Australian Government
Department of Health and Ageing

Communicable Diseases Intelligence

Volume 33 Number 1 Quarterly report March 2009

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ISSN 0725-3141
ISSN 1445-4866 Online

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

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This journal is indexed by *Index Medicus*, Medline and the Australasian Medical Index

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Printed by Union Offset, Canberra

Annual reports

Annual report of the Australian Meningococcal Surveillance Programme, 2007 – Amended

The Australian Meningococcal Surveillance Programme

Abstract

In 2007 there were 281 laboratory-confirmed cases of invasive meningococcal disease analysed by the National Neisseria Network, a nationwide network of reference laboratories. The phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility of 154 isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were determined and an additional 127 cases were confirmed by non-culture based methods. Nationally, 223 (85%) confirmed cases where a serogroup was determined were infected with serogroup B and 17 (6.5%) with serogroup C meningococci. The total number of confirmed cases was 10 more than the 271 cases identified in 2006. Queensland and New South Wales recorded slight increases in case numbers and Victoria recorded a decline. Typical primary and secondary disease peaks were observed in those aged 4 years or less and in adolescents and young adults respectively. Serogroup B cases predominated in all age groups and jurisdictions. The common phenotypes circulating in Australia were B:15:P1.7, B:4:P1.4 and C:2a:P1.5. No evidence of meningococcal capsular 'switching' was detected. About three-quarters of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06–0.5 mg/L). All isolates remained susceptible to rifampicin. A single serogroup B isolate had decreased susceptibility to ciprofloxacin (MIC 0.06 mg/L). This was the first local isolate of this type since the original report of this phenomenon in Australia in 2000. *Commun Dis Intell* 2009;33:1–9 (replacement for *Commun Dis Intell* 2008;32:299–307).

Keywords: disease surveillance, meningococcal disease, *Neisseria meningitidis*

Introduction

There has been a significant reduction in the number of cases of invasive meningococcal disease (IMD) following the completion, in 2004, of a publicly-funded program of selective vaccination with conjugate serogroup C meningococcal vaccine. However, IMD remains an issue of public health concern in Australia, including the continuing need for analysis of the subtypes of *Neisseria meningitidis* responsible for current cases.

A national laboratory-based program for the examination of *N. meningitidis* from cases of IMD, the National Neisseria Network (NNN), has operated since 1994 through the collaboration of reference laboratories in each jurisdiction. The NNN supplies information on the phenotype and/or the genotype of invasive meningococci, and their antibiotic susceptibility and these data supplement those from clinical notification schemes. The characteristics of the meningococci responsible for IMD are important both for individual patient management and to tailor the public health response. Annual reports summarising data gathered since the inception of the program were published in *Communicable Diseases Intelligence*.[1,2] The following is an amended version of the report analysing the characteristics of meningococci isolated in the calendar year 2007 published earlier.[3] This report includes additional data from 1 jurisdiction.

Methods

The NNN continues as a long-term collaborative program for the laboratory surveillance of the pathogenic Neisseria, *N. meningitidis* and *N. gonorrhoeae*. A network of reference laboratories in each state and territory performs and gathers laboratory data on cases of IMD throughout Australia. A list of reference laboratories is contained in the acknowledgements.

Isolate based invasive meningococcal disease cases

Each case confirmation was based upon isolation of a meningococcus from a normally sterile site and defined as IMD according to Public Health Laboratory Network criteria.[4] Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was sought. The isolate-based subset of the program categorised cases on the basis of site of isolation of the organism. Where an isolate was grown from

both blood and cerebrospinal fluid (CSF) cultures in the same patient, the case was classified as one of meningitis. It is recognised that the total number of cases and particularly the number of cases of meningitis e.g. where there was no lumbar puncture or else where lumbar puncture was delayed and the culture sterile, is underestimated. However the above approach has been used since the beginning of this program[1] and is continued for comparative purposes.

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 National Institute for Public Health, The Netherlands. Increasingly, sequencing of products derived from amplification of the porin genes *porA* and *porB* has been used to supplement and supplant serotyping analyses based on the use of monoclonal antibodies. For the purposes of continuity and comparability, the typing data from both approaches has been unified in the accompanying tables by converting sequence data to the more familiar serotyping/serosubtyping nomenclature.

Antibiotic susceptibility was assessed by determining the minimal 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/resistance when determined by a standardized agar plate dilution technique.[5]

sensitive, MIC ≤ 0.03 mg/L

less sensitive, MIC 0.06–0.5 mg/L

relatively resistant MIC ≥ 1 mg/L

Strains with MICs which place them in the category of 'sensitive' or 'less sensitive' would be considered to be amenable to penicillin therapy when used in currently recommended doses. However, precise MIC/outcome correlations are difficult to obtain because of the nature of IMD.

Non-culture-based laboratory-confirmed cases

Additional laboratory confirmation of suspected cases of IMD was obtained by means of non-culture based methods including nucleic acid agglutination testing (NAAT) and serological techniques. NAAT testing is essentially by polymerase chain reaction (PCR) techniques[6] and has been progressively introduced in the different jurisdictions. Data from the results of these investigations were included for the first time in the 1999 report.[1] The serological results are based on results of tests performed using the methods and test criteria of the Manchester Public Health Laboratory Service reference laboratory, United Kingdom as assessed for Australian conditions.[7–10] Where age, sex and outcome data for patients with non-culture based diagnoses are available these were also recorded. The site of a sample of a positive NAAT is also used to define the clinical syndrome. This separation is not possible for cases diagnosed serologically.

Results

Aggregated data on cases confirmed by culture based and non-culture based methods

Number of laboratory confirmed cases

There were 281 laboratory confirmed cases of IMD in 2007 (Table 1) compared with 271 in 2006, 345 in 2005 and 361 in 2004. In 154 cases (54.8%), a positive culture was obtained with or without a positive non-culture based test and 127 cases were confirmed by a non-culture based method alone. The total number of all laboratory confirmed cases increased in New

Table 1. Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2007, by state or territory and serogroup

State or territory	Serogroup						NG	Total
	B	C	A	X	Y	W135		
ACT	4	0				1		5
NSW	78	6			5	1	11	101
NT	1	1						2
Qld	61	7		1	2	3	1	75
SA	11	1			1	0	1	14
Tas	3	0			1	1		5
Vic	46	2			4	3	4	59
WA	19	0					1	20
Australia	223	17	0	1	13	9	18	281

NG Not serogrouped.

South Wales where numbers detected rose to 101 from 84 after a decrease in 2006 and in Queensland from 68 in 2006 to 75. Small or no numerical differences were noted in other jurisdictions with the exception of Victoria where numbers decreased from 75 to 59.

Seasonality

Forty cases occurred between 1 January and 31 March 2007, 53 between 1 April and 30 June, 108 between 1 July and 30 September and 80 between 1 October and 31 December. A winter peak of meningococcal disease is more usual.

Age distribution

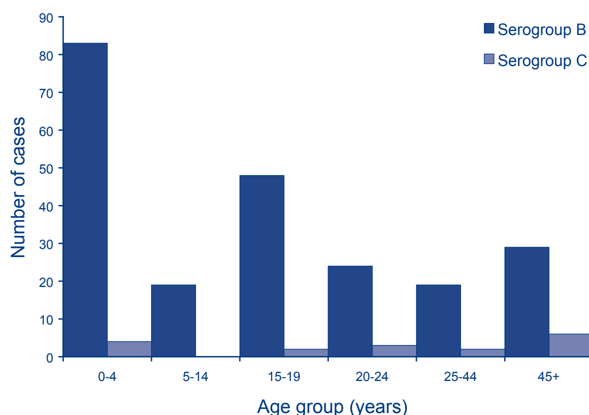
Nationally, the peak incidence of meningococcal disease was again in those aged 4 years or under (Table 2, Figure). Those aged less than 1 year or in the 1–4 year age group together accounted for 100 cases (35.5% of the total) in 2007. There were also 100 cases confirmed in these age groups (37%) in 2006. A secondary disease peak is also usual in the adolescent/young adult age group. The total of 56 cases (19.9% of all confirmed cases) in those aged 15–19 years was a little more than the 49 cases (18%) in this age group in 2006 (49, 18%). Those aged 15–24 years accounted for 87 cases (31%) in 2007 and 79 cases (29%) in 2006.

Table 2. All laboratory confirmed serogroup B and C cases of invasive meningococcal disease by age, Australia, 2007, by state or territory

State or territory	Serogroup	Age group										Total
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
ACT	B	1	2						1			4
	C											0
	Total	2	2						1			5
NSW	B	10	21	3	4	7	9	12	8	4	0	78
	C	1	1	0	0	0	1	0	3	0		6
	Total	15	24	3	6	8	10	15	12	8	0	101
NT	B						1					1
	C					1						1
	Total					1	1					2
Qld	B	10	14	4	2	15	4	5	5	2		61
	C		2			1	2	1		1		7
	Total	10	19	5	4	16	7	6	5	3		75
SA	B	2	3	1		4				1		11
	C								1			1
	Total	2	3	1		5	1		1	1		14
Tas	B		1		1						1	3
	C											0
	Total		1		1	1				1	1	5
Vic	B	8	6	1	3	17	7	0	3	1		46
	C							1	1			2
	Total	9	8	2	3	20	9	3	4	1		59
WA	B	2	2			5	3	2	4		1	19
	C											0
	Total	2	3			5	3	2	4		1	20
Australia	B	33	49	9	10	48	24	19	21	8	2	223
	C	1	3	0	0	2	3	2	5	1	0	17
	Total B+C	34	52	9	10	50	27	21	26	9	2	240
	other	6	8	2	4	6	4	5	1	5	0	41
	Total	40	60	11	14	56	31	26	27	14	2	281
	% of all	14.2	21.4	3.9	5	19.9	11	9.3	9.6	5	0.7	

NS Not stated. [Totals include cases due to other serogroups (23) and cases where the serogroup was not determined (18).]

Figure. Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2007, by age



Serogroup data

The serogroup of the meningococci causing disease was determined in 264 of the 281 laboratory confirmed cases of IMD. Of these 264 cases where a serogroup was determined, 223 (85%) were serogroup B and 17 (6.5%) were serogroup C. In 2006, 217 (83.8%) cases were serogroup B and 26 (10%) were serogroup C. In 2007, an additional 9 cases (3.4%) were of W135 and 13 (4.9%) of serogroup Y. A serogroup X meningococcus was detected in Queensland. With the continuing decline in numbers of serogroup C infections, serogroup B meningococci predominated in all age groups and jurisdictional differences in serogroup distribution were not evident. Eight of the 17 cases of serogroup C disease in 2007 were in those aged 25 years or more, 2 cases were recorded in those aged 15–19 years and a further three in those aged 20–24 years. Seven serogroup C cases were identified in those aged 15–24 years in 2006. Queensland and New South Wales each accounted for 7 serogroup C cases.

Table 3 shows a comparison of the number and proportion of serogroup B and C cases by age from 2004 to 2007. In those aged 14 years or less, there was a decrease in total case numbers and in serogroup B cases in 2007. Serogroup C case numbers were always low in these age groups. In those aged 15–19 years and 20–24 years, the number of serogroup B cases has remained relatively unaltered, but the proportion of serogroup B cases increased as serogroup C cases declined. In older (25 years or more) age groups there was an increase in serogroup B cases in 2007 but a continuing decrease in serogroup C cases, so that again the proportion of serogroup B IMD increased over time.

Phenotypes of invasive meningococcal isolates

The typical heterogeneity of serogroup B meningococci was again seen in 2007 when the phenotypes of invasive isolates, based on a determination of their serogroup, serotype and serosubtype were analysed. The predominant serotypes/serosubtypes in each state and territory are shown in Table 4. Serogroup B meningococci are in general also more difficult to characterise by serological methods and a number could not be phenotyped. A total of 27 isolates were of serotype 4. Twelve of these were

Table 3. A comparison of the number and proportion of serogroup B and serogroup C laboratory-confirmed cases, 2004 to 2007, by age

Year	Serogroup	Age									
		< 4 years		5–14 years		15–19 years		20–24 years		25+ years	
		n	%	n	%	n	%	n	%	n	%
2007	B	83	90	19	83	48	91	24	80	49	75
	C	4	4	0		2 4		3	10	8	12
	All*	92		23		53		30		65	
2006	B	93	93	21	84	40	82	21	70	38	61.3
	C	2	2	3	12	4	8.2	7	23	10	16.1
	All	100		25		49		30		62	
2005	B	99	90	38	75	39	81	22	67	51	50
	C	6	5.5	5	10	4	8	8	24	27	27
	All	110		51		48		33		101	
2004	B	97	88	27	77	40	65	20	57	59	50
	C	6	5.5	5	14	17	28	11	31	32	27
	All	110		35		61		35		117	

* All cases where a serogroup was determined.

Table 4. Common serotypes and serosubtypes of isolates from culture positive cases of *Neisseria meningitidis* infection, 2007, by state or territory

State or territory	Serogroup B				Serogroup C			
	Serotype	n	Serosubtype	n	Serotype	n	Serosubtype	n
ACT	4	1	1.4	1				
NSW	4	20	1.4	8	2a	4	1.5	2
			1.15	4			1.2	1
			1.7	2			1.4	1
			1.14	2				
			1.5	1				
			1.5,2	1				
			1.22,14	1				
			nst	1				
	15	5	1.5	5				
	1	3	1.4	1				
			nst	2				
	nt	14	1.9	3				
			1.14	1				
			1.15	1				
			1.16	1				
			1.4	1				
			nst	7				
NT					2a	1	1.5	1
Qld	1	7	1.14	4	2a	5	1.5	3
			1.4	2			nst	2
	15	4	1.7	3				
	nt	21	1.14	5				
			1.15	1				
			nst	12				
Tas	nt	1	1.17	1				
Vic	4	6	1.4	3	2a	2	1.16	2
			1.15	2				
			nst	1				
	15	6	1.7,16	4				
			1.4	1				
			1.5,10	1				
	nt	9	1.5,2	3				
			1.14	3				
			1.5,15	1				
			1..15	2				
			1..16	1				
			nst	2				
WA	14	1	1.5	1				
	15	1	nst	1				
	1	1	1.14	1				
	nt	6	diverse					

nt Not serotypable.

nst Not serosubtypable.

of serosubtype P1.4: eight from New South Wales, three from Victoria and one from the Australian Capital Territory. Fourteen serogroup B strains with this subtype/serosubtype were seen in 2006. This phenotype has been circulating in New Zealand at high rates for many years. Another 16 serogroup B isolates were of serotype 15 and included 5 of serosubtypes 1.5 and 10 of serosubtype 1.7. The latter phenotype has been circulating in Australia for many years.

There is continuing interest in the presence of any serogroup B or serogroup C meningococci of serotypes that indicate the possibility of genetic recombination events. Among serogroup C strains, phenotype C:2a:P1.4 is of particular interest. This phenotype has figured prominently in Victorian data in former years. Nationally, there were 29 cases of serogroup C isolates of this serotype/serosubtype detected in 2003; 21 in 2004, and eight in 2005. Only a single isolate with this phenotype was seen in 2007 (in New South Wales). All of the serotypeable serogroup C isolates were of serotype 2a.

Outcome data for invasive meningococcal disease for laboratory confirmed cases

Outcome data (survived or died) were available for 100 (35%) of the 281 laboratory confirmed cases (Table 5). Four deaths were recorded in this group (4%). Outcomes were available for 73 of 223 of serogroup B infections and five of 17 serogroup C infections. There was a single death from each of serogroup B and serogroup Y infections and two attributable to serogroup C. There were 2 deaths among 39 patients with meningitis, one due to a serogroup B and the other to a serogroup C meningococcus. Two deaths were recorded among 56 bacteraemic patients, one each due to serogroup C and serogroup Y infection. There were 39 cases of serogroup B meningococcal bacteraemia with no deaths. The single fatality with serogroup Y disease was in a group of 6 bacteraemic cases where outcomes were recorded. The septicaemic fatality due to serogroup C meningococci was recorded in 4 instances of bacteraemia with this serogroup.

Anatomical source of samples for laboratory confirmed cases

Table 6 shows the source of clinical samples by which laboratory confirmation of IMD was obtained. Those diagnoses shown as culture positive may have had positive PCR and/or serology, those shown as PCR positive were culture negative with or without positive serology and those shown as serologically positive were culture and PCR negative. There were 38 isolates from CSF either alone or with a blood culture isolate and 111 from blood cultures alone. There were 4 other isolates from synovial fluid and one, most unusually from the peritoneal fluid of a patient undergoing peritoneal dialysis.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

One hundred and fifty-three isolates were available for determination of their susceptibility to penicillin and other antibiotics. Using defined criteria, 121 isolates (79%) were less sensitive to penicillin in the MIC range 0.06–0.5 mg/L and the remainder (21%) fully sensitive (MIC 0.03 mg/L or less). The proportion of less sensitive strains is higher than that reported in recent years (67% in 2006). Seven iso-

Table 5. Outcome data (survived, died) for laboratory confirmed cases of invasive meningococcal disease, 2007, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	32	0	2	2	1	37
	Died	1	1	0	0	0	2
	Total	33	1	2	2	1	39
Septicaemia	Survived	39	3	5	2	5	54
	Died	0	1	1	0	0	2
	Total	39	4	6	2	5	56
All cases*	Survived	72	3	7	5	8	96
	Died	1	2	1	0	0	4
	Total	73	5	8	5	8	100

* Includes 3 cases of joint infection, one each of serogroup B and W135 and 1 non-sero-groupable and 1 case of septicaemia serogroup X, all of whom survived.

NG Not groupable.

Table 6. Anatomical source of samples positive for a laboratory confirmed case of invasive meningococcal disease, Australia, 2007

Specimen type	Isolate of MC	PCR positive*	Total
Blood	111	64	175
CSF +/– blood	38	57	95
Other†	5	1	6
Serology alone‡			5
Total	154	122	281

* Polymerase chain reaction (PCR) positive in the absence of a positive culture.

† Joint and fluid samples (4 isolates from joints and 1 by PCR of joint fluid; 1 culture from peritoneal fluid).

‡ Serology positive in the absence of positive culture or PCR.

lates had MICs of 0.5 mg/L. Six of these were found in New South Wales. Four were of serogroup B and three of serogroup Y.

Other antibiotics

All isolates were fully susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins). A single serogroup B strain from Queensland had a slightly elevated MIC for rifampicin of 1 mg/L. Another serogroup B isolate from New South Wales had reduced susceptibility to ciprofloxacin at an MIC of 0.06 mg/L.

Discussion

Overall, the number of cases has stabilised in 2007 with a small rise to 281 following the fall to 271 cases in 2006 after recording 345 cases in 2005. Much of the interpretation of these surveillance data needs to be in the context of the recently completed program of vaccination of children and adolescents with the serogroup C conjugate vaccine. The only jurisdictions to show small rises in the number of laboratory confirmed cases were Queensland and New South Wales. Cultures were obtained from sterile sites in 154 cases, the lowest number of isolates detected over the duration of the program that commenced in 1994 and a further decline from the 166 cases from whom isolates were obtained in 2006. Non-culture based diagnoses were used to confirm a further 127 (45%) of cases as IMD.

Only 17 serogroup C infections were identified nationally in 2007, 13 of these in Queensland and New South Wales combined, so that serogroup B disease accounted for 85% of all infections where a serogroup was determined. Only small numbers of infections due to serogroups Y and W135 were encountered, and this is usual for Australia. A serogroup X case was identified in Queensland. The NNN has not identified serogroup X cases previously, but prior to its inception serogroup X infection has been identified in Australia. A primary peak in IMD infection rates was again evident in younger age groups with a secondary peak in adolescents and young adults. In contrast to data from the earlier years of this program, serogroup C disease was infrequently encountered in the latter age group in 2007. Also of interest is the continuing decline in numbers of IMD in those aged 25 years or more (Table 3). A decrease in serogroup C cases in essentially unvaccinated age groups has been noted elsewhere. It is attributed to the secondary benefit of herd immunity accruing to the wider community following vaccination of those age groups where disease was formerly highly concentrated.[11]

The continuing absence of any substantial number of meningococci showing evidence of genetic recombination in phenotyping and genotyping data is reassuring and also consistent with data from the United Kingdom.[11] Analysis of meningococcal subtypes and any evidence for the expansion of 'new' subtypes will continue as part of the NNN program. Mortality data were assessable in only a low proportion of cases and must be interpreted with caution. The NNN does not attempt collection of morbidity data associated with IMD.

NNN trend data show an upward shift in penicillin MICs insofar as the proportion of invasive isolates with reduced susceptibility to penicillins increased from 67% to 79% in 2007. However penicillins remain a suitable treatment for IMD in Australia. All isolates were susceptible to the third generation cephalosporins and to the 'clearance' antibiotic rifampicin. Of particular interest was a serogroup B isolate from New South Wales with reduced susceptibility to ciprofloxacin (0.06 mg/L). The first ever reported case of an invasive *N. meningitidis* with reduced susceptibility to fluoroquinolones and where the molecular basis of the resistance mechanism involved was also described arose from surveillance conducted by this program.[12] Subsequently, other sporadic cases of meningococci

with reduced quinolone susceptibility have been reported in several countries, and more recently, clusters of quinolone less-susceptible meningococci have also been described in India[13] and the United States of America.[14] Serogroups A, B, C and Y have all exhibited this decreased quinolone susceptibility and a number of different resistance mechanisms are now known to be involved. Invasive meningococci possess the potential to develop full resistance to quinolones (similar to MIC levels now seen in quinolone-resistant *Neisseria gonorrhoeae*)[15] so that antimicrobial resistance surveillance remains an important component of Australian Meningococcal Surveillance Programme activities.

Acknowledgements

Isolates were received in the reference centres from many laboratories throughout Australia. The considerable time and effort involved in forwarding these strains is recognised and these efforts are greatly appreciated. These data could not have been provided without this assistance and the help of clinical colleagues and public health personnel.

The Australian Government Department of Health and Ageing provided a grant to the National Neisseria Network.

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Prevalence of MRSA strains among *Staphylococcus aureus* isolated from outpatients, 2006

Report from the Australian Group for Antimicrobial Resistance

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

Abstract

Biennial community-based *Staphylococcus aureus* antimicrobial surveillance programs have been performed by the Australian Group for Antimicrobial Resistance (AGAR) since 2000. Over this time the percentage of *S. aureus* identified as methicillin resistant has increased significantly from 10.3% in 2000 to 16% in 2006. This increase has occurred throughout Australia and has been due to the emergence of community-associated MRSA (CA-MRSA) clones. However, healthcare associated MRSA were still predominant in New South Wales/ Australian Capital Territory and Victoria/Tasmania. In the 2006 survey CA-MRSA accounted for 8.8% of community-onset *S. aureus* infections. Although multiple CA-MRSA clones were characterised, the predominate clone identified was Queensland (Qld) MRSA (ST93-MRSA-IV) a Panton-Valentine leukocidin (PVL) positive MRSA that was first reported in Queensland and northern New South Wales in 2003 but has now spread throughout Australia. Several international PVL-positive CA-MRSA clones were also identified including USA300 MRSA (ST8-MRSA-IV). In addition, PVL was detected in an EMRSA-15 (ST22-MRSA-IV) isolate; a hospital associated MRSA clone that is known to be highly transmissible in the healthcare setting. With the introduction of the international clones and the transmission of Qld MRSA throughout the country, over 50% of CA-MRSA in Australia are now PVL positive. This change in the epidemiology of CA-MRSA in the Australian community will potentially result in an increase in skin and soft tissue infections in young Australians. As infections caused by these strains frequently results in hospitalisation their emergence is a major health concern. *Commun Dis Intell* 2009;33:10–20.

Keywords: *Staphylococcus aureus*, MRSA, healthcare-acquired infection, antimicrobial resistance, epidemiology

Introduction

The emergence of methicillin-resistant *Staphylococcus aureus* (MRSA) causing community-onset infections has represented a major change in the epidemiology of *S. aureus*. Community-onset MRSA (CO-MRSA) is a worldwide phenomenon with epidemics reported in many regions including Canada,[1] the United States of America[2] and Europe.[3] These reports have a number of findings in common including: lack of association with risk factors for healthcare-associated acquisition of MRSA; lack of resistance to non-β-lactam antibiotics; frequent association with Indigenous populations; and association with subcutaneous abscess formation and necrotising pneumonia.[3] The latter clinical conditions have been shown to correlate strongly with possession of the genes for Panton-Valentine leukocidin (PVL)+, an extracellular toxin that destroys leucocytes and causes tissue necrosis. Furthermore, unlike hospital-onset MRSA (HO-MRSA) epidemics, which are due to a relatively small number of MRSA clones, CO-MRSA epidemics are polyclonal. Although global transmission of some community-associated MRSA (CA-MRSA) clones has occurred, most clones are believed to have evolved independently of one another with little or no evidence of global transmission.

Australia has had a unique experience with CO-MRSA in that the first epidemic in Western Australia was documented earlier than in most countries.[5,6] It was due initially to a PVL-negative clone and subsequently to a great variety of clones, including some that are PVL-positive.[7] Epidemics initially developed quite separately with distinct clones in different parts of the country. This might be expected in a country with relatively few dense concentrations of population separated by large areas, often desert, with very sparse population.

The Australian Group for Antimicrobial Resistance (AGAR) has previously established that the major CA-MRSA clones circulating the community were WA MRSA-1 (ST1-MRSA-IV), the 'south-west Pacific' (SWP) clone (ST30-MRSA-IV), and the Qld clone (ST93-MRSA-IV), which were widely dispersed geographically.[8] Both the SWP and Qld clones usually carry PVL genes and are associated with abscess formation, bacteraemia and necrotising pneumonia.[9]

In this paper we report the prevalence of antimicrobial resistance in clinical isolates of *S. aureus* throughout Australia in an outpatient population, and describe changes in prevalence and geographic range of MRSA clones and the extent of PVL gene carriage in these strains.

Methods

Thirty laboratories from all 6 states, the Australian Capital Territory and the Northern Territory participated in the survey. Commencing on 10 June 2006, each laboratory collected up to 100 consecutive significant clinical isolates from patients attending primary care clinics, outpatient clinics, emergency departments or other outpatient settings, or residing in long-term care facilities. Dialysis and day surgery patients were excluded. Only 1 isolate per patient was tested and no isolates from screening swabs or from specimens received for the purpose of gathering surveillance data were included.

Species identification

S. aureus was identified by morphology and positive results of at least two of 3 tests: slide coagulase test, tube coagulase test, and demonstration of deoxyribonuclease production.[10] Additional tests such as fermentation of mannitol or growth on mannitol-salt agar may have been performed for confirmation.

Susceptibility testing methodology

Participating laboratories performed antimicrobial susceptibility tests using the Vitek2® AST-P545 card (BioMerieux, Durham, NC). Antimicrobials tested were benzylpenicillin, oxacillin, cefazolin, vancomycin, rifampicin, fusidic acid, gentamicin, erythromycin, clindamycin, tetracycline, trimethoprim/sulphamethoxazole (cotrimoxazole), ciprofloxacin, quinupristin/dalfopristin (Synercid®), teicoplanin, linezolid, imipenem, and nitrofurantoin. Results were interpreted for non-susceptibility according to Clinical and Laboratory Standards Institute (CLSI) breakpoints.[11] Penicillin susceptible strains were tested for β-lactamase production using nitrocefin. A cefoxitin disc diffusion test was used to confirm methicillin-resistance. Mupirocin and cefoxitin were tested by disc diffusion using the CLSI or calibrated dichotomous sensitivity (CDS) methods.[11,12] The tigecycline minimum inhibitory concentration of all isolates was determined by Etest® (AB Biodisk, Solna, Sweden).

Characterisation of methicillin-resistant *Staphylococcus aureus*

Resistogram typing was performed by disk diffusion against a panel of 6 chemicals and dyes as previously described.[13,14] Coagulase gene restriction fragment length polymorphism typing was performed as described elsewhere.[15] Pulsed-field gel electrophoresis (PFGE) of chromosomal DNA was performed using the CHEF DRIII System (Bio-Rad Laboratories, Sydney, NSW) and interpreted as described elsewhere.[16,17] Representative isolates were characterised by multilocus sequence typing (MLST) and staphylococcal chromosomal cassette *mec* (SCC*mec*) typing with results interpreted as described previously.[8,18,19]

Clones are reported with their common names (e.g. WA MRSA-I) followed by the sequence type (ST), methicillin resistance phenotype, and SCC*mec* type (I to V) (e.g. ST1-MRSA-IV). Clones are classified into 2 groups on the basis of previously published evidence: those implicated in healthcare-associated infection and those implicated in community-associated infection.

MRSA isolates were assayed for the presence of PVL genes using polymerase chain reaction (PCR) primers for a 1554-bp region from the *lukS-PV* and *lukF-PV* genes.[20]

Statistical analysis

The proportions and 95% confidence intervals (CI) were calculated for MRSA by laboratory, state or territory, age, source, invasiveness of infection (blood, sterile site or cerebrospinal fluid isolates) and antibiogram. Odds ratio for the association of age and MRSA was examined after age of patient was categorised into one of 5 age groups. All descriptive and inferential statistics were calculated using Epi Info version 6.0.4 (Centers for Disease Control and Prevention, Atlanta, Georgia, USA) with the alpha level set at the 5% level for 2-sided tests for significance.

Results

Participating laboratories (26 public and 4 private) were located in New South Wales (8), the Australian Capital Territory (1), Queensland (5), Victoria (6), Tasmania (2), the Northern Territory (1), South Australia (3) and Western Australia (4). To ensure institutional anonymity data have been combined for New South Wales and the Australian Capital Territory, Victoria and Tasmania and for Queensland and the Northern Territory (Table 1). There were 2,979 isolates included in the survey with the majority (76.7%) of isolates contributed by New South Wales/Australian Capital Territory (30.0%), Victoria/Tasmania (26.5%) and Queensland/Northern Territory (20.1%).

Specimen source

Skin and soft tissue infections (SSTI) specimens contributed the majority (81.0%, 95% CI 79.6–

Table 1. Number of institutions and *Staphylococcus aureus* isolates collected in state or territory

Region	Number of institutions	Total	%
New South Wales/ Australian Capital Territory	9	895	30.0
Queensland/ Northern Territory	6	600	20.1
South Australia	3	299	10.0
Victoria/Tasmania	8	788	26.5
Western Australia	4	397	13.3
Total	30	2,979	100.0

Table 2. Number and proportion of isolates associated with specimen types (where known)

Specimen source	Number	% (95% CI)
Skin and soft tissue	2,414	81.0 (79.6–82.4)
Respiratory	177	5.9 (5.1–6.9)
Ear	109	3.7 (3.0–4.4)
Blood	106	3.6 (2.9–4.3)
Urine	96	3.2 (2.6–3.9)
Eye	48	1.6 (1.2–2.1)
Sterile site	28	0.9 (0.96–1.4)
Total	2,978	
Invasive	134	4.5 (3.8–5.3)
Non-invasive	2,844	95.5 (94.7–96.2)

CI – confidence interval

82.4%) of isolates followed by respiratory specimens (5.9%, 95% CI 5.1%–6.9%) while blood culture isolates contributed only 3.6% (95% CI 2.9%–4.3%) of the total with significantly ($P<0.0001$) more isolates causing non-invasive (95.5%) than invasive (4.5%) infections (Table 2).

Susceptibility and typing results

The proportion of MRSA was 16.0% (95% CI 14.7%–17.3%) nationally (Table 3), which was not significantly different from the proportion identified in 2004 (15.3%) ($P = 0.55$). At a regional level the proportions of MRSA identified in 2004 and 2006 were stable in New South Wales/Australian Capital Territory (19.8% in 2004 to 23.0%, NS), South Australia (10.3% to 12.0%, NS), Victoria/Tasmania (10.7% to 12.7%, NS) and Western Australia (13.0% to 11.3%, NS), while Queensland/Northern Territory showed a significant decrease (19.8%–14.8%, $P = 0.0494$).[9] The proportion of invasive isolates (blood/sterile sites) that were MRSA was 10.4% overall and did not vary significantly ($P = 0.6563$) between regions. Urinary isolates included a significantly ($P<0.0001$, $X^2=42.59$) greater proportion of MRSA (33.3%, 95% CI 24.2%–43.8%) than any other specimen types (Table 4).

Of the 476 *S. aureus* identified as MRSA, 462 were referred to the WA Gram-positive Bacteria Typing and Research Unit for epidemiological typing.

The proportion of MRSA that were healthcare-associated MRSA (HA-MRSA) clones varied markedly between regions, ranging from 11.4% in Western Australia to 57.1% in Victoria/Tasmania ($P<0.0001$) (Figure 1). More than half of all MRSA in Victoria/Tasmania (56/98, 57.1%) and New South Wales/Australian Capital Territory (110/198, 55.6%) were HA-MRSA, whereas a quarter or less were HA-MRSA in South Australia (9/36, 25.0%), Queensland/Northern Territory (20/86, 23.3%) and Western Australia (5/44, 11.4%). Of HA-MRSA, Eastern Australian (EA)-MRSA (ST239-MRSA-III) predominated in the eastern regions ranging from 52.7% in New South Wales/Australian Capital Territory to

Table 3. Proportion of *Staphylococcus aureus* that are methicillin-resistant, by region and source

	% (95%Confidence interval) (n/N)						Difference across regions
	NSW/ACT	Qld/NT	SA	Vic/Tas	WA	Aus	X^2 *P*
All	23.0 (20.3–25.9) (206/895)	14.8 (12.1–18.0) (89/600)	12.0 (8.7–16.4) (36/299)	12.7 (10.5–15.3) (100/788)	11.3 (8.5–15.0) (45/397)	16.0 (14.7–17.3) (476/2,979)	49.79 <0.0001
Invasive	15.4 (5.0–35.7)| (4/26)	12.5 (4.1–29.9) (4/32)	0.0 (0.0–40.2) (0/8)	10.9 (4.5–22.9) (6/55)	0.0 (0.0–28.3) (0/13)	10.4 (6.0–17.2) (14/134)	3.284 0.6563
Non–invasive	23.2 (20.5–26.2) (202/869)	15.0 (12.2–18.2) (85/568)	12.4 (8.9–16.8) (36/291)	12.8 (10.5–15.5) (94/733)	11.5 (8.6–15.2) (44/383)	16.2 (14.9–17.6) (461/2,844)	47.95 <0.0001

X2 – chi-square *P* – probability

80.0% in Queensland/Northern Territory but was not isolated in Western Australia. Queensland/Northern Territory had the lowest proportion (4/20, 20.0%) of EMRSA-15 (ST22-MRSA-IV).

Table 4. Proportion of *Staphylococcus aureus* that are methicillin-resistant, by source (where known)

Specimen source	% MRSA	95% CI
Skin and soft tissue	16.0 (387/2,414)	14.6–17.6
Respiratory	19.8 (35/177)	14.3–26.6
Ear	3.7 (4/109)	1.2–9.7
Blood	11.3 (12/106)	6.2–19.3
Urine	33.3 (32/96)	24.2–43.8
Eye	6.3 (3/48)	1.5–17.5
Sterile site	7.1 (2/28)	1.9–23.7

CI – confidence interval

Among the CA-MRSA strains, the Qld clone (ST93-MRSA-IV) was predominant in New South Wales/Australian Capital Territory (49/88, 55.7%) and Queensland/Northern Territory (20/66, 30.3%), while Western Australia MRSA-1 (ST1-MRSA-IV) accounted for approximately half of the isolates in Western Australia (19/39, 48.7%) and South Australia (15/27, 55.6%) (Figure 2). Victoria/Tasmania had the greatest diversity of community clones and was unique in having a large proportion (19/39, 21.4%) of the Victorian MRSA clone (ST45-MRSA-IV).

Resistance in *S. aureus* to non-β-lactam antimicrobials with the exception of rifampicin and fusidic acid varied significantly between states (Table 5). Resistance to gentamicin, tetracycline and trimethoprim/sulphamethoxazole was highest in New South Wales/Australian Capital Territory, Victoria/Tasmania, and Queensland/Northern Territory, reflecting the higher proportion of MRSA isolates in these regions that were EA-MRSA (Figure 1).

Resistance to non-β-lactam antimicrobials in MRSA clones with more than 30 isolates is shown in Table 6. EA-MRSA had very high levels of resistance to erythromycin, cotrimoxazole, tetracycline, ciprofloxacin and gentamicin, but low levels of

Figure 1. Proportion of healthcare associated methicillin-resistant *Staphylococcus aureus* clones, by regions

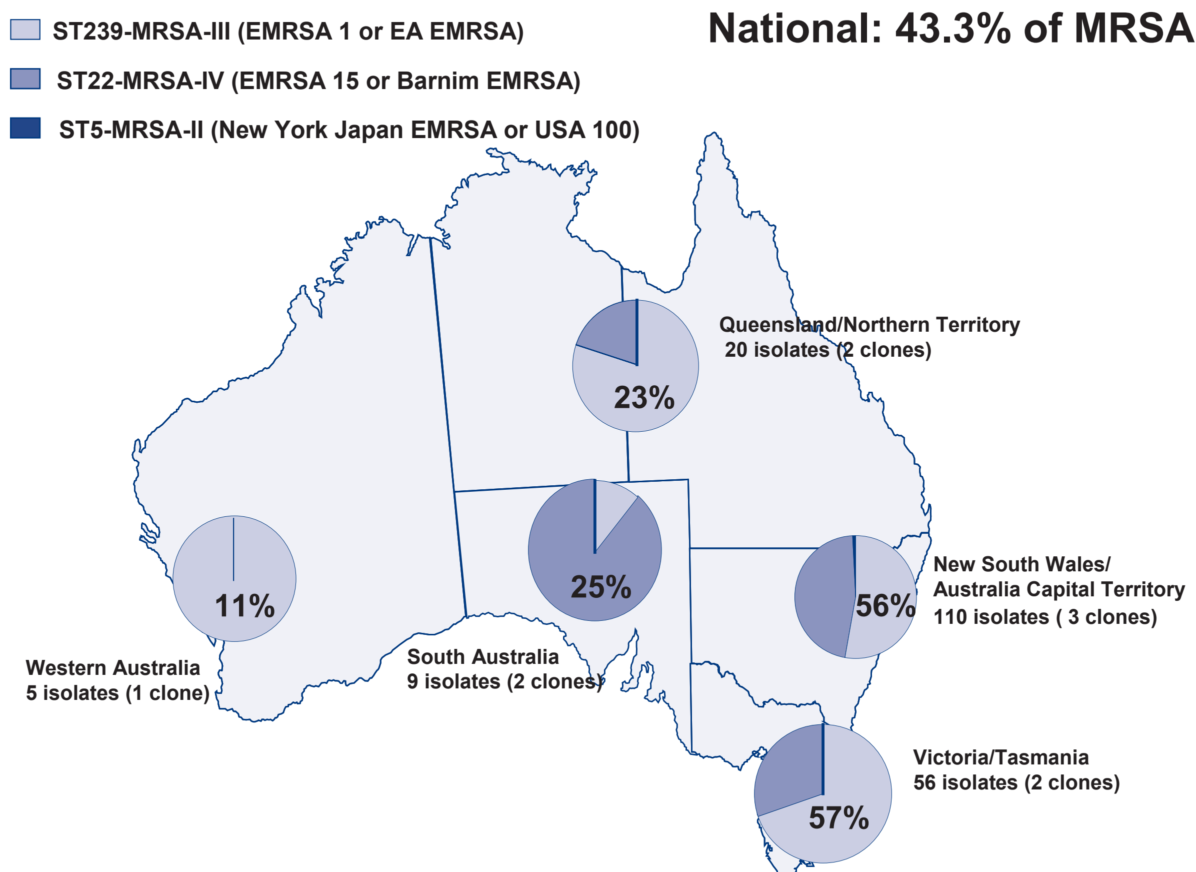


Figure 2. Proportion of community associated methicillin-resistant *Staphylococcus aureus* clones, by region

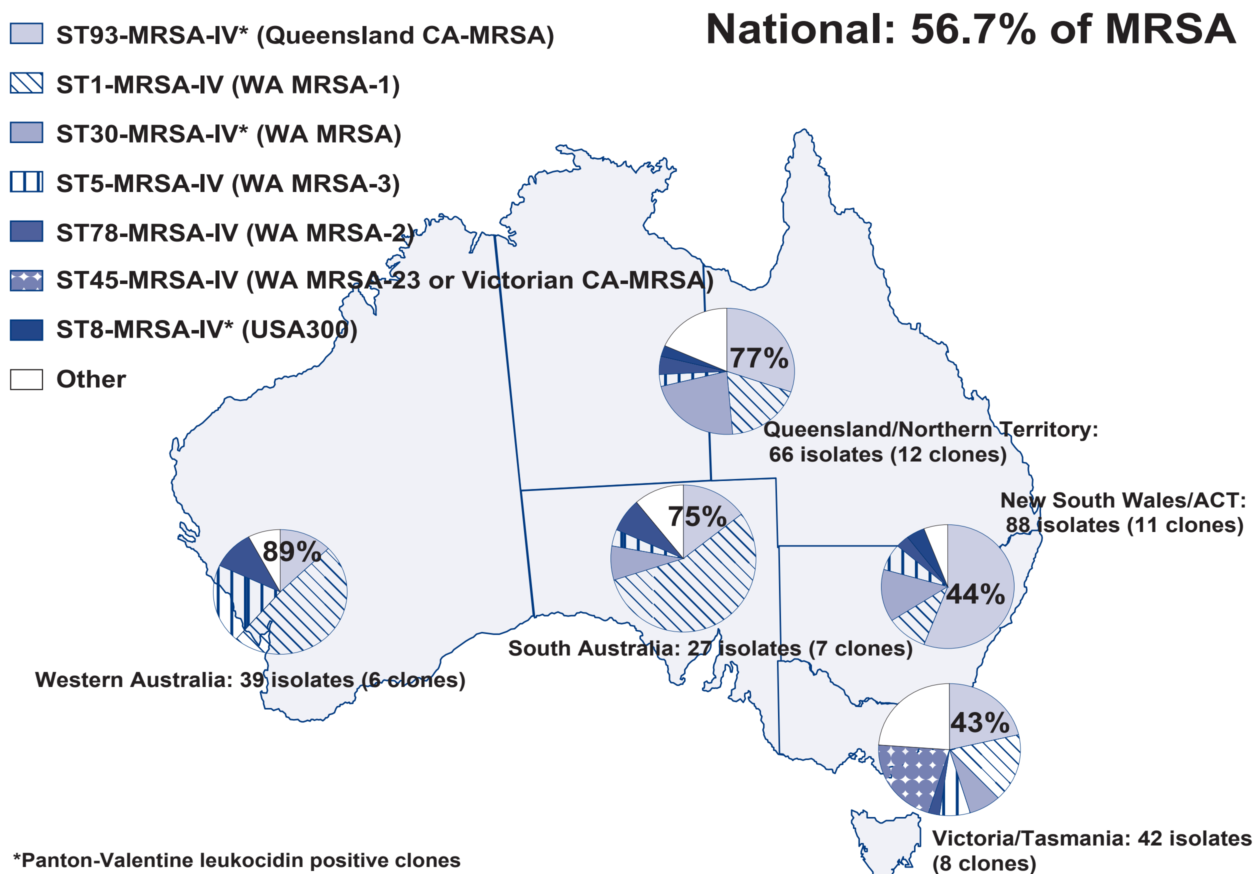


Table 5. Proportion (and number) of *Staphylococcus aureus* non-susceptible to non-β-lactams

Drug	NSW/ACT		Qld/NT		SA		Vic/Tas		WA		Aus		Difference across regions	
	%	n	%	n	%	n	%	n	%	n	%	n	X^2	*P*
Total isolates	895		600		299		788		397		2,979			
Erythromycin	20.1	180	16.3	98	16.7	50	16.4	129	13.1	52	17.1	509	10.79	0.0557
Tetracycline	10.4	93	5.5	33	2.0	6	8.9	70	3.3	13	7.2	215	40.71	<0.0001
Trimethoprim-sulphamethoxazole	8.9	80	3.8	23	1.3	4	7.4	58	0.8	3	5.6	168	54.56	<0.0001
Ciprofloxacin	14.6	131	4.5	27	4.3	13	10.2	80	2.0	8	8.7	259	84.65	<0.0001
Gentamicin	7.6	68	4.3	26	0.7	2	5.8	46	0.3	1	4.8	143	46.61	<0.0001
Fusidic acid	3.5	31	5.8	35	4.3	13	4.3	34	4.5	18	4.4	131	4.831	0.4369
Mupirocin	1.3	12	3.3	20	0.7	2	1.1	9	2.3	9	1.7	52	14.00	0.0156
Rifampicin	1.0	9	1.2	7	0.0	0	0.4	3	0.0	0	0.6	19	9.848	0.0797

X^2 – chi-square *P* - probability

resistance to rifampicin, fusidic acid and mupirocin. For EMRSA-15 almost all isolates were ciprofloxacin resistant and approximately half were erythromycin resistant, while resistance to other agents was uncommon. Of the major community-associated clones, the Qld clone was unique in being invariably susceptible to non-β-lactam antimicrobials except erythromycin. Approximately a third of WA MRSA-1 isolates were resistant to erythromycin and fusidic acid. Both WA MRSA-1 and SWP MRSA had much higher levels of resistance to mupirocin than was seen in other clones. The prevalence of clindamycin resistance approximates that of erythromycin as most erythromycin resistance in *S. aureus* in Australia is due to the inducible MLS_B mechanism.

Table 6. Methicillin-resistant *Staphylococcus aureus* clones: proportion non-susceptible and mean age

Clone	MLST/SCC*mec*$^{\Sigma}$	n	Ery	Tet	Tmp-SXT	Cip	Gen	Fus	Mup	Rif	Mean age (95% CI)
EA-EMRSA	ST239/III	114	99.1	96.5	97.4	94.7	93.9	1.8	0.9	4.4	64.7 (61.0–68.4)
Qld MRSA	ST93/IV	87	9.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	30.5 (26.5–34.5)
EMRSA-15	ST22/IV	85	52.9	2.0	0.0	97.6	1.2	0.0	1.2	2.4	71.9 (67.9–75.9)
WA MRSA-1	ST1/IV	62	33.9	1.6	0.0	3.2	8.1	33.9	8.1	1.6	49.1 (41.6–56.5)
SWP MRSA	ST30/IV	31	3.2	3.2	0.0	0.0	0.0	0.0	6.0	0.0	31.5 (23.9–39.0)
Other clones	various	83	43.4	12.0	3.6	20.5	6.0	2.4	2.4	3.6	47.8 (41.4–54.2)

MLST – multi-locus sequence type, SCCmec – staphylococcal chromosomal cassette, mec Ery – erythromycin, Tet – tetracycline Tmp-SXT – trimethoprim-sulphamethoxazole, Cip – Ciprofloxacin, Gen – gentamicin, Fus – fusidic acid, Mup – mupirocin, Rif – rifampicin, CI – confidence interval

No resistance was detected to vancomycin, teicoplanin, quinupristin-dalfopristin or linezolid. Seven of 2,979 (0.2%) isolates were classified as resistant to tigecycline using the US FDA and EUCAST breakpoints of 0.5mg/L. The regional level of resistance to penicillin and non-β-lactam antimicrobials in methicillin-susceptible *Staphylococcus aureus* (MSSA) is shown in Table 7.

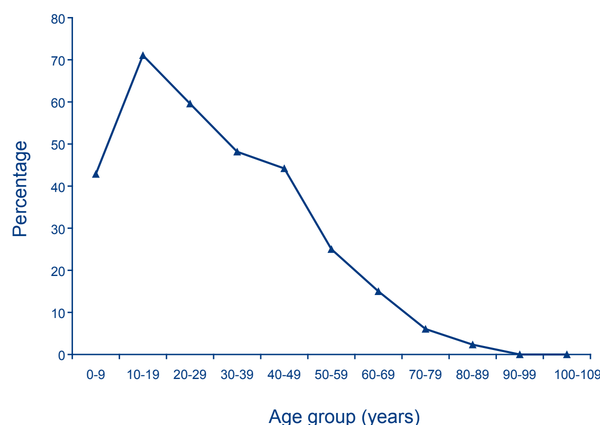
Prevalence of Panton-Valentine leukocidin

One isolate belonging to a HA-MRSA clone (EMRSA-15) was PVL positive (Table 8). The Qld clone, which was the predominant community-associated clone overall with 87 isolates, was invariably PVL positive. Other PVL positive clones included SWP, WA MRSA-1 (indistinguishable from USA400 by standard typing methods), USA300 (ST8-MRSA-IV), the European clone (ST80-MRSA-IV) and the Taiwanese clone (ST59-MRSA-V_T).

Relationship of age to methicillin-resistant *Staphylococcus aureus* prevalence

The mean age of patients with MRSA (52.2 (95% CI 49.7–54.7)) was significantly higher (P<0.0001) than that for patients with MSSA (44.7 (95% CI 43.6–45.7)). The mean age of patients with various MRSA clones also differed significantly: the mean ages for HA-MRSA clones (EA-MRSA and EMRSA-15) were highest, those for the PVL positive CA-MRSA clones (Qld and SWP) were lowest and the PVL negative community-associated WA MRSA-1 and the minor clones were in between (Table 6). The proportion of MRSA that was PVL positive was much higher in the young, peaking in the 10–19 year age group and falling steadily to zero in the 90–99 year age group (Figure 3). Of those patients presenting with SSTIs due to MRSA, a PVL positive clone was the most likely cause for those patients in the 10–19 (26/35 74.3% (95% CI 57.6%–86.0%) and the 20–29 year age groups (26/41 63.4% (95% CI 48.0%–76.5%).

Figure 3. Proportion of methicillin-resistant *Staphylococcus aureus* that are Panton-Valentine leukocidin positive, by age group



Discussion

Biennial community-based *S. aureus* antimicrobial surveillance programs have been performed in Australia by AGAR since 2000.[9] Over this time the number of participating laboratories has varied from

Table 7. Methicillin-susceptible *Staphylococcus aureus*: number and proportion (%) non-susceptible

Drug	NSW/ACT		Qld/NT		SA		Vic/Tas		WA		Aus		Difference across region	
	n	%	n	%	n	%	n	%	n	%	n	%	X^2	*P*
Total isolates	689		511		263		688		352		2,503			
Penicillin	582	84.5	445	87.1	224	85.2	588	85.5	296	84.1	2,135	85.3	2.104	0.8346
Erythromycin	70	10.2	65	12.7	39	14.8	69	10.0	35	9.9	278	11.1	6.956	0.2239
Clindamycin*	7	1.0	3	0.6	0	0.0	7	1.0	4	1.1	21	0.8	3.512	0.6216
Tetracycline	29	4.2	15	2.9	5	1.9	27	3.9	13	3.7	89	3.6	3.822	0.5752
Trimethoprim-sulphamethoxazole	23	3.3	6	1.2	3	1.1	16	2.3	3	0.9	51	2.0	11.57	0.0412
Ciprofloxacin	17	2.5	5	1.0	3	1.1	15	2.2	3	0.9	43	1.7	6.899	0.2283
Gentamicin	9	1.3	5	1.0	1	0.4	7	1.0	1	0.3	23	0.9	3.625	0.6046
Fusidic acid	25	3.6	27	5.3	9	3.4	32	4.7	13	3.7	106	4.2	2.988	0.7018
Rifampicin	4	0.6	5	1.0	0	0.0	1	0.1	0	0.0	7	0.3	4.635	0.4620
Mupirocin	10	1.5	13	2.5	2	0.8	5	0.7	9	2.6	39	1.6	9.770	0.0820

X^2 – chi-square, *P* - probability

* Constitutive resistance

No resistance was detected to vancomycin, teicoplanin, quinupristin-dalfopristin or linezolid.

24 to 30 institutions with the percentage of *S. aureus* identified as MRSA increasing significantly from 10.3% in 2000[9] to 16% in 2006 ($P<0.0001$).

In the 2006 program the percentage of *S. aureus* identified as MRSA ranged from 11.3% in Western Australia to 23% in New South Wales/Australian Capital Territory. When compared with the 2004 program a significant decrease was observed in Queensland/Northern Territory (19.8% to 14.8%), which may be related to an increase in regional participation to 6 laboratories in 2006 with a resulting change in the study population. The increase in MRSA between 2000 and 2006 has primarily been due to the emergence of CA-MRSA clones in the Australian community. Of the 462 MRSA referred to the WA Gram-positive Bacteria Typing and Research Unit in 2006, 200 (6.7% of all *S. aureus*) and 262 (8.8%) were classified as HA-MRSA and CA-MRSA clones, respectively. When compared with the 2000 program the percentage of *S. aureus* characterised as HA-MRSA has not significantly increased (5.6% to 6.7%). However the percentage of *S. aureus* characterised as CA-MRSA has almost doubled increasing from 4.7% to 8.8% ($P<0.0001$). Consequently, the emergence of CA-MRSA has resulted in a significant increase in the burden of CO-MRSA disease in Australia.

Three HA-MRSA clones were identified in this survey: EA EMRSA (ST239-MRSA-III), EMRSA-15 (ST22-MRSA-IV) and New York/Japan MRSA (ST5-MRSA-II). EA-MRSA, initially reported in eastern Australia in the late 1970s[21, 22] and possibly the most successful international HA-MRSA clone, accounted for approximately 25% of CO-MRSA infections. Although EA-MRSA continues to be the most prevalent MRSA isolated in the Australian community, since the 2000 survey (when it accounted for almost half of CO-MRSA isolated[9]) it has decreased significantly in most regions of Australia; including New South Wales/Australian Capital Territory (50.4% to 29.3%), South Australia (40.0% to 2.8%) and Victoria/Tasmania (80.6% to 39.8%). However in Queensland/Northern Territory the percentage was similar ranging from 22.2% in 2000 to 18.6% in 2006, which once again may be due to a major change in the study population in the region in 2006. As in previous surveys, little or no EA-MRSA was isolated in Western Australia. This is due to the WA 'MRSA search and destroy' policy introduced in 1982, which has prevented EA-MRSA from becoming established in the state's hospitals and spilling over into the community.

In contrast to EA-MRSA, the percentage of MRSA identified as EMRSA-15 has increased in most regions of Australia. EMRSA-15 is an international HA-MRSA associated with hospital infection and was first documented in Australia in Perth in 1997, where it was detected during the pre-employment screening of healthcare workers coming from the United Kingdom (UK) and Ireland.[23] This clone is now well established throughout Australia increasing from 11.7% of CO-MRSA infections in 2000 to 18.4% in 2006.[9] During this time, significant increases have been reported in New South Wales/Australian Capital Territory (18.4% to 25.8%),

Table 8. Methicillin-resistant *Staphylococcus aureus* clones: Panton-Valentine leukocidin polymerase chain reaction results

Clone	MLST/SCCmec	Number of isolates	PVL PCR positive	%
EA-EMRSA	ST239/III	114	0	0
Qld MRSA	ST93/IV	87	87	100
EMRSA-15	ST22/IV	85	1	1
WA MRSA-1	ST1/IV	62	5	8
SWP MRSA	ST30/IV	31	30	97
WA MRSA-3	ST5/IV	20	0	0
WA MRSA-2	ST78/IV	13	0	0
Victorian MRSA	ST45/IV	9	0	0
NT MRSA	ST75/IV	7	0	0
USA300	ST8/IV	6	6	100
WA MRSA-4	ST45/V	5	0	0
WA MRSA-5	ST8/IV	3	0	0
European MRSA	ST80/IV	2	2	100
Taiwan MRSA	ST59/V_T	2	2	100
---------	ST5/V	2	0	0
New York/Japan	ST5/II	1	0	0
WA MRSA-13	ST584/IV	1	0	0
WA MRSA-15	ST59/IV	1	0	0
WA MRSA-35	ST5/V	1	0	0
WA MRSA-69	ST12/IV	1	0	0
SWP variant	ST30/IV	1	1	100
---------	ST1/V	1	0	0
---------	ST20/V	1	0	0
---------	ST30slv*/IV	1	1	100
---------	ST338/V	1	1	100
---------	ST361/IV	1	0	0
---------	ST72/V	1	0	0
---------	ST7/V	1	0	0
---------	ST8/IV	1	0	0
Total MRSA		462	136	29

MLST multi-locus sequence type
SCCmec staphylococcal chromosomal cassette mec
PVL Panton-Valentine leukocidin
PCR polymerase chain reaction
* slv – single locus variant

South Australia (12% to 22.2%), Victoria/Tasmania (0% to 17.4%) and Western Australia (6.8% to 11.4%). The percentage has not significantly altered in Queensland/Northern Territory (3.7% to 4.7%).

In Western Australia EMRSA-15 has become a successful coloniser in many of the state's long term care facility residents, who with healthcare workers from the UK and Ireland have become the major source of EMRSA-15. The mean ages of EMRSA-15 and EA-MRSA patients in this survey were significantly higher than that seen with other MRSA clones, which is consistent with their known association with healthcare related infection and with long term care facilities.

A single isolate of New York/Japan MRSA (ST5-MRSA-II) was isolated in 2006. Although this clone is a major HA-MRSA in the United States of America (where it is also known as USA100) in recent years it has become a major cause of CO-MRSA infections.[24] In Australia, a single strain outbreak of

New York/Japan was recently reported in regional Western Australia.[25] The outbreak which involved several hospitals and long term care facilities was linked to an Australian healthcare worker who had previously worked and received surgery in a New York city hospital. Although the outbreak was able to be contained, the strain continues to be recovered from residents living in the region's long term care facilities. From the Western Australia experience it has been demonstrated that New York/Japan MRSA is highly transmissible and therefore may become a major MRSA strain in both the hospital and community setting if the opportunity arises.

As has been reported overseas, CA-MRSA in Australia is polyclonal. In this survey 25 different clones were characterised by MLST/SCC*mec* typing. Using BURST analysis these clones can be grouped into 13 clonal clusters and 2 singletons. Within some clonal clusters more than 1 clone was identified. In addition, both SCC*mec* IV and V were described. This suggests that CA-MRSA clones have evolved on multiple occasions in Australia with the emergence of new clones due to the horizontal and vertical transfer of SCC*mec* into *S. aureus* with diverse genetic backgrounds. Although multiple CA-MRSA clones were identified, as in previous surveys over 85% of CA-MRSA can be classified into 6 clones; Qld MRSA (ST93-MRSA-IV) (18.8% of MRSA), WA MRSA-1 (ST1-MRSA-IV) (13.4%) SWP MRSA (ST30-MRSA-IV) (6.9%), WA MRSA-3 (ST5-MRSA-IV) (4.3%), WA MRSA-2 (ST78-MRSA-IV) (2.8%) and the Victorian MRSA (ST45-MRSA-IV) (1.9%) In contrast to previous surveys, ST93-MRSA-IV (a PVL positive clone), which is now found throughout Australia, has become the predominant CA-MRSA clone in Australia.

The predominance of the Qld MRSA clone has resulted in a significant change in the percentage of CA-MRSA in Australia that are PVL positive. Initially, CO-MRSA infections in Australia were dominated by PVL negative strains including WA MRSA-1, WA MRSA-2, WA MRSA-3, NT MRSA (ST75-MRSA-IV) and the Victorian MRSA (ST45-MRSA-IV). However with the emergence of the Qld MRSA and the introduction of several international PVL-positive community-associated MRSA strains, including SWP (ST30-MRSA-IV), USA300 (ST8-MRSA-IV), USA400 (ST1-MRSA-IV), European MRSA (ST80-MRSA-IV) and the Taiwan MRSA (ST59-MRSA-V_T), over 50% of CA-MRSA in Australia are now PVL positive. Although the mean age of patients with MRSA was significantly older compared with MSSA (52 vs 45 years; $P<0.001$) 70% of PVL-positive MRSA infections occurred in patients 10–19 years of age.

Although transmission of PVL-positive CA-MRSA in the community has been reported, outbreaks of these strains within the hospital environment have not. In this survey a PVL-positive EMRSA-15 strain was identified. EMRSA-15 is a highly transmissible HA-MRSA frequently isolated in many Australian hospitals and long term care facilities.

In conclusion, the AGAR 2006 *S. aureus* surveillance program has shown that the proportion of community-onset *S. aureus* infections due to MRSA is increasing throughout Australia, and that this increase is due to the spread of the PVL-positive Qld MRSA clone as well as the introduction of several international PVL-positive clones including USA300 (ST8-MRSA-IV). This shift in the molecular epidemiology of MRSA clones in the Australian community will potentially increase the number of SSTI in young Australians. As SSTI caused by PVL-positive *S. aureus* frequently results in hospitalisation the emergence of these strains in the community as well as the detection of a PVL-positive healthcare-associated MRSA strain (EMRSA-15) is a major health concern.

A full detailed report of this study may be found on the Australian Group on Antimicrobial Resistance website: http://www.antimicrobial-resistance.com/ under 'AMR surveillance'.

Acknowledgements

This study was partially supported by a grant from the Commonwealth Department of Health and Ageing.

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Peer reviewed articles

Vaccine failures and vaccine effectiveness in children during measles outbreaks in New South Wales, March–May 2006

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Abstract

During March to May 2006 the highest incidence of measles in New South Wales since 1998 provided an opportunity to estimate the effectiveness of the measles-mumps-rubella (MMR) vaccination program in preventing childhood measles, and describe any differences in clinical presentation between vaccinated and unvaccinated children. We reviewed records of all 33 notified cases of measles in children aged 1–14 years during a state-wide outbreak in New South Wales from March – May 2006. Six of the children had a confirmed history of vaccination with at least 1 dose of MMR. The children with previous vaccination tended to have milder disease than those without vaccination as judged by their reported number of symptoms and hospitalisation rates. The vaccinated children were less likely to have a typical measles rash. Two of the cases in previously vaccinated children may be due to secondary vaccine failure, although a lack of complete diagnostic testing limits our ability to confirm this. Vaccine effectiveness after receiving at least 1 dose of MMR is estimated to be 96% (95% CI 77.8–99%). MMR vaccination was effective in preventing measles in children during these outbreaks. *Commun Dis Intell* 2009;32:21–26.

Keywords: measles-mumps-rubella vaccine, vaccine effectiveness, disease outbreaks, child

Introduction

From March to May 2006, there were 59 cases of measles in New South Wales.[1] There were 2 distinct outbreaks during this period – the first was associated with transmission in a hospital emergency department, and the second with an Australian tour by a spiritual group from a measles-endemic country. This represented the highest incidence of measles in New South Wales since the Measles Control Campaign (primary school catch-up) in 1998 and the introduction in the same year of the 2nd scheduled dose of measles vaccine (MMR) at 4 years of age.[2] From 1999 to 2005 there was an average of only 20 cases annually in New South Wales.[3]

Since 1999 New South Wales has maintained high vaccination rates, with approximately 93% of children receiving the 12-month dose of MMR, and 85% receiving the recommended 2 doses before school entry (measured at age 6 years).[4]

It has been reported that previously vaccinated children who develop measles may have a milder course, or have different characteristics compared to vaccine naive children.[5–7] Primary vaccine failure (a failure to mount an immune response to MMR) is well recognised and thought to occur in about 5%–10% of cases after 1 dose of measles vaccine given at 12 months of age.[8] However, the entity of secondary vaccine failure (clinical infection despite a prior immune response to vaccination) is less well understood for measles.[8]

We sought to determine whether the clinical presentation of children with measles differed according to vaccination status; the reason for vaccine failure; and to estimate MMR vaccine efficacy among children in New South Wales.

Methods

Setting

New South Wales is the most populous state in Australia. Most of its 7 million residents live in the Sydney metropolitan areas (population >4 million). Public health services are mainly provided by public health units located in 8 Area Health Services, coordinated by the NSW Department of Health. Public health unit surveillance officers (PHUSO) investigate cases of notifiable diseases and enter details into the state's Notifiable Disease Database (NDD).

Case definition

In Australia, a confirmed case of measles is defined as either: positive measles-specific IgM serology; or detection of measles virus by immunofluorescence (IF), polymerase chain reaction (PCR) or culture in the presence of a compatible illness; or clinical measles (fever and/or cough and/or coryza and/or conjunctivitis and maculopapular rash) with an epidemiological link to a laboratory confirmed case.[9]

Data collection

In New South Wales measles is notifiable under the *Public Health Act 1991* by laboratories, hospitals, clinicians, school principals and childcare centre operators, hence it is assumed that all confirmed cases of measles in New South Wales are reported to the NDD. We obtained data on all cases of confirmed measles in Australian residents aged 1–14 years reported to the New South Wales NDD between 1 March and 31 May 2006.

Data on symptoms and signs were obtained by PHUSO from cases' parents and/or guardians and their health care providers and recorded on a standardised reporting form.[10] A rash was classified as typical measles when described as maculopapular and spreading from the head to the trunk then extremities.

The vaccination status of cases aged 1–7 years was confirmed against the Australian Childhood Immunisation Register.[4] For older cases, vaccination status was confirmed by parent-held or general practitioner childhood vaccination records.

Data on immunisation rates in New South Wales were obtained from the Australian Childhood Immunisation Register (ACIR), for children born between April 1998 and March 2005 (1–7 years of age during the outbreak).[4]

Laboratory testing

All positive IgM serology tests were either performed initially or confirmed using the Enzygnost Anti-Measles-Virus IgM immunoassay for measles virus-specific IgM method (Dade Behring, Marburg, Germany) at one of 4 New South Wales reference laboratories. Measles virus was detected using either IF or PCR on nasal or pharyngeal specimens as previously described.[11]

Statistical analysis

Odds ratios (comparing vaccinated and unvaccinated cases) and significance levels were calculated using Epi Info version 3.2.[12] Levels of significance were obtained using Fisher's exact test. Age-corrected vaccine effectiveness was calculated for New South Wales resident children aged 1–7 years according to the screening method described by Farrington.[13] We tested for confounding by location by calculating vaccine effectiveness by Area Health Service of residence.

Results

Between 1 March and 31 May 2006 there were 59 notifications of measles in New South Wales. Figure 1 demonstrates the onset of cases by age group during the study period. Thirty-three of these notifications met the case definition for this study. Age distribution by vaccination status is illustrated in Figure 2. Only six of the 33 children (18%) had received at least 1 dose of MMR.

Figure 1. All measles cases onset, New South Wales, 1 March to 31 May 2006, by age group

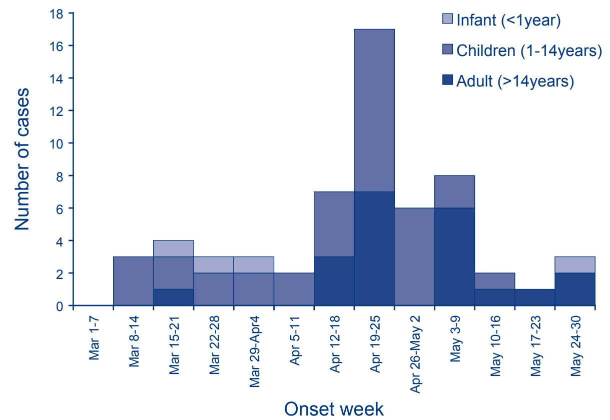
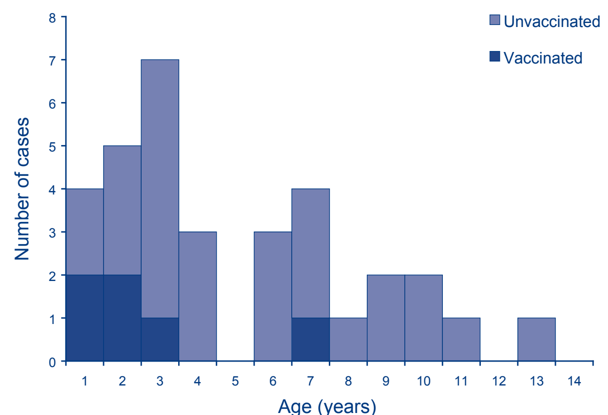


Figure 2. Age of children with measles, by vaccination status



The 6 previously vaccinated children ranged in age from 13 months to 7 years. All had received their first MMR vaccine at 12 months of age. Only one was old enough to have received a scheduled second MMR vaccine (at 4 years of age). The time elapsed since vaccination varied from 1 month to 3 years. All cases apart from one were residents of the Sydney Metropolitan region. Their characteristics are summarised in Table 1.

Table 2 compares the clinical characteristics by vaccination status of all 33 children, and also separately provides characteristics of the group of unvaccinated children aged 1–7 years for comparison, as all vaccinated children were in this age group.

Compared to the presence of atypical or no rash in four of the 6 vaccinated children, only three of the 27 unvaccinated children were reported to have an atypical rash ($\chi^2_{1df} = 9.13$, $P = 0.003$). Vaccinated children tended to have a shorter prodrome, although this was not statistically significant.

Diagnostic tests

Results of diagnostic testing on the 6 vaccinated children are summarised in Table 1. Of the 27 unvaccinated children, 10 had positive IF tests for measles antigen, six were confirmed by positive measles-specific IgM serology and 11 were diagnosed on the basis of clinical features and epidemiological links.

Calculation of vaccine effectiveness

Population rates of measles were estimated for vaccinated and unvaccinated New South Wales resident children aged between one and 7 years (excluding 1 case from this age group, resident in Queensland). There were 605,623 children born during the period 1 April 1998 to 31 March 2005 in New South Wales with records on the ACIR. The proportion of this population reported as vaccinated (PPV) is 92.9% for 1 dose and 86.6% for 2 doses of MMR (Table 3). The age-corrected vaccine effectiveness of at least 1 dose of MMR is estimated at 96% (95% CI 78.1–99.3). Cases arose in six of the 8 Area Health Services. For the Area Health Services where vaccine effectiveness could be calculated the point estimates were within these confidence intervals.

Table 1. Characteristics of measles cases in previously vaccinated children 1–14 years of age

Age	Lifetime doses MMR	Time between MMR and onset	Days from symptom onset to serum sample	IgM	IgG	Nasal swab IF
13 months	1	4 weeks	2	negative	equivocal	+
14 months	1	7 weeks	n/a	not done	not done	+
2 years	1	11 months	2	negative	+	+
2 years	1	10 months	6	low +	+	not done
3 years	1	2 years	4	negative	+	+
7 years	2	3 years	12	+	not done	not done

Table 2. Comparison of clinical characteristics of children (1–14 years), by vaccination status

	Vaccinated n=6		Unvaccinated n=27		Unvaccinated (1–7 years, n=20)		Odds ratio*	*P* value
Mean age	2.7 years		5.4 years		3.8 years			
Age range	1–7 years		1–13 years		1–7 years			
Sex (female)	3 (50%)		17 (63%)		11 (55%)		0.59	0.66
Signs and symptoms								
Fever	5	83%	27	100%	20	100%	0	0.18
Cough	4	67%	22	81%	17	85%	0.45	0.58
Coryza	3	50%	18	67%	13	65%	0.5	0.64
Conjunctivitis	3	50%	21	78%	15	75%	0.29	0.31
Koplik's spots	0	0%	4	15%	3	15%	0	1.00
Typical rash	2	33%	24	88%	20	100%	0.06	0.01†
≤2 prodromal symptoms‡	3	50%	4	15%	3	15%	5.75	0.09†
Median duration prodrome; range	2 days; 1–7 days		3 days; 0–7 days		3 days; 0–7 days			NS
Hospitalised	0	0%	4	15%	3	15%	0	1
Median number of visits§ range	2.5; 1–3		1; 0–4		1; 0–4			NS

* Comparing vaccinated and all unvaccinated children.

† Chi-squared, Fisher's exact test.

‡ Presence of only one or two of symptoms: fever, cough, coryza, conjunctivitis.

§ Visits to emergency department or general practitioner.

NS Not significant.

Table 3. Rate of measles, New South Wales resident children aged 1–7 years, by vaccination status

	Number in New South Wales	PPV	Number of cases	Rate per 100,000
One MMR (age 1–3 years)	260,738	93.3%	5	1.9
Two MMR (age 4–7 years)	344,884	86.6%	1	0.3
No MMR	42,949	0	19	44.2
Total population (age 1–7)	605,623		25	

MMR Measles-mumps-rubella

PPV Proportion of population reported as vaccinated.

Discussion

In these outbreaks we observed a differing clinical presentation in children with and without a history of MMR vaccination. The previously vaccinated children were significantly more likely to have fewer symptoms, had a non-significant shorter duration of prodrome, were significantly more likely to have an atypical rash, and none were hospitalised. This demonstrates that previously vaccinated children experienced milder disease.

Many previous case series comparing vaccinated and unvaccinated children are from developing countries where other determinants, such as malnutrition, may be important in influencing the clinical course of measles. Of the hospital-based case series, two found no difference between vaccinated and unvaccinated children in the clinical presentation or complication rate[14,15] whereas Adu in Nigeria, found that signs and symptoms were more severe among the unvaccinated children[5] and Aurangzeb in Pakistan,[6] found that mortality was significantly associated with unvaccinated status. The only community-based series from developing countries, Ibrahim in Khartoum, found that severe measles was as common in vaccinated as unvaccinated children.[16]

In developed countries, however, there have been reports of differences in disease severity related to vaccination status. In a large case series from Wisconsin, the authors found a significant association between milder measles (categorised by fewer symptoms and lower fever) in previously vaccinated children with presumed secondary vaccine failure, compared with previously vaccinated children with primary vaccine failure or unvaccinated children.[17] A case series from The Netherlands where 33 of 37 cases were vaccinated noted that measles infection was detected in patients with relatively few or atypical symptoms.[7]

In this case series the number of visits to primary care providers does not appear to be a good indicator of disease severity. The median number of visits to GPs or emergency departments per case was significantly greater for the vaccinated group of children, which may reflect the difficulty in making a diagnosis in this group of patients rather than the severity of disease. The range of number of visits was greater for the unvaccinated group. Some of this latter group who were contacts of other cases, had no visits at all to health care providers and were managed at home, whereas others repeatedly presented due to ongoing or increasing symptoms. The greater severity of disease in the unvaccinated group was demonstrated by the significantly increased number and duration of prodromal symptoms, and that 15% required hospital admission, compared to none of the vaccinated children.

Measles infection after immunisation is thought to largely arise from primary vaccine failure. In primary vaccine failure the patient fails to develop an immune response to the vaccination. This is reported to occur in 5%–10% of children after 1 vaccination at 12 months, and reduces to 1% after a second vaccination.[8] The causes of primary vaccine failure include failure of the cold chain, inadequate viral dose, and host immune factors, such as persistence of passively acquired maternal immunity.[18]

Secondary vaccine failure has also been postulated, where the patient develops an initial immune response to the vaccine and has detectable IgG antibody to measles. When challenged with wild virus however the immune response is inadequate to prevent disease. The incidence of secondary vaccine failure is not known, but has been reported as developing in 5% of children after 10 to 15 years.[8] Erdman observed 57 measles cases with a prior history of vaccination, and 55 (96%) had detectable IgM antibodies. Of these, 30 (55%) were classified as having a primary antibody response and 25 (45%) a secondary antibody response based on their ratios of IgM to IgG being greater than one (primary failure) or less than one (secondary failure). Differences in the severity of clinical symptoms between these 2 groups were consistent with this classification scheme, with cases categorised as secondary vaccine failure having significantly fewer and less severe symptoms.[19]

The interpretation of the type of vaccine failure in this case series is limited as only routine laboratory tests were available. Quantitative complement fixation on acute and convalescent serology would have assisted in determining whether vaccine failure was primary or secondary, and IgG avidity testing has also been used in this context, but neither are routinely undertaken in New South Wales.[20,21]

The study is however strengthened by the detection of measles antigen by immunofluorescence in four of the 6 vaccinated cases. Detection of measles antigen by immunofluorescence is assumed to have a specificity of 90%–95%, whereas the sensitivity depends on the quality of the specimen and is similar to culture at around 50%.[22,23] Two of the vaccinated cases were diagnosed on the basis of positive measles-specific IgM serology. Specificity of the Dade Behring method in a reference laboratory should be 97%, nevertheless these cases could be false positives. Both cases had atypical prodromal symptoms but developed classical measles rashes.

Cases 3 and 5 who had received MMR 11 months and 2 years previously may be examples of secondary vaccine failure as IgG was present at days 2 and 4 of disease respectively and IgM was not detected. The expected immunological response to primary exposure to the measles virus is a rapid rise in IgM from the appearance of the rash, peaking after 1 week. The rise in IgG is slower, and the peak occurs approximately 2 weeks after the rash.[24] The Dade Behring indirect enzyme immunoassay is reported to have a sensitivity of 88.6%, which increases from 70% in the first few days from onset of symptoms to 100% between six and 14 days after onset of symptoms.[20] We are unable to determine if the negative IgM results are false negatives as both these cases' sera were taken early in the course of the disease. False negative indirect IgM assays can also result from insufficient removal of high levels of measles-specific IgG from a test specimen.[25] Due to these factors, where measles is suspected in vaccinated children, specimens should be obtained for viral testing to allow confirmation of the diagnosis.

The comparison of the number of symptoms between the 2 groups should be viewed with caution. Symptoms were not independently verified by the investigators, but were reported by clinicians, or surveillance officers based on patient reports. Knowledge of previous vaccination against measles may have influenced reporting of symptoms, with a tendency for those with previous vaccination to be less likely to report symptoms known to be consistent with measles. The reported duration of a shorter prodrome in vaccinated cases may represent a more robust measure of milder disease as symptom and rash onset dates may be more accurately reported than the actual symptoms. Surveillance officers did use a standard form to record symptom information for each case that may have assisted in reducing measurement error.

It is interesting to note that none of the cases in vaccinated children presented as typical measles and were only confirmed by diagnostic testing. It may be that such cases are not routinely diagnosed in New South Wales, but were detected during this period due to heightened awareness of measles arising from information sent to clinicians and mass media releases.

Use of field observations in outbreaks to monitor vaccine program effectiveness is recommended.[26] The calculated measles incidence rates indicate that the vaccine failure rate in New South Wales children is low. Children who had received at least 1 dose of MMR developed measles infection at only 2.4% of the rate in the unvaccinated population, and children who received 2 doses developed measles at less than 1% of that observed in unvaccinated children. The calculated vaccine effectiveness rate of 96% compares favourably with that observed in an outbreak in Leeds of 95.1%,[13] however the impact of a 2nd dose of vaccine at 4 years cannot be accounted for by this method. The calculation of vaccine effectiveness utilised vaccination rates recorded at the Australian Childhood Immunisation Registry. As survey data indicate that ACIR records underestimate actual vaccination rates by 5%–13% the true vaccine effectiveness may be higher than we have estimated.[27,28]

Use of the screening method to assess vaccine effectiveness can also be confounded by age and location. We tested for confounding by age cohort and by area of residence (data not shown) and found that confounding was not present, indicating similar effectiveness over time and place.

Modelling of expected susceptible population numbers against the epidemic threshold predicted that there would be sufficient unimmunised children in Sydney between 2002 (in the lowest immunisation rate areas) and 2006 (in the highest immunised areas) to sustain a measles epidemic.[29] The introduction of measles to New South Wales in 2006 with a resultant epidemic has illustrated the usefulness of modelling in predicting disease control failure and underlines the importance of improving our current measles immunisation rates.

Acknowledgements

Ms Behnoosh Khalaj provided biostatistical expertise to calculate age-corrected vaccine effectiveness. The NSW Public Health Network, particularly communicable disease surveillance officers, Ken McPhie and the Virology Section of the Institute for Clinical Pathology and Medical Research and NSW Department of Health Communicable Diseases Control and Immunisation units all contributed extensively.

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Surveillance summaries

Supplementary report: surveillance of adverse events following immunisation among children aged <7 years in Australia, 1 January to 30 June 2008

Glenda L Lawrence, Deepika Mahajan, Ilnaz Roomiani

Introduction

This report summarises national passive adverse events following immunisation (AEFI) surveillance data notified to the Therapeutic Goods Administration (TGA) at 30 September 2008 for children aged less than 7 years who received vaccines between 1 January and 30 June 2008. The report includes all vaccines administered to children in this age group, with a focus on the vaccines included in the funded National Immunisation Program (NIP) schedule.[1]

There were recent changes to both the AEFI surveillance system and funded immunisation program that influence the interpretation of AEFI data for vaccines administered to children aged <7 years during January to June in 2008 compared with the same reporting period in 2007 and previous years. During mid-2007 Victoria implemented an enhanced AEFI surveillance system across the state,[2] while a pilot project of enhanced hospital-based surveillance for selected AEFI commenced in 4 tertiary paediatric hospitals (in Sydney, Melbourne, Adelaide and Perth).[3]

Changes to the immunisation program occurred on 1 July 2007 when rotavirus vaccine[4] was added to the NIP schedule for all infants, and in March 2008 when, due to an international shortage of the Pedvax® and Comvax® formulations of *Haemophilus influenza* type b (Hib) vaccine,[5] 3 states changed from using 2 combination vaccines (i.e. DTPa-IPV and Hib-HepB) to the single hexavalent DTPa-IPV-Hib-HepB formulation for children at 2, 4 and 6 months of age.[6–9] This occurred in Queensland, South Australia, and Victoria. The hexavalent vaccine has been used in all other jurisdictions since November 2005 except for the Northern Territory, which uses a pentavalent DTPa-IPV-HepB and monovalent Hib vaccine for the infant immunisation schedule.

The data reported here are provisional only. It is important to note that an AEFI is defined as a medical event that is temporally associated with immunisation but not necessarily causally associated with immunisation. Readers are referred to previous reports for a description of the national AEFI passive surveillance system,[10] methods used to analyse the data and information regarding limitations and interpretation of the data.[10–12] Often, several vaccines and reaction codes are listed in an AEFI record so the number of vaccines and reaction codes will exceed the total number of AEFI records. For the purpose of this report, an AEFI is defined as 'serious' if there is a code of life-threatening severity or an outcome code indicating recovery with sequelae, admission to hospital, prolongation of hospitalisation, or death.

Average annual population-based AEFI reporting rates were calculated using mid-2007 population estimates. Reporting rates per 100,000 doses were calculated for 10 vaccines on the NIP schedule using denominator data from the Australian Childhood Immunisation Register (ACIR).

Results

There was a total of 346 AEFI records (annualised reporting rate of 31.0 per 100,000 population) for children aged <7 years for vaccines administered in the first 6 months of 2008. This was a 32% increase on the 235 records (19.7 per 100,000 population) for the corresponding 6-month period in 2007 and the highest since 2003 when there were 485 AEFI records.

Forty-one per cent (n=143) of the 346 AEFI records for the 2008 reporting period were for children aged <1 year; 12% (n=40) for those aged 1 to <2 years; and 47% (n=163) were for the 2 to <7 year age group. Although there was an overall increase in the total number of AEFI records for the first 6 months of 2008, the distribution across age groups was similar to that seen in recent years.[11,13] The male to female ratio was 1.2:1, the same as the previous year.[11]

Twelve per cent (n=42) of the 346 AEFI records were defined as 'serious', slightly more than reported for the same period in 2007 (10%). Of the 42 serious AEFI records, one reported that the child had recovered with sequelae and 41 children were admitted to hospital. Serious and other significant AEFIs reported included anaphylaxis (n=1), seizure (n=14) and hypotonic-hyporesponsive episode (HHE; n=19)

Of the 346 AEFI records, 24 listed one or more vaccines where reporting rates could not be estimated from ACIR data due to some incomplete recording of doses on the ACIR. These were influenza (n=14), 23-valent pneumococcal polysaccharide (n=7), hepatitis A (n=4), combined hepatitis A-typhoid (n=1) and bacille Clamette-Guérin (n=1) vaccines.

AEFI reporting rates per 100,000 doses were calculated for 10 vaccines on the current NIP schedule for children aged 2 months or older (Table). These vaccines were recorded as suspected of involvement in the reported adverse event for 322 (93%) of the 346 records analysed. This is an overall AEFI reporting rate of 16.1 per 100,000 doses recorded on the ACIR with 2.0 'serious' AEFI records per 100,000 doses. AEFI reporting rates were higher than for the same period in 2007 and 2006 for most age groups,

Table. Reporting rates of adverse events following immunisation (AEFI) per 100,000 vaccine doses,* children aged less than 7 years, TGA database, January to June 2008

	AEFI records‡ (n)	Vaccine doses* (n)	Reporting rate per 100,000 doses§		
			Jan–June 2008	Jan–June 2007	Jan–June 2006
Vaccine†					
DTPa-containing vaccines	237	539,656	43.9	28.5	36.0
DTPa-IPV	165	212,159	77.8	39.1	46.2
Pentavalent (DTPa-IPV-HepB)	1	8,642	11.6	41.9	41.0
Hexavalent (DTPa-IPV-HepB-Hib)	71	318,855	22.3	8.8	16.8
Haemophilus influenzae type b	8	61,311	13.0	17.9	21.4
Haemophilus influenzae type b-hepatitis B	48	123,369	38.9	23.8	25.9
Measles-mumps-rubella	87	269,472	32.3	17.0	23.3
Meningococcal C conjugate	19	144,647	13.1	8.3	17.8
Pneumococcal conjugate	106	411,722	25.7	17.4	17.9
Varicella	20	131,775	15.2	15.3	15.8
Rotavirus	107	316,004	33.9	–	—
Age group					
<1 year	141	1,206,326	11.7	8.1	9.3
1 to <2 years	33	501,707	6.6	4.9	8.5
2 to <7 years	148	289,923	51.0	34.1	41.3
AEFI category†					
Total	322	1,997,956	16.1	11.7	15.1
'Certain' or 'probable' causality rating	104	1,997,956	5.2	4.2	6.2
'Serious' outcome	40	1,997,956	2.0	1.0	1.6

TGA Therapeutic Goods Administration

* Number of vaccine doses recorded on the Australian Childhood Immunisation Register (ACIR) and administered between 1 January and 30 June 2008.

† Records where at least one of the 10 vaccines shown in the table was suspected of involvement in the reported adverse event. AEFI category includes all records (i.e. total), those assigned 'certain' or 'probable' causality ratings, and those with outcomes defined as 'serious'. Causality ratings were assigned using the criteria described previously.[10] A 'serious' outcome is defined as recovery with sequelae, hospitalisation, life-threatening event or death.

‡ Number of AEFI records in which the vaccine was coded as 'suspected' of involvement in the reported adverse event and the vaccination was administered between 1 January and 30 June 2008. More than 1 vaccine may be coded as 'suspected' if several were administered at the same time.

§ The estimated AEFI reporting rate per 100,000 vaccine doses recorded on the ACIR.

reaction categories and vaccines (Table), while the rates of AEFI with certain or probably causality ratings remained stable.

The largest changes were for children aged 2 to <7 years and <1 year, and for DTPa-IPV, hexavalent (DTPa-IPV-HepB-Hib), Hib-HepB and measles-mumps-rubella (MMR) vaccines. Observed changes in AEFI reporting rates for the first 3 vaccines were at least partly related to a large increase in the use of hexavalent vaccine and reduction in the use of DTPa-IPV and Hib-HepB vaccines in children aged <1 year in Queensland, South Australia and Victoria in early 2008 (see Introduction). The increase in AEFI reporting rates for children aged <1 year is also likely to relate to the implementation of the rotavirus immunisation program in July 2007. The vaccine is co-administered with 7-valent pneumococcal conjugate vaccine (7vPCV) and combination vaccines containing DTPa, IPV, Hib and HepB antigens.

One of the more significant AEFI reported for children aged <1 year is HHE. For the first 6 months of 2008, the reporting rate of HHE following DTPa-IPV containing vaccines plus co-administered vaccines was 9.9 per 100,000 doses of quadrivalent DTPa-IPV vaccine and 2.9 per 100,000 doses of hexavalent DTPa-IPV-HepB-Hib vaccine. While the reporting rate of HHE following DTPa-IPV vaccine was similar to that seen in 2007 (7.6 per 100,000 doses)[12] the rate following DTPa-IPV-HepB-Hib increased substantially in 2008 from 0.6 per 100,000 doses in 2007. The increase in reporting of HHE following DTPa-IPV-HepB-Hib and co-administered vaccines mainly occurred in Victoria, with little change for other states and territories (data not shown).

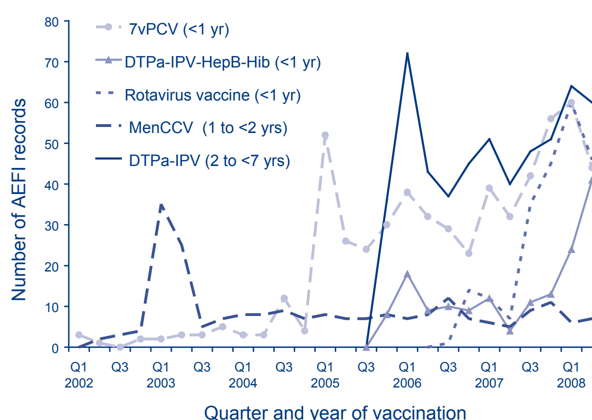
The very high reporting rate for DTPa-IPV vaccine of 77.8 per 100,000 doses (Table) includes reports for children aged <1 year (46.8 per 100,000) and children aged 2 to <7 years (98.6 per 100,000 doses). The majority of AEFI reports for the older age group listed injection site reaction (ISR; reporting rate of 92.7 per 100,000 doses). This is the highest reporting rate for ISR following DTPa-containing vaccines since 2002. The increase in the AEFI reporting rate for MMR vaccine in 2008 may relate to increased reporting of ISR following DTPa-IPV vaccine as the vaccines are administered at the same time in the 2 to <7 years age group and may have been administered in the same limb, or the actual injection sites not reported to the TGA.

Discussion

There was an increase in AEFI notified to the TGA for vaccines administered to children aged <7 years in the first 6 months of 2008 compared with the corresponding period in 2007, with the highest number of notifications since 2003. The majority of AEFI notifications described mild, transient and expected AEFI.

The increase in 2008 compared with the 2007 reporting period is likely to be due to several factors. There were fewer AEFI reports in 2007 compared with recent years, possibly related to there being no new vaccines added to the NIP schedule in the first 6 months of 2007 compared with 2005 and 2006.[11–13] Immunisation providers are more likely to report milder less serious AEFI for vaccines they are not familiar with. In Australia, it is evident that initial high levels of AEFI reporting occur each time a new vaccine is introduced into the NIP schedule, followed by a reduction and stabilisation of reporting over time (Figure). Corresponding peaks are also seen in reporting of co-administered vaccines (e.g. 7vPCV in Figure). The increase in AEFI reporting in 2008 appears to relate to an expected increase following the change to hexavalent vaccines in several jurisdictions in early 2008 as well as the introduction of the national infant rotavirus immunisation program in July 2007 (Figure). Major change in AEFI surveillance practices in Victoria where enhanced AEFI surveillance was implemented during 2007[2] has also

Figure. Reports of adverse events following immunisation, Therapeutic Goods Administration database, 1 January 2002 to 30 June 2008, for vaccines recently introduced into the funded National Immunisation Program*



* Meningococcal C conjugate vaccine (MenCCV) was introduced into the National Immunisation Program schedule on 1 January 2003; 7-valent pneumococcal conjugate vaccine (7vPCV) on 1 January 2005; DTPa-IPV and DTPa-IPV-HepB-Hib vaccines in November 2005; and rotavirus (RotaTeq® and Rotarix®) vaccines 1 July 2007. In early 2008, Queensland, South Australia and Victoria changed from DTPa-IPV to DTPa-IPV-HepB-Hib for children at 2, 4 and 6 months of age.

contributed to the increase in AEFI reporting for children aged <7 years for the first 6 months of 2008 compared with the same period in 2007.

Of particular interest are trends in reporting of ISR following acellular pertussis-containing vaccines among children aged 2 to <7 years and HHE among children aged <1 year. As noted previously,[12] the reporting rate of HHE following receipt of hexavalent DTPa-IPV-Hib-HepB has been lower than that following receipt of the antigens as 2 separate vaccines (i.e. DTPa-IPV and Hib-HepB). This may be related to a real difference in the occurrence of HHE following these 2 vaccines, or to surveillance factors. Interestingly, the significant increase in reporting of HHE following receipt of hexavalent vaccine in 2008, compared with 2007 (2.9 versus 0.6 per 100,000), suggests that surveillance is likely to play a major role as the increase mainly occurred for Victoria, which implemented a new AEFI surveillance system during 2007.[2,3] No increase was observed for Queensland or South Australia, which also changed to the hexavalent vaccine in early 2008.[7–9]

It is also unclear whether the observed rise in reporting of ISR following DTPa-IPV among children aged 2 to <7 years is related to changes in surveillance methods or a real increase in ISR, including extensive limb swelling. This AEFI is known to occur among children receiving a 4th and 5th dose of acellular pertussis-containing vaccine.[10,12,14,15] The reporting rate of ISR in this age group appeared to decline in recent years, as was expected following the removal from the NIP schedule in September 2003 of the dose due at 18 months of age. Children entering school in 2008 would have received their fourth dose of an acellular pertussis-containing vaccine at 4–5 years of age, whereas children in earlier birth cohorts would have received their 5th dose prior to school entry. Reporting of ISR following acellular pertussis-containing vaccines will continue to be monitored through the AEFI surveillance system.

Conclusion

This report further demonstrates that changes to the NIP schedule and to surveillance practices are reflected in the national passive AEFI surveillance data.[6,8,10] The majority of AEFI reported to the TGA were mild transient events and indicate the high safety level of the vaccines included in the NIP schedule. Close monitoring of passive AEFI surveillance data for vaccines administered to children continues through the TGA, in consultation with the Adverse Drug Reactions Advisory Committee and state and territory health departments.

Acknowledgements

We thank Mike Gold (Adverse Drug Reactions Advisory Committee), and Kristine Macartney and Nick Wood (NCIRS) for their contribution to this report. The National Centre for Immunisation Research and Surveillance is supported by the Department of Health and Ageing, the New South Wales Health Department and the Children's Hospital at Westmead.

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Short reports

Outbreaks of dengue in north Queensland, 1990–2008

Jeffrey N Hanna, Scott A Ritchie

The (recognised) outbreaks of dengue that have occurred in north Queensland from 1990 to 2008 (inclusive) are summarised in the following Table.

Year(s)	Location	Reported cases	Suburbs / islands predominantly affected	Duration of outbreak	Serotype
1990–91	Cairns, Townsville, Torres Strait	27		8 weeks	Dengue 1
1992–93[1,2]	Townsville, Charters Towers	900		64 weeks	Dengue 2
1995[3]	Cairns	4	Manunda	14 weeks	Dengue 2
1996–97[4]	Torres Strait, Cairns	208	Mer, Erub, Masig, Iama, Thursday Island, Westcourt	28 weeks	Dengue 2
1997–98	Cairns	12	Whitfield	11 weeks	Dengue 2
1997–99[5]	Cairns, Mossman, Port Douglas	498	Cairns North, Parramatta Park, Stratford, Machans Beach, Holloways Beach, Bungalow	70 weeks	Dengue 3
2000[6]	Cairns	49	Machans Beach	6 weeks	Dengue 2
2001[7]	Townsville	9	Mysterton, North Ward	3 weeks	Dengue 2
2002[8]	Kuranda	21		10 weeks	Dengue 2
2002[8]	Townsville	2	Railway Estate	2 weeks	Dengue 1
2002[8]	Cairns	2	Smithfield	3 weeks	Dengue 4
2003	Cairns	3	Parramatta Park	2 weeks	Dengue 1
2003[9]	Mareeba	1		1 week	Dengue 1
2003[10]	Cairns	5	Manunda	3 weeks	Dengue 2
2003–04[10]	Cairns, Townsville, (Torres Strait)	536	Parramatta Park, Bungalow, Stratford, Aeroglen, Edge Hill, Whitfield, Townsville CBD, Cranbrook, Currajong, North Ward, Thursday Island	69 weeks	Dengue 2
2003–04[10, 11]	Torres Strait, Cairns	356	Iama, Thursday Island, Mer	41 weeks	Dengue 2
2004	Torres Strait	1	Masig	1 week	Dengue 2
2005	Torres Strait	56	Mer, Thursday Island, Erub	7 weeks	Dengue 4
2005	Townsville	18	Currajong	22 weeks	Dengue 4
2005–06	Townsville	8	Cranbrook	6 weeks	Dengue 3
2006	Cairns	29	Gordonvale, Manunda	18 weeks	Dengue 2
2007	Townsville	46	South Townsville, West End	12 weeks	Dengue 3
2008	Port Douglas/ Mossman	22		10 weeks	Dengue 3
2008	Cairns	1	Manoora?	1 week	Dengue 2
2008	Cairns, Townsville	99 (with onset in 2008)	Cairns North, Clifton Beach, Whitfield, Parramatta Park, Edge Hill, Belgian Gardens (with more suburbs in 2009)	8 weeks in 2008 – ongoing into 2009	Dengue 3
2008	Townsville	5 (with onset in 2008)	North Ward, City	2 weeks in 2008 – ongoing into 2009	Dengue 1

It is obvious that outbreaks of dengue have become more frequent in north Queensland in recent years. This is a reflection upon the deteriorating dengue situation in the Asia–Pacific region and elsewhere,[12] as every outbreak in north Queensland is initiated by a viraemic traveller to the region.

However, increased surveillance for dengue in north Queensland and the use of very powerful molecular techniques that are able to discern significant differences between isolates of dengue viruses belonging to the same serotype[10] have also contributed to this increase, particularly in more recent years. It is quite possible that some small outbreaks, or outbreaks caused by (unrecognised) differing genotypes of the same serotypes of dengue viruses, were not detected, and therefore the Table may under-enumerate the actual number of outbreaks that occurred in north Queensland over the 18 years.

A detailed review of the recognised outbreaks that occurred in 2005–2008 (inclusive) is in preparation.

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An apparent recent decline in importations of dengue from Papua New Guinea into north Queensland

Jeffrey N Hanna, Scott A Ritchie

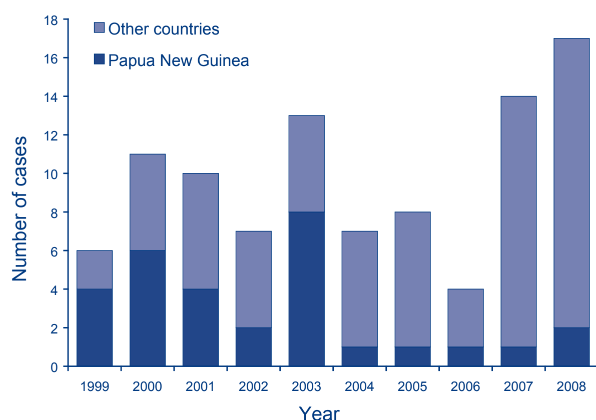
Although there is a paucity of information about dengue in Papua New Guinea, information gathered through surveillance in north Queensland in the early 1990s clearly documented that multiple serotypes of dengue viruses were circulating in that country.[1,2] Indeed, several importations from Papua New Guinea have initiated large outbreaks of dengue in north Queensland.[3,4]

Although the dengue viruses are not endemic in north Queensland, the principal vector, *Aedes aegypti*, is endemic. This means that the region is prone to outbreaks of dengue: each one being initiated by a traveller from abroad who is viraemic (i.e. infectious to *Ae. aegypti* mosquitoes) whilst in north Queensland. For this reason, surveillance for viraemic importations of dengue is a priority disease control activity in the region.[2]

Over the last decade, 1999–2008, 97 viraemic importations of dengue into north Queensland were notified, with a mean of 9.7 (range 4–17) cases per year (Figure). Of note, importations from Papua New Guinea predominated in the years 1999–2003, being responsible for 24 (51%) of the 47 importations over those 5 years. However, in the most recent 5 years, 2004–2008, importations from Papua New Guinea have been much less frequent, being responsible for only 6 (12%) of the 50 notifications (Figure).

This apparent recent decline in dengue importations from Papua New Guinea into north Queensland could be quite coincidental. For example, perhaps in the recent years there could have been fewer susceptible travellers or expatriates in Papua New Guinea, or there may have been milder disease, not necessitating travel to Australia for diagnosis and management. Perhaps there could have been a trough in the periodicity of hyperendemic dengue in these years.

Figure. Viraemic importations of dengue into north Queensland, 1999–2008



Nevertheless, it is possible that there is another explanation. An exotic mosquito, *Aedes albopictus*, was recognised as being established in several Torres Strait islands for the first time in 2005.[5] *Ae. albopictus* is an aggressive periurban Asian mosquito that has displaced local populations of *Ae. aegypti* in many locations.[6] However, although *Ae. albopictus* is able to transmit dengue viruses, it is a considerably less efficient vector of dengue than *Ae. aegypti*.[7,8]

The apparent decline in dengue importations from Papua New Guinea began in 2004, and *Ae. albopictus* was first recognised in the Torres Strait the following year. Container surveys in coastal villages in the Western Province of Papua New Guinea indicate that *Aedes albopictus* is prevalent in these locations (Ritchie SA, van den Hurk AF, unpublished data).

Therefore a plausible hypothesis is that *Ae. albopictus* has displaced *Ae. aegypti* in urban centres in Papua New Guinea frequented by travellers and expatriates (e.g. Port Moresby, Lae) from about 2004. This would result in these urban centres being populated with a considerably less efficient vector of dengue, thereby lowering the risk of travellers and expatriates in these centres acquiring dengue (and subsequently importing it into north Queensland).

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Outbreak of pertussis, 1 January to 31 March 2009

Surveillance Branch, Office of Health Protection

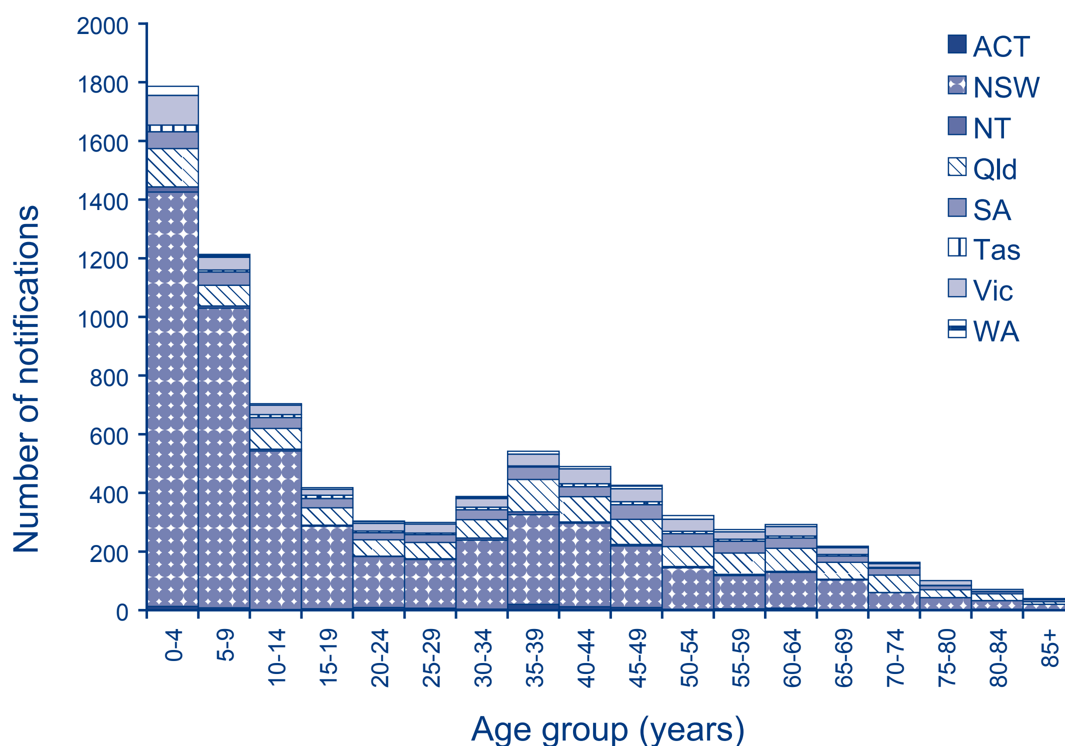
Pertussis (whooping cough) is an acute bacterial infection of the respiratory tract cause by *Bordetella pertussis*. The initial catarrhal stage has an insidious onset with an irritating cough that gradually becomes paroxysmal, usually within 1–2 weeks and lasting for 1–2 months or longer. Paroxysms can be followed by a characteristic high-pitched inspiratory whoop.[1] Transmission is by direct contact with droplets from respiratory mucous membranes of infected persons. In highly vaccinated populations, adults and adolescents are recognised as significant reservoir of infection due to waning immunity,[2,3] with parents having been identified as the source of infection in more than 50% of cases in infant pertussis cases.[4,5,6] As maternal antibodies do not provide reliable protection against pertussis, the maximal risk of infection and severe morbidity is for those infants too young to have received at least 2 vaccine doses.[7]

Between 1 January and 31 March 2008, 8,058 cases of pertussis were reported to the National Notifiable Diseases Surveillance System (NNDSS). Cases were reported from all states and territories, with the majority reported by New South Wales (n=5,265) and Queensland (n=1,187). The number of notifications for this quarter was 5.2 times more than in the same period in 2008 (n=1,554) and 4.8 times the 5 year-to-date mean for this quarter (1,677). The annualised notification rate for this quarter of 153 cases per 100,000 population was significantly higher than for the same period in 2008 (30 cases per 100,000 population, with a rate ratio of 5.1.

The highest number of notifications was observed in the 0–4 year age group (n=1,786; 22%), followed by the 5–9 year age group (n=1,213; n=15%) (Figure). Infants aged less than 6 months continue to represent just over 3% of total notifications.

Fifty-six per cent of cases were female (n=4,548) and 43% were male (n=3,482). There were 24 cases for which no gender was recorded. The average age in this quarter was 26 years with ages ranging from 3 weeks of age to 88 years at the time of diagnosis.

Figure: Notifications of pertussis, Australia, 1 January to 31 March 2009, by state or territory and age group



There were 3 infant deaths recorded in NNDSS for this quarter. The youngest, from New South Wales, was 4 weeks of age at onset of illness, was admitted to hospital and died in intensive care.[8] The infant was too young to be vaccinated. The other 2 infants that died in Australia were eligible for vaccination and each had received 1 dose of pertussis-containing vaccine.

In December 2008 and January 2009 NSW Health issued public health alerts to healthcare providers and the public to raise awareness about the increase in pertussis. On 10 March 2009 NSW Health announced that in order to help protect babies, it had arranged for a free vaccination through GPs for all new parents, grandparents and people who care for new babies.

An information mail out about the free vaccine was prepared for all new parents across New South Wales and GPs were contacted to encourage vaccination.

This follows the introduction in the Northern Territory in October 2008 of a funded booster vaccination for new mothers who gave birth in a hospital. More recently, in mid-April 2009, the Australian Capital Territory commenced a funded program for 3 months providing a booster vaccination for all parents and grandparents of children aged less than 12 months of age.

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Outbreak of human metapneumovirus infection in a residential aged care facility

Maggi Osbourn, Kenneth A McPhie, V Mala Ratnamohan, Dominic E Dwyer, David N Durrheim

Abstract

Summer outbreaks of respiratory illness in residential aged care facilities are uncommonly reported in New South Wales. A respiratory illness outbreak in an aged care facility during January 2008 prompted a response to contain the outbreak by implementing infection control measures, including cohorting of symptomatic residents, cohorting nursing care, closure to new admissions and the use of personal protective equipment by staff. In addition, respiratory tract specimens were collected to determine the causative agent. Human metapneumovirus (hMPV) was detected by polymerase chain reaction assay in 3 specimens with no other respiratory pathogens found. This is the 1st reported outbreak of hMPV in an aged care facility in Australia. hPMV should be considered as the possible cause of outbreaks in aged care facilities when influenza and respiratory syncytial virus have been excluded. *Commun Dis Intell* 2009;33:39–41.

Keywords: human metapneumovirus, outbreak

Background

Human metapneumovirus (hMPV) was first described as a respiratory pathogen in children in 2001[1] in The Netherlands, and has subsequently been recognised worldwide in children, including Australia.[2] There is a growing recognition of its capacity to cause illness in adults, particularly the elderly. A Canadian study found that 45.9% of hMPV isolates from patients hospitalised with respiratory illnesses were in people aged over 65 years.[3]

Elderly people living in residential aged care facilities (RACFs) are vulnerable to outbreaks of respiratory pathogens, particularly influenza, and hPMV has been implicated in winter outbreaks in RACFs in Japan with hMPV detected by nucleic acid testing in eight of 23 patients[4] and Canada (6 of 13 patients).[5] A summer outbreak has been previously reported in the United States of America (USA).[6]

In the summer months, reports of respiratory virus outbreaks from RACFs and other 'closed' environments in Australia are uncommon,[7] although it should be noted that respiratory outbreaks are only notifiable to New South Wales public health units if influenza virus infection is confirmed.[8] National guidelines for the management of influenza outbreaks in RACFs have been available since 2005 and are currently being revised.[9] We report on a summer outbreak of hMPV infection in a RACF in New South Wales.

Methods

In early January 2008, Hunter New England Population Health (HNEPH) received a report from a general practitioner of mild respiratory illness affecting 8 residents in a RACF in the Lake Macquarie area of New South Wales. The initial cases had not responded to cephalexin, and pathology specimens had not been collected.

HNEPH staff undertook an investigation, with respiratory tract swabs collected from the most recently symptomatic residents to ascertain the causative organism, while appropriate infection control measures, including isolation/cohorting of sick residents, cohorting of the care of sick residents and closure of the affected wing of the RACF to admissions and visitors, were instituted. A line-listing of affected residents and their symptoms was initiated.

Within 2 days hMPV was detected by direct immunofluorescence on a throat swab from a resident (influenza A and B, adenovirus, respiratory syncytial virus (RSV) and parainfluenza virus types1–3 were negative). Throat swabs were submitted from an additional 9 residents who had developed respiratory symptoms during the outbreak, with a further 2 cases of hMPV identified using an in-house polymerase chain reaction (PCR) assay. No isolates were recovered for further typing. No other respiratory virus pathogens were detected by PCR in other samples.

Results

From 29 December 2007, 16 of 73 residents in the affected RACF experienced respiratory symptoms meeting the case definition (cough, with one or more of: elevated temperature ≥ 37.5°C, sore throat, lethargy, myalgia or shortness of breath) (Table).

Twelve of the 16 affected residents, including the 3 positive hMPV cases, experienced three or more symptoms. These include pyrexia and moist cough (12), sore throat (6), myalgia (5) and lethargy (4). This clinical picture is not dissimilar to influenza in the elderly.

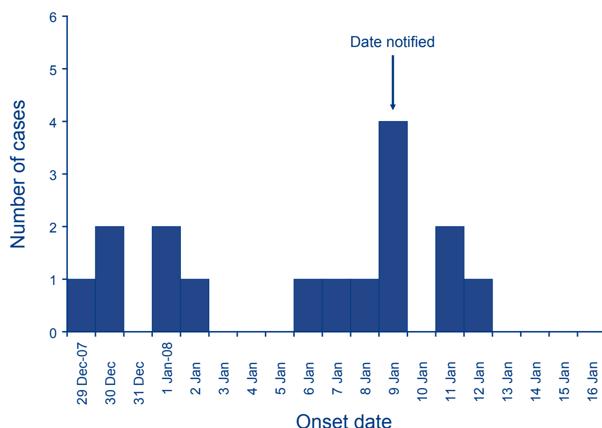
A survey of the 80 staff members to determine respiratory symptoms in the 7 days prior to onset of illness in the 1st resident found no symptomatic staff members.

Table. Symptoms experienced by cases with human metapneumovirus infection, New South Wales, 29 December 2007 to January 2008

Symptom	Number of residents with symptoms n=16
Cough	16
Elevated temperature	13
Sore throat	6
Myalgia	5
Lethargy	4
Shortness of breath	1

Affected residents were aged from 64 to 102 years, with a median of 89 years. All had underlying medical conditions. Seven of the affected residents had received influenza vaccination in 2007. Fourteen symptomatic residents did not use the communal dining area or participate in group activities, however two of the symptomatic residents with dementia were mobile, and their wandering within the RACF may have played a role in transmission. The last case was recorded with onset on 12 January 2008 (Figure).

Figure. Cases of human metapneumovirus infection, New South Wales, 29 December 2007 to 16 January 2008



For 11 residents where data were available, the duration of illness ranged from 3 to 18 days with a median of 6 days. Two residents died, 8 and 22 days after onset of respiratory symptoms, respectively, with the principal cause of death listed on their death certificates as bronchopneumonia. Although neither fatality had laboratory confirmed hMPV infection, or any other respiratory pathogen detected, the timing and clinical presentation suggests that the deaths were precipitated by hMPV infection.

Discussion

This report describes the first reported outbreak of hMPV infection in a RACF in Australia.

When a respiratory virus outbreak occurs in a RACF it is important to immediately introduce stringent infection control procedures to minimise transmission. Influenza virus infection specifically needs exclusion as antiviral treatment and prophylaxis may be appropriate.

Every year in the Hunter New England area there are a number of respiratory outbreaks in RACFs where influenza or other respiratory pathogens cannot be isolated from laboratory specimens (4/15 in 2007). Human metapneumovirus may be an important contributor to these outbreaks. It is diagnosed by antigen detection (e.g. immunofluorescence) or detection of hMPV RNA by nucleic acid testing using PCR on respiratory tract samples. Serological testing for hMPV is not widely available.

Outbreaks in RACFs are much less commonly reported during the summer months in New South Wales. This summer outbreak of hPMV was characterised by relatively mild disease in most residents, although 2 non-laboratory confirmed cases succumbed to bronchopneumonia, with a relatively low attack rate of 16.4%. The clinical picture and attack rate is not dissimilar to the reported summer outbreak in the USA, although there were no deaths during the latter.

The attack rate is similar to outbreaks of RSV, reported as between 3%–20%,[10] whereas attack rates during influenza outbreaks have been reported as high as 27%.[11] Introduction of infection control procedures occurred relatively late in the outbreak and thus it is difficult to judge their effectiveness in controlling hPMV outbreaks in RACF settings.

When influenza and RSV have been excluded as the causative organism for a RACF respiratory outbreak, infection with hMPV should be considered.

Acknowledgements

Management and staff of the residential care facility, and attending medical practitioners are warmly thanked for their collaboration in this outbreak investigation.

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Enhanced Q fever risk exposure surveillance may permit better informed vaccination policy

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Abstract

The association between farming risks and Q fever is not well documented in Australia. In a review of New South Wales notifications, data were analysed using 3-year study periods from 1993 to 2007 to investigate possible trends and explore reported risk exposures. A retrospective case series was also conducted using acute Q fever cases notified during 2007 from a rural area of New South Wales. Occupation was recorded for less than 50% of Q fever notifications in New South Wales during the study period. A significant decline in the proportion of notifications occurred in the occupational group reported as 'Abattoir/Meat' worker and a significant increase occurred in the 'Farmer/Livestock' category. The case series found that in the month prior to illness onset 78% (42/54) reported direct contact with animals. In the month prior to becoming ill with Q fever 71% (31/51) of employed cases had contact with newly introduced livestock in their workplace. As a result of their Q fever illness 93% of cases required time off work or school, with a median of 21 days. At the time of the structured interviews 63% had not fully recovered. The epidemiology of Q fever disease in New South Wales has changed and amongst notified cases the relative importance of non-abattoir contact with livestock, wildlife or feral animals appears to be increasing. The surveillance field 'Occupation' no longer alone adequately describes risk exposure for many of the people notified with Q fever and a new field that better describes risk exposures is required. This may allow more finely tuned vaccination policy. *Commun Dis Intell* 2009;33:42–46.

Keywords: Q fever, surveillance, rural, exposure, risk, occupation

Introduction

Q fever is an acute febrile illness caused by the intracellular gram-negative bacteria *Coxiella burnetii*[1] and is the most common zoonotic disease in Australia.[2] Transmission usually occurs because of direct or indirect contact with infected animals, their tissues or products.[3] There are several clinical syndromes of Q fever including a self-limited febrile illness, pneumonia, endocarditis, hepatitis and osteomyelitis.[4] The case-fatality rate among untreated cases may be as high as 2.4% but is usually less than 1%.[5]

Since the 1930s Q fever has been strongly associated with Australian abattoirs.[6,7] In a review of Q fever notifications in New South Wales, for the period 1991–2000, where data on occupation were recorded, 51% of the cases were recorded as abattoir or meat workers, and agriculture related occupations represented 29% of the cases.[7] Queensland and Victoria have reported abattoir worker as the occupation in 40%–45% of notifications.[8]

The association between farming and Q fever is less well documented. In south-west Queensland the majority of recent notifications have been associated with an occupation of farming.[9] In north-western New South Wales a Q fever cluster was described in a shearing team.[10] During a Q fever vaccination program on the north coast of New South Wales, over 27% of cattle workers had laboratory evidence of pre-existing immunity to Q fever.[11]

There is an effective, safe vaccine against Q fever[12] and vaccination of people at risk of Q fever is the main disease prevention strategy available in Australia.[13] Abattoir- and other meat industry workers were the main focus of the National Q Fever Management Program conducted in Australia from 2001–2004.[14] Since the end of the National Q Fever Management Program, cases of Q fever continue to be reported despite the availability of an effective vaccine. In New South Wales annual notified cases have increased from 143 in 2005 to 175 in 2006 and 215 cases in 2007.[15]

In New South Wales Q fever is a Category 3 scheduled medical condition under the provisions of the *NSW Public Health Act 1991* and is notifiable to public health units. In accordance with NSW Department of Health (NSW Health) policy, Q fever is followed up by public health units for the purpose of monitoring the epidemiology to inform the development of better prevention strategies.[16]

The aim of this investigation was to describe the changing epidemiology of Q fever in New South Wales and to survey notified individuals in the Hunter New England region, a rural area of New South Wales, to better understand current risk exposures.

Methods

New South Wales Q fever notifications recorded in the NSW Notifiable Diseases Database (NDD)

were sourced from NSW Health's HOIST (Health Outcomes Information and Statistical Toolkit). Data were analysed for New South Wales and the Hunter New England region.

Initially, New South Wales notifications were analysed using five 3-year study periods from 1993–2007 to investigate possible trends and explore reported exposures. The occupation recorded for each notification of Q fever was grouped for analysis into 'Abattoir/Meat' work and a small range of other occupational categories. Data were also described by gender, Indigenous status, Area Health Service of residence and hospitalisation. Analysis was conducted using SPSS® Graduate Pack 15.0 for Windows® (version 15, SPSS Inc, Chicago, Ill, USA). Chi square for trend analysis was conducted on gender and occupation variables over the study periods using Epi Info (version 6, Centers for Disease Control and Prevention, Atlanta, Georgia).

A retrospective case series was also conducted using acute Q fever cases from the 2007 notifications of Q fever from a rural area of New South Wales (Hunter New England), to gain a better understanding of Q fever risk exposures. This group was selected because of recent increased notifications in the area. Routine follow-up of notified cases had already occurred, however additional information on occupation, the nature of potential Q fever risk exposures and morbidity were obtained using a structured telephone survey of those that met the case definition for acute Q fever. Acute Q fever was defined according to the NSW Response Protocol for Public Health Units as: definitive laboratory evidence for acute Q fever; or laboratory suggestive evidence and a compatible clinical history.[16] Analysis for the retrospective case series was conducted using SAS V9.1 and Microsoft Office Excel, 2003. Ethics approval was not required.

Results

For the period 1993–2007 there were 3,447 notifications of Q fever in New South Wales residents with the highest number of notifications occurring in the period 1993–1995 (Figure 1). Most Q fever notifications (90%; n=3123) occurred in the working age group, 15–64 years, and less than 3% (n=81) were in children aged under 15 years (Figure 2).

Over the whole study period more than 80% (2,764 of 3,446) of notifications were males but an increasing proportion of females were notified with Q fever; 12.8% in 1993–1995 to 28.4% in 2005–2007 ($P<0.0001$). Across New South Wales, the large majority (94.9%) of notifications occurred in residents of rural Area Health Services. Only 43% (1,494 of 3,446) of notifications over the study period had valid data for the hospitalisation variable. Among notifications with valid data, 24% (358/1494) were reported to have been hospitalised.

Figure 1. Notifications of Q fever, New South Wales, 1993 to 2007, by 3-year groupings

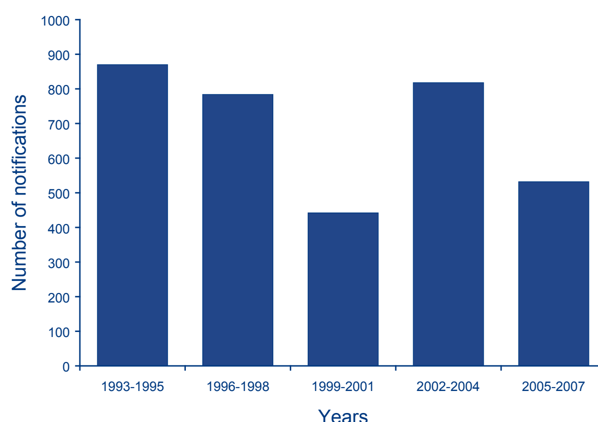
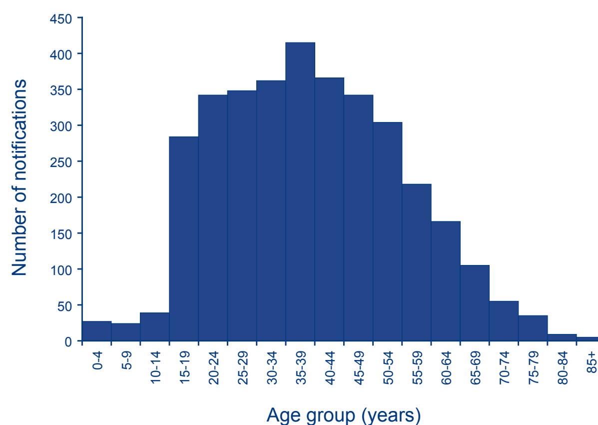


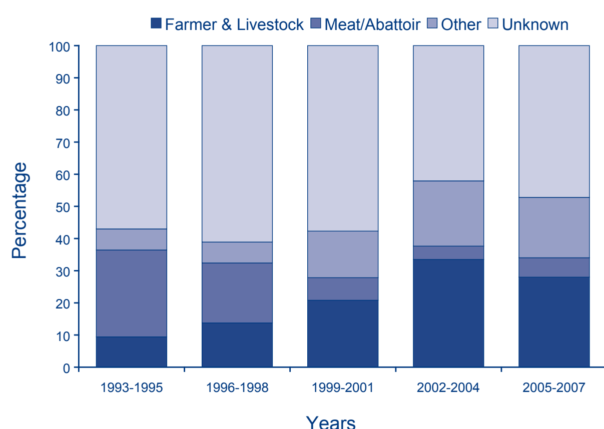
Figure 2. Age distribution of Q fever notifications, New South Wales, 1993 to 2007



Occupation was recorded for less than 50% of Q fever notifications in New South Wales. The highest reported occupation groups were 'Farmer/Livestock' (16.1%) and 'Abattoir/Meat' (13.9%). A significant decline in the proportion of notifications in the occupational group 'Abattoir/Meat' worker ($P<0.0001$) occurred over the study periods (Figure 3). The proportion in the 'Farmer/Livestock' occupational group increased over the study period ($P<0.0001$).

For the period 1 January 2007 to 31 December 2007 there were 75 notifications of Q fever in people resident in the Hunter New England area. On serological and clinical review, 61 were found to have acute Q fever and 12 (20%) of these were female. Structured interviews were completed with 54 of the 61 notifications (89%).

Figure 3. Notifications of Q fever, New South Wales, 1993 to 2007, by occupation group

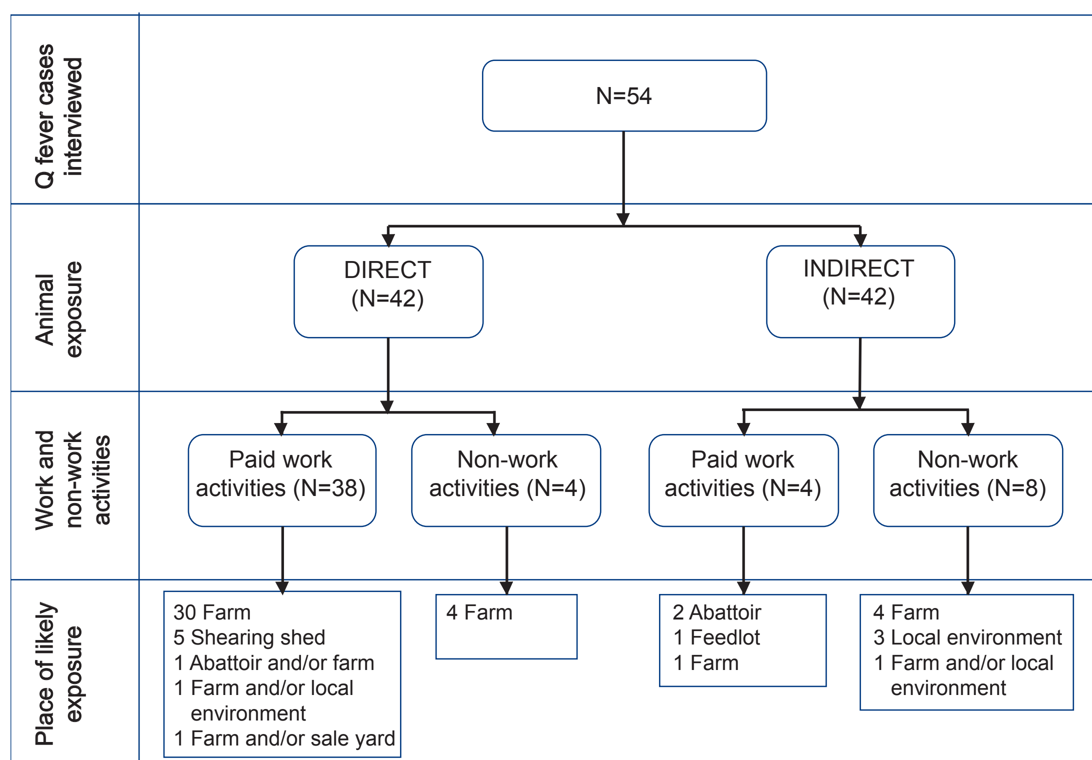


Of those surveyed 42 (78%) described themselves as living on a farm, or in a semi-rural area or village. Most worked (94%; n=51) in the month prior to illness onset with 18 occupations reported. Abattoir work was uncommon (6%; 3), while the occupations of farmer, farm manager and farm worker predominated (70%; 36). In the month prior to becoming ill with Q fever, 31 (61%) of those working had contact with newly introduced livestock as part of their work.

In the month prior to illness onset, 42 (78%) of the cases surveyed reported direct contact with animals, their tissues or products with 38 (90%) of these occurring during work activities. The remaining 12 (22%) reported indirect contact with dusts that were contaminated by animals tissues, products or excreta, with 4 (33%) occurring during work activities. Direct exposure to cattle was reported by 81% of respondents, exposure to sheep reported by 38% and kangaroos or wallabies exposure reported by 26%. The most common place where exposure to animals occurred was on a farm (Figure 4) although many respondents reported exposure to multiple animal species in different settings. Of those who worked with animals 31% of activities described involved contact with animal blood or body fluids, 32% involved assisting animals with parturition and 46% participated in activities that involved general handling of animals.

As a result of their Q fever illness 50/54 (93%) people had time off work or school, with a median of 21 days off work or school and a range of 2–296 days. Twenty-nine respondents were hospitalised for a median of 6 days and a range of 1–42 days. At the time of the structured interviews (conducted 28 to 93 weeks after illness onset) 34 (63%) people reported they had not fully recovered. Table 1 describes the most frequent ongoing issues reported by respondents. Of those reporting full recovery, the median time to full recovery was 12 weeks with a range of 1–35 weeks.

Figure 4. Description of likely Q fever exposures among interviewed Hunter New England residents notified with acute Q fever in 2007



None of the respondents reported being vaccinated against Q fever. Thirty-eight (70%) people reported that they knew about the vaccine before their illness and the most common reasons provided for not being immunised were: believing that they were not at risk or problems with access (Table 2).

Table 1. Ongoing health conditions in people notified with acute Q fever in the Hunter New England area, 2007

Issue	n*	%*
Fatigue	32	94
Athralgia or myalgia	20	59
Fevers and sweats	9	26
Endocarditis	1	3
Total with ongoing issues	34	100

* Number and per cent is greater than the total as certain respondents reported more than 1 ongoing issue.

Table 2. Reasons provided for not being vaccinated against Q fever in people notified with Q fever from Hunter New England in 2007

Issue	n	%
Thought not at risk	14	37
Access problems	9	24
Not got around to it	5	13
Told not at risk	2	5
Child	2	5
Not provided by employer	2	5
Other	4	11
Total aware of Q fever vaccine	38	100

Discussion

This study of people notified with Q fever confirms that it is a serious illness that commonly produces considerable morbidity, emphasising the importance of prevention. The high proportion of people with ongoing health issues many weeks after illness onset has not previously been reported in Australia. The comparison of hospitalisation rates from routinely collected surveillance data and data gathered during the retrospective survey highlights the underestimation in routinely collected notification data. This would be important to consider when conducting an economic evaluation of Q fever vaccination strategies.

Cases of Q fever continue to be reported in New South Wales despite the availability of an effective vaccine. The National Q Fever Management Program which operated from 2001–2004 provided free vaccine to some groups at risk. The large reduction in the number of notifications amongst people reporting work in an abattoir is likely to reflect a good outcome from this program, but many people in rural New South Wales who are potentially exposed to Q fever remain susceptible to this disease.

The epidemiology of Q fever disease in New South Wales has changed and amongst notified cases the relative importance of non-abattoir contact with livestock, wildlife or feral animals appears to be increasing. A fifth of notified rural residents described participating in activities that exposed them directly or indirectly to animals, their tissues and products in a non-work setting. The surveillance field ‘Occupation’ no longer alone adequately describes risk exposure for many of the people notified with Q fever and a new field that describes risk exposures is required. This would allow a more finely tuned focus of future vaccination policy.

Considering awareness of Q fever vaccination was reasonable at 70% (38/54), the barriers to immunisation described in this case series need to be confirmed in a larger sample of people and actions taken to address the underlying reasons for non vaccine uptake. Given the marked step in the age distribution of notified Q fever cases it would be valuable to confirm whether there is an opportunity of targeting Q fever vaccination to rural children, and confirm vaccine safety and efficacy in this group.

Acknowledgements

April Worley, Kylie Taylor and Bradley Moylan who assisted with data collection, recording and data cleaning.

Dr Stephen Graves, Australian Rickettsial Reference Laboratory, for providing expert microbiological advice.

The Australian Centre for Agricultural Health and Safety for their assistance in developing the survey tool.

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Hepatitis A outbreak epidemiologically linked to a food handler in Melbourne, Victoria

Stacey L Rowe, Kirsten Tanner, Joy E Gregory

Abstract

Hepatitis A is caused by the hepatitis A virus (HAV). Transmission occurs by the faecal-oral route, either by direct contact with an HAV-infected person or by ingestion of HAV-contaminated food or water. Hepatitis A outbreaks are uncommon in Australia. In 2008, Victoria experienced an outbreak of hepatitis A due to an infected food handler. *Commun Dis Intell* 2009;33:47–49.

Keywords: disease outbreak, foodborne illness, hepatitis A

Introduction

Hepatitis A is caused by the hepatitis A virus (HAV), of which humans, chimpanzees and other primates are the only reservoir. Transmission occurs by the faecal-oral route, through the ingestion of contaminated food or water,[1] or through direct hand-to-mouth contact with the faeces of an infectious case. After ingesting the virus, it can take between 15 and 50 days to become symptomatic. Symptoms may include jaundice and/or dark urine, fever, headache or vomiting. A person with hepatitis A is infectious during the 2 weeks prior to and 1 week after the onset of symptoms.

HAV contamination of a food product can occur at any point during cultivation, harvesting, processing, distribution, or preparation.[2] The source of most reported foodborne hepatitis A outbreaks has been HAV-infected food handlers present at the point of sale or who have prepared food for social events.[2]

In Victoria, medical practitioners and laboratories are required to notify the Department of Human Services of cases of hepatitis A under *Health (Infectious Diseases) Regulations 2001*.[3] Public health action is centred around the identification of a source, control of the environment and to minimise the risk of secondary cases through the provision of immunoglobulin and/or vaccine. Approximately 60 cases of hepatitis A are notified each year in Victoria, most of which are acquired overseas. Few outbreaks of hepatitis A occur in Victoria: only 8 hepatitis A outbreaks have been reported since 2002, most of these occurring among children in child care centres or primary schools. To our knowledge, a foodborne outbreak of hepatitis A has not previously been identified in Victoria, although in 1997 eighty Victorian cases linked to the consumption of contaminated oysters from Wallis Lakes in New South Wales were identified. Six sporadic cases of hepatitis A among food handlers have been notified since 2003.

Follow up of apparently sporadic cases of hepatitis A can lead to the identification of outbreaks. This report describes an outbreak of hepatitis A epidemiologically linked to a food handler in a Melbourne café, Victoria, Australia.

Background

On 24 April 2008, a medical practitioner from a Melbourne hospital notified the Victorian Department of Human Services of a case of hepatitis A, whose onset of jaundice was 24 April 2008. A routine interview with the case and the case's wife ascertained that he was also a co-owner of a café situated within the central business district of Melbourne, and occasionally worked as a food handler at the premises. The case also worked as a cleaner for serviced apartments. A source of the infection could not be determined, although occupational exposure to faecal matter was concluded as the likely source. All household contacts of the case were given normal human immunoglobulin within 2 weeks of their last contact with the case in accordance with *The Australian Immunisation Handbook*.[4] The department was advised by the case's wife (a co-owner and manager of the café) that the case had not worked at the café since 2 April 2008.

Three subsequent hepatitis A notifications were received by the department between 20 May 2008 and 22 May 2008. Sources of illness for 2 cases were not ascertained, and travel to a country where hepatitis A is endemic was believed to be the cause of illness for the third case.

On 26 and 28 May 2008, 2 additional hepatitis A notifications were received by the department. At interview, no risk factors for hepatitis A were identified, however, both cases nominated eating regularly at the café co-owned by the case notified in April (index case). On 28 May 2008, it was hypothesised that foodborne transmission of HAV may have occurred at the implicated food premises.

Methods

Epidemiological investigation

On 28 May 2008, a case series investigation was commenced, incorporating prospective and retrospective follow up of hepatitis A cases notified between 24 April and 19 June 2008, and in whom no other hepatitis A risk factors were identified. This time series took into account the minimum and maximum incubation periods of hepatitis A following potential exposure of HAV from the index case. Structured interviews were carried out with all identified cases to ascertain onset, symptoms, and possible source of illness. All cases were questioned about their consumption of ready-to-eat foods such as sandwiches and salads from 'inner city cafés'.

On 30 May 2008, the department issued a media release to the general public, advising people who had eaten food from the café prior to 25 April 2008 to be aware of symptoms of hepatitis A, and to seek urgent medical attention if symptoms were present. Cases notified after the media release were questioned with the standard hepatitis A questionnaire, and were additionally asked if they had eaten food at the named café.

Onset dates were plotted on a time-series figure, and incubation and infectious periods were calculated to epidemiologically link the cases that nominated eating at the implicated premises prior to 25 April 2008.

Environmental investigation

An inspection of the premises was carried out by an environmental health officer at the local council in which the premises was located.

Results

Epidemiological investigation

A total of 15 cases were identified in the case series, five of which were notified subsequent to the media release being issued. Ten cases were deemed to have acquired their illness through the consumption of contaminated food from the implicated café. One case was the partner of a case who had eaten at the café. It was hypothesised that he acquired his infection through person-to-person transmission. Four additional cases were identified during the case series investigation but had not eaten at the café, and therefore were not epidemiologically linked to the outbreak and were excluded from further analysis.

Of the 10 cases identified in the outbreak, the median age was 38 years (range 23–65 years). The male to female ratio was 2.3:1. The most common symptoms experienced by cases were fever (100%), jaundice (80%), and nausea (80%). Other symptoms reported were vomiting, headache, diarrhoea, dark urine and abdominal pain. All cases reported eating uncooked ready-to-eat foods such as salads, and sandwiches containing salad ingredients. At least 7 cases ate at the premises regularly (multiple times during their infectious period).

Standard public health responses were offered to each case, which in Victoria includes:

- education on the HAV and its transmission;
- work exclusions as necessary during the case's infectious period; and
- arranging post-exposure prophylaxis to close contacts of an infectious case.

Environmental investigation

Inspections of the premises were conducted on 28 April and 28 May 2008. A precautionary clean up of the premises was conducted on the weekend of 25–26 April 2008, and this was verified by the council officer during the site visit. During the second site visit, the local council officer reviewed the Food Safety Program, undertook a Food Safety Compliance Check, and reviewed hygiene, cleaning and food handling procedures. At the time of the inspections, food handling procedures were found to be good, with adequate hand washing facilities available. The staff illness register was also observed, which indicated that the index case had worked at the food premises from 20 April to 19 May 2008.

No food or environmental samples were collected as no representative samples were available due to the long incubation period of hepatitis A.

Additional public health responses

Having ascertained that the index case had worked at the premises during his infectious period, the department advised the other food handlers at the café to be aware of the symptoms of hepatitis A; and to discontinue work and seek urgent medical attention if symptoms were recognised. On 2 June 2008, the food handlers were requested to have a blood test for hepatitis A. Results showed that all 3 food handlers had immunity to hepatitis A, indicating that they were neither incubating hepatitis A nor at risk of contracting hepatitis A in the coming weeks and so posed no risk to ongoing HAV transmission from this café.

Discussion

This report describes an outbreak of hepatitis A involving 10 cases epidemiologically linked to eating ready-to-eat foods at a café in Melbourne, Victoria.

It was suspected that the vehicle for this outbreak was faecally contaminated uncooked ready-to-eat foods such as salads and sandwiches for the following reasons:

- all cases consumed these types of foods;
- multiple handling of these foods during preparation was likely;
- hepatitis A virus is killed during the cooking process.

Additional case finding facilitated by the issuing of a media release was an appropriate public health response to this outbreak. This lead to the identification of an additional 5 hepatitis A cases associated with the outbreak. Close contacts of the additional cases were subsequently offered immunoglobulin to prevent tertiary transmission of HAV to others. Only 1 tertiary case was identified in the outbreak.

The environmental inspections identified no major concerns relating to the manner in which food was prepared and served at the premises. The inspection did, however, reveal that the index case had worked during his infectious period, which was contrary to the advice that the department had received from the case as well as the co-owner of the food premises. This was only ascertained during the second inspection of the premises, approximately 1 month after the index case was initially questioned during routine follow up. Whilst this delay did not alter the outcome of the investigation, the information may have expedited the media release, and therefore may have lead to more timely notification of outbreak cases. The department's protocol for investigating cases of hepatitis A specifies that where the case's occupation is a food handler and the case worked during their infectious period, co-workers should be provided with information about hepatitis A and recommended to have immunoglobulin delivered within 14 days of exposure to the infectious case. Fortuitously, the food handling co-workers in this outbreak were already immune to hepatitis A, and therefore posed no risk to the public in handling food. If however, the food handling co-workers were not immune, and had contracted hepatitis A from the index case, it is plausible that they may have been infectious whilst working as a food handler.

Two lessons learnt arose from this investigation, with the following recommendations currently under consideration:

1. that all staff illness registers, if available, are inspected during routine follow-up of hepatitis A cases among food handlers; and
2. that routine hepatitis A questionnaires are updated to capture foods that are not cooked such as salads or sandwiches.

The outbreak described in this report was recognised through a series of apparently sporadic case investigations, and is a timely reminder of the importance of routine investigation of this disease of public health importance.

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Egg-associated *Salmonella* outbreak in an aged care facility, New South Wales, 2008

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Abstract

Salmonellosis is a bacterial disease that causes acute gastroenteritis, with sudden onset of headache, abdominal pain, diarrhoea, nausea and sometimes vomiting. Infection is often associated with the consumption of foods prepared using raw eggs. During July to August 2008 an outbreak at an aged care facility (ACF) in New South Wales was confirmed as *Salmonella* Typhimurium phage type 44 (Stm 44) in eight of 45 residents. Two additional probable cases also occurred. Cases were located in each unit of the ACF and for 5 cases, onset of diarrhoea was between 45 to 64 hours (median of 46 hours) after consumption of a dessert containing raw eggs. Onset for 5 further cases occurred up to 9 days after this meal. Eggs were supplied to the ACF from a local farm. Stm 44 was detected on an egg in an unopened box at the ACF from this supplier. The raw-egg dessert was epidemiologically implicated as the likely source of the *Salmonella* and delayed onset cases may have resulted from ingestion of a smaller dose of *Salmonella*, or ongoing transmission through cross-contamination of kitchen machinery or surfaces. This outbreak demonstrates that inadequate cooking of eggs continues to pose a risk for *Salmonella* infection in settings with vulnerable populations. The findings of the investigation provide support for the importance of food safety regulations and demand further advocacy for measures to reduce the risks associated with the distribution, storage and preparation of shell eggs. *Commun Dis Intell* 2009;33:50–53.

Keywords: disease outbreak, *Salmonella* Typhumirium, aged care, eggs

Introduction

In Australia, between 7,000 and 9,500 *Salmonella* infections are notified each year to state and territory health departments. In 2007, of the 9,484 notified cases of *Salmonella*, 1,039 (11.0%) occurred within recognised foodborne outbreaks.[1] The most common *Salmonella* serotype notified in Australia is *S*. Typhimurium. In 2007, eggs and egg based dishes were the most common food vehicles implicated in outbreaks of *S*. Typhimurium and were responsible for 15% (22/149) of reported foodborne outbreaks.[1] *Salmonella* Typhimurium phage type 44 (Stm 44) has commonly been associated with eggs, with 12 of 18 Stm 44 outbreaks reported in Australia between 2001 and 2007 associated with raw egg exposure.[2]

On 7 August 2008, the OzFoodNet site at Hunter New England Population Health (HNEPH) was notified that seven of 45 residents in an aged care facility (ACF) had gastroenteritis. On 8 August, when *Salmonella* was isolated in stool samples from 2 cases, HNEPH and the New South Wales Food Authority (NSWFA) launched an investigation into the cause of the outbreak.

Methods

A site investigation was conducted at the ACF from 10 to 12 August. Case finding was conducted by interviewing staff and by reviewing each resident's clinical notes for evidence of diarrhoea or vomiting during the outbreak period. Clinical notes provided the cases' symptom profiles. Through heightened surveillance from 8 August, all residents with loose stools had a sample submitted for parasitic, bacterial and viral pathogen examination.

A probable case of *Salmonella* was defined as a resident having at least 1 episode of diarrhoea between 31 July and 11 August 2008. A confirmed case also had a positive stool culture for Stm 44.

HNEPH conducted a cohort study, obtaining details from the nursing manager or residents' clinical notes Details included residential unit, room number, age, sex, nature of diet, dietary practices, nutritional supplements, medications and attendance at group activities. Interviews to obtain staff and resident food histories were attempted. Infection control practices were reviewed using records in clinical notes and by direct observation. Data were entered into a Microsoft Excel database. STATA™ (version 8.0, Stata Corporation, College Station, Texas, USA) was used to calculate relative risks using the Fisher's exact test.

The menu for residents was reviewed for high risk food and ingredients and a site inspection for hygiene and food preparation practices was conducted.

The NSWFA collected samples for microbiological testing on 11 and 12 August. These included: food samples (2 cooked chicken meals, fruit salad, roast lamb meal, red lentil soup, frozen mixed berries);

eggs with intact, cracked, or dirty shells from both opened and unopened bulk cartons; and 14 environmental swabs (5 different food mixer appliances, ice cream scoops, utensil drawer, stainless steel food preparation bench tops and frequently used handles).

A trace-back investigation of the eggs used by the ACF resulted in visits to the egg farm and grading/distribution facility on 20 and 21 August, and 17 September. Egg samples were selected from all 4 chicken sheds, and from first and second quality eggs rejected through the grading (candling) process. Chicken feed, filtered bore water supplied to the sheds, and chicken faecal matter were sampled and environmental swabs were taken in each shed. At the egg grading facility, swabs of the grading equipment and a cleaning cloth were obtained for testing. All samples were tested for *Salmonella*.

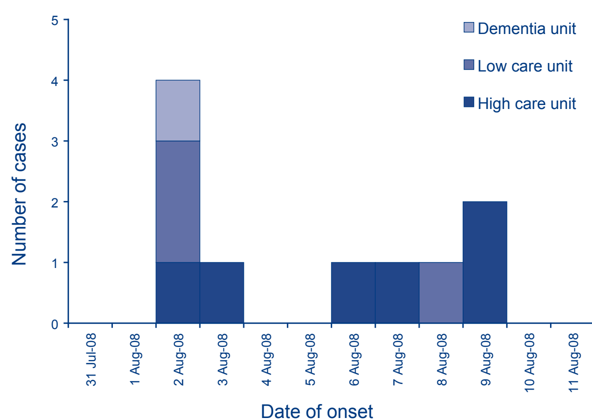
Results

Descriptive epidemiology

Ten of 45 residents (22%) of the ACF met the definition for a case, with Stm 44 confirmed in eight. The median age of cases was 81.5 years and eight were female. Nine cases experienced more than 1 diarrhoeal episode and one had bloody diarrhoea. Four cases experienced vomiting, one reported fever and one reported nausea. For the 4 cases where date of symptom cessation was recorded, the median symptom duration was 73 hours (range 30 minutes – 6.25 days).

A point-source outbreak with ongoing transmission was suggested by the epidemic curve of diarrhoea onset (Figure). The 5 initial cases occurred on 2 and 3 August, between 45 to 64 hours (median 46 hours) after consumption of a chocolate mousse dessert containing raw eggs. 5 subsequent cases had onset dates between 6 and 9 August. The median incubation period from consuming mousse to onset for all cases was 103 hours (5.4 days). The confirmed case with onset on 9 August had only 1 episode of mild diarrhoea.

Figure. Epicurve of diarrhoea onset in residents at the New South Wales aged care facility, 31 July to 11 August 2008, by residential care unit



No cases were admitted to hospital. Two residents with confirmed *Salmonella* and complex pre-existing medical conditions died during the fortnight after their symptoms resolved. Both residents' treating clinicians determined that salmonellosis was not an important factor contributing to their death.

The ACF houses residents in 3 units, according to the level of care required. The first 5 residents affected were distributed across all units in the ACF (Figure). Four of the last 5 cases occurred in the high care unit. The attack rate in residents in the high care unit was 46% (6/13) compared with 12.5% in both the low care (3/24) and dementia (1/8) units.

Between 2 and 10 August, there were at least 3 documented times when a nurse cared for both well residents and residents affected by gastroenteritis during the same shift; strict cohorting of residents and staff was fully implemented on 10 August.

Analytical epidemiology

Cases were not able to be interviewed about their food history due to very poor recall and/or cognitive impairment. Staff were unable to reliably report case food histories and the ACF did not accurately record the foods consumed by each resident. It was recommended that these records be kept in the future.

Twenty-two of 45 residents ate pureed or soft food and 15 required assistance with feeding. Cases were more likely to have eaten pureed foods (RR=4.2, 95% C.I. 1.0, 17.6, $P = 0.029$) and to have had assistance with feeding (RR =8.0, 95% C.I. 1.9, 33.1, $P = 0.009$) than non-cases.

Fourteen residents (including 3 cases) were taking protein pump inhibitors (PPIs) to reduce gastric acidity. PPIs have previously been associated with increased susceptibility to gastrointestinal infections.[3,4] Eight residents received nutritional supplements. There was no association between disease and use of PPIs, supplements or attendance at exercise or bingo sessions. No cases had received laxative medications in the 24 hours prior to diarrhoea onset.

Environmental investigation

Meals at the ACF were prepared in a central, on-site kitchen according to a rotating 4 week menu. Between 27 July and 2 August, one particularly high

risk meal was identified; a chocolate mousse containing raw egg yolks and whites was served at lunch on 31 July. This dessert was prepared on 30 July by 1 kitchen staff member from 9 whole eggs, whipped cream, margarine and melted chocolate. This staff member indicated that they had been well, all equipment had been clean and that food was under temperature control. Prior to serving for lunch on 31 July, the mousse was plated into individual serving dishes using an ice cream scoop and tinned fruit was added. The covered plates were stored in the cool room. The mousse was served to all residents except 2 lactose intolerant residents (who were not cases). Nursing staff reported that desserts are usually eaten by all residents to whom they are served, although there was no accurate record of individual consumption or amount of mousse consumed. The kitchen mixer used for mousse preparation was also utilised in the preparation of other ready-to-eat foods, particularly foods for residents on a pureed diet.

Each week, the ACF used approximately 174–270 free-range second-grade eggs, supplied by a local farm. Between one and 3 eggs in each tray of 30 were observed to be cracked or visibly dirty in an unopened carton of eggs. No raw eggs were reported to have been included in other dishes served prior to or during the outbreak. The ACF was advised to immediately cease serving raw egg foods or undercooked eggs, to only purchase first grade eggs and reject cracked and/or dirty eggs, and to use different mechanical mixing devices for raw eggs or products containing raw eggs and for ready-to-eat foods. A subsequent visit demonstrated compliance with an official NSWFA Improvement Notice.

Microbiology

All 8 residents with confirmed *Salmonella* were infected with Stm 44, with an identical MLVA pattern (3-10-9-9-523). The rinse of an uncracked egg in an unopened carton of eggs supplied from the local farm on 7 August 2008, yielded Stm 44 with the same MLVA pattern identified in human isolates. There were no remaining eggs from batches received by the ACF on 22 or 29 July for sampling. No other samples had evidence of Stm contamination.

Discussion

Chocolate mousse made with contaminated raw eggs was implicated as the likely source of infection with Stm 44 during this outbreak in an ACF. Whilst there were no remaining eggs of the batch used by the ACF in the implicated mousse, Stm 44 was isolated from the shell of an egg that was sampled from an unopened carton of a subsequent batch supplied to the ACF by the same local farm. Initial cases occurred within a compatible incubation period following consumption of the mousse and occurred in each ACF unit, suggesting a point-source foodborne infection.

In the kitchen, the same equipment was used for both raw and ready-to-eat foods, and there were a number of unsafe food handling practices that could facilitate cross-contamination, as well as some inadequate cleaning practices. All eggs used were 'seconds', with evidence of some being cracked and dirty, and there was no procedure for rejecting cracked or dirty eggs at time of receipt. Using cracked and dirty eggs increases the risk of *Salmonella* contamination of foods, the environment and persons handling the eggs.[5,6]

New cases occurred over 9 days following consumption of the mousse on 31 July. The initial cases occurred following the usual incubation period of *Salmonella*, of 6 to 72 hours.[7] There are 3 possible explanations for the occurrence of cases with symptom onsets between 6 and 9 August. Longer median incubation periods of up to 8 days[8–10] have been observed during *Salmonella* outbreaks when the infectious dose is low. Extremely long incubation periods for *S.* Typhimurium of up to 27 days have occasionally been observed.[8] Neonates, the elderly and immunocompromised individuals are more likely to become infected and symptomatic when exposed to a lower infectious dose than other population groups.[4] The later cases may have ingested a lower dose of *Salmonella* if it was not well distributed through the mousse, if they ate a smaller portion, or if other ready-to-eat food was cross contaminated with Stm 44 through a shared-use kitchen implement or machine. It is also possible that cases were exposed to *Salmonella* through food served on multiple days by this latter mechanism. Blenders may be difficult to clean, so may provide a reservoir for bacteria to accumulate.[4,11] The lever ice cream scoops used for serving foods such as chocolate mousse and mashed potato, pumpkin or ice cream, may also be difficult to clean providing an avenue for cross contamination. However, environmental swabs of the blenders and ice cream scoops did not detect *Salmonella* and they were clean on visual inspection. Kitchen staff stated they were cleaned and sanitised directly after each use.

In addition to being used in the mousse, eggs from the same batch were used in other cooked meals possibly until 6 August. Presuming this batch of eggs was contaminated with Stm 44, contamination of other foods or surfaces/implements/machines in the kitchen may have occurred.

Finally, it is possible that person-to-person spread occurred within the facility.[7,12] Initially, nursing staff were exposed to stools from symptomatic residents during the same shifts they worked with well resi-

dents. Transmission of *Salmonella* from nurses, food handlers or visitors to residents may have been possible although there was no evidence to support this hypothesis.

Conclusion

This outbreak highlights the need to avoid high risk foods, including raw-egg foods and undercooked eggs, in settings with vulnerable populations—particularly aged care facilities—and the potential value of clearer guidelines relating to egg risks for these facilities. The need for routine optimal food preparation and infection control practices to prevent the spread of pathogens in food is reinforced, and the findings of the investigation provide support for the importance of regulations such as Standard 3.3.1 – Food Safety Programs for Food Service to Vulnerable Populations[13] and demand further advocacy to reduce the risk associated with the distribution, storage and preparation of shell eggs.

Acknowledgements

This work was funded by OzFoodNet, Department of Health and Ageing, Canberra and Hunter New England Population Health.

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Quarterly reports

OzFoodNet quarterly report, 1 October to 31 December 2008

The OzFoodNet Working Group

Introduction

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally to investigate foodborne disease. 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, occurring in Australia from 1 October to 31 December 2008.

Data were received from OzFoodNet epidemiologists in all Australian states and territories. The data in this report are provisional and subject to change, as the results of outbreak investigations can take months to finalise.

During the 4th quarter of 2008, OzFoodNet sites reported 244 outbreaks of enteric illness, including those transmitted by contaminated food. Outbreaks of gastroenteritis are often not reported to health agencies or the reports may be delayed, meaning that these figures under-represent the true burden of enteric illness. In total, these outbreaks affected 3,575 people, of which 84 were hospitalised. There were 9 deaths reported during these outbreaks. The majority (77%, n=188) of outbreaks were due to person-to-person transmission (Table 1).

Table 1. Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet sites, 1 October to 31 December 2008

Transmission mode	Number of outbreaks	Per cent of total
Foodborne	31	13
Person-to-person	188	77
Unknown – *Salmonella* cluster	7	3
Unknown – other pathogen cluster	1	0
Unknown	17	7
Total	244	100

Foodborne disease outbreaks

There were 31 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table 2). These outbreaks affected 349 people and resulted in 15 hospitalisations. There was 1 death reported during these outbreaks. This compares with 27 foodborne outbreaks for the 4th quarter of 2007[1] and 17 outbreaks for the 3rd quarter of 2008.[2]

Salmonella was responsible for 12 outbreaks during this quarter, with *S.* Typhimurium being the most common serotype (83%, n=10). There were 3 outbreaks each due to *S.* Typhimurium phage types 9 and 126, two due to *S.* Typhimurium 44 and one each due to *S.* Typhimurium 170 and 170 var. There was 1 outbreak each due to *S.* Thompson and *S.* Virchow 8.

Of the remaining 19 outbreaks, 12 were due to foodborne toxins, including 3 *Staphylococcus aureus* outbreaks, 1 ciguatera fish poisoning outbreak, 1 *Clostridium perfringens* outbreak, and 7 suspected bacterial toxin outbreaks. There was 1 outbreak each due to *Campylobacter*, *Escherichia coli*, and norovirus. The remaining 4 outbreaks were of unknown aetiology.

Eighteen outbreaks (58%) reported in this quarter were associated with food prepared in restaurants. There were four outbreaks associated with food prepared in camps and three associated with food prepared in bakeries. Individual outbreaks were associated with food prepared at an aged care facility, commercial caterer, grocery store/delicatessen, national franchised fast food restaurant, primary produce, and a private residence.

To investigate these outbreaks, sites conducted 8 cohort studies, 2 case control studies, and collected descriptive case series data for 18 investigations. As evidence for the implicated vehicle, investigators collected microbiological evidence in 4 outbreaks, analytical epidemiological evidence in 7 outbreaks, and descriptive evidence in 22 outbreaks.

The following jurisdictional summaries describe key outbreaks and public health actions which occurred in this quarter. Tasmania and Western Australia did not report any foodborne outbreaks during this quarter.

Table 2. Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 October to 31 December 2008 (n=31)

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence†	Responsible vehicle(s)
ACT	December	Restaurant	*Salmonella* Typhimurium 44	23	A	Eggs, hollandaise sauce
NSW	November	Camp	*Campylobacter*	6	D	Chicken schnitzel
	November	Restaurant	*Clostridium perfringens*	4	AM	Suspected curry
	November	Restaurant	*Salmonella* Typhimurium 170 var	33	AM	Raw eggs in Caesar salad dressing
	November	Bakery	*Salmonella* Typhimurium 126	16	AM	Chocolate mousse cake
	November	Bakery	*Salmonella* Typhimurium 126	10	AM	Chocolate mousse cake
	November	Grocery store/delicatessen	*Salmonella* Typhimurium 126	2	D	Cabanossi or pepperoni
	December	National franchised fast food restaurant	*Salmonella* Typhimurium 44	3	D	Eggs
	December	Aged care facility	*Salmonella* Typhimurium 9	6	D	Unknown
	December	Restaurant	Suspected bacterial toxin	5	D	Lamb korma
	December	Restaurant	Suspected bacterial toxin	20	D	Unknown
	December	Restaurant	Suspected bacterial toxin	25	D	Unknown
	December	Restaurant	Unknown	5	D	Suspected aioli
	November	Restaurant	Unknown	6	D	Unknown
	October	Restaurant	Unknown	6	D	Unknown
	November	Restaurant	Suspected bacterial toxin	4	D	Suspected pasta with tomato sauce
	November	Restaurant	Suspected bacterial toxin	3	D	Unknown
	December	Restaurant	Unknown	3	D	Unknown
NT	December	Camp	*Salmonella* Thompson	8	D	Unknown
	December	Restaurant	*Salmonella* Typhimurium 9	2	D	Suspected raw egg mayonnaise
	November	Restaurant	Suspected bacterial toxin	3	D	Suspected meat lovers pizza
Qld	December	Primary produce	Ciguatera Fish Poisoning	3	D	Cod
	November	Camp	*Esherichia coli* – multiple serotypes	7	M	Untreated tank water
	October	Private residence	*Salmonella* Virchow 8	3	D	Chicken curry
	October	Camp	*Staphylococcus aureus*	23	D	Roast chicken
	November	Restaurant	*Staphylococcus aureus*	4	D	Unknown
	November	Commercial caterer	*Staphylococcus aureus*	16	M	Multiple foods

Table 2. Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 October to 31 December 2008, *continued*

State	Month of outbreak	Setting prepared	Infection	Number affected	Evidence[†]	Responsible vehicle(s)
SA	December	Bakery	*Salmonella* Typhimurium 9	15	A	Sweet bakery products
Vic	December	Restaurant	Norovirus	26	D	Multiple foods
	October	Restaurant	*Salmonella* Typhimurium 170	18	D	Tiramisu
	October	Restaurant	Suspected bacterial toxin	41	A	Buffet meal meats (lamb, pork, chicken)

* No foodborne outbreaks were reported by Tasmania or Western Australia during the quarter.

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

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

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

Australian Capital Territory

The Australian Capital Territory reported 1 outbreak of foodborne or suspected foodborne illness during the quarter.

An outbreak of *S.* Typhimurium 44 affected 23 people (16 laboratory confirmed cases) at a restaurant. A case control study showed strong associations with illness and having eaten eggs or hollandaise sauce. There were no positive food or environmental samples collected during the environmental investigation. Traceback of eggs was undertaken, with links to a New South Wales supplier established. A voluntary recall of eggs was undertaken by the supplier.

New South Wales

New South Wales reported 17 outbreaks of foodborne or suspected foodborne illness during this quarter: 6 *Salmonella* outbreaks, 1 *Campylobacter* outbreak, 1 outbreak of *Clostridium perfringens*, 5 outbreaks due to suspected bacterial toxins, and 4 outbreaks of unknown aetiology.

There were 3 outbreaks of *S.* Typhimurium 126 (MLVA 3-17-16-13-523), two of which were associated with a chocolate mousse cake containing raw eggs sourced from a farm associated with a *S.* Typhimurium 126 outbreak earlier in 2008. Two groups of people were ill after consuming this chocolate mousse cake, and the NSW Food Authority (NSW FA) prohibited the owners of the cake shop from selling cakes with raw egg mousse. *S.* Typhimurium 126 was identified in samples from birds and rats collected by the NSW FA at the egg farm. The 3rd outbreak was linked to cabanossi and pepperoni, however a different exposure may be possible as the outbreak occurred at the same time as the previous 2 egg-associated outbreaks and had a matching MLVA pattern.

An outbreak of *S.* Typhimurium 170 var (MLVA 3-9-7-13-523) that affected 33 cases was associated with foods containing raw eggs and chicken eaten at a café. Fouteen of the 33 cases were laboratory-confirmed. *S.* Typhimurium 170 was identified in pesto and in chopped parsley, however there was no epidemiological association between illness and eating these foods. Environmental sampling from the egg farm identified several different *Salmonella* serotypes.

An outbreak of *S.* Typhimurium 9 (MLVA 3-10-14-11-496) in a combined nursing home and hostel facility affected six of 60 residents. All cases had an unmodified diet. *S.* Kiambu was detected on raw chicken in the kitchen while samples of eggs, cooked meals and devon were negative for *Salmonella*. Environmental swabs were also negative for bacteria. A review of the menu and the NSW FA investigation of the central kitchen did not reveal high risk foods or practices to explain the point-source outbreak.

An outbreak of *S.* Typhimurium 44 (MLVA 3-10-7-9-523) affected 3 people who ate meals containing eggs from a national franchised fast food restaurant. The eggs were sourced from a large producer but traceback to farm level was not possible.

The outbreak of *C. perfringens* affected 4 people who consumed an Indian takeaway meal. *C. perfringens* was isolated from the curry and it is suspected that rapid cooling of the curry to safe temperatures after cooking did not occur.

The outbreak of *Campylobacter* occurred at a school camp where six from 43 people developed gastroenteritis after eating chicken schnitzel that had been prepared by camp members. *Campylobacter* was identified in the stools of 2 cases.

In 1 outbreak of suspected bacterial toxin poisoning, 4 people fell ill after consuming pasta and tomato sauce in a restaurant. The NSW FA inspected the premises and observed inadequate cleaning of equipment and slow cooling of prepared pasta.

There were 4 outbreaks of unknown aetiology suspected to be caused by food reported in this quarter. All four occurred at restaurants and involved a variety of foods, including a raw egg aioli, a seafood hotpot, and a lamb pizza.

Northern Territory

The Northern Territory reported 3 outbreaks of foodborne or suspected foodborne illness during the quarter.

An outbreak of *S.* Thompson affected 8 people at a remotely located farm/work camp; no food vehicle was identified. An outbreak of *S.* Typhimurium 9 affected 2 people at a restaurant after consumption of a raw egg mayonnaise. A suspected bacterial toxin outbreak affected 3 people following consumption of a 'meat-lovers' pizza at a restaurant.

Queensland

Queensland reported 6 outbreaks of foodborne or suspected foodborne illness during this quarter: 3 *Staphylococcus aureus* outbreaks, 1 ciguatera fish poisoning outbreak, 1 *S.* Virchow 8 outbreak and a Shiga toxin producing *Escherichia coli* (STEC) outbreak.

An outbreak of *Staphylococcus aureus* affected 22 of 29 students and 1 of 4 teachers after consuming a common meal containing roast chicken at a school camp. Gross temperature abuse of roast chicken was the suspected cause of the outbreak. Nine raw chickens were stored overnight at room temperature, cooked the following day and the cooked meat removed using bare hands. The cooked chicken was subsequently stored in large ice cream containers overnight and not served until the following evening for dinner.

Another outbreak of *S. aureus* affected 4 people after consuming a meal consisting of meatballs, rice and chicken souvlaki. While some foods collected in the investigation tested positive for other bacteria (*Bacillus cereus* and *C. perfringens*), no *S. aureus* was detected. Environmental Health provided the restaurant with an audit report with appropriate corrective actions and follow up inspections were conducted.

The third outbreak of *S. aureus* affected 16 people following a catered event. *Staphylococcus* enterotoxin was detected in chicken meat samples and *S. aureus* was cultured from chicken, pavlova and cream; six of 11 stool specimens were culture positive for *S. aureus* and stools were also positive for *Staphylococcus* enterotoxin.

Three cases of suspected ciguatera fish poisoning were notified in Townsville following the consumption of a cod fish caught off Lucinda (Truck Reef).

An STEC outbreak in a school camp affected 7 people. Multiple serotypes were identified among the cases: O26, O112ab:H2, O153:H21, O88:H25, Ont:H7, and O174:H-. Six of the 7 cases were school children attending the camp; the 7th case was the father of one of the infected children. Both the father and son were asymptomatic so mode of transmission of infection to the father could not be determined, however, the father had consumed untreated tank water during a short visit to the camp; this was identified as the potential source of the infection. STEC genes (stx1, ehxA, saa) were identified in the water sample and 2 types of shiga toxin-producing *E. coli* were cultured (serotypes pending). The serotypes of the water isolates are pending. Bottled water is now being used at the camp for drinking purposes until a decision has been made regarding the most appropriate method of water treatment for the tanks.

South Australia

South Australia reported 1 outbreak of foodborne or suspected foodborne illness during the quarter.

An outbreak of *S.* Typhimurium 9 affected 15 people and was associated with a bakery. A case control study showed a statistically significant association between the consumption of sweet products from the bakery with illness. Environmental investigation at the bakery reported satisfactory conditions of the premises and food handling procedures. Food and environmental swabs from the bakery were all negative for *Salmonella*.

Victoria

Victoria reported 3 outbreaks of foodborne or suspected foodborne illness this quarter.

A suspected *C. perfringens* outbreak affected 41 people who ate at a large buffet restaurant. Cases were identified from a booking list obtained after illness was reported in 3 separate groups of patrons. Thirteen cases submitted faecal samples but many were collected several days after symptoms resolved. *C. perfringens* enterotoxin was detected in 1 specimen. Analysis of hypothesis generating questionnaires showed a statistically significant association with consumption of 3 different foods and illness – lamb tenderloin (RR 4.0; 95% CI 2.3-7.0); chicken cacciatore (RR 2.0; 95% CI 1.4-2.8) and roast pork (2.4; 95% CI 1.4-4.0).

An outbreak of S. Typhimurium 170 affected 18 people associated with 2 different restaurants with the same proprietor. During the investigation it was discovered that foods were transferred between the restaurants. It was confirmed that the 3 initially reported cases had consumed tiramisu at these restaurants on the same weekend. In total, 18 cases (15 laboratory confirmed cases) had eaten tiramisu from either of these 2 restaurants. The tiramisu was made using raw eggs. The proprietor was advised about the risks associated with preparing and selling ready to eat foods containing raw eggs and as a result they now prepare tiramisu without eggs.

An outbreak of norovirus affected 26 people at a restaurant. Cases were identified from a booking list obtained after illness was reported in 1 group; subsequently illness was identified in 5 separate groups who dined at the restaurant on one of 2 consecutive days. Thirty-one attendees were interviewed and 26 people met the case definition. Ten faecal specimens were collected from cases and norovirus was detected in eight of these. During the investigation it was discovered that a food handler at the restaurant had an onset of diarrhoea whilst working at the restaurant on the same evening that the original group had dined. The food handler was responsible for preparation of the antipasto platters, which most of the cases consumed. A cohort study was not conducted so it was not possible to determine which foods were associated with illness, however, six ready to eat foods were sampled during

the investigation and four had either high bacterial counts, coliforms or *E. coli* detected, which is indicative of poor storage and handling.

Comments

During the quarter, the number of outbreaks (n=244) reported was lower than previous quarters in 2008, although the number of foodborne outbreaks (n=31) reported was the highest for 2008. There were 12 foodborne outbreaks of salmonellosis reported this quarter, compared with 3 foodborne outbreaks of salmonellosis last quarter and 11 outbreaks each for the 1st and 2nd quarters of 2008.

There were increased numbers of toxin-related outbreaks reported during the quarter; of the 12 toxin related outbreaks, three were confirmed as *Staphylococcus aureus,* an organism that is most likely present in foods as a result of contamination by food handlers. There were 7 outbreaks that were suspected to be due to bacterial toxins; the exact aetiology of these outbreaks was not elucidated either because samples were not collected or there was no quantification of detected enterotoxin.

Of the 12 salmonellosis outbreaks this quarter, 10 were due to *S.* Typhimurium (various phage types/MLVA types). While these outbreaks are reported here by their phage type, in New South Wales, they are, for the most part, being detected by routine MLVA surveillance. Of the 10 *S.* Typhimurium outbreaks, eight of these were associated with eggs, highlighting the ongoing role of uncooked or raw eggs in foodborne illness; the food items implicated in egg-associated outbreaks this quarter included hollandaise sauce, Caesar salad dressing, chocolate mousse cake, raw egg mayonnaise, bakery products, and tiramisu.

The outbreak of STEC associated with untreated drinking water at a camp in Queensland highlights the need to consider treating tank water if it is to be used for consumption.

Acknowledgements

OzFoodNet thanks the investigators in the public health units and state and territory departments of health, as well as public health laboratories and local government environmental health officers who provided data used in this report. We would also like to thank laboratories conducting serotyping, molecular typing and phage typing of *Salmonella* for their continuing work during this quarter.

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Communicable diseases surveillance

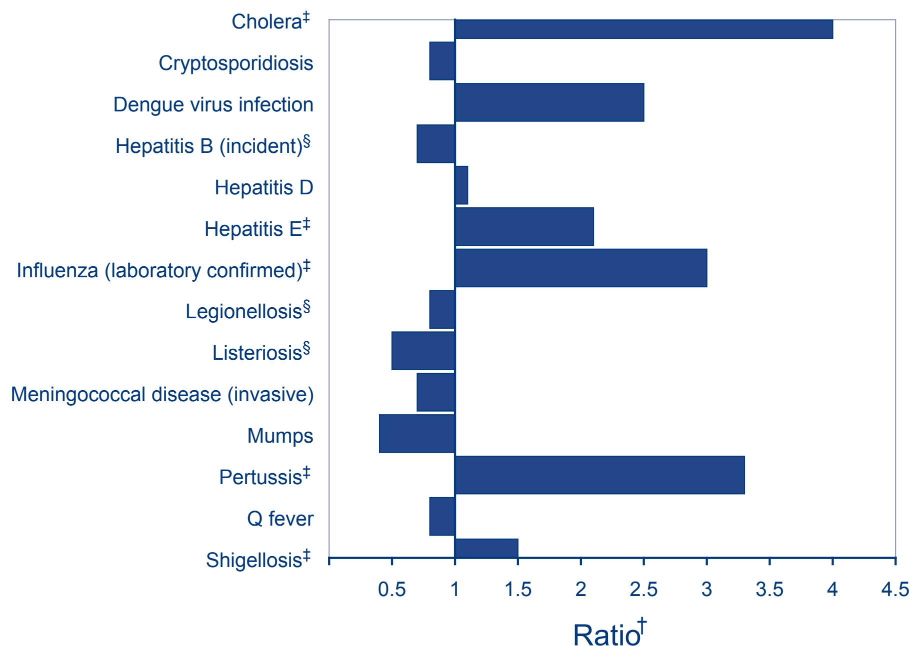
Highlights for 4th quarter, 2008

Communicable diseases surveillance highlights report on data from various sources, including the National Notifiable Diseases Surveillance System (NNDSS) and several disease specific surveillance systems that provide regular reports to Communicable Diseases Intelligence. These national data collections are complemented by intelligence provided by state and territory communicable disease epidemiologists and/or data managers. This additional information has enabled the reporting of more informative highlights each quarter.

The NNDSS is conducted under the auspices of the Communicable Diseases Network Australia. NNDSS collates data on notifiable communicable diseases from state and territory health departments. The Virology and Serology Laboratory Reporting Scheme (LabVISE) is a sentinel surveillance scheme which collates information on laboratory diagnosis of communicable diseases. In this report, data from the NNDSS are referred to as 'notifications' or 'cases' while data from the LabVISE scheme are referred to as 'laboratory reports'.

Figure 1 shows the changes in selected disease notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a diagnosis in the 4th quarter (October to December) 2008, in comparison with the 5-year mean for the same period. Notifications were above the 5-year mean for the same period and exceeded 2 standard deviations from the 5-year mean for: cholera, hepatitis E, influenza (laboratory confirmed), pertussis and shigellosis. Notifications were below the 5-year mean by more than 2 standard deviations for: hepatitis B (incident), legionellosis, listeriosis and tetanus.

Figure 1. Selected diseases* from the National Notifiable Diseases Surveillance System, comparison of provisional totals for the period 1 October to 31 December 2008 with historical data†



* Selected diseases are chosen each quarter according to current activity. Five year averages and the ratios of notifications in the reporting period in the 5-year mean should be interpreted with caution. Changes in surveillance practice, diagnostic techniques and reporting, may contribute to increases or decreases in the total notifications received over a 5-year period. Ratios are to be taken as a crude measure of current disease activity and may reflect changes in reporting rather than changes in disease activity.

† Ratio of current quarter total to mean of corresponding quarter for the previous 5 years.

‡ Where the number of notifications of the current quarter exceeds the mean of the corresponding quarter for the previous 5 years by more than 2 standard deviations.

§ Where the number of notifications of the current quarter is below the mean of the corresponding quarter for the previous 5 years by more than 2 standard deviations.

Gastrointestinal diseases

Hepatitis E

Between 1 October and 31 December 2008, there were 8 notifications of hepatitis E in Australia, 2.1 times the 5-year mean of 4 notifications for the same period. Hepatitis E cases in Australia are commonly imported, and all of the 8 notifications of hepatitis E in Australia during the quarter were overseas acquired.

Shigellosis

Between 1 October and 31 December 2008, there were 206 notifications of shigellosis in Australia, a 27% increase over the number reported during the same quarter of 2007 (162 notifications), and 1.5 times the 5-year mean of 134 notifications for the same period.

The highest notification rate was in the Northern Territory, where 44 cases were notified during the quarter, for an annualised rate of 81.9 cases per 100,000 population, compared with annualised rates of less than 6.0 cases per 100,000 population in each of the other jurisdictions during the quarter. Notification rates for shigellosis in the Northern Territory are usually high compared with other Australian states and territories, with an annual rate of 80.5 cases per 100,000 population in 2007 compared with 2.8 cases per 100,000 population nationwide.[1]

Continuing clusters of shigellosis during the 4th quarter of 2008 contributed to the observed increase in notifications compared with previous years. These clusters were reported from 3 states (Queensland, New South Wales and Victoria) amongst adult men who frequently report sex with other men as a risk factor.

Quarantinable diseases

Cholera

Between 1 October and 31 December 2008, there were 4 notifications of cholera, which was 4 times the 5-year mean of 1 notification for the same period. Two of these cases were reported from New South Wales and two from Western Australia. All of these cases were acquired overseas, two in India and one each in the Philippines and Singapore.

Vaccine preventable diseases

Influenza (laboratory confirmed)

Laboratory-confirmed influenza is a nationally notifiable disease in all states and territories in Australia. Data are reported from state and territory health departments to the NNDSS.

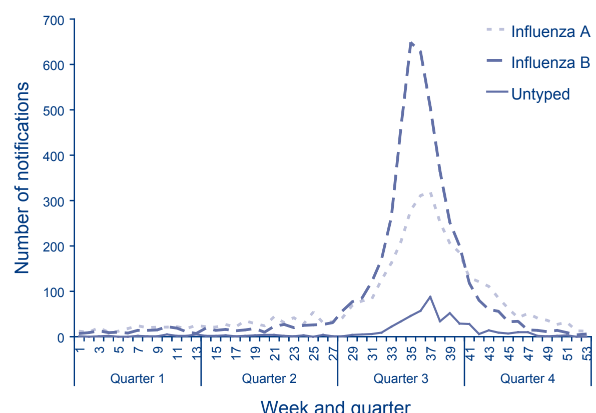
The influenza 2008 season commenced approximately 5 weeks late compared with the 2007 season. During the 4th quarter of 2008, notifications declined from the seasonal peak in week 36 and returned to baseline levels.

The total number of laboratory-confirmed influenza notifications to NNDSS for the 4th quarter was 1,545 cases (approximately 17% of year-to-date notifications). The majority of notifications during this quarter were from New South Wales with 429 cases (28%) and Queensland with 383 cases (25%).

Notifications in the 4th quarter of 2008 were 3 times the 5-year mean for the same period, this is due to the approximate 5-week delay in the start and end of the season and overall high seasonal numbers in 2008 (1.9 times the 5-year mean).

During the first 2 quarters of 2008, influenza notifications to NNDSS were predominantly type A while during the third quarter, notifications were predominantly type B. As overall notifications continued to decline during the 4th quarter (week 40 onwards), there appeared to be an increase in the predominance of type A (Figure 2) as type B notifications decreased more rapidly.

Figure 2. Typing characteristics of notifications of laboratory-confirmed influenza to the National Notifiable Diseases Surveillance System, Australia, 1 January to 31 December 2008, by week of diagnosis



Most jurisdictions experienced a slight increase in notifications between weeks 44 and 49. South Australia had a significant increase during week 44, followed by a decline, and another slight increase in week 48. The Northern Territory experienced an increase in notifications in weeks 46 and 47, Tasmania in week 46, Victoria in week 47 and Western Australia in week 49.

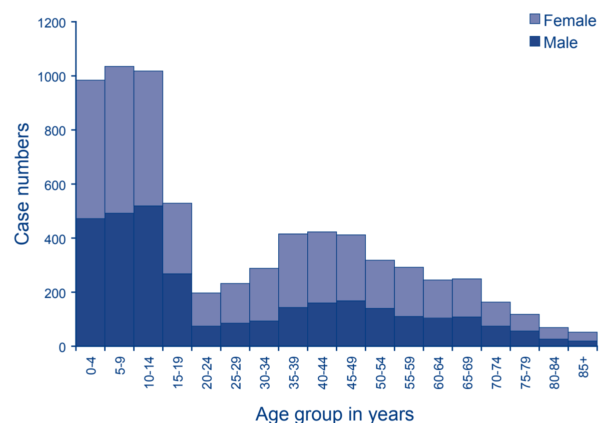
Pertussis

Between 1 October and 31 December 2008, 7,050 cases of pertussis were reported to the NNDSS. The majority of cases were reported in New South Wales (n=4,424) followed by Queensland (n=1,017) and Victoria (n=571), with South Australia (n=560), Western Australia (n=177), the Northern Territory (n=122), Tasmania (n=115) and the Australian Capital Territory (n=64) also reporting cases in this quarter. Case numbers in the 4th quarter (Q4) were 4.7 times more than in the same period in 2007 (n=1,487) and 3.3 times the 5-year mean for this period. Pertussis notifications in Q4 made up 50% of the total notifications in 2008 with numbers increasing with each quarter as follows Q1 (1,538 cases), Q2 (2,032 cases), Q3 (3,498 cases). The annualised notification rate for this quarter of 134 cases per 100,000 population was significantly higher than for the same period in 2007 (28 cases per 100,000). Fifty-six per cent of cases in Q4 were female (n=3,928) and 44% were male (n=3,111).

The total number of cases with a diagnosis date in 2008 was 14,118, which exceeded both the same period in 2007 (n=5,333) and the year-to-date 5-year mean (n=8,274).

A high proportion (29%) of the total case numbers in this quarter were aged between 0 and 9 years. Of these, there were 985 cases (14% of the total) in the 0–4 years age group, which can be further broken down to show 224 cases (3% of the total) in the 0–6 month age group and 219 cases (3% of the total) in the 7–23 months age group with 542 cases (or 8% of the total) in the 2–4.9 years age group. There were another 1,039 cases (15%) in the 5–9 year age group (Figure 3). Other key age groups include those aged 15–19 years (who under the National Immunisation Program are recommended to receive a booster dose of dTpa) where an additional 530 cases (18%) were reported; and the traditional child bearing 20–44 years in which 1,558 cases (22%) were reported.

Figure 3. Pertussis notifications, Australia, 1 October to 31 December 2008, by sex and age group



Vectorborne diseases

Mosquito-borne diseases under national surveillance in Australia include alphaviruses (Barmah Forest virus and Ross River virus), flaviviruses (dengue virus, Japanese encephalitis, Kunjin, Murray Valley encephalitis, yellow fever), arbovirus not elsewhere classified, and malaria.

Dengue virus infection

Dengue virus infection presents as an acute febrile illness of sudden onset and characterised by fever (biphasic), intense headache, myalgia, particularly backache, arthralgia, retro-orbital pain, anorexia, vomiting, diarrhoea gastrointestinal disturbance and rash. Dengue virus (DENV) has 4 serotypes: 1, 2, 3 and 4.[2]

On 1 December 2008 Queensland Health declared an outbreak of dengue serotype 3 in Cairns. The outbreak began after a resident who had visited Kalimantan, Indonesia returned to the city. Of the 198 cases of dengue virus infection reported during the quarter, 111 were overseas acquired cases and 87 were locally acquired in the Cairns outbreak. At the time of writing, the number of cases of locally acquired dengue in Cairns (serotype 3) since the outbreak was declared on 1 December 2008, has risen to over 300 and a further 2 outbreaks have been reported in Townsville. Of the 55 cases reported in Townsville since 1 January 2009, 40 cases were infected with serotype 1, 11 cases were infected with serotype 3 and four are unknown. More severe forms of the disease dengue haemorrhagic fever/dengue shock syndrome (DHF/DSS) occur when someone who has been infected at some time in the past becomes infected with a virus of a different serotype. DHF/DSS occurs most frequently in infants and young children. Having 2 strains of the virus circulating in Townsville increases the risk of a case of DHF/DSS.

Queensland health authorities are experienced in responding to outbreaks of the disease and have

implemented the Dengue Fever Management Plan. A major focus of the response is raising public awareness of the need for all members of the public to take responsibility for reducing mosquito breeding opportunities around their homes and for those people living in areas where dengue fever is known to occur to seek medical advice if feeling unwell.

State Emergency Service volunteers have door knocked house to house in Cairns warning residents about the dangers of dengue fever. The Queensland Government has also distributed free insect repellent to all schools in Cairns and Townsville.

Outbreaks of dengue in north Queensland are not unprecedented; in 2003 and 2004 there were over 700 cases of locally acquired dengue reported in Queensland and in 1998 over 500 cases of dengue recorded.

Acknowledgements

Thanks go to staff of the Surveillance Branch of the Australian Government Department of Health and Ageing and all our state and territory data managers.

References

1. The OzFoodNet Working Group. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia: Annual report of the OzFoodNet Network, 2007. *Commun Dis Intell* 2008;32:400–424.
2. Heymann DL, ed. *Control of Communicable Diseases Manual*. 18th edn. Washington: American Public Health Association; 2004.

Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 42,693 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 October to 31 December 2008 (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 (incident)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (incident)	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
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
STEC, VTEC	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 (all)	
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions except South Australia
Syphilis - congenital	All jurisdictions

Table 1. Reporting of notifiable diseases by jurisdiction, *continued*

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
Lyssaviruses (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

* Notifiable in South Australia as of 1 May 2008.

† Flavivirus (NEC) replaced Arbovirus (NEC) from 1 January 2004. Arbovirus (NEC) replaced Flavivirus (NEC) from 2008.

NEC Not elsewhere classified.

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 October to 31 December 2008, by date of diagnosis*

Disease	State or territory								Total 4th quarter 2008†	Total 3rd quarter 2008	Total 4th quarter 2007	Last 5 years mean 4th quarter	Year to date 2008	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	2	0	2	0	0	0.0	2	0.2	0.0
Hepatitis B (incident)	0	11	2	8	1	2	20	2	46	75	72	67.0	245	293.8	0.7
Hepatitis B (unspecified)	11	498	59	244	89	11	447	162	1,521	1,921	1,624	1,520.0	6,812	6,205.6	1.0
Hepatitis C (incident)	0	8	1	NN	9	6	38	0	62	81	80	88.2	306	366.2	0.7
Hepatitis C (unspecified)	58	946	60	684	131	77	567	322	2,845	3,112	2,850	3,024.8	11,938	12,427.2	0.9
Hepatitis D	0	3	0	0	0	0	2	1	6	9	7	5.6	42	30.2	1.1
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	0	0	0.2	0	1.4	0.0
Campylobacteriosis§	104	NN	53	1,315	479	126	1,410	555	4,042	3,376	4,711	4,534.4	15,481	15,970.2	0.9
Cryptosporidiosis	1	71	35	178	9	6	74	42	416	315	810	544.0	1,966	2,426.0	0.8
Haemolytic uraemic syndrome	0	7	0	2	0	0	1	0	10	6	7	7.0	30	16.8	1.4
Hepatitis A	0	18	0	8	1	1	13	4	45	57	35	65.6	274	304.6	0.7
Hepatitis E	0	4	0	1	0	0	2	1	8	10	3	3.8	43	22.4	2.1
Listeriosis	0	4	0	3	0	0	0	1	8	18	16	15.8	68	60.2	0.5
Salmonellosis	50	585	147	580	141	27	361	227	2,118	1,326	2,196	2,064.8	8,291	8,209.8	1.0
Shigellosis	2	37	44	31	17	0	47	28	206	202	162	134.2	832	568.0	1.5
STEC, VTEC‖	0	5	0	16	13	0	3	0	37	16	42	22.2	103	72.8	1.7
Typhoid	0	13	0	0	1	0	7	2	23	19	13	14.4	103	69.4	1.6
Quarantinable diseases															
Cholera	0	2	0	0	0	0	0	2	4	0	2	1.0	4	3.2	4.0
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Yellow fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0

Table 2. Notifications of diseases received by state and territory health authorities in the period 1 October to 31 December 2008, by date of diagnosis,* *continued*

Disease	State or territory								Total 4th quarter 2008[†]	Total 3rd quarter 2008	Total 4th quarter 2007	Last 5 years mean 4th quarter	Year to date 2008	Last 5 years YTD mean	Ratio[‡]
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection[¶]	226	3,284	512	3,792	822	351	3,060	2,113	14,160	14,654	12,900	10,296.4	58,403	41,501.2	1.4
Donovanosis	0	0	0	1	0	0	0	0	1	0	0	2.2	2	9.6	0.5
Gonococcal infection	7	312	335	425	124	6	253	374	1,836	1,752	1,798	1,817.6	7,717	7,657.2	1.0
Syphilis (all)	7	306	50	85	9	4	196	51	707	826	774	649.3	3,178	2,608.0	1.1
Syphilis < 2 years duration	0	75	16	35	9	2	73	32	241	311	323	230.8	1,249	921.3	1.0
Syphilis >2 years or unspecified duration	7	231	34	50	NDP	2	123	19	466	515	451	418.5	1,929	1,686.8	1.1
Syphilis - congenital	0	0	1	2	0	0	0	0	3	2	2	2.2	7	12.4	1.4
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	1	2	0	0	2	0	5	4	6	4.6	25	18.0	1.1
Influenza (laboratory confirmed)	35	429	60	383	203	67	192	176	1,545	6,425	712	511.2	9,109	4,775.2	3.0
Measles	0	0	0	0	0	0	0	1	1	5	1	9.4	65	57.0	0.1
Mumps	0	8	13	8	2	0	1	4	36	55	298	89.2	286	256.0	0.4
Pertussis	64	4,424	122	1,017	560	115	571	177	7,050	3,498	1,487	2,151.0	14,118	8,274.0	3.3
Pneumococcal disease (invasive)	6	95	15	60	36	12	80	37	341	633	295	385.8	1,613	1,857.4	0.9
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.2	0.0
Rubella	0	7	0	1	0	0	1	2	11	14	6	6.4	39	42.0	1.7
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.2	0	1.4	0.0
Tetanus	0	0	0	0	0	0	0	0	0	0	1	1.2	4	3.4	0.0
Varicella zoster (chickenpox)	1	0	39	117	217	5	214	148	741	449	519	415.0	1,738	1,612.5	1.8
Varicella zoster (shingles)	2	0	32	106	228	33	165	138	704	465	383	267.0	2,174	1,326.5	2.6
Varicella zoster (unspecified)	27	0	0	922	96	17	120	203	1,385	1,166	1,195	751.3	4,523	3,981.5	1.8
Vectorborne diseases															
Arbovirus infection (NEC)	0	1	0	8	0	0	2	0	11	6	3	7.6	27	40.4	1.4
Barmah Forest virus infection	1	97	20	240	7	0	5	53	423	376	377	297.0	2,112	1,530.0	1.4
Dengue virus infection	2	45	1	125	6	0	3	16	198	93	76	78.8	541	386.6	2.5
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	1	0	0.0	1	0.4	0.0
Kunjin virus infection	0	0	0	0	0	0	0	0	0	1	1	0.8	1	3.6	0.0
Malaria	3	26	8	49	5	3	13	21	128	153	137	142.0	534	657.4	0.9
Murray Valley encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	2	0.8	0.0
Ross River virus infection	3	177	70	347	57	2	32	228	916	784	1,148	684.0	5,641	4,068.4	1.3

Table 2. Notifications of diseases received by State and Territory health authorities in the period 1 October to 31 December 2008, by date of diagnosis,* *continued*

Disease	State or territory								Total 4th quarter 2008†	Total 3rd quarter 2008	Total 4th quarter 2007	Last 5 years mean 4th quarter	Year to date 2008	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.4	0.0
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Brucellosis	0	0	0	12	0	0	0	0	12	15	7	12.0	49	37.6	1.0
Leptospirosis	0	3	1	16	0	0	2	0	22	15	18	22.6	111	136.8	1.0
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	6	0	0	0	0	7	1	14	28	27	43.6	99	172.2	0.3
Q fever	1	42	0	38	0	0	5	3	89	88	105	107.0	357	445.4	0.8
Tularaemia	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Other bacterial infections															
Legionellosis	1	26	0	7	6	0	10	20	70	59	95	89.6	266	326.2	0.8
Leprosy	0	1	0	1	0	0	1	0	3	1	3	1.2	9	8.6	2.5
Meningococcal infection**	0	15	1	24	5	1	16	6	68	115	78	95.8	289	395.6	0.7
Tuberculosis	1	124	5	57	15	2	103	27	334	296	364	323.4	1,211	1,100.4	1.0
Total	613	11,642	1,687	11,245	3,290	874	8,048	5,295	42,693	43,063	36,062	31,520.3	162,278	129,114.6	1.4

* Date of diagnosis = true onset date, or where not available, the earliest of (i) specimen date, (ii) notification date, or (iii) notification receive date. Hepatitis B and C unspecified were analysed by the notification receive date.

† 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.

‡ Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter. Note: Ratios for syphilis <2 years; syphilis >2 years or unspecified duration based on 2 years data

§ Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

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

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens; Northern Territory and Queensland, which exclude ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

** 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.

NDP No data provided.

Table 3. Notification rates of diseases, 1 October to 31 December 2008, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0
Hepatitis B (incident)	0.0	0.6	3.7	0.8	0.3	1.6	1.5	0.4	0.9
Hepatitis B (unspecified)	13.0	28.9	109.8	23.3	22.5	8.9	34.4	30.8	29.0
Hepatitis C (incident)	0.0	0.5	1.9	NN	2.3	4.9	2.9	0.0	1.5
Hepatitis C (unspecified)	68.3	54.9	111.7	65.4	33.1	62.4	43.6	61.2	54.2
Hepatitis D	0.0	0.2	0.0	0.0	0.0	0.0	0.2	0.2	0.1
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	122.4	NN	98.6	125.8	120.9	102.2	108.4	105.4	112.2
Cryptosporidiosis	1.2	4.1	65.1	17.0	2.3	4.9	5.7	8.0	7.9
Haemolytic uraemic syndrome	0.0	0.4	0.0	0.2	0.0	0.0	0.1	0.0	0.2
Hepatitis A	0.0	1.0	0.0	0.8	0.3	0.8	1.0	0.8	0.9
Hepatitis E	0.0	0.3	0.0	0.1	0.0	0.0	0.2	0.2	0.2
Listeriosis	0.0	0.2	0.0	0.3	0.0	0.0	0.0	0.2	0.2
Salmonellosis	58.9	34.0	273.6	55.5	35.6	21.9	27.7	43.1	40.3
Shigellosis	2.4	2.1	81.9	3.0	4.3	0.0	3.6	5.3	3.9
STEC, VTEC‡	0.0	0.3	0.0	1.5	3.3	0.0	0.2	0.0	0.7
Typhoid	0.0	0.8	0.0	0.0	0.3	0.0	0.5	0.4	0.4
Quarantinable diseases									
Cholera	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.4	0.1
Highly pathogenic avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sexually transmissible infections									
Chlamydial infection§	266.1	190.7	952.9	362.7	207.5	284.6	235.2	401.3	269.6
Donovanosis	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	8.2	18.1	623.5	40.7	31.3	4.9	19.4	71.0	35.0
Syphilis (all)	8.1	17.6	90.9	7.9	2.2	3.2	14.8	9.4	13.2
Syphilis <2 years duration	0.0	4.3	29.1	3.3	2.2	1.6	5.5	5.9	4.5
Syphilis >2 years or unspecified duration	8.1	13.3	61.8	4.7	NDP	1.6	9.3	3.5	9.4
Syphilis - congenital	0.0	0.0	1.9	0.2	0.0	0.0	0.0	0.0	0.1
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	0.0	1.9	0.2	0.0	0.0	0.2	0.0	0.1
Influenza (laboratory confirmed)	41.2	24.9	111.7	36.6	51.3	54.3	14.8	33.4	29.4
Measles	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0
Mumps	0.0	0.5	24.2	0.8	0.5	0.0	0.1	0.8	0.7
Pertussis	75.3	256.9	227.1	97.3	141.4	93.2	43.9	33.6	134.2
Pneumococcal disease (invasive)	7.1	5.5	27.9	5.7	9.1	9.7	6.1	7.0	6.5

Table 3. Notification rates of diseases, 1 October to 31 December 2008, by state or territory. (Annualised rate per 100,000 population), *continued*

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Vaccine preventable diseases, *continued*									
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.4	0.0	0.1	0.0	0.0	0.1	0.4	0.2
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	1.2	NN	72.6	11.2	54.8	4.1	16.4	28.1	20.6
Varicella zoster (shingles)	2.4	NN	59.6	10.1	57.6	26.8	12.7	26.2	19.5
Varicella zoster (unspecified)	31.8	NN	0.0	88.2	24.2	13.8	9.2	38.6	38.5
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.1	0.0	0.8	0.0	0.0	0.2	0.0	0.2
Barmah Forest virus infection	1.2	5.6	37.2	23.0	1.8	0.0	0.4	10.1	8.1
Dengue virus infection	2.4	2.6	1.9	12.0	1.5	0.0	0.2	3.0	3.8
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	3.5	1.5	14.9	4.7	1.3	2.4	1.0	4.0	2.4
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ross River virus infection	3.5	10.3	130.3	33.2	14.4	1.6	2.5	43.3	17.4
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australian 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	1.1	0.0	0.0	0.0	0.0	0.2
Leptospirosis	0.0	0.2	1.9	1.5	0.0	0.0	0.2	0.0	0.4
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.3	0.0	0.0	0.0	0.0	0.5	0.2	0.3
Q fever	1.2	2.4	0.0	3.6	0.0	0.0	0.4	0.6	1.7
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	1.2	1.5	0.0	0.7	1.5	0.0	0.8	3.8	1.3
Leprosy	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Meningococcal infection[‖]	0.0	0.9	1.9	2.3	1.3	0.8	1.2	1.1	1.3
Tuberculosis	1.2	7.2	9.3	5.5	3.8	1.6	7.9	5.1	6.4

* Rates are subject to retrospective revision.

† Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

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

§ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens; Northern Territory and Queensland, which exclude ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

|| 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.

NDP No data provided.

Laboratory Serology and Virology Reporting Scheme

There were 6,952 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 October to 31 December 2008 (Tables 4 and 5).

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 October to 31 December 2008, and total reports for the year†

	State or territory								This period 2008	This period 2007	Year to date 2008	Year to date 2007
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	0	0	0	1	0	0	0	1	2	1	32	19
Mumps virus	0	0	0	1	0	0	0	1	2	12	41	53
Rubella virus	0	0	0	1	1	0	0	0	2		14	15
Hepatitis viruses												
Hepatitis A virus	0	1	0	4	0	0	0	1	6	10	50	41
Hepatitis D virus	0	0	0	1	3	0	0	0	4	2	24	22
Hepatitis E virus	0	0	0	1	0	0	0	0	1		9	1
Arboviruses												
Ross River virus	0	4	4	141	31	0	0	2	182	203	1,448	1,090
Barmah Forest virus	0	5	0	85	4	0	1	0	95	97	578	506
Flavivirus (unspecified)	0	10	0	39	0	0	0	0	49	16	104	97
Adenoviruses												
Adenovirus type 2	0	1	0	0	0	0	0	0	1		1	
Adenovirus type 3	0	1	0	0	0	0	0	0	1		1	
Adenovirus type 8	0	1	0	0	0	0	0	0	1		1	
Adenovirus not typed/ pending	1	97	0	248	67	2	10	0	425	309	1,724	1,202
Herpesviruses												
Herpes virus type 6	0	0	0	0	0	0	2	0	2		3	2
Cytomegalovirus	1	48	0	147	27	2	1	0	226	253	1,196	1,198
Varicella-zoster virus	4	82	0	561	76	3	6	1	733	684	2,928	2,809
Epstein-Barr virus	0	18	19	378	96	0	2	66	579	499	2,416	2,464
Other DNA viruses												
Contagious pustular dermatitis (orf virus)	0	2	0	0	0	0	0	0	2		2	
Parvovirus	0	3	0	73	8	0	5	1	90	124	297	413
Picornavirus family												
Coxsackievirus A9	1	9	0	0	0	0	0	0	10	2	14	2
Coxsackievirus A16	0	1	0	0	0	0	0	0	1		5	
Echovirus type 6	0	1	0	0	0	0	0	0	1	5	3	16
Echovirus type 30	0	2	0	0	0	0	0	0	2	1	2	4
Poliovirus type 1 (uncharacterised)	0	2	0	0	0	0	0	0	2		2	
Poliovirus type 2 (uncharacterised)	0	1	0	0	0	0	0	0	1		1	
Rhinovirus (all types)	0	62	0	0	1	0	0	0	63	80	201	326
Enterovirus type 71 (BCR)	0	1	0	0	0	0	0	0	1		1	
Enterovirus not typed/ pending	0	10	0	12	0	1	1	0	24	52	146	180
Picornavirus not typed	0	0	0	0	0	3	0	0	3	2	12	9

Table 4. Virology and serology laboratory reports by state or territory* for the reporting period 1 October to 31 December 2008, and total reports for the year,† *continued*

	State or territory								This period 2008	This period 2007	Year to date 2008	Year to date 2007
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Ortho/paramyxoviruses												
Influenza A virus	10	55	1	74	15	0	12	1	168	66	813	2,293
Influenza B virus	4	13	0	32	10	0	8	0	67	34	935	165
Parainfluenza virus type 1	0	0	0	0	1	0	0	0	1	11	182	50
Parainfluenza virus type 2	0	1	0	0	1	0	0	0	2	2	27	61
Parainfluenza virus type 3	2	96	0	66	14	1	13	2	194	134	392	507
Respiratory syncytial virus	0	48	0	48	19	3	7	0	125	175	1,916	2,238
Other RNA viruses												
HTLV-1	0	0	0	0	25	0	0	0	25	2	67	14
Rotavirus	0	13	0	0	12	7	3	0	35	253	310	619
Norwalk agent	1	14	0	0	1	0	0	0	16	371	90	1,138
Other												
Chlamydia trachomatis not typed	6	309	1	1,440	153	20	11	1	1,941	1,515	8,533	7,841
Chlamydia psittaci	0	2	0	0	0	0	11	0	13	27	92	66
Mycoplasma pneumoniae	0	15	0	152	19	6	41	10	243	167	972	1,156
Coxiella burnetii (Q fever)	0	1	0	21	5	0	8	0	35	96	240	279
Orientia tsutsuganushi	0	0	0	0	2	0	0	0	2	2	10	10
Rickettsia - spotted fever group	0	3	0	8	0	0	0	0	11	16	111	114
Streptococcus group A	0	10	0	237	0	0	0	0	247	281	1,015	1,106
Brucella species	0	0	0	4	0	0	0	0	4	3	32	10
Bordetella pertussis	0	372	1	323	186	4	0	0	886	163	1,924	829
Legionella pneumophila	0	2	0	0	0	0	6	0	8	2	20	30
Legionella longbeachae	0	0	0	0	1	0	0	0	1	2	10	8
Legionella species	0	0	0	1	0	0	1	0	2		3	3
Cryptococcus species	0	2	0	2	2	0	0	0	6	3	30	45
Leptospira species	0	0	0	12	1	0	0	0	13	9	83	61
Treponema pallidum	0	54	0	275	51	1	2	0	383	411	2,012	2,196
Entamoeba histolytica	0	0	0	1	0	0	0	0	1	2	9	8
Toxoplasma gondii	0	1	0	4	0	0	1	0	6	8	18	29
Echinococcus granulosus	0	0	0	0	5	0	1	0	6	8	34	24
Total	30	1,373	26	4,393	837	53	153	87	6,952	6,115	31,136	31,369

* State or territory of postcode, if reported, otherwise state or territory of reporting laboratory.

† Data presented are for reports with reports dates in the current period.

– No data received this period.

Table 5. Virology and serology reports by laboratories for the reporting period 1 October to 31 December 2008*

State or territory	Laboratory	October 2008	November 2008	December 2008	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	190	228	241	659
	New Children's Hospital, Westmead	98	85	57	240
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	44	50	31	125
	South West Area Pathology Service, Liverpool	34	20	15	69
Queensland	Queensland Medical Laboratory, West End	1,775	1,476	1,496	4,747
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	–	67	763	830
Tasmania	Northern Tasmanian Pathology Service, Launceston	15	20	17	52
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Australian Rickettsial Reference Laboratory	–	–	–	–
	Monash Medical Centre, Melbourne	25	7	6	38
	Royal Children's Hospital, Melbourne	–	–	–	–
	Victorian Infectious Diseases Reference Laboratory, Fairfield	73	17	–	90
Western Australia	PathWest Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	33	44	25	102
Total		2,287	2,014	2,651	6,952

* The complete list of laboratories reporting for the 12 months, January to December 2008, will appear in every report regardless of whether reports were received in this reporting period. Reports are not always received from all laboratories.

– No data received this period.

Additional reports

Australian Sentinel Practice Research Network

The Australian Sentinel Practices Research Network (ASPREN) is a national surveillance system that is 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. Electronic, web-based data collection was established in 2006.

The list of conditions is reviewed annually by the ASPREN management committee. In 2009, 4 conditions are being monitored. They include influenza like illness (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 *2009;33:83–84.*

Reporting period 1 October to 31 December 2008

Sentinel practices contributing to ASPREN were located in all jurisdictions other than the Northern Territory. A total of 104 general practitioners contributed data to ASPREN in the 4th quarter of 2008. Each week an average of 75 general practitioners provided information to ASPREN at an average of 6,955 (range 3,560 to 7,870) consultations per week.

ILI rates reported from 1 October to 31 December 2008 were lower (4–11 cases per 1,000 consultations) compared with the same reporting period in 2007 (4–14 cases per 1000 consultations. The ILI rates reported to ASPREN decreased more rapidly from the season's peak in 2008 than the decline in rates from the 2007 peak (Figure 1).

Reports of gastroenteritis from 1 October to 31 December 2008 were lower compared with the same period in 2007 (Figure 2). During this reporting period, consultation rates for gastroenteritis ranged from 1 to 8 cases per 1,000 consultations.

Figure 1. Consultation rates for influenza-like illness, ASPREN, 1 January 2007 to 31 December 2008, by week of report

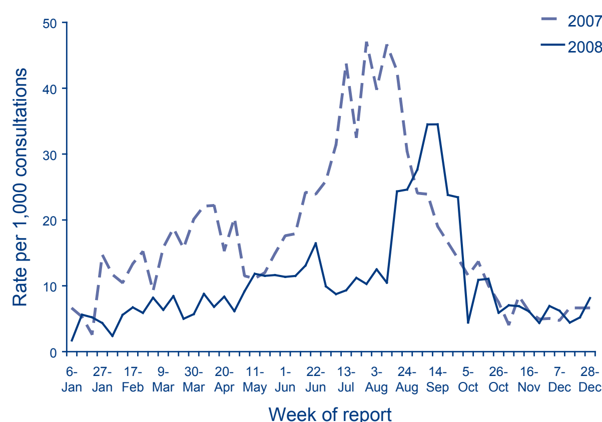
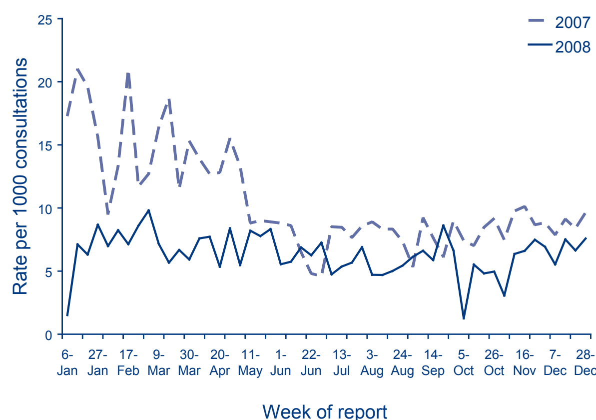


Figure 2. Consultation rates for gastroenteritis, ASPREN, 1 January 2007 to 31 December 2008, by week of report



Reports of varicella infections were reported at a similar rate for the 4th quarter of 2008 compared with the same period in 2007. From 1 October to 31 December 2008, recorded rates for chickenpox were between zero and 1 cases per 1,000 consultations (Figure 3).

In the 4th quarter of 2008, reported rates for shingles were between less than one to 1 case per 1,000 consultations (Figure 4).

Figure 3. Consultation rates for chickenpox, ASPREN, 1 January 2007 to 31 December 2008, by week of report

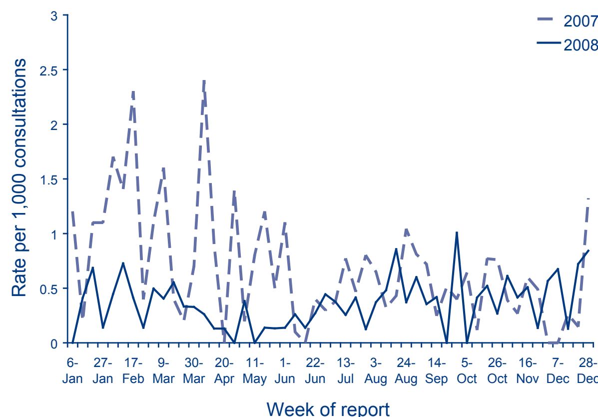
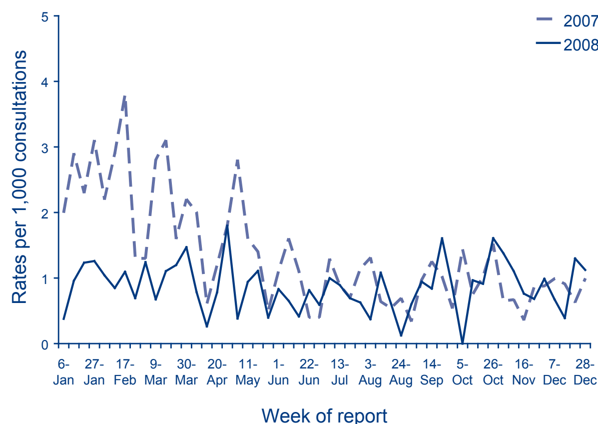


Figure 4. Consultation rates for shingles, ASPREN, 1 January 2007 to 31 December 2008, by week of report



Australian childhood immunisation coverage

Tables 1, 2 and 3 provide the latest 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 of age for the cohort born between 1 July and 30 September 2007, at 24 months of age for the cohort born between 1 July and 30 September 2006, and at 5 years of age for the cohort born between 1 July and 30 September 2002 according to the National Immunisation Program Schedule. However from March 2002 to December 2007, coverage for vaccines due at 4 years of age was assessed at the 6-year milestone age.

For information about the Australian Childhood Immunisation Register see Surveillance systems reported in CDI, *published in* Commun Dis Intell *2008;32:134–135 and for a full description of the methodology used by the Register see* Commun Dis Intell *1998;22:36-37.*

Commentary on the trends in ACIR data is provided by the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases (NCIRS). For further information please contact the NCIRS at telephone: +61 2 9845 1435, Email: brynleyh@chw.edu.au

'Fully immunised' at 12 months of age is defined as a child having a record on the ACIR of 3 doses of a diphtheria (D), tetanus (T) and pertussis-containing (P) vaccine, 3 doses of polio vaccine, 2 or 3 doses of *Haemophilus influenzae* type b (Hib) vaccine, and 2 or 3 doses of hepatitis B vaccine. '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 Hib vaccine, 2 or 3 doses of hepatitis B vaccine and 1 dose of a measles, mumps and rubella (MMR)-containing vaccine. 'Fully immunised' at 5 years 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.

Immunisation coverage for children 'fully immunised' at 12 months of age for Australia increased slightly by 0.1 of a percentage point to 91.3% (Table 1). There were no important changes in coverage for any individual vaccines due at 12 months of age or by jurisdiction.

Immunisation coverage for children 'fully immunised' at 24 months of age for Australia increased by 0.2 of a percentage point to 92.7 (Table 2). There were no important changes in coverage for any individual vaccines due at 24 months of age or by jurisdiction.

Immunisation coverage for 'fully immunised' at 5 years of age for Australia increased for the first time in 3 quarters, by 1.5 percentage points, to 88.3% (Table 3). This increase nationally was driven by significant increases in coverage for all individual vaccines due at 4 years of age in the Northern Territory (5.4 percentage points), Western Australia (3 percentage points) and Queensland (2.2 percentage points). There are a couple of possible explanations for the significant increases in these jurisdictions. The Health Kids Check initiative, implemented nationally in July 2008, may have had a disproportionately greater effect in these 3 jurisdictions. Further, various jurisdictional-specific strategies and local efforts including data quality improvements through data cleaning may also have had an effect.

Table 1. Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2007; assessment date 31 December 2008

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,202	24,663	924	15,794	4,965	1,691	18,144	7,589	74,972
Diphtheria, tetanus, pertussis (%)	93.9	91.7	90.5	91.3	92.4	92.3	92.6	90.6	91.8
Poliomyelitis (%)	93.8	91.6	90.6	91.2	92.4	92.3	92.6	90.6	91.7
Haemophilus influenzae type b (%)	95.9	94.6	93.3	93.7	94.9	94.6	94.9	93.9	94.4
Hepatitis B (%)	95.8	94.6	93.8	93.7	94.8	94.6	94.8	93.8	94.4
Fully immunised (%)	93.7	91.4	90.3	90.8	91.8	92.0	91.8	89.9	91.3
Change in fully immunised since last quarter (%)	+0.2	-0.1	+0.5	+0.1	+0.5	+0.4	+0.2	-0.1	+0.1

Table 2. Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2006; assessment date 31 December 2008*

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,213	24,494	856	15,064	4,865	1,721	18,171	7,691	74,075
Diphtheria, tetanus, pertussis (%)	96.1	94.9	95.6	94.3	94.8	96.2	95.7	92.9	94.8
Poliomyelitis (%)	96.2	94.8	95.4	94.3	94.8	96.2	95.7	92.9	94.8
Haemophilus influenzae type b (%)	96.1	95.2	93.6	93.4	93.7	95.9	94.7	92.9	94.4
Measles, mumps, rubella (%)	95.3	93.9	95.2	93.4	93.9	95.6	94.9	92.0	93.9
Hepatitis B (%)	96.5	95.7	96.6	95.0	95.5	96.9	96.4	94.0	95.6
Fully immunised (%)	94.5	92.7	93.0	92.2	92.7	94.7	93.8	89.9	92.7
Change in fully immunised since last quarter (%)	-0.4	+0.3	-0.6	+0.3	+0.3	+1.2	+0.5	-1.3	+0.2

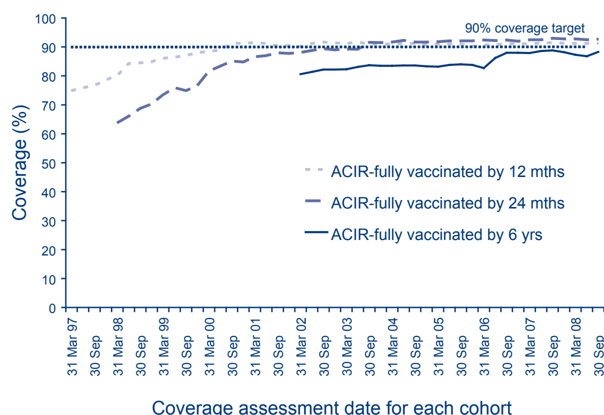
* The 12 months age data for this cohort was published in *Commun Dis Intell* 2008;32:122.

Table 3. Percentage of children immunised at 5 years of age, preliminary results by disease and state or territory for the birth cohort 1 July to 30 September 2003; assessment date 31 December 2008

Vaccine	State or territory								Australia
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,106	23,056	815	14,277	4,807	1,494	16,903	6,706	69,164
Diphtheria, tetanus, pertussis (%)	90.4	88.0	92.5	89.4	85.9	88.0	91.7	87.3	89.0
Poliomyelitis (%)	90.6	87.9	92.4	89.3	85.9	87.9	91.6	87.2	89.0
Measles, mumps, rubella (%)	90.6	87.7	91.9	89.1	85.8	87.7	91.3	87.0	88.8
Fully immunised (%)	90.0	87.2	91.8	88.7	85.5	87.2	90.9	86.4	88.3
Change in fully immunised since last quarter (%)	-0.6	+1.5	+5.3	+2.2	-0.2	-2.0	+1.0	+3.0	+1.5

Figure 5 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 5 years, although the rate of increase has slowed over the past few years for all age groups. It should also be noted that currently, coverage for the vaccines added to the National Immunisation Program since 2003 (varicella at 18 months, meningococcal C conjugate at 12 months, and rotavirus and pneumococcal conjugate at 2, 4, and 6 months) are not included in the 12 or 24 months coverage data, respectively.

Figure 5. Trends in vaccination coverage, Australia, 1997 to 30 September 2008, by age cohorts



Gonococcal surveillance

John Tapsall, The Prince of Wales Hospital, Randwick NSW 2031 for the Australian Gonococcal Surveillance Programme.

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various states and territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] *Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and 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. For more information see* Commun Dis Intell *2008;32:134.*

Reporting period 1 July to 30 September 2008

The AGSP laboratories received a total of 746 gonococcal isolates of which 727 remained viable for susceptibility testing. This was about 10% more than the 651 gonococci reported for the same period in 2007. About 29% of this total was from New South Wales, 18% from Queensland, 16% from each of Victoria and the Northern Territory, 13% from Western Australia and 8% from South Australia. There was 1 isolate from the Australian Capital Territory and no isolates from Tasmania.

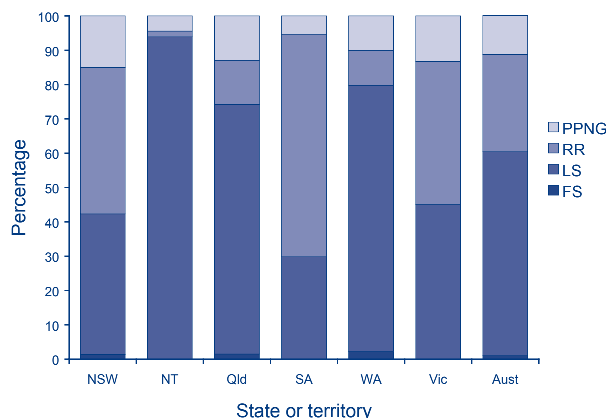
Penicillins

Two hundred and eighty-eight (39.6%) of the 727 isolates examined were penicillin resistant by one or more mechanisms. Eighty-two (11.3%) were penicillinase producing *Neisseria gonorrhoeae* (PPNG) and 206 (28%) resistant by chromosomal mechanisms, (CMRP). The proportion of all strains resistant to the penicillins by any mechanism ranged from 11.7% in the Northern Territory to 70.2% in South Australia. High rates of penicillin resistance were also found in New South Wales (58%), Victoria (55%), Queensland (25.8%) and in Western Australia (20.2%). The 1 gonococcus isolated in the Australian Capital Territory, was not penicillin resistant.

Figure 6 shows the proportions of gonococci fully sensitive (MIC ≥ 0.03 mg/L), less sensitive (MIC 0.06–0.5 mg/L), relatively resistant (MIC ≥ 1 mg/L) or else penicillinase producing, aggregated for Australia and by state and territory. A high proportion those strains classified as PPNG or else resistant by chromosomal mechanisms fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

New South Wales had the highest number of the penicillin resistance with 91 CMRP (42.7%) and 32 PPNG (15%). Victoria followed with 16 PPNG (13.3%) and 50 CMRP (42%). In Queensland CMRP and PPNG were 12.9% each. In South Australia PPNG were 5.3% and CMRP a record high at 64.9%. In Western Australia PPNG and CMRP were 10.1% each. CMRP and PPNG were also present in the Northern Territory (2 and 5 isolates, respectively), but there were no CMRP or PPNG in the Australian Capital Territory or Tasmania. All the penicillin resistant strains in the Northern Territory were from Darwin.

Figure 6. Categorisation of gonococci isolated in Australia, 1 January to 30 September 2008, by penicillin susceptibility and region



FS Fully sensitive to penicillin, MIC ≤0.03 mg/L.

LS Less sensitive to penicillin, MIC 0.06–0.5 mg/L.

RR Relatively resistant to penicillin, MIC ≥1 mg/L.

PPNG Penicillinase producing *Neisseria gonorrhoeae.*

Ceftriaxone

Seven isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected; four in New South Wales, two in Queensland and one in South Australia. It is emphasised that no treatment failures have been documented locally when a 250 mg IM dose of ceftriaxone has been used.

Spectinomycin

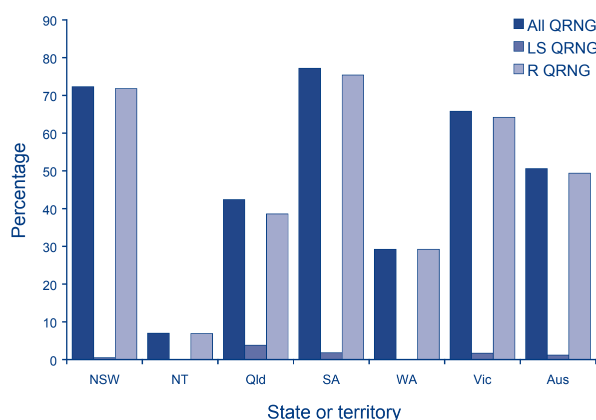
All isolates were susceptible to this injectable agent.

Quinolone antibiotics

Nationally, the 368 quinolone resistant *N. gonorrhoeae* (QRNG) detected in this quarter represented 50.6% of all isolates tested. In the 3rd quarter of 2007, the 321 QRNG also represented 50.5% of all isolates, in 2006 there were 38.0% QRNG and in the 3rd quarter of 2005 QRNG were 35.5% of all gonococci tested. The majority of QRNG (319 of 368, 86.7%) had higher level resistance to ciprofloxacin of 1 mg/L or more. QRNG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≤1 mg/L) groups.

QRNG were detected in all states and territories with the exception of Tasmania where no gonococci were isolated. The highest proportion of QRNG was found in South Australia where 44 QRNG represented 77.2% of isolates tested, while in New South Wales there were 154 QRNG (72.3%) (Figure 7). In Victoria there were 79 QRNG (65.8% of isolates). In the other states and territories, Queensland had 56 (42.4%) QRNG; Western Australia had 26 (29.2%) and the Northern Territory had 8 (7%) QRNG. The single isolate tested in the Australian Capital Territory was QRNG.

Figure 7. The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 January to 30 September 2008, by jurisdiction



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.

R QRNG Ciprofloxacin MICs ≥1 mg/L.

High level tetracycline resistance

The number (128) and proportion (17.6%) of high level tetracycline resistance (TRNG) detected was slightly lower than that recorded in this quarter in 2007 (129, 20.3%). TRNG were found in all states and territories except for Tasmania and the Australian Capital Territory and represented between 12.9% (Queensland); 13.9% (Northern Territory) and 30.3% (Western Australia) of all isolates tested.

Reference

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

National Enteric Pathogens Surveillance System

The National Enteric Pathogens Surveillance System (NEPSS) collects, analyses and disseminates data on human enteric bacterial infections diagnosed in Australia. Communicable Diseases Intelligence *NEPSS quarterly reports include only* Salmonella. *NEPSS receives reports of* Salmonella *isolates that have been serotyped and phage typed by the 5* Salmonella *typing laboratories in Australia.* Salmonella *isolates are submitted to these laboratories for typing by primary diagnostic laboratories throughout Australia.*

A case is defined as the isolation of a Salmonella *from an Australian resident, either acquired locally or as a result of overseas travel, including isolates detected during immigrant and refugee screening. Second and subsequent identical isolates from an individual within 6 months are excluded, as are isolates from overseas visitors to Australia. The date of the case is the date the primary diagnostic laboratory isolated* Salmonella *from the clinical sample.*

Quarterly reports include historical quarterly mean counts. These should be interpreted cautiously as they may be affected by outbreaks and by surveillance artefacts such as newly recognised and incompletely typed Salmonella.

NEPSS may be contacted at the Microbiological Diagnostic Unit, Public Health Laboratory, Department of Microbiology and Immunology, The University of Melbourne; by telephone: +61 3 8344 5701, facsimile: +61 3 8344 7833 or email joanp@unimelb.edu.au

Scientists, diagnostic and reference laboratories contribute data to NEPSS, which is supported by state and territory health departments and the Australian Government Department of Health and Ageing.

Reports to the National Enteric Pathogens Surveillance System of Salmonella *infection for the period 1 October to 31 December 2008 are included in Tables 4 and 5. Data include cases reported and entered by 4 February 2009. Counts are preliminary, and subject to adjustment after completion of typing and reporting of further cases to NEPSS. For more information see* Commun Dis Intell *2008;32:137.*

1 October to 31 December 2008

There were 1,682 reports to NEPSS of human *Salmonella* infection in the 4th quarter of 2008, approximately 65% more than in the preceding quarter. Limited data from Western Australia were available at the time of preparing this report. Taking this into account and some incompleteness of data from late 2008, the overall count of cases for the remainder of Australia was similar to the recent historical mean number of reports to NEPSS for this time of each year. The incidence of human salmonellosis in Australia typically begins to increase late each year, before peaking around March.

During the 4th quarter of 2008, the 25 most common *Salmonella* types in Australia accounted for 1,095 cases, 65% of all reported human *Salmonella* infections. Eighteen of the 25 most common *Salmonella* infections in the 4th quarter of 2008 were also among those most commonly reported in the preceding quarter.

S. Typhimurium phage type 170 was the most commonly reported *Salmonella* for the quarter, with counts in New South Wales and Victoria significantly greater than the recent historical mean. Increases in the following salmonellae were also evident: *S.* Typhimurium phage type 44 (Australian Capital Territory), *S.* Typhimurium phage type 126 (in New South Wales and Victoria), *S.* Newport (mostly eastern states) and *S.* Typhimurium phage type 29 (New South Wales and South Australia). The number of reports of *S.* Enteritidis phage type 6a was markedly elevated this quarter. This particular *Salmonella* infection is typically associated with travel to Bali or Thailand.

Acknowledgement: We thank scientists, contributing laboratories, state and territory health departments, and the Australian Government Department of Health and Ageing for their contributions to NEPSS.

Table 4. Reports to the National Enteric Pathogens Surveillance System of *Salmonella* isolated from humans during the period 1 October to 31 December 2008, as reported to 4 February 2009

	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA*	Australia
Total all *Salmonella* for quarter	45	513	107	480	136	28	350	23	1,682
Total contributing *Salmonella* types	18	116	37	107	56	14	85	15	224

* Limited data from Western Australia were available at the time of preparing this report.

Table 5. Top 25 *Salmonella* types identified in Australia, 1 October to 31 December 2008, by state or territory

National rank	*Salmonella* type	State or territory								Total 4th quarter 2008	Last 10 years mean 4th quarter	Year to date 2008	Year to date 2007
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA*				
1	*S.* Typhimurium PT 170	2	126	0	18	0	0	53	0	199	79	430	290
2	*S.* Typhimurium PT 135	2	27	0	49	0	2	48	2	130	165	858	682
3	*S.* Typhimurium PT 9	4	42	0	15	20	1	29	0	111	101	479	688
4	*S.* Saintpaul	0	14	13	42	3	0	8	0	80	98	271	375
5	*S.* Typhimurium PT 44	20	18	0	9	8	0	19	0	74	61	360	481
6	*S.* Birkenhead	0	22	0	37	0	0	0	0	59	63	204	232
7	*S.* Typhimurium PT 126	1	31	0	0	1	0	9	0	42	25	134	40
8	*S.* Chester	0	5	5	18	5	0	3	2	38	38	151	161
9	*S.* Enteritidis PT 6a	0	12	0	8	2	0	15	1	38	10	89	69
10	*S.* Virchow PT 8	0	3	8	23	1	0	0	0	35	60	183	258
11	*S.* Newport	1	9	0	8	2	1	9	0	30	12	71	75
12	*S.* Infantis	1	4	4	1	6	0	7	0	23	33	167	200
13	*S.* Typhimurium PT 197	1	6	1	9	2	0	3	0	22	34	107	199
14	*S.* Stanley	1	6	0	4	1	2	8	0	22	20	112	136
15	*S.* Muenchen	0	7	2	11	0	0	1	0	21	30	97	144
16	*S.* Montevideo	0	7	0	5	1	0	7	0	20	14	88	113
17	*S.* Aberdeen	0	0	1	17	0	0	0	0	18	23	85	145
18	*S.* Weltevreden	0	6	0	5	1	0	6	0	18	13	90	67
19	*S.* Waycross	0	7	0	9	1	0	0	0	17	20	89	101
20	*S.* Typhimurium untypable	0	4	0	2	4	0	4	3	17	15	86	94
21	*S.* subsp I ser 16:l,v:-	0	1	10	3	2	0	1	0	17	13	54	56
22	*S.* Typhimurium PT 135a	0	0	4	0	13	0	0	0	17	10	60	70
23	*S.* Hvittingfoss	0	1	0	15	0	0	0	0	16	20	74	115
24	*S.* Typhimurium PT 29	0	8	1	0	7	0	0	0	16	4	80	156
25	*S.* Anatum	1	3	0	5	0	0	5	1	15	20	78	77

* Limited data from Western Australia were available at the time of preparing this report.

Administration

Surveillance systems reported in *CDI*, 2009

This article describes the surveillance schemes that are routinely reported on in *Communicable Diseases Intelligence* (*CDI*).

Communicable disease surveillance in Australia operates at the national, state and local levels. Primary responsibility for public health action lies with the state and territory health departments. The role of communicable disease surveillance at a national level includes:

- detecting outbreaks and identifying national trends;
- guidance for policy development and resource allocation at a national level;
- monitoring the need for and impact of national disease control programs;
- coordination of response to national or multi-jurisdictional outbreaks;
- description of the epidemiology of rare diseases, that occur infrequently at state and territory levels;
- meeting various international reporting requirements, such as providing disease statistics to the World Health Organization; and
- support for quarantine activities, which are the responsibility of the national government.

State and territory health departments collect notifications of communicable diseases under their public health legislation. In September 2007, the *National Health Security Act 2007* (*National Health Security Act,* No 174) received royal assent.[1] This Act provides a legislative basis for and authorises the exchange of health information, including personal information, between jurisdictions and the Commonwealth. The Act provides for the establishment of the *National Health Security (National Notifiable Disease List) Instrument 2008*,[2] which specifies the diseases about which personal information can be provided. The *National Health Security Agreement,*[3] which was drafted in 2007 and signed by Health Ministers in April 2008, establishes operational arrangements to formalise and enhance existing surveillance and reporting systems, an important objective of the Act. States and territories voluntarily forward de-identified data on a nationally agreed set of communicable diseases to the Department of Health and Ageing for the purposes of national communicable disease surveillance.

Surveillance has been defined by the World Health Organization as the 'continuing scrutiny of all aspects of the occurrence and spread of disease that are pertinent to effective control.' It is characterised by 'methods distinguished by their practicability, uniformity, and frequently by their rapidity, rather than complete accuracy.'[3] Although some surveillance schemes aim for complete case ascertainment, others include only a proportion of all cases of the conditions under surveillance, and these samples are subject to systematic and other biases. Results generated from surveillance schemes must be interpreted with caution, particularly when comparing results between schemes, between different geographical areas or jurisdictions and over time. Surveillance data may also differ from data on communicable diseases gathered in other settings.

The major features of the surveillance schemes for which *CDI* publishes regular reports are described below.

Other surveillance schemes for which *CDI* publishes annual reports include tuberculosis notifications (*Commun Dis Intell* 2008;32:1–11), the Australian Mycobacterium Reference Laboratory Network (*Commun Dis Intell* 2008;32:12–17), invasive pneumococcal disease surveillance (*Commun Dis Intell* 2008;32:18–30), the National Arbovirus and Malaria Advisory Committee (*Commun Dis Intell* 2008;32:31–47), and the Australian Rotavirus Surveillance Program (*Commun Dis Intell* 2008;32:425–429).

National Notifiable Diseases Surveillance System

National compilations of notifiable diseases have been published intermittently in a number of publications since 1917.[5] The National Notifiable Diseases Surveillance System (NNDSS) was established in 1990 under the auspices of the Communicable Diseases Network Australia (CDNA).

Sixty-five communicable diseases agreed upon nationally are reported to NNDSS, although not all 65 are notifiable in each jurisdiction. Data are sent electronically from states and territories daily or several times a week. The system is complemented by other surveillance systems, which provide information on various diseases, including four that are not reported to NNDSS (AIDS, HIV, and the classical and variant forms of Creutzfeldt-Jakob disease).

The NNDSS core dataset includes data fields for a unique record reference number; notifying state or territory; disease code; age; sex; Indigenous status; postcode of residence; date of onset of the disease; death, date of report to the state or territory health department and outbreak reference (to identify cases linked to an outbreak). Where relevant, information on the species, serogroups/subtypes and phage types of organisms isolated, and on the vaccination status of the case is collected. Data quality is monitored by DoHA and the National Surveillance Committee (NSC) and there is a continual process of improving the national consistency of communicable disease surveillance.

While not included in the core national dataset, enhanced surveillance information for some diseases (hepatitis B (newly acquired), hepatitis C (newly acquired, invasive pneumococcal disease and tuberculosis) is obtained from states and territories.

Aggregated data are presented on the department's Internet site under *Communicable Diseases Surveillance* and updated daily (www.health.gov.au/nndssdata). A summary report and data table are also published on the Internet each fortnight (www.health.gov.au/cdnareport).

Data are published in *CDI* every quarter and in an annual report. The reports include numbers of notifications for each disease by state and territory, and totals for Australia for the current period, the year to date, and for the corresponding period of the previous year. The national total for each disease is compared with the average number of notifications over the previous 5 years in the same period. A commentary on the notification data is included with the tables in each issue of *CDI* and graphs are used to illustrate important aspects of the data.

Australian Childhood Immunisation Register

Accurate information on the immunisation status of children is needed at the community level for program management and targeted immunisation efforts. A population-based immunisation register can provide this need. The Australian Childhood Immunisation Register (ACIR) commenced operation on 1 January 1996 and is now an important component of the *Immunise Australia Program*. It is administered and operated by Medicare Australia (formerly the Health Insurance Commission). The Register was established by transferring data on all children under the age of 7 years enrolled with Medicare to the ACIR. This constitutes a nearly complete population register, as approximately 99% of children are registered with Medicare by 12 months of age. Children who are not enrolled in Medicare are added to the Register when a recognised immunisation provider supplies details of an eligible immunisation. Immunisations are generally notified to Medicare Australia either by electronic means, the Internet or by paper ACIR notification forms. Immunisations recorded on the Register must have been given in accordance with the guidelines for immunisation determined by the National Health and Medical Research Council.

From the data finally entered onto the ACIR, Medicare Australia provides regular quarterly coverage reports at the national and state level. Coverage for these reports is calculated using the cohort method described in *Commun Dis Intell* 1998;22:36–37. With this method, a cohort of children is defined by date of birth in 3-month groups. This birth cohort has the immunisation status of its members assessed at the 3 key milestones of 12 months, 24 months and 6 years of age. Analysis of coverage is undertaken 3 months after the due date for completion of each milestone, so that time is available for processing notifications and the impact on coverage estimates of delayed notification to the ACIR is minimised. Only children enrolled with Medicare are included in order to minimise inaccuracies in coverage estimates due to duplicate records.

Medicare Australia coverage reports for the 3 milestones are published in *CDI* each quarter. Coverage estimates are provided for each state and territory and Australia as a whole and for each individual vaccine assessed at each milestone. Changes in 'fully immunised' coverage from the previous quarter are also included in the tables.

A commentary on ACIR immunisation coverage estimates is included with the tables in each issue and graphs are used to provide trends in immunisation coverage.

Australian Gonococcal Surveillance Programme

The Australian Gonococcal Surveillance Programme (AGSP) is a continuing program to monitor antimicrobial resistance in *Neisseria gonorrhoeae* and includes the reference laboratories in all states and territories. These laboratories report data on sensitivity to an agreed core group of antimicrobial agents on a quarterly basis and provide an expanded analysis as an annual report in *CDI* (*Commun Dis Intell* 2008;32:227–231). The antibiotics that are currently routinely surveyed are the penicillins, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens. One main purpose of the AGSP is to help define standard protocols for antibiotic treatment of gonococcal infection. When *in vitro* resistance to a recommended agent is demonstrated in 5% or more of isolates, it is usual to reconsider the inclusion of that agent in current treatment schedules. Additional

data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level resistance to the tetracyclines and intermittent surveys of azithromycin resistance are conducted. Comparability of data is achieved by means of a standardised system of MIC testing and a program-specific quality assurance process.

Australian Meningococcal Surveillance Programme

The reference laboratories of the Australian Meningococcal Surveillance Programme report data of laboratory-confirmed cases confirmed either by culture or by non-culture techniques. Culture-positive cases where a *Neisseria meningitidis* is grown from a normally sterile site or skin, 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 are reported annually and quarterly in *CDI*. Data in the quarterly reports are restricted to a description of the number of cases per jurisdiction, and serogroup where known. A full analysis of laboratory-confirmed cases of IMD, including phenotyping and antibiotic susceptibility data are published annually (*Commun Dis Intell* 2009;33:1–9).

Australian Paediatric Surveillance Unit

The Australian Paediatric Surveillance Unit (APSU) is an active surveillance mechanism for prospective, national identification and study of children (<15 years) with uncommon conditions of childhood, including rare infectious and vaccine preventable diseases, genetic disorders, child mental health problems, and rare injuries. Each month the APSU sends an e-mail or paper report card to approximately 1,300 paediatricians and other child health clinicians. Clinicians are asked to indicate whether or not they have seen a child newly diagnosed with a condition listed, and clinicians reporting cases are asked to provide details about demographics, diagnosis, treatments and short-term outcomes. The report card return rate has been maintained at over 90% over the last 15 years.

Communicable diseases currently under surveillance include: acute flaccid paralysis (to identify potential cases of poliovirus infection); congenital cytomegalovirus infection; congenital rubella; perinatal exposure to HIV, HIV infection and AIDS; neonatal herpes simplex virus infection; neonatal varicella, congenital varicella, severe complications of varicella, intussusception and its causes (e.g. rotavirus infection), and acute rheumatic fever (group A *Streptococcus* infection). APSU is occasionally used for short-term rapid response surveillance e.g. severe complications of influenza during the 2007 and 2008 influenza season.

APSU is a unit of the Royal Australasian College of Physicians, and its activities are supported by the Department of Health and Ageing; the Faculty of Medicine, University of Sydney; and the National Health and Medical Research Council Enabling Grant 402784. For further information please contact the APSU Director, Professor Elizabeth Elliott on telephone: +61 2 9845 3005, facsimile +61 2 9845 3082 or email: apsu@chw.edu.au

Australian National Creutzfeldt-Jakob Disease Registry

The surveillance for CJD in Australia is conducted through the Australian National Creutzfeldt-Jakob Disease Registry (ANCJDR). CJD has been scheduled as a notifiable disease in a number of Australian states and territories. The ANCJDR is under contract to the Commonwealth to determine all suspect cases of transmissible spongiform encephalopathies (TSE) in Australia. An annual update is published in *CDI (Commun Dis Intell* 2008;32:232–236).

Australian Sentinel Practice Research Network

The Royal Australian College of General Practitioners and the Department of General Practice at the University of Adelaide operate the Australian Sentinel Practice Research Network (ASPREN). ASPREN is a national network of general practitioners who report presentations of defined medical conditions each week. The aim of ASPREN is to provide an indicator of the burden of disease in the primary health care setting and to detect trends in consultation rates.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2009, 4 conditions are being monitored; all of which are related to communicable diseases. These include influenza like illness, gastroenteritis, chickenpox and shingles.

There are currently 96 general practitioners participating in the network from all jurisdictions other than the Northern Territory. Sixty-eight per cent of these are in metropolitan areas, 26% in rural and 14% in remote areas of Australia. Approximately 6,000 consultations are recorded each week.

Data for communicable diseases are published in *CDI* every quarter. Data are presented in graphic format as the rate of reporting per 1,000 consultations per week. The conditions are defined as follows:

Influenza-like illness – record once only per patient

Must have the following: fever, cough and fatigue

Gastroenteritis – record once only per patient

Three or more loose stools, and/or 2 vomits in a 24 hour period excluding cases who have a known cause, for example bowel disease, alcohol, pregnancy.

Chickenpox – record once only per patient

An acute, generalised viral disease with a sudden onset of slight fever, mild constitutional symptoms and a skin eruption which is maculopapular for a few hours, vesicular for three to 4 days and leaves a granular scab.

Shingles – record once only per patient

Recurrence, recrudescence or re-activation of chickenpox infection. Vesicles with any erythematous base restricted to skin areas supplied by sensory nerves of a single or associated group of dorsal root ganglia. Lesions may appear in crops in irregular fashion along nerve pathways, are usually unilateral, deeper seated and more closely aggregated than those of chickenpox.

Note: Those conditions which show 'record once only per patient' are to have each occurrence of the condition only recorded on 1 occasion no matter how many patient contacts are made for this condition. If the condition occurs a second or subsequent time, it is to be recorded again. Conversely, for other conditions each attendance at which they are addressed in some way is to be recorded.

HIV and AIDS surveillance

National surveillance for HIV and AIDS is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with state and territory health authorities, the Australian Government Department of Health and Ageing, the Australian Institute of Health and Welfare and other collaborating networks in surveillance for HIV/AIDS.

Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, either by the diagnosing laboratory (Australian Capital Territory and Tasmania), by doctor notification (Western Australia) or by a combination of laboratory and doctor sources (New South Wales, Northern Territory, Queensland, South Australia and Victoria). Cases of AIDS are notified through the state and territory health authorities to the National AIDS Registry. Diagnoses of both HIV infection and AIDS are notified with the person's date of birth and name code, to minimise duplicate notifications while maintaining confidentiality.

Currently, 2 tables presenting the number of new diagnoses of HIV infection, AIDS and deaths following AIDS are published in each issue of *CDI*. The tabulations are based on data available 3 months after the end of the reporting period, to allow for reporting delay and to incorporate newly available information.

Each year from 1997, the NCHECR has published the *HIV/AIDS, Viral Hepatitis and Sexually Transmissible Infections in Australia Annual Surveillance Report*. The annual surveillance report, available through www.med.unsw.edu.au/nchecr/, provides a comprehensive analysis and interpretation of surveillance data on HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia.

Laboratory Virology and Serology Reporting Scheme

The Laboratory Virology and Serology Reporting Scheme (LabVISE) began operating in 1977. The scheme currently comprises 17 laboratories from all states and the Australian Capital Territory. Contributors submit data fortnightly on the laboratory identification of viruses and other organisms. Each record includes mandatory data fields (laboratory, specimen collection date, a patient identifier code and organism), and optional fields (patient's sex, date of birth or age, postcode of residence, specimen source, clinical diagnosis and the method of diagnosis). Reports are collated, analysed and published quarterly in *CDI*. Each report includes summary tables of total numbers of organisms identified by state or territory and numbers of reports by month and participating laboratory. Monthly updates of LabVISE data are also published on the *Communicable Diseases Surveillance* website.

LabVISE data should be interpreted with caution. The number and type of reports received are subject to a number of biases. These include the number of participating laboratories, which has varied over time. The locations of participating laboratories also create bias, as some jurisdictions are better represented than others. Also changes in diagnostic practices, particularly the introduction of new testing methodologies, may affect laboratory reports. The ability of laboratory tests to distinguish acute from chronic or past infection must also be considered in interpretation of the data. Although changes in incidence cannot be determined with precision from this data, general trends can be observed, for example with respect to seasonality and the age-sex distribution of patients. See review in *Commun Dis Intell* 2002;26:323–374).

National Enteric Pathogens Surveillance System

Since 1980, the National Enteric Pathogens Surveillance Scheme (NEPSS) has collected, analysed and disseminated data on human enteric bacterial infections diagnosed in Australia. These pathogens include *Salmonella*, *Escherichia coli*, *Vibrio*, *Yersinia*, *Plesiomonas*, *Aeromonas* and *Campylobacter*.

Communicable Diseases Intelligence NEPSS quarterly reports include only *Salmonella*. NEPSS receives reports of *Salmonella* isolates submitted from primary diagnostic laboratories throughout Australia to any of the 5 serotyping laboratories, two of which (MDU and IMVS) also perform phage typing.

A case is defined as the isolation of a *Salmonella* from an Australian resident, either acquired locally or as a result of overseas travel, including isolates detected during immigrant and refugee screening. Second and subsequent identical isolates from an individual within 6 months are excluded, as are isolates from overseas visitors to Australia. The date of the case is the date the primary diagnostic laboratory isolated a *Salmonella* from the clinical sample.

NEPSS is operated by the Microbiological Diagnostic Unit — Public Health Laboratory, Department of Microbiology and Immunology, The University of Melbourne; and is overseen by a Steering Committee of state, territory and Commonwealth stakeholders. NEPSS may be contacted at the Microbiological Diagnostic Unit, by telephone +61 3 8344 5701, facsimile +61 3 8344 7833 or email joanp@unimelb.edu.au

Scientists, diagnostic and reference laboratories, clinicians and public health professionals generate and contribute data to NEPSS, which is supported by state and territory health departments and the Australian Government Department of Health and Ageing.

National Influenza Surveillance Scheme

Influenza surveillance in Australia is based on several schemes collecting a range of data that can be used to measure influenza activity.

- Since 2001, laboratory-confirmed influenza has been a notifiable disease in all Australian states and territories (except South Australia) and reported in the National Notifiable Diseases Surveillance System.
- In 2009, 6 sentinel general practitioner schemes contribute reports of influenza-like illness: the Australian Sentinel Practice Research Network, the Tropical Influenza Surveillance from the Northern Territory, the New South Wales Sentinel General Practice Scheme, the Victorian Sentinel General Practice Scheme, Queensland and Western Australian sentinel general practices.
- The Laboratory Virology and Serology Reporting Scheme laboratory reports of influenza diagnoses including virus type.

The results of each of the schemes are published together fortnightly throughout the influenza season (May to October) on the *Communicable Diseases Australia* Website as the Australian Influenza Report.

Annual reports on influenza in Australia are published in *CDI* each year (*Commun Dis Intell* 2008;32:208–226). These reports include the above data as well as absenteeism data from a major national employer, hospitalisation and mortality data and influenza typing data from the WHO Collaborating Centre for Influenza Reference and Research.

OzFoodNet: enhanced foodborne disease surveillance

The Australian Government Department of Health and Ageing established the OzFoodNet network in 2000 to collaborate nationally in the investigation of foodborne disease. OzFoodNet conducts studies on the burden of illness and coordinates national investigations into outbreaks of foodborne disease.

OzFoodNet reports quarterly on investigations of gastroenteritis outbreaks and clusters of disease potentially related to food. Annual reports have been produced and published in *CDI* (*Commun Dis Intell* 2008;32:400–424) since 2001. Data are reported from all Australian jurisdictions.

Sentinel Chicken Surveillance Programme

The Sentinel Chicken Surveillance Programme is used to provide an early warning of increased flavivirus activity in Australia. The main viruses of concern are Murray Valley encephalitis (MVEV) and Kunjin viruses. MVEV causes the disease Murray Valley encephalitis (formerly known as Australian encephalitis), a potentially fatal disease in humans. Encephalitis is less frequent in cases of Kunjin virus infection and these encephalitis cases have a lower rate of severe sequelae.

These viruses are enzootic in parts of the north-east Kimberley region of Western Australia and the Top End of the Northern Territory but are epizootic in other areas of the Kimberley, Pilbara, Gascoyne

Murchison and Mid-west regions of Western Australia, in north Queensland and in Central Australia. MVEV is also responsible for occasional epidemics of encephalitis in eastern Australia. Since 1974, a number of sentinel chicken flocks have been established in Australia to provide an early warning of increased MVEV activity. These programs are supported by individual state health departments. Each state has a contingency plan that will be implemented if one or more chickens in a flock seroconverts to MVEV.

Currently, flocks are maintained in the north of Western Australia, the Northern Territory, New South Wales and in Victoria. The flocks in Western Australia and the Northern Territory are tested all year round but those in New South Wales and Victoria are tested only in the summer months, during the main MVEV risk season. Results are posted on the National Arbovirus Surveillance Website by state representatives. A yearly summary is presented in *CDI* (*Commun Dis Intell* 2008;32:31–47).

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Communicable Diseases Intelligence
instructions for authors

Communicable Diseases Intelligence (*CDI*) is published quarterly (March, June, September and December) by the Surveillance Branch, Office of Health Protection, Australian Government Department of Health and Ageing.

The aim of *Communicable Diseases Intelligence (CDI)* is to disseminate information on the epidemiology of communicable disease in Australia, including surveillance, prevention and control.

The objectives of *CDI* are:

- to report on surveillance of communicable diseases of relevance to Australia;
- to publish other articles relevant to communicable disease epidemiology in Australia; and
- to provide information on other activities relevant to the surveillance, prevention and control of communicable disease in Australia.

CDI invites contributions dealing with any aspect of communicable disease epidemiology, surveillance, prevention or control in Australia. Submissions can be in the form of original articles, short reports, or letters to the editor.

CDI will invite guest editorials and review articles on occasion and publish guidelines and position papers from the Communicable Diseases Network Australia (CDNA) and its expert sub-committees.

Manuscripts for submission

Manuscripts submitted to *CDI* must be offered exclusively to the journal. All manuscripts should be accompanied by a covering letter that should include:

- a list of all authors;
- confirmation that the manuscript content (in part or in full) has not been submitted or published elsewhere; and
- whether the manuscript is being submitted as an article, short report, surveillance summary, outbreak report or case report.

In addition, manuscripts should include a title page that should contain the following information:

- title (e.g. Prof, Dr, Ms, Miss, Mrs, Mr), full name including middle initial, position held, and institution at the time the article was produced, of each author;
- name of corresponding author, including current postal address, telephone, facsimile and email; and
- word count of the main text and of the abstract.

On receipt of a manuscript, authors will be sent a brief acknowledgment. Accepted manuscripts are edited for style and clarity and final proofs are returned to the corresponding author for checking prior to printing.

Authorship

Authorship should be based on substantial contribution to the article. Each author should have participated sufficiently to take public responsibility for the article. Others contributing to the work should be recognised in the acknowledgments.

Types of manuscript

Original articles

The text of articles must be structured to contain an abstract, introduction, methods, results, discussion, acknowledgments and references. Manuscripts submitted as articles must be 3,000 words or less and are peer-reviewed. Occasionally, reports of urgent public health importance may be published immediately, at the discretion of the Editor.

Short reports

Short reports are not subject to peer review and should be of less than 2,000 words. Types of short reports include:

Surveillance summaries

A report of 1,000 words or less which briefly reports on changes in the local epidemiology of communicable disease, changes in surveillance systems, or new interventions, such as implementing vaccination in an at-risk group. Surveillance summaries should provide a brief description of the setting and a discussion of the significance of the events, changes or interventions.

Outbreak reports

Unstructured reports of communicable disease outbreaks of 500 to 1,000 words will be considered for publication based on their public health significance. Reports should include details of the investigation, including results of interventions and

the significance of the outbreak for public health practice. More comprehensive reports on outbreaks should be submitted as articles.

Case reports

Brief unstructured reports of 500 to 1,000 words on unique cases of communicable disease will be considered based on their public health significance. Authors must note the instructions on the protection of patient's right to privacy (see Ethics committee approvals and patient's right to privacy below). Some discussion of the significance of the case for communicable disease control should be included.

Letters to the Editor

The editors welcome comments on articles published in *CDI* in the form of letters to the Editor. Letters should normally be less than 500 words, include no more than a single chart and less than 6 references.

Document preparation

Authors are asked to provide an electronic copy of the manuscripts. Microsoft Word for Windows 2003 or an earlier version is preferred. Alternatively files should be saved as Rich Text Format (rtf).

In addition:

- Arial font is preferred but if not available use Times New Roman.
- Abstracts should not exceed 250 words. Do not cite references in abstracts. Structured abstracts are not acceptable.
- Include up to 10 keywords.
- Avoid too many abbreviations.
- Do not use numbered paragraphs.
- Do not use page numbering.
- Do not use headers or footers.

Final manuscripts should not include any field codes such as automatic numbering for references. Electronic referencing software (e.g. Endnote) field codes should be embedded before submission of the final version.

Tables

- Tables and table headings should be provided in the manuscript at the end of the text and should be referred to within the results section.
- Information in tables should not be duplicated in the text.
- Headings should be brief.
- Simplify the information as much as possible, keeping the number of columns to a minimum.
- Separate rows or columns are to be used for each information type (e.g. percentage and number should be in separate columns rather than having one in parentheses in the same column).
- If abbreviations are used these should be explained in a footnote.
- Footnotes should use the following symbols in sequence: * † ‡ § || ¶ ** †† ‡‡
- Do not use borders, or blank rows or blank columns for spacing.

Figures and illustrations

Figures and illustrations, including headings, should be provided in the manuscript at the end of the text and should be referred to within the results section. In addition, they should also be provided as a separate file in accordance with the following requirements.

Examples of each of the following can be found in the on-line version of Instructions to authors at: http://www.health.gov.au/internet/wcms/publishing.nsf/Content/cda-pubs-cdi-auth_inst.htm

Charts

- Use Microsoft Excel for Windows.
- Each figure should be created on a separate worksheet rather than as an object in the datasheet (use the 'as new sheet' option for chart location).
- The numerical data used to create each figure must be included on a separate worksheet.
- Worksheets should be appropriately titled to distinguish each graph.
- Do not include the graph heading on the Excel worksheet.

Illustrations

- Black and white illustrations or flow charts can be included if required.
- Images should preferably be at least 300 dpi.
- Electronic copies of computer-generated illustrations should preferably be saved in a vector image program such as Adobe Illustrator but other similar graphic software is acceptable. Files should be saved in one of the following graphic formats (in preferential order): AI, EMF, TIFF, EPS, or GIF.
- Use a sans serif font for figures (e.g. Arial). Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

Photographs

- Photographs may be submitted if required.
- Photos need to be at least 300 dpi.
- Electronic copies should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats (in preferential order): PSD, TIFF, EPS or JPEG (JPG).

Maps

- Electronic copies of black and white (outline) maps should be saved in Adobe Photoshop, or similar graphic software in one of the following graphic formats (in preferential order): PSD, TIFF, EPS, or GIF.
- Thermal maps created by mapping programs such as MapInfo or Arc GIS should be saved at 300 dpi and in one of the following graphic formats (in preferential order): EMF, TIFF, EPS, or JPEG (JPG). Shading of map areas should be distinguishable when printed in black and white.
- Use a sans serif font for text. Symbols, lettering and numbering should be clear and large enough to be legible when reduced in size.

References

References should be identified consecutively in the text by the use of superscript numbers without brackets. Any punctuation should precede the reference indicators.

The accuracy of references is the responsibility of authors. Use the Vancouver reference style (see International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Ann Intern Med* 1997;1126:36–47 available from: http://www.nlm.nih.gov/bsd/uniform_requirements.html) and abbreviate journal names as in Medline (e.g. *Commun Dis Intell*). The Medline journal database is available from: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=journals. Include the surnames and initials of all authors (or only the first 6 authors, et al, if there are more than six). Cite the first and last page numbers in full, and specify the type of reference (e.g. a letter, an editorial, an abstract, or supplement).

Cite personal communications and unpublished papers in the text, not in the reference list, with the exception of material that has been accepted for publication (in press). Obtain written permission from people cited, and include their title, position and affiliation.

Ethics committee approvals and patients' rights to privacy

All investigations on human subjects must include a statement that the subjects gave their written informed consent, unless data collection was covered by public health legislation or similar studies have been considered by a relevant ethics committee and a decision made that its approval was not required. The name of the ethics committee that gave approval for the study should be included in the text. Alternatively, if approval is not required a statement to this effect should appear in the manuscript.

When informed consent has been obtained this should be included in the text.

Ethical approval and patient consent may also be required for case reports. Identifying details about patients should be omitted if they are not essential, but data should never be altered or falsified in an attempt to attain anonymity.

Review process

Articles provisionally accepted for publication undergo a peer review process. Manuscripts are reviewed by 2 experts in the topic area. Authors may be asked to revise articles as a result of the review process before the final decision about publication is made by the Editor. Revised articles are to be returned with a covering letter addressing each comment made by each reviewer.

Occasionally, reports of urgent public health importance may be published immediately without peer review, at the discretion of the Editor. Articles may also be rejected without peer review.

Short reports and reports from national committees are not subject to peer review.

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All authors are asked to transfer copyright to the Commonwealth before publication. A copyright form will be sent to the corresponding author. All authors are required to sign the copyright release. The Commonwealth copyright will be rescinded if the article is not accepted for publication.

Submission of manuscripts

Manuscripts should be provided electronically by email to: cdi.editor@health.gov.au

Requests for further information can be obtained either by telephone to (02) 6289 2717, by facsimile: (02) 6289 2600 or by email to the address above.