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Editorial

ANTIMICROBIAL RESISTANCE

Chris Baggoley, Aaliya Ibrahim

Antimicrobial resistance (AMR) has been identified by the World Health Organization as a global health concern, threatening to undo decades of advances in our ability to treat disease.[1] This is a serious issue as AMR contributes to increased patient morbidity and mortality, the complexity and duration of treatments and hospital stay, and results in substantial increases to health care system costs and financial burden to the community. The prevalence of AMR, including resistance to clinically important antibiotics, is increasing both in Australia and internationally. Globalisation increases the vulnerability of Australians to disease. Today, infectious diseases are travelling faster and further than ever before as a result of international travel and medical tourism.

AMR spans both animal and human health and as such requires a nationally coordinated, whole-of-system One Health response. Australia is committed to preventing the emergence and containing the spread of AMR. With this in mind, the Australian Antimicrobial Resistance Prevention and Containment (AMRPC) Steering Group was established in early 2013 to provide high-level governance and leadership on AMR in Australia. The AMRPC Steering Group is jointly chaired by the Secretaries of the Commonwealth Departments of Health and Agriculture, with the Commonwealth Chief Medical Officer and Commonwealth Chief Veterinary Officer also being members. The Steering Group is overseeing the development and implementation of a National AMR Strategy, which will coordinate Australia's efforts across human and animal health to reduce, monitor and respond to the threat of AMR.

An important first step for AMR intervention, which has also been identified by the Steering Group as a key priority area, is establishing an integrated national surveillance system for antibiotic resistance and antibiotic usage across human and animal health, food and agricultural sectors. Currently, there are a number of AMR surveillance systems that are active in Australia, including the Australian Group for Antimicrobial Resistance (AGAR), that provide data on AMR patterns for a number of key pathogens, and the National Antimicrobial Utilisation Surveillance Program, that reports on trends in antimicrobial usage in Australian hospitals. The Australian Commission on Quality and Safety in Health Care (ACSQHC) also manages a number of initiatives to further expand and support improvements in the quality of AMR monitoring and surveillance data, including the National Cumulative Antibiogram, standardisation of laboratory reporting, the Second National Survey of *Clostridium difficile* Infection and the Central line Associated Bloodstream Infection Prevention Project.

The AMR Standing Committee (AMRSC) was established under the Australian Health Protection Principal Committee (AHPPC) in 2012. This committee advises the AHPPC on matters relating to AMR; provides expert advice and assistance on issues relating to AMR; and recommends national priorities relating to AMR for action. In 2013, AMRSC completed a report detailing the current surveillance activities for antibiotic use and resistance in human health and recommended priorities for future action. This report provides an excellent overview of the current situation and will be invaluable in informing the development of a comprehensive national surveillance system. The AHPPC has endorsed the report as has the Australian Health Ministers' Advisory Council.

This issue of *Communicable Diseases Intelligence* contains annual reports of various surveillance systems: AGAR; the Australian Gonococcal Surveillance Programme; and the Australian Meningococcal Surveillance Programme, which provide data on the prevalence of resistance to key antibiotics in major pathogens in Australia. In their report, Coombs et al have referred to the importance of such data for informing infection control practices, antibiotic prescribing policies and drug regulatory matters, illustrating this with data that highlight an increasing trend of vancomycin resistance in enterococci.[2] These annual reports also exemplify the importance of surveillance programs being targeted and species-specific in order to effectively monitor changing antibiotic susceptibility patterns.

The Australian Government has committed $11.9 million over 3 years, starting in 2013–14, for the development of a National AMR Strategy. This includes funding for the ACSQHC to provide the foundations for a national AMR and antibiotic usage surveillance system.

While effective surveillance is a vital component of tackling AMR, it is just one part of the broader multifaceted approach that is required. The AMRPC Steering Group has agreed that in addition to surveillance, key activities under the National AMR Strategy will include the provision of strong central leadership and governance nationally and internationally; implementation of infection prevention and control activities to reduce the spread of infection in general and of resistant infections in particular; development of and streamlined regulatory mechanisms across all sectors; regular engagement with international organisations; effectively communication AMR issues (including national education initiatives, active engagement of key stakeholders and partnerships with governments, non-government organisations, professional societies and international agencies); and strategic AMR related research.

AMR is not confined to human health, but also extends across animal health and agriculture. The development of resistance by bacteria is an unavoidable consequence of bacterial evolution; but greater use of antibiotics in humans, livestock and the environment present greater opportunities for bacteria to become resistant. Due to the cross-sectoral nature of AMR, which impacts on industry, educators, health and veterinary professionals, and the community, it is vital to maintain a One Health approach: a coordinated, collaborative, multidisciplinary and cross-sectoral approach in the development of health strategies for people, animals and the environment.[3] This approach allows for an interdisciplinary dialogue between doctors, veterinarians, farmers, industry and the community, and ensures a consistent approach is undertaken to address health, science, social, environmental and economic issues related to AMR.

In July 2013, the Australian One Health Antimicrobial Resistance Colloquium was hosted by the AMRPC Steering Group. The forum assembled over 60 medical, veterinary and agricultural professionals and policy makers to collaboratively exchange views about AMR and to provide advice on One Health priorities and strategies to address AMR in Australia. Participants discussed key issues to inform development of the National AMR Strategy, with particular reference to surveillance requirements, regulatory reform and the most significant zoonotic AMR risks. The AMRPC Steering Group will continue to consult with stakeholders during the development of the National AMR Strategy. The Colloquium was the first step for establishing an ongoing dialogue.

Forums such as the One Health Colloquium highlight the commitment and cooperation of all stakeholders in tackling the threat of AMR in Australia. However, due to the globalised nature of our world, any efforts by Australia to combat the spread of resistant organisms will be of limited value if such efforts are not emulated internationally. Therefore, in addition to combatting AMR on a national scale, Australia is actively engaging in key international collaborations to elevate the issue of AMR and drive the need for action by all countries. By working together, this threat can be combatted.

Acknowledgements

The authors would like to acknowledge the contribution of Cristal Jones in the Australian Government Department of Health for assisting with the preparation of this editorial.

About the authors

Professor Chris Baggoley is Australia's Chief Medical Officer. Professor Baggoley is a member of the AMR Prevention and Containment Steering Group, the Antimicrobial Resistance Standing Committee and is Chair of the Australian Health Protection Principal Committee.

Aaliya Ibrahim is a graduate in the Office of Health Protection, Australian Government Department of Health.

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Annual reports

Australian Group on Antimicrobial Resistance Enterococcus Surveillance Programme annual report, 2010

Geoffrey W Coombs, Julie C Pearson, Keryn Christiansen, Thomas Gottlieb, Jan M Bell, Narelle George, John D Turnidge for the Australian Group on Antimicrobial Resistance

Abstract

In 2010, 15 institutions around Australia conducted a period prevalence study of key resistances in isolates of *Enterococcus* species associated with a range of clinical disease amongst in- and outpatients. Each institution collected up to 100 consecutive isolates and tested these for susceptibility to commonly used antimicrobials using standardised methods. Vancomycin-resistant *Enterococcus faecium* and *Enterococcus faecalis* were characterised by pulsed-field gel electrophoresis. Multilocus sequence typing was performed on representative pulsotypes of *E. faecium*. Susceptibility results were compared with similar surveys conducted in 1995, 1999, 2003, 2005, 2007 and 2009. In the 2010 survey, *E. faecalis* (1,201 isolates) and *E. faecium* (170 isolates) made up 98.9% of the 1,386 isolates tested. Ampicillin resistance was very common (85.3%) in *E. faecium* and absent in *E. faecalis*. Non-susceptibility to vancomycin was 36.5% in *E. faecium* (similar to the 35.2% in 2009 but up from 15.4% in the 2007 survey) and 0.5% in *E. faecalis*. There were significant differences in the proportion of vancomycin-resistant *E. faecium* between the states ranging from 0% in Western Australia to 54.4% in South Australia. The vanB gene was detected in 62 *E. faecium* and 3 *E. faecalis* isolates. The vanA gene was detected in 1 *E. faecium* isolate. All vancomycin-resistant *E. faecium* belonged to clonal complex 17. The most common sequence type (ST) was ST203, which was found in all regions that had reports of vancomycin resistant enterococci. ST341 was detected only in New South Wales/Australian Capital Territory and ST414 only in South Australia and Victoria. High-level resistance to gentamicin was 34.1% in *E. faecalis* and 66.1% in *E. faecium*. A subset of isolates was tested against high-level streptomycin, linezolid and quinupristin/dalfopristin. High-level streptomycin resistance was found in 8.2% of *E. faecalis* isolates and 43.8% of *E. faecium* isolates. Linezolid non-susceptibility was more common in *E. faecalis* (5.8%) than *E. faecium* (0.9%). Overall 9.4% of *E. faecium* were resistant to quinupristin/dalfopristin (*E. faecalis* is intrinsically resistant). *Commun Dis Intell* 2013;37(3):E199–E209.

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

Introduction

Enterococci are part of the normal flora of the gastrointestinal tract. They can give rise to endogenous infections such as urinary tract infections outside of hospitals. Enterococci are recognised as significant nosocomial pathogens causing urinary tract, blood stream, sterile site and wound infections. In hospitals, enterococci can be transmitted through poor infection control practices and can give rise to a wide variety of infections usually in patients with co-morbidities. The two main species causing infections in humans are *Enterococcus faecalis* and *Enterococcus faecium* with only a very small number of other species being isolated from clinical specimens.

In the 1980s, enterococci were generally susceptible to amoxycillin and vancomycin. Since then *E. faecium* has become increasingly resistant to ampicillin/amoxycillin making vancomycin the treatment of choice for severe infections caused by this organism. The first vancomycin resistant enterococci (VRE) were described in the United Kingdom and Europe in 1988[1] and in the United States of America (USA) in the early 1990s.[2] The first VRE was reported in Australia in 1994[3] and a report on the emergence and epidemiology of VRE in Australia was described in 1998 when 69 isolates were documented.[4]

Multilocus sequence typing (MLST) of *E. faecium* has revealed that clonal complex (CC) 17 strains have become predominant in hospitals in many countries and are characterised by ampicillin resistance and the presence of several genetic elements (e.g. *esp* and *hyl*) not present in colonising variants in humans and animals.[5–9] There is some evidence that this additional genomic content assists in adaptation to the hospital environment and the ability to spread, therefore when CC17 strains acquired the *vanA* or *vanB* gene encoding vancomycin resistance, they were already primed for transmission in the hospital setting.

Prevalence and incidence rates for VRE in Australian hospitals are not routinely collected although there have been reports of individual hospital outbreaks of VRE infections and associated colonisation of other patients.[9–13] The clinical impact of vancomycin resistance in enterococci has been reported to include increases in mortality, length of stay and hospital costs.[14,15] Serious infections caused by vancomycin-resistant *E. faecium* are difficult to treat, and rely on recently introduced antimicrobials such as linezolid, quinupristin-dalfopristin, tigecycline and daptomycin which are not approved for all indications. Further complicating the treatment of infections caused by VRE are reports of isolates that are resistant even to these newer agents.[16,17]

It is important to have an understanding of the occurrence of enterococcal infection and antibiotic resistance in Australia to guide infection control practices, antibiotic prescribing policies and drug regulatory matters.

The objective of the 2010 surveillance program was to determine the proportion of clinical isolates of *Enterococcus* species demonstrating antimicrobial resistance with particular emphasis on:

1. assessing susceptibility to ampicillin;
2. assessing susceptibility to glycopeptides; and
3. assessing changes in resistance patterns over time using data collected in previous Australian AGAR surveys,
4. determining which VRE clones are circulating within Australia.

The Australian Group on Antimicrobial Resistance (AGAR) commenced surveillance of antimicrobial resistance in *Enterococcus* species in 1995. Similar surveys were conducted in 1999, 2003, 2005, 2007 and 2009 (www.agargroup.org).

Methods

Fifteen laboratories from all mainland Australian states and the Australian Capital Territory participated in the 2010 AGAR survey *Enterococcus*. To ensure institutional anonymity the New South Wales and the Australian Capital Territory data were combined.

From 1 January to 30 June 2010 each laboratory collected up to 100 consecutive clinically significant isolates of enterococci. Only 1 isolate per patient was tested unless subsequent isolates had a different antibiogram to the original isolate.

Species identification

All isolates were tested for pyrrolidonyl arylamidase with optional testing for growth in 6.5% sodium chloride, esculin hydrolysis in the presence of bile, Group D antigen and growth at 45°C. Isolates were identified to species level by either API® 20S (bioMérieux, Marcy l'Etoile, France), Vitek® 2 (bioMérieux, Marcy l'Etoile, France), Phoenix™ (BD, New Jersey, USA), polymerase chain reaction (PCR), or conventional biochemical tests. If biochemical testing was performed, the minimum tests necessary for identification were: motility, pigment production, methyl-α-D-glucopyranoside, fermentation of 1% raffinose, 1% arabinose, 1% xylose and utilisation of pyruvate.

Susceptibility methodology

Participating laboratories performed antimicrobial susceptibility tests according to each laboratory's routine standardised methodology (Clinical and Laboratory Standards Institute (CLSI) disc diffusion, Vitek® 2, Phoenix™, agar dilution or Etest® (bioMérieux, Marcy l'Etoile, France)). Ampicillin and vancomycin were tested by all laboratories. Vancomycin resistance was confirmed by PCR. Overall, 1,378 (99.4%) isolates were screened for high level gentamicin resistance, 932 (67.2%) were tested against linezolid, 503 (36.3%) were tested against quinupristin/dalfopristin and 146 (10.5%) were screened for high level streptomycin resistance. CLSI breakpoints were utilised for all antimicrobials.[18] Isolates with an intermediate and resistant category have been classified as non-susceptible.

Of the 178 invasive isolates, 116 (65.2%) were tested for ß-lactamase production using a chromogenic cephalosporin nitrocefin.

Epidemiological typing of vancomycin resistant enterococci

Pulsed-field gel electrophoresis (PFGE) of *SmaI*-digested DNA agarose plugs was performed as previously described on all VRE isolates.[19] MLST was performed as previously described on a representative of each PFGE pulsotype of vancomycin resistant *E. faecium*.[20]

Statistical analysis

The difference between proportions was tested using Chi-square test with alpha set at the 5% level and Fisher's exact test for 95% confidence limits (GraphPad® Prism Software).

Results

Both public (n=13) and private (n=2) laboratories participated in the survey. Participants included New South Wales (n=3), the Australian Capital Territory (n=1), Queensland (n=4), Victoria (n=1), South Australia (n=3), and Western Australia (n=3). In 2010 there were 1,386 isolates from 15 institutions (Table 1). *E. faecalis* was the most frequently isolated species (86.7%) followed by *E. faecium* (12.3%) (Table 2).

The majority of isolates (70.9%) were from the urinary tract (Table 3). They were predominately *E. faecalis* (91.3%). Invasive (blood, cerebrospinal fluid (CSF) and sterile body cavity) isolates comprised 12.8% of the total number of isolates collected. *E. faecium* was disproportionately represented in the invasive group (28.7%). Of the *E. faecalis* isolates, 9.9% were invasive compared with 30.0% of *E. faecium* isolates.

Susceptibility

Resistance to ampicillin was common in the *E. faecium* isolates (Table 4). Resistance in *E. faecium* was due to penicillin binding protein changes. No ß-lactamase positive *E. faecium* were detected amongst the subset (30/51, 59%) of invasive isolates tested. Resistance in invasive isolates was lower than for non-invasive isolates (72.9% and 90.7% respectively, P=0.004). Ampicillin resistance was not detected for *E. faecalis* and none of the 81 invasive isolates tested for ß-lactamase were positive.

Trend data for *E. faecium* show that from 1995 to 1999, there was an increase in ampicillin resistance (P=0.002) with a plateau from 1999 to 2005 (Figure 1). Between 2005 and 2010, resistance has once again increased significantly (P=0.005). The gap between resistance in non-invasive versus invasive isolates narrowed over time, however in

Table 1: *Enterococcus* isolates in Australia, 2010, by region

Region	Number of institutions	Isolates	%
NSW/ACT	4	380	27.4
Qld	4	400	28.9
SA	3	207	14.9
Vic	1	100	7.2
WA	3	299	21.6
Total	15	1,386	100.0

Table 2: *Enterococcus* species isolated in Australia, 2010, by region

Region	*E. faecalis*	*E. faecium*	Other spp. or unspeciated	Total
NSW/ACT	334	41	5	380
Qld	381	18	1	400
SA	145	57	5	207
Vic	76	23	1	100
WA	265	31	3	299
Total	1,201 (86.7%)	170 (12.3%)	15 (1.1%)	1,386

Table 3: *Enterococcus* species isolated in Australia, 2010, by source

Source	*E. faecalis*	*E. faecium*	Other spp. or unspeciated	Total
Urine	897	82	3	982 (70.9%)
Wound	173	37	4	214 (15.4%)
Blood/CSF	77	34	5	116 (8.4%)
Sterile body cavity	42	17	3	62 (4.5%)
Other	12	0	0	12 (0.9%)
Total	1,201	170	15	1,386
Invasive*	119	51	8	178 (12.8%)
Non-invasive	1,082	119	7	1,208 (87.2%)

CSF Cerebrospinal fluid

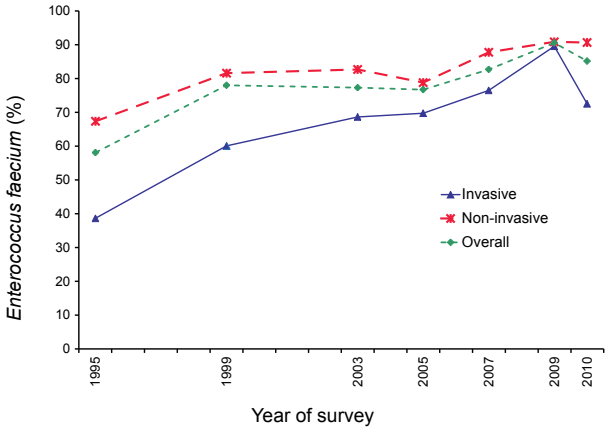
* Blood/cerebrospinal fluid/sterile body cavity

2010 there was a reversal of this trend with rates of resistance in invasive isolates falling significantly (P=0.04) compared to 2009 levels.

Vancomycin non-susceptibility was uncommon in *E. faecalis* (0.5%) (Table 5). Of the 6 non-susceptible *E. faecalis*, two harboured the *vanB* gene and four did not possess either *vanA* or *vanB*.

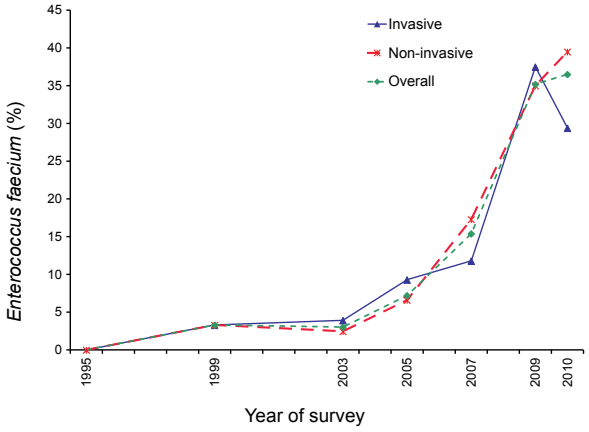
A total of 36.5% of *E. faecium* were vancomycin non-susceptible; a similar proportion to the 2009 survey (35.2%) but more than double that of the 2007 survey (15.4%, P<0.0001) (Figure 2). Vancomycin non-susceptible *E. faecium* were detected in all regions except Western Australia. Vancomycin non-susceptibility in the other regions ranged from 16.7% in Queensland to 54.4% in South Australia (Table 5). All of the vancomycin non-susceptible *E. faecium* were confirmed as VRE by PCR and were predominantly of the *vanB* genotype (61/62, 98.4%). In 2010, more than one third of urine, wound and blood *E. faecium* were vancomycin resistant. Trend data for *E. faecium* show there has been a marked increase in vancomycin resistance since 1995 (Figure 2). Vancomycin resistant *E. faecium* have occurred in all 5 regions over the 6 survey periods, with all regions except Western Australia showing increases in VRE over time (Figure 3).

Figure 1: Percentage of *Enterococcus faecium* resistant to ampicillin, by survey year



1995: invasive n=26, non-invasive n= 55, overall n=81.
1999: invasive n=30, non-invasive n= 152, overall n=182.
2003: invasive n=51, non-invasive n= 81, overall n=132.
2005: invasive n=43, non-invasive n= 137, overall n=180.
2007: invasive n=51, non-invasive n= 98, overall n=156.
2009: invasive n=48, non-invasive n= 165, overall n=213.
2010: invasive n=51, non-invasive n= 119, overall n=170

Figure 2: Percentage of *Enterococcus faecium* non-susceptible to vancomycin, by survey year



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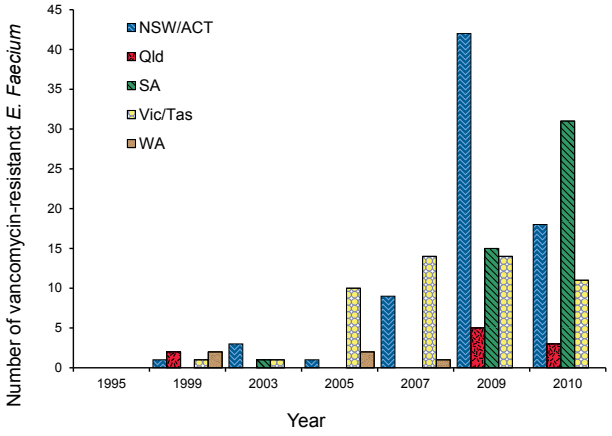
1999: invasive n=30, non-invasive n= 152, overall n=182.
2003: invasive n=51, non-invasive n= 81, overall n=132.
2005: invasive n=43, non-invasive n= 137, overall n=180.
2007: invasive n=51, non-invasive n= 98, overall n=156.
2009: invasive n=48, non-invasive n= 165, overall n=213.
2010: invasive n=51, non-invasive n= 119, overall n=170

Figure 3: Regional location of vancomycin-resistant *Enterococcus faecium*, by survey year



* Tasmania did not contribute isolates in 2009 or 2010.

High level gentamicin (HLG) resistance was seen in *E. faecalis* (34.1%) and *E. faecium* (66.1%) (Table 6). Trend data (Figures 4 and 5) show significant increases for *E. faecium* from 1995 to 1999 (P<0.001) and again from 2003 to 2010 (P<0.0001). The increase from 2003 to 2010 was driven by resistance in non-invasive isolates as rates of resistance remained stable in invasive isolates during that time period despite year-to-year fluctuations (Figure 4) (P=0.09). HLG resistance in *E. faecalis* invasive and non-invasive isolates continued to increase until 2005 and then stabilised.

In this survey, high level streptomycin resistance (HLS) was tested only in New South Wales/Australian Capital Territory and South Australia. HLS resistance is more common for *E. faecium* than *E. faecalis* (Table 7), similar to HLG resistance. The trend from 1995 to 2010 for *E. faecium* was for relatively stable resistance despite year to year fluctuations (Figures 6 and 7). In *E. faecalis*,

Table 4: Number of ampicillin resistant *Enterococcus* species isolated in Australia, 2010, by region

	NSW/ACT			Qld			SA			Vic			WA			Aus		
	n	N	%	n	N	%	n	N	%	n	N	%	n	N	%	n	N	%
E. faecalis all	0	334	0.0	0	381	0.0	0	145	0.0	0	76	0.0	0	265	0.0	0	1,201	0.0
Invasive	0	46	0.0	0	14	0.0	0	22	0.0	0	8	0.0	0	18	0.0	0	108	0.0
E. faecium all	34	41	82.9	17	18	94.4	51	57	89.5	19	23	82.6	23	31	77.4	145	170	85.3
Invasive	12	18	66.7	1	2	50.0	17	20	85.0	3	3	100.0	4	8	50.0	37	51	72.5

Table 5: Number of vancomycin non-susceptible *Enterococcus* species isolated in Australia, 2010, by region

	NSW/ACT			Qld			SA			Vic			WA			Aus		
	n	N	%	n	N	%	n	N	%	n	N	%	n	N	%	n	N	%
E. faecalis all	2	334	0.6	1	381	0.3	0	145	0.0	2	76	2.6	1	265	0.4	6	1,201	0.5
Invasive	2	41	4.9	0	12	0.0	0	40	0.0	0	5	0.0	1	21	4.8	3	119	2.5
E. faecium all	18	41	43.9	3	18	16.7	31	57	54.4	10	23	43.5	0	31	0.0	62	170	36.5
Invasive	6	18	33.3	0	2	0.0	8	20	40.0	1	3	33.3	0	8	0.0	15	51	29.4

Table 6: Number of high level gentamicin resistant *Enterococcus* species isolated in Australia, 2010, by region

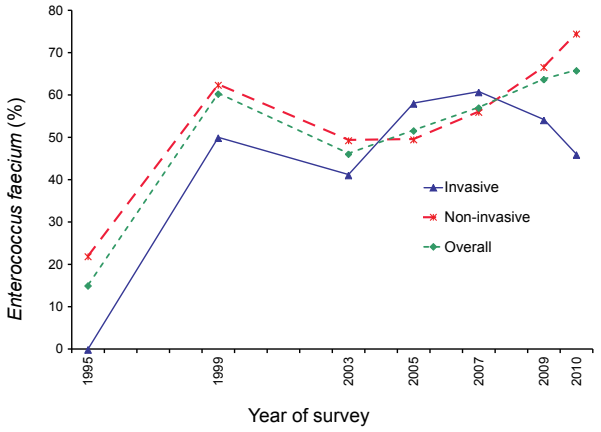
	NSW/ACT			Qld			SA			Vic			WA			Aus		
	n	N	%	n	N	%	n	N	%	n	N	%	n	N	%	n	N	%
E. faecalis all	141	334	42.2	150	381	39.4	38	142	26.8	18	76	23.7	62	265	23.4	409	1,198	34.1
Invasive	11	41	26.8	4	12	33.3	15	37	40.5	1	5	20.0	12	21	57.1	43	116	37.1
E. faecium all	30	41	73.2	16	18	88.9	30	52	57.7	16	23	69.6	17	31	54.8	109	165	66.1
Invasive	7	18	38.9	1	2	50.0	10	19	52.6	2	3	66.7	3	8	37.5	23	50	46.0

the HLS decreased significantly from 1995 to 2003 but has been relatively stable since then with lower rates of expression than HLG (Figures 5 and 7).

Linezolid non-susceptibility was present in 5.8% of *E. faecalis* (up from 4.0% in 2009) and in 0.9% of *E. faecium* (down from 2.1% in 2009) (Table 8). Forty-six of the 48 non-susceptible isolates had an minimum inhibitory concentration (MIC) in the intermediate resistant category; only two were classified as resistant (MIC ≥8 mg/L). The 2 resistant isolates were *E. faecalis* from Queensland.

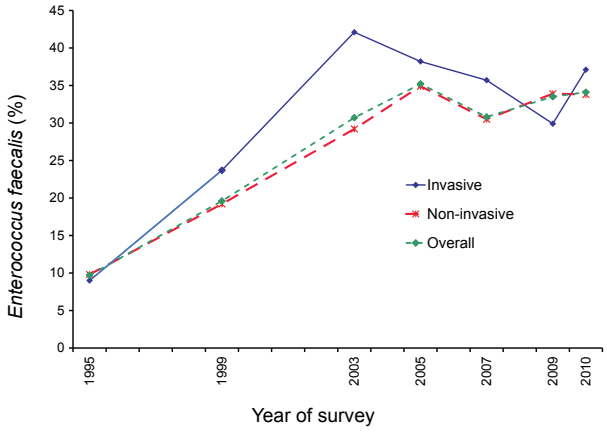
E. faecalis are intrinsically resistant to quinupristin/dalfopristin. Only 9.4% of the *E. faecium* were non-susceptible (down from 21.9% in 2009) with

Figure 4: Percentage of *Enterococcus faecium* resistant to high-level gentamicin, by survey year



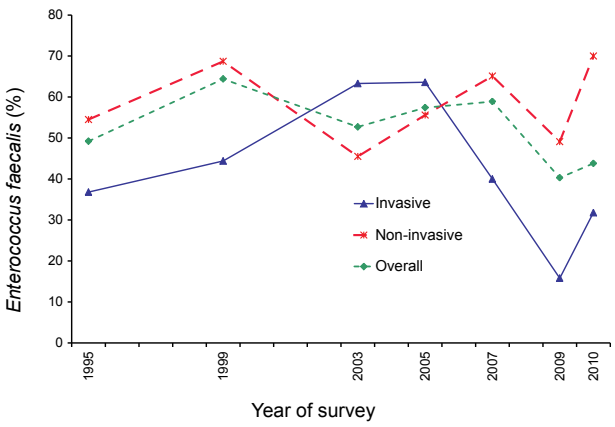
1995: invasive n=23, non-invasive n= 50, overall n=73.
1999: invasive n=30, non-invasive n= 152, overall n=182.
2003: invasive n=51, non-invasive n= 81, overall n=132.
2005: invasive n=43, non-invasive n= 137, overall n=180.
2007: invasive n=51, non-invasive n= 98, overall n=156.
2009: invasive n=48, non-invasive n= 165, overall n=213.
2010: invasive n=50, non-invasive n= 115, overall n=165.

Figure 5: Percentage of *Enterococcus faecalis* resistant to high-level gentamicin, by survey year



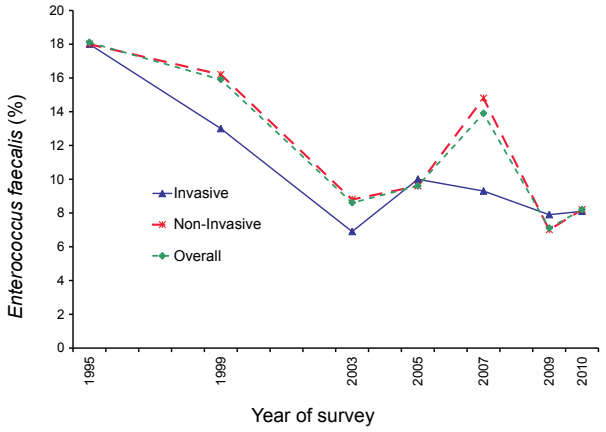
1995: invasive n=100, non-invasive n= 1109, overall n=1,211.
1999: invasive n=135, non-invasive n= 1,442, overall n=1577.
2003: invasive n=190, non-invasive n=1,432, overall n=1,622.
2005: invasive n=170, non-invasive n= 1,816, overall n=1,986.
2007: invasive n=143, non-invasive n= 1,333, overall n=1,520.
2009: invasive n=107, non-invasive n= 1005, overall n=1,112.
2010: invasive n=116, non-invasive n= 1082, overall n=1,198

Figure 6: Percentage of *Enterococcus faecium* resistant to high-level streptomycin, by survey year



1995: invasive n=19, non-invasive n= 44, overall n=63.
1999: invasive n=18, non-invasive n= 83, overall n=101.
2003: invasive n=30, non-invasive n= 44, overall n=74.
2005: invasive n=22, non-invasive n= 72, overall n=94.
2007: invasive n=25, non-invasive n= 43, overall n=73.
2009: invasive n=19, non-invasive n=53, overall n=72.
2010: invasive n=22, non-invasive n=10, overall n=32

Figure 7: Percentage of *Enterococcus faecalis* resistant to high-level streptomycin, by survey year



1995: invasive n=61, non-invasive n= 916, overall n=979.
1999: invasive n=92, non-invasive n= 916, overall n=1008.
2003: invasive n=102, non-invasive n=715, overall n=817.
2005: invasive n=80, non-invasive n= 1012, overall n=1092.
2007: invasive n=197, non-invasive n= 783, overall n=913.
2009: invasive n=38, non-invasive n= 229, overall n=267.
2010: invasive n=37, non-invasive n= 73, overall n=110.

four of the five non-susceptible isolates having an MIC in the resistant range (MIC >2 mg/L). All quinupristin/dalfopristin non-susceptible cases were identified in isolates originating in New South Wales/Australian Capital Territory, as was the case in 2007 and 2009 (Table 9).

Cross resistance to other agents was examined in vancomycin resistant isolates of enterococci (Table 10). Resistance to ampicillin and high levels of gentamicin was more common in vancomycin resistant *E. faecium*. Resistance to high levels of streptomycin, quinupristin/dalfopristin and linezolid was similar for VRE and non-VRE ($P>0.05$).

Table 7: Number of high level streptomycin resistant *Enterococcus* isolated in Australia, 2010, by region

	NSW/ACT			Qld	SA			Vic	WA	Aus		
	n	N	%		n	N	%			n	N	%
E. faecalis all	6	73	8.2	–	3	37	8.1	–	–	9	110	8.2
Invasive	1	9	11.1	–	2	28	7.1	–	–	3	37	8.1
E. faecium all	2	7	28.6	–	12	25	48.0	–	–	14	32	43.8
Invasive	1	5	20.0	–	6	17	35.3	–	–	7	22	31.8

Table 8: Number of linezolid non-susceptible *Enterococcus* isolated in Australia, 2010, by region

	NSW/ACT			Qld			SA			Vic	WA			Aus		
	n	N	%	n	N	%	n	N	%		n	N	%	n	N	%
E. faecalis all	18	334	5.4	28	341	7.3	1	90	1.1	–	0	5	0.0	47	810	5.8
Invasive	2	41	4.9	0	12	0.0	1	32	3.1	–	0	5	0.0	3	90	3.3
E. faecium all	1	41	2.4	0	18	0.0	0	52	0.0	–	0	2	0.0	1	113	0.9
Invasive	1	18	5.6	0	2	0.0	0	18	0.0	–	0	1	0.0	1	39	2.6

Table 9: Number of quinupristin/dalfopristin non-susceptible *Enterococcus* isolated in Australia, 2010, by region

	NSW/ACT			Qld			SA			Vic	WA			Aus		
	n	N	%	n	N	%	n	N	%		n	N	%	n	N	%
E. faecalis all	246	258	95.3	154	177	87.0	2	3	66.7	–	5	5	100.0	407	443	91.9
Invasive	33	34	97.1	6	6	100	0	1	0.0	–	5	5	100.0	44	46	95.7
E. faecium all	5	33	28.1	0	16	0.0	0	2	0.0	–	0	2	0.0	5	53	9.4
Invasive	4	15	26.7	0	2	0.0	–			–	0	1	0.0	4	18	22.2

Table 10: Cross resistant *Enterococcus* isolated in Australia, 2010

		Ampicillin			Gentamicin			Streptomycin			Linezolid			Quinupristin/ dalfopristin		
		n	N	%	n	N	%	n	N	%	n	N	%	n	N	%
E. faecalis	Not VRE	0	1,198	0.0	407	1,195	34.1	9	110	8.2	47	809	5.8	407	443	91.9
	VRE	0	3	0.0	2	3	66.7	–			0	1	0.0	–		
E. faecium	Not VRE	82	107	76.6	58	107	54.2	5	16	31.3	1	62	1.6	4	34	11.8
	VRE	63	63	100.0	51	58	87.9	9	16	56.3	0	51	0.0	1	19	5.3

VRE Vancomycin resistant enterococci

Vancomycin resistant enterococci characterisation

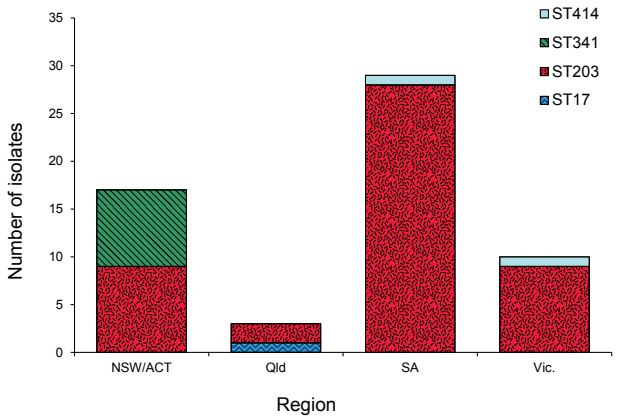
Three (100%) *vanB E. faecalis*, 1 (100%) *vanA E. faecium* and 59/62 (95%) *vanB E. faecium* isolates were available for molecular typing.

Two of the *vanB E. faecalis* were classified as pulsotype A and one was classified as pulsotype B (Table 11). The *vanA E. faecium* was a pulsotype C and sequence type (ST) 117. Six pulsotypes and 4 STs were identified in the *vanB E. faecium*. ST203 was the most common ST (comprising 81% of *vanB E. faecium*) and was found in all regions that reported VRE. ST341 was found only in New South Wales/Australian Capital Territory, ST414 only in South Australia and Victoria and ST17 only in Queensland (Figure 8). The *E. faecium* isolates belonged to CC 17.

Discussion

It is clear from this study and the examination of trends over the last 15 years that antimicrobial resistance has increased significantly in *E. faecium*.

Figure 8: Distribution of vanB *Enterococcus faecium* multi-locus sequence types, by region



Treatment options for this species are becoming ever more limited as resistance to ampicillin and other penicillins is now very frequent, and glycopeptide resistance is increasing. In some instances only expensive and/or potentially toxic treatment options such as linezolid, quinupristin-dalfopristin, tigecycline or daptomycin are available.

Ampicillin resistance in *E. faecuim* is the result of changes in penicillin-binding proteins. This is also true for most isolates of *E. faecalis*, although ß-lactamase production has been seen rarely (3 known instances in Australia in the last 2 decades).[21] This survey has shown that ampicillin resistance is now usual in *E. faecium* but is rare in *E. faecalis*. Ampicillin resistance in enterococci presents considerable challenges when infections are serious, as the isolates will not be susceptible to any ß-lactam antibiotic, and the drug of choice becomes vancomycin, which is only slowly bactericidal. Further, for endocarditis the combination of vancomycin with an aminoglycoside creates significant toxicity problems.

Unfortunately vancomycin resistance in enterococci is increasing in Australia particularly over the past 5 years. It has been seen in all states and territories although rates in each region vary considerably. It is widely recognised that rates of colonisation far exceed the rates of infection with VRE, and thus the amount of VRE seen in this survey does not truly reflect the size of the VRE reservoir. The survey results are also consistent with the previous Australian experience that the dominant type of resistance is encoded by the *vanB* complex[4,22] in contrast with the situation in Europe and the USA where *vanA* dominates. Vancomycin-resistant isolates causing serious infection are very challenging to treat. The choices are linezolid, quinupristin-dalfopristin, tigecycline and daptomycin. Each of these agents presents its own challenges for treatment.

Table 11: Molecular characterisation of vancomycin-resistant enterococci isolated in Australia, 2010

van Gene	Species	PFGE	MLST	NSW/ACT	Qld	SA	Vic
vanB	*E. faecalis*	A	n.d.				2
vanB	*E. faecalis*	B	n.d.			1	
vanA	*E. faecium*	C	ST117	1			
vanB	*E. faecium*	D	ST203	5	1	28	9
vanB	*E. faecium*	E	ST203	2	1		
vanB	*E. faecium*	F	ST203	2			
vanB	*E. faecium*	G	ST341	8			
vanB	*E. faecium*	H	ST414			1	1
vanB	*E. faecium*	I	ST17		1		
Total				18	3	30	12

High-level resistance to gentamicin has increased in recent years after apparently reaching a plateau in the early 2000s. This greatly compromises the ability to treat enterococcal endocarditis effectively.

Molecular characterisation of the VRE isolates in this study has revealed that *E. faecium* belonging to CC17 are now established in Australia. CC17, including ST203 and ST414 both found in this study are considered to be hospital-associated clones and have been responsible for outbreaks in several countries including Australia.[9,23,24] Containing additional genetic content thought to assist in survival and spread in the hospital environment, CC17 poses a challenge for hospital infection control as standard measures may not be enough to control spread in the long term. Extensive screening of patients, confinement of colonised or infected patients, antimicrobial restrictions and additional cleaning protocols are often required to reduce VRE in the hospital environment.[7,10,24,25] In addition, VRE belonging to CC17 are causing severe infections, in particular bacteraemia, in increasing numbers.[9,23]

The data provided by this survey will be useful in informing microbiologists, infectious diseases physicians and infection control practitioners about the increasing importance of VRE in Australia. It will help to guide prescribers treating presumptive enterococcal infections in empirical choices; e.g. ampicillin/amoxycillin still being active against the vast majority of isolates of *E. faecalis* when treating infections caused by this organism. Finally, the data will assist regulators and the pharmaceutical industry on the growing importance of VRE in Australia, and guide decision makers about controls that might be required on the prescribing of reserve antibiotics.

Limitations of the study

The enterococci in this study were tested against a limited range of antimicrobials. In part, this was driven by the presence of intrinsic resistances in this genus. Enterococci are intrinsically resistant to cephalosporins, macrolides, lincosamides and conventional therapeutic levels of aminoglycosides when used alone. Other agents which are usually active against enterococci in urinary tract infection, including fluoroquinolones and nitrofurantoin, were not examined, largely because few clinical treatment problems have been encountered up to now with enterococcal urinary tract infection.

It is likely that the number of wound isolates in this study under-represents the true proportion, as it is common for microbiology laboratories not to proceed with identification or susceptibility testing of enterococci when they are found in mixed cultures from wound infections.

Only a maximum of 100 isolates were collected per institution, therefore only a portion of actual clinical isolates are represented.

There have been changes in participating laboratories in the AGAR Enterococcus surveys over time from 1995 through to 2010 with the more recent inclusion of a number of private pathology laboratories. This may have influenced trend data.

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Australian Group on Antimicrobial Resistance Hospital-onset *Staphylococcus aureus* Surveillance Programme annual report, 2011

Geoffrey W Coombs, Graeme R Nimmo, Julie C Pearson, Peter J Collignon, Jan M Bell, Mary-Louise McLaws, Keryn J Christiansen, John D Turnidge for the Australian Group on Antimicrobial Resistance

Abstract

In 2011, the Australian Group on Antimicrobial Resistance (AGAR) conducted a period-prevalence survey of clinical *Staphylococcus aureus* isolated from hospital inpatients. Twenty-nine microbiology laboratories from all states and mainland territories participated. Specimens were collected more than 48 hours post-admission. Isolates were tested by Vitek2® antimicrobial susceptibility card (AST-P612 card). Nationally, the proportion of *S. aureus* that were methicillin-resistant *S. aureus* (MRSA) was 30.3%; ranging from 19.9% in Western Australia to 36.8% in New South Wales/Australian Capital Territory. Resistance to the non-ß-lactam antimicrobials was common except for rifampicin, fusidic acid, high-level mupirocin and daptomycin. No resistance was detected for vancomycin, teicoplanin or linezolid. Antibiotic resistance in methicillin susceptible *S. aureus* (MSSA) was rare apart from erythromycin (13.2%) and there was no resistance to vancomycin, teicoplanin or linezolid. Inducible clindamycin resistance was the norm for erythromycin resistant, clindamycin intermediate/susceptible *S. aureus* in Australia with 90.6% of MRSA and 83.1% of MSSA with this phenotype having a positive double disc diffusion test (D-test). The proportion of *S. aureus* characterised as being healthcare-associated MRSA (HA-MRSA) was 18.2%, ranging from 4.5% in Western Australia to 28.0% in New South Wales/Australian Capital Territory. Four HA-MRSA clones were characterised and 98.8% of HA-MRSA isolates were classified as either ST22-IV [2B] (EMRSA-15) or ST239-III [3A] (Aus-2/3 EMRSA). Multiclonal community-associated MRSA (CA-MRSA) accounted for 11.7% of all *S. aureus*. In Australia, regional variation in resistance is due to the differential distribution of MRSA clones between regions, particularly for the major HA-MRSA clone, ST239-III [3A] (Aus-2/3 EMRSA), which is resistant to multiple non-ß-lactam antimicrobials. *Commun Dis Intell* 2013;37(3):E210–E218.

Keywords: antimicrobial resistance surveillance; *Staphylococcus aureus;* hospital-onset infections; methicillin susceptible, methicillin resistant

Introduction

Staphylococcus aureus is a major pathogen in the hospital environment, causing a wide variety of infections that are associated with considerable mortality. Several studies have indicated that mortality is higher for patients infected with methicillin-resistant *S. aureus* (MRSA) than methicillin-susceptible *S. aureus* (MSSA)[1–4] and that MRSA infections are associated with increased costs due to longer hospital stays and the need for treatment with costly antimicrobials.[5–7]

The Australian Group on Antimicrobial Resistance (AGAR) has undertaken antimicrobial resistance period-prevalence surveys in Australia since 1986.[8] Hospital inpatient surveys have been conducted biennially since 2005.[9] The objectives of the hospital inpatient survey was to determine the prevalence of antimicrobial resistance in clinical isolates of *S. aureus* throughout Australia in hospital inpatients admitted for 48 hours or more and to describe the molecular epidemiology of the MRSA isolates.

The findings of the 2011 AGAR hospital inpatients survey are presented in this report.

Methods

Twenty-nine laboratories from all states and territories participated in the 2011 *S. aureus* AGAR survey. In the Northern Territory and the Australian Capital Territory only 1 laboratory participated in each region and in Tasmania only 2 laboratories participated. To ensure institutional anonymity data were combined as follows: New South Wales with the Australian Capital Territory, Victoria with Tasmania, and Queensland with the Northern Territory.

From 1 July to 30 November 2011 each laboratory collected up to 100 consecutive *S. aureus* isolates from hospital inpatients (hospital stay greater than 48 hours at the time of specimen collection). Only 1 isolate per patient was tested. Each *S. aureus* isolate was judged to come from a potentially infected

site. Each hospital laboratory only collected from one institution. The three private laboratories collected from the multiple institutions that they serviced.

Susceptibility methodology

All isolates were tested using the Vitek2® antimicrobial susceptibility card (AST-P612). All isolates with a penicillin minimum inhibitory concentration of ≤0.125 mg/L were screened for the presence of ß-lactamase using nitrocefin discs. To detect inducible clindamycin resistance a double disc diffusion test (D-test) was performed on all erythromycin resistant and clindamycin intermediate or susceptible *S. aureus* isolates. Clinical and Laboratory Standards Institute breakpoints[10] were utilised for all antimicrobials excluding fusidic acid (http://www.eucast.org/clinical_breakpoints/). Isolates with an MIC in the intermediate resistance category have been called resistant in this report.

Epidemiological typing of methicillin-resistant *Staphylococcus aureus*

Of the 713 MRSA identified, 703 (98.6%) were referred to the Australian Collaborating Centre for *Enterococcus* and *Staphylococcus* Species (ACCESS) Typing and Research for epidemiological typing.

Electrophoresis of chromosomal DNA using a contour-clamped homogeneous electric field DRIII System (Bio-Rad Laboratories Pty Ltd) was performed as previously described[11] on all MRSA isolates. Multilocus sequence typing (MLST) and SCC*mec* typing was performed as previously described[12–14] on selected MRSA isolates.

PCR for the detection of Panton–Valentine leucocidin (PVL) determinants was performed as previously described[15] on all MRSA isolates.

Methicillin-resistant *Staphylococcus aureus* nomenclature

MRSA clones were defined by the combination of the MLST and the SCC*mec* type.[16] Clones were reported with their ST and SCC*mec* type followed by their colloquial name in parenthesis; e.g. ST22-IV [2B] (EMRSA-15). Clones were classified into 2 groups on the basis of previously published evidence; those implicated in healthcare-associated infection (HA-MRSA) and those implicated in community-associated infection (CA-MRSA).

Clones that diverged at no more than one of the 7 MLST loci were considered to belong to the same clonal complex. Double locus variants were included in the same clonal complex if the linking single locus variant was present in the MLST database (http://www.mlst.net/).

Statistical analysis

Differences between proportions were tested using a Chi-square test with alpha set at 5% and Fisher's exact test for 95% confidence limits (GraphPad® Prism Software). Relative risk and 95% confidence intervals were calculated using VassarStats (http://vassarstats.net).

Results

There were 2,357 isolates included in the survey (Table 1). Skin and soft tissue infection specimens contributed the majority of isolates (70.5%) followed by respiratory specimens (17.1%). Blood culture isolates contributed 6.5% of the total. Significantly ($P<0.0001$) more isolates caused non-invasive (91.3%) than invasive (8.7%) infections (Table 2).

Table 1: *Staphylococcus aureus* isolates, Australia, 2011, by region

Region	Number of institutions	Number of isolates	Per cent of total
NSW/ACT	8	639	27.1
Qld/NT	7	591	25.1
SA	3	254	10.8
Vic/Tas	7	541	22.9
WA	4	332	14.1
Total	29	2,357	100.0

Table 2: Site of *Staphylococcus aureus* isolates, Australia, 2011

Specimen site	Number of isolates	Per cent of total	95%CI
Skin and soft tissue	1,661	70.5	68.6–72.3
Respiratory	404	17.1	15.6–18.7
Blood	153	6.5	5.5–7.6
Urine	88	3.7	3.0–4.6
Sterile body cavity	49	2.1	1.5–2.7
Cerebrospinal fluid	2	0.1	0.01–0.3
Total	2,357	100.0	
Invasive*	204	8.7	7.5–9.9
Non-invasive	2,153	91.3	90.1–92.4

* Blood/cerebrospinal fluid/sterile body cavity

Table 3: Proportion of *Staphylococcus aureus* that were methicillin-resistant, Australia, 2011, by region and source

Region	All isolates			Invasive isolates*			Non-invasive isolates		
	n/N	%	95%CI	n/N	%	95%CI	n/N	%	95%CI
NSW/ACT	235/639	36.8	33.1–40.6	29/65	44.6	33.2–56.7	206/574	35.9	32.1–39.9
Qld/NT	180/591	30.5	26.9–34.3	11/41	26.8	15.7–41.9	169/550	30.7	27.0–34.7
SA	55/254	21.7	17.0–27.1	10/28	35.7	20.7–54.2	45/226	19.9	15.2–25.6
Vic/Tas	177/541	32.7	28.9–36.8	9/42	21.4	11.7–35.9	168/499	33.7	29.7–37.9
WA	66/332	19.9	15.9–24.5	4/28	14.3	5.7–31.5	62/304	20.4	16.2–25.3
Aus	713/2,357	30.3	28.4–32.1	63/204	30.9	24.9–37.5	650/2,153	30.2	28.3–32.2

* Blood/cerebrospinal fluid/sterile body cavity

Methicillin-resistant *Staphylococcus aureus*

The proportion of *S. aureus* isolates that were MRSA was 30.3% nationally (Table 3) with significantly different ($P<0.0001$) proportions across Australia ranging from 19.9% in Western Australia to 36.8% in New South Wales/Australian Capital Territory. The proportion of *S. aureus* isolates that were MRSA at each institution ranged from 7% to 56%. The proportion of invasive *S. aureus* that were MRSA (30.9%) was not significantly higher than for non-invasive isolates (30.2%) ($P=1$). The proportion of MRSA isolated in the 5 sites of infection was similar ($P=0.24$) with MRSA ranging from 29.0% in skin and soft tissue infections to 36.4% in urine (Table 4). MRSA was not isolated from cerebrospinal fluid specimens.

Amongst the MRSA isolates, resistance to the non-ß-lactam antimicrobials was common except for fusidic acid, rifampicin, high-level resistance to mupirocin and daptomycin, where resistance was below 4% nationally (Table 5). Resistance was not detected for vancomycin, teicoplanin or linezolid. Resistance levels varied significantly between regions with Victoria/Tasmania having the highest proportions for the top 6 antimicrobials.

Table 4: Proportion of *Staphylococcus aureus* that were methicillin-resistant, by specimen type

Site of infection	All Isolates		
	n/N	%	95%CI
Skin and soft tissue	482/1,661	29.0	26.8–31.3
Respiratory	136/404	33.7	29.1–38.5
Blood/cerebrospinal fluid	46/155	29.7	22.6–37.5
Urine	32/88	36.4	26.4–46.7
Sterile body cavity	17/49	34.7	17.0–49.6

Table 5: Number and proportion of methicillin-resistant *Staphylococcus aureus* isolates resistant to the non-ß-lactam antimicrobials, Australia, 2011, by region

Drug	NSW/ACT (n=235)		Qld/NT (n=180)		SA (n=55)		Vic/Tas (n=177)		WA (n=66)		Aus (n=713)		Differences across regions	
	n	%	n	%	n	%	n	%	n	%	n	%	X^2	*P*
Erythromycin	164	69.8	103	57.2	28	50.9	131	74.0	30	45.5	456	64.0	28.63	<0.0001
Clindamycin*	89	37.9	43	23.9	10	18.2	68	38.4	2	3.0	212	29.7	42.82	<0.0001
Tetracycline	91	38.7	59	32.8	12	21.8	76	42.9	1	1.5	239	33.5	43.66	<0.0001
Co-trimoxazole	87	37.0	50	27.8	11	20.0	69	39.0	2	3.0	219	30.7	37.54	<0.0001
Ciprofloxacin	188	80.0	85	47.2	34	61.8	150	84.7	20	30.3	477	66.9	115.7	<0.0001
Gentamicin	85	36.2	58	32.2	7	12.7	66	37.3	1	1.5	217	30.4	42.07	<0.0001
Fusidic acid	5	2.1	12	6.7	1	1.8	6	3.4	2	3.0	26	3.6	6.844	0.14
Rifampicin	3	1.3	7	3.9	0	0.0	3	1.7	1	1.5	14	2.0	5.279	0.26
Mupirocin†	4	1.7	5	2.8	0	0.0	0	0.0	0	0.0	9	1.3	7.492	0.11
Daptomycin	0	0.0	2	1.1	0	0.0	0	0.0	0	0.0	2	0.3	5.939	0.20

* Constitutive resistance

† High-level resistance

Methicillin-susceptible *Staphylococcus aureus*

Resistance to non-ß-lactams amongst MSSA was rare apart from resistance to erythromycin (13.2% nationally) (Table 6). Resistance was not detected for vancomycin, teicoplanin or linezolid. Resistance levels between regions varied significantly for penicillin and high-level mupirocin. South Australia had the highest rate of resistance for penicillin and Queensland/Northern Territory was highest for high-level mupirocin. Multi-resistance was uncommon in MSSA (36/1,644 2.2%).

Inducible clinidamycin resistance

Overall, 348 of the 2,357 isolates (14.8%) were erythromycin resistant and clindamycin intermediate/susceptible *S. aureus* isolates. Of these, 306 (87.9%) were D-test positive indicating inducible clindamycin resistance. For MRSA the number that were D-test positive was 203/224 (90.6%) and for MSSA 103/124 (83.1%).

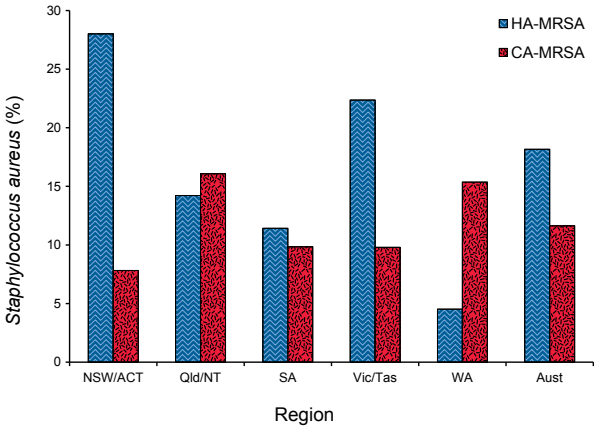
Molecular typing

Based on molecular typing, of the 703 MRSA referred to ACCESS Typing and Research, 428 (60.9%) and 275 (39.1%) were classified as HA-MRSA and CA-MRSA strains respectively.

Healthcare-associated methicillin-resistant *Staphylococcus aureus*

Throughout Australia the percentage of *S. aureus* characterised as HA-MRSA was 18.2% ranging from 4.5% in Western Australia to 28.0% in the New South Wales/Australian Capital Territory region (Figure).

Figure: Percentage of *Staphylococcus aureus* characterised as HA-MRSA and CA-MRSA strains, by region



Four HA-MRSA clones were identified: ST22-IV [2B] (EMRSA-15) (49.5% of HA-MRSA); ST239-III [3A] (Aus-2/3 EMRSA) (49.3%); 3 isolates of ST5-II [2A] (New York Japan MRSA/USA100) and 2 isolates of ST36-II [2A] (EMRSA-16/USA200).

ST22-IV [2B] (EMRSA-15) has become the predominant HA-MRSA clone in Australia accounting for 30.2% of MRSA ranging from 15.6% in Queensland/Northern Territory to 40.2% in

Table 6: Number and proportion of methicillin-susceptible *Staphylococcus aureus* isolates resistant to the non-ß-lactam antimicrobials, Australia, 2011, by state or territory

	NSW/ACT (n=404)		Qld/NT (n=411)		SA (n=199)		Vic./Tas. (n=364)		WA (n=266)		Aus (n=1,644)		Differences across regions	
Drug	n	%	n	%	n	%	n	%	n	%	n	%	X^2	*P*
Penicillin	346	85.6	355	86.4	179	89.9	321	88.2	212	79.7	1,413	85.9	12.8	0.01
Erythromycin	49	12.1	64	15.6	25	12.6	52	14.3	27	10.2	217	13.2	5.0	0.29
Clindamycin*	10	2.5	8	1.9	3	1.5	9	2.5	7	2.6	37	2.3	1.0	0.91
Tetracycline	13	3.2	9	2.2	2	1.0	13	3.6	9	3.4	46	2.8	4.3	0.37
Co-trimoxazole	9	2.2	8	1.9	3	1.5	8	2.2	5	1.9	33	2.0	0.5	0.98
Ciprofloxacin	15	3.7	12	2.9	5	2.5	15	4.1	8	3.0	55	3.3	1.6	0.81
Gentamicin	5	1.2	7	1.7	1	0.5	3	0.8	2	0.8	18	1.1	1.1	0.29
Fusidic Acid	11	2.7	24	5.8	8	4.0	3	0.8	9	3.4	55	3.3	1.8	0.18
Rifampicin	0	0.0	0	0.0	1	0.5	0	0.0	0	0.0	1	0.1	7.3	0.12
Mupirocin†	3	0.7	20	4.9	0	0.0	0	0.0	3	1.1	26	1.6	4.2	0.04
Daptomycin	1	0.2	0	0.0	0	0.0	0	0.0	0	0.0	1	0.1	3.1	0.55

* Constitutive resistance

† High-level resistance

New South Wales/Australian Capital Territory (Table 7). ST22-IV [2B] are typically PVL negative, and 99% and 66% of these were resistant to ciprofloxacin and erythromycin respectively.

ST239-III [3A] (Aus-2/3 EMRSA) accounted for 30.0% of MRSA ranging from 0% in Western Australia to 36.2% in Victoria/Tasmania (Table 7). PVL negative ST239-III [3A] (Aus -2/3 EMRSA was typically resistant to tetracycline (100%), erythromycin (97%), ciprofloxacin (96%), cotrimoxazole (94%), and gentamicin (94%).

Community-associated-methicillin-resistant *Staphylococcus aureus*

Throughout Australia the percentage of *S. aureus* characterised as CA-MRSA was 11.7% ranging from 7.8% in New South Wales/Australian Capital Territory to 16.1% in Queensland/Northern Territory. Thirty-two CA-MRSA clones were identified by pulsed field gel electrophoresis, corresponding to 25 MLST/SCC*mec* clones (Table 8). Overall, 79.6% of CA-MRSA were classified into 6 clones.

ST1-IV [2B] (WA1) accounted for 9.0% of MRSA ranging from 3.5% in Victoria/Tasmania to 31.8% in Western Australia (Table 9). Typically PVL negative, 95.2% of isolates were non-multi-resistant. Eighty- nine per cent of isolates were resistant to the ß-lactam antimicrobials only or additionally to erythromycin (16%) or fusidic acid only (6%), or to both (8%). Two isolates were resistant to mupirocin, gentamicin and erythromycin, and a single isolate resistant to mupirocin, gentamicin and fusidic acid.

ST93-IV [2B] (Qld CA-MRSA) accounted for 7.3% of MRSA ranging from 1.7% in Victoria/Tasmania to 14.5% Queensland/Northern Territory (Table 9). PVL positive ST93-IV (Qld CA-MRSA) were typically resistant to the ß-lactams only (41/51) or additionally to erythromycin (10/51).

ST5-IV [2B] (WA3) and ST78-IV [2B] (WA2) although predominantly isolated in Western Australia (9.1% and 18.2% of MRSA respectively), were also isolated in most regions of Australia. ST45-V [5C2] (WA84) and ST30-IV [2B] (SWP MRSA) were predominantly isolated in Victoria/Tasmania (11.5% of MRSA) and Queensland/Northern Territory (8.4% of MRSA) respectively.

Overall, 92.7% of CA-MRSA were non-multi-resistant and 50.9% of isolates were resistant to ß-lactam antimicrobials only. However, 20 isolates (7.3% of CA-MRSA) were multiresistant including 3 PVL positive ST772-V [5C2] (Bengal Bay MRSA) isolates, which in addition to ß-lactam antimicrobials were resistant to gentamicin, erythromycin, ciprofloxacin and cotrimoxazole. One CA-MRSA (ST7-V [5C2]) isolate was resistant to 5 non-ß-lactam antimicrobials; gentamicin, erythromycin, ciprofloxacin, cotrimoxazole and tetracycline.

Panton–Valentine leucocidin

In 2011, 13.5% of MRSA were PVL positive. Eighty-seven (31.6%) CA-MRSA (Table 8) and 8 ST22-IV [2B] were PVL positive. PVL-positive CA-MRSA clones included the international clones ST8-IV [2B] (USA300) and ST772-V [5C2] (Bengal Bay MRSA).

Discussion

This survey demonstrates that MRSA remains a significant burden in Australian hospitals. For 2011, the national proportion of *S. aureus* that were MRSA was 30.3%, which was not significantly different to the proportions seen in past AGAR hospital inpatient surveys (X^2 for trend 0.7527, $P=0.3856$).[9] Differences between regions in the 2011 survey were significant with South Australia and Western Australia having a lower proportion than other regions. Although the proportion of MRSA amongst the different specimen types was similar, the high proportion of MRSA in invasive isolates is of concern as MRSA bacteraemia is associated with increased mortality compared with MSSA.[1–4]

More than 60% of the MRSA in the 2011 study were resistant to erythromycin and ciprofloxacin, and more than 30% were resistant to tetracycline, co-trimoxazole and gentamicin. Regional differences were again common due to different MRSA clones circulating in Australia. Erythromycin and ciprofloxacin resistance was more widespread in

Table 7: Proportion of methicillin-resistant *Staphylococcus aureus* isolates characterised as ST22-IV [2B] (EMRSA-15) and ST239-III [3A], Australia, 2011, by state or territory

	NSW/ACT	Qld/NT	SA	Vic/Tas	WA	Aust
ST22-IV [2B]	40.2%	15.6%	34.5%	33.3%	22.7%	30.2%
ST239-III [3A]	35.8%	31.3%	18.2%	36.2%	0%	30.0%

Table 8: Proportion of community-associated-methicillin-resistant *Staphylococcus aureus*, Australia, 2011, by clone and Panton–Valentine leucocidin carriage

Clone	Clonal complex	Alternative name	n	%	PVL Pos
ST1-IV [2B]	1	WA-1	63	22.9	3 (4.8%)
ST93-IV [2B]	Singleton	Queensland MRSA	51	18.6	51 (100%)
ST5-IV [2B]	5	WA-3	34	12.4	2 (5.9%)
ST78-IV [2B]	88	WA-2	25	9.1	0
ST45-V [5C2]	45	WA-84 (Vic CA-MRSA)	25	9.1	0
ST30-IV [2B]	30	SWP MRSA	21	7.6	18 (85.7%)
ST73-IV [2B]	5	WA-65	10	3.6	0
ST8-IV [2B]	8	USA300	8	2.9	8 (100%)
ST772-V [5C2]	1	Bengal Bay	3	1.1	3 (100%)
ST835-IV [2B]	5	WA-48	3	1.1	0
ST45-V [5C2]	45	WA-4	3	1.1	0
ST45-IV [2B]	45	WA-75	3	1.1	0
ST1-V [5C2]	1		2	0.7	0
ST5-V [5C2]	5	WA-90	2	0.7	0
ST59-IV [2B]	59	WA-15	2	0.7	0
ST72-IV [2B]	72	WA-44	2	0.7	0
ST75-IV [2B]	75	WA-8	2	0.7	0
ST45-V [5C2]	45		2	0.7	0
ST188-IV [2B]	1	WA-38	1	0.4	0
ST573-V [5C2]	1	WA-10	1	0.4	1 (100%)
ST5-V [5C2]	5	WA-14	1	0.4	0
ST575-IV [2B]	5	WA-25	1	0.4	0
ST5-V [5C2]	5	WA-35	1	0.4	0
ST5-V [5C2]	5	WA-108	1	0.4	0
ST5-V [5C2]	5	WA-109	1	0.4	0
ST1756-V [5C2]	5		1	0.4	0
ST7-V [5C2]	7		1	0.4	0
ST45-IV [2B]	45	WA-23	1	0.4	0
ST1970-V [5C2]	45	WA-106	1	0.4	0
ST59-IV [2B]	59	WA-55	1	0.4	1 (100%)
ST1304-IV [2B]	75	WA-72	1	0.4	0
ST953-IV [2B]	97	WA-54	1	0.4	0
Total			275		87 (31.6%)

Percentage figures in parenthesis relate to community-associated-methicillin-resistant *Staphylococcus aureus* isolates.
PVLPanton–Valentine leucocidin.

Table 9: Proportion of methicillin-resistant *Staphylococcus aureus* characterised as ST1-IV [2B] (WA1) and ST93-IV [2B], Australia, 2011, by region

	NSW/ACT	Qld/NT	SA	Vic/Tas	WA	Aus
ST1-IV [2B]	4.8%	11.2%	9.1%	3.5%	31.8%	9.0%
ST93-IV [2B]	5.7%	14.5%	9.1%	1.7%	6.1%	7.3%

this survey with at least 30% of MRSA with this profile in any region. Erythromycin and ciprofloxacin resistance is common in ST239-III [3A] (Aus 2/3 EMRSA) isolates but is also characteristic of ST22-IV [2B] (EMRSA-15). ST22-IV [2B] (EMRSA-15) is a frequently isolated HA-MRSA in Australia and was found in all regions. Resistance was not detected for vancomycin, teicoplanin or linezolid. Compared with previous AGAR hospital inpatient surveys, the proportion of MRSA resistant to erythromycin, clindamycin, tetracycline, co-trimoxazole, ciprofloxacin, gentamicin and rifampicin has decreased nationally with significant decreases in New South Wales/Australian Capital Territory and Victoria/Tasmania. The proportion of *S. aureus* that are MRSA has remained stable in all regions and nationally. This finding is due to non-multi-resistant CA-MRSA increasing in Australian hospitals at the expense of the long-established multi-resistant ST239-III [3A].

Given that reports of outbreaks of CA-MRSA in Australian hospitals are thought to be rare[17,18] it is likely that many infections in hospital inpatients are caused by the patients' own colonising strains acquired prior to admission. Community clones such as PVL negative ST1-IV [2B] (WA1) and PVL positive ST93-IV [2B] (Qld CA-MRSA) are well established in Australia,[19,20] and therefore it is important to monitor antimicrobial resistance patterns to MRSA over time as this information will guide therapeutic practices.

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Australian Group on Antimicrobial Resistance Community-onset Gram-negative Surveillance Program annual report, 2010

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Abstract

The Australian Group on Antimicrobial Resistance (AGAR) performs regular period-prevalence studies to monitor changes in antimicrobial resistance in selected enteric Gram-negative pathogens. The 2010 survey focussed on community-onset infections, examining isolates from urinary tract infections from patients presenting to outpatient clinics, emergency departments or to community practitioners. Two thousand and ninety-two *Escherichia coli,* 578 *Klebsiella* species and 268 *Enterobacter* species were tested using a commercial automated method (Vitek 2, BioMérieux) and results were analysed using Clinical and Laboratory Standards Institute breakpoints from January 2012. Of the key resistances, non-susceptibility to the third-generation cephalosporin, ceftriaxone, was found in 3.2% of *E. coli* and 3.2%–4.0% of *Klebsiella* spp. Non-susceptibility rates to ciprofloxacin were 5.4% for *E. coli,* 1.0%–2.3% for *Klebsiella* spp., and 2.5%–6.6% in *Enterobacter* spp, and resistance rates to piperacillin-tazobactam were 2.8%, 3.2%–6.9%, and 16.8%–18.0% for the same 3 groups respectively. Only 3 strains, 2 *Klebsiella* spp. and 1 *Enterobacter* spp, were shown to harbour a carbapenemase (IMP-4). *Commun Dis Intell* 2013;37(3):E219–E223.

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

Introduction

Emerging resistance in common pathogenic members of the Enterobacteriaceae is a worldwide phenomenon, and presents therapeutic problems for practitioners in both the community and in hospital practice. The Australian Group on Antimicrobial Resistance commenced surveillance of the key Gram-negative pathogens, *Escherichia coli* and *Klebsiella* species in 1992. Surveys have been conducted biennially until 2008 when annual surveys commenced alternating between community– and hospital-onset infections (http://www.agargroup.org/surveys). In 2004, another genus of Gram-negative pathogens in which resistance can be of clinical importance, *Enterobacter* species, was added. *E. coli* is the most common cause of community-onset urinary tract infection, while *Klebsiella* species are less common but are known to harbour important resistances. *Enterobacter* species are less common in the community, but of high importance due to intrinsic resistance to first-line antimicrobials in the community. Taken together, the 3 groups of species surveyed are considered to be valuable sentinels for multi-resistance and emerging resistance in enteric Gram-negative bacilli.

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

The objectives of the 2010 surveillance program were to:

1. determine the proportion of resistance to the main therapeutic agents in *Escherichia coli, Klebsiella* species and *Enterobacter* species in a subset of Australian diagnostic laboratories;
2. examine the extent of co-resistance and multi-resistance in these species; and
3. detect emerging resistance to newer last-line agents such as carbapenems. Isolates from the urinary tract were selected for this program.

Methods

Source of isolates

Isolates were collected from non-hospitalised patients with urinary tract infections, including those presenting to emergency departments, outpatient departments or to community practitioners. Each institution collected up to 70 *E. coli,* 20 *Klebsiella* spp. and 10 *Enterobacter* spp. Urinary tract isolates were selected because of their high frequency and high rates of exposure to antimicrobial agents in the community.

Species identification

Isolates were identified by one of the following methods: Vitek®; Phoenix™ Automated Microbiology System, Microbact; ATB®; or agar replication. In addition, some *E. coli* isolates were identified using chromogenic agar plus spot indole (DMACA).

Susceptibility testing

Testing was performed by a commercial semi-automated method, Vitek® 2 (BioMérieux), which is calibrated to the ISO reference standard method of broth microdilution. Commercially available Vitek® AST-N149 cards were utilised by all participants throughout the survey period. The most recent Clinical and Laboratory Standards Institute breakpoints from 2012[1] have been employed in the analysis. *E. coli* ATCC 25922 and *E. coli* ATCC 35218 were the quality control strains for this survey. For analysis of cefazolin, breakpoints of ≤4 for susceptible, ≥8 for resistant were applied due to the minimum inhibitory concentration (MIC) range available on the Vitek card, recognising that the January 2012 breakpoint is actually susceptible ≤2 mg/L. Ertapenem MICs were performed using Etest™ strips (BioMérieux).

Molecular confirmation of resistances

E. coli and *Klebsiella* isolates with ceftazidime or ceftriaxone MIC >1 mg/L, or cefoxitin MIC >8 mg/L; *Enterobacter* spp. with cefepime MIC >1 mg/L; and all isolates with ertapenem MIC >0.5 mg/L or meropenem MIC >0.25 mg/L were referred to a central laboratory for molecular confirmation of resistance.

All isolates were screened for the presence of the bla_{TEM} and bla_{SHV} genes using a real-time polymerase chain reaction (PCR) platform (LC-480) and published primers.[2,3] A multiplex real-time TaqMan PCR was used to detect CTX-M-type genes.[4] Strains were probed for plasmid-borne AmpC enzymes using the method described by Pérez-Pérez and Hanson,[5] and subjected to molecular tests for MBL (bla_{VIM}, bla_{IMP} and bla_{NDM}), bla_{KPC} and $bla_{OXA-48-like}$ genes using real-time PCR.[6,7]

Results

The species isolated, and the numbers of each are listed in Table 1. Major resistances and non-susceptibilities are listed in Table 2. Non-susceptibility, (which includes both intermediately resistant and resistant strains), has been included for some agents because these figures provide information about important emerging acquired resistances. Multiple acquired resistances by species are shown in Table 3. Multi-resistance was detected in 7.3% of *E. coli* isolates, 4.3% of *Klebsiella* species, and 8.6% of *Enterobacter* species. A more detailed breakdown of resistances and non-susceptibilites by state and territory is provided in the online report from the group (http://www.agargroup.org/surveys). By way of summary, there were no substantial differences across the states and territories in resistance patterns in contrast to what is seen with resistance patterns in *Staphylococcus aureus* and *Enterococcus* spp.

Table 1: Species tested

Group	Species	Total
E. coli	*E. coli*	2,092
Klebsiella	*K. pneumoniae*	475
	K. oxytoca	101
	K. pneumoniae subsp *ozaenae*	2
	Total	578
Enterobacter	*E. cloacae*	137
	E. aerogenes	122
	E. asburiae	7
	E. sakazakii	1
	Enterobacter not speciated	1
	Total	268

Escherichia coli

Moderately high levels of resistance to ampicillin (and therefore amoxycillin) were observed (43.4%), with lower rates for amoxycillin-clavulanate (14.8% intermediate, 6.2% resistant). Non-susceptibility to third-generation cephalosporins is low but appears to be increasing slowly compared with the 2008 survey (ceftriaxone 3.2%, ceftazidime 1.9%). In line with international trends among community strains of *E. coli*, most of the strains with extended-spectrum ß-lactamase (ESBL) genes harboured genes of the CTX-M type (51/65 = 78%). Moderate levels of resistance were detected to cefazolin (15.2%) and trimethoprim (21.2%). Ciprofloxacin non-susceptibility was found in 5.4% of *E. coli* isolates. Ciprofloxacin resistance was found in 60.3% and gentamicin resistance was found in 49.2% of ESBL-producing strains. Resistance to ticarcillin-clavulanate, piperacillin-tazobactam, cefepime, and gentamicin were below 5%. No isolates had elevated meropenem MICs (≥0.5 mg/L) but 28 (1.3%) strains had ertapenem MICs above wild-type (>0.06 mg/L), 85% of which contained CTX-M or plasmid-borne AmpC genes.

Klebsiella species

These showed slightly higher levels of resistance to cefazolin, ceftriaxone and piperacillin-tazobactam

compared with *E. coli*, but lower rates of resistance to amoxycillin-clavulanate, ticarcicllin-clavulanate, ciprofloxacin, gentamicin, and trimethoprim. ESBLs were present in all 17 presumptively ESBL-positive isolates of *K. pneumoniae*, 12 of which proved to be of the CTX-M type. Two of 3 strains of *K. pneumoniae* with elevated meropenem MICs (≥ 0.5 mg/L) harboured bla_{IMP-4}, while 13 additional strains had elevated ertapenem MICs (> 0.06 mg/L), but none of these harboured a known carbapenemase.

Enterobacter species

Acquired resistance was common to ticarcillin-clavulanate (19.8%), piperacillin-tazobactam (17.2%), ceftriaxone (24.6%), ceftazidime (20.9%) and trimethoprim (12.3%). Rates of resistance to cefepime, ciprofloxacin, and gentamicin were all less than 5%. Five of 12 strains tested for extended-spectrum ß-lactamases based on a suspicious phenotype, harboured ESBL-encoding genes. Three strains had elevated meropenem MICs (≥ 0.5 mg/L) one of which harboured bla_{IMP-4}, while 37% of strains had ertapenem MICs above wild-type (> 0.125 mg/L), which appeared to bear some relationship to stably-derepressed chromosomal AmpC ß-lactamase.

Discussion

The Australian Group on Antimicrobial Resistance has been tracking resistance in sentinel enteric Gram-negative bacteria since 1992. Until 2008, surveillance was segregated into hospital- versus community-onset infections. The first year of community-onset only surveillance was 2008.[8] Comparing results from that year with 2010, the next community-onset surveillance year, shows a small but noticeable increase in resistance rates to some reserve antibiotics. For example, rates of resistance in *E. coli* for ceftriaxone rose from 2.1% to 3.2% and for non-susceptibility to ciprofloxacin rose from 4.2% to 5.4%. Further surveys will establish whether this is a genuine trend or simply a sampling issue.

Overall though, there are worrying trends in the emergence of CTX-M-producing *E. coli* and *Klebsiella* species and ciprofloxacin-resistant *E. coli* now presenting in or from the community. Other resistance patterns appear stable. Compared with many other countries in our region, resistance rates in Australian Gram-negative bacteria are still relatively low. [9]

Table 2: Non-susceptibility and resistance rates for the main species tested

Antimicrobial	Category*	*E. coli* (%)	*K. pneumonia* (%)	*K. oxytoca* (%)	*E. cloacae* (%)	*E. aerogenes* (%)
Ampicillin	I	1.3	†	†	†	†
Ampicillin	R	43.4	†	†	†	†
Amoxycillin-clavulanate	I	14.8	2.3	4.0	†	†
Amoxycillin-clavulanate	R	6.2	2.3	5.0	†	†
Ticarcillin-clavulanate	R	4.5	2.3	3.0	22.6	17.2
Piperacillin-tazobactam	R	2.8	3.2	6.9	16.8	18.0
Cefazolin	R	15.2	6.7	68.3	†	†
Cefoxitin	R	1.8	2.7	2.0	†	†
Ceftriaxone	NS	3.2	3.4	4.0	27.7	22.1
Ceftazidime	NS	1.9	1.9	0.0	22.6	19.7
Cefepime	NS	0.7	0.0	0.0	1.5	0.0
Meropenem	NS	0.0	0.2	0.0	0.0	0.0
Ertapenem	NS	0.1	0.6	0.0	16.2	5.8
Ciprofloxacin	NS	5.4	2.3	1.0	6.6	2.5
Norfloxacin	NS	5.2	2.1	1.0	3.6	2.5
Gentamicin	NS	4.2	2.3	0.0	4.4	0.8
Trimethoprim	R	21.2	10.5	5.9	16.1	7.4
Nitrofurantoin	NS	0.5	†	†	†	†

* R = resistant, I = intermediate, NS = non-susceptible (intermediate + resistant)

† Considered largely intrinsically resistant due to natural β-lactamases

Table 3: Multiple acquired resistances, by species

Species	Total	Non-multi-resistant				Cumulative (%)	Number of acquired resistances				Multi-resistant				Cumulative (%)
		0	1	2	3		4	5	6	7	8	9	10		
E. coli	2,092	1,073	407	357	105		66	32	26	13	8	3	2		
%		51.3	19.5	17.1	5.0	92.8	3.2	1.5	1.2	0.6	0.4	0.1	0.1		7.2
Klebsiella spp.*	578	292	209	37	15		13	6	2	3		1			
%		50.5	36.2	6.4	2.6	95.7	2.2	1.0	0.3	0.5		0.2			4.3
Enterobacter spp.†	268	108	92	20	25		14	7	2						
		40.3	34.3	7.5	9.3	91.4	5.2	2.6	0.7						8.6

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

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

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Australian Meningococcal Surveillance Programme annual report, 2012

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Abstract

In 2012, there were 208 laboratory-confirmed cases of invasive meningococcal disease (IMD) analysed by the National Neisseria Network, and 222 cases notified to the National Notifiable Diseases Surveillance System, thus laboratory data were available for 93.7% of cases of IMD in Australia in 2012. Isolates of *Neisseria meningitidis* from 116 invasive cases of meningococcal disease were available for testing, and the phenotype (serogroup, serotype and serosubtype) and/or genotype, and antibiotic susceptibility were determined. Molecular typing was performed for the 92 cases confirmed by nucleic acid amplification testing (NAAT). Typing information was available for 194 of the 208 laboratory confirmed cases and 83% (161 cases) were serogroup B infections, 5.7% (11 cases) were serogroup C infections, 3.6% (11 cases) were serogroup W135, and 7.7% (15 cases) were serogroup Y meningococci. The number of laboratory confirmed IMD cases in 2012 was the lowest since laboratory surveillance data have been reported. Primary and secondary disease peaks were observed in those aged 4 years or less and in adolescents (15–19 years) and young adults (20–24 years), respectively. Serogroup B cases predominated in all age groups and jurisdictions. In 2012, the most common porA genotype circulating in Australia was P1.7-2,4. Serogroup C, W135 and Y cases were numerically low, similar to previous years. Decreased susceptibility to the penicillin group of antibiotics was observed in 81.9% of isolates, and 1 isolate exhibited resistance to penicillin. All isolates remained susceptible to ceftriaxone, ciprofloxacin and rifampicin. *Commun Dis Intell* 2013;37(3):E224–E232.

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

Introduction

The National Neisseria Network (NNN) is a long-standing collaborative association for the laboratory surveillance of the pathogenic *Neisseria* species (*N. meningitidis* and *N. gonorrhoeae*). Since 1994 the NNN has operated through a network of reference laboratories in each state and territory to provide a national laboratory-based program for the examination of *N. meningitidis* from cases of invasive meningococcal disease (IMD).[1]

The NNN supplies data on the phenotype and/or the genotype of invasive meningococci, and their antibiotic susceptibility for the AMSP. The AMSP data supplement the clinical notification data from the National Notifiable Diseases Surveillance System (NNDSS). The NNN receives samples for analysis from about 90% of IMD cases notified to NNDSS.[2] The AMSP annual reports are published in *Communicable Diseases Intelligence*.[3]

The characteristics of the meningococci responsible for IMD are important both for individual patient management, contact management, and to tailor the public health response for outbreaks or case clusters locally and nationally. The introduction of publicly funded conjugate serogroup C meningococcal vaccine onto the National Immunisation Program in 2003 (with a catch-up program for those aged 1–19 years that ran until May 2007) has seen a significant and sustained reduction in the number of cases of IMD evident after 2004.[2] However, IMD remains an issue of public health concern in Australia. The success of any further vaccine initiatives in Australia is dependent upon detailed analysis of the *N. meningitidis* isolates circulating locally. This report provides relevant details of cases of IMD confirmed by laboratory testing in Australia in 2012.

Methods

Case confirmation of invasive meningococcal disease cases

Case confirmation was based upon isolation of, or positive nucleic acid amplification testing (NAAT) for, *N. meningitidis* from a normally sterile site and defined as IMD according to Public Health Laboratory Network criteria.[4] Information regarding the site of infection, the age and sex of the patient, and the outcome of the infection (survived/died) was collated.

Cases were categorised on the basis of the site from which *N. meningitidis* was isolated or from which meningococcal DNA was detected. When *N. meningitidis* was grown from both blood and cerebrospinal fluid (CSF) cultures from the same patient, the case was classified as one of meningitis. It is recognised that the total number of IMD cases, and particularly the number of cases of meningitis,

may be underestimated if a lumbar puncture was not performed or was delayed and the culture was sterile. However, the above approach has been used since the beginning of this program and is continued for comparative purposes. Where the diagnosis is made by serology, it is not possible to definitively classify a case as meningitis or septicaemia.

Phenotyping and genotyping of *Neisseria meningitidis*

Phenotyping of invasive isolates of meningococci by serotyping and serosubtyping was based on the detection of outer membrane protein (porin) antigens using a standard set of monoclonal antibodies obtained from The Netherlands National Institute for Public Health. Genotyping of isolates and DNA extracts from NAAT diagnosis is performed by sequencing of products derived from amplification of the porin genes *porA, porB* and *FetA*.

Antibiotic susceptibility testing

Antibiotic susceptibility was assessed by determining the minimum inhibitory concentration (MIC) to antibiotics used for therapeutic and prophylactic purposes. This program uses the following parameters to define the various levels of penicillin susceptibility or resistance when determined by a standardised agar plate dilution technique.[5]

Sensitive: MIC ≤ 0.03 mg/L
Less sensitive: MIC 0.06–0.5 mg/L
Resistant: MIC ≥ 1 mg/L

Meningococcal serology

Laboratory diagnosis of suspected cases of IMD can be made serologically based on the demonstration of IgM antibody by enzyme immunoassay to *N. meningitidis* outer membrane protein using the methods and test criteria of the Health Protection Agency, United Kingdom, as assessed for Australian conditions.[6–8]

Results

Aggregated data on laboratory confirmed invasive meningococcal disease cases

In 2012, there were 208 laboratory-confirmed cases of IMD analysed by the National Neisseria Network, and 222 cases notified to the NNDSS, thus laboratory data were available for 93.7% of cases of IMD in Australia in 2012 (Table 1). This was the lowest annual total of IMD cases recorded by the NNDSS and the AMSP since surveillance data was collated (Figure 1).

In 2012, a positive culture was obtained for 116 of 208 (56%) cases of which 92 (44%) cases were confirmed by NAAT testing alone. There were no IMD cases diagnosed serologically in 2012.

The highest number of laboratory-confirmed cases was from New South Wales (62 cases), slightly lower than the 67 cases in 2011. Victoria had 33 cases, markedly lower than the 53 cases in 2011. Numbers for the other states were similar to 2011 (Table 1).

Seasonality and age distribution

As in previous years, the peak incidence for IMD continues to be late winter and early spring (1 July to 30 September) (Table 2).

Nationally, the peak incidence of IMD was in children aged less than 5 years, which was similar to previous years. Between 2007 and 2011, 28% to 36% of cases were in this age group. In 2012, 62 of

Table 1: Number of laboratory-confirmed cases of invasive meningococcal disease, Australia, 2012, by serogroup and state or territory

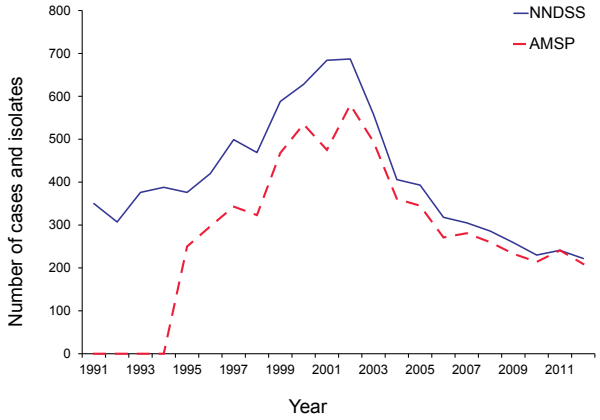
State or territory	Serogroup						Total
	B	C	Y	W135	NG	ND	
ACT	1	0	0	0	0	0	1
NSW	43	2	5	4	7	1	62
NT	2	1	0	0	0	1	4
Qld	45	3	4	3	0	4	59
SA	23	1	0	0	0	0	24
Tas	4	1	1	0	0	1	7
Vic	28	1	4	0	0	3	33
WA	15	2	1	0	0	0	18
Australia	161	11	15	7	7	7	208

NG Non-groupable.

ND Non-determined (samples were examined by nucleic acid amplification test).

208 (30%) IMD cases occurred in this age group, as shown in Table 3. A secondary disease peak has also been observed in previous years amongst adolescents and young adults aged 15 to 24 years. Of all cases 13.5% (28 confirmed cases) in those aged 15 to 19 years in 2012 was lower than the number reported for the years 2007 to 2011 (between 17% and 20%). There were 26 cases of IMD (12.5%) in the 20 to 24 years age group, which was similar to 2011, but lower than the 22% to 31% reported in this age group in the years 2007 to 2010.

Figure 1: Number of invasive meningococcal disease cases reported to the NNDSS compared with laboratory confirmed data from the AMSP, Australia, 2012



Serogroup data

The serogroup was determined for 194 of the 208 laboratory-confirmed cases of IMD in 2012 (Table 1). Of these, 161 (83%) were serogroup B and 11 (5.7%) were serogroup C. The proportion of cases that were serogroup B was similar to the proportion reported between 2006 and 2011 (between 84% and 88%). The number and proportion of cases of serogroup C in 2012 was slightly higher than in 2011 (9 cases; 3.7%). In 2012 there were 7 (3.6%) cases of serogroup W135, which was less than the previous year (11 cases; 5.2%). There were 15 (7.7%) cases of serogroup Y, a slightly higher proportion than in 2011 (6.2%) and higher than the proportions in 2009 and 2010 (3.5% and 3.9% respectively). With the continuing low number of serogroup C infections, serogroup B meningococci predominated in all age groups and jurisdictional differences in serogroup distribution were not evident.

In 2012, total IMD cases, the number of cases due to serogroup B, and the proportion of serogroup B cases

Table 2: Laboratory-confirmed cases of invasive meningococcal disease, Australia, 2012, by quarter

Serogroup	Qtr 1	Qtr 2	Qtr 3	Qtr 4	Total 2012
B	24	47	56	34	161
C	3	1	3	4	11
Y	1	3	9	2	15
W135	0	1	4	2	7
NG/ND	3	3	8	0	14
Total	31	55	80	42	208

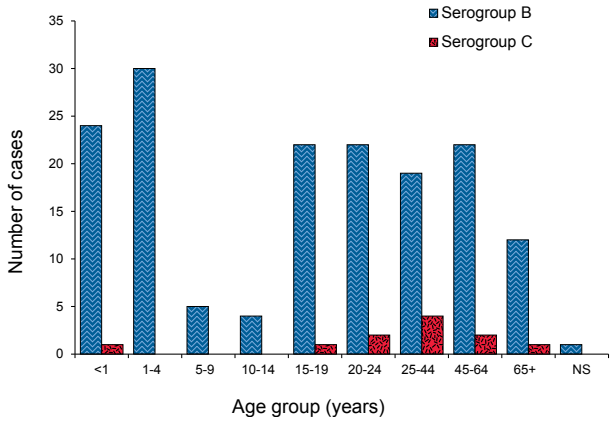
Table 3: Laboratory-confirmed cases of invasive meningococcal disease, Australia, 2012, by age and serogroup

	Age group										
Serogroup	<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	Total
B	24	30	6	4	22	22	19	21	12	1	161
C	1	0	0	0	1	2	4	2	1	0	11
Y	1	0	0	0	1	2	4	3	4	0	15
W135	0	2	0	0	2	0	0	2	1	0	7
NG/ND	1	3	4	3	2	0	0	1	0	0	14
Total	27	35	10	7	28	26	27	29	18	1	208
% of B within age group	88. 9	85.7	60.0	57.1	78.6	84.6	70.4	72.4	66.7	100.0	

NS Age not stated
NG Non-groupable
ND Non-determined (samples were examined by nucleic acid amplification test).

from the total was lower in each of the age categories less than 20 years (Table 2, Figure 2). The proportion of serogroup B cases in the 20 to 24 years age group (84.6%) was higher than the previous year (61%) but similar to 2007 to 2010 (between 80% and 88%). In people aged 25 years or over, there was a modest increase in the proportion of serogroup B cases from 2011. This may in part be explained by an increase in the number of serological IMD diagnoses (and thus serogroup not determined) in this age category for 2011. The peak number of serogroup C cases occurred in the 25 to 44 years age category, as reported for 2011. There were 2 serogroup C cases in those aged less than 20 years in 2012, but no cases in 2011.

Figure 2: Number of serogroup B and C cases of confirmed invasive meningococcal disease, Australia, 2012, by age



NS Not serotyped

Phenotypes of invasive meningococcal isolates

Serogroup B meningococci are typically of heterogeneous phenotypes. In 2012, the phenotypes of invasive isolates, based on a determination of their serogroup, serotype and serosubtype, were analysed for New South Wales (Table 4). Serogroup B meningococci are in general more difficult to characterise by serological methods and a number could not be phenotyped. All 35 New South Wales IMD isolates were phenotyped, the most common being B:4:P1.4 followed by B:15:P1.7.

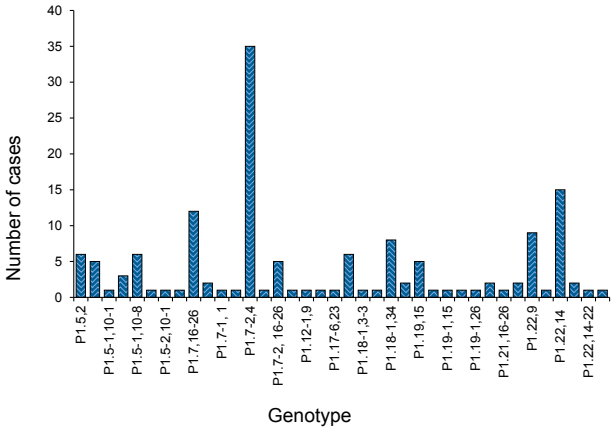
Genotyping data of invasive meningococcal samples (culture or nucleic acid amplification test products)

Sequencing products derived from amplification of the variable region *porA, porB* and *FetA* genes is used in an increasing number of jurisdictions in place of serotyping using monoclonal antibodies. In 2012, genotyping data were available from all states and territories for 146 of 208 (70%) IMD cases (Table 5). The predominant *porA* genotypes for serogroup B isolates were again P1.7-2,4 (35 cases, compared with 21 in 2011), P1.22,14 (15 cases, compared with 7 in 2011) and P1.7,16-26 (12 cases, compared with 19 in 2011 (Table 6 and Figure 3).

Table 4: Laboratory confirmed cases of invasive meningococcal disease, New South Wales and the Australian Capital Territory, 2012, by phenotype

Serotype	Subtype	Serogroup	Total
4	P1.4	B	5
NT	P1.5	Y	3
15	P1.7	B	3
1	P1.14	B	3
NT	NST	B	2
4	P1.14	B	2
15	NST	B	2
NT	P1.4	B	2
1	P1.4	B	1
NT	P1.5,2	B	1
15	P1.7,1	B	1
4	P1.9	B	1
NT	P1.9	B	1
4	NST	B	1
2a	P1.2	W135	1
NT	P1.3	W135	1
NT	P1.5	B	1
NT	P1.6,3	W135	1
NT	P1.7	Y	1
NT	P1.16	W135	1
NT	NST	ND	1

Figure 3: Number of *porA*-genotypes* for serogroup B in cases of invasive meningococcal disease, Australia, 2012



* Where genotype data available.

The predominant *porA* genotype for serogroup C isolates was P1.5-1,10-8 (6 cases, compared with 4 in 2011). The AMSP was not aware of any epidemiological link between any of the cases reported where genotyping was available.

Outcome data for invasive meningococcal disease for laboratory-confirmed cases

For 69% of IMD cases (143/208), outcome data (survived or died) were available from the referring laboratories (Table 7). Nine deaths were recorded amongst the 143 cases for whom outcome data were available. Eight of these deaths were attributable to serogroup B infections, and one to serogroup C infection.

Anatomical source of samples for laboratory confirmed cases

There were 69 diagnoses of meningitis based on cultures or NAAT examination of CSF either alone or with a positive blood sample. There were 133 diagnoses of septicaemia based on cultures or NAAT examination from blood samples alone (Table 8). There were no IMD cases diagnosed by serology in 2012. Sites other than blood, CSF or serum from which diagnoses were made were tissue (1 by polymerase chain reaction), and joint fluid (5 by culture).

Antibiotic susceptibility testing of invasive meningococcal isolates

Penicillins

Susceptibility to penicillin and other antibiotics was determined for 116 of 208 (56%) cases in 2012. Using defined criteria, 95 (82%) isolates were less sensitive to penicillin in the MIC range 0.06–0.5 mg/L; and 19 (16%) isolates were fully sensitive (MIC 0.03 mg/L or less). One isolate was resistant (MIC = 1.0 mg/L). The proportion of less sensitive strains was lower than in 2011 (86.4%) but higher than that reported in 2007 to 2010 (range 67% to 80%).

Other antibiotics

All isolates were fully susceptible to ceftriaxone and by extrapolation to other third generation cephalosporins. All isolates were fully susceptible to ciprofloxacin. There were 2 isolates with altered susceptibility to rifampicin (MIC = 0.5 mg/L).

Table 5: Laboratory confirmed cases of invasive meningococcal disease, Australia (excluding New South Wales and the Australian Capital Territory), 2012, by *porA* genotype

Genotype *porA*	B	C	W135	Y	Total
P1.7-2,4	26	0	0	0	26
P1.22,14	9	1	0	1	11
P1.7,16-26	8	0	0	0	8
P1.22,9	7	0	0	0	7
P1.5-1,10-8	0	6	0	0	6
P1.18-1,34	6	0	0	0	6
P1.5,2	1	0	2	2	5
P1.7-2,16-26	5	0	0	0	5
P1.19,15	5	0	0	0	5
P1.5-1,2-2	3	0	0	1	4
P1.18-1,3	2	0	0	2	4
P1.19-3,15	1	1	0	0	2
P1.22,14-6	2	0	0	0	2
P1.5-1,10-1	0	0	0	1	1
P1.5-1,10-4	0	0	0	1	1
P1.5-2,10-1	0	0	0	1	1
P1.5-8,2-48	1	0	0	0	1
P1.7,30	1	0	0	0	1
P1.7-1,1	1	0	0	0	1
P1.7-1,13-1	0	0	0	1	1
P1.7-2,13-1	1	0	0	0	1
P1.7-11,16-26	1	0	0	0	1
P1.12-1,9	1	0	0	0	1
P1.17-6,23	1	0	0	0	1
P1.18-1,3-3	1	0	0	0	1
P1.18-7,9	1	0	0	0	1
P1.19-1,10-8	1	0	0	0	1
P1.19-1,15	1	0	0	0	1
P1.21,16-26	1	0	0	0	1
P1.22,10-8	1	0	0	0	1
P1.22-1,14	1	0	0	0	1
Total	89	8	2	10	109

Discussion

In 2012 there were 208 IMD cases laboratory-confirmed by the NNN, representing 93.7% of notifications to the NNDSS.[2] This was the lowest number of confirmed IMD cases and notifications since surveillance data began in 1991. It was also one-third of the number of confirmed cases and notifications of IMD in Australia in 2002 (580 confirmed IMD cases of 687 notifications) when the number of cases of IMD peaked. A primary peak in IMD infection rates is evident in children aged less than 5 years, as reported in previous years, with a secondary peak in adolescents and young adults.

The proportion of cases with serogroup B IMD cases is essentially the same as that reported between 2006 and 2011. The proportion of cases

Table 6: Laboratory-confirmed cases of invasive meningococcal disease, Australia (excluding New South Wales and the Australian Capital Territory), 2012, by state or territory

	NT		Qld		SA		Tas		Vic		WA	
Genotype *porA*	Serogroup	n	Serogroup	n	Serogroup	n	Serogroup	n	Serogroup	n	Serogroup	n
P1.7-2,4			B	14	B	12						
P1.22,14			B	1	C	1	B	1	B	5	B	2
P1.22,14			Y	1								
P1.7,16-26			B	4	B	1	B	1	B	2		
P1.22,9			B	1					B	6		
P1.5-1,10-8	C	1	C	3					C	1	C	1
P1.18-1,34			B	2					B	3	B	1
P1.5,2			W135	2					B	1	B	1
P1.5,2									Y	1		
P1.7-2,16-26			B	4							B	1
P1.19,15			B	2					B	3		
P1.5-1,2-2			B	1					B	2	Y	1
P1.18-1,3			B	1			Y	1	B	1		
P1.18-1,3									Y	1		
P1.19-3,15							B	1			C	1
P1.22,14-6			B	1							B	1
P1.5-1,10-1			Y	1								
P1.5-1,10-4									Y	1		
P1.5-2,10-1			Y	1								
P1.5-8,2-48									B	1		
P1.7, 30											B	1
P1.7-1,1					B	1						
P1.7-1,13-1											Y	1
P1.7-2,13-1									B	1		
P1.7-11,16-26											B	1
P1.12-1,9			B	1								
P1.17-6,23			B	1								
P1.18-1,3-3									B	1		
P1.18-7,9			B	1								
P1.19-1,10-8			B	1								
P1.19-1,15			B	1								
P1.21,16-26			B	1								
P1.22,10-8									B	1		
P1.22-1,14			B	1								

Table 7: Outcome data of infection for laboratory confirmed cases of invasive meningococcal disease, Australia, 2012, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	35	0	2	0	2	39
	Died	3	0	0	0	0	3
	Unknown	20	1	2	0	5	28
	Total	58	1	4	0	7	70
Septicaemia	Survived	72	5	8	3	2	90
	Died	5	1	0	0	0	6
	Unknown	24	3	2	3	5	37
	Total	101	9	10	6	7	133
Other	Survived	2	1	1	1	0	5
	Died	0	0	0	0	0	0
	Unknown	0	0	0	0	0	0
	Total	2	1	1	1	0	5
All cases	Survived	109	6	11	4	4	134
	Died	8	1	0	0	0	9
	Unknown	44	4	4	3	10	65
	Total	161	11	15	7	14	208

NG Serogroup not groupable or not determined.

Table 8: Anatomical source of samples positive for laboratory-confirmed cases of invasive meningococcal disease, Australia, 2012

Specimen type	Isolate of meningococci	PCR positive*	Serology alone†	Total
Blood	93	40	0	133
CSF +/– blood	18	51	0	69
Other‡	5	1	0	6
Total	116	92	0	208

* Nucleic acid amplification test (NAAT) positive in the absence of a positive culture.

† Serology positive in the absence of positive culture or NAAT.

‡ Joint fluid (n=5), tissue (n=1).

PCR Polymerase chain reaction.

with serogroup C cases continues to be low across all age groups, following the decline as a result of the introduction of the serogroup C vaccine in 2003. As in previous years, there were only a small number of serogroup C cases in those aged 25 years or over, which may reflect the secondary benefit of herd immunity accruing to the wider community following vaccination of those age groups where disease was formerly highly concentrated.[9] Low numbers of infections with serogroups Y and W135 is usual for Australia, however there was a proportional increase in serogroup Y disease in 2011 to 2012 compared with previous years. This will continue to be monitored to determine whether it is the beginning of an increasing trend.

As in previous years, phenotypic and genotypic data found no evidence of substantial numbers of cases of IMD caused by *N. meningitidis* that have undergone genetic recombination. There have been concerns that the emergence of new and invasive subtypes following extensive vaccine use would occur given the capacity for genetic reconfiguration within meningococci.[9] Monitoring of meningococcal genotypes will continue as part of the NNN program.

Outcome data were assessable for 69% of the cases reported by laboratories, and thus should be interpreted with caution. Eight of the 9 fatal cases of IMD were associated with serogroup B infection

and one with serogroup C. The NNN does not attempt active collection of morbidity data associated with IMD.

The proportion of IMD isolates with penicillin MICs in the less sensitive category (0.06–0.5 mg/L) for 2012 was 82%. This was lower than that reported in 2011, but higher than in previous years indicating a continuing shift in penicillin MICs of IMD isolates from sensitive to less sensitive category. All isolates were susceptible to the third generation cephalosporins and ciprofloxacin. Strains with decreased susceptibility to quinolone antibiotics have been the subject of ongoing international interest following their first description by the AMSP in 2000.[10–13] There were 2 isolates with altered susceptibility to rifampicin from Queensland.

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Australian Gonococcal Surveillance Programme annual report, 2012

Monica M Lahra for the Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme has continuously monitored antimicrobial resistance in clinical isolates of *Neisseria gonorrhoeae* from all states and territories since 1981. In 2012, 4,718 clinical isolates of gonococci from public and private sector sources were tested for *in vitro* antimicrobial susceptibility by standardised methods. Variation in antibiotic susceptibility patterns were reported between jurisdictions and regions. Resistance to the penicillins and quinolones was high in all jurisdictions except the Northern Territory and Tasmania. Penicillin resistance ranged from 21% in Western Australia to 53% in Victoria. Quinolone resistance ranged from 17% in Queensland to 46% in Victoria, and the resistance was mostly high level. Decreased susceptibility to ceftriaxone (MIC 0.06–0.25 mg/L or greater) was found nationally in 4.4% of isolates, an increase from 3.2% in 2011, but lower than in 2010. To date, there has not been an isolate of *N. gonorrhoeae* with a ceftriaxone MIC value greater than 0.125 mg/L reported in Australia. Azithromycin susceptibility testing was performed in all jurisdictions and resistance ranged from 0.3% in the Northern Territory to 2.7% in Victoria. The highest reported azithromycin MIC value was 16 mg/L and azithromycin resistant gonococci were not detected in the Australian Capital Territory or Tasmania. Nationally, all isolates remained susceptible to spectinomycin. *Commun Dis Intell* 2013;37(3):E233–E239.

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

Introduction

The World Health Organization (WHO) estimates that 106 million new *Neisseria gonorrhoeae* infections occur amongst men and women aged 15–49 years annually worldwide,[1] and 62.7% (67.4 million) of these occur in the Asia Pacific Region. In Australia, the rate of gonorrhoea increased from 35.8 per 100,000 in 2005 to 60.0 per 100,000 in 2012.[2] The increased rate of infection is coupled with a global increase in the prevalence of antimicrobial resistance (AMR) in *N. gonorrhoeae*. The potential impact of this on gonococcal disease control is a growing concern, as effective antibiotic treatment is fundamental to disease control at the population level.[3–5]

Over time, the emergence of resistance to the penicillins, tetracyclines, macrolides and fluoroquinolone antibiotics has necessitated the removal of these agents from standard treatment regimens for gonorrhoea.[6] This was followed by replacement with extended-spectrum cephalosporin (ESCs) antibiotics as the recommended first line treatment for gonorrhoea in Australia and elsewhere.[7] Importantly in Australia however, treatments based on the penicillins remain effective in many rural centres where high disease rates persist.[8]

In Australia, the injectable extended spectrum cephalosporin ceftriaxone is recommended for use in high doses.[7] However, over time there has been an increasing proportion of gonococcal isolates with raised ceftriaxone minimum inhibitory concentration (MIC) values.[8] Pharyngeal gonorrhoea treatment failures have been reported in Australia following 250 mg and 500 mg doses of ceftriaxone.[9,10] The infecting gonococcal isolates had raised ceftriaxone MIC values (range 0.016–0.06 mg/L). In 2010, the first ceftriaxone-resistant strain (MIC value: 2.0 mg/L), the H041 strain, was reported from Japan, followed by the ceftriaxone resistant F89 strain initially reported from France (MIC value 2.0 mg/L)[9,11] and subsequently from Spain.[12] In large centres in urban Australia, AMR in *N. gonorrhoeae* has long been influenced by the introduction of multi-resistant strains from overseas.[6]

Strategies for treating and controlling gonorrhoea are based on regimens effecting cure in a minimum of 95% of cases, and the formulation of these regimens is reliant on data derived from continuous AMR monitoring of gonococci to the antibiotics in clinical use.[5] The increase in ceftriaxone MIC values globally, in the absence of an ideal alternate treatment for gonococcal infection, has escalated fears for gonococcal disease treatment and control.[13] The WHO has called for enhanced surveillance as a fundamental component of the Global Action Plan to control the spread and impact of gonococcal AMR.[14]

The Australian Gonococcal Surveillance Programme (AGSP) has continuously monitored the susceptibility of *N. gonorrhoeae* since 1981 making it the longest, continually running national surveillance system for gonococcal AMR in the world.[15]

This report of the analysis of AMR in *N. gonorrhoeae* in Australia was derived from data collated by the AGSP during the 2012 calendar year.

Methods

The National Neisseria Network (NNN) of Australia comprises reference laboratories in each state and territory that collaborate to monitor clinical isolates of pathogenic *Neisseria* species nationally from as wide a section of the community as possible. The data for gonococcal isolates is collated for the AGSP, which is a product of the collaboration of the NNN laboratories. Both public and private sector laboratories refer isolates to regional testing centres. The increasing use of non-culture based methods of diagnosis has reduced the number of isolates available for testing. The number of isolates is a proportion of the number of cases of gonococcal disease notified to the National Notifiable Diseases Surveillance System (NNDSS).

Gonococci isolated in, and referred to, the NNN laboratories are examined for antibiotic susceptibility to the penicillins, quinolones, spectinomycin, third generation cephalosporins, and azithromycin and for high-level resistance to the tetracyclines. Testing is performed using previously described standardised methodology to determine the MIC values.[15,16] The MIC value is the least amount of antibiotic that inhibits *in vitro* growth under defined conditions. The AGSP conducts a program-specific quality assurance program, to ensure that data are valid and comparable for surveillance.[17]

Antibiotic susceptibility data from each jurisdiction are submitted quarterly to the coordinating laboratory, which collates the results and provides individual feedback. Additionally, where available, the AGSP collects data on the gender of the patient and site of isolation of gonococcal strains. In this report the data are further divided into urban versus rural data. Data from isolates from all jurisdictions are predominantly from urban centres, excepting the Northern Territory. Where available, data on the geographic source of acquisition of antibiotic-resistant isolates are included in analyses.

Results

Number of isolates

There were 4,784 gonococcal isolates tested in NNN laboratories in 2012, representing 35.3% of the 13,539 cases of gonococcal infection notified to the NNDSS in 2012 (Table 1).[2] This is proportionally the same as in 2011, but lower than the 40%–42% referred between 2008 and 2010.

Source of isolates

There were 3,860 isolates from men (81%) and 924 (19%) from women (Table 2). The proportion of gonococcal isolates from males and females tested by the AGSP has remained stable over recent years; ranging between 18% and 20% for women and 80% to 82% for men between 2009 and 2011.

The infected site reported as 'other' or 'not specified' for 47 isolates from males and 16 isolates from females (Table 2). Isolates from urine samples were regarded as genital tract isolates.

Antibiotic susceptibility patterns

In 2012, 4,718 of the 4,784 referred gonococcal isolates (99%) remained viable for antibiotic susceptibility testing. These were examined by the AGSP reference laboratories for susceptibility to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone

Table 1: Number of clinical gonococcal isolates tested and proportion of National Notifiable Diseases Surveillance System notifications, Australia 2012, by state and territory

State or territory	Number of isolates tested	Number of cases notified*	% isolates tested AGSP/ NNDSS
ACT	56	93	60.2
NSW	1,712	4,129	41.5
Northern Territory	335	1,539	21.8
Queensland	719	2,699	26.6
South Australia	151	479	31.5
Tasmania	14	35	40.0
Victoria	1,249	2,450	50.9
Western Australia	548	2,115	25.9
Total	4,784	13,539	35.3

Table 2: Gonococcal isolates, Australia, 2012, by sex, site of isolate and jurisdiction

	Site	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	Aust.
Male	Urethra	26	877	214	392	83	9	547	313	2,461
	Rectal	18	282	1	68	34	2	340	31	776
	Pharynx	10	279	0	37	12	2	197	25	562
	DGI	0	1	4	2	0	0	1	6	14
	Other/NS	0	10	2	3	0	0	31	1	47
	Total	54	1,449	221	502	129	13	1,116	376	3,860
Female	Cervix	2	187	108	195	19	1	112	162	786
	Rectal	0	3	0	3	0	0	2	2	10
	Pharynx	0	66	0	4	2	0	16	4	92
	DGI	0	0	6	11	0	0	0	3	20
	Other/NS	0	7	0	4	1	0	3	1	16
	Total	2	263	114	217	22	1	133	172	924
Total		56	1,712	335	719	151	14	1,249	548	4,784

DGI Disseminated gonococcal infection

NS Not specified

antibiotics), azithromycin and spectinomycin, and for high level resistance to tetracycline. As in past years the patterns of gonococcal antibiotic susceptibility differed between the states and territories, thus the data are presented by region as well as aggregated for Australia as a whole (Table 3).

Penicillins

In gonococci, resistance to the penicillin group of antibiotics (penicillin, ampicillin and amoxycillin with or without clavulanic acid), is a result of penicillinase-production by the *N. gonorrhoeae* (PPNG) and /or *N. gonorrhoeae* that are chromosomally resistant to penicillin (CMRP). Resistance in the PPNG group results from the production of beta-lactamase, and in the CMRP group by the aggregation of chromosomally-controlled resistance mechanisms.[6] Chromosomal resistance is defined by an MIC to penicillin of 1 mg/L or more.[6,16] Infections with gonococci classified as fully sensitive (FS: MIC ≤0.03 mg/L) or less sensitive (LS: MIC 0.06–0.5 mg/L) would be expected to respond to standard penicillin treatments, although response to treatment may vary at different anatomical sites.

In 2012, in total, there were 1,513 (32%) penicillin-resistant strains, a slight increase in the proportion recorded in 2010–2011 (25%–29%) but lower than the proportion in 2008–2009 (36%–44%). In 2012, there were 815 (17%) CMRP and 698 (15%) with PPNG compared with 579 (14%) CMRP and 474 (11%) PPNG in 2011. The increase in penicillin

Table 3: Proportion of isolates with resistance to penicillin, ciprofloxacin and azithromycin and decreased susceptibility to ceftriaxone reported, Australia, 2012, 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	56	2	3.6	19	33.9	0	0.0	8	14.3
New South Wales	1,701	76	4.5	539	31.7	9	0.5	482	28.3
Northern Territory	324	0	0.0	9	2.8	1	0.3	10	3.1
Queensland	708	17	2.4	120	16.9	15	2.1	183	25.8
South Australia	150	1	0.7	49	32.7	1	0.7	53	35.3
Tasmania	14	0	0.0	5	35.7	0	0.0	5	35.7
Victoria	1,249	105	8.4	572	45.8	34	2.7	666	53.3
Western Australia	516	6	1.2	123	23.8	3	0.6	106	20.5
Australia	4,718	207	4.4	1,428	30.3	63	1.3	1,513	32.1

resistance nationally in 2012 was due to increased proportions of both CMRP and PPNG, but the proportion of CMRP was similar to that of 2010.

In the Northern Territory 10 of 324 (3.1%) gonococci tested were penicillin resistant: (1 CMRP and 9 PPNG) (Table 3). This was the lowest proportion of penicillin resistance reported from the Northern Territory in recent years. Data on geographic location of acquisition were available for 57 (8.2%) of the 698 infections with PPNG. Thirty-four (4.9%) of the infections with PPNG were acquired locally, and 23 (3.3%) were acquired by overseas contact. These external contacts were principally in Western Pacific or South East Asian countries with those reported from Thailand, the Philippines, Indonesia, Vietnam, Malaysia, Papua New Guinea and Taiwan.

Ceftriaxone

From 2001 onwards, a low number of gonococcal isolates with decreased susceptibility to ceftriaxone (MIC 0.06 to 0.25 mg/L) have been found in Australia. The proportion has increased incrementally from 0.6% in 2006 to 4.8% in 2010 and decreased to 3.2% (134/4,129) in 2011. In 2012, 4.4% (207/4718) of isolates tested had decreased susceptibility to ceftriaxone (Table 3). In 2011, there was a decrease from 2010 in gonococci with decreased susceptibility to ceftriaxone in all jurisdictions except the Northern Territory and Tasmania (Table 4). This was essentially unchanged in 2012 with the exception of Victoria where there was an increase in the proportion from 5.3% to 8.4%. There were no gonococci with decreased susceptibility to ceftriaxone in Tasmania. There has not been an isolate of *N. gonorrhoeae* with an MIC value greater than 0.125 mg/L reported in Australia.

Spectinomycin

In 2012, all isolates from all jurisdictions were susceptible to this injectable antibiotic.

Quinolone antibiotics

The AGSP uses ciprofloxacin as the representative quinolone and defines altered susceptibility as an MIC of 0.06 mg/L or more.[16] In quinolone resistant *N. gonorrhoea* (QRNG), resistance is defined as a MIC value of ≥ 1 mg/L and thus far has been mediated only by chromosomal mechanisms so that incremental changes in MIC values are observed.

In 2012, 1,428 of gonococci examined (30.3%) had some level of resistance to quinolones (QRNG). The proportion reported by the AGSP in 2011 was lower (27%), but there has been a trend of decreasing proportions since 2008 when 54% isolates were reported as resistant. The majority in 2012 (1,407; 98.5%) had MICs values ≥ 1 mg/L and many of these had MIC levels of 8–64 mg/L, a similar proportion to that reported in 2010 and 2011.

During 2012 information on country of acquisition of QRNG was available for 91 (6.4%) of the 1,428 cases reported. Of these, 64 (4.5%) were acquired locally and 27 (3.7%) overseas from the same countries reported for PPNG acquisition and with contacts additionally reported from the United States of America.

Azithromycin

In 2012, data on azithromycin susceptibility was available from all states and territories. Nationally, the proportion of isolates exhibiting any resistance

Table 4: Number and percentage of gonococcal isolates with decreased susceptibility to ceftriaxone,* Australia, 2009 to 2012, by state or territory and year

	Decreased susceptibility to ceftriaxone							
	2009		2010		2011		2012	
State or territory	n	%	n	%	n	%	n	%
Australian Capital Territory	2	5.3	2	6.7	2	3.1	2	3.6
New South Wales	16	1.7	74	5.6	58	4.4	76	4.5
Northern Territory	1	0.2	1	0.2	2	0.4	0	0.0
Queensland	10	1.8	26	3.2	18	2.3	17	2.4
South Australia	9	5.3	19	11.6	1	0.7	1	0.7
Tasmania	0	0.0	0	0.0	0	0.0	0	0.0
Victoria	17	2.2	52	5.7	50	5.3	105	8.4
Western Australia	9	3.1	17	5.2	3	0.7	6	1.2
Australia	64	2.0	191	4.8	134	3.2	207	4.4

* MIC value 0.06–0.125 mg/L

was low (1.3%) similar to that reported in 2011 (1.1%) (Table 3). No isolates exhibiting high level resistance were reported.

High-level tetracycline resistance

High-level tetracycline resistant *N. gonorrhoeae* (TRNG) is used as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea and are rarely, if ever used for treatment of gonorrhoea in Australia. The proportion of TRNG detected increased between 2006 and 2010 from 12% to 21% and decreased to 18% in 2011. In 2012, there was a further decrease in TRNG nationally to 13.6% (641/4,718) of isolates reported.

TRNG were present in all jurisdictions in 2012, with the highest proportions in Western Australia (133 TRNG, 25.8%), Queensland (151, 21.3%), South Australia (25, 16.7%) and the Northern Territory (44 TRNG, 13.6%).

Discussion

Gonococcal disease control is contingent on the availability of effective treatment strategies that are informed by surveillance data. The WHO recommendations for standardised treatment regimens for gonorrhoea are based on data from epidemiological surveillance of the distribution and extent of AMR in gonococci.[5] An antimicrobial resistance rate of 5% or more in gonococci isolated from a general population is the 'threshold' for removal of an antibiotic from treatment schedules and substitution with another.[5] Programs such as the AGSP are conducted to determine the proportion of antimicrobial resistance in gonococcal strains isolated in a defined patient population and relate these findings to the likely efficacy of current treatment schedules.[5,7,16] The quality of the AMR data and the size and representativeness of the sample is imperative. For quality assurance and quality control of gonococcal AMR data, the AGSP distributes the 2008 WHO *N. gonorrhoeae* reference strain panel for use in internal quality control practices and provides the AGSP External Quality Assurance Programme.[17,18] In 2012, the AGSP examined clinical isolates from both the public and private health sectors, constituting a comprehensive sample comprising 35% of all notifications nationally. Of concern for gonococcal AMR surveillance programs is the increasing use of nucleic acid amplification testing for diagnosis of gonorrhoea in Australia and elsewhere. Currently, molecular testing strategies are unable to provide definitive data for predicting AMR, thus the continued support of surveillance programs such as the AGSP is a critical component of disease control strategies in the current context of emerging ESC resistance globally.[14]

The overall number of gonococcal strains examined by the AGSP in 2012 (4,784) was higher than the number examined in previous years. However, the proportion of isolates received from notified cases in Australia was the same as 2011 (35%) but lower than the 40%–41% examined in the years 2008 to 2010.

In 2012, 32% of gonococci nationally were resistant to penicillin, and 30% were resistant to quinolone antibiotics. These proportions were higher than those reported in 2011, where there was 25% resistance to penicillin, and 27% to the quinolone antibiotics. Over the period 2008 to 2011 there was a reduction in penicillin and quinolone resistance whereas prior to 2008 resistance to both classes of antibiotics had been increasing annually since 2003.[6] Fluctuations in the proportions of penicillin and quinolone resistance have been reported over time by the AGSP. Since 2003, aggregated data have shown a predominant clone of CMRP coupled with high-level quinolone resistance circulating with increasing frequency annually.[6,8] In 2012, the increase in the proportion of isolates with penicillin and quinolone resistance is likely to be a further reflection of the clonal shift in gonococcal isolates nationally.

The proportion of gonococci with high-level tetracycline resistance in Australia increased over the period 2006 to 2008 then stabilised at 21% in 2009 to 2010. In 2011, the proportion of TRNG decreased to 18%, with a further decrease to 13.6% in 2012.

Low rates of penicillin and ciprofloxacin resistance in the Northern Territory underscore the continued need for disaggregated surveillance data, as these are used to define treatment regimens appropriate for the various jurisdictions. Remote areas in some jurisdictions with high disease rates continue to be able to use penicillin-based treatments, but effective use of this inexpensive and acceptable treatment is contingent on continued, timely and vigilant monitoring of resistance patterns.

Decreased susceptibility to ceftriaxone is quantified by the determination of the MIC value, and encompasses the range 0.06–0.25 mg/L. The emergence and spread of gonococci with decreased susceptibility to ceftriaxone has been documented in AGSP reports.[18] These gonococci have also been found in rapidly increasing numbers in the WHO Western Pacific Region.[19] Ceftriaxone is now the standard treatment for gonorrhoea in the majority of public sector clinics. Decreased susceptibility

to the ESCs has been accompanied by increasing numbers of reports of treatment failures,[9,10,20–22] and concerns are escalating locally and globally as increasing proportions of strains with decreased susceptibility are reported.[23] To date, there have been no strains of *N. gonorrhoeae* reported in Australia with a ceftriaxone MIC value greater than 0.125 mg/L.

In Australia in 2012, the proportion of gonococcal isolates with decreased susceptibility to the ESCs (4.4%) was higher than 2011 (3.1%) but lower than the 4.8% reported in 2010. The trend has been that of incremental increases annually from the proportion reported in 2009 (2%). Surveillance to monitor *N. gonorrhoeae* with elevated MIC values coupled with sentinel site surveillance in high risk populations is critically important to inform therapeutic strategies and to detect instances of treatment failure. Sentinel site surveillance programs involve patient follow up and test of cure cultures after treatment of *N. gonorrhoeae* infections, in particular those in oropharyngeal sites. This is currently conducted in a very limited number of settings in Australia, and needs to be expanded throughout all jurisdictions as a matter of priority.

All gonococcal isolates tested in Australia in 2012 were susceptible to spectinomycin, including those with altered cephalosporin susceptibility. A low proportion of gonococci were found to be resistant to azithromycin in 2012. Recently, the United States Centers for Disease Control and Prevention, and United Kingdom gonococcal treatment guidelines have moved to recommend a dual therapy strategy of ceftriaxone with oral azithromycin for uncomplicated gonococcal infection.[24,25] Resistance to azithromycin, (which is widely used as an anti-chlamydial agent in conjunction with gonococcal treatment), has been frequently reported with very high MIC levels overseas,[26,27] but these strains have not been detected in Australia.

The continued emergence and spread of AMR in *N. gonorrhoeae* cannot be ignored. The evolution of gonococcal AMR is complex, and widely recognised as a global public health threat that requires attention to broad based disease control strategies including the rational use of antibiotics.[4,5,24,28] It is critical that disease control strategies and the understanding of the global scope of AMR continue to be informed by surveillance programs of AMR nationally and internationally.[14] Continuing maintenance of culture-based systems and commitment to surveillance of AMR in *N. gonorrhoeae* is fundamental to gonococcal disease control.

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Original articles

Impacts of a measles outbreak in western Sydney on public health resources

Kristina L Flego, Daniel A Belshaw, Vicky Sheppeard, Kathryn M Weston

Abstract

During February and March 2011, an outbreak of 26 confirmed cases of measles was reported to the Parramatta Public Health Unit (PHU) in western Sydney. This paper describes the impact of the outbreak on PHU resources. A retrospective review of information obtained from case notification forms and associated contact tracing records was carried out for each of the confirmed cases. Seven cases (27%) required hospital admission for more than 1 day and 10 (38%) cases required management within a hospital emergency department. There were no cases of encephalitis or death. The number of contacts was determined for each case as well as the number who required post-exposure prophylaxis. In total, 1,395 contacts were identified in this outbreak. Of these, 79 (5.7%) required normal human immunoglobulin and 90 (6.5%) were recommended to receive the measles-mumps-rubella vaccine. A case study detailing the PHU costs associated with the contact management of a hospitalised measles case with 75 identified contacts is also included and the estimated total cost to the PHU of containing this particular case of measles was A$2,433, with staff time comprising the major cost component. Considerable effort and resources are required to manage measles outbreaks. The total cost of this outbreak to the PHU alone is likely to have exceeded A$48,000. *Commun Dis Intell* 2013;37(3):E240–E245.

Keywords: measles, disease outbreaks, contact tracing, measles vaccine, health costs, immunisation programs

Introduction

Although Australia has declared itself to have eliminated measles,[1] imported cases continue to occur with occasional outbreaks involving local transmission amongst under-immunised groups. An outbreak of 26 cases of measles occurred within 1 local government area in western Sydney, New South Wales in February and March of 2011.

This paper describes the impact of a measles outbreak on public health unit (PHU) resources. A case study that estimates the monetary costs to the PHU associated with contact management for 1 measles case is included.

Background

On 4 February 2011, a case of measles in a 12-year-old girl was reported to the Parramatta PHU. She had not travelled recently and no source case was identified; however guests from Samoa had stayed with her family in the preceding weeks. Eleven days later, measles was notified in an 18-month-old girl, also of Samoan background but without recent travel, and with whom the index case had occasional social contact although apparently not within the estimated infectious period for the index case. Two additional cases were notified at this time; one was a 20-year-old non-Samoan woman with no recent travel history and the other was a 15-year-old boy of Samoan background. Neither of these cases reported any epidemiological connection to either of the other 2 cases.

During the next 5 weeks a further 22 confirmed cases of measles were reported, three of which were acquired overseas (Philippines). None of the cases had documented evidence of having received 2 doses of a measles-containing vaccine (MCV). Two cases were recorded on the Australian Childhood Immunisation Register (ACIR) as having received 1 dose of the measles-mumps-rubella (MMR) vaccine. In 4 other cases, a parent stated that their child had been vaccinated against measles but this could not be verified.[2]

Methods

A review of the notification and case-investigation records for each confirmed case and their contacts was conducted, specifically examining the impact on public health resources. According to the NSW Health guidelines, a confirmed case of measles requires laboratory evidence (measles virus isolation or detection by nucleic acid testing or measles IgG seroconversion or measles-virus specific IgM antibody detection) or clinical evidence and an epidemiological link.[3]

The Measles Investigation Forms completed for each case during the outbreak were reviewed. The forms contained information obtained from cases, their carers, other associated contacts, and clinicians. Details recorded included patient demographics, symptoms and onset dates, illness outcomes

(hospitalisation or death), potential exposures, and details of contacts (usually household) and their management. Complication and hospitalisation rates for cases were calculated using information recorded on the Measles Investigation Form.

Contact tracing records were also reviewed. Contact tracing and management was performed by PHU staff as per the NSW Health guidelines.[3] Whenever a healthcare setting was identified as the site of exposure, any person who shared a waiting area with a case or was in the waiting or consultation room up to 2 hours after the case during the infectious period for the case was classified as a contact. Names and contact details for staff and patients meeting the above definition were collected by staff from the healthcare setting and provided to the PHU.

Contacts were telephoned and asked about their measles vaccination status and any history of measles infection. Those born after 1965 who were not age-appropriately vaccinated against measles and who had not previously been infected with measles virus were offered post-exposure prophylaxis (PEP). This was either the MMR vaccine (up to 72 hours post-exposure) or normal human immunoglobulin (NHIG) (4 to 6 days post-exposure or for contacts with contraindications to MMR vaccine). PEP is not considered effective after 6 days (144 hours) post-exposure and so generally is not offered after this time[4] but efforts were still made to inform all contacts about their potential exposure regardless of whether prophylaxis was warranted. For infants under 6 months of age whose mother's measles immunity status was unclear, a maternal measles serum immunoglobulin G (IgG) level was urgently requested and results followed-up by PHU staff. A positive measles IgG negated the use of NHIG in both mother and child.

The total number of contacts for each case (including household contacts) and the number who required NHIG were recorded. PHU staff followed-up each contact to ensure that PEP was administered as per the protocol. An estimate of the number of contacts for whom MMR was recommended is provided; but some contacts chose the option of double-checking their vaccination history before seeking MMR vaccination. In these cases, there was no follow-up to confirm receipt of the vaccine. Wherever possible, NHIG and MMR were provided at the place where contacts were exposed to the infectious case (i.e. hospital emergency department or general practice).

Contacts that were unable to be contacted by phone were sent a letter informing them of their potential exposure to measles, advising on measles signs and symptoms and what to do if they suspected they were developing the disease.

A case study was conducted to describe the costs of following up contacts of 1 particular hospitalised case of measles. Data were prospectively gathered on the time spent by PHU staff identifying and telephoning contacts, arranging testing and PEP, and other tasks associated with contact tracing. Staff costs were calculated according to their level and it was assumed that all staff members were paid at the top increment for their award. A log of letters, faxes, and phone calls was kept and costed according to the standard rate charged by the local health district. Pathology costs were actual costs billed by the laboratory for tests ordered by the PHU. A follow-up telephone call to each contact was made 2 weeks later to determine whether any illness had arisen. Hospital medical records were reviewed to identify any visit to hospital made by contacts who could not be reached for follow-up by telephone.

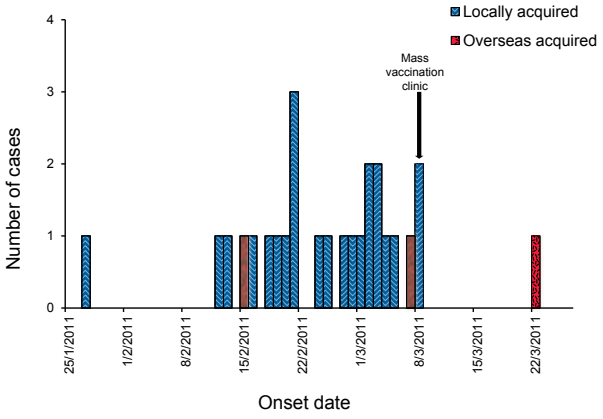
The cost to follow up 1 contact was estimated by dividing the costs for following up contacts of this case by the number of contacts for the case. The total cost of contact follow up to the PHU for the outbreak was estimated by multiplying the cost for 1 contact by the total number of identified contacts and adding the staffing cost for a high school vaccination clinic that formed part of the outbreak response.

As this work was conducted as part of routine public health control activities, review by a human research ethics committee was not required.

Results

Twenty-six confirmed cases of measles were reported to the PHU between 4 February and 29 March 2011 (Figure). The age of cases ranged between 8 months and 35 years and all cases resided within a single local government area of New South Wales. The mean and median delay

Figure: Epidemic curve of measles outbreak, western Sydney, January to March 2011, by onset date and place of acquisition



between onset of rash and notification was 2 days (range 2–8 days) and the mean and median delay between onset of illness and notification was 5 days (range 1–9 days).

Seven (27%) cases required admission to hospital for longer than 1 day. A further 10 (38%) cases were managed within a hospital emergency department. For all cases, the most common presenting symptom in addition to fever, cough, coryza, conjunctivitis and rash, was diarrhoea (23%). One case was diagnosed with pneumonitis. Another case experienced recurrent epistaxis requiring nasal packing although this was a pre-existing problem and was most likely exacerbated by, rather than a direct consequence of, measles. There were no other serious complications, including no deaths.

In total, 1,395 contacts were identified and managed by PHU staff. The average number of contacts per case was 54 (median 28). The maximum number of contacts for a single case was 206. Most contacts resulted from cases visiting large and busy general practice clinics, often on multiple occasions. Many contacts attended healthcare facilities with one or more companions, increasing the total number of contacts beyond those included on the initial list supplied by the healthcare facility (Table 1).

Table 1: Number of contacts identified during the measles outbreak, by exposure category

Household	General practice	Emergency department	Other*	Total
161	889	283	62	1,395

*

Other includes settings such as airplane and social.

Of the identified contacts, 1,241 were contacted by telephone and 154 who could not be contacted by telephone were sent a letter. Interviews identified 169 potentially susceptible contacts, of whom 79 were given NHIG and 90 were recommended to receive the MMR vaccine. There were no secondary cases amongst the contacts given PEP.

Ten cases attended a single high school and had onset dates covering a 15-day period. The absence of any other epidemiological link between these cases suggested that the entire school population was at risk. Further, none of these cases had documentation of having received a MCV, raising concern that under-vaccination was widespread in this population. In collaboration with the school, a decision was made after notification of the 4th case to hold a mass MMR immunisation clinic at the school for all staff and students who did not have documented evidence of 2 doses of a MCV. The clinic was held 7 days later and was run by 10 nursing staff and 2 medical staff from the PHU. Of the total population of 1,150 students and 100 staff, 492 students (43%) and 42 staff (42%) were vaccinated. The major cost of this clinic from the PHU perspective was attributed to nursing staff salaries (A$2,590) although the total cost of vaccine was almost 3 times this figure. However, the cost of the vaccine was not met by the PHU as it was funded by the NSW Ministry of Health.

A case study conducted for a single case estimated that the cost to the PHU of following this case was A$2,433 (Box). It can be estimated that the total cost to the PHU of contact management for the entire outbreak was at least A$48,000.

Box: Case study on Public Health Unit costs for responding to a single case of measles

The PHU received a call at 15:15 hours, 8 March 2011 about a 35-year-old pregnant patient with measles serology IgM positive and IgG negative, who had been an inpatient on the antenatal ward for the past 7 days. Six PHU staff were assigned to manage the response to the case. Seventy-five patient contacts were identified by the hospital for follow-up by PHU staff.

All contacts of the case were notified through: telephone (42), letter (25), email (7) and fax (1). Five contacts required serology to confirm current immunity, 1 contact required MMR vaccine and two required urgent NHIG. A total of 49 hours personnel time over 3 days was spent to follow-up the contacts of this single case of measles. This ranged from 3.5 hours to 17 hours per staff member assigned to the case. The average time spent per contact was 38 minutes.

Seventy per cent of the 75 contacts (n=52) were reached by a follow-up telephone call 2 weeks later. The medical records of the remaining 30% were reviewed. No secondary cases of measles were identified among the contacts.

The costs of all components of the response are listed in Table 2. Only costs borne by the PHU are included and other costs such as MMR vaccine and NHIG which are borne by other parts of the health sector (New South Wales Government) are excluded. Staff represented 90% of the total cost to the PHU of responding to this case. This is similar to the estimate derived in Iowa in 2004, where once overheads and the costs of the MMR vaccine and NHIG were removed, over 90% of measles containment costs to the PHU were attributed to staff.[5]

The cost to the PHU for this 1 case of measles was A$2,433 with staff representing the major cost component of a public health response.

Table 2: Public health unit costs for responding to a single case of measles

Item	Cost (A$2011)	% of total
Pathology	158	6.5
Telephone/mobile	59	2.4
Stationery and mail	24	1.0
Staff	2,194	90.1
Total	2,434	100.0

Genotyping of measles viruses was performed by the Victorian Infectious Diseases Research Laboratory in Melbourne, Australia. The D9 genotype (prevalent in South East Asia, Japan and Turkey in 2011[6]) was isolated from 7 cases, all of whom had definite epidemiological links to 15 other cases where genotyping was not requested. D8 (prevalent in India and the Arabian peninsula in 2011[6]) was isolated from 1 case, indicating it was an unrelated sporadic case. No source could be identified for this case, and no secondary cases arose. None of the imported cases (all from The Philippines) were able to be genotyped.

Following containment of this outbreak in March 2011, no further cases of measles were notified in western Sydney until measles was re-introduced by a returning traveller in April 2012.

Discussion

This outbreak was largely sustained by the clustering of susceptible people within a single high school. The lack of evidence of any doses of a MCV for most cases underscores the importance of MMR vaccine in controlling this disease.

Timeliness of notification, and in some cases, the time required to prepare lists of contacts directly impacted the type of control measures that could be undertaken. Late notifications increased the potential need for NHIG and very late notifications precluded the use of any PEP. The poor specificity of prodromal signs and symptoms coupled with clinician inexperience with measles resulted in delayed notification, whilst the moderately severe nature of the illness resulted in recurrent presentations to healthcare facilities, generating more potential contacts.

A significant issue for PHU management was poor documentation of vaccination history as well as confusion arising from changes to the measles vaccination schedule over the past 3 decades. Since 1996, the ACIR has been used to record the vaccination history of all Australian children up to their 7th birthday, and this was used to check the vaccination history of all contacts up to 15 years of age.[7] No centralised vaccination recording system exists for older individuals, including for vaccines administered in school-based measles control programs. Contacts may have remembered 'getting all their needles at school' but their immunisation documents were not always accessible to PHU staff. A reported history of measles infection was assumed to be correct. Contacts born before 1966 were assumed to have experienced natural infection but still needed to be contacted to determine whether they might be immunosuppressed and whether they had been accompanied by younger (and potentially susceptible) people at the time. With the frequent lack of documentation, it is possible that some recipients of measles PEP had previously received 2 doses of a MCV.

There has been recent discussion in Australia about considering the use of childcare and primary school entry as a trigger to review vaccination status. Such a measure may be helpful in future measles contact tracing activities, as children who had attended childcare or school in New South Wales would be more likely to have up-to-date ACIR records.

A 2-dose MMR schedule has been officially recommended in Australia since 1992.[8] In addition, between 1992 and 2000 a number of schedule changes and catch-up programs targeting school-aged children and young adults were undertaken. These programs complicated the risk assessment for contacts who could not provide written vaccination records. A useful tool for assessing vaccination status was a table that listed annual birth cohorts from 1966 until 1994 and which drew on historical knowledge of vaccination policy to predict whether a 2nd dose of a MCV had been offered and in which setting. If a contact stated that they received all of their school vaccinations and a 2nd dose MMR vaccine had been offered to their birth cohort when they were in school then receipt of a 2nd dose of a MCV was assumed. These strategies of assuming 2-dose vaccination or a history of disease appeared to be justified when evidenced by the lack of secondary cases reported amongst identified contacts that were assessed as not requiring PEP.

On several occasions, pregnant contacts required PEP. Measles infection during pregnancy has been associated with an increased risk of maternal and foetal complications including pre-term delivery and foetal loss.[9] Pregnant women who have received 2 doses of a MCV in their lifetime are considered protected and do not require any form of PEP under normal circumstances; however, for those with uncertain vaccination history or known susceptibility to measles, NHIG is indicated as MCVs are contraindicated in pregnancy.

Since many women of childbearing age are in the age group that is most likely to be susceptible to measles in Australia (born after 1965, but prior to introduction of the 2-dose schedule), the addition of routine antenatal testing for measles immune status along with rubella immunity, would be useful. If undertaken prior to pregnancy, vaccination could then be provided if required.

One hundred and fifty-four contacts (11%) required a letter to inform them of measles exposure. The delay in providing these contacts with information would have excluded them from the possibility of receiving PEP if it was required. Despite this, no secondary cases amongst this group were notified to the PHU, probably reflecting high levels of immunity in the general population.

The case study provides an approximation of the monetary costs incurred by the PHU in response to a single case of measles. By calculating a 'cost per contact' and multiplying this figure by the total number of contacts, then adding the cost of staffing the mass vaccination clinic held at the high school, it can be estimated that the total cost of contact management for the entire outbreak was in excess of A$48,000 (2011 A$) from the PHU perspective alone. In reality, the 'cost per contact' as calculated from this case study probably underestimates the true 'cost per contact' compared with situations where exposure occurred at a general practice or hospital emergency department. This is because many of the contacts were antenatal patients and would have been vaccinated previously or discussed vaccination. The hospital was more likely to have up-to-date contact details. Contacts may have been more co-operative and receptive to advice given that they were pregnant and have an ongoing relationship with the hospital. This contrasts with the more usual situation where contacts of measles cases are identified from an emergency department or waiting room exposure or, for instance, airplane contact. In such situations it can take considerable time to obtain lists of contacts, then even more time for PHU staff to follow–up. Such contacts may be less willing or able to cooperate with public health measures than those in this case study, as they may not have an established relationship with the health service, or they may not have a particular focus on possible risks to their health, compared with that experienced by pregnant women.

The total cost of containment efforts of an Iowa measles outbreak was US$142,452 (2004 US$). Excluding costs that were not accounted for in this case study (overheads, MMR vaccine and NHIG, transport, the costs of the public information campaign and the costs incurred by the Public Health Laboratory for Iowa) the figure for Iowa comes to US$78 734.[5] Contributors to this high cost were 2 secondary cases and 3 vaccination clinics. Over 1,000 potential contacts were traced compared with almost 1,400 contacts for the western Sydney outbreak. Although no direct comparison can be made, this calculation does suggest that the figure of A$48,000 is an underestimate. The costs did not include incident control team meetings, vehicle and transport, overtime and time-in-lieu, liaison with public health staff in other offices, preparation of clinician and media alerts, time liaising with infection control, clinical or laboratory staff, nor the costs associated with post-outbreak activities to finalise the investigations. Moreover, the total figure would be much higher if the costs incurred by all sectors of the health system including general practitioners, maternity unit staff, hospital infection control, laboratory staff and emergency departments contributing to the identification and management of contacts were included.

Enhanced surveillance contributes to the increased workload (and cost) created by a measles outbreak. During the period 25 February to 16 April 2011, 16 suspected but subsequently excluded cases of measles were reported to the Parramatta PHU. In the context of enhanced surveillance, these were considered sufficiently suspicious to be extensively followed-up. In addition, it is worth noting that many other suspected cases were reported to the Parramatta PHU (as well as to the Penrith PHU, which services the immediately adjacent population) but were excluded after initial investigation. The time taken to deal with the results of the increased awareness and reporting of suspect cases adds to the overall costs of the outbreak, particularly as heightened awareness can last for some time beyond the final case.

Neither this case study nor the Iowa study investigated opportunity costs in regards to resources utilised in responding to an infectious disease outbreak. Clinical staff administering PEP were removed from attending to other patients and PHU infectious diseases staff were occupied with measles cases and contact management with little time to work on other tasks or projects.

Conclusions

Costs associated with public health interventions should be assessed to ensure value for money, appropriate resource allocation and value for the community. Measles is a re-emerging disease of public health significance in Australia and considerable time and resources are invested in striving to control an outbreak. Maintaining Australia's measles elimination status requires extensive effort in outbreak control to reduce the number of secondary cases, their consequent morbidity and health care costs. However, of much more importance is

a robust measles vaccination strategy that achieves high level coverage, thus preventing outbreaks in the event of an imported case. Recent initiatives to improve 2-dose measles vaccine coverage, including amendments to the *Public Health Act 2010* in New South Wales to require the presentation of immunisation documentation for entry into childcare and bringing forward the 2nd MMR dose to 18 months of age should improve population immunity for measles and reduce the risk of future outbreaks.

The PHU was unable to immediately contact more than 10 per cent of the notified contacts, but through examining records of hospital admission and attendance it was determined that there were no secondary cases amongst these contacts during the period in which PEP would have been effective, suggesting that current population immunity is high enough to prevent sustained transmission. One benefit of the outbreak has been collaboration between members of the local Pacific Islander community and the PHU to provide catch-up vaccination and community education about immunisation in Australia.

Acknowledgements

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Influenza epidemiology, vaccine coverage and vaccine effectiveness in sentinel Australian hospitals in 2012: the Influenza Complications Alert Network (FluCAN)

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Abstract

Influenza is mostly a mild, self-limiting infection and severe infection requiring hospitalisation is uncommon. Immunisation aims to reduce serious morbidity and mortality. The Influenza Complications Alert Network (FluCAN) is a sentinel hospital-based surveillance program that operates at 15 sites across all states and territories in Australia. This study reports on the epidemiology of hospitalisation with confirmed influenza, estimate vaccine coverage and influenza vaccine protection against hospitalisation with influenza during the 2012 influenza season. In this observational study, cases were defined as patients admitted to one of the sentinel hospitals with influenza confirmed by nucleic acid detection. Controls were patients who had acute respiratory illnesses who were test-negative for influenza. Vaccine effectiveness was estimated as 1 minus the odds ratio of vaccination in case patients compared with control patients, after adjusting for known confounders. During the period 9 April to 31 October 2012, 1,231 patients were admitted with confirmed influenza at the 15 FluCAN sentinel hospitals. Of these, 47% were more than 65 years of age, 8% were Indigenous Australians, 3% were pregnant and 76% had chronic co-morbidities. Influenza A was detected in 83% of patients. Vaccination coverage was calculated from the vaccination status of 1,216 test negative controls and was estimated at 77% in patients 65 years or over and 61% in patients with chronic comorbidities. Vaccination effectiveness was estimated at 41% (95% CI: 28%, 51%, $P<0.001$). Vaccine coverage was incomplete in at-risk groups, particularly non-elderly patients with medical comorbidities. The study results suggest that the seasonal influenza vaccine was moderately protective against hospitalisation with influenza during the 2012 season. *Commun Dis Intell* 2013;37(3):E246–E252.

Keywords: influenza; vaccine effectiveness

Introduction

Hospitalisation due to influenza is an uncommon complication, and the case hospitalisation ratio has been estimated in the United States of America at around 0.45%.[1] However, because infection with influenza virus is relatively widespread and estimated to affect 5%–10% of the population, the incidence of hospitalisation is of significance to public health. Influenza vaccination is recommended in Australia for high risk groups, including the elderly, patients with chronic comorbidities, pregnant women and Indigenous Australians.[2] The National Immunisation Program, funded by the Australian Government and implemented by state and territory departments of health, provides public funding for influenza immunisation to reduce serious morbidity and mortality from influenza. Hospital-based surveillance is able to detect a dimension of severity not captured in a timely manner by other surveillance systems for influenza and influenza-like illnesses. This study aimed to describe the epidemiology of hospitalisation with confirmed influenza, estimate vaccine coverage in hospitalised patients with acute respiratory illnesses but without influenza, and estimate influenza vaccine protection against hospitalisation with influenza during the 2012 influenza season.

Methods

The Influenza Complications Alert Network (FluCAN) has operated since 2009.[3] In the 2012 season, the participating sites were The Alfred Hospital (Vic), Royal Melbourne Hospital (Vic), Canberra Hospital (ACT), Calvary Hospital (ACT), Monash Medical Centre (Vic), Geelong Hospital (Vic), Royal Perth Hospital (WA), Royal Adelaide Hospital (SA), Royal Hobart Hospital (Tas.), Mater Hospital (Qld), Princess Alexandra Hospital (Qld), Cairns Base Hospital (Qld), Alice Springs Hospital (NT), Westmead Hospital (NSW), and John Hunter Hospital (NSW). Ethics approval has been obtained at all participating sites and the Australian National University.

An influenza case was defined as a patient admitted to hospital with influenza confirmed by polymerase chain reaction (PCR). Surveillance was conducted from 9 April to 31 October 2012. Test negative controls (up to two for each case) were the next tested patients with acute respiratory

symptoms who were negative for influenza by PCR. Admission or transfer to the intensive care unit (ICU) included patients managed in a high dependency unit (HDU). The onset date was defined as the date of admission except for patients where date of test is more than 7 days after admission, where the onset date was the date of the test. Admissions that are listed as influenza A includes both untyped and seasonal strains, and may include infections involving the pandemic H1N1/09 strain if not specifically typed.

Vaccination coverage was estimated separately in two groups of patients. Prior to the onset of the influenza season (from study commencement on 9 April), vaccine status was collected in patients with radiologically-confirmed pneumonia. This was different by state/territory and was defined by National Notifiable Diseases Surveillance System data as follows: Australian Capital Territory (to 15 June), New South Wales (to 25 May), Northern Territory (not included, as influenza activity evident from 30 March), Queensland (22 June), South Australia (1 June), Tasmania (28 June), Victoria (8 June), and Western Australia (22 June). Subsequent to the commencement of the season, vaccine status was collected from patients admitted with influenza like illness but who were negative on influenza testing. Patients were defined as being vaccinated if they reported (as documented in the medical record or from self-report) receiving the 2012 trivalent seasonal vaccine more than 2 weeks prior to presentation. In Australia, only unadjuvanted vaccines are available under the National Immunisation Program although 1 adjuvanted vaccine is approved for use.

Vaccine effectiveness was estimated by comparing the odds of a confirmed case being vaccinated with the odds of a test negative control being vaccinated, assuming that vaccination would have no effect on admissions with non-influenza respiratory infections. This was calculated as 1 minus the odds ratio of vaccination using methods previously described.[4] A multivariate model was constructed from factors known to be associated with vaccination, and therefore potential confounders. Where the vaccine is assumed to only partially protect vaccinated individuals, the odds ratio of vaccination in cases compared with controls can be shown to be arithmetically equivalent to the relative rate of disease in vaccinated vs. unvaccinated individuals, as long as the time at risk is the same.[5–6] This has led to the development of the incidence density test design, where controls are selected from patients without influenza contemporaneous to a case.[7] A convenience sample of controls can be obtained from patients tested for influenza but who are negative for influenza using a sensitive and specific assay; this assumes that influenza vaccination has no effect on the prevention of non-influenza influenza-like illnesses (i.e. those due to other respiratory viral infections), and that these patients are generally representative of the population at risk.

Results

During the period 9 April to 31 October 2012, 1,231 patients were admitted with confirmed influenza at the 15 FluCAN sentinel hospitals. In most jurisdictions, the peak number of hospitalised cases occurred during July 2012 (Figure 1). The majority

Figure 1: Date of admission in patients hospitalised with confirmed influenza

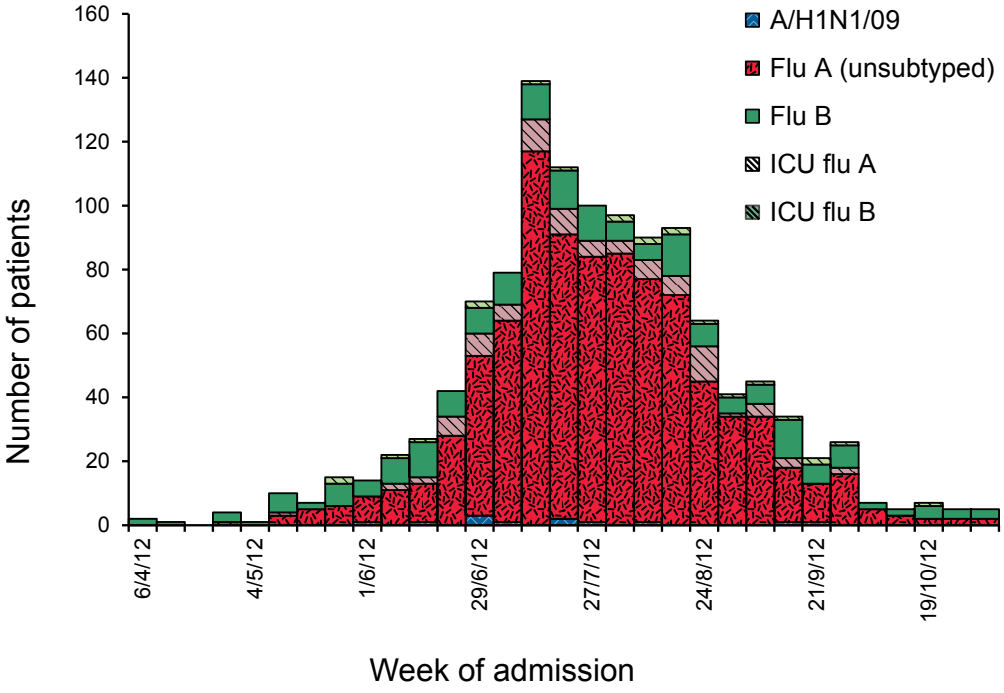

Table 1: Demographics, risk factors and outcomes in hospitalised patients with confirmed influenza

Variable	Confirmed influenza		Test negative controls	
	n	%	n	%
Total	1,231	100.0	1,694	100.0
Influenza strain				
H1N1/09	12	1.0	–	–
Flu A (unknown/ seasonal)	1,006	81.7	–	–
Flu B	213	17.3	–	–
Age group				
<18 years	148	12.0	25	1.5
18–39 years	229	18.6	185	10.9
40–64 years	281	22.8	340	20.1
65–79 years	307	24.9	398	23.5
>80 years	266	21.6	746	44.0
Male	614	49.9	869	51.3
Indigenous	99	8.0	168	9.9
State or territory				
ACT	105	8.5	30	1.8
NSW	84	6.8	137	8.1
NT	83	6.7	155	9.1
Qld	167	13.6	311	18.4
SA	200	16.2	275	16.2
Tas.	99	8.0	103	6.1
Vic.	390	31.7	546	32.2
WA	103	8.4	137	8.1
Risk factors				
Pregnancy	39	3.2	13	0.8
Nursing home resident	68	5.5	107	6.3
Medical co-morbidities*	944	76.7	1,410	83.2
Chronic respiratory disease	446	36.2	743	43.9
Diabetes	260	21.1	350	20.7
Chronic liver disease	38	3.1	71	4.2
Immunosuppressed	217	17.6	452	26.7
Chronic cardiac disease	353	28.7	495	29.2
Chronic neurological disease	175	14.2	260	15.3
Chronic renal disease	116	9.4	193	11.4
Other characteristics				
Received 2012 trivalent seasonal vaccine	437/963	45.4	689/1,216	56.7
Days from onset of illness (median, Interquartile range)	3 (2, 5) days (n=1097)		4 (2, 7) days (n=1519)	
Admitted to intensive care unit	123	10.0	272	16.1
Treated with oseltamivir	665/1,120	59.4	264/1,465	18.0
In-hospital mortality	40/1,157	3.5	49/1,413	3.4

* Multiple co-morbidities possible

of cases were due to influenza A, with 213 (17%) due to influenza B. Influenza B was more common in patients admitted to Alice Springs Hospital in the Northern Territory, accounting for 59 of 83 (71%) admitted cases.

Of these 1,231 patients, 573 (47%) were more than 65 years of age, 99 (8%) were Indigenous Australians, 39 (3%) were pregnant and 944 (77%) had chronic co-morbidities (Table 1). Of the 963 (78%) patients where influenza vaccination status was ascertained, 437 (45%) had been vaccinated. Of all cases, 108 (9%) were initially admitted to ICU and a further 15 patients were subsequently transferred to ICU after initial admission to a general ward. Of the 1,157 patients where discharge status was known, 40 (4%) patients died during the hospital admission, of which 15 (38%) patients died in intensive care.

During the surveillance period, 1,694 control patients were enrolled; of which vaccination status was ascertained for 1,216 (72%). Based on the vaccination status of patients admitted with pneumonia prior to the commencement of the season, vaccination coverage was estimated at 71% in patients aged more than 65 years and 64% in patients with chronic comorbidities. In test negative controls during the season, vaccination coverage was estimated at 78% and 61% in the elderly and those with medical comorbidities respectively (Table 2).

The effectiveness of the 2012 trivalent seasonal influenza vaccine in reducing the risk of hospitalisation with influenza was estimated at 41% (95% CI: 28%, 51%, $P<0.001$) in the 2012 influenza season (Table 3). Vaccine effectiveness was estimated to be lower for elderly patients and in those with medical comorbidities (Figure 2).

Discussion

In 2012, FluCAN recorded more than 1,200 admissions to the 15 hospitals that participate in this surveillance network, in a year where the A/H3N2 strain predominated but the vaccine match to circulating strains was good.[8] As the hospitals

Table 2: Estimated vaccine coverage in pre-season pneumonia and test-negative groups

	Test negative acute respiratory illness*		Pre-season pneumonia†	
	n/N	%	n/N	%
All patients	690/1,216	56.7	222/370	60.0
Age >65 years	420/541	77.6	171/241	71.0
Medical comorbidities	397/506	78.5	159/217	73.3
No medical comorbidities	23/35	65.7	12/24	50.0
Age <65 years	270/675	40.0	51/129	39.5
Medical comorbidities	238/532	44.7	42/95	44.2
No medical comorbidities	32/143	22.4	9/34	26.5

* The 9 April to 31 October 2012 cohort.

† Radiologically confirmed pneumonia prior to influenza season

Figure 2: Estimated vaccine effectiveness against hospitalisation in all patients, in specified subgroups and against infection with influenza subtypes

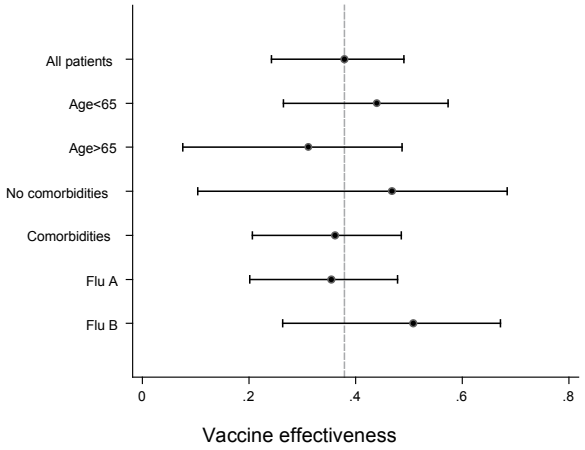

Table 3: Factors associated with hospitalisation with influenza compared with admission with non-influenza acute respiratory illnesses

Variable	Crude odds ratio	*P*	Adjusted odds ratio	*P*
Primary outcome				
Influenza vaccination	0.67 (0.56, 0.80)	<0.001	0.62 (0.51, 0.76)	<0.001
Potential confounders				
Age ≥ 65 years	1.13 (0.97, 1.32)	0.12	1.65 (1.34, 2.03)	<0.001
Medical comorbidities	0.70 (0.58, 0.85)	<0.001	0.65 (0.51, 0.83)	0.001
Pregnancy	4.99 (2.50, 9.95)	<0.001	6.05 (2.68, 13.66)	<0.001
Indigenous	1.04 (0.70, 1.54)	0.86	1.59 (0.89, 2.84)	0.12

represented in this network represent approximately 12% of the national hospital bed capacity, the cases detected here are likely to reflect approximately 10,000 admissions nationally. It is difficult to compare this with previous years as a different number of hospitals participated in 2010 and 2011, but it was noted that the age of patients was older than in the H1N1/09 dominant seasons in 2010 and 2011, and case numbers were higher.[9] It should be noted that the relative number of cases between jurisdictions does not reflect true influenza activity, due to differences in the number and size of sentinel hospitals in each jurisdiction.

The FluCAN surveillance system was established in 2009 to fill a gap between long-running, established surveillance systems based in the community and primary care, and mortality statistics.[8] We have previously demonstrated that sentinel surveillance broadly reflects population level data from notifications and ICU surveillance,[3] and had used these data to describe the clinical features of infection with the H1N1/09 strain,[10] and have previously estimated vaccine effectiveness against influenza in the 2010 and 2011 seasons.[4,11]

Influenza vaccine coverage has only been estimated infrequently in hospitalised patients in Australia.[12] An important issue is the degree to which these patients represent the population at risk of hospitalisation with respiratory illness. This study therefore estimated vaccine coverage in 2 distinct groups: patients with pneumonia prior to the influenza season, and in patients during the influenza season who had tested negative for influenza. Previous Australian studies that aggressively pursued a microbiological diagnosis have only found that influenza was implicated in only 7% of patients with pneumonia, and this is likely to be much lower outside the influenza season,[13] suggesting that influenza vaccine is not likely to be protective against pneumonia prior to the influenza season. The study found that the estimates of vaccine coverage were consistent in both groups. Self-reported vaccination status has been shown to slightly overestimate true influenza vaccination status.[12,14,15] Community-based estimates of influenza vaccine coverage, last reported in 2009, have shown that the proportion vaccinated has remained stable in periodic surveys since 2002.[16]

The effectiveness of influenza vaccines in preventing influenza has most commonly been considered in the primary care setting. A systematic review which included studies where PCR confirmation was the outcome measure has suggested that vaccine effectiveness against influenza presenting to primary care was 59%.[17] In that review, it was found that most randomised controlled trials enrolled healthy adults or children, but a smaller number of observational studies have specifically examined vaccine effectiveness in the elderly. Only 1 study was identified in this review that estimated vaccine effectiveness against hospitalisation,[18] and we are aware of a few other studies published since.[4,11,19,20] In general, results from these studies have been consistent and have shown that protection against hospitalisation ranges from 49%–61%.[4,18,19] We note that estimated vaccine effectiveness was lower in patients with comorbidities and in the elderly but this difference was not statistically significant.

There are several limitations to this study. Despite the diagnosis of influenza having both infection control and therapeutic implications in hospital, it is likely that not all patients with influenza are diagnosed. Additionally, some patients with acute respiratory infections due to influenza may test negative due to delayed presentation or secondary bacterial pneumonia after clearance of the primary infection with influenza. There also may be unmeasured confounding of the association between vaccination and admission with influenza, a bias that has plagued studies of influenza mortality.[21] In a sentinel surveillance system it is not possible to define the denominator population and therefore the true incidence of hospitalisation. Although previous studies have suggested that self-reported influenza vaccination status only slightly overestimates vaccination coverage, we have not validated this in our population.[12,14,15] In particular, differential recall bias between cases and control patients may bias estimates of vaccine effectiveness. Finally, it is difficult to reconcile studies based on diagnosed influenza with those that indirectly estimate the burden of disease from excess seasonal hospitalisations or mortality.[22]

In summary, this study detected a large number of hospital admissions with confirmed influenza in a national observational study in 2012. Vaccine coverage was low in at-risk groups, particularly non-elderly patients with medical comorbidities. The results suggest that the seasonal influenza vaccine was moderately protective against hospitalisation with influenza.

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Enhanced surveillance for gonorrhoea in two diverse settings in Queensland in the 2000s: comparative epidemiology and selected management outcomes

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Abstract

Gonorrhoea is an important sexually transmitted notifiable condition. This paper describes findings from two gonorrhoea enhanced surveillance programs operating during the 2000s in Queensland: one in the remote Torres and Northern Peninsula Area (T&NPA); the other in an urban region. The overall response rate in the T&NPA (2006–2011) was 82% (723 of 879), and in Brisbane Southside and West Moreton (BSWM) (2003–2011), it was 62% (1,494 of 2,401 notifications). In the T&NPA, cases were young (80% <25 years), Indigenous (97%) and 44% were male. In the BSWM, cases were predominantly male (76%), non-Indigenous (92%) and 42% were aged less than 25 years. Co-infection with chlamydia was found in 54% of males and 60% of females in the Torres, and in 18% of males and 35% of females in the BSWM. In the BSWM 35% of the men without a syphilis test recorded had reported sexual contact with men; similarly 34% of the men without an HIV test recorded had reported sexual contact with men. Compliance with recommended treatment (ceftriaxone) was greater than 90% in all years except 2008 (84%) in the T&NPA. Treatment compliance increased significantly, from 40% in 2003 to 84.4% in 2011 ($P<0.0001$) in the BSWM cohort. The proportion of contacts with a documented treatment date increased significantly in the T&NPA from 56% in 2009 to 76% in 2011 ($P= 0.019$), after a system for follow-up with the clinician became routine. Gonorrhoea epidemiology and management challenges vary across Queensland populations. Enhanced surveillance allows public health authorities to monitor epidemiology and reminds clinicians to prioritise effective sexually transmitted infection treatment for their clients. *Commun Dis Intell* 2013;37(3):E253–E259.

Keywords: Aboriginal and Torres Strait Islander, sexually transmitted infection, gonorrhoea, enhanced surveillance programs, epidemiology, management

Introduction

Genital gonorrhoea (*Neisseria gonorrhoeae*) is an important bacterial sexually transmitted infection (STI). It can cause acute illness and long-term morbidity (infertility, adverse pregnancy outcomes and chronic pelvic pain).[1–4] In addition, the presence of gonorrhoea (as with other STIs) increases the risk of the acquisition and transmission of HIV.[5]

Notification of gonococcal infection in Queensland rose from 1,567 cases (37.3 per 100,000 population) in 2006 to 2,697 (58.9 per 100,000 population) in 2012.[6] The burden of gonococcal infection amongst Aboriginal and Torres Strait Islander people is disproportionately high compared with other populations. In 2011, the age standardised rate of gonococcal notifications in the Queensland Aboriginal and Torres Strait Islander population was 673 per 100,000 compared with 22 per 100,000 in the non-Indigenous population.[7] In the Torres and Northern Peninsula Area (T&NPA) region of north Queensland, 176 cases of gonococcal infection were notified in 2011 (1,608 per 100,000 population).[8] In the broader Australian population notifications are concentrated among men who have sex with men while in the Aboriginal and Torres Strait Islander population cases are concentrated among youth (both males and females).

The Queensland surveillance system for *N. gonorrhoeae* infection consists of the passive notification of all laboratory confirmed cases in accordance with the (Queensland) *Public Health Act 2005*.

Quinolone resistance in gonorrhoea isolates from most regions in Australia is common. The Australian Gonococcal Surveillance Programme has reported that in 2011, 27% of all isolates nationally, and 18% of those from Queensland, demonstrated quinolone resistance.[9] Consequently, ceftriaxone rather than ciprofloxacin has been recommended as first line treatment for gonorrhoea in Australia since 2003. Co-treatment for chlamydia, testing for other STIs and treatment of sexual partners are also recommended.[10]

The T&NPA region is remote and has a population of 10,949. Of these, 83% are Torres Strait Islander and Aboriginal people[11] living in a cluster of five villages near the tip of Cape York and on 15 islands in the Torres Strait. The T&NPA

stands at the most northern tip of Queensland and borders the Western Province of Papua New Guinea (PNG) where an HIV/AIDS epidemic is established.[12] In recognition of close family ties and cultural activities across the region, the Torres Strait Treaty between Australia and PNG allows free movement for local residents to conduct traditional activities across this international border.[13] Brisbane Southside and West Moreton (BSWM) Public Health Unit (PHU) region has a largely urban population numbering 1,275,703 of whom 28,552 or 2.2% identify as Aboriginal and/or Torres Strait Islander.[11]

The objective of this paper is to describe and highlight the differences between the epidemiological and clinical management findings of gonorrhoea enhanced surveillance in these two diverse settings in Queensland. The paper illustrates how gonorrhoea enhanced surveillance may be utilised as a public health measure to monitor disease epidemiology and, potentially, to inform improvements in case management.

Methods

In the T&NPA, cases of gonorrhoea are managed in the primary care setting by nurses, medical officers and sometimes health workers. The recommended clinical management is described in the Primary Clinical Care Manual (PCCM).[14] In the BSWM, cases are managed in general practice, specialist sexual health units and public hospitals. Clinical management information is sourced from the Therapeutic Guidelines – Antibiotic,[15] or from local sexual health physicians. Enhanced surveillance for gonorrhoea notifications has been functioning in the BSWM PHU region since 2002, and in the T&NPA district of the Tropical Public Health Unit (TPHU) region from late 2005. The T&NPA enhanced surveillance program was reviewed in 2008 and refinements directed at improving contact tracing outcomes, were subsequently implemented.

In the T&NPA district, all notifications of oropharyngeal and anogenital *N. gonorrhoeae* between 1 January 2006 and 31 December 2011 were included. Clinicians were requested to complete a one page case report when treating those with a positive pathology result, and on presentation for symptomatic cases and contacts of gonorrhoea, and then to fax the report to the TPHU in Cairns. On receipt of a gonorrhoea notification, TPHU staff forwarded a case report form to the treating clinician, if one had not already been received. In the absence of a response, the treating clinician was reminded by email or telephone after 1 and 2 months. TPHU staff checked case reports for data completeness, confirmed the pathology diagnosis of gonorrhoea, checked results of other tests and (from early 2009) contacted the treating clinician after 3 to 4 weeks to document the outcomes of contact tracing.

In the BSWM PHU region, all notifications of oropharyngeal and anogenital *N. gonorrhoeae* infection between 1 January 2003 and 31 December 2011 were included. On receipt of a gonorrhoea notification, BSWM PHU staff forwarded a pack to the treating clinician containing treatment recommendations, a fact sheet for clinicians, a contact tracing letter for partners and a case report form, with a request that the latter be completed and returned to the PHU. In the absence of a response, the treating clinician was telephoned once, one month later.

The enhanced surveillance programs for the T&NPA District and the BSWM PHU region collected similar data covering case demographics and clinical presentation, sexual contact history, tests collected, treatment provided and arrangements for tracing sexual contacts. Data collected for the T&NPA cases included more detail regarding gonorrhoea culture, time to treatment and (from 2009) treatment of contacts.

This enhanced surveillance was conducted under the provisions of the (Queensland) *Public Health Act 2005*. Human research ethics committee approval was not required.

The enhanced data analysed for this report included only laboratory confirmed cases. In the TPHU, data from the case report forms of pathology positive gonorrhoea cases were entered into a Microsoft Access database. Analysis was conducted using Microsoft Excel and EpiInfo V3.3.2. In the BSWM PHU, de-identified data were stored in a Structured Query Language server database and collated for analysis using Crystal Reports. The chi-square test was used to assess the statistical significance of differences in proportions and trends at the $P<0.05$ level.[16]

Results

Response rate

There were 879 gonorrhoea notifications in the T&NPA Health Service District between 1 January 2006 and 31 December 2011. Seven hundred and twenty-three case reports were received giving a response rate of 82%. There were 2,401 gonorrhoea notifications in the BSWM PHU area between 1 January 2003 and 31 December 2011. Fourteen hundred and ninety-four case reports were received giving a response rate of 62%. The case report dataset did not differ significantly

from the total notification dataset by age group (P=0.60), gender (P=0.39) or ethnicity (P=0.99) for the T&NPA cohort, nor by age group (P=0.13) or gender (P=0.75) for the BSWM cohort.

Demographics and epidemiology

Cases

Aboriginal and Torres Strait Islander people were over-represented in both case cohorts, constituting 97% of the cases and 83% of the population in the T&NPA and 7.9% of the cases and 2.2% of the population in the BSWM. The median age of a case in the T&NPA cohort was 20 years, and in the BSWM cohort it was 26 years. In the T&NPA, the burden of diagnosed infection mainly affected young Aboriginal and Torres Strait Islander men and women compared with that in BSWM where the affected population was largely male, older and non-Indigenous (Table). Of the 23 non-Indigenous cases in the T&NPA between 2006 and 2011, 18 were Australians of PNG ethnicity living in the Torres Strait and the remainder were non-Indigenous caucasians or PNG national visitors from PNG. There was no significant change in the annual proportion of cases from PNG over the enhanced surveillance period (P=0.23).

Sexual contacts

Of the 583 contacts with stated ethnicity identified by the T&NPA cases, 556 (95%) were Aboriginal and/or Torres Strait Islander, 17 (3%) were from PNG and 10 (2%) were non-Indigenous. Between 2006 and 2011, only 10 cases identified PNG national visitor contacts. There was no change in the annual proportion of cases identifying PNG National contacts over the enhanced surveillance period (P=0.36). Prior to 2008, fewer than 10% of contacts (for whom residence was reported) came from outside remote Far North Queensland. This proportion increased significantly (approximately doubling) in each of the years 2009–11 (P<0.001). Of the 1,198 contacts identified by the BSWM cohort, the majority were non-Indigenous (947, 79%), 108 (9%) were Aboriginal and Torres Strait Islander and 143 (11.9%) were of unknown ethnicity.

Clinical presentation

In the T&NPA, being symptomatic was the most frequently reported reason for testing among males (58%,185) while females were most likely to be tested as a screen (49%, 198). In the BSWM being symptomatic was the most frequently reported reason for testing for both males (72%, 812) and females (39%, 141). Among males, reported same sex partners were uncommon in the T&NPA region: 3% (9 cases) compared with 44% (498 cases) in the BSWM cohort.

Management

Sexually transmitted infection testing

Chlamydia tests were recorded for 720 of the 723 T&NPA cases (99.6%). Of the 316 males and 404 females tested, 171 (54%) and 244 (60%) respectively returned positive chlamydia results. This compared with 1,271 (85%) chlamydia tests being recorded for the BSWM cohort, of which 178 (18.4%) males and 106 (35%) females returned a positive chlamydia result.

The proportion of cases tested for HIV in the T&NPA region in 2006 was low at 31%, but had increased significantly to 83% in 2011 (P<0.001). Over the same period, the proportion tested for syphilis also significantly increased (from 64% to 83%) (P<0.001). There was no significant difference in testing for syphilis or HIV by gender in the T&NPA cohort.

In the BSWM cohort, 50.7% and 52.4% of males were reported to have had tests for syphilis and HIV, respectively, while among females, 48% and 46% were reported to have had tests for syphilis and HIV, respectively. Many males who did not

Table: Characteristics of enhanced surveillance cases of *Neisseria gonorrhoeae* infection in the Torres and Northern Peninsular Area (2006–2011) and the Brisbane Southside and West Moreton region (2003–2011), Queensland

	Number of enhanced surveillance cases	% total notifications	% male	Number <25 years of age	% <25 years of age	Number Indigenous	% Indigenous
T&NPA	723	82.0	44.0	579	80.0	700	97.0
BSWM PHU	1,494	62.0	76.0	633	42.0	116	7.9

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T&NPA Torres and Northern Peninsular Area
BSWM PHU Brisbane Southside and West Moreton region

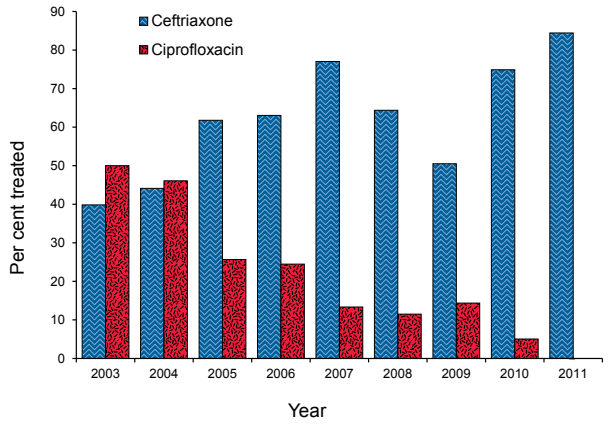
have a syphilis test recorded reported male sexual contact (195, 35%). Similarly, 184 (34%) males who did not have an HIV test recorded reported male sexual contact.

In the T&NPA cohort, 29% of males and 50% of females had a swab collected for microscopy, culture and sensitivity. Of these 296 specimens, 130 (44%) recorded *N. gonorrhoeae* growth. Isolates from only 2 cases were resistant to ciprofloxacin. One was sensitive to ceftriaxone, the other (in 2011, from a non-Indigenous male with contacts from outside the T&NPA), was also the only isolate documented as being less than fully sensitive to ceftriaxone. Data on the findings of culture from specimens from the BSWM cohort are not available.

Antibiotic treatment

Documented treatment with the recommended first line treatment (ceftriaxone) in the T&NPA region was greater than 90% in all years other than 2008 (84%). Reported co-treatment for chlamydia was greater than 90% in all years other than 2006 (84%). For the BSWM cohort, there was a significant increase in the proportion of cases treated with ceftriaxone during the study period, from 40% in 2003 to 84.4% in 2011 ($P<0.0001$), while at the same time ciprofloxacin (quinolone) use decreased from 50% (54 of 108 cases) in 2003 to zero of 199 cases in 2011 ($P<0.0001$) (Figure). In the BSWM, females were slightly more likely to receive ceftriaxone (RR 1.1, 95% CI 1.01–1.18, $P=0.05$) and ceftriaxone use did not vary by Indigenous status (RR 1.08; 95% CI 0.97–1.3).

Figure: Treatment for genital gonorrhoea over time for the Brisbane Southside and West Moreton Public Health Unit gonorrhoea enhanced surveillance cohort



Time to treatment

Of the 247 T&NPA screening cases (i.e. were not symptomatic or a contact of a case), 159 (64%) were treated within 14 days and 211 (85%) within a month of testing. The proportion of T&NPA cases presenting as a contact or with symptoms, and treated for both gonorrhoea and chlamydia at presentation in accordance with the regional recommendation,[14] was greater than 70% in all years other than 2006 (60%, range 60%–85%). Within this group, symptomatic women were the least likely to be treated correctly at presentation. Time to treatment data was not collected for the BSWM cohort.

Contact tracing

Contact tracing in the T&NPA cohort was almost universally initiated by the treating clinician. The annual proportion of T&NPA cases who identified zero contacts ranged from 5% to 11% with no significant trend. Treatment dates for contacts of T&NPA cases were recorded from 2009. Documented treatment of at least 1 contact for each case increased from 56% in 2009, and 60% in 2010, to 76% in 2011 ($P=0.019$). In contrast, in the BSWM PHU region, contact tracing was predominantly initiated by the case (1,024; 72.6%) with contact tracing being undertaken by the treating clinician in only 43 (2.9%) cases overall. The annual proportion of contact tracing initiated by either case (range: 65%–81%) or clinician (range: 1.5%–5.5%) did not change over the study period ($P=0.53$).

Discussion

These findings highlight the diverse epidemiology of gonorrhoea across these two Queensland populations, and illustrate the role that enhanced surveillance programs can play in monitoring epidemiological and management challenges. In remote areas to the north, gonorrhoea (often in combination with chlamydia) is common and largely affects Indigenous youth, while in the south it is more frequently diagnosed in older non-Indigenous males. Aboriginal and Torres Strait Islander people were over-represented in the enhanced surveillance cohorts in both the T&NPA and BSWM regions. This is consistent with findings from across Australia[7] and reflects the global disadvantage experienced by Indigenous youth in this area. In north Queensland, this disadvantage is evidenced in low levels of knowledge and awareness of personal risk,[17] poor access to condoms,[18] and inadequate implementation of school based sexuality and relationships education in Indigenous majority schools.[19]

The enhanced surveillance findings reiterate the importance of early detection and high quality clinical management of STI in the primary care setting for remote-living Australian youth. The findings for the BSWM cohort suggest that despite the clinical recommendation to test for co-infection (with syphilis or HIV), many men who have sex with men who are at increased risk are not being tested. The extent to which the enhanced surveillance process contributed to observed management improvements in either the T&NPA or BSWM regions is arguable. However, the process itself regularly reminded clinicians of the correct management for gonorrhoea and of the importance of following through on contact tracing processes. It also sent a message that public health services prioritised effective management of the common STI.

Given the high prevalence of quinolone resistance nationally, it is noteworthy that the strain of *N. gonorrhoea* circulating in remote Far North Queensland where ciprofloxacin has not been widely used to treat gonorrhoea over the last decade, remained largely sensitive to quinolones, suggesting fairly closed sexual networks. There was however, a significant increase in the proportion of contacts from further south on the Australian mainland in the last few years of the enhanced surveillance period. This increasing mobility of remote area youth within Australia increases the likelihood of quinolone (and other antibiotic) resistant strains, as indeed it also increases the risk of HIV entering the T&NPA population from mainland Australian sexual contact. The movement of local residents across the border with PNG presents further potential for spread of communicable diseases across the Torres Strait. In this context, monitoring the epidemiology of STIs, especially in an era when PNG is enduring a significant epidemic of HIV/AIDS,[12] is important. In this period of enhanced surveillance, there was no significant change in the proportion of cases of gonorrhoea that were diagnosed in PNG national visitors each year, a finding that may reflect no change in local sexual network behaviours or varying testing practices over time. Over the same period, there was no significant change in the annual proportion of cases naming PNG national visitor contacts.

Gonorrhoea is a common clinical presentation in the T&NPA and since the early 2000s, regional treatment guidelines (the PCCM) have recommended (both) ceftriaxone as first line treatment, and co-treatment for chlamydia. This is likely to explain the high level of adherence to the recommended antibiotic regime. On the other hand, the proportion of gonorrhoea cases tested for syphilis and for HIV improved significantly, as did reported contact tracing outcomes. There was a significant increase in the proportion of cases with a documented treatment date for at least 1 contact once the public health nurses established the system for a follow-up telephone call to request a contact treatment date. In the BSWM region there was a highly significant improvement in adherence to the antibiotic recommendations for the treatment of gonorrhoea infections. However, in the BSWM, key areas for targeted education and improvement remain, most particularly in relation to co-treatment for chlamydia and testing for concurrent infection, especially for HIV and syphilis, and contact tracing.

Limitations

While noting that ethnicity could not be assessed in the BSWM, the response rates and demographic similarities between the T&NPA and the BSWM enhanced surveillance cohorts and all gonorrhoea notifications, are satisfactory. This suggests that the findings are likely to be representative of gonorrhoea notifications in each region during the study period – a view supported for the T&NPA by the similarity of our findings with those of a recent analysis of the epidemiology of gonorrhoea notifications in Indigenous Australians nationally for 2000–2009.[20] The extent to which either cohort was representative of gonorrhoea infection in each population is unknown. In the T&NPA, testing and treatment information was usually verifiable, but data about contacts relied on the information provided by the case and recorded by the treating clinician, and is likely to be of variable quality. For example, same sex partnerships may have been under-estimated for the T&NPA cohort.

The levels of recorded testing for other STI (chlamydia, syphilis and HIV) in the T&NPA and BSWM regions were likely to have been influenced by differences in data collection methods. For example, staff in the TPHU complemented the case report data by checking the single pathology database in use in the region for any test results. The presence of multiple pathology providers in the BSWM suggests that this would not have been possible for BSWM staff and is likely to have resulted in lower estimates for the latter region.

Conclusion

This analysis illustrates the different epidemiology and management challenges posed by genital gonorrhoea across two diverse populations in Queensland. The required health system responses can be informed by the enhanced surveillance data. It also demonstrates that despite its remoteness, the T&NPA district was able to achieve substantial

improvements and ultimately a high standard of clinical management for gonorrhoea by the end of the surveillance period.

In addition to continuing efforts to reduce the morbidity caused by chlamydia and gonorrhoea, Far North Queensland is now confronting an era of increasing vulnerability to more serious STIs, both syphilis[21] and HIV. In the face of economic constraints affecting health expenditure at the regional, state and national levels, renewing efforts in surveillance, and enhancing prevention and treatment services in remote areas should remain a high priority.

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Conflicts of interest

None declared.

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Quarterly report

OzFoodNet quarterly report, 1 July to 30 September 2012

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. In each Australian state and territory, OzFoodNet epidemiologists investigate outbreaks of enteric infection. 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 occurred in Australia between 1 July and 30 September 2012.

Data were received from OzFoodNet epidemiologists in all Australian states and territories. The data in this report are provisional and subject to change, as additional outbreak data may have been collected following the collation of data for this report.

During the 3rd quarter of 2012, OzFoodNet sites reported 614 outbreaks and clusters of enteric illness, including those transmitted by contaminated food. In total, these outbreaks affected 11,560 people, of whom 268 were hospitalised. There were 49 deaths reported during these outbreaks. This compares with a 5 year mean between 2007 and 2011 of 610 outbreaks affecting 11,825 people, 275 hospitalisations and 39 deaths for the 3rd quarter. 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. The majority of outbreaks during the 3rd quarter of 2012 (86%, n=531) were due to person-to-person transmission (Table 1), with 57% (304/531) of these occurring in residential aged care facilities.

Foodborne and suspected foodborne disease outbreaks

There were 31 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as being the primary mode of transmission. These outbreaks affected 241 people, and resulted in 16 hospitalisations and 1 death. This compares with 31 outbreaks in the 2nd quarter of 2012[1] and a 5 year mean of 30 outbreaks for the 3rd quarter between 2007 and 2011.

Salmonella Typhimurium was identified as the aetiological agent in 9 outbreaks (29%) during this quarter (Table 2). Of the remaining outbreaks, 3 (10%) were due to norovirus, 2 (6%) were due to ciguatera fish poisoning, and 1 each due to scombroid poisoning, Shiga toxin-producing *Escherichia coli* (STEC), *Clostridium perfringens*, *Listeria monocytogenes,* and *Campylobacter*. In 12 outbreaks (39%), the aetiological agent was unknown.

Fifteen outbreaks (48% of foodborne or suspected foodborne outbreaks) reported in this quarter were associated with food prepared in restaurants (Table 3).

To investigate the 31 outbreaks, sites conducted 4 cohort studies, 1 case control study and collected descriptive case series data for 24 investigations. No individual patient data were collected for 2 outbreaks. The evidence used to implicate food included analytical evidence in 3 outbreaks and microbiological evidence in 3 outbreaks. Descriptive evidence alone was obtained in 25 outbreak investigations.

Table 1: Outbreaks and clusters of gastrointestinal illness reported by OzFoodNet, 1 July to 30 September 2012, by mode of transmission

Transmission mode	Number of outbreaks and clusters	Per cent of total
Foodborne and suspected foodborne	31	5
Waterborne and suspected waterborne	1	<1
Person-to-person	531	86
Unknown (*Salmonella* cluster)	7	1
Unknown (other pathogen cluster)	4	1
Unknown	40	7
Total	614	100*

* Percentages do not add up due to rounding.

Table 2: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites,* 1 July to 30 September 2012 (n=31)

State	Month†	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
NSW/Vic.	July	Commercially manufactured	*Listeria monocytogenes* ST 1/2a,3a, BT 58, PFGE 18A:17A:10	3	3	A	Suspected smoked salmon
NSW	July	Restaurant	Unknown	9	0	D	Unknown
NSW	August	Restaurant	Unknown	10	0	D	Mushroom sauce
NSW	August	Commercial caterer	*Salmonella* Typhimurium PT 170 / MLVA profile 03-09-08-14-523	14	0	D	Raw egg mayonnaise
NSW	August	Restaurant	Unknown	3	0	D	Unknown
NSW	August	Unknown	*S.* Typhimurium MLVA profile 03-27-08-21-496	9	0	D	Unknown
NSW	August	Restaurant	*S.* Typhimurium PT 135 / MLVA profile 03-17-09-12-523	2	0	M	Unknown
NSW	August	Aged care	*S.* Typhimurium PT 170 / MLVA profile 03-09-07-13-523	3	2	D	Unknown
NSW	August	Restaurant	Unknown	5	0	D	Unknown
NSW	September	Restaurant	Unknown	10	0	D	Unknown
NSW	September	Restaurant	Unknown	5	0	D	Unknown
NT	September	Camp	Shiga toxin-producing *Escherichia coli*	5	1	D	Kangaroo meat
Qld	July	Restaurant	Norovirus genotype II	6	0	D	Oysters
Qld	July	Restaurant	Unknown	5	0	D	Unknown
Qld	July	Private residence	Scombroid	4	0	M	Fresh mullet fillets
Qld	July	Restaurant	*Clostridium perfringens*	7	0	A	Lamb curry
Qld	August	Restaurant	*S.* Typhimurium PT 16 / MLVA profile 03-13-11-11-524	3	3	D	Chicken Caesar salad with raw egg dressing
Qld	August	Private residence	Ciguatera fish poisoning	2	0	D	Coral trout
Qld	September	Private residence	Ciguatera fish poisoning	2	0	D	Coral trout
Qld	September	Aged care	*S.* Typhimurium PT 16 / MLVA profile 03-13-10-11-524	14	0	D	Unknown
SA	July	Restaurant	*Campylobacter*	15	1	A	Chicken liver pâté
SA	July	Bakery	*S.* Typhimurium PT 9	8	3	D	Unknown
SA	September	Restaurant	*S.* Typhimurium PT 9	11	1	M	Fried ice cream made using raw eggs

Table 2 *continued*: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet sites,* 1 July to 30 September 2012 (n=31)

State	Month†	Setting prepared	Agent responsible	Number affected	Hospitalised	Evidence	Responsible vehicles
Tas.	July	Commercial caterer	Unknown	5	0	D	Unknown
Tas.	July	Commercial caterer	Unknown	10	0	D	Unknown
Vic.	July	Private residence	*S.* Typhimurium PT 135a	7	0	D	Chocolate mousse containing raw eggs
Vic.	August	Restaurant	Unknown	5	0	D	Unknown
Vic.	August	Unknown	Unknown	2	1	D	Unknown
Vic.	September	Restaurant	Norovirus	43	0	D	Unknown
WA	September	Bakery	Norovirus	4	1	D	Multiple foods
WA	September	Camp	Unknown	10	0	D	Unknown
Totals				241	16		

* No foodborne or suspected foodborne outbreaks were reported by the Australian Capital Territory

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

A Analytical epidemiological association between illness and 1 or more foods

BT Binary type

D Descriptive evidence implicating the suspected vehicle or suggesting foodborne transmission

M Microbiological confirmation of agent in the suspected vehicle and cases

MLVA Multi-locus variable number tandem repeat analysis

PFGE Pulsed-field gel electrophoresis

PT Phage type

ST Serotype

Table 3: Outbreaks of foodborne or suspected foodborne disease reported by OzFoodNet, 1 July to 30 September 2012 by food preparation setting

Food preparation setting	Outbreaks
Restaurant	15
Private residence	4
Commercial caterer	3
Aged care	2
Bakery	2
Camp	2
Unknown	2
Commercially manufactured	1
Total	31

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

Australian Capital Territory

There were no reported outbreaks of foodborne or suspected foodborne illness during the quarter.

New South Wales

There were 10 reported outbreaks of foodborne or suspected foodborne illness during the quarter. The aetiological agent was identified in 4 outbreaks; all were due to *S.* Typhimurium. The aetiology remains unknown for the other 6 outbreaks.

Description of key outbreaks

Over an 8 week period, a cluster of cases of *S.* Typhimurium multi-locus variable number tandem repeat analysis (MLVA) profile* 03-09-08-14-523 [phage type (PT) 170] infection were notified to authorities. Nine cases reported eating a beef, egg and mustard sandwich from an event kiosk on the day before their onset of illness. Three other cases reported eating egg sandwiches or an egg salad with mayonnaise at a catered work function. The same catering company supplied food for both events. A further 2 cases reported working directly or indirectly for the catering company and had consumed the foods prepared. The New South Wales Food Authority (NSWFA) inspected the catering premises on 2 occasions and found that the sandwiches contained mayonnaise made by the caterer using raw egg and that it was possible that all the sandwiches consumed by cases contained mayonnaise from the same batch. The caterers are now using a commercial mayonnaise. No batch code details were available to enable trace-back to the farm.

Investigators identified a potential point source outbreak during a review of a cluster of salmonellosis (*S.* Typhimurium PT 135 MLVA profile 03-17-09-12-523). A group of 49 people attended a function at a restaurant where the menu was buffet style with a variety of poultry, other meat dishes and salads. Two of the 9 people interviewed became ill with nausea, vomiting, abdominal pain and diarrhoea 48 hours after consuming the meal. Both cases were confirmed as having *S.* Typhimurium (MLVA profile 03-17-09-12-523) but otherwise did not know each other. The NSWFA conducted an inspection of the premises and did not identify any problems. NSWFA attempted to trace back to the chicken suppliers; however this did not yield any extra information. See also *Cluster investigations*.

Northern Territory

There was one reported outbreak of foodborne or suspected foodborne illness during the quarter.

A group of 7 people from a remote Indigenous community all ate kangaroo that was killed, cooked and eaten in a bush setting.[3] Five of the group experienced diarrhoea and vomiting, with three suffering from bloody diarrhoea and subsequently required hospital attention. One patient was admitted to hospital. Stool samples were taken from the 3 cases with bloody diarrhoea and 1 case tested positive for the stx_2 toxin gene produced by STEC. Multiplex polymerase chain reaction testing confirmed this result. No food samples were collected for testing.

Queensland

There were 8 reported outbreaks of foodborne or suspected foodborne illness during the quarter. The aetiological agent was identified in seven of these outbreaks: two each were due to *S.* Typhimurium and ciguatera fish poisoning; and one each due to *C. perfringens,* norovirus and scombroid fish poisoning.

Description of key outbreaks

Seven of a group of 12 people who shared a common meal at a restaurant developed diarrhoea and stomach cramps between 3 and 15 hours following a meal that included lamb curry, chicken, beef rendang, and pork. Food samples and environmental swabs from the restaurant kitchen tested negative for bacterial pathogens. A retrospective cohort study identified that persons who consumed lamb curry had an elevated risk of illness [relative risk (RR) 3.0;

* MLVA profiles are reported using the Australian coding convention agreed at a MLVA typing harmonisation meeting in Sydney in November 2011.[2]

95% confidence interval (CI) 1.0 to 9.3; $P=0.06$]. Four stool samples were positive for *C. perfringens* with vegetative cell counts ranging between 3.8×10^5 and 5.7×10^7 cells/g and faecal spore counts ranging between 2.1×10^6 and 3.9×10^7 spores/g. *C. perfringens* isolates were confirmed as Type A, but the *C. perfringens* enterotoxin gene was not detected. Control measures included thorough cleaning of the restaurant and advice to improve handling and temperature monitoring of cooked foods.

Fourteen residents of an aged care facility experienced illness with symptoms including diarrhoea, vomiting, stomach cramps and fever. *S.* Typhimurium PT 16 (MLVA profile 03-13-10-11-524) was isolated from the stools of 8 residents. No hospitalisations were reported. A wide variety of food items were consumed in the week prior to illness (including vitamised meals). Extensive environmental samples were collected for microbiological testing as well as food samples including eggs. All samples tested negative for *Salmonella*. Investigators were unable to identify a food vehicle or source of infection. The MLVA profile and phage types of case isolates suggests that this outbreak may have had a common source of infection with another outbreak investigated during the quarter in which 3 cases of salmonellosis (*S.* Typhimurium PT 16 MLVA profile 03-13-11-11-524) all consumed Caesar salad. This was prepared using a raw egg dressing at a sporting club restaurant. No common exposures between the 2 outbreaks were found.

South Australia

There were 3 outbreaks of foodborne or suspected foodborne illness investigated during the quarter. The aetiological agents were identified as *S.* Typhimurium PT 9 for 2 outbreaks and *Campylobacter* for the remaining outbreak.

An outbreak of *Campylobacter* occurred following a private function in a commercial restaurant. A retrospective cohort study was conducted amongst the 57 party guests.[4] All of the guests completed the questionnaire, 15 of whom met the case definition. Analysis of the data showed a significant association between illness and the consumption of a chicken liver pâté (RR 16.7; 95% CI 2.4 to 118.6; $P<0.001$). No other food or beverage served at the party was associated with illness. Three guests submitted stool samples, all tested positive for *Campylobacter* and one was further identified as *Campylobacter jejuni*. The environmental investigation noted that the cooking process used in the preparation of chicken liver pâté may have resulted in some portions not being cooked adequately to inactivate potential *Campylobacter* contamination.

Investigators identified an outbreak of *S.* Typhimurium PT 9 after 11 cases of gastrointestinal illness were found to be associated with dining at or receiving takeaway food from a particular restaurant. All 11 cases had consumed fried ice cream at the restaurant. Nine of 11 cases submitted faecal samples and all 9 were confirmed as having *S.* Typhimurium PT 9 infection. An environmental investigation was conducted and *S.* Typhimurium PT 9 was identified from the breadcrumb plate used to prepare the fried ice cream. Ice cream with raw egg coating was rolled in these breadcrumbs.

Tasmania

There were 2 reported outbreaks of foodborne or suspected foodborne illness during the quarter affecting a total of 15 people. The outbreaks were clustered and were linked to the same catering company but no pathogen could be identified.

Victoria

There were 4 reported outbreaks of foodborne or suspected foodborne illness during the quarter. The aetiological agent was identified for two of these outbreaks with one each due to *S.* Typhimurium and norovirus.

Investigators were notified of an outbreak of salmonellosis in a family who became ill after sharing a meal that consisted of roast beef, vegetables and chocolate mousse containing raw eggs. All 7 attendees ate the mousse and all became ill approximately 24 hours later. The mousse contained free range eggs. There were no leftover eggs for testing and no batch code details were available to enable trace back to the farm.

An outbreak of gastroenteritis affecting 6 people from a group of 12 who dined at a hotel was reported to a council in regional Victoria. Authorities received a second complaint of illness affecting a large group of 38 people who had dined at the same hotel restaurant a week later. In total, 7 separate groups reported illness after dining at the hotel. Sixty-two interviews were conducted with 3 affected groups and food handlers, and 43 people were identified as cases. Illness was not associated with the consumption of any specific food or drink items. Eight food handlers were interviewed and 6 reported having a gastrointestinal illness but stated that they remained away from work for the recommended time. However, 1 food handler became ill whilst at work and is known to have prepared some food just prior to his illness onset. This food was then served over subsequent days. One faecal sample was submitted by a case and was positive for norovirus. Despite being unable to definitively identify the mode of

transmission for this cluster of related outbreaks, the incubation period for each of the affected groups and the high attack rate (74%) for the large group of 38 suggests that cases were exposed to a common source such as food that may have been intermittently contaminated by an infectious food handler or a contaminated environment. Findings that support this hypothesis include the absence of adequate handwashing facilities in the kitchen, and no documented clean up after the first outbreak.

Western Australia

There were 2 reported outbreaks of foodborne or suspected foodborne illness during the quarter. Norovirus was identified as the aetiological agent for one of these outbreaks.

An increase in gastroenteritis in a rural town was reported by hospital staff. Of the 17 people investigated, 12 were from the community and 5 were relatives of a food business owner. Of the 12 community cases, 9 were interviewed and all reported diarrhoea and/or vomiting. Four of these 9 cases had reported eating a range of food items from a food business prior to illness, including bread rolls containing salad (n=3) and a meat pie (n=1). None of the 4 community cases reported contact with an ill person prior to their illness. The median incubation period was 30 hours (range 20–40 hours) and the median duration of diarrhoea was 24 hours. Two cases had faecal specimens positive for norovirus, including one who had eaten food from the food business. Of the 5 ill relatives of the food business proprietor, three presented at hospital with diarrhoea and/or vomiting and at least one of the five was an employee at the implicated food business. The food business owner was given advice on cleaning and sanitising the food preparation areas and exclusion of ill workers. The evidence suggested that illness was due to norovirus and the 4 community cases had suspected foodborne illness due to an ill food handler preparing food. However, as norovirus was also circulating in the community at the time, it is possible that the 4 cases who ate food from the implicated food business may have acquired their illness via person-to-person transmission.

Cross-jurisdictional investigation

Through its National Enhanced Listeriosis Surveillance System (NELSS), OzFoodNet identified an outbreak of suspected foodborne illness involving 3 cases of *Listeria monocytogenes* serotype (ST) 1/2a,3a, binary type (BT) 58, pulsed-field gel electrophoresis (PFGE) pattern 18A:17A:10. NELSS commenced in January 2010 and contained no record of this strain being previously isolated from humans. Cases occurred in January (New South Wales), late May (Victoria) and early June (Victoria). One case died. A case–case analysis using data from NELSS involved 3 cases and 56 controls. In univariate analysis, only smoked salmon had a statistically significant association between consumption and illness; however, as all 3 cases consumed smoked salmon in the 28 days prior to illness onset, an odds ratio (OR) could not be calculated (OR undefined; 95% CI 2.2 to undefined; $P=0.02$). All 3 cases consumed the same brand of smoked salmon but 2 of the cases also consumed other brands of smoked salmon. The Microbiological Diagnostic Unit at the University of Melbourne has maintained PFGE typing data of *Listeria* isolates collected from environmental and food samples since 1995. While the dataset is not complete and does not contain a systematic record of food testing, there was no historical laboratory evidence of *L. monocytogenes* ST 1/2a,3a, BT 58, PFGE 18A:17A:10 in smoked salmon.

Health authorities met with the company of interest and concluded that it had an extensive program for *Listeria* identification and control in place. The company's test and hold criteria for fish products exceeded the current microbiological requirements of the Australia New Zealand Food Standards Code.[5] Twenty samples of ready-to-eat cold smoked salmon of varying brands, batches and date coding sampled from retail sites were all negative for *Listeria*.

Cluster investigations

During the quarter, OzFoodNet sites investigated multiple clusters of illness due to a range of pathogens, with five being due to *S.* Typhimurium, two due to other *Salmonella* serotypes, and one each due to *Campylobacter*, *Cryptosporidium*, *Giardia* and *Yersinia enterocolitica*. No particular source or transmission mode could be identified in the clusters.

An investigation into a *S.* Typhimurium cluster with a novel MLVA profile of 03-17-09-12-523 was commenced in New South Wales in July. Phage typing of clinical samples from the cluster identified the organism as *S.* Typhimurium PT 135. Sixty-nine cases were reported in the quarter, with a total of 41 cases interviewed using a hypothesis generating questionnaire. A trawling questionnaire was completed for 35 cases. Place of residence for cases varied across New South Wales. Frequently consumed foods for cases included fresh pre-cut chicken (88%), fresh beef cuts (60%), carrots (71%), cooked onions (63%), apples (63%) and bananas (57%). Fresh chicken purchased from large supermarket retailers was a feature of the cluster. The NSWFA conducted a trace back investigation based on place of purchase information provided

by cases, and identified 3 predominant chicken suppliers. NSWFA approached the 3 suppliers, and one supplier noted that they had seen the same phage type in samples collected during the year and supplied the isolates for MLVA typing. These isolates were found to have the same MLVA pattern as the clinical isolates (MLVA profile 03-17-09-12-523). The supplier subsequently introduced a series of changes in the production process and the number of new cases decreased. One outbreak was identified as a result of this cluster investigation (reported above and in Table 2).

Comments

The majority of reported outbreaks of gastrointestinal illness in Australia are due to person-to-person transmission, and in this quarter, 86% of outbreaks (531/614) were transmitted via this route. The number of foodborne outbreaks this quarter (n=31) was the same as the previous quarter and consistent with the 5-year mean (2007–2011) of 30 outbreaks. *S.* Typhimurium was identified as the aetiological agent in 9 (29%) of the foodborne or suspected foodborne outbreaks during this quarter (Table 2). In fact, all (9/9) outbreaks with known *Salmonella* aetiology were due to this serotype. Of the 9 outbreaks where *Salmonella* was implicated as the responsible agent, 44% (4/9) were associated with raw or undercooked egg products (including raw egg dressings, raw/undercooked egg dessert and fried ice cream).

NELSS was established in 2010 in response to a *L. monocytogenes* outbreak affecting both an airline and catering company in 2009.[6,7] The surveillance scheme involves serotyping, binary typing and further molecular characterisation, including PFGE, of all clinical isolates of *L. monocytogenes*. An exposure history for each case is also recorded where available. In this quarter, NELSS allowed the identification of a small *Listeria* outbreak with just 3 cases in 2 jurisdictions with consumption of a smoked salmon brand common to all cases, indicating that the surveillance system is sensitive.

A limitation of the outbreak data provided by OzFoodNet sites for this report was the potential for differences in how investigators interpreted circumstances and classified and categorised features of the outbreaks. Changes in the number of foodborne outbreaks should be interpreted with caution due to the small number each quarter.

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National Notifiable Diseases Surveillance System, 1 April to 30 June 2013

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 104,327 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 April and 30 June 2013 (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	
Arbovirus infection (NEC)	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
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 April to 30 June 2013, by date of diagnosis*

Disease	State or territory								Total this quarter 2013	Total last quarter 2012	Total this quarter 2012	Last 5 years mean this quarter	Ratio	Year to date 2013	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)†	1	9	2	10	2	0	14	6	44	45	34	55.4	0.8	89	113.0
Hepatitis B (unspecified)‡	19	567	155	194	77	18	457	280	1,767	1,645	1,583	1,624.0	1.1	3,402	3,305.4
Hepatitis C (newly acquired)†	2	2	0	NN	10	6	22	24	66	108	116	106.0	0.6	172	207.8
Hepatitis C (unspecified)‡	48	835	98	594	86	48	469	306	2,484	2,464	2,248	2,581.2	1.0	4,937	5,234.0
Hepatitis D	0	3	0	5	1	0	6	1	16	14	10	11.2	1.4	30	20.6
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	1	0	1	2	0	0.2	5.0	3	0.4
Campylobacteriosis	70	NN	61	792	432	136	1,338	364	3,193	3,515	3,270	3,591.4	0.9	6,667	8,131.6
Cryptosporidiosis	12	290	33	210	49	19	379	125	1,117	1,736	919	684.0	1.6	2,842	1,930.2
Haemolytic uraemic syndrome	0	0	1	0	0	0	1	0	2	4	5	4.0	0.5	6	9.2
Hepatitis A	0	11	0	10	1	0	14	6	42	70	37	74.2	0.6	110	142.6
Hepatitis E	0	4	0	1	0	0	1	0	6	15	8	9.6	0.6	21	24.0
Listeriosis	0	7	0	3	1	0	4	2	17	29	21	17.2	1.0	46	44.6
STEC, VTEC§	0	6	0	5	11	0	3	1	26	45	23	18.2	1.4	71	50.6
Salmonellosis	114	844	93	608	270	54	712	334	3,029	4,190	2,454	2,423.6	1.2	7,180	6,225.2
Shigellosis	2	20	32	22	5	0	25	9	115	145	119	138.8	0.8	258	332.2
Typhoid	0	17	0	5	0	0	6	1	29	69	21	24.0	1.2	97	67.2
Quarantinable diseases															
Cholera	0	0	0	0	0	0	1	0	1	0	4	1.8	0.6	1	2.6
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	1.0	0.0	0	1.0

Table 2 *continued:* Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2013, by date of diagnosis*

Disease	State or territory								Total this quarter 2013	Total last quarter 2012	Total this quarter 2012	Last 5 years mean this quarter	Ratio	Year to date 2013	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection[‖,¶]	334	5,099	745	4,778	1,346	429	4,992	3,019	20,742	21,045	20,359	18,272.0	1.1	41,633	36,763.6
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	0.6
Gonococcal infection[‖]	22	1,042	523	717	262	22	775	472	3,835	3,768	3,454	2,713.2	1.4	7,564	5,361.2
Syphilis – congenital	0	2	0	0	0	0	0	0	2	1	0	0.6	3.3	3	1.8
Syphilis < 2 years duration[‖]	1	121	3	61	20	4	142	23	375	443	395	337.4	1.1	815	688.8
Syphilis > 2 years or unspecified duration[‡,‖]	4	105	49	50	26	3	135	64	436	403	308	329.0	1.3	834	664.0
Vaccine preventable diseases															
Diphtheria	0	0	0	0	0	0	0	0	0	1	0	0.8	0.0	1	0.8
Haemophilus influenzae type b	0	2	0	2	0	0	1	0	5	3	4	6.8	0.7	8	11.2
Influenza (laboratory confirmed)	58	269	38	649	421	12	494	209	2,510	2,341	7,338	5,624.6	0.4	4,824	6,687.4
Measles	0	9	0	4	1	0	3	0	17	10	31	22.4	0.8	27	65.2
Mumps	1	33	0	12	0	1	2	9	58	74	69	46.4	1.3	131	105.4
Pertussis	37	501	28	834	137	88	589	322	2,536	3,634	5,384	5,709.4	0.4	6,137	12,435.8
Pneumococcal disease (invasive)	5	135	12	86	35	7	104	54	438	214	505	462.0	0.9	652	678.0
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	4	0	1	1	0	1	0	7	2	9	10.2	0.7	9	23.0
Rubella – congenital	0	0	0	0	1	0	0	0	1	0	0	0.0	0.0	1	0.0
Tetanus	0	1	0	0	0	0	0	0	1	3	0	0.6	1.7	4	2.4
Varicella zoster (chickenpox)	1	NN	19	48	101	8	176	80	433	373	458	401.2	1.1	804	755.0
Varicella zoster (shingles)	20	NN	66	8	490	51	290	339	1,264	1,210	1,119	805.8	1.6	2,460	1,646.8
Varicella zoster (unspecified)	30	NN	1	1,196	34	23	658	297	2,239	2,279	2,037	1,630.2	1.4	4,480	3,284.6
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	5	0	0	0	0	5	5	3	2.4	2.1	10	5.8
Barmah Forest virus infection	1	133	168	828	23	2	24	361	1,540	1,427	332	387.8	4.0	2,959	1,012.4
Dengue virus infection	1	68	14	182	15	3	94	118	495	489	419	220.4	2.2	980	697.6
Japanese encephalitis virus infection	0	0	0	0	1	0	0	1	2	0	0	0.0	0.0	2	0.2
Kunjin virus infection	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	1.0
Malaria	2	14	2	24	2	5	18	18	85	137	73	104.6	0.8	222	212.6
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	2.0	0.0	0	4.4
Ross River virus infection	0	197	72	595	36	1	34	363	1,298	1,349	1,199	1,345.0	1.0	2,642	3,617.4

Table 2 *continued:* Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2013, by date of diagnosis*

Disease	State or territory								Total this quarter 2013	Total last quarter 2012	Total this quarter 2012	Last 5 years mean this quarter	Ratio	Year to date 2013	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	1	0	0.0	0.0	1	0.0
Brucellosis	0	0	0	4	0	0	0	0	4	6	3	8.0	0.5	9	15.8
Leptospirosis	0	3	2	32	0	0	1	1	39	16	47	43.4	0.9	54	104.0
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	4	0	0	0	0	11	0	15	8	15	18.8	0.8	22	36.0
Q fever	0	45	0	77	3	0	15	2	142	97	81	81.6	1.7	238	177.0
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.5
Other bacterial infections															
Legionellosis	0	29	1	29	13	2	15	22	111	99	89	89.8	1.2	208	164.0
Leprosy	0	0	1	0	0	0	0	2	3	2	1	2.0	1.5	5	3.4
Meningococcal infection**	1	6	1	9	5	0	3	3	28	39	67	61.4	0.5	67	106.4
Tuberculosis	3	104	9	43	9	0	83	31	282	309	270	278.4	1.0	589	580.2
Total	**789**	**10,901**	**2,229**	**12,733**	**3,927**	**942**	**12,113**	**7,269**	**50,903**	**53,884**	**54,941**			**104,327**	

* Date of diagnosis = the true diagnosis. If this is not available, the 'date of onset' is equivalent to the earliest of two dates: (i) specimen date of collection, or (ii) the date of notification to the public health unit. Hepatitis B and C unspecified were analysed by the date of notification.

† Newly-acquired hepatitis includes cases where the infection was determined to have been acquired within 24 months prior to diagnosis.

‡ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined.

§ Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli.*

ll In the national case definitions for chlamydial, gonococcal and syphilis infection, the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode.

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for in South Australia where only genital tract specimens are reported, and the Northern Territory and Western Australia where ocular specimens are excluded, and Western Australia also excludes perinatal infection.

** Only invasive meningococcal disease is nationally notifiable. However, New South Wales, the Australian Capital Territory and South Australia 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 April to 30 June 2013, 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)‡	1.1	0.5	3.4	0.9	0.5	0.0	1.0	1.0	0.8
Hepatitis B (unspecified)§	20.3	31.1	263.6	17.0	18.6	14.1	32.5	46.0	31.1
Hepatitis C (newly acquired)‡	2.1	0.1	0.0	NN	2.4	4.7	1.6	3.9	1.5
Hepatitis C (unspecified)§	51.2	45.7	166.7	52.0	20.8	37.5	33.3	50.3	43.8
Hepatitis D	0.0	0.2	0.0	0.4	0.2	0.0	0.4	0.2	0.3
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Campylobacteriosis	74.7	NN	103.7	69.4	104.3	106.2	95.1	59.9	82.9
Cryptosporidiosis	12.8	15.9	56.1	18.4	11.8	14.8	26.9	20.6	19.7
Haemolytic uraemic syndrome	0.0	0.0	1.7	0.0	0.0	0.0	0.1	0.0	0.0
Hepatitis A	0.0	0.6	0.0	0.9	0.2	0.0	1.0	1.0	0.7
Hepatitis E	0.0	0.2	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Listeriosis	0.0	0.4	0.0	0.3	0.2	0.0	0.3	0.3	0.3
STEC,VTEC‖	0.0	0.3	0.0	0.4	2.7	0.0	0.2	0.2	0.5
Salmonellosis	121.6	46.2	158.2	53.3	65.2	42.2	50.6	54.9	53.4
Shigellosis	2.1	1.1	54.4	1.9	1.2	0.0	1.8	1.5	2.0
Typhoid fever	0.0	0.9	0.0	0.4	0.0	0.0	0.4	0.2	0.5
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
Human pathogenic avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmitted infections									
Chlamydial infection¶,**	356.4	279.4	1,267.1	418.6	325.1	334.9	354.7	496.4	365.3
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection¶	23.5	57.1	889.5	62.8	63.3	17.2	55.1	77.6	67.5
Syphilis – congenital	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syphilis < 2 years duration¶	1.1	6.6	5.1	5.3	4.8	3.1	10.1	3.8	6.6
Syphilis > 2 years or unspecified duration¶,§	4.3	5.8	83.3	4.4	6.3	2.3	9.6	10.5	7.7
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.2	0.0	0.0	0.1	0.0	0.1
Influenza (laboratory confirmed)	61.9	34.5	64.6	56.9	101.7	9.4	35.1	34.4	44.2
Measles	0.0	0.5	0.0	0.4	0.2	0.0	0.2	0.0	0.3
Mumps	1.1	1.8	0.0	1.1	0.0	0.8	0.1	1.5	1.0
Pertussis	39.5	27.4	47.6	73.1	33.1	68.7	41.9	52.9	44.7
Pneumococcal disease (invasive)	5.3	7.4	20.4	7.5	8.5	5.5	7.4	8.9	7.7
Poliomyelitis	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	0.1	0.2	0.0	0.1	0.0	0.1
Rubella – congenital	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0
Tetanus	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Table 3 *continued:* Notification rates of diseases, 1 April to 30 June 2013, by state or territory. (Annualised rate per 100,000 population)*,†

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Vaccine preventable diseases, *cont'd*									
Varicella zoster (chickenpox)	1.1	NN	32.3	4.2	24.4	6.2	12.5	13.2	11.2
Varicella zoster (shingles)	21.3	NN	112.3	0.7	118.3	39.8	20.6	55.7	32.8
Varicella zoster (unspecified)	32.0	NN	1.7	104.8	8.2	18.0	46.8	48.8	58.1
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.1
Barmah Forest virus infection	1.1	7.3	285.7	72.5	5.6	1.6	1.7	59.4	27.1
Dengue virus infection	1.1	3.7	23.8	15.9	3.6	2.3	6.7	19.4	8.7
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.2	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	0.8	3.4	2.1	0.5	3.9	1.3	3.0	1.5
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	0.0	10.8	122.5	52.1	8.7	0.8	2.4	59.7	22.9
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	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.1
Leptospirosis	0.0	0.2	3.4	2.8	0.0	0.0	0.1	0.2	0.7
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.2	0.0	0.0	0.0	0.0	0.8	0.0	0.3
Q fever	0.0	2.5	0.0	6.7	0.7	0.0	1.1	0.3	2.5
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	0.0	1.6	1.7	2.5	3.1	1.6	1.1	3.6	2.0
Leprosy	0.0	0.0	1.7	0.0	0.0	0.0	0.0	0.3	0.1
Meningococcal infection††	1.1	0.3	1.7	0.8	1.2	0.0	0.2	0.5	0.5
Tuberculosis	3.2	5.7	15.3	3.8	2.2	0.0	5.9	5.1	5.0

* Date of Diagnosis = the true diagnosis. If this is not available, the 'date of onset' is equivalent to the earliest of two dates: (i) specimen date of collection, or (ii) the date of notification to the public health unit. Hepatitis B and C unspecified were analysed by the date of notification.

† 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.

§ Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined.

|| Infection with Shiga-like toxin (verotoxin) producing *Escherichia coli*.

¶ In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode (e.g. perinatal infections, epidemic gonococcal conjunctivitis).

** Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for in South Australia where only genital tract specimens are reported, and the Northern Territory and Western Australia where ocular infections are excluded, and Western Australia also excludes perinatal infection.

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

NEC Not elsewhere classified.

NN Not notifiable.

Australian childhood immunisation coverage, 1 April to 30 June cohort, assessed as at 30 September 2012

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

Introduction

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

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months, for 3-month birth cohorts of children at the stated ages between 1 April and 30 June 2012. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, pneumococcal conjugate, varicella, and meningococcal C conjugate vaccines, and is outlined in more detail below.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of three doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of all other hepatitis B vaccines. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 or 4 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 or 4 doses of Comvax hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 or 5 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

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

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

Results

The percentage of children 'fully immunised' by 12 months of age for Australia decreased marginally from the previous quarter by 0.1 of a percentage point to 91.8% (Table 1). There were no important changes in coverage for any individual vaccines due at 12 months of age or by jurisdiction.

The percentage of children 'fully immunised' by 24 months of age for Australia increased marginally from the previous quarter by 0.5 of a percentage point to 92.8% (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

Table 1. Percentage of children immunised at 12 months of age for the birth cohort 1 April to 30 June 2011, preliminary results, by disease and state or territory; assessment date 30 September 2012

	State or territory								
Vaccine	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Total number of children	1,329	24,738	1,001	15,954	4,945	1,523	17,807	8,223	75,520
Diphtheria, tetanus, pertussis (%)	94.1	92.1	93.1	92.5	92.5	92.5	92.9	90.9	92.3
Poliomyelitis (%)	94.1	92.0	93.1	92.5	92.4	92.5	92.9	90.9	92.3
Haemophilus influenzae type b (%)	94.0	91.9	93.1	92.4	92.4	92.3	92.7	90.7	92.2
Hepatitis B (%)	93.2	91.7	93.0	92.2	92.1	92.5	92.5	90.4	91.9
Fully immunised (%)	93.1	91.6	92.8	92.1	92.0	92.3	92.3	90.3	91.8
Change in fully immunised since last quarter (%)	-0.0	-0.1	-1.4	+0.4	-0.6	-0.8	-0.4	+0.2	-0.1

Table 2. Percentage of children immunised at 24 months of age for the birth cohort 1 April to 30 June 2010, preliminary results, by disease and state or territory; assessment date 30 September 2012*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,327	24,735	934	15,925	4,896	1,494	18,152	8,150	75,613
Diphtheria, tetanus, pertussis (%)	96.3	95.0	95.8	94.5	95.3	95.4	95.7	93.7	95.0
Poliomyelitis (%)	96.3	95.0	95.9	94.5	95.3	95.4	95.7	93.6	95.0
Haemophilus influenzae type b (%)	96.0	95.4	96.0	94.5	95.3	95.6	95.7	93.8	95.1
Measles, mumps, rubella (%)	94.7	94.1	94.9	94.0	94.4	94.7	94.7	92.6	94.1
Hepatitis B (%)	95.6	94.7	95.7	94.1	94.9	95.1	95.2	92.7	94.5
Fully immunised (%)	93.4	92.8	93.8	92.6	93.1	93.9	93.6	90.6	92.8
Change in fully immunised since last quarter (%)	+0.7	+0.8	-1.9	+0.0	+0.9	+0.3	+0.6	+0.5	+0.5

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2011;35(4):331.

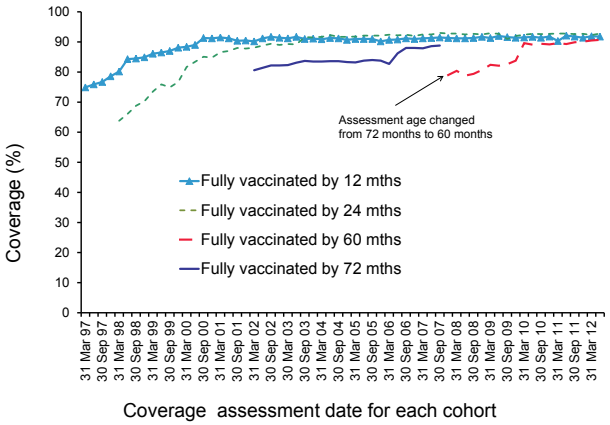
Table 3. Percentage of children immunised at 60 months of age for the birth cohort 1 April to 30 June 2007, preliminary results, by disease and state or territory; assessment date 30 September 2012

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,232	24,830	946	16,142	4,901	1,548	17,895	8,186	75,680
Diphtheria, tetanus, pertussis (%)	93.2	91.2	90.0	91.1	89.7	93.1	92.4	89.5	91.2
Poliomyelitis (%)	93.0	91.1	90.1	91.1	89.5	93.0	92.3	89.5	91.2
Measles, mumps, rubella (%)	92.8	91.0	90.1	91.0	89.5	93.1	92.2	89.2	91.0
Fully immunised (%)	92.5	90.7	89.5	90.5	89.1	92.9	91.8	88.9	90.7
Change in fully immunised since last quarter (%)	+1.6	+0.1	-0.8	-0.5	+0.3	+2.1	+0.2	+1.3	+0.2

The percentage of children 'fully immunised' by 60 months of age for Australia increased from the previous quarter by 0.2 of a percentage point to 90.7% (Table 3). This continues the upward trend in coverage for this age milestone. There were no important changes in coverage for any individual vaccines due at 60 months of age or by jurisdiction.

There is a clear trend of increasing vaccination coverage over time for children aged 12 months, 24 months and 60 months (from December 2007) (Figure). Coverage at 60 months of age is close to the coverage levels attained at 12 and 24 months.

Figure: Trends in vaccination coverage, Australia, 1997 to 30 June 2012, by age cohorts



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

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

Introduction

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

The data show the percentage of children 'fully immunised' at 12 months, 24 months and 60 months, for 3-month birth cohorts of children at the stated ages between 1 October and 31 December 2012. 'Fully immunised' refers to vaccines on the National Immunisation Program Schedule, but excludes rotavirus, pneumococcal conjugate, varicella, and meningococcal C conjugate vaccines, and is outlined in more detail below.

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of three doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of PRP-OMP containing *Haemophilus influenzae* type b (Hib) vaccine or 3 doses of any other Hib vaccine, and 2 or 3 doses of Comvax hepatitis B vaccine or 3 doses of all other hepatitis B vaccines. 'Fully immunised' at 24 months of age is defined as a child having a record on the ACIR of 3 or 4 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 or 4 doses of Comvax hepatitis B vaccine or 4 doses of all other hepatitis B vaccines, and 1 dose of a measles, mumps and rubella-containing (MMR) vaccine. 'Fully immunised' at 60 months of age is defined as a child having a record on the ACIR of 4 or 5 doses of a DTP-containing vaccine, 4 doses of polio vaccine, and 2 doses of an MMR-containing vaccine.

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

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

Results

The percentage of children 'fully immunised' by 12 months of age for Australia decreased marginally from the previous quarter by 0.1 of a percentage point to 91.5% (Table 1). There were no important changes in coverage for any individual vaccines due at 12 months of age or by jurisdiction.

The percentage of children 'fully immunised' by 24 months of age for Australia decreased marginally from the previous quarter by 0.4 of a percentage point to 92.2% (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

Table 1. Percentage of children immunised at 12 months of age for the birth cohort 1 October to 31 December 2011, preliminary results, by disease and state or territory; assessment date 31 March 2013

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	1,343	23,908	864	14,854	4,962	1,558	18,439	7,791	73,719
Diphtheria, tetanus, pertussis (%)	92.9	91.5	91.0	92.7	91.9	92.8	92.5	91.5	92.1
Poliomyelitis (%)	92.9	91.4	91.0	92.7	91.9	92.7	92.4	91.4	92.0
Haemophilus influenzae type b (%)	92.7	91.3	91.0	92.7	91.7	92.5	92.3	91.3	91.9
Hepatitis B (%)	92.6	91.0	91.1	92.4	91.5	92.4	92.0	90.7	91.6
Fully immunised (%)	92.4	90.9	91.0	92.3	91.4	92.4	91.8	90.6	91.5
Change in fully immunised since last quarter (%)	-0.0	-0.3	+0.4	+0.2	-0.2	-0.6	-0.4	+0.6	-0.1

Table 2. Percentage of children immunised at 24 months of age for the birth cohort 1 October to 31 December 2010, preliminary results, by disease and state or territory; assessment date 31 March 2013*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,218	23,671	887	14,639	4,858	1,509	17,842	7,908	72,532
Diphtheria, tetanus, pertussis (%)	95.7	94.3	95.2	94.5	94.4	96.3	95.2	93.3	94.5
Poliomyelitis (%)	95.7	94.2	95.2	94.5	94.4	96.3	95.1	93.3	94.5
Haemophilus influenzae type b (%)	95.9	94.8	95.6	94.6	94.4	96.4	95.2	93.4	94.8
Measles, mumps, rubella (%)	94.2	93.6	94.4	94.1	93.5	95.8	94.1	92.4	93.7
Hepatitis B (%)	95.1	93.9	94.5	94.0	94.1	96.2	94.7	92.4	94.0
Fully immunised (%)	92.9	91.9	93.1	92.6	92.3	94.9	92.7	90.4	92.2
Change in fully immunised since last quarter (%)	-0.3	-0.4	-0.4	-0.1	+0.3	+1.0	-0.8	-0.4	-0.4

* The 12 months age data for this cohort were published in *Commun Dis Intell* 2012;36(2):E203.

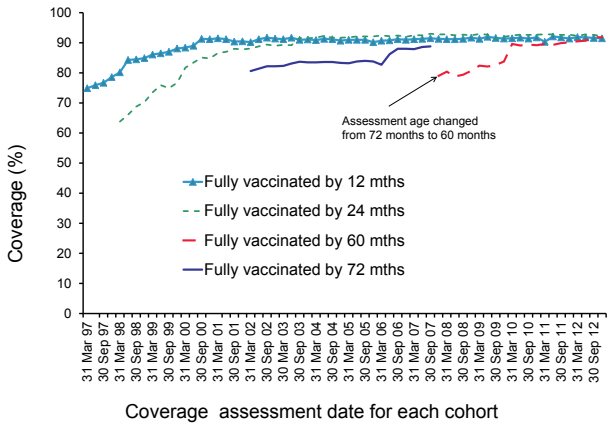
Table 3. Percentage of children immunised at 60 months of age for the birth cohort 1 October to 31 December 2007, preliminary results, by disease and state or territory; assessment date 31 March 2013

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas.	Vic.	WA	
Total number of children	1,248	24,807	884	15,537	5,054	1,639	18,797	8,273	76,239
Diphtheria, tetanus, pertussis (%)	94.0	92.3	91.7	92.4	91.9	92.1	93.5	90.0	92.4
Poliomyelitis (%)	94.0	92.3	91.7	92.3	91.8	92.0	93.4	89.9	92.3
Measles, mumps, rubella (%)	94.0	92.0	91.7	92.3	91.6	92.2	93.2	89.8	92.1
Fully immunised (%)	93.7	91.7	91.4	91.9	91.1	91.8	92.9	89.3	91.8
Change in fully immunised since last quarter (%)	+1.7	+0.1	+0.9	-0.1	-0.4	-1.6	-0.3	-0.5	-0.1

The percentage of children 'fully immunised' by 60 months of age for Australia decreased marginally from the previous quarter by 0.1 of a percentage point to 91.8% (Table 3). This maintains the improvement in coverage for this age milestone. There were no important changes in coverage for any individual vaccines due at 60 months of age or by jurisdiction.

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

Figure: Trends in vaccination coverage, Australia, 1997 to 31 December 2012, by age cohorts



Australian Gonococcal Surveillance Programme, 1 January to 31 March 2013

Monica M Lahra for the Australian Gonococcal Surveillance Programme

Introduction

The Australian National Neisseria Network reference laboratories in each state and territory report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly for the Australian Gonococcal Surveillance Programme (AGSP). The antibiotics routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, which are current or potential agents used for the treatment of gonorrhoea. Azithromycin testing is now performed by all states and territories as it has a role as part of a dual therapy regimen in 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 are also provided on other antibiotics from time to time. The AGSP has a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. These data are presented quarterly in tabulated form, below, as well as in the AGSP annual report. For more information see *Commun Dis Intell* 2013;37(1):E61.

Results

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. Total penicillin resistance includes penicillinase producing NG (PPNG); and chromosomally mediated resistance to penicillin (CMRP). Quinolone resistant NG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 1.0 mg/L.

Azithromycin resistance is reported as a MIC to azithromycin equal to or greater than 1.0 mg/L. Of note in this quarter 2013, there were 2 isolates reported in Australia with high level resistance (HLR) with azithromycin resistance (MIC value >256 mg/L). There was 1 isolate reported from Victoria, acquired in China; and the other from Queensland where information regarding acquisition was not available. These are the first reports of HLR to azithromycin reported in Australia.

Ceftriaxone MIC values in the range 0.06–0.25 mg/L are reported as having decreased susceptibility (DS). To date there has not been an isolate reported in Australia with a ceftriaxone MIC value >0.125 mg/L. In this quarter 2013 there was a marked increase in the proportion of NG isolates with DS to ceftriaxone nationally, (8.5%), compared with 3.5% in 2012 and 2.7% in 2011, and 6.1% during the same quarter in 2010. This increase was predominantly from Victoria where the proportion of NG isolates with DS to ceftriaxone rose from 6.7% during the same quarter of 2012 to 15% in

Table: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to ciprofloxacin, azithromycin and penicillin, Australia, 1 January to 31 March 2013, by state or territory

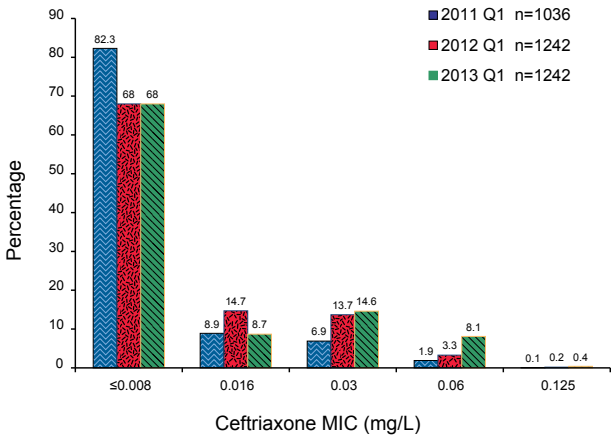
State or territory	Number of isolates tested	Decreased susceptibility		Resistance					
		Ceftriaxone		Ciprofloxacin		Azithromycin		Penicillin	
		n	%	n	%	n	%	n	%
ACT	18	0	0.0	3	17.0	1	5.6	1	5.6
NSW	394	38	9.6	125	32.0	11	2.8	147	37 .0
NT	103	1	0.97	10	9.7	0	0.0	10	9.7
Qld	154	3	1.9	40	26.0	6	3.9	56	36 .0
SA	34	1	2.9	12	35.0	3	8.8	11	32.0
Tas.	9	0	0.0	4	44.0	0	0.0	2	22.0
Vic.	389	60	15 .0	181	46.0	6	1.5	200	51.0
WA	141	3	2.1	27	19.0	0	0.0	36	25.0
Aust	1,242	106	8.5	402	32.0	27	2.1	463	37.0

2013; and New South Wales where the increase was from 3.8% in the 1st quarter of 2012 to 10% in this quarter 2013. Of the 60 strains from Victoria with ceftriaxone DS there were 59 (98%) that were multi-drug resistant (resistant to penicillin and high level resistance to ciprofloxacin); all were from males; and 23/60 (38.3%) were isolated from extra genital sites (rectal and pharyngeal). From New South Wales, there were 38 strains with decreased susceptibility to ceftriaxone and of those, 29/38 (76%) were multi-drug resistant; 33/38 (87%) were from males and 16/38 were isolated from extra genital sites (rectal and pharyngeal). The majority of strains from Victoria and New South Wales that had decreased susceptibility to ceftriaxone had a multi-drug resistant phenotype suggesting clonal spread, although this would need to be confirmed by typing studies.

There are recent reports of ceftriaxone 500 mg treatment failure in Victoria and New South Wales. These patients had pharyngeal infections where the gonococcal strains had ceftriaxone MIC values in the range 0.03–0.06 mg/L.[2,3] Patients with infections in extra genital sites, where the isolate has decreased susceptibility to ceftriaxone, are recommended to have a test to confirm a cure.

In South Australia, 2.9% of isolates were reported with DS in the first quarter 2013, compared with no isolates in the same quarter of 2012. In Western Australia and the Australian Capital Territory the proportion of strains with DS was lower than in the same quarter 2012; and in Queensland there was a small increase from 1.4% in this quarter in 2012 to 1.9% in this quarter 2013. The proportion of NG strains at each MIC value is shown in the Figure, where it can be seen that the greatest increase was in the proportion of isolates with an MIC value of 0.06–0.125 mg/L.

Figure: Distribution of ceftriaxone MIC values in gonococcal isolates tested in the AGSP, 1 January to 31 March, 2011 to 2013



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2. Read PJ, Limnios EA, McNulty A, Whiley D, Lahra MM. One confirmed and one suspected case of pharyngeal gonorrhoea treatment failure following 500 mg ceftriaxone in Sydney, Australia. *Sex Health* 2013;10(5):460–462.
3. Chen M, Stevens K, Tideman R, Zaia A, Fairley CK, Lahra MM, Hogg G. Failure of 500 mg of ceftriaxone to eradicate pharyngeal gonorrhoea, Australia. *J Antimicrob Chemother* 2013;68(6):1445–1447.

Australian Gonococcal Surveillance Programme, 1 April to 30 June 2013

Monica M Lahra for the Australian Gonococcal Surveillance Programme

Introduction

The Australian National Neisseria Network reference laboratories in each state and territory report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly for the Australian Gonococcal Surveillance Programme (AGSP). The antibiotics routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, which are current or potential agents used for the treatment of gonorrhoea. Azithromycin testing is now performed by all states and territories as it has a role as part of a dual therapy regimen in 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 are also provided on other antibiotics from time to time. The AGSP has a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. These data are presented quarterly in tabulated form, below, as well as in the AGSP annual report. For more information see *Commun Dis Intell* 2013;37(1):E61.

Results

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. Total penicillin resistance includes penicillinase producing NG (PPNG); and chromosomally mediated resistance to penicillin (CMRP). Quinolone resistant NG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 1.0 mg/L.

Azithromycin resistance is reported as a MIC to azithromycin equal to or greater than 1.0 mg/L. There were no isolates reported in Australia with high level resistance with an azithromycin (MIC value >256 mg/L) in this quarter.

Ceftriaxone MIC values in the range 0.06–0.25 mg/L are reported as having decreased susceptibility (DS). To date there has not been an isolate reported in Australia with a ceftriaxone MIC value >0.125 mgL. In the 2nd quarter 2013 there was a further increase to 10.9% in the proportion of NG isolates with DS to ceftriaxone nationally, compared with 3.4% in 2012 and 3.6% in 2011, and 5.1% during the same quarter in 2010. An increase in the proportion of NG isolates with DS was seen in all jurisdictions with the exception of the Australian Capital Territory. This increase was predominantly from New South Wales where the proportion of NG isolates with DS to ceftriaxone rose to 15% from 3.8% during the same quarter 2012; and Victoria with an increase to 12% in this quarter 2013 from 5.9% in the 2nd quarter 2012. The majority of isolates with decreased susceptibility to ceftriaxone were also multi-drug resistant,

Table: Gonococcal isolates showing decreased susceptibility to ceftriaxone and resistance to ciprofloxacin, azithromycin and penicillin, Australia, 1 April to 30 June 2013, by state or territory

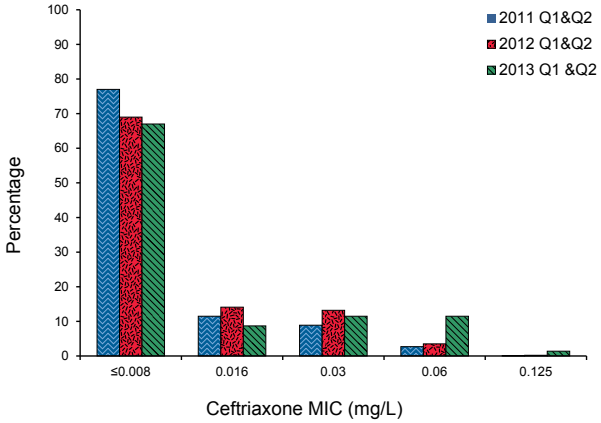
State or territory	Number of isolates tested	Decreased susceptibility		Resistance					
		Ceftriaxone		Ciprofloxacin		Azithromycin		Penicillin	
		n	%	n	%	n	%	n	%
ACT	8	0	0.0	0	0.0	0	0.0	1	12.0
NSW	412	63	15.0	168	41.0	3	0.7	156	38.0
NT	73	1	1.4	4	5.5	0	0.0	5	6.9
Qld	184	15	8.2	58	31.0	10	5.4	60	33.0
SA	63	2	3.2	16	25.0	0	0.0	11	18.0
Tas.	15	5	33.0	6	40.0	0	0.0	6	40.0
Vic.	423	49	12.0	180	43.0	16	3.8	161	38.0
WA	102	4	3.9	28	28.0	0	0.0	25	25.0
Aust	1,280	139	10.9	460	36.0	29	2.3	425	33.0

with resistance to penicillin and high level resistance to ciprofloxacin. From New South Wales, there were 63 strains with decreased susceptibility to ceftriaxone and of those, 51/63 (81%) were multi-drug resistant; 57/63 (91%) were from males; and 33/63 (58%) were isolated from extra genital sites (rectal and pharyngeal). In Victoria, there were 49 strains with decreased susceptibility to ceftriaxone. All were from males with 30/49 (61%) isolated from extra genital sites. There were recent reports of ceftriaxone 500 mg treatment failure in Victoria and New South Wales. These patients had pharyngeal infections where the gonococcal strains had ceftriaxone MIC values in the range 0.03–0.06 mg/L.[2,3] Patients with infections in extra genital sites, where the isolate has decreased susceptibility to ceftriaxone are recommended to have a test to confirm a cure.

In Queensland, there were 15 (8%) isolates reported with DS to ceftriaxone compared with 4.6% during the same quarter in 2012. Western Australia reported 4 (3.9%) isolates; an increase from 0.8%. South Australia reported 2 (3.2%) from 63 NG isolates with DS during this quarter in 2012. The Northern Territory reported 1 (1.4%) isolate in this quarter; an increase from 0%. Although numbers were low, Tasmania reported 5 of 15 (33%) isolates with decreased susceptibility, whereas there were none during the 2nd quarter of 2012. No gonococci with DS to ceftriaxone were reported from the Australian Capital Territory.

The greatest increase is in the proportion of isolates with a ceftriaxone MIC value of 0.06–0.125 mg/L (Figure).

Figure: Distribution of ceftriaxone MIC values in gonococcal isolates tested in the AGSP, 1 April to 30 June, 2011 to 2013



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3. Chen M, Stevens K, Tideman R, Zaia A, Fairley CK, Lahra MM, Hogg G. Failure of 500 mg of ceftriaxone to eradicate pharyngeal gonorrhoea, Australia. *J Antimicrob Chemother* 2013;68(6):1445–1447.

Australian Meningococcal Surveillance Programme quarterly report, 1 April to 30 June 2013

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

Introduction

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of cases confirmed by laboratory testing using culture and by non-culture based techniques. Culture positive cases, where *Neisseria meningitidis* is grown from a normally sterile site or skin lesions, and non-culture based diagnoses, derived from results of nucleic acid amplification assays and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in quarterly reports are restricted to a description of the number of cases by 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 annual reports of the programme published in *Communicable Diseases Intelligence*. For more information see *Commun Dis Intell* 2013;37(1):E61.

Results

Laboratory confirmed cases of invasive meningococcal disease for the period 1 April to 30 June 2013 are shown in the Table.

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2013, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD
Australian Capital Territory	2013	0	0	0	0	0	0	1	1	0	0	0	0	1	1
	2012	0	0	1	2	0	0	0	0	0	0	0	0	1	2
New South Wales	2013	0	0	5	10	0	2	1	1	0	0	1	1	7	14
	2012	0	0	14	20	0	0	1	1	0	0	2	4	17	25
Northern Territory	2013	0	0	1	1	0	0	0	0	0	0	0	0	1	1
	2012	0	0	2	2	1	1	0	0	0	0	0	1	3	4
Queensland	2013	0	0	8	15	1	1	1	2	1	2	0	0	11	20
	2012	0	0	10	20	0	1	0	0	1	1	0	0	11	22
South Australia	2013	0	0	6	10	0	0	1	1	0	1	0	0	7	12
	2012	0	0	7	7	0	1	0	0	0	0	0	0	7	8
Tasmania	2013	0	0	0	0	0	0	0	0	0	0	0	0	0	1
	2012	0	0	1	1	0	0	0	0	0	0	1	1	2	2
Victoria	2013	0	0	2	8	0	1	0	0	0	0	1	1	3	10
	2012	0	0	6	13	0	0	2	2	0	0	0	0	8	15
Western Australia	2013	0	0	1	8	1	1	0	0	1	1	0	0	3	10
	2012	0	0	6	7	0	1	0	1	0	0	0	0	6	9
Australia	2013	0	0	23	52	2	5	4	5	2	4	2	2	33	69
	2012	0	0	47	72	1	4	3	4	1	1	3	6	55	87

Australian Sentinel Practices Research Network, 1 July to 30 September 2012

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

Introduction

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

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

Since 2010, ASPREN general practitioners have been collecting nasal swab samples for laboratory testing, allowing for viral testing of 25% of influenza like illness (ILI) patients for a range of respiratory viruses including influenza A, influenza B and H1N1(2009).

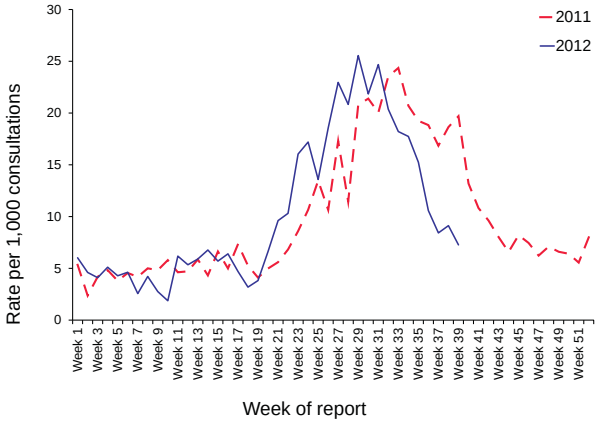
The list of conditions reported is reviewed annually by the ASPREN management committee. In 2012, 4 conditions were being monitored. They included ILI, gastroenteritis and varicella infections (chickenpox and shingles). Definitions of these conditions are described in Surveillance systems reported in CDI, published in *Commun Dis Intell* 2013;37(1):E62.

Results

Sentinel practices contributing to ASPREN were located in all 8 states and territories in Australia. A total of 236 general practitioners contributed data to ASPREN in the 3rd quarter of 2012. Each week an average of 194 general practitioners provided information to ASPREN at an average of 20,235 (range 15,070–23,235) consultations per week and an average of 429 (range 241–571) notifications per week.

ILI rates reported from 1 July to 30 September 2012 averaged 17 cases per 1,000 consultations (range 7–26 cases per 1,000 consultations), which was lower compared with rates in the same reporting period in 2011, which averaged 19 cases per 1,000 consultations (range 11–24 cases per 1,000 consultations, Figure 1). This can be attributed to the 2012 season beginning and peaking earlier than the 2011 season with the peak in 2012 occurring at week 29 compared with week 33 in 2011.

Figure 1: Consultation rates for influenza-like illness, ASPREN, 1 January 2011 to 30 September 2012, by week of report



ILI swab testing continued during 2012. The most commonly reported virus during this reporting period was influenza A (untyped) (28% of all swabs performed, Figure 2), with the second most common virus being influenza B (12% of all swabs performed).

From the beginning of 2012 to the end of week 39, 1,251 cases of influenza were detected, the majority of these being influenza A (untyped) (28% of all swabs performed), influenza B (12% of all swabs performed) and the remainder H1N1(2009) (0.5% of all swabs performed). As influenza A H3N2 was

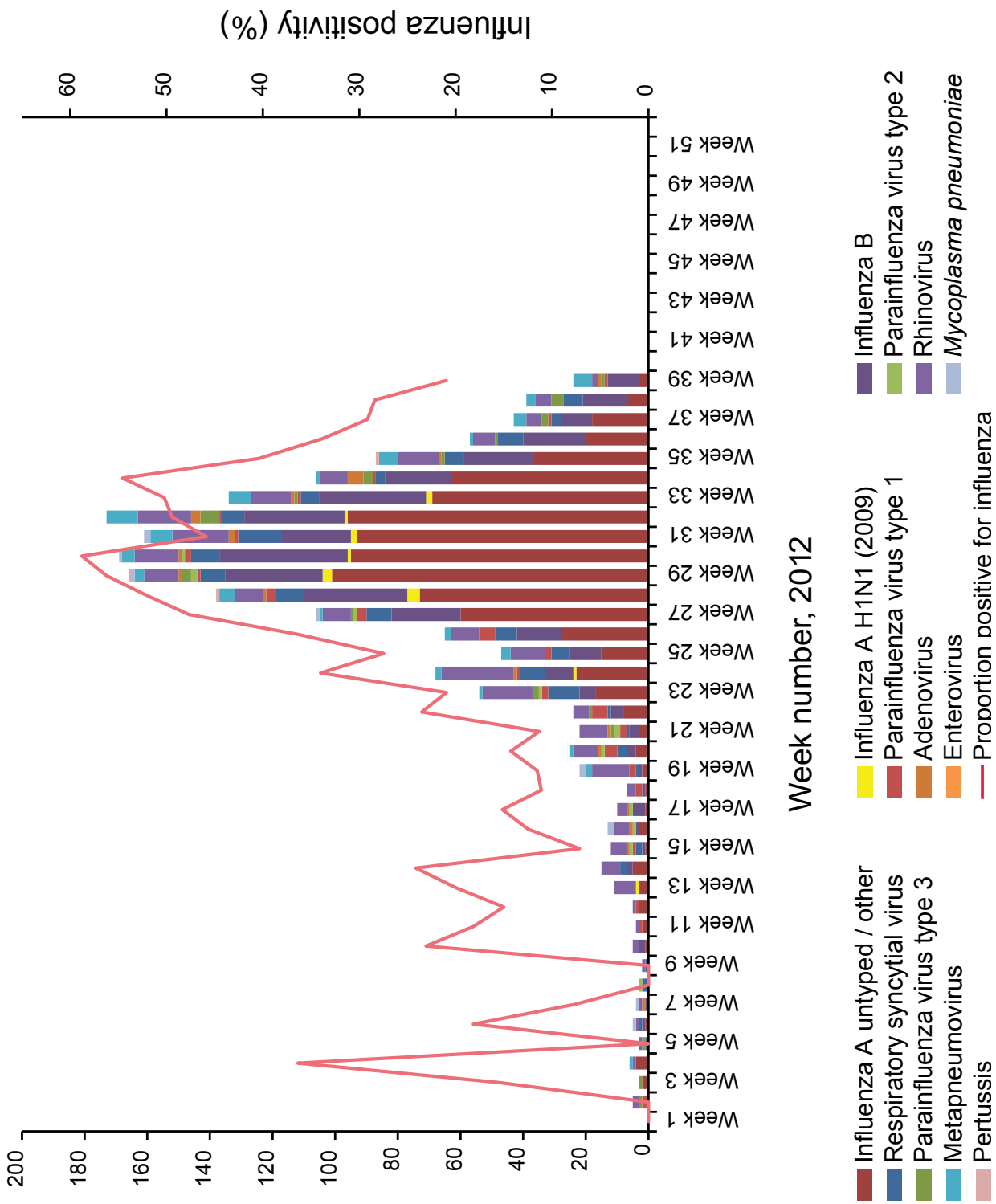
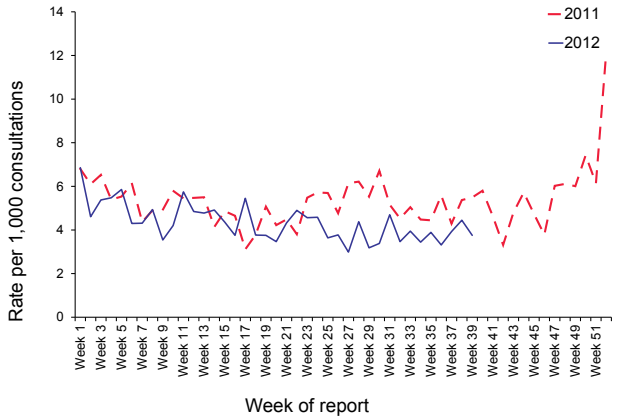


Figure 2: Influenza-like illness swab testing results, ASPREN, 1 January to 30 September 2012, by week of report

the predominantly circulating strain in Australia in 2012, it is assumed that the vast majority of the influenza A (untyped) are of the H3N2 subtype.

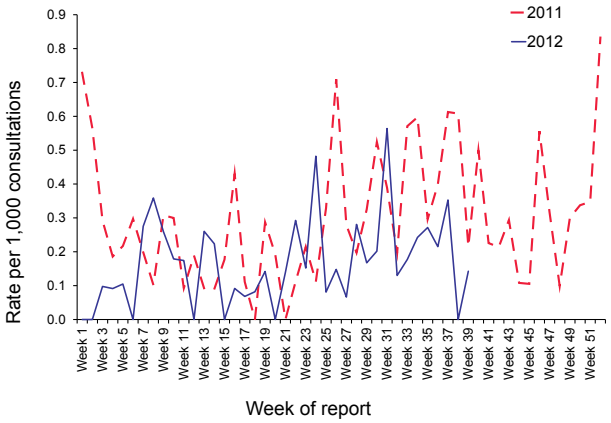
During this reporting period, consultation rates for gastroenteritis averaged 4 cases per 1,000 consultations (range 3–5 cases per 1,000 consultations, Figure 3). This was lower compared with rates in the same reporting period in 2011 where the average was 5 cases per 1,000 consultations (range 4–7 cases per 1,000 consultations).

Figure 3: Consultation rates for gastroenteritis, ASPREN, 1 January 2011 to 30 September 2012, by week of report



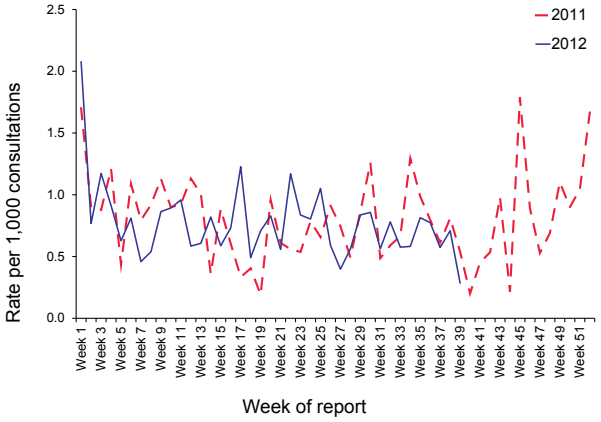
Varicella infections were reported at a lower rate for the 3rd quarter of 2012 compared with the same period in 2011. From 1 July to 30 September 2012, recorded rates for chickenpox averaged 0.22 cases per 1,000 consultations (range 0–0.56 cases per 1,000 consultations, Figure 4).

Figure 4: Consultation rates for chickenpox, ASPREN, 1 January 2011 to 30 September 2012, by week of report



In the 3rd quarter of 2012, reported rates for shingles averaged 0.64 cases per 1,000 consultations (range 0.29–0.86 cases per 1,000 consultations, Figure 5), slightly lower compared with the same reporting period in 2011 where the average shingles rate was 0.78 case per 1,000 consultations (range 0.49–1.29 cases per 1,000 consultations).

Figure 5: Consultation rates for shingles, ASPREN, 1 January 2011 to 30 September 2012, by week of report



Invasive pneumococcal disease surveillance Australia, 1 April to 30 June 2013

Christina Bareja for the Enhanced Invasive Pneumococcal Disease Surveillance Working Group

Introduction

Invasive pneumococcal disease (IPD) is caused by the bacterium *Streptococcus pneumoniae* and results in illnesses such as pneumonia, bacteraemia and meningitis. There are currently more than 90 serotypes recognised worldwide and IPD has been a nationally notifiable disease in Australia since 2001. The Communicable Diseases Network Australia established the Enhanced Invasive Pneumococcal Disease Surveillance Working Group (EIPDSWG) in 2000 to assist in developing and implementing a nationally standardised approach to the enhanced surveillance of IPD in Australia. This quarterly report documents trends in notified cases of IPD occurring in Australia in the 2nd quarter of 2013.

Notification data are collected by all Australian states and territories under jurisdictional public health legislation and are forwarded to the Commonwealth under the *National Health Security Act 2007*. Notified cases are collated nationally in the National Notifiable Diseases Surveillance System (NNDSS). The data in this report are provisional and subject to change as laboratory results and additional case information become available. The data are analysed by diagnosis date, which is the onset date, or where the onset date was not known, the earliest of the specimen collection date, the notification date, and the notification receive date. Data for this report were extracted on 14 August 2013. Crude rates were calculated using the Australian Bureau of Statistics estimated resident populations for Australia at 30 June of each year. Consideration of vaccination status of cases is outside the scope of this report. For more detailed reports readers are referred to the regular *Communicable Diseases Intelligence* supplements *Vaccine Preventable Diseases in Australia*.

In Australia, pneumococcal vaccination is recommended as part of routine immunisation for children, the medically at risk and older Australians. 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 older in 1999 and for non-Indigenous Australians aged 65 years or older from January 2005.

Results

There were 437 cases of IPD reported to the NNDSS in the 2nd quarter of 2013, bringing the year to date total to 651 cases (Table). While the number of cases notified in the reporting period is more than twice the number in the 1st quarter of 2013 (n=214), it was a 13% reduction on the number of cases reported during the same period in 2012 (n=505), and a seasonal increase is not unexpected.

Overall, Aboriginal and Torres Strait Islander status was reported for 84% (n=365) of cases, ranging from 58% of cases reported by Victoria to 100% of cases reported by the Australian Capital Territory, the Northern Territory, Tasmania and Western Australia. Victoria and New South Wales only actively follow up notified cases of IPD aged 5 years and under and 50 years and older for core and enhanced data, whereas follow up of all cases is undertaken in other states and territories. This may account for missing data among cases falling outside these age groups. Of cases with a reported Indigenous status, Aboriginal and Torres Strait peoples accounted for 16% (n=58) of all cases notified in the quarter (Table).

Serotype information was available for 95% (n=417) of all cases reported in the quarter (Table). There were 5 cases reported in the quarter that were deemed by the reference laboratory as non-typable; these cases are included in the vaccine serotype group in figures of this report as serotype not specified.

In the 2nd quarter of 2013, notified cases were highest in the under 5 years age group (n=59), followed by the 65–69 years age group (n=41) and the 85 years or older age group (n=38). This age distribution was evident in cases reported as non-Indigenous Australian (Figure 1). However in cases reported as Indigenous, the most prevalent age groups were those under 5 years (n=8) and the 35–39 years age group (n=8). Three groups have been selected for focused analyses in this quarterly report. These groups carry the greatest burden of disease and are those at which the NIP is targeted.

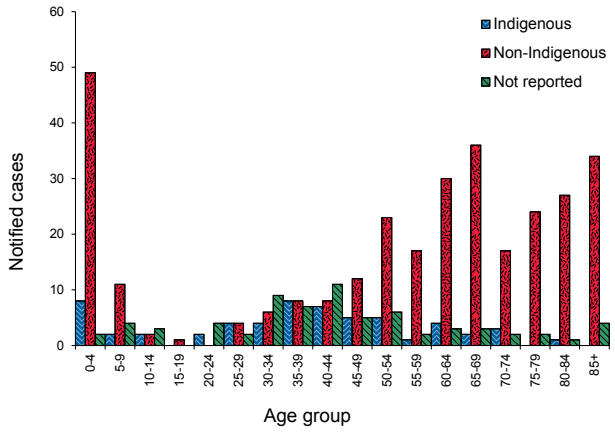
Table: Notified cases of invasive pneumococcal disease, Australia, 1 April to 30 June 2013, by Indigenous status, serotype and state or territory

Indigenous status	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Total qtr 2 2013	Total qtr 1 2013	Total qtr 2 2012	Year to date 2013
Indigenous	0	6	10	17	3	0	1	21	58			
Non-Indigenous	5	107	2	65	31	7	59	33	309			
Not stated/ unknown	0	22	0	4	1	0	43	0	70			
Total	5	135	12	86	35	7	103	54	437	214	505	651
Indigenous status completeness* (%)	100	84	100	95	97	100	58	100	84			
Serotype completeness† (%)	100	90	100	100	91	100	98	96	95			

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

† Serotype completeness is the proportion of all cases of IPD that were reported with a serotype or as non-typable. Serotype incompleteness may include when no isolate was available as diagnosis was by polymerase chain reaction and no molecular typing was performed; 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. Figures in this report present the category 'Serotype not specified', this includes notified cases reported with an incomplete serotype or non-typable.

Figure 1: Notified cases of invasive pneumococcal disease Australia, 1 April to 30 June 2013, by Indigenous status and age group



Invasive pneumococcal disease in children aged less than 5 years

In the 2nd quarter of 2013, 14% (n=59) of notified cases were aged less than 5 years. This was more than twice the number of cases reported in the previous quarter (n=21) but similar to the number reported during the same period of 2012 (n=51) (Figure 2).

The majority (88%, n=52) of cases aged less than 5 years were reported with serotype information. Of these, 40% (n=21) were reported with a serotype included in the 7vPCV or the 13vPCV.

Notified cases aged less than 5 years with disease caused by the 6 additional serotypes targeted by the 13vPCV increased steadily over the period 2007 to 2011, particularly those caused by serotype 19A (Figure 3). However, cases of this type have decreased since the 4th quarter of 2011, reflecting the introduction of the 13vPCV on the universal childhood immunisation program in mid-2011. In the 2nd quarter of 2013, there were 10 cases aged less than 5 years with disease due to serotype 19A, 4 cases due to serotype 3 and 1 case each due to serotypes 1 and 7F. No cases in this age group were reported with disease caused by serotypes 5 or 6A.

Invasive pneumococcal disease in Indigenous Australians aged 50 years or older

In the 2nd quarter of 2013, 4% (n=16) of notified cases were reported in Indigenous Australians aged 50 years or over. This was twice the number of cases reported in the previous quarter (n=8), but a 38% decrease on the number reported during the same period in 2012 (n=26) (Figure 4). The annual rate of IPD in this group has tended to increase over time, with an outbreak of disease caused by serotype 1 in Central Australia that commenced in late 2010 contributing in part to this increase.[1]

All but one of the cases were reported with serotype information. Of these, 87% (n=13) were reported with disease due to serotypes targeted by the 23vPPV; the remaining reported disease due to a non-vaccine serotype (n=2).

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

In the 2nd quarter of 2013, 32% (n=138) of notified cases were reported as non-Indigenous Australians aged 65 years or over. This was more than twice

Figure 2: Notified cases and rates of invasive pneumococcal disease in those aged less than 5 years, Australia, 2002 to 30 June 2013, by vaccine serotype group

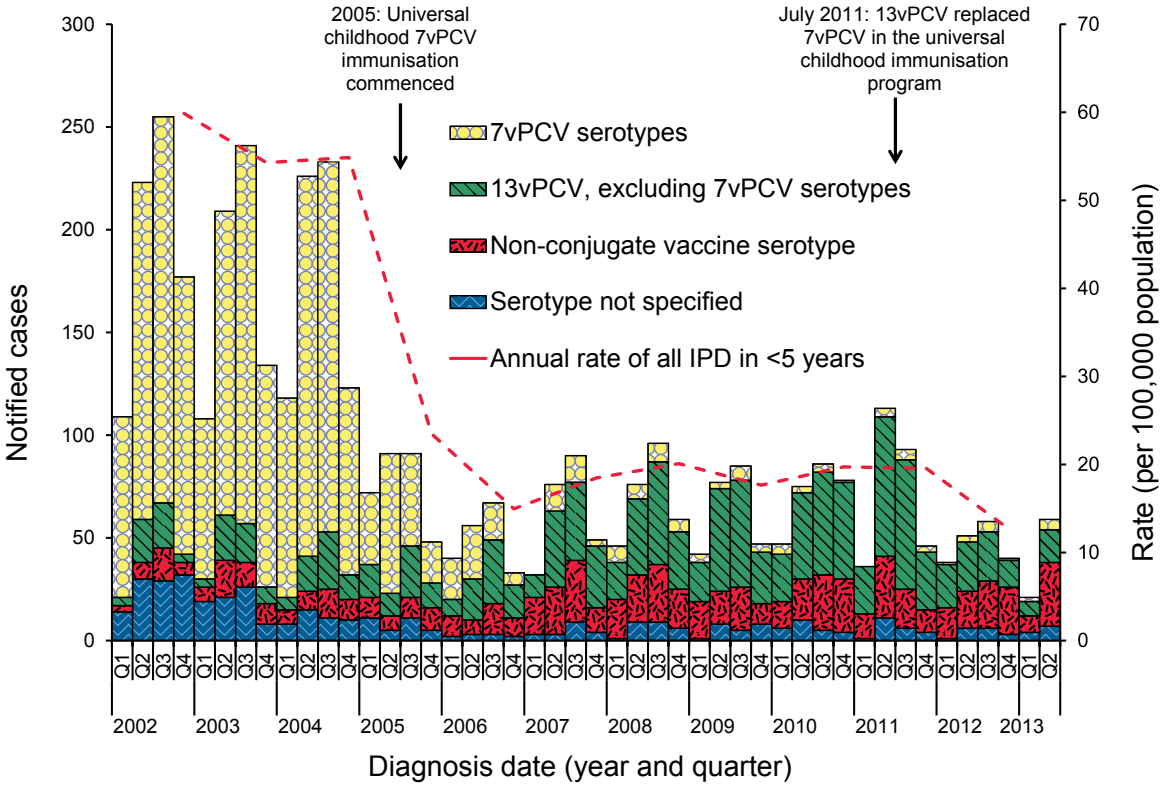
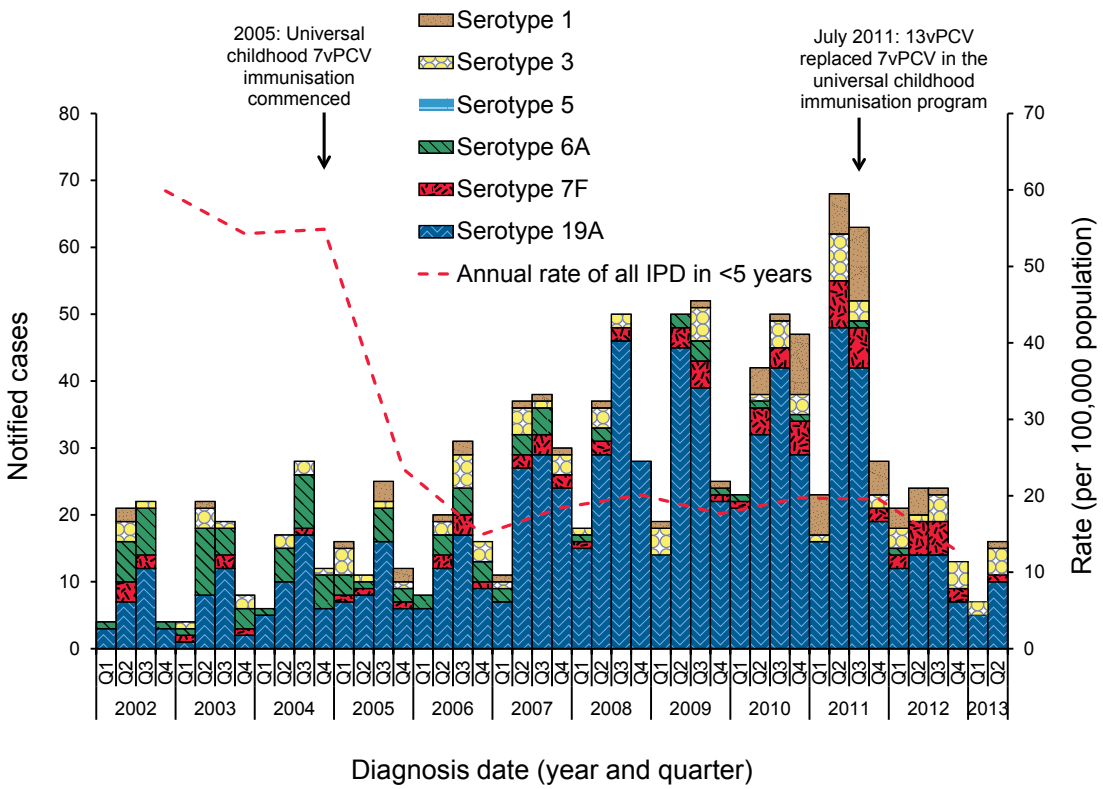


Figure 3: Notified cases of invasive pneumococcal disease caused by serotypes targeted by the 13-valent pneumococcal conjugate vaccine (excluding those targeted by 7-valent pneumococcal conjugate vaccine) and rates of all invasive pneumococcal disease, aged less than 5 years, Australia, 2002 to 30 June 2013



the number of cases reported in the previous quarter (n=60), but a 19% decrease on the number reported during the same period of 2012 (n=166) (Figure 5).

The majority (96%, n=133) of cases reported in this quarter were reported with serotype information. Of these cases, 57% (n=76) 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 effects from the childhood immunisation program.

Conclusion

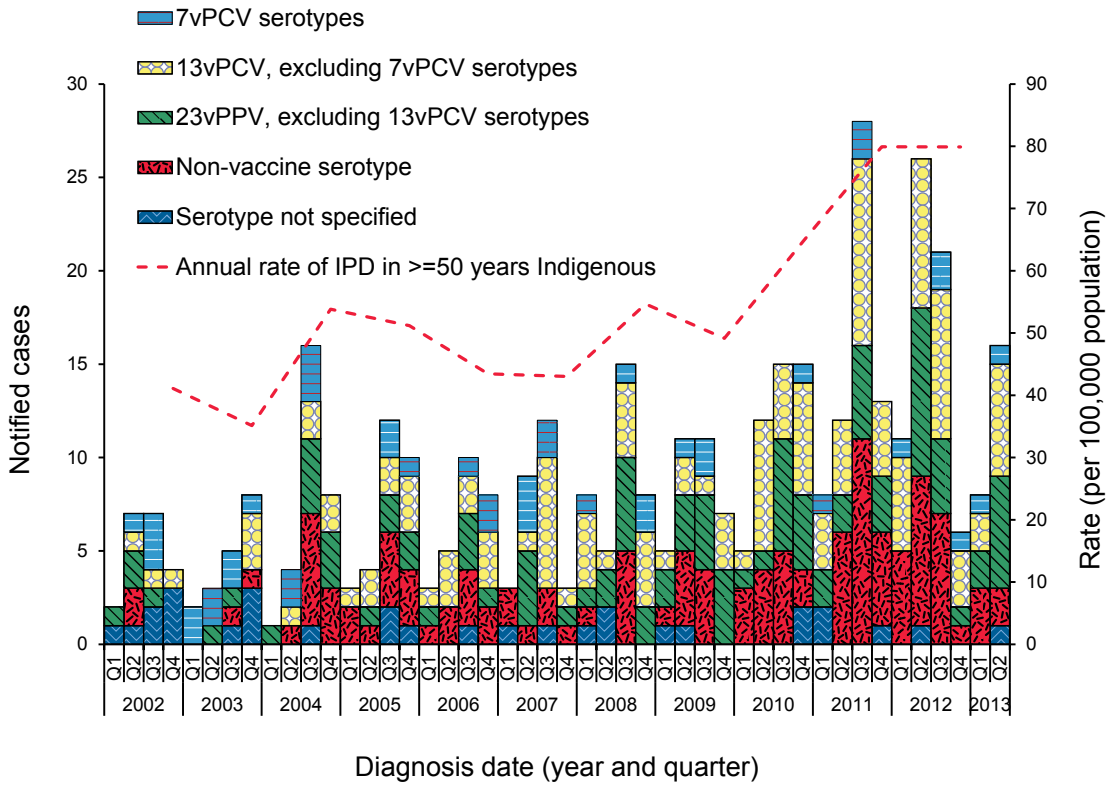
While the number of notified cases of IPD in the 2nd quarter of 2013 was more than double the number reported in the previous quarter, it represented a decline compared with the number reported during the same quarter in 2012. Disease due to the serotypes targeted by the 13vPCV has continued to decline since the 13vPCV replaced the 7vPCV in the childhood immunisation program from July 2011. Notified cases of IPD in Indigenous Australians aged 50 years or older have tended to increase over time, whereas disease in non-Indigenous Australians aged 65 years or older has remained relatively stable but the profile of serotypes causing disease has diversified.

This quarter the EIPDSWG noted a localised increase in the number and severity of IPD cases due to serotype 3 and will continue to monitor the occurrence of such cases in future reporting periods. The group is also actively monitoring the occurrence of vaccine failures following a full course of 13vPCV.

Acknowledgements

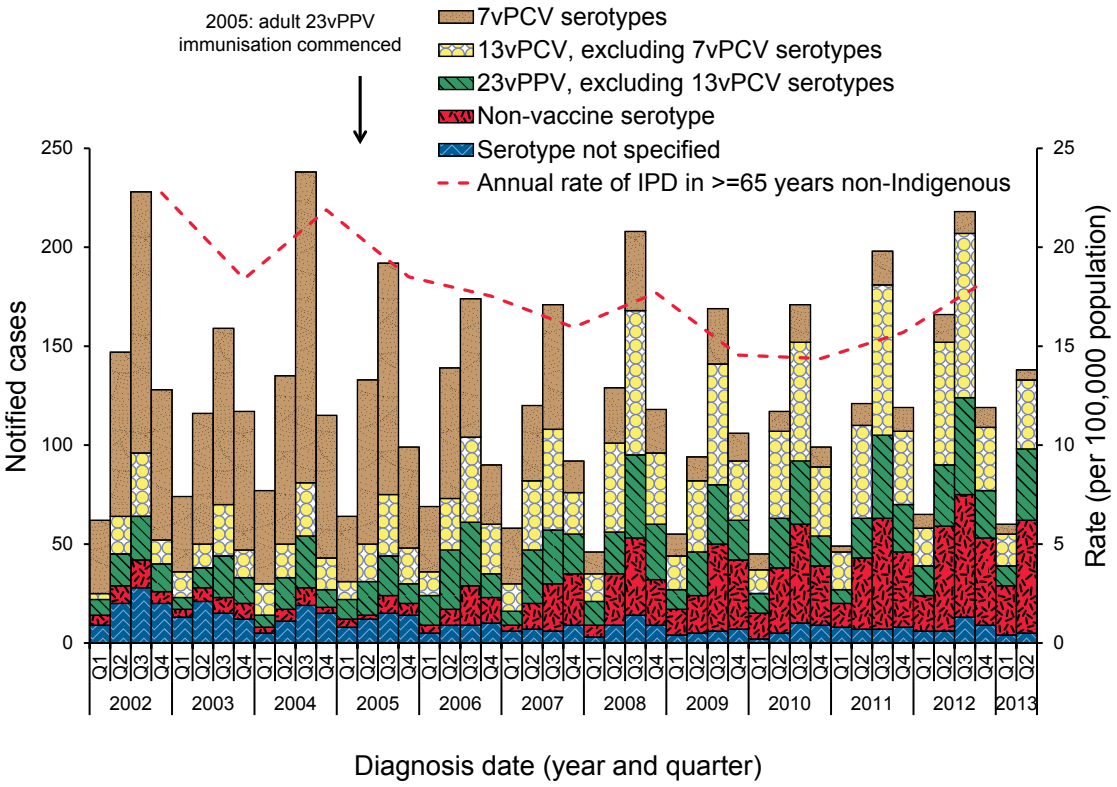
EIPDSWG contributors to this report include (in alphabetical order): Christina Bareja (Health), David Coleman (Tas), Heather Cook (NT), Lucinda Franklin (Vic), Carolien Giele (WA), Robin Gilmour (NSW), Michelle Green (Tas), Geoff Hogg (Microbiological Diagnostic Unit, University of Melbourne), Vicki Krause (NT), Rob Menzies (NCIRS), Shahin Oftadeh (Centre for Infectious Diseases and Microbiology, Public Health, Westmead Hospital), Sue Reid (ACT), Stacey Rowe (Vic), Vitali Sintchenko (Centre for Infectious Diseases and Microbiology, Public

Figure 4: Notified cases and rates of invasive pneumococcal disease in Indigenous Australians aged 50 years or older, Australia, 2002 to 30 June 2013, by vaccine serotype group



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

Figure 5: Notified cases and rates of invasive pneumococcal disease in non-Indigenous Australians aged 65 years or older, Australia, 2002 to 30 June 2013, by vaccine serotype group



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1. Centre for Disease Control Northern Territory. Comments on notifications. *Northern Territory Disease Control Bulletin* 2012;19(1):29.