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Original article

Supplemental use of an interferon-gamma release assay in a state-wide tuberculosis contact tracing program in Victoria: a six-year review

Karen M Goebel, Ee L Tay, Justin T Denholm

Abstract

Introduction: Tuberculin skin testing (TST) has been the accepted Australian standard for investigating contacts following exposure to infectious tuberculosis (TB). In recent years, the availability of the interferon-gamma release assays (IGRA) has introduced a potential alternative test but data on its use in this context are limited.

Methods: A prospective longitudinal cohort study was conducted from 2008–2013 to review the use of IGRA and subsequent TB disease following testing in a state-wide contact tracing program. Additional information on the experience and acceptability of IGRA in this context was also obtained through program staff surveys following implementation.

Results: IGRA testing was performed on 643 contacts, with a mean follow-up of 3.7 years. IGRA was primarily used to supplement TST, most commonly due to borderline TST reactivity in individuals who had bacille Calmette-Guérin vaccination. Where both TST and IGRA were performed, correlation of test results was poor (kappa=0.35). The negative predictive value for later development of active TB was 99.5%.

Conclusions: Our experience suggests that IGRA are able to be incorporated safely and effectively as a supplement to TST-based contact tracing. *Commun Dis Intell* 2015;39(2):E191–E196.

Keywords: tuberculosis, public health, contact tracing, interferon-gamma release assay, tuberculin skin test

Introduction

The State of Victoria, Australia has a population of approximately 5.4 million people, with 75% residing in Melbourne. In 2010, there were 7.8 notifications of tuberculosis (TB) per 100,000 population in Victoria, with 96% of laboratory-confirmed cases occurring in people born outside of Australia.[1] State-wide public health management of TB in Victoria, including contact tracing for approximately 2,000 people per year following exposure to pulmonary TB, is performed by the Victorian Tuberculosis Program, based within the Victorian Department of Health (and now within the Peter Doherty Institute, Melbourne Health), during the period of this report.

Historically, the approach to contact tracing in Victoria has been through standardised interviews regarding extent of contact (infectiousness, duration and intensity). Break-of-contact (BOC) tuberculin skin testing (TST) is performed routinely 3 months following exposure, with additional TST performed at baseline for household or other high-risk contacts. However, in certain settings the use or interpretation of TST has been considered problematic, such as baseline reactivity in a bacille Calmette-Guérin (BCG)-vaccinated contact, or a non-reactive test in an immunocompromised individual.[2] Alternative testing options, including the use of interferon-gamma release assays (IGRA) for latent TB, have emerged in recent years, and have been increasingly well-validated for diagnosis of latent tuberculosis infection (LTBI) in a variety of contexts.[3,4] IGRA testing can also be performed in a single visit, offering a potential advantage over the TST, which requires a second visit for recording a result. Increasing availability of IGRA has introduced the possibility that this may offer a suitable alternative in some situations, and these tests have been reported to function effectively in contact tracing settings internationally.[5–7] However, experience in an Australian contact tracing context has been limited to date and existing national guidelines call for local programmatic evaluation.[8]

This article reports a prospective longitudinal cohort study that was initiated to review experience using IGRA within a public health contact tracing program in Victoria. From 2008, IGRA has been available as an alternative to, or supplement for TST on an individual contact assessment basis. Funding provided by a charitable trust enabled testing to be provided without cost to contacts, as

the use of IGRA for contact tracing has not been a previously funded part of programmatic activities. In this paper we describe the use of IGRA by the Victorian Tuberculosis Program from 2008–2013, including comparison of results where both TST and IGRA tests were performed and subsequent re-notification with active TB, and discusses the pragmatic experience of IGRA testing for contact tracing in a low-prevalence setting.

Methods

Prospective data was collected from 1 January 2008 to 31 December 2013 on all contacts in Victoria who were tested with IGRA by the Victorian Tuberculosis Program (hereafter termed IGRA recipients). At the time of substituting IGRA, public health nursing staff were required to document the circumstances of use, including reasons for selecting IGRA. Results of TST (using 5 IU tuberculin) were also recorded where performed. Demographic information, contact investigation results and referral outcomes were routinely collected as part of the Victorian Tuberculosis Program contact tracing records. Data on all other contacts registered in the notification database during the same time period (hereafter termed other TST recipients) were extracted to enable comparisons of basic demographic details and BCG vaccination status between IGRA and other TST recipients. All IGRA recipients were also checked against the notification database to assess re-notifications with active TB.

A single experienced pathology service provided all IGRA (Quantiferon Gold In-Tube assay [Qiagen, Victoria]) testing for this program state-wide. The Victorian Tuberculosis Program records were checked against the pathology service database to ensure reliability of records.

Data were analysed using Microsoft® Office Excel 2010 and Stata (version 12.0, Stata Corp., College Station, TX, USA). TST results reported as induration in mm were categorised as <10 mm, 10–<15 mm and ≥ 15 mm. TST >10 mm are reported as positive unless otherwise stated. Contacts with equivocal or indeterminate IGRA (as per manufacturer's instructions) were categorised as having a negative IGRA result for analysis. Descriptive analysis incorporating means, medians and proportions were performed. Comparison of proportions was conducted with chi-square tests for categorical variables, and for continuous variables, the unpaired *t*-test or Mann-Whitney Wilcoxon rank-sum test. The kappa score was calculated for TST/IGRA agreement where both tests were performed for IGRA recipients. We also calculated the positive and negative predictive value (PPV and NPV respectively) of IGRA and subsequent re-notification of active TB. PPV was derived by the following: true positive/(true positive + false positive), where true positive was defined as those with IGRA positive and active disease, and false positive were IGRA negative and active disease. NPV was derived by the following: true negative/(true negative + false negative), where true negative was defined as those with negative IGRA and non-disease and false negative were those with positive IGRA and active disease.

All data included in this report were collected and recorded as part of routine Victorian Tuberculosis Program activities under the *Public Health and Wellbeing Act 2008*. According to the rules of the Department of Health Victoria, human research ethics committee approval or additional informed consent from participants was not required.

Results

During the period of 1 January 2008 to 31 December 2013, 643 IGRA were performed, with an average of 107 IGRA given annually (range 60–145). During this period, 10,604 TST were performed on contacts registered in the database.

Demographic details, including BCG vaccination status, are presented in Table 1. IGRA recipients were more likely to be aged 25 years or above, people living in rural Victoria, overseas-born and those with a past history of BCG vaccination. In IGRA recipients 83% of overseas-born contacts had a past history of BCG vaccination, compared with 23% of Australia-born (chi-squared $P<0.0001$).

Justification for the use of IGRA is presented in the Figure. IGRA were most commonly used as an adjunct to TST in BCG vaccinated individuals with a 10–15 mm TST reaction at BOC (40%). This was followed by IGRA replacing TST in 198 contacts (30%) due a variety of reasons listed in Table 2.

Comparison of interferon-gamma release assay and tuberculin skin test results

Of the 643 contacts with IGRA performed during the study period, 33% had a positive IGRA result. BOC TST testing was performed in 72% (463/643) of contacts and of these, 17% (78/463) also had baseline TST. Breakdown of TST and IGRA results are outlined in Table 3.

Of 463 contacts where both tests were performed, the kappa coefficient between IGRA and TST was 0.35.

Outcomes of interferon-gamma release assay testing

A chest x-ray (CXR) was performed in 90% of IGRA positive contacts compared with 14% of

Table 1: Demographic profile and bacille Calmette-Guérin vaccination status of interferon-gamma release assays and other tuberculin skin testing recipients, Victoria, 2008 to 2013

	IGRA recipients (n=643)		Other TST recipients (n=10,604)		*P* value‡
Median age in years (Inter-quartile range)	37 (26–50)		22 (16–36)		<0.0001
Age group (years)*	**n**	**%**	**N**	**%**	
0–4	1	0.2	857	8.1	
5–14	24	3.9	1,379	13.1	
15–24	106	16.5	3,693	34.8	
25–44	257	40.0	2,909	27.4	
45–64	196	30.5	1,475	13.9	
65 or over	29	4.5	242	2.3	<0.0001
Sex*					
Males	306	49.6	5,504	47.7	
Females	322	50.1	5,054	52.1	0.638
Area of residence*					
Metropolitan Melbourne	522	81.2	9,659	91.1	
Rural Victoria	106	16.5	902	8.5	<0.0001
Other Victoria†	1	0.2	7	0.1	
Interstate postcodes	0	0.0	34	0.3	
Country of birth*					
Australia	208	32.4	5,257	49.6	
Overseas	408	63.5	5,206	49.1	<0.0001
BCG vaccination status					
Yes	446	69.4	4,776	45.0	
No	51	7.9	2,814	26.5	
Unknown	146	22.71	3,014	28.4	<0.0001

* Percentages do not add up to 100% due to missing data

† Residents of Victoria with unknown postcodes of residence

‡ *P*-value calculated using Mann-Whitney rank-sum test for median age and chi-squared test for other categorical variables

IGRA Interferon-gamma release assays

TST Tuberculin skin test.

BCG Bacille Calmette-Guérin.

IGRA negative contacts (chi-squared $P<0.0001$). The 85% of those with a positive IGRA were referred for consideration of chemo-preventative therapy compared with only 4% of those with a negative result.

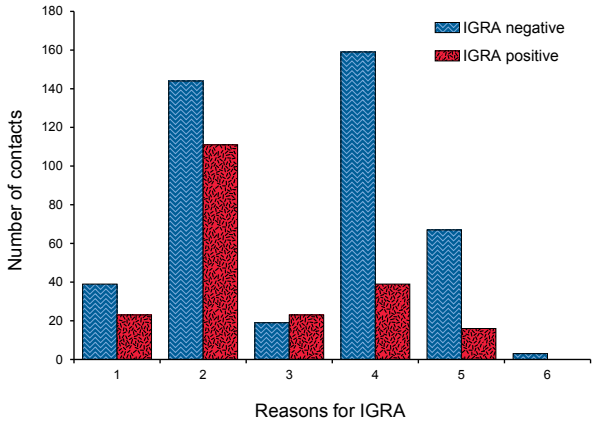
Notification with active tuberculosis

We followed up 633 (98%) of the 643 contacts to ascertain if any developed active TB. All contacts were followed up until 30 June 2014 and the mean length of follow up time was 3.7 years (range 0.6 to 7.3 years). There were no differences in the length of follow up between those with positive and negative IGRA. Six contacts were subsequently re-notified with active TB, four in those with positive IGRA and two in contacts with negative IGRA. The positive and negative predictive value of IGRA was 2% and 99% respectively (Table 4). The median duration for re-notification in IGRA positive contacts was 5.3 months (range 2.5–6.2 months).

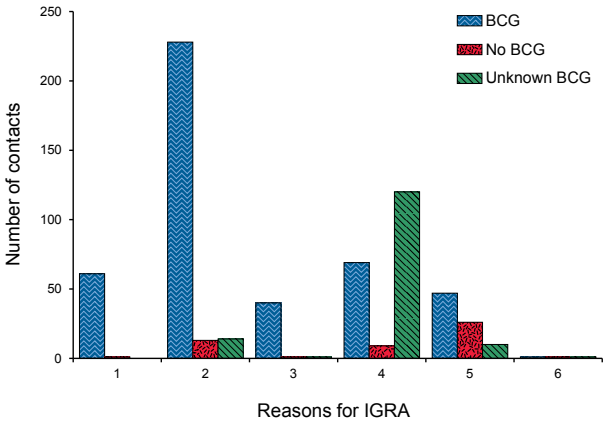
Of the 2 contacts with negative IGRA who subsequently developed active disease, the 1st case was a 22-year-old close household contact born overseas from a high TB burden country and was notified 18 months later with culture-confirmed pulmonary and genitourinary TB. The case had a concurrent TST result of 0 and 10 mm at baseline and BOC respectively, in the context of previous BCG vaccination. The 2nd case was a 21-year-old household contact born overseas, who was notified 10 months later with culture negative pulmonary TB and was on treatment for presumptive TB.

Figure: Indications for interferon-gamma release assays where used in contact investigations, by interferon-gamma release assays positivity (A) and bacille Calmette-Guérin vaccination status (B)

(A) By interferon-gamma release assays positivity



(B) By bacille Calmette-Guérin vaccination status



1. Conversion at break of contact (e.g. 8–14 mm / 0 mm–6 mm – when the tuberculin skin test is positive to increase specificity, mainly in bacille Calmette-Guérin (BCG) vaccinated individuals).
2. 10 mm–15 mm or at break of contact (straightforward adjunct / increase specificity, mainly in BCG vaccinated individuals).
3. 16 mm and BCG (to increase specificity, mainly in BCG vaccinated individuals).
4. Interferon-gamma release assays (IGRA) only replacing the tuberculin skin test.
5. Other reasons: Tuberculin skin test < 10 mm; Anxiety or peace of mind, to validate not infected, validate boost at break of contact; No BCG however ill-defined bleb < 10 mm.
6. Indeterminate.

Table 2: Reasons for interferon-gamma release assays replacing tuberculin skin testing in contact investigations, 2008 to 2013

Reason	n	%
1. Conversion at BOC to increase specificity, mainly in BCG vaccinated	62	9.6
2. 10 mm – 15 mm at BOC (straightforward adjunct / increase specificity, mainly in BCG vaccinated)	255	39.7
3. 16 mm and BCG (to increase specificity, mainly in BCG vaccinated)	42	6.5
4. IGRA only replacing the TST for the following reasons:	198	30.8
Distance	44	22.2
Failed or unable to attend a TST reading	30	15.2
Patient preference or time constraints	25	12.6
Airline contacts	17	8.6
Immunocompromised	14	7.1
BCG vaccination status	13	6.6
Past history of positive TST or active TB disease	9	4.5
Referral from another interstate TB program	6	3.0
Work contact	5	2.5
Elderly age	4	2.0
Health care worker screening	1	0.5
Past history Steven Johnsons Syndrome	1	0.5
No details documented	29	14.6
5. Other reasons: Other TST < 10 mm, Anxiety / peace of mind, to validate not infected, validate boost at BOC, No BCG however ill-defined bleb < 10 mm	83	12.9
6. Indeterminate	3	0.5

BOC – Break of contact; IGRA – Interferon-gamma release assays; TST – Tuberculin skin test; BCG – Bacille Calmette-Guérin.

Table 3: Tuberculin skin testing and interferon-gamma release assays results, 2008 to 2013, by bacille Calmette-Guérin vaccination status

	Total	IGRA positive		IGRA negative	
	n	n	%	n	%
All contacts	643	212	33.0	431	67.1
Contacts with IGRA only	180	34	18.9	146	81.1
Contacts with IGRA and TST	463	178	38.4	285	61.6
<10 mm	109	18	16.5	91	83.5
10–<15 mm	238	105	44.1	133	55.9
≥15 mm	116	55	47.4	61	52.6
Past history of BCG	389				
<10 mm	70	10	14.3	60	85.7
10–<15 mm	208	87	41.8	121	58.2
≥15 mm	111	50	45.1	61	55.0
No past history of BCG	43				
<10 mm	24	4	16.7	20	83.3
10–<15 mm	18	12	66.7	6	33.3
≥15 mm	1	1	100.0	0	0.0
BCG status unknown	31				
<10 mm	15	4	26.7	11	73.3
10–<15 mm	12	6	50.0	6	50.0
≥15 mm	4	4	100.0	0	0.0

IGRA Interferon-gamma release assays
TST Tuberculin skin test
BCG Bacille Calmette-Guérin

Table 4: Notification with active tuberculosis, 2008 to 2013, by interferon-gamma release assays positivity

	IGRA positive (n=212)		IGRA negative (n=431)	
	n	%	n	%
Follow up	209	98.6	424	98.4
Mean follow up (range) in years	3.7 (0.6–7.3)		3.8 (0.6–6.7)	
Re-notification with active tuberculosis				
Yes	4	1.9	2	0.5
No	205	96.7	42	97.9
Unknown	3	1.4	7	1.6

IGRA Interferon-gamma release assays.

The case had a TST result of 0 mm and 6 mm at baseline and BOC, and reported travelling home regularly to a high-burden country during the period between initial exposure and active disease. Neither case met existing guidelines for further assessment at the time of contact investigations.

Discussion

Over the first 6 years since implementation, the use of IGRA has developed as a routine component of contact tracing for TB in Victoria. In the selected group reported here, we found that the test performed well, with good positive and negative predictive values for the later development of active TB. Informally, our experience has been that IGRA have provided a useful adjunct test for contact tracing, and that program staff have become comfortable with their use and interpretation in this setting.

Programmatic recommendations regarding the use of IGRA internationally have tended to suggest that IGRA may be substituted for TST in most circumstances, including contact tracing.[9] Despite the generally positive experience of introduction, however, only a small proportion of contacts in Victoria are currently tested with IGRA. This reflects several factors, including the cost of IGRA testing. The data presented here, both in terms of actual usage and perceived benefits, tend to support the use of IGRA as an adjunct to TST, rather than as a replacement tool. In our study, this was most commonly used in BCG vaccinated individuals to improve the specificity of a positive TST result which explained the poor agreement between both tests, largely because of discordant TST positive / IGRA negative results. The programmatic use of IGRA may change in future, particularly if the cost of IGRA testing is reduced or Medicare-funded indications expand. However, uncertainties regarding the use of IGRA in some contacts (particularly children aged < 5 years) suggest that it is unlikely to replace TST entirely.[10]

In keeping with international experience, the data presented here suggest that IGRA performs well in predicting risk of future development of tuberculosis. This report has some limitations. This report has not considered health economic aspects of test selection, such as a cost-benefit analysis. These analyses are an important part of programmatic decision-making, and future research to evaluate cost-benefit is planned. While contact tracing services are provided centrally, chemoprophylaxis for latent tuberculosis is delivered in a decentralised fashion within public hospital networks. Accordingly, some patients found to have a positive TST or IGRA may not have subsequently completed therapy. Reinfection, particularly in the setting of overseas travel, is also possible, and so precise assessment of risk of TB disease following exposure is not possible. However, this report also has the advantage of complete statewide contact

tracing and laboratory data with longitudinal follow up, and provides additional information suggesting that IGRA may be incorporated within public health programs in Australia.

In a low-prevalence region such as Victoria, experience with performing and interpreting TST results is relatively focused within the Victorian Tuberculosis Program. Accordingly, testing contacts of pulmonary TB in rural districts requires considerable travel time and expense associated with TST. The logistic difficulties of performing TST in rural locations are likely to account for the higher proportion of rural contact tested with IGRA. In our setting, a state-wide pathology provider was able to offer a local blood collection service for IGRA, allowing considerable improvement in efficiency in these circumstances. In other regions with varying capacity for pathology services and TST, logistic experiences may be different. Services with different background populations, including those with high proportions of HIV-infected contacts, may require different approaches to screening and treatment following TB exposure.[11]

Overall, while TST are likely to remain in common use for Australian TB programs, our experience has been that IGRA are able to be incorporated safely and effectively as a supplement to TST-based screening. We hope the results presented here will assist other TB programs in evaluating the utility of incorporating IGRA into local contexts. Further research into the impact of IGRA in this setting, including formal cost-effectiveness evaluation, will provide additional insights into their optimal programmatic use.

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Short report

Interim estimates of male human papillomavirus vaccination coverage in the school-based program in Australia

Julia ML Brotherton, Michael R Batchelor, Michelle O Bradley, Scott A Brown, Simone M Duncombe, Dennis Meijer, Lauren E Tracey, Maureen Watson, Rosalind J Webby

Introduction

In February 2013, following the successful establishment of the National Human Papillomavirus (HPV) Vaccination Program for females in Australia in 2007,[1,2] the program was extended to males. This followed a recommendation by the Pharmaceutical Benefits Advisory Committee that extension of the quadrivalent HPV vaccine program to males would be acceptably cost-effective compared with female only vaccination,[3] and subsequent listing on the National Immunisation Program of quadrivalent HPV vaccine for males. The program extends routine school-based HPV vaccination offered during the first year of high school (at age approx 12–13 years) to males, with a 2 year catch-up program for males aged 14–15 years delivered in 2013 and 2014. The 3 dose coverage (completed course) in the female program has been consistently around 71% by age 15 years, with higher 1 (~81%) and 2 (~79%) dose coverage (National HPV Vaccination Program coverage data (http://www.hpvregister.org.au/research/coverage-data)). In this report we present interim estimates of male HPV vaccination coverage achieved in the school-based program in 2013.

Methods

The Department of Health funds the purchase of HPV vaccine for the national school program, whilst the states and territories are responsible for managing the distribution and administration of the vaccine.

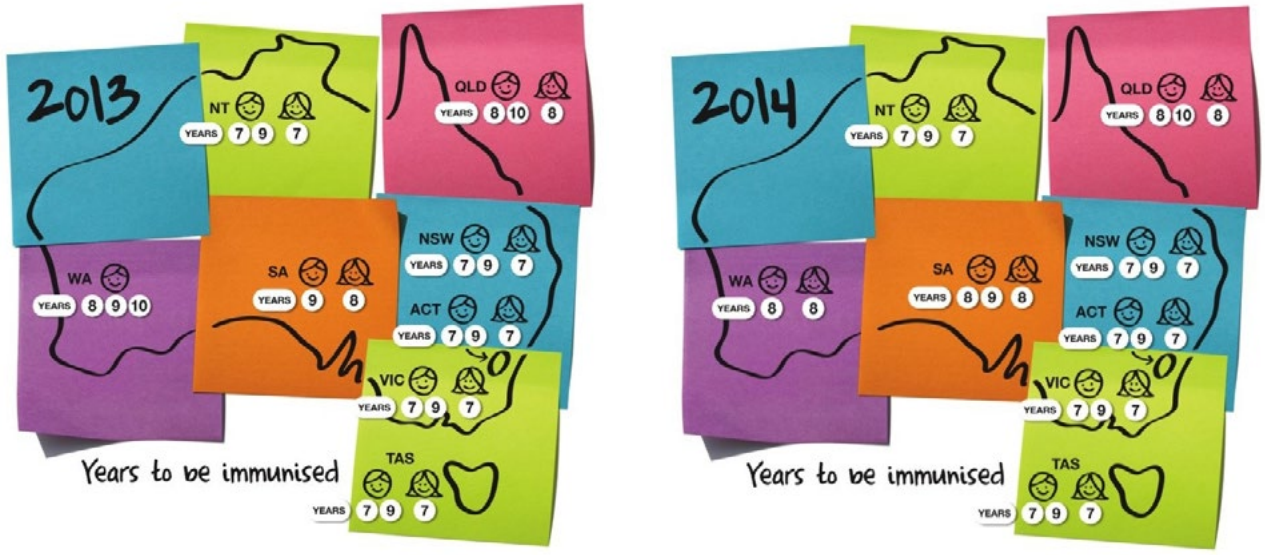
Each of the 6 states and 2 territories are responsible for delivering the school-based vaccination program to their population. As described elsewhere, programs vary somewhat in terms of organisational infrastructure, data collection systems, co-administration of other vaccines, frequency of reporting of data to the National HPV Vaccination Program Register ('the Register') and coverage achieved.[1,4,5] The Register uses estimated resident populations from the Australian Bureau of Statistics (ABS) as the denominator for estimating population based HPV vaccination coverage.[5] Numerator data include doses delivered in the school setting as well as doses delivered in general practice and by other community providers.

In the first year of the male HPV vaccination program (2013), target populations by age and gender (i.e. year level) varied by jurisdiction, as the timeline for the delivery of the catch-up component varied (Figure). Because of this variation in target age group, meaningful national level coverage data by age is not yet available from the Register.

For this report, all jurisdictions except the Northern Territory, utilised available data from their respective departments of education to estimate the eligible enrolled school populations targeted within their jurisdiction during 2013, as the denominators. The numerators comprised the number of doses administered by the school program by dose number. Doses administered in general practice were generally excluded except in the Northern Territory and in South Australia, where general practitioners must notify the health department when giving vaccine to an age eligible recipient. Doses administered in 2014 to complete courses commenced in 2013 in the targeted cohorts were included up to the time of data extraction (September 2014) in New South Wales, Western Australia, South Australia and the Northern Territory. Thus in Victoria, Queensland, the Australian Capital Territory and Tasmania in particular, the presented estimates will be an underestimate of the actual coverage achieved within the school cohorts as 2014 doses are not included, and comparisons across jurisdictions for dose 2 and 3 estimates should not be made.

Data for the Northern Territory utilised all doses administered to targeted age groups in 2013 calculated at 1 October 2014 and recorded in the Northern Territory Immunisation Register as the numerator data. The denominator data were age and sex based population estimates from the Northern Territory health population estimates based on ABS data.

Figure: Human papillomavirus vaccination delivery schedule, Australia, 2013 to 2014, by year level, sex and state or territory



Results and interpretation

Based on dose orders placed by the states and territories, a total of 1,281,350 doses of the HPV vaccine were purchased by the Australian Government in 2013.

In jurisdictions where the same high-school year level was targeted for boys and girls in 2013 (New South Wales, Queensland, Victoria, the Australian Capital Territory and Tasmania), male coverage for dose 1 was only slightly lower than for females (1%–6% lower) with the exception of Tasmania (Table). Male coverage in Tasmania was 3% higher, although not all councils offered HPV vaccine to girls in Year 7 in 2013 (girls in some council areas had been offered vaccine in Year 6 in 2012). Coverage in older boys appears lower, which is consistent with the lower coverage achieved in adolescents in school programs with increasing age.[1,4]

These preliminary estimates suggest a high community acceptance of the extension of the program to males, although coverage appears to be slightly lower than for females (for whom the program is in its 8th year of HPV vaccination in 2014). Over 837,000 doses have been reported to the Register from the male program as at 30 September 2014. Comparison of coverage across jurisdictions is not appropriate from these data due to variations in completeness of data and methods used. Jurisdictional variability in reported school coverage for dose 1 has been previously observed for HPV vaccination in females, and other adolescent vaccines.[1,4]

Final immunisation data following the completion of the male catch-up program at the end of 2014 are awaited, following which a more complete assessment of the population coverage achieved in the targeted cohorts will be undertaken. We need to continue to encourage the notification of HPV vaccination doses delivered by general practitioners and other immunisation providers outside of the school program to ensure that coverage estimates are as accurate as possible. The provision by the Register of overdue dose reports for providers and of history statements or reminder letters to those yet to complete their vaccine course are effective methods to improve both the reporting of doses administered and actual coverage.[6] Accurate coverage estimates will facilitate the on-going monitoring of the impact of our world-leading vaccination program on HPV-related disease and cancers, with results from the female program already showing substantial reductions in HPV infection, genital warts and cervical pre-cancers.[7,8,9]

Postscript: National estimates from the National HPV Vaccination Program Register are now available for males on the Register's web site (http://www.hpvregister.org.au/research/coverage-data).

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Table: Human papillomavirus vaccination coverage preliminary estimates for 2013,* Australia, by state or territory school–based programs

State or territory	Male coverage School year level in 2013, Dose 1/2/3 (%)	Female coverage School year level in 2013, Dose 1/2/3 (%)
Australian Capital Territory	Year 7: 84/82/67 Year 9: 77/73/55	Year 7: 87/86/71
New South Wales	Year 7: 80/78/75 Year 9: 70/67/56	Year 7: 86/84/81
Northern Territory	Age 13: 81/71/51 Age 14: 71/63/46 Age 15: 66/58/41	Age 13: 87/79/61
Queensland	Year 8: 75/71/63 Year 10: 65/61/52	Year 8: 78/74/67
South Australia	Year 9: 79/76/71	Year 8: 89/86/80
Tasmania	Year 7: 69/64/57 Year 9: 66/60/51	Year 7: 68/61/55†
Victoria	Year 7: 83/77/68 Year 9: 71/70/63	Year 7: 84/81/74
Western Australia	Year 8: 75/73/66	2012 Year 7: 85/82/78‡

* Vaccination programs ongoing, including opportunity to complete courses in 2014. Vaccinations given in general practice are not routinely captured in school program estimates, except in South Australia, so actual coverage will be higher.

† In Tasmania, the 2013 female coverage estimate excludes 2013 data from individual school programs that offered human papillomavirus vaccination to Year 6 females during 2012.

‡ Females were not vaccinated in 2013 in Western Australia. Female coverage in the same age cohort who were vaccinated in Year 7 in 2012 is provided as a comparison.

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Legionella pneumophila: probable transmission from a contaminated respiratory device

Jacqueline H Stephens, Douglas D Shaw, Ann P Koehler

Clinical record

A 77-year-old man, who lived alone but with good social support, presented to his general practitioner with a 1 day history of fever and productive cough, with a background of increasing shortness of breath during the previous 2 weeks. He was prescribed a course of amoxycillin but did not commence treatment until the following day. After 3 days of antibiotic treatment he was admitted to his local hospital with persistent fever and cough. On examination he had left lower lobe consolidation, decreased oxygen saturation (SpO_2) and an increased respiratory rate. A chest x-ray revealed increased opacity in the left lower lobe consistent with a pneumonia. Arterial blood gas analysis found his SpO_2 to be 75.4% with a pH of 7.25. The case had a white cell count of 18.3 x 10^9 cells/L with 95.6% neutrophils and 2.4% lymphocytes. His creatinine was elevated (127 μmol/L), as were his fasting glucose (10.2 mmol/L) and liver function tests (alanine transaminase 105 U/L, aspartate aminotransferase 145 U/L). He was diagnosed with community-acquired pneumonia and treated with broad spectrum antibiotics and non-invasive ventilation. Multiplex polymerase chain reaction testing of a throat swab did not detect *Bordetella pertussis, Mycoplasma pneumoniae,* or any respiratory viruses. Three sets of sputum cultures were negative, however ,the patient was already on antibiotics when these were collected. A urinary antigen immunochromatographic assay (BinaxNOW® *Legionella* Urinary Antigen Card, Alere, Maine, USA) confirmed the patient had *Legionella pneumophila* serogroup 1 (LP1). He died of respiratory failure 4 days after admission.

The patient's medical history included obstructive sleep apnoea syndrome, bronchiectasis, and interstitial lung disease. These respiratory conditions were diagnosed in 2007 following a sleep study, lung function testing, and computed tomography scans. He was a non-smoker who only occasionally consumed alcohol. He had no history of cardiac illness. He had used a continuous positive airway pressure (CPAP) machine (ResMed S8 Autoset Spirit™ ANZ Limited edition system with an integrated HumidAire 3i™ humidifier) for 4 years for the management of his obstructive sleep apnoea.

A public health investigation was commenced within 1 hour of receipt of the positive LP1 result. The investigation identified numerous locations visited by the patient during the 10-day incubation period of his illness. Air conditioning cooling towers identified at, or in the vicinity of, these locations were investigated. Environmental samples taken from these towers did not reveal the presence of any LP1. Environmental health officers visited the case's house and found the CPAP machine in a poorly maintained state (Figures 1 and 2). The device was retrieved and swabs of the internal chamber, filter, and mouth piece were positive for LP1. All environmental samples were tested using the Australian Standard Method AS/NZS 3896:2008. This method isolates *Legionella* species by the spread plate technique, with further characterisation using rapid latex slide agglutination.

Figure 1: Respiratory device retrieved from case with biofilm on the internal filter

Figure 2: Respiratory device retrieved from case with biofilm on the face mask

The user manual for the CPAP machine recommends daily cleaning of the mask, with the tubing air dried between uses. Both the mask and tubing should be cleaned weekly with detergent. The humidifier user manual has guidelines for the daily and weekly cleaning of the water chamber. Neither the CPAP or humidifier device manuals explain the importance of, or reasons for, cleaning and maintenance.

Discussion

The acquisition of *Legionella* infection has previously been associated with the use of respiratory equipment.[1–3] A case of humidifier-acquired legionellosis had not been reported in detail in the literature since 1991.[1] A short report in 2013 highlighted the potential association between the use of CPAP devices and 2 non-fatal cases of legionellosis, though in neither of the cases discussed was evidence provided of contamination of the CPAP devices.[3] Without prominent reporting of cases in the literature, the importance of respiratory devices as potential reservoirs of *Legionella* species may not be fully appreciated by a new generation of medical practitioners and respiratory physicians. Furthermore, the majority of previous reports on the risk of these devices described equipment that is now outdated. With modern advances in medical technology it may be wrongly assumed that bacterial colonisation of respiratory devices is no longer a risk. Clearly however, modern respiratory devices remain a potential reservoir of *Legionella* bacteria. It is important, therefore, to highlight this fatality and reaffirm that respiratory devices are a potential reservoir of LP1 and other pathogens, and may be implicated in the acquisition of Legionnaire's disease. This is particularly important for patients with underlying respiratory illnesses.

A respiratory specimen could not be collected, so confirmation of the diagnosis of LP1 by culture was not possible. Furthermore, immunochromatographic urinary antigen testing has been shown to produce false positive results.[4,5] While the sensitivity of the immunochromatographic assay used is estimated at 74%–79%,[4,6] the specificity of the test is estimated as 99.1%.[5] This case also meets the Australian case definition for legionellosis, which is that a confirmed case requires the detection of *Legionella* urinary antigen with clinical evidence, such as fever or pneumonia.[7]

Medical practitioners must ensure their respiratory patients who are advised to use CPAP devices are taught the importance of good device maintenance. Patients need to fully appreciate the potential risks associated with not adhering to the cleaning instructions provided in the instruction manual. A position paper released by the Australian Sleep Association in 2009 states that patients should be provided with device information, including cleaning and safety.[8] This is particularly important for patients living in the community, including those who may have strong social supports. Individual or group education sessions on the use of CPAP devices have been shown to improve compliance with treatment.[9,10] These sessions, which might be conducted by respiratory therapists, provide an opportunity to discuss device maintenance issues. Instruction manuals have detailed information on the process of cleaning and maintaining the device's components, however these instructions read more as a guide to ensuring the longevity of the device components and manufacturer warranty, rather than mentioning health implications of poor maintenance. Ideally, important information on the reasons for cleaning respiratory devices should be included in instruction manuals and highlighted by medical practitioners during any discussions about use of the device. Finally, the inclusion of the patient's social supports in these discussions will raise the profile of, and reinforce, the importance of regular cleaning to ensure that contamination of respiratory devices is avoided.

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Influenza outbreak preparedness: lessons from outbreaks in residential care facilities in 2014

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Abstract

This report describes 6 influenza outbreaks in residential care facilities during the 2014 influenza season in the Sydney Local Health District. Vaccination rates were high among residents (95%) and low among staff (39%). The majority of residents with laboratory confirmed influenza (67%) did not meet the influenza-like illness case definition. Positive influenza specimens were subtyped as H3N2 (40%), H1N1 (5%) or not subtyped (55%). We illustrate the implications of low vaccine effectiveness and antigenic drift, and provide recommendations for the effective management of future influenza outbreaks. *Commun Dis Intell* 2015;39(2):E204–E207.

Keywords: influenza A virus, H3N2 subtype, residential care facility, vaccine effectiveness, antigenic drift, outbreak preparedness

Introduction

Influenza can spread rapidly through residential care facilities (RCFs) resulting in significant morbidity and mortality.[1,2,3] Nationally, 2014 was notable for the highest number of influenza notifications since the 2009 pandemic.[4] New South Wales experienced a more severe season than other states, characterised by higher prevalence of influenza A(H3N2) than the nationally predominant A(H1N1) subtype.[5] One hundred and eleven influenza outbreaks in RCFs were reported in New South Wales in 2014, the highest number in the past decade.[5,6]

Influenza A(H3N2) is associated with a higher burden of disease in elderly populations, and also affects highly vaccinated populations.[1,7] Vaccine effectiveness against H3N2 ranges from 13% to 26%,[8,9] and protection against H3N2 wanes significantly both from the time of vaccination and within single influenza seasons.[8,10] RCFs are thus at high risk of influenza outbreaks and require robust prevention and control measures.

This report describes the management of influenza outbreaks in RCFs by the Sydney Local Health District Public Health Unit (PHU) during the 2014 influenza season (May to October). We discuss the importance of outbreak preparedness by RCFs, and the implications of low vaccine effectiveness and antigenic drift on effective outbreak response.

Methods

Influenza is a scheduled medical condition in New South Wales, and RCF outbreaks are notifiable to the NSW Ministry of Health.[11] The Communicable Diseases Network Australia (CDNA) guidelines define influenza–like illness (ILI) as a triad of fever ≥38°C, respiratory symptoms and systemic symptoms, and a potential outbreak as 'three or more cases of ILI in residents or staff of the facility within a period of 72 hours'.[12]

RCFs contacted the PHU according to the above definition, or when a large number of residents were hospitalised with ILI. RCFs provided a cumulative record of resident and staff symptoms, testing, hospitalisation and deaths on a daily basis using electronic line lists. Information regarding hospital presentations was obtained from the New South Wales electronic medical records system (PowerChart).

The PHU monitored outbreak progression and provided recommendations on infection control, laboratory testing and use of antivirals. Two RCF site visits were conducted where local resources were inadequate; the PHU performed point of care testing, specimen collection, staff vaccination, and delivered antivirals.

Nasopharyngeal swabs were collected from symptomatic individuals for nucleic acid testing. Complement fixation testing was performed on one pre–mortem specimen. Four swabs were sent for strain detection to the World Health Organization Collaborating Centre for Reference and Research on Influenza.

Outbreak description and results

Six influenza outbreaks were notified to the PHU between 4 July and 8 September 2014 affecting 90 residents and 43 staff. The median vaccination rate was 95% among residents and 39% among staff (Table 1). The median influenza attack rate among residents was 24% and median outbreak

duration was 16 days. The median delay in notification to the PHU was 3 days. The proportion of symptomatic residents and staff meeting the ILI case definition was low (median 8% and 10% respectively). The majority of residents with laboratory confirmed influenza did not meet the ILI case definition (median 67%). Among the 3 ILI criteria, fever was least often recorded and respiratory symptoms (cough, coryza or sore throat) were most often recorded.

All laboratory confirmed cases were typed as influenza A. Fifty–five per cent of specimens were not subtyped, 40% were subtyped as H3N2 and 5% as H1N1 (Table 2). Results of specimens sent for strain detection were not available for this report. The majority of residents received the influenza vaccine in March (range January to April), with a maximum interval of 8 months between vaccination and influenza outbreak.

Discussion

Our experience during the 2014 influenza season demonstrates the importance of robust outbreak preparedness in the context of low vaccine effectiveness and antigenic drift.

Insufficient awareness of the CDNA guidelines prevented RCFs from instituting adequate outbreak preparedness measures. A key CDNA recommendation is to ensure a 90% staff vaccination rate;[12] this was not met by our RCFs (median 39%). It is likely that this low vaccination rate was a nidus for ongoing disease transmission. Other authors have also shown that nursing staff contribute to transmission during influenza outbreaks in RCFs.[13] Although there was high vaccine coverage among residents (median 95%), some RCFs vaccinated residents in January 2014. This was prior to the release of the 2014 Southern Hemisphere influenza vaccine in March and is unlikely to have provided effective protection during this season. Better RCF awareness of CDNA guidelines could

Table 1: Summary of influenza outbreaks in residential care facilities, Sydney Local Health District, July to September 2014

	Facility 1	Facility 2	Facility 3	Facility 4	Facility 5	Facility 6
Number of residents	65	101	61	46	68	63
Number of staff	60	100	64	18	90	77
Proportion of residents vaccinated*	95%	38%	98%	91%	100%	95%
Proportion of staff vaccinated*	42%	24%	39%	67%	Unknown†	22%
Outbreak commencement	July 2014	August 2014	August 2014	August 2014	August 2014	September 2014
Duration of outbreak (days)	21	14	18	11	23	14
Delay in notification (days)‡	11	–1	4	7	1	2
Number of symptomatic residents§	28	6	24	17	8	7
Attack rate among residents	43%	6%	39%	37%	12%	11%
Number of symptomatic staff§	10	5	20	1	3	4
Attack rate among staff	17%	5%	31%	6%	3%	5%
Proportion of symptomatic residents meeting ILI case definition	25%	17%	0%	0%	0%	100%
Proportion of symptomatic staff meeting ILI case definition	30%	0%	20%	0%	0%	100%
Proportion of residents with laboratory confirmed influenza not meeting ILI case definition	57%	50%	100%	80%	78%	0%

* Number of residents and staff vaccinated was provided by residential care facilities (RCFs) on line lists.

† This RCF employed regular and casual nursing staff and failed to maintain complete staff vaccination records.

‡ Calculated as time from three or more symptomatic cases noted on line list to public health unit notification. Facility 2 notified the public health unit 1 day prior to 3 symptomatic residents being identified.

§ Symptomatic cases were defined as any residents or staff included on RCF line lists.

Table 2: Laboratory testing results during influenza outbreaks in residential care facilities in Sydney Local Health District, July to September 2014

	Facility 1	Facility 2	Facility 3	Facility 4	Facility 5	Facility 6
Number of specimens collected (staff and residents)	11	9	7	6	11	9
Number of specimens collected (residents only)	11	5	6	6	8	8
Total number of positive influenza specimens	7	6	6	5	9	7
Influenza A, not subtyped	1	4	5	0	8	4
Influenza A, H3N2	4	2	1	5	1	3
Influenza A, H1N1	2	0	0	0	0	0

have improved vaccine coverage among both residents and staff, and thus bolstered outbreak preparedness.

Early outbreak recognition and notification to PHUs is an important outbreak control measure.[12] A very low proportion of symptomatic residents and staff met the ILI case definition (8% and 10% respectively). It is known that elderly populations mount a poor febrile response,[3] and that over half of all influenza infections are asymptomatic.[9] In our outbreaks two–thirds of residents with a positive influenza test result did not have ILI symptoms. It is likely that this led to the use of alternative, late triggers for PHU notification (for example, hospital transfer of symptomatic residents). Thus low sensitivity of the ILI definition and RCFs' difficulty in interpreting CDNA guidelines may have delayed public health action.

The 2014 influenza outbreaks in the Sydney Local Health District occurred during a more severe season and involved highly vaccinated populations where H3N2 was the predominant subtype. This led to the consideration of antigenic drift. H3N2 has been shown to undergo near–constant antigenic drift from season to season.[14,15] In addition to evolutionary drift in the circulating H3N2 virus, it has been suggested that low vaccine effectiveness against H3N2 results from mutations introduced during the egg–based vaccine production process.[16] The World Health Organization confirmed antigenic drift of H3N2 in 2014, and has recommended updating the H3N2 component in the 2015 influenza vaccine.[17] In this context of low vaccine effectiveness and antigenic drift, effective application of outbreak prevention and control measures remains paramount, and requires effective collaboration between RCFs, PHUs, general practice and laboratories. Further intervention research is needed to evaluate stakeholders' understanding of their responsibilities according to CDNA guidelines.

Limitations

Data quality (timely notification and completeness of line lists) remained a problem during the 2014 influenza season. Incomplete data affected the PHU's decision–making ability in the outbreak setting. Laboratories often provided positive influenza results without subtyping results (55% of specimens), resulting in a limited understanding of the extent to which different subtypes contributed to outbreaks.

Conclusion

High vaccine coverage of residents and staff is an important component of influenza preparedness in RCFs. However, low vaccine effectiveness and the potential for antigenic drift highlights the need for greater RCF awareness and application of national guidelines. We recommend the promotion of the CDNA guidelines to RCFs at the start of each influenza season, and interventions to improve RCF staff vaccination rates. Collaboration between key stakeholders to address these limitations will enable more effective management of future influenza outbreaks.

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Annual report

Australian National Enterovirus Reference Laboratory annual report, 2013

Jason A Roberts, Linda K Hobday, Aishah Ibrahim, Thomas Aitken, Bruce R Thorley

Abstract

Australia conducts surveillance for cases of acute flaccid paralysis (AFP) in children less than 15 years of age as the main method to monitor its polio-free status in accordance with the World Health Organization (WHO) recommendations. Cases of AFP in children are notified to the Australian Paediatric Surveillance Unit or the Paediatric Active Enhanced Disease Surveillance System and faecal specimens are referred for virological investigation to the National Enterovirus Reference Laboratory. In 2013, no cases of poliomyelitis were reported from clinical surveillance and Australia reported 1.4 non-polio AFP cases per 100,000 children, meeting the WHO performance criterion for a sensitive surveillance system. Non-polio enteroviruses can also be associated with AFP and enterovirus A71 was identified from nine of the 61 cases classified as non-polio AFP in 2013, which was part of a larger outbreak associated with this virus. A Sabin poliovirus was detected in an infant recently returned from Pakistan and who had been vaccinated while abroad. Globally, 416 cases of polio were reported in 2013, with the 3 endemic countries: Afghanistan; Nigeri;a and Pakistan, accounting for 38% of the cases. To safeguard the progress made towards polio eradication, in May 2014, WHO recommended travellers from the 10 countries that are currently reporting wild poliovirus transmission have documented evidence of recent polio vaccination before departure. *Commun Dis Intell* 2015;39(2):E208–E216.

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

Introduction

Australia has established clinical and virological surveillance schemes to monitor its polio-free status. The clinical surveillance follows the World Health Organization (WHO) recommendation of investigating cases of acute flaccid paralysis (AFP) in children less than 15 years of age. AFP cases are ascertained either by clinicians notifying the Australian Paediatric Surveillance Unit (APSU) via a monthly report card or through the Paediatric Active Enhanced Disease Surveillance System (PAEDS) at 5 sentinel tertiary paediatric hospitals.[1,2,3] The WHO recommends that 2 faecal specimens be collected for virological investigation at least 24 hours apart and within 14 days of the onset of paralysis from cases of AFP to exclude poliovirus as the causative agent. It is a requirement of the WHO polio eradication program that the specimens are tested in a WHO accredited laboratory, which for Australia is the National Enterovirus Reference Laboratory (NERL) at the Victorian Infectious Diseases Reference Laboratory (VIDRL). The clinical and laboratory data from AFP cases in children is reviewed by the Polio Expert Panel (PEP) and reported to the WHO as evidence of Australia's continued polio-free status.

Enterovirus and environmental surveillance programs were established as virological surveillance for poliovirus to complement the clinical surveillance program focussed on AFP cases in children. Enteroviruses other than poliovirus have been associated with AFP and poliovirus infection may manifest clinically without paralysis. The Enterovirus Reference Laboratory Network of Australia (ERLNA) involves public diagnostic virology laboratories reporting enterovirus typing results from clinical specimens to exclude poliovirus and establish the epidemiology of non-polio enteroviruses (NPEVs) in Australia. WHO supports environmental surveillance as a sensitive means of detecting poliovirus through the testing of sewage samples. In 2013, Israel reported the detection of wild poliovirus type 1 in sewage samples without reports of cases of poliomyelitis. The importation and sustained transmission of the virus occurred despite the national polio vaccine coverage being over 90%.[4] Pakistan was identified as the original source of the wild poliovirus importation by genetic sequencing.

The number of wild polio cases worldwide increased from 223 in 2012 to 416 in 2013.[5] This was mainly due to an outbreak of 194 cases in Somalia that originated from Nigeria and led to a further 23 cases in Ethiopia and Kenya, while 160 cases were reported in the three remaining polio endemic countries: Afghanistan, Nigeria and Pakistan. All wild polioviruses detected were serotype 1 with the most recent detections of type 3 in November 2012 in Nigeria and April 2012 in Pakistan. The last detection of wild poliovirus serotype 2 was in India in 1999 leading WHO to

recommend that this serotype be removed from the oral polio vaccine from 2016.[6] All 3 serotypes will still be incorporated in the inactivated polio vaccine. In May 2014, the WHO declared the transmission of polio during the low transmission season in the Northern Hemisphere to be a public health emergency of international concern and recommended travellers from the 10 countries reporting detection of wild poliovirus to have documented evidence of recent polio vaccination.[7]

This report summarises the polio surveillance program in Australia for 2013 encompassing clinical surveillance for AFP cases in children and virological surveillance for poliovirus.

Methods

Acute flaccid paralysis surveillance

Paediatricians reviewing a patient less than 15 years of age presenting with AFP, or clinicians reviewing a patient of any age with suspected poliomyelitis, are requested to notify the NERL (telephone 03-9342 9607, email enterovirus@mh.org.au). Paediatricians also notify the AFP case to the APSU (http://www.apsu.org.au/) via a monthly report card. Upon receipt of the notification, the AFP National Surveillance Co-ordinator based at VIDRL forwards a clinical questionnaire for the clinician to complete. Alternatively, AFP cases are ascertained by PAEDS nursing staff from medical records and are enrolled in the surveillance program with parental or guardian consent.

According to the WHO surveillance criterion 2 faecal specimens must be collected more than 24 hours apart due to intermittent virus shedding, and within 14 days of the onset of paralysis, while the virus titre remains high, to be classified as adequate. The faecal specimens are tested free of charge by the NERL.

The PEP, a subcommittee of the Communicable Diseases Network Australia, reviews the clinical and laboratory data for all notified cases of AFP, irrespective of whether they are an eligible or ineligible case. An eligible case is an Australian child 14 years or younger with AFP (including Guillain-Barré syndrome and transverse myelitis) or an Australian of any age with suspected polio. Ineligible cases include patients aged 15 years or over, overseas residents and cases notified in error or later determined not to be AFP.

The PEP classifies cases of AFP as:

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

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

The WHO AFP surveillance performance indicator for a polio non-endemic country is 1 case of non-polio AFP per 100,000 children aged less than 15 years. For Australia in 2013, this equated to 43 cases per year, based on the Australian Bureau of Statistics data released in December 2012. An AFP surveillance scheme that satisfies the WHO surveillance performance indicator is deemed sufficiently sensitive to detect a wild poliovirus importation in children of that country. The WHO surveillance performance indicator for laboratory testing is that at least 80% of notified AFP cases have adequate faecal specimens collected and tested in a WHO accredited laboratory.

At the end of each calendar year, a number of AFP notifications remain pending as there is insufficient clinical and laboratory data for the PEP to report a final classification. The PEP classifies such notifications as 'polio compatible-zero evidence' if a final review reveals no evidence of clustering among the cases.

Virus culture

Upon receipt at the NERL, faecal specimens are treated with minimum essential medium containing Hank's salts, chloroform (9.1% v/v) and foetal bovine serum (2%). The suspension is clarified and the supernatant inoculated onto a series of mammalian cell lines. Two WHO recommended cell lines are used for the isolation of poliovirus, L20B (a transgenic mouse epithelial cell line expressing the human poliovirus receptor, CD155) and RD-A (human rhabdomyosarcoma).[8,9] Diagnostic laboratories in Australia are encouraged to refer enteroviruses of unknown serotype to the NERL for further characterisation as poliovirus infection can lead to clinical presentations without paralysis such as aseptic meningitis.

Two WHO real time reverse transcription polymerase chain reaction (RT-PCR) tests are used to determine whether a poliovirus is a wild strain, oral poliomyelitis vaccine (OPV) strain (Sabin-

like) or a VDPV, in a process known as intratypic differentiation (ITD).[10] The NERL sequences the complete poliovirus viral protein 1 (VP1) genomic region, which contains a major neutralising antibody binding site. The VP1 genomic sequence provides valuable biological information, including the number of mutations within a significant region of the OPV virus strain and it enables phylogenetic analysis of wild poliovirus to rapidly determine the likely source of the virus, as utilised in the 2007 wild poliovirus importation.[11]

Enterovirus surveillance

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

The NERL encourages members of ERLNA to perform their own enterovirus typing. It has advised members of ERLNA on enterovirus detection, supplied laboratory and computer analysis protocols and performed tests in parallel with other laboratories for quality assurance purposes. The NERL receives untyped enteroviruses from 3 laboratories for typing on a regular basis. The other laboratories perform their own enterovirus typing and report the results to the NERL for inclusion in the national enterovirus database.

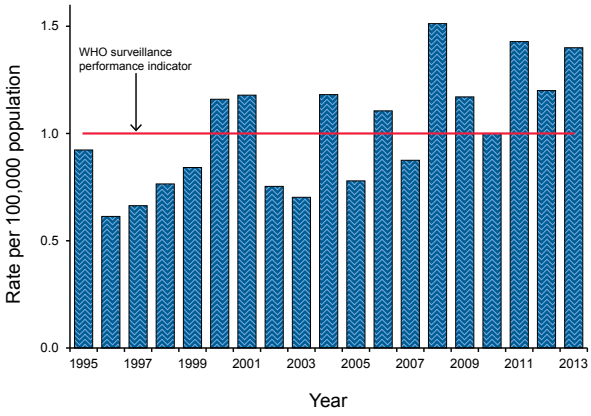
The NERL screens clinical specimens for enterovirus using a semi-nested RT-PCR directed to highly conserved sequence in the 5' non-translated region.[12] Enterovirus typing is primarily performed by amplifying a fragment of the VP1 genomic region according to a published method,[13] but the complete nucleotide sequence of VP1 is required to type some enteroviruses. The enterovirus typing RT-PCR is directed to a region of sequence divergence that allows differentiation between enterovirus genomes. As a consequence, the enterovirus sequence based typing assay is not as sensitive as the pan-enterovirus detection assay. This can result in an enterovirus being detected by pan-enterovirus RT-PCR in a clinical specimen without subsequent identification by the VP1 enterovirus typing assay.

Results

Classification of acute flaccid paralysis cases

A total of 100 notifications of AFP cases were received in 2013 (Table 1). The PEP classified 61 cases as non-polio AFP, which equated to a rate of 1.4 cases per 100,000 children less than 15 years of age, exceeding the WHO AFP surveillance performance criterion for a polio-free country of 1 case of non-polio AFP per 100,000 children (Table 2, Figure 1).

Figure 1: Non-polio acute flaccid paralysis rate after final classification, 1995 to 2013, by the Polio Expert Panel



* The World Health Organization acute flaccid paralysis surveillance performance indicator for a polio non-endemic country is 1 case per 100,000 children <15 years of age.

An enterovirus A71 (EV-A71) outbreak occurred along the eastern seaboard of Australia in 2013, with most cases reported in Sydney that included at least 2 deaths associated with EV-A71 infection in children.[14] Between February and September, EV-A71 subgenogroup C4a was isolated by virus culture or detected by RT-PCR from the stool specimens of 9 AFP cases and a further 3 non-polio AFP cases were associated with EV-A71 based on the clinical evidence, representing 20% of AFP cases with onset of paralysis in 2013. Ten of the cases were from New South Wales and two in Victoria and involved children less than 6 years of age. NPEVs were isolated from another 4 AFP cases in New South Wales and Victoria and identified as coxsackievirus A4 (CV-A4), coxsackievirus A10 (CV-A10), echovirus 7 (E7) and echovirus 11 (E11). In total, NPEVs were reported from 16 cases; a quarter of the non-polio AFP cases classified by the PEP in 2013.

Table 1: Notification of acute flaccid paralysis cases, 2013 by state or territory

State or territory	Estimated population aged <15 years*	Expected number of AFP cases in 2013	Total number of notifications	Ineligible notifications	Duplicate notifications	Polio compatible	Eligible cases with final classification by PEC	Non-polio AFP rate per 100,000 children
ACT	68,177	0.5	0	0	0	0	0	0.0
NSW	1,367,952	14	37	3	11	1	22	1.6
NT	52,914	0.5	1	0	0	0	1	2.0
Qld	902,387	9	9	2	1	0	6	0.7
SA	291,942	3	3	1	0	0	2	0.7
Tas.	94,805	1	1	0	0	0	1	1.0
Vic.	1,024,646	10	39	2	15	0	22	2.2
WA	464,882	5	10	2	1	0	7	1.4
Australia	4,267,705	43	100	10	28	1	61	1.4

* Australian Bureau of Statistics, Estimated population at 30 June 2012 (www.abs.gov.au).

AFP Acute flaccid paralysis

PEC Polio Executive Committee

Table 2: Surveillance for acute flaccid paralysis cases in Australia, 2013, compared with the main World Health Organization performance indicators

WHO surveillance performance indicator for AFP cases in children <15 years	Performance of Australia's AFP surveillance	
≥1.0 non-polio AFP case / 100,000 children (43 cases for Australia in 2013).	61 cases classified as non-polio AFP.	1.4 (61 / 43) non-polio AFP cases / 100,000 children <15 years.
≥80% of classified AFP cases with adequate specimens (2 faecal specimens collected at least 24 hours apart and within 14 days of onset of paralysis.	25 AFP cases with adequate specimens collected.	41% (25 / 61) classified non-polio AFP cases with adequate specimens.

AFP Acute flaccid paralysis

A patient fully immunised against polio was diagnosed with anterior myelitis due to an enteroviral infection. Stool specimens were not collected for testing at the NERL and the case was classified as polio compatible by the PEP as polio could not be excluded based on the available clinical evidence (Table 1). Twenty-eight AFP cases were notified by more than 1 source: either 2 clinicians returned a clinical questionnaire about the same case or a clinician returned a clinical questionnaire about a case also enrolled in the PAEDS system, and were regarded as duplicate notifications (Table 1). A further 10 AFP notifications did not meet the criteria for an eligible case as the patients were greater than 14 years of age, the patient's clinical condition was later considered not to be consistent with AFP or consent to enrol the case through PAEDS was not granted.

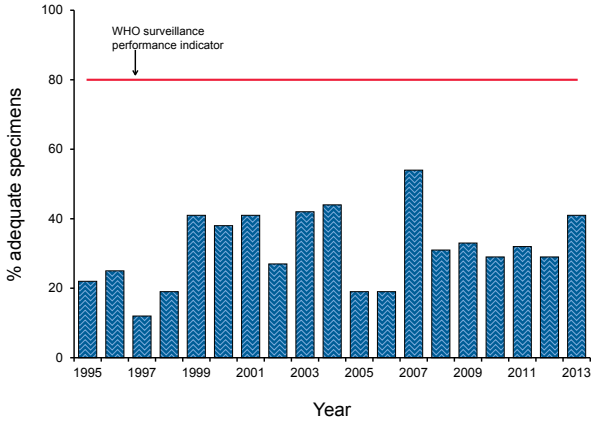
Notification of acute flaccid paralysis cases by state and territory

In 2013, AFP cases were notified from all jurisdictions in Australia except the Australian Capital Territory (Table 1). The non-polio AFP rates for eligible cases per jurisdiction exceeded the WHO AFP surveillance performance indicator of 1 case per 100,000 children in New South Wales, the Northern Territory, Tasmania, Victoria and Western Australia. Queensland did not achieve the expected rate of non-polio AFP cases despite the syndrome being included in the state's Notifiable Conditions System since 2001.[15]

Faecal collection from acute flaccid paralysis cases

A total of 81 faecal specimens from 44 of the 61 eligible cases were tested at the NERL but only 25 AFP cases had two specimens collected within 14 days of the onset of paralysis in 2013 (Tables 2 and 3). While the proportion of cases that met the WHO criteria for specimen collection was 41% compared with the target of 80% (Figure 2), 61% of AFP cases had at least 1 specimen collected within 14 days of onset, and 72% of cases had a specimen collected at any time after the onset of paralysis. Five different types of NPEV (CA-A4, CA-A10, E7, E11 and EV-A71) were isolated by virus culture from 15 of the 81 stool specimens, an isolation rate of 19%. EV-A71 was isolated from 5 AFP cases by virus culture and detected direct in the stool extract by RT-PCR of a further 4 AFP cases.

Figure 2: Percentage of acute flaccid paralysis cases with adequate faecal specimens, 1995 to 2013*



* The WHO surveillance performance indicator is 2 faecal specimens collected more than 24 hours apart and within 14 days of the onset of paralysis from 80% of classified non-polio AFP cases.

Enterovirus surveillance

A poliovirus type 2 was referred through the ERLNA for ITD in March 2013 (Tables 3 and 4). An infant was hospitalised in Perth with diarrhoea after returning from Pakistan where the patient was vaccinated with OPV and also likely to have acquired a rotavirus infection. The laboratory in Perth identified rotavirus and poliovirus type 2

Table 3: Test results of acute flaccid paralysis cases with onset in 2013, and specimens referred to the Australian National Enterovirus Reference Laboratory from within Australia

Result	Specimens from AFP cases involving children < 15 years of age	Specimens from AFP cases involving patients ≥15 years of age	Specimens from sources other than AFP	Total
Sabin poliovirus type 2	0	0	1	1
Non-polio enterovirus	22	0	133	155
Rhinovirus	0	0	9	9
No enterovirus identified	59	1	136	196
Total	81	1	279	361

AFP Acute flaccid paralysis.

and referred the latter to the NERL for further investigation. The poliovirus type 2 was Sabin-like by the WHO recommended test that differentiates between wild and vaccine strains. The full VP1 genomic region had 100% sequence identity to prototype Sabin poliovirus type 2, consistent with recent vaccination with OPV.

The ERLNA typed 461 NPEVs, of which the NERL contributed 242 identifications (Table 4). The most common serotypes identified by ERLNA were, in order of decreasing frequency, echovirus 6, coxsackievirus A6, echovirus 9 and EV-A71.

Regional reference laboratory activities

The following activities were performed as a Polio Regional Reference Laboratory in 2013.

- Specimens from AFP cases were referred from Brunei Darussalam (2 cases), Pacific Island countries (8 cases) and Papua New Guinea (19 cases). No poliovirus was isolated from any of the specimens but NPEVs were reported from 5 cases from the Pacific Islands and 14 cases from Papua New Guinea.
- Twelve poliovirus type 1, 2 poliovirus type 2 and 2 poliovirus type 3 isolates were referred from AFP cases in the Philippines for ITD and all were characterised as Sabin-like. The national polio laboratory of Malaysia referred a poliovirus type 1 and a type 2 isolate from environmental samples for sequencing after the ITD tests indicated both had significant mutations that required further investigation. The VP1 genomic regions were sequenced at VIDRL and both were confirmed as Sabin-like polioviruses.

Table 4: Summary of enterovirus testing at the Australian National Enterovirus Reference Laboratory for samples referred within Australia 1995 to 2013

Year	Poliovirus		Non-poli enterovirus	No enterovirus detected	EVID results referred[||]	Total samples reviewed
	Sabin-like	Non-Sabin-like				
1995	190	0	200	13	0	403
1996	224	0	198	9	0	431
1997	124	0	76	0	0	200
1998	52	0	15	4	0	71
1999*	60	1	9	9	0	79
2000	45	0	44	47	0	136
2001*	46	5	33	75	0	159
2002	36	0	21	49	0	106
2003	9	0	15	47	0	71
2004	6	0	26	61	0	93
2005	18	0	10	39	0	67
2006	2	0	6	71	29	108
2007†	0	2	32	115	107	256
2008	0	0	20	92	77	189
2009‡	1	0	63	78	113	255
2010	0	0	170	39	108	317
2011	0	0	174	61	205	440
2012	0	0	155	97	123	375
2013§	1	0	242	189	219	671

* Untyped enterovirus or uncharacterised poliovirus isolates were referred for further testing after completion of a laboratory inventory. The 6 isolates tested as non-Sabin-like and were subsequently identified as wild type poliovirus prototype strains and were destroyed.

† Wild poliovirus type 1 was imported from Pakistan.

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

§ A Sabin-like poliovirus type 2 was identified from an infant who was immunised overseas with oral polio vaccine and hospitalised with diarrhoea upon return to Australia.

|| Enterovirus identification (EVID) results include retrospective data made available via the Enterovirus Reference Laboratory Network of Australia.

Quality assurance programs

In 2013, the NERL was accredited as a WHO Polio Regional Reference Laboratory through participation in the annual WHO poliovirus quality assurance panels for isolation by cell culture, RT-PCR for ITD, vaccine derived poliovirus and sequencing and an on-site visit in October. The laboratory also participated in the Royal College of Pathologists of Australasia quality assurance panel for enterovirus detection by RT-PCR.

Discussion

In 2013, Australia reached the WHO surveillance target of ≥1 non-polio AFP cases per 100,000 children, for the 6th year in a row. The combination of clinicians notifying AFP cases via the APSU monthly report card and nurses ascertaining cases through the PAEDS system provided Australia with a polio surveillance system that meets the international standard to detect an imported case of polio in children less than 15 years of age through these well-established schemes.[3,16]

Australia has never met the strict WHO surveillance target for adequate stool collection from 80% of AFP cases, with 41% of cases having 2 specimens tested in 2013, and 61% of cases with at least a single specimen. In 2014, the Chair of the National Polio Certification Commission published a letter reminding paediatricians that it was critical to notify cases of AFP and arrange for collection of 2 stool specimens as part of Australia's vigilance for poliovirus importations.[17]

Virological investigation of AFP cases is important to confirm the presence of poliovirus and also serves to establish an association between NPEVs and AFP cases. This was reinforced in 2013, with 5 different types of NPEV reported from AFP cases by the NERL, including EV-A71 from 9 AFP cases. This was part of a larger EV-A71 outbreak first reported in Sydney that included at least 2 fatalities.[14,18] In 2010, WHO undertook a risk assessment of EV-A71 infection in the Western Pacific region and concluded that outbreaks will increase in frequency due to the virus's continued evolution and production of novel recombinants with severe cases likely to occur due to the introduction of novel strains.[19] WHO noted that the level of uncertainty for the risk assessment was moderate due to incomplete surveillance such as determining the EV-A71 subgenogroup, which would provide more information on virus transmission. The sudden increase in association of EV-A71 with AFP cases in 2013, coincided with a switch from subgenogroup B5 being the predominant strain detected in Australia, as reported by the Enterovirus Reference Laboratory Network of Australia,[20] to subgenogroup C4a. This highlights the value of routine enterovirus typing to establish the epidemiology of enterovirus circulation in Australia.

Outbreaks of hand, foot and mouth disease due to EV-A71 with instances of fatal neurological complications have occurred in many Asian countries since the late 1990s, which has led to research and development of candidate vaccines.[21,22] Two independently produced, inactivated whole-virus vaccines were reported to be safe, immunogenic and protective against EV-A71 associated hand, foot and mouth disease in phase 3 clinical trials in China.[23,24] Both vaccines were produced from the C genogroup strain and cross-protection against the other EV-A71 genogroups needs to be determined.

The referral of a poliovirus detected from a patient recently returned from Pakistan, through the ERNLA, demonstrates the additional value of virological surveillance to complement the clinical surveillance program. While the reporting of a type 2 Sabin-like poliovirus in Australia in 2013, may be considered inconsequential, it is important that all polioviruses are fully characterised by the NERL. VDPVs can evolve in areas with low oral polio vaccine coverage through person-to-person transmission leading to loss of attenuation, and were reported in 8 countries worldwide in 2013.[25] The NERL is accredited by WHO to perform specific tests that detect VDPVs, which must be reported under the International Health Regulations (2005), the same as for a wild poliovirus detection. Seven of the 8 VDPV outbreaks in 2013, involved poliovirus type 2 with only 1 event involving poliovirus type 3. The risk of type 2 VDPV outbreaks in the absence of wild poliovirus type 2, which was last detected in 1999, has led WHO to recommend the Sabin 2 poliovirus strain be removed from OPV by the end of 2016, as part of the polio eradication and endgame strategic plan.[26] As a safeguard, the plan recommends at least 1 dose of trivalent inactivated polio vaccine (IPV) be introduced into all routine immunisation schedules prior to the switch from trivalent to bivalent OPV. Australia ceased use of OPV from November 2005 and IPV is available as a paediatric combination vaccine and as an individual vaccine for booster immunisations.[27]

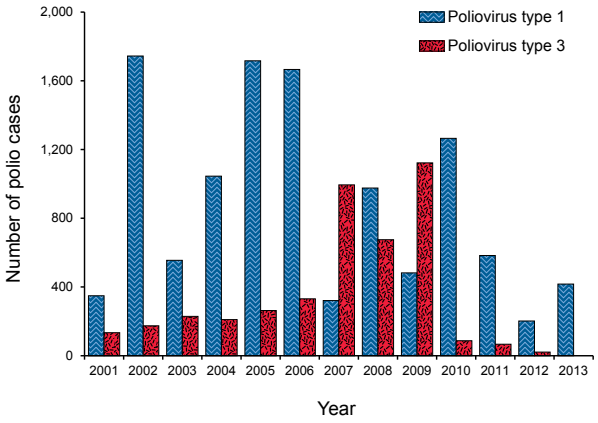
In 2012, the National Polio Certification Commission for Poliomyelitis Eradication undertook a review of polio surveillance that found support for the existing systems that were deemed to be appropriate for Australia.[28] Gaps in surveillance were identified with regard to the detection of adult cases, ensuring clinicians would recognise a case of polio, the risk of polio importations, the need to improve stool collection rates and the importation and storage of biological samples containing poliovirus. The review made 10 recommendations:

1. Australia should continue to undertake active polio surveillance;
2. the existing active surveillance systems should occur for 3 years post-eradication and enterovirus surveillance should continue post-eradication;
3. the purpose, objectives and activities of the Australian polio surveillance system should be documented by the Department of Health;
4. AFP surveillance should continue in its current form;
5. stool collection rates should be improved including through enhancing the effectiveness of the PAEDS program;
6. polio should remain a nationally notifiable condition but AFP should not be nationally notifiable;
7. sentinel environmental surveillance sites should be maintained and trialed in a major metropolitan area;
8. communication to raise awareness of the importance of completing global poliovirus eradication and highlighting the need for clinicians to remain vigilant for cases of poliomyelitis should be developed by the Department of Health;
9. the Department of Health should review current vaccination policies to determine if they are adequate to address the risk of polio importations by immigrants refugees and travellers to and from endemic countries; and
10. a review of biosecurity arrangements for the laboratory containment of polioviruses should be conducted.

Despite an increase in the number of polio cases worldwide from 223 in 2012, to 416 in 2013, it is significant that the last reports of the 2 remaining genetic lineages of wild poliovirus type 3 were in November 2012 in Nigeria and April 2012 in Pakistan (Figure 3).[5] While three years of surveillance will be required to confirm the eradication of another poliovirus serotype, the data looks promising that this important milestone will be achieved.

Three countries remain endemic for wild poliovirus, having never interrupted transmission: Afghanistan, Nigeria, Pakistan, and another seven have wild poliovirus due to importations; Cameroon, Equatorial Guinea, Ethiopia, Iraq, Israel, Somalia and Syria. In May, WHO announced that the continued spread of wild poliovirus from Cameroon, Pakistan and Syria to neighbouring countries during January to April 2014, considered as the low season for transmission, is considered a public health emergency of international concern.[7] In response, WHO issued temporary recommendations under the International Health Regulations (2005) that people travelling from the 3 countries linked to the recent importations should have documented evidence of polio vaccination within 4 weeks to 12 months of departure and travellers from the other 7 countries are encouraged to be vaccinated with documented evidence before departure. The Australian Government Department of Health issued recommendations for Australian travellers in light of this development.[29] The new international recommendations will be reviewed by WHO after 3 months but such a proactive international stance may be needed to herald the end of the last remaining reservoirs of wild poliovirus.

Figure 3: Number of wild polio cases, worldwide, 2000 to 2013, by poliovirus serotype



Source: Global Polio Elimination Initiative [online]. Accessed on 4 May 2014 (http://www.polioeradication.org/Dataandmonitoring/Poliothisweek/Wildpolioviruslist.aspx).

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Tuberculosis notifications in Australia, 2012 and 2013

Cindy Toms, Richard Stapledon, Justin Waring, Paul Douglas and the National Tuberculosis Advisory Committee, for the Communicable Diseases Network Australia, and the Australian Mycobacterium Reference Laboratory Network

Abstract

The National Notifiable Diseases Surveillance System received 1,317 tuberculosis (TB) notifications in 2012 and 1,263 notifications in 2013. This represents a rate of 5.8 per 100,000 population in 2012 and 5.5 per 100,000 population in 2013 and a reversal of the upward trend in TB incidence reported since 2007. In 2012 and 2013, Australia's overseas-born population continued to represent the majority of TB notifications with an incidence rate of 19.5 per 100,000 and 18.4 per 100,000 respectively. The incidence of TB in the Australian-born Indigenous population has fluctuated over the last decade; however, it remained reasonably steady in 2012 and 2013 with an incidence rate of 4.5 per 100,000 and 4.6 per 100,000 respectively. The incidence of TB in the Australian-born non-Indigenous population has continued to remain low at 0.7 per 100,000 in 2012 and 0.8 per 100,000 in 2013. Australia continued to record only a small number of multidrug resistant TB cases nationally (2012: n=20; 2013: n=22) of which nearly all were identified in the overseas-born population. This report demonstrates excellent and sustained control of TB in Australia and reflects Australia's commitment to reducing the global burden of TB. *Commun Dis Intell* 2015;39(2):E217–E235.

Keywords: Australia, *Mycobacterium tuberculosis* complex, communicable disease surveillance, epidemiology, annual report

Introduction

Despite the progress made toward global tuberculosis (TB) control and the achievement of the 2015 Millennium Development Goal (MDG) of halting and reversing incidence, TB continues to pose a significant global public health challenge with the World Health Organization (WHO) estimating that in 2013, 9.0 million people developed TB and 1.5 million died from the disease.[1] Domestically, Australia has achieved good TB control since the mid-1980s with rates ranging from 5.2 to 7.0 per 100,000 population per year, although a slight overall increase has been observed over the last decade. Controlling TB in Australia's current and future migrant population continues to pose the biggest challenge to domestic TB control, with improvements to pre-migration screening and the ongoing success of the Stop TB Strategy in the region likely to be the main contributors to further reductions in TB incidence in Australia.

Surveillance of TB in Australia is overseen by the National Tuberculosis Advisory Committee (NTAC), a subcommittee of the Communicable Diseases Network Australia (CDNA). NTAC has the key role of providing strategic, expert advice to CDNA, and subsequently the Australian Government, on a coordinated national approach to TB control. NTAC also develops and reviews nationally agreed strategic and implementation plans for the control of TB in Australia.

This report describes the epidemiology of notified cases of TB in Australia in 2012 and 2013 and includes some discussion on the factors that impact on the control of TB in Australia.

Methods

TB is a nationally notifiable disease in Australia and is monitored using the National Notifiable Diseases Surveillance System (NNDSS). Medical practitioners, public health laboratories and other health professionals are required under state and territory public health legislation to report cases of TB to jurisdictional health authorities. The *National Health Security Act 2007* provides the legislative basis for the national notification of communicable diseases and authorises the exchange of health information between the Australian Government and state and territory governments. State and territory health departments transfer these notifications regularly to the NNDSS. The primary responsibility for public health action resulting from a notification resides with state and territory health departments.

The Tuberculosis Data Quality Working Group (TBDQWG), a working group of NTAC, has representation from states and territories, the Australian Government and the Australian Mycobacterium Reference Laboratory Network. It ensures routine and timely reporting of trends and emerging issues in TB. The TBDQWG is also responsible for maintaining national consistency and currency in data standards and systems for TB surveillance that are relied upon to produce this report.

With the exception of the pre-migration screening data, the data presented in this report represent a point-in-time analysis of notified cases of TB in Australia. This report presents data extracted from NNDSS during October 2014. Due to the dynamic nature of the NNDSS, data in this report may vary from data reported in other NNDSS reports and reports of TB notifications at the state or territory level. Detailed notes on case definition, data collection, quality control and the categorisation of population subgroups are available in the 2007 annual report.[2]

In accordance with the Torres Strait Treaty, some Torres Strait Islanders and coastal people from Papua New Guinea (PNG) are allowed free movement (without passports or visas) within the northern Torres Strait Islands of Australia and PNG. This free movement is to allow for traditional activities to take place and does not include visits for health treatment.[3] However, at times PNG nationals do still present with TB to Queensland health care clinics in the Torres Strait. In these instances, the patient's diagnosis of TB is notified in Australia and identified in the NNDSS as 'Residents of the Torres Strait Protection Zone (TSPZ) accessing TB treatment in Queensland', but the patient is transferred back to PNG for treatment providing they are well enough to travel.

This report presents data analysed by date of diagnosis, a derived field within the NNDSS. The methodology for date of diagnosis for TB changed in January 2014 and was applied to notifications retrospectively. The diagnosis date for TB is now equivalent to the notification receive date*. Reported rates were calculated using population data described in the Australian Bureau of Statistics' (ABS) *Australian Demographic Statistics* and *Migration, Australia, 2011–12 and 2012–13* datasets.[4,5] Overall population rates were calculated using the 2012 and 2013 mid-year estimated resident population data, while rates for population subgroups (i.e. overseas-born), age and country of birth were calculated using 2011 mid-year estimated resident population data.

The pre-migration screening data represents a calendar year analysis of TB cases detected through the offshore pre-migration screening process. Cases of TB identified through this process are not included in the NNDSS as they are identified prior to entry to Australia. Pre-migration screening data are provided by the Australian Government Department of Immigration and Border Protection (DIBP).

* The date the notification of the disease was received by the communicable disease section of the health authority (i.e. the date the notification was received by the state or territory health department).

Results

Epidemiological situation in 2012 and 2013

In 2012, 1,317 cases of TB were reported to the NNDSS, representing a rate of 5.8 cases per 100,000 (Table 1). This was a 5% decrease on the number of TB notifications reported in 2011 (n=1,385). In 2013, 1,263 cases were reported, representing a rate of 5.5 per 100,000 and a 4% decrease on the number of notifications reported in 2012 (Table 1).

A case classification (whether new or relapse) was reported in almost all cases in both 2012 and 2013 (2012: n=1,315; 2013: n=1,261). Of those with a case classification, the majority of cases in both 2012 and 2013 were classified as new (2012: 96%, 1,261/1,317; 2013: 97%, 1,218/1,263), that is a patient who has never been treated for TB or a patient treated previously for less than one month. Relapse was reported in 54 cases in 2012 and 43 cases in 2013 with the majority of those cases (2012: 78%, 42/54; 2013: 63%, 27/43) having a treatment history of full or partial treatment overseas (Table 2).

Whilst Australia has maintained a low rate of TB since the mid-1980s, over the last 2 decades rates have been steadily increasing with Australia recording its highest rate since 1985 in 2011 (6.2 per 100,000). The decrease in rates observed in 2012 and 2013 are promising for Australian TB control; however, these lower rates will need to be sustained to have an impact on the overall trend in incidence (Figure 1).

Figure 1: Notification rates for tuberculosis, Australia, 1960 to 2013, by year

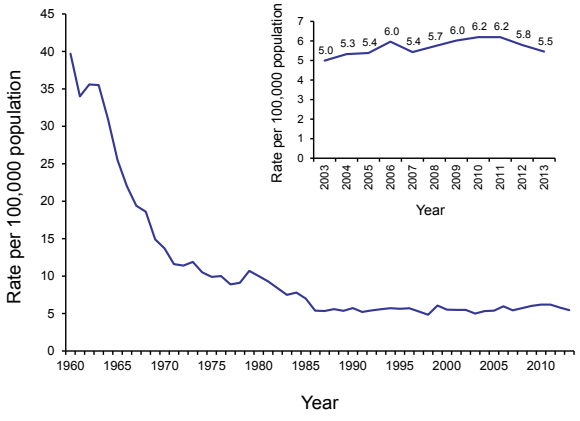


Table 1: Notified cases and notification rate for tuberculosis, Australia, 2012 and 2013, by case classification and state or territory

State or territory	New cases		Relapse cases		Total cases*	
	Notifications (n)	Rate per 100,000	Notifications (n)	Rate per 100,000	Notifications (n)	Rate per 100,000
2012						
ACT	15	4.0	3	0.8	18	4.8
NSW	439	6.0	28	0.4	469	6.4
NT	27	11.5	1	0.4	28	11.9
Qld	170	3.7	2	0.0	172	3.8
SA	83	5.0	0	0.0	83	5.0
Tas.	6	1.2	0	0.0	6	1.2
Vic.	352	6.3	17	0.3	369	6.6
WA	169	6.9	3	0.1	172	7.1
Australia	1,261	5.5	54	0.2	1,317	5.8
2013						
ACT	16	4.2	2	0.5	18	4.7
NSW	426	5.8	14	0.2	440	5.9
NT	41	17.1	1	0.4	42	17.5
Qld	149	3.2	4	0.0	154	3.3
SA	66	4.0	3	0.1	69	4.1
Tas.	8	1.6	0	0.0	8	1.6
Vic.	366	6.4	15	0.3	382	6.7
WA	146	5.8	4	0.2	150	6.0
Australia	1,218	5.3	43	0.2	1,263	5.5

* Total includes 2 cases reported in New South Wales without a case classification in 2012 and 2 cases reported without a case classification (Queensland: 1; Victoria: 1) in 2013.

Table 2: Notified cases of tuberculosis cases classified as relapse, Australia, 2012 and 2013, by treatment history

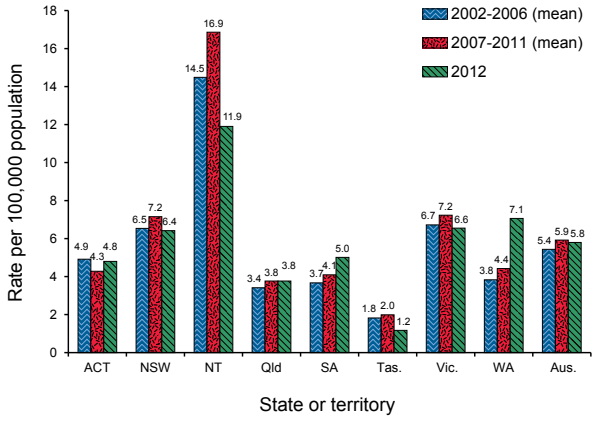
Treatment history	2012		2013	
	Notifications (n)	Percentage of relapse cases (%)	Notifications (n)	Percentage of relapse cases (%)
Relapse following full treatment only in Australia	8	15	12	28
TB following partial treatment only in Australia	4	7	4	9
Relapse following full or partial treatment overseas	42	78	27	63
Total	54	–	43	–

Geographic distribution

Similar to previous years, in 2012 and 2013 New South Wales accounted for the largest number of cases notified by a state or territory (2012: n=469; 2013: n=440) while the Northern Territory recorded the highest jurisdiction-specific rates (2012: 11.9 per 100,000; 2013: 17.5 per 100,000). Also similar to previous years, Tasmania continued to record the lowest number of cases notified (2012: n=6; 2013: n=8) and the lowest jurisdiction-specific rates (2012: 1.2 per 100,000; 2013: 1.6 per 100,000) for both years (Table 1). In 2012, South Australia and Western Australia recorded a jurisdiction-specific rate higher than the 5-year mean rate of the 2 preceding 5-year intervals (Figure 2). Western Australia recorded a rate of 7.1 per 100,000 in 2012, the highest rate recorded in Western Australia since the collection

of NNDSS data commenced in 1992 (Table 1). It is postulated that the large influx of irregular maritime arrivals peaking over both these years was a sizeable contributor to the increase in notifications in Western Australia and to a lesser extent in the Northern Territory.

Figure 2: Notification rates of tuberculosis, Australia, 2002 to 2012, by state or territory



In 2013, the Australian Capital Territory, Western Australia and the Northern Territory all recorded a jurisdiction-specific rate higher than the 5-year mean jurisdiction-specific rate of the 2 preceding 5-year intervals (Figure 3). In 2013, the Northern Territory reported 42 cases, which was a 50% increase on the number of cases reported in 2012 (n=28) (Table 1). New South Wales recorded its lowest rate since 2003, which was also 5.9 per 100,000 (Table 1).

Tuberculosis in the Australian-born population

In 2012 and 2013, the rate of TB in the Australian-born population was 0.9 per 100,000 and 1.0 per 100,000 respectively (Table 3). Indigenous Australians continued to experience a greater TB burden when compared with Australian-born non-Indigenous Australians and in both 2012 and 2013, the rate of TB in the Australian-born Indigenous population (2012: 4.5 per 100,000; 2013: 4.6 per 100,000) was approximately 6 times that of the rate of TB in the Australian-born non-Indigenous population (2012: 0.7 per 100,000; 2013: 0.8 per 100,000). The rate of TB in the Australian-born non-Indigenous population continues to remain relatively stable with a rate ranging from 0.6 per 100,000 to 0.9 per 100,000 since 2002, while the rate in the Australian-born Indigenous population demonstrates no clear trend with rates ranging from 3.1 per 100,000 to 6.3 per 100,000 (Figure 4).

Figure 3: Notification rate for tuberculosis, Australia, 2003 to 2013, by state or territory

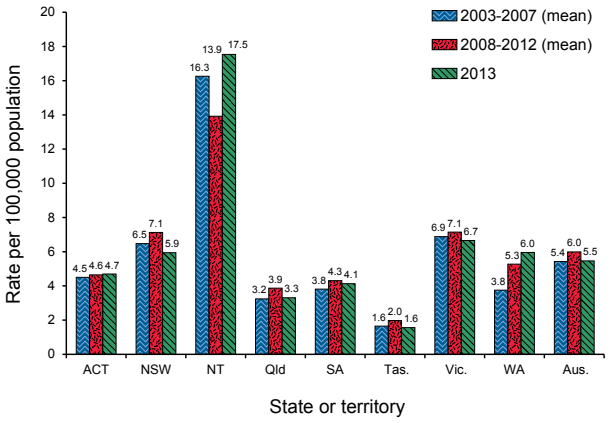
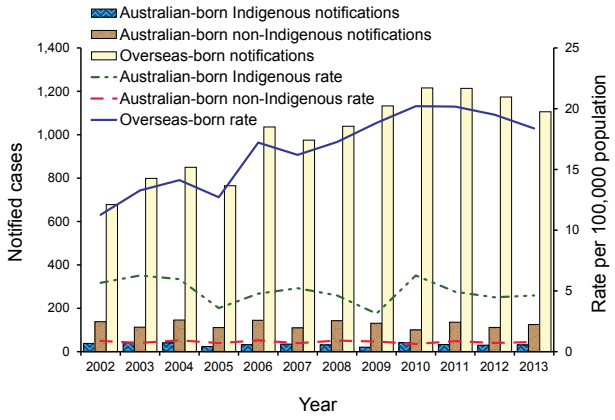


Figure 4: Notified cases and notification rate for tuberculosis, Australia, 2002 to 2013, by population subgroup



Tuberculosis in the overseas-born population

All but 1 case in 2012 and 1 case in 2013 were reported with country of birth information, with 89% (n=1,174) of notifications in 2012 and 88% (n=1,106) of notifications in 2013 being reported as overseas-born (Table 3). In 2012, the proportion of cases reported as being overseas-born ranged from 71% of cases in the Northern Territory (n=20) to 100% of cases in the Australian Capital Territory (n=18) and Tasmania (n=6). While in 2013, the proportion of cases reported as being overseas-born ranged from 55% in the Northern Territory (n=23) to 100% of cases in Tasmania (n=8).

In 2012, the rate of TB in the overseas-born population (19.5 per 100,000) was just over 22 times the rate in the Australian-born population, while the rate in the overseas-born population in 2013 (18.4 per 100,000) was just over 19 times the rate in the Australian-born population. The rate of TB in the overseas-born population has decreased by 3% in 2012 and by 6% in 2013, with the 2013 rate being the lowest recorded rate in this population sub-

Table 3: Notified cases and notification rate for tuberculosis, Australia, 2012 and 2013, by population subgroup and state or territory

State or territory	Australian-born						Overseas-born	
	Indigenous		Non-Indigenous		Total			
	Notifications (n)	Rate per 100,000	Notifications (n)	Rate per 100,000	Notifications (n)	Rate per 100,000	Notifications (n)	Rate per 100,000
2012								
ACT	0	0.0	0	0.0	0	0.0	18	18.8
NSW	7	3.4	42	0.8	49	0.9	419	20.5
NT	6	8.7	2	1.7	8	4.3	20	45.9
Qld	13	6.9	19	0.6	32	0.9	140	13.9
SA	1	2.7	9	0.7	10	0.8	73	18.8
Tas.	0	0.0	0	0.0	0	0.0	6	9.3
Vic.	2	4.2	27	0.7	29	0.7	340	21.4
WA	1	1.1	13	0.9	14	0.9	158	20.1
Australia	30	4.5	112	0.7	142	0.9	1,174	19.5
2013								
ACT	0	0.0	5	1.9	5	1.8	13	13.5
NSW	3	1.4	36	0.7	39	0.8	401	19.6
NT	15	21.8	4	3.4	19	10.1	23	52.7
Qld	9	4.8	12	0.4	21	0.6	132	13.1
SA	1	2.7	8	0.7	9	0.7	60	15.4
Tas.	0	0.0	0	0.0	0	0.0	8	12.5
Vic.	0	0.0	45	1.2	45	1.1	337	21.2
WA	3	3.4	15	1.0	18	1.1	132	16.8
Australia	31	4.6	125	0.8	156	1.0	1,106	18.4

group since 2008 (Figure 4). This figure should be interpreted with caution, given that completeness of reporting country of birth has improved over time.

Similar to recent years, the most frequently reported country of birth for TB cases in both 2012 and 2013 was India, followed by Vietnam, the Philippines, China and Nepal (Table 4). In both 2012 and 2013, people born in these 5 countries contributed to just over half of all the overseas-born cases (2012: 54%, 634/1,174; 2013: 51%, 568/1,106). Of the most frequently reported countries of birth, those born in Nepal (183 per 100,000), Ethiopia (177 per 100,000) and Papua New Guinea (150 per 100,000) recorded the highest estimated rates of TB in 2012, while those born in Somalia (243 per 100,000), Nepal (205 per 100,000) and Myanmar (160 per 100,000) recorded the highest estimated rates of TB in 2013 (Table 4).

Residency status was available for 94% of TB cases reported as overseas-born in 2012 and 95% of cases in 2013. Residency status is self-reported at the time of diagnosis and is not verified against migration records. In both 2012 and 2013, the majority of overseas-born cases reported with a residency status were reported as permanent residents (2012: 59%, 653/1,104) 2013: 60%, 636/1,056) (Table 5). International students continue to be the 2nd most reported category of residency status (2012: 14%, 155/1,104; 2013: 12%, 130/1,056). Of the most frequently reported countries of birth, the proportion of cases who were international students was highest among cases born in Nepal in both 2012 (39%, 20/51) and 2013 (37%, 21/57) followed by Pakistan (33%, 7/21), China (31%, 24/77) and Indonesia (28%, 12/43) in 2012, and Indonesia (25%,14/56), the Republic of Korea (South) (25%, 4/16) and China (22%, 16/74) in 2013 (Table 4).

There were 22 cases of TB notified among PNG nationals accessing health care in the TSPZ in 2012; a 53% decrease on the 47 cases reported in 2011. Just 3 cases were reported in 2013, which was an 86% decrease on the number of cases reported in 2012 (Table 5). In 2012 and 2013, PNG nationals being diagnosed with TB in the TSPZ accounted for 13% (22/172) and 2% (3/154) respectively of Queensland's TB cases.

Data on year of arrival were available for 97% of overseas-born cases in both 2012 (n=1,142) and 2013 (n=1,073). Of these, almost half (2012: 49%, 560/1,142; 2013: 47%, 501/1,073) were diagnosed with active TB within 4 years of arrival in Australia.

Table 4: Notified cases and notification rate for tuberculosis for frequently reported countries of birth, Australia, 2012 and 2013, by residency status

Country of birth	Residency status: International students (n)	Residency status: Permanent residents (n)	Residency status: Other* (n)	Total cases (n)	Estimated resident population†	Estimated rate per 100,000	WHO incidence rate per 100,000‡
2012							
India	40	130	102	272	337,120	81	176
Vietnam	11	100	21	132	207,620	64	147
Philippines	4	73	25	102	193,030	53	265
China§	24	38	15	77	387,420	20	73
Nepal	20	17	14	51	27,810	183	163
Papua New Guinea	2	15	29	46	30,650	150	348
Indonesia	12	16	15	43	73,060	59	185
Afghanistan	0	10	29	39	32,970	118	189
Myanmar	2	13	16	31	24,430	127	377
Sudan	0	17	10	27	22,000	123	114
Cambodia	2	15	4	21	32,510	65	411
Pakistan	7	8	6	21	34,150	61	231
Malaysia	3	13	3	19	134,140	14	80
New Zealand	0	17	1	18	543,950	3	8
Ethiopia	0	13	4	17	9,630	177	247
Thailand	3	8	5	16	52,990	30	119
Other overseas-born	25	150	67	242	–	–	–
Total overseas-born	155	653	366	1,174			
Australian-born	–	–	–	142			
Total‖	–	–	–	1,317			
2013							
India	31	101	87	219	337,120	65	176
Vietnam	6	92	12	110	207,620	53	147
Philippines	8	80	20	108	193,030	56	265
China§	16	43	15	74	387,420	19	73
Nepal	21	20	16	57	27,810	205	163
Indonesia	14	21	21	56	73,060	77	185
Afghanistan	0	13	29	42	32,970	127	189
Myanmar	1	20	18	39	24,430	160	377
Papua New Guinea	3	16	16	35	30,650	114	348
Pakistan	5	6	19	30	34,150	88	231
Sri Lanka	0	19	6	25	99,740	25	66
Cambodia	1	18	3	22	32,510	68	411
Sudan	0	14	6	20	22,000	91	114
Thailand	3	10	5	18	52,990	34	119
Korea, Republic of (South)	4	8	4	16	85,930	19	108
Somalia	0	11	5	16	6,590	243	286
Other overseas-born	17	144	58	219			
Total overseas-born	130	636	340	1,106			
Australian-born	–	–	–	156			
Total‖	–	–	–	1,263			

* Total includes cases reported without a residency status.

† Population data are sourced from the Australian Bureau of Statistics estimated resident population, at 30 June 2011, Table 9.1, Cat 3412.0.

‡ Rates for countries of birth, taken from the World Health Organization TB Burden Estimates, 2012.

§ China excludes Special Administrative Regions and Taiwan.

‖ Total includes 1 case without a reported country of birth in 2012 and 1 case without a reported country of birth in 2013.

Note that these estimated rates must be interpreted with caution as temporary residents are included in Australia's TB notifications (the numerator) but may not be included in the ABS' estimated resident population (the denominator).

Table 5: Notified cases of tuberculosis in overseas-born people, Australia, 2012 and 2013 by residency status and state or territory

Residency status	ACT	NSW	NT	Qld	SA	Tas.	Vic	WA	Aus.
2012									
Refugee/humanitarian	0	8	0	6	12	1	14	32	73
Permanent resident	13	282	6	57	9	4	201	81	653
Overseas visitor	1	28	0	17	12	0	9	6	73
Overseas student	4	59	2	15	11	0	44	20	155
Unauthorised person	0	1	9	0	0	0	1	5	16
Other	0	35	3	18	0	1	46	9	112
Illegal foreign fisher	0	0	0	0	0	0	0	0	0
Residents of the TSPZ accessing TB treatment in Queensland	N/A	N/A	N/A	22	N/A	N/A	N/A	N/A	22
Unknown or not reported	0	6	0	5	29	0	25	5	70
Total overseas-born cases	18	419	20	140	73	6	340	158	1,174
2013									
Refugee/humanitarian	0	5	0	5	6	4	17	31	68
Permanent resident	9	284	7	79	3	3	198	53	636
Overseas visitor	2	23	0	8	12	1	18	15	79
Overseas student	1	52	0	12	6	0	45	14	130
Unauthorised person	0	2	15	0	0	0	1	4	22
Other	1	34	1	15	2	0	52	13	118
Illegal foreign fisher	0	0	0	0	0	0	0	0	0
Residents of the TSPZ accessing TB treatment in Queensland	N/A	N/A	N/A	3	N/A	N/A	N/A	N/A	3
Unknown or not reported	0	1	0	10	31	0	6	2	50
Total overseas-born cases	13	401	23	132	60	8	337	132	1,106

Of those diagnosed within 4 years of arrival in Australia, the proportion of these being international students has reduced from 31% (185/593) in 2011 to 24% (133/560) in 2012 and 21% (103/501) in 2013 (Figure 5 and Figure 6). It is unclear why the proportion of cases who are international students has reduced but it may reflect more robust pre-migration screening practices.

Figure 5: Notified cases of tuberculosis in the overseas-born population, Australia, 2012, by residency status and number of years since arrival in Australia

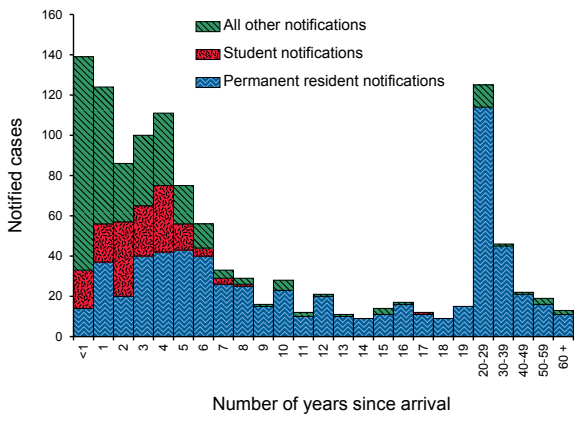
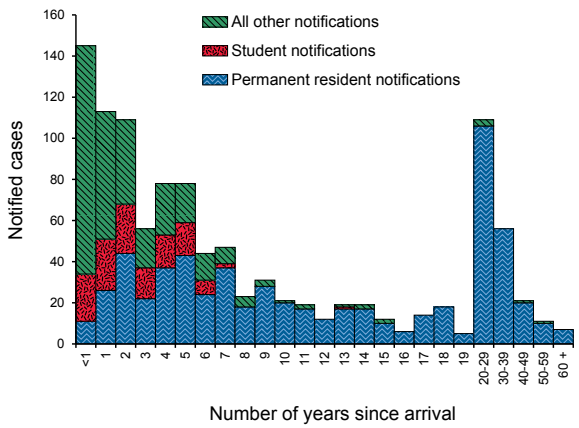


Figure 6: Notified cases of tuberculosis in the overseas-born population, Australia, 2013, by residency status and number of years since arrival in Australia



Pre-migration health screening

The Migration Regulations 1994, enabled by the *Migration Act 1958,* stipulate that visa applicants must meet certain Public Interest Criteria; and these criteria include a stipulation that visa applicants must be "[…] free from TB" and/or not be a "[…] threat to public health in Australia or a danger to the Australian community".[6] Therefore, permanent resident visa applicants, and some temporary resident visa applicants are required to undergo offshore pre-migration screening, which includes a medical examination and a chest x-ray to screen for active TB. Children aged less than 11 years of age are required to undergo a physical examination. Visa applicants who are identified as having active TB during pre-migration screening are required to undergo treatment for the disease prior to entry to Australia.[7]

In 2012, there was a 44% increase in the number of TB cases detected through offshore pre-migration screening when compared with 2011. In 2013, the number of cases continued to increase with a 13% rise compared with the number recorded in 2012 (Table 6). In both 2012 and 2013, the highest number of TB cases was identified in adults aged 21–30 years, closely followed by adults aged 31–40 years.

Table 6: Number of cases and notification rate for tuberculosis identified through offshore pre-migration health screening, 2011 to 2013

Year	Number of cases*	Estimated rate per 100,000 offshore medical examinations
2011	287	80
2012	412	116
2013	467	88

* The number of cases includes cases newly diagnosed through the pre-migration screening process and cases that were already on treatment for tuberculosis at the time of screening.

In 2012 and 2013, the majority of TB cases identified through offshore pre-migration screening (approximately 90%) were in visa applicants from countries in the South East Asian and Western Pacific World Health Organization (WHO) regions, with the Philippines, Vietnam and China contributing 61% of all cases in 2012 and 55% of all cases in 2013. Approximately 60% of all cases identified through offshore pre-migration screening in 2012 and 55% of all cases in 2013 were identified in temporary visa applicants and approximately 80% of those cases were detected in short-term visitor (less than 12 months) or student visa applicants.

The diagnostic capacity in the offshore setting has improved with nearly two-thirds of cases recorded on the database being diagnosed with laboratory confirmation; up from 50% in 2011. There was increased number of multi-drug resistant TB (MDR-TB) with 3.4% of cases with drug susceptibility testing (DST) results available in 2012 and 11.2% in 2013. Some form of resistance was observed in 26% of cases overall. This is potentially a result of both a growing resistance problem and improvements to laboratory standards in the offshore environment. In 2013, 9 separate countries had a MDR-TB case, with the majority coming from India, the Philippines and Vietnam.

Since mid-2013, DIBP has implemented an automated pre-migration screening data collection process resulting in more accurate data collection than previous years. Therefore, the comparison of pre-migration screening data to previous years should be interpreted with some caution. Further information on the pre-migration health screening process and related statistics can be obtained from DIBP's Immigration Health Branch.†

Age and sex distribution

Age and sex were reported for all TB cases notified in 2012 and 2013. Similar to previous years, there were more males than females notified with TB, with a male to female ratio of 1.3:1 in both 2012 and 2013.

As for previous years, TB was predominantly seen in young adults aged 25–34 years in both 2012 and 2013 (2012: 13.3 per 100,000; 2013: 12.0 per 100,000), and again this was driven by the high rates observed in overseas-born cases in this age group (Table 7).

Tuberculosis in children aged under 15 years

One of the most important measures of TB control is the incidence in children aged less than 15 years because these cases represent recent TB infection. Similar to the past 5 years, children aged less than 15 years contributed 4% of all TB cases in both 2012 and 2013 (2012: n=48, 1.1 per 100,000; 2013: n=48, 1.1 per 100,000). In the last decade, the number of cases in children aged less than 15 years has ranged from 37 in 2003 to 66 in 2006, and on average just over half of these notifications are recorded in overseas-born children.

In 2012 and 2013, there were three cases in children reported as Australian-born Indigenous (2012: n=1; 2013: n=2). Two of these reported

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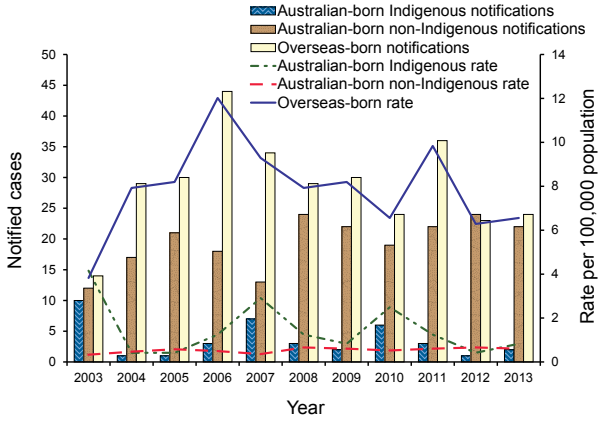
Table 7: Notification rate for tuberculosis, Australia, 2012 and 2013, by population subgroup and age group

Age group	Australian-born Indigenous		Australian-born non-Indigenous		Overseas-born		Total	
	2012	2013	2012	2013	2012	2013	2012	2013
0–4	–	–	1.4	1.3	9.4	10.9	1.6	1.6
5–14	0.6	1.3	0.3	0.2	5.6	5.6	0.9	0.9
0–14	0.4	0.8	0.7	0.6	6.3	6.6	1.1	1.1
15–24	3.7	3.0	0.7	0.7	29.1	29.9	6.5	6.7
25–34	4.4	–	0.8	0.5	38.8	35.7	13.3	12.0
35–44	6.1	9.8	0.3	0.7	17.6	19.0	5.8	6.5
45–54	11.1	19.0	0.4	0.8	12.0	9.7	4.5	4.1
55–64	19.0	8.2	0.7	0.7	10.6	11.4	4.4	4.6
65+	4.4	8.8	1.4	1.8	16.1	12.7	6.8	5.8

a 'household member or close contact with TB' and no risk factor information was provided for the 3rd case. Of the Australian-born non-Indigenous cases in children, 46% in 2012 (11/24) and 41% (9/22) in 2013 reported a 'household member or close contact with TB' as the only risk factor.

The rate of TB in Australian-born non-Indigenous children has remained relatively stable over the past decade (range: 0.3 per 100,000 to 0.7 per 100,000), whilst the rate in Australian-born Indigenous (range: 0.4 per 100,000 to 4.2 per 100,000) and overseas-born children (range: 3.8 per 100,000 to 12.0 per 100,000) has fluctuated over that time (Figure 7).

Figure 7: Notified cases and notification rate for tuberculosis in children aged less than 15 years, Australia, 2003 to 2013, by population subgroup



Selected risk factors for tuberculosis

Selected risk factor data were provided for 92% (n=1,212) of notified cases in 2012 and 92% (n=1,162) in 2013. Of those cases assessed for risk factors, the most frequently reported risk factor in both 2012 and 2013 was 'past travel to or residence in a high-risk country' (2012: 84%, n=1,014; 2013: 81%, n=947) (Table 8). Since 2011, the proportion of cases with information available who had this risk factor increased from approximately 60% to approximately 80%. The increase is in part due to Victoria initiating reporting of this risk factor to the national dataset from 2011. Interpretation of this risk factor in overseas-born cases is problematic as at the time these data were collected there were inconsistent practices across states and territories as to the inclusion of a case's country of birth in the assessment of this risk factor. NTAC has agreed that this risk factor is to identify travel-related TB and as such is intended to be exclusive of a case's country of birth. The reporting and interpretation of this risk factor should be clearer post-2013.

In 2012, the most frequently reported risk factor among overseas-born cases and Australian-born non-Indigenous cases was 'past travel to or residence in a high-risk country' (n=978 and n=36, respectively). In 2013, the most frequently reported risk factor among overseas-born cases was again 'past travel to or residence in a high-risk country' (n=913) whilst the Australian-born non-Indigenous cases more frequently reported 'none of the above risk factors' (n=29). In both 2012 and 2013, the most frequently reported risk factor among Australian-born Indigenous cases was a 'household member or close contact with TB' (2012: n=16; 2013: n=15) (Table 8).

A total of 67 cases of TB in 2012 and 77 in 2013 were reported in people who were currently or had previously worked in a health care setting. Of these, 27 in 2012 and 24 in 2013 were working in a health care setting in Australia at the time of diagnosis or within 12 months of diagnosis. 63% (n=17) of

Table 8: Notified cases of tuberculosis, Australia, 2012 and 2013, by population subgroup and selected risk factors

Risk factor*	Australian-born Indigenous	Australian-born non-Indigenous	Overseas-born	Total
2012				
Household or other close contact with tuberculosis	16	26	89	131
Ever resided in a correctional facility†	0	1	9	10
Ever resided in an aged care facility†	0	0	3	3
Ever employed in an institution†‡	0	0	9	9
Currently or previously† employed in health industry in Australia or overseas	0	4	63	67
Ever homeless	1	1	5	7
Past travel to or residence in a high-risk country	0	36	978	1,014
Chest X-ray suggestive of old untreated tuberculosis	0	1	19	20
Currently receiving immunosuppressive therapy	0	4	27	31
Australian-born child with one or more parent born in a high-risk country	0	9	0	9
None of the above risk factors	7	29	75	111
Total cases assessed for risk factors	23	100	1,089	1,212
2013				
Household or other close contact with tuberculosis	15	24	89	128
Ever resided in a correctional facility†	1	0	12	13
Ever resided in an aged care facility†	0	3	3	6
Ever employed in an institution†‡	0	2	5	7
Currently or previously† employed in health industry in Australia or overseas	3	4	70	77
Ever homeless	0	2	6	8
Past travel to or residence in a high-risk country	0	34	913	947
Chest X-ray suggestive of old untreated tuberculosis	0	3	34	37
Currently receiving immunosuppressive therapy	0	7	27	34
Australian-born child with one or more parent born in a high-risk country	0	13	1	14
None of the above risk factors	8	38	67	113
Total cases assessed for risk factors	26	109	1,027	1,162

* More than one risk factor may be reported for each notified case of tuberculosis.

† Within the preceding 5 years.

‡ Institution is defined as a correctional facility, aged care facility or homeless shelter.

those cases in 2012 and 50% (n=12) in 2013 were reported as having extrapulmonary disease only, which is generally not communicable.

Tuberculosis and HIV status

According to Australia's *2011 National HIV Testing Policy version 1.3*, '…all people with HIV should be tested for tuberculosis, and all people with tuberculosis should be tested for HIV…'[8]. The HIV testing history‡ of notified cases of TB were reported in 97% of cases in 2012 (n=1,281) and 96% of cases in 2013 (n=1,215). Of those cases, just over 80% were tested for HIV (2012: 81%, n=1,033; 2013: 83%, n=1,004) (Table 9).

More than half of the cases with a known HIV test history in both 2012 and 2013 were reported with a known HIV status (2012: 58%, 741/1,281; 2013: 56%, 683/1,215), of which 1.5% in 2012 (n=11) and 3.4% in 2013 (n=23) were reported as being HIV positive (Table 9).

Approximately a quarter of cases in 2012 (23%, n=292) and 2013 (26%, n=321) with a known

‡ HIV test history means knowing whether or not the person was tested for HIV, not tested for HIV or refused testing for HIV.

HIV testing history were reported as being tested for HIV but the result of that test has not been reported to the NNDSS. Nearly all these cases were reported by Victoria (2012: n=287; 2013: n=310) where policy has prevented the HIV status of an individual being reported against their TB notification.

Anatomical site of disease

The anatomical site of TB disease was recorded in nearly all notified cases in 2012 (n=1,315) and 2013 (n=1,262). Similar to previous years, pulmonary disease was the most frequently reported site of disease in both 2012 (60%, n=796) and 2013 (58%, n=734), with most of these cases being reported as having pulmonary disease only. Extrapulmonary disease only was reported in 39% (n=519) of all cases in 2012 and 42% (n=528) in 2013, with the most frequently reported extrapulmonary site of disease being lymph nodes (2012: n=259; 2013: n=268) (Table 10). In both 2012 and 2013, children aged less than 15 years made up only 3% of the total cases of extrapulmonary TB (2012: 16/519; 2013: 18/528) and interestingly have a lower proportion of cases with this form of the disease than found overall for the population (2012: 33%, 16/48; 2013: 38%, 18/48. Of the more severe forms of TB, 4 cases in 2012 and 9 cases in 2013 were classified as miliary, while 9 cases in 2012 and 6 cases in 2013 were classified as meningeal. This is similar to the number of miliary and meningeal cases reported in previous years.

Bacteriologically confirmed cases

The majority of cases in 2012 (n=1,146, 87%) and 2013 (n=1,068, 85%) were bacteriologically and/or histological confirmed as TB. The remaining 13% (n=171) and 15% (n=195) of cases in 2012 and 2013 respectively were diagnosed using clinical and radiological evidence.

Of the total number of cases with pulmonary disease§, 79% (632/796) in 2012 and 75% (547/734) in 2013 were either sputum culture positive or bronchoscopy washings/aspirate culture positive with nearly half of these cases also being smear positive (2012: 47%, 294/632; 2013: 44%, 243/547). Smear positive cases of pulmonary TB can be up to 10 times more infectious than smear negative cases and are usually the main source of TB transmission in the community.[9,10]

Of the extrapulmonary only cases, 60% (311/519) in 2012 and 55% (289/528) in 2013 were culture positive. Cases with extrapulmonary disease only are generally not infectious and rarely are a source of transmission.[9] Of the extrapulmonary only cases reported in children aged less than 15 years, 63% (10/16) in 2012 and 50% (9/18) in 2013 were bacteriologically confirmed. The WHO recommends that wherever possible, a diagnosis of TB in a child should be bacteriologically confirmed. [11]

§ Pulmonary cases include both pulmonary only cases and pulmonary cases that also have extrapulmonary sites detected.

Table 9: Notified cases of tuberculosis, Australia, 2012 and 2013, by population subgroup and HIV status

HIV testing history	Australian-born Indigenous	Australian-born non-Indigenous	Overseas-born	Unknown population subgroup	Total
2012					
HIV positive	0	3	8	0	11
HIV negative	25	54	651	0	730
HIV tested, result unknown	1	14	277	0	292
Not tested	2	33	207	1	243
Refused testing	1	1	3	0	5
HIV testing history unknown	0	7	28	1	36
Total	29	112	1,174	2	1,317
2013					
HIV positive	0	3	19	1	23
HIV negative	24	58	578	0	660
HIV tested, result unknown	0	21	300	0	321
Not tested	4	37	164	0	205
Refused testing	1	0	5	0	6
HIV testing history unknown	2	6	40	0	48
Total	31	125	1,106	1	1,263

Table 10: Notified cases of tuberculosis, Australia, 2012 and 2013, by case classification and site of disease

Site	New cases	Relapse cases	Total cases*	Percentage of cases (%)
2012				
Pulmonary				
Pulmonary only	630	28	659	50.0
Pulmonary plus other sites	129	8	137	10.4
Pulmonary - total	759	36	796	60.4
Extrapulmonary only†				
Pleural	82	1	83	6.3
Lymph nodes	247	11	259	19.7
Bone/joint	36	4	40	3.0
Genitourinary	19	0	19	1.4
Miliary	4	0	4	0.3
Meningeal	9	0	9	0.7
Peritoneal	26	0	26	2.0
Other	153	4	157	11.9
Unknown extrapulmonary site	1	0	1	0.1
Extrapulmonary – total	500	18	519	39.4
Unknown site of disease – total	2	0	2	0.2
Total	1261	54	1317	100.0
2013				
Pulmonary				
Pulmonary only	589	26	616	48.8
Pulmonary plus other sites	117	1	118	9.3
Pulmonary – total	706	27	734	58.1
Extrapulmonary only†				
Pleural	63	3	66	5.2
Lymph nodes	255	12	268	21.2
Bone/joint	35	1	36	2.9
Genitourinary	27	0	27	2.1
Miliary	9	0	9	0.7
Meningeal	6	0	6	0.5
Peritoneal	35	0	35	2.8
Other	102	0	102	8.1
Unknown extrapulmonary site	1	0	1	0.1
Extrapulmonary – total	511	16	528	41.8
Unknown site of disease – total	1	0	1	0.1
Total	1,218	43	1,263	100.0

* Total includes 1 pulmonary case and 1 extrapulmonary case reported without a case classification in 2012 and 1 pulmonary case and 1 extrapulmonary case reported without a case classification in 2013.

† More than one extrapulmonary site may be reported for each notified case of tuberculosis.

It is recommended that a bronchoscopy should not be performed on patients with known active tuberculosis (i.e. sputum smear positive) unless absolutely necessary as this presents an increased risk of TB transmission to future bronchoscope patients and clinical staff.[12,13] Of the bacteriologically confirmed cases, 17% (191/1,146) of cases in 2012 and 20% (208/1,068) of cases in 2013 recorded a positive microscopy or culture result on a bronchoscopy obtained washing or aspirate. Of these cases, 14% (27/191) in 2012 and 34% (71/208) in

2013 also recorded a sputum smear positive result with one of those cases in 2013 being identified as MDR-TB.

Of the bacteriologically confirmed cases, 2% in 2012 (28/1,146) and 5% (54/1,068) in 2013 were reported as being confirmed using a nucleic acid testing (NAT) method only. In Australia, culture remains the gold standard diagnostic for confirming TB cases as it is more sensitive than NAT and it provides a bacterial isolate for DST and molecular typing.[14]

Drug resistant tuberculosis in Australia

The results of DST were available for 3 quarters of the TB cases notified in 2012 (75%, 988/1,317) and 2013 (73%, 928/1,263) and of those cases, resistance to at least one of the standard first line anti-tuberculosis agents was identified in 13% of cases (2012: 129/988; 2013: 120/928). Resistance to rifampicin only (mono-resistance) remains low and is reported in only 0.3% cases with DST results available in both 2012 (3/988) and 2013 (3/928). Mono-resistance to isoniazid was more common than rifampicin mono-resistance but still relatively low and was reported in 4% (42/988) of cases in 2012 and 5% (48/928) of cases in 2013. Similar to previous years, resistance to at least rifampicin and isoniazid, known as MDR-TB, was identified in approximately 2% of cases with DST results in both 2012 (20/988) and 2013 (22/928). There were no cases of extensively drug resistant TB (XDR-TB) reported in 2012 or 2013 (Table 11).

The majority of MDR-TB cases were reported in the overseas-born population (2012: n=18; 2013: n=20) and of those, 7 cases in 2012 and 1 case in 2013 were identified as residents of the TSPZ accessing TB treatment in Queensland. The remaining MDR-TB cases (2012: n=2; 2013: n=2) were reported in the Australian-born non-Indigenous population. Of these cases, 1 case in 2012 was recorded as having completed treatment, while the other case in 2012 was reported as defaulting from treatment. Both cases in 2013 are currently reported as being still under treatment.

Treatment outcomes of 2011 and 2012 tuberculosis patient cohorts

The treatment outcomes of an annual patient cohort are reported in the following year's annual report. This allows adequate time for all cases notified in a single year to begin treatment and for the treatment outcomes to be recorded in the NNDSS. Treatment outcomes for the 2011 and 2012 patient cohorts are reported in this annual report; and treatment outcomes for the 2013 patient cohort will be reported in the 2014 annual report.

2011 tuberculosis patient cohort

In 2011, treatment success, which includes those bacteriologically confirmed as cured and those who completed treatment, was reported in 96% (1,178/1,233) of cases with assessable outcomes (Table 12). Treatment success ranged from 77% in Australian-born Indigenous cases to 96% in overseas-born cases. In 2011, there were 2 cases of a treatment failure reported, one in an Australian-born Indigenous case and one in an overseas-born case, and 20 (1.6%) cases were reported to have died due to TB.

Table 11: Notified cases of tuberculosis, Australia, 2012 and 2013, by drug susceptibility testing profile

Drug susceptibility testing (DST) profile	2012		2013	
	Notifications (n)	Percentage of cases with DST results (%)	Notifications (n)	Percentage of cases with DST results (%)
Total cases with DST results	988	–	928	–
Resistance to at least one first line anti-tuberculosis agents*	129	13.1	120	12.9
Mono-resistance to rifampicin	3	0.3	3	0.3
Mono-resistance to isoniazid	42	4.3	48	5.2
MDR-TB†	20	2.0	22	2.4
XDR-TB‡	0	0.0	0	0.0

* Isoniazid, rifampicin, pyrazinamide, ethambutol and streptomycin.

† Multi-drug resistant tuberculosis: resistance to isoniazid and rifampicin.

‡ Extensively drug resistant tuberculosis: resistance to isoniazid and rifampicin, and any of the fluoroquinolones, and to at least one of the 3 injectable second-line drugs.[15]

Table 12: Notified cases of tuberculosis, Australia, 2011, by population subgroup and treatment outcome

Treatment outcome	Australian-born Indigenous cases		Australian-born non-Indigenous		Overseas-born		Total cases*	
	Notifications (n)	Percentage assessable (%)	Notifications (n)	Percentage assessable (%)	Notifications (n)	Percentage assessable (%)	Notifications (n)	Percentage assessable (%)
Assessable outcomes								
Treatment success	20	76.9	117	94.4	1,040	96.1	1,178	95.5
Cured (bacteriologically confirmed)†	3	11.5	10	8.1	35	3.2	48	3.9
Completed treatment	17	65.4	107	86.3	1,005	92.9	1,130	91.6
Interrupted treatment‡	1	3.8	0	0.0	2	0.2	3	0.2
Died of tuberculosis	1	3.8	5	4.0	14	1.3	20	1.6
Defaulted§	0	0.0	1	0.8	18	1.7	19	1.5
Failure‖	1	3.8	0	0.0	1	0.1	2	0.2
Not followed up, outcome unknown	3	11.5	1	0.8	7	0.6	11	0.9
Total assessable	26	100.0	124	100.0	1,082	100.0	1,233	100.0
Non-assessable outcomes								
Transferred out of Australia	0	0.0	2	1.5	106	8.7	108	7.8
Died of other causes	7	21.2	9	6.6	25	2.1	41	3.0
Still under treatment	0	0.0	1	0.7	1	0.1	2	0.1
Total	33	100.0	136	100.0	1,214	100.0	1,384	100.0

* Total includes 1 case reported with an unknown population subgroup.

† Cured is defined as the bacteriologically confirmed sputum smear and culture positive at the start of treatment and culture negative in the final month of treatment and on at least one previous occasion.

‡ Interrupted treatment is defined as treatment interrupted for 2 months or more but completed

§ Defaulted is defined as failed to complete treatment.

‖ Failure is defined as sputum culture positive at 5 months or later.

2012 tuberculosis patient cohort

In 2012, treatment success, which includes those bacteriologically confirmed as cured and those who completed treatment, was reported in 95% (1,124/1,181) of cases with assessable outcomes (Table 13). Treatment success ranged from 86% in Australian-born Indigenous cases to 96% in overseas-born cases. In 2012, there were 2 cases of a treatment failure reported, one in an Australian-born Indigenous case and one in an overseas-born case, and 14 (1.2%) cases were reported to have died due to TB.

National performance indicators

In 2011, 2012 and 2013, the performance criterion for annual incidence (less than 1 per 100,000) was met only in the Australian born non-Indigenous cases and incidence rates in Australian born children continue to exceed the performance criteria of less than 0.1 per 100,000. The reporting of HIV testing history has improved over time but still remains just short of the reaching the target of 100%. In 2011 and 2012, outcome reporting fell just short of the performance criteria with 1% of cases with assessable outcomes in 2011 and 2% in 2012 reported with an unknown outcome (Table 14). Overall the performance indicators for treatment success and treatment failure were achieved in both 2011 and 2012; however, at a sub-population level these indicators were only achieved in the Australian-born non-Indigenous and overseas born cases, and not in the Australian born Indigenous cases (Table 13).

Discussion

Although Australia has maintained a low incidence of TB, over the past decade, the rate of TB had been showing a steady upward trend, largely attributed to the growth in immigration from high TB burden countries. The rate had increased from 5 per 100,000 in 2003 to highs of 6.2 per 100,000 population in 2010 and 2011.

For this 2 year reporting period, the trend has reversed with successive annual declines amounting to a net reduction of 8.8% in overall case numbers. The case rate of 5.5 per 100,000 in 2013 is the lowest since 2007. This decline mirrors a fall in the number of persons from high TB burden countries notified with TB. The substantial increase in TB detected offshore through the pre-migration screening process may be a contributing factor to this change (44% increase 2011 to 2012; 13% increase 2012 to 2013). Also during this period the absolute number of overseas students and the proportion of cases they contribute to the overseas-born TB case-load fell from 224 in 2011 (18.5%) to 155 in 2012 (13.2%) and 130 in 2013 (11.8%). The reasons for the latter are unclear.

New South Wales and Victoria between them accounted for approximately two-thirds of all cases in 2012 and 2013. This disproportionate share of the TB burden has not changed significantly since 2010 although New South Wales has experienced a notable decline in notifications and rate from 541 (7.5 per 100,000) in 2011 to 469 (6.4 per 100,000) in 2012 and 440 (5.9 per 100,000) in 2013. In contrast the Northern Territory reported the highest rates (11.9 and 17.5 per 100,000), although significantly lower notification numbers, that predominantly relate to the Indigenous Australian and overseas-born groups. Western Australia and South Australia also have experienced higher rates than previously. These geographic fluctuations may reflect changing patterns in migrant and temporary resident placements across the states and territories. In Queensland, the number of notifications detected in PNG nationals in the TSPZ with TB and drug resistant TB has fallen dramatically from 47 cases in 2011 to three in 2013. In mid-2012, the policy regarding the management of cross-border patients in the Torres Strait was amended and resulted in a more stringent enforcement of the Torres Strait Treaty rules regarding health treatment. This likely contributed to the decreased number of PNG nationals diagnosed over that time period.

Australian-born Indigenous people continue to experience TB at a rate about 6 times that of Australian-born non-Indigenous people, although the actual rate by international standards is low (< 5 per 100,000) and the proportion of all notifications small (2.5%–3%). The key issue however, is that socioeconomic conditions in some Indigenous communities remain conducive for TB to prosper and in such settings, TB can be more problematic to manage. In contrast, TB rates in Australian-born non-Indigenous people have remained stable and consistently below 1 per 100,000 over the past decade at the pre-elimination level. Of these cases, when examined by risk factors, household or close TB contact was identified in 26% (2012) and 22% (2013) of cases and past travel or residence in a high risk country in 36% (2012) and 31% (2013) of cases. Approximately 10% had at least 1 overseas-born parent but in a 3rd of cases no risk factors were identified. While many of these cases may be unavoidable, if the goal of elimination is to be achieved (<1 per million) then further TB preventive initiatives are required.

Overseas-born persons continue to drive the epidemiology of TB in Australia, contributing nearly 90% of all cases. The overall rates of disease are

Table 13: Notified cases of tuberculosis, Australia, 2012, by population subgroup and treatment outcome

Treatment outcome	Australian-born Indigenous cases		Australian-born non-Indigenous		Overseas-born		Total cases*	
	Notifications (n)	Percentage assessable %)	Notifications (n)	Percentage assessable (%)	Notifications (n)	Percentage assessable (%)	Notifications (n)	Percentage assessable (%)
Assessable outcomes								
Treatment success	24	85.7	98	90.7	1,002	96.0	1,124	95.2
Cured (bacteriologically confirmed)†	4	14.3	3	2.8	50	4.8	57	4.8
Completed treatment	20	71.4	95	88.0	952	91.2	1,067	90.3
Interrupted treatment‡	0	0.0	1	0.9	1	0.1	2	0.2
Died of tuberculosis	1	3.6	1	0.9	11	1.1	14	1.2
Defaulted§	0	0.0	3	2.8	13	1.2	16	1.4
Failure‖	1	3.6	0	0.0	1	0.1	2	0.2
Not followed up, outcome unknown	2	7.1	5	4.6	16	1.5	23	1.9
Total assessable	28	100.0	108	100.0	1,044	100.0	1,181	100.0
Non-assessable outcomes								
Transferred out of Australia	0	0.0	1	0.9	86	7.3	87	6.6
Died of other causes	1	3.4	2	1.8	32	2.7	36	2.7
Still under treatment	0	0.0	1	0.9	12	1.0	13	1.0
Total	29	100.0	112	100.0	1,174	100.0	1,317	100.0

* Total includes 2 cases reported with an unknown population subgroup.

† Cured is defined as the bacteriologically confirmed sputum smear and culture positive at the start of treatment and culture negative in the final month of treatment and on at least one previous occasion.

‡ Interrupted treatment is defined as treatment interrupted for 2 months or more but completed

§ Defaulted is defined as failed to complete treatment.

‖ Failure is defined as sputum culture positive at 5 months or later.

Table 14: National tuberculosis performance indicators, performance criteria* and the current status of tuberculosis, Australia, 2011 to 2013

National tuberculosis performance indicator	Performance criteria	2013	2012	2011
Annual incidence of TB (cases per 100,000 population)				
Total	<6.0†	5.5	5.8	6.2
Australian-born Indigenous Australians	<1.0	4.6	4.5	4.9
Australian-born non-Indigenous Australians	<1.0	0.8	0.7	0.9
Overseas-born persons	*	18.4	19.5	20.2
Incidence in children <15 years, by risk group (per 100,000 population)				
Australian-born Indigenous Australians	<0.1	0.8	0.4	1.2
Australian-born non-Indigenous Australians	<0.1	0.6	0.7	0.4
Overseas-born persons	*	6.6	6.3	9.0
Collection of HIV testing history				
Collection of HIV testing history in all tuberculosis cases	100%	96%	97%	98%
Treatment outcome measures (%)				
Cases evaluated for outcomes	100%	TBA‡	98%	99%
Cases that have treatment completed and are cured (treatment success)	>90%	TBA‡	95%	96%
Cases recorded as treatment failures	<2%	TBA‡	0.2%	0.2%

* Performance criteria currently under review.

† This performance criterion is based on the key performance indicator published in the DIBP 2012–13 and 2013–14 Portfolio Budget Statements under Program 1.1: Visa and Migration.

‡ TBA is to be assessed: 2013 patient cohort outcomes to be reported in 2014 annual report.

more than 20 times higher than for the Australian-born population and reflect rates in the countries of origin. The highest proportions of cases were found in permanent residents (54% in 2012, 57% in 2013) and overseas students. Individuals from India, Vietnam, the Philippines, China and Nepal, as in recent years, contributed more than 50% of cases. In most instances, TB is attributed to reactivation of 'imported' infection rather than from recent transmission within Australia. Given the expected high rates of TB infection in the overseas-born and that nearly 50% of cases are occurring more than 5 years after arrival, without increased attempts at identifying latent TB infection the rate of decline of TB will be slow and the pool of infected persons in the Australian population will continue to increase with future implications for TB control.

Cases classified as 'relapse' are a potential indicator of TB program performance (past or recent). The proportion of cases in this group remains low and in both years was lower than the 2011 figure of 4.8% (2012: 54, 4.1%; 2013: 43, 3.4%). The majority of these cases occurred in persons previously treated overseas and hence do not reflect on the quality of TB management in Australia. If these cases are excluded, then for those previously treated in Australia, very low numbers were reported (2012: 12, 0.9%; 2013: 16, 1.2%). From the surveillance data, it is not possible to determine whether these cases related to recent or more distant previous treatment or to classify possible contributing factors such as treatment quality or non-adherence, drug resistance or possible re-infection.

The proportion of cases that are smear positive for TB in a sample obtained by bronchoscopy but also in a diagnostic sputum specimen may be an indicator of suboptimal clinical practice. Approximately 20% of pulmonary cases in both 2012 and 2013 were bacteriologically confirmed from bronchoscopy specimens. The concern is that of these cases, 14% and 34% in 2012 and 2013 respectively were also confirmed smear positive from diagnostic sputum specimens. Risk to staff is an important consideration particularly when multidrug-resistance is involved. However, as it is often common practice to obtain post-bronchoscopy spontaneous sputum samples for smear and culture, further investigation is required to confirm whether the proportions of cases above were post bronchoscopy collections and/or whether the collection of a pre-bronchoscopy sputum sample was considered. If there is difficulty in obtaining spontaneous

sputum specimens for diagnosis, then induced sputum in a negative pressure setting is strongly encouraged as the next step.

The proportion of TB occurring in children is an important indicator of recent transmission of infection in the community. The proportion of cases reported to have occurred in people less than 15 years of age remained steady at 4% of all cases, with overseas-born children contributing approximately half of these. In both Australian-born Indigenous and Australian-born non-Indigenous children, case rates were less than 1 per 100,000 and in nearly half of the cases, close contact with TB was reported as a risk factor. These cases may have represented a missed opportunity for earlier intervention with TB preventive therapy. In the Australian-born Indigenous population, although case numbers were small, the ability of TB programs to respond in a timely manner to a new case remains paramount.

Acquired drug resistance is a marker of TB program performance. Drug resistant TB in Australia remains at low levels. MDR-TB (resistance to at least isoniazid and rifampicin) was reported in only 2% of cases, of which 90% of cases were in overseas-born persons. Mono-resistance to rifampicin is considered as significant as MDR-TB in terms of treatment requirements but was only found in 0.3% of cases. No cases of XDR-TB were reported. Future cases are inevitable, but the low number of these cases and their problematic nature mean that management should be undertaken by, or in close collaboration with, the specialised TB services at state level.

The incidence of TB in health care workers continues to be of interest because of the increased reliance on overseas health care workers from high burden regions. The proportion of cases reported has remained relatively stable since 2010, contributing 5.5% of all cases in 2012 and 6.6% in 2013. As indicated in the 2011 report, more than 90% of these cases occurred in overseas-born people and invariably reflect reactivation of infection acquired in their country of origin. Only about a third of these cases however, were working in a health institution at the time or within 12 months of diagnosis and greater than 50% of cases were reported as extrapulmonary suggesting that the overall risk of transmission in the workplace is not high. Nevertheless, this requires continued monitoring.

Testing of HIV status in all TB cases is recommended nationally, although until recently, data reporting has varied between states and territories. Although high rates of reporting of testing history have been achieved, only about 80% were tested for HIV and of these, HIV results were obtained for 58% and 56% of cases in 2012 and 2013 respectively. Despite this, HIV co-infection appears to have minimal impact on TB control in Australia.

Monitoring treatment outcomes provides an important measure of the quality of treatment programs. Despite the slight upward trend in the TB case-load observed over the last decade, high rates of treatment success have been maintained and few adverse outcomes (treatment failure, deaths) have been reported, supporting overall good TB control efforts across the country. Treatment success rates remain at high levels (≥ 95%) by international standards and consistently exceed the national performance and WHO criteria of greater than 90%. However, in the Australian-born Indigenous population, success rates were only 76.9% and 85.7% in 2011 and 2012 respectively. These figures need to be interpreted with caution, because of the small case numbers involved. In all population groups the rate of adverse outcomes (treatment failure, treatment default or TB related deaths) was low at approximately 3% in total, with cases classified as treatment failure only 0.2%.

Overall, this report, as in previous years, demonstrates that Australia's jurisdictional TB programs achieve a very high level of diagnostic and treatment success, and thereby maintenance of one of the lowest rates of TB in the world. In addition, Australia is probably improving pre-migration detection of TB, which is leading to a fall in the incidence of TB in Australia. However, in 2014 the Australian Government endorsed the World Health Assembly's *Global Strategy and targets for tuberculosis prevention, care and control after 2015,* which sets targets for ending the global TB epidemic.[16] NTAC is currently drafting a new strategic plan for 2016–2020, which will align with the WHO's framework for low-incidence countries and primarily consider the actions needed to achieve TB elimination targets.[17]

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Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet network, 2011

The OzFoodNet Working Group

Abstract

This report summarises the incidence of diseases potentially transmitted by food in Australia and details outbreaks associated with food in 2011. OzFoodNet sites reported 30,957 notifications of 9 diseases or conditions that may be transmitted by food. The most commonly notified infections were *Campylobacter* (17,733 notifications) followed by *Salmonella* (12,271 notifications). The most frequently notified *Salmonella* serotype was *Salmonella* Typhimurium, accounting for 48% of all *Salmonella* notifications. OzFoodNet sites also reported 1,719 outbreaks of gastrointestinal illness affecting 29,839 people and resulting in 872 people being hospitalised and 103 associated deaths. The majority of outbreaks (79% 1,352/1,719) were due to person-to-person transmission, 9% (151/1,719) were suspected or confirmed to be foodborne, 11% (192/1,719) were due to an unknown mode of transmission, 19 were due to community based *Salmonella* clusters, four were due to waterborne or suspected waterborne transmission and 1 outbreak was due to animal-to-person transmission. Foodborne and suspected foodborne outbreaks affected 2,104 persons and included 231 hospitalisations. There were 5 deaths reported during these outbreaks. *Salmonella* was the most common aetiological agent identified in foodborne outbreaks and restaurants were the most frequently reported food preparation setting. A single food source of infection was identified for 49 outbreaks, 26 of which were associated with the consumption of dishes containing raw or minimally cooked eggs and all of these outbreaks were due to *S.* Typhimurium. These data assist agencies to document sources of foodborne disease, develop food safety policies, and prevent foodborne illness. *Commun Dis Intell* 2015;39(2):E236–E264.

Introduction

In Australia, an estimated 4.1 million domestically acquired cases of foodborne gastroenteritis occur annually, costing an estimated $1.2 billion per year.[1–3] Many of these illnesses are preventable by appropriate interventions. Foodborne disease surveillance can be used to gather evidence to help inform appropriate control measures.[4] Health departments conduct surveillance for foodborne diseases and diseases potentially transmitted by food to monitor trends in illness, detect outbreaks, inform preventative measures and to evaluate the efficacy of public health interventions.[5–7]

Most foodborne diseases manifest as mild self-limiting gastroenteritis, with around 28% of affected people seeking medical attention.[1] Consequently, surveillance data collected by health departments underestimate the true burden of disease. In Australia, for every case of salmonellosis notified to a health department there are an estimated 7 infections that occur in the community, while there are approximately 8 cases in the community for every notified case of Shiga toxin-producing *Escherichia coli* (STEC) and 10 cases in the community for every notified case of campylobacteriosis.[8–10]

In Australia, state and territory health departments conduct surveillance for between 10 and 15 different diseases that may be transmitted through food. Most of these are also transmitted by the faecal–oral route and as such may be transmitted by contact with infected animals, environments, or people, and may be acquired domestically or overseas. They may also be transmitted by contaminated food preparation equipment or surfaces, or through the consumption of contaminated water. Health departments collect summary data on notified outbreaks of foodborne diseases, providing robust information on the contaminated foods that are causing illness in Australia.

The Australian Government established OzFoodNet—Australia's enhanced foodborne disease surveillance system—in 2000 to improve national surveillance and conduct applied research into the causes of foodborne illness.[11] OzFoodNet aggregates and analyses national-level information on the incidence of diseases caused by pathogens commonly transmitted by food, as well as investigating foodborne disease outbreaks. The OzFoodNet network in 2011 included foodborne disease epidemiologists from each state and territory health department and collaborators from the Public Health Laboratory Network (PHLN), Food Standards Australia New Zealand

(FSANZ), the then Department of Agriculture, Fisheries and Forestry, and the National Centre for Epidemiology and Population Health at the Australian National University. OzFoodNet is a member of the Communicable Diseases Network Australia (CDNA), which is Australia's peak body for communicable disease control.[12] This is the 11th annual report for the OzFoodNet network and summarises the 2011 surveillance data including a comparison with data from previous years.

Methods

Population under surveillance

In 2011, the OzFoodNet network covered the whole of the Australian population, which was estimated to be 22,618,294 persons as at 30 June 2011.[13]

Data sources

Notified infections

All Australian states and territories have public health legislation requiring doctors and pathology laboratories to notify cases of infectious diseases that are important to public health. State and territory health departments record details of notified cases on surveillance databases. These surveillance datasets are aggregated into a national database, the National Notifiable Diseases Surveillance System (NNDSS),[14] under the auspices of the *National Health Security Act 2007*.[15] For this 2011 report, OzFoodNet aggregated and analysed data from NNDSS and enhanced surveillance data from OzFoodNet sites on the following 9 diseases or conditions:

- *Salmonella* infections (including paratyphoid);
- *Campylobacter* infections (except in New South Wales);
- *Listeria* infections;
- *Shigella* infections;
- *Salmonella* Typhi (typhoid) infections;
- hepatitis A infections;
- botulism;
- STEC infections; and
- haemolytic uraemic syndrome (HUS).

There may be differences when comparing OzFoodNet enhanced data state totals and NNDSS derived notifications. This is due to continual adjustments to NNDSS data made by states and territories after the date of data extraction, to improve data quality. Also, some jurisdictions report by notification date rather than onset date. Data for this report were extracted from NNDSS in May 2012 and were analysed by the date of diagnosis within the reporting period 1 January to 31 December 2011. Date of diagnosis was derived for each case from the earliest date supplied by the jurisdiction, which could be the date of onset of the case's illness, the date a specimen was collected or the date that a health department received the notification. Estimated resident populations for each state or territory as at June 2011 were used to calculate rates of notified infections.[13]

Enhanced surveillance for listeriosis

Commencing in 2010, OzFoodNet collected enhanced surveillance data on all notified cases of listeriosis in Australia via the National Enhanced Listeriosis Surveillance System (NELSS). This enhanced surveillance system adds to the routinely collected data within NNDSS. NELSS includes a centralised national database that contains detailed information regarding the characterisation of *Listeria monocytogenes* isolates by molecular subtyping methods, food histories and exposure data on all notified listeriosis cases in Australia since 2010. The overall aim of this enhanced surveillance is to enable timely detection of clusters and to initiate a public health response. Local public health unit staff interview all cases using a standard national listeriosis questionnaire. Interviews are conducted at the time individual cases are reported to improve accurate recall of foods consumed during the incubation period. Data are collated nationally via an online database using NetEpi Case Manager, a secure web-based reporting system used by OzFoodNet epidemiologists for the enhanced surveillance of listeriosis and multi-jurisdictional outbreaks in Australia. NetEpi allows data to be entered from multiple sites and promotes nationally consistent data collection and analysis by OzFoodNet epidemiologists.[16–18]

Supplementary surveillance

OzFoodNet sites also collected supplementary data on infections that may be transmitted by food. Information on travel status was collected for cases of *Salmonella* Enteritidis infection, hepatitis A, shigellosis, paratyphoid, and typhoid. Locally-acquired infection includes people acquiring their infection in Australia from overseas-acquired cases, from unknown sources of infection, and possible false positives where no clinically compatible illness was reported.

To examine the quality of surveillance data collected across Australia, OzFoodNet sites provided data on the completeness of notifications data for *Salmonella* regarding serotype and phage type. Data from Western Australia, New South Wales, and the Australian Capital Territory were excluded from the analysis of phage type completeness, as

pulsed-field gel electrophoresis (PFGE) is predominantly used for typing *S.* Typhimurium in Western Australia, multiple-locus variable number tandem repeat analysis (MLVA) is predominantly used in New South Wales and the Australian Capital Territory employs either phage typing or MLVA depending on to which reference laboratory the specimen is sent. To assess completeness, data were analysed using the date a notification was received by a health department.

Outbreaks of gastrointestinal disease including foodborne disease outbreaks

OzFoodNet sites collected summary information on gastrointestinal disease outbreaks that occurred in Australia during 2011, including those transmitted via the ingestion of contaminated food (foodborne outbreaks). A foodborne outbreak was defined as an incident where two or more persons experienced a similar illness after consuming a common food or meal and analytical epidemiological and/or microbiological evidence implicated the food or meal as the source of illness. A suspected foodborne outbreak was defined as an incident where two or more persons experienced illness after consuming a common meal or food and descriptive epidemiological evidence implicated the food or meal as the suspected source of illness, including outbreaks where food-to-person-to-food transmission occurred. A cluster was defined as an increase in infections that were epidemiologically related in time, place or person where there is no common setting and investigators were unable to implicate a vehicle or determine a mode of transmission.

Summary information for foodborne and suspected foodborne outbreaks has been combined for the analysis. Information collected on each outbreak included the setting where the outbreak occurred, where the food was prepared, the month the outbreak investigation began, the aetiological agent, the number of persons affected, the type of investigation conducted, the level of evidence obtained, and the food vehicle responsible for the outbreak. To summarise the data, outbreaks were categorised by aetiological agent, food vehicle and the setting where the implicated food was prepared. Data on outbreaks due to waterborne transmission and data from clusters investigated by jurisdictional health departments were summarised. The number of outbreaks and documented causes reported here may vary from summaries previously published by individual jurisdictions as these can take time to finalise.

Data analysis

Microsoft Excel and Stata version 10.1 were used for all analyses.

Results

Rates of notified enteric infections

In 2011, OzFoodNet sites reported 30,957 notifications of 9 diseases or conditions that may be transmitted by food (Table 1), which was a 15% increase compared with the mean of 26,953 notifications per year for the previous 5 years (2006–2010).

Salmonella infections

In 2011, Australian jurisdictions reported 12,271 notifications of *Salmonella* infection, at a rate of 54.3 cases per 100,000 population. This is a 23% increase compared with the mean rate for the previous 5 years (44.1 cases per 100,000) (Table 1) and 1% higher than for 2010 (53.7 cases per 100,000) (Figure 1). Salmonellosis rates in 2011 were lower in the Northern Territory, Tasmania and the Australian Capital Territory compared with the 5-year mean (Table 1). The remaining jurisdictions had higher rates compared with the 5-year mean, with South Australia having the largest percentage increase (48%), followed by Victoria (47%) and New South Wales (26%). Notification rates ranged from 38.2 cases per 100,000 in Tasmania to 174.5 cases per 100,000 in the Northern Territory, which usually has the highest rate of salmonellosis (Table 1). Most cases of salmonellosis in the Northern Territory are thought to be due to infection from environmental sources.[19]

In 2011, 51% of notified cases were females. The highest notification rates were in children aged 0–4 years for both males and females (228.1 and 194.7 cases per 100,000 respectively) with the next highest rates in the 5–9 years age group for both sexes (68.7 and 64.9 cases per 100,000 respectively) (Figure 2).

Salmonella *serotyping and phage typing*

In 2011, *Salmonella* serotype information was available for 98.6% of all notified cases. Nationally during 2011, the most commonly notified *Salmonella* serotype was *S.* Typhimurium, which was responsible for approximately 48% (5,940/12,271) of all notified infections (Table 2). Rates of *S.* Typhimurium notifications in 2011 increased by 50% compared with the 5-year mean (2006–2010). *S.* Enteritidis, *S.* Virchow and *S.* Paratyphi B biovar Java also had large percentage increases compared with the 5-year mean (Table 2).

Phage typing was conducted for 99% of *S.* Typhimurium isolates from South Australia, Victoria, Tasmania, Queensland and the Northern Territory. The top 5 most common phage types

Table 1: Number of notified cases, crude rate and 5-year mean (2006–2010) rate per 100,000 population of diseases or infections commonly transmitted by food, Australia, 2011, by disease and state or territory

Disease or infection		State or territory								
		ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Botulism	Notified cases, 2011	0	2	0	0	0	0	0	0	2
*Campylobacter**	Notified cases, 2011	497	NN	157	5,139	2,119	862	6,783	2,176	17,733
	Crude rate, 2011	135.9	NN	68.2	112.2	127.9	168.8	120.7	92.6	115.8
	Mean rate, 2006–2010	128.4	NN	103.0	105.1	133.6	126.4	114.3	99.1	111.9
Haemolytic uraemic syndrome	Notified cases, 2011	0	4	1	1	3	0	4	0	13
	Crude rate, 2011	0.0	0.1	0.4	0.0	0.2	0.0	0.1	0.0	0.1
	Mean rate, 2006–2010	0.1	0.1	0.1	0.1	0.1	0.0	0.0	0.0	0.1
Hepatitis A	Notified cases, 2011	3	57	3	25	6	4	34	12	144
	Crude rate, 2011	0.8	0.8	1.3	0.5	0.4	0.8	0.6	0.5	0.6
	Mean rate, 2006–2010	1.1	1.2	3.6	1.1	1.2	0.7	2.1	1.6	1.4
Listeria	Notified cases, 2011	1	21	1	10	6	2	22	7	70
	Crude rate, 2011	0.3	0.3	0.4	0.2	0.4	0.4	0.4	0.3	0.3
	Mean rate, 2006–2010	0.3	0.4	0.0	0.2	0.2	0.4	0.3	0.4	0.3
Salmonella	Notified cases, 2011	161	3,479	403	2,923	1,058	195	2,734	1,318	12,271
	Crude rate, 2011	44.0	47.8	174.5	64.0	63.7	38.2	48.7	56.1	54.3
	Mean rate, 2006–2010	46.9	38.1	217.2	57.9	43.1	41.1	33.2	46.3	44.1
Shiga toxin-producing *Escherichia coli*	Notified cases, 2011	5	10	1	16	49	2	9	3	95
	Crude rate, 2011	1.4	0.1	0.4	0.3	3.0	0.4	0.2	0.1	0.4
	Mean rate, 2006–2010	0.1	0.2	0.5	0.5	2.6	0.0	0.2	0.2	0.4
Shigella	Notified cases, 2011	9	131	77	63	34	2	95	84	495
	Crude rate, 2011	2.5	1.8	33.4	1.4	2.1	0.4	1.7	3.6	2.2
	Mean rate, 2006–2010	1.2	1.5	54.7	2.3	4.3	0.6	1.8	5.8	2.9
Typhoid	Notified cases, 2011	2	45	3	21	9	3	36	15	134
	Crude rate, 2011	0.5	0.6	1.3	0.5	0.5	0.6	0.6	0.6	0.6
	Mean rate, 2006–2010	0.2	0.5	0.8	0.3	0.2	0.2	0.6	0.4	0.4

* *Campylobacter* is notifiable in all jurisdictions except New South Wales.

NN Not notifiable

Figure 1: Notification rate for salmonellosis in Australia, by year of diagnosis

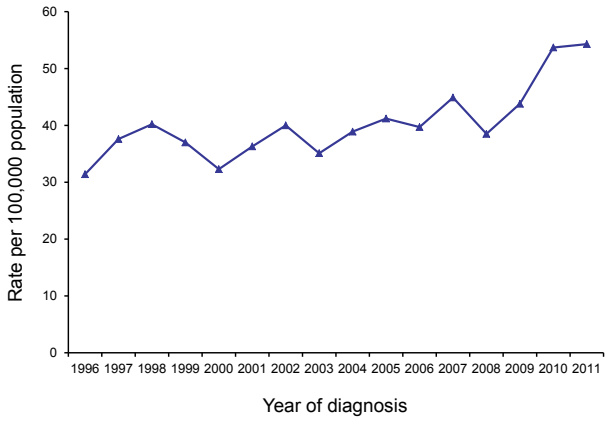


Figure 2: Notification rate for salmonellosis in Australia, 2011, by age group and sex

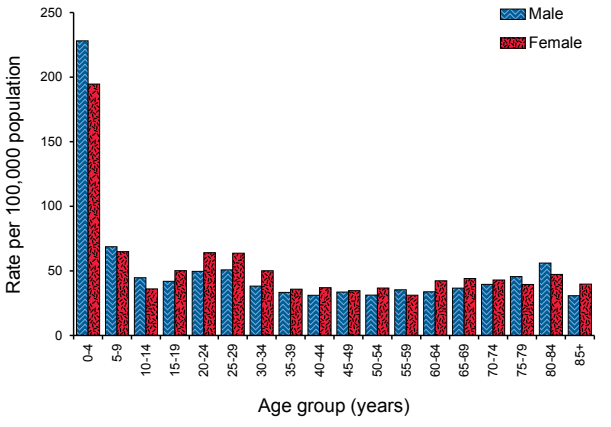


Table 2: Top 5 most common *Salmonella* serotypes, in Australia, 2011, by state or territory, compared with the 5-year mean

State or territory		*Salmonella* Typhimurium	*Salmonella* Enteritidis	*Salmonella* Virchow	*Salmonella* Saintpaul	*Salmonella* Paratyphi B biovar Java
ACT	Notified cases, 2011	116	9	3	3	3
	Mean (2006–2010)	100	9	4	3	3
	% change	16%	0%	–25%	0%	0%
NSW	Notified cases, 2011	1,977	169	160	51	72
	Mean (2006–2010)	1,415	105	77	54	60
	% change	40%	61%	108%	–6%	20%
NT	Notified cases, 2011	48	9	38	48	7
	Mean (2006–2010)	47	10	37	45	12
	% change	2%	–10%	3%	7%	–42%
Qld	Notified cases, 2011	969	116	317	215	45
	Mean (2006–2010)	611	100	273	216	36
	% change	59%	16%	16%	0%	25%
SA	Notified cases, 2011	664	59	15	14	15
	Mean (2006–2010)	383	34	15	11	11
	% change	73%	74%	0%	27%	36%
Tas.	Notified cases, 2011	58	9	5	0	4
	Mean (2006–2010)	73	6	5	3	1
	% change	–21%	50%	0%	N/A	300%
Vic.	Notified cases, 2011	1,681	160	82	38	55
	Mean (2006–2010)	1,001	94	38	34	32
	% change	68%	70%	116%	12%	72%
WA	Notified cases, 2011	427	281	15	37	60
	Mean (2006–2010)	324	169	17	48	33
	% change	32%	66%	–12%	–23%	82%
Australia	Notified cases, 2011	5,940	812	635	406	261
	Mean (2006–2010)	3,953	528	466	413	188
	% change	50%	54%	36%	–2%	39%

N/A Not available.

all had significant increases in notifications compared with the 2-year mean (2009–2010) notifications (Table 3). Phage type (PT) 60 notifications increased significantly, most of these notifications (86%) were notified in Victoria. The *S.* Typhimurium phage type that was associated with the most foodborne disease outbreaks in 2011 was PT 170/108* (n=15 outbreaks), followed by PT 9 (n=12 outbreaks), PT 135 (n=8 outbreaks) and PT 135a (3 outbreaks).

OzFoodNet also monitors the completeness of 6 serotypes that are routinely phage typed: Bovismorbificans; Enteritidis; Hadar; Heidelberg; Typhimurium; and Virchow. In 2011, phage typing was greater than 90% complete for *S.* Heidelberg and *S.* Virchow only. Across these 6 serotypes, phage type completeness declined from 86% in 2010 to 65% in 2011. This decline was predominantly attributable to the change to MLVA typing for Typhimurium in New South Wales.

Salmonella *Enteritidis*

S. Enteritidis is a globally important *Salmonella* serotype that can infect the internal contents of eggs, but is not endemic in Australian egg layer flocks.[20, 21] The majority of cases in Australia are associated with overseas travel. To monitor incidence of this serotype in Australia, OzFoodNet conducts enhanced surveillance of locally-acquired infections of *S.* Enteritidis in humans.

* Classification of this organism differs between laboratories, with the Microbiological Diagnostic Unit using PT 170 to classify this type of *S.* Typhimurium and SA Pathology using PT 108 due to a difference in the interpretation of 1 phenotypic characteristic.

Table 3: The number of notifications for the top 5 most common *Salmonella* Typhimurium phage types, Australia, 2011, compared with the average of 2009–2010*

Phage types	2011	Average 2009–2010	Ratio†
170/108§	690	518	1.3
9	689	387	1.8
135a	537	372	1.4
135	297	145	2.0
60	281	14	20.1

* Data from jurisdictions that phage type more than 90% of isolates in 2011. Excludes New South Wales, the Australian Capital Territory and Western Australia.

† Ratio of the number of cases in 2011 compared with the average of 2009 and 2010 notifications.

§ Classification of this organism differs between laboratories, with the Microbiological Diagnostic Unit using PT 170 to classify this type of *Salmonella* Typhimurium and SA Pathology using PT 108 due to a difference in the interpretation of 1 phenotypic characteristic.

During 2011, OzFoodNet sites reported 816 cases of *S.* Enteritidis infection (Table 4) compared with 835 notifications in 2010 and 589 notifications in 2009. Travel histories were obtained for 94% (771) of cases in 2011, similar to 2010 (792/835, 95%). Of those cases in 2011 with travel history information recorded, 89% (690) had travelled overseas and 11% (81) were locally-acquired. Western Australia reported the highest number of notified cases compared with other jurisdictions in 2011. Queensland reported the largest number of locally-acquired cases.

In 2011, South East Asia (86%, 591) was the most common region of overseas acquisition for *S.* Enteritidis. Similarly to previous years, the most common overseas country of acquisition was Indonesia, (62%, 431). Thailand was the second most common overseas country of acquisition (8%, 55), followed by Malaysia (5%, 37).

Phage typing was performed for 67% (544) of the *S.* Enteritidis cases with travel history and the most common phage types among overseas-acquired cases were PT 1 (21%), 6a (13%), 13 (9%), 1b (9%) and 21 (8%). Locally-acquired cases were sporadic with no clusters detected by person, place, or time. The most common phage types among locally-acquired isolates were PT 26 (21%), 4b (17%), RDNC (9%), 1 (6%) and 6a (6%) (Table 5). In addition, PT 13, 1b and 21, which were common among overseas-acquired isolates, were less common among locally-acquired isolates accounting for 3%, 3% and 2% of phage typed isolates respectively.

Table 5: Top 5 most common phage types of locally and overseas-acquired *Salmonella Enteritidis* infections, Australia, 2011

Overseas-acquired cases			Locally-acquired cases		
Phage type	n	% of total typed (n=391)	Phage type	n	% of total typed (n=66)
1	81	21	26	14	21
6a	51	13	4b	11	17
13	36	9	RDNC	6	9
1b	35	9	1	4	6
21	32	8	6a	4	6

RDNC Reactions do not conform.

Campylobacter infections

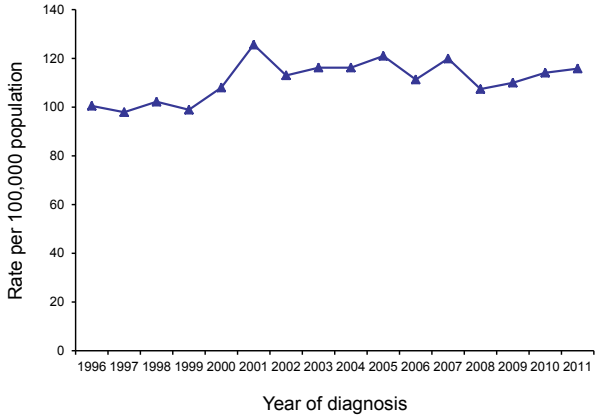
In 2011, OzFoodNet sites (excluding New South Wales where *Campylobacter* infection is not notifiable) reported 17,733 notifications of *Campylobacter* infection, the highest number of notifications recorded in the NNDSS database since records began in 1991.[14] This equates to a rate of 115.8 notifications per 100,000 population (Figure 3, Table 1). This is a 3.5% increase compared with the 5-year mean of 111.9 per 100,000. The Northern Territory

Table 4: Number of *Salmonella Enteritidis* infections, Australia, 2011, by travel history and state or territory

State or territory	Locally-acquired	Overseas-acquired	Unknown	Total
WA	17	263	1	281
NSW	23	137	8	168
Vic.	9	150	1	160
Qld	25	64	32	121
SA	2	55	2	59
NT	0	8	1	9
ACT	2	7	0	9
Tas.	3	6	0	9
Total	81	690	45	816

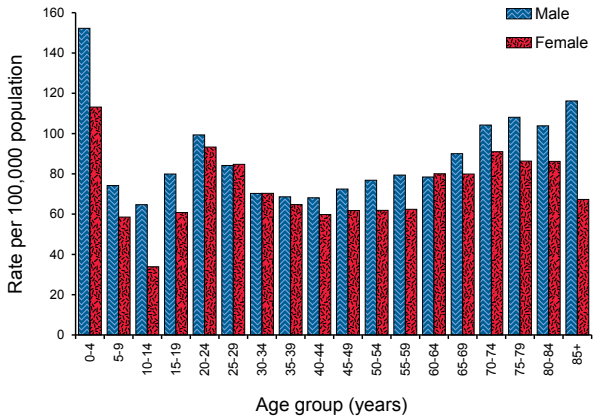
reported a rate of 68.2 cases per 100,000, 34% below the 5-year mean rate and Tasmania reported a rate of 168.8 cases per 100,000, a 34% increase above the 5-year mean (Table 1).

Figure 3: Notification rate for campylobacteriosis, Australia, by year of diagnosis



Overall, 54% of notified cases were males. Notification rates were highest in children aged 0–4 years for both males and females (152 and 113 notifications per 100,000, respectively) with additional peaks in the 20–24 years age group and in the 65s or over year age group (Figure 4).

Figure 4: Notification rate for campylobacteriosis, Australia, 2011, by age group and sex



Listeria infections

There were 70 notifications of *L. monocytogenes* infection reported in 2011 (0.3 cases per 100,000 population), consistent with the 5-year historical mean rate of 0.3 cases per 100,000 (Table 1). State and territory rates ranged from 0.2 to 0.4 cases per 100,000. Of the 70 notifications in 2011, 74% (n=52) were in people aged 60 years or more and males accounted for 59% (41) of all notifications. Four cases in 2011 were pregnant women with 1 associated neonatal case. The most commonly reported *L. monocytogenes* isolates were serotype 1/2b, 3b, 7; binary type 158 (14%, 10/70) and serotype 4b, 4d, 4e; binary type 254 (14%, 10) (Table 6). These were also the most common types in 2010.

Table 6: Top 5 most common *Listeria monocytogenes* strains, Australia, 2011, by molecular serotype and binary type

Serotype	Binary type	Number of cases
4b, 4d, 4e	254	10
1/2b, 3b, 7	158	10
4b, 4d, 4e	190	9
1/2a, 3a	131	6
1/2a, 3a	155	6

Source: OzFoodNet National Enhanced Listeriosis Surveillance System.

No multi-jurisdictional clusters or outbreaks of listeriosis were detected in 2011. Six cases sharing an identical molecular serotype, binary type, multilocus sequence typing profile but slightly different PFGE types were detected in Victoria. Interviews did not detect any common exposures.

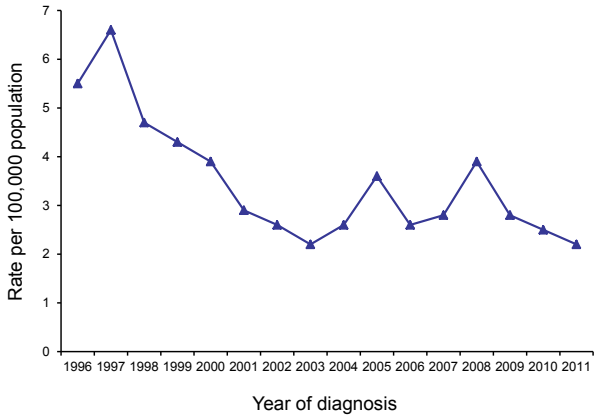
Shigella infections

There were 495 notifications of *Shigella* infection in Australia in 2011, a rate of 2.2 notifications per 100,000 population compared with the 5-year historical mean rate of 2.9 per 100,000 (Figure 5, Table 1). In 2011, compared with the 5-year mean there was a decline (ranging from 6%–51%) in rates of *Shigella* infection for all states and territories apart from New South Wales and the Australian Capital Territory. As in previous years, the highest notification rate was in the Northern Territory, with 33.4 per 100,000 followed by Western Australia with a rate of 3.6 per 100,000.

In 2011, notification rates for shigellosis were highest in males and females aged 0–4 years, with 6.4 and 7.5 notifications per 100,000 population respectively (Figure 6). The overall rate for males was 2.4 per 100,000 in 2011 compared with the female rate of 2.0 per 100,000. Indigenous status was recorded for 87% (432) of shigellosis cases. Of these, 32% (137) identified as Aboriginal and/or Torres Strait Islander people. The Northern Territory and Western Australia reported the most

cases of shigellosis in people who identified as Aboriginal and/or Torres Strait Islander, (49%, 67) and (28%, 39) respectively.

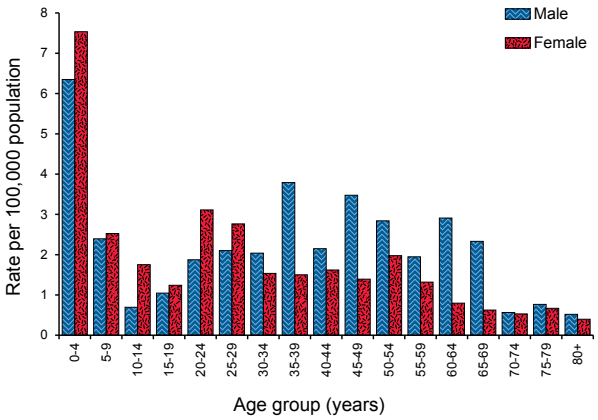
Figure 5: Notification rate for shigellosis, Australia, by year of diagnosis



Travel history information was available for 69% (342/495) of shigellosis notifications in 2011 and of these, 46% (158) acquired their illness overseas. The most common overseas country of acquisition was Indonesia (31%, 49).

Nearly all *Shigella* isolates were typed (98%, 483) and *Sh. sonnei* was the most frequent species notified (71%, 344), followed by *Sh. flexneri* (27%, 130). There were also 7 notifications of *Sh. boydii* and 2 notifications of *Sh. dysenteriae*. *Sh. sonnei* biotype a was the most frequently notified biotype in 2011 (33.1%, 164), 41% higher than the total for 2010 (Table 7).

Figure 6: Notification rate for shigellosis, Australia, 2011, by age and sex



Typhoid

In 2011, there were 134 notifications of *S.* Typhi infection (typhoid) in Australia, a rate of 0.6 notifications per 100,000 population and a 50% increase above the 5-year historical mean rate (2006–2010) of 0.4 per 100,000 (Table 1). Most cases were notified in New South Wales (n=45) and Victoria (n=36). In 2011, 60% (81) of cases were male. Travel history was known for 99% (132) of cases, with 96% (127) of these infections likely to have been acquired overseas. For the remaining 5 cases, four had spent time in a typhoid endemic country from between 8 and 18 months prior to the onset of their illness. The maximum incubation period of typhoid is up to 60 days, thus these cases may be detections of chronic infections. The 5th case was believed to have contracted their infection from a known typhoid case.

Table 7: Number, percentage and ratio of the top 10 *Shigella* infections, Australia, 2010 to 2011

Biotype	2010 n	2010 %*	2011 n	2011 %†	Ratio‡
Shigella sonnei biotype a	116	21.0	164	33.1	1.4
Shigella sonnei biotype g	191	34.6	139	28.1	0.7
Shigella sonnei untyped	32	5.8	34	6.9	1.1
Shigella flexneri 2a	36	6.5	27	5.5	0.8
Shigella flexneri 4a	38	6.9	18	3.6	0.5
Shigella flexneri 4	22	4.0	18	3.6	0.8
Shigella flexneri 3a	37	6.7	15	3.0	0.4
Shigella flexneri 2b	18	3.3	12	2.4	0.7
Shigella untyped	6	1.1	12	2.4	2.0
Shigella flexneri untyped	13	2.4	10	2.0	0.8

* Proportion of total shigellosis notified in 2010.

† Proportion of total shigellosis notified in 2011.

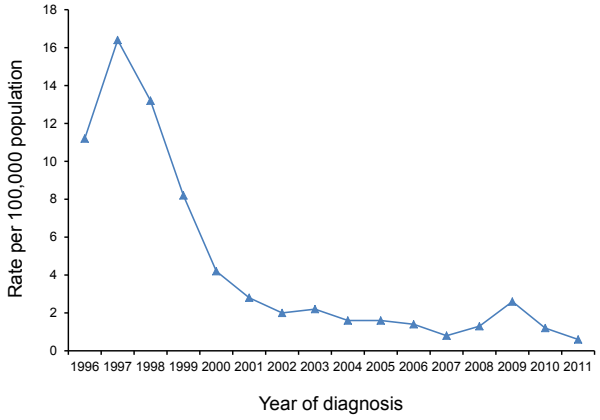
‡ Ratio of the number of cases in 2011 compared with the number in 2010.

Most overseas-acquired cases of typhoid in 2011 had travelled to India (58%, 74) and Indonesia (9%, 12). The most commonly notified phage type was E1 and these infections were mostly acquired in India. Three of the 5 cases without a history of overseas travel were also PT E1 (Table 8).

Hepatitis A

In 2011, there were 144 hepatitis A notifications with a rate of 0.6 notifications per 100,000 population, the lowest total number of notifications and annual rate recorded in the NNDSS database since records began in 1991,[14] and 57% below the 5-year historical mean rate of 1.4 notifications per 100,000 (Table 1). There was a large decrease in hepatitis A notifications between 1997 and 2001 and then a more gradual decrease from 2002 to 2011, noting an increase in 2009 due to la arge multi-jurisdictional outbreak associated with the consumption of semi-dried tomatoes[22,23] (Figure 7). The median age of cases in 2011 was 29 years (range 2–90 years) with 59% being male (85).

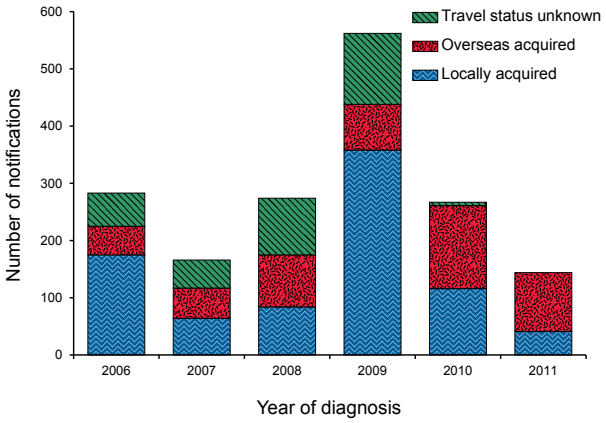
Figure 7: Notification rate for hepatitis A infections, Australia, by year of diagnosis



Indigenous status was known for 93% (134) of hepatitis A cases in 2011. Two cases were identified as Aboriginal and/or Torres Strait Islander people (1%), compared with one in 2010.

In 2011, 72% (103/144) of hepatitis A infections were acquired overseas (Figure 8). Regions of acquisition included South Asia (34%, 35), South East Asia (25%, 26) and Africa (15%, 15). In 2011, 28% (41) of hepatitis A cases were locally-acquired, the lowest number and proportion since supplementary surveillance began in 2006.

Figure 8: Place of acquisition for hepatitis A cases in Australia, by year of diagnosis



Shiga toxin-producing *Escherichia coli* infection

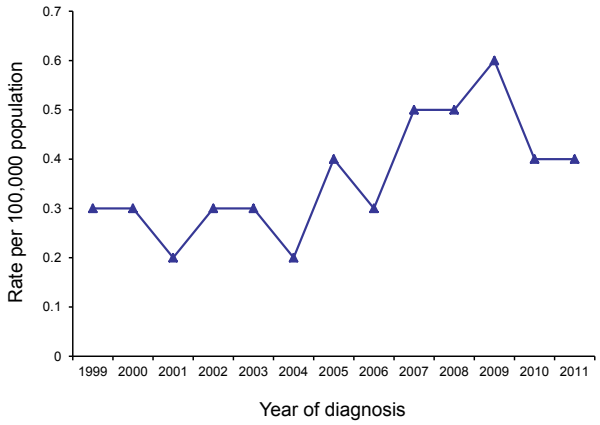
In 2011, there were 95 notifications of STEC infection in Australia; a rate of 0.4 notifications per 100,000 population, equivalent to the 5-year mean rate (Table 1) and the rate from 2010 (Figure 9). Seven of these cases were also diagnosed with HUS. Under the Australian national notifiable disease surveillance case definitions, (http://www.health.gov.au/casedefinitions), these conditions are

Table 8: Notifications of *Salmonella Typhi* infection, Australia, 2011, by phage type and country of acquisition

Phage type	Australia	Bangladesh	India	Indonesia	Other countries	Unknown	Total
E1	3	1	21	0	5	0	30
E9	0	3	16	0	4	0	23
A	1	0	3	2	2	0	8
D2	0	0	0	2	1	0	3
Other types	0	0	5	0	4	1	10
Untypable	0	4	4	1	3	1	13
Unknown	1	3	25	7	11	0	47
Total	5	11	74	12	30	2	134

notified separately. In 2011, 58% (55) of cases were male. The median age of cases was 26 years (range <1–85 years).

Figure 9: Notification rate for Shiga toxin-producing *Escherichia coli* infections, Australia, by year of diagnosis*



* Shiga toxin-producing *Escherichia coli* became nationally notifiable in Australia in 1999.

Notified cases of STEC infection are strongly influenced by jurisdictional practices regarding the screening of stool specimens.[24] In particular, South Australian public health laboratories routinely test all bloody stools with a polymerase chain reaction (PCR) assay specific for genes coding for Shiga toxins, making rates for this state typically the highest in the country. In 2011, South Australia had the highest rate of notifications with 3 cases per 100,000 population (n=49) followed by the Australian Capital Territory with 1.4 cases per 100,000 (n=5). The increase in the notification rate for the Australian Capital Territory relates to the commencement of an STEC screening study in October 2011 based in a local laboratory.

In 2011, serogroup information was available for 61% of STEC cases (58/95). The most common serogroups identified were: O157 (38%, 22); O111 (17%, 10); O26 (12%, 7); and O128 (7%, 4). Serotype information was obtained by serotyping cultured isolates or by PCR targeting serotype-specific genes. The remaining 37 isolates either could not be serotyped or were Shiga toxin positive by PCR only. In 2010, O157 accounted for 59% (30/51) and O111 10% (5/51) of serogrouped specimens.

Haemolytic uraemic syndrome

In 2011, OzFoodNet sites reported 13 cases of HUS with a rate of 0.1 cases per 100,000, equating with the 5-year historical mean rate (Table 1). There were 7 male and 6 female cases and the median age was 30 years (range <1 to 84 years). In contrast to previous years, the majority of cases were in adults with only 3 cases in children aged 0–4 years and 2 cases in children aged 5–10 years.

Not all diagnoses of HUS are related to enteric pathogens (including those potentially transmitted by food). In 2011, 54% of HUS cases (7) were positive for STEC with serotypes O157 (n=1), O111 (n=1) and O41:H4 (n=1) detected in 3 cases. The remaining 4 cases were Shiga toxin positive but the isolates were unable to be serotyped.

Botulism

Four forms of naturally occurring botulism are recognised; adult, infant (intestinal), foodborne and wound.[25] Intestinal botulism mostly affects infants less than 1 year of age and occurs when *Clostridium botulinum* spores are ingested, germinate in the infant's intestine and the organism produces botulinum toxin. It does not include cases where the preformed toxin is ingested: these are considered foodborne.

There were 2 cases of intestinal botulism reported in 2011, affecting 1 male infant and one female infant. Both cases were diagnosed in New South Wales and both were due to botulinum toxin A but the cases were not clustered in time or place and no source was identified. There were no notifications in 2010 and 1 case reported in 2009.[23]

Outbreaks of gastrointestinal illness

In 2011, OzFoodNet sites reported 1,719 outbreaks of gastrointestinal illness (including foodborne disease), affecting 29,839 people, of whom 872 were hospitalised (Table 9). There were 103 deaths during these outbreaks. This compares with a 5-year mean (2006–2010) of 1,686 outbreaks.

Outbreaks spread person-to-person

In 2011, 79% of all reported gastrointestinal outbreaks were transmitted from person-to-person (1,352). These outbreaks affected 25,432 people, 537 people were hospitalised and 95 deaths were reported during these outbreaks (Table 9). Aged care facilities (53%, 720) were the most frequently reported setting for person-to-person outbreaks, followed by hospitals (15%, 204). Outbreaks were most commonly due to norovirus (43%, 575) or a suspected viral agent (34%, 458), with 227 of unknown aetiology (17%).

Outbreaks spread animal-to-person

One outbreak was reported to have been transmitted from animal-to-person. The aetiological agent was

Table 9: Outbreaks of gastrointestinal illness reported to state and territory health departments, Australia, 2011

Transmission mode	Number of outbreaks	Number of ill	Number hospitalised	Number died
Foodborne and suspected foodborne	151	2,104	231	5
Person-to-person	1,352	25,432	537	95
Animal-to-person	1	10	4	0
Waterborne or suspected waterborne	4	100	5	0
Unknown	211	2,193	95	3
Total	1,719	29,839	872	103

identified as STEC. The outbreak affected 10 people, with four being hospitalised including 2 cases of HUS, following contact with animals at a petting zoo at an agriculture show in South Australia (Table 9). The aetiological agent for seven of the cases was identified as STEC O111 and for one of the cases identified as STEC O157. STEC isolates from the remaining 2 cases were unable to be serotyped. STEC O111 was detected in environmental samples collected from the two areas of the petting zoo believed to be the source of the infection.

Waterborne outbreaks

There were 4 outbreaks reported to be waterborne or suspected to be waterborne. These outbreaks affected 100 people, with 5 people hospitalised (Table 9). Each outbreak was attributed to a different aetiological agent: *Giardia*, *S.* Typhimurium, *Campylobacter* and *Cryptosporidium*. The source of infection was not confirmed for these outbreaks but was suspected to be the rural community water supply for the first 2 outbreaks (bore water and reticulated supply respectively), a private supply (rainwater tank) and several public swimming pools were implicated in the final outbreak.

Outbreaks with unknown mode of transmission

There were 211 outbreaks in which cases were clustered in time, place or person, but investigators were unable to develop an adequate hypothesis for the mode of transmission. These outbreaks affected 2,193 people, 95 of whom were hospitalised. There were 3 deaths reported during these outbreaks. Aged care facilities were the most frequently reported settings for these outbreaks (51%, 107), followed by child care facilities (13%, 27) and the community (9%, 20). In 176 (83%) of these outbreaks, both the aetiological agent and transmission mode remained unknown. In 12 (6%) outbreaks the aetiological agent was identified as *S.* Typhimurium and in 6 (3%) outbreaks the agent was norovirus.

Foodborne and suspected foodborne outbreaks

In 2011, OzFoodNet sites reported 151 outbreaks of foodborne and suspected foodborne illness. These outbreaks affected 2,104 people, with 231 hospitalised. Five people were reported to have died during these outbreaks (Table 9). This compares with a 5-year mean (2006–2010) of 137 outbreaks. The overall rate of foodborne disease outbreaks in 2011 was 6.7 outbreaks per million population (Table 10). The highest rates were in the Northern Territory (30.4 outbreaks per million) and the Australian Capital Territory (13.7 outbreaks per million), although these jurisdictions reported only 7 and 5 outbreaks respectively. The largest number of outbreaks (55) was reported by Victoria.

Aetiologies

More than a third of all foodborne and suspected foodborne outbreaks (37%, 56/151) were due to *S.* Typhimurium (Table 11). Other frequently reported pathogens were *Clostridium perfringens* (11%, 16) and *Campylobacter* (6%, 9). There were 47 outbreaks of unknown aetiology (31%), which was similar to 2010 (36%, 55/154).

Food vehicles

Outbreaks were categorised as being attributable to one of 18 food commodities (17 as described by Painter et al[26] with an additional category for lamb) if a single contaminated ingredient was identified or if all ingredients belonged to that food category. Outbreaks that could not be assigned to one of the 18 categories, or for which the report contained insufficient information for food category assignment, were not attributed to any food category.[27]

In 49 foodborne and suspected foodborne outbreaks (32%), investigators attributed the outbreak to a single food commodity, in another 35 outbreaks (23%), the implicated dish contained a mix of ingredients, and no single ingredient was impli-

Table 10: Outbreaks of foodborne and suspected foodborne disease, Australia, 2011 by OzFoodNet site

State or territory	Number of outbreaks	Number ill	Mean size (persons)	Number hospitalised	Outbreak rate per million population
ACT	5	70	14.0	8	13.7
NSW	45	694	15.4	50	6.2
NT	7	28	4.0	2	30.4
Qld	18	227	12.6	37	3.9
SA	10	148	14.8	34	6.0
Tas.	1	5	5.0	0	2.0
Vic.	55	692	12.6	74	9.8
WA	9	224	24.9	23	3.8
Multi-jurisdictional	1	16	16.0	3	N/A
Total	151	2,104	13.9	231	6.7

N/A Not applicable.

Table 11: Number of reported foodborne disease outbreaks and of people number affected, Australia, 2011, by aetiology and food category

Agent category	Total		Attributed to a single food category		Attributed to >1 food category		Not attributed to a food category	
	Number of outbreaks	Number ill	Number of outbreaks	Number ill	Number of outbreaks	Number ill	Number of outbreaks	Number ill
Bacillus cereus	1	12	0	0	1	12	0	0
Campylobacter	9	118	3	80	0	0	6	38
Ciguatera fish poisoning	5	17	5	17	0	0	0	0
Clostridium perfringens	16	207	2	58	1	3	13	146
Norovirus	7	216	1	15	2	47	4	154
Salmonella Typhimurium	56	815	30	500	11	126	15	189
Other *Salmonella* serotypes	5	102	3	60	1	37	1	5
Histamine fish poisoning	2	6	2	6	0	0	0	0
Staphylococcus aureus	2	66	0	0	2	66	0	0
Suspected viral	1	3	0	0	0	0	1	3
Unknown	47	542	3	16	17	121	27	405
Total	151	2,104	49	752	35	412	67	940

cated. The majority of outbreaks (44%, 67/151) could not be definitively attributed to a particular food or foods due to a lack of evidence (Table 11).

Of the outbreaks attributed to a single food (n=49), the foods most frequently implicated were eggs (53%, 26), poultry (16%, 8) and fish (16%, 8). From these outbreaks, 752 people were affected, 108 people were hospitalised and 1 person was reported to have died (Table 12).

There were 84 outbreaks with a known food vehicle or vehicles and of these more than one-third (35%, 29) were suspected or confirmed to have been associated with the consumption of eggs and egg-based dishes (Table 13). These egg-associated outbreaks comprised 19% (29/151) of all foodborne outbreaks, just under half (48%, 29/61) of all foodborne *Salmonella* outbreaks, and more than half (59%, 29/49) of the outbreaks that were attributed to a single commodity. In these egg-associated outbreaks, eggs were served in desserts (12 outbreaks), in sauces and dressings such as Caesar salad dressing and mayonnaise (11 outbreaks), in pastries (1 outbreak), in raw dough or batter (3 outbreaks), as a component of meals that were suspected to be undercooked (2 outbreaks), as a binding ingredient of prawn dumplings (1 outbreak) and added to soup

after cooking (1 outbreak). One outbreak occurred in multiple settings and in multiple foods all microbiologically linked to eggs from a single supplier.

Settings

In 2011, foods implicated in foodborne and suspected foodborne outbreaks were most commonly prepared in restaurants (33%, 50/151), in aged care facilities (15%, 22), or private residences (12%, 18) (Table 14). This represents an absolute and proportional decrease from 2010 levels for both restaurants (39%, 60/154)[33] and aged care facilities (21%, 33/154). However, private residences demonstrated an absolute and proportional increase from 2010 (9%, 14/154).

Investigative methods and levels of evidence

To investigate foodborne outbreaks, epidemiologists in the states and territories conducted 28 cohort studies and 6 case-control studies. Descriptive case series investigations were conducted for 97 outbreaks. In 20 outbreaks, no formal study was conducted (Appendix).

There was an analytical association between illness and the implicated food as well as microbiological evidence of the aetiological agent in the epidemiologically implicated food for 8 outbreaks. Investigators relied on analytical evidence alone for 10 outbreaks and microbiological (or toxicological for non-microbial outbreaks) evidence alone for 16 outbreaks. These confirmed foodborne outbreaks comprised 23% (34/151) of all foodborne outbreaks (Appendix).

Contributing factors

Investigators collect information about factors that are likely to have contributed to a foodborne outbreak occurring. Contamination factors are those contributing factors that led to the food becoming contaminated or to contaminated products being consumed. Contamination factors for the 34 confirmed foodborne outbreaks were most commonly stated to have been unknown (53%, 18) (Table 15). The contamination factors for the remaining confirmed outbreaks were based on measured evidence (15%, 5), verbal confirmation during inspections (6%, 2), postulated based on detection of the aetiological agent in a food vehicle (9%, 3), or investigator suspicion (18%, 6). Contamination factors varied by the aetiology of outbreaks. In the 2 *Staphylococcus aureus* outbreaks, investigators reported that person-to-food-to-person contamination and unknown contamination were involved, respectively. In the 2 *Campylobacter* outbreaks, ingestion of raw products and unknown contamination were involved, respectively. Of the 16 *S.* Typhimurium outbreaks, ingestion of raw products (8), unknown contamination (4), a combination of ingestion of contaminated raw products and cross contamination from raw ingredients (2), cross-contamination from raw ingredients (1) and inadequate cleaning of equipment (1) were reported.

Significant foodborne and suspected foodborne outbreaks

In 2011, OzFoodNet sites responded to 151 foodborne or suspected foodborne outbreaks (including a multi-jurisdictional *S.* Typhimurium PT 135a outbreak). There were 12 outbreaks that each affected more than 40 people. Five outbreaks were due to *S.* Typhimurium, two were due to norovirus and one each was due to a *Campylobacter*/*S.* Singapore mixed infection, *Cl. perfringens* and *S.* Singapore and 2 outbreaks were of unknown aetiology. These outbreaks affected at least 752 people of whom 81 were hospitalised. There were no reported deaths.

An outbreak of *S.* Typhimurium PT 44 with MLVA profile 03-10-08-09-523 was investigated in New South Wales in January following an increase in hospital emergency department presentations with gastrointestinal symptoms. Case data were suggestive of a point source of infection from pork/

Table 12: Foodborne disease outbreaks attributed to a single food vehicle, Australia, 2011

Food commodities (Painter et al)	Number of outbreaks	Number affected	Number hospitalised	Number of fatalities
Eggs	26	471	88	1
Poultry	8	159	7	0
Fish	8	27	7	0
Beef	2	58	0	0
Fruits-nuts	2	20	0	0
Pork	2	9	2	0
Grains-beans	1	8	4	0
Total	49	752	108	1

Table 13: Outbreaks of foodborne illness associated with egg-based dishes, Australia, 2011 (n=29)

State or territory	Setting prepared	Agent responsible	Number affected	Evidence	Responsible vehicles
ACT	Bakery	*S.* Typhimurium PT 170/108*, MLVA 03-09-07-14-523†	41	M	Chicken Caesar salad roll containing raw egg mayonnaise
NSW	Private residence	*S.* Typhimurium MLVA 03-13-12-10-523	3	D	Homemade hollandaise sauce and semifreddo
	Restaurant	*S.* Typhimurium MLVA 03-11-11-10-523	10	D	Dessert containing raw egg custard
	Restaurant	*S.* Typhimurium PT 3, MLVA 03-13-14-09/10-523	11	M	Caesar salad dressing containing raw egg
	Restaurant	*S.* Typhimurium MLVA 03-09-08-14-523	13	D	Tiramisu containing raw egg
	Restaurant	*S.* Typhimurium MLVA 03-10-08-09-523	8	AM	Chicken and corn soup containing raw egg
	Restaurant	*S.* Typhimurium PT 135, MLVA 03-13-11-09-523	4	D	Prawn dumplings containing raw egg
	Restaurant	*S.* Typhimurium PT 170/108, MLVA 03-09-07-14-523	6	M	Fried ice cream
	Restaurant	*S.* Typhimurium MLVA 03-09-07-13-523	6	D	Raw egg dressing
	Restaurant	*S.* Typhimurium MLVA 03-09-07-15-523	3	D	Raw egg mayonnaise
	Takeaway	*S.* Typhimurium PT 44, MLVA 03-10-08-09-523	85	M	Vietnamese pork, chicken and salad rolls containing raw egg butter
Qld	Multiple settings	*S.* Typhimurium PT 135a, MLVA 03-14-11-11-524	49	M	Multiple foods made with eggs from a single supplier
SA	Bakery	*S.* Typhimurium PT 135	6	M	Pies glazed with raw egg
Vic.	Private residence	*S.* Typhimurium PT 135	4	A	Raw pasta dough
	Private residence	*S.* Typhimurium PT 135a	9	D	Potato salad containing raw egg mayonnaise
	Private residence	*S.* Typhimurium PT 141	2	D	Chocolate mousse containing raw egg
	Private residence	*S.* Typhimurium PT 170/108	2	M	Raw pancake batter
	Private residence	*S.* Typhimurium PT 170/108	2	D	Raw muffin batter
	Private residence	*S.* Typhimurium PT 44	12	D	Tiramisu containing raw egg
	Private residence	*S.* Typhimurium PT 9	4	D	Chocolate mousse containing raw egg
	Private residence	*S.* Typhimurium PT 44	5	D	Tiramisu containing raw egg
	Private residence	*S.* Typhimurium PT 9	7	D	Chocolate mousse containing raw egg
	Restaurant	*S.* Typhimurium PT 170/108	14	D	Chocolate mousse containing raw egg
	Restaurant	*S.* Typhimurium PT 170/108	15	AM	Fried ice cream
	Takeaway	*S.* Typhimurium PT 170/108	15	D	Vietnamese mixed dish including egg
	Takeaway	*S.* Typhimurium PT 170/108	26	D	Sushi containing raw egg mayonnaise
	Takeaway	*S.* Typhimurium PT 170/108	37	M	Pizza with egg and chocolate mousse containing raw egg
	Takeaway	*S.* Typhimurium PT 9	84	M	Sushi containing raw egg mayonnaise
WA	Takeaway	*S.* Typhimurium PT 9	15	D	Vietnamese pork roll made with raw egg butter

D Descriptive evidence implicating the vehicle.

A Analytical epidemiological association between illness and vehicle.

M Microbiological confirmation of aetiology in vehicle and cases.

* Classification of this *Salmonella* Typhimurium phage type differs between laboratories, with the Microbiological Diagnostic Unit using PT 170 and SA Pathology using PT 108. This is due to a difference of interpretation of 1 phenotypic characteristic.

† Multiple-locus variable number tandem repeat analysis (MLVA) profiles are reported using the Australian coding convention agreed at a MLVA typing harmonisation meeting in Sydney in November 2011.

Table 14: Food preparation setting implicated in foodborne disease outbreaks, Australia, 2011

Setting	Number of outbreaks	Per cent of outbreaks	Number affected
Restaurant	50	33	542
Aged care facility	22	15	220
Private residence	18	12	126
Takeaway	13	9	291
Commercial caterer	13	9	388
Bakery	8	5	142
Hospital	5	3	36
Primary produce*	5	3	17
Unknown†	4	2	164
Camp	3	2	14
Grocery store/ delicatessen	2	1	55
Fair/festival/mobile service	2	1	42
Institution	2	1	13
Private caterer	1	1	17
School	1	1	17
Cruise/airline	1	1	16
National franchised fast food	1	1	4
Total	151	100	2,104

* The 5 outbreaks associated with primary produce were all ciguatera fish poisoning. The implicated fish species were Coral Trout (2 outbreaks) and Spanish Mackerel, Red Bass and an unknown reef fish were implicated in the other 3 outbreaks.

† An outbreak was assigned a setting of unknown when the implicated food was prepared in multiple settings (n=2) or not enough detail was provided to determine the setting (n=2).

Table 15: Factors reported as leading to the contamination of food vehicles in confirmed foodborne disease outbreaks, Australia, 2011, by aetiology

Agent	Contamination factor	Total
Bacillus cereus	Unknown	1
Campylobacter	Ingestion of contaminated raw products	1
	Unknown	1
Clostridium perfringens	Unknown	2
Norovirus	Unknown	2
Other *Salmonella* serotypes	Cross contamination from raw ingredients	2
	Unknown	1
Salmonella Typhimurium	Ingestion of contaminated raw products	8
	Unknown	4
	Ingestion of contaminated raw products and cross contamination from raw ingredients	2
	Cross contamination from raw ingredients	1
	Inadequate cleaning of equipment	1
Histamine fish poisoning	Unknown	1
Staphylococcus aureus	Person-to-food-to-person	1
	Unknown	1
Unknown	Unknown	5
Total		34

chicken/salad rolls with raw egg mayonnaise from a Vietnamese bakery in the area. Of 147 cases who presented to emergency departments and general practitioners, 58 were interviewed and provided information on a further 27 people who were ill. Forty-nine people submitted a stool sample and 47 were positive for *S.* Typhimurium PT 44 (MLVA profile 03-10-08-09-523). The bakery was inspected by the New South Wales Food Authority and shut down for cleaning and disinfection. Thirteen of 21 food samples including raw egg butter, pâté, chicken, pork and salad items and 5 of 11 environmental swabs were positive for *S.* Typhimurium PT 44 (MLVA profile 03-10-08-09-523). Lack of records or supplier information prevented an egg trace back.

Two outbreaks of *S.* Singapore associated with buffet functions on a cruise boat were investigated in New South Wales in February. The first was an 80th birthday party, with 45 of 57 people reporting a gastrointestinal illness. *S.* Singapore was isolated from 5 stool specimens, and *Salmonella* species detected from a 6th specimen. Roast chicken pieces (relative risk [RR] 5.7, 95% confidence intervals [CI] 0.9–35.2), silverside (RR 1.3, 95% CI 1.0–1.8) and potato salad (RR 1.6, 95% CI 1.1–2.4) were found to have an association with illness, but in a multivariate analysis only roast chicken had a statistically significant association with illness (odds ratio [OR] 26.4, 95% CI 2.9–244.4). The 2nd outbreak investigated involved a function held the previous day, with 10 of 35 attendees becoming ill (one with laboratory confirmed *S.* Singapore infection). Similar foods were served at both functions. Five of 7 food handlers were also ill with a similar illness and all 5 cases reported consuming food at both functions. The chicken for both functions was purchased pre-cooked from a supermarket and then plated and stored for use. *S.* Virchow PT 34 was isolated from a sample of chicken obtained from the supermarket; however other food samples and swabs taken from both the supermarket and the cruise owner's premises were negative for pathogens. It is suspected that the outbreak was caused through cross contamination between raw and cooked product, and temperature abuse of the cooked product.

In late January in South Australia, 2 outbreaks of *S.* Typhimurium PT 9 were investigated following a sharp increase in notifications. Through hypothesis-generating interviews, it was found that bakery products were frequently consumed food items. A case-control study identified that custard filled items from 2 different bakeries were significantly associated with illness in a multivariate analysis: custard Berliners (OR 55.9, 95% CI 11.1–282.1) and cannolis (OR 16.8, 95% CI 1.8–157.2). Bakery A made the custard Berliners eaten by 43 cases, 19 of whom were hospitalised. Samples of product, raw materials and environmental swabs collected from bakery A were all negative for *Salmonella*. Bakery B made the cannolis that were eaten by 15 cases, three of whom were hospitalised. Products, raw materials and environmental swabs were collected from bakery B and product samples tested positive for *S.* Typhimurium PT 9. Investigators were unable to identify a common link to both bakeries via staff members, ingredients, processes, distribution chains or suppliers. Several months after the point source outbreaks had ceased, sporadic cases of the MLVA profiles observed during the outbreak were still being reported from the community. Further trace-back of ingredients supplied (but not used in implicated products) to the 2 premises investigated in the January outbreaks, supplemented with information from interviews with sporadic cases, found a common supplier of eggs. An investigation conducted at the egg farm found 3 of 26 samples collected to be positive for *S.* Typhimurium PT 9.

In Victoria in February, a large outbreak of *S.* Typhimurium PT 9 associated with the consumption of sushi containing raw egg mayonnaise was detected through routine surveillance. A number of cases were notified from the same pathology service located at a metropolitan hospital and 4 patients at the same hospital, reported eating sushi from the same premises prior to becoming ill. Three further cases were found through council food poisoning complaints. A total of 8 cases of gastroenteritis, including 59 confirmed cases of *S.* Typhimurium PT 9, were found to have eaten sushi from this premises. Two of the confirmed cases were food handlers at the premises and 19 (23%) cases were hospitalised with their illness. Twenty-five of 59 food samples and 5 of 17 environmental swabs were positive for *S.* Typhimurium PT 9. The mayonnaise used in the sushi hand rolls was made using raw eggs and a sample of the mayonnaise as well as environmental swabs of the blender used to make the mayonnaise were positive for *S.* Typhimurium PT 9. The eggs were traced back to a farm but samples taken on the farm were negative.

During the 1st quarter of 2011 in Queensland, 49 cases of *S.* Typhimurium PT 135a with MLVA profile 03-14-11-11-524 were reported with 6 hospitalisations. Interviews were conducted with 34 cases via a telephone-administered structured questionnaire. A sushi outlet located in a suburban shopping precinct was associated with 7 cases, while others were associated with a restaurant (3 cases), a café (5 cases) and another sushi venue (1 case and 1 epidemiologically-linked case). The remaining cases were community-acquired and not associated with a particular venue. Investigations at each of the premises identified that all were sourcing eggs from the same farm. No other common links were identified

among the food establishments. Food preparation, handling and storage procedures in each of these premises were investigated and environmental sampling conducted. An extensive environmental audit of the implicated farm detected the outbreak strain as well as *S.* Montevideo, *S.* Anatum, *S.* Kottbus and *S.* monophasic Subsp 1. *S.* Montevideo was also detected in eggs sampled at the retail level. No *Salmonella* were detected in environmental samples taken from the sushi outlet that was epidemiologically linked to seven of the cases.

In Queensland in September a suspected foodborne outbreak affecting 38 of 115 guests who attended a catered wedding was investigated. A retrospective cohort study identified multiple foods served at the reception (fried rice, egg fu yung, chicken and mussels) were associated with an increased risk of illness (RR 1.9–2.1). High levels of coagulase positive *Staphylococcus aureus* and emetic and diarrhoeal strains of *Bacillus cereus* were detected in mixed leftover samples of multiple dishes served at the function. High levels of *Cl. perfringens* were reported in samples of fried rice, staphylococcal enterotoxin was detected in samples of fried rice and chicken, and high levels of *Escherichia coli* were detected in corned meat. Six faecal specimens from cases grew coagulase positive staphylococci. Staphylococcal enterotoxin and *B. cereus* were not detected in any of the clinical specimens. Inappropriate timing of food preparation resulting in long holding times, inadequate food storage, inappropriate defrosting of food and lack of knowledge in safe food handling practice were considered major contributing factors in this outbreak.

In Victoria in September an outbreak of gastroenteritis in 41 of 184 attendees at a sports club dinner was investigated. Analysis of food history information for 66 attendees showed a statistically significant association between illness and consumption of roast beef (RR 12.7, 95% CI 3.3–48.0). *Cl. perfringens* enterotoxin was detected in 11 of 12 faecal specimens collected. The beef was roasted the day before the dinner, and then kept in the cool-room. The following day the meat was sliced thinly on a slicing machine and then placed into a warmer, without being re-heated. The meat slicer was found to be unclean with pieces of meat and meat juices behind the blade. No leftover food was available for testing.

In September in Western Australia, an outbreak of gastroenteritis caused by *Campylobacter* and *Salmonella* affected 65 of 705 attendees (attack rate of 9.2%) at a gala dinner at a function centre. Of 6 confirmed cases, two were positive for both *Campylobacter* and *S.* Typhimurium, one was positive for both *Campylobacter* and *S.* Infantis, and 3 were positive for *Campylobacter* only. A self-administered questionnaire regarding illness and food consumed was completed by 136 attendees and multivariate analysis of significant food exposures found that illness was statistically associated with consumption of duck parfait (OR 13, 95% CI 1.9–91.5, $P=0.01$). None of the parfait served at the dinner was available to be tested at the time of the investigation. A sample of frozen duck livers from a different batch to that used in the parfait served at the function was positive for *S.* Orion and *Campylobacter*.

An outbreak of *S.* Typhimurium PT 170/108 with a common MLVA profile 03-09-07-14-523 was identified in the Australian Capital Territory in November following reports of admissions for *Salmonella* gastroenteritis by a local hospital, with the notifying clinician also advising that the cases had eaten at a common bakery. Hypothesis generating interviews revealed most cases had eaten chicken Caesar salad rolls containing raw egg mayonnaise. In total, 41 cases of gastroenteritis were linked to the bakery, including 23 laboratory confirmed cases. The outbreak strain was identified in samples of the raw egg mayonnaise used by the bakery. A trace-back investigation of the eggs used by the bakery identified an egg producer in New South Wales. The outbreak strain was also identified in specimens collected at the farm.

Cluster investigations

In August 2011, in response to a national increase in *S.* Typhimurium PT 193 notifications and *S.* Typhimurium notifications with an MLVA pattern traditionally associated with PT 193, OzFoodNet commenced a national cluster investigation. The aim of this investigation was to form a hypothesis as to the source of the increase. Cases were interviewed using a hypothesis generating questionnaire, which included a trawling section on pork and beef related foods, contact with cows and pigs, contact with dogs and cats and food eaten by cats or dogs including pet treats. The investigation continued into late 2012 and results will be summarised in the 2012 report.

Multi-jurisdictional outbreak investigations

On 17 March 2011, OzFoodNet commenced multi-jurisdictional outbreak investigations into *S.* Virchow PT 34 and *S.* Typhimurium PT 170/108. The *S.* Virchow PT 34 investigation was commenced after a temporal cluster of 13 cases was detected in Victoria (the Victorian 5-year average for the same time period was 2 cases). Cases were also notified in South Australia, Tasmania, Queensland, New South Wales and the Australian Capital Territory during the same period. Jurisdictions conducted hypothesis-generating

interviews with notified cases of *S.* Virchow PT 34 using a standardised *Salmonella* questionnaire developed in Victoria. Data from the interviews were entered onto a national database and analysed for common exposures to develop food frequencies. Victoria also conducted targeted sampling of some high risk foods identified during case interviews. Forty-nine cases of *S.* Virchow PT 34 were interviewed by jurisdictions during the investigation (26 from Victoria). Two of these were considered to have been secondary cases. The median age of cases was 11 years (range 4 months to 90 years). *S.* Virchow 34 was isolated from the external surface of one batch of eggs sampled in Victoria during the investigation. A trace back investigation of this brand of eggs identified the source farm but no samples taken at the farm were positive for S. Virchow 34. While a range of foods such as eggs were consumed by the majority of cases, the products were from a range of retailers and were different brands, and no source of infection was definitively identified. The investigation was stood down on 1 June 2011 due to notifications returning to background levels.[28]

In 2011, *S.* Typhimurium PT 170/108 was observed to be the largest single contributor to a substantial increase in *Salmonella* notifications nationally and warranted further investigation. From January to May 2011, OzFoodNet epidemiologists investigated 13 point source *S.* Typhimurium PT 170/108 outbreaks that affected at least 124 people with 35 hospitalisations (hospitalisation rate 28.2%) and 1 death (case fatality rate 0.8%). A food vehicle was identified for nine of the 13 foodborne outbreaks. Seven of the 9 (77%) outbreaks with a known food vehicle were suspected to be due to eggs, or a food containing raw or lightly cooked eggs (Table 13, Appendix). As this phage type was the largest single contributor to the increase in *Salmonella* notifications nationally and because these point source outbreaks only accounted for 12% of the cases of *S.* Typhimurium PT 170/108 notified during this period, the investigation also included interviewing sporadic cases not linked to any of the identified point source outbreaks. Investigation of the sporadic cases of *S.* Typhimurium PT 170/108 notified during this period did not provide any additional evidence of the source(s) of infection. As cases demonstrated poor recall of food histories, associations between illness and the consumption of specific food items were difficult to establish for commonly eaten foods such as chicken and eggs. The investigation ceased on 1 June 2011 with declining notifications.[28]

A multi-jurisdictional outbreak investigation was initiated in December following reports of gastroenteritis in passengers (from New South Wales, Victoria, South Australia and Western Australia) and crew aboard a West Australian-owned ship cruising Papua New Guinea (PNG). There were 3 confirmed *S.* Typhimurium PT 135a cases (1 case each from South Australia, Victoria and Western Australia) (Appendix). Sixteen of the 31 passengers and crew reported illness. Of these, questionnaires were completed for 7 passengers and 7 crew members. Two crew members and 1 passenger were hospitalised. There was no epidemiological association between illness and eating a particular food item. The majority of food consumed on the ship was supplied from Australia, mostly from Queensland. All meat was from Western Australia. Some produce from PNG was used on board, including eggs, fruits, cucumber, pumpkin and coconut. A number of sauces, (mayonnaise, hollandaise and anglaise) and desserts (ice cream and tiramisu) contained raw eggs. An inspection of the vessel was conducted, but no samples were collected and the food vehicle was not identified. The investigation was completed in early 2012.

Discussion

This report documents the incidence of gastrointestinal diseases that may be transmitted by food in Australia during 2011. The OzFoodNet surveillance network concentrates its efforts on the surveillance and outbreak investigation of foodborne diseases. This is based on partnerships with a range of stakeholders, including state and territory health departments, food safety regulators, public health laboratories, and government departments of primary industries. These partnerships and the analysis of data on notified cases and outbreaks contribute to public health action, the prevention of disease and the assessment of food safety policies and campaigns. A national program of surveillance for foodborne diseases and outbreak investigation such as OzFoodNet has many benefits including identifying foods that cause human illness through investigation of outbreaks that occur across state and territory borders. Continuing to strengthen the quality of these data will ensure their use by agencies to develop food safety policy contributing to the prevention of foodborne illness. This aims to reduce the cost of foodborne illness to the community, such as healthcare costs and lost productivity, and those to industry such as product recalls and loss of reputation.

Campylobacter continues to be the most frequently notified enteric pathogen under surveillance by OzFoodNet despite not being notifiable in New South Wales. In fact, 2011 saw the highest number of notifications for campylobacteriosis since the commencement of the NNDSS in 1991. *Campylobacter* species were only implicated in 10 of 151 (7%) foodborne or suspected foodborne outbreaks in 2011, similar to 2010 (9/154, 6%).

Subtyping of *Campylobacter* species is not routinely performed in Australia hampering outbreak detection, but many previous OzFoodNet outbreak investigations have identified consumption of undercooked poultry livers as a particular risk for outbreaks of campylobacteriosis. It is important that poultry livers are handled in such a way as to avoid cross-contamination and are cooked thoroughly before eating.[29] As a result of the increasing notifications of campylobacteriosis in Australia, OzFoodNet put this issue to the Food Safety Information Council (http://www.foodsafety.asn.au/); a non-government organisation that produces and disseminates community food safety information. The Food Safety Information Council made campylobacteriosis prevention a major focus for their Australian Food Safety Week 2012 campaign. FSANZ also published a fact sheet on how to cook poultry liver safely (http://www.foodstandards.gov.au/consumer/safety/poultryliver/pages/default.aspx).

In 2011, both total *Salmonella* notifications (12,271) and the national notification rate of 54.3 cases per 100,000 population were also at the highest levels since the commencement of the NNDSS in 1991, surpassing 2010 the previous highest year (11,992 notifications, 53.7 cases per 100,000) and a 23% increase on the 5-year historical mean rate (44.1 cases per 100,000). OzFoodNet sites reported 151 foodborne or suspected foodborne outbreaks, including a multi-jurisdictional outbreak investigation. *Salmonella* continued to be the leading cause of reported outbreaks of foodborne illness in Australia, with 40% of outbreaks (61/151) due to this pathogen and 92% of these (56/61) due to *S*. Typhimurium. Of these 56 *S*. Typhimurium outbreaks, including community clusters, 52% (29/56) were associated with egg-based dishes.

OzFoodNet has been monitoring a national increase in *Salmonella* outbreaks associated with the consumption of raw or minimally cooked eggs since 2008. *S*. Typhimurium PT 170/108 and related MLVA types have most frequently been associated with these outbreaks. In 2011, *S*. Typhimurium PT 170/108 was the aetiological agent identified in 9 of these outbreaks across a range of settings and food vehicles, compared with 13 in 2010. Food vehicles identified during outbreak investigations included raw egg mayonnaise and dressings, desserts containing raw eggs such as tiramisu and chocolate mousse and raw cake batter. Notifications of salmonellosis peaked in January and the multi-jurisdictional investigation was launched in March because *S*. Typhimurium PT 170/108 was recognised as the largest contributor to the national increase.

OzFoodNet established a working group to describe the national epidemiology of egg-associated salmonellosis outbreaks and state and territory food safety authorities developed communication and education programs in relation to the use of raw egg products in commercial settings. The *Primary Production and Processing Standard for Eggs and Egg Products* was also gazetted in May 2011 and in force from 26 November 2012.[20] This Standard places legal obligations on egg producers and processors to introduce measures to reduce food safety hazards. It also includes traceability of individual eggs for sale or used to produce egg pulp. Further information on the implementation of the egg standard at the state and territory level is available on the Department of Health web site (http://www.health.gov.au/internet/main/publishing.nsf/Content/foodsecretariat-isc-model.htm).

This was the 1st full year of the OzFoodNet NELSS. Typing, demographic and exposure data for all nationally notified listeriosis cases were entered by OzFoodNet epidemiologists into a centralised national database from which fortnightly and ad hoc reports were generated and shared through all OzFoodNet sites. Creating a standardised national database of typing and risk exposures allowed rapid cluster detection and facilitated case–case analysis.[30] No multi-jurisdictional clusters or outbreaks were detected, but a cluster of 6 cases was identified and investigated in Victoria. An evaluation found that NELSS was meeting its objectives of monitoring the epidemiology of invasive listeriosis infections over time and detecting clusters and outbreaks.[31]

The largest recorded international outbreak of STEC occurred in northern Germany in 2011. Up to 15 other countries also recorded cases among people who had travelled to northern Germany during the outbreak. With a total of 3,816 cases, including 845 cases of HUS and 54 deaths, the outbreak was attributed to consumption of contaminated fenugreek sprouts.[32] On 3 June 2011, CDNA held an emergency teleconference that included OzFoodNet, the PHLN and FSANZ that confirmed that Australia has the surveillance and testing capacity to detect any possible cases of the outbreak strain (STEC O104:H4) if they arose in Australia. OzFoodNet monitored the outbreak closely and assessed Australia's capacity to respond to an outbreak of similar magnitude from the epidemiological perspective. OzFoodNet also collaborated closely with FSANZ to ensure that the implicated foods had not been imported into Australia.

South Australia experienced a significant STEC outbreak in 2011 associated with a petting zoo at an agricultural show. As a result of the inves-

tigation into this outbreak, OzFoodNet funded a review of the existing *South Australia Petting Zoo Infection Control Guidelines* with a view to formulating national guidelines.

Hepatitis A notifications (144) and the notification rate (0.6 per 100,000 population) reached the lowest levels in 2011 ever recorded in the NNDSS. There were also only 2 notified cases of hepatitis A infection in Aboriginal and Torres Strait Islander people in Australia in 2011, which represented only 1.4% of the total notifications. This compares with rates of 10%–15% in the early 2000s.[33] This is further evidence of the success of the staged introduction of hepatitis A vaccination programs targeted at young Aboriginal and Torres Strait Islander children from 1999 onwards in Queensland, the Northern Territory, South Australia and Western Australia.[34, 35]

OzFoodNet recognises some of the limitations of the data used in this report. Where there are small numbers of notifications, caution must be used in comparisons between jurisdictions and over time. Some of the most common enteric pathogens such as norovirus and *Cl. perfringens* are not notifiable in any Australian jurisdiction, and *Campylobacter* is not notifiable in New South Wales, which is why investigation of outbreaks is important. A further limitation relates to the outbreak data provided by OzFoodNet sites for this report and the potential for variation in categorising features of outbreaks depending on investigator interpretation and circumstances. State and territory representatives are involved in a continuous program aimed at harmonising the collection and recording of the outbreak data via the Outbreak Register Working Group.

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Appendix: Summary of foodborne and suspected foodborne disease outbreaks reported by OzFoodNet sites, Australia, 2011 (n=151)

State or territory	Month*	Setting prepared	Agent responsible	Ill	Hospitalised	Deaths	Evidence	Epidemiological study	Responsible vehicles	Commodity	Contamination factor
MJOI	Nov	Cruise/ airline	*S.* Typhimurium PT 135a, PFGE 0039, MLVA 03-08-10-14/16-523	16	3	0	D	Point source cohort	Unknown	Not attributed	Unknown
ACT	Feb	Takeaway	*S.* Typhimurium PT 197	9	1	0	D	Case series	Kebabs	Poultry, lamb	Other source of contamination and inadequate cleaning of equipment
ACT	Jun	Restaurant	Unknown	6	0	0	D	Case series	Burgers, schnitzels, chips and salad	Multiple	Unknown
ACT	Jun	Private residence	*S.* Typhimurium PT 135	5	1	0	D	No formal study	Spit roasted pig	Pork	Cross contamination from raw ingredients and ingestion of contaminated raw products
ACT	Oct	Commercial caterer	Unknown	9	0	0	D	Case series	Mixed sandwiches	Not attributed	Person to food to person
ACT	Nov	Bakery	*S.* Typhimurium PT 170/108, MLVA 03-09-07-14-523	41	6	0	M	Case series	Chicken caesar roll containing raw egg mayonnaise	Eggs	Ingestion of contaminated raw products
NSW	Jan	Grocery store/ delicatessen	*S.* Singapore	45	2	0	AM	Point source cohort	Roast chicken pieces served cold	Poultry	Cross contamination from raw ingredients
NSW	Jan	Grocery store/ delicatessen	*S.* Singapore	10	0	0	M	Case series	Roast chicken pieces served cold	Poultry	Cross contamination from raw ingredients
NSW	Jan	Restaurant	*S.* Typhimurium PT 3, MLVA 03-13-14-9/10-523	11	1	0	M	Case series	Caesar salad dressing – raw egg	Eggs	Ingestion of contaminated raw products
NSW	Jan	Restaurant	*S.* Typhimurium, MLVA 03-11-11-10-523	10	0	0	D	Case series	Dessert containing raw egg custard	Eggs	Unknown
NSW	Jan	Bakery	*S.* Typhimurium PT 135, MLVA 03-12-09-10-550	9	0	0	D	Case series	Unknown	Not attributed	Cross contamination from raw ingredients and inadequate cleaning of equipment
NSW	Jan	Takeaway	*S.* Typhimurium PT 44, MLVA 03-10-08-09-523	85	17	0	M	Case series	Vietnamese pork/ chicken/salad rolls containing raw egg butter	Eggs	Ingestion of contaminated raw products and cross contamination from raw ingredients
NSW	Jan	Restaurant	Unknown	7	0	0	D	No formal study	Unknown	Not attributed	Unknown
NSW	Jan	School	*S.* Typhimurium PT 170/108, MLVA 03-09-08-13-523	17	1	0	D	Point source cohort	Apple turnover	Multiple	Unknown
NSW	Jan	Restaurant	Unknown	5	0	0	D	No formal study	Unknown	Not attributed	Unknown

Appendix, *continued*: Summary of foodborne and suspected foodborne disease outbreaks reported by OzFoodNet sites, Australia, 2011 (n=151)

State or territory	Month*	Setting prepared	Agent responsible	Ill	Hospitalised	Deaths	Evidence	Epidemiological study	Responsible vehicles	Commodity	Contamination factor
NSW	Feb	Restaurant	*Campylobacter*	11	0	0	AM	Point source cohort	Chicken liver pâté on toast	Poultry	Unknown
NSW	Feb	Restaurant	Unknown	3	0	0	D	Case series	Unclear	Not attributed	Unknown
NSW	Feb	Restaurant	*S.* Typhimurium PT 170/108, MLVA 03-09-07-14-523	6	2	0	M	Case series	Fried ice cream	Eggs	Ingestion of contaminated raw products
NSW	Feb	Restaurant	Unknown	36	0	0	D	No formal study	Suspected dessert	Not attributed	Unknown
NSW	Mar	Restaurant	Unknown	7	0	0	D	Case series	Unknown	Not attributed	Unknown
NSW	Mar	Takeaway	*S.* Typhimurium PT 170/108, MLVA 03-09-07-14-523	2	0	0	D	No formal study	Unknown	Not attributed	Unknown
NSW	Mar	Takeaway	*S.* Typhimurium PT 9, MLVA 03-10-14-12-496	5	2	0	D	No formal study	Beef kebab with onion, lettuce, tomato, cheese, BBQ & garlic sauce	Not attributed	Cross contamination from raw ingredients
NSW	Apr	Restaurant	Unknown	3	0	0	D	Case series	Suspect prawn and pesto pizza	Not attributed	Unknown
NSW	Apr	Private residence	*S.* Typhimurium MLVA 03-13-12-10-523	3	0	0	D	Case series	Homemade hollandaise sauce and semifreddo	Eggs	Unknown
NSW	Apr	Restaurant	Unknown	6	0	0	D	Case series	Unknown	Not attributed	Unknown
NSW	Apr	Other	Unknown	80	0	0	D	Point source cohort	Unknown	Not attributed	Unknown
NSW	May	Commercial caterer	Norovirus G II-6	23	0	0	A	Point source cohort	Suspect chocolate and mandarin pie	Not attributed	Unknown
NSW	May	Restaurant	*S.* Typhimurium MLVA 03-10-08-09-523	8	0	0	M	Case series	Chicken and corn soup with raw egg added	Poultry, eggs	Cross contamination from raw ingredients
NSW	May	Takeaway	Unknown	4	0	0	D	Case series	Unknown	Not attributed	Unknown
NSW	May	Restaurant	Norovirus	79	12	0	D	Point source cohort	Person-to-person transmission via infected food handler	Not attributed	Food handler contamination
NSW	May	Restaurant	*S.* Typhimurium PT 135, MLVA 03-13-11-09-523	4	2	0	D	Case series	Suspect prawn dumplings with egg to bind	Crustaceans, leafy vegetables, eggs	Unknown
NSW	Jul	Takeaway	Unknown	3	1	0	D	No formal study	Unknown	Not attributed	Unknown

Appendix, *continued*: Summary of foodborne and suspected foodborne disease outbreaks reported by OzFoodNet sites, Australia, 2011 (n=151)

State or territory	Month*	Setting prepared	Agent responsible	Ill	Hospitalised	Deaths	Evidence	Epidemiological study	Responsible vehicles	Commodity	Contamination factor
NSW	Jul	Restaurant	Unknown	2	0	0	D	No formal study	Unknown	Not attributed	Unknown
NSW	Jul	Restaurant	Unknown	2	0	0	D	No formal study	Unknown	Not attributed	Unknown
NSW	Jul	Restaurant	*S.* Typhimurium, MLVA 03-09-08-14-523	13	1	0	D	Case series	Tiramisu containing raw egg	Eggs	Ingestion of contaminated raw products
NSW	Aug	Restaurant	*S.* Typhimurium, MLVA 03-09-07-13-523	6	0	0	D	Case series	Raw egg dressing	Eggs	Ingestion of contaminated raw products
NSW	Aug	Restaurant	*S.* Typhimurium, MLVA 03-09-07-15-523	3	0	0	D	Case series	Raw egg mayonnaise	Eggs	Ingestion of contaminated raw products
NSW	Aug	Commercial caterer	Unknown	25	0	0	D	Point source cohort	Unknown	Not attributed	Unknown
NSW	Aug	Restaurant	Unknown	11	0	0	D	Case series	Unknown	Not attributed	Unknown
NSW	Sep	Restaurant	Unknown	3	0	0	D	Case series	Unknown	Not attributed	Unknown
NSW	Sep	Restaurant	*Campylobacter*	2	0	0	D	No formal study	Unknown	Not attributed	Unknown
NSW	Sep	Commercial caterer	Unknown	87	0	0	A	Point source cohort	Salad of poached prawns with Thai herbs	Multiple	Unknown
NSW	Sep	Restaurant	Unknown	4	0	0	D	Case series	Unknown	Not attributed	Unknown
NSW	Sep	Restaurant	Unknown	6	0	0	D	No formal study	Madras chicken curry with rice	Not attributed	Unknown
NSW	Oct	Camp	Unknown	8	4	0	D	No formal study	Cooked pasta	Grains-beans	Other source of contamination
NSW	Oct	Bakery	Unknown	3	0	0	D	Case series	Unknown	Not attributed	Unknown
NSW	Oct	Restaurant	*S.* Typhimurium PT 9	3	1	0	D	Case series	Unknown	Not attributed	Unknown
NSW	Nov	Commercial caterer	Unknown	16	0	0	AM	Point source cohort	Suspect lamb curry	Not attributed	Unknown
NSW	Nov	Restaurant	Unknown	4	4	0	D	Case series	Tuna	Fish	Toxic substance or part of tissue
NSW	Nov	Restaurant	Unknown	12	0	0	D	No formal study	Unknown	Not attributed	Unknown
NSW	Nov	Restaurant	*Campylobacter*	2	0	0	D	Case series	Unknown	Not attributed	Unknown
NT	Jan	Camp	*S.* Typhimurium PT 9	3	0	0	D	No formal study	Unknown	Not attributed	Unknown
NT	Feb	Camp	Suspected viral	3	1	0	D	No formal study	Unknown	Not attributed	Unknown
NT	Feb	Aged care	Unknown	4	1	0	D	No formal study	Unknown	Not attributed	Unknown
NT	May	Commercial caterer	*S.* Typhimurium PT 141	5	0	0	D	No formal study	Unknown	Not attributed	Unknown
NT	Jul	Private residence	*S.* Saintpaul	5	0	0	D	No formal study	Unknown	Not attributed	Unknown

Appendix, *continued*: Summary of foodborne and suspected foodborne disease outbreaks reported by OzFoodNet sites, Australia, 2011 (n=151)

State or territory	Month*	Setting prepared	Agent responsible	Ill	Hospitalised	Deaths	Evidence	Epidemiological study	Responsible vehicles	Commodity	Contamination factor
NT	Sep	Fair/festival/ mobile service	*S.* Saintpaul	5	0	0	D	Case series	Suspect mango	Fruits-nuts	Unknown
NT	Oct	Takeaway	Unknown	3	0	0	D	Point source cohort	Unknown	Not attributed	Unknown
Qld	Jan	Unknown	*S.* Typhimurium PT 135a, MLVA 03-14-11-11-524	49	6	0	M	Case series	Eggs	Eggs	Ingestion of contaminated raw products
Qld	Mar	Primary produce	Ciguatera fish poisoning	3	0	0	D	Case series	Red Bass	Fish	Toxic substance or part of tissue
Qld	Jun	Private residence	*Campylobacter*	4	0	0	D	Case series	Chicken kebabs	Poultry	Ingestion of contaminated raw products
Qld	Jul	Primary produce	Ciguatera fish poisoning	3	0	0	D	Case series	Reef fish (unknown species)	Fish	Toxic substance or part of tissue
Qld	Jul	Restaurant	*Clostridium perfringens*	3	0	0	M	Case series	Chicken curry	Poultry, vegetables, grains	Unknown
Qld	Aug	Primary produce	Ciguatera fish poisoning	3	0	0	D	Case series	Coral trout	Fish	Toxic substance or part of tissue
Qld	Sep	Hospital	*Campylobacter*	5	5	0	D	Case series	Unknown	Not attributed	Unknown
Qld	Sep	Commercial caterer	*Staphylococcus aureus*	38	1	0	AM	Point source cohort	Fried rice; chicken; egg fu yung; mussels	Not attributed	Person to food to person
Qld	Sep	Commercial caterer	*S.* Typhimurium PT 170/108, MLVA 03-09-07-12-524	14	11	0	D	Case series	Unknown	Not attributed	Inadequate cleaning of equipment
Qld	Oct	Restaurant	Unknown	3	Unknown	0	D	Case series	Unknown	Not attributed	Unknown
Qld	Oct	Restaurant	Norovirus	6	0	0	D	Case series	No vehicle identified	Not attributed	Person to food to person
Qld	Nov	Restaurant	Histamine fish poisoning	3	3	0	M	Case series	Yellow-tail kingfish	Fish	Toxic substance or part of tissue
Qld	Nov	Fair/festival/ mobile service	*S.* Birkenhead	37	9	0	D	Point source cohort	Pumpkin or potato curry	Multiple	Unknown
Qld	Nov	Restaurant	Unknown	19	0	0	D	Point source cohort	No vehicle identified	Not attributed	Unknown
Qld	Nov	Primary produce	Ciguatera fish poisoning	6	0	0	D	Case series	Spanish mackerel	Fish	Toxic substance or part of tissue

Appendix, *continued*: Summary of foodborne and suspected foodborne disease outbreaks reported by OzFoodNet sites, Australia, 2011 (n=151)

State or territory	Month*	Setting prepared	Agent responsible	Ill	Hospitalised	Deaths	Evidence	Epidemiological study	Responsible vehicles	Commodity	Contamination factor
Qld	Dec	Primary produce	Ciguatera fish poisoning	2	0	0	D	Case series	Coral trout	Fish	Toxic substance or part of tissue
Qld	Dec	Restaurant	*S.* Typhimurium 197, MLVA 04-15-09-09-490	25	2	0	D	Point source cohort	Unknown	Not attributed	Unknown
Qld	Dec	National franchised fast food	Unknown	4	0	0	D	Case series	Unknown	Not attributed	Unknown
SA	Jan	Bakery	*S.* Typhimurium PT 9, MLVA 03-24-11-10/11-523	43	19	0	A	Case control study	Custard berliner bun	Multiple	Unknown
SA	Jan	Bakery	*S.* Typhimurium PT 9 MLVA 03-24-11-10/11-523	15	3	0	AM	Case control study	Custard cannolis	Multiple	Unknown
SA	Feb	Bakery	*S.* Typhimurium PT 44	8	0	0	D	Case series	Unknown	Not attributed	Unknown
SA	Feb	Bakery	*S.* Typhimurium PT 135	6	2	0	M	Case series	Egg glaze	Eggs	Cross contamination from raw ingredients
SA	Mar	Private residence	Unknown	16	1	0	D	No formal study	Unknown	Not attributed	Unknown
SA	Jul	Institution	*S.* Typhimurium PT 170/108 and *Campylobacter*	4	0	0	D	Case series	Unknown	Not attributed	Unknown
SA	Aug	Institution	*Campylobacter*	9	0	0	D	Point source cohort	Unknown	Not attributed	Unknown
SA	Sep	Private residence	Norovirus	16	0	0	D	Point source cohort	Unknown	Not attributed	Unknown
SA	Sep	Restaurant	*S.* Typhimurium PT 126	4	2	0	D	Case series	Unknown	Not attributed	Unknown
SA	Nov	Commercial caterer	*S.* Typhimurium PT 9, MLVA 03-24-13-11-523	27	7	0	D	Case series	Multiple foods	Not attributed	Cross contamination from raw ingredients and inadequate cleaning of equipment
Tas.	Nov	Unknown	*Campylobacter*	5	0	0	D	No formal study	Unknown	Not attributed	Unknown
Vic.	Jan	Aged care	Unknown	9	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Jan	Takeaway	*S.* Typhimurium PT 9	84	19	0	M	Case series	Mayonnaise (raw eggs)	Eggs	Ingestion of contaminated raw products
Vic.	Jan	Takeaway	*S.* Typhimurium PT 170/108	15	6	0	D	Case series	Salty fish, pork and egg dish	Fish, pork, eggs	Unknown
Vic.	Jan	Private residence	*S.* Typhimurium PT 44	5	0	0	D	Case series	Tiramisu	Eggs	Ingestion of contaminated raw products
Vic.	Feb	Aged care	*Clostridium perfringens*	23	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Feb	Aged care	*Clostridium perfringens*	7	0	0	D	Case series	Unknown	Not attributed	Unknown

Appendix, *continued*: Summary of foodborne and suspected foodborne disease outbreaks reported by OzFoodNet sites, Australia, 2011 (n=151)

State or territory	Month*	Setting prepared	Agent responsible	Ill	Hospitalised	Deaths	Evidence	Epidemiological study	Responsible vehicles	Commodity	Contamination factor
Vic.	Feb	Hospital	*S.* Typhimurium PT 135	7	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Feb	Takeaway	*S.* Typhimurium PT 170/108	26	6	0	D	Case series	Mayonnaise (raw eggs)	Eggs	Ingestion of contaminated raw products
Vic.	Feb	Aged care	*Campylobacter*	15	2	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Feb	Private residence	*S.* Typhimurium PT 170/108	2	1	0	D	Case series	Uncooked muffin batter	Eggs	Ingestion of contaminated raw products
Vic.	Mar	Aged care	Unknown	9	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Mar	Aged care	*Clostridium perfringens*	9	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Mar	Bakery	*S.* Typhimurium PT 135	17	3	0	M	Case series	Chicken liver pâté	Poultry	Unknown
Vic.	Mar	Aged care	Unknown	9	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Mar	Aged care	*S.* Typhimurium PT 170/108	6	5	1	D	Case series	Unknown	Not attributed	Unknown
Vic.	Mar	Private residence	*S.* Typhimurium PT 141	2	1	0	D	Case series	Chocolate mousse containing raw eggs	Eggs	Ingestion of contaminated raw products
Vic.	Apr	Restaurant	*S.* Typhimurium PT 170/108	15	2	0	AM	Point source cohort	Fried ice cream	Eggs	Ingestion of contaminated raw products
Vic.	Apr	Private residence	*S.* Typhimurium PT 170/108	2	2	0	M	Case series	Pancake batter	Eggs	Ingestion of contaminated raw products
Vic.	Apr	Private residence	*S.* Typhimurium PT 135a	9	5	0	D	Case series	Raw egg mayonnaise on potato salad	Eggs	Ingestion of contaminated raw products
Vic.	Apr	Aged care	*Clostridium perfringens*	5	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Apr	Private residence	*S.* Typhimurium PT 9	9	1	0	D	Point source cohort	Unknown	Not attributed	Unknown
Vic.	May	Aged care	*Clostridium perfringens*	13	0	1	D	Case series	Unknown	Not attributed	Unknown
Vic.	May	Aged care	Unknown	10	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	May	Aged care	*Clostridium perfringens*	8	0	1	D	Case series	Unknown	Not attributed	Unknown
Vic.	May	Aged care	Unknown	12	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	May	Aged care	Unknown	6	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	May	Restaurant	Norovirus	24	4	0	D	Point source cohort	Chicken parmigiana	Not attributed	Unknown
Vic.	Jun	Hospital	*Clostridium perfringens*	11	Unknown	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Jun	Restaurant	Unknown	9	0	0	D	Case series	Curries	Not attributed	Unknown
Vic.	Jun	Aged care	Unknown	8	0	0	A	Point source cohort	Vitamised food	Multiple	Unknown

Appendix, *continued*: Summary of foodborne and suspected foodborne disease outbreaks reported by OzFoodNet sites, Australia, 2011 (n=151)

State or territory	Month*	Setting prepared	Agent responsible	Ill	Hospitalised	Deaths	Evidence	Epidemiological study	Responsible vehicles	Commodity	Contamination factor
Vic.	Jun	Aged care	Unknown	5	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Jun	Aged care	Unknown	5	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Jun	Restaurant	Norovirus	15	0	0	A	Point source cohort	Fruit	Fruits-nuts	Unknown
Vic.	Jun	Restaurant	Unknown	7	0	0	D	Case series	Beef rendang or pork satay	Multiple	Unknown
Vic.	Jun	Private residence	*S.* Typhimurium PT 9	7	0	0	D	Case series	Chocolate mousse containing raw eggs	Eggs	Ingestion of contaminated raw products
Vic.	Jul	Aged care	Clostridium perfringens	11	1	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Aug	Restaurant	*S.* Typhimurium PT 170/108	14	1	0	A	Point source cohort	Chocolate mousse	Eggs	Ingestion of contaminated raw products
Vic.	Aug	Private residence	*S.* Typhimurium PT 135	4	0	0	A	Point source cohort	Uncooked pasta dough containing eggs	Eggs	Unknown
Vic.	Aug	Aged care	*Clostridium perfringens*	7	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Sep	Aged care	*Clostridium perfringens*	14	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Sep	Commercial caterer	*Staphylococcus aureus*	28	1	1	AM	Point source cohort	Lamprias	Fish, beef, vine-stalks	Unknown
Vic.	Sep	Commercial caterer	*Clostridium perfringens*	41	0	0	A	Point source cohort	Roast beef	Beef	Unknown
Vic.	Sep	Private residence	*S.* Typhimurium PT 44	12	0	0	D	Case series	Tiramisu containing raw eggs	Eggs	Ingestion of contaminated raw products
Vic.	Oct	Hospital	*Clostridium perfringens*	8	Unknown	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Oct	Restaurant	Histamine fish poisoning	3	0	0	D	Case series	Tuna	Fish	Unknown
Vic.	Oct	Hospital	*Clostridium perfringens*	5	Unknown	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Oct	Private caterer	*Clostridium perfringens*	17	0	0	D	Case series	Roast beef suspected	Beef	Unknown
Vic.	Nov	Private residence	*S.* Typhimurium PT 9	4	0	0	D	Case series	Chocolate mousse	Eggs	Unknown
Vic.	Dec	Aged care	*Clostridium perfringens*	25	0	0	D	Case series	Unknown	Not attributed	Unknown
Vic.	Dec	Takeaway	*S.* Typhimurium PT 9	3	1	0	D	Case series	Chicken hand rolls	Poultry	Cross contamination from raw ingredients
Vic.	Dec	Restaurant	*Bacillus cereus*	12	0	0	M	Case series	Multiple foods	Multiple	Unknown

Appendix, *continued*: Summary of foodborne and suspected foodborne disease outbreaks reported by OzFoodNet sites, Australia, 2011 (n=151)

State or territory	Month*	Setting prepared	Agent responsible	Ill	Hospitalised	Deaths	Evidence	Epidemiological study	Responsible vehicles	Commodity	Contamination factor
Vic.	Dec	Restaurant	Unknown	4	1	0	A	Point source cohort	Moroccan chicken salad	Poultry	Unknown
Vic.	Dec	Restaurant	Unknown	14	0	0	A	Point source cohort	Mango sticky rice	Dairy, eggs, grains-beans, oils-sugars, fruits-nuts	Unknown
Vic.	Dec	Private residence	*S.* monophasic (I:4,5,12:I:-) PT 193	4	1	0	M	Case series	Pork salami	Pork	Unknown
Vic.	Dec	Takeaway	*S.* Typhimurium PT 170/108	37	11	1	M	Case series	Pizza and chocolate mousse containing raw egg	Eggs	Unknown
WA	Jan	Restaurant	*S.* Typhimurium PT 170/108, MLVA 03-10-07-13-526, PFGE 0011	4	1	0	D	Case series	Unknown	Not attributed	Unknown
WA	Jan	Takeaway	*S.* Typhimurium PT 9	15	5	0	D	Case series	Vietnamese pork roll made with raw egg butter	Eggs	Unknown
WA	Jan	Restaurant	*S.* Typhimurium PT 135, PFGE 0003	23	15	0	D	Case series	Unknown	Not attributed	Unknown
WA	Apr	Other	*S.* Typhimurium PT 193, PFGE 386	30	2	0	D	Case control study	Unknown	Not attributed	Unknown
WA	Jul	Restaurant	Norovirus	53	0	0	D	Case control study	Salad	Not attributed	Person to food to person
WA	Sep	Commercial caterer	*Campylobacter* and *S.* Infantis	65	0	0	AM	Case control study	Duck liver parfait	Poultry	Ingestion of contaminated raw products
WA	Nov	Private residence	Unknown	17	0	0	D	Case series	Chicken biryani	Multiple	Unknown
WA	Dec	Restaurant	Unknown	7	0	0	D	Case series	Unknown	Not attributed	Unknown
WA	Dec	Commercial caterer	Unknown	10	0	0	D	Case control study	Unknown	Not attributed	Unknown

* Month of outbreak is the month of onset of first case or month of notification/investigation of the outbreak. The number of people affected and hospitalised relate to the findings of the outbreak investigation at the time of writing and not necessarily in the month specified.

MJOI Multi-jurisdictional investigation

D Descriptive evidence implicating the vehicle

A Analytical epidemiological association between illness and vehicle

M Microbiological confirmation of aetiology in vehicle and cases

AM Analytical association and microbiological confirmation of aetiology

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Invasive pneumococcal disease in Australia, 2009 and 2010

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Abstract

Enhanced surveillance for invasive pneumococcal disease (IPD) was conducted in all Australian states and territories in 2009 and 2010 with comprehensive comparative data available since 2002. There were 1,556 cases of IPD notified to the National Notifiable Diseases Surveillance System in Australia in 2009, a notification rate of 7.2 cases per 100,000 population. In 2010 there were 1,640 cases, a notification rate of 7.4 cases per 100,000. The overall rate of IPD in Indigenous Australians was almost 6 times the rate in non-Indigenous Australians in both 2009 and 2010. In 2009 and 2010, notification rates of IPD caused by serotypes included in the 7-valent pneumococcal conjugate vaccine (7vPCV) continued to decrease across all age groups. Rates of IPD caused by non-7vPCV serotypes continued to show an increasing trend in both Indigenous and non-Indigenous children aged less than 5 years. In Indigenous adults (≥50 years), rates of IPD caused by both 23-valent pneumococcal polysaccharide vaccine (23vPPV) serotypes and non-23vPPV serotypes continued to show an overall increase, particularly in 2010. There were 110 deaths attributed to IPD in 2009 and 137 in 2010, although it should be noted that deaths may be under-reported. The number of invasive pneumococcal isolates with reduced penicillin susceptibility remained low and reduced susceptibility to third generation cephalosporins was rare. *Commun Dis Intell* 2015;39(2):E265–E279.

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

Introduction

Streptococcus pneumoniae infection is a major cause of vaccine preventable disease worldwide.[1,2] The organism colonises the nasopharynx of healthy carriers who show no symptoms of disease. In susceptible groups, the bacterium can spread to the respiratory tract and sterile sites, such as the blood, cerebrospinal fluid or pleural fluid, and cause disease ranging from mild, such as otitis media and sinusitis, to more severe, such as pneumonia, septicaemia and meningitis.[3] The burden of disease is greatest in infants and the elderly. The 23-valent pneumococcal polysaccharide vaccine (23vPPV) was first recommended in Australia prior to 1991 for certain high risk groups. A 7-valent pneumococcal conjugate vaccine (7vPCV) program with a 3+0 schedule (i.e. 2, 4 and 6 month schedule without a conjugate vaccine booster) was first funded by the National Immunisation Program (NIP) for Aboriginal and Torres Strait Islander infants in mid-2001 with children in areas of very high incidence also funded for a 23vPPV booster at 18–24 months.[4] The 23vPPV is currently funded nationally for all individuals aged 65 years or over and Aboriginal and Torres Strait Islanders aged 50 years or over. From January 2005, NIP-funded 7vPCV was extended to all infants nationally, together with catch-up vaccination for all children aged less than 2 years. High vaccination uptake of over 90% has been maintained since the implementation of universal infant pneumococcal vaccination.[4] Enhanced surveillance of risk factors, invasive pneumococcal disease (IPD)-specific clinical details, microbiological and vaccination history is carried out for IPD, which has been nationally notifiable in Australia since 2001. Some states and territories hold data from earlier years. Surveillance reports have been published in *Communicable Diseases Intelligence* for 2002 to 2008.[5–10] This report describes epidemiological, microbiological and disease trends for the years 2009 and 2010.

Methods

Data collection

IPD is a nationally notifiable disease in Australia and is monitored using the National Notifiable Diseases Surveillance System (NNDSS). Complete data have been reported to the NNDSS from all states and territories since 2002. To varying degrees across jurisdictions, medical practitioners, public health laboratories and other health professionals are required under state and territory public health legislation to report cases of IPD to state and territory health authorities. The *National Health Security Act 2007* provides the legislative basis for the national notification of communicable diseases and authorises the exchange of health information between the Commonwealth and the states and territories.[11] Notified cases of IPD that meet the national surveillance case definition are transferred by state and territory health departments to the NNDSS regularly.[12] The primary responsibility for public health action resulting from notification resides with state and territory health departments.

The Communicable Diseases Network Australia (CDNA) established the Enhanced Invasive Pneumococcal Disease Surveillance Working Group (EIPDSWG) in 2000 to assist in the development and implementation of a nationally standardised approach to the enhanced surveillance of IPD in Australia. In 2009 and 2010, core organism and diagnosis data were collected for all notified cases, whereas enhanced data, which includes clinical categories and risk factors, were collected to varying degrees across states and territories (Table 1).

Data presented in this report represent a point in time analysis of notified cases of IPD. This report presents data extracted in July 2014 and analysed by date of diagnosis. Date of diagnosis is a derived field within the NNDSS and represents the onset date, or when the onset date was not known, the earliest of the specimen collection, notification, or notification receive dates. Due to the dynamic nature of the NNDSS, data in this report may vary from data reported in other NNDSS reports and reports of IPD notifications at the state or territory level.

Australian Bureau of Statistics mid-year estimated resident populations were used to calculate notification rates.[13]

Case definition

Cases of IPD were notified according to the CDNA case definition for IPD.[14] A confirmed case requires definitive evidence only. Laboratory definitive evidence for IPD is the isolation from or detection by nucleic acid amplification test of *S. pneumoniae* in blood, cerebrospinal fluid or other sterile site.

Indigenous status

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

Cases reported with an unknown Indigenous status (2009:195/1,556, 13%; 2010: 193/1,640, 12%) were excluded from the analyses in this report relating to Indigenous status.

Vaccination

Pneumococcal vaccination for various specified age groups and other high risk populations has been recommended within Australia since before 1991. In 2009–2010, primary pneumococcal vaccination and specified boosters were recommended and funded under the NIP for the following groups:

- all infants;
- children aged under 10 years with specified underlying medical conditions;
- Aboriginal and Torres Strait Islander children aged 18–24 months and living in high risk areas;
- Aboriginal and Torres Strait Islander people aged 15–50 years with specified underlying medical conditions;
- all Aboriginal and Torres Strait Islander people aged 50 years or over; and
- all adults aged 65 years or over.

NIP pneumococcal vaccination schedules for these groups were unchanged from those used in 2008.[10] From October 2009, the 10-valent pneumococcal conjugate vaccine (10vPCV) was funded as a replacement to the 7vPCV in the Northern Territory for all children. However, in this report, Northern Territory cases were not analysed separately. There are now 4 vaccines available in Australia with each targeting multiple serotypes (Table 2); however, the13-valent pneumococcal conjugate vaccine (13vPCV) was not yet introduced to the Australian NIP during the years of this study, 2009 and 2010.

More information on the scheduling of the pneumococcal vaccination can be found on the Immunise Australia web site (www.immunise.health.gov.au). A detailed history on vaccination recommendations and practices is available through the National Centre for Immunisation Research and Surveillance (NCIRS).[15]

The evaluation of vaccination status in this report is described in Table 3. These definitions are applied to the vaccination fields reported to the NNDSS and are agreed to by the EIPDSWG.

Table 1: Enhanced invasive pneumococcal disease surveillance data collection performed by states and territories in 2009 and 2010

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

Table 2: Serotypes targeted by pneumococcal vaccines

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

Table 3: Definitions of vaccination status and vaccine failure used in this report

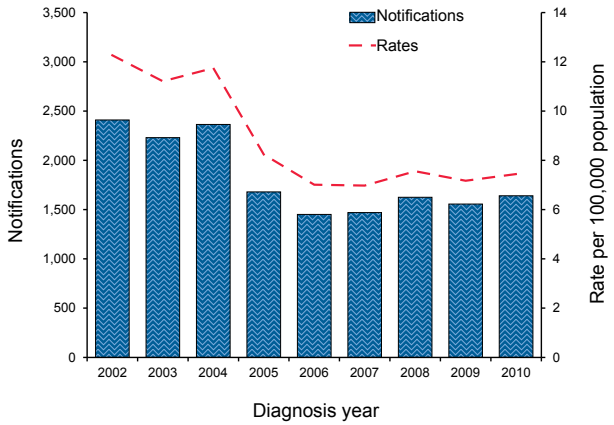
Category	Definition
Fully vaccinated	Those who have completed the primary course of the relevant vaccine(s) required for their age according to the most recent edition of *The Australian Immunisation Handbook,* at least 2 weeks prior to disease onset with at least 28 days between doses of vaccine. This includes the following; • a child who received a vaccine as 'catch up' and therefore did not require a full 3 dose primary schedule. Providing they have had the number of doses required for the age they were at first dose they should be considered fully vaccinated. 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 assessed as fully vaccinated.
Vaccination validation	Written confirmation of vaccination through the Australian Childhood Immunisation Register, state or territory immunisation register or health record.
Vaccine failure	A fully vaccinated child (as defined above) with disease due to a serotype found in the corresponding vaccine.

Results

Invasive pneumococcal disease notifications

There were 1,640 cases of IPD notified in 2010, representing an annual notification rate of 7.4 per 100,000 population. This was a 5% increase on the 1,556 cases reported in 2009 (7.2 per 100,000). The number of cases of IPD notified to the NNDSS has been stable since 2005, when the universal pneumococcal conjugate vaccine program for young children was introduced (Figure 1).

Figure 1: Notification and notification rate for invasive pneumococcal disease, Australia, 2002 to 2010



A summary of the number and rates of notifications by jurisdiction is shown in Table 4. As in previous years, New South Wales accounted for the largest number of cases notified by a state or territory in 2009 (n=478) and 2010 (n=499) and the Northern Territory reported the highest notification rate in both 2009 (38.5 per 100,000) and 2010 (24.4 per 100,000). The Australian Capital Territory reported the smallest number of cases in both 2009 (n=30) and 2010 (n=25) and Queensland recorded the lowest notification rate in both 2009 (6.0 per 100,000) and 2010 (6.2 per 100,000).

The number of cases of IPD was greatest in the winter months with the peak number of notifications for both 2009 (n=226) and 2010 (n=218) occurring in July. The effect of season was more evident in the distribution of cases aged 5 years or over compared with younger children (Figure 2).

Invasive pneumococcal disease by age and sex

In almost all age groups, there was a greater notification rate of IPD in males than females. Consistent with 2007–2008, the highest rates in 2009 and 2010 combined were again among the elderly aged 85 years or over (34.4 per 100,000) and in children aged 1 year (30.8 per 100,000) (Figure 3).

Table 4: Notified cases and notification rate for invasive pneumococcal disease, Australia, 2009 and 2010 by state or territory, age group and Indigenous status

Age and Indigenous status	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
2009									
Notified cases aged <5 years									
Indigenous	0	3	16	3	0	0	0	4	26
Non-Indigenous	7	70	3	37	21	1	40	26	205
Total*	7	73	19	55	21	1	45	30	251
Notified cases aged 5–64 years									
Indigenous	1	5	57	27	12	3	8	29	142
Non-Indigenous	16	138	4	102	55	25	157	56	553
Total*	17	225	61	145	68	28	196	85	825
Notified cases ≥ 65 years									
Indigenous	0	2	5	3	1	0	0	1	12
Non-Indigenous	6	178	2	39	55	9	102	33	424
Total*	6	180	7	59	56	10	128	34	480
Total									
Indigenous	1	10	78	33	13	3	8	34	180
Non-Indigenous	29	386	9	178	131	35	299	115	1,182
Total*	30	478	87	259	145	39	369	149	1,556
Rate (per 100,000 population)	8.5	6.8	38.5	6.0	9.0	7.7	6.9	6.7	7.2
Indigenous status completeness	100%	83%	100%	81%	99%	97%	83%	100%	87%
2010									
Notified cases aged <5 years									
Indigenous	0	4	10	6	2	0	1	14	37
Non-Indigenous	5	91	2	32	22	4	47	32	235
Total*	5	95	12	42	24	4	58	46	286
Notified cases aged 5–64 years									
Indigenous	2	9	32	29	16	1	7	52	148
Non-Indigenous	14	124	9	115	58	18	183	63	584
Total*	16	224	41	164	74	19	216	115	869
Notified cases ≥ 65 years									
Indigenous	0	4	2	2	0	0	0	3	11
Non-Indigenous	4	176	1	44	41	23	109	34	432
Total*	4	180	3	65	41	23	132	37	485
Total									
Indigenous	2	17	44	37	18	1	8	69	196
Non-Indigenous	23	391	12	191	121	45	339	129	1,251
Total*	25	499	56	271	139	46	406	198	1,640
Rate (per 100,000 population)	6.9	7.0	24.4	6.2	8.5	9.0	7.4	8.6	7.4
Indigenous status completeness	100%	82%	100%	84%	100%	100%	85%	100%	88%

* Total includes cases reported with a not stated or not reported Indigenous status.

Figure 2: Notifications of invasive pneumococcal disease, Australia, 2009 and 2010, by month and year of diagnosis and age group

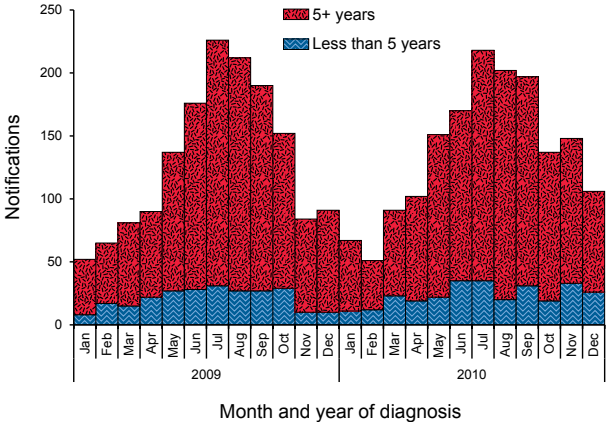
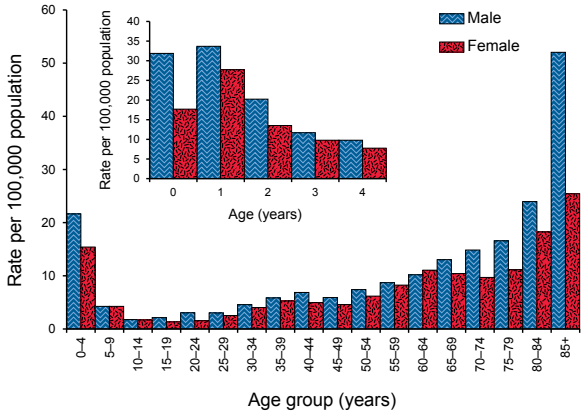


Figure 3: Notification rate for invasive pneumococcal disease, Australia, 2009 and 2010, by age group and sex



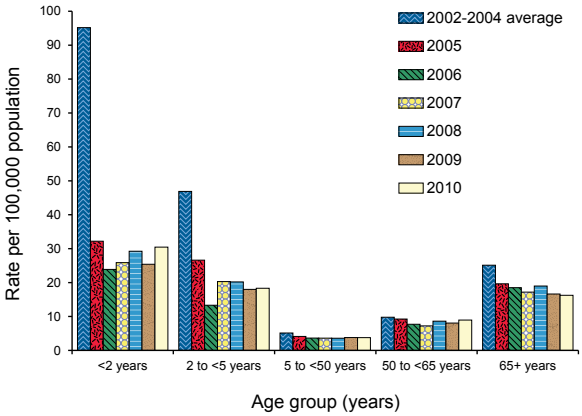
In 2009, the rate of IPD in children aged under 2 years was 25.4 per 100,000 (Figure 4). In 2010, the rate in this age group increased to 30.5 per 100,000; however, overall the rate maintains the large decrease experienced in this age group as a result of the introduction of the universal 7vPCV immunisation program in 2005. Prior to the vaccination program the notification rate in this age group was close to 100 cases per 100,000 (Figure 4).

The overall rate of IPD in adults aged 65 years or over continued to slowly decline in 2009 (16.6 per 100,000) and 2010 (16.2 per 100,000).

Invasive pneumococcal disease in Aboriginal and Torres Strait Islander people

Indigenous status was reported in 87% of notifications in 2009 and in 88% of notifications in 2010 (Table 4). In 2009, there were 180 cases of IPD reported as Indigenous (11.6% of all cases). This represents a rate of 33 cases per 100,000; a rate almost 6 times that seen in the non-Indigenous population (6.0 per 100,000). The rate of IPD among Indigenous people in 2010 was similar to 2009 with 196 cases representing 12.0% of all cases. Further analyses of the Indigenous population group are provided throughout this report.

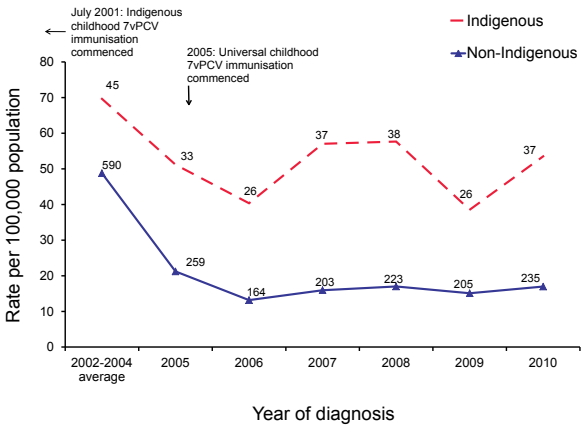
Figure 4: Notification rate for invasive pneumococcal disease, Australia, 2002 to 2010, by age group



Invasive pneumococcal disease in children

The rate of IPD in Indigenous children aged less than 5 years in 2009 was 38.5 cases per 100,000 (n=26) and in 2010 was 53.5 cases per 100,000 (n=37) (Figure 5). The rate of IPD in non-Indigenous children aged less than 5 years in 2009 was 15.1 cases per 100,000 (n=205) and in 2010 was 17.0 cases per 100,000 (n=235).

Figure 5: Notification rate for invasive pneumococcal disease in children aged less than 5 years, Australia, 2002 to 2010, by Indigenous status

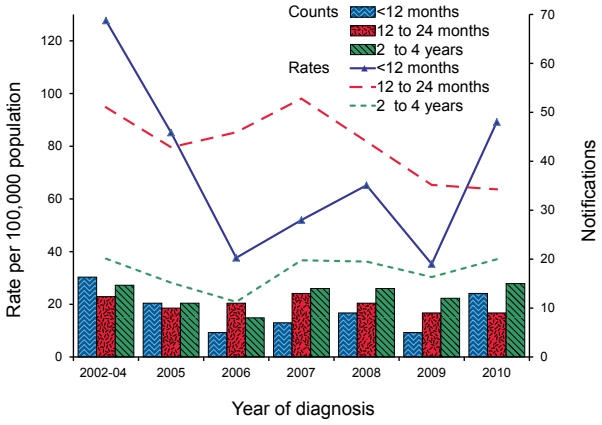


Data point labels represent the number of notifications.

The rate of IPD in Indigenous children aged:

- less than 12 months was 35 cases per 100,000 (n=5) in 2009 and 89 cases per 100,000 in 2010 (n=13) (Figure 6a);
- 12 to 23 months was 65 cases per 100,000 (n=9) in 2009 and 64 cases per 100,000 in 2010 (n=9); and
- 24 months to less than 60 months was 30 cases per 100,000 (n=12) in 2009 and 37 cases per 100,000 in 2010 (n=15).

Figure 6a: Notification rate for invasive pneumococcal disease in Indigenous children aged less than 5 years, Australia, 2002 to 2010, by Indigenous status and age group



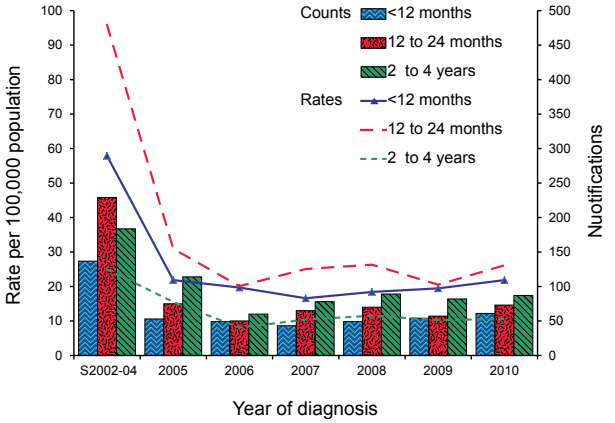
Simple average of data from 2002 to 2004.

The rate of IPD in Indigenous children fluctuated over the period due to the small number of notifications.

Less variability was seen in non-Indigenous children. Despite a slight increase in the rate of IPD in non-Indigenous children between 2009 and 2010, there has been an overall decrease in the rate since the implementation of the universal 7vPCV immunisation program in 2005. The IPD rate in non-Indigenous children aged:

- less than 12 months was 20 cases per 100,000 (n=54) in 2009 and 22 cases per 100,000 in 2010 (n=61) (Figure 6b);
- 12 to 23 months was 21 cases per 100,000 (n=57) in 2009 and 26 cases per 100,000 in 2010 (n=73); and
- 24 to less than 60 months was 10 cases per 100,000 (n=82) in 2009 and 11 cases per 100,000 in 2010 (n=87).

Figure 6b: Notification rate for invasive pneumococcal disease in non-Indigenous children aged less than 5 years, Australia, 2002 to 2010, by Indigenous status and age group



Simple average of data from 2002 to 2004.

Mortality of invasive pneumococcal disease cases

Mortality data were reported for 63% (n=987) of IPD cases notified in 2009 and 62% (n=1,013) in 2010 (Table 5). One hundred and ten and 137 deaths associated with IPD were reported in 2009 and 2010, respectively.

Overall, case fatality rates (CFR) in notifications reported as non-Indigenous were higher than in those reported as Indigenous. In 2009, death associated with IPD was reported in 9 Indigenous cases (CFR=5.0%) and in 96 non-Indigenous cases (CFR=8.1%). In 2010, death associated with IPD was reported in 10 Indigenous cases (CFR=5.1%) and in 122 non-Indigenous cases (CFR=9.8%).

In those aged less than 5 years, there were 3 deaths associated with IPD in 2009 and 10 deaths in 2010 giving case fatality rates of 1.2% and 3.5% respectively. Three of the deaths that occurred in 2010 were in cases reported as Indigenous children. Further details, including serotype and vaccination history, of the 13 children aged less than 5 years whose deaths were associated with IPD are shown in Table 6.

Risk factors for invasive pneumococcal disease

Risk factor data were provided for 69% (2,221/3,196) of cases reported in 2009 and 2010. Of the cases with risk factor data reported, 76% (1,696) of cases reported at least 1 risk factor and 11% (243) of cases reported that no risk factors were identified.

Table 5: Deaths and case fatality rates* for invasive pneumococcal disease, Australia, 2009 and 2010, by age group, Indigenous status and state or territory

Age group	State or territory								
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
2009									
Notified cases aged <5 years									
Deaths	0	0	1	1	0	0	1	0	3
Case fatality rate*	0.0	N/A	5.3	1.8	N/A	0.0	2.2	0.0	1.2
Notified cases aged 5–64 years									
Deaths	0	12	2	8	2	1	11	4	40
Case fatality rate*	0.0	N/A	3.3	5.5	2.9	3.6	5.6	4.7	4.8
Notified cases ≥ 65 years									
Deaths	0	41	1	3	3	3	12	4	67
Case fatality rate*	0.0	N/A	14.3	N/A	5.4	30.0	9.4	11.8	14.0
Total									
Indigenous	0	0	4	2	1	0	0	2	9
Non-Indigenous	0	53	0	10	4	4	19	6	96
Total†	0	53	4	12	5	4	24	8	110
Death reporting completeness (%)	100	11	100	54	87	100	98	100	63
2010									
Notified cases aged <5 years									
Deaths	0	1	0	2	1	0	1	5	10
Case fatality rate*	0.0	N/A	0.0	N/A	4.2	0.0	1.7	10.9	3.5
Notified cases aged 5–64 years									
Deaths	0	14	1	6	3	1	10	6	41
Case fatality rate*	0.0	N/A	2.4	N/A	4.1	5.3	4.6	5.2	4.7
Notified cases ≥ 65 years									
Deaths	2	40	1	1	5	7	22	8	86
Case fatality rate*	50.0	N/A	33.3	N/A	12.2	30.4	16.7	21.6	17.7
Total									
Indigenous	0	1	2	1	0	0	1	5	10
Non-Indigenous	2	54	0	8	9	8	27	14	122
Total†	2	55	2	9	9	8	33	19	137
Death reporting completeness (%)	100	11	100	32	100	100	100	100	62

* Jurisdictional specific case fatality rates have not been presented for those jurisdictions where completeness of data was less than 50%; denoted as 'N/A'. Rates shown should be interpreted with caution given the proportion of cases without mortality data reported to the NNDSS, as well as the variability across jurisdictions in reporting death as primary and secondary causes.

† All notified cases include cases reported with a not stated or not reported Indigenous status.

Table 7 shows data on the risk factors for IPD in specified population sub-groups for 2009 and 2010 combined.

In children aged less than 5 years, the most frequently reported risk factor in the Indigenous population was premature birth (< 37 weeks gestation, 25% of cases with a risk factor reported), while childcare attendance was the most frequently reported risk factor in the non-Indigenous population (35% of cases with risk factor data reported). Among the adult population groups, Indigenous cases aged greater than 50 years and non-Indigenous cases aged greater than 65 years, chronic illness was the most frequently reported risk factor, with 46% and 41% of cases with a risk factor reported respectively.

Table 6: Characteristics of deaths from invasive pneumococcal disease in children aged less than 5 years of age, Australia, 2009 and 2010

Case	Year of diagnosis	Sex	Age (months)	Indigenous status	Serotype	Doses of 7vPCV	Risk factors
Deaths potentially preventable by 7vPCV							
1	2009	Male	16	Non-Indigenous	19F	3	Other*
Deaths not preventable by 7vPCV							
2	2009	Male	10	Indigenous	16F	2	Chronic illness and other
3	2009	Female	16	Non-Indigenous	8	0	Unknown
4	2010	Male	5	Indigenous	1	1	Anatomic or functional asplenia
5	2010	Female	55	Non-Indigenous	11A	0	Unknown
6	2010	Female	10	Non-Indigenous	18A	3	Unknown
7	2010	Female	6	Non-Indigenous	19A	3	No risk factor identified
8	2010	Male	12	Non-Indigenous	19A	3	Information not supplied
9	2010	Male	1	Non-Indigenous	23B	0	No risk factor identified
10	2010	Female	1	Non-Indigenous	35B	0	No risk factor identified
11	2010	Male	2	Non-Indigenous	7F	0	Premature (<37 weeks gestation) and Congenital or chromosomal abnormality
12	2010	Male	23	Non-Indigenous	Non-typable	3	Unknown
13	2010	Female	3	Indigenous	Untyped	1	No risk factor identified

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

Mortality caveat:

NNDSS is generally a passive surveillance system that contains a minimum dataset including whether or not the case 'died of the notifiable condition'. The specific manner in which these data items are collected has not been standardised and therefore varies from jurisdiction to jurisdiction. There is consensus that data reported here includes deaths within the first one to two weeks of diagnosis.

Table 7: Numbers of risk factors* for invasive pneumococcal disease population sub-groups, Australia, 2009 and 2010

Risk factor	Children aged less than 5 years		Indigenous aged 50 years or over	Non-Indigenous aged 65 years or over
	Indigenous	Non-Indigenous		
Premature (<37 weeks gestation)	14	29	N/A	N/A
Congenital or chromosomal abnormality	5	14	1	0
Anatomic or functional asplenia	1	1	2	12
Immunocompromised	3	18	14	203
Chronic illness	7	14	56	386
Childcare attendee	4	61	N/A	N/A
Previous episode of IPD	2	1	5	7
Other†	21	34	43	344
No risk factor identified	14	88	3	29
Unknown or not reported	14	217	13	186
Total	63	440	81	856

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

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

N/A Not applicable.

Pneumococcal serotypes causing invasive disease

While serotype information does not influence initial clinical care, it is necessary to monitor the effectiveness of vaccination programs and inform future policy. It is also recognised that specific serotypes may be associated with certain disease presentation and severity and may have identifiable antibiotic susceptibility patterns. Pneumococcal serotypes were identified for 95% (1,483/1,556) of all notified cases in 2009 and for 94% (1,546/1,640) in 2010.

Of all the cases reported with a serotype in 2009 and 2010, 12% (370/3,029) were due to serotypes covered by the 7vPCV. This ranged from 2% (1/61) of Indigenous cases aged less than 5 years to 15% (81/554) of non-Indigenous cases aged 5–49 years. The 3 additional serotypes (1, 5 and 7F) covered by the 10vPCV accounted for an additional 10% (300/3,029) of cases in 2009 and 2010, which ranged from 3% (27/815) of non-Indigenous adults aged 65 years or over to 19% (103/554) of non-Indigenous cases aged 5–49 years. The 3 additional serotypes (3, 6A and 19A) covered by the 13vPCV accounted for an additional 34% (1,027/3,029) of cases, ranging from 9% (19/220) of Indigenous cases aged 5–49 years to 57% (234/409) of non-Indigenous cases aged less than 5 years.

There are an additional 16 serotypes covered by the 23vPPV, in addition to the seven covered by the 7vPCV. Of all the cases reported with a serotype in 2009 and 2010, 65% (1,982/3,029) of all cases were due to these additional 16 serotypes. This ranged from 53% (431/815) of non-Indigenous adults aged 65 years or over to 74% (303/409) of non-Indigenous cases aged less than 5 years.

Table 8 and the remainder of the analyses included in this section consider the number and proportions of IPD cases due to serotypes covered by the various pneumococcal vaccines and their target age groups. Note that 13vPCV is included in Table 8 pending its introduction to the NIP in 2011.

7-valent pneumococcal conjugate vaccine serotypes

Overall, the notification rate of IPD due to 7vPCV serotypes has continued to decrease across all age groups since 2002 (Figure 7). Since the 2005 introduction of the universal 7vPCV immunisation program, the notification rate of IPD in all age groups decreased by 78% in 2009 (4.5 to 1.0 per 100,000) and 84% in 2010 (4.5 to 0.7 per 100,000).

Figure 8 shows rates of IPD caused by 7vPCV serotypes in Indigenous and non-Indigenous children aged less than 5 years since 2002. The rate of IPD due to 7vPCV serotypes in Indigenous children remained low in 2009 (0 per 100,000) and 2010 (1.4 per 100,000). Similarly, rates also remained low in non-Indigenous children in 2009 (1.1 per 100,000) and 2010 (0.9 per 100,000).

Figure 7: Notification rate for invasive pneumococcal disease caused by 7vPCV serotypes, Australia, 2002 to 2010, by age group

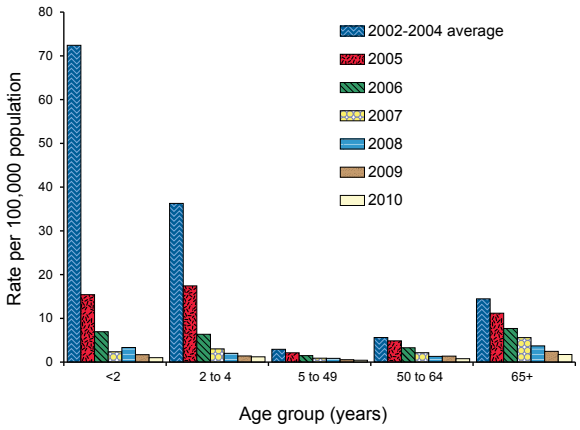
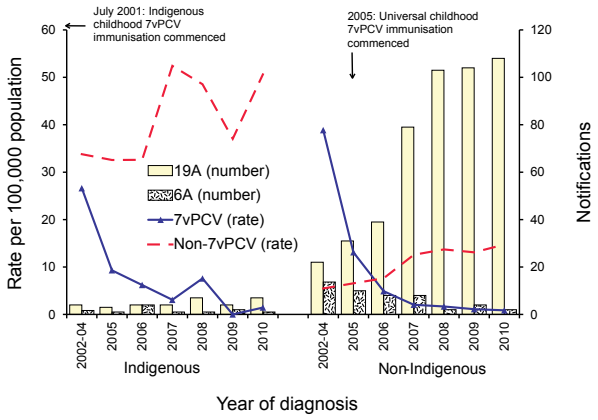


Figure 8: Notification rate for 7vPCV and non-7vPCV serotypes causing cases of invasive pneumococcal disease in children aged less than 5 years, 2002 to 2010, by Indigenous status



Simple average of data from 2002 to 2004.

	Indigenous		Non-Indigenous	
	7vPCV (n)	Non-7vPCV (n)	7vPCV (n)	Non-7vPCV (n)
2002-04*	17	22	470	66
2005	6	21	160	80
2006	4	21	61	95
2007	2	34	26	160
2008	5	32	22	180
2009	0	25	15	178
2010	1	35	12	204

Table 8: Notified cases of invasive pneumococcal disease, Australia, 2009 and 2010, by pneumococcal vaccine serotypes

Age group	Vaccine type	Indigenous			Non-Indigenous		
		Number	%	Cumulative (%)	Number	%	Cumulative (%)
<5 years	7vPCV	1	2	2	27	7	7
	10vPCV (non-7vPCV)	11	18	20	25	6	13
	13vPCV (non-10vPCV)	15	25	44	234	57	70
	Non-conjugate vaccine serotypes	34	56	100	123	30	100
	Total	61	100		409	100	
	23vPPV (non-7vPCV)	45	74		303	74	
5–49 years	7vPCV	17	8	8	81	15	15
	10vPCV (non-7vPCV)	34	15	23	103	19	33
	13vPCV (non-10vPCV)	19	9	32	161	29	62
	Non-conjugate vaccine serotypes	150	68	100	209	38	100
	Total	220	100		554	100	
	23vPPV (non-7vPCV)	140	64		366	66	
50–64 years	7vPCV	3	5	5	76	14	14
	10vPCV (non-7vPCV)	8	14	19	47	9	23
	13vPCV (non-10vPCV)	11	19	39	181	34	57
	Non-conjugate vaccine serotypes	35	61	100	227	43	100
	Total	57	100		531	100	
	23vPPV (non-7vPCV)	37	65		342	64	
65+ years	7vPCV	1	5	5	112	14	14
	10vPCV (non-7vPCV)	2	10	14	27	3	17
	13vPCV (non-10vPCV)	4	19	33	268	33	50
	Non-conjugate vaccine serotypes	14	67	100	408	50	100
	Total	21	100		815	100	
	23vPPV (non-7vPCV)	12	57		431	53	
Total	7vPCV	22	6	6	296	13	13
	10vPCV (non-7vPCV)	55	15	21	202	9	22
	13vPCV (non-10vPCV)	49	14	35	844	37	58
	Non-conjugate vaccine serotypes	233	65	100	967	42	100
	Total	359	100		2,309	100	
	23vPPV (non-7vPCV)	234	65		571	25	

Notifications with Indigenous status and/or serotype reported as unknown are excluded.

Since 2002, the rate of IPD disease caused by non-7vPCV serotypes has increased overall for both Indigenous and non-Indigenous children aged less than 5 years. The rate of IPD due to non-7vPCV serotypes in Indigenous children was 37 per 100,000 in 2009 and 51 per 100,000 in 2010, and in non-Indigenous children was 13 per 100,000 in 2009 and 15 per 100,000 in 2010.

Increasing rates of invasive pneumococcal disease caused by serotypes not contained in the 7vPCV has been observed since 2005 with serotypes 19A and 6A the more commonly occurring replacement serotypes. The number of cases due to serotype 19A has continued to show an overall increase since 2002 in both Indigenous and non-Indigenous children aged less than 5 years. The number of cases due to the 19A serotype in Indigenous children was four in 2009 and seven in 2010. The number of cases due to the 19A serotype in non-Indigenous children was 104 in 2009 and 108 in 2010. The number of cases due to serotype 6A has

remained steady in Indigenous children aged less than 5 years with two cases notified in 2009 and a single case notified in 2010. The number of cases due to the 6A serotype in non-Indigenous children was four in 2009 and two in 2010.

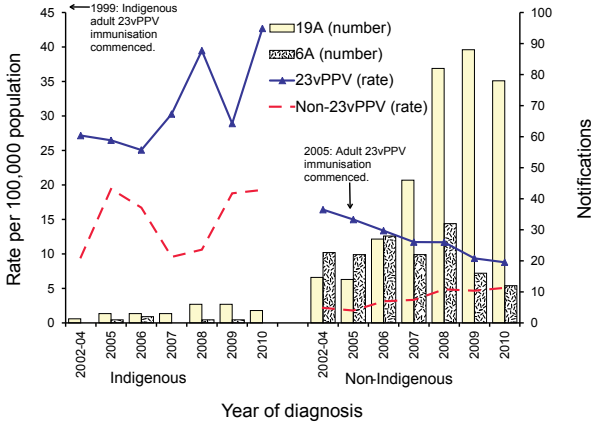
23-valent pneumococcal polysaccharide vaccine serotypes

Figure 9 shows rates of IPD caused by 23vPPV serotypes in the groups targeted to receive the vaccine including Indigenous adults aged 50 years or over and non-Indigenous adults aged 65 years or over. In Indigenous adults, the rate of disease caused by 23vPPV serotypes continued to show an overall increase with a rate of 29 per 100,000 in 2009 and 43 per 100,000 in 2010. Conversely, in non-Indigenous adults the rate continued to show a decreasing trend with a rate of 9.4 per 100,000 in 2009 and 8.8 per 100,000 in 2010.

The rate of disease caused by non-23vPPV serotypes continued to show an overall increase in both Indigenous and non-Indigenous adults. The rate of IPD disease caused by non-23vPPV was 19 per 100,000 in both 2009 and 2010 for Indigenous adults and 4.7 per 100,000 in 2009 and 5.1 per 100,000 in 2010 for non-Indigenous adults.

The number of cases due to serotype 19A in Indigenous adults showed a slight overall increase since 2002 with 6 cases in 2009 and 4 cases in 2010. The number of cases due to serotype 19A in non-Indigenous adults showed a more marked increase since 2002 with 88 cases in 2009 and 78 cases in 2010. The number of cases due to serotype 6A has remained steady in Indigenous adults with 1 case in 2009 and no cases in 2010. The number of cases due to serotype 6A showed an overall increase from 2002 to 2008; however, have since decreased with 16 cases in 2009 and 12 cases in 2010.

Figure 9: Notification rate for 23vPPV and non-23vPPV serotypes causing cases of invasive pneumococcal disease in Indigenous adults (aged 50 years or over) and non-Indigenous adults (aged 65 years or over), 2002 to 2010



Simple average of data from 2002 to 2004.

	Indigenous		Non-Indigenous	
	23vPPV (n)	Non-23vPPV (n)	23vPPV (n)	Non-23vPPV (n)
2002-04*	14	5	413	55
2005	15	11	393	47
2006	15	10	358	84
2007	19	6	322	92
2008	26	7	329	137
2009	20	13	269	134
2010	31	14	261	151

Vaccine failures

In 2009 and 2010, a total of 17 children who were considered fully vaccinated, were notified with disease due to 7vPCV serotypes (Table 9). Sixteen of the 17 cases were reported as non-Indigenous and 1 case did not have Indigenous status identified. Serotype 19F was reported in 76% (n=13) of these cases.

Antibiotic resistance

Antimicrobial resistance in invasive pneumococci is an emerging problem in Australia.[16] Antibiotic susceptibility testing was performed across all jurisdictions by a range of different methods. Penicillin and ceftriaxone/cefotaxime susceptibility data were analysed only for jurisdictions that reported susceptibility data for more than 50% of cases. Penicillin susceptibility completeness was suitable for reporting for all jurisdictions in 2009 and all jurisdictions, excluding Victoria in 2010. Similarly, ceftriaxone/cefotaxime susceptibility completeness was suitable for reporting for all jurisdictions, in 2009 and for all jurisdictions, excluding Victoria in 2010.

Throughout the report period, the proportion of isolates with reduced susceptibility to penicillin increased from 10% (2009: 133/1,306) to 16% (2010: 180/1,139) of total isolates tested, mainly due to a 50% increase in the number of isolates with intermediate susceptibility (Table 10). This compares with a steady rate of 11% for 2007 and 2008. Of the isolates in 2009 with reduced susceptibility to penicillin, 131 were serotyped, with 22% (29/131) of these cases due to a serotype in the 7vPCV and 85% (113/131) due to a serotype in the 23vPPV; in 2010, 176 were serotyped, with 18% (32/176) of these cases due to a serotype in the 7vPCV and 72% (130/180) due to a serotype in the 23vPPV.

Table 9: Characteristics of 7vPCV vaccine failures in children aged less than 5 years, Australia, 2009 and 2010

Case	Year of diagnosis	Age (months)	Indigenous status	Serotype	Doses of 7vPCV	Clinical category	Risk factors
1	2009	28	Non-Indigenous	19F	3	Bacteraemia	Unknown
2	2009	31	Non-Indigenous	19F	3	Pneumonia	Yes
3	2009	37	Non-Indigenous	19F	3	Bacteraemia	Unknown
4	2009	58	Not reported	14	3	Pneumonia	No
5	2009	16	Non-Indigenous	19F	3	Bacteraemia	Yes
6	2009	30	Non-Indigenous	19F	3	Bacteraemia	Unknown
7	2009	22	Non-Indigenous	19F	3	Bacteraemia	Unknown
8	2009	43	Non-Indigenous	23F	3	Bacteraemia	Yes
9	2009	51	Non-Indigenous	18C	3	Pneumonia	Unknown
10	2010	13	Non-Indigenous	23F	3	Bacteraemia	Unknown
11	2010	51	Non-Indigenous	19F	3	Meningitis	Yes
12	2010	56	Non-Indigenous	19F	3	Pneumonia	Unknown
13	2010	25	Non-Indigenous	19F	3	Pneumonia	Yes
14	2010	34	Non-Indigenous	19F	3	Pneumonia	Unknown
15	2010	45	Non-Indigenous	19F	3	Bacteraemia	Unknown
16	2010	47	Non-Indigenous	19F	3	Pneumonia	Unknown
17	2010	10	Non-Indigenous	19F	3	Pneumonia	No

Of the isolates with reduced susceptibility to penicillin in 2009, 77 were serotype 19A, eight were 9V, and 11 were 19F, accounting for 72% (96/133) of isolates with reduced penicillin susceptibility and with a known serotype. Of the isolates with reduced susceptibility to penicillin in 2010, 110 were serotype 19A, three were 9V, and 13 were 19F, which together account for 70% (126/180) of isolates with reduced penicillin susceptibility and with a known serotype.

With respect to ceftriaxone/cefotaxime, the proportion of isolates with reduced susceptibility has remained consistent at 2% (2009: 21/1,090) to 3% (2010: 27/986). These results were similar to 2007 and 2008, which were 3% (25/834) and 2% (16/910) respectively. All of the isolates in 2009 with reduced susceptibility to ceftriaxone/cefotaxime were serotyped, with 29% (6/21) due to a serotype in the 7vPCV and 62% (13/21) to a serotype in the 23vPPV. Of the isolates in 2010 with reduced susceptibility to ceftriaxone/cefotaxime, 26 were serotyped, with 38% (10/26) due to a serotype in the 7vPCV and 69% (18/26) due to a serotype in the 23vPPV.

Of the isolates with reduced susceptibility to ceftriaxone/cefotaxime in 2009, 12 were serotype 19A, one was 9V, and five were 19F, accounting for 86% (18/21) of isolates with reduced ceftriaxone/cefotaxime susceptibility and with a known serotype. Of the isolates with reduced susceptibility to ceftriaxone/cefotaxime in 2010, 15 were serotype 19A, one was 9V, and six were 19F, accounting for 85% (22/26) of isolates with reduced ceftriaxone/cefotaxime susceptibility and with a known serotype.

Discussion

In 2009, 1,556 cases of IPD were notified in Australia compared with 1,640 in 2010. The overall rate of IPD in Australia has been stable since 2006, the first year following introduction of the universal 7vPCV program in children. During the study period, the highest notification rates were among adults aged 80 years or over (27 per 100,000) and among children under 5 years of age (19 per 100,000). However, small increases in notification rates across most age groups were observed in 2010.

The number of IPD notifications due to serotypes included in the 7vPCV has continued to decline since the introduction of the universal infant (3+0 schedule) vaccination program in 2005. Since 2005, the most marked reduction has been in children aged less than 5 years, where disease burden due to 7vPCV serotypes has declined by 97%. In contrast, notifications of disease due to non-7vPCV serotypes increased by 71% in this age group over the same period suggesting serotype replacement. Substantial impacts on IPD due to serotypes covered by vaccines in vaccinated age groups, herd immunity impacts on adults, as well as variable levels of serotype replacement have

Table 10: *Streptococcus pneumoniae* susceptibility to penicillin and ceftriaxone/cefotaxime,* for selected states and territories, 2009 and 2010

	9V	19F	All 7vPCV serotypes	19A	All 23vPPV	Not specified	All isolates
2009							
Penicillin							
Resistant	4	5	14	14	29	1	36
Intermediate	4	6	15	63	84	1	97
Sensitive	9	33	151	230	861	40	1,173
Total tested	17	44	180	307	974	42	1,306
Total isolates with reduced susceptibility (%)	8 (47%)	11 (25%)	29 (16%)	77 (25%)	113 (12%)	2 (5%)	133 (10%)
Cetriaxone							
Resistant	0	2	2	3	3	0	5
Intermediate	1	3	4	9	10	0	16
Sensitive	11	34	142	245	752	25	1,069
Total tested	12	39	148	257	765	25	1,090
Total isolates with reduced susceptibility (%)	1 (8%)	5 (13%)	6 (4%)	12 (5%)	13 (2%)	0 (0%)	21 (2%)
2010							
Penicillin							
Resistant	3	6	14	13	18	1	29
Intermediate	5	7	18	97	112	3	151
Sensitive	4	12	69	164	653	38	959
Total tested	12	25	101	274	783	42	1,139
Total isolates with reduced susceptibility (%)	8 (67%)	13 (52%)	32 (32%)	110 (40%)	130 (17%)	4 (10%)	180 (16%)
Cetriaxone							
Resistant	0	1	2	3	4	0	5
Intermediate	1	5	8	12	14	1	22
Sensitive	9	17	78	216	667	25	959
Total tested	10	23	88	231	685	26	986
Total isolates with reduced susceptibility (%)	1 (10%)	6 (26%)	10 (11%)	15 (6%)	18 (3%)	1 (4%)	27 (3%)

* Susceptibility data are restricted to jurisdictions with completeness suitable for reporting (greater than 50% completeness). Penicillin and ceftriaxone/cefotaxime susceptibility completeness was suitable for reporting in both 2009 for all jurisdictions. However, penicillin and ceftriaxone/cefotaxime susceptibility completeness was not suitable for reporting in 2010 in Victoria.

also been seen in other countries with established 7vPCV programs including England, Wales and the United States of America.[17, 18]

During 2009–2010 the most prevalent serotypes reported to cause IPD were 19A, 22F, 3, 7F and 6C, which together accounted for almost half of all notifications. Across all age categories, increases in the number of IPD infections due to serotypes 19A and 22F in particular were noted.

For the 2009–2010 study period, the proportion of cases due to infection with serotype 6C (6.7% of cases for which information on serotype was available) was a marked increase compared with 2008 (2.5%) and 2007 (1.9%). The majority of cases were in adults aged 65 years or over. The increased prevalence in nasopharyngeal carriage of serotype 6C after vaccination has been previously described in a study of seven cohorts of Massachusetts children between 1994 and 2007.[19] Currently, serotype 6C is not covered by any of the licensed vaccines. However, there is evidence that 13vPCV has the potential to confer cross-protection against serotypes not directly covered by the vaccine, namely serotypes 6C and 7A.[20, 21] These findings support the idea of introducing 13vPCV into national vaccination schemes.

Among the most prevalent serotypes, serotypes 19A (n=55) and 3 (n=22) accounted for the highest number of deaths. Serotype 19A has emerged worldwide to be an important serotype associated with IPD, pneumonia, acute otitis media, and haemolytic uremic syndrome.[22,23] It has been proposed that the high incidence of multidrug-resistant 19A provides it with an advantage over other serotypes through selective pressure.[24] Serotype 3 has been associated with more severe disease and increased mortality in other countries, but the mechanisms underlying this are currently unknown.[25] Serotype 6B isolates frequently exhibit resistance to antibiotics and accounted for the second highest serotype-specific fatality rate (23%) during the study period.[26,27] However, the relationship between antibiotic resistance and mortality has not been formally evaluated. In Australia, analysis of serotype-specific fatality rates should be interpreted with caution due to the small number of cases for which serotype and death are known (n=214).

One of the major objectives of IPD surveillance is in assessment of serotype replacement, which is defined as the reduction of serotypes included in the vaccines and the rise of non-vaccine serotypes. This phenomenon has been widely described [17,28–30] and in Australia, was particularly evident in children aged less than 5 years and in non-Indigenous adults.

Penicillin and ceftriaxone/cefotaxime are the first and second line antibiotics recommended for use in Australia because of high levels of reported resistance to macrolides, especially erythromycin (17.6%).[31] The proportion of IPD cases with reduced susceptibility to penicillin did not change between 2006 and 2009 (10–11% in each year), but in 2010 the overall rate of resistance increased to 16% with resistance most common in serotypes 19A and 19F.

Post-immunisation surveillance in Australia is essential to monitor trends in IPD, to inform future control strategies, including the targeting of existing and new vaccines and the best options for antibiotic treatment. The Australian Government through the National IPD Laboratory Surveillance Project, provides funding to support the serotyping of all *S. pneumoniae* isolates causing invasive disease.

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Quarterly report

OzFoodNet quarterly report, 1 July to 30 September 2013

The OzFoodNet Working Group

Introduction

OzFoodNet is Australia's enhanced foodborne disease surveillance network, funded since 2000 by the Australian Government Department of Health to collaborate nationally to investigate foodborne disease. In each Australian state and territory OzFoodNet epidemiologists investigate outbreaks of enteric infection. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease. This quarterly report documents investigations of outbreaks of gastrointestinal illness and clusters of disease potentially related to food, which commenced in Australia between 1 July and 30 September 2013.

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

During the 3rd quarter of 2013, OzFoodNet sites reported 613 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports may be delayed, meaning that these figures under-represent the true burden of enteric disease outbreaks. In total, these outbreaks affected 10,458 people, of whom 315 were hospitalised. There were 51 deaths reported during these outbreaks. The majority of outbreaks (497) were due to person-to-person transmission (Table 1), with 51% (251/497) of these occurring in residential aged care facilities and 26% (129/497) occurring in child care centres.

Foodborne and suspected foodborne disease outbreaks

There were 27 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission (Appendix). These outbreaks affected 315 people and resulted in 25 hospitalisations. There were 2 deaths reported during these outbreaks. This compares with 31 outbreaks in the 3rd quarter of 2012[1] and a 5-year mean of 29 outbreaks for the 3rd quarter between 2008 and 2012. A limitation of the outbreak data provided by OzFoodNet sites for this report was the potential for variation in the categorisation of the features of outbreaks depending on circumstances and investigator interpretation. Changes in the number of foodborne outbreaks should be interpreted with caution due to the small number each quarter.

Salmonella Typhimurium was identified as, or suspected to be, the aetiological agent in 11 (41%) foodborne or suspected foodborne outbreaks during this quarter. This was two more than the number reported in the same quarter in 2012. The aetiological agents for the remaining outbreaks included: *Clostridium perfringens* in 3 outbreaks (11%); norovirus in 2 outbreaks (7%); and *S.* Virchow phage type (PT) 23 and *Campylobacter*

Table 1: Outbreaks and clusters of gastrointestinal illness reported by OzFoodNet, 1 July to 30 September 2013, by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total
Foodborne and suspected foodborne	27	4.4
Waterborne and suspected waterborne	6	1.0
Person-to-person	497	81.1
Animal-to-person	1	0.2
Unknown (*Salmonella* cluster)	13	2.1
Unknown (other pathogen cluster)	1	0.2
Unknown	68	11.1
Total	613	100*

* Percentages do not add to 100 due to rounding.

jejuni in 1 outbreak each (4%). For 9 outbreaks (33%), the aetiological agent was unknown. The 3 *Cl. perfringens* outbreaks affected 58 people including 2 hospitalisations. In comparison, there was only 1 *Cl. perfringens* outbreak recorded in the 3rd quarter of 2012 affecting 7 people, with no hospitalisations reported.[1]

Eighteen outbreaks (67% of all the foodborne or suspected foodborne outbreaks) reported in this quarter were associated with food prepared in restaurants (Table 2), compared with 15/31 (48%) foodborne or suspected foodborne outbreaks in the 3rd quarter of 2012.[1]

Table 2: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet, 1 July to 30 September 2013, by food preparation setting

Food preparation setting	Outbreaks
Restaurant	18
Private residence	3
Hospital	2
Commercial caterer	1
Aged care	1
Takeaway	1
School	1
Total	27

To investigate these outbreaks, sites conducted 6 cohort studies, 2 case control studies and collected descriptive case series data for 14 investigations, while for 5 outbreaks no individual patient data were collected. In outbreak investigations where a food vehicle was implicated, the evidence used to implicate the food included analytical evidence in 4 outbreaks and descriptive evidence in 6 outbreaks.

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

Australian Capital Territory

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

New South Wales

There were 10 outbreaks of foodborne or suspected foodborne illness reported in New South Wales during this quarter. The aetiological agents were identified as norovirus for 2 outbreaks and *Ca. jejuni* for 1 outbreak. The aetiological agent was unable to be determined for 7 outbreaks.

Description of key outbreak

An outbreak of campylobacteriosis was reported in September associated with a wedding in the Hunter Valley region of New South Wales that took place in July. Seventeen of 50 attendees were affected. One attendee was hospitalised. One stool specimen had been collected and confirmed positive for *Ca. jejuni*. A retrospective cohort study found that consumption of a duck entrée, including duck liver parfait, was significantly associated with illness (relative risk [RR] 4.3, 95% confidence intervals [CI] 1.2–15.5). Fifteen of the 17 cases (88.2%) ate the duck entrée. The NSW Food Authority reviewed the preparation and handling of foods served at the reception and provided advice on cooking temperatures required to render poultry livers free from bacterial pathogens. A full report on this outbreak is published in a previous issue of *Communicable Diseases Intelligence*.[2]

Northern Territory

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

Queensland

There were 2 outbreaks of foodborne or suspected foodborne illness reported in Queensland during this quarter. The aetiological agents were identified as *S.* Typhimurium PT 16 (multi-locus variable number tandem repeat analysis [MLVA] profile 03-13-10-11-524) and *S.* Typhimurium PT 135 (MLVA 03-13-10-11-524) respectively.

Description of key outbreak

An outbreak of gastrointestinal illness was reported in July affecting 30 people who had eaten at the same Brisbane café. A total of 22 cases were laboratory confirmed with *S.* Typhimurium PT 16 (MLVA 03-13-10-11-524). Three people were hospitalised. A case control study was performed using food histories from multiple groups who had attended the café. Persons who had consumed eggs Benedict (odds ratio [OR] undefined,* 95% CI undefined, $P<0.001$) and any meals that contained eggs (OR 6.0, 95% CI 1.1–34.1, $P=0.05$)

* Undefined: this occurs when there is a zero (0) in the denominator of an odds ratio calculation. This happens when all cases report the exposure of interest or none of the controls report the exposure of interest. In this case all the ill patrons reported consuming eggs Benedict rendering the odds ratio incalculable.

were significantly more likely to have developed illness. However, an environmental investigation at the premises did not detect *Salmonella* species.

South Australia

There were 5 outbreaks of foodborne or suspected foodborne illness reported in South Australia during this quarter. The aetiological agents were identified as *S.* Typhimurium PT 9 (MLVA 03-24-11/12-10-523) in 2 outbreaks and *S.* Virchow PT 23, *S.* Typhimurium PT 135a (MLVA 03-14-10-10-523), and *S.* Typhimurium PT 135 (MLVA 03-12-09-11-523) in 1 outbreak each.

Description of key outbreak

An outbreak of gastroenteritis was detected in September following a medical notification of a confirmed case of salmonellosis and a report of several others ill after attending a private function at a restaurant. Foods at the function were prepared and supplied by the restaurant, with a cake from a separate commercial caterer. A retrospective cohort study was conducted with 52/60 (87%) attendees. Diarrhoea was reported by 15/52 (29%) attendees. Three faecal samples were submitted and all were confirmed as *S.* Typhimurium PT 9. Multivariate analysis found consumption of coleslaw to be significantly associated with illness, (adjusted odds ratio [AOR] 5.3, 95% CI 1.2–23.1, $P=0.03$). The coleslaw was prepared with a raw egg aioli. An environmental investigation identified several issues with the preparation and storage of the raw egg aioli.

Tasmania

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

Victoria

There were 5 outbreaks of foodborne or suspected foodborne illness reported in Victoria during this quarter. The aetiological agents were identified as *S.* Typhimurium PT 126, *S.* Typhimurium PT 135, *S.* Typhimurium PT 135a, and *Cl. perfringens* for 1 outbreak each. The aetiological agent was unable to be determined for 1 outbreak.

Description of key outbreak

An outbreak of gastroenteritis was reported in September among attendees of a wedding reception. Illness was reported by 24/245 attendees. A case control study was conducted with a random sample of 75/245 attendees interviewed, based on a reported attack rate of approximately 20%. Analysis of foods consumed identified two foods as having statistically significant associations with consumption and illness. Consumption of hot savouries (OR 3.3, 95% CI 1.1–10.9, $P=0.023$) and chicken volau-vents (OR 4.3, 95% CI 1.3–15.2, $P=0.006$) were significantly associated with illness. Combining these foods increased the association (OR 6.3, 95% CI, 1.5–35.6, $P=0.004$). The incubation period, symptoms, duration of illness and the identification of secondary cases among family members who did not attend the reception, were consistent with a point-source outbreak of viral gastroenteritis. However, the mode of transmission was not confirmed as no faecal specimens were submitted for testing. The source of contamination of the food was not identified.

Western Australia

There were 5 outbreaks of foodborne or suspected foodborne illness reported in Western Australia during this quarter. The aetiological agents were identified as *Cl. perfringens* for 2 outbreaks and *S.* Typhimurium PT 170/108† (pulsed-field gel electrophoresis [PFGE] type 11) and *S.* Typhimurium PT 135a (PFGE type 39) for 1 outbreak each. The aetiological agent was unable to be determined for 1 outbreak.

Description of key outbreak

An outbreak of gastroenteritis was reported in August among students from a high school. Approximately 34 students reported symptoms of gastroenteritis. Information was obtained for 21 students who reported diarrhoea, including 3 students who reported diarrhoea and vomiting. The median duration of diarrhoea was 12 hours. No information was able to be obtained for students who remained well after the meal. All ill students reported eating a Thai chicken curry prepared at the school. No other specific food item had been consumed by more than 50% of cases. The median incubation period was 12 hours from consumption of the chicken curry. Faecal specimens were submitted by 3 cases with 1 positive for *Cl. perfringens*. An environmental investigation at the school did not identify any major food safety risks.

Multi-jurisdictional investigations

There were no multi-jurisdictional outbreak investigations conducted during this quarter.

† Classification of this organism differs between laboratories, with the Microbiological Diagnostic Unit using PT 170 to classify this type of *Salmonella* Typhimurium and SA Pathology using PT 108 due to a difference in the interpretation of one phenotypic characteristic.

Cluster investigations

During the quarter, OzFoodNet sites conducted investigations into 14 clusters of infection for which no common food vehicle or source of infection could be identified. Aetiological agents identified during the investigations included 7 *S.* Typhimurium clusters, 2 monophasic *S.* Subsp I clusters and 1 cluster each of: *S.* Infantis; *S.* Oslo; *S.* Hadar; *S.* Kiambu; and Shiga toxin-producing *Escherichia coli*.

Comments

The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission, and in this quarter 81% of outbreaks (n=497) were transmitted via this route. The number of foodborne outbreaks this quarter (n=27) was lower than for the 3rd quarter of 2012 (n=31) and the 3rd quarter 5-year mean (2008–2012) of 29 outbreaks.

Salmonella species were identified as the aetiological agent in 12 (44%) of the 27 foodborne or suspected foodborne outbreaks during the quarter (Appendix), with 11/12 outbreaks being due to *S.* Typhimurium and 1 outbreak due to *S.* Virchow. Of the 3 confirmed foodborne outbreaks where there was an analytical association between illness and the implicated food, two-thirds (2/3, 67%) were due to *S.* Typhimurium and associated with the consumption of raw or minimally cooked egg dishes.

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

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1. The OzFoodNet Working Group. OzFoodNet quarterly report, 1 July to 30 September 2012. *Commun Dis Intell* 2013;37(3):260–266.
2. Hope KG, Merritt TD, Durrheim DN. Short incubation periods in *Campylobacter* outbreaks associated with poultry liver dishes. *Commun Dis Intell* 2014;38(1):20–23.

Appendix: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites,* 1 July to 30 September 2013 (n=27)

State or territory	Month†	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
NSW	July	Takeaway	Unknown	6	0	D	Hamburger with salad
NSW	July	Restaurant	Unknown	12	0	D	Unknown
NSW	July	Restaurant	Unknown	8	0	D	Unknown
NSW	August	Restaurant	Unknown	38	0	D	Unknown
NSW	August	Restaurant	Unknown	6	0	D	Unknown
NSW	August	Restaurant	Unknown	3	0	D	Unknown
NSW	August	Restaurant	Norovirus	16	0	D	Unknown
NSW	September	Restaurant	Norovirus	5	0	D	Unknown
NSW	September	Restaurant	*Campylobacter jejuni*	17	1	A	Duck liver parfait
NSW	September	Restaurant	Unknown	2	0	D	Unknown
Qld	July	Restaurant	*S.* Typhimurium PT 16, MLVA 03-13-10-11-524	30	3	A	Eggs Benedict
Qld	August	Restaurant	*S.* Typhimurium PT 135, MLVA 03-13-10-11-524	10	0	D	Unknown
SA	July	Restaurant	*S.* Virchow PT 23	6	1	D	Unknown
SA	August	Restaurant	*S.* Typhimurium PT 135a, MLVA 03-14-10-10-523	9	3	D	Tartare sauce
SA	August	Private residence	*S.* Typhimurium PT 9, MLVA 03-24-11-10-523	4	0	D	Unknown
SA	September	Restaurant	*S.* Typhimurium PT 135, MLVA 03-12-09-11-523	4	2	D	Unknown
SA	September	Restaurant	*S.* Typhimurium PT 9, MLVA 03-24-12-10-523	15	1	A	Coleslaw made with raw egg
Vic.	July	Private Residence	*S.* Typhimurium PT 126	4	2	D	Suspected BBQ chicken
Vic.	July	Restaurant	*S.* Typhimurium PT 135a	6	0	D	Suspected bacon and egg pide
Vic.	August	Hospital	*Clostridium perfringens*	12	2	D	Unknown
Vic.	September	Commercial caterer	Unknown	24	0	A	Hot savouries and/or chicken vol-au-vents
Vic.	September	Hospital	*S.* Typhimurium PT 135	9	0	D	Unknown
WA	July	Private residence	*S.* Typhimurium PT 170/108, PFGE 11	8	6	D	Unknown
WA	July	Restaurant	*S.* Typhimurium PT 135a, PFGE 39	12	4	D	Eggs
WA	July	Restaurant	Unknown	3	0	D	Unknown
WA	August	School	*Cl. perfringens*	34	0	D	Chicken curry
WA	September	Aged care	*Cl. perfringens*	12	0	D	Unknown

* No foodborne or suspected foodborne outbreaks were reported by the Australia Capital Territory, the Northern Territory or Tasmania.

† Month of outbreak is the month of onset of first case or month of notification/investigation of the outbreak. The number of people affected and hospitalised relate to the findings of the outbreak investigation at the time of writing and not necessarily in the month specified or in this quarter.

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

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

MLVA Multi-locus variable number tandem repeat analysis.

PFGE Pulsed-field gel electrophoresis.

PT Phage type.

National Notifiable Diseases Surveillance System, 1 January to 31 March 2015

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 67,831 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 January to 31 March 2015 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

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

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
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Varicella zoster (chickenpox)	All jurisdictions except New South Wales
Varicella zoster (shingles)	All jurisdictions except New South Wales
Varicella zoster (unspecified)	All jurisdictions except New South Wales
Vectorborne diseases	
Barmah Forest virus infection	All jurisdictions
Chikungunya virus infection	All jurisdictions
Dengue virus infection	All jurisdictions
Flavivirus infection (NEC)	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

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

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2015, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2015	Total 4th quarter 2014	Total 1st quarter 2014	Last 5 years mean 1st quarter	Ratio	Year to date 2015	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Hepatitis B (newly acquired)†	0	8	2	11	0	0	10	9	40	34	58	56.0	0.7	40	56.0
Hepatitis B (unspecified)‡	18	584	51	255	90	6	454	134	1,592	1,517	1,613	1,628.2	1.0	1,592	1,628.2
Hepatitis C (newly acquired)†	3	2	0	0	17	4	39	37	102	86	107	112.0	0.9	102	112.0
Hepatitis C (unspecified)‡	51	889	50	660	107	49	546	251	2,603	2,472	2,481	2,511.6	1.0	2,603	2,511.6
Hepatitis D	0	3	0	3	2	0	3	0	11	15	13	11.2	1.0	11	11.2
Gastrointestinal diseases															
Botulism	0	1	0	0	0	0	0	0	1	0	0	0.4	2.5	1	0.4
Campylobacteriosis	122	NN	97	1,987	396	246	1,955	697	5,500	5,558	4,800	4,440.4	1.2	5,500	4,440.4
Cryptosporidiosis	4	398	27	595	160	0	209	111	1,504	455	859	1,039.6	1.4	1,504	1,039.6
Haemolytic uraemic syndrome	0	2	0	0	0	0	2	0	4	2	9	5.6	0.7	4	5.6
Hepatitis A	1	38	2	20	5	0	12	10	88	51	86	68.8	1.3	88	68.8
Hepatitis E	0	2	0	0	1	1	4	2	10	12	10	13.0	0.8	10	13.0
Listeriosis	0	8	1	3	1	0	2	1	16	20	22	25.8	0.6	16	25.8
STEC, VTEC§	0	8	0	10	6	0	3	0	27	19	40	34.0	0.8	27	34.0
Salmonellosis	92	1,589	139	2,519	409	99	1,054	556	6,457	4,113	5,206	4,416.4	1.5	6,457	4,416.4
Shigellosis	0	53	52	51	26	1	108	44	335	278	306	193.0	1.7	335	193.0
Typhoid	0	13	0	11	2	0	13	4	43	28	46	51.6	0.8	43	51.6
Quarantinable diseases															
Cholera	0	0	0	0	0	0	0	0	0	0	1	0.4	0.0	0	0.4
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0

Table 2 continued: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2015, by date of diagnosis*

Disease	State or territory								Total 1st quarter 2015	Total 4th quarter 2014	Total 1st quarter 2014	Last 5 years mean 1st quarter	Ratio	Year to date 2015	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection[‖,¶]	337	6,098	758	5,437	1,484	484	4,622	2,980	22,200	19,623	22,972	21,303.6	1.0	22,200	21,303.6
Donovanosis	0	0	0	0	0	0	0	0	0	0	1	0.2	0.0	0	0.2
Gonococcal infection[¶]	42	1,453	515	823	219	18	1,092	510	4,672	3,638	4,267	3,440.2	1.4	4,672	3,440.2
Syphilis – congenital	0	0	0	0	0	0	0	0	0	1	0	1.2	0.0	0	1.2
Syphilis < 2 years duration[¶]	3	125	38	128	17	7	231	27	576	491	452	383.4	1.5	576	383.4
Syphilis > 2 years or unspecified duration[‡,¶]	4	124	41	70	31	6	187	20	483	452	478	367.8	1.3	483	367.8
Vaccine preventable diseases															
Diphtheria	0	0	0	1	0	0	0	0	1	1	0	0.2	5.0	1	0.2
Haemophilus influenzae type b	0	0	1	1	0	0	0	0	2	5	3	2.8	0.7	2	2.8
Influenza (laboratory confirmed)	84	1,082	38	1,377	756	38	667	515	4,557	5,719	3,834	2,111.6	2.2	4,557	2,111.6
Measles	1	4	0	7	1	0	12	2	27	36	175	57.4	0.5	27	57.4
Mumps	1	13	0	13	18	3	3	8	59	40	63	46.2	1.3	59	46.2
Pertussis	87	1,621	13	316	164	4	1,491	374	4,070	4,115	2,339	5,912.2	0.7	4,070	5,912.2
Pneumococcal disease (invasive)	1	50	12	30	16	4	56	19	188	337	216	219.6	0.9	188	219.6
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	3	0	2	0	0	0	0	5	5	6	11.6	0.4	5	11.6
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Tetanus	0	0	0	0	0	0	0	0	0	2	1	1.4	0.0	0	1.4
Varicella zoster (chickenpox)	15	NN	16	46	121	23	219	89	529	548	497	388.8	1.4	529	388.8
Varicella zoster (shingles)	40	NN	92	15	592	65	418	367	1,589	1,379	1,398	1,099.4	1.4	1,589	1,099.4
Varicella zoster (unspecified)	28	NN	1	1,498	37	28	1,159	360	3,111	3,099	3,033	2,365.2	1.3	3,111	2,365.2
Vectorborne diseases															
Barmah Forest virus infection	2	73	12	168	0	0	6	9	270	97	331	699.8	0.4	270	699.8
Chikungunya virus infection	0	18	3	12	0	0	17	6	56	48	21	18.2	3.1	56	18.2
Dengue virus infection	8	121	22	147	33	3	118	283	735	255	658	475.0	1.5	735	475.0
Flavivirus infection (NEC)	0	1	0	6	0	0	0	0	7	3	14	5.4	1.3	7	5.4
Japanese encephalitis virus infection	0	1	0	0	0	0	1	0	2	0	0	0.2	10.0	2	0.2
Kunjin virus infection**	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Malaria	2	12	2	19	0	1	14	13	63	61	92	104.4	0.6	63	104.4
Murray Valley encephalitis virus infection**	0	0	1	0	0	0	0	0	1	0	0	1.8	0.6	1	1.8
Ross River virus infection	7	894	179	4,062	53	2	137	388	5,722	1,485	1,616	1,987.4	2.9	5,722	1,987.4

Table 2 continued: Notifications of diseases received by state and territory health authorities, 1 January to 31 March 2015, by date of diagnosis

Disease	State or territory								Total 1st quarter 2015	Total 4th quarter 2014	Total 1st quarter 2014	Last 5 years mean 1st quarter	Ratio	Year to date 2015	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	0.2	0.0	0	0.2
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.2	0.0	0	0.2
Brucellosis	0	2	0	0	0	0	0	0	2	3	6	6.8	0.3	2	6.8
Leptospirosis	1	4	0	12	0	2	1	0	20	11	26	46.0	0.4	20	46.0
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	0	0	0	0	0	1	0	1	14	11	12.8	0.1	1	12.8
Q fever	0	54	1	82	5	0	7	1	150	105	125	101.2	1.5	150	101.2
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.3	0.0	0	0.3
Other bacterial infections															
Legionellosis	2	26	2	19	5	1	14	23	92	110	88	86.2	1.1	92	86.2
Leprosy	0	0	0	0	0	0	0	0	0	2	3	1.4	0.0	0	1.4
Meningococcal infection††	1	6	0	6	1	0	10	3	27	45	25	40.0	0.7	27	40.0
Tuberculosis	5	89	6	41	19	4	80	37	281	363	313	317.8	0.9	281	317.8
Total	**962**	**15,472**	**2,174**	**20,463**	**4,794**	**1,099**	**14,977**	**7,890**	**67,831**	**56,753**	**58,721**			**67,831**	

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

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

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

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

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

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

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

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

NN Not notifiable

NEC Not elsewhere classified

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

Table 3: Notification rates of diseases, 1 January to 31 March 2015, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)‡	0.0	0.4	3.3	0.9	0.0	0.0	0.7	1.4	0.7
Hepatitis B (unspecified)§	18.5	31.5	84.0	21.5	21.6	4.7	31.4	20.5	27.3
Hepatitis C (newly acquired)‡	3.1	0.1	0.0	0.0	4.1	3.2	2.7	5.7	1.7
Hepatitis C (unspecified)§	52.5	48.1	82.4	55.7	25.7	38.7	37.7	38.4	44.6
Hepatitis D	0.0	0.2	0.0	0.3	0.5	0.0	0.2	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis	125.7	NN	159.9	167.6	95.0	194.4	135.1	106.8	137.9
Cryptosporidiosis	4.1	21.5	44.5	50.2	38.4	0.0	14.4	17.0	25.8
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Hepatitis A	1.0	2.1	3.3	1.7	1.2	0.0	0.8	1.5	1.5
Hepatitis E	0.0	0.1	0.0	0.0	0.2	0.8	0.3	0.3	0.2
Listeriosis	0.0	0.4	1.6	0.3	0.2	0.0	0.1	0.2	0.3
STEC,VTEC‖	0.0	0.4	0.0	0.8	1.4	0.0	0.2	0.0	0.5
Salmonellosis	94.8	85.8	229.1	212.5	98.1	78.2	72.8	85.2	110.6
Shigellosis	0.0	2.9	85.7	4.3	6.2	0.8	7.5	6.7	5.7
Typhoid fever	0.0	0.7	0.0	0.9	0.5	0.0	0.9	0.6	0.7
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Human pathogenic avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmitted infections									
Chlamydial infection¶,**	347.1	329.3	1,249.2	458.8	356.1	382.5	319.4	456.5	380.2
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	43.3	78.5	848.7	69.4	52.5	14.2	75.5	78.1	80.0
Syphilis – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years duration**	3.1	6.7	62.6	10.8	4.1	5.5	16.0	4.1	9.9
Syphilis > 2 years or unspecified duration§,**	4.1	6.7	67.6	5.9	7.4	4.7	12.9	3.1	8.3
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.0	1.6	0.1	0.0	0.0	0.0	0.0	0.0
Influenza (laboratory confirmed)	80.3	58.4	62.6	116.2	181.4	30.0	46.1	78.9	77.9
Measles	1.0	0.2	0.0	0.6	0.2	0.0	0.8	0.3	0.5
Mumps	1.0	0.7	0.0	1.1	4.3	2.4	0.2	1.2	1.0
Pertussis	89.6	87.5	21.4	26.7	39.1	3.2	103.1	57.3	69.7
Pneumococcal disease (invasive)	1.0	2.7	19.8	2.5	3.8	3.2	3.9	2.9	3.2
Poliomyelitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.2	0.0	0.2	0.0	0.0	0.0	0.0	0.1
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 continued: Notification rates of diseases, 1 January to 31 March 2015, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, *cont'd*									
Varicella zoster (chickenpox)	15.5	NN	26.4	3.9	28.8	18.2	15.1	13.6	13.2
Varicella zoster (shingles)	41.2	NN	151.6	1.3	142.1	51.4	28.9	56.2	39.9
Varicella zoster (unspecified)	28.8	NN	1.6	126.4	9.1	22.1	80.1	55.1	78.0
Vectorborne diseases									
Barmah Forest virus infection	2.1	3.9	19.8	14.2	0.0	0.0	0.4	1.4	4.6
Chikungunya virus infection	0.0	0.9	4.9	1.0	0.0	0.0	1.2	0.9	0.9
Dengue virus infection	8.2	6.5	36.3	12.4	7.9	2.4	8.2	43.3	12.6
Flavivirus infection (NEC)	0.0	0.1	0.0	0.5	0.0	0.0	0.0	0.0	0.1
Japanese encephalitis virus infection	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Kunjin virus infection††	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	2.1	0.6	3.3	1.6	0.0	0.8	1.0	2.0	1.1
Murray Valley encephalitis virus infection††	0.0	0.0	1.6	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	7.2	48.3	295.0	342.7	12.7	1.6	9.5	59.4	98.0
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Leptospirosis	1.0	0.2	0.0	1.0	0.0	1.6	0.1	0.0	0.3
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Q fever	0.0	2.9	1.6	6.9	1.2	0.0	0.5	0.2	2.6
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	2.1	1.4	3.3	1.6	1.2	0.8	1.0	3.5	1.6
Leprosy	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Meningococcal infection‡‡	1.0	0.3	0.0	0.5	0.2	0.0	0.7	0.5	0.5
Tuberculosis	5.2	4.8	9.9	3.5	4.6	3.2	5.5	5.7	4.8

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

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

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

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

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

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

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

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

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

NEC Not elsewhere classified.

NN Not notifiable.

Australian childhood immunisation coverage, 1 July to 30 September cohort, assessed as at December 2014

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

Introduction

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

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

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months, for 3-month birth cohorts of children assessed at the stated ages between 1 July and 30 September 2014 using ACIR data as at 31 December 2014 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, varicella, and meningococcal C conjugate vaccines.

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

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

Results

The percentage of children 'fully immunised' by 12 months of age for Australia decreased from the previous quarter by 0.9 of a percentage point to 90.6% (Table 1). Most jurisdictions experienced small decreases in the percentage of children 'fully immunised' by 12 months of age. For individual vaccines due by 12 months of age a majority of jurisdictions achieved coverage greater than 90%, except for Tasmania where coverage decreased by more than 2 percentage points for all individual vaccines.

The percentage of children 'fully immunised' by 24 months of age for Australia decreased significantly from the previous quarter by 5.5 percentage points to 87.3% (Table 2) There were also significant decreases in fully immunised coverage at 24 months of age in all jurisdictions (range: 2.6–9.4 percentage points). This drop was likely to be due to the inclusion of the varicella vaccine into the algorithm to calculate fully immunised coverage for this age group for the December 2014 quarter. Coverage for

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

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,399	24,806	923	15,934	4,990	1,488	19,180	8,507	77,227
Diphtheria, tetanus, pertussis (%)	93.9	90.9	90.8	92.3	91.2	89.9	91.3	91.9	91.5
Poliomyelitis (%)	93.8	90.8	90.8	92.3	91.1	89.8	91.3	91.9	91.4
Haemophilus influenzae type b (%)	93.6	90.7	90.8	92.1	91.0	89.7	91.2	91.6	91.3
Hepatitis B (%)	93.7	90.6	90.8	92.0	90.9	89.6	90.9	91.4	91.1
Pneumococcal	93.7	90.5	90.6	92.0	90.8	89.4	91.0	91.2	91.1
Fully immunised (%)	93.1	90.1	90.2	91.7	90.4	89.0	90.4	90.7	90.6
Change in fully immunised since last quarter (%)	+0.6	–1.0	+2.5	–0.4	–1.0	–2.7	–1.3	–1.0	–0.9

all individual vaccines that are due by 24 months remained high in all jurisdictions except for MMR, which experienced decreases in all jurisdictions (range: 2.5–7.3). This was likely due to the introduction of the MMRV vaccine onto the National Immunisation Program Schedule in July 2013.

The percentage of children 'fully immunised' by 60 months of age for Australia remained the same as the previous quarter at 92.2% (Table 3). This maintains the improvement in coverage for this age milestone. There were also only marginal changes in fully immunised coverage at 60 months of age in all jurisdictions. Coverage for individual vaccines due by 60 months remained greater than 90% in all jurisdictions.

The Figure shows the trends in vaccination coverage from the first ACIR-derived published coverage estimates in 1997 to the current estimates. There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007). However, in late 2014 coverage at 24 months of age dropped below coverage at 12 and 60 months of age. This is likely due to the change in the immunisation coverage assessment algorithm for the 24 month milestone age, which was amended to include meningococcal dose 1 and varicella dose 1 in the assessment of 'fully immunised'. The 24 month assessment algorithm has also been amended to look for MMR dose 2 instead of MMR dose 1.

Figure: Trends in vaccination coverage, Australia, 1997 to September 2014, by age cohorts

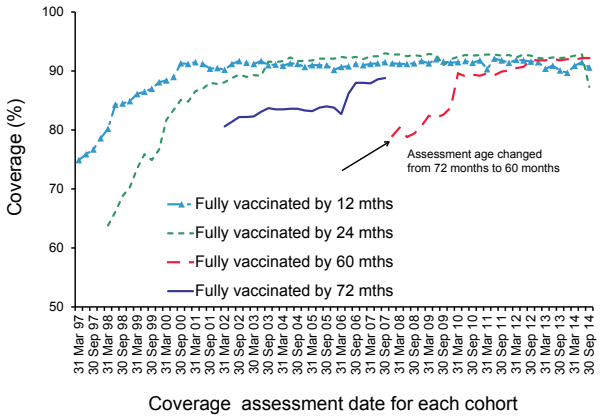

Table 2. Percentage of children immunised at 24 months of age for the birth cohort 1 July to 30 September 2012, preliminary results, by disease and state or territory; assessment date 31 December 2014*

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,433	25,729	891	16,018	5,177	1,484	19,330	8,519	78,581
Diphtheria, tetanus, pertussis (%)	95.8	95.0	94.8	94.8	94.4	94.9	95.5	94.4	95.0
Poliomyelitis (%)	95.9	94.9	94.8	94.8	94.3	94.7	95.4	94.4	94.9
Haemophilus influenzae type b (%)	94.6	93.0	95.2	93.9	92.3	92.1	93.9	92.9	93.4
Measles, mumps, rubella (%)	93.2	89.2	91.4	90.7	88.5	87.2	89.7	86.9	89.4
Hepatitis B (%)	95.7	94.6	95.1	94.4	94.1	94.5	95.1	94.0	94.6
Meningococcal C (%)	94.5	93.0	94.3	93.9	92.4	92.5	93.6	92.4	93.3
Varicella (%)	94.8	90.9	92.0	91.6	89.7	87.5	91.8	88.8	91.0
Fully immunised (%)	91.1	86.7	87.2	89.2	86.0	83.6	87.8	85.1	87.3
Change in fully immunised since last quarter (%)	–2.6	–5.7	–7.5	–4.1	–6.3	–9.4	–5.5	–6.9	–5.5

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2014;38(2):E158.

Table 3. Percentage of children immunised at 60 months of age for the birth cohort 1 July to 30 September 2009, preliminary results, by disease and state or territory; assessment date 31 December 2014

Vaccine	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,439	25,810	844	16,353	5,147	1,612	19,187	8,500	78,892
Diphtheria, tetanus, pertussis (%)	93.6	93.2	92.4	92.4	91.4	92.2	93.1	91.3	92.7
Poliomyelitis (%)	93.7	93.1	92.5	92.5	91.4	92.1	93.1	91.2	92.6
Measles, mumps, rubella (%)	93.4	93.1	93.2	92.5	91.3	92.4	93.1	91.2	92.7
Fully immunised (%)	93.3	92.6	91.9	92.1	90.8	91.5	92.6	90.6	92.2
Change in fully immunised since last quarter (%)	+0.3	0.0	+0.3	–0.4	–0.8	–1.4	0.0	+0.5	0.0

Australian Gonococcal Surveillance Programme, 1 July to 30 September 2014

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

Introduction

The Australian National Neisseria Network (NNN) comprises reference laboratories in each state and territory that report data on sensitivity to an agreed group of antimicrobial agents for the Australian Gonococcal Surveillance Programme (AGSP). The antibiotics are penicillin, ceftriaxone, azithromycin and ciprofloxacin, which are current or potential agents used for the treatment of gonorrhoea. Azithromycin testing has been recently introduced by all states and territories as it is part of a dual therapy regimen with ceftriaxone recommended for the treatment of gonorrhoea in the majority of Australia. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. In certain remote regions of the Northern Territory and Western Australia gonococcal antimicrobial resistance rates are low and an oral treatment regimen comprising amoxycillin, probenecid and azithromycin is recommended for the treatment of gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5% or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatments.[1] Additional data on other antibiotics are reported in the AGSP annual report. The AGSP has a program-specific quality assurance process. The AGSP data are presented quarterly in tabulated form, as well as in the AGSP annual report. For more information see *Commun Dis Intell* 2015;39(1):E178–E179.

Results

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

Penicillin

Penicillin resistant *Neisseria gonorrhoeae* (NG) are defined as those isolates with a minimum inhibitory concentration (MIC) to penicillin equal to or greater than 1.0 mg/L. Penicillin resistance includes penicillinase producing NG (PPNG), and NG that have chromosomally mediated resistance to penicillin (CMRP). In certain areas of the Northern Territory and Western Australia, which are classified as remote, a treatment regimen based on oral amoxycillin, probenecid and azithromycin is used. Due to the distance specimens must travel to a laboratory from these remote regions, low numbers of cultures are collected, and thus, by necessity, nucleic acid amplification testing (NAAT) is used. In remote Western Australia, the introduction of a targeted NAAT, developed by the NNN to detect PPNG, is in use to enhance surveillance.[2,3]

Table 1: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to ciprofloxacin, azithromycin and penicillin, Australia, 1 July to 30 September 2014, by state or territory

State or territory	Number of isolates tested	Decreased susceptibility		Resistance					
		Ceftriaxone		Ciprofloxacin		Azithromycin		Penicillin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	4	0	0.0	1	25.0	0	0.0	1	25.0
New South Wales	429	23	5.4	181	42.0	13	3.0	187	44.0
Queensland	143	5	3.5	39	27.0	9	6.0	34	24.0
South Australia	41	0	0.0	14	34.0	0	0.0	5	12.0
Tasmania	5	2	40.0	2	50.0	0	0.0	1	25.0
Victoria	356	19	5.3	153	43.0	8	2.2	67	20.0
Northern Territory/Urban and Rural	23	2	8.7	7	30.0	0	0.0	6	26.0
Northern Territory/Remote	30	1	3.3	1	3.3	0	0.0	0	0.0
Western Australia/Urban and Rural	104	5	4.8	22	21.0	4	3.8	21	20.0
Western Australia/Remote	27	0	0.0	0	0.0	0	0.0	0	0.0
Australia	1,162	57	4.9	420	36.0	34	2.9	322	28.0

Ciprofloxacin

Ciprofloxacin resistance includes isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L.

Azithromycin

Azithromycin resistance is defined as a MIC to azithromycin equal to or greater than 1.0 mg/L. In 2013, 4 gonococcal strains with azithromycin high level resistance were reported from Victoria and Queensland.[4] There was 1 strain reported from New South Wales with high level resistance (azithromycin MIC value >256 mg/L) in this quarter of 2014.

Ceftriaxone

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

In the 1st quarter of 2014 there was a decrease in the proportion of NG isolates with DS to ceftriaxone, predominantly from New South Wales and Victoria, when compared with the same quarter in 2013; and the annual data for 2013.[4] The highest proportions of isolates with decreased susceptibility to ceftriaxone were reported from New South Wales and Victoria.

From New South Wales, there were 23 of 429 strains with DS to ceftriaxone. Off those, 16 (70%) were multi-drug resistant (MDR); 20 (87%) were from males; and 6 (26%) were isolated from extragenital sites (rectal and pharyngeal). From Victoria, there were 19 356 strains with DS to ceftriaxone and, of those, 10 (53%) were MDR; 8 (50%) were from males; and 7 (36%) were isolated from extragenital sites (rectal and pharyngeal).

From urban and rural Western Australia, there were 5 of 104 strains with DS to ceftriaxone. Of those, 3 (60%) were MDR; 5 (100%) were from males; and 3 (60%) were isolated from extragenital sites (rectal and pharyngeal). From Queensland, there were 5 of 143 strains with DS to ceftriaxone and, of those, all (100%) were MDR; 1 (20%) was from a male; and none were isolated from extragenital sites (rectal and pharyngeal) In contrast, there were no gonococci with DS to ceftriaxone reported from Australia Capital Territory, South Australia, or remote Western Australia; and low numbers were reported from Tasmania, and urban and remote Northern Territory.

The proportion of strains with DS to ceftriaxone is of increasing concern in Australia and overseas, as this is phenotypic of the genotype with the key mutations that are the precursor to ceftriaxone resistance.[5] There are recent reports of ceftriaxone 500 mg treatment failures in patients from Victoria and New South Wales in patients with pharyngeal gonococcal infections. In these patients the infecting gonococcal strains had ceftriaxone MIC values in the range 0.03–0.06 mg/L.[6,7] Until 2014, there had not been an isolate reported in Australia with a ceftriaxone MIC value > 0.125 mg/L.[4] In late December 2013, there was a new multi-drug-resistant gonococcal strain (A8806) with a ceftriaxone MIC of 0.5 mg/L, the highest ever reported in Australia, which was isolated from a female traveller from Central Europe. This infection was acquired in Sydney from another traveller, also from Europe. The patient was tested in the Northern Territory, but had travelled to north-eastern Queensland before the results were available, and was treated there. To date there has been no evidence of spread of this strain.[8]

The category of ceftriaxone DS as reported by the AGSP includes the MIC values 0.06 and 0.125 mg/L. The right shift in the distribution of ceftriaxone MIC values over recent years (Table 2), 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 –1.62], and 2012–2013 [P<0.0001, 95% CI: 1.70–2.38]). In 2010, the proportion of strains with ceftriaxone DS was higher than that reported in 2011. This proportion has subsequently increased as shown in Table 2. The proportion of strains with a ceftriaxone MIC 0.125 mg/L has also increased from 0.1% in 2010 and 2011, to 0.3% in 2012 and to 0.6% in 2013. These differences were not significant, however this may be attributable to the low number of strains in this MIC category.[4] In the first 2 quarters of 2014 there were lower proportions of strains with MIC values at 0.06 and 0.125 mg/L than reported in 2013. In the 3rd quarter of 2014, the proportion of strains with ceftriaxone MIC of

Table 2: Percentage of gonococcal isolates with decreased susceptibility to ceftriaxone MIC 0.06–0.125 mg/L, Australia, 2010 to 2013, and 1 July to 30 September 2014, by state or territory

Ceftriaxone MIC mg/L	2010	2011	2012	2013	2014 Q1	2014 Q2	2014 Q3
0.06	4.6%	3.2%	4.1%	8.2%	6.4%	5.4%	4.2%
0.125	0.1%	0.1%	0.3%	0.6%	0.4%	0.3%	0.7%

0.06 mg/L was lower than in the previous 2 quarters but there was an increase in the proportion of strains with an MIC of 0.125 mg/L, which will continue to be monitored.

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

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1. Surveillance of antibiotic susceptibility of *Neisseria gonorrhoeae* in the WHO Western Pacific Region 1992–4. WHO Western Pacific Region Gonococcal Antimicrobial Surveillance Programme. 1997.
2. 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.
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Australian Gonococcal Surveillance Programme, 1 January to 31 March 2015

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

Introduction

The Australian National Neisseria Network (NNN) comprises reference laboratories in each state and territory that report data on sensitivity to an agreed group of antimicrobial agents for the Australian Gonococcal Surveillance Programme (AGSP). The antibiotics are penicillin, ceftriaxone, azithromycin and ciprofloxacin, which are current or potential agents used for the treatment of gonorrhoea. Azithromycin testing has been recently introduced by all states and territories as it is part of a dual therapy regimen with ceftriaxone recommended for the treatment of gonorrhoea in the majority of Australia. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. In certain remote regions of the Northern Territory and Western Australia gonococcal antimicrobial resistance rates are low and an oral treatment regimen comprising amoxycillin, probenecid and azithromycin is recommended for the treatment of gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5% or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatments.[1] Additional data on other antibiotics are reported in the AGSP annual report. The AGSP has a program-specific quality assurance process. The AGSP data are presented quarterly in tabulated form, as well as in the AGSP annual report. For more information see *Commun Dis Intell* 2015;39(1):E178–E179.

Results

A summary of the proportion of isolates with extragenital to ceftriaxone, and the proportion resistant to penicillin, ciprofloxacin and azithromycin are shown in Table 1.

Penicillin resistant *Neisseria gonorrhoeae* (NG) are defined as those isolates with a minimum inhibitory concentration (MIC) to penicillin equal to or greater than 1.0 mg/L. Penicillin resistance includes penicillinase producing NG (PPNG), and NG that have chromosomally mediated resistance to penicillin (CMRP). In certain areas of the Northern Territory and Western Australia, which are classified as remote, a treatment regimen based on oral amoxycillin, probenecid and azithromycin is used. Due to the distance specimens must travel to a laboratory from these remote regions, low numbers of cultures are collected, and thus, by necessity, nucleic acid amplification testing (NAAT) is used. In remote Western Australia the introduction of a targeted NAAT, developed by the NNN to detect PPNG, is in use to enhance surveillance.[2,3]

Ciprofloxacin

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

Table 1: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to ciprofloxacin, azithromycin and penicillin, Australia, 1 January to 31 March 2015, by state or territory

State or territory	Number of isolates tested	Decreased susceptibility Ceftriaxone		Resistance Ciprofloxacin		Azithromycin		Penicillin	
		n	%	n	%	n	%	n	%
Australian Capital Territory	14	0	0.0	6	43.0	0	0.0	4	29.0
New South Wales	568	17	3.0	200	35.0	8	1.4	156	28.0
Queensland	206	0	0.0	54	26.0	12	6.0	54	26.0
South Australia	39	0	0.0	16	41.0	0	0.0	8	20.5
Tasmania	7	0	0.0	0	0.0	1	14.0	0	0.0
Victoria	485	9	1.9	112	23.0	8	1.6	71	15.0
Northern Territory/Urban and Rural	21	0	0.0	2	9.5	0	0.0	3	14.0
Northern Territory/Remote	55	0	0.0	3	5.5	0	0.0	3	5.5
Western Australia/Urban and Rural	78	0	0.0	16	21.0	8	10.0	18	23.0
Western Australia/Remote	11	0	0.0	0	0.0	0	0.0	0	0.0
Australia	1,484	26	1.7	409	28.0	37	2.5	317	21.0

Azithromycin

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

Ceftriaxone

Ceftriaxone MIC values in the range 0.06–0.125 mg/L have been reported in the category extragenital (DS) since 2005.

In the 1st quarter of 2015 the only states that reported isolates with DS to ceftriaxone were New South Wales and Victoria. Both reported a decrease in the proportion of NG isolates with DS to ceftriaxone when compared with the same quarter in 2014; and the annual data for 2014.[4]

From New South Wales there were 17 of 568 strains with DS to ceftriaxone. Of those, 12 (71%) were multi-drug resistant (MDR); 12 (71%) were from males; and 8 (47%) were isolated from extragenital sites (rectal and pharyngeal). From Victoria, there were 9 of 485 strains with DS to ceftriaxone. Of those, all (100%) were MDR; 8 (89%) were from males; and 4 (44%) were isolated from extragenital sites (rectal and pharyngeal).

The proportion of strains with DS to ceftriaxone is of increasing concern in Australia and overseas, as this is phenotypic of the genotype with the key mutations that are the precursor to ceftriaxone resistance.[5] There are recent reports of ceftriaxone 500 mg treatment failures in patients from Victoria and New South Wales in patients with pharyngeal gonococcal infections. In these patients the infecting gonococcal strains had ceftriaxone MIC values in the range 0.03–0.06 mg/L.[6,7] Until 2013 there had not been an isolate reported in Australia with a ceftriaxone MIC value >0.125 mg/L.[4] In late December 2013, there was a new multi-drug-resistant gonococcal strain (A8806) with a ceftriaxone MIC of 0.5 mg/L, the highest ever reported in Australia, which was isolated from a female traveller from Central Europe. This infection was acquired in Sydney from another traveller, also from Europe. The patient was tested in the Northern Territory, but had travelled to north-eastern Queensland before the results were available, and was treated there. To date there has been no evidence of spread of this strain.[8]

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

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

References

1. Surveillance of antibiotic susceptibility of *Neisseria gonorrhoeae* in the WHO Western Pacific Region 1992–4. WHO Western Pacific Region Gonococcal Antimicrobial Surveillance Programme. 1997.
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8. Australian Sexual Health Alliance. The Australian Sexually Transmitted Infection Management Guidelines 2014. [Online]. Available from: www.sti.guidelines.org.au

Table 2: Percentage of gonococcal isolates with decreased susceptibility to ceftriaxone MIC 0.06–0.125 mg/L, Australia, 2010 to 2014, and 1 January to 31 March 2015, by state or territory

Ceftriaxone MIC mg/L	2010	2011	2012	2013	2014	2015 Q1
0.06	4.6%	3.2%	4.1%	8.2%	4.8%	1.6%
0.125	0.1%	0.1%	0.3%	0.6%	0.6%	0.1%

Australian Meningococcal Surveillance Programme quarterly report, 1 January to 31 March 2015

Monica M Lahra, Ratan Kundu for the Australian Meningococcal Surveillance Programme

Introduction

The reference laboratories of the Australian Meningococcal Surveillance Programme (AMSP) report data on the number of cases confirmed by laboratory testing using culture and by non-culture based techniques. Culture positive cases, where *Neisseria meningitidis* is grown from a normally sterile site or skin lesions, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in quarterly reports are restricted to a description of the number of cases by jurisdiction and serogroup, where known. Some minor corrections to data in the Table may be made in subsequent reports if additional data are received. A full analysis of laboratory confirmed cases of IMD in each calendar year is contained in the AMSP annual reports published in *Communicable Diseases Intelligence*. For more information see *Commun Dis Intell* 2015;39(1):E179.

Results

Laboratory confirmed cases of invasive meningococcal disease for the period 1 January to 31 March 2015 are shown in the Table.

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 January to 31 March 2015, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD	Q1	YTD
Australian Capital Territory	2015	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2014	0	0	0	0	0	0	0	0	0	0	0	0	0	0
New South Wales	2015	0	0	3	3	1	1	0	0	1	1	1	1	6	6
	2014	0	0	3	3	0	0	0	0	2	2	2	2	7	7
Northern Territory	2015	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2014	0	0	1	1	0	0	0	0	0	0	0	0	1	1
Queensland	2015	0	0	5	5	0	0	1	1	0	0	0	0	6	6
	2014	0	0	6	6	0	0	0	0	1	1	0	0	7	7
South Australia	2015	0	0	2	2	0	0	0	0	0	0	0	0	2	2
	2014	0	0	5	5	0	0	0	0	0	0	0	0	5	5
Tasmania	2015	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	2014	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Victoria	2015	0	0	7	7	0	0	0	0	1	1	1	1	9	9
	2014	0	0	3	3	0	0	0	0	1	1	0	0	4	4
Western Australia	2015	0	0	1	1	2	2	0	0	0	0	0	0	3	3
	2014	0	0	2	2	0	0	0	0	0	0	0	0	2	2
Total	2015	0	0	18	18	3	3	1	1	2	2	2	2	26	26
	2014	0	0	20	20	0	0	0	0	4	4	2	2	26	26

Australian Sentinel Practices Research Network, 1 January to 31 March 2015

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

Introduction

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

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

Since 2010, ASPREN GPs have been collecting nasal swab samples for laboratory testing, allowing for viral testing of 20% of influenza-like illness (ILI) patients for a range of respiratory viruses including influenza A, influenza B and A(H1N1) pdm09.

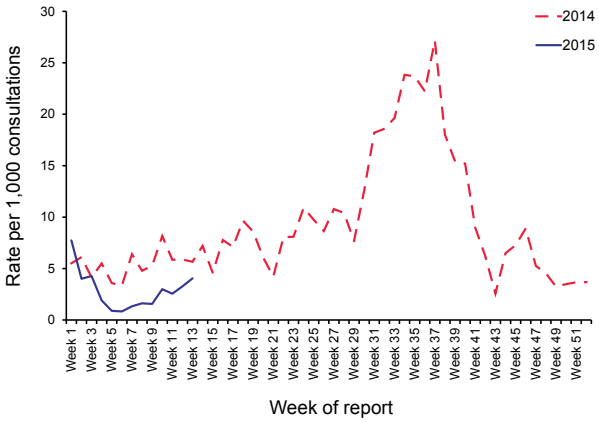
The list of conditions reported is reviewed annually by the ASPREN management committee. In 2015, 4 conditions are being monitored. They include ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, published in *Commun Dis Intell* 2015;39(1):E180.

Results

Sentinel practices contributing to ASPREN were located in all 8 states and territories in Australia. A total of 213 general practitioners regularly contributed data to ASPREN in the 1st quarter of 2015. Each week an average of 185 general practitioners provided information to ASPREN at an average of 14,919 (range 9,032–16,551) consultations per week and an average of 124 (range 91–171) notifications per week.

ILI rates reported from 1 January to 31 March 2015 averaged 3 cases per 1,000 consultations (range 1–8 cases per 1,000 consultations). This was lower compared with rates in the same reporting period in 2014, which averaged 5 cases per 1,000 consultations (range 3–8 cases per 1,000 consultations, Figure 1).

Figure 1: Consultation rates for influenza-like illness, ASPREN, 2014 and 1 January to 31 March 2015, by week of report



The ASPREN ILI swab testing program continued in 2015 with 95 tests being undertaken from 1 January to 31 March. The most commonly reported virus during this reporting period was rhinovirus (27.3% of all swabs performed, Figure 2), with the 2nd most common virus being influenza A (5.3% of all swabs performed).

From the beginning of 2015 to the end of week 13, 8 cases of influenza have been detected with five of these typed as influenza A (27.3% of all swabs performed) and the remaining three being influenza B (3.2% of all swabs performed) (Figure 2).

During this reporting period, consultation rates for gastroenteritis averaged 6 cases per 1,000 consultations (range 4–11 cases per 1,000, Figure 3). This was the same compared with rates in the same reporting period in 2014 where the average was 6 cases per 1,000 consultations (range 4–8 cases per 1,000).

Varicella infections were reported at a lower rate for the 1st quarter of 2015 compared with the same period in 2014. From 1 January to 31 March 2015,

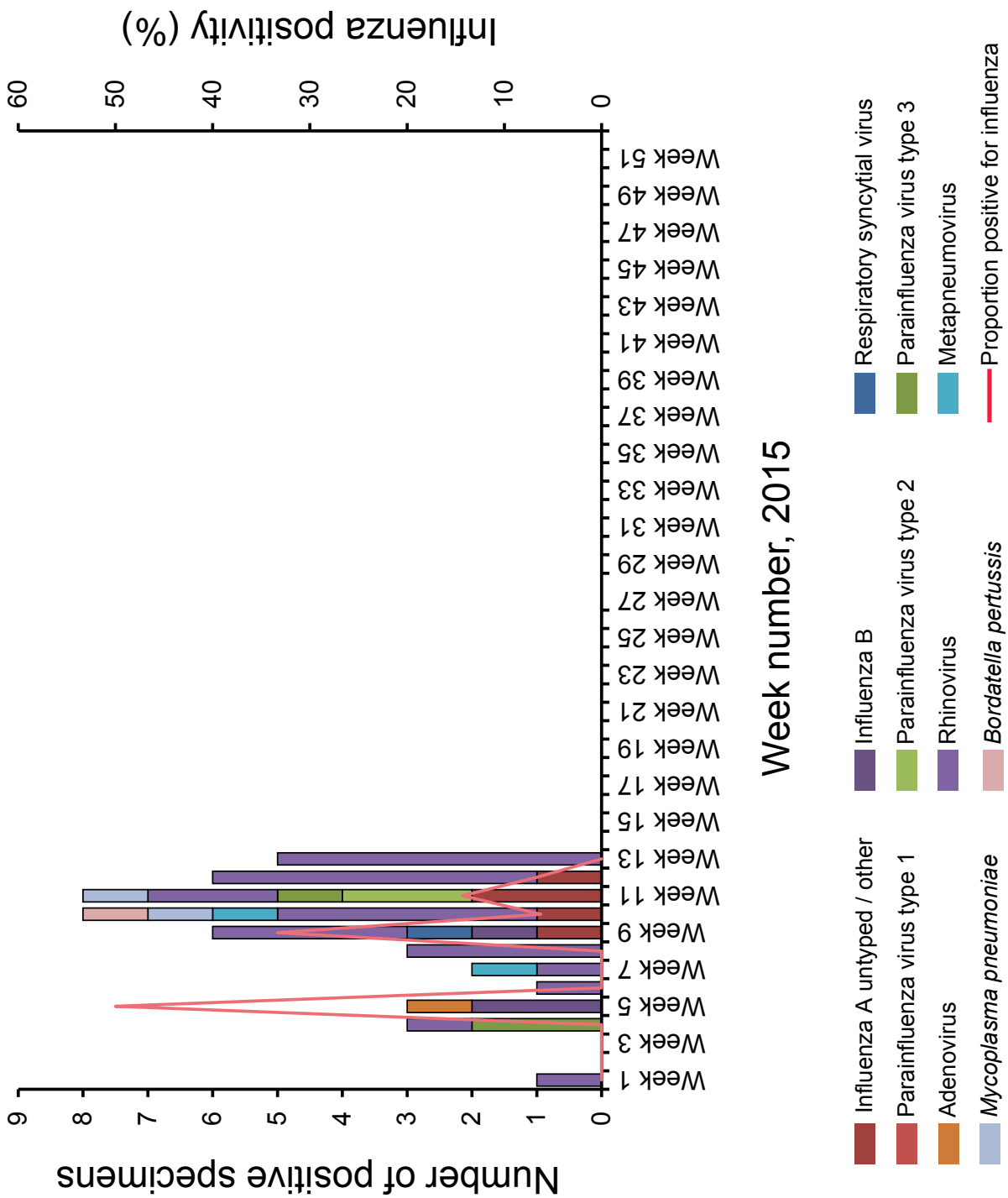


Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January to 31 March 2015, by week of report

recorded rates for chickenpox averaged 0.07 cases per 1,000 consultations (range 0.00–0.25 cases per 1,000, Figure 4).

In the 1st quarter of 2015, reported rates for shingles averaged 1.5 cases per 1,000 consultations (range 0.90–2.52 cases per 1,000 consultations, Figure 5), which was higher compared with the same reporting period in 2014 where the average shingles rate was 1.11 cases per 1,000 consultations (range 0.53–2.14 cases per 1,000 consultations).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 2014 and 1 January to 31 March 2015, by week of report

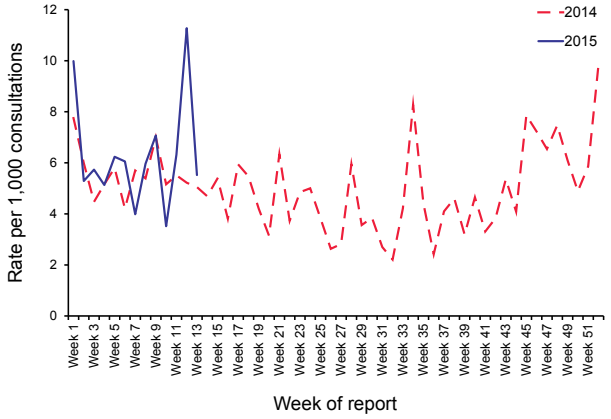


Figure 4: Consultation rates for chickenpox, ASPREN, 2014 and 1 January to 31 March 2015, by week of report

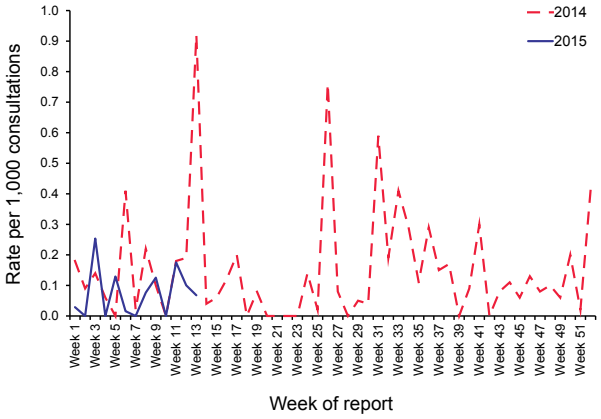
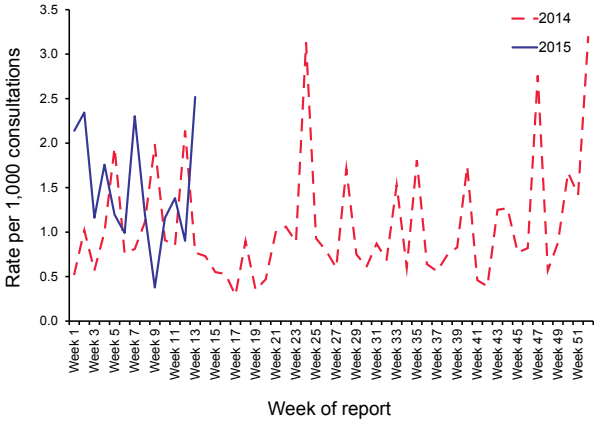


Figure 5: Consultation rates for shingles, ASPREN, 2014 and 1 January to 31 March 2015, by week of report



Invasive pneumococcal disease surveillance Australia, 1 October to 31 December 2014

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

- There were 349 cases of IPD reported to the NNDSS in the 4th quarter of 2014, bringing the year to date total to 1,558 cases (Table).
- The total number of cases in the year to date was similar to the number of cases reported for the same period in 2013 (n=1,543).
- Aboriginal and Torres Strait Islander peoples accounted for 13% (n=39) of all cases with a reported Indigenous status, in the quarter (Table).

Introduction

Invasive pneumococcal disease (IPD) is caused by the bacterium *Streptococcus pneumoniae* and results in illnesses such as pneumonia, bacteraemia and meningitis. There are currently more than 90 serotypes recognised worldwide, approximately half of which are found in Australia where IPD has been a nationally notifiable disease since 2001. This quarterly report documents trends in notified cases of IPD occurring in Australia in the 4th quarter of 2014. In this quarterly report, 3 age groups have been selected for focused analysis. These age groups align with groups that carry the greatest burden of disease and against which the National Immunisation Program is targeted. The data in this report are provisional and subject to change as laboratory results and additional case information become available.

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

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk or Australians age 65 years or over.*

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

Results

There were 349 cases of IPD reported to the NNDSS in the 4th quarter of 2014, bringing the year to date total to 1,558 cases. The number of cases notified in the reporting period decreased to almost half that of the 3rd quarter (n=587). The large reduction in notifications from the 3rd quarter to the 4th quarter is consistent with the usual peak in the number of cases of IPD in the winter months (Table). For the report period and the calendar year, the total number of cases was similar to the number of cases reported in 2013 (4th quarter n=342; calendar year n=1,543).

Overall, and for cases reported as non-Indigenous, the numbers of notified cases were highest in the under 5 years age group followed by the over 85 years age group (Figure 1). In cases reported as Indigenous, the most prevalent age group was the under 5 years age group (n=9) followed by the 35–39 and 55–59 years age groups (n=5 each age group).

Data completeness

During the reporting period, Indigenous status was reported for 87% (n=305) of cases and serotype information was available for 94% (n=328) of all cases reported (Table).

Invasive pneumococcal disease in children aged less than 5 years

In the 4th quarter of 2014, 15% (n=54) of notified cases were aged less than 5 years, which was a small increase in this proportion compared with the 3rd quarter (11%, n=66) (Figure 2). For the 2014 calendar year, there was a small increase in the total number of cases aged less than 5 years (n=211) compared with 2013 (n=191). The annual rate of notified cases in children less than 5 years of age was 14 per 100,000, which was similar to the 2012–2013 historic low of 13 per 100,000.

The majority (87%, n=47) of cases aged less than 5 years were reported with serotype information. Of these, 36% (n=17) were reported with a serotype included in the 7vPCV or the 13vPCV.

Over the period 2007 to 2011, notified cases aged less than 5 years with disease caused by the

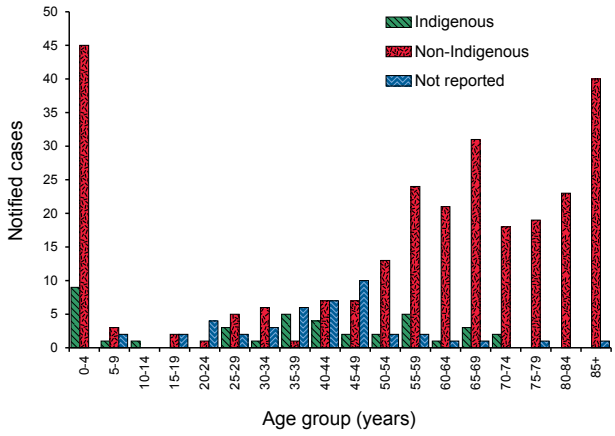
Table: Notified cases of invasive pneumococcal disease, Australia, 1 October to 31 December 2014, by Indigenous status, serotype completeness and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	4th qrt 2014	3rd qrt 2014	4th qrt 2013	Year to date 2014
Indigenous	0	2	8	6	3	0	0	20	39	62	39	101
Non-Indigenous	3	96	1	35	19	11	68	33	266	468	277	734
Not stated/ unknown	0	23	0	0	1	0	20	0	44	57	26	101
Total	3	121	9	41	23	11	88	53	349	587	342	1,558
Indigenous status completeness* (%)	100	81	100	100	96	100	77	100	87	–	–	–
Serotype completeness† (%)	100	93	100	100	91	100	89	100	94	–	–	–

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

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

Figure 1: Notifications of invasive pneumococcal disease, Australia, 1 October to 31 December 2014, by Indigenous status and age group



6 additional serotypes (1, 3, 5, 6A, 7F and 19A) that would be covered by the 13vPCV, increased steadily, particularly those caused by serotype 19A (Figure 3). Since the introduction of the 13vPCV into the universal childhood immunisation program in mid-2011, cases of disease caused by the 6 additional serotypes have generally decreased. In the 4th quarter of 2014, there were 6 cases aged less than 5 years with disease due to serotype 19A, 2 cases due to serotype 3, 1 case of serotype 1 and no cases of 6A. In this age group, 3 cases were reported with disease caused by serotype 7F, a previously common serotype in Australia. For 2014, the number of notified cases aged less than 5 years with disease caused by the 6 additional serotypes (1, 3, 5, 6A, 7F and 19A) that would be covered by the 13vPCV (n=59) was similar to 2013 (n=60).

Invasive pneumococcal disease in Indigenous Australians aged 50 years or over

In the 4th quarter of 2014, 4% (n=13) of notified cases were reported as Indigenous Australians aged 50 years or over (Figure 4), which was similar to the proportion of notifications in the 3rd quarter (3%).

All of the cases notified in the 4th quarter of 2014 were reported with serotype information. Of these, 62% (n=8) were reported with disease due to serotypes targeted by the 23vPPV. The remaining cases reported disease due to a non-23vPPV serotype (n=5). During 2014, the annual rate decreased to 62 per 100,000, a 24% reduction from the peak rate of 2012 (82 per 100,000 population).

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

In the 4th quarter of 2014, 38% (n=134) of all notified cases were reported as non-Indigenous and aged 65 years or over (Figure 5). This was similar to the proportion of cases reported in the previous quarter (34%) and in the 4th quarter of 2013 (35%). In the 2014 calendar year, the annual rate was 15 per 100,000, a 40% reduction from the peak rate of 2004 (25 per 100,000 population) and a small reduction on 2013 (16 per 100,000).

The majority (97%, n=130) of cases reported in this quarter were reported with serotype information.

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

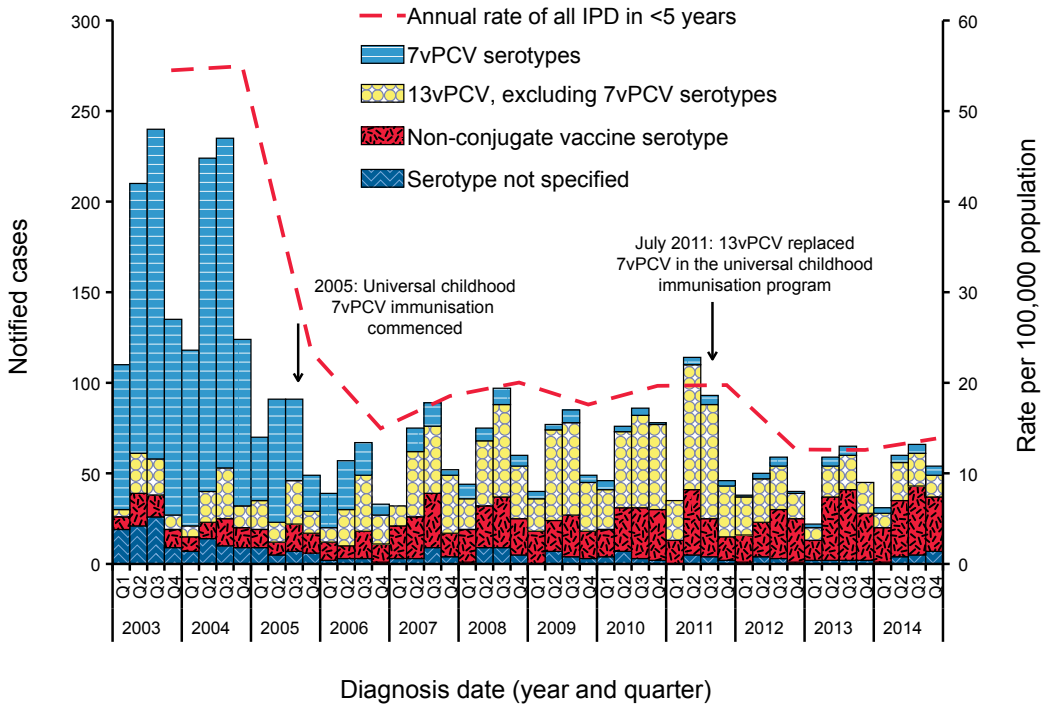
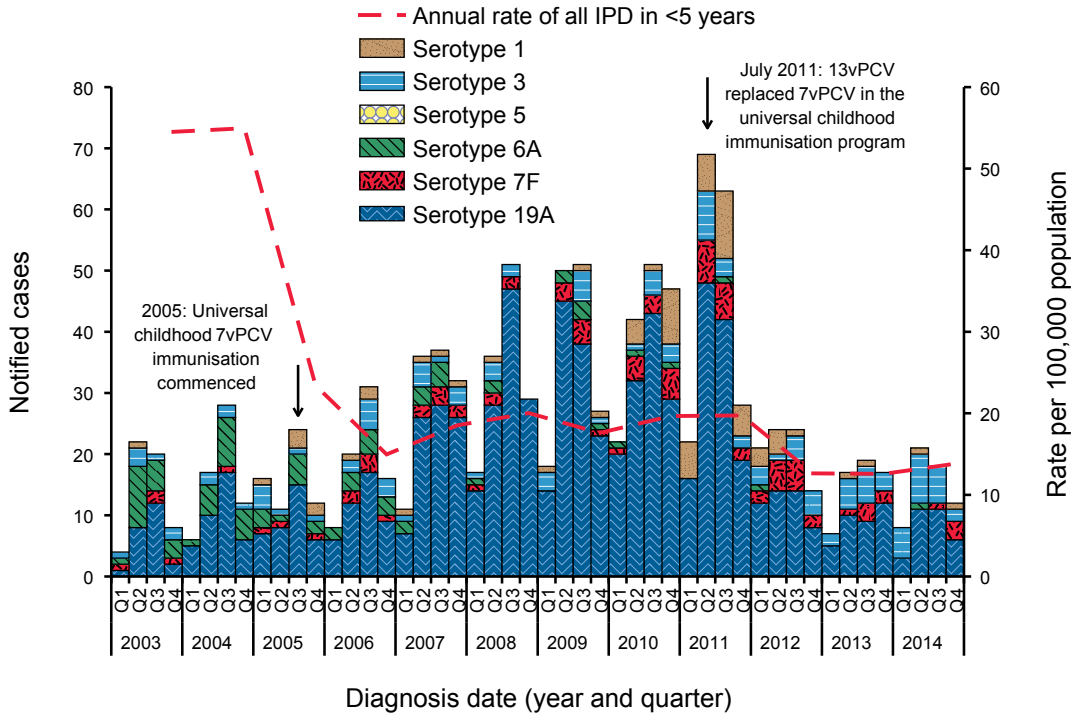
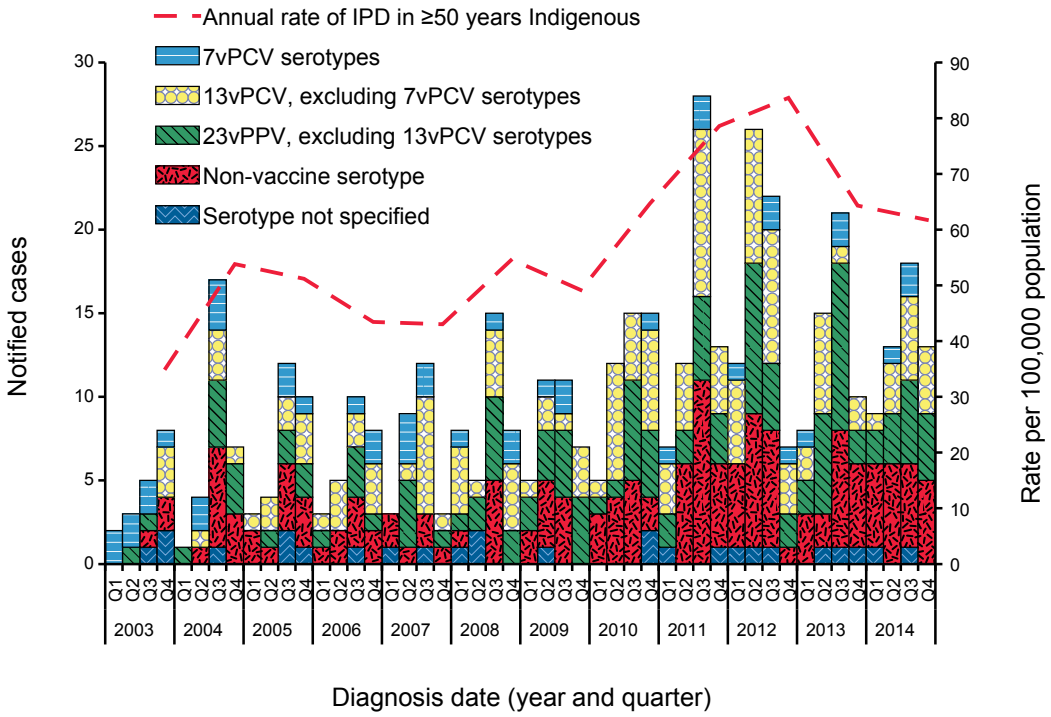


Figure 3: Notifications of invasive pneumococcal disease caused by serotypes targeted by the 13-valent pneumococcal conjugate vaccine* and annual rates of all invasive pneumococcal disease, aged less than 5 years, Australia, 2003 to 2014



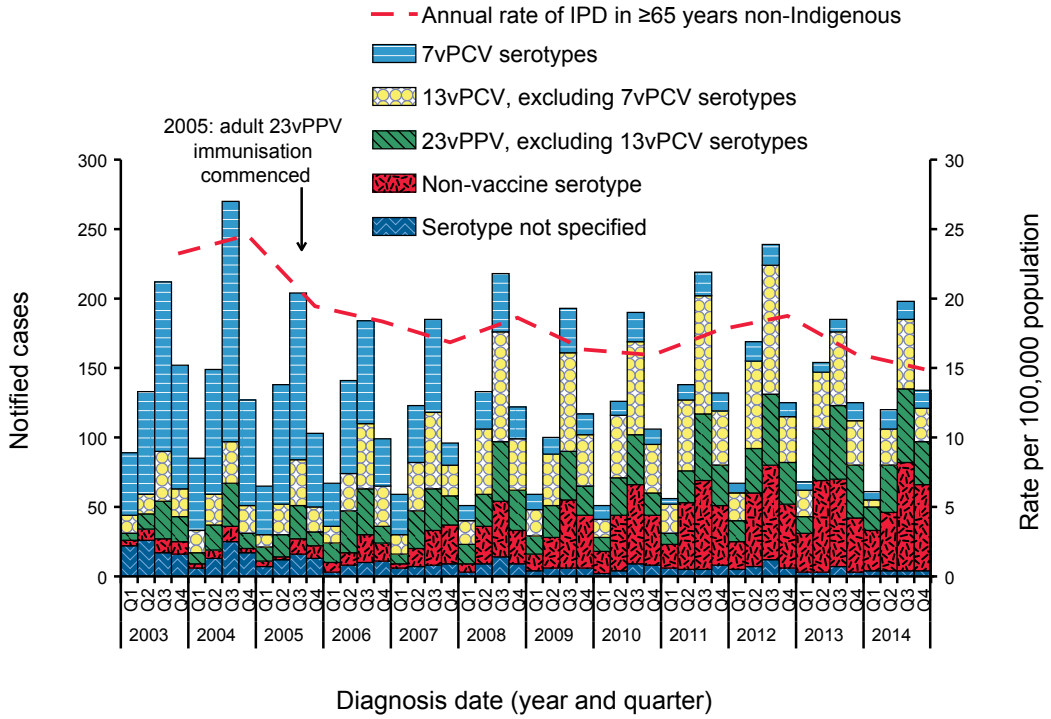
* Excludes those targeted by 7-valent pneumococcal conjugate vaccine.

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



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

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



Of these cases, 52% (n=68) were reported with a serotype targeted by the 23vPPV. While the burden of disease in this age group has remained relatively stable, the profile of serotypes causing disease has changed over time. Disease due to serotypes targeted by the 7vPCV has reduced substantially in this age group, which is likely to be due to herd immunity impacts from the childhood immunisation program.

Mortality due to invasive pneumococcal disease

Nationally, there were 31 deaths attributed to 17 different IPD serotypes during this reporting period. There were 2 deaths in the under 5 years age group, neither of which were due to serotypes included in the 7vPCV or 13vPCV.

Conclusion

The number of notified cases of IPD in the 4th quarter of 2014 was almost half that of the 3rd quarter. The large reduction in notifications from the 3rd quarter to the 4th quarter is consistent with the usual peak in the number of cases of IPD in the winter months. For the report period and the calendar year, the total number of cases was similar to the number of cases reported in 2013. The decline in disease due to the serotypes targeted by vaccines has been maintained and IPD associated with non-vaccine serotypes has remained stable in all groups targeted for IPD vaccination. Disease in non-Indigenous Australians aged 65 years or over has remained relatively stable but the profile of serotypes causing disease in this cohort has diversified.

Acknowledgements

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Invasive pneumococcal disease surveillance Australia, 1 January to 31 March 2015

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

Summary

The number of notified cases of invasive pneumococcal disease (IPD) in the 1st quarter of 2015 was less than the previous quarter and less than the number of notified cases in the 1st quarter of 2014. Overall, the decline in disease due to the serotypes targeted by the 13-valent pneumococcal conjugate vaccine (13vPCV), across all age groups, has been maintained since the 13vPCV replaced the 7-valent pneumococcal conjugate vaccine (7vPCV) in the childhood immunisation program from July 2011.

Key points

In the 1st quarter of 2015, there were 188 cases of IPD reported to the NNDSS. This was a 13% reduction on the number of cases reported for the same period in 2014 (n=215) (Table 1). Most serotypes affect all age groups, with serotype 19A being the most common cause of IPD overall (Table 2).

In non-Indigenous Australians, the number of notified cases was highest in the under 5 years age group followed by the 65–69 years age group. In Aboriginal and Torres Strait Islander people, the number of notified cases was highest in the under 5 years age group followed by the 50–54 years age group (Table 3). The distribution of IPD cases across age groups and by Indigenous status was similar to the 1st quarter of 2014 (http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-cdi3802n.htm).

There were 30 cases of IPD reported in children under 5 years of age, of which 39% (n=11) were due to a serotype included in either the 7vPCV or the 13vPCV (Figure 1). The most common serotypes affecting this age group were 19A and 19F, both of which are included in the 13vPCV (Table 2). The number of cases in this age group and serotype distribution is similar to the 1st quarter of 2014.

There were 10 cases of IPD reported in Indigenous Australians aged 50 years or over. Of those cases with a reported serotype, 44% (n=4) were due to a serotype included in the 23-valent polysaccharide pneumococcal vaccine (23vPPV) (Figure 2). The number of notified cases of IPD in this age group was similar to the 1st quarter of 2014 (n=9) and a small reduction from the previous quarter (n=14). The proportion of 23vPPV serotypes also remained stable (2014 quarter 4, 38%).

There were 55 cases of IPD reported in non-Indigenous Australians aged 65 years or over. Of

Table 1: Notified cases of invasive pneumococcal disease, Australia, 1 January to 31 March 2015, by Indigenous status, serotype completeness and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Total Q1 2015	Total Q4 2014	Total Q1 2014	Year to date 2015
Indigenous	0	6	10	7	2	0	0	8	33	40	45	33
Non-Indigenous	1	29	2	21	14	4	44	11	126	272	148	126
Not stated/ unknown	0	14	0	2	0	0	13	0	29	41	22	29
Total	1	49	12	30	16	4	57	19	188	353	215	188
Indigenous status completeness* (%)	100	71	100	93	100	100	77	100	85			–
Serotype completeness† (%)	100	94	100	93	95	100	98	98	96			–

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

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

Table 2: Frequently notified serotypes of invasive pneumococcal disease, Australia, 1 January to 31 March 2015, by age group

Serotype	Age group			Serotype total*
	Under 5 years	5 to 64 years	Over 65 years	
19A	5	6	6	17
23B	3	5	5	13
19F	4	5	3	12
6C	0	4	8	12
3	2	6	1	9
9N	2	6	1	9
16F	0	5	3	8
8	1	6	1	8
11A	0	5	2	7
15A	0	2	4	6
22F	0	2	4	6
23A	0	2	4	6
15B	2	2	1	5
33F	0	3	2	5
7F	0	5	0	5
Other	8	18	17	43
Serotype unknown†	3	10	4	17
Total	30	92	66	188

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

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

Figure 1: Notifications (2004 to 31 March 2015) and annual rates (2004 to 2014) of invasive pneumococcal disease in children aged less than 5 years, Australia, by vaccine serotype group

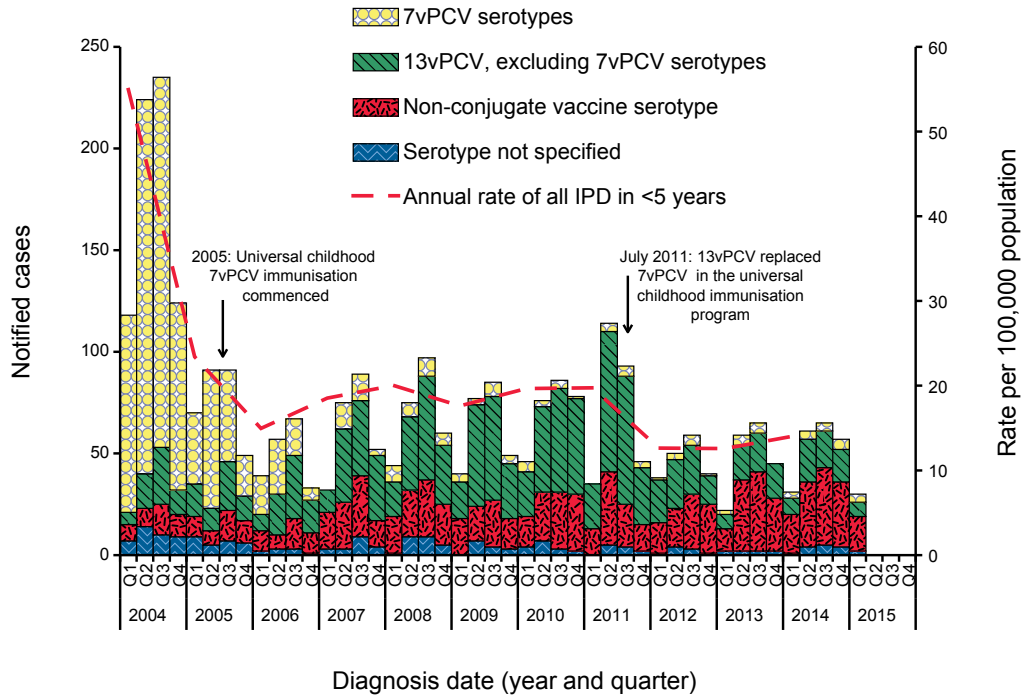


Table 3 Notified cases of invasive pneumococcal disease, Australia, 1 January to 31 March 2015, by Indigenous status and age group

Age group	Indigenous status			
	Indigenous	Non-Indigenous	Not reported	Total
0–4	8	20	2	30
5–9	2	4	2	8
10–14	2	2	1	5
15–19	1	1	2	4
20–24	1	0	0	1
25–29	0	0	0	0
30–34	1	5	0	6
35–39	1	2	2	5
40–44	4	2	2	8
45–49	3	2	5	10
50–54	5	13	2	20
55–59	2	10	0	12
60–64	1	10	2	13
65–69	0	14	4	18
70–74	1	10	1	12
75–79	0	13	1	14
80–84	1	6	0	7
85+	0	12	3	15
Total	33 (18%)	126 (67%)	29 (15%)	188

those cases with a reported serotype, 40% (n=21) were due to a serotype included in the 23vPPV (Figure 3). The number of cases in this age group was similar to the same quarter of the previous 5 years however the proportion of vaccine serotypes has steadily decreased over the same period.

In this quarter there were 12 deaths attributed to 11 different IPD serotypes. No deaths in children aged under 5 years were reported.

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* (CDI).

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk 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).

Figure 2: Notifications (2004 to 31 March 2015) and annual rates of all invasive pneumococcal disease (2004 to 2014) in Indigenous Australians aged 50 years or over, Australia, by vaccine serotype group

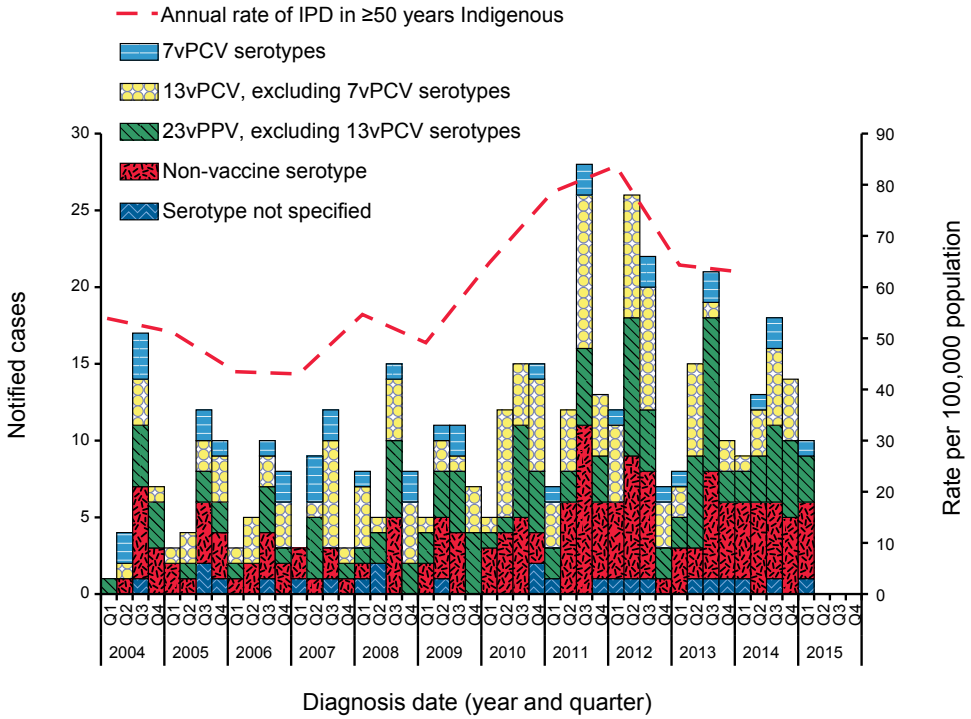
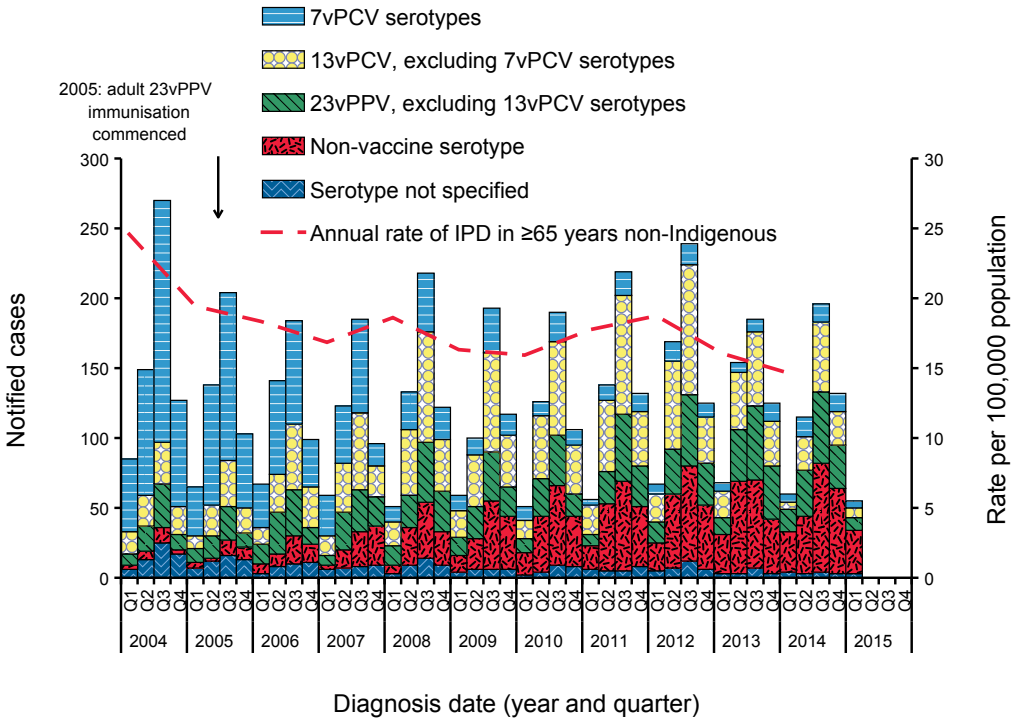


Figure 3: Notifications (2004 to 31 March 2015) and annual rates of all invasive pneumococcal disease (2004 to 2014) in non-Indigenous Australians aged 65 years or over, Australia, by vaccine serotype group



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Policy and guidelines

New surveillance case definition: Avian influenza in humans (AIH)

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

The following new case definition has been reviewed by CDWG and endorsed by CDNA.

This case definition was implemented on 1 July 2015.

Avian influenza in humans (AIH)

Reporting

Both confirmed cases and probable cases should be notified. Suspected cases shouldn't be notified.

Confirmed case

A confirmed case requires laboratory definitive evidence AND clinical evidence

Laboratory definitive evidence

Isolation of an Avian Influenza (AI) virus

OR

Detection of AI by nucleic acid testing using two different targets, e.g. primers specific for influenza A and AI haemagglutinin (genetic sequencing should be employed to confirm diagnosis);

OR

A fourfold or greater rise in antibody titre to the AI virus detected in the outbreak (or AI virus suspected of causing the human infection), based on testing of an acute serum specimen (collected 7 days or less after symptom onset) and a convalescent serum specimen. The convalescent neutralising antibody titre must also be 80 or higher.

OR

An antibody titre to the AI virus detected in the outbreak (or AI virus suspected of causing the human infection) of 80 or greater in a single serum specimen collected at day 14 or later after symptom onset. The result should be confirmed in at least two different serological assays (i.e haemagglutinin-inhibition, microneutralisation, positive Western blot, etc).

Note: Tests must be conducted in a national, regional or international influenza laboratory whose Avian Influenza in Humans (AIH) test results are accepted by WHO as confirmatory

Clinical evidence

An acute illness characterised by:

a. Fever (>38ºC) or history of fever AND one or more of; cough OR rhinorrhoea OR myalgia OR headache OR dyspnoea OR diarrhoea;

OR

b. Conjunctivitis

OR

c. infiltrates or evidence of an acute pneumonia on chest radiograph plus evidence of acute respiratory insufficiency (hypoxaemia, severe tachypnoea).

Probable case

A probable case requires laboratory suggestive evidence AND Clinical evidence AND Epidemiological evidence

Laboratory suggestive evidence

Confirmation of an influenza A infection but insufficient laboratory evidence for AIH infection.

Clinical evidence

As with confirmed case

Epidemiological evidence

One or more of the following exposures in the 10 days prior to symptom onset:

a. Close contact (within 1 metre) with a person (e.g. caring for, speaking with, or touching) who is a probable, or confirmed AIH case;
b. Exposure (e.g. handling, slaughtering, defeathering, butchering, preparation for consumption) to poultry or wild birds or their remains or to environments contaminated by their faeces in an area where AI infections in animals or humans have been suspected or confirmed in the last month;
c. Consumption of raw or undercooked poultry products in an area where AI infections in animals or humans have been suspected or confirmed in the last month;
d. Close contact with a confirmed AI infected animal other than poultry or wild birds (e.g. cat or pig);
e. Handling samples (animal or human) suspected of containing AI virus in a laboratory or other setting.

Suspected case

A suspected case requires clinical evidence AND epidemiological evidence

Clinical evidence for suspected case

As with confirmed case

Epidemiological evidence

As with probable case.

Note: For overseas exposures, an AI-affected area is defined as a region within a country with confirmed outbreaks of AI strains in birds or detected in humans in the last month (seek advice from the National Incident Room when in doubt). With respect to the H5N1 AI outbreak that commenced in Asia in 2003, information regarding H5-affected countries is available from the World Health Organization Global Health Observatory Map Gallery (http://gamapserver.who.int/mapLibrary/). With respect to the H7N9 outbreak that commenced in eastern China in 2013, information regarding H7-affected countries is available from the World Health Organization Avian influenza web site (http://www.who.int/influenza/human_animal_interface/influenza_h7n9/en/).

Revised surveillance case definitions

This report provides the revised Surveillance case definitions approved by the Communicable Diseases Network Australia (CDNA) since 1 January 2015.

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

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

These case definitions were implemented on 1 July 2015 and supersede any previous versions.

Hepatitis B – newly acquired

Reporting

Only confirmed cases should be notified.

Confirmed case

A confirmed case requires laboratory definitive evidence only.

Laboratory definitive evidence

Detection of hepatitis B surface antigen (HBsAg) in a patient shown to be negative within the last 24 months

OR

Detection of HBsAg and IgM to hepatitis B core antigen, except where there is prior evidence of hepatitis B infection

OR

Detection of hepatitis B virus by nucleic acid testing, and IgM to hepatitis B core antigen, except where there is prior evidence of hepatitis B infection

Note:

Transient HBsAg positivity can occur in patients following HBV vaccination. This occurs more commonly in dialysis patients and is unlikely to persist beyond 14 days post-vaccination.

Hepatitis B – newly acquired	**Laboratory definitive evidence** For clarity, remove “in the absence of prior evidence of hepatitis B infection” and insert “except where there is prior evidence of hepatitis B infection”. **Note** To caution about the influence of recent vaccination, add note: “Transient HBsAg positivity can occur in patients following HBV vaccination. This occurs more commonly in dialysis patients and is unlikely to persist beyond 14 days post-vaccination”

Hepatitis B – unspecifed

Reporting

Only confirmed cases should be notified.

Confirmed case

A confirmed case requires laboratory definitive evidence AND that the case does not meet any of the criteria for a newly acquired case.

Laboratory definitive evidence

Detection of hepatitis B surface antigen (HBsAg), or hepatitis B virus by nucleic acid testing, except where there is prior evidence of hepatitis B infection.

Note:

Transient HBsAg positivity can occur in patients following HBV vaccination. This occurs more commonly in dialysis patients and is unlikely to persist beyond 14 days post-vaccination.

Hepatitis B – unspecified	**Laboratory definitive evidence** For clarity, remove "in the absence of prior evidence of hepatitis B infection" and insert "except where there is prior evidence of hepatitis B infection". **Note** To caution about the influence of recent vaccination, add note: "Transient HBsAg positivity can occur in patients following HBV vaccination. This occurs more commonly in dialysis patients and is unlikely to persist beyond 14 days post-vaccination"

Hepatitis E

Reporting

Only confirmed cases should be notified.

Confirmed case

A confirmed case requires laboratory definitive evidence

OR

Laboratory suggestive evidence AND clinical evidence

Laboratory definitive evidence

Detection of hepatitis E virus by nucleic acid testing

OR

Detection of hepatitis E virus in faeces by electron microscopy

OR

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

Laboratory suggestive evidence

Detection of IgM or IgG to hepatitis E virus.

Clinical evidence

A clinically compatible illness without other apparent cause.

Hepatitis E	**Confirmed case** Remove requirement for epidemiological evidence so that a positive IgM or IgG in combination with clinical evidence can constitute a confirmed case Remove **Epidemiological evidence** section

Syphilis – congenital

Reporting

Both confirmed cases and probable cases should be notified, including syphilis-related stillbirth[1]

Confirmed case

A confirmed case requires laboratory definitive evidence.

Laboratory definitive evidence

Mother and child both seropositive by a treponemal specific test[2]

AND

One or more of the following:

Direct demonstration of *Treponema pallidum* by any of the following: nucleic acid amplification (NAA) test, dark field microscopy, fluorescent antibody or silver stain - in specimens from lesions, nasal discharge, placenta, umbilical cord, cerebrospinal fluid (CSF), amniotic fluid or autopsy material

OR

Detection of *Treponema pallidum* specific IgM in the child

OR

The child's serum non-treponemal[3] serology titre at birth is at least fourfold greater than the mother's titre.

Probable case

A probable case requires laboratory suggestive evidence AND clinical evidence.

Laboratory suggestive evidence

Direct demonstration of *Treponema pallidum* as described under laboratory definitive evidence (above), but without serological confirmation in the child.

OR

Child seropositive on non-treponemal testing in the absence of IgM testing

OR

A reactive CSF non-treponemal test (VDRL or RPR) in a child.

OR

A child who remains seropositive by a treponemal specific test at 15 months of age, which is confirmed either by another, different reactive treponemal specific test or a reactive non-treponemal test, in the absence of post-natal exposure to *Treponema pallidum*, including the non-venereal subspecies *Treponema pallidum* subsp. pertenue (Yaws) or subsp. endemicum (Bejel, endemic syphilis).

Clinical evidence

1. Any evidence of congenital syphilis on physical examination

OR

2. Any evidence of congenital syphilis on radiographs of long bones

OR

3. An elevated CSF cell count or protein (without other cause)

OR

4. The mother is seropositive in the perinatal period AND has no documented evidence of adequate treatment.[4]

Notes:

1. A stillbirth where the foetal death has occurred after a 20 week gestation or in a foetus which weighs greater than 500 g should be counted as clinical evidence towards a case where laboratory suggestive or definitive evidence exists.
2. Treponemal-specific tests are:

   Treponema pallidum immunoassays, *Treponema pallidum* haemagglutination assay (TPHA), *Treponema pallidum* particle agglutination assay (TPPA), Fluorescent Treponemal Antibody Absorption (FTA-Abs) and various IgM assays including 19S-IgM antibody test, or IgM immunoassay.

   IgM assays should not be used for screening purposes.

   Treponema pallidum-specific rapid immunochromatography (ICT) assays for use as point-of-care tests are now becoming available, but their performance has not yet been fully established. Positive ICT results should be confirmed with a second treponemal specific assay.

3. Non-treponemal tests are the agglutination assays Rapid Plasma Reagin (RPR) and Venereal

Disease Research Laboratory (VDRL). Any positive sera should be tested by serial dilution to provide an end-titre. Non-treponemal tests may be used to monitor efficacy of treatment. Mother and child sera should be collected contemporaneously and tested in parallel and cord blood should not be used for the investigation of congenital syphilis.

4. Treatment is considered adequate if

   - a stage-appropriate penicillin-containing regimen was used 30 days or more prior to delivery AND

   - all antenatal and delivery pathology investigations were performed and results verified AND

   - there is no evidence of reinfection.

4.1 Treatment with macrolides alone during pregnancy in penicillin-allergic women is no longer regarded as adequate therapy as resistance to macrolides in *T. pallidum* is increasingly common and may arise during therapy.

4.2 Although the risk of congenital syphilis is much higher in early-stage disease, in the presence of untreated syphilis the birth of an unaffected child does not guarantee that subsequent children will not be affected.

Syphilis – congenital	**Reporting** Inclusion of a syphilis-related stillbirth where this was previously a note for the 'Laboratory definitive evidence' section. **Laboratory definitive evidence** Inclusion of detection of *Treponema pallidum* specific IgM in the child. Inclusion of a nucleic acid amplification (NAA) test as a means of direct demonstration of *Treponema pallidum* . **Notes** Removal of the serological criterion for proof of treatment in point 4. This is also reflected in the last sentence of the 'Clinical evidence' section.

Infectious syphilis – less than two years duration (includes primary, secondary and early latent)

Reporting

Confirmed and probable cases should be notified.

Confirmed case

A confirmed case requires either:

1. Laboratory definitive evidence

OR

2. Laboratory suggestive evidence **AND** clinical evidence.

Laboratory definitive evidence

1. Seroconversion in past two years: treponemal specific test[a] reactive when previous treponemal specific test non-reactive within past two years and the latest result is confirmed by either a reactive non-treponemal test[b] or a different reactive treponemal specific test

OR

2. A fourfold or greater rise in non-treponemal antibody titre compared with the titre within past two years, and a reactive treponemal specific test

Laboratory suggestive evidence

1. Demonstration of *Treponema pallidum* by darkfield microscopy (not oral lesions), direct fluorescent antibody microscopy (direct antigen test), equivalent microscopic methods (e.g. silver stains), or DNA methods (e.g. nucleic acid testing)

OR

2. A reactive treponemal specific test confirmed by either a reactive non-treponemal test or a different reactive treponemal specific test

OR

3. A reactive non-treponemal test confirmed by a treponemal specific test.

Clinical evidence

1. Presence of a primary chancre (or ulcer)

OR

2. Clinical signs of secondary syphilis.

Probable case

A probable case requires that the case does not meet the criteria for a confirmed case AND

Either:

a. In a person with no known previous reactive serology: no history of adequate treatment of syphilis, or endemic treponemal disease, and

    1. Contact with an infectious case **AND** laboratory suggestive evidence.

OR

    2. Laboratory suggestive evidence **AND** RPR ≥16.

OR

    3. Positive syphilis IgM **AND** laboratory suggestive evidence.

OR

b. In a person with previous reactive serology: a fourfold or greater rise in non-treponemal antibody titre when the previous serology was done more than two years ago.

AND

    1. Contact with an infectious case, or

OR

    2. Positive syphilis IgM

Notes:

a. Treponemal specific tests are: IgG immunoassay, *Treponema pallidum* haemagglutination assay, *Treponema pallidum* particle agglutination assay, Fluorescent Treponemal Antibody Absorption, 19S-IgM antibody test, or IgM immunoassay

b. Non-treponemal tests are; Rapid Plasma Reagin (RPR), Venereal Disease Research Laboratory (VDRL)

Infectious syphilis – less than two years duration (includes primary, secondary and early latent)	Change name from 'Syphilis – less than 2 years duration (infectious - primary, secondary and early latent)' to '**Infectious** Syphilis – less than two years duration (includes primary, secondary and early latent)' Include new case definition for **infectious syphilis, probable case**. **Reporting** Both confirmed and probable cases should be notified. **Laboratory definitive evidence** Move details regarding treponemal tests to notes section.

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