, 1 October to 31 December 2016

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 69,209 notifications to the National Notifiable Diseases Surveillance System (NNDSS) between 1 October and 31 December 2016 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

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

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

Disease	Data received from:
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease – invasive	All jurisdictions
Poliovirus infection	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Varicella zoster (chickenpox)	All jurisdictions except New South Wales
Varicella zoster (shingles)	All jurisdictions except New South Wales
Varicella zoster (unspecified)	All jurisdictions except New South Wales
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Chikungunya virus infection	All jurisdictions except Australian Capital Territory
Dengue virus infection	All jurisdictions
Flavivirus infection (unspecified)	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
West Nile/Kunjin 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 October to 31 December 2016, by date of diagnosis*

Disease	State or territory								Total 4th quarter 2016	Total 3rd quarter 2016	Total 4th quarter 2015	Last 5 years mean 4th 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)†	0	5	0	10	2	0	13	6	36	26	27	41.8	0.9	151	173.6
Hepatitis B (unspecified)‡	21	533	23	263	57	9	380	148	1,434	1,561	1,531	1,567.4	0.9	6,466	6,461.8
Hepatitis C (newly acquired)†	3	2	1	98	8	7	25	28	172	149	127	107.6	1.6	698	443.8
Hepatitis C (unspecified)‡	27	1,121	54	569	79	69	555	276	2,750	2,600	2,757	2,519.6	1.1	11,278	10,044.8
Hepatitis D	0	5	0	4	2	0	3	0	14	14	5	11.0	1.3	58	48.4
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	1	0.2	0	0	2.0
Campylobacteriosis	168	NN	85	2,037	1,169	223	2,463	945	7,090	5,947	6,585	5,015.6	1.4	24,242	18,118.2
Cryptosporidiosis	12	358	23	295	35	8	178	33	942	645	1,108	600.6	1.6	5,412	3,055.4
Haemolytic uraemic syndrome	0	2	0	0	0	1	0	2	5	5	4	4.4	1.1	15	17.4
Hepatitis A	0	13	0	8	3	0	12	4	40	23	34	41.4	1	142	182.0
Hepatitis E	1	4	0	2	1	0	5	0	13	8	17	11.8	1.1	41	41.2
Listeriosis	0	6	0	4	2	0	9	0	21	13	22	21.4	1	84	77.8
Paratyphoid	2	4	1	2	1	0	8	0	18	13	24	19.4	0.9	76	72.6
STEC§	0	34	2	4	77	2	12	13	144	91	51	34.2	4.2	337	127.8
Salmonellosis	50	954	156	1,075	304	51	882	502	3,974	3,120	3,906	3,429.4	1.2	18,197	13,877.0
Shigellosis	1	78	39	71	7	4	140	21	361	324	216	185.6	1.9	1,398	730.0
Typhoid fever	2	8	0	3	3	0	6	3	25	21	24	30.6	0.8	104	127.8
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	1	0	1	0	1	0.6	1.7	1	3.6
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	0.4

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

Disease	State or territory								Total 4th quarter 2016	Total 3rd quarter 2016	Total 4th quarter 2015	Last 5 years mean 4th quarter	Ratio	Year to date 2016	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA							
Sexually transmissible infections															
Chlamydial infection[‖,¶]	296	6,374	657	5,223	1,304	324	700	2,727	17,605	17,916	16,647	19,432.6	0.9	74,105	81,419.6
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.4
Gonococcal infection[¶]	56	1,791	430	1,141	235	25	1,067	895	5,640	5,634	4,703	3,766.2	1.5	23,045	15,030.0
Syphilis <2 years duration[¶]	2	174	76	147	23	3	231	75	731	831	712	476.4	1.5	3,188	1,874.4
Syphilis >2 years or unspecified duration[‡,¶]	4	93	15	69	29	9	204	24	447	480	473	400.6	1.1	1,882	1,639.2
Syphilis – congenital	0	0	0	0	0	0	0	0	0	2	0	0.6	0	2	4.0
Vaccine preventable diseases															
Diphtheria	0	0	0	3	0	0	0	0	3	1	0	0.4	7.5	8	2.2
Haemophilus influenzae type b	0	1	1	2	0	0	0	0	4	5	2	3.4	1.2	18	17.2
Influenza (laboratory confirmed)	122	3,699	218	4,078	3,097	194	2,654	999	15,061	62,513	6,691	5,077.0	3	90,868	53,675.8
Measles	2	8	0	8	2	0	2	7	29	14	9	42.4	0.7	99	192.8
Mumps	0	23	41	19	8	0	5	13	109	158	287	89.2	1.2	800	280.4
Pertussis	186	2,870	114	481	778	3	710	385	5,527	4,702	8,634	6,493.4	0.9	20,037	21,925.4
Pneumococcal disease – invasive	5	120	16	56	37	15	99	47	395	637	314	349.2	1.1	1,664	1,663.0
Poliovirus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Rubella	0	1	0	0	0	0	0	1	2	3	5	5.8	0.3	17	30.6
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0	0.8
Tetanus	0	0	0	1	0	0	0	0	1	2	1	1.4	0.7	7	3.8
Varicella zoster (chickenpox)	35	NN	33	79	128	14	37	193	519	657	719	631.6	0.8	2,369	2,159.2
Varicella zoster (shingles)	66	NN	86	24	589	84	33	468	1,350	1,499	1,654	1,351.0	1	6,388	5,080.2
Varicella zoster (unspecified)	47	NN	0	1,849	113	33	5	400	2,447	3,480	3,640	2,963.2	0.8	13,500	10,880.2
Vectorborne diseases															
Barmah Forest virus infection	0	7	4	50	2	0	0	4	67	44	89	330.4	0.2	322	1,840.0
Chikungunya virus infection	0	17	0	2	2	0	13	10	44	34	11	18.6	2.4	111	82.4
Dengue virus infection	4	100	14	50	19	4	45	69	305	414	292	266.4	1.1	2,181	1,527.6
Flavivirus infection (unspecified)	1	7	0	5	0	0	0	1	14	29	3	1.6	8.8	115	12.8
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	1	0.2	0	0	1.8
Malaria	2	17	4	15	0	2	24	9	73	82	59	83.4	0.9	301	347.2
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0	3.8
Ross River virus infection	6	261	29	170	86	2	280	204	1,038	263	1,063	999.8	1	3,550	5,800.4
West Nile/Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	1	0.8	0	0	1.2

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

Disease	State or territory								Total 4th quarter 2016	Total 3rd quarter 2016	Total 4th quarter 2015	Last 5 years mean 4th 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	0.0	0	0	0.2
Brucellosis	0	1	1	2	0	0	0	2	6	6	2	5.4	1.1	18	23.6
Leptospirosis	0	2	1	10	1	1	6	4	25	18	15	15.6	1.6	136	114.8
Lyssavirus infection (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0	0.0
Ornithosis	0	8	0	0	0	0	2	0	10	6	9	19.4	0.5	19	53.8
Q fever	0	59	0	59	11	0	8	3	140	128	136	111.6	1.3	537	459.0
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.2	0	0	0.4
Other bacterial infections															
Legionellosis	0	32	0	11	9	1	21	23	97	90	86	106.6	0.9	372	407.4
Leprosy	0	3	0	1	0	0	0	2	6	7	4	3.0	2	21	10.8
Meningococcal infection – invasive**	0	15	1	15	7	1	35	10	84	79	41	42.2	2	254	193.2
Tuberculosis	3	160	4	55	21	3	102	42	390	367	353	362.6	1.1	1,376	1,312.2
Total	1,124	19,345	2,147	18,368	8,340	1,095	10,975	8,661	69,209	114,661	63,096			316,010	

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

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

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

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

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

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

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

NN Not notifiable

NEC Not elsewhere classified

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

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

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Bloodborne diseases									
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0
Hepatitis B (newly acquired)‡	0	0.3	0	0.8	0.5	0	0.9	0.9	0.6
Hepatitis B (unspecified)§	21.5	28	37.6	22	13.4	7	25.6	22.9	24.1
Hepatitis C (newly acquired)‡	3.1	0.1	1.6	8.2	1.9	5.4	1.7	4.3	2.9
Hepatitis C (unspecified)§	27.6	58.8	88.3	47.6	18.6	53.4	37.4	42.6	46.2
Hepatitis D	0	0.3	0	0.3	0.5	0	0.2	0	0.2
Gastrointestinal diseases									
Botulism	0	0	0	0	0	0	0	0	0
Campylobacteriosis	171.9	NN	139.1	170.4	275.2	172.6	165.8	145.9	175.4
Cryptosporidiosis	12.3	18.8	37.6	24.7	8.2	6.2	12	5.1	15.8
Haemolytic uraemic syndrome	0	0.1	0	0	0	0.8	0	0.3	0.1
Hepatitis A	0	0.7	0	0.7	0.7	0	0.8	0.6	0.7
Hepatitis E	1	0.2	0	0.2	0.2	0	0.3	0	0.2
Listeriosis	0	0.3	0	0.3	0.5	0	0.6	0	0.4
Paratyphoid	2	0.2	1.6	0.2	0.2	0	0.5	0	0.3
STEC‖	0	1.8	3.3	0.3	18.1	1.5	0.8	2	2.4
Salmonellosis	51.1	50.1	255.2	89.9	71.6	39.5	59.4	77.5	66.8
Shigellosis	1	4.1	63.8	5.9	1.6	3.1	9.4	3.2	6.1
Typhoid fever	2	0.4	0	0.3	0.7	0	0.4	0.5	0.4
Quarantinable diseases									
Avian influenza in humans	0	0	0	0	0	0	0	0	0
Cholera	0	0	0	0	0	0	0.1	0	0
Middle East respiratory syndrome coronavirus	0	0	0	0	0	0	0	0	0
Plague	0	0	0	0	0	0	0	0	0
Rabies	0	0	0	0	0	0	0	0	0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0
Smallpox	0	0	0	0	0	0	0	0	0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0
Yellow fever	0	0	0	0	0	0	0	0	0
Sexually transmitted infections									
Chlamydial infection¶,**	302.8	334.6	1074.8	437	307	250.8	47.1	421.1	296.1
Donovanosis	0	0	0	0	0	0	0	0	0
Gonococcal infection**	57.3	94	703.5	95.5	55.3	19.4	71.8	138.2	94.8
Syphilis < 2 years duration**	2	9.1	124.3	12.3	5.4	2.3	15.6	11.6	12.3
Syphilis > 2 years or unspecified duration§,**	4.1	4.9	24.5	5.8	6.8	7	13.7	3.7	7.5
Syphilis – congenital	0	0	0	0	0	0	0	0	0
Vaccine preventable diseases									
Diphtheria	0	0	0	0.3	0	0	0	0	0.1
Haemophilus influenzae type b	0	0.1	1.6	0.2	0	0	0	0	0.1
Influenza (laboratory confirmed)	124.8	194.2	356.6	341.2	729.1	150.2	178.7	154.3	253.3
Measles	2	0.4	0	0.7	0.5	0	0.1	1.1	0.5
Mumps	0	1.2	67.1	1.6	1.9	0	0.3	2	1.8
Pertussis	190.3	150.7	186.5	40.2	183.2	2.3	47.8	59.4	92.9
Pneumococcal disease – invasive	5.1	6.3	26.2	4.7	8.7	11.6	6.7	7.3	6.6
Poliovirus infection	0	0	0	0	0	0	0	0	0

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

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Vaccine preventable diseases, cont'd									
Rubella	0	0.1	0	0	0	0	0	0.2	0
Rubella – congenital	0	0	0	0	0	0	0	0	0
Tetanus	0	0	0	0.1	0	0	0	0	0
Varicella zoster (chickenpox)	35.8	NN	54	6.6	30.1	10.8	2.5	29.8	12.8
Varicella zoster (shingles)	67.5	NN	140.7	2	138.7	65	2.2	72.3	33.4
Varicella zoster (unspecified)	48.1	NN	0	154.7	26.6	25.5	0.3	61.8	60.5
Vectorborne diseases									
Barmah Forest virus infection	0	0.4	6.5	4.2	0.5	0	0	0.6	1.1
Chikungunya virus infection	0	0.9	0	0.2	0.5	0	0.9	1.5	0.7
Dengue virus infection	4.1	5.2	22.9	4.2	4.5	3.1	3	10.7	5.1
Flavivirus infection (unspecified)	1	0.4	0	0.4	0	0	0	0.2	0.2
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0
Malaria	2	0.9	6.5	1.3	0	1.5	1.6	1.4	1.2
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0
Ross River virus infection	6.1	13.7	47.4	14.2	20.2	1.5	18.9	31.5	17.5
West Nile/Kunjin virus infection	0	0	0	0	0	0	0	0	0
Zoonoses									
Anthrax	0	0	0	0	0	0	0	0	0
Australia bat lyssavirus infection	0	0	0	0	0	0	0	0	0
Brucellosis	0	0.1	1.6	0.2	0	0	0	0.3	0.1
Leptospirosis	0	0.1	1.6	0.8	0.2	0.8	0.4	0.6	0.4
Lyssavirus infection (NEC)	0	0	0	0	0	0	0	0	0
Ornithosis	0	0.4	0	0	0	0	0.1	0	0.2
Q fever	0	3.1	0	4.9	2.6	0	0.5	0.5	2.4
Tularaemia	0	0	0	0	0	0	0	0	0
Other bacterial diseases									
Legionellosis	0	1.7	0	0.9	2.1	0.8	1.4	3.6	1.6
Leprosy	0	0.2	0	0.1	0	0	0	0.3	0.1
Meningococcal infection – invasive††	0	0.8	1.6	1.3	1.6	0.8	2.4	1.5	1.4
Tuberculosis	3.1	8.4	6.5	4.6	4.9	2.3	6.9	6.5	6.6

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

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

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

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

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

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

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

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

NEC Not elsewhere classified.

NN Not notifiable.

Australian childhood immunisation coverage, 1 July 2015 and 30 June 2016 cohort, assessed as at 30 September 2016

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 Australian Immunisation Register (AIR) 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 AIR.

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

'Fully immunised' at 12 months of age is defined as a child having a record on the AIR 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 AIR 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 AIR of 4 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

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

Results

The rolling annualised percentage of all children 'fully immunised' by 12 months of age for Australia increased marginally from the previous report by 0.2 of a percentage point to 93.2% (Table 1). All states and territories, except Tasmania, experienced small increases in the percentage of children 'fully immunised' by 12 months of age. Coverage for 'fully immunised' by 12 months of age is now greater than 93% in all jurisdictions, apart from Western Australia. Coverage for individual vaccines due by 12 months of age also remained greater than 93% in all jurisdictions.

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

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Total number of children	5,680	97,874	3,807	61,898	19,800	5,737	76,915	34,238	305,949
Diphtheria, tetanus, pertussis (%)	95.7	94.1	93.8	94.1	93.8	93.8	94.3	93.6	94.1
Poliomyelitis (%)	95.6	94.1	93.7	94.1	93.8	93.8	94.2	93.6	94.1
Haemophilus influenzae type b (%)	95.2	93.8	93.6	93.9	93.5	93.6	93.8	93.3	93.8
Hepatitis B (%)	95.6	94.0	94.0	94.2	93.7	93.9	94.1	93.3	94.0
Pneumococcal	95.5	93.7	93.7	93.9	93.5	93.7	93.8	93.2	93.7
Fully immunised (%)	94.9	93.2	93.2	93.5	93.1	93.3	93.2	92.7	93.2

The rolling annualised percentage of all children 'fully immunised' by 24 months of age for Australia increased by 0.4 percentage points to reach 91.1% (Table 2). Coverage for individual vaccines due by 24 months of age is now above 94.5% in all jurisdictions, except for the measles, mumps and rubella vaccine and varicella vaccine. Coverage for these antigens at 24 months of age have however continued to improve from the previous report with measles, mumps and rubella coverage increasing by 0.3 of a percentage point to 92.6% and varicella coverage also increasing by 0.3 of a percentage point to 92.7%.

The rolling annualised percentage of all children 'fully immunised' by 60 months of age for Australia increased marginally from the previous report by 0.2 of a percentage point to 93.1% (Table 3). Coverage for individual vaccines due by 60 months of age remained greater than 92% in all jurisdictions.

The Figure shows the trends in vaccination coverage from the first published coverage estimates in 1997 to the current AIR estimates. Overall there is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007).

Coverage by 24 months did fall below the 12 and 60 month coverage estimates following the change in the 24 month coverage assessment algorithm to include MMR dose 2 instead of MMR dose 1, varicella dose 1, and meningococcal C dose 1. However, 'fully immunised' coverage by 24 months has been steadily increasing since this change and as at 30 June 2016 reached 91.9%, which was an increase of 0.6 of a percentage point from the previous quarterly report. A similar increase has been seen in the 'fully immunised' coverage by 12 months of age, reaching 93.9% and the 'fully immunised' coverage by 60 months increased by 0.4 of a percentage point to 93.4%.

Disclaimer

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

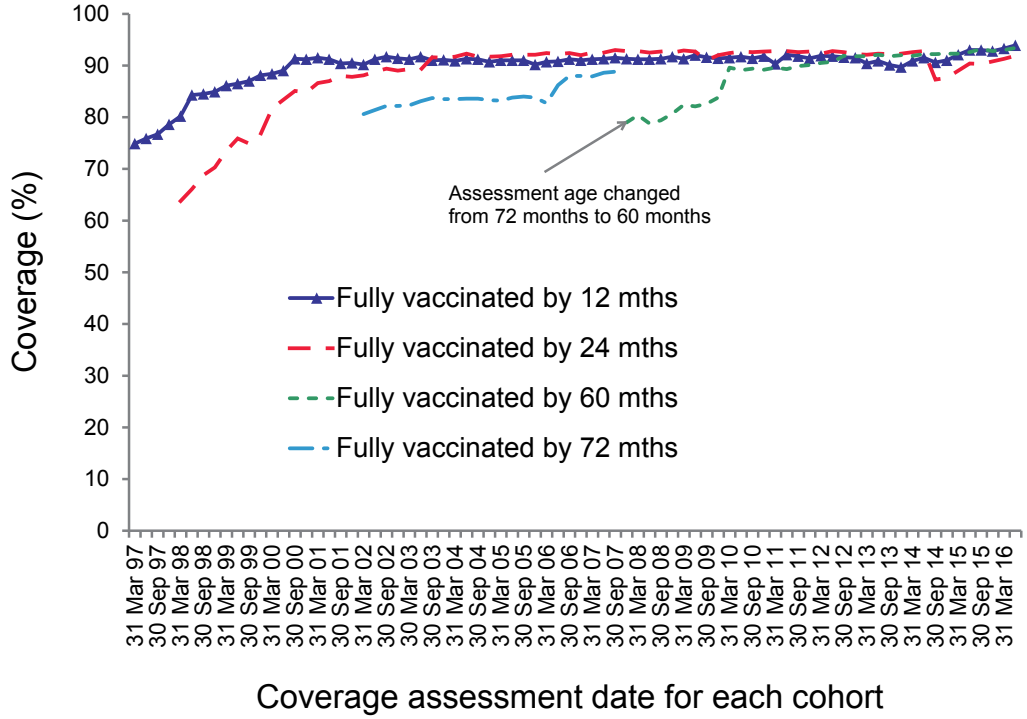
Table 2. Percentage of children immunised at 24 months of age for the birth cohort 1 July 2013 to 30 June 2014, preliminary results, by disease and state or territory; assessment date 30 September 2016

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	5,697	99,525	3,566	62,419	19,993	5,923	77,228	34,259	308,610
Diphtheria, tetanus, pertussis (%)	97.1	95.7	96.1	95.8	96.0	96.3	96.3	95.9	96.0
Poliomyelitis (%)	97.1	95.7	96.0	95.8	95.9	96.4	96.2	95.9	95.9
Haemophilus influenzae type b (%)	95.9	94.7	95.1	95.2	95.0	95.4	95.4	94.9	95.1
Measles, mumps, rubella (%)	93.6	92.4	91.7	93.1	92.9	93.6	92.9	91.6	92.6
Hepatitis B (%)	96.9	95.5	96.3	95.7	95.7	96.3	96.1	95.6	95.8
Meningococcal C (%)	95.6	94.6	94.9	95.1	95.0	95.4	95.1	94.5	94.9
Varicella (%)	93.6	92.5	91.0	92.9	92.8	93.2	93.1	91.5	92.7
Fully immunised (%)	91.9	90.6	89.4	91.9	91.2	91.4	91.4	90.1	91.1

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

Vaccine	State or territory								Aust.
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	5,577	101,428	3,552	65,148	20,148	6,269	76,256	34,587	312,965
Diphtheria, tetanus, pertussis (%)	94.0	94.0	93.1	93.6	93.3	94.7	94.2	92.2	93.7
Poliomyelitis (%)	94.1	94.1	93.2	93.6	93.3	94.7	94.2	92.2	93.8
Measles, mumps, rubella (%)	94.3	94.1	93.4	93.6	93.4	94.7	94.2	92.5	93.8
Fully immunised (%)	93.4	93.5	92.3	92.9	92.7	94.0	93.5	91.5	93.1

Figure: Trends in vaccination coverage, Australia, 1997 to 30 June 2016, by age cohorts



Australian Gonococcal Surveillance Programme, 1 July to 30 September 2016

Monica M Lahra, Rodney P Enriquez, The Prince of Wales Hospital, Randwick, for The National Neisseria Network

Introduction

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

Results

A summary of the proportion of isolates with decreased susceptibility to ceftriaxone, and the proportion resistant to azithromycin, penicillin, and ciprofloxacin for the 3rd quarter of 2016 are shown in Table 1.

Ceftriaxone MIC values in the range 0.06–0.125 mg/L have been reported by the AGSP in the category decreased susceptibility since 2005. There was no isolate with a ceftriaxone MIC value greater than 0.125 mg/L in this quarter. A summary of the proportion of isolates with decreased susceptibility to ceftriaxone for 2011 to 2015, and the first 3 quarters of 2016 is shown in Table 2.

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

State or territory	Number of isolates tested	Decreased susceptibility		Resistance					
		Ceftriaxone MIC ≥0.06–0.125 mg/L		Azithromycin MIC ≥1.0 mg/L		Penicillin* MIC ≥1.0 mg/L		Ciprofloxacin MIC ≥1.0 mg/L	
		n	%	n	%	n	%	n	%
Australian Capital Territory	32	0	0.0	1	3.1	4	12.5	3	9.4
New South Wales	532	23	4.3	32	6.0	272	51.1	172	32.3
Queensland	235	8	3.4	4	1.7	59	25.1	36	55.4
South Australia	65	0	0.0	5	7.7	28	43.1	36	55.4
Tasmania	5	0	0.0	1	20.0	2	40.0	2	40.0
Victoria	441	5	1.1	29	6.6	204	46.3	175	39.7
Northern Territory Urban & Rural	22	0	0.0	0	0.0	1	4.5	5	22.7
Northern Territory Remote	34	0	0.0	0	0.0	0	0.0	0	0.0
Western Australia Urban & Rural	168	3	1.8	19	11.3	26	15.5	38	22.6
Western Australia Remote	39	0	0.0	0	0.0	3	7.7	3	7.7
Australia	1,573	39	2.5	91	5.8	599	38.1	491	31.2

* Penicillin resistance includes MIC value of ≥1.0 mg/L, or penicillinase production.

Table 2: Percentage of gonococcal isolates with decreased susceptibility to ceftriaxone MIC 0.06-0.125 mg/L, Australia, 2011 to 30 September 2016

Ceftriaxone MIC mg/L	2011	2012	2013	2014	2015	2016 Q1	2016 Q2	2016 Q3
0.06	3.2%	4.1%	8.2%	4.8%	1.7%	1.5%	3.4%	2.2%
0.125	0.1%	0.3%	0.6%	0.6%	0.1%	0.0%	0.1%	0.3%

Ceftriaxone

In the 3rd quarter of 2016, the jurisdictions that reported isolates with decreased susceptibility to ceftriaxone were New South Wales, Queensland, Victoria, and urban/rural Western Australia.

Those *Neisseria gonorrhoeae* isolates that have decreased susceptibility to ceftriaxone and are penicillin and ciprofloxacin resistant are referred to as multidrug-resistant (MDR) for the purposes of the AGSP. From New South Wales there were 23/532 strains with decreased susceptibility to ceftriaxone and of those 10 (44%) were MDR, 17/23 (74%) were from males and 8 (35%) were isolated from extragenital sites (rectal and pharyngeal). From Queensland, there were 8/235 strains with decreased susceptibility to ceftriaxone and of those 8 (100%) were MDR, 7 (88%) were from males and 2 (25%) were from extragenital sites. From Victoria, there were 5/441 strains with decreased susceptibility to ceftriaxone and of those all were MDR, all were from males and none were isolated from extragenital sites. From urban/rural Western Australia there were 3/168 strains with decreased susceptibility to ceftriaxone and of those 2 (67%) were MDR, all were from males and 2 (67%) were isolated from an extragenital site.

Azithromycin

In the 3rd quarter of 2016, all states, with the exception of the Northern Territory and remote Western Australia, reported isolates with resistance to azithromycin. Notably, the reported proportion of *N. gonorrhoeae* isolates with resistance to azithromycin in South Australia for the 3rd quarter 2016 was 5/65 (7.7%). This was lower than that reported in the first 2 quarters of 2016: 26/88 (29.5%) and 25/110 (22.7%) respectively; and compares with 7/251 (2.8%) for 2015. None of the South Australian strains had high level azithromycin resistance (MIC ≥ 256 mg/L). Also of note, there was an increase in azithromycin resistance reported from urban/rural Western Australia, (11.3%) with 1 isolate being of high level resistance (MIC ≥ 256 mg/L).

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The Australian Sentinel Practices Research Network, 1 October to 31 December 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 influenza and infectious diseases surveillance system that is funded by the Australian Government Department of Health. ASPREN was established by the Royal Australian College of General Practitioners in 1991 and is currently directed through the Discipline of General Practice at the University of Adelaide.

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

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

The list of conditions reported is reviewed annually by the ASPREN management committee. In 2016, 4 conditions were being monitored. They included 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.

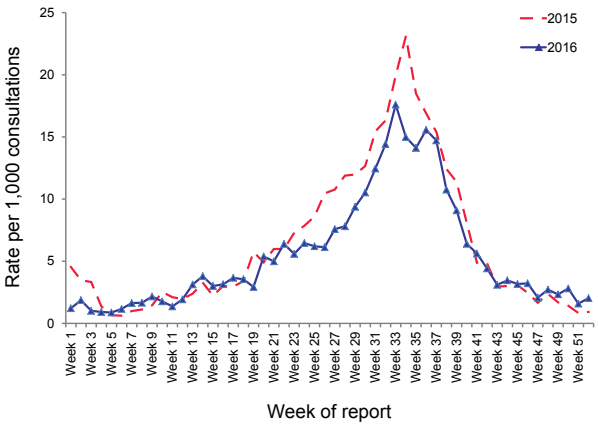
Results

Sentinel practices contributing to ASPREN were located in all 8 states and territories in Australia. A total of 203 general practitioners regularly contributed data to ASPREN in the 4th quarter of 2016.

Each week an average of 176 general practitioners provided information to ASPREN at an average of 13,435 (range 5,901 to 15,461) consultations per week and an average of 113 (range 51 to 151) notifications per week (all conditions).

ILI rates reported from 1 October to 31 December 2016 averaged 3.3 cases per 1,000 consultations (range 1.6 to 6.4 cases). This was similar to the rates in the same reporting period in 2015, which averaged 2.9 cases per 1,000 consultations (range 0.8 to 8.1 cases, Figure 1). Overall, ILI rates reported in 2016 were lower than in 2015.

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



The ASPREN ILI swab testing program continued in 2016 with 196 tests being undertaken from 1 October to 31 December. The most commonly reported virus during this reporting period was rhinovirus (20.4% of all swabs performed), with the second most common virus being influenza A (11.2% of all swabs performed, Figure 2). It is important to note that virological data from week 34 (week ending 4 September 2016) onwards is inclusive of data from the Sentinel Practitioners Network of Western Australia, who were formally merged into ASPREN.

In 2016, a total of 457 cases of influenza were detected with 362 of these typed as influenza A (20.8% of all swabs performed) and the remaining 95 being influenza B (5.5% of all swabs performed) (Figure 2).

During this reporting period, consultation rates for gastroenteritis averaged 4 cases per 1,000 consultations (range 2.2 to 6.9 cases per 1,000, Figure 3). This was lower than the rates in the same reporting period in 2015 where the average was 5.7 cases per 1,000 consultations (range 3.2 to 8.1 cases).

Varicella infections were reported at a lower rate for the 4th quarter of 2016 compared with the same period in 2015. From 1 October to 31 December 2016, recorded rates for chickenpox averaged 0.1 case per 1,000 consultations (range 0.0 to 0.4 cases, Figure 4).

In the 4th quarter of 2016, reported rates for shingles averaged 1 case per 1,000 consultations (range 0.4 to 2 cases, Figure 5). This was slightly lower than the rates in the same reporting period in 2015 where the average shingles rate was 1.2 cases per 1,000 consultations (range 0.4 to 1.8 cases).

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

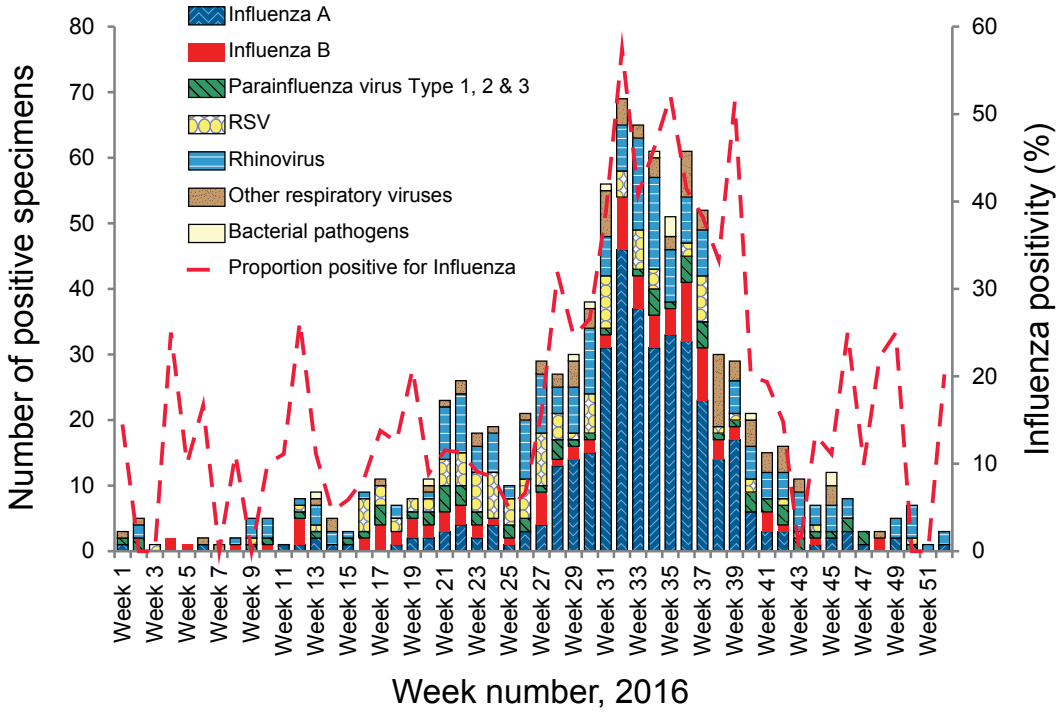


Figure 3: Consultation rates for gastroenteritis, ASPREN, 2015 and 1 January to 31 December 2016, by week of report

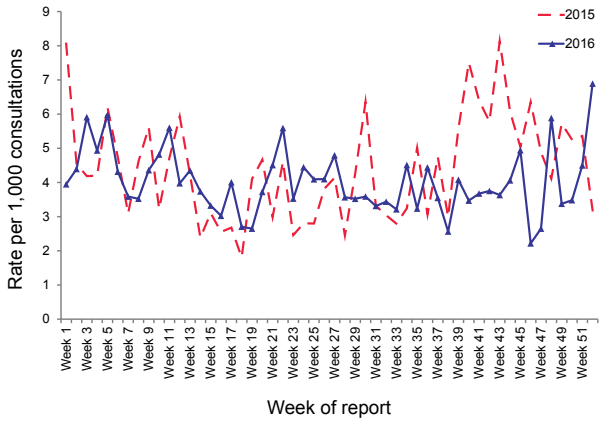


Figure 4: Consultation rates for chickenpox, ASPREN, 2015 and 1 January to 31 December 2016, by week of report

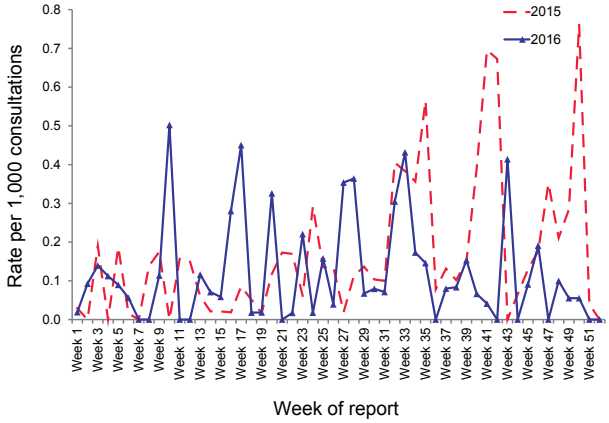
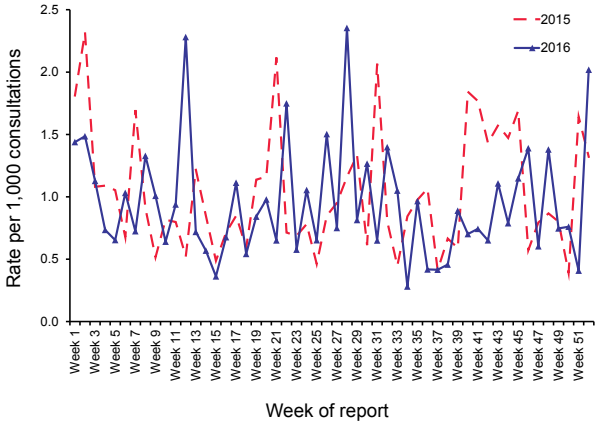


Figure 5: Consultation rates for shingles, ASPREN, 2015 and 1 January to 31 December 2016, by week of report



Invasive Pneumococcal Disease Surveillance, 1 October to 31 December 2016

Kate Pennington and the Enhanced Invasive Pneumococcal Disease Surveillance Working Group, for the Communicable Diseases Network Australia

Summary

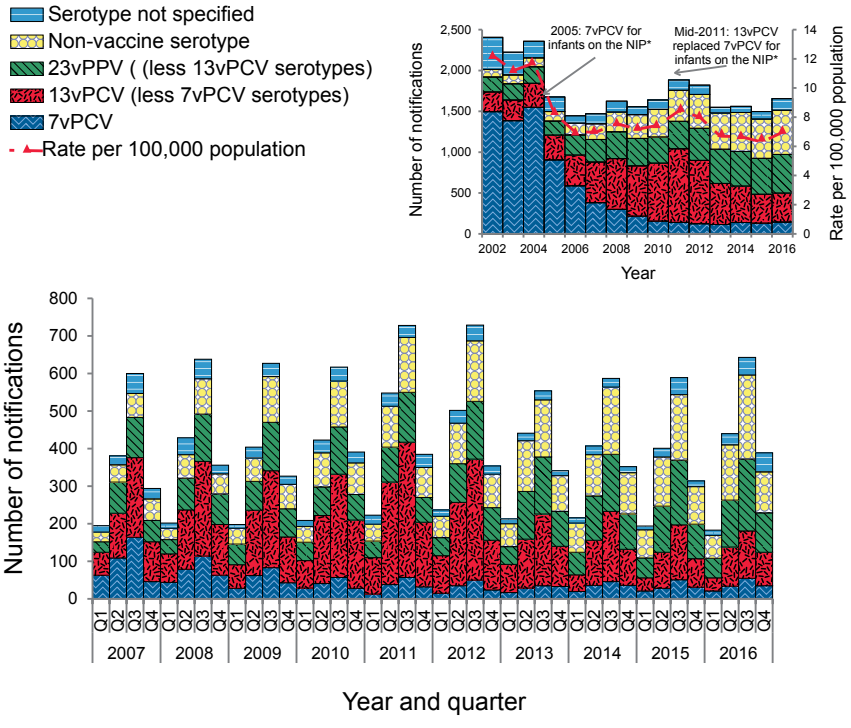
The number of notified cases of invasive pneumococcal disease (IPD) in the 4th quarter of 2016 (n = 389) was less than the previous quarter (n = 643), but more than the number of notified cases in the 4th quarter of 2015 (n = 314). Overall, the decline in disease due to the serotypes targeted by the 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 (Figure 1).

Key points

In the 4th quarter of 2016, there were 389 cases of IPD reported to the National Notifiable Diseases Surveillance System (NNDSS). This represented a 40% decrease compared with the 3rd quarter of 2016 (n = 643) and a 23% increase when compared with the same period in 2015 (n = 314) (Table 1).

For the 2016 calendar year, there were 1,655 notified cases, which was 10% higher when compared with 2015 (n = 1,498). In the 4th quarter of 2016 the most common pneumococcal serotypes causing IPD were 3 (11.3%), 19A (8.2%), 9N (8.0%) and 22F (7.2%) (Table 2). For the reporting quarter and 2016 calendar year, serotypes 3, 19A, 9N and 22F were the most common serotypes, which together accounted for 32% of annual cases (523/1,655).In non-Indigenous Australians this quarter, the number of notified cases was highest in children aged less than 5 years and older adult age groups, especially those aged 60 years or over (Table 3). In Indigenous Australians, cases were highest in children aged less than 5 years and in the 40–44 years age group. The proportion of cases reported as Indigenous this quarter (11%; 42/389) was lower compared with what was observed in the 4th quarter of 2015 (17%; 53/314), and similar to the proportion reported in the 3rd quarter of 2016 (12%; 74/643).

Figure 1: Notifications of invasive pneumococcal disease, Australia, 2002 to 2016, year and quarter, by vaccine serotype group



NIP = National Immunisation Program

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

Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total 4th qtr 2016	Total 3rd qtr 2016	Total 4th qtr 2015	Year to date 2016
Indigenous	0	4	13	6	2	0	1	16	42	74	53	174
Non-Indigenous	5	87	3	50	33	15	69	30	292	509	233	1,314
Not stated / Unknown	0	25	0	0	1	0	29	0	55	60	28	167
Total	5	116	16	56	36	15	99	46	389	643	314	1,655
Indigenous status completeness* (%)	100	78	100	100	97	100	71	100	86	91	91	90
Indigenous status completeness in targeted groups *† (%)	100	86	100	100	96	92	86	100	91	97	100	96
Serotype completeness‡ (%)	100	81	94	93	61	93	93	96	87	93	96	92

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

† Targeted groups for follow-up by almost all jurisdictions and public health units are cases aged less than 5 years and 50 years or over.

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

Table 2: Distribution of serotypes causing invasive pneumococcal disease in notified cases, Australia, 1 October to 31 December 2016, by age group

Serotype	Vaccine type	Age groups			Serotype total
		Under 5 years	5–64 years	Over 65 years	
19F	7vPCV	2	9	7	18
4	7vPCV	–	6	–	6
3	13vPCV non-7vPCV	9	16	19	44
19A	13vPCV non-7vPCV	8	14	10	32
7F	13vPCV non-7vPCV	–	11	1	12
9N	23vPPV non-13vPCV	1	16	14	31
22F	23vPPV non-13vPCV	1	17	10	28
33F	23vPPV non-13vPCV	3	5	6	14
11A	23vPPV non-13vPCV	1	6	5	12
8	23vPPV non-13vPCV	–	6	4	10
23A	Non-vaccine type	1	7	15	23
23B	Non-vaccine type	2	12	3	17
6C	Non-vaccine type	–	5	9	14
35B	Non-vaccine type	–	5	7	12
15A	Non-vaccine type	2	3	7	12
15C	Non-vaccine type	3	5	1	9
16F	Non-vaccine type	1	3	1	5
31	Non-vaccine type	–	3	2	5
Unknown	–	14	25	12	51
Other	–	5	18	11	34
Total		53	192	144	389

* 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 October to 31 December 2016, by Indigenous status and age group

Age group	Indigenous status			Total
	Indigenous	Non-Indigenous	Not reported*	
0–4	6	46	1	53
5–9	2	5	4	11
10–14	1	3	1	5
15–19	5	2	2	9
20–24	1	1	1	3
25–29	1	1	3	5
30–34		6	3	9
35–39	3	7	4	14
40–44	9	9	6	24
45–49	3	12	7	22
50–54	1	20	3	24
55–59	4	23	3	30
60–64	2	31	3	36
69–69	2	25	6	33
70–74	1	27	5	33
75–79		19	4	23
80–84		19		19
85+	1	35		36
Total	42	291	56	389

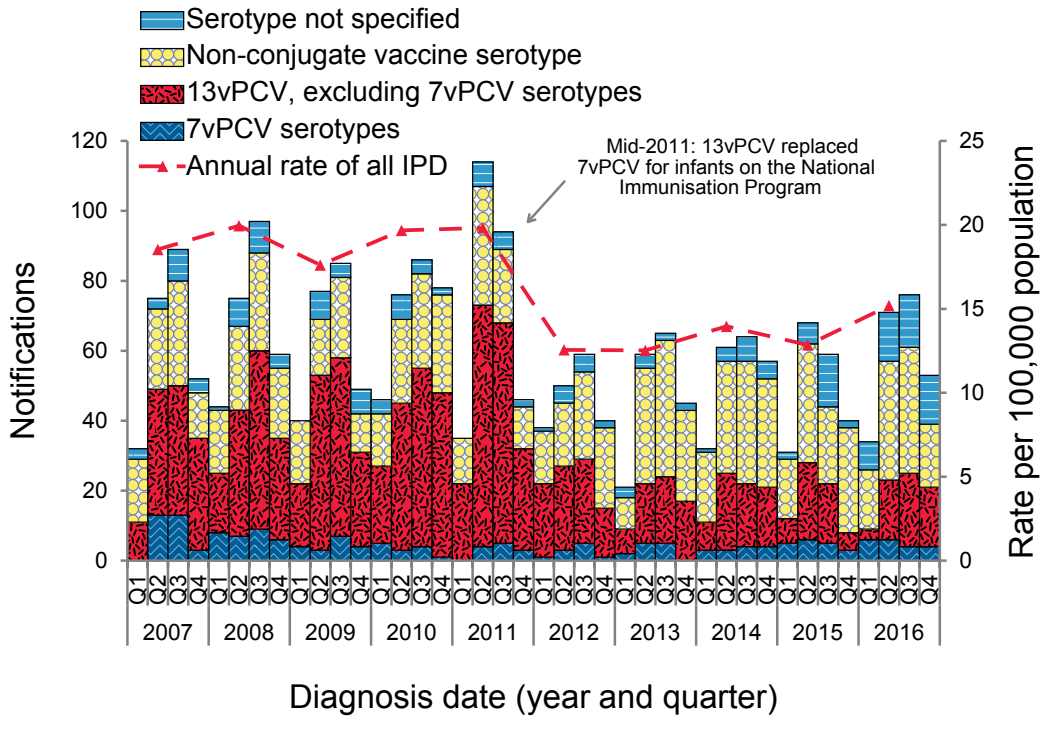
* Not reported is defined as not stated, blank or unknown Indigenous status.

In children aged less than 5 years, there were 53 cases of IPD reported, representing 14% of all cases reported in this quarter. The proportion of cases notified in this age group was similar in this reporting period when compared with the 4th quarter of 2015 (13%; 40/314). Of those cases with a known serotype, 54% (21/39) were due to a serotype included in the 13vPCV compared with 22% (36/40) of cases in the 4th quarter of 2015 (Figure 2). Serotypes 3 and 19A were the most common serotypes affecting this age group in this quarter, noting that both of these serotypes are included in the 13vPCV (Table 2).

In the 4th quarter of 2016, there were 13 cases reported in fully vaccinated children aged less than 5 years who were considered to be 13vPCV failures. Serotype 3 (n=4) was the most common serotype associated with 13vPCV failure reported this quarter (n=7), followed by serotype 19A (n=4) (Table 4).

Among Indigenous Australians aged 50 years or over, there were 11 cases of IPD reported this quarter. Of those cases with a reported serotype, whilst no particular serotype was dominant, 80% (8/10) were due to a serotype included in the 23-valent pneumococcal polysaccharide vaccine (23vPPV) (Figure 3). The number of notified cases of IPD in this age group were less than half of the number reported in the previous quarter (n=26) and

Figure 2: Notifications and annual rates* of invasive pneumococcal disease in children aged less than 5 years, Australia, 2007 to 2016, by vaccine serotype group



* Annual rates are shown on the 2nd quarter.

the 4th quarter of 2015 (n=23). Compared with the previous quarter, the proportion of cases in this population group that were due to serotypes included in the 23vPPV increased markedly from 63% to 80% among cases with a known serotype.

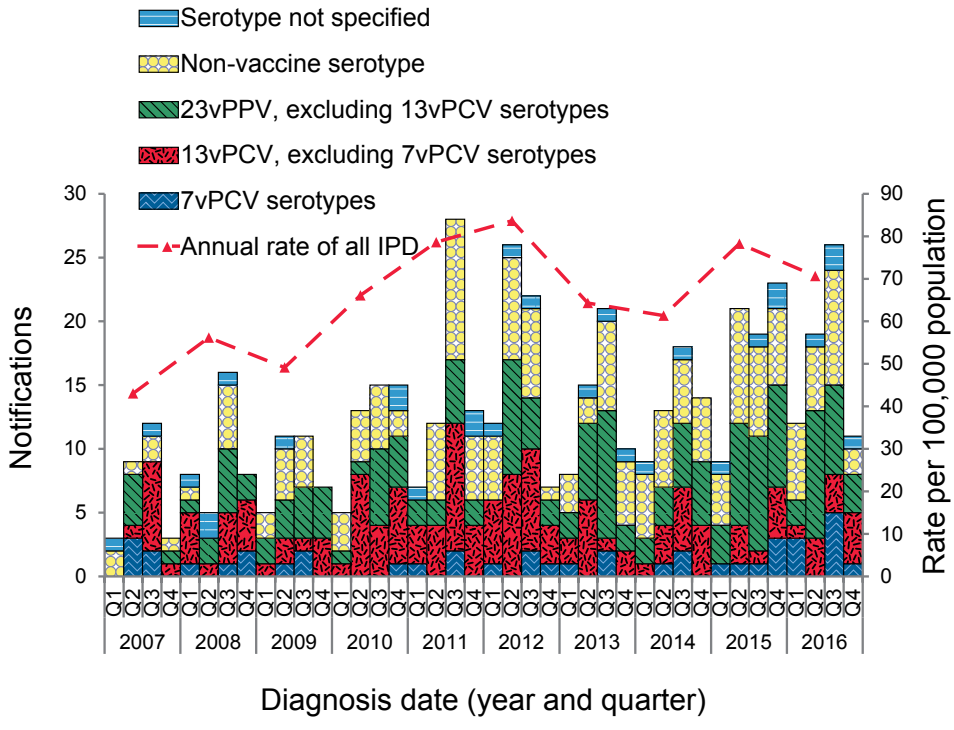
Among non-Indigenous Australians* aged 65 years or over there were 140 cases of IPD reported this quarter. The number of notified cases of IPD in this age group decreased by 40% when compared with the previous quarter (n=232) but was 25% higher than the number reported in the 4th quar-

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

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

Age	Indigenous status	Serotype	Clinical category	Risk factor/s
10 months	Non-Indigenous	3	Pneumonia	Childcare attendee
1 year	Non-Indigenous	19A	Bacteraemia	No data available
1 year	Non-Indigenous	3	Pneumonia	No data available
1 year	Non-Indigenous	19A	Pneumonia	Childcare attendee
2 years	Non-Indigenous	19F	Bacteraemia	No risk factor identified
2 years	Indigenous	3	Pneumonia	Other
2 years	Non-Indigenous	14	Pneumonia and other (other sterile site)	Childcare attendee
2 years	Non-Indigenous	19A	Pneumonia	Other
2 years	Non-Indigenous	3	Pneumonia	Childcare attendee
3 years	Non-Indigenous	3	Pneumonia and other (pleural empyema)	Childcare attendee
3 years	Non-Indigenous	19A	Pneumonia	Other
3 years	Non-Indigenous	3	Pneumonia	No data available
4 years	Non-Indigenous	3	Meningitis	Childcare attendee

Figure 3: Notifications and annual rates* of all invasive pneumococcal disease in Indigenous Australians aged 50 years or over, Australia, 2007 to 2016, by vaccine serotype group



* Annual rates are shown on the 2nd quarter.

ter of 2015 (n=112). Of those cases with a reported serotype, 63% (80/128) were due to a serotype included in the 23vPPV (Figure 4), which represented an increase of 50% when compared with the previous quarter (114/227). For this quarter, serotypes 3 (n=18), 23A (n=15) and 9N (n=14) were the predominant serotypes for this population group, noting that serotype 23A is not included in the 23vPPV.

During this quarter there were 18 deaths attributed to a variety of IPD serotypes, with serotypes 3 (n=6) and 35B (n=3) predominant. Almost all of the reported deaths (17/18) occurred in non-Indigenous Australians. The median age of those who died was 71 years (range 0 to 97 years). Two deaths were reported in children aged less than 5 years, which were associated with serotypes 3 and 10A.

Notes

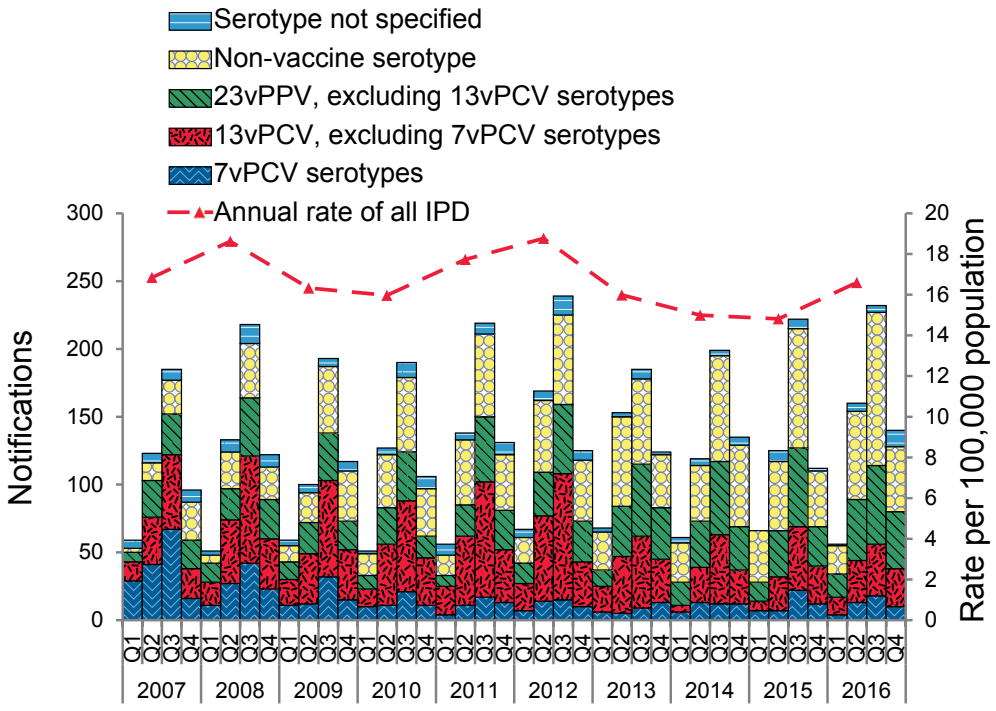
The data in this report are provisional and subject to change as laboratory results and additional case information become available. More detailed data analysis of IPD in Australia and surveillance methodology are described in the IPD annual report series published in *Communicable Diseases Intelligence*.

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

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

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

Figure 4: Notifications and annual rates* of all invasive pneumococcal disease in non-indigenous Australians† aged 65 years or over, Australia, 2007 to 2016, by vaccine serotype group



* Annual rates are shown on quarter 2.

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

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

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

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

Acknowledgements

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Administration

Communicable Diseases Intelligence
INSTRUCTIONS FOR AUTHORS

Communicable Diseases Intelligence (CDI) is a peer-reviewed scientific journal published quarterly by the Office of Health Protection, Department of Health. The journal aims to disseminate information on the epidemiology, surveillance, prevention and control of communicable diseases of relevance to Australia. The objectives of CDI are to:

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

CDI is listed on MEDLINE and indexed by PubMed, an online searchable index of published articles and authors. CDI is open access. All articles published are made available free of charge.

CDI encourages submissions consistent with the objectives from practitioners in all disciplines across the public health field. Advanced trainees and post graduate students are also encouraged to submit to CDI. CDI publishes original articles, short reports, annual reports and quarterly reports, letters to the Editor and editorials. Original articles and short reports are peer-reviewed.

Manuscripts for submission

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

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

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

Authorship

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

Authors details and affiliations of each author should be included in the manuscript before the References. Details should include:

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

Types of manuscript

Original articles

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

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

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

Letters to the Editor and invited editorials

The editorial team welcome comments on articles published in CDI in the form of letters to the Editor. Editorials on a particular topic may be commissioned at the discretion of the Editor. Letters should normally be less than 500 words, while the length of editorials will be negotiated. Letters and editorials should include no more than a single chart and less than six references.

Short reports

Short reports may be submitted for peer review or for publication without peer review, depending on the content. Articles of particular relevance for rapid dissemination (such as timely outbreak reports) may be fast-tracked for early release prior to the next issue of CDI. Please discuss your requirements with the editorial team. Short reports may include an abstract. Types of short reports include:

Surveillance summaries

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

Case reports

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

Outbreak reports

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

An outbreak report may be structured as below (the subheadings can be adjusted to suit), or may be unstructured if very brief.

Most outbreak reports will present only the descriptive epidemiology of the outbreak, with suspected risk factors for infection. The findings of any analytic study would usually be presented in an article at a later date, though authors may choose to present preliminary analyses from analytic studies.

Suggested structure

Abstract

A very brief unstructured abstract should be included.

Background and methods

Include initial detection of the outbreak, case finding and interview techniques, study design and any statistical methods.

Description of outbreak

Case definition, number of cases, number laboratory confirmed, symptoms. Time, place and person, epidemic curve.

A maximum of 2 tables and/or figures is suggested.

Laboratory, trace back and environmental investigations

Details of the proportion of laboratory confirmation of cases.

Public health response

A very brief description of any actions taken to prevent further cases may be included.

Discussion

Including the significance of the outbreak for public health practice.

References

A maximum of 20 references is suggested.

Peer review process

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

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

Authors may be asked to revise articles as a result of the review process before the final decision about publication is made by the Editor. Revised articles are to be returned with a covering letter addressing each comment made by each reviewer. All co-authors should be cc'd when resubmitting revisions to indicate their agreement to the changes.

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

Document preparation

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

Articles are only accepted in electronic form, in Microsoft Word and Microsoft Excel. Graphics may be provided in a range of other formats. In addition:

- Abstracts should not exceed 250 words. Do not cite references in abstracts.
- Structured abstracts are acceptable.
- Include up to 10 keywords.
- Avoid too many abbreviations. Use only accepted acronyms. Do not make up acronyms.
- Use sentence case for all headings.

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

Tables

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

Information in tables should not be duplicated in the text.

Headings should be brief.

Simplify the information as much as possible, keeping the number of columns to a minimum and avoid merged cells as much as possible.

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

Do not use blank rows or blank columns for spacing.

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

Footnotes should use the following symbols in sequence:

* † ‡ § ¶ ** †† ‡‡ ¶

A short summary of each table should be included to satisfy government accessibility requirements.

Figures and illustrations

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

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

A long text description should be included to satisfy government accessibility requirements.

Figures

Use Microsoft Excel.

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

The numerical data used to create each figure must be included on a separate worksheet (example on the Department of Health web site).

Worksheets should be appropriately titled to distinguish each graph (e.g. Figure 1, Figure 2; Figure 1 data, Figure 2 data).

Do not include the graph heading on the Excel worksheet.

Graphs should be formatted to CDI requirements as much as possible. These requirements are available on the Department of Health web site (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-cdi-auth_excel_fig.htm).

Illustrations

Illustrations or flow charts can be included if required.

Images should preferably be at least 300 dpi.

Electronic copies of computer-generated illustrations should preferably be saved in a vector

image program such as Adobe Illustrator or other similar graphic but charts created in either Word or PowerPoint are acceptable. Use a sans serif font for figures (e.g. Arial). Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

Photographs

Photographs may be submitted if required.

Photos need to be at least 300 dpi.

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

- PSD
- TIFF
- EPS
- JPEG (JPG).

Maps

Maps created by mapping programs such as MapInfo or ArcGIS should be saved at 300 dpi and in one of the following graphic formats (in preferential order) to allow editing of font size and colours:

- AI
- EMF

If this is not possible the following graphic formats should be used (in preferential order):

- TIFF
- EPS
- GIF.

Other images

Other images may be submitted in one of the following graphic formats (in preferential order):

- PSD
- TIFF
- EPS
- GIF.

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

Web accessibility requirements

The Australian Government is required to meet level AA of the Web Content Accessibility Guidelines version 2.0 (WCAG 2.0). These guidelines include the need for alternate methods of presenting the information depicted in images—including figures and maps—for readers with vision impairment and other disabilities who use text readers. Complex tables also present challenges for text readers.

Articles and reports should be submitted with a separate Word document that includes:

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

Keep in mind that the description should be sufficient for a sight impaired person to understand what the information image is conveying.

Samples of descriptors for tables and figures can be found on the Department of Health web site (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-pubs-cdi-auth_web.htm).

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

References

References should be identified consecutively in the text using the Vancouver reference style. Any punctuation should precede the reference indicators.

Abbreviate journal names as in the PubMed journal database (http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=journals) (e.g. Commun Dis Intell). Include the surnames and initials of all authors (or only the first six authors, et al, if there are more than six). Cite the first and last page numbers in full, and specify the type of reference (e.g. letter, editorial).

Examples of the Vancouver reference style are available on the Department of Health web site. See also the International Committee of Medical Journal Editors Uniform requirements for manuscripts submitted to biomedical journals Ann Intern Med 1997;1126:36–47 (http://www.nlm.nih.gov/bsd/uniform_requirements.html).

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

The accuracy of references is the responsibility of authors.

Ethics committee approvals and patients' rights to privacy

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

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

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

All co-authors should be cc'd when submitting new or revised manuscripts, to indicate their approval of the content.

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

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