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Annual reports

Annual report of the Australian Meningococcal Surveillance Programme, 2008

The Australian Meningococcal Surveillance Programme

Abstract

In 2008, there were 260 laboratory-confirmed cases of invasive meningococcal disease (IMD) analysed by the National Neisseria Network, a nationwide network of reference laboratories. One hundred and forty-nine isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were available for which the phenotypes (serogroup, serotype and serosubtype) and antibiotic susceptibility were determined. An additional 111 cases were confirmed by non-culture based methods. Nationally, 223 (85%) laboratory-confirmed cases where a serogroup was determined were infected with serogroup B and 17 (6.5%) infected with serogroup C meningococci. Nationally, the total number of confirmed cases has remained relatively stable since 2006, but the number of cases in each jurisdiction may vary from year to year. Queensland had the highest number of recorded cases in 2008. 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 again B:15:P1.7 and B:4:P1.4. Although serogroup C cases were numerically low, phenotype C:2a:P1.5 predominated in this group. 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 ceftriaxone. One isolate had reduced susceptibility to rifampicin and two to ciprofloxacin. *Commun Dis Intell* 2009;33(3):259–267.

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

Introduction

The National Neisseria Network (NNN) is a long-term collaborative program for the laboratory surveillance of the pathogenic Neisseria, *Neisseria meningitidis* and *N. gonorrhoeae*. NNN has operated since 1994 through a network of reference laboratories in each state and territory to provide a national laboratory-based program for the examination of *Neisseria meningitidis* from cases of invasive meningococcal disease (IMD).[1] The NNN supplies national data on the phenotype and/or the genotype of invasive meningococci, and their antibiotic susceptibility, in annual reports published in *Communicable Diseases Intelligence*.[2] 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 for outbreaks or case clusters locally and nationally. Despite the significant reduction in the number of cases of IMD since 2004 when a publicly-funded program of selective vaccination with conjugate serogroup C meningococcal vaccine was completed, IMD remains an issue of public health concern in Australia. The success of further vaccine initiatives in Australia is dependent upon detailed analysis of the *N. meningitidis* isolates circulating locally. This report provides relevant details of cases of IMD confirmed by laboratory testing in 2008.

Methods

Isolate based invasive meningococcal disease cases

Case confirmation was based upon isolation of a meningococcus from a normally sterile site or demonstrated and defined as IMD according to Public Health Laboratory Network criteria.[3] Information on the site of infection, the age and sex of the patient and the outcome of the infection (survived/died) 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 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 have been approximated 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 standardised agar plate dilution technique.[4]

sensitive, MIC $\leq$ 0.03 mg/L;

less sensitive, MIC 0.06 – 0.5 mg/L;

relatively resistant MIC $\geq$ 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 primarily by NAAT and occasionally by serological techniques. NAAT testing is essentially by polymerase chain reaction (PCR) techniques[5] that demonstrate the presence of meningococcal-specific nucleic acid in appropriate samples and has been progressively introduced and updated in the different jurisdictions. Data from the results of these investigations were included for the first time in the 1999 report. 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.[6–9] 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.

Results

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

Number of laboratory confirmed cases

There were 260 laboratory confirmed cases of IMD in 2008 (Table 1) compared with 281 in 2007, 271 in 2006, 345 in 2005 and 361 in 2004. In 149 cases (57%), a positive culture was obtained with or without a positive non-culture based test and 111 (43%) cases were confirmed by a non-culture based method alone. The total number of all laboratory confirmed cases increased in Queensland from 75 in 2007 to 83 in 2008 and this jurisdiction had the highest number of laboratory confirmed cases. In New South Wales, numbers

Table 1: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2008, by serogroup and state or territory

State or territory	Serogroup					Total
	B	C	Y	W135	NG*	
ACT	4	1	0	0	0	5
NSW	46	6	4	5	1	62
NT	4	2	0	0	0	6
Qld	73	4	1	2	3	83
SA	18	0	0	1	0	19
Tas	1	0	0	0	0	1
Vic	51	2	2	0	6	61
WA	22	0	0	0	1	23
Australia	219	15	7	8	11	260

* Not serogrouped.

detected decreased to 62 from 101 in 2007. There were 61 cases in Victoria, which was little changed from the 59 cases in 2007. Small or no numerical differences were noted in other jurisdictions.

Seasonality

Thirty-six cases occurred between 1 January and 31 March, 61 between 1 April and 30 June, 98 between 1 July and 30 September and 65 between 1 October and 31 December. A winter peak of meningococcal disease is more usual, but the above pattern was also present in 2007.

Age distribution

Nationally, the peak incidence of meningococcal disease was again in those aged 4 years or under (Table 2). Those aged less than one year or in the 1–4 year age group, together accounted for 94 cases (36.1% of the total) in 2008. There were 100 cases confirmed in these age groups (35.5%) in 2007. A secondary disease peak is also usual in the adolescent or young adult age group. The total of 50 cases (19.2% of all confirmed cases) in those aged 15–19 years was a little less than the 56 cases (19.9%) in this age group in 2007. Those aged 15–24 years accounted for 71 cases (27.2%) in 2008 and 87 cases (31%) in 2007.

Table 2: All laboratory confirmed cases of invasive meningococcal disease, Australia, 2008, by age, state or territory and B and C serogroups

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	2	1	1	0	0	0	0	0	0	0	4
	C	0	0	0	0	0	0	1	0	0	0	1
	Total	2	1	1	0	0	0	1	0	0	0	5
NSW	B	8	8	1	2	10	2	8	6	0	1	46
	C	0	1	0	0	0	1	0	3	1	0	6
	Total	10	10	1	2	13	4	9	9	3	1	62
NT	B	1	1	0	0	0	0	1	0	0	1	4
	C	0	0	0	0	1	0	1	0	0	0	2
	Total	1	1	0	0	1	0	2	0	0	1	6
Qld	B	20	13	3	7	13	3	5	7	2	0	73
	C	1	1	0	0	0	1	0	1	0	0	4
	Total	23	15	3	8	14	5	5	8	2	0	83
SA	B	0	3	0	0	5	4	5	1	0	0	18
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	0	4	0	0	5	4	5	1	0	0	19
Tas	B	0	0	1	0	0	0	0	0	0	0	1
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	0	0	1	0	0	0	0	0	0	0	1
Vic	B	10	6	3	5	12	2	7	5	1	0	51
	C	1	0	0	0	0	0	0	0	1	0	2
	Total	11	6	3	5	15	4	7	8	2	0	61
WA	B	6	3	0	0	2	4	3	3	1	0	22
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	6	4	0	0	2	4	3	3	1	0	23
Australia	B	47	35	9	14	42	15	29	22	4	2	219
	C	2	2	0	0	1	2	2	4	2	0	15
	Total B+C	49	37	9	14	43	17	31	26	6	2	234
	other	4	4	0	1	7	4	1	3	2	0	26
	Total	53	41	9	15	50	21	32	29	8	2	260
	% of all	20.4	15.7	3.5	5.8	19.2	8.1	12.3	11.1	3.1	0.8	

NS Not stated.

Totals include cases due to other serogroups (15) and cases where the serogroup was not determined (11).

Serogroup data

The serogroup of the meningococci causing disease was determined in 249 of the 260 laboratory confirmed cases of IMD. Of these 249 cases where a serogroup was determined, 219 (88%) were serogroup B and 15 (6%) serogroup C. In 2007, 223 (85%) were serogroup B and 17 (6.5%) serogroup C. In 2008, an additional 8 cases (3.2%) were of W135 and 7 cases (2.8%) were of serogroup Y. With the continuing decline in the number of serogroup C infections, serogroup B meningococci predominated in all age groups (Figure) and jurisdictional differences in serogroup distribution were not evident. The 15 serogroup C cases of IMD were distributed in 5 jurisdictions: New South Wales (6), Queensland (4), Victoria and the Northern Territory (2 each) with a single case in the Australian Capital Territory. Eight of the 15 cases of serogroup C disease in 2008 were in those aged 25 years or more, 4 cases were reported in those aged 4 years or less, a single case in those aged 15–19 years and a further two in those aged 20–24 years.

Figure: Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2008, by age

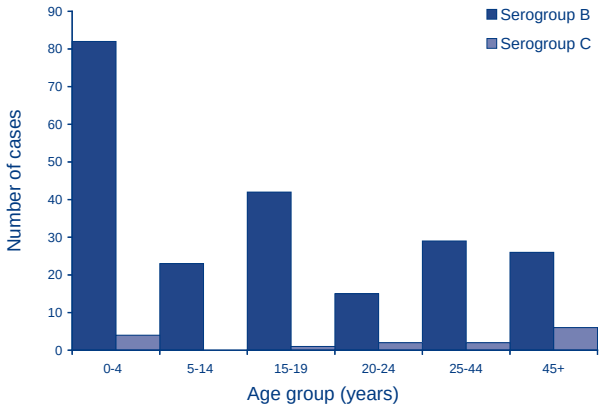

Table 3 shows a national comparison of the number and proportion of serogroup B and C cases by age from 2004 to 2008. In those aged 14 years or less, there was a decrease in total case numbers and in serogroup B cases in 2007, but there was no further change noted in case numbers in these age groups in 2008. 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. Again, the relative proportion of serogroup B and C IMD cases was unaltered in 2008 from that observed in 2007. In older (25 years or more) age groups there was a further increase in the number and proportion of serogroup B cases in 2008 whereas the number of serogroup C cases in these age groups was unaltered.

Table 3: A comparison of the number and proportion of serogroup B and serogroup C laboratory-confirmed cases, 2004 to 2008, by known age

Year	Serogroup	Age									
		< 4 years		5-14 years		15 –19 years		20-24 years		25+ years	
		n	%	n	%	n	%	n	%	n	%
2008	B	82	89.1	23	95.8	42	91.3	15	83.3	57	81.4
	C	4	4.4	0	0	1	2.2	2	11.1	8	11.4
	All*	92		24		46		18		67	
2007	B	83	90	19	83	48	91	24	80	49	75
	C	4	4	0	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.

Phenotypes of invasive meningococcal isolates

Serogroup B meningococci are typically of heterogeneous phenotypes. In 2008, the phenotypes of invasive isolates, based on a determination of their serogroup, serotype and serosubtype were analysed and again showed this diversity. 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 20 isolates were of serotype 4 and nine of these were from New South Wales, five from Victoria and three from Queensland with

Table 4: Common serotypes and sero-subtypes of isolates from culture positive cases of *Neisseria meningitidis* infection, 2008, by state or territory

State or territory	Serogroup B				Serogroup C			
	serotype	n	serosubtype	n	serotype	n	serosubtype	n
ACT	4	1	1.14	1	2a	1	1.14	1
	15	1	1.7	1				
NSW	4	9	1.15	2	2a	4	1.5	2
			1.4	2			NST	2
			1.6,3	1	NT	2	1.3	1
			nst	4			NST	1
	15	6	1.7	5				
			nst	1				
	14	2	1.4	1				
			1.5,2	1				
	1	2	1.14	1				
			1.6,1.3	1				
	nt	6	nst	2				
			Diverse:1.3/.4/.5/.9	1 ea				
NT	4	1	1.4					
	nt	3	nst	3				
Qld	15	5	1.7	5	2a	2	1.5	1
	1	4	1.14	2			NST	1
			nst	2	15	1	1.9	1
	4	3	1.4	2				
			nst	1				
	14	2	1.4	1				
			nst	1				
	nt	23	1.4	7				
			Diverse:1.13/.14/.15/5	1 ea				
			nst	12				
Tas								
Vic	7	7	1.19	3	2a	2	1.4	2
			1.5	3				
			1.17,9	1				
	15	5	1.7	4				
			1.4	1				
	19.1	6	1.18	3				
			1.22,14	1				
	4,7	5	1.4	3				
			1.5	1				
			1.18	1				
WA	1	4	nst	2				
			1.14/1.6	1 ea				
	14	2	nst	2				
	15	2	1.7	1				
	4	1	1.4	1				
	nt	6	nst	4				
			1.14/1.4	1 ea				

nt Not sero-typable.

nst Not sero-subtypable.

single isolates from the Australian Capital Territory, the Northern Territory and Western Australia. Eight of these 20 were of serosubtype P1.4, which has been circulating in New Zealand at high rates for many years. Another 19 serogroup B isolates were of serotype 15, and 16 of these were of serosubtype 1.7, which has been circulating in Australia for many years.

The 12 serogroup C strains that were phenotyped were predominantly of serotype 2a (9 strains) and this phenotype has predominated in serogroup C meningococci in Australia for many years. Two strains could not be serotyped and one was of serotype 15, usually found within serogroup B meningococci. 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 has been of particular interest. This phenotype has figured prominently in Victorian data previously. For example, in 2003 there were 29 serogroup C isolates of this serotype/serosubtype detected nationally, 21 in 2004, and eight in 2005. Only a 2 isolates with this phenotype were seen in 2008, both in Victoria. Three of the serogroup C:2a isolates were of sero-subtype 1.5 and one of 1.14.

Outcome data for invasive meningococcal disease for laboratory confirmed cases

Outcome data (survived or died) were available for 76 (29%) of the 260 laboratory confirmed cases (Table 5). Four deaths were recorded in this group (5.2%), all with serogroup B infection for which outcomes were available for 64 of 219 cases. Three of the cases were attributable to septicaemia and the fourth to meningitis. No deaths were recorded in 12 infections caused by other serogroups. A single death in 25 patients with meningitic IMD and 3 deaths in 51 bacteraemic patients were recorded.

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, while those shown as PCR positive were culture negative with or without positive serology. There were 85 diagnoses of meningitis based on cultures or PCR examination of CSF either alone or with a positive blood sample (including 2 PCR based diagnoses on post-mortem brain samples and 170 from blood samples (cultures or PCR) alone. There were three other isolates from synovial fluid and in 2 cases the

Table 6: Anatomical source of samples positive for a laboratory confirmed case of invasive meningococcal disease, Australia, 2008

Specimen type	Isolate of MC	PCR positive*	Total
Blood	116	54	170
CSF +/– blood	30	55‡	85
Other†	3	2	5
Total	149	111	260

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

† Other samples: 3 isolates from joints and 2 PCR diagnoses from an unknown source.

‡ 2 diagnosed by PCR of brain tissue.

Table 5: Outcome data (survived, died) for laboratory confirmed cases of invasive meningococcal disease, 2008, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG	
Meningitis	Survived	21	0	2	1	0	24
	Died*	1	0	0	0	0	1
	Total	22	0	2	1	0	25
Septicaemia	Survived	39	2	1	1	5	48
	Died	3	0	0	0	0	3
	Total	42	2	1	1	5	51
All cases	Survived	60	2	3	2	5	72
	Died	4	0	0	0	0	4
	Total	64	2	3	2	5	76

* Clinical sample from post-mortem brain tissue.

NG Not groupable.

source of the clinical sample was not disclosed. No cases that were serologically positive were culture and PCR negative.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

One hundred and forty-nine isolates were available for determination of their susceptibility to penicillin and other antibiotics. Using defined criteria, 108 isolates (72%) 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 was slightly less than that reported in 2007 (79%).

Other antibiotics

All isolates were fully susceptible to ceftriaxone (and by extrapolation to other third generation cephalosporins) A single isolate had altered susceptibility to rifampicin and two to ciprofloxacin (MIC, 0.25 mg/L). All three were reported from Queensland.

Discussion

The total number of laboratory-confirmed cases of IMD nationally has remained relatively stable from 2006 to 2008 (range 260–281) after recording 345 cases in 2005. However, there have been fluctuations in the frequency of detection of cases between jurisdictions over this period with Queensland recording the highest number of cases in 2008 (83) with a reduction in numbers from New South Wales. These changes in case distribution were essentially attributable to altered numbers of serogroup B cases in 2008 and little change was detected in serogroup C numbers. Cultures were obtained from sterile sites in 149 cases, the lowest number of isolates detected over the duration of the program that commenced in 1994, and a further slight decline from the 154 cases seen in 2007 and the 166 cases from which isolates were obtained in 2006. Non-culture based diagnoses were used to confirm a further 111 (43%) cases as IMD (127 [45%] in 2007). Attention is specifically drawn to earlier AMSP reports that explain differences between the number of clinically notified cases and laboratory confirmed cases.[10] It should also be remembered that surveillance systems rarely capture all cases in any given period so that small differences in numbers of cases should be expected.

Only 15 serogroup C infections were identified nationally in 2008 so that serogroup B disease accounted for 88% of all infections where a serogroup was determined. No serogroup C cases were identified in South Australia, Western Australia or Tasmania with only small numbers present in the other jurisdictions. Only low numbers of infections due to serogroups Y and W135 were encountered, and this is usual for 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 again infrequently encountered in the latter age group in 2008. The reduced and low number of serogroup C cases in those aged 25 years or more (Table 3) was also maintained, and may be attributable 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]

Phenotypic and genotypic data again found no evidence of substantial numbers of cases of IMD caused by *N. meningitidis* that have undergone genetic recombination, although sporadic instances of this occurrence have been detected in Australia. There were some concerns expressed that the documented capacity for genetic reconfiguration within meningococci may lead to the emergence of new and invasive subtypes following extensive vaccine use.[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. All of the small number of fatal cases of IMD were associated with serogroup B infections. The NNN does not attempt collection of morbidity data associated with IMD.

The distribution NNN of penicillin MICs in invasive isolates showed that the proportion with decreased susceptibility to penicillins was 72%, a little less than that observed in 2007 (79%). It is emphasised that this decreased susceptibility does not affect clinical outcomes and penicillins remain a suitable treatment for IMD in Australia. All isolates were susceptible to the third generation cephalosporins and to the 'clearance' antibiotics rifampicin and ciprofloxacin with the exception of a small number of isolates from Queensland with decreased susceptibility to rifampicin (1) and ciprofloxacin (2). The latter group of strains with decreased susceptibility to quinolone antibiotics is the subject of on-going international interest following their first description from the AMSP group in 2000.[12–15] A single isolate with decreased susceptibility to quinolone antibiotics was detected in 2007 in AMSP data.

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Annual report of the Australian Gonococcal Surveillance Programme, 2008

The Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme monitors the antibiotic susceptibility of *Neisseria gonorrhoeae* isolated in all states and territories. In 2008 the *in vitro* susceptibility of 3,110 isolates of gonococci from public and private sector sources was determined by standardised methods. Different antibiotic susceptibility patterns were again seen in the various jurisdictions and regions. Resistance to the penicillins nationally was at 44% and ranged between 25% in Queensland and 73% in South Australia with the exception of the Northern Territory, where the proportion of drug resistant strains was 4%. Quinolone resistance in gonococci isolates also continued to increase so that nationally 54% of all isolates were ciprofloxacin-resistant, and most of this resistance was at high minimal inhibitory concentrations (MIC) levels. The proportions of quinolone resistant gonococci detected ranged between 80% in South Australia and 31% in Western Australia. All isolates remained sensitive to spectinomycin. Approximately 1.1% of isolates showed some decreased susceptibility to ceftriaxone (MIC 0.06 mg/L or more) and azithromycin resistance was also present in low numbers of gonococci with MICs up to 16 mg/L. A high proportion of gonococci examined in larger urban centres were from male patients and rectal and pharyngeal isolates were common in men. In other centres and in rural Australia the male to female ratio of cases was lower, and most isolates were from the genital tract. *Commun Dis Intell* 2009;33(3):268–274.

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

Introduction

Gonorrhoea remains a major disease of public health importance in many countries including Australia. Effective antibiotic treatment is an essential component of gonococcal disease control at the population level so that the impact of antimicrobial resistance (AMR) in *Neisseria gonorrhoeae* on the outcome of treatment is a major concern, and one of long standing.[1] AMR in gonococci has emerged and spread in many parts of the world so that resistance to the penicillins, tetracyclines and macrolides has seen the widespread removal of these cheap, oral agents from standard treatment regimens. Over many years the high levels of resistance to fluoroquinolone antibiotics documented in urban centres in Australia[2] and nearby countries have also compromised the efficacy of this group of antibiotics at both an individual and population health level. This has lead to their widespread replacement with extended-spectrum cephalosporin antibiotics as the recommended treatment for gonorrhoea in Australia and elsewhere.[3] Unusually, but importantly in Australia however, treatments based on the penicillins remain effective in many rural centres where extremely high disease rates persist.[2]

AMR in *Neisseria gonorrhoeae* isolated in large urban centres in Australia is heavily influenced by the continuing introduction of multi-resistant gonococci from overseas.[2] Increasing numbers of reports of treatment failures with orally administered extended-spectrum cephalosporins have appeared from overseas sources.[4,5] In Australia, only injectable, but not oral, extended-spectrum cephalosporin antibiotics are available (ceftriaxone) and are recommended for use in high doses.[3] No treatment failures have yet been reported following ceftriaxone treatment of genital-tract gonorrhoea. Recently, 2 instances of failure of treatment of pharyngeal gonorrhoea were recorded in Sydney,[6] where elimination of intercurrent genital-tract infection with the same organism was achieved. The gonococci involved in both instances had raised minimal inhibitory concentrations (MICs) for ceftriaxone.

Strategies for treating and controlling gonorrhoea are based on the use of single dose treatments that cure a minimum of 95% of cases,[1] but formulation of these standard treatment regimens relies on data derived from continuous monitoring of the susceptibility of gonococci to recommended antibiotics.[1,7] Recently, following reports of treatment failures with orally administered extended-spectrum cephalosporins,[4,5] calls have been made for enhanced surveillance of all forms of gonococcal AMR in order to optimise gonococcal antibiotic treatment.[8]

The Australian Gonococcal Surveillance Programme (AGSP) has monitored the susceptibility of *N. gonorrhoeae* continuously since 1981[9] making it the longest continually running national surveillance system for gonococcal AMR. The emergence and spread of penicillin and quinolone resistant gonococci in major cities in Australia has

been well documented.[2] This analysis of AMR in *N. gonorrhoeae* in Australia was derived from data generated by the AGSP during the 2008 calendar year. It includes analyses and commentary arising from the increasing concerns consequent upon the presence in Australia of gonococcal isolates showing resistance to multiple antibiotics including those with decreased susceptibility to ceftriaxone.[2,10]

Methods

The AGSP is a component of the National Neisseria Network of Australia and conducts ongoing monitoring of AMR in gonococci through a collaborative network of reference laboratories in each state and territory. This collaborative network of laboratories obtains isolates for examination from as wide a section of the community as possible and both public and private sector laboratories refer isolates to regional testing centres. The increasing use of non-culture based methods of diagnosis has the potential to reduce the size of the sample of isolates available for testing. Details of the numbers of organisms examined are thus provided in order to indicate the AGSP sample size.

Gonococci, isolated in and referred to the participating laboratories, were examined for antibiotic susceptibility to the penicillins, quinolones, spectinomycin and third generation cephalosporins and for high-level resistance to the tetracyclines by a standardised methodology.[9,11] The AGSP also conducted a program-specific quality assurance (QA) program.[12] Antibiotic sensitivity data were submitted quarterly to a coordinating laboratory, which collated the results and also conducted the QA program. Additionally, the AGSP received data on the sex of the patient and site of isolation of gonococcal strains. Where available, data on the geographic source of acquisition of antibiotic-resistant isolates were included in analyses.

Results

Number of isolates

There were 3,192 gonococcal isolates referred to or else isolated in AGSP laboratories in 2008, little changed overall from the 3,103 examined in 2007. The source and site of infection of these isolates are shown in Table 1. Eight hundred and fifty-seven gonococci (27% of the Australian total) were isolated in New South Wales, 567 (17.8%) in Victoria, 542 (17%) in Queensland, 410 (12.8%) in Western Australia, 403 (12.6%) in the Northern Territory, and 391 (12.3%) in South Australia, with small numbers in Tasmania (13) and the Australian Capital Territory (9). Numbers decreased in New South Wales (from 973), Victoria (from 625) and Queensland (from 542) from those reported in 2007, but increased from 366 in Western Australia and 240 in South Australia. The number of isolates from the Northern Territory was little changed. Three thousand one hundred and ten isolates remained viable for susceptibility testing, representing approximately 40% of the notifications made to NNDSS during 2008.

Source of isolates

There were 2,509 isolates from men and 682 from women, with a male to female (M:F) ratio of 3.7:1, lower than the 4.7:1 ratio for 2007. The number of isolates from men decreased slightly from 2,560

Table: Source and number of gonococcal isolates, Australia, 2008, by sex, site and state or territory

Gender	Site	State or territory						Aust*
		NSW	NT	Qld	SA	Vic	WA	
Male	Urethra	457	257	317	215	308	270	1,835
	Rectal	181	1	54	23	110	15	386
	Pharynx	99	0	20	31	69	9	229
	Other/NS	3	8	12	17	6	10	59
	Total	740	266	403	286	493	304	2,509
Female	Cervix	102	131	126	73	62	101	600
	Other/NS	15	6	13	31	12	5	82
	Total	117	137	139	104	74	106	682
Unknown	Total	0	0	0	1	0	0	1
Total*		857	403	542	391	567	410	3,192

NS Not stated.

* Includes isolates from Tasmania (13) and the Australian Capital Territory (9).

The site of isolation and sex of some infected patients was not known.

in 2007, but the number of isolates from women increased from 541. Isolates from females increased in all larger jurisdictions except for the Northern Territory. The M:F ratio remained high in New South Wales (6.3:1) and Victoria (6.6:1) where strains were more often obtained from urban populations than in Queensland (2.9:1), Western Australia (2.9:1), South Australia (2.7:1) and the Northern Territory (1.9:1) where there is a large non-urban component of gonococcal disease. Male rectal and pharyngeal isolates were most frequently found in New South Wales (together 38% of isolates obtained from men) and Victoria (36%), with about half this proportion in South Australia and Queensland. For 141 isolates in Table 1, the site is shown as 'other' or 'not stated'. Included in this total were 24 cases of disseminated gonococcal infection, 13 in men (0.5% of all infections) and 11 (1.6%) in women. Another 39 of these gonococci were pharyngeal and 18 rectal isolates from women. Other infected sites included ophthalmic infection in adults and children (6), placenta (2), peritoneal fluid in women (2) and a wound isolate (1) and several isolates from unspecified abscesses. Although not all infected sites were identified, isolates from urine samples were regarded as genital tract isolates and most of the other unidentified isolates were probably from this source, although they were not so specified.

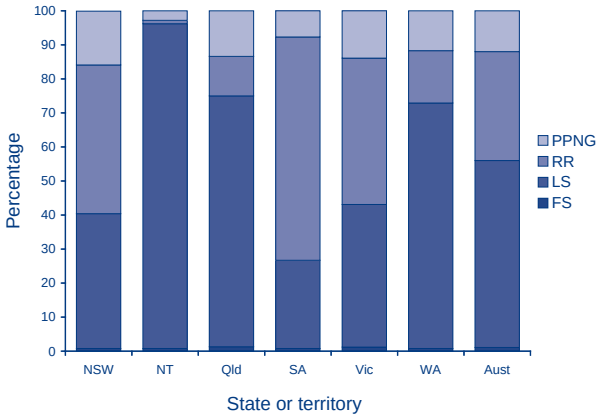
Antibiotic susceptibility patterns

In 2008 the AGSP reference laboratories examined 3,110 gonococcal isolates for sensitivity to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics), spectinomycin and for high level resistance to tetracycline (TRNG). As in past years the patterns of gonococcal antibiotic susceptibility differed between the states and territories. For this reason data are presented by region as well as aggregated for Australia as a whole.

Penicillins

The categorisation of gonococci isolated in Australia in 2008 by penicillin MIC is shown in Figure 1. Infections unlikely to respond to the penicillin group of antibiotics (penicillin, ampicillin, amoxycillin, with or without clavulanic acid) are those caused by gonococci shown as 'penicillinase-producing' *N. gonorrhoeae* (PPNG) and 'RR - relatively resistant'. Resistance in the PPNG group results from the production of beta-lactamase. In those 'relatively resistant' through the aggregation of chromosomally-controlled resistance mechanisms[1] (CMRP), chromosomal resistance is defined by an MIC to penicillin of 1 mg/L or more.[1,11] (The MIC in mg/L is the least amount of antibiotic which inhibits *in vitro* growth under defined conditions.) Infections with gonococci classified as fully sensitive (FS, MIC ≤ 0.03 mg/L) or less sensitive (LS, MIC 0.06–0.5 mg/L) would be expected to respond to standard penicillin treatments, although response to treatment may vary at different anatomical sites.

Figure 1: Penicillin resistance of gonococcal isolates, Australia, 2008, by state or territory



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.*

Nationally, 1,367 (44%) gonococci were penicillin resistant by one or more mechanisms in 2008, a further increase in the proportion of isolates resistant to this group of antibiotics recorded in 2007 (38.2%), 2006 (34%) and 2005 (29.5%). Of these, 994 (32% of all isolates) were CMRP and 373 (12%) PPNG. In 2007, 796 (26.2%) were CMRP and 369 (12.1%) PPNG, so that the increase in penicillin resistance nationally was solely due to increased chromosomally mediated resistance. The proportion of penicillin-resistant gonococci of all gonococcal isolates was highest in South Australia, 73.2% (PPNG 7.6%, CMRP 65.6%); New South Wales, 59.6% (PPNG 15.8%, CMRP 43.8%); Victoria, 56.8% (PPNG 13.9%, CMRP 43%); and Western Australia 27.1% (PPNG 11.7%, CMRP 15.4%). All these jurisdictions showed increased proportions of penicillin resistant gonococci. In Queensland the proportion of penicillin resistant gonococci decreased to 25% (PPNG 13.4%, CMRP 11.6%). One CMRP was identified in the Australian Capital Territory but no PPNG was detected. In Tasmania there were 5 PPNG and 6 CMRP. In the Northern Territory there were 15 penicillin resistant gonococci; 11 PPNG (7 from Darwin and 4 from Alice Springs) and 4 CMRP (2 each from Darwin and Alice Springs) resulting in a total of 3.9% of strains that were penicillin resistant in 2008 (4.1% in 2007; 4.6% in 2006). Data on acquisition of PPNG were available in 81 (22%) infections. Forty-three

(53%) of these infections with PPNG were acquired locally and 38 (47%) by overseas contact. These external contacts were principally in Western Pacific or South East Asian countries with those reported from Thailand (12), the Philippines (7) and Indonesia (Bali) (4) the most numerous. Additionally, China, Malaysia, Singapore and more widely Europe, the United Kingdom and the United Arab Emirates were named as countries of acquisition.

Ceftriaxone

From 2001 onwards, low numbers of isolates with slightly raised ceftriaxone MICs have been found in Australia. In 2008, 34 (1.1%) gonococci 'non-susceptible' to ceftriaxone were identified with ceftriaxone MICs in the range 0.06 to 0.25 mg/L. In 2006, there were 23 (0.6%) gonococci isolates examined of this type and 25 (0.8%) in 2007. Seventeen of these were present in New South Wales (2% of isolates there), four (0.8%) in Queensland, 11 (2.9%) in South Australia and one each in Western Australia and the Northern Territory.

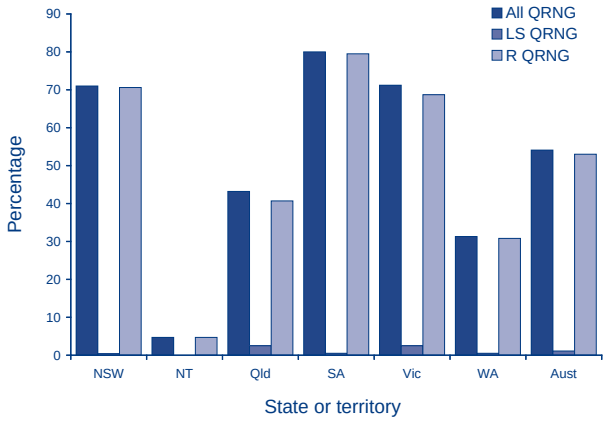
Spectinomycin

All isolates were again susceptible to this injectable antibiotic.

Quinolone antibiotics

Figure 2 shows the distribution of gonococci with altered susceptibility to quinolones nationally and by jurisdiction. Thus far, resistance to the quinolone antibiotics in *N. gonorrhoeae* is mediated only by chromosomal mechanisms so that incremental increases in MICs are observed. The AGSP uses ciprofloxacin as the representative quinolone and defines altered susceptibility as an MIC of 0.06 mg/L or more.[11] Treatment with currently recommended doses of 500 mg of ciprofloxacin is effective for strains with a lower level of resistance, viz. 0.06–0.5 mg/L, in about 90% of cases, but lower doses of the antibiotic will result in treatment failure more often. At higher levels of resistance i.e. an MIC of 1 mg/L or more, rates of failed treatment rise rapidly. At MIC levels of 4 mg/L or more treatment failure approaches 100%, even with higher ciprofloxacin doses.

Figure 2: Percentage of gonococcal isolates which were less sensitive to ciprofloxacin or with higher level ciprofloxacin resistance and all strains with altered quinolone susceptibility, Australia, 2008, by state or territory



LS QRNG MIC 0.06–0.5 mg/L.
R QRNG MIC 1 mg/L or more.

Nationally in 2008, 1,685 (54%) of gonococci examined had some level of resistance to quinolones (QRNG), again showing an increase over the 1,493, (49%) detected in 2007 and the 1,455 (37.8%) in 2006. In 2005, there were 1,190 (30.6%) QRNG reported compared with 825 (23.3%) in 2004. Most of the QRNG in 2008 (1,651 or 98.9%) had resistance at a higher level i.e. MICs ≥ 1mg/L and many of these had MIC levels of the order of 8–64 mg/L. High proportions of QRNG were seen in South Australia, where 304 QRNG represented 80% of all isolates examined. Victoria had 401 (71.2%) QRNG and New South Wales had 606 (71%). In Queensland (228 QRNG, 43.2%) and Western Australia (118 QRNG, 31.3%), lower proportions of QRNG were maintained than in the other mainland states. In other jurisdictions, the number of QRNG remained low with 18 in the Northern Territory, 8 in Tasmania, and 2 in the Australian Capital Territory.

Information on the acquisition of QRNG was available in 408 of the 1,685 (24%) cases. Three hundred and thirty-eight of these (83%) were acquired locally and 70 (17%) were acquired overseas. The most prominent overseas sources of QRNG acquisition were Thailand (17 cases), the Philippines (8) and Indonesia (Bali) (7). Others were from sources referred to under PPNG acquisition with contacts additionally reported in Egypt, Hong Kong, Germany, Poland and South Africa.

High-level tetracycline resistance

The spread of high-level tetracycline resistance in *N. gonorrhoeae* (TRNG) is examined as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea and are rarely, if ever, used for treatment of gonorrhoea in Australia. Despite this lack of use of this antibiotic group, the proportion of TRNG detected continues to increase. In 2006, 12% of isolates were TRNG. In 2007, 505 (16.6%) of gonococci examined were TRNG, the highest proportion of TRNG detected in this series at that time. In 2008, 553 (18%) of isolates tested were TRNG.

TRNG were present in all jurisdictions except the Australian Capital Territory, with the highest proportion in Western Australia (111 TRNG, 27%). Lower proportions of TRNG were present in New South Wales (172, 20.2%), Queensland (98, 18.6%), Victoria (95, 16.9%) and South Australia (32, 8.4%). There were 39 (10.1%) TRNG found in the Northern Territory and five in Tasmania.

Discussion

The World Health Organization has long-established strategies designed to optimise standardised treatment regimens for gonorrhoea on the basis of epidemiological surveys of the distribution and extent of gonococcal AMR[1] and these have been recently revised.[13] For public health purposes, AMR at a rate of 5% or more in gonococci sampled in a general population is the 'threshold for action' for removal of an antibiotic from treatment schedules and for substitution of another effective agent.[1,13] Programs such as the AGSP therefore seek to determine the proportion of strains in a defined patient population that are resistant to antibiotics relevant to the treatment of gonorrhoea and relate these findings to likely efficacy of current treatment schedules.[1,2,7,11,13] These strategies require quality AMR data, and the special requirements for *in vitro* growth and AMR testing of the fastidious *N. gonorrhoeae* complicate this requirement. One aspect that requires attention is the ability to obtain and examine a sufficient and representative sample of isolates.[1,11,13] The 3,110 strains examined by the AGSP and their source from public and private health sectors constitutes a comprehensive sample that meets these requirements despite the increasing use of nucleic acid amplification assays for diagnosis of gonorrhoea in Australia. The AGSP also distributes reference panels of gonococci for use in internal quality control and external quality assurance schemes[14] necessary for the validation of gonococcal AMR data.

The proportion of *N. gonorrhoeae* resistant to multiple antibiotics continued to increase in urban Australia in 2008, and nationally, approximately 44% of gonococci were resistant to the penicillins and 54% to the quinolone antibiotics, together with an historical high in Australia of the presence of gonococci with high-level tetracycline resistance. This increase in quinolone and tetracycline resistance occurred despite low exposure to these antibiotics in Australia.[2] The 'rural–urban divide'[2] in gonococcal resistance rates was maintained (Figures 1 and 2) insofar as remote areas in some jurisdictions with high disease rates continue to be able to use penicillin-based treatments. Effective use of this cheap and acceptable treatment requires continuing close monitoring of resistance patterns. This dichotomy also illustrates the need for disaggregated information rather than pooled national data to define treatment regimens appropriate for the various jurisdictions.

Specific comment has been made in recent reports regarding gonococci with decreased susceptibility to ceftriaxone.[2,10] In 2008, the number of these isolates remained low at about 1% of all isolates tested, but in most cases they were also resistant to quinolones and penicillins. However, the parameters for laboratory recognition of altered susceptibility to the cephalosporins are poorly defined and differ for the oral and injectable extended-spectrum cephalosporins. Proper comparisons of the frequency of their isolation in Australia and other countries are thus difficult to obtain at present. However, regional surveys and local studies have confirmed the wider distribution of these gonococci in countries in close proximity to Australia and also locally.[2,4,5]

The mechanism of resistance to ceftriaxone in these isolates is not fully elucidated, although alterations in the *penA* gene, including the presence of mosaic PBP2, are regarded as pivotal.[15,16] The presence of a mosaic PBP2 can be detected by molecular methods[17] and *N. gonorrhoeae* with this mosaic PBP2 are present in local isolates. Additionally, molecular typing methods have shown that multiple gonococcal sequence types (STs) may harbour these mosaic PBP2.[18] Gonococci of STs associated with treatment failures following cephalosporin therapy in Hong Kong have now been found in Australia, as have gonococci of other STs that also harbour mosaic PBP2. The presence of a mosaic PBP2-containing lesion does not however of itself equate with clinical resistance, and other gene polymorphisms are required to increase MIC levels to those that may impact on treatment efficacy.[16] Other non-mosaic allele changes in *penA* are also associated with ceftriaxone non-susceptibility, but have no impact on the equivalent oral agents. Additionally, ceftriaxone treatment failures have now been documented in pharyngeal gonorrhoea in Australia where the infection was due to gonococci with *penA* alterations other than those associated with a mosaic gene.[6]

Thus considerable further clarification is required regarding the laboratory detection of these gonococci and the interpretation of their likely clinical impact. AGSP reports have also consistently emphasised that the previous local recommendation for a minimum dose of 250 mg of ceftriaxone was prudent given the presence of these isolates and the propensity for resistance to develop in *N. gonorrhoeae*. It is thus reassuring to note that current treatment recommendations are for an increased 500 mg dose of ceftriaxone.

All gonococci tested in Australia in 2008, including those with altered cephalosporin susceptibility, were

susceptible to spectinomycin. A low proportion of gonococci was also found to be resistant to azithromycin in 2008. Azithromycin has been suggested as a possible component of treatment for gonorrhoea that uses dual antibiotic treatment.[19] Resistance to azithromycin, widely used as an anti-chlamydial agent in conjunction with gonococcal treatment, has been reported with increasing frequency overseas. MIC levels in azithromycin-resistant gonococci have reached very high levels in Europe, but these strains have not been detected in Australia.

These recent data showing emergence and spread of anti-microbial resistance in *N. gonorrhoeae* indicate a continuing need for surveillance of antimicrobial resistance in this organism both at a national and international level. The problems of emergence and spread of resistance are complex and require attention to both disease control as well as rational use of antibiotics.[8,15,20] A continuing commitment to surveillance of AMR in *N. gonorrhoeae,* which is an essential component of these control measures,[1,13] means that maintenance of culture-based systems will be required while this surveillance is still based on testing of gonococcal isolates.

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TRACHOMA SURVEILLANCE ANNUAL REPORT, 2008

A report by the National Trachoma Surveillance and Reporting Unit

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Abstract

The National Trachoma Surveillance and Reporting Unit has reported data for trachoma endemic regions and communities in the Northern Territory, South Australia and Western Australia for 2006 to 2008. Aboriginal children aged 1–9 years were examined using the World Health Organization grading criteria. Screening in the Northern Territory was conducted by the primary health care staff from the Healthy School Age Kids program, the Australian Government Emergency Intervention and Aboriginal Community Controlled Health Services. Forty-three of 92 communities in 6 regions were screened and reported data (2,462 children). In South Australia, the Eye Health and Chronic Disease Specialist Support Program and a team of eye specialists visited 11 of 72 communities in regions serviced by 6 Aboriginal Community Controlled Health Services (365 children). In Western Australia, population health unit and primary health care staff screened and reported data for 67 of 123 communities in 4 regions (1,823 children). Prevalence rates of active trachoma varied between the regions with reported prevalence ranging from 4%–67% in the Northern Territory, 0%–13% in South Australia and 8%–25% in Western Australia. Statistical comparisons must be viewed with caution due to the year-to-year variation in the coverage of children examined and the small numbers. Comparisons of 2006, 2007 and 2008 regional prevalence of active trachoma showed that many communities had no change in prevalence, though there were a few statistically significant increases and decreases ($P < 0.05$). The number of communities screened and the number of children examined has improved but still remains low for some regions. The implementation of the World Health Organization Surgery (for trichiasis), Antibiotics (with azithromycin), Facial cleanliness and Environmental improvement (SAFE) strategy has been variable. Few data continue to be reported for the surgery and environmental improvement components. In general, the availability of the community programs for surgery, antibiotic treatment, and facial cleanliness has improved. Reporting of antibiotic treatment has improved from 2006 to 2008. No significant changes were noted in bacterial resistance reported by pathology services from 2007 to 2008; these rates are comparable to national data collected by the Advisory Group on Antibiotic Resistance in 2005. *Commun Dis Intell* 2009;33(3):275–290.

Keywords: active trachoma, antibiotic resistance, facial cleanliness, Northern Territory, SAFE trachoma control strategy, South Australia, trachoma control activities, trachoma endemic, Western Australia

Introduction

This is the third report of the National Trachoma Surveillance and Reporting Unit (NTSRU). This report aims to compare 2008 data with results from the screening in 2006 and 2007 conducted in the Northern Territory, South Australia and Western Australia in regions with endemic trachoma.[1,2] It comments on jurisdictions' implementation of the Communicable Diseases Network Australia (CDNA) trachoma guidelines 'minimum best-practice approach', and makes recommendations regarding future reporting and management.[3]

Methods

Presented below is a summary of the data collection methods used by the jurisdictions and the data analysis and reporting methods used by the NTSRU. A detailed description of the 2008 report is included in the full 2008 report.[4]

Screening and data collection

Key representatives from each jurisdiction categorised communities that were 'At Risk' or 'Not At Risk' for trachoma, and further categorised each group into screened or not screened. Communities considered At Risk were determined using historical reports of trachoma in their regions. In most cases this did not include the large urban regions. For many communities in South Australia there was no information on prior screening for trachoma; these communities have been reported as At Risk and should have been screened.

According to the CDNA guidelines, screening should be conducted annually in Communities At Risk until prevalence of active trachoma is less than 5% for 5 consecutive years. The World Health Organization (WHO) has set the criteria for the elimination of blinding endemic trachoma in a community as being a prevalence of active trachoma greater than 5% in children aged 1–9 years or a prevalence of operable trichiasis of less than 0.1% in the population.

The WHO simplified trachoma grading system was used to report results of screening.[5] Active trachoma includes WHO grades trachomatous inflammation follicular (TF) and/or trachomatous inflammation intense (TI).

In brief, data were reported for prevalence of active trachoma, antibiotic treatment of children, their household contacts and community members, facial cleanliness, trachomatous trichiasis (TT) and surgery for trichiasis. The implementation of the Surgery, Antibiotics, Facial cleanliness and Environmental improvements (SAFE) components of the SAFE trachoma control strategy were also reported. This report focuses on the data for Aboriginal children aged 1–9 years and Aboriginal adults aged 30 years or more—unless otherwise specified—to comply with CDNA guidelines.

The NTSRU monitored antibiotic resistance in Aboriginal communities for 2 years (2007 and 2008). Three pathology services collected and reported data: the Institute of Medical Veterinary Science (IMVS), the Northern Territory Government Pathology Service (NTGPS) and the Western Diagnostics Pathology Service (WDPS). The participating laboratories and health services reported azithromycin resistance (defined as both intermediate and high level resistance) for any invasive and non-invasive isolates of *Streptococcus pneumoniae* specimens collected from Aboriginal people in trachoma endemic regions. Specimens were collected over a 6 month period in 2008 (1 July to 30 December) because too few results were reported within the 3 month collection period in 2007. Information on indigenous status was only reported from the NTGPS. IMVS and WDPS provided data for specimens from those regions or health services that predominately service Aboriginal people.

Northern Territory

Screening for trachoma was conducted between February and November 2008 in 6 regions. The Healthy School Age Kids (HSAK) program conducted most of the screening in the Top End and in Central Australia with collaboration with primary health care staff from the Aboriginal Community Controlled Health Services (ACCHS). In 2008 the HSAK program was fully implemented in Central Australia.

Screening was conducted in an Alice Springs town camp for the first time by the trachoma coordinator in conjunction with the Australian Government Emergency Intervention (AGEI) at the Central Australian Aboriginal Congress. Previously, Alice Springs town camps had not been screened because they were not regarded as At Risk for trachoma and because the HSAK program is responsible for remote areas.

In 2007, the AGEI conducted Child Health Checks throughout the Northern Territory. The AGEI clinical advisory panel decided that trachoma screening was only to be conducted by members of the intervention teams who had appropriate skills and training to do so. During Phase 2 of the AGEI in 2008, some children in the Northern Territory were examined for trachoma during the Child Health Check. The screening reported was not regarded as reliable or consistent by the Northern Territory authorities so has not been included in this report. The communities that were visited by the AGEI (n=14) were not revisited by the HSAK program and this contributed to the smaller number of communities reporting active trachoma data for 2008.

Ophthalmologists examined Aboriginal adults for trichiasis when they conducted outreach visits in the regions.

South Australia

Screening for trachoma was conducted between April and December 2008 in regions serviced by 6 ACCHSs. The Ceduna/Koonibba region includes communities in the Eyre school district (located south-east of the Ceduna/Koonibba Health Service). This incorporates communities serviced by the Port Lincoln ACCHS region where screening has not been conducted. The Pika Wiya region includes communities from within the Flinders school district, and 2 communities from the Northern Country school district, which were reassigned by the Eye Health and Chronic Disease Specialist Support Program (EH&CDSSP) coordinator. For this reason the Australian Bureau of Statistics Census data for the Aboriginal population in the Ceduna/Koonibba and Pika Wiya regions appear larger than what would be expected for some of these regions as serviced by the ACCHS.

In 2006 communities in regions serviced by Oak Valley ACCHS were reported with communities from the Tullawon ACCHS; these data have been combined together in Table 1 so comparisons can be made for each year between 2006 and 2008.

A widespread screening program was not implemented as the EH&CDSSP only visits selected communities serviced by some ACCHS. The project coordinator of the EH&CDSSP assisted a screening team of ophthalmologists and optometrists in recording information on active trachoma. Some communities were visited twice over a 1 year period, however only 1 round of data was reported. Aboriginal children who were identified for screening were seen in schools and others brought to the clinics by family members, Aboriginal health workers or clinic staff.

Table 1: Community coverage, screening coverage and active trachoma prevalence of Aboriginal children aged 1 to 9 years, 2006 to 2008, by state or territory, region and Aboriginal Community Controlled Health Service

State or territory and region	Number of Communities At Risk	Community coverage Number of communities screened (% of Communities At Risk)						Screening coverage Number of children examined (% of children in Communities At Risk)						Prevalence of active trachoma Children 1 to 9 years (% prevalence)					
	2008	2006		2007		2008		2006		2007		2008		2006		2007		2008	
	n	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Northern Territory																			
Alice Springs	1	–		–		1	100	–		–		45	22	–		–		18	40
Alice Springs Remote	30	25	83	19	63	18	60	530	35	231	15	459	29	94	18	46	20	157	34*
Barkly†	8	6	67	6	67	2	25	105	20	68	13	87	26	22	21	18	26	58	67*
Darwin Rural	16	15	94	12	75	11	69	522	27	377	19	907	45	84	16	25	7	183	20*
East Arnhem	12	12	100	12	100	4	33	879	78	465	41	232	20	22	3	23	5	10	4
Katherine†	20	11	52	11	52	7	35	218	12	562	31	732	50	65	30	104	19	287	39*
Total	87	69	78	60	67	43	49	2,254	33	1,703	24	2,462	36	287	13	216	13	713	29*
South Australia																			
Ceduna/Koonibba	21	1	5	1	5	1	5	18	1	16	1	121	6	1	6	1	6	0	0
Nganampa	10	8	80	4	40	6	60	27	8	76	23	167	50	5	19	10	13	4	2*
Oak Valley‡	2	2	100	2	100	2	100	28	108	34	131	25	93	7	25	7	21	2	8
Pika Wiya	33	5	15	–		1	3	51	1	–		37	1	6	12	–		0	0*
Umoona Tjutagku	6	1	17	1	17	1	17	6	7	2	2	15	17	1	17	0	0	0	0
Total	72	17	24	8	11	11	15	130	1	128	1	365	4	20	15	18	14	6	2*
Western Australia																			
Goldfields	20	6	30	10	50	13	65	231	24	227	23	238	23	43	19	8	4	18	8*
Kimberley†	34	28	82	25	83	32	94	1,048	51	1,006	58	1,169	55	192	18	164	16	175	15
Midwest	6	6	100	5	83	6	100	167	90	127	68	122	64	32	19	28	22	12	10*
Pilbara§	16	9	56	14	88	16	100	273	36	306	40	294	37	146	53	50	16	73	25*
Total	76	49	64	54	75	67	88	1,719	43	1,666	45	1,823	44	413	24	250	15	278	15*
Australia	235	135	57	122	52	121	51	4,103	21	3,497	18	4,650	23	720	18	484	14	997	21*

– Data not reported.

* $P < 0.05$ = statistical significant change found between 2006 and 2008 using chi-square test.

† Barkly had 9 Communities At Risk of trachoma in 2006 and 2007; Katherine had 21 Communities At Risk in 2006 and 2007; and the Kimberley had 30 Communities At Risk in 2007.

‡ Communities in regions serviced by the Oak Valley Aboriginal Community Controlled Health Services (ACCHS) were reported with communities from the Tullawon ACCHS.

§ Change in grading from 2007.

Source: Data were collected by the Healthy School Age Kids program in Northern Territory, the Eye Health and Chronic Disease Specialist Support Program in South Australia and population health units in Western Australia.

Data from the Pika Wiya region were collected by the mainstream Health Service and forwarded to the EH&CDSSP coordinator to be included in this report.

The screening team of eye specialists also visited ACCHS clinics twice in the year to examine adults for trichiasis.

Western Australia

Screening for trachoma was conducted between August and September 2008 in 4 regions. Population Health Units collected data in partnership with primary health care staff from state government ACCHS. In most regions letters were sent to parents in order to gain permission for the screening of their children.

Adults were examined for trichiasis as part of an annual influenza vaccination program.

Data analysis and reporting

Comparisons between jurisdictions need to be interpreted with caution because of the variation in methods, data collection and reporting.

In 2008, a community was defined as a group of people where there is a school; larger communities where two or more schools are located were counted as a single community instead of reporting data for each school separately. Community coverage was calculated using the number of communities that were screened as a proportion of those that were identified by each jurisdiction as At Risk. Communities that were reported as Not At Risk and were not screened are not included.

The 2006 Australian Bureau of Statistics (ABS) census data of the number of Aboriginal people resident in a region were used to calculate 2008 high and low series population projections.[6,7] Screening coverage was calculated using the number of children who were examined for trachoma in 2008 as a proportion of those who were estimated by the ABS to be resident in Communities At Risk.

The prevalence of active trachoma in Aboriginal children aged 1–9 years was calculated using the number of children examined as the denominator and 95% confidence intervals were calculated.

CDNA guidelines recommend providing azithromycin treatment to affected children, their households and community members. In some communities the treatment strategy was not reported, although some treatment was distributed. In other communities, treatment was reported to have been distributed where active trachoma was found in children aged 10–14 years without being detected in children aged 1–9 years. Where the data indicated that treatment was only given to affected children, without providing household or community treatment, these communities were regarded as not following the CDNA guidelines.

Comparisons must be viewed with caution due to the year-to-year variation in methods, data collection and reporting, and the small numbers of children examined. For comparisons to be made eligible communities had to report comparable data for at least 2 years. Chi-square tests were used to detect significant differences ($P < 0.05$) in the prevalence of active trachoma for communities that examined 10 or more children in two or more years. Where numbers were less than five in any cell, a Fisher's exact test was used. Analysis could not be conducted on 2006 data for 2 regions in Western Australia: the Kimberley region (where the number of children examined from each community was not reported) and the Pilbara region (where follicular trachoma was not graded according to the WHO grading system). In 2008, comparisons of prevalence of active trachoma were not possible for 4 of the 7 communities in the Katherine region of the Northern Territory where data were provided for children aged 0–15 years.

Results

National overview

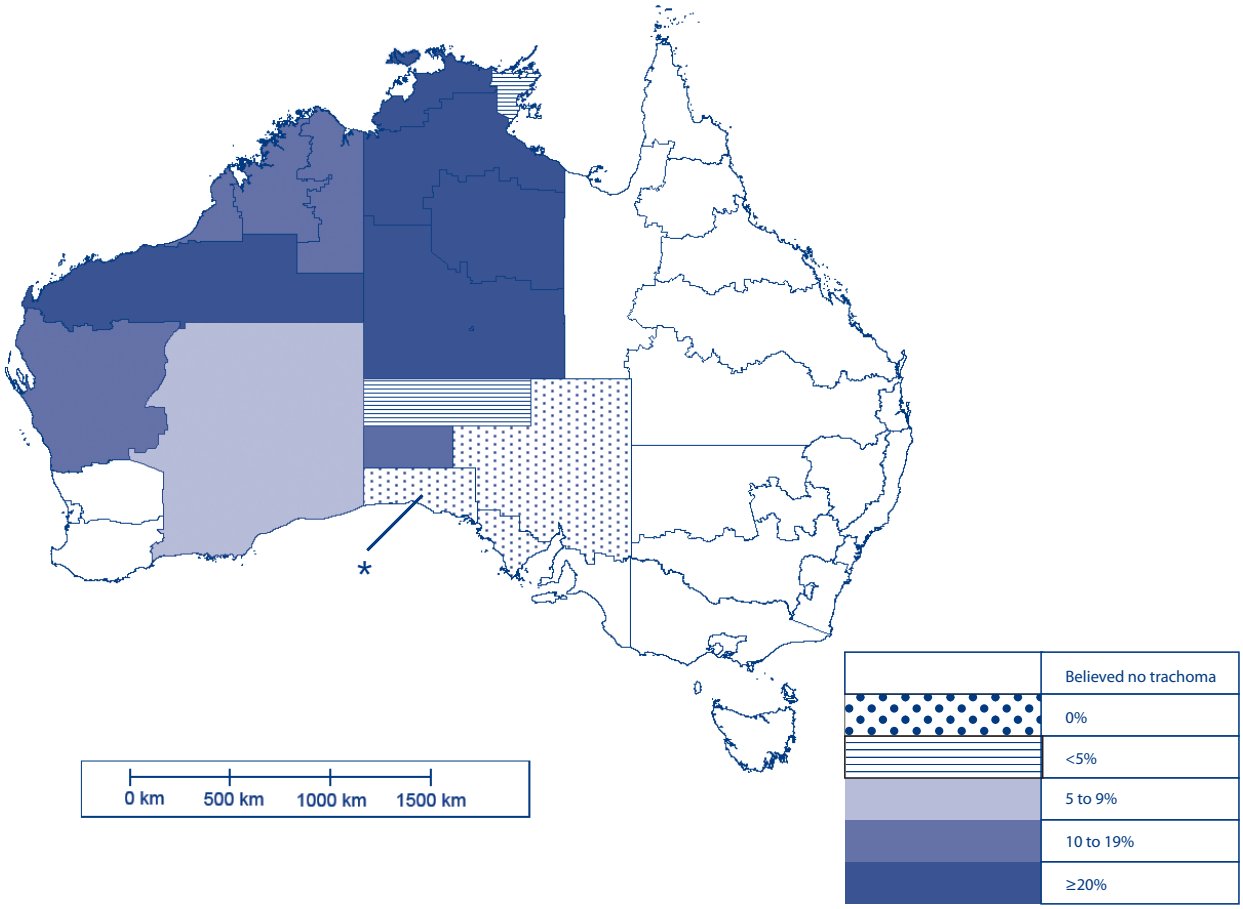
A total of 16 regions in the Northern Territory, South Australia and Western Australia conducted screening. Other jurisdictions were not included in this project (Map).

Data were reported for 121 of the 235 Communities At Risk (51%) in 2008 (Table 2). The overall prevalence of active trachoma in Aboriginal children aged 1–9 years for whom data were reported was 21% (Table 1). A total of 82 communities (68%) had a prevalence of active trachoma ≥ 5% (Table 3), and this occurred in 10 of the 15 regions (67%) (Table 1).

Of the communities where data were reported for the screening of active trachoma, facial cleanliness data were reported by 93 (76%) of 123 communities in 2007 and 108 (89%) of 121 in 2008. Many communities have tried to break the cycle of re-infection by promoting facial cleanliness through the use of programs (70%) and resources (49%) (Table 4).

Treatment was reported to have been distributed in 90 (92%) of the 98 communities in which treatment for trachoma was indicated (Table 5), including 4 communities where active trachoma was found in children aged 10–14 years. Overall, 76 (78%) communities were treated according to CDNA guidelines, this included children found to have active trachoma, their household contacts and community members.

Map: Prevalence of active trachoma in Australia, 2008, by region



* No active trachoma was found, however few children were examined in this area (n=9).

Table 2: Number of communities screened for trachoma by trachoma risk, state and territory, 2008

Communities	Number and percentage of communities						Total	
	Northern Territory		South Australia		Western Australia			
	n	%	n	%	n	%	n	%
Not At Risk								
Screened	0		0		0		0	
Not screened	5	100	0		47	100	52	100
Total Not At Risk	5		0		47		52	
At Risk								
Screened with no trachoma found	4	5	7	10	16	21	27	11
Screened with trachoma found	39	45	4	6	51	67	94	40
Reported screened but no data received	19	22	0		2	3	21	9
Not screened	25	29	61	85	7	9	93	40
Total At Risk	87		72		76		235	
Total communities	92		72		123			

Community coverage varied between each state and territory with higher coverage in Western Australia and consistently low coverage in South Australia (Table 1). Comparisons must be viewed with caution due to the year-to-year variation in methods, data collection and reporting, and the small num-

Table 3: Community prevalence of active trachoma in Aboriginal children aged 1 to 9 years, 2006 to 2008, by state or territory

Community prevalence	Number and percentage of communities where trachoma data were reported						Total	
	Northern Territory		South Australia		Western Australia			
	n	%	n	%	n	%	n	%
2006 data								
0	30	42	0		5	9	35	26
1 to <5	7	10	0		3	6	10	8
5 to <10	7	10	2	25	8	15	17	13
10 to <20	6	8	3	38	6	11	15	11
20 to <50	12	17	3	38	19	36	34	26
≥ 50	10	14	0		12	23	22	17
Total	72	100	8	100	53	100	133	100
2007 data								
0	29	48	2	25	20	36	51	41
1 to <5	7	12	0		0		7	6
5 to <10	4	7	2	25	5	9	11	9
10 to <20	8	13	2	25	12	22	22	18
20 to <50	11	18	2	25	16	29	29	24
≥ 50	1	2	0		2	4	3	2
Total	60	100	8	100	55	100	123	100
2008 data								
0	4	9	7	64	16	24	27	22
1 to <5	4	9	1	9	7	10	12	10
5 to <10	4	9	2	18	8	12	14	12
10 to <20	6	14	1	9	7	10	14	12
20 to <50	16	37	0		21	31	37	31
≥ 50	9	21	0		8	12	17	14
Total	43	100	11	100	67	100	121	100

Table 4: Implementation of trachoma control activities (SAFE strategy), 2008, by state or territory

SAFE trachoma control activities	Number and percentage of communities where trachoma control activities were reported						Total	
	Northern Territory		South Australia		Western Australia			
	n	%	n	%	n	%	n	%
Surgery referral process for trichiasis available	39	91	4	36	26	39	69	57
Antibiotics distributed	35	81	5	45	50	75	90	74
Facial cleanliness resources used	27	63	1	9	31	46	59	49
Facial cleanliness programs implemented	32	74	0		53	79	85	70
Good environmental condition	2	5	0		9	13	11	9
Total number of communities where trachoma screening data were reported	43		11		67		121	

bers of children examined. A comparison between 2006 and 2008 regional prevalence data found there was no change in prevalence in 6 regions, a statistically significant increase ($P < 0.05$) in prevalence was found in 4 regions, and a decrease ($P < 0.05$) in 5 regions (Table 1). Of the 77 communities where comparable data were provided, 53 (69%) communities had no significant change (Table 6).

Trichiasis screening was carried out only in a small proportion of Communities At Risk (62/235), but the overall prevalence in the adults examined was 4% (52/1,407) (Table 7, 8 and 9). Not all communities where data were reported are implementing the components of the SAFE strategy according to the CDNA guidelines, but antibiotic treatment and facial cleanliness programs are being reported by the majority, 74% (90/121) and 70% (85/121) respectively (Table 4).

Northern Territory

Of the 92 communities in 6 regions of the Northern Territory, 87 (95%) communities were categorised as being At Risk for trachoma (Table 2). Included in these communities was a community (town camp) in Alice Springs, previously categorised Not At Risk. After finding a 40% prevalence of active trachoma in children, this community was then re-categorised as At Risk (Table 1). Of the 62 (71%) communities that were screened in 2008, data were reported from 43 (69%) (Table 2). Four (9%) communities had no active trachoma while 35 (81%) had a prevalence of active trachoma of ≥ 5% (Table 3).

Table 5: Reported treatment for trachoma, 2008, by state or territory

Communities	Northern Territory		South Australia		Western Australia		Total	
	n	%	n	%	n	%	n	%
Treated in compliance with CDNA guidelines*								
Community-based	9	26	0		20	49	29	38
Household-based	19	54	0		21	51	40	53
Strategy not reported†	7	20	0		0		7	9
Total treated	35		0		41		76	
Not treated in compliance with CDNA guidelines								
Children only	0		5	100	9	82	14	64
No treatment reported	6	100	0		2	18	8	36
Total not following CDNA	6		5		11		22	
Total communities	41		5		52		98	

* Includes 2 communities in the Northern Territory, 1 in South Australia and 1 in Western Australia, where active trachoma was found in children aged 10 to 14 years without being detected in children aged 1 to 9 years.

† Communities carried out treatment but the strategy was not reported.

The Communicable Diseases Network Australia (CDNA) guidelines recommend that treatment of children and household or community contacts aged over 6 months be completed in as short a time frame as possible where population mobility is high.

Table 6: Changes in the prevalence of active trachoma in Aboriginal children aged 1 to 9 years in communities where ≥10 children were examined, 2006 to 2008, by state or territory

Change in prevalence of active trachoma 2006–2008	State or territory						Total	
	Northern Territory		South Australia		Western Australia			
	n	%	n	%	n	%	n	%
Significant decrease*	2	6	0		8	21	10	13
No change	21	64	6	100	26	68	53	69
Significant increase*	10	30	0		4	11	14	18
Total communities	33		6		38		77	

* Fisher's test used to evaluate change; significant at P < 0.05.

Source: Data were collected by the Healthy School Age Kids program in the Northern Territory, the Eye Health and Chronic Disease Specialist Support Program coordinator and the screening team in South Australia, and population health units in Western Australia.

Table 7: Trichiasis screening reported for Aboriginal adults aged ≥ 30 years in the Northern Territory, 2008, by region

	Alice Springs	Alice Springs Remote	Barkly	Darwin Rural	East Arnhem	Katherine	Total
Regional population (ABS)							
Adults resident:							
In region*	1,838	3,521	1,301	3,297	3,309	3,041	16,307
In Communities At Risk*	514	3,010	542	3,173	2,315	2,038	11,592
Trichiasis							
Communities from which data were reported/Communities At Risk	0/1	12/30	2/8	0/16	0/12	0/20	14/87
Adults examined	–	183	23	–	–	–	206
With trichiasis	–	23	3	–	–	–	26
Prevalence of trichiasis (%)	–	13	13	–	–	–	13
Trichiasis surgery within 12 months prior to the date of reporting	–	36	3	7	–	–	46

– Data not reported.

* Projected 2008 population data for the whole region based on the Australian Bureau of Statistics (ABS) 1.4% low series population growth rate in the Northern Territory.

Table 8: Trichiasis screening reported for Aboriginal adults aged ≥30 years in South Australia, 2008, by Aboriginal Community Controlled Health Service

	Ceduna/ Koonibba*	Nganampa	Oak Valley (Maralinga Tjarutja)	Pika Wiya†	Tullawon	Umoona Tjutagku	Total
Regional population (ABS)							
Adults resident							
In region‡	3,568	673	34	11,772	28	206	16,281
In Communities At Risk‡	3,568	673	34	11,772	28	206	16,281
Trichiasis							
Communities from which data were reported/ Communities At Risk	0/21	6/10	0/1	1/33	0/1	1/6	8/72
Adults examined	–	221	–	26	–	51	298
With trichiasis	–	1	–	0	–	0	1
Prevalence of trichiasis (%)	–	0.5	–	0	–	0	0.3
Trichiasis surgery within 12 months prior to the date of reporting	–	1	–		–		1

– Data not reported.

* Regional population data of Aboriginal adults and the number of Communities At Risk include adults and communities in the Eyre school district in South Australia and incorporates those serviced by the Port Lincoln Aboriginal Community Controlled Health Service region where screening has not been conducted.

† Regional population data of Aboriginal adults, and the number of Communities At Risk, include adults and communities in the Flinders school district in South Australia and two communities from the Northern Country school district which were reassigned by the Eye Health and Chronic Disease Specialist Support Program coordinator.

‡ Projected 2008 population data for the whole region based on the Australian Bureau of Statistics (ABS) 1.9% low series population growth rate in South Australia.

All communities in South Australia were considered At Risk, therefore the number of adults resident in the region and in Communities At Risk is the same.

Table 9: Trichiasis screening reported for Aboriginal adults aged ≥ 30 years in Western Australia, 2008, by region

	Goldfields	Kimberley	Midwest	Pilbara	Total
Regional population (ABS)					
Adults resident:					
In region*	2,063	3,551	2,185	2,384	10,183
In Communities At Risk*	1,761	3,321	406	1,585	7,073
Trichiasis					
Communities from which data were reported/ Communities At Risk	11/20	15/34	5/6	9/16	40/76
Adults examined	67	442	210	184	903
With trichiasis	3	21	1	0	25
Prevalence of trichiasis (%)	4	5	0.5	0	3
Trichiasis surgery within 12 months prior to the date of reporting	–	2	–		2

– Data not reported.

* Projected 2008 population data for the whole region based on the Australian Bureau of Statistics (ABS) 1.8% low series population growth rate in Western Australia.

Of the 6,747 children aged 1–9 years reported by the ABS to be resident in Communities At Risk (Table 10), 2,462 (36%) were examined for trachoma, and 713 had active trachoma (prevalence = 29%, 95% CI, 27%–31%) (Table 1). Comparisons for prevalence of active trachoma were not possible for four of the 7 communities in the Katherine region, where data were provided for children aged 0–15 years instead of 1–9 years, and age breakdowns were not provided. Of the 1,493 children examined for facial cleanliness, 1,004 (67%) had clean faces (Table 10).

Treatment was reported to have been distributed according to the CDNA guidelines in 35 (85%) of the 41 communities in which treatment for trachoma was indicated (Table 5). This included 2 communities where active trachoma was found in children aged 10–14 years.

Comparison of prevalence of active trachoma was made for 33 of 64 communities where data were reported for at least two of the years between 2006 and 2008. No change in prevalence was found in 21 (64%) communities, a statistically significant increase ($P < 0.05$) was found in 10 (30%) and a decrease ($P < 0.05$) was found in 2 (6%) (Table 6).

Data on trichiasis were reported for the Alice Springs Remote and Barkly regions only and 26 Aboriginal adults (13%) aged ≥ 30 years were found to have

Table 10: Number of resident Aboriginal children aged 1 to 9 years, and number of communities and children examined for facial cleanliness in the Northern Territory, 2008, by region

	Alice Springs	Alice Springs Remote	Barkly	Darwin Rural	East Arnhem	Katherine	Total
Regional population (ABS)							
Children resident:							
In region*	954	1,817	661	2,146	1,916	1,991	9,485
In Communities At Risk*	201	1,577	341	2,013	1,155	1,460	6,747
Facial cleanliness							
Communities from which data were reported/ Communities At Risk	1/1	18/30	2/8	5/16	2/12	2/20	30/87
Children examined	45	468	87	627	133	133	1,493
Prevalence of clean faces (%)	12	47	48	79	84	89	67

* Projected 2008 population data for the whole region based on the Australian Bureau of Statistics (ABS) 1.4% low series population growth rate in the Northern Territory.

trichiasis (Table 7). Forty-six adults were reported to have undergone surgery for trichiasis within 12 months prior to the date of reporting.

South Australia

Of the 72 communities in 6 ACCHS of South Australia, all were categorised as being At Risk for trachoma, of which 11 communities (15%) were visited and data reported (Table 2). Seven (64%) communities had no active trachoma while 3 (27%) had a prevalence of active trachoma of ≥ 5% (Table 3).

Of the 9,218 children aged 1–9 years reported by the ABS to be resident in Communities At Risk (Table 11), 365 (4%) were examined for trachoma, and 6 had active trachoma (prevalence = 2%, 95% CI, 1%–4%) (Table 1); 260 (71%) had clean faces (Table 11).

Treatment was reported to have been distributed in all five of the communities in which treatment for trachoma was indicated, including 1 community where active trachoma was found in 1 child aged 10–14 years without being detected in children aged 1–9 years (Table 5). Treatment was given to 7 children (6 aged 1–9 and another aged 14 years) who were examined and found to have active trachoma. CDNA treatment guidelines were not followed as household treatment was not given irrespective of the presence of trachoma. This was similar to 2006 and 2007.

Comparison of prevalence of active trachoma was made for 6 of 11 communities where data were reported for at least two of the years between 2006 and 2008, however no statistically significant changes were found (Table 6).

Data for trichiasis were reported for three of the 6 ACCHSs only, and 1 (0.3%) adult was found to have trichiasis. One adult was reported to have undergone surgery for trichiasis within 12 months prior to the date of reporting (Table 8).

Western Australia

Of the 123 communities in 4 regions of Western Australia, 76 (62%) communities were categorised as being At Risk for trachoma, of which 69 (91%) were screened in 2008 (Table 2). Data were reported from 67 (97%) of these communities. Sixteen (24%) communities had no active trachoma while 44 (66%) had a prevalence of active trachoma ≥ 5% (Table 3).

Of the 4,112 children aged 1–9 years reported by the ABS to be resident in Communities At Risk (Table 12), 1,823 (44%) were examined for

Table 11: Number of resident Aboriginal children aged 1 to 9 years and number of communities and children examined for facial cleanliness in South Australia, 2008, by Aboriginal Community Controlled Health Service

	Ceduna/ Koonibba*	Nganampa	Oak Valley (Maralinga Tjarutja)	Pika Wiya†	Tullawon	Umoona Tjutagku	Total
Regional population (ABS)							
Children resident:							
In region‡	2,083	334	9	6,687	18	87	9,218
In Communities At Risk‡	2,083	334	9	6,687	18	87	9,218
Facial cleanliness							
Communities from which data were reported/ Communities At Risk	1/21	6/10	1/1	1/33	1/1	1/6	11/72
Children examined	121	167	16	37	9	15	365
Prevalence of clean faces (%)	100	47	0	100	100	100	71

* Regional population data of Aboriginal children and the number of Communities At Risk include children and communities in the Eyre school district in South Australia, and incorporates those serviced by the Port Lincoln Aboriginal Community Controlled Health Service region where screening has not been conducted.

† Regional population data of Aboriginal children and the number of Communities At Risk include children and communities in the Flinders school district in South Australia and 2 communities from the Northern Country school district, which were reassigned by the Eye Health and Chronic Disease Specialist Support Program coordinator.

‡ Projected 2008 population data for the whole region based on the Australian Bureau of Statistics (ABS) 1.9% low series population growth rate in South Australia.

All communities in South Australia were considered At Risk, therefore the number of children resident in the region and in Communities At Risk is the same.

trachoma, and 278 had active trachoma (prevalence = 15%, 95% CI, 13%–17%) (Table 1). Of the 1,833 children examined for facial cleanliness, 1,433 (78%) had clean faces (Table 12).

Treatment was reported to have been distributed according to the CDNA guidelines in 41 of the 52 communities (79%) in which treatment for trachoma was indicated (Table 5), including one community where active trachoma was found in children aged 10–14 years.

Comparisons must be viewed with caution due to the year-to-year variation in methods, data collection and reporting, and the small numbers of children examined. Comparison of prevalence of active trachoma was made for 38 out of 62 communities where data were reported for at least two of the years between 2006 and 2008; 11 between 2006 and 2008, and 27 between 2007 and 2008. No change in prevalence was found in 26 communities (68%), a statistically significant increase ($P < 0.05$) was found in 4 (11%) and a decrease ($P < 0.05$) was found in 8 (21%) (Table 6).

Data on trichiasis were reported for all 4 regions, and 25 adults (3%) were found to have trichiasis. Two adults were reported to have undergone surgery for trichiasis within 12 months prior to the date of reporting (Table 9).

Antibiotic resistance

The reporting of azithromycin antibiotic treatment in trachoma endemic jurisdictions has improved from 2006 to 2008 (Table 13).

Overall, 53 of the 261 *S. pneumoniae* isolates (20.3%, 95% CI, 16%–26%) were reported to be resistant or have intermediate resistance to azithromycin (Table 14).

Table 12: Number of resident Aboriginal children aged 1 to 9 years, and number of communities and children examined for facial cleanliness in Western Australia, 2008, by region

	Goldfields	Kimberley	Midwest	Pilbara	Total
Regional population (ABS)					
Children resident:					
In region*	1,184	2,875	1,239	1,199	6,497
In Communities At Risk*	1,017	2,116	192	787	4,112
Facial cleanliness					
Communities from which data were reported/ Communities At Risk	13/20	32/34	6/6	16/16	67/76
Children examined	235	1,182	122	294	1,833
Prevalence of clean faces (%)	72	81	82	72	78

* Projected 2008 population data for the whole region based on the Australian Bureau of Statistics (ABS) 1.8% low series population growth rate in Western Australia.

Table 13: Percentage of people treated with azithromycin (total treated/total requiring treatment) in jurisdictions where trachoma is regarded as endemic, 2006 to 2008

	2006*		2007		2008†	
Northern Territory	–/287		328/533	(62%)	3,069/4,860	(63%)
South Australia‡	19/20	(95%)	18/18	(100%)	7/7	(100%)
Western Australia§	396/471	(84%)	1,675/2,084	(80%)	2,917/3,013	(97%)
Total	415/778	(53%)	2,235/2,635	(85%)	5,993/7,880	(76%)

– Data not reported.

* No jurisdiction reported the number of household or community contacts treated.

† An additional 871 people were treated in 4 communities in the Katherine region (Northern Territory), they have not been included in the total because the number of people requiring treatment was not provided.

‡ Number of children found to have active trachoma at the first screening have been reported, no household or community contacts were treated irrespective of the presence of trachoma.

§ Treatment data were reported for only two out of the 4 regions in 2006.

Table 14: Azithromycin resistance and susceptibility to *Streptococcus pneumoniae* isolates collected from Aboriginal people, 2008, by pathology service and region

Pathology service/ region	Number and percentage of isolates						Total	%
	Resistant	%	Intermediate	%	Susceptible	%		
Institute of Medical Veterinary Science								
Goldfields	0		0		1	100	1	100
Nganampa	3	27	0		8	73	11	100
Pika Wiya	0		0		2	100	2	100
Subtotal	3	21	0		11	79	14	100
Northern Territory Government Pathology Service								
Alice Springs	11	38	1	3	17	59	29	100
Alice Springs Remote	11	30	0		26	70	37	100
Darwin	1	5	0		18	95	19	100
Darwin Rural	4	40	0		6	60	10	100
East Arnhem	3	27	0		8	73	11	100
Goldfields	1	50	0		1	50	2	100
Katherine	3	16	0		16	84	19	100
Kimberley	0		0		1	100	1	100
Nganampa	1	14	0		6	86	7	100
Queensland	0		0		1	100	1	100
Unknown	0		0		5	100	5	100
Subtotal	35	25	1	1	105	74	141	100
Western Diagnostics Pathology Service								
Alice Springs	1	100	0		0		1	100
Alice Springs Remote	4	20	0		16	80	20	100
Darwin	2	10	0		19	90	21	100
Darwin Rural	4	12	0		29	89	33	100
East Arnhem	2	10	0		18	90	20	100
Katherine	1	10	0		9	90	10	100
Perth	0		0		1	100	1	100
Subtotal	14	13	0		92	87	106	100
Total	52	20	1	0.4	208	80	261	100

The 27.4% (95% CI, 18%–40%) and 20.3% (95% CI, 16%–26%) resistance found in isolates reported in this study in 2007 and 2008 are comparable to the 22.7% (95% CI, 21%–25%) resistance found in isolates in Australia reported in the Australian Group on Antimicrobial Resistance survey in 2005 (Table 15).[8] No detectable increase in resistance to azithromycin in the *S. pneumoniae* bacteria was found.

Discussion

Surveillance data presented to the NTSRU clearly indicate that trachoma is still endemic in Australia.

In 2008, 235 of the 287 (82%) communities in the Northern Territory, South Australia and Western Australia were categorised as being At Risk for trachoma in 16 regions. A similar proportion of communities were screened in 2008 (51%) compared with 2006 (57%) and 2007 (52%). However, Western Australia showed a significant increase ($P < 0.05$) in community coverage, 64% in 2006 and 88% in 2008, and Northern Territory showed a significant decrease ($P < 0.05$) in community coverage, 78% in 2006 and 49% in 2008. The community coverage in South Australia has been consistently lower than the other jurisdictions; there was no significant difference between the community coverage in 2006 (24%) and 2008 (15%). A trachoma workshop organised for April 2009 discussed ways in which screening can be implemented to maximise the number of communities screened and the number of children examined in South Australia.

Table 15: Comparison of azithromycin resistance (resistant and intermediate) to invasive and non-invasive *Streptococcus pneumoniae* isolates collected from Aboriginal people (number resistant/total tested), 2005 to 2008, by state or territory

State or territory	AGAR monitoring		NTSRU monitoring			
	2005		2007		2008	
	%	Number resistant/ total tested	%	Number resistant/ total tested	%	Number resistant/ total tested
New South Wales/ACT	27.8	162/583		NR		NR
Northern Territory		NR	23.4	11/47	20.9	48/230
Queensland	28.2	80/284		NR	0	0/1
South Australia	20.9	82/392	40.0	6/15	20.0	4/20
Victoria	14.5	35/221		NR		NR
Western Australia	16.2	48/296		NR	20.0	1/5
Unknown		0		0	0	0/5
Australia	22.7	404/1,776	27.4	17/62	20.3	53/261
(95%CI)	21,25		18,40		16,26	

AGAR Australian Group on Antimicrobial Resistance.
NR Not reported.

The decrease in community coverage in the Northern Territory was in part due to the non-inclusion of communities examined during the AGEI. Data collected by the AGEI have been presented with caution in government reports due to limited training of staff collecting the data. For this reason they have not been presented in this report. The HSAK program, which provided data for other Northern Territory communities, did not re-visit communities screened by the AGEI.

In the years 2006 to 2008 there has been much discussion regarding the best way to report screening coverage of children. The NTSRU has explored the reporting of ABS regional population data of resident children, ABS population data for children resident in Communities At Risk, and the estimated number of children in communities where screening was conducted as provided by health care workers in the communities. Community statements of the children resident in each community vary compared with the ABS data, in part due to the high mobility of Aboriginal people. The ABS population data for children resident in Communities At Risk of trachoma was used to enumerate the children in communities where screening should have been conducted but was not. For example, in South Australia, the majority of communities have not been screened so it is not known whether they have trachoma or not; by including the number of children in these communities there will be a better understanding of the number of children who are not being examined. There were increases and decreases in the regional screening coverage, although the overall coverage for each jurisdiction was similar across the three years. Less than half of the children residing in Communities At Risk are being examined, emphasising that there are still many gaps in the screening.

In each jurisdiction there are regions with endemic trachoma. Across all the jurisdictions the average prevalence of active trachoma in communities from which data were reported, was 21% compared with the 14% reported for 2007 ($P < 0.05$) but there are no consistent changes in regional prevalence. Caution must be exercised due to variable coverage and small numbers. The majority of communities, 53 (69%) of the 77 where comparisons could be made, showed no change. Overall, a decrease ($P < 0.05$) in prevalence was found in South Australia (15% in 2006 and 2% in 2008) and Western Australia (24% in 2006 and 15% in 2008), and an increase ($P < 0.05$) was found in the Northern Territory (13% in 2006 and 29% in 2008). Reports of no active trachoma within some South Australia ACCHS should also be taken with caution because in many of these regions only very small numbers of children were examined.

Screening all children and providing azithromycin treatment as appropriate to household and community members is a necessary component of trachoma control. The surveillance data indicate that household and community treatment has improved from 2006 to 2008 according to the CDNA guidelines and possibly due to the operation of the NTSRU. Treatment was reported to have been distributed according to the CDNA guidelines in 35 communities (85%) in the Northern Territory and 41 (79%) in Western Australia; most of the regions within these

jurisdictions treated more than 80% of the people who required treatment. However, South Australia has consistently examined few children at the schools and continues to treat children found to have active trachoma without providing household or community treatment. Family members can cause a cycle of ongoing re-infection. The issue of re-infection is something that has been considered by CDNA that proposes cross-regional and cross-state treatment where people are known to move frequently across borders because of strong family/cultural links.[3] Western Australia has implemented this coordinated approach to treatment distributing azithromycin to most people who required treatment.

Poor facial hygiene is an important risk factor for trachoma and the promotion of facial cleanliness is a key component of the SAFE strategy. This has improved in 2008, with many communities reporting the promotion of facial cleanliness through the use of programs and resources to integrate behavioural change regarding hygiene.

The reporting of trichiasis data has improved, although still only 4% of those At Risk were examined. In 2007, only Western Australia reported the systematic screening for trichiasis. In 2008, almost every region reported data on trichiasis screening although in many regions this was still incomplete. The inclusion of trichiasis screening into existing programs such as the Adult Health Check and influenza vaccination programs has made it possible to assess the later stages of trachoma. This should lead to appropriate referrals for surgery when trichiasis is identified.

Improvements have been made for the reporting of the Surgery, Antibiotics and Facial Cleanliness activities of the SAFE trachoma control strategy, however these components still need to be strengthened. In 2008, 57% of communities reported having an existing referral process for trichiasis surgery for adults (Table 4), a marked increase compared with the 4% that reported this information in 2007.[2] In 2008, three quarters (74%) of the communities distributed antibiotics to children with active trachoma, but not necessarily as needed to their household and community contacts (Table 4). It is apparent that activities for the Environmental Improvement component of the SAFE strategy have either not been comprehensively implemented or reported. However, there have been reports of the installation of new swimming pools in some of the remote Aboriginal communities. While research has shown considerable health and social benefits of the pools, efforts should also be made to improve housing sanitation, nutrition, education and access to health care.[9]

While the reporting of treatment has improved from 2006 to 2008, no change in antibiotic resistance of *S. pneumoniae* has been detected over this time. It was not possible to make comparisons for Western Australia as PathWest pathology service was not able to provide the NTSRU with antibiotic resistance data in either year due to difficulties in obtaining the necessary clearances.

The trachoma surveillance process has enabled key representatives involved in trachoma programs from each jurisdiction to share successes and ideas relating to trachoma screening and management. A cross-regional 'health blitz' focusing on outreach screening and treatment of multiple conditions has been discussed by some jurisdictions. This will assist with the collection of data from communities that share borders while also aiming to deal with the cycle of re-infection caused by population mobility.

Future control activities in all jurisdictions would benefit from incorporating simple health messages such as keep your face clean as part of existing programs aimed at children and families. Future activities should also consider the responsibilities of members of the screening teams. For example, an efficient team might include at least 2 people responsible for the examination of trachoma, a nurse to administer treatment, and a health worker to assist in engaging with the community. It is important for all health workers and organisations involved in the monitoring of trachoma to be accountable and to take responsibility for their roles.

In summary, jurisdictions have attempted to collate data from both state based and independent data collection authorities where trachoma is still thought to be present. There are still gaps and limitations in the reporting of data, however considerable improvements have been made over the last 3 years. Recommendations for the future include reviewing assumptions that Aboriginal children in urban communities are Not At Risk, screening all Communities At Risk, examining at least all children aged 5 to 9 years in these communities, whether they are attending school or not, and strengthening the implementation of trachoma control activities. Additional effort is required to ensure that azithromycin is appropriately distributed, facial cleanliness is actively promoted and that adults with trichiasis are detected and operated on. An increase in community and screening coverage will enable more stable and reliable estimates of the prevalence and distribution of trachoma, and strengthening the implementation of all four components of the WHO SAFE strategy will lead to the elimination of blinding endemic trachoma.

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The organisations that collected and/or reported data were:

Northern Territory

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Australian Government Emergency Intervention

Centre for Disease Control, Northern Territory Department of Health and Families, Northern Territory

Healthy School Age Kids program: Top End and Central Australia

South Australia

Aboriginal Health Council of South Australia, Eye Health and Chronic Disease Specialist Support Program

Country Health South Australia

Ceduna/Koonibba Health Service

Nganampa Health Council

Oak Valley (Maralinga Tjarutja) Health Service

Pika Wiya Health Service

Tullawon Health Service

Umoona Tjutagku Health Service

Western Australia

Aboriginal Community Controlled Health Services staff

Communicable Diseases Control Directorate, Western Australian Department of Health

Goldfields Population Health Unit

Kimberley Population Health Unit

Midwest Population Health Unit

Pilbara regions Population Health Unit

Antibiotic resistance

Institute of Medical Veterinary Science

Northern Territory Government Pathology Service

Western Diagnostics Pathology Service

National Trachoma Surveillance Reference Group

The NTSRU is advised by the National Trachoma Surveillance Reference Group, members of which include representatives from the following organisations:

Centre for Disease Control, Alice Springs, Northern Territory Department of Health and Families

Centre for Disease Control, Darwin, Northern Territory Department of Health and Families

Communicable Diseases Control Directorate, Western Australian Department of Health

Country Health South Australia

Eye Health and Chronic Disease Specialist Support Program, Aboriginal Health Council of South Australia

Kimberley Population Health Unit, Western Australia

National Aboriginal Community Controlled Health Organisation

Office for Aboriginal and Torres Strait Islander Health, Australian Government Department of Health and Ageing

Surveillance Policy and Systems Section, Office of Health Protection, Australian Government Department of Health and Ageing

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Abbreviations

ABS	Australian Bureau of Statistics
ACCHS	Aboriginal Community Controlled Health Service(s)
AGEI	Australian Government Emergency Intervention
AHCSA	Aboriginal Health Council of South Australia
CDNA	Communicable Diseases Network Australia
CI	confidence interval
EH&CDSSP	Eye Health and Chronic Disease Specialist Support Program
HSAK	Healthy School Age Kids program
IMVS	Institute of Medical Veterinary Science
NR	not reported
NTGPS	Northern Territory Government Pathology Service
NTSRU	National Trachoma Surveillance and Reporting Unit
SAFE	Surgery, Antibiotics, Facial cleanliness, and Environmental improvement
TF	Trachomatous inflammation – follicular
TI	Trachomatous inflammation – intense
TT	Trachomatous trichiasis
WDPS	Western Diagnostics Pathology Service
WHO	World Health Organization

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Annual report of the Australian National Poliovirus Reference Laboratory, 2008

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Abstract

The Australian National Poliovirus Reference Laboratory (NPRL) is accredited by the World Health Organization (WHO) for the testing of stool specimens from cases of acute flaccid paralysis (AFP), a major clinical presentation of poliovirus infection. The NPRL, in collaboration with the Australian Paediatric Surveillance Unit, co-ordinates surveillance for cases of AFP in children in Australia, according to criteria recommended by the WHO. Clinical specimens are referred from AFP cases in children and suspected case of poliomyelitis in persons of any age. The WHO AFP surveillance performance indicator for a polio-free country such as Australia, is 1 non-polio AFP case per 100,000 children less than 15 years of age. In 2008, the Polio Expert Committee (PEC) classified 62 cases as non-polio AFP, or 1.51 non-polio AFP cases per 100,000 children aged less than 15 years. Poliovirus infection is confirmed by virus culture of stool specimens from AFP cases as other conditions that present with acute paralysis can mimic polio. While no poliovirus was reported in Australia from any source in 2008, the non-polio enteroviruses echovirus 25, coxsackievirus B2 and echovirus 11 were isolated from stool specimens of AFP cases. The last report of a wild poliovirus in Australia was due to an importation from Pakistan in 2007. With 4 countries remaining endemic for poliomyelitis–Afghanistan, India, Nigeria and Pakistan–and more than 1,600 confirmed cases of wild poliovirus infection in 18 countries in 2008, Australia continues to be at risk of further importation events. *Commun Dis Intell* 2009;33(3):291–297.

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

Introduction

The global disease burden of poliomyelitis was reduced from 350,000 cases in 1988, when the World Health Assembly passed a resolution to eradicate polio, to less than 1,700 in 2008. The World Health Organization (WHO) polio eradication program is based on maintaining high levels of polio vaccine coverage, clinical surveillance for cases of acute flaccid paralysis (AFP) in children less than 15 years of age and laboratory confirmation of poliovirus infection by testing stool specimens from AFP cases at a laboratory accredited by the WHO for the purpose. In Australia, the National Polio Reference Laboratory (NPRL) is located at the Victorian Infectious Diseases Reference Laboratory (VIDRL).

Surveillance for AFP cases in children less than 15 years of age in Australia is co-ordinated by the NPRL in collaboration with the Australian Paediatric Surveillance Unit (APSU). Clinicians who treat a case of AFP are requested to arrange for collection of 2 stool specimens, due to intermittent virus shedding, within 14 days of the onset of paralysis, notify the case and complete a clinical questionnaire. The Polio Expert Committee (PEC) reviews the clinical and laboratory data from AFP cases in children less than 15 years of age, and suspected polio in persons of any age, to determine if the case is compatible with poliovirus infection.

Countries no longer endemic for polio, such as Australia, will continue to report cases of AFP as other clinical conditions mimic poliovirus infection. WHO considers an AFP surveillance scheme sufficiently sensitive to detect a wild poliovirus importation if 1 case of non-polio AFP per 100,000 children less than 15 years of age is reported each year. Based on Australia's population in 2008, the WHO AFP surveillance performance indicator was 41 AFP cases in children less than 15 years of age.

It is important that Australia maintains high levels of polio vaccine coverage to avoid a resurgence of poliomyelitis in the event of a wild poliovirus importation. The National Immunisation Program of Australia recommends immunisation with inactivated polio vaccine at 2, 4 and 6 months of age, with a booster dose at 4 years of age.[1] Multiple vaccinations ensure seroconversion occurs to each of the 3 poliovirus serotypes present in the vaccine. People travelling to polio endemic countries and countries with recent wild poliovirus importations should receive a booster polio vaccine prior to departure, or a full course of vaccination if they are unsure of their vaccination history. Individuals who are at continuing risk of infection, such as health care workers, are recommended to have a booster polio vaccine every 10 years. The WHO provides weekly updates of global wild poliovirus cases at http://www.polioeradication.org/

With the removal of the live, attenuated Sabin oral poliovirus vaccine (OPV) from the Australian immunisation schedule from November 2005, virol-

ogy laboratories in Australia are no longer expected to routinely isolate OPV-derived polioviruses from clinical specimens. Any poliovirus isolated within Australia may be an importation event and requires further investigation.

This report summarises the activities of the Australian NPRL and the performance of AFP surveillance in Australia in 2008.

Methods

Notification of cases of suspected poliomyelitis is mandatory in all Australian states and territories, whereas AFP in children less than 15 years of age is only notifiable in Queensland. Australia follows the criteria recommended by the WHO for AFP surveillance. Namely, cases of AFP in children less than 15 years of age are notified and stool specimens arranged for collection and testing at a WHO accredited laboratory. AFP surveillance is co-ordinated by the NPRL in collaboration with the Australian Paediatric Surveillance Unit and is implemented as follows:

- In keeping with WHO guidelines, clinicians are requested to notify all cases of AFP in children less than 15 years of age and cases of suspected poliomyelitis in patients of all ages.
- Clinicians notify AFP cases by contacting the NPRL (telephone: 03-9342 2607, email: polio@mh.org.au) while paediatricians also complete a monthly report card submitted to the APSU (http://www.apsu.org.au/).
- Two faecal specimens should be collected 24 to 48 hours apart, due to intermittent shedding of virus, and within 14 days of onset of paralysis for optimal virus isolation.
- Faecal specimens are referred to the NPRL at VIDRL for testing without charge.
- Clinicians are supplied with a clinical questionnaire immediately upon notification of an AFP case.
- The PEC, convened by the Australian Government Department of Health and Ageing, reviews clinical and laboratory data for all notified cases of AFP, regardless of case eligibility.
  - The PEC case definition for AFP is: Any child under 15 years of age with acute flaccid paralysis (including Guillain-Barré syndrome), or any person of any age with a paralytic illness if poliomyelitis is suspected.
  - In accordance with the WHO guidelines an ineligible case for Australia involves a patient aged 15 years or greater, a resident of another country, or a case notified as AFP in error by a clinician.
- The PEC case classifications are as follows:
  1. AFP as poliomyelitis due to poliovirus (wild type or vaccine);
  2. non-polio AFP or;
  3. non-AFP.
- If the PEC requires more information regarding an AFP case before a final classification can be made, a follow-up clinical questionnaire is sent to the notifying clinicians 60 days after the onset of paralysis.
- Australian AFP data are forwarded to WHO for inclusion in the global AFP surveillance data published in the Weekly Epidemiological Report, (available from http://www.who.int/wer/en/).

Upon receipt at the NPRL, faecal specimens are treated with Minimum Essential Medium containing Hank's Salts, chloroform (9.1% v/v) and foetal bovine serum (2%). The suspension is clarified and the supernatant inoculated onto a series of mammalian cell lines. Following the WHO recommendation, the cell lines used for the isolation of poliovirus are L20B (a transgenic mouse epithelial cell line expressing the human poliovirus receptor, CD155)[2] and RD-A (human rhabdomyosarcoma). The NPRL utilises 2 additional cell lines for the isolation of poliovirus and non-polio enteroviruses: BGMK (buffalo green monkey kidney) and HEL (human embryonic lung). Diagnostic laboratories in Australia are encouraged to refer enteroviruses of unknown serotype to the NPRL for further characterisation as poliovirus infection can lead to clinical presentations without paralysis such as aseptic meningitis.

All polioviruses, whether isolated from AFP cases or other sources, are further characterised to distinguish between wild and vaccine strains of poliovirus, a process known as intratypic differentiation. Polioviruses are characterised by reverse transcriptase polymerase chain reaction (RT-PCR) and sequencing the VP1 genomic region. The VP1 genomic region encodes one of the virus capsid proteins containing a major antigenic determinant. One per cent or more change in this region of the genome compared with the respective prototype OPV strain is, by definition, a vaccine-derived poliovirus (VDPV). A fragment of the 3D genomic region is often sequenced in order to determine whether the poliovirus has undergone a recombination event with another poliovirus serotype or non-polio enterovirus. The VP1 nucleotide sequence is also used following published methods to identify non-polio enteroviruses.[3,4]

The NPRL is also accredited, through proficiency testing and periodic on-site inspections by WHO staff as a Regional Reference Laboratory for the Western Pacific Region.

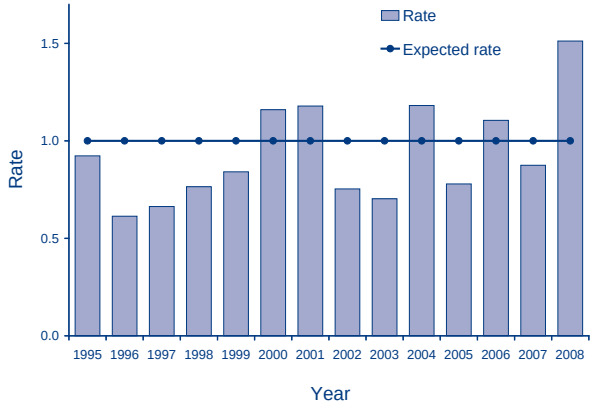
Results

Notification of acute flaccid paralysis cases and Polio Expert Committee case classifications

A total of 71 AFP cases with onset of symptoms in 2008, were notified to the APSU or the NPRL. Sufficient data were available to classify 62 cases involving children less than 15 years of age as non-polio AFP. In 2008, the non-polio AFP rate for Australia was 1.51 (62/41) per 100,000 children aged less than 15 years, which exceeds the WHO AFP surveillance performance indicator (Table 1). The classification of eligible AFP cases from 1995 to 2008 is presented in the Figure. Nine cases did not meet the WHO criteria; 8 cases involved Australian residents aged 15 years or more while 1 case was a patient from the United Kingdom.

In addition to the 71 AFP cases for which the date of onset is known, a further 8 cases remain pending classification by the PEC due to insufficient information being available for the committee to review. The 8 cases were notified as AFP cases with no further details supplied, such as the patient's name and date of symptom onset, which would allow more thorough investigation.

Figure: Classification of eligible acute flaccid paralysis cases by the Polio Expert Committee from 1995 to 2008



World Health Organization (WHO) acute flaccid paralysis (AFP) surveillance performance indicator = 1 non-polio AFP case per 100,000 population <15 years.

WHO AFP surveillance performance indicator for Australia in 2008 = 41 AFP cases in children aged less than 15 years of age.

Notification of acute flaccid paralysis cases by state and territory

In 2008, the AFP case notification rates for all states and territories exceeded the AFP surveillance performance indicator of 1 case per 100,000 children except for the Northern Territory and Tasmania, who did not notify any AFP cases. (Table 2). The 2 most populous states, New South Wales and Victoria, which account for more than 56% of expected AFP cases in Australia, met the surveillance performance indicator based on final classification of cases by the PEC. Queensland is the only jurisdiction in Australia where AFP is notifiable. While the notification rate of AFP cases in Queensland was 2.0 per 100,000 children less than 15 years of age, sufficient information was available to classify eight of the 18 cases notified, which represented a non-polio AFP rate of 0.9.

Faecal specimen collection from acute flaccid paralysis cases

The WHO has a further surveillance performance indicator that adequate faecal specimens be collected from 80% of eligible AFP cases. The WHO

Table 1: Surveillance for acute flaccid paralysis cases in children aged less than 15 years, Australia 2008, compared with the World Health Organization acute flaccid paralysis surveillance performance indicators

WHO surveillance performance indicator for AFP cases in children less than 15 years*	Australia's surveillance for AFP cases in children with onset of paralysis in 2008	Australia's AFP surveillance performance in 2008
Non-polio AFP case rate of 1.0 per 100,000 children (41 cases for Australia in 2008).	71 unique cases of AFP notified 62 cases classified by the PEC as non-polio AFP	AFP notification rate: 1.93 per 100,000 children. Non-polio AFP case rate: 1.51 per 100,000 children.
More than 80% of classified AFP cases with 2 adequate faecal specimens collected at least 24 hours apart and within 14 days of onset of paralysis.	19 AFP cases with 2 or more adequate specimens	Referral of adequate specimens from AFP cases: 31% (19/61) of the eligible cases.

* Based on data supplied by the Australian Bureau of Statistics (ABS), estimated resident population, preliminary – 30 June 2006. ABS publication 3201.0, December 2006.

AFP Acute flaccid paralysis.

PEC Polio Expert Committee.

defines adequate specimens for poliovirus culture, as 2 faecal specimens collected 24 to 48 hours apart and within 14 days of onset of symptoms.

In 2008, faecal specimens from 37 of the 62 eligible cases were tested at the NPRL:

- 19 (31%) cases had adequate specimens;
- 13 (21%) cases had at least 1 specimen collected within 14 days of onset of symptoms;
- 5 (8%) cases had 2 specimens collected after 14 days of onset of symptoms;
- no faecal specimens were received from the remaining 25 (40%) eligible cases.

While the number of AFP cases with adequate faecal specimen collection did not meet the WHO AFP surveillance performance criterion, at least 1 specimen was collected within 14 days of the onset of symptoms from 52% of the eligible cases.

Laboratory testing of specimens

Acute flaccid paralysis cases

Between 1 January and 31 December 2008, 99 specimens were referred to the NPRL from AFP cases involving patients of all ages with onset of paralysis in 2007–2008. No poliovirus was isolated from any specimens. Seventy-nine specimens were referred from the 37 cases classified as non-polio AFP involving Australian children less than 15 years of age with onset of paralysis in 2008. The non-polio enteroviruses, coxsackievirus B2, echovirus 11 and echovirus 25, were identified from 3 separate AFP cases by sequencing a fragment of the VP1 genomic region. Adenovirus type 1 and type 5 were identified from 2 separate AFP cases by sequencing a fragment of the hexon gene. No enterovirus was isolated from the remaining faecal specimens (Table 3).

Table 2: Unique notifications of eligible acute flaccid paralysis cases, onset of symptoms between 1 January and 31 December 2008, by state or territory of residence

State or territory	Estimated population aged <15 years*	Expected number of cases per year	Total number of notifications	Eligible cases classified by PEC 1 January to 31 December 2008	Non-polio AFP rate per 100,000 population aged <15 years
ACT	63,874	0.5	2	1	2.0
NSW	1,332,066	13	30	23	1.8
NT	52,253	0.5	0	0	0.0
Qld	861,002	9	18	8	0.9
SA	289,309	3	8	5	1.7
Tas	97,012	1	0	0	0.0
Vic	995,096	10	22	16	1.6
WA	426,476	4	11	9	2.3
Australia	4,117,612	41	91	62	1.5

* Australian Bureau of Statistics, estimated resident population, preliminary – 30 June 2007. ABS publication 3201.0, December 2007.

AFP Acute flaccid paralysis.

PEC Polio Expert Committee.

Table 3: Results from specimens referred to the Australian National Poliovirus Reference Laboratory from within Australia, 1 January to 31 December 2008

Result	Isolations from AFP cases*	Isolations from non-AFP referred samples	Total
Non-polio enterovirus†	8	12	20
Adenovirus‡	5	1	6
No enterovirus isolated	86	0	86
Total	99	13	112

* Includes specimens from patients of all ages and nationalities referred from within Australia.

† Genetic sequence results of non-polio enterovirus isolates from acute flaccid paralysis (AFP) and non-AFP sources identified coxsackieviruses B2 and B5 and echoviruses 6, 11, 18, 21, 25 and 30.

‡ Genetic sequence results of adenovirus isolates from AFP and non-AFP sources identified adenovirus types 1, 2 and 5.

A total of 10 faecal specimens involving Australian patients greater than 15 years of age were referred from 6 AFP cases with onset of paralysis in 2008. A total of 4 specimens from 2 cases of AFP with onset of symptoms in 2007 were referred in January 2008. No enterovirus was isolated from any of the specimens.

Sources other than acute flaccid paralysis

Thirteen specimens and isolates were received by the NPRL from sources other than AFP in 2008 (Table 3). Coxsackievirus B5 was identified from an adult with aseptic meningitis. Adenovirus type 2 was identified from a case with unspecified aetiology. Untyped enteroviruses were referred from a virology laboratory and the following non-polio enteroviruses were identified by nucleotide sequencing: coxsackievirus B5, echovirus 6, echovirus 11, echovirus 18, echovirus 21 and echovirus 30.

A summary of laboratory testing at the NPRL for the period 1995 to 2008 is presented in Table 4.

Polio serology

Poliovirus serology is only performed for cases with a clinical suspicion of acute poliovirus infection. Sixty-two requests for polio serology were cancelled after discussion with the referring doctor, as the requests related to the patient's immune status for work or travel purposes.

Regional reference laboratory activities

In addition to the Australian samples, 239 specimens and isolates were received from countries of the Western Pacific Region in 2008. The specimens referred for testing included 73 faecal specimens from 36 cases of AFP from Pacific island countries, Papua New Guinea and Brunei Darussalam; 103 specimens and isolates from Malaysia; 54 specimens and isolates from the Philippines; and 9 enterovirus isolates from the Republic of Korea.

Quality assurance program

The laboratory retained full accreditation status as a WHO Polio Regional Reference Laboratory for 2008 after an on-site review by WHO headquarters in September 2007. The main recommendation from the review was to implement all aspects of the new WHO test algorithm introduced to the regions with endemic wild poliovirus in 2006. The new algorithm was designed to shorten the time for issuing laboratory reports from 28 to 14 days, mainly by reducing the period for virus isolation by cell culture incubation from 14 to 10 days and for poliovirus intratypic differentiation from 14 to 7 days.

The NPRL retained full accreditation for poliovirus isolation from and identification of specimens and poliovirus diagnostic RT-PCR after completing the respective WHO proficiency panels for 2008. The poliovirus isolation and identification proficiency

Table 4: Summary of enterovirus testing at the Australian National Poliovirus Reference Laboratory, 1995 to 2008

Year	Poliovirus		Non-polio enterovirus	No enterovirus detected	Total samples tested
	Sabin-like	Non-Sabin-like			
1995	190	0	200	13	403
1996	224	0	198	9	431
1997	124	0	76	0	200
1998	52	0	15	4	71
1999*	60	1	9	9	79
2000	45	0	44	47	136
2001*	46	5	33	75	159
2002	36	0	21	49	106
2003	9	0	15	47	71
2004	6	0	26	61	93
2005	18	0	10	39	67
2006	2	0	6	71	79
2007†	0	2	32	115	149
2008	0	0	20	92	112

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

† Wild poliovirus type 1 was imported from Pakistan.

panel was subsequently distributed to the national polio reference laboratories throughout the Western Pacific Region by the Australian NPRL, as part of the terms of reference as a WHO regional reference laboratory.

The NPRL was accredited to AS 4633:2004 (ISO 15189:2003) for quality and competence as a medical testing facility as part of an institutional review by the National Association of Testing Authorities (NATA) in October 2008.

Discussion

In 2008, Australia met the WHO AFP surveillance performance indicator for the 5th time since the program commenced in 1995. The non-polio AFP rate of 1.51 in 2008, is the highest reported by Australia. The establishment of the Paediatric Active Enhanced Disease Surveillance (PAEDS) pilot study in 2007, co-ordinated by the APSU and the National Centre for Immunisation Research and Surveillance of Vaccine Preventable Diseases contributed to the high number of cases classified by the PEC in 2008. PAEDS is being trialled as a hospital based surveillance system in 4 major hospitals to collect clinical data and specimens for four childhood conditions including AFP.[5] While the system led to more cases classified by the PEC, the proportion of cases with adequate stool collection (31%) still did not reach the WHO performance indicator of 80%. It is important that stool specimens are tested from cases of AFP to exclude poliovirus infection as part of the differential diagnosis; however these results indicate the difficulties faced in establishing a system for the efficient referral of specimens.

At the end of each calendar year a small number of AFP notifications remain unclassified by the PEC as no clinical or laboratory data are available for the cases. After consultation with the WHO, the PEC resolved that AFP notifications remaining pending after an appropriate period, would be classified as 'polio compatible-zero evidence' and to consider them as surveillance failures. As a consequence, the PEC classified a total of 12 cases notified from 2005 to 2007 as 'polio compatible-zero evidence' in December 2008. The 8 cases listed as pending in this report may be classified according to this criterion if further information is not forthcoming before the end of 2009.

The WHO AFP surveillance performance indicator of 1 case of non-polio AFP per 100,000 children less than 15 years of age was based on the incidence of Guillain-Barré syndrome as the most common cause of AFP in the absence of wild poliovirus circulation in the Americas.[6] The year-to-year fluctuation in Australia meeting this surveillance indicator led to a 2-source capture-recapture investigation of the incidence of AFP from 1998 to 2000 in the state of Victoria, which had consistently not met the WHO criteria.[7] Whereas 0.5 AFP cases per 100,000 children less than 15 years of age were reported in Victoria via the national surveillance scheme, the retrospective study determined the average annual incidence to be 1.4 per 100,000 children less than 15 years of age. The improved performance of AFP notifications in 2008, through the establishment of a hospital based surveillance system in New South Wales, South Australia, Victoria and Western Australia, supports the validity of the WHO AFP surveillance performance indicator and that it should be considered a minimum at the national level. Thus, the years that Australia does not meet the AFP surveillance performance indicator represent gaps in surveillance for the detection of imported cases of polio.

The identification of enteroviruses from clinical specimens is important. Apart from excluding poliovirus, non-polio enteroviruses are also associated with the onset of AFP and their serotype identification will ascertain the viral aetiology of non-polio AFP. Furthermore, poliovirus infection can cause clinical symptoms without paralysis and may not be considered as part of the differential diagnosis unless techniques to identify the specific serotype are employed. Therefore, identification of enteroviruses from clinical specimens is important as part of the laboratory surveillance for poliovirus and also to establish the epidemiology of enteroviruses in Australia. With this in mind, the NPRL has been in discussion with other virology laboratories to establish an enterovirus reference laboratory network. The NPRL has introduced the CODEHOP method for the identification of non-polio enteroviruses isolated from clinical specimens and untyped enteroviruses referred from other virology laboratories.[4] This method has the benefit of being able to identify enteroviruses from original clinical specimens or cell culture isolates, which in 2008 included 2 enterovirus 71 and a coxsackievirus A16 that could not be identified by standard PCR methods.

It is well established that in areas of poor sanitation and low vaccine coverage, the Sabin live attenuated oral polio vaccine can produce vaccine derived polioviruses, defined as having more than 1% variation from the prototype serotype nucleotide sequence.[8,9] In such settings, sustained person-to-person transmission results in the accumulation of viral genome mutations with a loss of attenuation and the potential to cause outbreaks of paralytic polio. In 2008, the largest vaccine derived poliovirus outbreak was reported from Nigeria, which stands at 276 cases as at July 2009.[10] The outbreak is all the more concerning since it involves oral polio vac-

cine type 2 poliovirus, whereas wild poliovirus type 2 was last isolated in 1999 and its eradication was considered a success of the global polio eradication program.[11]

Australia last reported a wild poliovirus importation in 2007.[12] Since 2005, the number of polio cases worldwide caused by wild poliovirus has not dropped below 1,300.[13] The continued presence of type 1 and type 3 wild poliovirus in the four endemic countries (Afghanistan, India, Nigeria and Pakistan) and importations in 17 countries so far in 2009, coupled with the circulation of type 2 vaccine derived poliovirus in Nigeria underscores the need for continued awareness for imported cases of polio in Australia.

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Tuberculosis in Australia: Bacteriologically confirmed cases and drug resistance, 2007

A report of the Australian Mycobacterium Reference Laboratory Network

Richard Lumb, Ivan Bastian, Robyn Carter, Peter Jelfs, Terillee Keehner, Aina Sievers

Abstract

The Australian Mycobacterium Reference Laboratory Network collects and analyses laboratory data on new cases of disease caused by the *Mycobacterium tuberculosis* complex. In 2007, a total of 872 cases were identified by bacteriology; an annual reporting rate of 4.1 cases per 100,000 population. Isolates were identified as *M. tuberculosis* (n = 867), *M. africanum* (n = 4) and *M. bovis* (n = 1). Fifteen children aged under 10 years had bacteriologically-confirmed tuberculosis. Results of *in vitro* drug susceptibility testing were available for 871 of 872 isolates for isoniazid (H), rifampicin (R), ethambutol (E), and pyrazinamide (Z). A total of 98 (11.3%) isolates of *M. tuberculosis* were resistant to at least one of these anti-tuberculosis agents. Resistance to at least H and R (defined as multi-drug resistance, MDR) was detected in 24 (2.8%) isolates, all from overseas-born patients; 17 were from the respiratory tract (sputum n=16, endotracheal aspirate n=1). Thirteen patients with MDR-TB were from the Papua New Guinea–Torres Strait Islands zone. Of the 98 *M. tuberculosis* isolates resistant to at least one of the standard drugs, 54 (55.1%) were from new cases, 9 (9.2%) from previously treated cases, and no information was available on the remaining 35 cases. Seven were Australian-born, 90 were overseas- born, and the country of birth of 1 was unknown. Of the 90 overseas-born persons with drug resistant disease, 66 (73.3%) were from 5 countries: India (n=16); Papua New Guinea (n=15); the Philippines (n=12); Vietnam (n=12); and China (n=11). No XDR-TB was detected in 2007. *Commun Dis Intell* 2009;33(3):298–303.

Keywords: *Mycobacterium tuberculosis*, *Mycobacterium bovis*, laboratory diagnosis, drug resistance

Introduction

Australia continues to record one of the lowest notification rates (5–6 cases per 100,000 population) of tuberculosis (TB) in the world. Australian TB services continue to ensure that treatment success rates remain high, there are low rates of relapse, a complete absence of treatment failure cases and a low case fatality rate.[1,2] Drug resistant TB has emerged as a global problem that threatens TB control programs in many countries. Drug resistance is mainly associated with people born in high-burden TB countries within the Western Pacific and South East Asia regions.[3,4] Multidrug-resistant TB (MDR-TB) (resistance to at least isoniazid and rifampicin) has remained low in Australia, although the 2.4% reported in 2006 was the highest recorded figure since data collection began in 1986.[5]

There are 2 sources of TB-related data for Australia. Since 1991, the National Notifiable Diseases Surveillance System (NNDSS) has provided statistics on TB notifications reported to public health authorities in Australia's states and territories. The Australian Tuberculosis Reporting Scheme has been conducted by the Australian Mycobacterium Reference Laboratory Network (AMRLN) since 1986. Statistics compiled by the AMRLN relate to cases of bacteriologically-confirmed tuberculosis, whereas NNDSS data also include cases that are identified on the basis of clinical and epidemiological information, or on non-bacteriological laboratory investigations. This report describes the bacteriologically-confirmed TB diagnoses for the year 2007.

Methods

The data are based on clinical specimens that were culture-positive for *Mycobacterium tuberculosis* complex (MTBC). Although the bacille Calmette-Guérin strain of *Mycobacterium bovis* is a member of the MTBC, no information on this organism is included in the present report. Almost all isolates of MTBC were referred to one of the 5 laboratories comprising the AMRLN for species identification and drug susceptibility testing. Comparable methodologies are used in the reference laboratories. Relapse cases, as defined by the *National Strategic Plan for TB Control in Australia Beyond 2000* prepared by the National TB Advisory Committee,[6] were included in the laboratory data as laboratories are generally unable to differentiate relapse cases from new cases.

Data include temporary visitors to Australia, illegal immigrants, or persons detained in Australia in correctional services facilities, and asylum seekers. For each new bacteriologically-confirmed case, the following information was collected where available: demography: patient identifier, age, sex, HIV status

and state of residence; specimen: type, site of collection, date of collection and microscopy result; isolate: *Mycobacterium* species and results of drug susceptibility testing; nucleic acid amplification testing results; and for drug resistant isolates: patient country of origin, and history of previous TB treatment to determine whether resistance was initial or acquired. Data from contributing laboratories were submitted in standard format to the AMRLN coordinator for collation and analysis. Duplicate entries (indicated by identical patient identifier and date of birth) were deleted prior to analysis. Rates were calculated using mid-year estimates of the population for 2007 supplied by the Australian Bureau of Statistics.[7] For each case, the nature of the first clinical specimen that yielded an isolate of MTBC was used to record the nominal site of disease. Culture-positive specimens collected at bronchoscopy or by gastric lavage were counted as pulmonary disease. Patients with isolates recovered from multiple sites were counted as pulmonary disease (the most important category for public health purposes) if a sputum, bronchoscopy, or lung biopsy specimen was culture positive.

Drug resistance among new cases (proxy for primary resistance) was defined as the presence of resistant isolates of *M. tuberculosis* in patients who, in response to direct questioning, denied having received any prior anti-TB treatment (for more than 1 month) and, in countries where adequate documentation is available, for whom there is no evidence of such a history.[8] Drug resistance among previously treated cases (proxy for acquired resistance) is defined as the presence of resistant isolates of *M. tuberculosis* in cases who, in response to direct questioning, admit having been treated for 1 month or more or, in countries where adequate documentation is available, for whom there is evidence of such a history.[8]

Results

There were 872 bacteriologically-confirmed cases of tuberculosis in 2007, representing an annual rate of 4.1 cases per 100,000 population. State-specific reporting rates varied from 1.6 (Tasmania) to 15.4 (Northern Territory) cases per 100,000 population (Table 1).

Causative organism

Almost all isolates were identified as *M. tuberculosis* (n=867), the remaining isolates being *Mycobacterium africanum* (n=4) and *Mycobacterium bovis* (n=1).

Distribution by sex, age and site of disease

Complete information for sex and age was available for 863 (99.0%) patients. Of the 863 MTBC isolates, 402 (46.6%) were from females, 461 (53.4%) were from males, and sex was not recorded for 9 cases. The site of disease was dependent upon age and sex. The overall male:female ratio was 1.15:1. For respiratory isolates, the male:female ratio was 1.24:1. For TB lymphadenitis, the male:female ratio was 1:1.5. For males, there were 2 distinct peak age groups in bacteriologically-confirmed rates: a rise to 10.9 cases of TB per 100,000 population at 25–29 years and a second peak in elderly males aged 75 years or over (up to 12.2 cases of TB per 100,000 population). The age distribution of female cases was similar with 9.5 and 6.9 bacteriologically-confirmed TB cases per 100,000 population at the 25–29 and >84 year age groups, respectively. The median age group for patients with bacteriologically-confirmed disease was 35–39 years for both males and females. The predominant culture-positive specimen type was sputum (n=393, 45.1%); a further 132 (15.1%) were obtained from bronchoscopy, 10 were aspirates, 8 were from lung biopsies, and a single specimen of pus (Table 2). Fifty-two pleural specimens

Table 1: Bacteriologically confirmed cases of tuberculosis, Australia, 1997 and 2005 to 2007, cases and rate per 100,000 population, by state or territory

State or territory	2007		2006*		2005*		1997*	
	n	Rate	n	Rate	n	Rate	n	Rate
New South Wales†	343	5.0	342	4.8	346	4.9	329	5.0
Victoria	279	5.4	263	5.2	261	5.2	193	4.2
Queensland	118	2.8	120	3.0	91	2.3	74	2.2
Western Australia	45	2.1	93	4.5	42	2.1	51	2.8
South Australia	46	2.9	51	3.3	36	2.3	39	2.6
Tasmania	8	1.6	9	1.8	10	2.1	8	1.8
Northern Territory	33	15.4	27	12.9	24	11.9	28	15.0
Total	872	4.1	905	4.4	810	4.0	722	3.9

* Data from previous reports of the Australian Mycobacterium Reference Laboratory Network.

† Data from the Australian Capital Territory are included with those from New South Wales.

(36 fluid, 16 biopsy/tissue) were culture positive. The most commonly encountered extrapulmonary culture-positive specimen was lymph tissue (n=175, 20.0%) followed by pleural (n=52, 6.0%), peritoneal (n=28, 3.2%), bone/joint (n=26, 3.0%), and genitourinary tract (n=13, 1.5%).

Fifteen children aged under 10 years (male n=9, female n=6) had bacteriologically-confirmed tuberculosis (sputum n=4, gastric aspirate n=3, lymph node n=3, oropharyngeal aspirate n=2, and one each from pleural, cerebrospinal fluid, and pus).

Association with HIV

The AMRLN database recorded the HIV status of only 48 (5.5%) patients. No patient was identified as HIV-seropositive.

Table 2: Site of specimens smear– and culture-positive for *Mycobacterium tuberculosis* complex, 2007

	n*	Smear positive (%)*
Sputum	393	211 (54.8)
Bronchoscopy	128	46 (35.9)
Lymph node	175	33 (18.9)
Pleural	52	2 (3.8)†
Genito-urinary	13	‡
Bone/joint	26	‡
Peritoneal	28	‡
Skin	0	‡
Cerebrospinal fluid	7	‡

* Based on specimens that reported a microscopy result and excludes (i) microscopy not performed or (ii) result unknown.

† One pleural biopsy and 1 pleural fluid was smear positive.

‡ Percentage of specimens smear positive not calculated due to the small number of cases.

Microscopy

Of the 872 bacteriologically-confirmed cases in 2007, the results of microscopy were available for 851 (97.6%); microscopy was not performed on 5 specimens and no result was provided for the remaining 16 specimens. Smears were positive in 211 of 393 (53.7%) sputum and 46 of 128 (35.9%) bronchoscopy specimens respectively (Table 2). Of 52 pleural specimens (16 biopsy and 36 fluids) that were culture-positive for *M. tuberculosis*, only 1 biopsy and 1 fluid was smear-positive. Lymph node specimens were smear-positive in only 33 of 175 (18.9%) cases.

Drug susceptibility testing

Results of *in vitro* drug susceptibility testing were available for 871 of 872 isolates for isoniazid (H), rifampicin (R), ethambutol (E), and pyrazinamide (Z). A total of 98 (11.3%) isolates of *M. tuberculosis* were resistant to at least one of these anti-tuberculosis agents. Resistance to at least H and R (defined as MDR) was detected in 24 (2.8%) isolates (Table 3). All of the MDR isolates were *M. tuberculosis*. Of the 24 MDR-TB isolates, 18 were from the respiratory tract (sputum n=17, endotracheal aspirate n=1), lymph node (n=5) and a single isolate from pleural tissue. Eleven of the MDR-TB-positive sputum specimens were smear-positive, as were single samples from lymph node and pleural tissue.

None of the MDR-TB cases were extensively drug resistant resistant-TB (XDR-TB). The revised definition of XDR-TB is an isolate that has resistance to at least isoniazid and rifampicin (MDR-TB) plus additional resistance to a fluoroquinolone and an injectable (kanamycin, amikacin, capreomycin).[8] In 2007, one of the 24 MDR-TB isolates also had resistance to ofloxacin, and another MDR-TB isolate had resistance to amikacin.

Table 3: Drug resistance patterns in multi-drug resistant strains, Australia, 1995 to 2007

Resistance pattern (standard drugs)*	2007	2006	2005	2004	2003	2002	2001	2000	1999	1998	1997	1996	1995
H+R only	16	16	5	7	4	8	8	3	2	2	6	10	3
H+R+E	2	1	3	2	2	1	1	1	1	1	1	1	1
H+R+Z	5	0	1	1	1	1	3	3	1	2	5	4	1
H+R+E+Z	1	5	3	2	0	2	0	1	0	1	2	0	0
XDR-TB	0	0	0	1	0	0	0	0	0	0	0	0	0
Total (%)	24 (2.8)	22 (2.4)	12 (1.5)	12 (1.5)	7 (0.9)	12† (1.7)	12 (1.6)	8 (1.0)	4 (0.5)	6 (0.9)	14 (1.9)	15 (2.0)	5 (0.7)

* The streptomycin result was not considered for this table.

† Excludes the 1 extensively drug-resistant strain (XDR-TB), which was included in the multi-drug resistant strains.

H = isoniazid, R = rifampicin, E = ethambutol, Z = pyrazinimide.

Thirteen patients with MDR-TB were from the Papua New Guinea (PNG) – Torres Strait Islands (TSI) cross-border region who access health services in outer TSI and are eligible to receive treatment in Australia. MDR-TB was also isolated from patients born in India (n=4), China (n=3), and Vietnam (n=2), with a single case each from Timor Leste and Sierra Leone. The impact of MDR-TB arising from the PNG-TSI zone is demonstrated in the Figure. In the past 3 years (2005–07), the impact of MDR-TB cases from the PNG-TSI zone has lifted the proportion of MDR-TB cases above the 0.5%–2.0% range.

Mono-resistance to isoniazid (H) was detected in 52 isolates, with some mono-resistance to rifampicin (n=3) and to ethambutol (n=3) There was no mono-resistance to pyrazinamide (Z). Ninety-two isolates demonstrated resistance to H at a concentration of 0.1 mg/L. Of these, 44 (47.8%) demonstrated resistance to H at the higher level of 0.4 mg/L. Among MDR-TB strains, 13/24 (54.2%) demonstrated H resistance at the higher concentration (0.4 mg/L). Forty-one of 98 (41.8%) specimens culture-positive for drug resistant strains, including 30 of 56 (53.6%) sputum or bronchoscopy specimens, were smear-positive for acid-fast bacilli. The single *M. bovis* isolate, which is inherently Z-resistant, was not included in the above results.

Results of testing for streptomycin (S) were available for 259 of 871 (29.7%) isolates with 36 demonstrating resistance to at least S; 7 had mono-resistance, 11 were resistant to S and H, and 18 MDR-TB strains were also S-resistant.

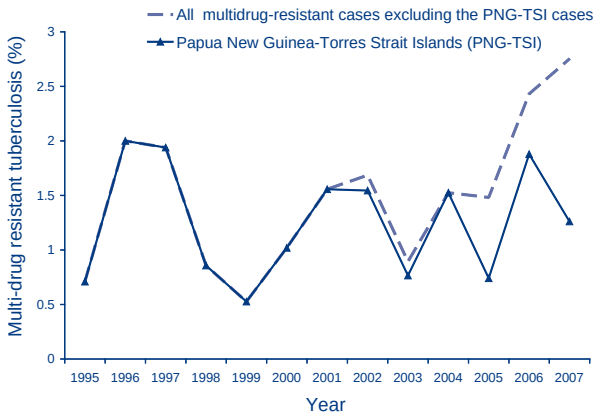
New or previously treated cases, and country of birth

Of the 98 *M. tuberculosis* isolates resistant to at least one of the standard drugs, 54 (55.1%) were from new cases, 9 (9.2%) from previously treated cases, and no information was available on the remaining 35 cases. Seven were Australian-born, 90 were overseas-born, and the country of birth of one was unknown. The 90 overseas-born persons with drug resistant disease were from 21 countries; 66 (73.3%) were from 5 countries; India (n=16), Papua New Guinea (n=15), the Philippines (n=12), Vietnam (n=12), and China (n=11).

Discussion

In 2007, there were 872 cases of bacteriologically-confirmed tuberculosis representing 4.1 cases per 100,000 population, a similar rate to that found in 2006 and consistent with the results dating back to 1986.[5,9] *M. tuberculosis* was the predominant species reported with only 4 isolates of *M. africanum* and 1 strain of *M. bovis* identified in 2007 respectively.

Figure: Percentage of multi-drug resistant tuberculosis in Australia: the impact of cases from the Papua New Guinea–Torres Strait Island zone



For 2007, the NNDSS reported 1,183 notifications, a difference between the 2 datasets of 311 (26.3%) cases.[1] The NNDSS has consistently recorded a higher number of notifications, typically in the range of 20%–30%, than the AMRLN dataset.

The level of drug resistance in *M. tuberculosis* isolates remains at a relatively constant level; excluding resistance to streptomycin, 11.3% of strains had resistance to one or more anti-tuberculosis drugs. This finding is at the high end of the range 7.4%–11.0% for resistance to one or more anti-tuberculosis drugs for the years 2000–2006. Most cases with drug-resistant strains occurred in the overseas-born as observed in previous years.

There is increasing concern regarding the rise in the proportion of bacteriologically confirmed cases with MDR-TB. During the years 1995 to 2005, the level of MDR-TB has averaged 1.3% and stayed within a range of 0.5%–2.0%. However, in the past 2 years, the proportion of MDR-TB isolates has risen from 1.5% in 2005 to 2.4% and 2.8% for years 2006 and 2007 respectively.[5,9] A substantial contributor has been from the movement of persons across the PNG–TSI zone with TB being diagnosed and then managed within Australia's borders.[10] When the 13 MDR-TB cases from PNG–TSI region are excluded and the MDR-TB percentages adjusted, the MDR-TB rates fall to 0.7% (2005), 1.9% (2006), and 1.3% (2007) respectively.[5,9] These revised figures lie within the long-standing range of 0.5%–2.0% for the bacteriologically confirmed cases of MDR-TB in Australia.

Although the population movement in the PNG–TSI zone is relatively small, the financial and epidemiological implications for the Queensland TB services and for the Commonwealth are substantial. The implications for the PNG government are equally

obvious. The emergence of MDR-TB, in apparently increasing numbers, bodes ill for PNG and highlights critical weaknesses in their TB control program. The World Health Organization (WHO) estimates that more than 900 MDR-TB cases occurred in PNG in 2006, including 563 among new TB cases and 352 cases among retreatment cases.[3] At present, the MDR-TB strains from PNG–TSI patients remain susceptible to second-line TB drugs and no XDR-TB has been identified, yet.

The WHO estimated that 489,139 new cases of MDR-TB emerged in 2006, and that the global proportion of MDR-TB among all cases was 4.8%. China and India account for almost 50% of the global burden with the Russia Federation contributing a further 7%.[8] Where there is MDR-TB, XDR-TB will also be lurking because XDR-TB is promoted by the same deficiencies in TB control programs and suboptimal care. Some 41 countries have already reported XDR-TB within their borders[11]and there are countries yet to formally report the presence of XDR-TB within their borders. Drug-resistant TB has emerged independently in all countries treating TB, and the emergence of MDR-TB is merely the stepwise accumulation of mutations for drug resistance to anti-TB drugs in a single TB organism that then amplifies and spreads. XDR-TB is a logical extension whereby MDR-TB has acquired mutations for resistance to fluoroquinolones (FQ) and injectable agents, the 2 most effective second-line drug groups for the management of MDR-TB.[12]

Many low-income countries, such as PNG, do not have ready access to laboratory culture and drug susceptibility testing facilities. Australian laboratories can provide sentinel data for these countries through the testing of their ex-patriate migrants and refugees who develop TB after arrival in Australia. India, PNG, the Philippines, Vietnam, and China accounted for nearly three-quarters of the drug-resistant cases occurring in Australia in 2007. Our experience mirrors a WHO report on TB in the Western Pacific region which estimated that China, Vietnam and the Philippines account for almost 98% of the MDR-TB cases in the region in 2006 (i.e. 82,087 new and 70,601 retreatment cases).[3]

Another worrying international phenomenon of which Australian healthcare professionals must be aware is 'pre-XDR-TB' (MDR-TB with resistance to either a fluoroquinolone or second-line injectable agent but not both).[13] Two such isolates were recognised in Australia in 2007: 1 MDR-TB strain resistant to ofloxacin, and another MDR-TB isolate was resistant to amikacin. Pre-XDR-TB is more commonly encountered than XDR-TB. An increasing incidence of FQ-resistant MDR-TB is occurring globally and represents a dangerous threat to TB control programs. For example, an Indian study from a tertiary care hospital and a referral centre in Mumbai for non-responding TB cases, determined that the proportion of *M. tuberculosis* isolates with FQ-resistance had risen from 2.6% in 1996 to 35.2% in 2004. Unfortunately, the proportion of MDR-TB with FQ-resistance was not documented although the proportion of isolates being MDR-TB had risen from 26.5% to 56.5% in the years 1996–2004 respectively.[14] High rates of quinolone resistance and pre-XDRTB have also been reported from the Philippines and China, and among foreign-born persons and recent migrants in California.[13,15,16] The increasing rates of quinolone resistance have been attributed to their wide use as first-line agents for community-acquired infections or in ineffective regimens for failed TB treatment in these countries.[17] Banerjee et al reported that the cost of inpatient treatment for a single XDR-TB case was US$600,000 in California.[13]

Fortunately, in Australia, access to FQs is restricted and fluoroquinolone use in TB cases is undertaken in a limited manner and with the benefit of drug susceptibilities performed by quality assured laboratories. In fact, all bacteriologically confirmed MDR-TB isolates and any rifampicin-resistant or multi-resistant strain of *M. tuberculosis* in Australia has second-line DST performed including against an FQ and an injectable agent. All 5 Australian Mycobacterium Reference Laboratories use the BACTEC MGIT 960 automated broth culture system for primary culture and DST for first-and second-line drugs. Recently-published WHO guidelines for second-line DST have highlighted that critical concentrations for kanamycin, cycloserine, *P*-aminosalicylic acid, thioacetazone, clarithromycin and clofazamine have not been determined for the MGIT 960 system, though, another recent study has suggested a breakpoint for kanamycin testing in a broth-based system.[18,19] Clinicians must desist from requesting laboratories to perform DST on the other second-line antibiotics listed above because the results are likely to be invalid.

The last AMRLN report mentioned that the NNDSS and AMRLN databases would be combined and a single report would describe the findings for 2007.[5] However, computing issues have confounded the roll-out of a standardised database and have lead to significant delays in several jurisdictions. A unified report will hopefully occur next year.

In conclusion, Australia remains the 'lucky country' in terms of TB incidence and level of drug resistance. The increasing number of MDR-TB cases from the PNG-TSI region has resulted in a rise in the proportion of isolates that are MDR-TB. After accounting for the influx of MDR-TB cases from PNG, the level of MDR-TB has remained stable within the 0.5%–2.0% band over the last decade. Although no XDR-TB was detected in 2007, and only 1 case has

been reported in the period 1986–2007, it is only a matter of time before further cases are identified. Importantly, 2 pre-XDR-TB isolates were identified in 2007. The AMRLN continues to be a vital part of the national TB control effort.

Acknowledgements

The Australian Mycobacterium Reference Laboratory Network comprises the Mycobacterium Reference Laboratories at the following facilities:

Institute of Medical and Veterinary Science, Adelaide, South Australia

Microbiology and Infectious Diseases, SA Pathology

Queensland Health Pathology Services, Herston Hospitals Complex, Herston, Queensland

Victorian Infectious Diseases Reference Laboratory, North Melbourne, Victoria

PathWest Laboratory Medicine WA – QEIIMC, Hospital Avenue, Nedlands, Western Australia

Institute of Clinical Pathology and Medical Research, Westmead Hospital, Westmead, New South Wales

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Tuberculosis notifications in Australia, 2007

Christina Barry, Anastasios Konstantinos and the National Tuberculosis Advisory Committee

Abstract

The National Notifiable Diseases Surveillance System received 1,135 tuberculosis (TB) notifications in 2007, of which 1,086 were new cases and 48 were relapsed cases. The incidence of TB in Australia in 2007 was 5.4 cases per 100,000 population, similar to rates since 1986. In 2007, 86.4% of cases occurred in the overseas-born population. The incidence in the Indigenous Australian population was 6.6 cases per 100,000 population. By contrast, the incidence of TB in the non-Indigenous population was 0.9 cases per 100,000 population. Household or other close contact with TB or past residence in a high risk country were the most commonly reported risk factors for TB infection. In 2007, 31 cases of TB were reported in health care workers, 29 of which were in health care workers born overseas. There were no reports of TB transmission in Australian health care settings. Outcome data of the 2006 TB cohort indicate that treatment success was attained in more than 95% of cases. As Australia continues to contribute to global TB control it is important to maintain good centralised reporting of TB to identify populations at risk and for early detection of reversal in trends in TB. *Commun Dis Intell* 2009;33(3):304–315.

Keywords: disease surveillance, tuberculosis

Introduction

Previous reports on tuberculosis (TB) notifications in Australia have highlighted that TB remains a major global health problem, with important implications for TB control locally.[1] The global burden of disease is such that international travel and mass movement of people, combined with the natural history of TB, make the goal of TB elimination in any 1 country optimistic.

For Australia, the global burden of TB has 2 major implications. The first is the maintenance of health policy and health services that have ensured rates of TB in Australia remain low, despite the global burden of TB. Experience elsewhere has shown that premature dismantling of TB services can have perverse effects on TB control.[2] The global situation and natural history of TB is such that cases of TB will continue to arise in migrants from high TB incidence countries for many years. If uncontrolled, this can lead to ongoing transmission within the general community. It is important that early diagnosis and effective management of TB cases remain a priority.

A subsidiary strategy is targeted screening and treatment of latent TB infection (LTBI). This strategy is limited because current tools available for diagnosis of LTBI provide a poor and unreliable prediction of risk versus benefits for individuals.[3] Indigenous Australians continue to have higher rates of TB than the non-Indigenous Australian population, although rates are falling. It is important that the health system remains vigilant in diagnosing reactivation cases of TB in Indigenous Australians as they age. This population also has higher rates of other chronic diseases, such as renal disease and diabetes, than the general population.[4] These chronic diseases also increase the risk for reactivation of TB and also of chronic lung diseases that confound the diagnosis of TB.

The second implication for Australia is the need to contribute to global TB control. This report looks at the impact of the overseas-born population on the burden of TB in Australia. It shows that TB control will not be achieved within Australia until TB is controlled throughout the world. Additionally, the advent of drug resistant TB internationally has implications for TB control efforts everywhere and this has already been felt in Australia, particularly with regard to the high rates of multi-drug resistant TB occurring in Papua New Guinea (PNG) nationals accessing health care in the Torres Strait Treaty Zone.[1]

The *Tuberculosis notifications in Australia* series of annual reports serve to review TB control in Australia through existing TB surveillance mechanisms. Additionally, the agreement for all jurisdictions within Australia to contribute to these reports ensures they maintain a health system capable of monitoring TB control. These reports should be viewed in conjunction with the Australian Mycobacterium Reference Laboratory Network reports on bacteriologically proven cases.

Methods

Data collection

TB is a nationally notifiable disease in Australia. Medical practitioners, public health laboratories and other health professionals are legally required to report cases of TB to state and territory health authorities. Information on notified cases is collated by state and territory jurisdictions under jurisdictional public health legislation. The *National Health Security Act 2007*[5] provides the legislative basis for

and authorises the exchange of health information between jurisdictions and the Commonwealth. The Act provides for the establishment of the National Notifiable Diseases List,[6] which specifies the diseases about which personal information can be provided. TB is one of the diseases specified in this list. De-identified data on notified cases are sent by jurisdictions electronically to the National Notifiable Diseases Surveillance System (NNDSS), managed by the Australian Government Department of Health and Ageing. National data standards ensure consistency and comparability of data collected across Australia. Enhanced data are collected for each notified case of TB, including information relating to cases' risk factors, clinical diagnostics and treatment outcomes.

A subcommittee of the Communicable Diseases Network Australia (CDNA), the National Tuberculosis Advisory Committee (NTAC) and its subcommittee, the Tuberculosis Data Quality Working Group were responsible for finalising the 2007 dataset reported to the NNDSS. Data presented in this report were analysed by date of diagnosis, a derived field within the NNDSS that is the earliest of the reported fields of notification date and notification received date.

Data presented in this report represent a point in time analysis of cases of TB notified to the NNDSS. Analyses of these cases were finalised in October 2009. Due to the dynamic nature of the NNDSS, data in this report may vary from data reported in other NNDSS reports and reports of TB notifications at the jurisdictional level.

TB drug susceptibility data on bacteriologically confirmed cases are collected, analysed and reported by the Australian Mycobacterial Reference Laboratory Network in an accompanying report.[7] In future reports, it is hoped to combine notification and laboratory data into 1 report.

Data processing and quality control

Data on all TB notifications and outcomes of treatment received from state and territory jurisdictions were examined for completeness and accuracy. Any invalid or missing data were returned to the jurisdictions for review and correction. Invalid or missing data that were unable to be resolved are discussed along with the relevant data analyses in the results section.

It is believed that almost all cases of TB in Australia are reported to the NNDSS. Reasons for the high level of reporting include adherence to the *National Health Security Act 2007,*[5] the presence of effective TB screening programs, a high standard of health care, and specialised and multi-disciplinary TB services in each jurisdiction. The terms 'notification rate' and 'incidence rate' may therefore be used interchangeably in this report.

Indigenous status and country of birth

Three population subgroups are referred to in this report, 'Indigenous Australians', 'non-Indigenous Australians' and 'overseas-born'. These population subgroups are derived from a combination of the 2 distinct data fields indigenous status and country of birth that require completing for each individual notified case of TB. Cases with a reported indigenous status of Indigenous (Aboriginal and/or Torres Strait Islander) and a reported country of birth of Australia or unknown are assumed, for the purposes of this report, to be 'Indigenous Australians'. Cases with a reported indigenous status of non-Indigenous or unknown and a country of birth of Australia are assumed to be 'non-Indigenous Australians'. Cases with a country of birth of a country other than Australia, regardless of indigenous status, are assumed to be 'overseas-born'.

In 2007, the indigenous status field was 99.9% complete (1,134 of 1,135) for cases of TB reported to the NNDSS. The country of birth field was 97.9% complete (1,111 of 1,135) for all cases reported to the NNDSS. Generally, rates specific to the 3 population subgroups presented in this report exclude the 24 cases reported with an invalid or incomplete country of birth and indigenous status fields. Adjusted rates, where these cases with a country of birth of unknown and a reported indigenous status of unknown are proportionately distributed amongst the categories 'non-Indigenous' and 'overseas-born' are presented through this report where relevant.

Case definitions

Cases of TB were determined to be notified for national reporting according to the CDNA case definition for TB.[8] A confirmed case requires a diagnosis accepted by the Director of Tuberculosis Control (or equivalent) in the relevant jurisdiction, based on either laboratory definitive evidence or clinical evidence. Laboratory definitive evidence includes either the isolation of *Mycobacterium tuberculosis* complex (*M. tuberculosis*, *M. bovis* or *M. africanum*) from a clinical specimen by culture; or nucleic acid amplification testing indicating *M. tuberculosis* complex, except where it is likely to be due to previously treated or inactive disease. Clinical evidence is defined according to the case definition as a clinical diagnosis of tuberculosis, made by a clinician experienced in tuberculosis, including a clinical follow-up assessment to ensure a consistent clinical course.

TB cases were classified as new or relapsed at the time of notification. A new case required a diagnosis accepted by the Director of TB Control (or equivalent) in the relevant state or territory, based on either laboratory or clinical evidence, and in the absence of any previous treated or untreated TB diagnosis.

A relapsed TB case is defined as a case of active TB diagnosed bacteriologically, radiologically or clinically, having been considered inactive or quiescent following previous treatment (as deemed by the state or territory Director of Tuberculosis). Relapses refer to re-treatment cases of which some may be reinfections rather than a true relapse of prior disease. Relapse cases are disaggregated into relapse after full or partial treatment, in Australia or overseas.

It is important to note that this report only considers cases of active TB disease. Latent TB infection, when a person is infected with *M. tuberculosis* but does not have the disease and is non-infectious, is not a notifiable disease under the *National Health Security Act 2007*.[5]

National Performance Indicators

The performance criteria for the National Performance Indicators were set by NTAC in 2002 as part of the *National Strategic Plan for TB Control in Australia Beyond 2000*.[9] In TB annual reports before 2005, the performance criteria for incidence in people born overseas applied to people who had been living in Australia for more than 5 years. In this report the criteria have been applied to all cases regardless of length of residence.

Population estimates for 2007

Notification rates were calculated using the estimated 2007 mid-year resident population supplied by the Australian Bureau of Statistics.[10] Rates specific to the Indigenous Australian population were based on projections from the 2001 census estimate of the Indigenous population in Australia.[11] As data on the state and territory composition of Australia's estimated resident population by country of birth were only available for census years, rates specific to the Australian born population by state or territory were based on 2006 census counts where country of birth was Australia.[12] The preliminary 2007 estimated resident population by country of birth was used to calculate incidence rates of TB in the overseas-born population.[13]

Results

Tuberculosis notification rates

The incidence of TB in Australia has remained at a stable rate since 1986 (Figure 1). A total of 1,135 cases of TB was reported in Australia in 2007, representing a crude rate of 5.4 cases per 100,000 population (Table 1).

In 2007 there were 1,086 new cases of TB notified and 48 relapse cases notified (Table 1). A single case was notified to the NNDSS without this information. Of the 48 relapsed cases, 9 relapsed after full treatment in Australia, 9 following partial treatment in Australia, 18 following full treatment overseas and 12 following partial treatment overseas.

Tuberculosis notifications by state or territory

In 2007, New South Wales reported the largest number of cases of TB (454). However the highest notification rate was recorded in the Northern Territory (25.1 cases per 100,000 population) because there were relatively few cases (54 cases) in a smaller population (Table 1).

Table 1: Notifications of new and relapsed cases of tuberculosis and notification rate per 100,000 population, Australia, 2007, by state or territory

State or territory	New cases		Relapse cases		Total cases	
	Notifications	Rate	Notifications	Rate	Notifications*	Rate
ACT	10	2.9	0	0.0	10	2.9
NSW	437	6.3	17	0.2	454	6.6
NT	52	24.2	1	0.5	54	25.1
Qld	125	3.0	12	0.3	137	3.3
SA	55	3.5	4	0.3	59	3.7
Tas	8	1.6	0	0.0	8	1.6
Vic	345	6.6	8	0.2	353	6.8
WA	54	2.6	6	0.3	60	2.8
Australia	1,086	5.2	48	0.2	1,135	5.4

* Total includes 1 case reported with no new/relapse case information.

Figure 1: Tuberculosis notification rate per 100,000 population, Australia, 1960 to 2007

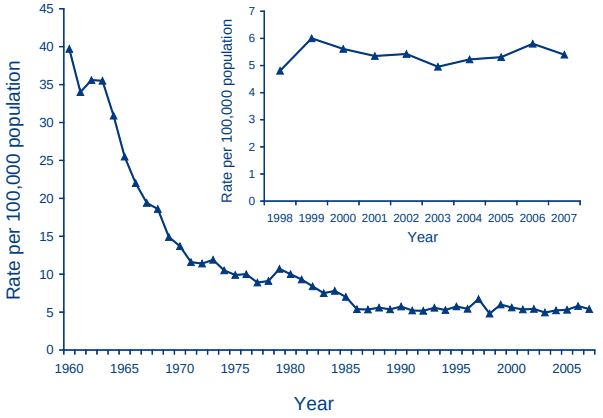


Figure 2 presents TB notification rates, on a semi-logarithmic scale, for the previous decade for each state and territory. The highest notification rates over the 10 year period were seen in the Northern Territory. Notification rates in 2007 for the Australian Capital Territory and Western Australia were their lowest in 10 years.

The largest increases in the number of cases of TB over the 10 year period were seen in the Northern Territory (39% increase), Victoria (36% increase) and Queensland (24% increase). The largest decrease in the number of cases of TB over the same period was seen in the Australian Capital Territory (70% decrease). Notification rates in the Australian Capital Territory varied greatly over the 10 year period. The volatility in the rate is due, in part, to small changes in the number of cases of TB over time proportionate to a relatively small estimated residential population.

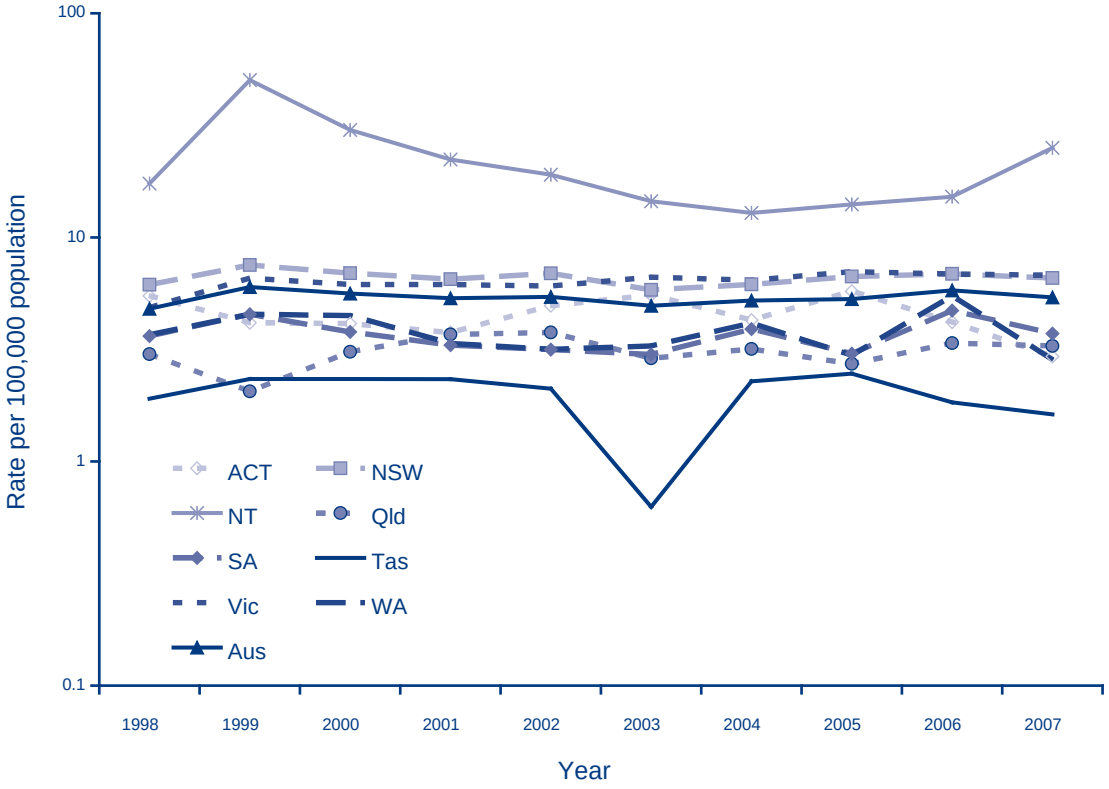
Tuberculosis in non-Indigenous Australians

Indigenous status was reported for all 151 Australian-born cases (Table 2). The incidence rate of TB in non-Indigenous Australians in 2007 was 0.9 cases per 100,000 population (116 cases), with the trend over time for this population group suggesting continued decline (Figure 3). The adjusted rate for this population subgroup, accounting for cases reported with an invalid or incomplete country of birth and indigenous status, remained at 0.9 cases per 100,000 population.

Tuberculosis in Indigenous Australians

In 2007, the TB incidence rate in the Indigenous Australian population was 6.6 cases per 100,000 population (35 cases) (Table 2). There has been variability in incidence rates in the Indigenous Australian population over time, but overall rates

Figure 2: Tuberculosis notification rate per 100,000 population, Australia, 1998 to 2007, by state or territory



are decreasing with time (Figure 3). Nevertheless, the crude TB incidence rate in Indigenous Australians in 2007 was greater than 7 times the rate in non-Indigenous people. The adjusted rate for this population subgroup, accounting for cases reported with an invalid or incomplete country of birth and indigenous status, remained at 6.6 cases per 100,000 population.

Tuberculosis notifications in the overseas-born population

In 2007, the country of birth was reported for 1,111 of the 1,135 cases. Table 3 shows notifications and incidence rates by country of birth.

Of the cases reported with a country of birth, overseas-born people contributed 86.4% (960 cases) of the total tuberculosis case-load in 2007 (Table 3). The TB incidence rate in the overseas-born population was 18.3 cases per 100,000 population (960 cases) in 2007. This rate is more than 20 times

Figure 3: Tuberculosis notification rate per 100,000 population, Australia, 1996 to 2007, by population subgroup

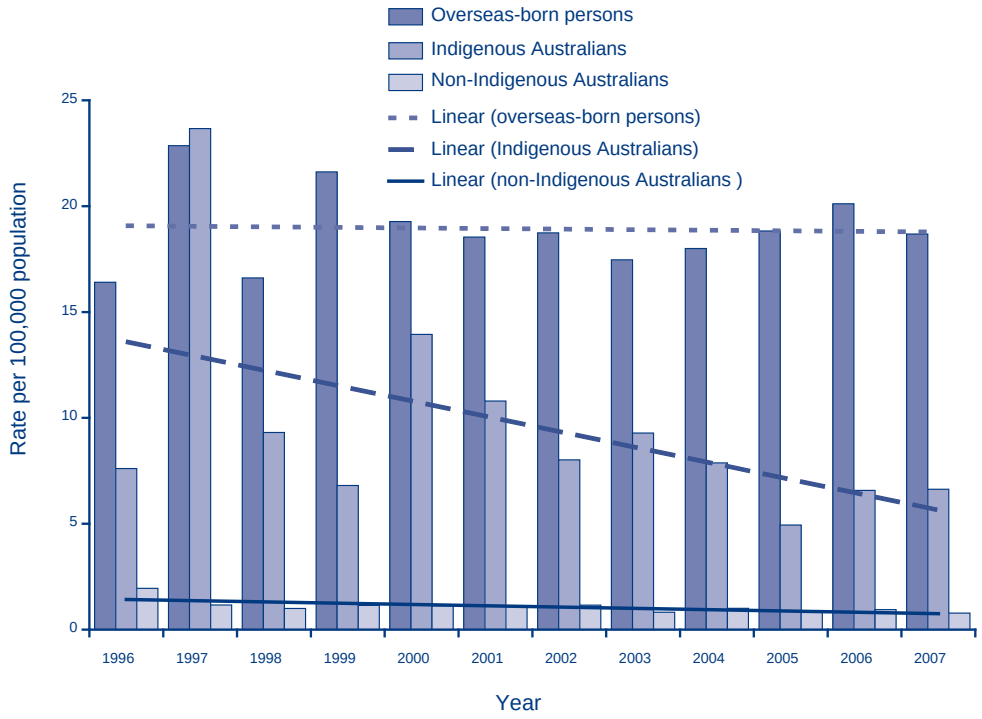


Table 2: Notifications of tuberculosis and notification rate per 100,000 population in all Australian-born cases, Australia, 2007, by state or territory and indigenous status

State or territory	Indigenous Australians		Non-Indigenous Australians		Total Australian-born	
	Notifications	Rate	Notifications	Rate	Notifications	Rate*
ACT	0	0.0	2	0.9	2	0.8
NSW	3	1.9	52	1.2	55	1.2
NT	21	32.2	2	2.4	23	15.5
Qld	7	4.7	12	0.4	19	0.6
SA	3	10.5	9	0.8	12	1.1
Tas	0	0.0	3	0.8	3	0.8
Vic	1	2.9	30	0.9	31	0.9
WA	0	0.0	6	0.5	6	0.5
Australia	35	6.6	116	0.9	151	1.0

* Australian-born rates by state or territory were based on 2006 census counts where country of birth was Australia.[12]

the incidence rate experienced by non-Indigenous Australians. The adjusted rate for this population subgroup, accounting for cases reported with an invalid or incomplete country of birth and indigenous status, increased to 18.7 cases per 100,000 population.

The rate of notification of overseas-born cases was similar to rates in this population in the previous 2 years (20.1 and 18.8 per 100,000 population in 2006 and 2005 respectively, (Figure 3). The rate of notification of TB in the overseas-born population has remained relatively stable since 1996.

While the highest notification rates were among those born in Nepal (19 notifications; 293.9 cases per 100,000 population), Somalia (15 notifications; 283.8 cases per 100,000 population) and Eritrea (6 notifications; 245.1 cases per 100,000 population), these represent a relatively small number of cases in a small resident population (Table 3). As in previous years, the largest numbers of TB cases were detected in those born in India (193 notifications; 96.6 cases per 100,000 population), Viet Nam (88 notifications; 46.8 cases per 100,000 population), China (82 notifications; 29.2 cases per 100,000 population),

Table 3: Notifications of tuberculosis and estimated rate per 100,000 population for selected countries of birth, Australia, 2007

Country of birth	New cases	Relapse cases	Total cases	Estimated resident population 2007	Rate per 100,000 population in Australia*	WHO incidence rate per 100,000 population 2007†
Nepal	19	0	19	6,465	293.9	173.0
Somalia	14	1	15	5,286	283.8	248.7
Eritrea	6	0	6	2,448	245.1	95.4
Sierra Leone	5	0	5	2,434	205.4	573.9
Libya	3	0	3	1,794	167.2	17.2
Ethiopia	11	0	11	6,981	157.6	378.2
Papua New Guinea	34	8	42	28,531	147.2	249.5
Myanmar	20	1	21	15,103	139.0	170.9
Sudan	26	1	27	23,100	116.9	243.3
India	189	4	193	199,696	96.6	167.8
Liberia	2	0	2	2,123	94.2	277.1
Bangladesh	16	0	16	19,530	81.9	222.5
Albania	2	0	2	2,467	81.1	16.9
Pakistan	16	1	17	21,117	80.5	181.3
Indonesia	43	1	45	63,060	71.4	228.0
Timor-Leste	6	1	7	10,584	66.1	322.0
New Caledonia	1	0	1	1,542	64.9	21.6
Nigeria	2	0	2	3,161	63.3	310.7
Kenya	7	0	7	12,361	56.6	352.6
Philippines	77	3	80	144,340	55.4	290.0
Solomon Islands	1	0	1	1,885	53.1	127.8
Viet Nam	85	3	88	188,038	46.8	171.2
China‡	76	6	82	281,009	29.2	98.3
Other overseas-born	419	19	268	4,210,797	6.4	
Total overseas-born	1,080	49	960	5,253,852	18.3	
Australian-born	144	7	151	15,763,370	1.0	
Total§	1,086	48	1,135	21,017,222	5.4	

* The Australian Bureau of Statistics estimated resident population at June 2007.[13]

† Rates from the World Health Organization 2009 Global Tuberculosis Report.[14]

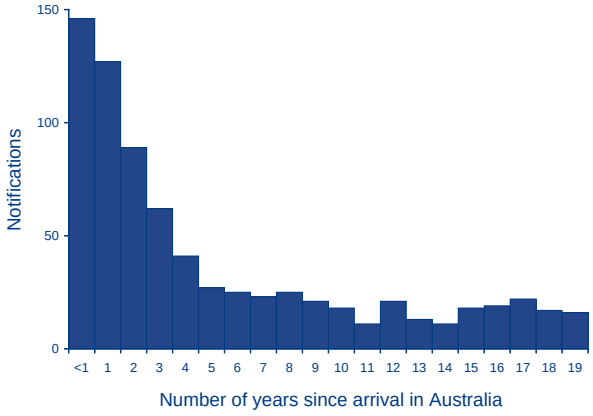
‡ China excludes Hong Kong SAR and Taiwan.

§ Total includes 24 cases reported with an invalid or incomplete country of birth and indigenous status.

the Philippines (80 notifications; 55.4 cases per 100,000 population) and Indonesia (45 notifications; 71.4 cases per 100,000 population).

Data on the year of arrival were available for 935 of the 960 overseas-born cases in 2007. Of the overseas-born cases that were reported with a year of arrival, 29.2% (273 cases) presented within 2 years of arrival in Australia and 80.4% (752 cases) within 20 years of arrival (Figure 4).

Figure 4: Tuberculosis notifications in the overseas-born population, Australia, 2007, by number of years since arrival in Australia



The Australian immigration status was available for 508 (52.9%) of the 960 overseas-born cases in 2007. The majority of these cases were permanent residents (291 cases, 57.3%), with refugees, overseas visitors and overseas students representing a similar proportion within the subgroup (51 cases, 10.0%; 48 cases, 9.4%; 48 cases, 9.4%, respectively.)

Unauthorised entrants made up 3.9% nationally, with 18 of the 20 cases reported by the Northern Territory. These cases were illegal fisherpersons detained by Australian Customs, diagnosed with TB and commenced on TB treatment. There was a total of 33 notifications of TB in Queensland among Papua New Guinea nationals accessing health care in the Torres Strait Treaty Zone, representing an increase on previous years.

Tuberculosis notifications by age and sex

Information on the sex and age of TB cases was available for all cases. The male to female ratio in TB notifications was 1:0.8.

One of the most important measures of TB control is the incidence in children aged less than 15 years because these cases represent recent TB infection. TB was notified in 58 children aged less than 15 years in 2007, 5.1% of the total number of notified cases (Table 4). Of these, 20 were Australian-born and 38 were born overseas. Of the 20 Australian-born children, 7 were identified as Indigenous Australians.

The NTAC target performance indicator for rates of TB in children aged less than 15 years is less than 0.1 case per 100,000 population for all groups. The age group-specific notification rate for children under 15 years of age in 2007 was 1.4 cases per 100,000 population (Table 4). The rate was highest in overseas-born children (12.9 cases per 100,000 population), followed by the Indigenous Australian children (3.6 cases per 100,000 population). The notification rate remained low in non-Indigenous children (0.4 cases per 100,000 population, Table 4).

The age group incidence rates for TB in overseas-born, Indigenous Australian and non-Indigenous

Table 4: Notifications of tuberculosis and estimated rate per 100,000 population, Australia, 2007, by age group and population subgroup

Age group	Indigenous Australians		Non-Indigenous Australians		Overseas-born		Total*	
	Notifications	Rate	Notifications	Rate	Notifications	Rate	Notifications	Rate
0–4	3	4.7	9	0.7	4	8.6	16	1.2
5–14	4	3.1	4	0.2	34	13.7	42	1.5
Subtotal	7	3.6	13	0.4	38	12.9	58	1.4
15–24	3	3.0	14	0.6	151	27.9	173	5.9
25–34	6	8.2	9	0.4	295	38.4	314	10.8
35–44	7	10.8	6	0.3	138	15.1	153	5.0
45–54	5	11.1	18	0.9	116	12.4	141	4.8
55–64	4	16.6	14	0.9	77	9.2	99	4.2
65+	3	18.9	42	2.4	145	15.0	197	7.1

* Total includes 24 cases reported with an invalid or incomplete country of birth and indigenous status.

Australian populations are shown in Table 4 and Figure 5. TB incidence in the overseas-born population was highest in the 25–34 year age group (295 cases, 38.4 cases per 100,000 population). In the Indigenous Australian and non-Indigenous Australian cases, rates increased throughout adult life with the highest notification rates in the 65 years or over age group (3 cases, 18.9 cases per 100,000 population; 42 cases, 2.4 cases per 100,000 population, respectively).

Tuberculosis and selected risk factors

Information on risk factors for TB, excluding HIV, is reported in Table 5. One case may report more than one associated risk factor. Information on risk factors for TB, excluding HIV, was reported in 658 (58.0%) of the 1,135 cases.

Of the cases that reported risk factors in 2007, household or other close contact with a TB patient was a common risk factor in all 3 population subgroups (163 total cases). A total of 31 TB cases were reported in people who had previously worked or were currently working in a health care setting, with over 90% of these cases being overseas-born. Past residence of 3 months or longer in duration in a TB high risk country was reported in the majority of cases (481 total cases), including 14 Australian-born cases.

The number of TB cases reported in health care workers has varied between 12 (2005) and 42 (2006) cases per year since 2001 with 31 cases reported in 2007 (Figure 6). Since 2001, the proportion born overseas has varied between 50.0% in 2001 to 93.5% in 2007. At diagnosis, most health care workers were or had been working in the previous 12 months in an Australian health care setting. None of the cases were deemed to have acquired TB in an Australian

Figure 5: Tuberculosis notification rate per 100,000 population, Australia, 2007, by age group and population subgroup

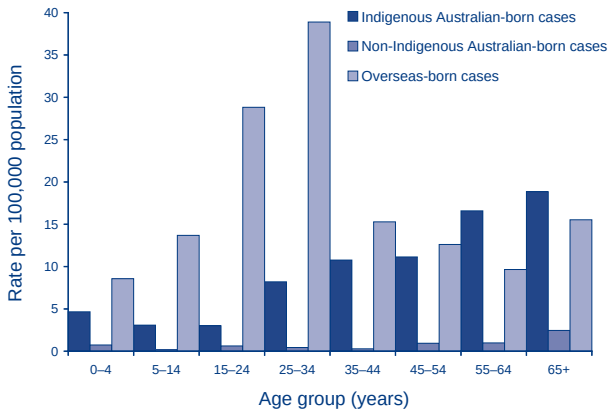


Figure 6: Tuberculosis notifications reported in health care workers, Australia, 2001 to 2007, by country of birth

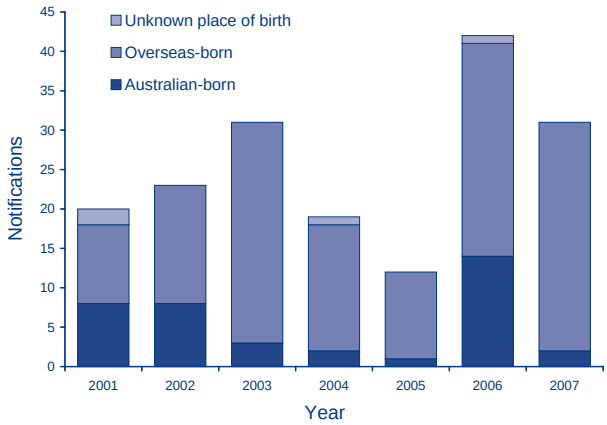


Table 5: Notifications of tuberculosis, Australia, 2007, by selected risk factors*,† and population subgroup

Risk factor	Indigenous Australians notifications	Non-Indigenous Australians notifications	Overseas-born notifications	Total notifications‡
Household or other close contact with a TB patient	15	22	111	163
Currently or recently resident in correctional facility§	1	0	1	2
Currently or recently residing in aged care facility§	0	1	3	4
Currently or recently employed in an institution§	0	6	6	13
Currently or previously employed in health industry§	0	2	29	31
Past residence in high risk country	1	14	465	481

* Excludes HIV status.

† More than 1 risk factor may be reported for each notified case.

‡ Total includes cases reported with risk factor and with an invalid or incomplete country of birth and indigenous status.

§ Within the preceding 5 years.

health care setting, nor were there any reports of active TB transmission to patients from health care workers in Australia in 2007.

Tuberculosis and HIV status

Information on HIV status was reported in 486 of the 1,135 (42.8 %) cases. Of these cases, 13 were identified with HIV infection at the time of diagnosis (10 Overseas-born, and 3 non-Indigenous). The proportion of cases with HIV status reported increased from 35.2% in 2006 to 42.8% in 2007.

Anatomical site of disease

The anatomical site of TB infection was recorded in all notified cases (Table 6). Pulmonary disease was reported in 694 cases, of which 556 cases were reported as having pulmonary disease only and the remaining 138 cases were reported as having pulmonary disease plus disease at an extrapulmonary site. Extrapulmonary disease only was reported in 441 notifications, with lymph nodes reported as the most frequent extrapulmonary site.

Treatment outcomes of 2006 tuberculosis patient cohort

Treatment outcome data for all TB cases reported in 2006 were received by August 2009 (Table 7).

Treatment success, including those with bacteriologically confirmed cure and those who completed treatment, was reported in more than 95% of cases with assessable outcomes in the non-Indigenous and overseas-born populations. In contrast, only 83.9% of Indigenous Australian cases were reported with treatment success. There was 1 case of a treatment failure; this was reported in a non-Indigenous case.

National Performance Indicators

Performance criteria for incidence (less than 1 per 100,000 population) were met only for the crude incidence rates in non-Indigenous cases (Table 8). Incidence rates in the age groups under 15 years exceeded the performance criteria (less than 0.1 case per 100,000 population) in all population groups. The completeness of HIV data collection remains well below the goal of 100%, however has improved upon the completeness of HIV data recorded for 2006 notifications. Outcome reporting did not meet the target of 100% for the 2006 patient cohort, with 1.7% of cases with assessable outcomes reported with an unknown outcome. While overall the performance indicator for cases that reported treatment success was met, the performance indicator was not met by the Indigenous Australian subset of the population, with 83.9% of cases reporting treatment success.

Discussion

The annual incidence of reported TB cases has remained reasonably constant in Australia since 1986. In 2007, the crude rate of total cases (including new and relapse cases) was 5.4 cases per 100,000 population. However, the incidence of total cases in the Australian born population continues to show a downward trend with 1 case per 100,000 population observed in 2007. Notifications of TB cases in Australia that were overseas-born comprised 86.4% of all notified cases in 2007 (Table 3), with over one-quarter of these having lived in Australia for 2 years or less. Thus, most TB cases currently seen in Australia arise from transmission overseas or transmission in Australia within the overseas-born population. Currently, molecular typing of TB isolates is carried out in various Australian jurisdictions by the Mycobacterial Reference Laboratory Network. Such testing should provide data to support whether the transmission within overseas-born populations is occurring within Australia or not.

Among Australian-born populations, much disparity still exists between Indigenous and non-Indigenous Australians, with crude rates of TB in Indigenous Australians being more than 7 times that of non-Indigenous Australians in 2007 (Table 2). Table 4 shows that the difference in younger age groups is much greater than this, suggesting that age-adjusted rates would show that the disparity is much greater than suggested by crude rates. Variations in rates between jurisdictions may reflect differences in the proportion of migrants in the population or, in the case of the Northern Territory, the proportion of Indigenous Australians in the population. However, in the case of the Northern Territory, rates among non-Indigenous Australians are higher than in other states (Table 2).

While it is felt that TB transmission is rare in Australian-born people generally, we suggest that transmission is still a problem among the Australian Indigenous community. This ongoing transmission could then explain the higher rate among non-Indigenous Australians in an Australian jurisdiction with the highest proportional population of Indigenous people. Molecular studies in the future will allow greater elucidation of this. However, the Queensland TB Control Centre (QTBCC) has noted that the incidence of TB among non-Indigenous Australians in Far North Queensland (where there is again a relatively high proportion of Indigenous Australians) is higher than in those from other areas of the State (QTBCC, personal communication). The large variation of TB in Indigenous Australians throughout Australia (Table 2) reflects both the low numbers of Indigenous people and the fact that incidence seems to occur within various community groups and is subject to case-finding

Table 6: New and relapsed cases of tuberculosis, Australia, 2007, by site of disease

Site	New cases	Relapse cases	Total cases	Per cent of total cases
Total pulmonary disease	656	37	694	61.1
Pulmonary only	530	25	556	49.0
Pulmonary plus other sites	126	12	138	12.2
Extrapulmonary only*	430	11	441	38.9
Pleural	58	2	60	5.3
Lymph nodes	130	3	133	11.7
Bone/joint	31	2	33	2.9
Genito/urinary	20	0	20	1.8
Milliary	2	0	2	0.2
Meningeal	12	0	12	1.1
Peritoneal	3	0	3	0.3
Other	51	2	53	4.7

* Extrapulmonary only' includes 153 cases reported without an extrapulmonary site further categorised. More than 1 extrapulmonary site may be reported for each notified case.

Table 7: Notifications of tuberculosis, Australia, 2006, by population subgroup and treatment outcomes

	Indigenous Australians		Non-Indigenous Australians		Overseas-born		Total cases	
	Cases	% assessable	Cases	% assessable	Cases	% assessable	Cases	% assessable
Assessable outcomes								
Treatment success	26	83.9	119	96.7	854	95.4	999	95.2
Cured (bacteriologically confirmed)*	13	41.9	13	10.8	39	4.3	65	6.2
Completed treatment†	13	41.9	105	85.9	816	91.1	934	89.0
Interrupted treatment‡	0	0.0	0	0.0	0	0.0	0	0.0
Died of tuberculosis§	2	6.5	2	1.6	10	1.1	14	1.3
Defaulted‖	0	0.0	1	0.8	16	1.8	17	1.6
Failure¶	0	0.0	1	0.8	0	0.0	1	0.1
Not followed up, outcome unknown	3	9.7	0	0.0	15	1.7	18	1.7
Total assessable	31	100.0	123	100.0	895	100.0	1,049	100.0
Non-assessable outcomes								
Transferred out of Australia§	0	0.0	1	0.7	77	7.5	78	6.5
Died of other causes§	2	6.1	14	9.9	43	4.2	59	4.9
Still under treatment	0	0.0	4	2.8	5	0.5	9	0.8
Total	33		142		1,020		1,195	

* Cured is defined as the bacteriologically confirmed cure of smear or culture positive pulmonary cases.

† 80% of standard regimen completed.

‡ Interrupted treatment means treatment interrupted for two months or more but completed.

§ During treatment phase.

‖ Defaulted means failed to complete treatment.

¶ Failed means treatment completed but failed to be cured.

Table 8: National tuberculosis performance indicators, performance criteria and the current status of tuberculosis, Australia, 2006 and 2007

National tuberculosis performance indicator	Performance criteria	2006†	2007
Annual incidence of TB (cases per 100,000 population)			
Crude incidence			
Indigenous Australians	<1	6.8	6.6
Non-Indigenous persons	<1	0.9	0.9
Overseas-born persons	*	20.1	18.3
Relapse cases initially treated in Australia	<2% treated cases	0.9%	0.8%
Incidence in children <15 years, by risk group (per 100,000 population)			
Indigenous children	<0.1	1.7	3.6
Non-Indigenous children	<0.1	0.5	0.4
Overseas-born children	*	19.8	12.9
Collection of HIV status in tuberculosis cases (% of cases with data collected)	100%	35%	43%
Treatment outcome measures (%)			
Cases evaluated for outcomes	100%	98.3%	TBA
Cases that have treatment completed and are cured	>90%	95.2%	TBA
Cases recorded as treatment failures	<2%	0.1	TBA

* Performance criteria currently under review.

† Evaluation of outcomes of 2006 patient cohort re-assessed in August 2009

TBA To be assessed: 2007 patient cohort outcomes to be reported in 2008 annual report.

activities. Ongoing molecular typing will help elucidate these issues as well as explore further the reason for increasing rates in Queensland and Victoria over the last 10 years. Victoria has a high migrant intake (as does New South Wales) while Queensland has had increasing numbers reported among people from PNG over the last 10 years.[1,15]

As in previous reports, the data show that most TB in Australia occurs in migrants. Table 3 is useful in identifying the source countries for many of these migrants and can be useful for informing health services about risk groups for TB. However, the estimates of rates are biased by the fact that temporary visitors are included among the cases but are not necessarily enumerated within the base population. Nevertheless, it is important to recognise that migrants from high incidence TB countries will be a source of reactivation of TB as they age and develop other medical conditions that increase the risk for reactivation. It is important that health services remain alert to this possibility now that TB has become rare even in the oldest non-Indigenous people (Table 4) and thus rarely encountered as a cause of morbidity among the aged by the general medical workforce.

It is important that good centralised reporting of TB continues for Australia to identify populations at risk and to detect promptly any reversal in trends in TB incidence. This is particularly important as the full impact of poor TB control often emerges only after many years of neglect when reversal of trends can become costly. Due to the low rates of TB in Australia, important risk groups for TB are often overlooked because of the perception that latent infection and newly acquired TB infection are rare in the community at large. However, this can lead to unwarranted complacency and failure to recognise risk for those populations with higher rates of TB (overseas-born and Indigenous Australians). In this respect, it is unfortunate that HIV infection status is still being reported for only a small percentage of TB cases, despite the aims of the NTAC to achieve 100% reporting for many years. There is a need to explore other avenues for achieving greater compliance, for example, initiating HIV testing by TB control unit clinical staff after TB has been notified or increasing reminders to clinicians managing TB about the need for HIV data. It is also possible that the reported data are under-reporting the true extent of HIV testing if the test results are only available after the collection of data or if the data collection process initiates testing for HIV after data have been provided. Such information could be better elucidated without the barriers of the administrative and political constraints to linking data in a manner that protects individual confidentiality.

Unfortunately, data on bacteriology and drug resistance testing are provided separately at the national level. It is planned that these databases will be

linked at the national level in the near future, which will allow more complete reporting. However, the data provided do suggest that TB is currently well-controlled within Australia. The challenge for the future is to maintain such high level control in the face of the continuing high incidence for much of the world's population, by maintaining effective diagnostic and treatment services that are readily and freely accessible to all population groups within the Australian setting. Even among Australia's health care workers, the major risk for TB is birth overseas. Reporting on molecular typing of TB strains in the future will provide better data to determine whether disease in the overseas-born population was acquired overseas or the result of transmission within clusters of overseas-born people within Australia.

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Peer-reviewed articles

Flutracking: A weekly Australian community online survey of influenza-like illness in 2006, 2007 and 2008

Craig Dalton, David Durrheim, John Fejsa, Lynn Francis, Sandra Carlson, Edouard Tursan d'Espaignet, Frank Tuyl

Abstract

Surveillance for influenza is an important public health function as it allows initiation and evaluation of public health measures. Flutracking is a weekly online survey of influenza-like illness (ILI) completed by community members that has been trialled in the 2006, 2007, and 2008 winter influenza seasons. The online survey allows participants to record their past and current influenza immunisation status and they receive a weekly email prompt to answer questions on the previous week's experience of cough, fever and time absent from normal activities. The weekly survey takes participants less than 15 seconds to complete. Symptom rates of Flutracking participants were compared by influenza vaccination status to estimate the incidence and severity of influenza and the field effectiveness of influenza vaccine. Participation rates increased from 394 in 2006 to 982 in 2007 and 4,827 in 2008. In 2008, 56% of participants were from New South Wales and 26% were from Tasmania. Greater than 70% of respondents replied within 24 hours of the survey being sent in 2007 and 2008. The 2008 influenza season appeared milder than 2007 with the peak weekly rate of cough and fever among all unvaccinated participants at 7% in 2008 compared with 15% in 2007. The peak week of influenza activity detected by Flutracking in 2008 was the week ending 31 August, which was contemporaneous with the peak week in other syndromic and laboratory influenza surveillance systems. Participation in the survey continues to grow and appears sustainable. A more balanced recruitment across jurisdictions will provide a more national perspective. *Commun Dis Intell* 2009;33(3):316–322.

Keywords: influenza, surveillance, syndromic surveillance, influenza-like illness, survey

Background

There are approximately 3,000 deaths per year due to influenza and its complications in Australia.[1] Community-based surveillance of influenza-like illness (ILI) is recommended by the World Health Organization (WHO) as part of a comprehensive surveillance system during inter-pandemic and pandemic periods.[2,3] Surveillance for influenza can serve the following public health objectives:

1. early detection of epidemics to enable the implementation of public health measures, such as the vaccination of high risk groups, outbreak control campaigns, enhanced laboratory testing and infection control measures, and provision of clinical services;
2. characterisation of the nature of the epidemic;
3. isolation and antigenic characterisation of circulating influenza viruses to assist in the formulation of the following season's vaccine and to provide new vaccine strains; and
4. evaluation of the impact of the epidemic and associated public health measures.[4]

In Australia, surveillance for influenza is conducted through sentinel general practices and locum services, emergency department surveillance for ILI, worksite absenteeism and laboratory surveillance for influenza infection.[4] These systems complement each other by drawing information from different parts of the surveillance pyramid.

To contribute broader population information on ILI, we piloted an online community survey and assessed its acceptability and feasibility for detecting inter-pandemic and, potentially, pandemic influenza in a regional health service with a population of 800,000 in south-eastern Australia during 2006 and expanded the project nationally in 2007 and 2008.

The main aims of Flutracking are to develop a system that will:

1. compare ILI syndrome rates between vaccinated and unvaccinated participants to determine the utility of Flutracking for detection of influenza activity and early confirmation of vaccine effectiveness or failure;
2. determine whether Flutracking provides earlier warning of influenza activity than existing surveillance systems, including emergency department and general practice ILI surveillance, and laboratory testing for influenza infections.

Methods

In June 2006, an invitation to participate in the online survey was sent to approximately 7,000 email addresses on the Hunter New England area health service network with a clickable link to the survey. A media release was sent to all major newspapers and radio stations in the area health service region, which has a population of approximately 800,000 people. A short domain name (www.flutracking.net) was used to assist the memory of people hearing or reading the recruitment messages. Emails were sent to colleagues and friends of investigators and participants were able to forward the invitation email on to acquaintances to consider joining the study. Potential participants were directed to a web page providing information about the study and an online consent form. A confirmatory email response from the participant's email address was required to complete enrolment in the study.

In 2007, similar recruitment promotion activities were undertaken except that approval was received from the Hunter New England Human Research Ethics Committee to expand recruitment nationally within Australia. We received approval to allow a household member to respond to the survey on behalf of other members of their household of any age, and for children 12 years of age and above to complete their own survey online.

Every Monday morning from 19 June to 23 October 2006, from 4 June to 15 October 2007, and from 5 May to 20 October 2008 participants received an automatically generated weekly email link to the online questionnaire asking about their symptoms and absence from usual activities in the previous week. This questionnaire was modified slightly each year (Table 1), with the choice of symptoms based on a review of case definitions and predictors of influenza infection.[5–7] Participants who reported not being vaccinated against influenza in the current season were asked if they had received vaccination in the previous week during each weekly survey and if they responded in the affirmative the question was automatically deleted from their subsequent weekly surveys. Participants were allowed to join at any time during the surveillance period.

In the first online questionnaire participants were asked about:

1. month and year of birth;
2. receipt of influenza vaccine in the current and the preceding year;
3. working face to face with patients in hospitals, nursing homes, doctors' surgeries or as community health workers; and
4. postcode of residence.

Participants then received a weekly email, which contained a link to an online survey form asking about the presence of the typical flu-like symptoms listed in Table 1. The survey usually took less than 15 seconds to complete.

To explore the difference between vaccinated and unvaccinated participants, the weekly proportion of participants with ILI symptoms was calculated by vaccination status. These proportions were compared with influenza activity recorded in 2007 and 2008 by established influenza surveillance systems in New South Wales, including emergency department ILI presentations, and laboratory influenza A antigen tests (polymerase chain reaction and direct immunofluorescence) (NSW Department of Health unpublished data).

Table 1: Symptoms asked about in the weekly online survey, jurisdictional and respondent age restriction changes from 2006 to 2008

	2006	2007	2008
Fever	Y	Y	Y
Cough	Y	Y	Y
Number of days absent from normal duties	Y	Y	Y
Muscle aches	N	Y	N
Sudden onset of fever, muscle aches, and cough	Y	Y	N
Jurisdictions	Area health service	National	National
Respondent's age	18 years +	18 years +	12 years+*

* Parents could respond on behalf of children of any age in their household.

More information about the survey, including screenshots of the survey forms, is available from: www.flutracking.net

Results

Participants

Participation in 2008 increased more than 10-fold compared with 2006, and almost 5-fold compared with 2007 (Table 2).

During 2006, for the weeks ending 18 June to 22 October a total of 394 respondents completed at least 1 weekly survey (Table 2). Among the 162 participants who joined in the first 4 weeks of the survey, the median weekly participation rate was 93% and 58 (36%) did not miss a single weekly survey.

Recruitment appeared most effective via email invitations with clickable links to the online study consent and information page. Based on an analysis of email domain names, at least 222 of 394 respondents in 2006 were Hunter New England Area Health Service employees.

During 2007, for the weeks ending 3 June to 14 October a total of 982 respondents completed at least 1 weekly survey. Although national recruiting was undertaken in 2007, 770 (79%) participants were from New South Wales. In 2007, 93% of those who ever responded to the weekly email, responded within 7 days of the email dispatch and of these, 50% of participants answered the survey within 3 hours and 80% answered within 24 hours. Among the 412 participants who joined in the first 4 weeks of the survey, the median weekly participation rate was 95%, and 159 (38%) did not miss a single weekly survey.

During 2008, for the weeks ending 4 May to 19 October a total of 3,279 respondents completed at least 1 weekly survey for themselves and on behalf of 1,548 other household members – a total of 4,827 participants. Among the 3,649 participants who joined in the first 4 weeks of the survey, the median weekly participation rate was 96% and 1,416 (39%) did not miss a single weekly survey.

In 2008 the Flutracking survey further expanded participation in all Australian states and territories. Although New South Wales had the highest participant counts for 2008, Tasmania had the highest participation rate in 2008, followed by the Australian Capital Territory (Table 3).

Of the 4,827 who participated at least once in 2008, 283 first participated in 2006, another 509 first participated in 2007. The remaining 4,035 first participated in 2008.

Table 2: Comparison of 2006, 2007, and 2008 Flutracking participants

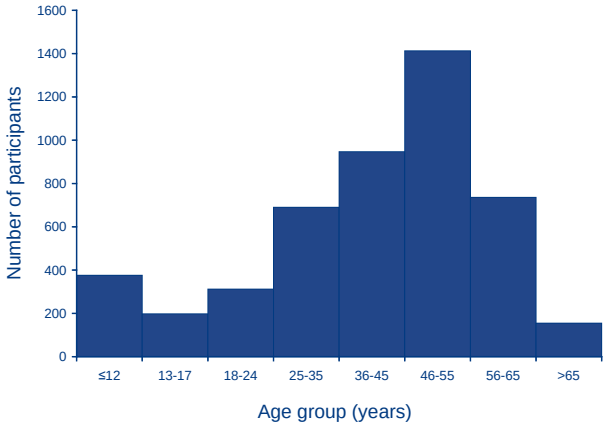
	2006	2007	2008
Number participating one or more times	394	982	4,827
Number of weeks survey conducted	19	20	25
Median number of surveys completed by participants	10	11	22
Number of participants withdrawing	14 (3.5%)	7 (0.7%)	72 (1.5%)
Peak weekly participation number	346	863	4,183
Number (%) working directly with patients	174 (44.0%)	285 (29.0%)	1,393 (28.9%)
Number (%) vaccinated within the calendar year	253 (64.0%)	505 (51.0%)	2,406 (49.8%)

Table 3: Number of Flutracking participants who responded to at least 1 survey, 2008, by state or territory

State or territory	Number of respondents	%	Rate per 100,000
ACT	159	3.3	46.4
NSW	2,689	55.7	38.7
NT	2	0.0	0.9
Qld	158	3.3	3.7
SA	52	1.1	3.3
Tas	1,235	25.6	248.3
Vic	404	8.4	7.7
WA	128	2.7	6.0
Total	4,827	100.0	22.7

Figure 1 shows that of participants who responded to at least 1 survey in 2008, 78.4% were aged 25 to 64 years. In 2008, persons aged under 18 years could participate for the first time. There has been a positive response to this initiative, with 11.9% of participants aged less than 18 years in 2008. In 2007, participants aged less than 18 years were not eligible to participate and 4.1% were aged 65 years or over.

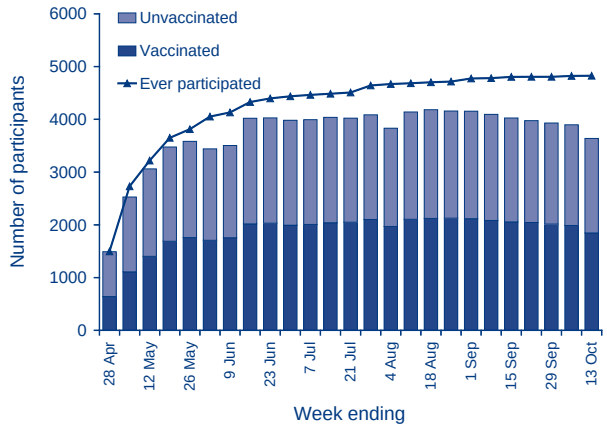
Figure 1: Age distribution of participants who responded to at least 1 survey in 2008



The mean weekly proportion of participants vaccinated against influenza in 2008 across all weeks in 2008 was 49.8% (Figure 2) – 70.1% among participants who worked with patients and 41.5% among those who did not.

In 2008, 95% of responses were received within 7 days of the email survey dispatch and of these 40% of participants answered the survey within 3 hours, while 73% answered within 24 hours.

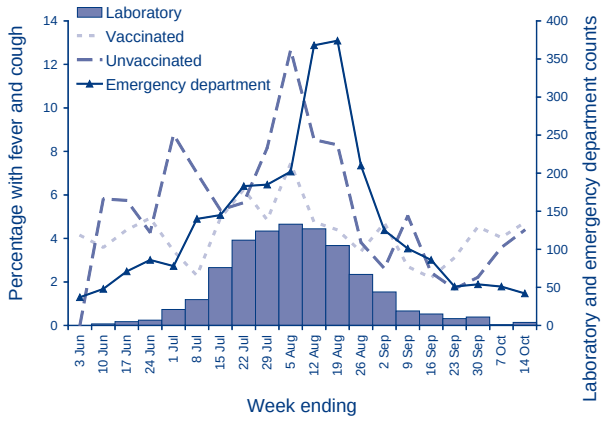
Figure 2: Number of participants who had ever participated in the survey in 2008 and weekly number of participants, by influenza vaccination status



Detection of influenza-like illness

Participant numbers in 2006 were considered insufficient to identify a relationship between influenza vaccination status and ILI symptoms (data not shown), however, in 2007 there was a marked divergence between cough and fever rates in influenza vaccinated and unvaccinated participants that was contemporaneous with the pattern of New South Wales laboratory influenza antigen test results and ILI reports from New South Wales emergency departments (Figure 3). Unvaccinated participants reported a peak of ILI of 13% in the week ending 5 August 2007 (Figure 3).

Figure 3: Comparison of fever and cough in influenza vaccinated and unvaccinated Flutracking participants compared with counts of positive influenza antigen test from laboratories and influenza-like illness counts in emergency departments, New South Wales, 2007



In 2008, the national data show a steady symptom rate of approximately 4% for fever and cough until 10 August when fever and cough rates increased for the season. The fever and cough rates diverged by vaccination status most markedly for the weeks ending 24 and 31 August with a peak of ILI of 7% in unvaccinated participants (Figure 4).

The divergence in rates of ILI between vaccinated and unvaccinated participants was most marked in New South Wales, which had the highest number of participants (Figure 5) and the divergence in symptom rates between influenza vaccinated and unvaccinated participants was contemporaneous with the rise in New South Wales influenza laboratory notifications and emergency department presentations for ILI.

Figure 4: Comparison of fever and cough in influenza vaccinated and unvaccinated Flutracking participants, Australia, 2008

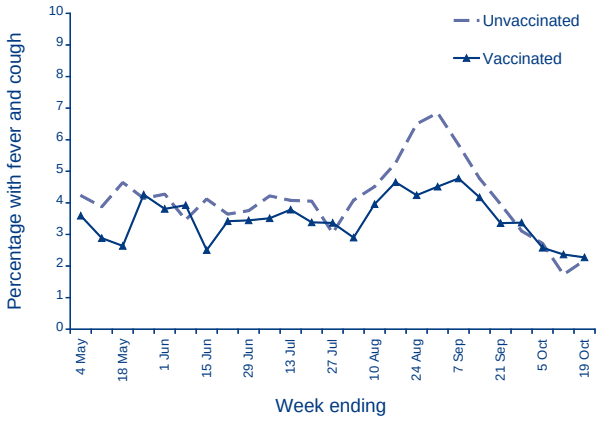
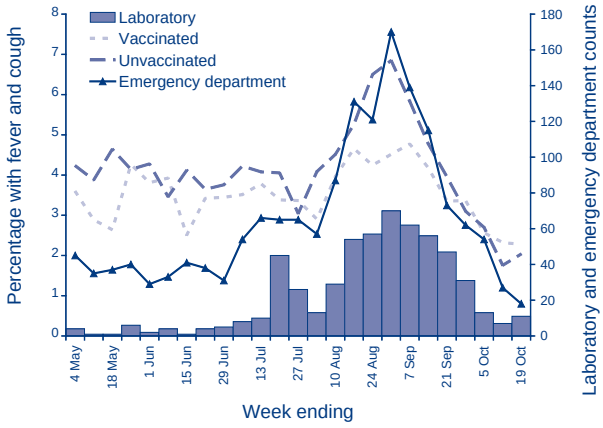


Figure 5: Comparison of fever and cough in influenza vaccinated and unvaccinated Flutracking participants compared with counts of positive influenza antigen tests from laboratories, and influenza-like illness counts in emergency departments, New South Wales, 2008



Discussion

This study confirms that a rapidly completed weekly online survey of ILI is feasible and appears acceptable to participants. Participation and retention rates were encouraging but may be improved in subsequent years through enhanced feedback to participants and support from large organisations to distribute invitations via employee email systems.

Recruitment was most successful when participants received an email with a clickable hyperlink leading them to the study information web site, perhaps because interested participants could immediately act upon the invitation rather than having to remember the web address to access at a later time.

These first years of the study were designed to test the methodology and we did not expect to recruit sufficient participants to identify influenza activity with any confidence. However, the following findings lend face validity to a relationship with actual influenza activity:

1. similar rates of illness among vaccinated and unvaccinated respondents prior to and after the 'influenza season' as defined by the laboratory surveillance data;
2. a divergence in rates occurring during the influenza activity period; and
3. the elevation in the proportion with symptoms among the unvaccinated compared with the vaccinated participants.

The finding that the absolute peak for unvaccinated participants and the peak difference in symptom prevalence between the vaccinated and unvaccinated respondents occurred contemporaneously with existing New South Wales influenza surveillance systems, provides reassurance that they are monitoring the same condition. Time series analysis of the relationship between 2007 Flutracking and New South Wales influenza laboratory notifications controlling for autocorrelation within the data, also support the premise that Flutracking is detecting actual influenza activity with the greatest cross correlation between the systems occurring in the same week.[8]

Although syndromic reporting has limited sensitivity and predictive value for influenza, the inclusion of information on vaccination status in this survey has the potential to enhance specificity. Findings from studies of the predictive value of influenza symptoms may differ due to different settings and subject populations. The presence of cough and fever is usually a core component of ILI case definitions. The positive predictive value of cough and fever together may exceed 70% and is sometimes improved with the addition of symptoms such as myalgia or fatigue.[5–7]

This surveillance system addresses needs in routine influenza surveillance and potentially in surveillance for pandemic influenza. It complements laboratory confirmed influenza and general practice and emergency department ILI surveillance with the potential to rapidly detect influenza activity and provide a prompt to enhance infection control in institutions or rapidly vaccinate or exclude unvaccinated staff from high risk settings. Influenza surveillance may also provide prior warning of a community-wide influenza epidemic to allow clinical services to prepare for an increase in respiratory admissions. By directly monitoring community incidence of ILI, it may provide more reliable estimates of community attack rates compared with systems that may be biased by health seeking behaviour.

These findings suggest that Flutracking may provide a unique opportunity for monitoring seasonal vaccine efficacy or failure. Vaccine failure would be suggested by unvaccinated and vaccinated participants ILI symptom rates failing to diverge despite laboratory notification rates suggesting an influenza epidemic is underway. If the methodology is proven and broader community participation is sustained it may allow rapid monitoring of other syndromes to provide rapid assessment of illness from contaminated water supplies, new and emerging infectious diseases or bioterrorism related events. Response to the survey is rapid with 70% to 80% of participants responding within 24 hours and it is possible to vary the surveillance case definitions rapidly throughout the season if required. Data can be quickly analysed and a report can be finalised within 2 days of the end of the surveillance week; the email link is sent to survey participants on a Monday asking about symptoms up to and including the day before (Sunday) and a report is completed by Tuesday.

The impact of different recruitment settings and strategies, age related participation, regional variations, and biases due to surges in recruitment during periods of influenza activity will be examined in future analyses. Because influenza spreads across different regions over many months, comparing symptom rates in unvaccinated and vaccinated participants in different and disparate regions in the same week may artificially dilute the impact of influenza vaccination and suppress the divergence that would be seen in the geographic epicentres of influenza activity for that week.

Flutracking is the first online community ILI surveillance system in the Asia Pacific region and is similar to an influenza surveillance project being conducted in The Netherlands, Portugal, and Belgium which follows a combined cohort of over 20,000 participants throughout the winter months.[9,10]

The recruitment strategy in Tasmania in 2008 was extremely effective with more participants recruited in 4 weeks than were recruited during the first 2 years of Flutracking operation in all jurisdictions. Recruitment was enhanced by the circulation of an email memo from the Director of Public Health to all staff in the Tasmanian Department of Health and Human Services and through reinforcement with an internal newsletter article. With support from other jurisdictions and corporate entities it is likely that Flutracking could achieve more than 10,000 participants nationally by 2011.

Future analyses will focus on comparison of signals of influenza activity from other surveillance data sets and exploring measures of field vaccine effectiveness from Flutracking data to determine if the data can provide early warning of vaccine failure or early reassurance of its effectiveness. The analysis of vaccine effectiveness will be particularly important in 2009 due to the emergence of novel H1N1 influenza.[11] Jurisdictional data will be provided to jurisdictions with greater than 1,000 participants to support local surveillance, encourage collaborative analyses of data and explore new methods of analysis.

Flutracking appears to provide important ILI surveillance intelligence and situational awareness, complementing current national influenza surveillance systems in Australia. Influenza surveillance information will be best interpreted when all available information is integrated into public health decision making.

Acknowledgements

Craig Dalton conceived and designed the project, oversaw the statistical analysis, and drafted the manuscript. David Durrheim, contributed to the design of the project, statistical analysis, and writing of the manuscript. John Fejsa, contributed to the design of the project and had primary responsibility for the online software and database development, as well as questionnaire design. Lynn Francis, Frank Tuyl and Sandra Carlson contributed to the statistical analysis and writing of the manuscript. Edouard Tursan d'Espaignet contributed to the design of the project, statistical analysis, and writing of the manuscript.

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We would like to acknowledge the thousands of Flutracking participants who give their time freely each week to contribute to influenza surveillance.

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Flutracking surveillance: Comparing 2007 New South Wales results with laboratory confirmed influenza notifications

Sandra J Carlson, Craig B Dalton, Frank A Tuyl, David N Durrheim, John Fejsa, David J Muscatello, J Lynn Francis, Edouard Tursan d'Espaignet

Abstract

General practice and hospital surveillance for influenza-like illness (ILI) and laboratory influenza surveillance provide useful but incomplete information on influenza incidence. Flutracking is an Australian pilot of an Internet-based community ILI syndromic surveillance system designed to detect inter-pandemic and, potentially, pandemic influenza. Presence of fever and/or cough and absence from normal duties are collected weekly. Influenza vaccination status of respondents is recorded. New South Wales Flutracking data for 2007 were compared with New South Wales laboratory notifications for confirmed influenza to validate it's ability to provide alerts of influenza activity. Symptom rates amongst vaccinated and unvaccinated Flutracking respondents were compared using a variety of case definitions, with New South Wales laboratory influenza notifications. Time series methods were used to estimate the degree of correlation between each Flutracking case definition and the laboratory data. For the unvaccinated group, the correlations between all Flutracking case definitions and laboratory data were statistically significant, while for the vaccinated group no case definitions were significantly correlated with laboratory data. Thus Flutracking ILI data amongst unvaccinated participants correlated well with influenza laboratory surveillance. *Commun Dis Intell* 2009;33:323–326.

Keywords: influenza, surveillance, Flutracking, time series, ARIMA

Introduction

Seasonal influenza causes substantial morbidity and mortality each year.[1] Community-based surveillance of influenza-like illness (ILI) is therefore recommended by the World Health Organization (WHO) as part of a comprehensive influenza surveillance system during inter-pandemic and pandemic periods.[2,3] Influenza surveillance supports the detection and public health response to influenza transmission.[4]

It is acknowledged that while laboratory confirmed influenza surveillance data may be biased by testing activity, it is usually considered the most reliable indicator of the onset and peak of influenza activity. Therefore, laboratory data are often used as the default measure for comparing the performance of syndromal (or 'syndromic') influenza surveillance. Zheng et al compared emergency department visits assigned a clinical diagnosis of influenza to New South Wales influenza laboratory data to determine whether the former could offer earlier warning of an increase in influenza incidence in the New South Wales population.[5] Lau et al defined the start of peak influenza activity using laboratory isolation rates for their analysis of multiple streams of influenza surveillance data.[6]

Flutracking is a weekly community online survey of ILI that integrates syndromic information with participants' influenza immunity status. Flutracking aims to help fill the gap between laboratory and syndromal surveillance systems because it uniquely combines information on influenza symptom rates and vaccination status of participants. It has been piloted with approximately 900 participants predominantly in New South Wales in 2007 and this rose to over 4,000 nationwide in 2008.

The purpose of this study was to use sound time series methods to validate the 2007 New South Wales Flutracking data against New South Wales data for laboratory confirmed influenza.

Methods

Flutracking recruitment

Flutracking was initially piloted in 2006. Recruitment occurred as outlined in Dalton et al.[7] Potential participants were directed to a web page providing information about the study and an online consent form. A confirmatory email response from the participant's email address was required to complete enrolment. The study was approved by the Hunter New England Area Health Service Human Research Ethics Committee. Participants were allowed to join at any time during the surveillance period.

Flutracking data collection

Each Monday from 4 June to 15 October 2007, participants received an automatically generated weekly email link to the online questionnaire. In the 1st online questionnaire participants were asked

about their usual postcode of residence; whether they work face-to-face with patients in hospitals, nursing homes, doctors' surgeries or as community health workers; their month and year of birth; and whether they received an influenza vaccination in the previous or current year.

For each subsequent questionnaire, participants were asked whether during the prior week (ending Sunday) they had experienced fever and/or cough and/or muscle aches on any specific day/s, and whether they had been absent from usual activities on any specific day/s. Participants who reported not being vaccinated against influenza in the current season were asked if they had received vaccination in the prior week during each weekly survey. If they responded in the affirmative the question was automatically deleted from their subsequent weekly surveys.

Analysis

Data for New South Wales participants for the week ending 3 June 2007 to the week ending 14 October 2007 were included in the analysis. New South Wales data accounted for 76% of all participants in Australia who completed at least 1 survey during 2007. For the purpose of this analysis, the laboratory data was classified as the independent variable, and each of the Flutracking symptoms were classified as dependent variables.

For each of the vaccinated and unvaccinated groups, a time series of the proportion of respondents reporting any of 5 possible case definitions was created. The case definitions were:

- fever only;
- cough only;
- absence from work or normal duties;
- fever and cough; or
- fever, cough and absence from work or normal duties.

A time series of weekly counts of positive influenza antigen tests (polymerase chain reaction and direct immunofluorescence) were created from the NSW Department of Health notifiable diseases database.[8] Counts were aggregated into weeks based on the date of specimen collection.

We used autoregressive integrated moving average (ARIMA) time series and cross correlation analysis to determine whether there was an association between the laboratory time series and weekly proportions for each Flutracking case definition. As the Flutracking data used for analysis were proportions the variance stabilising transformation for binomial data was applied.[9] This is an arcsine transformation, $\gamma_a = \arcsin\sqrt{\gamma}$, where γ_a is the transformed Flutracking data, and γ is the proportion of participants with the particular Flutracking symptom/s specified by each case definition. Similarly, the laboratory data were counts, and the variance stabilising transformation for a Poisson distribution was applied: $x_a = \sqrt{x}$, where x is the original laboratory data, and x_a is the transformed laboratory data.[9]

In time series modelling, the assumption that model residuals are independent is typically violated due to the residuals being autocorrelated (i.e. the current values of a series correlate with past values of the same series).[10] If autocorrelation is not removed, then the relationship between 2 time series could be overestimated.[11] Any comparisons made between laboratory data and Flutracking data potentially require correction for autocorrelation.

For the vaccinated and unvaccinated groups, we calculated raw correlations and used ARIMA models to estimate the association between weekly proportions of respondents reporting each case definition and weekly counts of positive influenza isolates. ARIMA modelling is a well established time series analysis technique that can be used to model an autocorrelated variable.[10] Adding an independent variable to the usual ARIMA model (called transfer function analysis)[12] allows the relationship between 2 time series to be measured, while correcting for autocorrelation. The SAS ARIMA[13] procedure was used to compute cross correlations between the 2 data series at various time differences, after both series had been 'prewhitened' (that is, filtered by an ARIMA model that was originally fitted to the independent variable).

Results

Descriptive statistics

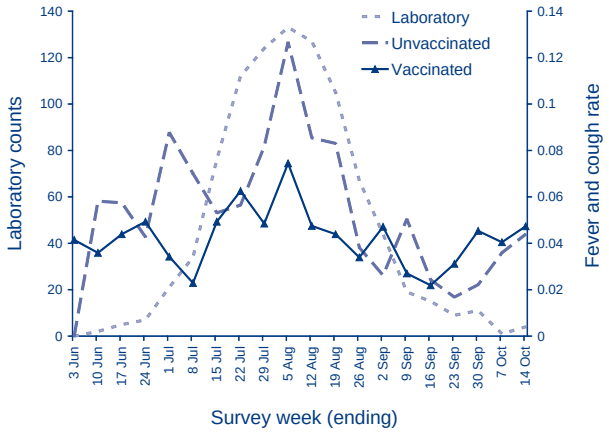
In New South Wales, for the 20 week period between 3 June and 14 October 2007, there was an average of 502 participants per week who completed the survey. Over that period, a weekly average of 65% of participants reported being vaccinated.

Visual inspection of the time series of each Flutracking case definition against laboratory data suggested that the peaks in laboratory data corresponded to periods of high Flutracking symptom rates for the unvaccinated group compared with the vaccinated group. A graph for the 'fever and cough' case definition is shown in the Figure.

Raw correlation analysis

Using raw correlation analysis (i.e. without autocorrelation correction), we found that the correlation values were generally highest when Flutracking

Figure: Flutracking symptom rates for 'fever and cough' case definition, compared with influenza laboratory notification counts, New South Wales, 2007, by influenza vaccination status



symptom rates and laboratory data were compared in the same week (i.e. a lag of 0), but similar values also occurred at other differences in time (or lags).

Each Flutracking case definition in both the vaccinated and unvaccinated groups showed a statistically significant relationship with the laboratory data at a lag of zero (all *P* values for the correlation coefficients were less than 0.05). However, it was important to further analyse the relationship between the two time series using ARIMA analysis.

Autoregressive integrated moving average analysis

Results from an autocorrelation check for white noise using ARIMA analysis indicated that laboratory data showed significant autocorrelation (at the level of $P = 0.05$), and that the model that fitted this data best was $\gamma_t = 1.6\,\gamma_{t-1} - 0.6\,\gamma_{t-2} + \varepsilon_t$ where γ_t is the laboratory data at time t (in weeks), and ε are the residuals from the model. This model was used to pre-whiten both the Flutracking and laboratory data.

Cross correlations for the residuals from the ARIMA model applied to the laboratory data and each of the Flutracking data series are summarised in the Table. Only cross correlation values at a lag of zero for each case definition related to laboratory data are reported.

In the unvaccinated group, all cross correlations at a lag of 0 weeks were statistically significant at a level of $P = 0.05$. The cross correlation analysis did not provide evidence of a substantive difference between the case definitions, except for 'absence from work or normal activities,' which at 0.442, did not have as high a cross correlation as the other symptoms. In the vaccinated group no case definitions at a lag of zero were statistically significant at a level of $P = 0.05$. The results from the ARIMA analysis for the vaccinated group were not consistent with results from raw correlation analysis, where there were statistically significant relationships between every case definition for the vaccinated group and the laboratory data.

Discussion

There was a statistically significant correlation between time series of laboratory confirmed influenza and Flutracking data for unvaccinated participants in New South Wales for all 5 case definitions (fever; cough; absence; fever and cough; fever, cough and absence) at a lag of 0 weeks. This indicates that Flutracking responds contemporaneously with laboratory surveillance of disease caused by influenza that leads to a specimen being col-

Table: Cross correlation and corresponding probability values from the ARIMA analysis for each Flutracking case definition symptom rate compared with influenza laboratory notifications, New South Wales, 2007, by vaccination status

Vaccination status	Case definition	Cross correlation value	Probability value for cross correlation (using a one-tailed t statistic)
Vaccinated	Fever	–0.006	1
Vaccinated	Cough	0.302	0.097
Vaccinated	Absence	–0.054	1
Vaccinated	Fever and cough	0.203	0.188
Vaccinated	Fever, cough and absence	–0.072	1
Unvaccinated	Fever	0.654	0.005
Unvaccinated	Cough	0.623	0.006
Unvaccinated	Absence	0.442	0.032
Unvaccinated	Fever and cough	0.640	0.005
Unvaccinated	Fever, cough and absence	0.652	0.005

lected. For the vaccinated group who should have at least some protection against influenza infection, cross correlations were not statistically significant after correction for autocorrelation, indicating that Flutracking can discriminate between influenza and other causes of ILI disease.

For vaccinated participants, the change in statistical significance between raw correlation results and ARIMA modelling results demonstrates the importance of adjusting for autocorrelation, and using appropriate analysis techniques for time series data. Without controlling for autocorrelation, spurious results were obtained. However, after correcting for autocorrelation the 'true' relationship between the 2 data series could be seen.

A limitation when quantifying the relationship between the Flutracking and laboratory data was that there were only 20 continuous time points in the weekly Flutracking data series, when usually at least double that number are recommended for ARIMA analysis.[10] However, we confirmed by Monte Carlo simulation that a model of the type found for the laboratory data, nearly always generates data that are clearly autocorrelated, even when there are only 20 time points, based on checking by time series analysis.

In conclusion, this analysis of Flutracking results has provided support for its value in providing alerts of influenza activity. Distinguishing between vaccinated and unvaccinated participants offers further potential to determine the value of Flutracking in assessing the effectiveness of the annual influenza vaccine composition in real-time.

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Sandra Carlson drafted the manuscript and performed the statistical analysis. Craig Dalton conceived and designed the Flutracking project, contributed to the statistical analysis and contributed to the manuscript. Frank Tuyl oversaw the statistical analysis and contributed to the writing of the manuscript. David Durrheim contributed to the design of the Flutracking project and writing of the manuscript. John Fejsa contributed to the design of the Flutracking project and had primary responsibility for the online software and database development, as well as questionnaire design. David Muscatello contributed to the statistical analysis and writing of the manuscript. Lynn Francis contributed to the statistical analysis and writing of the manuscript. Edouard Tursan d'Espaignet contributed to the design of the Flutracking project, statistical analysis, and writing of the manuscript.

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High proportion of influenza B characterises the 2008 influenza season in Victoria

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Abstract

The 2008 influenza season in Victoria was distinctive because of the increased proportion of influenza-like illness (ILI) cases due to influenza B infection and the lateness of the season compared with preceding years. Influenza activity fell within the bounds of normal seasonal activity thresholds. The average rate of ILI reported by general practitioners participating in sentinel surveillance was 5.5 cases per 1,000 consultations, peaking at 13.4 cases per 1,000 consultations. The average ILI rate reported by the Melbourne Medical Deputising Service was 5.1 cases per 1,000 consultations over the season peaking at 16.2 cases per 1,000 consultations at the same time as peak rates were reported by rural geneal practitioners (GPs), with a secondary peak observed 2 weeks later (10.9 cases per 1,000 consultations). Metro GP rates peaked in week 35 (week beginning 25 August) at 15.2 cases per 1,000 consultations. Influenza B cases notified directly to the Victorian Department of Human Services (DHS) from other sources peaked in the 1st week of September with peak numbers of influenza A notifications occurring the following week. Overall 56% of notifications of laboratory confirmed influenza to DHS and 56% of influenza positive swabs from sentinel surveillance were influenza type B. *Commun Dis Intell* 2009;33(3):328–336.

Keywords: surveillance, epidemiology, influenza

Introduction

A sentinel general practice (GP) program for the surveillance of influenza like illness (ILI) has been conducted in Victoria by the Victorian Infectious Diseases Reference Laboratory (VIDRL) and the Victorian Department of Human Services (DHS) since 1993. VIDRL coordinates the sentinel GP ILI surveillance program and laboratory testing of cases has been conducted at VIDRL since 1998.[1] Additionally, VIDRL monitors diagnoses of ILI made by the locum medical practitioners through the Melbourne Medical Deputising Service (MMDS). The DHS coordinates the surveillance of all laboratory confirmed influenza in Victoria, a prescribed group B notifiable disease under the *Health (Infectious Diseases) Regulations 2001*.[2]

The objectives of the influenza surveillance system are to:

- monitor the epidemiology of laboratory confirmed influenza in Victoria;
- identify the onset, duration and relative severity of annual influenza seasons in Victoria;
- provide samples for the characterisation of circulating influenza strains in the community to assist in the evaluation of the current season's and formulation of the following season's vaccine; and
- provide a role in early recognition of new influenza viruses and new or emerging respiratory diseases.

Additionally, the World Health Organization (WHO) Collaborating Centre for Reference and Research on Influenza provides data on strain typing of influenza isolates or Victorian influenza positive specimens forwarded by VIDRL and 2 Melbourne hospital laboratories.

In this paper we summarise findings from the Victorian Influenza Surveillance System in 2008.

Methods

General Practice Sentinel Surveillance

In 2008, 47 GPs from 17 metropolitan practices and 20 GPs from 11 rural practices participated in the VIDRL General Practice Sentinel Surveillance (GPSS) program (Figures 1a and 1b), which is approved for continuing professional development points by the Royal Australian College of General Practitioners and the Australian College of Rural and Remote Medicine.

The GPSS program for 2008 operated between 28 April and 2 November (weeks 18–44). This 4 week extension of the surveillance period in comparison with previous years allowed full capture of ILI cases in the later than usual season and improved comparability of our data with other surveillance programs in Australia.

The 67 participating GPs reported the total number of consultations per week and age, sex and vaccination status of any patients presenting with ILI. The

Figure 1a: Distribution of sentinel surveillance sites in metropolitan Victoria, 2008

Figure 1b: Distribution of sentinel surveillance sites in rural Victoria, 2008

accepted case definition for ILI is defined as fever, cough and fatigue/malaise.[3] ILI rates were calculated using the number of ILI patients per 1,000 consultations and were compared with previously established thresholds for Victorian influenza seasons.[4]

Nose and throat swabs were collected from patients presenting within 3 days of the onset of symptoms. Data including age, sex, symptoms (fever, cough, fatigue, myalgia, other), vaccination status and date, and Aboriginal and Australian Torres Strait Islander status were collected. GPs were also asked to indicate their confidence in their clinical diagnosis ('almost certain,' 'probable' or 'less likely'). Formal consent was obtained from all patients from whom a swab was collected. Virus detection was performed in the Virus Identification Laboratory at VIDRL using the respiratory virus reverse-transcriptase-polymerase chain reaction (RT-PCR)-based assay, which detects influenza viruses (types A and B), respiratory syncytial virus, parainfluenza viruses (types 1, 2 and 3), adenoviruses, rhinoviruses and enteroviruses.[5]

All specimens positive for influenza were forwarded to the WHO Collaborating Centre for Reference and Research on Influenza, for virus isolation and identification.

Melbourne Medical Deputising Service

The MMDS, formerly the Melbourne Medical Locum Service, is the largest medical locum service in Australia and has contributed to Victorian influenza surveillance since 2003. The MMDS provides a 24-hour medical service to patients in their own home or aged care facility. VIDRL has password protected access to the clinical database maintained by the MMDS and conducts weekly searches on the terms 'influenza' and 'flu'. This provides weekly rates of influenza-related diagnoses by MMDS clinicians per 1,000 consultations.

Notifications of laboratory confirmed influenza to the Department of Human Services

Under the Health (Infectious Diseases) Regulations 2001,[2] medical practitioners and pathology services are required to notify laboratory confirmed influenza cases to the DHS within five days of the positive test. Records of all laboratory confirmed influenza cases with a 2008 notification date were extracted for analysis from the DHS Notifiable Infectious Diseases Surveillance database on 19 January 2009.

Data collation and reporting

An SQL database (S Long, SL Digital, Melbourne), tailored to the needs of the GPSS program, was developed to assist with data storage and analysis, as well as report generation. Further to GPSS data, the MMDS ILI rate and summary results of all respiratory viruses detected by routine diagnostic PCR assays at VIDRL, were collated and entered weekly.

Summary information was reported to the DHS Communicable Disease Prevention and Control Unit (CDPCU) and to the Australian Government Department of Health and Ageing (DoHA) on a weekly basis. Summary reports of laboratory confirmed influenza notifications were updated daily and posted on the CDPCU website (http://www.health.vic.gov.au/ideas/surveillance/daily.htm). Laboratory confirmed influenza notifications were also reported by the CDPCU to the National Notifiable Diseases Surveillance System (NNDSS) and were published in the DoHA influenza surveillance report at: http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-surveil-ozflu-flucurr.htm

In 2008, surveillance reports were prepared and distributed to all participating GPs, state and territory health departments, other interested health professionals and health agencies, and were also made available on the VIDRL website (http://www.vidrl.org.au) on a weekly basis. Reports included summary strain data on isolates from Australasia and the South East Asia region prepared by the WHO Collaborating Centre for Reference and Research on Influenza, as well as a summary of national influenza activity from the NNDSS website.

Results

Influenza-like illness surveillance

For the first 23 weeks of surveillance, an average of 98% (66/67) of GPs returned tally sheets to VIDRL each week (range 94% to 100%). During the four weeks of the extended season an average of 83% of GPs (range 76% to 87%) continued reporting.

GPs reported having conducted 159,110 consultations (117,719 metropolitan and 41,391 rural) and identified 876 ILI cases (652 metropolitan and 224 rural) during the season; a rate of 5.5 cases per 1,000 consultations for metropolitan GPs and 5.4 cases per 1,000 consultations for rural GPs.

Figure 2 shows the weekly ILI rates by reporting source. All sources reported a relative increase in ILI rates in week 33 (week commencing 11 August), after which seasonal peak rates were reported in weeks 35 (metropolitan; 15.2 ILI cases per 1,000 consultations) and 36 (rural; 15.7 ILI cases per 1,000 consultations, MMDS; 16.2 ILI cases per 1,000 consultations). ILI rates fell in week 37 but rose sharply to secondary peaks in weeks 38 (MMDS; 10.9 ILI cases per 1,000), 39 (metropolitan; 8.3 ILI cases per 1,000 consultations) and 41 (rural; 7.0 ILI cases per 1,000 consultations), before declin-

ing to a combined baseline level of 1.9 ILI cases per 1,000 consultations at the end of week 43. Among consultations conducted by the MMDS during the 2008 surveillance season, 210 patients (0.5%) were diagnosed with 'flu' or 'influenza.' Unlike previous years, in which the MMDS ILI rate was generally higher, rates from MMDS in 2008 were similar to the overall GP ILI rate.[6]

Among the total ILI cases reported by GPs, 52% (458/876) were female and 48% (418/876) were male. The median age of ILI cases was 35 years (range 1–99 years) and 85% (745/876) were reported as being unvaccinated for the season (Table 1).

Using the previously described thresholds for GP sentinel surveillance in Victoria, ILI rates in 2008 were in the mid to high range of normal seasonal activity (Figure 3).

General Practice Sentinel Surveillance laboratory surveillance

Sentinel surveillance GPs submitted swabs to VIDRL from 403 ILI patients. In total, 43% (173/403) of submitted swabs tested positive to one of the respiratory viruses included in the multiplex respiratory RT-PCR. Of these, 29% (51/173) were positive for influenza A, and 38% (65/173) were positive for influenza B (Table 2). Of all swabs in

Figure 2: Weekly influenza-like illness rates reported by rural and metropolitan practices and the Melbourne Medical Deputising Service compared with influenza notifications, Victoria, 2008

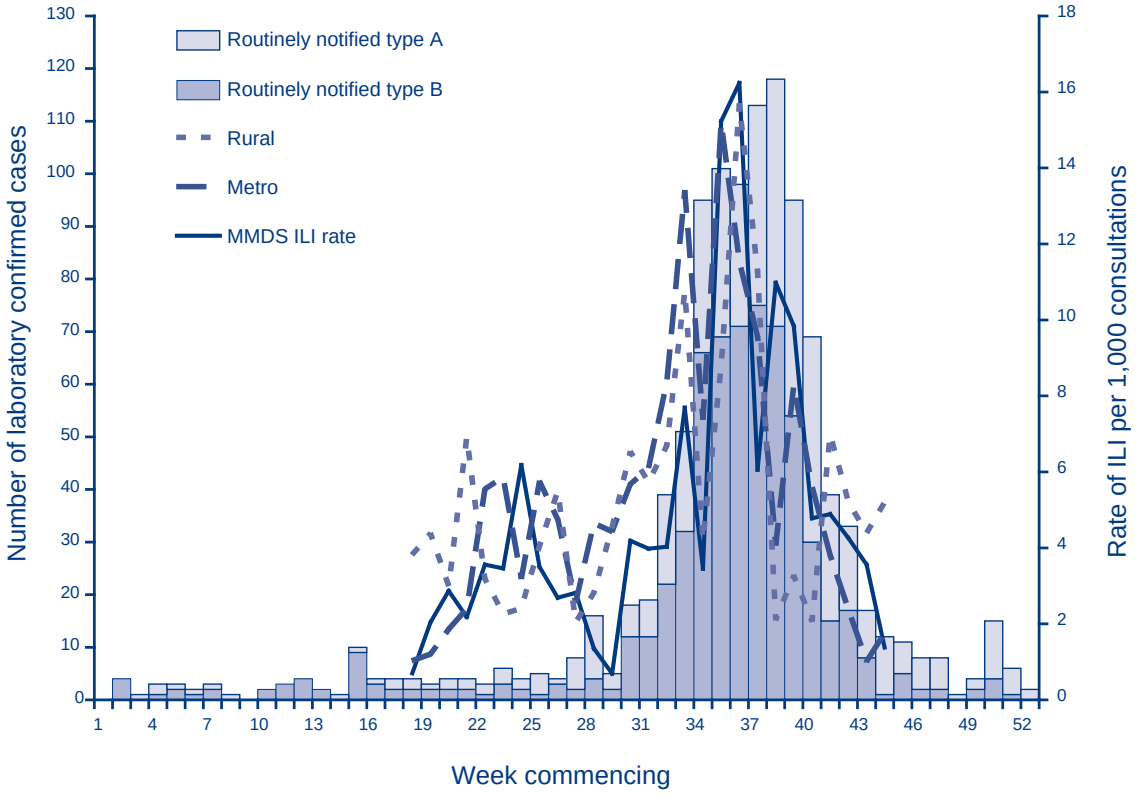


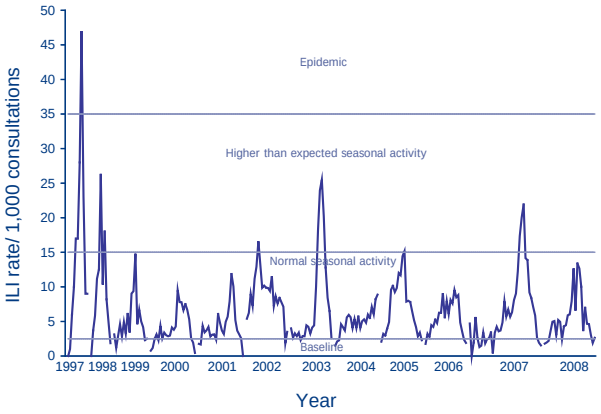
Table 1: Vaccination status for swabs and influenza-like illness patients with known vaccination status, by age group

Age group (years)	Total swabs				Total influenza-like illness patients			
	Vaccinated		Not vaccinated		Vaccinated		Not vaccinated	
	n	%	n	%	n	%	n	%
0–4	1	10.0	9	90.0	0	0.0	37	100.0
5–19	4	5.4	70	94.6	8	4.3	176	95.7
20–54	40	15.4	219	84.6	65	12.6	450	87.4
55–64	8	29.6	19	70.4	18	21.2	67	78.8
65+	21	75.0	7	25.0	40	74.1	14	25.9

which influenza was detected, 56% (65/116) were influenza B, which was higher than the proportion in 2007 (12%) (Table 2).

The positive predictive value (PPV) for the clinical diagnosis of influenza increased with increasing GP confidence in the diagnosis of influenza in 2008 and previous years (Table 3).

Figure 3: Fortnightly general practitioner sentinel surveillance influenza-like illness rates, Victoria, seasons 1997 to 2008



There was no significant difference in the proportion of influenza A or B positive patients by sex (females 53% (61), $P = 0.2$). Of the 116 influenza positive subjects 84% (98) reported being unvaccinated in 2008, 15% (17) being vaccinated and 0.9% (1) had unknown vaccination status.

Figure 4 the relative age distributions of notifications of influenza type A or B according to sentinel and non-sentinel GP notification. From GPSS influenza cases, the median age of patients with influenza A was 31 years (0–96 years) and of patients with influenza B was 20 years (0–58 years).

Notifications to the Department of Human Services

VIDRL was the most frequent notifier of laboratory confirmed cases in 2008, accounting for 30% of notifications. Gippsland Pathology Service and Melbourne Pathology were the next most common notifiers of influenza, comprising 24% and 13% of the total respectively. Eight other Victorian laboratories provided 30% of laboratory notifications and a further 3% of notifications were Victorian residents diagnosed by interstate laboratories.

Table 2: Respiratory viruses detected from general practitioner sentinel surveillance influenza-like illness patient swabs, Victoria, 2008

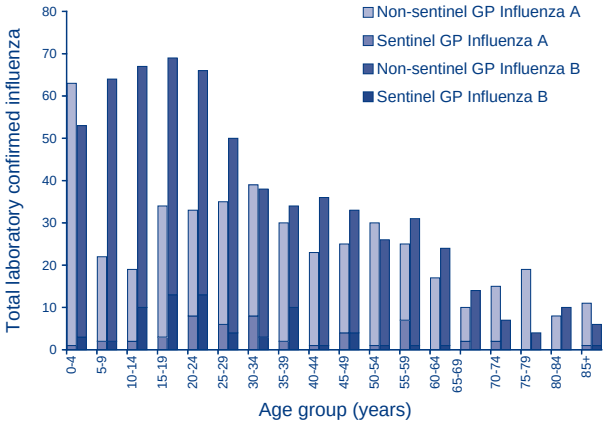
Respiratory virus	n detected	% detected (total swabs)	% detected (PCR positive swabs)
Influenza A	51	13	29
Influenza B	65	16	38
Picornavirus	42	10	24
Adenovirus	4	1	2
Parainfluenza virus	0	0	0
Respiratory syncytial virus	11	3	6
Total	173	43	100

PCR Polymerase chain reaction.

Table 3: Positive predictive value of clinical diagnoses of influenza, Victoria, 2002 to 2008, by general practitioner certainty of diagnosis

Year of surveillance	General practitioner certainty of diagnosis – number laboratory confirmed (PPV)									
	Almost certain		Probable		Less likely		Not stated		Total	
	n	PPV %	n	PPV %	n	PPV %	n	PPV %	n	PPV %
2003	87	45	73	29	4	9	20	38	184	34
2004	12	26	23	16	6	13	2	9	43	16
2005	74	61	90	41	8	15	10	43	182	43
2006	48	51	56	27	11	21	11	41	126	33
2007	85	64	91	44	5	13	8	38	189	47
2008	40	40	61	26	4	9	11	41	116	29

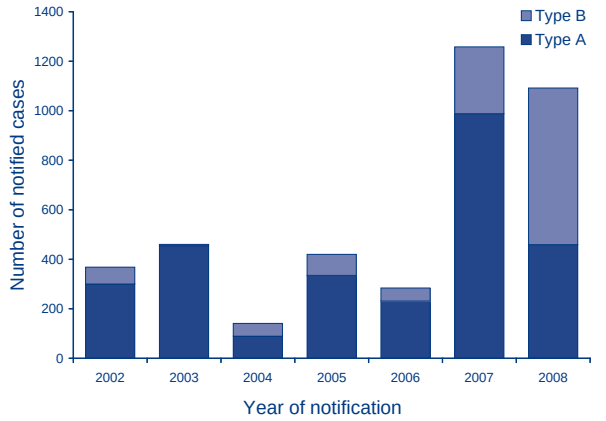
Figure 4: Laboratory confirmed influenza, sentinel and non-sentinel, Victoria, 2008, by age group and notification source



A total of 1,298 laboratory confirmed cases of influenza were notified to the DHS in 2008. Of these, 1,132 (87%) resulted from routine clinical presentations to Victorian GPs and hospitals, 116 (9%) were from sentinel GPs and 49 (4%) were identified from outbreak investigations. Of the total routine clinical presentation notifications in 2008, 90% (1,022/1,132) were identified during the surveillance period. There was a 1:1 male to female ratio among the routinely notified cases and the median age was 30–34 years for influenza A and 20–24 years for influenza B. Of the routinely notified cases, 41% (459/1,132) were influenza A, 56% (633/1,132) were influenza B, 38 (3%) were of an unknown type and two cases were notified with influenza A and B co-infection (Figure 5).

As shown in Figure 2, the trend of routine notifications of influenza A and B cases to DHS increased in parallel with MMDS and GPSS ILI rates. Peak

Figure 5: Notifications of laboratory confirmed influenza to the Department of Human Services from routine clinical presentations, Victoria, 2002 to 2009, by year and type



influenza A activity was observed in week 38 (week beginning 15 September) with 47 notifications and influenza B peak activity was observed in week 37 (week beginning 8 September) with 75 notifications. The total number of routine influenza notifications peaked in week 38 (week beginning 15 September) with 123 notifications, 3 weeks after the GP surveillance, and 2 weeks after the MMDS ILI peak rates were observed.

One case, an 89-year-old male, notified in week 47 (17 November) was reported to have died as a result of type A influenza virus infection. A patient, aged 96 years, was reported to have died with a type B influenza virus infection, but death was subsequently attributed to other causes.

World Health Organization Collaborating Centre for Reference and Research on Influenza

One hundred and sixty-seven specimens and 32 isolates collected in Victoria during 2008 were referred to the WHO Collaborating Centre for Reference and Research on Influenza. Of the 38% (75/199) that were propagated in tissue culture, 56% (42/75) were type A and 44% (33/75) were type B. One type A isolate was further characterised as an H1N1 strain that was A/Brisbane/59/2007-like. The remaining influenza A isolates were H3N2 strains, which were A/Brisbane/10/2007-like (including 8 low reactors). Forty-two per cent (14/33) of influenza type B isolates were designated as B/Florida/4/2006-like, of the B/Yamagata/16/88 lineage virus, and the remaining 58% (19/33) were characterised as B/Malaysia/2506/2004-like (low reactor) viruses, of the B/Victoria/2/87 lineage.

Outbreak investigations

Thirty respiratory outbreaks were notified to DHS in 2008 of which 27 were in aged care facilities. Among the aged care facility outbreaks, eight (30%) were determined to be caused by influenza type A virus, three (11%) by influenza type B virus, two (7%) each by respiratory syncytial virus and picornavirus, one (4%) by parainfluenza virus and the remaining 11 (41%) were of unknown aetiology. The 3 outbreaks not associated with an aged care facility all occurred in the same military facility: one in March was caused by influenza type B virus and there were concurrent outbreaks of influenza type A and type B viruses in August and September.

Discussion

The 2008 influenza season in Victoria was characterised by an unusually high proportion of influenza B virus circulation and a peak of influenza activity much later than previous years (Figure 6). The season remained within the normal seasonal

activity thresholds. The delay between the increased GP and MMDS ILI rates and notifications to DHS can be explained by the time between the visit to a GP, laboratory confirmation of diagnosis and subsequent notification.[7]

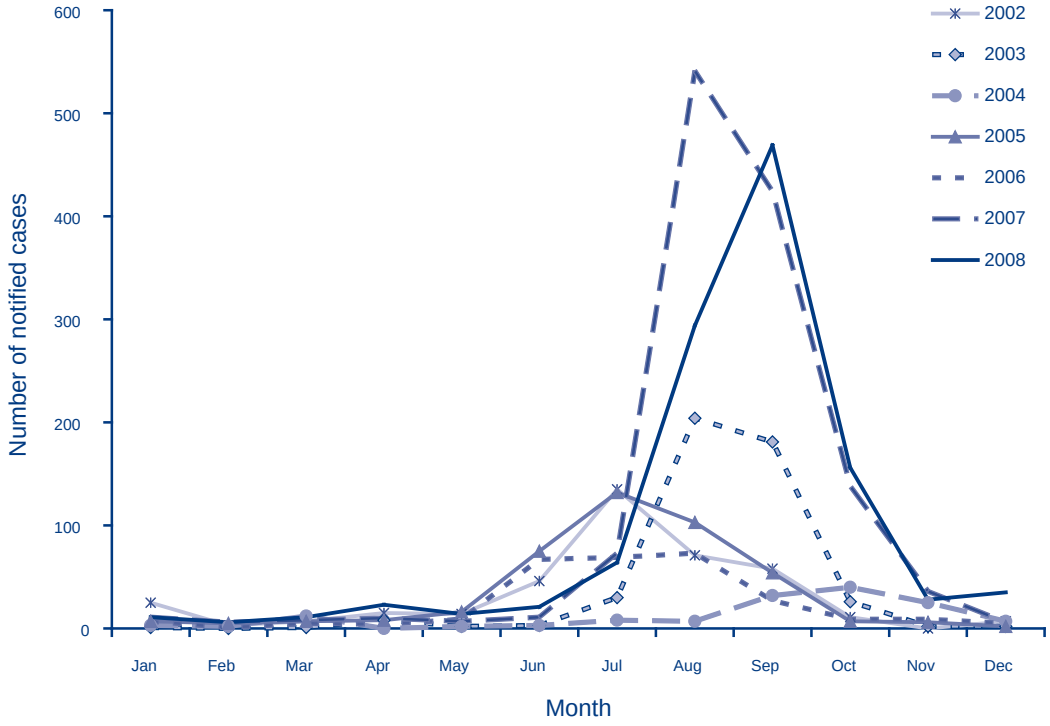
The average GP response rate of 98% was higher than previous years. GPs reported that participation in the program gave them a greater awareness of patterns of influenza symptoms and that feedback assisted with diagnosis, treatment and clinical decision making. This feedback was similar to previous years with weekly reporting further improving the relevance of the information for GPs.

Influenza type B was the predominant circulating seasonal influenza type in 2008 in Victoria. The proportion of influenza B from GPSS swabs was the highest observed since surveillance began, with previous highest percentages of influenza B detections in 2002 (33%) and in 2004 (23%). This increased proportion of influenza B was similarly reflected in laboratory confirmed notifications to DHS. According to NNDSS figures, there has not been an influenza season in Australia in which type B virus has been predominant since influenza became nationally notifiable in 2001.[8] This predominance of influenza B circulation in 2008 was also reported by the WHO in most Asian countries.[9]

Routine (non-sentinel) notifications to DHS of laboratory confirmed influenza are predominantly made from hospitalised patients who tend to be young children [10] and in those 65 years or over.[11] In contrast, workplace and university requirements for sick certificates, as well as greater compliance with swabbing procedures by sentinel surveillance participants, consistently result in a larger proportion of laboratory confirmed influenza from patients in the 15–44 year age group each year.[7]

The proportion of cases in older children and young adults relative to other groups was higher in 2008 compared with other recent influenza seasons.[12,13] This is largely explained by the younger median age of patients with influenza B infection.[14] During an influenza B outbreak in New Zealand in 2005, children in the 5–19 years age group had significant excess morbidity compared with the average from 1995 to 2004 for the same age group, with 3 influenza B associated deaths in children.[15] School absenteeism rates were also significant, with 1 school in Wellington temporarily closing.[16,17] Previous studies have demonstrated higher attack rates with influenza B among school aged children compared with other age groups.[18] An influenza B outbreak in 2006 in North Carolina, United States of America, resulted in an increase in student and school staff absenteeism and nine school closures.[19] In 2008, there were media reports of influenza outbreaks in schools in Australia, with a primary school in Tasmania reporting 250 students and 10 teachers absent during August.[20]

Figure 6: Notifications of laboratory confirmed influenza to the Department of Human Services from routine clinical presentations, 2002 to 2008, Victoria, by month and year



The Southern Hemisphere 2008 influenza vaccine contained A/Solomon Islands/3/2006 (H1N1), A/Brisbane/10/2007 (H3N2) and B/Florida/4/2006 influenza strains.[21] In 2008, the predominant circulating type A strain was H3 (A/Brisbane/10/2007-like – 98% (41/42)) matching the vaccine strain, with only 1 vaccine mismatch H1 strain (A/Brisbane/59/2007-like) identified from the type A circulating strains.

Since 1987 two antigenically distinct strains of influenza B, B/Victoria/2/87 and B/Yamagata/16/88, have co-circulated in varying proportions, causing seasonal outbreaks in Australia and the Asia–Pacific region.[22] In the 2008 influenza season in Victoria, these 2 distinct strains of influenza B (B/Florida/4/2006-like [14/33] of the B/Yamagata/16/88 lineage virus, and B/Malaysia/2506/2004-like [19/33] viruses, of the B/Victoria/2/87 lineage) co-circulated with the influenza A viruses. There is some suggestion that residual protective antibody may be produced against viruses from different influenza B lineages thus providing some residual immunity due to previous exposure to type B viruses of both lineages.[23] This low level cross protection may be due to the greater antigenic stability of influenza B and may explain the higher susceptibility of younger age groups not previously exposed to influenza B.

The 2008 national laboratory confirmed notifications were lower than in the previous year (9,121 notifications in 2008 compared with 10,446 notifications in 2007).[8] As with the previous 4 years the majority of national notifications in 2008 came from Queensland (41%), New South Wales (20%) and Victoria (14%). However, these figures may reflect good surveillance and data capture programs in those states, as well as larger populations rather than truly higher rates of influenza. Whilst the total number of notifications has increased during the last 2 years, with 2007 having higher than expected activity and several highly publicised influenza related deaths, notifications can be influenced year to year by outbreak investigations and testing propensity and may not necessarily reflect the levels of influenza in the community.

Threshold analysis indicated that the 2008 season remained within the normal seasonal activity levels, and highlights the importance of using several influenza and influenza-like illness surveillance systems to describe and assess the epidemiology of each season.

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Short reports

ROTAVIRUS VACCINATION ONE YEAR ON...

Daniel A Belshaw, David J Muscatello, Mark J Ferson, Alma Nurkic

Abstract

The rotavirus vaccine was incorporated into the Australian National Immunisation Program from 1 July 2007. To measure early impact of the vaccine on rotavirus disease and the burden of gastroenteritis in young children, we examined 2 surveillance data sources, rotavirus isolates from selected New South Wales laboratories, and New South Wales Emergency Department (ED) visits assigned a gastroenteritis-related diagnosis. Between 2001 and 2008, weekly time series were prepared for 2 age groups representing children young enough to have been offered vaccination prior to the 2008 seasonal epidemic (< 15 months) and older children (15 months to 5 years). In 2008, the seasonal increase in laboratory confirmed rotavirus infection and gastroenteritis related ED visits declined substantially in both age groups compared with earlier years. These data provide preliminary evidence of the effectiveness of the rotavirus vaccination program in New South Wales. Immunising the most susceptible population group, infants, against rotavirus may limit wider circulation of the virus in older children. *Commun Dis Intell* 2009;33(3):337–340.

Keywords: rotavirus, vaccine, immunisation, gastroenteritis, emergency department, surveillance

Introduction

Rotavirus is a leading cause of gastroenteritis in children, with an estimated mortality as high as 702,000 deaths per year internationally, including 440,000 deaths in children aged under 5 years.[1,2] Eighty-five per cent of deaths occur in low income countries in Africa and Asia.[2]

Fortunately, in Australia, rotavirus was estimated to account for less than 1 death per year during 1990 to 2004.[3] Nevertheless, during 1993 to 1996, rotavirus gastroenteritis resulted in 10,000 hospitalisations per year and accounted for up to 50% of hospitalisations for diarrhoea in Australian children aged under 5 years.[4] Between 1998 and 2006, there were an estimated 22,000 emergency department (ED) presentations annually attributable to rotavirus.[5] In 2006, the estimated cost to the Australian health care system for rotavirus was estimated to be $30 million.[5] In 1997, nearly half of the primary carers and three-quarters of secondary carers of children with confirmed rotavirus suffered from a loss of income or required leave from employment to care for their children.[6]

In Australia, the 0–5 year age group (inclusive) experiences the greatest burden of rotavirus gastroenteritis, with those aged under 2 years representing 72% of all positive laboratory results.[7] Like many gastrointestinal infections, symptoms of rotavirus infection include severe diarrhoea, vomiting and intolerance to large volumes of fluid. The younger the child, the more rapid is the onset of dehydration and electrolyte imbalances.[8]

Rotavirus is seasonal and, in the more temperate regions of Australia, the highest rates occur within the cooler months. In New South Wales this period can range between June to October with the peak occurring in August to September.[5,8,9]

As of 1 July 2007 rotavirus vaccination was incorporated into the National Immunisation Program in Australia.[10] The Program provides free voluntary vaccination to all children and is administered by the Australian Government Department of Health and Ageing and implemented by State and Territory health agencies. In New South Wales, rotavirus vaccination commenced on 1 July 2007, using the oral live attenuated vaccine, Rotarix®, given at 2 and 4 months of age.[11]

Recommendations from the Rotavirus surveillance in Australia report suggest utilising laboratory positive rotavirus results, and general practice and ED presentations, to monitor the effectiveness of the rotavirus vaccine.[12] A previous study in New South Wales in 1996 showed a direct correlation between laboratory surveillance for rotavirus and gastroenteritis in EDs in children aged under 5 years and further confirmed the seasonal pattern.[13]

Based on the above recommendations we examined both laboratory and ED surveillance data to assess the early impact of the vaccine on the burden of rotavirus in young children in New South Wales.

Methods

For the period 2001 to 2008, we examined weekly time series of counts of laboratory confirmed rotavirus infection from 2 public pathology providers, and visits to New South Wales EDs assigned a primary ED diagnosis of gastroenteritis or gastroenteritis symptoms, which include nausea, vomiting, and/or diarrhoea.

Children 2 months of age were eligible to be vaccinated on 1 July 2007 and would be aged 15 months on 1 August 2008, which would be the approximate mid-point of the seasonal increase. The time series were prepared for 2 age groups: children aged less than 15 months, who would have been offered and have completed the vaccine regime before the 2008 season; and those aged 15 months to 5 years, who were too old to have been included in the program.

Laboratory data were collected from 2 public pathology providers under the South Eastern Sydney Laboratory Surveillance Program. One of these provided services to a children's hospital and the second provided services to 4 adult and 1 children's hospital. Together, the laboratories provided services to both New South Wales paediatric hospitals.

Data for ED presentations were selected from the New South Wales Emergency Department Data Collection[14] based on primary ED diagnoses selected using International Classification of Diseases ICD-9, ICD-10 and SNOMED Clinical Terminology codes for gastroenteritis or gastroenteritis symptoms (codes available from the corresponding author). For the period analysed, this database contains reasonably complete data for 43 hospitals in both metropolitan and regional New South Wales, and these hospitals capture approximately two-thirds of all New South Wales ED presentations.

Catchment populations for New South Wales EDs cannot be determined, therefore disease incidence rates based on the New South Wales population will be under-enumerated due to the incomplete ED coverage. We nevertheless calculated population rates in order to account for changes in the population at risk over time. Rates were only calculated during the rotavirus season, June to October inclusive, and were annualised to be comparable to full year population rates.

Results

The laboratory and ED time series clearly demonstrate the seasonal nature of rotavirus infection in New South Wales, with incidence peaking during June to October each year. Increases in rotavirus activity clearly coincide with increases in gastroenteritis presentations to EDs in children aged 5 years and under. Prior to 2008, the burden of rotavirus varied from year to year, with the lowest burden in 2004 (Table 1 and Figure). Children under the age of 15 months showed a disproportionately high burden of disease, annually accounting for 30% to 50% of ED presentations in children aged 5 years and under during the rotavirus season.

In 2008, the lowest count in 8 years of rotavirus infections was recorded in the laboratory data, with 117 confirmed infections identified. This compares with a minimum of 178 in 2004. In children aged under 15 months, the count of 36 in 2008 was dramatically lower than in any of the previous 7 years (minimum 83 in 2007). In Children aged 15 months to 5 years, the 2008 count of 81 was the second lowest of the 8 years, with the previous minimum being 76 in 2004 (Table 1). While the weekly counts showed little seasonal activity in the laboratory data in 2008, a distinct but smaller seasonal increase was evident in the weekly ED presentations for gastroenteritis in both age groups (Figure).

In children aged under 15 months, the annualised rate of gastroenteritis ED presentations for the period June to October (75 per 1,000) was lower in 2008 than in any of the previous 7 years (80.6–131.0 per 1,000). A similar pattern was evident in the older age group, with a rate of 26.0 per 1,000 during June to October 2008 compared with 28.9–51.5 per 1,000 in the 7 previous years (Table 2).

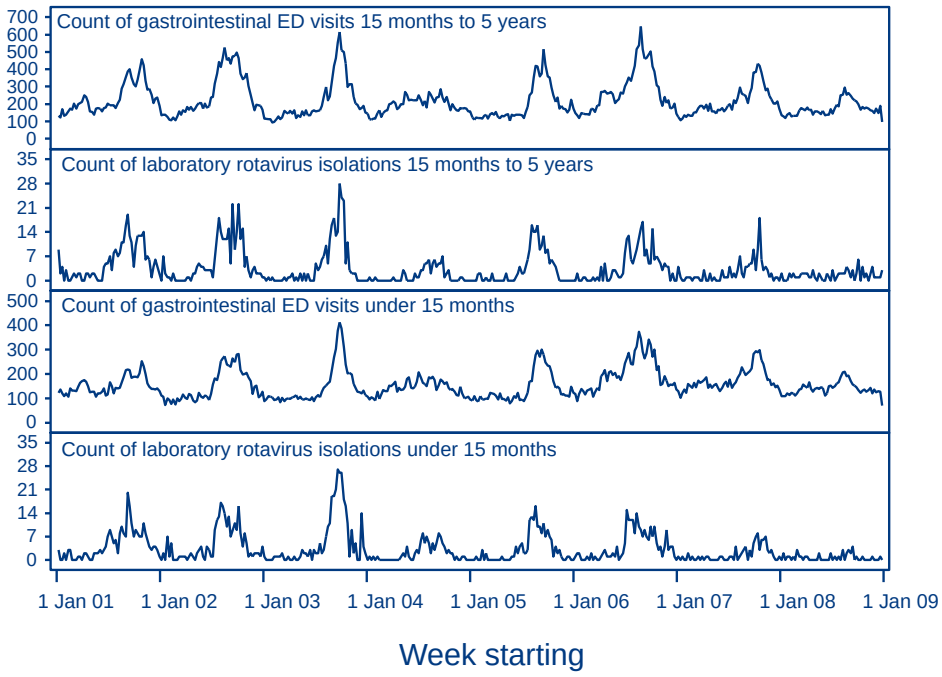
Discussion and conclusion

We found that introduction of the rotavirus vaccine was associated with the lowest counts of positive

Table 1: Counts of laboratory rotavirus isolations by year and age from two public pathology laboratories, 2001 to 2008

Year	Age under 15 months	Age 15 months to 5 years	Age 0 to 5 years
2001	239	276	515
2002	238	281	519
2003	285	229	514
2004	102	76	178
2005	165	170	335
2006	219	215	434
2007	83	122	205
2008	36	81	117
Total	1,367	1,450	2,817

Figure: Weekly counts of laboratory-positive rotavirus isolates and ED gastroenteritis presentations, children aged under 15 months and 15 months to 5 years, 2001 to 2008



Laboratory data are from 2 public pathology laboratories in New South Wales and the emergency department (ED) data are from 43 New South Wales hospitals, and therefore do not include the entire New South Wales population.

Table 2: Annualised counts and rates per 1,000 population of gastroenteritis presentations to 43 New South Wales emergency departments during and excluding the rotavirus season, June to October, New South Wales, 2001 to 2008

Year	Children aged under 15 months				Children aged 15 months to 5 years			
	Presentations June–October	Annualised rate 1,000 June–October	Presentations other months	Annualised rate per 1,000 other months	Presentations June–October	Annualised rate per 1,000 June–October	Presentations other months	Annualised rate per 1,000 other months
2001	3,795	84.0	4,129	65.3	6,163	35.6	5,937	24.5
2002	4,435	101.3	3,374	55.1	8,085	47.2	5,273	22.0
2003	4,267	80.6	3,409	46.0	6,557	38.1	4,695	19.5
2004	3,617	81.3	3,903	62.7	4,955	28.9	5,310	22.1
2005	4,014	90.0	3,845	61.6	5,918	34.6	5,145	21.5
2006	6,084	131.0	3,856	59.3	8,824	51.5	4,955	20.6
2007	4,609	100.0	4,537	70.3	5,866	34.5	5,202	21.9
2008	3,428	75.0	3,997	62.5	4,392	26.0	4,736	20.1

Rates underestimate the actual New South Wales population rate because not all New South Wales hospitals are included.

laboratory rotavirus isolates and presentations to EDs for gastroenteritis during the usual rotavirus season in the 8 years to 2008. While laboratory isolates were virtually absent during 2008, there remained evidence of residual seasonal activity in the ED time series. The declines occurred in the age group that would have been offered the vaccine prior to the 2008 season, and older children.

The apparent decrease in both the immunised and non-immunised age groups could be explained by unusually low community circulation of rotavirus in 2008 or by a beneficial effect of vaccination that had a flow-on effect to older children through reduced transmission in the most susceptible age group, infants. This phenomenon has been observed elsewhere,[15,16] but further studies will be required to confirm this apparent herd immunity.

Rotarix® vaccine contains a single, attenuated human rotavirus of serotype G1P[8] and has an efficacy of up to 84.7% against severe rotavirus

gastroenteritis caused by strains that carry the P[8] antigen.[17] In Australia during 2006 to 2007, the most common serotypes for rotavirus were the G1, G3, and G9 strains and each of these possess the P[8] antigen.[7] If rotavirus strains in Australia have not changed since 2006–2007, then Rotarix® should have been effective during the 2008 season. When more recent strain data become available, a clearer picture should emerge on whether a benefit of vaccination should have been expected.

A limitation of this study is that the ED presentation data does not discriminate between rotavirus gastroenteritis and gastroenteritis cause by other infectious and non-infectious agents. Nevertheless, the ED data collection covers a large proportion of the New South Wales population and provides a rapid means of assessing the burden of gastroenteritis in the New South Wales population. Laboratory data were restricted to only 6 hospitals, and this could explain the difference in apparent 2008 seasonal activity between the laboratory and ED time series – geographic variation in vaccine uptake might explain this difference.

As recommended by Ward et al,[12] both laboratory and non-laboratory surveillance of vaccine preventable diseases are valuable in evaluating the effectiveness of the rotavirus immunisation program. It appears likely that the introduction of the rotavirus vaccine in New South Wales has been associated with a marked decline in rotavirus and associated health utilisation activity. This will need to be confirmed by continued surveillance.

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Quarterly reports

OzFoodNet quarterly report, 1 April to 30 June 2009

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 April to 30 June 2009.

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 2nd quarter of 2009, 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 4,166 people, of whom 140 were hospitalised. There were 20 deaths reported during these outbreaks. The majority of outbreaks (64%, n=157) were due to person-to-person transmission (Table 1).

Table 1: Mode of transmission for outbreaks of gastrointestinal illness reported by OzFoodNet, 1 April to 30 June 2009

Transmission mode	Number of outbreaks	Per cent of total
Foodborne	27	11
Person-to-person	157	64
Salmonella cluster	5	2
Unknown – other pathogen cluster	3	1
Unknown	52	21
Total	244	100

Foodborne disease outbreaks

There were 27 outbreaks during this quarter where consumption of contaminated food was suspected or confirmed as the primary mode of transmission (Table 2). These outbreaks affected 419 people and resulted in 68 hospitalisations. There were 2 reported deaths during these outbreaks. This compares with 25 outbreaks for the 2nd quarter of 2008 and 43 foodborne outbreaks for the 1st quarter of 2009.

Salmonella was responsible for 12 outbreaks during this quarter, with *S.* Typhimurium being the most common serotype. There were 4 outbreaks due to *S.* Typhimurium phage type 170, and 1 each due to *S.* Typhimurium phage types 6, 29, 44, 135 and 135a. There was 1 outbreak of *S.* Typhimurium where phage typing was not reported and 1 outbreak each due to *S.* Litchfield and *S.* Virchow 34.

Of the remaining 15 outbreaks, two were due to foodborne toxins, including 1 *Clostridium perfringens* outbreak and 1 histamine poisoning outbreak associated with a fish meal. There were 3 outbreaks due to norovirus and 1 outbreak of hepatitis A infection. One outbreak was due to waxy esters in Escolar fish. The remaining 8 outbreaks were of unknown aetiology.

Ten outbreaks (37%) reported in this quarter were associated with food prepared in restaurants, four (15%) were associated with aged care facilities and two (7%) with bakeries and commercial caterers. Individual outbreaks were associated with primary produce, or food prepared at a fair or festival, a takeaway and a community event. Five outbreaks (21%) were associated with other settings.

To investigate these outbreaks, sites conducted 5 cohort studies, 2 case control studies, and collected descriptive case series data for 20 investigations. As evidence for the implicated food vehicle or foodborne transmission, investigators obtained microbiological evidence in 3 outbreaks, analytical epidemiological evidence in 2 outbreaks, and both analytical epidemiological and microbiological evidence in 2 outbreaks. Descriptive evidence only was obtained in 20 outbreaks.

Table 2: Outbreaks of foodborne disease reported by OzFoodNet sites,* 1 April to 30 June 2009 (n=28)

State or territory	Month of outbreak	Setting prepared	Agent	Number affected	Hospitalised	Evidence	Responsible vehicles
ACT	May	Contaminated primary produce	Waxy esters	3	0	D	Escolar fish
NSW	April	Aged care facility	*C. perfringens*	16	2	M	Unknown
	April	Restaurant	Unknown	5	0	D	Possible lasagne, chicken Caesar salad
	April	Other	Unknown	7	0	D	Unknown
	April	Restaurant	Norovirus	16	1	D	Unknown
	April†	Fair, festival, other temporary/mobile service	*S.* Typhimurium 170	8	2	D	Raw egg sauces
	June	Restaurant	Unknown	15	2	D	Unknown
	March	Bakery	*S.* Virchow 34	10	3	M	Suspected pork rolls
	March	Bakery	*S.* Typhimurium 170	8	1	D	Suspected chicken/pork rolls
	May	Other	Unknown	15	0	D	Unknown
	May	Restaurant	*S.* Typhimurium 29	3	1	D	Unknown
	May	Other	Unknown	4	1	D	Mixed sandwiches
Qld	April†	Restaurant	*S.* Typhimurium 170	3	0	D	Unknown
	May	Other	Histamine poisoning	6	0	M	Tuna
	May	Takeaway	Unknown	2	0	D	Prawn roll
	May	Restaurant	Norovirus	17	1	D	Unknown
SA	April	Commercial caterer	*S.* Typhimurium 44	8	0	AM	Aioli
	May	Restaurant	*S.* Typhimurium 135	10	0	A	Fried ice cream
Vic	April	Aged care facility	*S.* Typhimurium 170	12	0	D	Unknown
	April	Restaurant	Unknown	6	0	D	Unknown
	May	Aged care facility	Unknown	7	0	D	Unknown
	May	Aged care facility	Norovirus	17	1	D	Unknown
WA	June	Restaurant	*S.* Typhimurium 135a	7	1	D	Unknown
	May	Restaurant	*S.* Typhimurium	8	2	D	Unknown
	May	Commercial caterer	*S.* Typhimurium 6	5	0	D	Unknown
Multi-jurisdictional	April	Contaminated primary produce	Hepatitis A	125	50	AM	Semi-dried tomatoes
	June	Other	*S.* Litchfield	76	0	A	Suspected barramundi

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.

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

† These outbreaks were part of the multi-jurisdictional investigation into *S.* Typhimurium 170/108.

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

Australian Capital Territory

There was 1 small outbreak of escolar-induced keriorrhoea (oily diarrhoea due to consumption of waxy esters) in the Australian Capital Territory during the quarter. Eighteen hours after eating a home prepared fish meal, 2 adults and an infant experienced keriorrhoea, abdominal pain and nausea that lasted for 3 days. The fish was being sold as 'deep sea cod/escolar', which a local fishmonger had purchased from the Sydney fish markets.

New South Wales

New South Wales reported 11 foodborne or suspected foodborne disease outbreaks in the 2nd quarter of 2009, with four of these being due to *Salmonella*.

In the first of the *Salmonella* outbreaks, there were 4 notifications of *S.* Typhimurium multi-locus variable number of tandem repeats analysis (MLVA) 3-11-10-8-523, three of whom were employees at the same restaurant and 1 restaurant patron. Cases onsets occurred over 3 weeks. Person-to-person transmission was suspected as the cause of the 3 restaurant workers becoming ill.

An outbreak of *S.* Typhimurium 170, MLVA 3-9-7-13-532 affected 8 cases that were linked to a single Vietnamese bakery. Chicken and pork rolls were identified as the likely source of infection, but ingredients and environmental swabs tested negative for *Salmonella*. In a 2nd outbreak associated with a Vietnamese bakery, 11 cases of *S.* Virchow were reported through routine surveillance. The age of cases ranged between seven and 82 years. The suspected vehicle was raw egg in the egg butter used in the pork rolls. The New South Wales Food Authority inspected the premises and collected food and environmental samples, all of which tested negative except for the swab of the sink, which was positive for *S.* Virchow 34. A trace back of eggs used at the bakery was conducted and eggs collected at the commercial supplier were tested, but no *Salmonella* was detected.

During a multi-jurisdictional outbreak investigation of *S.* Typhimurium 170/108, OzFoodNet Hunter New England interviewed 18 cases (MLVA type 3-9-7-12-523). Eight of these cases were associated with a point source outbreak. This investigation is detailed further in the section on multi-jurisdictional outbreak investigations.

The other foodborne investigations included an investigation of *C. perfringens* at an aged care facility with 16 residents affected, two of whom subsequently died, and an outbreak of norovirus that affected 20 out of 64 guests at a wedding reception, one of whom was hospitalised.

Northern Territory

During June, the Northern Territory was notified of cases of gastroenteritis occurring among participants of a charity car rally. Six cases were notified with *S.* Litchfield infection, which is a common serotype in northern parts of Australia. OzFoodNet coordinated an investigation into the outbreak, which is detailed in the section on multi-jurisdictional outbreaks.

Queensland

Four outbreaks of foodborne or suspected foodborne illness were investigated in Queensland during the 2nd quarter of 2009.

During a multi-jurisdictional outbreak investigation of *Salmonella* Typhimurium 170, 4 cases of *S.* Typhimurium 170 with the same MLVA profile (1-13-3-21-3) were interviewed. Three of these cases were associated with a point source outbreak detailed further in the section on multi-jurisdictional outbreak investigations.

Queensland Health investigated 6 cases of histamine poisoning among 3 families in May 2009. Each had consumed fish from the same 35 kg tuna caught in waters off the Sunshine Coast. Reported symptoms included diarrhoea, fever and headaches. Five thousand milligrams per kilo of histamine was detected in a 2 kg sample taken from the implicated fish, which was approximately 10 times that which is known to cause illness (500 mg/kg). The fish was subsequently removed from sale.

An outbreak of gastroenteritis due to norovirus affected 17 patrons and staff members of a Brisbane hotel following a lunch meal in May. Several food handlers were ill with gastroenteritis prior to the event and a cohort investigation was unable to identify a specific food vehicle.

Two people who consumed prawn sushi rolls from a Brisbane store in May became ill with vomiting, diarrhoea and stomach cramps 2 hours after consuming the meal. No specimens were tested and the aetiology was unknown.

South Australia

South Australia reported 3 outbreaks of foodborne or suspected foodborne disease in the second quarter of 2009, including one that was part of a multi-jurisdictional outbreak of hepatitis A.

The Communicable Disease Control Branch (CDCB) investigated an outbreak of 37 cases of locally acquired hepatitis A identified in South Australia between March and June 2009. A case control study found semi-dried tomatoes were significantly associated with illness. See further detail under the section on multi-jurisdictional outbreak investigations.

In April 2009, CDCB investigated a report of gastrointestinal illness among attendees at a catered event in Adelaide. Thirty out of approximately 200 attendees reported illness of which nine were confirmed as being infected with *S.* Typhimurium phage type 44. A cohort study found a significant association between consumption of aioli, made from raw eggs, and illness among attendees (RR 5.4 95% CI: 1.6,18.1). An environmental investigation detected *S.* Typhimurium 44 in the aioli, but all other samples tested (including eggs) were negative.

An outbreak of *S.* Typhimurium phage type 135 was investigated in May 2009 with eight of 9 cases interviewed eating fried ice cream at the same restaurant. An environmental investigation was undertaken and samples collected. All samples were negative for *Salmonella*. Fried ice cream was removed from the menu and advice provided regarding the use of pasteurised egg for fried ice cream batter.

Victoria

Victoria reported 5 outbreaks of foodborne or suspected foodborne disease during the quarter, including 1 outbreak that was part of the multi-jurisdictional outbreak of hepatitis A.

In April, 12 residents of an aged care facility became unwell with gastroenteritis. *S.* Typhimurium 170 was isolated from faecal specimens of 7 cases and *S.* Typhimurium 44 from 1 further case. The source of this outbreak was unclear, however case patients became ill over a 1 month period suggesting that there may have been low dose sporadic contamination of some foods due to inadequately cleaned kitchen surfaces and/or equipment.

The Communicable Diseases Control Unit (CDCU) was notified of an outbreak of vomiting and diarrhoea in a group of 8 people attending meetings in April. The chef at the hotel where the meetings were held reported leaving work early on the 2nd day of the meeting due to a diarrhoeal illness. The aetiology of the outbreak could not be confirmed as no stool specimens were collected.

Seven residents of an aged care facility became ill with diarrhoea on the same day in May. Roast beef prepared on the previous day was suspected as the cause of this outbreak. One faecal specimen was collected and *C. perfringens* enterotoxin was detected.

In May, an outbreak of gastroenteritis due to norovirus was reported in 2 staff members and 15 residents of an aged care facility. A food handler at the facility became ill and returned to work sooner than 48 hours after symptoms resolved. Following this, 8 residents and another staff member became ill on the same day, with a further 3 residents on the following day. This outbreak was likely to have commenced as a point source foodborne outbreak rather than person-to-person transmission. The 4 remaining cases were likely to have become ill through secondary person-to-person transmission.

Between early March and late May, 86 cases of hepatitis A were notified in Victorian residents. This investigation is detailed further in the section on multi-jurisdictional outbreak investigations.

Western Australia

There were 3 outbreaks of foodborne or suspected foodborne disease investigated in the 2nd quarter of 2009 in Western Australia.

In the 1st outbreak, 17 cases of *S.* Typhimurium pulsed field gel electrophoresis (PFGE) type 0003 cases (phage type 135) were notified from April to early June. Of these cases, eight reported eating or attending the same Chinese food outlet. The cases ate a range of food from the outlet including noodles, honey chicken, satay beef, chicken on a stick and omelettes. An environmental investigation found the premises to have satisfactory food preparation and hygiene practices. Food samples and swabs were negative for *Salmonella*. No common exposure could be identified for the 9 cases that had not eaten at the food outlet.

In the 2nd outbreak, 2 cases of *S.* Typhimurium PFGE type 0018 (phage type 6) were part of an extended family party of 10 who attended a catered mother's day lunch in May at a European community function centre. Three other members of this party were also ill. Food at the lunch was served as a buffet and included lamb, pasta, pork, chicken, salads and cakes. As there was no booking list other cases could not be identified. No further cases of this PFGE type were notified in June or July.

In the 3rd outbreak, 7 cases of *S.* Typhimurium PFGE 0200 notified between late May and late June were investigated. Five cases had attended a wedding reception in early May and 2 cases attended a christening in mid-May, all at the same function centre. An Italian buffet with similar foods was served at both functions. Foods eaten by cases at both functions included: prosciutto, pizza, ice cream, squid and food garnished with parsley and snow pea sprouts. An environmental investigation found that the premises had satisfactory food storage, preparation and hand hygiene practices. Food samples were negative for *Salmonella*.

Multi-jurisdictional outbreak investigations

Salmonella *Typhimurium 170/108*

A large increase in notifications of *S.* Typhimurium 170/108 in the first 3 months of 2009 triggered a multi-jurisdictional investigation in Queensland, New South Wales, Victoria and the Australian Capital Territory during April 2009. The Microbiological Diagnostic Unit at the University of Melbourne developed an outbreak case definition based on 3 main outbreak strains of interest, referred to as MLVA 1, MLVA 2 and MLVA 3. In total, there were in excess of 600 cases in this outbreak. During this investigation, 2 point source outbreaks were identified:

- OzFoodNet Hunter New England OzFoodNet investigators interviewed 18 cases with *S.* Typhimurium 170 MLVA 3, eight of whom attended a food festival in Sydney and consumed a common dessert. Raw eggs used in the dessert were suspected as a possible source, although no pathogens were detected during environmental investigations. The restaurant was warned about the use of raw eggs in ready-to-eat foods. A subsequent inspection found that the restaurant was using pasteurised eggs in all foods containing raw eggs.
- Queensland identified 4 cases of *S.* Typhimurium 170 MLVA 3 in April 2009. All of the cases were females from Brisbane aged between 26 and 49 years. Three of the 4 cases had consumed different meals from the same Brisbane hotel during the same week. An environmental health inspection was undertaken, which identified the use of raw eggs during the preparation of large quantities of mayonnaise (10 litre containers). This was subsequently used as a base for a variety of other sauces and recipes which had no further cooking steps. Trace back identified that eggs used within the hotel were sourced both from Queensland and New South Wales companies. Visibly dirty eggs were observed in the hotel kitchen. The hotel managers were advised to cease using soiled eggs during the preparation of meals. Egg and mayonnaise samples collected from the premises tested negative for bacterial pathogens.

Locally-acquired hepatitis A

State and territory health departments participated in a multi-jurisdictional outbreak investigation of hepatitis A associated with semi-dried tomatoes during the 2nd quarter of 2009. All jurisdictions were asked to interview all cases of hepatitis A to determine if they had travelled and to administer hypothesis generating questionnaires. Increases in locally acquired hepatitis A cases were identified in Victoria, Queensland, South Australia and Western Australia.

Between 1 March and 27 May, 86 cases of hepatitis A were notified in Victorian residents. The majority of these cases (n=74) reported none of the usual risk factors for hepatitis A. In Queensland, a total of 72% (13/18) of cases notified between April and June 2009 were locally-acquired. Seven Queensland cases recalled consuming loose semi-dried tomatoes purchased from various delicatessens prior to illness. South Australia reported 37 locally-acquired cases between March and June 2009.

South Australia, conducted a case control study examining various risk factors for locally-acquired disease, which found semi-dried tomatoes were significantly associated with illness (OR 5.7, 95% CI: 1.5, 21.9). Trace back investigations conducted by South Australia identified a single company distributing the majority of product within the State. The distributor conducted a voluntary recall of potentially affected semi-dried tomatoes.

A multi-state case control study of cases from Victoria (34), Queensland (8) and New South Wales (2) confirmed the association between hepatitis A and semi-dried tomatoes (OR=3.6, 95% CI 1.6,8.3). The 2 New South Wales cases had exposures in Victoria during their incubation period. Trace back for semi-dried tomatoes was complicated and no specific source could be identified. Genotyping of hepatitis A confirmed that locally-acquired infections were part of an outbreak. Samples of semi-dried tomatoes were sent to an overseas laboratory for testing for hepatitis A by polymerase chain reaction-based tests. Hepatitis A genomic material was detected in some samples, but results were difficult to interpret. As a further precaution the South Australia and Victorian Health Departments issued a public health warning advising the public to avoid eating specified semi-dried tomatoes.

As part of the multi-state investigation, Western Australia investigated a cluster of 7 locally-acquired cases of hepatitis A. One of the cases was found to

have the outbreak genotype strain associated with the multi-state outbreak linked to semi-dried tomatoes. Five of the other cases were from 1 regional area of Western Australia, and genetic sequencing of hepatitis A virus from three of these cases showed that they had the same genetic strain as each other, which was different from the outbreak strain associated with semi-dried tomatoes. Four of the 5 cases reported that they had eaten frozen berries during the incubation period. Frozen raspberries were sampled from the home of 1 case and were positive for hepatitis A genomic material. Trace back could not identify a common supplier for frozen berries consumed by the cases.

Cases of hepatitis A declined nationally in June 2009 leading to the national investigation being closed. Since that time, case numbers increased markedly in the State of Victoria with some cases occurring in other jurisdictions, resulting in a re-opening of the investigation.

Salmonella Litchfield outbreak associated with a car rally

In June, the Northern Territory was informed of illness occurring during a charity car rally travelling through Queensland and the Northern Territory. OzFoodNet coordinated a multi-jurisdictional investigation to identify the cause of the outbreak. The investigation team contacted 286 participants by email inviting them to fill in an online survey. In total, 43% (76/176) of responses were ill with gastroenteritis, including 6 people who were notified with *S.* Litchfield. A variety of foods and meals were associated with illness, with barramundi having the highest relative risk for illness, although a source of illness was not definitively identified.

Cluster investigations

During the 2nd quarter of 2009, OzFoodNet sites investigated several clusters. A cluster is defined as an increase in a specific infection in terms of time, place or person where a source of infection is not obvious. The majority of these investigations involved *Salmonella* serotypes, including Singapore, Wangata, Typhimurium phage type 170 and Typhimurium PFGE type 0039 (not phage typed).

Comments

During the quarter, there were 3 multi-jurisdictional investigations of foodborne disease. The investigation of *S.* Typhimurium 170/108 was carried over from the previous quarter and was suspected to be largely due to eggs, which are a consistent cause of *Salmonella* outbreaks in Australia.[1]

During this quarter, OzFoodNet coordinated a multi-jurisdictional investigation into an outbreak of hepatitis A infection linked to consumption of semi-dried tomatoes. The investigation relied on cooperation between the jurisdictions in the investigation of cases and provision of public warnings, however trace back was complex and a recall of affected food could only be enacted in one jurisdiction where the supply chain was clear. The source of contamination for the affected products remains unclear. Produce items are frequently implicated as vehicles of gastroenteritis outbreaks in Australia.[1] This is the first hepatitis A outbreak associated with fruits and vegetables in Australia, although these foods have been associated with outbreaks overseas.[2,3] The investigation was complex due to the long incubation period for hepatitis A making it difficult for cases to recall specific exposures.

The 2 outbreaks of salmonellosis associated with pork and chicken rolls from Vietnamese bakeries are a concern. Bakeries producing these foods use several ingredients, including raw eggs, chicken, pork and liver, which may be high risk for *Salmonella.* Vietnamese rolls have been responsible for some of Australia's largest and most severe foodborne outbreaks.[4] Health departments and food authorities have developed specific programs to improve food safety in these food premises over the last 10 years. The regular occurrence of these outbreaks highlights the need for continued work with bakery proprietors.

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Communicable diseases surveillance

Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 56,246 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 April and 30 June 2009 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
STEC, VTEC	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis <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
Lyssavirus (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* 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 April to 30 June 2009, by date of diagnosis*

Disease	State or territory								Total 2nd quarter 2009†	Total 1st quarter 2009	Total 2nd quarter 2008	Last 5 years mean 2nd quarter	Year to date 2009	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.4	0.0
Hepatitis B (newly acquired)	2	8	1	14	4	2	25	0	56	40	66	71.4	96	141.6	0.8
Hepatitis B (unspecified)	17	820	39	251	133	18	464	164	1,906	2,017	1,553	1,551.2	3,923	3,127.6	1.2
Hepatitis C (newly acquired)	3	6	2	NN	5	0	47	0	63	67	90	94.0	130	188.2	0.7
Hepatitis C (unspecified)	28	1,427	45	672	106	72	580	281	3,211	3,221	2,652	2,914.0	6,432	5,998.0	1.1
Hepatitis D	0	2	0	4	0	0	5	0	11	10	15	8.0	21	16.6	1.4
Gastrointestinal diseases															
Botulism	0	0	0	0	0	0	0	0	0	1	0	0.2	1	0.8	0.0
Campylobacteriosis§	115	NN	65	1,116	474	137	1,259	632	3,798	4,030	3,420	3,449.0	7,828	7,785.4	1.1
Cryptosporidiosis	26	351	45	325	35	9	285	59	1,135	2,861	482	659.6	3,996	1,695.6	1.7
Haemolytic uraemic syndrome	0	2	0	1	0	0	1	0	4	3	6	3.2	7	8.6	1.3
Hepatitis A	0	24	1	17	39	0	73	12	166	69	87	71.8	235	157.0	2.3
Hepatitis E	0	4	0	1	0	0	3	2	10	16	10	6.8	26	18.2	1.5
Listeriosis	0	7	0	1	0	0	5	6	19	29	14	12.8	48	32.4	1.5
STEC, VTEC‖	0	4	0	6	11	0	5	1	27	53	19	18.2	80	41.0	1.5
Salmonellosis	44	549	148	645	211	42	336	251	2,226	3,390	1,966	2,021.6	5,616	4,996.8	1.1
Shigellosis	2	49	19	31	10	1	17	39	168	217	184	156.0	385	346.8	1.1
Typhoid	1	8	0	2	1	0	6	1	19	37	26	18.2	56	47.4	1.0
Quarantinable diseases															
Cholera	0	1	0	0	0	0	0	0	1	2	0	0.8	3	1.6	1.3
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 April to 30 June 2009, by date of diagnosis,* *continued*

Disease	State or territory								Total 2nd quarter 2009†	Total 1st quarter 2009	Total 2nd quarter 2008	Last 5 years mean 2nd quarter	Year to date 2009	Last 5 years YTD mean	Ratio‡
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶	258	3,895	672	4,375	1,078	397	3,698	2,375	16,748	15,613	15,126	11,973.0	32,361	23,920.2	1.4
Donovanosis	0	0	0	1	0	0	0	0	1	0	1	1.6	1	3.6	0.6
Gonococcal infection	14	382	500	434	140	5	361	394	2,230	2,248	2,163	2,131.0	4,478	4,170.8	1.0
Syphilis (all)	4	371	44	92	24	7	226	48	816	799	809	683.6	1,615	1,356.2	1.2
Syphilis < two years duration	1	110	12	31	24	2	102	27	309	320	332	254.2	629	485.6	1.2
Syphilis >two years or unspecified duration	3	261	32	61	NDP	5	124	21	507	479	477	417.8	986	845.8	1.2
Syphilis - congenital	0	1	0	0	0	0	0	0	1	2	2	4.6	3	7.2	0.2
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	2	0	3	1	0	0	2	8	8	11	4.2	16	8.2	1.9
Influenza (laboratory confirmed)	337	896	541	2,631	1,402	223	3,228	646	9,904	472	711	529.0	10,376	818.8	18.7
Measles	0	2	0	1	2	0	1	3	9	78	26	26.6	87	40.8	0.3
Mumps	0	13	1	11	5	0	9	4	43	56	54	67.2	99	128.2	0.6
Pertussis	59	3,410	64	1,624	1,012	230	833	203	7,435	8,509	2,033	1,965.0	15,944	3,640.0	3.8
Pneumococcal disease (invasive)	6	131	21	70	33	7	94	40	402	205	431	459.0	607	702.2	0.9
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Rubella	0	4	0	2	0	0	0	1	7	8	9	13.0	15	19.4	0.5
Rubella - congenital	0	0	0	0	0	0	0	0	0	0	0	0.4	0	0.6	0.0
Tetanus	0	0	0	0	0	0	0	0	0	3	1	0.8	3	2.0	0.0
Varicella zoster (chickenpox)**	0	NN	26	52	108	9	65	82	342	317	284	249.3	659	518.0	1.4
Varicella zoster (shingles)**	1	NN	29	122	221	29	268	157	827	659	502	209.2	1,486	450.0	4.0
Varicella zoster (unspecified)**	16	NN	1	904	118	17	265	214	1,535	1,747	968	554.2	3,282	1,136.4	2.8
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	4	0	0	1	0	5	14	3	6.6	19	22.8	0.8
Barmah Forest virus infection	1	108	33	174	8	0	0	35	359	569	481	493.6	928	1,032.4	0.7
Dengue virus infection	2	26	7	97	5	1	6	48	192	1,013	94	67.8	1,205	195.6	2.8
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.2	0.0
Kunjin virus infection	0	0	0	0	0	0	0	0	0	2	0	0.4	2	1.6	0.0
Malaria	0	29	5	56	11	0	33	17	151	135	127	159.8	286	351.0	0.9
Murray Valley encephalitis virus infection	0	0	0	1	0	0	0	1	2	2	1	0.4	4	1.2	5.0
Ross River virus infection	1	465	104	654	54	13	24	205	1,520	1,642	1,158	1,103.2	3,162	3,241.4	1.4

Table 2: Notifications of diseases received by state and territory health authorities in the period 1 April to 30 June 2009, by date of diagnosis,* *continued*

Disease	State or territory								Total 2nd quarter 2009†	Total 1st quarter 2009	Total 2nd quarter 2008	Last 5 years mean 2nd quarter	Year to date 2009	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	1	0	7	1	0	0	0	9	9	13	7.4	18	18.4	1.2
Leptospirosis	1	8	0	36	0	0	3	0	48	66	31	41.4	114	91.4	1.2
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.0	0.0
Ornithosis	0	8	0	0	1	0	4	1	14	16	34	41.6	30	82.2	0.3
Q fever	0	37	0	37	0	0	6	1	81	98	70	99.8	179	203.6	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	0	34	2	25	12	0	18	11	102	64	76	81.2	166	163.2	1.3
Leprosy	0	0	0	1	0	0	0	0	1	1	3	2.4	2	5.6	0.4
Meningococcal infection††	2	28	0	9	10	1	6	7	63	47	64	76.0	110	141.8	0.8
Tuberculosis	4	99	2	76	1	3	66	22	273	309	282	259.8	582	525.8	1.1
Total	944	13,213	2,417	14,760	5,276	1,235	12,326	6,075	56,246	51,063	36,391	32,509.7	107,309	67,851.4	1.7

* 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 are based on 5 years of 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; the Northern Territory and Queensland, which exclude ocular specimens; and Western Australia, which excludes ocular and perinatal infections.

** Ratio = ratio of current quarter total to the mean of last 5 years for the same quarter. Note: Ratios for varicella zoster (chickenpox), varicella zoster (shingles) and varicella zoster (unspecified) are based on 5 years of data

†† 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 April to 30 June 2009, by state or territory. (Annualised rate per 100,000 population)

Disease*	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)	2.3	0.5	1.8	1.3	1.0	1.6	1.9	0.0	1.0
Hepatitis B (unspecified)	19.8	47.1	70.9	23.5	33.2	14.5	35.0	30.3	35.7
Hepatitis C (newly acquired)	3.5	0.3	3.6	NN	1.2	0.0	3.5	0.0	1.2
Hepatitis C (unspecified)	32.5	81.9	81.8	62.8	26.5	57.8	43.8	52.0	60.1
Hepatitis D	0.0	0.1	0.0	0.4	0.0	0.0	0.4	0.0	0.2
Gastrointestinal diseases									
Botulism	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis†	133.6	NN	118.2	104.3	118.4	110.0	95.1	116.9	71.1
Cryptosporidiosis	30.2	20.2	81.8	30.4	8.7	7.2	21.5	10.9	21.2
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.1	0.0	0.0	0.1	0.0	0.1
Hepatitis A	0.0	1.4	1.8	1.6	9.7	0.0	5.5	2.2	3.1
Hepatitis E	0.0	0.2	0.0	0.1	0.0	0.0	0.2	0.4	0.2
Listeriosis	0.0	0.4	0.0	0.1	0.0	0.0	0.4	1.1	0.4
STEC, VTEC‡	0.0	0.2	0.0	0.6	2.7	0.0	0.4	0.2	0.5
Salmonellosis	51.1	31.5	269.2	60.3	52.7	33.7	25.4	46.4	41.7
Shigellosis	2.3	2.8	34.6	2.9	2.5	0.8	1.3	7.2	3.1
Typhoid	1.2	0.5	0.0	0.2	0.2	0.0	0.5	0.2	0.4
Quarantinable diseases									
Cholera	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
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
Sexually transmissible infections									
Chlamydial infection§	299.8	223.6	1,222.1	408.9	269.2	318.8	279.2	439.2	313.5
Donovanosis	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Gonococcal infection	16.3	21.9	909.3	40.6	35.0	4.0	27.3	72.9	41.7
Syphilis (all)	4.6	21.6	80.0	9.2	6.2	5.6	17.1	8.9	15.5
Syphilis <2 years duration	1.2	6.3	21.8	2.9	6.0	1.6	7.7	5.0	5.8
Syphilis >2 years or unspecified duration	3.5	15.0	58.2	5.7	NDP	4.0	9.4	3.9	9.5
Syphilis - congenital	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Haemophilus influenzae type b	0.0	0.1	0.0	0.3	0.2	0.0	0.0	0.4	0.1
Influenza (laboratory confirmed)	391.6	51.4	983.9	245.9	350.1	179.1	243.7	119.5	185.4
Measles	0.0	0.1	0.0	0.1	0.5	0.0	0.1	0.6	0.2
Mumps	0.0	0.7	1.8	1.0	1.2	0.0	0.7	0.7	0.8
Pertussis	68.6	195.8	116.4	151.8	252.7	184.7	62.9	37.5	139.2
Pneumococcal disease (invasive)	7.0	7.5	38.2	6.5	8.2	5.6	7.1	7.4	7.5

Table 3: Notification rates of diseases, 1 April to 30 June 2009, 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	0.2	0.0	0.2	0.0	0.0	0.0	0.2	0.1
Rubella - congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Varicella zoster (chickenpox)	0.0	NN	47.3	4.9	27.0	7.2	4.9	15.2	6.4
Varicella zoster (shingles)	1.2	NN	52.7	11.4	55.2	23.3	20.2	29.0	15.5
Varicella zoster (unspecified)	18.6	NN	1.8	84.5	29.5	13.7	20.0	39.6	28.7
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.4	0.0	0.0	0.1	0.0	0.1
Barmah Forest virus infection	1.2	6.2	60.0	16.3	2.0	0.0	0.0	6.5	6.7
Dengue virus infection	2.3	1.5	12.7	9.1	1.2	0.8	0.5	8.9	3.6
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	0.0	1.7	9.1	5.2	2.7	0.0	2.5	3.1	2.8
Murray Valley encephalitis virus infection	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.2	0.0
Ross River virus infection	1.2	26.7	189.1	61.1	13.5	10.4	1.8	37.9	28.4
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	0.1	0.0	0.7	0.2	0.0	0.0	0.0	0.2
Leptospirosis	1.2	0.5	0.0	3.4	0.0	0.0	0.2	0.0	0.9
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.5	0.0	0.0	0.2	0.0	0.3	0.2	0.3
Q fever	0.0	2.1	0.0	3.5	0.0	0.0	0.5	0.2	1.5
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial infections									
Legionellosis	0.0	2.0	3.6	2.3	3.0	0.0	1.4	2.0	1.9
Leprosy	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Meningococcal infection‖	2.3	1.6	0.0	0.8	2.5	0.8	0.5	1.3	1.2
Tuberculosis	4.6	5.7	3.6	7.1	0.2	2.4	5.0	4.1	5.1

* 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; the 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 12,613 reports received by the Virology and Serology Laboratory Reporting Scheme (LabVISE) in the reporting period, 1 April to 30 June 2009 (Tables 4 and 5).

Table 4: Virology and serology laboratory reports by state or territory* for the reporting period 1 April to 30 June 2009, and total reports for the year†

	State or territory								This period 2009	This period 2008	Year to date 2009	Year to date 2008
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Measles, mumps, rubella												
Measles virus	–	1	–	–	2	–	2	–	5	11	44	24
Mumps virus	–	1	1	2	5	–	8	–	17	15	30	34
Rubella virus	–	2	–	2	1	–	–	–	5	3	9	9
Hepatitis viruses												
Hepatitis A virus	–	–	–	6	14	–	–	–	20	12	28	35
Hepatitis D virus	–	–	–	–	7	–	–	–	7	9	14	18
Arboviruses												
Ross River virus	–	35	8	180	59	2	3	7	294	261	694	1,057
Barmah Forest virus	–	3	–	29	11	–	–	–	43	127	149	377
Flavivirus (unspecified)	–	4	–	17	–	–	1	–	22	12	171	42
Adenoviruses												
Adenovirus not typed/ pending	–	73	–	113	256	–	14	–	456	366	862	715
Herpesviruses												
Herpes virus type 6	–	–	–	–	–	–	1	–	1	1	1	1
Cytomegalovirus	–	42	–	90	137	1	6	–	276	254	635	620
Varicella–zoster virus	2	56	–	327	199	4	8	–	631	562	1,383	1,322
Epstein–Barr virus	–	7	14	219	266	4	3	34	547	564	1,159	1,219
Other DNA viruses												
Parvovirus	–	1	–	25	18	2	9	–	55	43	121	123
Picornavirus family												
Echovirus type 4	–	1	–	–	–	–	–	–	1		2	1
Echovirus type 30	–	1	–	–	–	–	–	–	1		8	
Rhinovirus (all types)	–	39	–	1	1	–	–	–	41	53	65	91
Enterovirus not typed/ pending	–	9	–	7	2	–	1	–	19	23	52	134
Picornavirus not typed	–	–	–	–	–	1	–	–	1	6	5	7
Ortho/paramyxoviruses												
Influenza A virus	28	1,792	–	305	898	1	110	–	3,134	89	3,220	138
Influenza A virus H1N1	–	1	–	–	–	5	–	–	6		6	
Influenza A virus H3N2	–	1	–	–	–	–	–	–	1		1	
Influenza B virus	3	31	–	42	30	–	–	1	107	70	142	92
Influenza virus – typing pending	–	4	–	–	–	–	1	–	5	–	5	
Parainfluenza virus type 1	–	4	–	3	–	–	1	–	8	86	11	149
Parainfluenza virus type 2	–	22	–	26	2	–	5	–	55	10	69	20
Parainfluenza virus type 3	–	19	–	20	73	–	7	–	119	17	163	24
Parainfluenza virus typing pending	–	1	–	–	–	–	–	–	1	–	1	
Respiratory syncytial virus	–	710	–	341	546	2	92	2	1,693	739	1,944	944

Table 4: Virology and serology laboratory reports by state or territory* for the reporting period 1 April to 30 June 2009, and total reports for the year,† *continued*

	State or territory								This period 2009	This period 2008	Year to date 2009	Year to date 2008
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA				
Other RNA viruses												
HTLV–1	–	–	–	–	59	–	–	–	59	14	142	18
Rotavirus	–	14	–	–	69	4	1	–	88	88	151	197
Norwalk agent	1	15	–	–	–	–	–	–	16	11	32	28
Other												
Chlamydia trachomatis not typed	6	314	–	1,155	679	18	10	1	2,183	2,039	4,559	4,396
Chlamydia pneumoniae	–	1	–	1	–	–	2	–	4	1	6	1
Chlamydia psittaci	–	1	–	–	–	–	15	–	16	33	39	55
Chlamydia spp typing pending	–	4	–	–	–	–	–	–	4		6	
Chlamydia species	–	–	–	–	–	–	2	–	2		7	2
Mycoplasma pneumoniae	–	10	1	132	68	7	93	9	320	217	573	424
Mycoplasma hominis	–	2	–	–	–	–	–	–	2	2	4	4
Coxiella burnetii (Q fever)	–	1	–	10	15	–	3	–	58	73	115	153
Rickettsia prowazeki	–	–	–	–	1	–	6	–	7		8	
Rickettsia – spotted fever group	–	2	–	21	1	–	3	–	37	63	75	91
Streptococcus group A	–	7	–	129	–	–	1	–	137	178	315	450
Brucella species	–	2	–	3	–	–	–	–	5	9	9	17
Bordetella pertussis	1	397	–	358	810	3	2	–	1,571	246	2,933	444
Legionella pneumophila	–	5	–	2	3	–	4	–	14	4	20	11
Legionella longbeachae	–	2	–	1	2	–	2	–	7	1	10	5
Legionella species	–	4	–	4	–	–	–	–	8		13	1
Cryptococcus species	–	5	–	3	4	–	–	–	12	8	21	15
Leptospira species	–	–	–	5	1	–	1	–	7	24	25	54
Treponema pallidum	–	62	–	193	213	1	5	–	474	543	962	1,108
Entamoeba histolytica	–	–	–	–	–	–	1	–	1	1	1	4
Toxoplasma gondii	–	–	–	2	1	–	1	–	4	5	11	6
Echinococcus granulosus	–	–	–	–	5	–	1	–	6	15	14	22
Total	41	3,708	24	3,774	4,458	55	425	54	12,613	6,908	21,045	14,702

* 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 April to 30 June 2009*

State or territory	Laboratory	April 2009	May 2009	June 2009	Total this period
Australian Capital Territory	The Canberra Hospital	–	–	–	–
New South Wales	Institute of Clinical Pathology and Medical Research, Westmead	300	404	1,593	2,297
	New Children's Hospital, Westmead	127	150	154	431
	Repatriation General Hospital, Concord	–	–	–	–
	Royal Prince Alfred Hospital, Camperdown	42	60	46	148
	South West Area Pathology Service, Liverpool	104	125	315	544
Queensland	Queensland Medical Laboratory, West End	124	1,797	2,204	4,125
	Townsville General Hospital	–	–	–	–
South Australia	Institute of Medical and Veterinary Science, Adelaide	925	1,389	2,140	4,454
Tasmania	Northern Tasmanian Pathology Service, Launceston	12	17	20	49
	Royal Hobart Hospital, Hobart	–	–	–	–
Victoria	Australian Rickettsial Reference Laboratory	25	23	26	74
	Monash Medical Centre, Melbourne	15	79	125	219
	Royal Children's Hospital, Melbourne	–	–	–	–
	Victorian Infectious Diseases Reference Laboratory, Fairfield	59	58	83	200
Western Australia	PathWest Virology, Perth	–	–	–	–
	Princess Margaret Hospital, Perth	–	–	–	–
	Western Diagnostic Pathology	–	72	–	72
Total		1,733	4,174	6,706	12,613

* The complete list of laboratories reporting for the 12 months, January to December 2009, 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 data collection was established in 2006 and currently, further development of ASPREN is in progress to create an automatic reporting system.

The list of conditions is reviewed annually by the ASPREN management committee and an annual report is published. In 2009, four 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 *2008;32:135.*

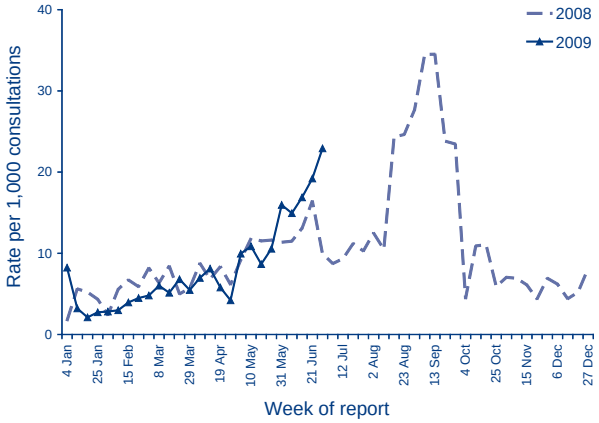
Data on influenza-like illness, gastroenteritis, chickenpox and shingles from 1 April to 30 June 2009 compared with 2008, are shown as the rate per 1,000 consultations in Figures 1, 2, 3 AND 4, respectively.

Reporting period 1 April to 30 June 2009

Sentinel practices contributing to ASPREN were located in all jurisdictions other than the Northern Territory. A total of 102 general practitioners contributed data to ASPREN in the 2nd quarter of 2009. Each week an average of 81 general practitioners provided information to ASPREN at an average of 8,089 (range 6,740–8,902) consultations per week.

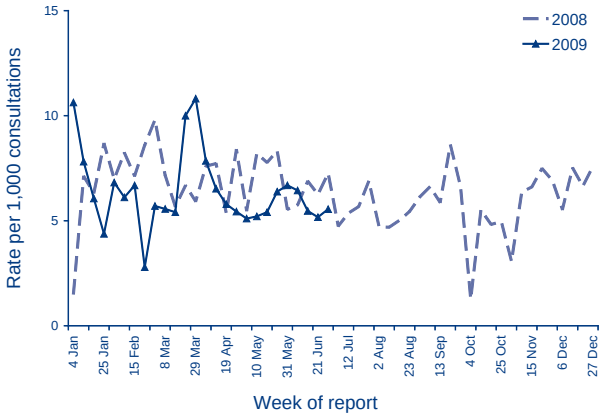
ILI rates reported from 1 April to 30 June 2009 were 4–23 cases per 1,000 consultations. The reported rates in April and May 2009 were similar compared with the same reporting period in 2008 (Figure 1). ILI rates reported in June 2009 (15–23 cases per 1,000 consultations) were higher than rates recorded in June 2008 (10–16 cases per 1,000 consultations).

Figure 1: Consultation rates for influenza-like illness, ASPREN, 1 January 2008 to 30 June 2009, by week of report



During this reporting period, consultation rates for gastroenteritis ranged from 5 to 8 cases per 1,000 consultations (Figure 2).

Figure 2: Consultation rates for gastroenteritis, ASPREN, 1 January 2008 to 30 June 2009, by week of report



Varicella infections were reported at a similar rate for the 2nd quarter of 2009 compared with the same period in 2008. From 1 April to 30 June 2009, recorded rates for chickenpox were between 0 and 1 case per 1,000 consultations (Figure 3).

In the second quarter of 2009, reported rates for shingles were between less than 1 to 1.4 cases per 1,000 consultations (Figure 4).

Figure 3: Consultation rates for chickenpox, ASPREN, 1 January 2008 to 30 June 2009, by week of report

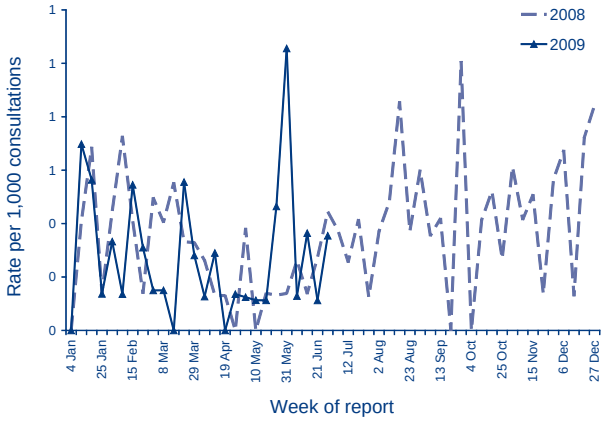
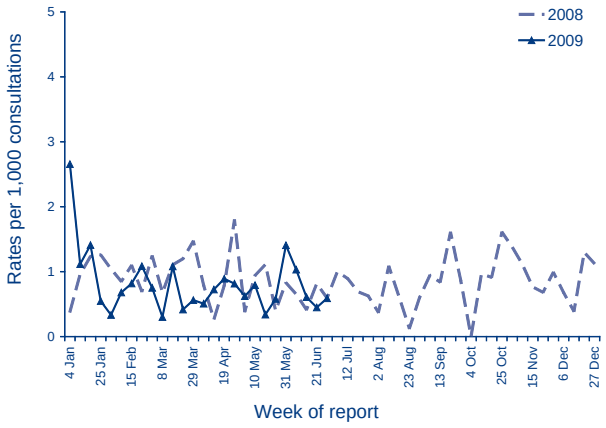


Figure 4: Consultation rates for shingles, ASPREN, 1 January 2008 to 30 June 2009, by week of report



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 January and 31 March 2008, at 24 months of age for the cohort born between 1 January and 31 March 2007, and at 5 years of age for the cohort born between 1 January and 31 March 2003 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 one dose of a measles, mumps and rubella-containing (MMR) 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 decreased slightly by 0.3 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 slightly by 0.3 of a percentage point to 92.9% (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 children 'fully immunised' at 5 years of age for Australia is currently at 82.4% (Table 3). In South Australia and Tasmania it is below 80%, at 75.6% and 78.6% respectively. Due to a calculation error by ACIR, the coverage estimates for the past 5 quarters for the 5-year age group have been incorrect. Assessment was made at 66 months rather than 60 months, which inflated the estimates. The age of assessment for vaccines due at 4 years of age makes a critical difference to coverage estimates for these vaccines. Comparing the current correct figures (assessment at 60 months) with the figures assessed in December 2007, the last time assessment was made at 72 months, a decrease of 6.4 percentage points in coverage for Australia was observed with an even greater decrease of 12 percentage points observed for South Australia.

Table 1: Percentage of children immunised at 1 year of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2008; assessment date 30 June 2009

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,164	24,006	966	15,531	4,864	1,610	17,597	7,873	73,611
Diphtheria, tetanus, pertussis (%)	94.0	92.2	90.6	91.3	91.8	90.6	92.3	89.5	91.7
Poliomyelitis (%)	94.0	92.1	90.5	91.3	91.7	90.6	92.3	89.4	91.7
Haemophilus influenzae type b (%)	95.5	94.7	94.8	94.2	94.8	93.0	94.9	93.3	94.5
Hepatitis B (%)	95.6	94.7	94.7	94.0	94.7	93.0	94.7	93.3	94.4
Fully immunised (%)	93.6	91.9	90.3	91.0	91.5	90.3	91.9	88.9	91.3
Change in fully immunised since last quarter (%)	-1.2	-0.3	+0.3	-0.2	0.0	-1.9	-0.5	-0.1	-0.3

Table 2: Percentage of children immunised at 2 years of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2007; assessment date 30 June 2009*

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,145	24,259	967	15,517	4,942	1,601	17,709	7,652	73,792
Diphtheria, tetanus, pertussis (%)	95.6	95.0	96.5	94.6	95.1	94.8	95.7	94.3	95.0
Poliomyelitis (%)	95.6	95.0	96.5	94.5	95.1	94.9	95.7	94.3	95.0
Haemophilus influenzae type b (%)	95.6	95.3	95.1	93.7	94.3	94.9	94.8	94.0	94.6
Measles, mumps, rubella (%)	94.5	93.8	96.2	93.4	94.5	93.8	94.9	93.3	94.0
Hepatitis B (%)	96.2	95.9	97.3	95.5	95.7	95.3	96.3	95.1	95.8
Fully immunised (%)	93.6	92.7	94.6	92.2	93.2	93.0	93.8	91.8	92.9
Change in fully immunised since last quarter (%)	-0.3	+0.4	+0.9	0.0	+1.3	-0.4	+0.2	+0.9	+0.3

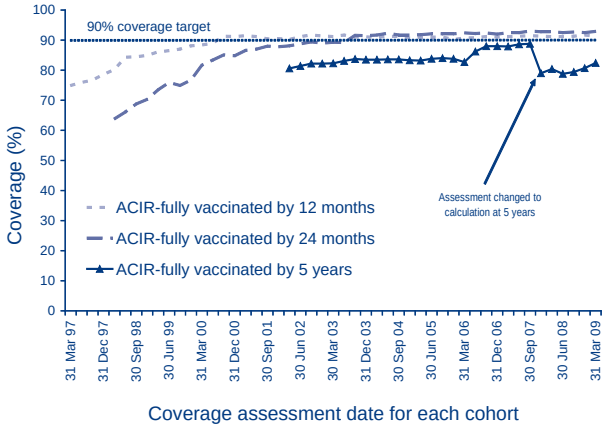
* The 12 months age data for this cohort were published in *Commun Dis Intell* 2008;32(3):358.

Table 3: Percentage of children immunised at 5 years of age, preliminary results by disease and state or territory for the birth cohort 1 January to 31 March 2004; assessment date 30 June 2009

Vaccine	State or territory								Aust
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
Total number of children	1,099	22,095	893	14,307	4,626	1,396	16,307	7,099	67,822
Diphtheria, tetanus, pertussis (%)	85.2	82.8	85.3	83.3	76.4	79.7	86.4	81.4	83.2
Poliomyelitis (%)	84.9	82.7	85.4	83.1	76.4	79.6	86.3	81.2	83.1
Measles, mumps, rubella (%)	85.1	82.5	85.1	83.2	76.2	79.2	86.1	81.1	82.9
Fully immunised (%)	84.4	82.0	84.8	82.5	75.6	78.6	85.8	80.3	82.4
Change in fully immunised since last quarter (%)	0.0	+3.6	+2.9	+1.7	+0.2	-3.6	+0.2	+1.7	+1.7

Figure 5 shows the trends in vaccination coverage from the 1st 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 6 years (5 years from March 2008), although coverage for vaccines due at 4 years decreased significantly due to the above-mentioned change in assessment age. It should also be noted that, currently, coverage for the vaccines added to the NIP since 2003 (varicella at 18 months, meningococcal C conjugate at 12 months 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 31 March 2009, 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 January to 31 March 2009

The AGSP laboratories received a total of 875 isolates in this quarter of which 856 underwent susceptibility testing. This number is 76 more than the 799 isolates reported in this period in 2008. About 27% of this total was from New South Wales, 25% from Victoria, 16% from Queensland, 12% each from Western Australia and the Northern Territory and 7% from South Australia. A small number of isolates were also received from Tasmania and the Australian Capital Territory.

Penicillins

In this quarter, 336 (39%) of all isolates examined were penicillin resistant by one or more mechanisms. One hundred and eleven (13%) were penicillinase producing (PPNG) and 223 (26%) penicillin resistant by chromosomal mechanisms, (CMRP). The proportion of all strains resistant to the penicillins by any mechanism ranged from 2% in the Northern Territory to 56% in New South Wales. In this quarter in 2008, 45% of isolates were penicillin resistant by any mechanism and 39% in 2007. The decrease in penicillin resistant strains to 2007 proportions was the result of decreased numbers of gonococci with chromosomally mediated resistance.

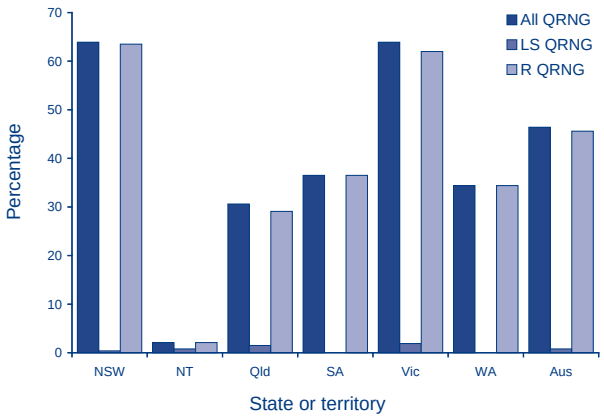
Figure 6 shows the proportions of gonococci fully sensitive (MIC $\leq$ 0.03 mg/L), less sensitive (MIC 0.06–0.5 mg/L), relatively resistant (MIC $\geq$ 1 mg/L) or else PPNG, aggregated for Australia and by state and territory. A high proportion of those strains classified as PPNG or else resistant by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

The highest number of PPNG and CMRP were found in New South Wales where there were 97 CMRP (41%) and 36 PPNG (15%). Victoria had 77 (36%) CMRP and 29 (13%) PPNG. Queensland had higher numbers of PPNG: 23 (17%), but fewer CMRP: 11 (8%). Western Australia also had higher numbers of PPNG: 18 (19%) than CMRP: 13 (14%). One CMRP and 1 PPNG strain were found in the Northern Territory. Two CMRP and 1 PPNG in the Australian Capital Territory and 2 CMRP and no PPNG reported from Tasmania. Of note was the decrease in penicillin resistant strains

Figure 6: Categorisation of gonococci isolated in Australia, 1 January to 31 March 2009, 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.*

Figure 7: The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 January to 31 March 2009, by jurisdiction



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.
R QRNG Ciprofloxacin MICs ≥1 mg/L.

in South Australia in this quarter to 36.5% comprising 20 CMRP (31.75%) and 3 PPNG (4.75%). Corresponding proportions in 2008 were 5% PPNG and 70.7% CMRP.

Ceftriaxone

Ten isolates with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected nationally, five in New South Wales, three in Queensland and two in South Australia. Eight were seen nationally in the 1st quarter of 2008.

Spectinomycin

All isolates were susceptible to this injectable agent. This antibiotic is no longer available in Australia.

Quinolone antibiotics

The total number (397) and proportion (46%) of quinolone resistant *N. gonorrhoeae* (QRNG) was lower than data reported in recent quarters that reported high levels of resistance to this group of antibiotics. In the equivalent period in 2008, there were 415 (53%) QRNG. All but seven of the 397 QRNG detected in this quarter had ciprofloxacin MICs of 1 mg/L or more and 340 had ciprofloxacin MICs of 4 mg/L or more. QRNG are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L (Figure 7). QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups.

QRNG were present in all jurisdictions. The highest number of QRNG was found in New South Wales (152) and this represented 64% of all isolates. One hundred and thirty-eight QRNG in Victoria also represented a high (64%) proportion of all isolates there. In Queensland there were 41 (31%), and in Western Australia 33 (34%) QRNG. The 23 (37%) QRNG in South Australia was a marked decrease in number compared with the 83 (84%) QRNG in the same quarter in 2008, and parallels the decrease in penicillin resistance also noted in that jurisdiction in this quarter. Six QRNG were detected in the Australian Capital Territory and two in Tasmania. A single QRNG was detected in the Northern Territory.

High level tetracycline resistance

Nationally, the number (157) and proportion (18%) of high level tetracycline resistance (TRNG) detected increased when compared with the 2008 data (135 TRNG, 17%). TRNG were found in all states and territories except Tasmania, and elsewhere represented between 2% (South Australia) and 33% of isolates (Western Australia).

Reference

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

Meningococcal surveillance

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The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of laboratory confirmed cases confirmed either by culture or by non-culture based 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 contained 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 is contained in the annual reports of the Programme, published in Communicable Diseases Intelligence. For more information see* Commun Dis Intell *2009;33:82.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 April to 30 June 2009, are included in this issue of Communicable Diseases Intelligence *(Table 4).*

Table 4: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2009, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD
Australian Capital Territory	09			3	3									3	3
	08			2	2									2	2
New South Wales	09			13	25	1	4	1	1	1	2	3	3	19	35
	08			9	13	2	3	1	2	1	1			13	19
Northern Territory	09			1	3	0	1							1	4
	08			0	0	1	2							1	2
Queensland	09			6	17	0	0							6	17
	08			25	41	0	2			1	1			26	44
South Australia	09			7	11			1	1					8	12
	08			5	7									5	7
Tasmania	09			1	1									1	1
	08			0	0									0	0
Victoria	09			5	10	0	1					0	2	5	13
	08			18	22			1	1			1	1	20	24
Western Australia	09			8	10	0	2							8	12
	08			5	8								1	5	9
Total	09			44	80	1	8	2	2	1	2	3	5	51	97
	08			64	93	3	7	2	3	2	2	1	2	72	107

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HIV and AIDS surveillance

National surveillance for HIV disease is coordinated by the National Centre in HIV Epidemiology and Clinical Research (NCHECR), in collaboration with State and Territory health authorities and the Commonwealth of Australia. Cases of HIV infection are notified to the National HIV Database on the first occasion of diagnosis in Australia, by either the diagnosing laboratory (Australian Capital Territory, New South Wales, Tasmania, Victoria) or by a combination of laboratory and doctor sources (Northern Territory, Queensland, South Australia, Western Australia). 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.

Tabulations of diagnoses of HIV infection and AIDS are based on data available 3 months after the end of

the reporting interval indicated, to allow for reporting delay and to incorporate newly available information. More detailed information on diagnoses of HIV infection and AIDS is published in the quarterly Australian HIV Surveillance Report, and annually in 'HIV/AIDS, viral hepatitis and sexually transmissible infections in Australia, annual surveillance report'. The reports are available from the National Centre in HIV Epidemiology and Clinical Research, 376 Victoria Street, Darlinghurst NSW 2010. Internet: http://www.med.unsw.edu.au/nchecr. Telephone: +61 2 9332 4648. Facsimile: +61 2 9332 1837. For more information see Commun Dis Intell 2009;33:83.

HIV and AIDS diagnoses and deaths following AIDS reported for 1 October to 31 December 2008, as reported to 31 March 2009, are included in this issue of Communicable Diseases Intelligence *(Tables 5 and 6).*

Table 5: New diagnoses of HIV infection, new diagnoses of AIDS and deaths following AIDS occurring in the period 1 October to 31 December 2008, by sex and state or territory of diagnosis

	Sex	State or territory								Totals for Australia			
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	This period 2008	This period 2007	YTD 2008	YTD 2007
HIV diagnoses	Female	1	9	0	7	0	0	8	3	28	36	136	135
	Male	0	82	3	38	13	0	55	15	206	218	859	909
	Not reported	0	0	0	0	0	0	0	0	0	0	0	0
	Total*	1	91	3	45	13	0	63	18	234	254	995	1,045
AIDS diagnoses	Female	0	0	0	0	0	0	1	2	3	6	8	16
	Male	0	0	1	4	2	0	11	4	22	36	91	144
	Total*,†	0	0	1	4	2	0	12	6	25	42	99	161
AIDS deaths	Female	0	0	0	0	0	0	0	0	0	1	0	8
	Male	0	0	1	0	0	0	7	0	8	12	24	45
	Total*,†	0	0	1	0	0	0	7	0	8	13	24	53

* Totals include people whose sex was reported as transgender.

† AIDS diagnoses and death following AIDS in New South Wales in 2008 not included.

Table 6: Cumulative diagnoses of HIV infection, AIDS, and deaths following AIDS since the introduction of HIV antibody testing to 1 October to 31 December 2008, and reported by 31 March 2009, by sex and state or territory

	Sex	State or territory								Australia
		ACT	NSW	NT	Qld	SA	Tas	Vic	WA	
HIV diagnoses	Female	35	965	25	329	116	13	435	240	2,158
	Male	273	14,148	146	3,069	1,021	115	5,749	1,332	25,853
	Not reported	0	228	0	0	0	0	22	0	250
	Total*	308	15,371	171	3,407	1,138	128	6,228	1,579	28,330
AIDS diagnoses	Female	10	265	4	73	32	4	121	45	554
	Male	94	5,513	47	1,080	418	55	2,103	448	9,758
	Total*,†	104	5,796	51	1,155	451	59	2,237	495	10,348
AIDS deaths	Female	7	138	1	43	20	2	64	29	304
	Male	73	3,598	32	679	280	34	1,443	299	6,438
	Total*,†	80	3,747	33	724	300	36	1,516	329	6,765

* Totals include people whose sex was reported as transgender.

† AIDS diagnoses and death following AIDS in New South Wales in 2008 not included.